LECTURES ON DISEASES OF CHILDREN

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ON
DISEASES OF CHILDREN

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PREFACE TO THE 9th EDITION

Originally this book consisted of lectures delivered by Sir Robert Hutchison in a systematic course at the London Hospital and published serially in the *Clinical Journal*. The approach has always been essentially practical and clinical with detailed reference to treatment. Since its first appearance in 1904, this book has won a growing popularity and passed through eight editions. Responsibility for the present edition, as for the eighth, is entirely the junior author's, and he well appreciates the difficulty of the task he has undertaken in carrying out a thorough revision. He has tried to leave as much as possible of the original work whilst at the same time bringing it up to date as regards advances, especially in diagnosis and treatment, of the last few years.

The whole book has been revised, some out-of-date matter omitted and some new sections introduced. The principal alterations will be found in relation to infections in the new-born, the treatment of infantile diarrhœa, the cause and treatment of cœliac disease, the treatment of thread worms, the types of congenital heart disease and the fascinating story of the Rhesus factor. Chapters XXV and XXVI of the last edition have been reversed in their order, with a new introduction to Chapter XXVI in the present book. A section on encephalitis has been added to Chapter XXIX, which previously dealt only with meningitis.

It has not been possible, owing to war-time conditions, to secure many new illustrations. Fig. 11 on cœliac disease, however, illustrating a child before and after treatment, was kindly made for me by Mr. R. N. G. Atherstone, and the new diagrams to illustrate congenital heart disease were drawn from another purpose some years ago by Mr. Charles Keogh, F.R.C.S. Of the illustrations carried over from the previous edition, I must point out again that some of these are from the collection at the Hospital for Sick Children, Great Ormond Street, and as far as possible permission of my colleagues has been sought and obtained for photographs of patients not under my own care. I am particularly indebted to

Mr. Charles Donald, F.R.C.S., for Fig. 79; to Mr. P. B. Ashcroft, F.R.C.S., for Fig. 82; and to Mr. E. W. Riches, F.R.C.S., for Fig. 98. For X-ray photographs of patients at the Middlesex Hospital, I am grateful to Dr. H. Graham Hodgson; and at the Hospital for Sick Children, to the late Dr. Bertram Shires.

Lastly, I must express my humble gratitude to Sir Robert Hutchison for having entrusted this book to my care. I eagerly read it from cover to cover when I was a medical student. May nothing I have deleted or added spoil a similar pleasure for the present generation whom it is my privilege to instruct.

A. M.

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LECTURES ON DISEASES OF CHILDREN

LECTURE I

THE CLINICAL EXAMINATION OF SICK CHILDREN

It will be generally admitted that the study of diseases of children, which will occupy us in these lectures, is one which is apt to be neglected in general hospitals. One reason for this probably is that adult patients are so numerous, and their claims on the staff so pressing, that there is very little time left for special teaching in the children's wards. And also, I think, it has its explanation in this further fact, that the teaching of general hospitals is, and necessarily must be, directed specially to the requirements of qualifying examinations. Now it is—in my judgment, at least—unfortunate, though I can hardly expect to carry your sympathies in this matter, that there is no demand made at the ordinary qualifying examinations for a special knowledge of the diseases of children, although instruction in diseases of children has now been made a compulsory part of the medical curriculum. The consequence is that many men when they qualify know almost nothing of many of the commonest ailments of child life, although in any large general practice children make up a great part, perhaps the greater part, of the patients, and skill in treating them will be an important factor in your professional success. This is the more to be regretted when one remembers that the care of infant and child life is attaining to an ever greater degree of importance in the public health work of this country. At no time, indeed, has the 'cult of the child' been more evident.

It will be the duty of many of you in your professional lives to conduct infant welfare centres, to carry out the medical inspection of schools, and in other ways to participate in this movement. At present much of this work is in the hands of people who are prac-

tically amateurs, and a great deal of it is in consequence very ill done. If you are to prepare yourselves rightly for it you can only do so by acquiring a wide knowledge of the principles of pædiatrics and as extensive a practical acquaintance as possible with all forms of disease in children. Only thus can you avoid the crankiness and the 'viewy' methods which at present so often mar the work of the well-meaning but ill-informed amateur in this field.

I should be sorry, however, if you were to embark upon this course of study merely from such utilitarian motives as those I have put forward, because I can promise you that on scientific grounds alone, alike pathological and therapeutic, there is no subject which will better repay your attention than the study of the diseases of children. You will find that pathological processes in children tend, if I may use the term, to be pure bred; they are not hybrids produced by a blending of different morbid factors. Disease in children also runs a less complicated course than it does in the adult, and tends to exhibit pathological processes in their simplest forms.

As regards therapeutics, you will find the treatment of children eminently satisfactory. I am bound to confess that when you study the results of the treatment of disease in adults in large hospitals, you are tempted to form a rather low opinion of the potentialities of medical therapeutics. Frequent failure is inevitable; it depends largely on the circumstances in which our patients are placed. But with children it is not so; they respond to treatment in a most surprising and gratifying fashion. One reason is, no doubt, that you can so easily modify your patient's surroundings. A child has not got to work; you can take him away from school for an indefinite number of weeks, and no one will object except the schoolmaster or the attendance officer. Moreover, in the case of children you are fighting with Nature instead of against her. In the case of most of our adult patients they are going down the hill, or are about to go down it; but children are going up the hill, and they have great reparative power to fall back upon, and hence it is that the treatment of their diseases is so encouraging. All the more reason, therefore, that you should know how to treat these diseases, for you will often find when you go into practice that the life of the child is literally in your hands, and that a mistake at a critical moment may be really a matter of life and death.

I may now turn to the subject of the first lecture of this course—the **methods of examining children** clinically. I must ask you

to bear with me if you find this not very interesting, but it is an unavoidable preliminary, for the methods of examining and handling a sick child do not come to you by intuition. It was pointed out by Dr. Charles West, who was one of the pioneers in this subject, that the man who starts for the first time to study the diseases of children is like a traveller in a foreign country ; he hears a strange language spoken which he does not understand. At all events, if the language is not absolutely strange, it is spoken with a foreign accent, for the physical signs of disease are often different in children from what they are in grown-up people, and so you have to devote special attention to their interpretation.

Begin with the **history.** The child, of course, will be unable to tell you anything, or but very little, and you have to fall back upon the information supplied by the mother or the nurse for a history of the illness. These are apt to be loquacious persons, but it is always worth while to listen patiently to all they have to say, for there is nobody in such a favourable position to observe small changes in the health of the child as those who are constantly attending to him. Listen attentively, then, to what the mother or the nurse has to say as to the mode of onset of the illness, and the symptoms which the patient exhibits.

With what are called ' subjective symptoms ' in children you are not in any way troubled, because there are none. Pædiatrics is like veterinary work in this, that the patient is unable to give you an account of his sufferings, and you are thrown back entirely on your own observation, which, of course, makes the necessity for careful examination all the greater. With regard to the history, I would say that nutrition and heredity play such a large part in the diseases of children that you must always make a special investigation into these points. You must ask, as regards the family history, how many other children there are, and whether any are dead, as well as the ages at which they died, and the cause of death in each case. You must inquire also about the health of the mother during pregnancy, whether the child was carried to term or not, and as to the nature of the labour by which it was ushered into the world. All these points have a direct bearing on the conclusions which you will draw as to the kind of heredity with which your patient is endowed. You will further inquire with regard to nutrition, noting the exact mode in which the child has been fed, as well as the particular food or foods which are being given now.

Answers to these questions will clear the ground, and will prepare you for proceeding to your **physical examination.** I need hardly emphasize here the importance of gentleness and tact. These things cannot be taught—they come naturally to some people—but everybody can acquire some degree of them by care and with opportunities for practice. Children are difficult to handle : they are easily frightened, and they distrust the presence of a stranger, and these things add to the difficulties which you will encounter. There is a certain amount of ' patter ' to be acquired by listening to a skilled physician making contact with a sick child. The methods which you adopt in your examination of children are the usual methods of inspection, palpation, percussion, and auscultation. These have not the same relative values, however, as in the case of adults. Inspection is immeasurably the most important method in a child. The next in importance is palpation, next auscultation ; percussion we will leave till last.

You have already, of course, gleaned much information by casual observation whilst you have been talking to the mother and getting the history. During that time the child has become accustomed to your presence. You will have noticed whether he looks seriously ill, whether he is interested in his toys or surroundings, and whether he presents any obvious signs of disease. Having done that, you have to proceed to more exact **inspection.** For that purpose it is important to have the child completely stripped, and this should be done if possible before you come into the room ; it is bad to get a baby to associate you in its mind with the taking off of its clothes. The next piece of advice I would give you is never to look a baby in the face. That, of course, does not apply to grown-up persons ; we feel inclined to distrust a man who does not look us in the face, but the baby distrusts the man who does. There is no surer way of making a baby cry, and therefore of making it far more difficult to examine, than by staring it out of countenance.

You should begin your inspection by noting the child's **expression.** Much has been written about the facies of disease, and you will find characteristic lines described as coming out on the child's face when it is ill. I do not think any good purpose would be served if I attempted to describe them to you. That is knowledge which you must pick up as you have opportunity here, where you will be able to see the facies of all sorts of diseases in children, and you will get familiar by and by with the facies of abdominal disease, of acute

respiratory disease, of cerebral disease, and so on. The importance of this subject, however, justifies all that has been written about it, because the face of the child is a mirror in a way in which that of a grown-up person is not. It is a clean sheet upon which disease can write ; it is not furrowed over with the lines of care and anxiety, nor does it show those changes so often to be seen in adults as a result of previous illness or bad living. Moreover, the signs of disease in a child are not masked by the emotions nor suppressed by the will.

Now, having noted these things, you cast your eye down over the child, and you will observe certain **peculiarities of configuration** which are really normal, but which the novice may think are abnormal. One of these is that the child has a relatively large abdomen. You will not be long in practice before babies are brought to you by anxious mothers with the complaint that ' the stomach is getting large.' I do not say that these mothers are not occasionally right, and that the abdomen is not pathologically enlarged sometimes, but it is well to note that a child's abdomen is naturally prominent, the main reason for which is that the liver is relatively large and occupies a considerable space in the abdomen.

Another peculiarity is that the chest is very round, and it is only after some time that it acquires the oval shape which is seen in a well-formed adult. There are certain consequences of this round-shaped chest in children which I shall have to point out to you when we come to study respiratory disease. Another point to note is the relatively large head of the child compared with its trunk. Here, again, anxious mothers come to you complaining that the child's head is getting large, and possibly the neighbours have been trying to make her believe that the baby has got ' water on the brain.' Remember in such an event that a child is about three years of age before the head and chest have the same relative size as in the adult. Of course, this is easily understood when you remember that during those years the child's brain is growing rapidly.

Another point to notice is the character of the respiratory movements—whether they are normal or rapid, irregular, or laboured. You will also notice whether there is any head retraction. You will observe the position of the limbs—whether they are held in an easy and natural posture, or kept rigid, as they are apt to be when the seat of painful disease, such as scurvy. Look also for the signs of rickets, which you will quickly learn to recognize when we come to speak of that disease. You will, at the same time, notice the

presence or absence of rashes, which are so important a diagnostic sign in the diseases of childhood.

Having run your eye rapidly over the child in this way you come to the next method of examination—namely **palpation.** The advice used to be given by an old teacher of mine that you should always 'paw your babies.' That was very sound advice; but I would remind you of the importance of a warm hand in doing so. Though it is now long ago I remember still, as a child, having been examined by a doctor with very cold hands, and I never forgot it or forgave him. I recommend you to begin by shaking hands or, in the case of a baby, by fondling its fingers. This gives an easy introduction to physical contact. Next proceed to palpation of the head. First go over the anterior fontanelle; notice whether it is closed or open, bulging or depressed. The fontanelle tells you as much in a baby as the pulse does in older patients, a depressed fontanelle having the same significance as a feeble pulse. Notice whether there is any softening of the bone, such as one finds in craniotabes, which is a frequent sign of rickets or syphilis. Observe also whether there is any bossing of the head, and whether there are any nodes or lumps upon the bones.

Having studied the head, you can proceed to the trunk, passing your hand over the skin, and noticing whether it is hot or cold, dry or moist, for the latter is information which no thermometer can give you. You will also notice whether there is beading of the ribs, which is an important and early sign of rickets. Next feel for the edge of the liver; you can generally make it out quite well a little below the costal margin. The spleen, of course, you ought not to be able to feel in health. Lastly, you should run your hands over the limbs and notice whether there is any tenderness of the legs. Some cases of obscure disease in children are to be explained by inflammatory affections of the bones, which can only be diagnosed by careful palpation.

We pass next to **auscultation.** One should perform this before percussion in children, reversing the rule which obtains in the case of older patients. Percussion is apt to frighten children, and so would make auscultation difficult if that were left to the last. In auscultating the chest it is best to have the child sitting up; he should not be lying on his face, for he is likely to have unpleasant associations with that position, and also because the pressure on the abdomen, if the child is prone, pushes up the abdominal organs and interferes with the expansion of the lungs.

It is often recommended that you should listen to the chest of a child without a stethoscope, by putting your ear to the back. Certainly by that means you frighten the child less than by any other method, but it is open to objection in patients who are not very clean. Do not have a stethoscope with a metal end-piece ; use vulcanite, or something which is not chilly when put upon the skin. It is an advantage to have a short chest-piece, so that you can get in between the mother and the child, and get round corners without interfering with the child's position. It is also of help to have it reversible with a narrow end to go into the small spaces between the ribs. You may think that the crying of the child is a great bar to auscultation, and you will find it so as a beginner ; but by and by when you get practice you will rather prefer that the child should cry a little, because crying ensures full expansion of the lungs, and is the equivalent in the adult to the taking of a deep breath.

You will be struck at first by the length of time that a child can hold his breath, and you may have to wait for quite a long time for another inspiration. This is not a sign of disease ; it is merely due to the enormous relative vital capacity of the infant chest. There is another peculiarity of respiration met with in children who are the subjects of serious respiratory disease which is likely to confuse you at first, and which is best described as an *inversion of the respiratory rhythm*. You and I, when we breathe, first fill the chest, then empty it, and then stop for a moment ; that is to say, the ordinary rhythm is inspiration, expiration, pause. But you will often notice in children in whom respiration is embarrassed that a short grunting expiration is made, followed by a full inspiration, and that by a pause. In other words, the ordinary rhythm has become inverted. What the advantage of this mode of breathing is it would be hard to say. It has been suggested that the result of it must be to facilitate free oxidation of the blood, for the pause after inspiration means that the lungs are kept full of pure air for a much longer period than by the normal method ; but it must be confessed that this explanation is not very convincing. Such a reversal of the usual mode may be possible to the child by reason of the greater plasticity of his respiratory apparatus, and by the fact that the mechanism of respiration is not so stereotyped as it becomes in the adult. At all events, whatever the explanation may be, you will find the occurrence of this inversion of respiration of considerable diagnostic value in many cases.

There are other points in connection with auscultation of the lungs in children which have to be noticed, and one of these is the harshness of the respiratory sound. This is the so-called puerile breathing, and it is so marked and constant that, if you hear loud breathing on one side of the chest and faint on the other, the loud is probably the normal. Towards the right apex, and between the scapulæ behind, the respiration in the young child is normally not only harsh, but almost bronchial, a point which it is well to remember if one is to avoid errors of diagnosis.

Another noticeable thing is the extreme ease with which sounds are conducted in a child's chest, so that if you hear a faint accompaniment on one side it is possible that it is really being produced on the other. In the chests of children, too, you meet with pathological conditions which you do not often encounter in grown-up persons; for example, collapse of the lung. It has always to be thought of in considering the abnormal sounds in a child's chest, whereas it is one of the last things to consider in the case of an adult.

Passing to the **auscultation of the heart,** I would remind you that the blood-pressure in the child is low. In a young infant the capacity of the heart is to the cross diameter of the arteries as 25 is to 20. At the age of puberty the proportion is as 290 is to 61. This means that there is a relatively large channel for the blood to pass into in the child, and that there is no great obstacle opposed to the heart; hence the low blood-pressure. In consequence of this the first sound is louder than the second in all the areas, for owing to the low blood-pressure the second aortic sound is comparatively feeble.

There is also a difference in the child between the relative loudness of the pulmonary and aortic second sounds at the base. In the adult the aortic sound is louder than the pulmonary. In the infant that is not the case. Below the age of four the pulmonary second sound is decidedly louder than the aortic. From twenty to forty, careful observation shows that the sounds are about equal. Above forty the aortic second sound becomes progressively louder than the pulmonary. This means that if you are listening to the heart in a young child you are to regard as accentuation of the aortic second sound anything which is equal to the pulmonary.

You will also note that the rhythm of the heart in little children is often irregular—the so-called sinus arrhythmia—and you must not attach any importance to that, particularly if the child is asleep. Note also the comparative frequency of congenital bruits. If you

hear a loud murmur below the age of two it is almost certainly congenital, because endocarditis hardly ever occurs at that age. Hæmic murmurs, however, may occur below two years. This has been denied by some writers, but there can be no doubt of the fact, and such murmurs may be mistaken for the results of organic disease.

The study of the **pulse** is of little importance in the case of young infants. If you want to count the rate of the heart you do it by listening with the stethoscope, and if you want to learn anything about the arterial tension you do it by observing the fontanelle.

Lastly, we come to **percussion,** and here let me say at once that it is not necessary to use a heavy stroke. *Do not percuss the chests of little children forcibly.* You learn more by light percussion, and you do not give the child the impression that you are punishing him. It is often an advantage to use three fingers, for by that means you throw a broad area into vibration. I ask you to remember also that you may get certain sounds normally in infants which are abnormal in adults, such, for example, as the cracked-pot sound. You can often hear a distinct cracked-pot sound on percussing the apices of a crying child as the air is driven out from the apex of the lung through the mouth. That is due to the great elasticity of the chest-wall, which yields before the percussing finger. Be careful also to percuss during the same phase of respiration when you are comparing the two sides of the chest. You will find that this makes far more difference in the case of children than it does in grown-up people, and I have known forgetfulness of this fact lead to the belief that there was dullness, whereas one was only percussing one lung during inspiration and the other during expiration. Sometimes you will detect dullness over the manubrium of the sternum ; and you must remember in such a case the possibility of an hypertrophied thymus or enlarged bronchial glands.

The investigation of the functions of the **nervous system** in little children is not an easy matter. *Motor paralysis* you must detect by observing whether or not the limb or group of muscles which you believe to be affected is made use of voluntarily. You cannot estimate the exact degree of paralysis by opposing passive resistance to movements as you do in the case of the adult. Remember, however, that a child may not make use of a limb from causes other than paralysis, such, for example, as pain. To this *pseudo-paralysis*, as it is called, I shall have occasion to refer in another lecture.

The *sensory functions* you must investigate as best you can on the same lines as those on which one proceeds in the case of adults, but fortunately sensory paralysis is not common in little children.

The *knee-jerks* are best tested in infants by placing the sole of the foot on the palm of your hand as on a stirrup, whilst with the other hand you tap over the patellar tendon. The *superficial reflexes* are normally rather brisk in childhood, and remember that it is normal for the sole of the foot to give an extensor response up till about the time when the child begins to walk. In other words, a Babinski reflex is normal throughout infancy.

Kernig's sign is one which is often referred to in descriptions of nervous disease in childhood. It is best elicited by placing the child on his back and keeping one leg fully extended whilst the other thigh is flexed to a right angle. If the knee on this side cannot now be extended beyond one and a half right angles (135°) the sign may be regarded as present. I should advise you, however, to be very careful about making inferences from the presence or absence of this sign.

In examining the **fundus of the eye** in young children it is best to use the direct method, and the use of an electric ophthalmoscope makes the examination much easier. No part of your investigation requires greater skill or patience than this, and you must often be content with a very fleeting glimpse of the optic disc.

The **ear** you will examine in the ordinary way, but it is well to remember the shortness of the auditory meatus in infancy and the great obliquity of the drum membrane.

Having carried out these methods of investigation, you will look lastly at the **tongue and throat**. The importance of examination of the throat of the child I cannot exaggerate, because so many diseases of children are associated with abnormal conditions of the fauces. Here you may have to use some force in order to open the child's mouth. There are many ways of coaxing a child to open his mouth, but you may not have any time to waste over him, and may have to proceed to more energetic measures. One of the best ways is to push the lower lip over the lower incisor teeth, and then to press downwards; in order to take the lip away from the teeth the child will open his mouth. Sometimes you may require to hold the nose till a gasping breath is taken. You examine the throat in the usual way, taking care that you get plenty of light.

Finally, you must not expect to be quite systematic in examining

children. I have spoken as if you proceed systematically, but when you come to investigate a case you have to seize the opportunity of examining an organ when you can. When the child is on his back, you examine the heart, the anterior aspects of the lungs, and the abdomen, and when he is sitting up you examine the back of the lungs and the spine. You have to run over everything quickly as opportunity offers.

There are still one or two special points to be mentioned. The first is the **weight and height** of the child. Here is a chart showing the curve of weight for the first year (fig. 1). There are certain turning-points of weight to which I would direct your special atten-

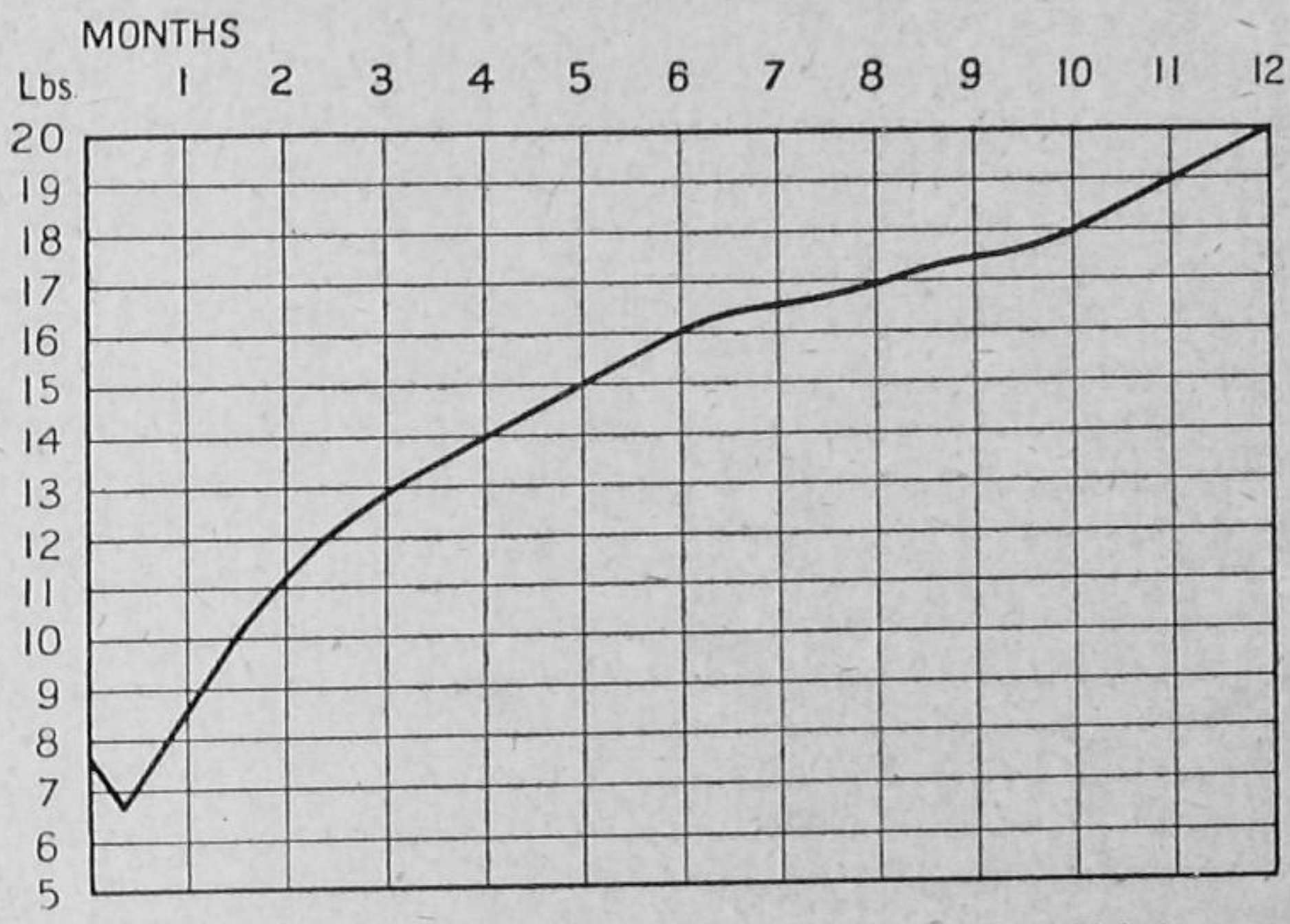

FIG. 1.—WEIGHT CURVE OF FIRST YEAR. (After Holt.)

tion, and which I hope you will be able to remember. At birth the average weight is 7 pounds. At five months old the weight is 14 pounds, at twelve months 21 pounds, at six years 42 pounds, at fourteen years 84 pounds. These weights, being multiples, can be easily kept in mind.

Another important point is the **circumference of the head.** You should remember that the normal is 13 inches at birth; at the ninth month it is 17 inches; at one year it is 18 inches; at five years it has only grown another 2 inches. This will show you the enormous rate at which the child's head grows during the first year of life—growth which is due to the rapid enlargement of the brain.

In conclusion, there are certain **milestones,** as I may call them,

in the development of the child to which I should like to draw your attention, because they are points with which mothers and nurses are familiar, and you will look foolish if you do not know them. The first is the time or age at which the child *cuts its teeth*. This begins at the sixth month. All the temporary teeth are present at the age of three years. The permanent teeth begin to come at the age of six years, and by the age of twelve all except the wisdom teeth are present. The second milestone is the *closure of the anterior on tanelle*, which occurs between the eighteenth and twenty-fourth months. If it is not closed by the twenty-fourth month there is something wrong. The third milestone is associated with the *assumption of the erect position*. A healthy baby will hold up his head at three to four months ; before this he merely waggles it about as if trying to balance it on the neck. If the child does not hold his head up by the age I have mentioned, you should suspect some impairment of mental development. The infant sits up at from nine to twelve months, and will walk at from twelve to eighteen months. He should talk fairly well when he is two years old.

Examination of the **urine** must never be forgotten, difficult although it may be to obtain a specimen. In boys a test-tube may be strapped to the penis : in girls a soap dish may be used as a receptacle.

The last thing I have to mention is the **character of the motions.** Alterations in the fæcal evacuations constitute another set of milestones in development. Remember that for the first eight weeks there should be three to four motions daily and you are not to regard this number at such an age as evidence of diarrhœa. Up to this period the motions resemble beaten-up eggs in colour and consistence, and are of somewhat sour but not feculent odour. From about the eighth month up to the end of two years there is an average of two motions daily. They become now browner in colour, more porridgy in consistence, and of a slightly feculent odour. After two years of age the motions are well formed and feculent.

LECTURE II

INFANT FEEDING

The subject of infant feeding has become unnecessarily complicated in recent years. Systems and methods abound, and the manufacturers of patent foods and dried milk circulate literature which helps to add to the confusion. On the other side of the picture must be placed attempts to feed babies according to a mathematical formula, and what is really an art is wrongly called a science. All these methods ignore the fact that every baby is an individual and will not necessarily conform to the most accurately devised rules and regulations. It is proposed in the following remarks to set out certain general principles which govern infant feeding and then to show how these should be applied in actual practice.

GENERAL PRINCIPLES

There are two fundamental requirements for the successful feeding of infants : the first is common sense and the second is a knowledge of mental arithmetic, so that the few calculations necessary can be easily carried out. There are a few fundamental figures which should be remembered. The first is the average weekly gain in weight. Provided that a baby is gaining weight in a more or less regular manner and at a satisfactory rate then in all probability the feeding is being correctly managed. The new-born baby loses weight in the first few days of life and this should be regained by the tenth to fourteenth day. After the first fortnight and up to about the sixth month (the period during which most difficulties in infant feeding arise)

the average weekly gain in weight is $4\frac{1}{2}$ *to* 6 *ounces.*

If this is achieved, if the baby is reasonably contented, sleeping well between feeds, passing motions daily and not vomiting excessively, then the food is probably correct in quality and quantity.

In general the weight gain is more important than any of the other considerations.

The **quality** of the infant's food will next be discussed. It must clearly be digestible, and here individual babies will show differences. What is easily digestible by one will be found to cause trouble in another. But babies can be trained to develop their digestions and it does not follow that because an infant has difficulty with one milk mixture at the age of a few weeks the same trouble will persist and be present at a few months. Indeed if a baby appears to be unable to digest a rational food at an early age it is of great importance that the digestion be trained by the introduction of small but increasing quantities of the offending part of the milk mixture. Nor must it be lightly assumed that milk is not being digested properly because of its composition. For example, human breast milk is naturally the most easily digestible food for the human infant and its qualitative variations are so rare that the statement 'the baby could not digest his mother's milk' is far more likely to indicate something wrong with the baby than with the milk. In other words, weaning to something else is quite wrong in such circumstances; what is necessary is to deal with the baby and correct whatever faults may be found, leaving the feed unaltered.

Next, as regards quality, the infant's food must contain adequate quantities of energy-supplying material, of body-building material and of those still somewhat mysterious substances, vitamins and minerals. The sugar and fat content of milk supply energy, the protein supplies the body-building materials, and whatever milk is supplied care must be taken that it contains adequate amounts of vitamins and minerals. Human milk, if the mother is herself being properly fed, supplies all the necessary elements required by the human baby, but it does not follow that substitutes for human milk can be satisfactorily achieved by slavishly following the composition of human milk, even if this can be done, which is doubtful. This point will be dealt with in more detail later when the use of cow's milk in infant feeding is discussed.

The general principles governing the **quantity** of food required by the baby will now be set out. It is customary in most of the books on infant feeding to deal in certain units of energy but this only makes the subject unnecessarily complicated. After all, the bulk of babies are fed on milk, human milk or cow's milk, and hence

it is more convenient and much simpler to set out the quantitative aspects of infant feeding in terms of milk. Here we come to the second fundamental figure which can be usefully memorized—

the average baby (7 *pounds at birth*) *requires* $2\frac{1}{2}$ *ounces of human milk or its equivalent per pound body weight per day.*

The details of feeding in the early days of life will be discussed later: the theoretical average amount of milk mentioned above will not be taken until about the tenth day of life. Babies under the average weight require relatively more per pound. For example, a baby of 4 to 5 pounds body weight may require as much as 3 or $3\frac{1}{2}$ ounces of milk per pound daily before a regular weight gain is achieved. Larger babies require relatively less. For example, a baby of three months weighing 10 pounds will probably require but $2\frac{1}{4}$ ounces of breast milk per pound daily and a baby of 6 months weighing 15 pounds will gain steadily on 2 ounces of breast milk per pound daily. These figures are only rough averages and too much attention should not be paid to them if all is going well. They are useful, however, to check the quantitative side of feeding if there appears to be difficulty.

The amount of breast milk obtained by a baby is ascertained by what is called **test-feeding.** On reasonably accurate baby scales (*not* a spring balance) which can usually be hired from the local chemist or from the large instrument firms, the baby is weighed before and after being put to the breast. So long as everything else as regards clothes and napkins is kept constant during the feed the gain in weight represents what the baby has obtained from the breast.

So long as the baby is doing well, as has been said, too much attention should not be paid to the theoretical requirements. Quiet, placid babies often gain weight on smaller feeds than noisy, restless babies for obvious reasons since food is required to supply energy. Difficulty is experienced when there has been trouble over feeding, symptoms of dyspepsia have occurred (such as colic or diarrhœa) and more particularly the infant has failed to gain weight or has gained far too much.

When working out the food requirements of such a baby ought one to reckon them according to the actual weight attained or according to some theoretical average weight for age? Here is

clearly an occasion for the application of common sense. Obviously if a baby has been half-starved and at three months of age is found to weigh only 6 pounds when, since it was average weight at birth, it should weigh 10 pounds, it will be no solution of the problem to continue to feed it on a 6 pound basis. On the other hand, if a feed is prescribed suitable in quantity for a normal baby of 3 months it may well prove too much for the feeble and under-exercised digestive powers of the ailing infant. Hence a good compromise is to take a figure somewhere between the weight that a baby has actually attained and the weight that ought to have been achieved. For example, in the imaginary case discussed the actual weight is 6 pounds and the theoretical weight is 10 pounds; an average between these two is 8 pounds. Similar consideration will suggest that an average between $2\frac{1}{2}$ ounces and $2\frac{1}{4}$ ounces of milk per pound should be given and mental arithmetic shows that a daily quantity of 19 ounces of breast milk or its equivalent is what the baby needs. This amount should be tried; if a gain in weight is obtained the quantity can be increased gradually. If no improvement is obtained, then a larger feed should be tried at once. By this process of careful trial the underweight baby can be safely brought to catch up the normal weight curve and by a similar process the grossly overfed baby can be taught to take smaller quantities, and its symptoms of overfeeding brought under control. It will be noted that in this example the baby at three months was actually less than its birth weight which represents an extreme case. A good working rule is never to feed a baby for less than its birth weight. This is especially important in the early weeks of life when an initial loss of half a pound or more may occur.

If every baby were breast fed then no more need be said on the subject of quantities but unfortunately resort to unnatural forms of infant feeding is only too common. As will be illustrated later when this is discussed in more detail, such artificial feeding mostly consists in the use of cow's milk, in some form or other (liquid, dried or condensed) suitably diluted with additions of sugar and sometimes of fat in the form of cream or cod-liver oil. For the purpose of setting out some more useful figures the following three statements can be introduced at this stage and amplified in later sections: (the word 'equivalent' is very loosely used and must not be misinterpreted; there is nothing to equal breast milk and *all* bottle feeding is second best):

One ounce of undiluted cow's milk is equivalent to one ounce of human milk.

One and a half level teaspoonfuls of sugar are equivalent to one ounce of human milk.

Three quarters of a level teaspoon of 'fat' is equivalent to one ounce of human milk.

(Actually 'fat' is seldom used save as an emulsion which is a dilution. If 25 per cent. cream or a 25 per cent. emulsion of cod-liver oil is used this last statement can be modified accordingly; e.g. $4 \times \frac{3}{4} = 3$ teaspoonfuls of 25 per cent. fat emulsion is equivalent to one ounce of human milk.)

BREAST-FEEDING

It is indubitably true that there has been a serious decline in breast-feeding in recent years in this country. It is commonly asserted that the will to feed their babies by natural means is still present among the mothers of Britain but that for some undiscovered reason they find themselves unable to succeed. This does not seem a very satisfactory explanation. It seems more likely that expert knowledge of the subject is lacking; possibly the mothers of large families in the last century gained this after a period of trial and error associated with a high infant mortality rate. Whatever the reason for the general decline in breast-feeding it is a serious matter, for there is no shadow of doubt that not only is there a higher mortality among bottle-fed babies but there is a greater incidence of illness in the early months of life when artificial feeding has to be resorted to. A greater attention to the details of breast-feeding on the part of the medical and nursing professions would go a long way to promoting a more widespread success in this important matter. With all its difficulties breast-feeding has the great merits of being safe and cheap.

Certain details concerned with the ante-natal care of the breasts and nipples are of great importance and they are dealt with in the textbooks on midwifery. From the point of view of securing successful breast-feeding there are, however, certain aspects of the anatomy and physiology of the breast which must be briefly summarized. In the first place the baby's action at the breast is not a simple process of suction; the baby 'milks' the breast by a

chewing mechanism in which there is active movement of the jaws, lips and tongue—of great importance in the formation of a well-shaped mouth. Although internal secretions play an important part in bringing the breast to a state of lactation, complete emptying of the breast by an actively sucking baby is probably the most important factor in keeping lactation going. The nervous control of lactation must also be remembered. Not only is it true that sudden fright will occasionally produce a sudden 'drying-up' of breast milk but the opposite, so to speak, is also true, and nothing so ensures successful breast-feeding as a genuine expectation of such success by all concerned—the mother (and all her relatives), her nurse and her medical attendant.

The baby should be put to the breast six to eight hours after

FIG. 2.—TWINS, THE ONE ON THE LEFT BREAST-FED; THE OTHER BOTTLE-FED.
A commentary on the advantages of breast-feeding.

delivery and thereafter every three hours. Both breasts should be used at each feeding for an increasing period, beginning on the first day with one minute at each breast and reaching ten minutes on each breast by about the end of the first week. Convenient times are 6 a.m., 9 a.m., 12 noon, 3 p.m., 6 p.m. and 10 p.m. For normal babies there should be no feed during the night and hence the last feed at night should be made as late as possible. The early morning feed can be anticipated by an hour or so if the baby is wakeful but thereafter the fixed times during the day should be adhered to. Once it is apparent that lactation has been well established and in no instance later than about the third month of the baby's life, a change should be made to a four-hourly programme, suitable times being 6 a.m., 10 a.m., 2 p.m., 6 p.m. and 10 p.m. If lactation is

so abundant that enough milk appears to be obtained from one breast then alternate breasts can be used instead of both breasts.

Three practical points in successful breast-feeding must be emphasized. First, the baby must be comfortable. Second, the nasal airways must be kept clear so that breathing can go on satisfactorily while the baby is feeding. Third, a stop must be made three or four times during the feeding period and the baby held upright while the back is patted to encourage the bringing up of ' wind,' that is, swallowed air.

During lactation the mother must drink extra fluid, best taken as a glass or two of water at each of the baby's feeding times. Otherwise there is no need for any special additions to the mother's diet provided that she is getting daily some of the following ; dairy produce (milk, butter, eggs, cheese, cream) ; fresh fruit or salad ; green vegetables ; and sea fish once or twice a week. The nursing mother should usually avoid anything containing vinegar, sour and stone fruit and any of the more exotic foods (lobsters !). If these are taken the baby may suffer from abdominal pain and pass green stools. This sometimes happens if the mother takes too many eggs or proprietary preparations containing egg-powder. Certain drugs may also pass through the milk and affect the baby ; these include phenolphthalein (a common ingredient of proprietary laxatives), arsenic, iodides and bromides.

Unless the mother's diet is deficient in certain substances (fat or iron, for example) there are no foods or drugs which can be counted as efficient galactogogues, or stimulators of the milk supply. A pituitary secretion called prolactin has recently been shown to have a variable effect in this connection and sometimes successfully increases the milk supply. It is given by intramuscular injection.

Every nursing mother should endeavour to get some walking exercise daily in the fresh air. It is also important that she should get some rest during the day, if possible, to compensate for her broken nights.

Within ordinary limits the quality of breast milk remains remarkably constant and uninfluenced by the mother's diet. During the first few weeks of lactation a change from a rather yellow milk (due to ' colostrum corpuscles ') to a pale blue milk takes place but there is no great variation in the percentage composition as regards protein, fat and carbohydrate. Statements to the effect that the milk is weak or watery are to be accepted with the greatest reserve.

Laboratory analysis fails to confirm them. Very rarely excessive fat may be present in the early weeks and cause vomiting. This can usually be cured by giving the infant water before the feeds and the milk soon regains a normal composition. Weaning a baby because of alleged irregularities in the composition of the milk is to be sternly condemned. If symptoms of dyspepsia occur it is far more likely that there is something wrong with the baby or with the method of feeding than with the milk.

During the first week or so of lactation the baby does not receive the full quantities of food necessary for growth and energy. Nature provides colostrum during the first two or three days and the milk 'comes in' about the fourth day, slowly (although sometimes quickly) increasing in quantity. It is advisable to give the baby extra fluid during this period, as weak saline ($\frac{1}{2}$ teaspoonful of salt to a pint of water) for the first two days and as plain boiled water during subsequent days, in quantities of about $\frac{1}{2}$ ounce, from a spoon or from a bottle using a large teat, either before or after the breast-feed. If the baby is restless at night a little water can also be given then. Save in exceptional circumstances when the mother is ill or the baby is small and feeble no cow's milk feeding should be added during the first week. At the end of this period the situation may be reviewed, if lactation appears to be deficient and some addition made. This is best done as a bottle feed after each breast-feed, called **complementary feeding.** The quantities can be ascertained by test-feeding the baby and making up the deficiency by a suitable mixture. The total amounts of food required and suitable mixtures will be discussed later in the section on artificial feeding. What is termed **supplementary feeding,** when a bottle is substituted for the breast at certain times, has nothing to commend it in the early days of life when the object is to secure frequent stimulation of the breasts and adequate emptying. If and when lactation is well established it may sometimes be permitted for economic or social reasons, as when a mother has to go out to work for part of the day.

Certain **difficulties** encountered in breast-feeding may be briefly summarized. **Overfeeding on the breast** should not be interfered with unless there are symptoms of indigestion or unless the weekly gain in weight is regularly excessive (e.g. 12 to 16 ounces for several weeks!). The usual symptoms are those of colic, generally with some diarrhœa, restlessness between feeds and at night, only too

often interpreted as due to hunger. Overfeeding is frequently associated with rapid emptying of the breasts so that the baby takes far too large a feed in a matter of five minutes, also sometimes erroneously interpreted as underfeeding. Vomiting is also a common symptom and may lead to so much loss of food that loss of weight may occur or the weekly gain slows up. In all such cases test-feeding will reveal the true cause of the trouble. Boiled water should be given before feeds to reduce greediness and the length of time at the breast reduced by two to three minutes or more until the disturbances are controlled. A sedative, such as chloral (1 grain) may usefully be given ten minutes before each feed to reduce greed and also to allay any pangs of what may be termed ' relative hunger ' when a baby accustomed to getting 7 to 8 ounces at a time is reduced to his proper ration of 4 or 5 ounces.

Underfeeding on the breast is much commoner than overfeeding and is often associated with air swallowing. It appears to be especially liable to occur with a nervous, over-anxious mother and the baby also soon becomes an over-anxious, unduly nervous, restless creature, crying after feeds and taking the breast in a jerky, ineffective manner. Because of the air swallowing vomiting is likely to occur, unless ' wind ' is carefully brought up, and this adds to the starved condition of the baby. Usually constipation is present because not sufficient bulk is being passed into the intestine, but sometimes a curious type of diarrhœa occurs, in which the infant passes ' hunger stools '—small, dark green stools with little fæcal matter and excess of mucus. These are generally passed during or just after a feed. Again test-feeding will reveal the cause of the symptoms, but the weight curve is sufficient guide in most instances and sometimes a glance at the baby is all that is required. Three things must be done : the mother must be reassured and her over-anxiety allayed by sedative drugs for the baby (again chloral is most useful), steps must be taken to attempt to increase the supply of milk, and, if necessary, complementary feeding must be arranged. With regard to increasing the supply of milk to the baby such measures as the use of both breasts instead of one, three-hourly feeding instead of four-hourly feeding and even an extra feed at night may all be instituted as immediate measures. The various suggestions made above about fluid, diet, rest and exercise should be reiterated. Further, the mother should be instructed to bathe the breasts each night with alternate hot and cold water. Com-

plementary feeding may be necessary and should consist of milk mixtures along the lines to be discussed later. Often a guess can be made at a suitable quantity for a complementary feed ; the mother is told to 'offer' 2 or 3 ounces, for example, after each breast-feed, leaving it to the baby to decide how much is required. It is often possible after a week or so to reduce and even discard the complementary feed. It is altogether too pessimistic a view and not in accord with experience in welfare clinic work that once a bottle is introduced breast-feeding always ceases. It is worth while continuing the breast-feeding when at least 50 per cent. of the daily requirements can be obtained in this way.

Breast-feeding should be continued until the infant is nine months of age. At about six months mixed feeding should be slowly introduced. The details of this process of slow weaning are the same as for bottle-fed infants and will be discussed later.

Theoretically the additions of vitamins to the diet of breast-fed infants would appear to be unnecessary. But such a theory presupposes that every nursing mother is herself adequately nourished, which is far from true. Severe cases of rickets are known to occur in certain circumstances among breast-fed infants, for example. As a working rule the following may be followed : give all babies, breast-fed or bottle-fed, some orange juice daily beginning with a few teaspoonfuls of equal parts of orange juice and water sweetened to taste at the age of one month and increasing up to the juice of a whole orange undiluted at the age of six months. (Tomato juice is a useful alternative ; grape juice is unreliable. During the war black currant purée and juice have been made available. Tablets of vitamin C can also be used if no fruit juice is available). Cod-liver oil is sometimes a useful addition if there is a slight deficiency in the quantity of breast-milk, and in any case should be begun at about three months if there is any doubt about the mother's health and at six months in all babies in Britain.

LECTURE III

ARTIFICIAL FEEDING

The basis of all artificial feeding of infants in ordinary circumstances is some form of cow's milk. Goat's milk and ass's milk are rarely available and various synthetic 'vegetable' milks, although of academic interest, scarcely come into the field of practical infant feeding. Before going into the varieties of cow's milk and their uses it is necessary to set out briefly some fundamental facts about cow's milk which are often misunderstood. The health of the nation—not only of infants—would be improved if there was less ignorance and prejudice on the whole subject.

COW'S MILK

1. *Cow's milk is a dangerous liquid.* Diseases can be and are conveyed from the cow to human beings. The chief of these is tuberculosis which in its bovine form accounts for 2,000 deaths annually in this country and 4,000 fresh cases. Of samples of milk taken in big towns 1 in 10 are found to contain living tubercle bacilli. Undulant fever is another disease of the bovine species which can be conveyed to humans and this is increasing in incidence. Haphazard methods of milking lead to the contamination of the milk by organisms found in the excreta of the cow, leading to diarrhœa in infants. Contamination of the milk by the milker or by those concerned in its collection and distribution is also only too common. Within recent years there have been epidemics of streptococcal sore throats with numerous deaths, traced to infected milk. An epidemic of typhoid fever at a seaside resort some years ago was traced to infected milk.

2. *Are the higher 'grades' of milk safe?* An attempt to get over this grave danger of cow's milk has been made by the official establishment of various grades such as 'certified,' 'grade A,' 'accredited' and so forth, but none of these can be regarded as absolutely safe. 'T.T.' milk is milk from cows which are tested

periodically for tuberculosis, but this is no absolute guarantee that tubercle bacilli are not present in certain samples of milk and no safeguard whatever against the presence of other dangerous organisms. One of the epidemics of sore throat referred to above (with 65 deaths) was traced to one of the highest grades of milk produced on a model farm! There is small wonder, therefore, that an eminent bacteriologist has pronounced the dictum 'No raw milk is safe.'

3. *Milk can be made safe by heat.* The disease-bearing organisms can be rendered harmless by heat, and milk can readily be made safe in this way. The process of pasteurization, if efficiently performed, provides milk which is practically free from the risk of conveying tuberculosis and most other milk-borne diseases. For children over the age of two this pasteurized milk is the best milk to use. Under two years—i.e. for the feeding of infants—there is still the risk that pasteurization may not have been efficiently performed and therefore it is best to *boil all milk for babies.* It is most convenient to do this with a double saucepan and all that is necessary is that the milk shall be definitely brought to the boil. Since all milk, even the most expensive grades, must be treated in this way, it is obviously absurd to buy such milks—which have the added disadvantage of often being too rich in fat—and if liquid milk is to be used for infant feeding the mother should buy ordinary milk, pasteurized or not, and boil it. Obviously the heat required to dry milk to a powder or to condense it will also effectively kill all the disease-bearing bacilli.

4. *Does heating milk affect it adversely?* This question can be simply answered by the statement that every piece of scientific evidence goes to show that children thrive as well on heat-treated milk as on raw milk, with a single exception to be noted. Indeed the effect of 'cooking' the milk, as with all cooking, is to render some of its constituents more digestible and therefore better utilized than in the raw state. The only substance definitely destroyed by heating is vitamin C, but this can very easily be added to the infant's diet in the form of orange juice or tomato juice. (See p. 22.)

FEEDING WITH LIQUID MILK

Cow's milk differs in composition from human milk. It contains less sugar—only about 4 per cent.—as compared with 6 per cent.

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in human milk, about the same amount of fat, although the fat emulsion is much coarser in cow's milk, and double as much protein. Very young infants are not able to digest undiluted cow's milk, principally because of the amount of protein present, and some modification is desirable. A superficial study of the subject would suggest that cow's milk might be so modified that the percentage composition is made to resemble human milk and this can be done as follows :

Take 10 ounces of boiled cow's milk and add 10 ounces of water. (This reduces the protein percentage by half and thus brings it to the same amount as in human milk.) Add 1 ounce of lactose (bringing the sugar content up to 6 per cent.) and 1 ounce of 50 per cent. cream (bringing the fat content up to 4 per cent.).

Such a mixture is often called ' humanized milk,' but it is necessary to examine this claim in more detail as it contains certain serious errors. The most important fallacy lies in the assumption that the protein of cow's milk is the same as the protein of human milk so that simple dilution gives a correct amount for the infant. This is not so. Cow's milk protein consists mostly of casein (strictly speaking it is caseinogen) with a small amount of lactalbumin. Human milk protein has a much higher proportion of lactalbumin. Both casein and lactalbumin are composed of various amino-acids and although the total protein of the modified milk mixture described above is similar to that of human milk the individual amounts of the lactalbumin and amino-acids are different from those present in human milk. That means that a baby fed for any length of time on such a mixture might be starved of certain essential amino-acids. It is not certain that this is the case and in fact many babies have been successfully reared on such mixtures. But the risk is there and it is better to avoid it. It must be accepted that cow's milk is inferior to human milk for rearing the human infant ; by no known method can human milk be manufactured save in the mother's breasts and it is better to abandon all pretence of ' humanizing ' cow's milk and use it intelligently in as strong a mixture as the infant's digestion can stand. There is no scientific evidence that this ever did any harm. It must also be remembered that dilution of cow's milk means that minerals and vitamins are also diluted and the baby may go short of some essential substance.

Regarding the other constituents of the milk, the sugar (lactose) is the same in both varieties, but lactose is expensive and ordinary cane sugar does just as well. If fermentation of sugar appears to

occur, i.e. if the infant has loose, acid stools, a non-fermentable variety such as dextri-maltose should be employed. Glucose should not be used in infant feeding. The fat of cow's milk is less digestible by the human infant than the fat of human milk and the addition of large quantities of cream to dilute milk mixtures is undesirable. It may even lead to an intolerance of fat in later infancy.

The feeding of a baby in the first ten days of life will be discussed later. Assuming that this has been accomplished by artificial means, then a suitable mixture made from liquid milk can be devised in the following manner:

According to the general principles set out previously, the infant weighing 7 pounds requires $2\frac{1}{2}$ ounces of human milk or its equivalent per pound body weight per day. This means $17\frac{1}{2}$ ounces a day and on a basis of six feeds this can be given as 3 ounces per feed. For an infant of ten days cow's milk should be diluted with equal parts of water. So each feed will consist of $1\frac{1}{2}$ ounces of boiled milk and $1\frac{1}{2}$ ounces of water. But this will only give the infant the equivalent of $1\frac{1}{2}$ ounces of human milk per feed. If $1\frac{1}{2}$ level teaspoonfuls of sugar (ordinary brown sugar) is added per feed this is equivalent to 1 ounce of breast milk. The feed is still short of $\frac{1}{2}$ ounce of breast milk equivalent, or 3 ounces short in the twenty-four hours. There are various ways in which this can be supplied. One is to add some fat emulsion to each feed (cream or one of the special artificial creams), but young infants often have some difficulty in digesting such fat. Cod-liver oil, in $\frac{1}{4}$ to $\frac{1}{2}$ teaspoonful doses three times a day (from a spoon) will nearly make up the missing values.

In practice it is best to start the infant on such a mixture (milk, water, sugar) and gradually add more milk so that after about a week the mixture would be boiled milk, 2 ounces, water $1\frac{1}{2}$ ounces, sugar $1\frac{1}{2}$ teaspoonfuls. The weight curve and general state of the infant must now be watched and usually the milk in the mixture must be gradually increased by about $\frac{1}{2}$ ounce a fortnight. Thus when the infant is six to eight weeks old a suitable milk mixture would be boiled milk, 3 ounces, water $1\frac{1}{2}$ ounces, sugar $1\frac{1}{2}$ teaspoonfuls. In addition, cod-liver oil (from a spoon) $\frac{1}{2}$ teaspoonful twice or three times a day and orange juice daily. If the infant is gaining weight satisfactorily, having regular stools, and seems generally contented, the feed is probably correct. The sugar can be increased to $1\frac{3}{4}$ or 2 teaspoonfuls per feed if there is any tendency to infrequency of stools and a slow weight gain. If there is a tendency to loose stools the sugar may have to be reduced slightly and the amount of milk slightly increased.

Thereafter the milk mixture can be made stronger. A change to four-hourly feeding (five feeds in the twenty-four hours) should be made at three months of age or 10 pounds in weight, whichever comes first. When the baby reaches 15 pounds in weight or six months of age the following position should have been reached: the infant requires 30 ounces a day of human milk or its equivalent (2 ounces per pound body weight). This means 6 ounces each feed and may be made up with $4\frac{1}{2}$ ounces of boiled milk and $1\frac{1}{2}$ ounces of water and $1\frac{1}{2}$ to 2 teaspoonfuls of sugar, plus cod-liver oil from a spoon (1 teaspoonful daily). Often by this age the strength of the mixture can be increased, the amount of water being diminished gradually so that undiluted boiled milk with added sugar is given. Some authorities recommend this from an earlier age. At six months mixed feeding is begun according to the method to be outlined later. Such milk feeds as are continued can be gradually increased up to 7 or 8 ounces per feed of undiluted milk.

Water has been suggested as the diluting fluid in the milk mixtures described and in general for the healthy infant there is no need to use lime water, barley water or to add citrate. Before it was realized that the only way to make milk safe is to boil it (and when it was feared that something vital was destroyed in boiling) one of the difficulties about using unboiled cow's milk was the large curds formed in the infant's stomach. The addition of citrate and the use of barley water were intended to produce a smaller curd. With boiled milk the position is different, as can be easily proved by comparing the results of the addition of rennet to unboiled milk at blood heat and to milk which has been boiled and cooled. In the unboiled milk the usual 'junket'—large curd—is formed and in the case of boiled milk a thick sort of paste of much smaller curd. Barley water and citrate do not improve this very much more. Lime water is a poor way of giving calcium and is unnecessary if dilute mixtures are avoided. Cow's milk is one of the best sources of calcium available and added lime water may actually contain less calcium than the milk it displaces. All water used in infant feeding should be boiled.

FEEDING WITH DRIED MILK

One of the difficulties about liquid cow's milk is that it is easily contaminated and greater safety is achieved in households where this risk exists by the use of dried milk, since in powder form storage

difficulties are overcome. Milk is made into powder by exposing it to great heat and practically all dried milks are reconstituted to make the original milk by taking 1 level teaspoonful (or the measure supplied with the packet of dried milk) and adding sufficient boiled water (not boiling) to make 1 ounce. Most of the brands of dried milk reconstituted in this way give 1 ounce of ordinary cow's milk so that we might add to the statements set out in the section on 'general principles'—

One level teaspoonful or measure of ordinary dried milk powder is equivalent to 1 *ounce of human milk.*[1]

It is not proposed here to discuss individual brands of dried milk by name. Popularity of certain brands varies in different parts of the country, and since the same principles apply to all, the matter can be dealt with in general terms. There are three varieties of dried milk to be considered in the feeding of normal babies. One is the ordinary full cream variety already mentioned which, when reconstituted 1 in 8 (1 teaspoon or measure to the ounce of water) gives milk of the same composition as undiluted liquid milk. Now whereas the drying process actually increases the digestibility of the protein elements of milk so that dried milk mixtures may be used in greater strength than with liquid milk, the fat present is made slightly less digestible. In consequence young infants may have some difficulty with full-cream dried milk. Hence a second variety of dried milk which has had some of the fat skimmed off before drying is available known as 'half-cream milk.' In its original form this, when reconstituted 1 in 8, gave a partially skimmed milk of which 1 ounce was equivalent to only about $\frac{4}{5}$ of an ounce of human milk. In some instances therefore manufacturers have added sugar to the dried milk powder to make up the deficiency. This is to be deprecated, as it is better for the amount of added sugar to be determined for each baby and varied according to conditions of weight-gain and the state of the stools. The third variety of dried milk available has been so modified before and after drying that when reconstituted 1 in 8 it gives a milk which resembles human milk in composition and hence this is called 'humanized dried milk'; 1 ounce of the reconstituted mixture is equivalent to 1 ounce of human milk.

[1] Teaspoons vary greatly in size, and it is perhaps unfortunate that they are so universally used for measurement. The '1 in 8 dilution' here mentioned means 60 grains to the ounce. It is better to use the measure supplied with the milk or a medicine glass graduated in teaspoons. Milk powder must never be heaped but always smoothed level.

It is convenient, because of possible trouble over fat, to use either humanized dried milk or half-cream dried milk (plain with added sugar) for the young baby and, omitting for the present details of feeding in the first ten days, a plan of feeding would be as follows :

Feeding with humanized dried milk all that is necessary is to calculate how many ounces of breast milk are required per feed and then give the same number of ounces of reconstituted humanized dried milk using 1 level teaspoonful or measure of the powder to make each ounce required. Thus the 7 pound baby requiring $17\frac{1}{2}$ ounces of breast milk a day or 3 ounces per feed receives 3 ounces at each feed of the humanized dried milk made up with 3 measures or teaspoonfuls of powder to 3 ounces of water. As the baby gets older the amount per feed is increased, the strength remaining the same. Thus at, say, 9 pounds body weight the baby requires something between $2\frac{1}{2}$ and $2\frac{1}{4}$ ounces per pound, say 21 ounces a day or $3\frac{1}{2}$ ounces per feed.

With half-cream dried milk it must be remembered that reconstituted 1 in 8 it yields a milk with practically the full protein content of undiluted cow's milk. Dilution of this to half is not necessary as the protein is relatively more digestible than with liquid milk. A suitable mixture with which to start the infant at, say, ten days of age is made by taking 3 measures (or level teaspoons) of half-cream dried milk powder and adding this to enough water to make 4 ounces. Sugar is then added (to the plain variety), $1\frac{1}{2}$ teaspoonfuls, and the infant is given 3 to $3\frac{1}{2}$ ounces of this per feed, increased gradually as required to produce the requisite gain in weight.

Neither of these two varieties of dried milk should be continued for more than a month or two. Certainly they should not be used for longer than three months or after the baby attains 10 pounds in weight. A change should then be made to the full cream variety of dried milk made up as follows :

The baby now requires $22\frac{1}{2}$ ounces of breast milk or its equivalent a day, divided into 5 feeds of $4\frac{1}{2}$ ounces. Each ounce of reconstituted full-cream dried milk is equivalent to 1 ounce of breast milk. Hence take 3 measures or level teaspoons of full cream dried milk and add to enough water to make 3 ounces. Then add another $1\frac{1}{2}$ ounces of water (in practice, of course, the whole quantity is added at once) to make $4\frac{1}{2}$ ounces in all. Add $1\frac{1}{2}$ teaspoonfuls of sugar and the feed is more or less identical in composition with that made from liquid milk at this weight and age (i.e. 3 ounces of milk, $1\frac{1}{2}$ ounces of water, $1\frac{1}{2}$ teaspoonfuls of sugar). It can gradually be increased in strength and size as the baby grows—e.g. 5 measures to 6 ounces of water with $1\frac{1}{2}$ teaspoonfuls of sugar at 15 pounds weight or six months of age.

The amount of sugar can be adjusted up or down, just as in feeding with liquid milk, if the state of the stools warrants such variations.

The change from one variety of dried milk to another should always be made gradually, substituting one bottle the first day, two bottles the next day, and so on. Additions of cod-liver oil and orange juice (see p. 22) should be made from about one month of age, even although some varieties of dried milk contain added synthetic vitamins.

Feeding with various patent foods and starch additions to liquid milk mixtures is quite unnecessary with the normal baby although of value in certain digestive disturbances. In any case such a method is expensive, as in fact is all feeding with dried milk at ordinary retail prices.

FEEDING WITH CONDENSED MILK

Two main varieties of condensed or evaporated milk are available, sweetened and unsweetened. The sweetened varieties should not be used for the feeding of normal babies as the sugar content is altogether too high and feeds containing dilutions of milk equivalent in value to human milk are too rich in sugar and too poor in protein. The unsweetened brands of condensed milk are, however, of great value. They make better mixtures than dried milk in which the fat often appears as globules on the surface of the reconstituted liquid. On the other hand, once the tin of condensed milk has been opened it is liable to contamination and thus difficulties of storage exist. The strength of the different brands of unsweetened condensed milk varies slightly but in general reconstitution of liquid cow's milk strength is obtained by taking 1 ounce of condensed milk and adding $1\frac{1}{2}$ ounces of water (1 in $2\frac{1}{2}$ dilution). It will be seen that 1 ounce of unsweetened condensed milk is equivalent to $2\frac{1}{2}$ ounces of human milk. Suitable mixtures can be made on this basis as follows :

For the baby at ten days of age weighing 7 pounds it is desirable to dilute the condensed milk below the strength of liquid cow's milk ; 1 ounce of condensed milk added to 4 ounces of water gives 5 ounces of a milk mixture which is half the strength of liquid cow's milk. To every 3 ounces of this (a suitable quantity per feed) add 1 to $1\frac{1}{2}$ teaspoonfuls of sugar. Give small amounts of cod-liver oil ($\frac{1}{2}$ to 1 teaspoonful) daily. The strength can gradually be increased so that at 10 pounds in weight, for example, with $4\frac{1}{2}$-ounce feeds the amount of unsweetened

condensed milk should be about $1\frac{1}{4}$ ounces, water $3\frac{1}{4}$ ounces and sugar $1\frac{1}{2}$ teaspoonfuls. At 15 pounds weight full strength reconstitution (1 in $2\frac{1}{2}$) can be employed with added sugar. Additions of cod-liver oil and orange juice should be given as described above for feeding with liquid or dried milk.

FEEDING IN THE FIRST TEN DAYS OF LIFE

If a baby must be fed artificially from birth the following programme should be adopted. The difficulty in the management of this period as regards infant feeding lies in the fact that increases in the size and the strength of feeds have to be made at the same time. The figures given are based upon an average baby of 7 pounds weight. The aim is to reach the full requirements of the equivalent of $2\frac{1}{2}$ ounces of breast milk per pound body weight by the seventh to tenth day. With smaller babies slight reduction in the size of the feeds should be made; with larger babies larger feeds can be given after the seventh day.

(*a*) *Quantities.* The baby is offered $\frac{1}{4}$ to $\frac{1}{2}$ ounce of half-strength normal saline ($\frac{1}{2}$ teaspoonful of salt to 1 pint of water) at each feeding time (i.e. every three hours for six feeds) on the first and second days of life. On the third day the milk feed is started, giving $\frac{1}{2}$ ounce at each feed and thereafter increasing the amount at each feed by $\frac{1}{2}$ ounce a day—thus 1 ounce per feed on fourth day, $1\frac{1}{2}$ ounces on fifth day and so on until 3 ounces is reached on the eighth day. These quantities must be regarded with common sense: the baby may refuse to take the full amount offered at each feed and in other instances may appear so hungry or thirsty that a little extra may be given at certain feeds.

(*b*) *Strength of feeds.* In the sections above on the use of liquid milk, dried milk and condensed milk, suitable dilutions for feeding the infant after the tenth day were given. Briefly summarized these standard mixtures were:

(i) *Liquid milk.* Boiled milk $1\frac{1}{2}$ ounces, water $1\frac{1}{2}$ ounces, sugar $1\frac{1}{2}$ teaspoonfuls.

(ii) *Dried milk.* Humanized variety, 1 measure to 1 ounce of water. Half cream variety, 3 measures to 4 ounces of water with $1\frac{1}{2}$ teaspoonfuls of sugar.

(iii) *Unsweetened condensed milk.* 1 ounce added to 4 ounces of water and 1 to $1\frac{1}{2}$ teaspoonfuls of sugar to 3 ounces.

When artificial feeding is introduced on the third day of life any of these standard mixtures can be chosen, according to the general

decision on the type of feed to be used during infancy, and given in $\frac{1}{4}$ strength, i.e. 1 part of the mixture added to 3 parts of water. On the fourth day the mixture should be given in $\frac{1}{2}$ strength (1 part with equal parts of water). On the fifth and sixth day it should be given in $\frac{3}{4}$ strength (3 parts of the mixture to 1 of water) and thereafter in full strength. Here again individual variations must be made. With smaller babies the increase in strength should be slowed up. If the stools appear loose and irritant the strength can be kept more dilute for a few days so that full strength is not reached until the ninth or tenth day. The important point is to make the changes, both in quantity and in quality, slowly and to be prepared to modify the general programme according to developments. In other words, like so much of infant feeding, the method is one of trial and error. The general principles here described will, however, tend to minimize the risk of dangerous error.

The foregoing remarks can be summarized in the following table :

FEEDING IN FIRST TEN DAYS OF LIFE

Day . . .	1	2	3	4	5	6	7	8	9	10
Size of feeds in ounces . .	$\frac{1}{4}$ $\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	1	$1\frac{1}{2}$	2	$2\frac{1}{2}$	3	3	3
Strength of feeds . .	saline	saline	$\frac{1}{4}$	$\frac{1}{2}$	$\frac{3}{4}$	$\frac{3}{4}$	Full	Full	Full	Full

The infant is fed every 3 hours making 6 feeds in the day. The strength of feeds refers to the standard mixture for use in the early weeks of life. For large infants the feeds on the ninth and tenth days may be made $3\frac{1}{2}$ and 4 ounces respectively.

COMPLEMENTARY AND SUPPLEMENTARY FEEDING

These terms were explained in the section on breast-feeding, but it is convenient to add a few remarks at this stage. The theoretical quantity of breast-milk required at each feed can be worked out according to the various principles already set out. If complementary feeding has to be undertaken in the first week of life (and as already explained this is undesirable for normal babies with normal mothers) the table just given shows the quantities of total food required at each feed. Test-feeding will indicate the amount of breast-milk obtained and the complementary feed necessary to bring the quantity up to the appropriate total is then given from

a bottle, the strength and nature of the feed depending on the baby's age as already set out. For example, a complementary feed (if necessary) on the fifth day of life would be $\frac{3}{4}$ strength of the standard mixture suitable for young infants. A complementary feed for an infant of three months of age would consist of, say, cow's milk 3 ounces, water $1\frac{1}{2}$ ounces, sugar $1\frac{1}{2}$ teaspoonfuls. Supplementary feeding, similarly, will depend on the age and weight of the infant for a correct ascertainment of the size and strength of the feeds.

This may all sound very complicated and it is not always easy to carry out test-feeding. Actually it is quite simple to plan a programme of complementary feeding without the use of test-feeding at all. Suppose the breast-fed baby appears to be underfed, a consideration of the severity of the symptoms and the actual weight compared with the theoretical weight for age will enable a guess to be made at the amount of the deficiency. The infant is then offered 1 ounce, 2 ounces or 3 ounces of a suitable complementary feed and the mother instructed not to force the infant to finish the bottle. After a few days on this programme the situation is reviewed and the quantity of complementary feed adjusted according to the results. Often it is found that the infant will refuse the bottle altogether after certain breast feeds and finish it completely or incompletely at others. Quantities can be therefore altered to suit the requirements. With an intelligent mother complementary feeding can be carried on successfully for many months.

BOTTLES AND TEATS

Either the simple single-ended bottle with rubber teat or the double-ended, boat-shaped bottle with rubber teat and small rubber valve should be used. Great care must be taken to keep the bottles clean ; a daily boiling of bottle and teats is best. If a bottle brush is used it must also be kept carefully clean, as thrush organisms may develop on any milk curds left attached to the base of the bristles. In general the baby should be induced to feed from as big a teat as possible. This is because with the small teats a pure sucking mechanism is likely to be employed whereas with a big teat the baby has to employ a 'champing' process, with good exercise for the jaws, similar to that necessary when feeding at the breast. The hole in the teat must be adjusted so that the feed is

taken in ten to fifteen minutes. Two or three halts for the bringing up of wind should be made just as with the breast-fed baby and with single-ended bottles the teat must be slipped off several times during the feed to allow air to enter the bottle. The bottle should be held nearly vertical so that the baby is always getting liquid only and not a mixture of air and liquid. All feeds should ideally be given at body temperature but as some cooling occurs during the feeds it is usual to begin with the feed at slightly higher temperature, say 100° F.

When liquid milk is being used it is sometimes convenient to make up all the feeds for the day at one time. With dried milk or condensed milk it is better to make up each bottle separately.

WEANING AND MIXED FEEDING

As already mentioned, a start with mixed feeding should conveniently be made when the infant reaches about six months of age. As far as possible weaning should be carried out slowly and the programme to be outlined here spreads out the whole process over about three months. Rapid weaning should certainly be avoided in hot weather or if the infant is showing any signs of illness of any sort. The first step is gradually to substitute a feed of bone and vegetable soup for the 2 p.m. feed—whether breast or bottle. The following recipe may be used for the bone and vegetable soup :

Bone and Vegetable Soup.

Required : (1) About two pennyworth of chopped fresh bones ; bones from a roast joint could be chopped up and used instead sometimes, but fresh are better. When possible add scraps of meat, especially bits of gristle which would otherwise be discarded. A small piece of liver is a good addition.

(2) A selection of three or four of the following (always try and include one green vegetable—coarse outer leaves are best) : Beans (fresh or dried), celery, carrots, lettuce, parsnip, peas (fresh or dried) swedes, turnips (small amount only), broccoli, cabbage, cauliflower (leaves or flower), sprouts, turnip tops, tomatoes, potato.

For instance—3 or 4 outer lettuce leaves, 2 medium carrots, 1 tomato, small piece of turnip, 1 tablespoonful of peas. Increase the amount of vegetables gradually as child gets older.

Method : Wash bones and scraps, cover with water and soak for one hour. Bring bones and steeping water slowly to the boil, either in a saucepan or in a stone jar standing in a saucepan. Keep sim-

mering for one hour. Add vegetables and simmer for another hour. Strain and when cold, take off all the fat (you can melt the fat down and use it for cooking). If there is any water in which vegetables have been cooked, add a small cupful to the soup; if not, put in a pinch of salt to improve the flavour. There should be $\frac{3}{4}$ to 1 pint of fluid at the end. A $\frac{1}{4}$ teaspoonful of marmite may be added.

Note. The soup should keep for 2 days, except perhaps in very hot weather, or 3 days in cold weather. Keep in the coolest place available.

The infant is offered one or two ounces of this soup from a cup and spoon and this is followed at first by a breast or a bottle feed, reduced in time to, say, 5 or 7 minutes at each breast, or a bottle feed of, say, 4 or 5 ounces of whatever the infant has been having. Few infants take to the new feed readily and patience is required to secure that the new taste and the new method of feeding are successfully achieved. The amount of soup is gradually increased and the amount of breast milk or cow's milk gradually decreased until the 2 p.m. feed is entirely soup. This can usually be reached in three or four weeks. Then at 7 months of age the next step in weaning is taken. This consists in substituting 'porridge' at the 10 a.m. feed. A suitable recipe is as follows:

PORRIDGE.

Mix one level tablespoon of groats or finest oatmeal into a smooth paste with cold water, add a pinch of salt and make up to $\frac{1}{4}$ pint (5 ounces) by stirring in boiling milk (for the first few days use half milk and half water). Boil gently for $\frac{1}{2}$ hour, or cook in double saucepan. Serve warm, with a little boiled milk poured over it. A small piece of butter may be added.

Fine oatmeal or groats is used at first and the porridge should be sieved for the first few months. Thereafter the sieving can be omitted and later a coarser variety of oatmeal substituted. The quantity of porridge is gradually increased and the amount of breast-milk or bottle-feed decreased just as with the soup at the 2 p.m. feed. During this seventh month the soup can be thickened somewhat by adding some of the vegetable pulp or some mashed potato.

The next stage takes place at the eighth month when a drink of milk from a cup and a crust of bread and butter, with honey or marmite, is substituted for the 6 p.m. feed. During this eighth month the baby is gradually weaned altogether from the breast or bottle. Generally the 6 a.m. feed can be dropped first, the infant being given orange juice on waking and the times of the three

substitute feeds all made earlier. In addition the 'breakfast' of porridge can now be elaborated by the addition of a second course and 'dinner' can also be changed from 'thickened soup' to 'vegetables and gravy' and a second course added. The process of weaning can be summarized in the following table.

	Age			
Time of Feeds.	6 months.	7 months.	8 months.	9 months.
6 a.m. . .	Breast or bottle	Breast or bottle	Breast or bottle	Orange juice on waking
10 a.m. . .	Breast or bottle	'Porridge'	Porridge, Fried bread or toast	8.30 a.m. Breakfast
2 p.m. . .	Soup	Thickened soup	Vegetables and gravy. Pudding	12.30 p.m. Dinner
6 p.m. . .	Breast or bottle	Breast or bottle	Cup of milk and crust	5 p.m. Tea-supper
10 p.m. . .	Breast or bottle	Breast or bottle	Breast or bottle	Breast or bottle

A hard crust to chew daily is advisable from the age of six months. The infant should also continue to have orange juice and cod-liver oil daily. The total amount of milk daily should be about 1 pint, including that used in cooking: it must still be boiled.

Milk puddings should be made with ground rice, cornflour, cream of wheat and similar fine cereals to start with. Coarser varieties can be added gradually —semolina or fine sago.

At the ninth month the following scheme of mixed feeding can be adopted and the remaining breast or bottle-feed (at 10 p.m.) omitted gradually.

Diet. Nine to Eighteen Months

On Waking:

Fruit juice or water.

Breakfast:

(1) Well cooked groats, or Quaker Oats, or fine oatmeal, made with milk, *or* boiled or scrambled egg.
(2) Toast with butter or dripping, or fried bread.
(3) Milk to drink.

[redacted]

Dinner :

(1) Soup or gravy with sieved vegetables (any variety), *or* steamed fish.
After 1 year, give small amounts of liver, brains or meat.
(2) Well-cooked milk pudding with fruit juice or stewed fruit or baked apple,
or egg custard,
or steamed 'cake' pudding or light suet pudding.
(3) Water to drink.

Tea-supper :

(1) Crusty bread or toast, with butter or dripping and honey, jelly jam, or golden syrup, or marmite, sieved tomato or lettuce. A little piece of raw apple or banana to be well chewed (after 1 year old).
(2) Milk or milky cocoa to drink.

10 *p.m.* :
Drink of milk at first until child learns to sleep through from 'tea-supper.'

During the second year of life the diet can be gradually elaborated and a suitable diet sheet from eighteen months onwards is as follows :

Diet. Children aged 1½ to 3 Years

Breakfast :

(1) Porridge, groats or 'Force' or other breakfast cereal, made or served with milk.
(2) Egg ; boiled, poached, scrambled, baked or omelette, *or* Fat bacon, lightly fried, *or* Fish (freshly cooked, if it can be bought fresh), kedgeree or fish cake, *or* in summer, stewed fruit.
(3) Bread fried in bacon fat or dripping.

Toast or bread (with crusts) and butter.
Honey or syrup, or jelly jam.
Fresh fruit or fruit juice, or sieved tomato.
Milk (may be coloured with cocoa).

Dinner :

1st course—
(*a*) Soup or broth, *or*
(*b*) Meat, including bacon and ham. Frozen meat is as good as English, if thawed slowly. Cheaper cuts are as nourishing as expensive joints, if suitably cooked. Stews are good, and may be served with dumplings, *or*
(*c*) Rabbit or chicken, *or*
(*d*) Fish (mackerel and shell fish are *un*suitable), or fish roe, *or*

(*e*) Brains, heart, tripe or liver, if *stewed, or*
(*f*) Cheese dishes.

Potatoes (boiled in the skin).
Vegetables.—All varieties, especially green vegetables. Peas and beans should be sieved unless very young. Vegetables should be cooked without soda and with the lid on the pan. The water in which they have boiled should be added to soups.

2nd course—
Milk puddings (cooked thoroughly) *or*
Milk shapes, *or*
Baked, steamed or boiled egg custard, *or*
Junket made with pasteurized milk (may be flavoured with cocoa or chocolate to make variety) *with*
Stewed fruit. (Fruits with seeds or skins should be sieved), *or*
Baked apple or raw fruits (not pineapple or nuts. Banana should be ripe and mashed) *or*
Steamed suet pudding with fruit or syrup, *or*
Egg jelly or milk jelly, *or*
'Sponge cake' pudding, *or*
Bread and butter pudding with cherries or seeded raisins.
Water, lemonade or orangeade (made with fresh oranges and lemons).

Tea:

(1) Toast or bread with butter or dripping or vitaminized margarine.
Rusks or plain biscuits or oatcake.
Plain cake or shortbread (do not give cheap pastries and buns).
(2) Honey, golden syrup or jelly jam.
Stewed fruit or watercress, lettuce or sieved tomato.
Home-made meat paste, for a change, marmite or cheese.
(3) Milk, or milky cocoa.

LECTURE IV

SOME DISEASES OF THE NEWLY-BORN

So soon as a child is born it meets with various adverse influences, which may render it the subject of disease before it has hardly begun to live. As many of these diseases are capable of being prevented if only one is alive to the possibility of their occurrence, I propose to devote this lecture to a brief consideration of some of the commonest of them, only premising that I shall not attempt to deal either with surgical ailments or with congenital malformations or deformities.

At the outset it may be well to recall to your recollection some of the outstanding physiological peculiarities of the newly-born child, some of which render it peculiarly vulnerable to the attacks of disease.

For the first few days at least of life the heat-regulating mechanism has not got into working order. In consequence of this, young infants, especially if at all premature, suffer severely from exposure to cold, and chill is a danger which must be sedulously guarded against in their case. For the same reason, rises of temperature are apt to occur upon slight provocation, and have not as much significance as they have later on. A slight rise of temperature in the first three days of life may, indeed, be considered as a normal occurrence. Should the rise be great (103° F. or so), it is to be regarded, in the absence of any other apparent cause, as an indication for the necessity of giving fluid: half-strength normal saline ($\frac{1}{2}$ teaspoonful of salt to the pint of water) should be offered freely. To this form of pyrexia the term **dehydration fever** is sometimes applied (fig. 3).

Difficulties in respiration are common in the early days of life, all generally grouped under the unsatisfactory term **asphyxia.** You will be instructed in the course of your midwifery studies about the importance of securing a clear passage for the first inspiration, and for a fascinating solution of an old problem I refer you to Sir Joseph Barcroft's work on the factors concerned in producing this

first breath. Once breathing is established certain forms of respiratory failure can still occur. Obstruction in the air passages may still be a problem. Not only is adequate aspiration of the pharynx of great importance but I suggest that you also pay some attention to the nasal passages. The new-born baby has such an intense instinctive urge to breathe through the nose (probably because breast-feeding can only be accomplished if the nose is used for breathing) that it will go blue in the face, struggle, cry and even stop breathing altogether rather than breathe easily through the

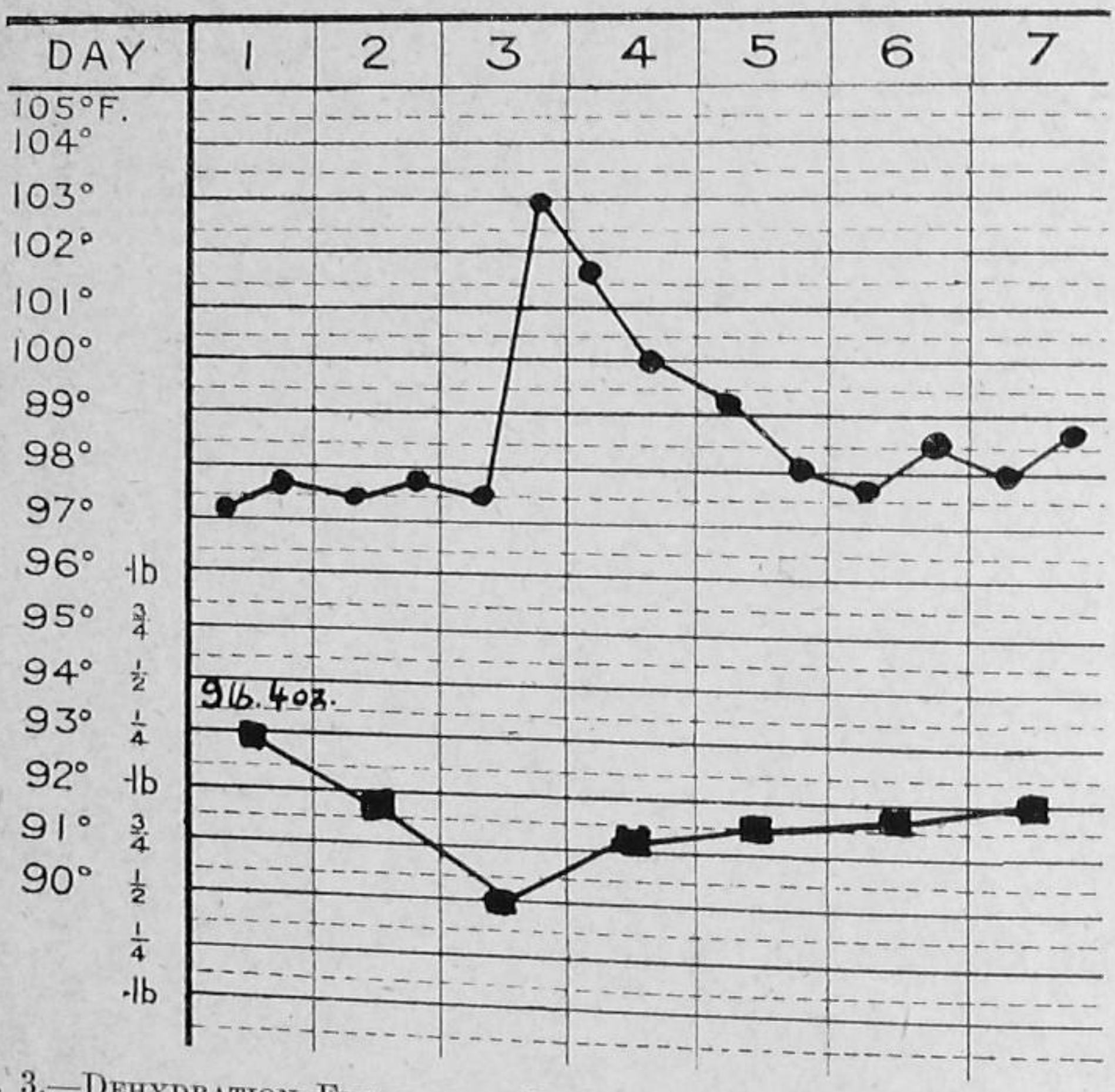

FIG. 3.—DEHYDRATION FEVER IN A BABY WEIGHING 9 LB. 4 OZ. AT BIRTH.
Note how rise in temperature (top line) coincides with the fall in weight (bottom line), both due to loss of fluid.

mouth. A soft rubber catheter (No. 5) lubricated in paraffin should be passed through each nostril into the naso-pharynx. Another difficulty with respiration in the early days of life may be due to the modern use of sedative drugs for the mother; the baby's respiratory centre under the influence of such narcotics fails to respond satisfactorily to the normal chemical and nervous regulators of the respiratory rhythm. For such babies the centre can be stimulated by injections of coramine (niketha mide) ($\frac{1}{2}$ to 1 c.c.) and

by inhalations of carbon dioxide in oxygen (5 to 7 per cent.) given either by nasal catheter or by a close-fitting rubber mask. With weak and premature babies, especially when obstruction in the respiratory tract has not been adequately abolished, full expansion of the lungs may be greatly delayed, and to such a condition the name **atelectasis** is applied. Weak air entry at the bases of the lungs, sometimes with added moist sounds, in a feeble, cyanosed baby, indicates the presence of this condition. Stimulation of respiration on the lines indicated for the narcotized baby should be carried out. In all babies with respiratory failure oxygen should be freely used.

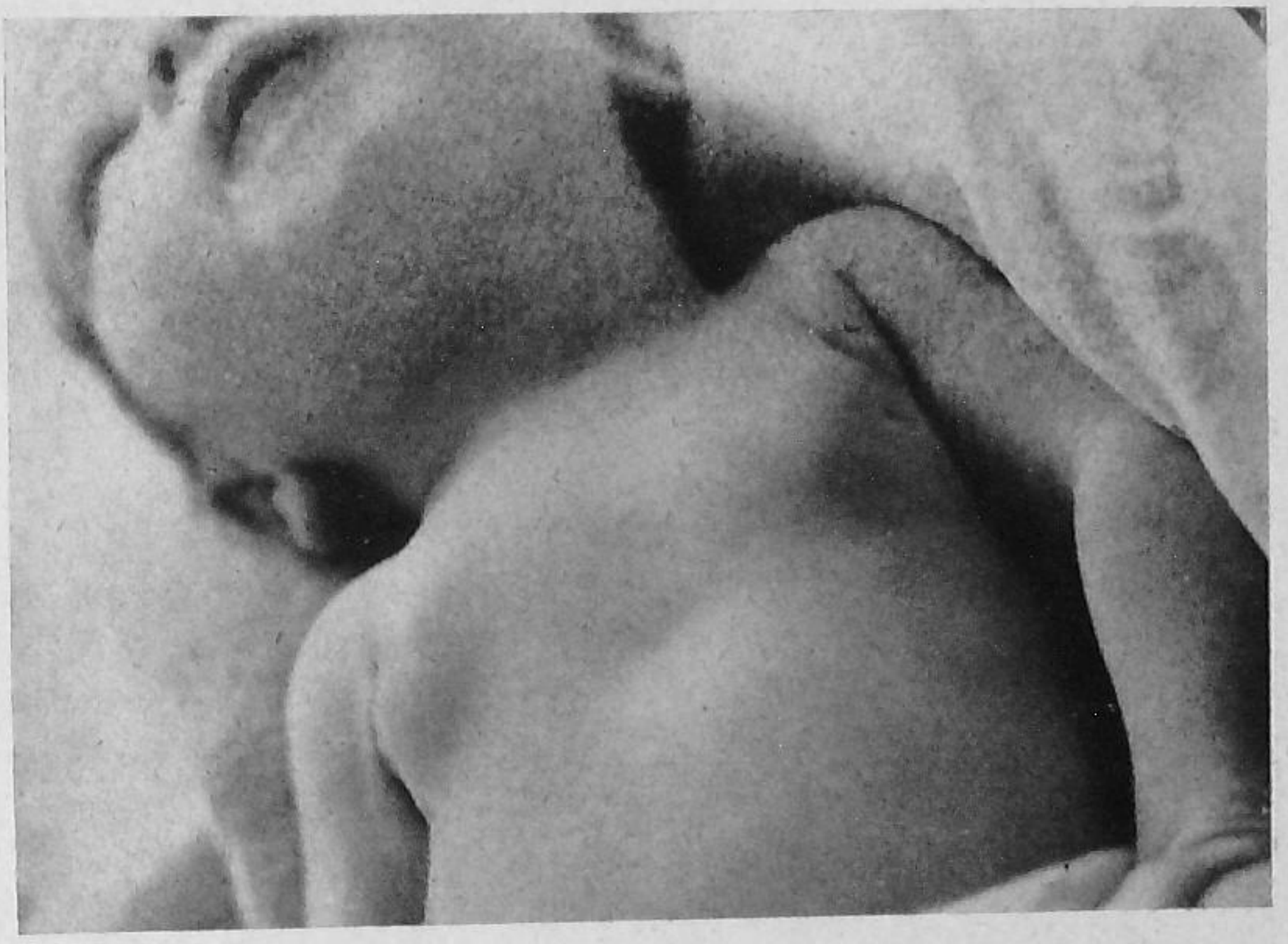

FIG. 4.—MASTITIS OF NEW-BORN. MALE INFANT, AGED 8 DAYS.

Sometimes when breathing has been satisfactorily established and all seems well there develop what have been termed **attacks of respiratory failure.** These may occur in babies who seem in every respect perfectly normal, and the attacks set in without any apparent exciting cause a few hours or, at most, a few weeks after birth. In an attack the breathing simply ceases suddenly and quietly, and the child is observed to be deadly pale or leaden-coloured. The heart goes on beating normally for some time after the attack has begun. Artificial respiration, if resorted to in time, will probably restart the breathing, but the attacks tend to recur at longer or shorter intervals, and sooner or later usually prove fatal.

The nature of the attacks is unknown, and there is no treatment for them except constantly watching the infant day and night so as to be able to start artificial respiration at any moment.

The existence of the stump of the umbilical cord is one of the most important facts in the anatomy of the newly-born child, and is a constant source of danger, as will be shown immediately. Meanwhile I would ask you to remember that it should fall off within five days, and that the raw surface left should be cicatrized by about eight days later. A single, red granulation sometimes persists and oozes serum; it should be touched with a crystal of copper sulphate on two or three occasions.

It may be worth while to remind you that **inflammation of the mammæ** is very common in children of both sexes in about the second week of life, and that sometimes it may go on to abscess formation (fig. 4). It is to be treated by simply covering the inflamed breast with cotton wool. Fomentations are to be avoided.

We may now pass on to consider some of the commoner diseases which affect the newly-born child, beginning with

INFECTIVE DISEASES

The commonest infections in the new-born period are the common infections at any time of life, so that we find the common cold, for example, and simple skin infections particularly prevalent. I shall discuss in a later lecture (see p. 53) the particular dangers of the common cold for the small baby, and here I want merely to call attention to certain other infections which, although rarer, are peculiar to the new-born period.

Diarrhœa in the new-born baby is particularly dangerous when it is the result of some infection spread round a nursery. Clearly, a high standard of hygiene in the preparation of foods, and in all utensils for giving drinks, is essential to prevent what may otherwise be a catastrophic outbreak. It has recently been suggested that sulphaguanidine is particularly valuable in the treatment of neonatal diarrhœa.

I have pointed out above that the existence of the umbilical wound is a constant menace to the health of the young infant, and it is through this channel that infection usually finds access. Its simplest form is a mere local inflammation (**omphalitis**), which causes a little redness and swelling, with slight purulent discharge,

but without any general symptoms. As a sequel to this **septic thrombosis** of the umbilical vessels may occur. This is most likely to happen in the case of premature infants, in whom closure of the vessels is apt to be slow and imperfect. From septic thrombosis it is but a step to **general septicæmia,** the supervention of which may be assumed when, in addition to umbilical sepsis there appear such symptoms as pyrexia, profound icterus, purpuric hæmorrhages, diarrhœa, and signs of inflammation of the internal organs or serous membranes. These symptoms, of course, are not necessarily all present in one case. In some even fever is absent, and in others the initial umbilical affection may be so slight as to escape detection. Icterus and hæmorrhages, when they occur, are the expression of a profound poisoning and destruction of the blood, and are very unfavourable signs. Of the internal inflammations, peritonitis and pleuro-pneumonia are the commonest.

Septicæmia in the newly-born is, as may be imagined, a very dangerous condition, and usually proves fatal in less than a week. It can only be treated symptomatically unless the offending bacteria can be identified, when the appropriate sulphonamide should be used.

Trismus neonatorum, as Cameron has pointed out, is almost certainly only a manifestation of sepsis affecting the brain and spinal cord and has nothing to do with true tetanus. It is accompanied always by some general muscular rigidity. If this view be correct, the use of the anti-tetanic serum is not indicated. It usually begins about the fifth day and is rarely seen after the second week.

In trismus, as in all the other umbilical infections, prevention is better than cure, and prophylaxis can only be secured by careful attention to the stump of the cord. It should therefore be enveloped from the outset in a dry sterile dressing, and if any signs of suppuration should appear after separation, local antisepsis should be vigorously carried out. It is only in this way that these fatal diseases can be prevented.

Of infections which find access to the body by another route than the umbilicus, I would mention **gonorrhœal septicæmia,** which may follow upon gonorrhœal conjunctivitis and manifests itself by synovitis, just like the so-called gonorrhœal rheumatism of adults. This I have seen more than once in newly-born babies. So far as I have observed, the prognosis is not unfavourable. Modern treatment by 'M. & B. 693' is stated to be eminently successful,

THE HÆMORRHAGIC DISEASE

Severe infections of any sort in the newly-born are apt to manifest themselves by hæmorrhages, chiefly of a purpuric character. They are in this respect directly comparable to the hæmorrhagic form of the acute specific fevers—e.g. hæmorrhagic smallpox—and have usually a fatal issue. Apart from these, there is a disease peculiar to newly-born infants, which manifests itself chiefly by a tendency to bleeding, and which, for want of a better name, is spoken of simply as the **hæmorrhagic disease.** Modern work has shown that it is associated with a drop in the prothrombin of the blood, but although this explains why bleeding continues once it starts because of a faulty coagulation mechanism, it still leaves unexplained why the bleeding starts at all.

The disease usually first manifests itself about the second day, and very rarely starts after the twelfth. As a rule, the first thing to be noticed is hæmorrhage from the alimentary canal. You must not expect, however, to find anything like a true hæmatemesis or melæna. The bleeding is more of an oozing than an actual hæmorrhage, and reveals itself by the vomiting of small quantities of brownish grumous-looking fluid, or by the presence of similar discharges on the napkins. Bleeding may also take place from the umbilicus, and may be profuse; and there may also be subcutaneous hæmorrhages, like those of purpura, or bleeding into the internal organs. The latter may be difficult to detect, unless so situated that they produce definite signs or symptoms. This took place in the following instances:

Case 1.—A boy, two days old, one of twins, seemed healthy at birth, but on the second day vomited a little brownish fluid. Soon after this he became collapsed and comatose, with a bulging fontanelle, contracted pupils, and rhythmical respiration. Hæmorrhage into the meninges was diagnosed, and puncture of the anterior fontanelle resulted in the escape of a good deal of blood. After this he brightened up a little (probably from the relief of pressure), but a few hours later slight convulsions set in, followed by death.

Case 2.—A boy, three days old, had been born at the full time by an easy forceps labour. He suffered from asphyxia after birth, and was only resuscitated by prolonged artificial respiration. He cried a great deal during the first two days, and then became rather collapsed. When I saw him he was deeply jaundiced, with a sunken fontanelle, but with no bleeding from the umbilicus or into the skin. That

morning he was said to have brought up a little blood from ' the throat.' The bowels had been opened with castor oil, and on the napkin there was a small tarry motion and a good deal of meconium. In the left side of the abdomen there was a dull swelling, difficult to define and not movable, which the doctor in attendance said had not been there that morning.

Death ensued a few hours later, and although a post-mortem could not be obtained, there can be little doubt that the swelling was due to a hæmorrhage into the neighbourhood of the kidney.

Apart from the hæmorrhages, the disease may run its course without any general symptoms, and prove fatal simply by a sort of general collapse. If the bleeding is entirely internal, or only from the alimentary canal, it may easily be overlooked, in which case the child may be believed to have died from ' convulsions,' or simply from ' prematurity.' There can be no doubt, indeed, that many cases really do pass unrecognized in this way, and that the disease causes more deaths than is properly realized.

The **prognosis** in the hæmorrhagic disease if untreated is always grave, and if the symptoms last for more than a day, is very bad indeed, although recovery *may* take place even in what seem the worst cases.

As regards **treatment,** vitamin K has proved of real value. An injection of 1 c.c. of one of the synthetic preparations, repeated after four hours, is generally all that is necessary. If the infant is very anæmic by the time this treatment is started, the question of a blood transfusion should be considered. Prevention is most satisfactory if the disease is anticipated, as for example, when there is a family history or in a premature infant. It is achieved by giving the mother some vitamin K before delivery of the child. Incidentally, an important practical point is that, as with all cases of internal hæmorrhage, an accurate pulse chart is a most important guide to the effectiveness of treatment. With a collapsed bleeding baby, the pulse can often only be counted at the heart apex beat.

There is no connection between the hæmorrhagic disease of the new-born and any form of bleeding in later life, so that when recovery does occur it is complete.

PEMPHIGUS NEONATORUM

It has been well remarked that pemphigus is not a disease, but a lesion, and in the newly-born child we can distinguish two varieties of it : (1) syphilitic, (2) non-syphilitic.

Syphilitic pemphigus is distinguished by the fact that the bullæ occur on the palms and soles, as well as on the trunk, whereas in non-syphilitic pemphigus the palms and soles escape.

Non-syphilitic pemphigus (fig. 5) is a coccal infection of the skin closely allied to contagious impetigo. In some cases at least it can be shown to be simply a cutaneous manifestation of general sepsis. Thus, of the cases which occurred at the London Hospital within ten years, three were associated with umbilical infection and two with sepsis consequent upon ritual circumcision. In institutions it may occur in the form of epidemics, which is another indication of its infective character.

Apart from the eruption, which may come out all at once or in successive crops, the general symptoms vary greatly. Sometimes there are practically none at all; at others there is high fever, prostration, and symptoms of general septicæmia.

The prognosis varies in accordance with the general symptoms, but in recent years has been less serious than formerly. It is always grave in syphilitic cases.

Treatment in the syphilitic cases is simple, and consists in push-

FIG. 5.—PEMPHIGUS.

ing mercury and salvarsan. In non-syphilitic cases the blisters should be snipped and the area dabbed with 4 per cent. silver nitrate in spirit. The surrounding skin should be treated by a non-irritant disinfectant solution such as Bonney's blue. The use of one of the sulphonamide preparations by mouth is claimed to be of value.

A special form of pemphigus is the condition known as **exfoliative dermatitis**—a rare disease sometimes met with in newly-born infants. The epidermis here peels off in great sheets, leaving the true skin denuded just as if it had been scalded, and producing a picture which, once seen, can never be forgotten. In spite of the extensive skin lesion, there may be few or no general symptoms, but sometimes there is high fever and prostration. I cannot tell you what the mortality rate is, as the disease is a rare one, but it is usually regarded as even more dangerous than the bulbous form. The treatment consists in the use of an antiseptic dusting powder and the use of a sulphonamide preparation by mouth.

BIRTH INJURIES

The infant is apt to suffer damage during delivery especially if the labour has been abnormal or protracted. I do not propose to deal with surgical injuries such as fractures or dislocations, and shall defer reference to injury of nerve trunks to a later lecture (p. 313), but I wish to refer now to injuries of the head—both extra and intra-cranial. Of the former **cephalhæmatoma** must be mentioned. It consists of an extravasation of blood between the periosteum and bone and forms a fluctuating swelling over the posterior part of a parietal which appears within a few days of birth. The swelling does not extend over the sutures and it soon develops a hard rim at its periphery. A cephalhæmatoma is best left alone and will be absorbed within three months. One should resist the temptation to aspirate owing to the risk of sepsis.

Intracranial injuries may be very serious in difficult labours and consists of hæmorrhages or even laceration of the brain or membranes. The symptoms are usually present immediately after birth, but are sometimes delayed for a few days. They are usually rather indefinite and such as may not at once suggest the nature of the trouble. Asphyxia, collapse, inability to suck and a tendency to convulsions are common. On the other hand, localizing signs are

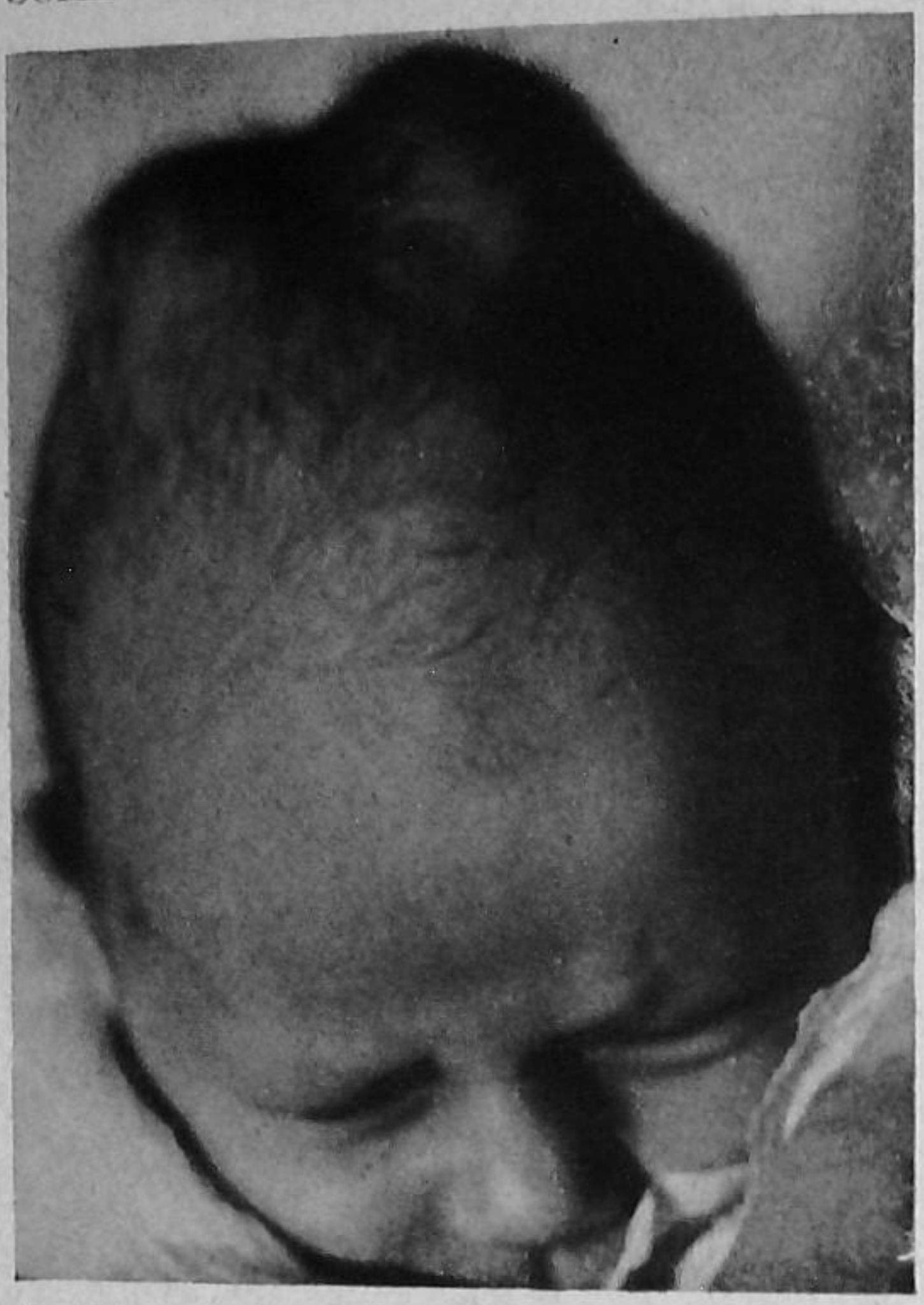

FIG. 6.—DOUBLE CEPHALHÆMATOMA IN NEW-BORN INFANT.

usually absent. It is easier, therefore, to suspect injury than to be sure about it. Prognosis, in any case, should be guarded, for although immediate recovery is likely enough if the child survives for a week, yet remote effects such as hydrocephalus, spastic paralysis, mental deficiency or fits may declare themselves later. Milder cases are probably due merely to congestion and œdema of the brain and respond well to treatment. This it must be admitted, is not very satisfactory, but if there is reason to suspect intracranial damage the infant should be kept as quiet as possible and not handled more than can be helped. Small doses of chloral may be given for restlessness. If the fontanelle is tense, rectal injections of 10 per cent. saline (1 teaspoon of salt to 2 ounces) repeated four hourly for three or four times, help in reducing cerebro-spinal pressure. Other indications must be met symptomatically.

I shall speak of the important subject of **convulsions** in the newly-born when I deal with convulsions in general (p. 296), and the different varieties of **jaundice** which occur at or shortly after birth will be described in a lecture on affections of the liver (p. 393).

LECTURE V

THE PREMATURE INFANT

I want to talk to you to-day about the care and management of the premature infant. It is a subject which is in danger of being neglected by the medical profession. These small and difficult infants are too often left to the undirected care of the nursing staff and it cannot be said that the results are altogether satisfactory. The mortality rate among the new-born is still as high as it was last century and half of the deaths in the first month of life are attributed to lack of maturity. We find a great deal of fuss being made—and quite rightly--because over 2,000 mothers die each year as a result of childbirth, but we find widespread ignorance of the fact that the number of infants dying in the first month of life runs into tens of thousands. Now the premature baby is essentially a good proposition and well repays study and trouble. I think that some of the neglect of our profession in relation to this subject is due to the common belief that by keeping premature babies alive we are likely to be rearing a lot of weaklings. This is quite untrue : except for certain special dangers (which I will deal with later under prognosis) the premature baby grows up into essentially a normal child.

Of course, everyone knows what we mean when we speak of a premature baby, but the moment you try and work out a definition —for example for statistical purposes—you will find certain difficulties. Therefore I think we may profitably first discuss

Clinical Features

The ' official ' *standard* of a premature baby is now expressed in terms of weight. We say that for the purposes of comparison of records we will class all babies of $5\frac{1}{2}$ pounds or less at birth as premature *or immature*. Now it may seem illogical to apply a weight standard to something which should be measured in time standards, but in actual practice it is found by obstetricians that

the mother's history as regards length of pregnancy is frequently unreliable. Moreover, however prematurely delivered a baby is alleged to be, if it weighs over $5\frac{1}{2}$ pounds it will probably behave as a mature baby. On the contrary a small baby born at full term —and this includes multiple births—may be very immature in all its functions. Another possible standard is one of length. The normal baby should be 20 inches in length at full term : before this the fœtal age in weeks is roughly twice the fœtal length in inches. The real difficulty about length is that it is a most unsatisfactory measurement to make in a small, wet and non-co-operative subject ! There are other ways of estimating the degree of prematurity such as X-ray examination of the lower end of the femur or estimation of the proportion of nucleated red cells in the blood. But I advise you to stick to the simple weight standard. Incidentally this is the standard generally used in continental literature (2,500 grammes), and also in America.

Apart from the question of size there are other features of the premature infant which are characteristic. The head seems big in proportion to the body, the vault of the skull seems big in proportion to the face, and the eyes and mouth seem big in proportion to the rest of the face. Subcutaneous fat is missing so that bony prominences appear exaggerated and the infant looks curiously shrivelled and wizened. The colour tends to be even redder than the normal new-born infant and nearly every premature baby becomes jaundiced. The skin is also covered with downy hair, known as lanugo and the nails often fail to reach to the end of the fingers.

The premature infant lies curled up on its back in the fœtal position. The baby is only roused with difficulty and has a feeble cry. Attacks of cyanosis occur frequently, especially after feeding. It is difficult to keep the infant warm and the temperature chart tends to be widely erratic. I have known such an infant (the child of a medical practitioner, of course) to swing through a range of ten degrees (107° F. to 97° F.) in a day without any obvious cause and no apparent harm came from it. Other features to be noted are the frequent absence of the testicles from the scrotum in boys, and the occurrence of œdema of legs and perineal region.

Prognosis

When an infant has been prematurely born the first thing that the parents naturally want to know is whether it is going to survive,

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and secondly, whether or not the baby will grow up normally if survival occurs. Now it is naturally not an easy matter to make an accurate forecast of the fate of a very small baby. Favourable factors are : weight—the bigger the baby the better the outlook ; a stable temperature ; an ability to take enough food ; steady daily progress ; and, as a point in obstetrics, what I may term a ' mechanical ' cause of the premature birth (e.g. a fall or fright on the part of the mother). Unfavourable factors are : a low weight ; persistent low temperature ; attacks of cyanosis ; lack of progress ; and a ' pathological ' cause for the premature labour such as a toxæmia in the mother. If you are asked what the chances of a small baby are, all you can do is to sum up the position along these lines and make an intelligent guess. In any case much will depend upon the success or otherwise of the general management and whether or not you can secure a first-class nurse or institutional facilities.

On the question of remote prognosis it can be asserted that apart from a very slightly increased risk—according to statistics—of mental defect and spastic paralysis among prematurely-born infants as compared with full-term infants, there is every prospect of the child growing up normally. Possibly prematurely born infants remain under the average as regards height and weight but this is by no means always the case and I have more than once seen huge, overweight children in an out-patient department who were alleged to have weighed incredibly small amounts at birth ! Premature babies are more likely to develop rickets and also anæmia, but these are both preventable diseases and with special care need never occur.

General Management

The most important factor in the management of a premature baby is the *nurse*. Such an infant requires a very special type of nursing, in which few nurses can, naturally, have had much experience. In most instances it is best to nurse the baby where the birth took place since moving a small infant to a hospital may mean exposure to changes of temperature and to infection. Nevertheless, unless the constant attention of a nurse in the home can be secured it will be better to move the infant, well wrapped up, to a small isolation room in a hospital or nursing home. The baby is nursed in an ordinary *cot or cradle*, well screened from draughts and kept warm by the use of hot water bottles or the most useful

electric blanket pads issued by 'Thermega.' The rectal temperature should be recorded every four hours and although in theory this should be normal, i.e. 99·4° F., in practice I think a nurse is doing well if she keeps the infant fairly steady between 97°–98° F. I do not like the incubator, as it tends to incubate bacteria as well as the baby and in any case will not be available in the ordinary house. The *temperature* of the room must be kept at about 70°–75° F. to start with and gradually reduced as the baby grows older. More important than the temperature is the *humidity* of the atmosphere in the room. Without some special source of water vapour a room kept at over 70° F. is apt to become very dry and premature babies are particularly prone to the effects of dehydration. So arrange that there shall be a bowl of water in front of the fireplace or a damp towel hanging over the radiator or a basin kept full of water all the time or steam a kettle into the room every hour for a few moments. Authorities who have studied the subject with elaborate air-conditioned rooms find that premature babies do best with a relatively high humidity (65 per cent. at 70° F.). You can keep a record of this if you like by using a wet and dry bulb thermometer or a simple device is a 'damp detector.' This is a pocket hygrometer, like a watch in shape, sold to keep commercial travellers from sleeping in damp beds! It crudely indicates 'wet' or 'dry' conditions. I often advise that one of these little gadgets be suspended at the head of the cot of a premature infant and steps taken to see that the hand on its dial always points to 'wet.'

After birth the premature baby must be kept most carefully covered to avoid exposure. It should never be bathed at birth. Careful cleaning with soap and water, exposing only a few square inches at a time can easily be carried out, followed by plenty of powder. I do not like olive oil as it is a most inefficient cleansing agent, is not always sterile and softens the skin at a time when, if anything, it needs hardening! The baby is usually clothed in gamgee—that is to say cotton wool in gauze—made into little jackets, bonnets, boots and gloves. I would only remind you, whilst approving of this costume, that it is highly inflammable. Another point is that from the very start one should plan to discard some of this extra clothing gradually. Similarly the temperature of the room should be gradually reduced. There is a tendency with premature babies to leave them too long in an over-coddled

state. You should start educating their skin and temperature-regulating function towards maturity right from the start.

A most important point in the management of these premature babies is the *avoidance of infection*. I want to say a special word about infection in young babies, with particular reference to premature infants. New-born babies have not, as is often supposed, a sort of general immunity to all diseases: they have only such immunity as the mother herself possesses. Now very few adults have any permanent immunity to the 'common cold' and therefore it is particularly this infection which is such a menace for the new-born baby. The clinical picture of the result of such infection is not at all like that in adults. The infant, lying on its back in bed, may have little or no anterior nasal discharge and may sneeze very seldom. The infection creeps down the back of the nose and the first evidence of the 'cold' may be a bronchitis or pneumonia, if the respiratory tract is involved, or green stools, if the alimentary tract is affected. When any of these disorders occur somewhat mysteriously in a young infant always suspect that someone in the immediate environment has infected them. Sometimes it is a sufferer from an ordinary 'cold' or sometimes a chronic sufferer from nasal sinus infection. If we apply these observations to the case of the premature infant it is obvious that great care must be taken to prevent adults with acute or chronic infections of the upper respiratory tract from contact with such infants. It is best to make everyone wear a simple mask when dealing with the premature infant for the earlier periods of life. After a week or so the restriction can be gradually relaxed as far as healthy contacts are concerned, although adults suffering from 'colds' must be barred for the early months.

Feeding

The main problem of management of the premature infant is concerned with feeding. The subject can be conveniently discussed under the three headings: how to feed the baby, how much to give, and what to give.

Methods.—The first step is to ascertain whether or not the premature baby can suck. Unless well over four pounds in weight it is unwise to put the small baby to the breast. A bottle and teat should be tried and you will find the 'Breck' feeder will be useful. This is merely a small narrow bottle like an enlarged fountain-pen

filler with a teat at one end where the baby sucks and a rubber bulb at the other, which can be gently squeezed to produce a little positive pressure in the bottle. With very small infants, say between two and three pounds or less, sucking will be poorly attempted or absent. In such cases fluid can be introduced into the mouth by means of a small spoon or dropped in by means of a pipette. Care must always be taken to see that such infants are able to swallow. The first fluid should consist of a few drops of sterile water, just in case it goes into the respiratory tract. If the swallowing reflex is absent or the infant is so feeble that exhaustion quickly follows attempts to swallow, recourse to tube feeding is necessary. A soft rubber catheter (No. 5) is passed into the œsophagus, either through the mouth or through the nose, and a suitable feed introduced. Whatever method of feeding has to be used it is important to make daily attempts to educate the infant towards a more advanced method. For example, the spoon-fed infant should be tried on a bottle daily and the bottle-fed baby should be tried at the breast. An attempt should be made to get the premature baby on to the breast by the time the weight has reached four pounds. I admit that this is seldom achieved because in the absence of the stimulus of the actively-sucking infant, the supply of breast-fed milk is apt to fall off and in any case the average mother of a premature baby is unwilling to remain in hospital or nursing home long enough to get breast-feeding properly established.

Quantities.—It is difficult to be exact when speaking in general terms because each premature baby is a special and individual problem. With very small infants it will be a matter of drops to start with, offering up to 30 minims every hour day and night. From this small beginning attempts are made to increase gradually to drachm doses until $\frac{1}{2}$ ounce is reached when the interval can be extended to every two hours. It may sound an easy process set out in this way, but it is in reality a difficult and tedious matter, calling for great skill on the part of the nurse. One of the difficulties is that for body weight the premature infant requires a relative higher intake than the larger infant and sometimes as much as 3 or 4 ounces of breast milk (or its equivalent) per pound body weight before an increase in weight is obtained. Thus a 3-pound baby may require almost as much in the twenty-four hours as the 5-pound baby and yet is infinitely harder to feed. After $\frac{1}{2}$ ounce feeds have been reached the worst is usually over, and the

stronger the baby becomes the larger the feeds taken. The amount can be gradually increased up to an ounce which can be given every three hours and further increases follow easily until the baby is gaining weight. Once the baby is strong enough to go to the breast, say at 4 pounds in weight, the amounts taken need no longer be accurately controlled. When tube feeding is used for very small infants it is best to give rather more at a time and make the intervals longer. A small baby, for example, is given three or four drachms every two hours through a tube at a period when it would have only taken one or two drachms every hour by a spoon had swallowing been possible.

Quality.—Two preliminary observations may be made as regards what food is suitable for the premature infant. First, every effort should be made to secure human milk to start with. Even if it is unlikely that lactation will be established in the premature baby's own mother, it is simpler and safer to secure human milk from some other source than to risk a digestive upset by starting with some form of artificial feeding. Even in the remote country district in which so many obstetrical emergencies appear to occur it should be possible to secure an ounce or so of breast-milk daily by a little organization. If brought to the boil breast-milk is rendered perfectly safe so that its 'source' need not be subject to rigid medical inspection. The second general observation is that if and when artificial feeding has to be started for the premature infant, even if it is very small and weakly, it is best to use some modification of cow's milk (very dilute, of course) rather than some of the curious concoctions of egg-yolk and olive oil which are still recommended!

For the first forty-eight hours the premature infant should be given half-strength normal saline ($\frac{1}{2}$ teaspoonful of salt to a pint of water) in quantities already outlined above. If the infant's mother has any colostrum which can be easily expressed it should be given in place of saline during this period. The breasts should be carefully massaged so as to help in establishing lactation. Steps may also be taken during this first two days of the infant's life to secure a supply of human milk as mentioned above. On the third day of life breast-milk should be given. In very small infants (under 2 pounds) a suitable dilution should be made with one part of milk to three parts of boiled water and this can be very cautiously strengthened. For infants between 2 and 4 pounds a beginning may be made with the same weak milk for one day, making it

equal parts of breast-milk and water for the fourth day of life and getting on to undiluted breast-milk by about the seventh to tenth day.

If it appears to be quite impossible to secure any breast-milk at all, what alternative should be used for the early days of life? There are several methods which can be adopted. Perhaps the most practical is to use unsweetened condensed milk and beginning with a dilution such as one part of the milk in ten of water for the average small baby. Very slow increases can be made until the standard dilutions of this type of milk as already described for young babies are reached (p. 26). Another method, suitable for very small infants, is to use ordinary liquid milk, boiled, diluted with four or six parts of water with added dextri-maltose (1 teaspoonful to every 3 ounces) and then predigested by peptonization for 30 minutes or even longer. The peptonizing time can be slowly reduced and the strength of milk mixture can be slowly increased towards the 'modified milk mixture' strength already described. A third method which is probably the simplest is to use a 'humanized' dried milk powder and begin with a very dilute strength such as one measure in 4 to 6 ounces, making it stronger very slowly. This is a thoroughly safe and useful method if it can be begun when the infant has already had several days of breast-milk. To sum up, I suggest that the ideal plan is as follows:

Give saline for two days. Then begin with diluted breast-milk. If possible get the mother's breast-milk supply going so that the infant can eventually be put to the breast. If this appears to be impossible or the supply of breast-milk gives out, get the infant gradually over on to a diluted unsweetened condensed milk or 'humanized' dried milk in a suitable dilution.

It will have been realized by now how complicated the feeding of the premature baby may be. The main difficulty is that three things are being done at once: increasing the size of the feeds, lengthening the intervals between feeds and strengthening the feeds. The great secret of success is to do all these things gradually but at the same time the whole process must be a progressive one.

'Treatment'

It is scarcely correct to speak of treatment in connection with the premature baby, but there are several points in the general care which remain to be discussed. One of the great troubles you will

have with these small babies is to get them to liven up. They tend to lie in a sort of moribund state with a subnormal temperature, being roused to take their feeds only with great difficulty. I always distrust the premature baby who is reported as being 'good.' This often means that the baby has not the vitality to be 'bad' if by bad is meant an occasional good hungry cry. The premature baby looks as if it needs some stimulus to wake it up. This can sometimes be achieved by the use of thyroid extract. For very small infants (under 3 pounds) you can give $\frac{1}{8}$ grain a day and over this weight $\frac{1}{4}$ grain a day for a week or so until a satisfactory feed is being taken. When the baby is older—a few weeks or so—gradual introduction to fresh air, the daily bath and a normal routine will help to establish a normal metabolism.

After the first month of life, cod-liver oil should be given to all premature infants and all artificially fed infants should receive some vitamin D concentrate in addition. Similarly an iron preparation (p. 366) should be given from about the third month onwards so as to prevent the development of anæmia.

LECTURE VI

DIET AFTER THE PERIOD OF INFANCY

The period of infancy may be taken as ending with the third year and thereafter the diet should conform more or less to that of an adult. A common mistake is to continue the infantile type of diet too long, an excess of sloppy food and especially of milk being given. The evil consequences of this in the way of producing digestive disorders, and especially ' biliousness ' in its various forms, should be recognized. In the present lecture I propose to consider with you the feeding of children after the period of infancy in more detail. I shall deal first with the scientific principles which should guide us in this matter and shall afterwards try to translate these into terms of practical dietetics.

As regards its scientific basis we have to consider (1) the total amount of energy (calories) which the diet should contain, and, (2) the amount of each nutritive constituent (protein, fat, etc.) to be supplied.

1. **The total energy** required in the child's diet is made up of various fractions. These are :

(*a*) The *basal requirement* or the amount of energy required to ' run the machine ' by supplying the essential needs of the circulation, respiration, digestion, and so forth. The amount of energy so required is fairly uniform for all children of the same weight. In other words, as regards its basal metabolism one child is not much more economical than another.

(*b*) The *growth requirement*. This fraction is very important and is what mainly distinguishes the energy-needs of children from those of adults. It is rather curious that growth demands not only a supply of building material but energy to make use of it. Indeed the amount of energy required for the purpose is very considerable, for it has been calculated that an intake of 80 calories per *day* is required to ensure a gain of 1 kilo of weight in a *year*.

It is obvious that the growth requirement will be greatest at the time when the body is developing most rapidly in height and weight. In boys this is between the ages of thirteen and seventeen—roughly coinciding with the public-school age; in girls the period of most rapid growth is about two years earlier, i.e. between eleven and fifteen.

(*c*) *Energy requirement.* This fraction represents the amount of food required to meet the demands of muscular energy. Obviously it will vary with the temperament of the child. In active children it will be great; in the lethargic, small. Some nervous, restless children expend so much in this way that it is impossible for them to become fat. As the mothers say, 'they live on their nerves.' Apart from this I think there is reason to believe that certain children, so far as muscular work is concerned, are more economical machines than others—they run more miles to the gallon as it were—but in all there is a constant competition between the demands of growth and those of the muscles. If growth is going on very rapidly the intake of food may not be sufficient to meet the needs of very active exertion as well and the child is then said to 'have outgrown his strength.' In these circumstances it may be necessary to curtail for a time the amount of exercise the child takes. Games or 'Gym' may have to be restricted and it is sometimes even advisable to prescribe an occasional day in bed. During complete rest in bed there is much more energy left over for growth and that is why children often gain so much in height during a long illness, especially if it is one that does not much interfere with the taking of food. There is, indeed, a real danger of over-exercising children during their years of most rapid growth and this may lead the doctor into antagonism with the schoolmaster, so that you must be prepared sometimes to exercise your authority and to insist upon the prior claims of growth.

The energy of the food, then, is used to 'run the machine' and to provide for growth as well as to meet the demands of muscular exertion in the form of games, and during the period of adolescence when the boy or girl is growing rapidly, and at the same time playing hard, the total intake required may amount to a very high figure. It has been calculated, indeed, that if the total daily caloric intake of a man doing a moderate amount of work is about 3,300 calories, that of a boy of about seventeen may amount to 4,100 calories, or if a woman needs 2,600 calories, a growing

active girl of fifteen will require about 3,300. Put in another way, three adolescent boys will eat as much as four full-grown men, and three girls as much as four women.

2. We have next to consider **the amount of each nutritive constituent which the diet should contain.** The nutritive constituents to be reckoned with are protein, fat, carbohydrate, mineral matter and vitamins.

Protein is the most important of these, for it is (along with mineral matter) the material out of which the body is built up and the demand for it during growth is therefore very great. It is calculated that the child requires about $2\frac{1}{2}$ grams of protein per kilo of body weight, half of which goes to growth and the rest to 'repair.' Now each gram of protein yields 4 calories of energy, so that put in another way 15 per cent. of the total intake of energy should be in the form of protein. In the case of the average boy of seventeen this will amount to about 150 grams ($4\frac{1}{2}$ ounces) of protein daily as compared with 100 grams for a fully grown man. A girl requires proportionately less.

Fat is used solely for the supply of heat and energy, and seeing that these are also supplied by carbohydrate, the question arises whether any fat in the diet is really necessary. This question is all the more important in view of the repugnance which many children exhibit to the taking of fat. There would seem to be no doubt, however, that fat cannot be entirely replaced by carbohydrate without detriment to health, for it has functions which the other nutritive constituents cannot fulfil. In the first place it is a better source of heat than carbohydrate and a shortage of it in the food is apt to lead to insufficient heat production. It is interesting in this connection that some children are cured of a tendency to chilblains by eating more fat. Fat seems also to have the power of increasing the power of resistance to certain infections—certainly to tuberculosis and perhaps also to pyogenic infections—and if it is insufficiently represented in the diet the liability to these forms of disease is increased. This was well seen during the fat shortage in the war of 1914–18.

There is some reason also to believe that fat is a 'nerve food' and that children who take plenty of it are less subject to functional nervous disturbances. Further, fat is a vehicle of some of the vitamins and it also favours the absorption and utilization of the calcium in the diet and so facilitates the formation of bone. For all

these reasons fat cannot be entirely replaced by carbohydrate and there must be added to them the consideration that an excessive consumption of carbohydrate, such as would be necessary if the great bulk of the energy required in the diet were taken in that form, is apt to result in the production of dyspepsia.

As to the optimum amount of fat which the diet of childhood should contain opinions vary, but it may be taken as about the same as the daily requirement of protein. The balance of the energy which is not supplied by the necessary intake of protein is derived from **carbohydrate** of which there will be needed nearly twice as much as of protein and fat taken together. In the case of a boy of seventeen this will amount to about 500 grams and will be sufficient to supply about one half of the total calories required daily.

Of the **mineral constituents** two alone are of importance—calcium and iron—and if the diet contains a reasonable proportion of milk, meat and green vegetables there is not likely to be a deficiency of either.

We have now to consider the **source from which each nutritive constituent should be derived.** In the case of **protein** we have the choice of animal or vegetable sources. It has been found, however, that the ' biological value ' of animal protein, that is to say its value as building material, is considerably higher than that of protein derived from vegetable sources and that to attain the same result a larger amount of vegetable protein must be consumed. In practice this means that it is difficult to ensure an adequate supply of building material on a basis of pure vegetarianism, and it is considered advisable by most physiologists that two-thirds of the protein in the child's diet should be ' first-class protein ' derived from animal foods, that is meat, milk or cheese.

Fats may also be obtained either from the animal or the vegetable kingdom, but in this country, where vegetable oils are not largely consumed, the greater part of the fat in the diet is, as a matter of fact, of animal origin, and the question resolves itself largely into one of butter *versus* margarine. As to this question I shall have something to say immediately.

In the case of **carbohydrates** there is no choice, for they are all obtained from vegetable products and the chief problem is, how much of them should be taken as starch and how much as sugar ? In modern times there has been a tendency for more and more of

the carbohydrate in the diet of children to be taken in the form of sugar, so that in America to-day sugar enters into the diet nearly as largely as starch. Now, there is no doubt that sugar is a valuable food in childhood and the craving that the young have for it is probably the sign of a physiological need. None the less it is not wise to allow sugar to replace starch too largely. To say nothing of its injurious effects upon the teeth and of the remoter danger of its favouring the development of diabetes, an excess of sugar certainly tends to produce dyspepsia and the efforts of the doctor will usually have to be directed to restricting it, though it is impossible to state in figures the exact amount which the diet should contain. It is certain, however, that the child himself will see to it that sugar is always adequately represented. There is no justification for the modern craze of stuffing the normal child with glucose.

We come next to the question of the **vitamins.** A great deal is said nowadays as to the importance of the vitamins in the diet of children and indeed to hear some people talk you would think that nothing else mattered in comparison. I am sure this is an exaggeration. No sensible person denies that vitamins are essential ingredients of a healthy diet, but I believe it to be true to say that if the diet is a suitable one in other respects the vitamins may safely be left to look after themselves.

There are, as you know, four chief vitamins—the anti-scorbutic (C), the anti-neuritic (B), and the two fat-soluble (A and D). As regards the two former, there is no reason to suspect a deficiency in the ordinary peace-time diet of children. Scurvy is almost never seen in childhood beyond the age of infancy and beri-beri is not seen in this country at all. Nor do I believe that there is any real clinical evidence that there are any ' non-specific ' effects due to a shortage rather than a total absence of these vitamins and resulting in vague disturbances of the general health as some experimentalists would have one believe. There is more to be said in favour of the importance of the fat-soluble vitamin (A) in the diet of childhood and adolescence, but there again it must be remembered that this vitamin, essential though it may be, is very widely diffused, being present not only in almost all animal fats but in green vegetables as well. It is not likely therefore that any reasonable diet will be deficient in it. As regards the anti-rachitic vitamin (D) it is more important in the diet of infancy than it is after the age

of three, and if the diet contains an ordinary amount of milk, eggs and butter there should be no risk of a shortage of it. From a practical point of view the vitamin question really resolves itself into the questions of white bread *versus* brown and of margarine *versus* butter. As to the first of these, there is no doubt that brown bread contains more of the anti-neuritic vitamin than white, and if a child had to live almost exclusively on bread it would be wise to choose brown. Where, however, as ought always to be the case, bread is only part of a mixed diet, the question is not of real importance and becomes one mainly of taste, though the balance of advantage is in favour of a bread made of flour derived from 80 per cent. of the wheat-berry.

As to the butter *versus* margarine controversy, it may be said that the caloric value of the two fats is practically equal, but the vitamin content (A and D) may vary. As a matter of fact winter butter is relatively poor in vitamins and now that most, if not all, brands of margarine contain added vitamins, it is often better to use a vitaminized margarine than winter butter.

Now a few words on the translation of all this into practical terms. The first statement one may make in expressing these scientific principles in practical requirements is, that the diet for the child must be an abundant one. That follows from the large demands that are made at this period for energy. Secondly, it must be a mixed diet containing some animal food, because of the difficulty of getting sufficient of the proper kind of protein from purely vegetable sources. In other words, there should be two meals containing animal food a day. Also there should be an adequate representation of milk, for milk appears to have a peculiar power of promoting growth. At the same time, as I pointed out before, one must not overdo the giving of milk after the period of infancy ; one pint a day in all forms should be quite a sufficient allowance in most cases. Further, the diet must contain fat, because fat cannot be replaced by starch. There is always the danger that, owing to the cheapness of starch, fat will be elbowed out of the dietary by it in the case of the poorer-class children. If the food fulfils these conditions, and if you add a proper representation of green vegetables and fresh fruit, then I think that the other constituents, that is to say, the minerals and the vitamins, may be left to look after themselves. Modern authorities point out the value of fish and it is a good plan to include this once a week.

[redacted]

LECTURE VII

THE DIGESTIVE DISORDERS OF INFANCY—COLIC AND VOMITING

The symptoms of digestive disorder in infancy are both local and general. The local symptoms are of three sorts—colic, vomiting, and diarrhœa—but I need hardly say that these different symptoms, although one describes them separately, very commonly coexist in the same case. The second or general group of symptoms may be comprised in the one term 'wasting.' To-day I shall deal only with the local symptoms. The general symptom—wasting—may be more conveniently deferred until we speak of marasmus as a special disease, digestive disorders being only one of many causes which may lead to it.

COLIC

Let us begin then with that very common disorder, griping, or colic. I think one can distinguish three causes as giving rise to the painful contraction of the intestines which we call colic. The first is acidity due to fermentation occurring in the milk, and producing lactic and other acids, which irritate the bowel; the second is the presence of undigested casein or curds in the intestine; the third is gas in the bowel. When the colic is of recent origin and the infant or young child has become rapidly ill, the possibility of some surgical condition (e.g. intussusception) must never be forgotten.

How are you to recognize colic? Its chief symptom is pain, and whenever you find constant and frequent **screaming** it should always make you think of the possibility of colic, for it is one of the commonest causes of persistent screaming.

There are, however, other causes for screaming which may lead you into error. One of these is *hunger*. Some infants, even although they are gaining weight, seem to feel dissatisfied unless they have something fairly solid in the stomach, and they express their dissatisfaction by persistent screaming. I remember such a case in which the neighbours thought the child was being ill-used

because of its continual lamentations, and complained to the Society for the Prevention of Cruelty to Children, but the addition of a little Benger's Food to the bottles instantly restored domestic peace. Another cause is *earache*. It is often difficult to be sure of the existence of earache in young infants; but in such a case the mother will probably tell you that the child has been in the habit of putting his hand to the ear or head, and this may indicate to you the seat of pain. Yet another cause of persistent screaming is *teething*—especially the cutting of the eye-teeth. Teething and earache seem often to go together.

Another cause, and one which is commoner than you think, just as teething is less common than you are apt to think, is *renal colic*. It is, perhaps, surprising to you to be told that little children suffer from renal colic at all, but the probability is that they suffer from it as frequently as grown-up persons do. Of course, I do not mean to say that children pass stones, but they do pass gravel. You may have seen in the post-mortem room the kidneys of quite young infants which contained uric acid infarcts, and even in the fœtus *in utero* you may find them. Screaming may also be due to *phimosis*, which gives rise to straining on passing water; and the pain which that entails, the result of what one may perhaps speak of as a colic of the bladder, expresses itself in screaming.

Another group of causes which you have to think of when a child is constantly screaming is *tenderness of the bones*, resulting from scurvy, or possibly from rickets in its acuter forms, or from congenital syphilis. In that case the screaming will be more noticeable when the child is handled, or when he is put into his bath, and that, along with a careful examination of the bones, will enable you to establish or exclude this cause. There remains one other cause of screaming which you ought to think of when you have excluded every other possibility, and that is *mental deficiency*. One of the signs of defective cerebral development in young infants is screaming without apparent reason. You will, therefore, bear in mind those possible causes before you conclude too hastily that the child is screaming because of colic. But you must also remember that colic is commoner than all these other causes put together except, perhaps, hunger.

The **signs by which you recognize colic** are, in the first place, hardness of the abdomen. You put your hand on the child's abdomen, and feel that it is unusually resistant; indeed, it may be

almost knotty. You may even feel little coils of firmly contracted bowel standing out. And you will observe, in the second place, that as the child screams he tends to draw up his legs. The screaming may even be so extreme that the child passes into a condition of convulsion, for there can be no doubt that the irritation caused by undigested food in the intestines may be an exciting cause of convulsions in young children. Another sign by which you can be quite certain that the pain is due to colic is the cessation of the screaming on the passage of flatus. If that happens you may be sure that colic was the cause.

Those being the signs and symptoms by which you recognize colic, we now come to consider **how you are to treat it.** I shall describe first of all what ought to be the immediate treatment of an attack of colic, and then I shall ask you to consider by what means you may prevent the attacks coming back. The first thing to be done in an attack is to apply warmth to the abdomen. You can do that by friction with warm oil, by warm fomentations, or by poultices of linseed or mustard; and in very extreme cases it may even be necessary to use the mustard bath. In addition to the external application of warmth you can apply it internally with advantage by means of an enema of warm water. Inject 2 or 3 ounces of water, as hot as the child will bear it, well up into the large intestine. In addition it is right to give carminatives by the mouth to aid in the expulsion of flatus; peppermint-water or dill-water are suitable for the purpose. Some prefer to give small doses of sweet spirit of nitre, say 10 drops.

By means such as these you will generally succeed in cutting short an attack of colic, and then you have to consider how you are to prevent its recurrence. One may divide cases for this purpose into two groups: first, those which are being fed by the breast; and, secondly, those which are being fed by the bottle. If a breast-fed child is suffering from colic you will probably find on inquiry that it is being fed too often or at irregular intervals. All that you require to do in such a case is to regulate the feeding—to feed every three hours instead of every two hours, or every four hours instead of every three, as the case may be. The tendency to overfeeding in those cases is a natural one, because the taking of warm milk into the stomach, just as the injection of warm water into the rectum, temporarily relieves the colic, and the mother finding the child is relieved by suckling, tends to go on giving the breast too

often, and so a vicious circle is set up ; and what you have to do is to break that vicious circle by seeing that the child is fed regularly by the clock, and not at irregular intervals.

In some cases the colic of breast-fed children seems to be due to excessive greediness, so that the first part of the feed is taken too quickly, or there may be a lot of air-swallowing. Some boiled water before the feed and care in getting up the ' wind ' will usually put things right. Further, you will remember to correct any constipation, which is so common in breast-fed children—a most important point, and one which I shall deal with in another lecture. Just occasionally there may be something wrong in the maternal diet or the mother may be taking a purgative.

If the colic still persists, the question will arise, Are you to wean the child or not ? Here I would remind you of what I said in a previous lecture, that mere digestive disorder is by itself no necessary indication for weaning ; it is only if the digestive disorder is accompanied by a persistent and steady loss of weight that you are justified in taking that step.

We will pass now to the commoner state of things which you have to deal with—namely, the prevention of colic in an infant who is being fed by the bottle. One great cause of **colic in bottle-fed babies** is a want of cleanliness in preparing the milk ; and your first duty is to see that the child has a proper and clean bottle ; and, secondly, just as in the case of breast-fed children, see that the feeds are regulated, and not given too often. Your third care will be to see that the mixture which the child gets is sufficiently digestible. You may therefore have to give a mixture which contains less casein, or in which the casein has been rendered more digestible in one of the ways to be described shortly. You may also require, as in the case of breast-fed children, to correct constipation ; but more commonly you will have to deal with diarrhœa, and the best ways of treating that we shall consider when we speak of diarrhœa as a symptom of digestive disorder.

In all cases of colic, both in breast-fed and bottle-fed children, you will find the administration of a **carminative** between the feeds of help. A carminative mixture for a young infant should contain certain definite ingredients. First of all, it should be alkaline, and therefore you use bicarbonate of soda ($2\frac{1}{2}$ grains) as the basis. It should be alkaline, because colic is so often due to acidity in the intestine. You supplement this with an aromatic

ingredient which is also alkaline; for instance, aromatic spirits of ammonia, $2\frac{1}{2}$ minims. Secondly, to make this pleasant, you will add a little glycerin (2 minims). It is always advisable if you can to make medicines pleasant for infants, and one of the best ways of doing this is to add glycerin. Syrupy preparations are less suitable, because they are apt to ferment, and as you have already got fermentation going on you do not want to do anything to increase it. Thirdly, you make the mixture up to a teaspoonful with peppermint or some other carminative water. This should be given between the feeds, and it will be found useful in all cases. In addition, you should see that a child who suffers from colic is kept warm. A common, but often unsuspected, cause of the disorder is chilling of the abdomen. Inquire, therefore, into the covering of the child's abdomen, and insist upon his having a broad flannel binder, even though he be beyond the age at which binders are used, and see that the binder does not slip up over the chest and leave the abdomen unprotected. See also that the feet and legs are kept warm, for cold feet are a very common cause of colic in young babies. The child should have warm socks, and, if necessary, a hot bottle to the feet.

If, in spite of all you do, the colic persists, and if at the same time the infant is gaining weight (which sometimes does happen), so that you are not justified in making any great alteration in the feeding, you may perhaps require to have recourse to some form of sedative. You should not give opium any oftener than you can help, but sometimes you are driven to use it owing to the persistent pain which the child suffers. The best preparation to use is codein, $\frac{1}{40}$ or $\frac{1}{30}$ grain, which, with glycerin and some carminative water, makes a very suitable opiate for a young child. A milder remedy, sufficient in some infants, is to order chloral, 1 grain before each feed.

VOMITING

I now pass on to the consideration of *vomiting* as a sign of dyspepsia in young infants. There are two varieties of vomiting which you will learn to recognize when you get into practice as signs of digestive disorder. There is (1) *acute vomiting* which is usually the result of an attack of gastric catarrh; and there is (2) *chronic vomiting*, which may be the result of chronic gastric catarrh, but is very commonly due merely to unsuitable feeding.

You will have no difficulty in recognizing the **acute type.** The child will be brought to you with the history that he began to vomit a few hours before. You will find that the skin is hot, the tongue thickly furred, and that there is a sour smell about the breath. The only difficulty in your diagnosis will be the exclusion of more serious disease. Pneumonia and many of the acute specific fevers in children often begin with vomiting; so do many intracranial diseases, and you may at first be doubtful whether you are not dealing with some such case, particularly as acute gastric catarrh is often accompanied by a high degree of pyrexia. The possibility of intestinal obstruction has also to be remembered. You may do wisely, therefore, to reserve your diagnosis at first.

The proper treatment of acute vomiting is by **starvation.** You need never be afraid to starve young children in the matter of solid food. It is said that infants bear starvation badly. That may be true of chronic underfeeding, but babies will stand the complete withdrawal of all nourishment for two or three, or even more, days without any disadvantage, and, indeed, often with great benefit, provided always that you fulfil two conditions. The first of these is that the child is kept warm. Children stand starvation badly simply because they lose so much heat from their surfaces, a fact which is true of all young animals. And the second condition is that water must on no account be withheld. Children stand the withdrawal of fluid very badly indeed. So when I say that they are to be starved it means that they are to be given nothing which can be described as nourishment, but they must have abundant liquid; and in such cases as we speak of that may take the form of boiled water, or, if you like, and in order to satisfy the mother, one may administer a thin decoction of barley or weak tea. The liquid, whatever it may be, should be given cold with a teaspoon.

You will find, also, great help in these cases from **washing out the stomach.** This is the first time I have had occasion to mention washing out of the stomach as a therapeutic measure in children. I can only say that you will find it of the very greatest use in all cases, acute or chronic, whether in breast-fed or bottle-fed infants, where vomiting is a prominent symptom. The method is so simple, it is so safe, and, compared with a similar proceeding in adults, so little disagreeable to the patient, that you need have no hesitation in employing it. You should use for the purpose a small-size red rubber œsophageal tube. The œsophagus in a baby is

bigger than you might think; it is certainly bigger than an ordinary pencil, and you will be able to get the smallest size of tube down it without difficulty. It is an advantage to use a tube which is rather large than otherwise, because the tendency of a small tube is to permit regurgitation of fluid alongside, which leads to coughing and choking, and to struggling on the part of the child. The œsophageal tube is connected by means of a piece of glass tubing to another piece of soft rubber tube, and that to a funnel such as I show you, which is really nothing more than the barrel part of a glass syringe from which the piston has been removed. A funnel of this shape has the advantage that you can blow down it if the eyelet of the œsophageal tube gets blocked with a mass of curd. The best fluid to use is normal saline and it can be used quite hot as it always tends to cool as it passes down. The chloride present will help to replace that lost by vomiting, and therefore having completed the washing, you will find it an advantage to leave an ounce or two of fluid behind, for this also helps to fulfil the condition which I have laid down, that the child must be kept supplied with a large quantity of liquid when he is being starved. The hot water also acts as a kind of internal poultice, and is soothing. After this manipulation a child will often go quietly off to sleep, though he may have been screaming for some hours before the washing out was done.

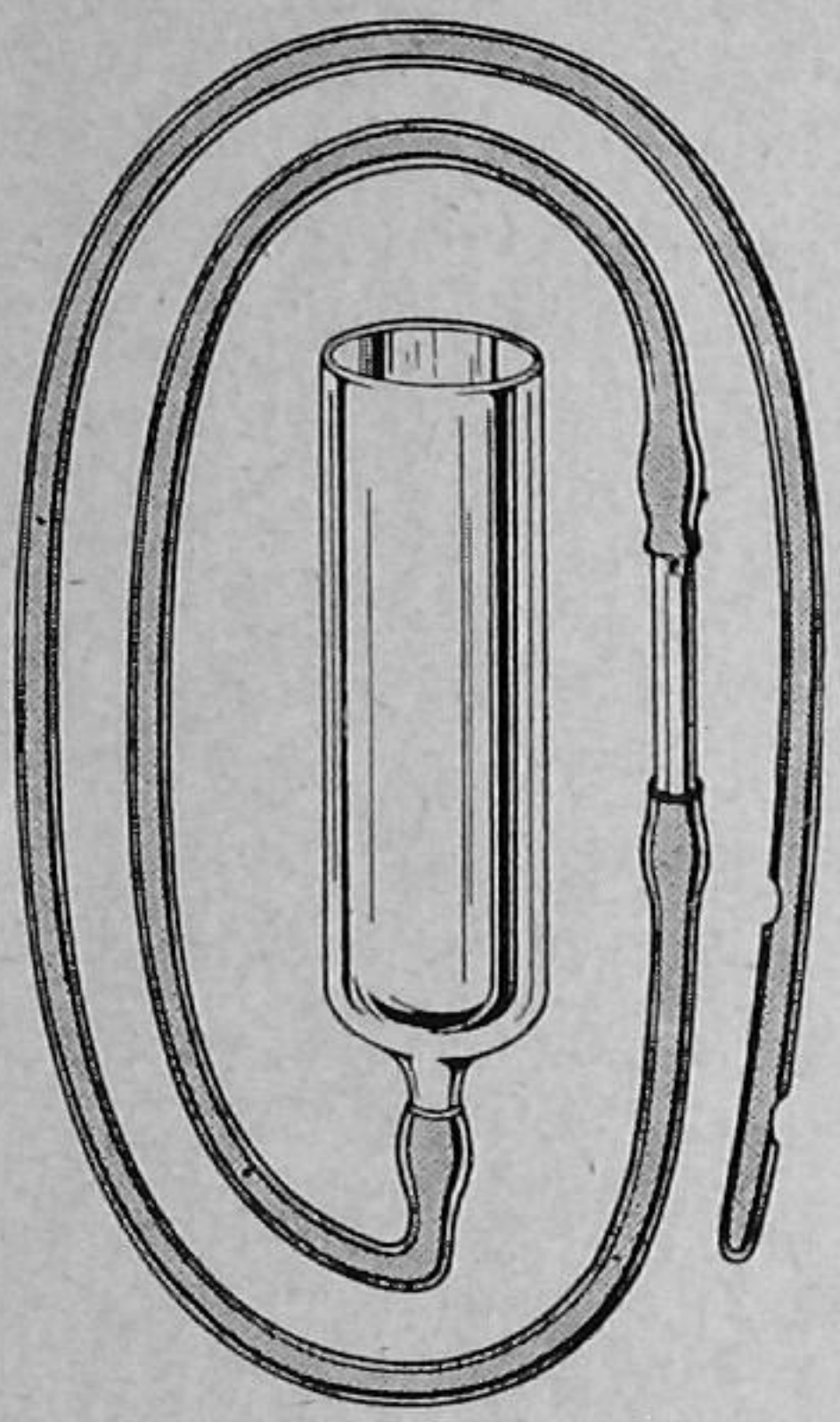

FIG. 7.—STOMACH-WASHING APPARATUS.

You will not find **drugs** of much assistance **in acute vomiting.** If you wish to have recourse to them, one would recommend especially small repeated doses of calomel, particularly if the tongue is much furred; $\frac{1}{10}$ to $\frac{1}{5}$ grain every hour for half a dozen doses is a suitable quantity for an infant, and that may be followed by the use of bismuth, either the carbonate or the subnitrate. Be sure always to give bismuth in large doses; that is to say, never less than 5

grains, and even in a young infant you can give more than that with advantage.

The **chronic vomiting** which is the result of digestive disorder is of quite a different nature to that I have been describing; and I would make this first remark about chronic vomiting in young infants, *that it is of no importance unless it is accompanied by progressive loss of weight.* You are not to get anxious, as nurses and mothers do, because a child vomits after his feeds. Many children, like the Romans of old, have a way of swallowing more than they want and rejecting the excess, and that is not a habit which need alarm you. Indeed, there is a saying amongst the nurses that 'sick babies always thrive.' It is only when the vomiting is accompanied by progressive loss of weight that you should take it seriously. The second remark I would make is, that if you are dealing with quite a young infant—that is to say, one a few days, or at most a few weeks old—and if the vomiting has dated from birth, you should make certain if you can that you are not dealing with a case of **congenital pyloric obstruction.** That, however, is a subject of so much importance that I shall devote a special lecture to its consideration.

There is a rare form of **habitual vomiting** met with in nervous infants which cannot be ascribed to any error of feeding or digestion. It usually occurs just after a meal and some degree of visible peristalsis may be seen, though no pyloric tumour can be felt. The infant is usually very restless and cries a great deal. The best treatment in such a case is to thicken the feeds with flour or fine oatmeal. About half an ounce of flour may be added to a pint of milk mixture, and the whole brought to the boil and allowed to simmer for ten minutes. This should produce a thin gruel which will flow through a large hole in the teat. The application of a chin-strap immediately after each feed is also of help in these cases.

The **treatment of chronic vomiting** depends to a large extent on the time at which it occurs. If the vomiting takes place immediately after a feed the indication is to reduce the size of the feed or to prolong the intervals. If, on the other hand, the vomit occurs half an hour or more after feeding and consists of masses of curd, one must give a more digestible mixture. Drugs are not of much assistance in the treatment of chronic vomiting, but if the vomited matter is very sour it is often an advantage to give 20 minims of milk of magnesia just before each feed. If this tends to open the

bowels too much, a few drops of saccharated solution of lime may be substituted. Bismuth is not of as much use as in the vomiting of adults, but if given in large doses immediately before the feed it is sometimes successful. In habitual vomiting regular washing out of the stomach is a method of treatment always worth trying.

MORE DIGESTIBLE MILK MIXTURES

In setting out the ways in which cow's milk can be used in the artificial feeding of infants (Lecture III) it was pointed out that *dried milk* offered certain advantages over liquid milk. Further, the half-cream variety of dried milk is more easily digestible than the full cream brands. There are also skimmed varieties of dried milk available with little or no fat, which are valuable when there is severe intolerance of fat. The *citration* of milk has already been mentioned and it was pointed out that the digestibility of boiled milk was very little improved by the addition of citrate. However, there is a type of baby who persistently vomits large curds and for such the addition of citrate may help: 1 grain of citrate of soda should be added to each ounce of a milk mixture.

Condensed milk is certainly more easily digested than ordinary cow's milk, even when the latter is well diluted. For infants with dyspepsia a useful temporary feed is a full-cream sweetened condensed milk (e.g. Nestlé's), and if 2 average teaspoonfuls [1] of such milk are added to 3 ounces of water one gets a mixture of approximately the following composition:

Protein	1·1	per cent.
Fat	1·2	,,
Sugar	6·7	,,

You will observe that the protein and fat percentage is rather low; that of sugar relatively high. It is probably the low proportion of protein and fat that makes such a preparation so easily digested. For the same reason, however, the continued use of condensed milk is apt to produce rickets, and it should not be used for more than a few weeks. For young and delicate infants, however, you will find it very useful, but it is advisable to increase the proportion of fat by giving some cod-liver oil emulsion at the same

[1] The spoon should not be dipped in the tin, but the milk poured into the spoon.

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time, or by adding a teaspoonful of cream to each feed, although in many ways I prefer the oil.

Peptonized milk is best prepared by using Fairchild's 'Peptogenic Milk Powder.' You will find that the directions supplied with it recommend that 10 ounces of milk should be diluted with an equal quantity of water, one 'measure' of the powder added, and the solution 'peptonized' for ten minutes. You are also told that cream should be added. I think it is better to omit the cream and to peptonize for a longer time. Indeed, I often allow the mixture to stand for as long as forty-five minutes before bringing it to the boil. You may also vary the proportions of milk and water, using, for example, eight of milk to twelve of water in very delicate infants, the essential being that the milk and water should together make up 20 ounces (1 pint).

Another useful method for infants with weak digestion is feeding on a *whey basis*. Suppose that even the most digestible of the above, peptonized milk, fails to agree, what are you to do? You are then up against one of those very difficult but fortunately rare cases in which there appears to be an inability on the part of the child to digest cow's milk in almost any form. In such an event I would advise you to make whey the basis of your feeding. Whey may be bought ready made at some dairies, or you may prepare it from milk by the use of rennet in the ordinary way, remembering to bring it to the boil to stop the action of the ferment before using it as a food. Dried whey powder can also be obtained. Whey has the following composition:

Protein	0·8	per cent.
Fat	0·2	,,
Sugar	4·6	,,

from which you will see that it is too weak to form a sufficient food by itself for anything but short periods. You must therefore enrich it, and this can be done by the addition of one of the pancreatized foods, such as Benger's or Savory and Moore's, or of a dextrinized food such as Mellin's. These foods may be prepared according to the directions on the tin,[1] but using whey instead of milk and water, and it is advisable in the case of the pancreatized foods to allow the mix-

[1] Details as to the preparation of such a food will be found on p. 122, where some further points in the feeding of delicate and wasting infants are discussed. Whey Powder ('Seeway') is supplied by Trufood Ltd.

ture to stand a good deal longer than is recommended before boiling it up, in order that the conversion of the starch may take place more thoroughly. The use of whey with one of these foods is often quite satisfactory, and as the child gets older and the digestive capacity increases you may gradually replace the whey by milk, beginning with only a teaspoonful or two of milk in each bottle, and gradually feeling your way until all the whey is replaced by milk.

Finally it should be remembered that *human milk* is the most digestible food for the human infant, and nowadays it is possible to obtain this from maternity hospitals in some instances.

LECTURE VIII

CONGENITAL PYLORIC STENOSIS

Congenital hypertrophy of the pylorus, or congenital hypertrophic stenosis, as it is sometimes termed, the subject of this lecture, is a condition which is probably very much commoner than is generally believed, and I am perfectly sure that anyone who is capable of diagnosing it will meet with cases in his practice not very infrequently. Inasmuch as the treatment of this condition, if it is to be successful, must be begun early, it is extremely important that you should be able to diagnose it when you see it, and that is why I am devoting a special lecture to it.

I shall not trouble you with the history of our knowledge of congenital pyloric stenosis, but shall begin at once with the **symptoms,** which, fortunately, are few and definite. The kind of case which exhibits congenital pyloric stenosis is this: The child is, usually—indeed, I may say four times out of five—a boy, often first in the family, and he is brought to you when a few weeks old because the mother says she cannot find any food to suit him. She will complain that, no matter what food she tries, it produces vomiting. You will usually find that a great many different foods have been tried—many of the patients, indeed, have run the whole gamut of the patent infant foods in the market—and yet the vomiting goes on. There is a very curious fact about the vomiting, and it is this: that when a new food is tried it will often seem to suit the child for a period of about twenty-four hours, or a little longer, and then the vomiting begins again, and will go on every day until a new food is tried, when it will again often cease for a day or two, only to recommence once more. At each of these occurrences the hopes of the mother are buoyed up; she believes she had at last found the kind of food that is going to suit; but they are immediately dashed to the ground again when the vomiting reasserts itself. Why it is that the new food is often borne for a time in these cases I cannot tell you; it is one of the many curious clinical facts that characterize this condition.

On careful inquiry, you will usually find that the vomiting has not strictly dated from the moment of birth. As a rule, you will be told that the child was born a fine, healthy child ; that he was fed from the breast at first, and all went well for a week, ten days, or a fortnight, but then vomiting began, and the child was taken off the breast, very often by medical advice, because of the continuous sickness, and put on some artificial food. As that was also followed by vomiting, another artificial food was tried, and so on.

I want you to observe—because it has some bearing upon the view that one takes of the pathology of the condition—that the vomiting is not, strictly speaking, congenital ; it is more usual for it to begin ten days or a fortnight after birth. In addition, the vomiting has certain peculiarities which distinguish it from the ordinary vomiting of dyspepsia. In the first place, it is often extremely violent, or what is termed ' projectile '—that is to say, the child will suddenly throw out the stomach contents forcibly, often projecting them quite a long way from itself and partly through the nose. The vomiting is also characterized by its abundance, and the mother will tell you that the child brings up much more food than it swallowed at its last feed. Further, you will often be told that the child brings up a quantity of ' slime ' with the vomit. Slime is what you and I know as mucus, and the bringing up of a quantity of mucus is another of the things which mark this particular kind of vomiting. Sometimes the mucus is streaked with blood.

Another prominent symptom that the child will exhibit besides the characteristic vomiting is *progressive loss of weight*. That, of course, is natural. The loss of weight may proceed until it reaches a very serious degree ; such children may waste away until they are literally mere skin and bone.

The third thing which characterizes a case of pyloric stenosis is that the child is usually—indeed, almost invariably—*constipated*. That is a sign of very great importance, because if a child is vomiting from unsuitability of food, instead of being constipated, it usually has diarrhœa ; and a combination of vomiting *plus* constipation should always arouse in your minds strong suspicions that you are dealing with a case of this sort. The reason for the constipation in this case is plain. If no food, or very little food, is entering the intestine, obviously there must be very little in the way of excreta

to go out of the intestine, and the constipation, therefore, is what is sometimes called the 'constipation of starvation.' It is the inevitable result of the small amount of nutriment which is finding its way through the pylorus into the bowel.

When you come to examine the child for **physical signs,** you will be struck at once by the *emaciation.* Observe that, although these children are emaciated, they are not cachectic, and that is a point worth noting. They show the thinness of starvation, but not the thinness of cachexia. In other words, although thin, often miserably so, they are not anæmic, and you know that it is a combination of emaciation with anæmia which makes up cachexia.

On examining the abdomen, you will usually find that there is a certain amount of fullness in the upper part above the umbilicus, whereas the rest of the abdomen is sunken. And then, having your suspicions aroused, you should look for what is, after all, the most characteristic sign of the condition—*the thickened pylorus.* You can feel it usually to the right of the umbilicus. Observe that sometimes the pylorus cannot be felt, even in undoubted cases, for the simple reason that it lies tucked up under the liver where you cannot get at it. But if you wish to give yourself every chance in feeling for it, there is a particular way of setting about it. You should sit at the left side of the child and palpate with your left hand (warmed !) in the right hypochondrium. You then 'hook' the middle finger round the right rectus, as it were, and you will feel a small hard knob—'like the end of the nose felt through a blanket' as someone once described it. It sometimes appears to harden and relax under your finger. It is best felt when the stomach is empty, e.g. just after a vomit.

In addition to the emaciation and the palpable tumour, you will find in most of these cases *visible stomach peristalsis.* If you are going to succeed in seeing visible stomach peristalsis, you must look for it when the stomach is full. There is no use in examining a child for gastric peristalsis shortly after it has been sick ; if you wish to make it out, you should always insist upon the child being fed in your presence, and immediately it is taking its food you should feel for the tumour. Then as the stomach fills, look for the peristalsis which usually assumes a very characteristic form. You will see appear below the left costal margin a swelling about the size of a golf-ball, which will begin to travel downwards to the right, sweeping above, or even over, the umbilicus, and which, before it has dis-

appeared, is followed by another swelling, so that a dumb-bell or hour-glass-like appearance is produced over the upper part of the abdomen. Sometimes in extreme cases you will see three such swellings—or, in other words, three waves of peristalsis—simultaneously visible over the stomach. Such peristalsis, *if it is well marked*, is absolutely pathognomonic of the condition, and sometimes it is extremely well marked. I have seen it so well marked that the swelling of the stomach could be seen through the child's clothing. Unless it is well marked you should not attach importance to it, because *slight* gastric peristalsis may be visible as a purely transitory phenomenon in circumstances other than those in which there is an actual constriction of the pylorus.

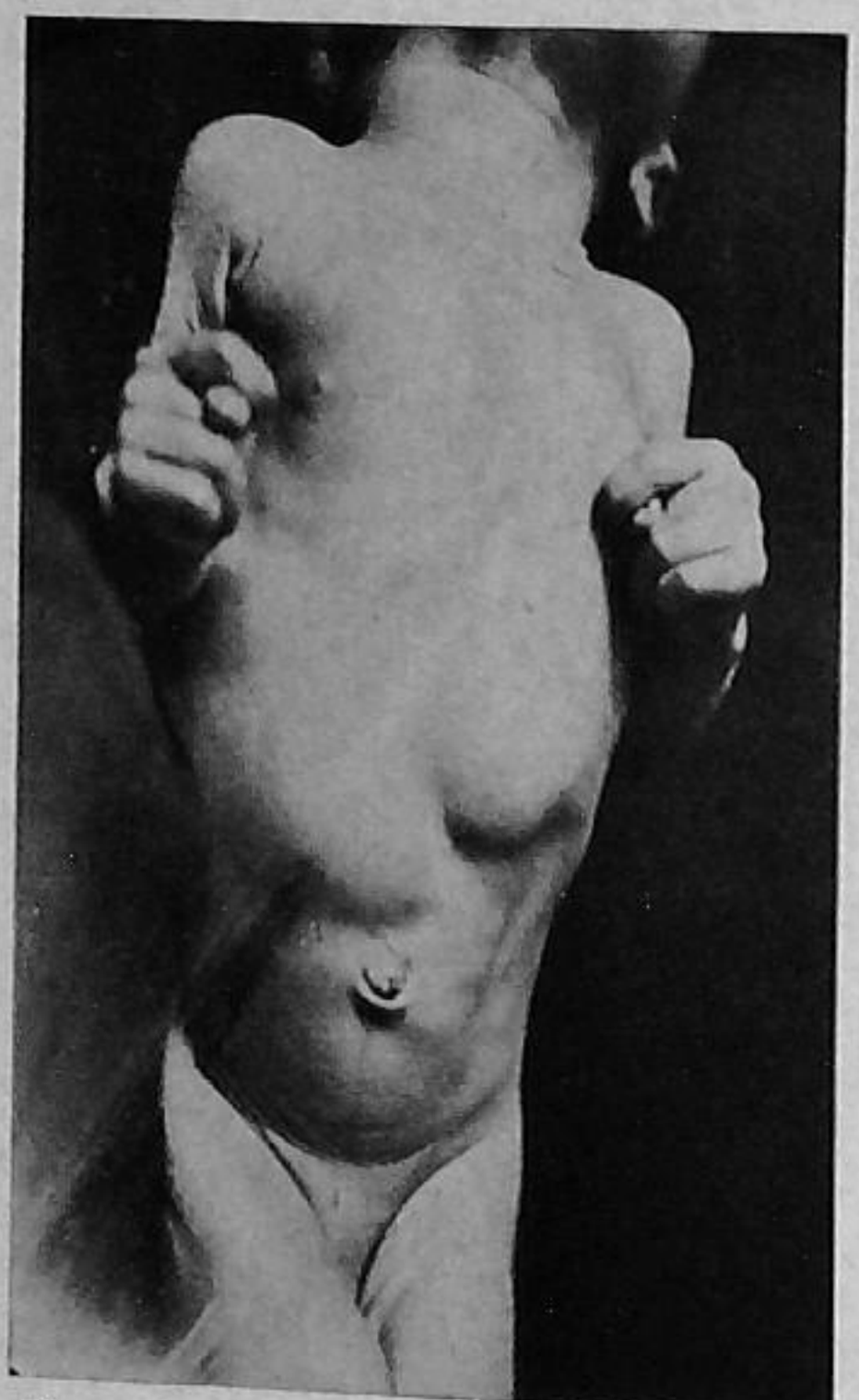

FIG. 8.—VISIBLE STOMACH PERISTALSIS IN PYLORIC STENOSIS.

If you proceed farther in your examination by washing out the stomach, you will usually be able to make out that the capacity of the organ is increased—that instead of holding, let us say, a couple of ounces or so, which is normal for that time of life, the stomach holds 3, 4, or even more ounces of fluid. In other words, you can prove the existence of actual *gastric dilatation*, and that in addition to the peristalsis and the palpable tumour, is the third purely stomach sign of this condition. So much, then, for the symptoms and signs.

As regards the **pathology of congenital pyloric stenosis**, I may say at once that we do not know what the real pathology of the condition is. What we do know is that in those cases which prove fatal, or in which operation has been performed and an opportunity has been given of inspecting the pylorus during life, there is present a great thickening of the pylorus, which section shows to be due almost exclusively to hypertrophy of the circular muscle fibres. I have here a few specimens which show this thickening very well. Here, for example, is one in which you can see quite well with the naked eye the ring of thick muscular tissue surrounding the pylorus, and which practically converts it into a tube of almost cartilaginous

consistence (fig. 9). That is one thing you can make out. Another thing you find is that the whole wall of the stomach is more or less hypertrophied owing to the efforts of the stomach to drive its contents through this narrow channel. Thirdly, you will find that the mucous membrane is in a state of chronic catarrh. That summarizes all there is to be said about the morbid anatomy of the condition: there is an hypertrophy or overgrowth of the circular fibres of the pylorus, there is a thickening of the whole wall of the stomach, and there is catarrh of the mucous membrane.

A FIG. 9. B

A. SECTION OF NORMAL PYLORUS.
B. SECTION OF CONGENITAL PYLORIC STENOSIS.
(EQUAL MAGNIFICATION.)

So far everyone is agreed, but when you come to attempt to explain how these anatomical changes are produced you at once enter the realm of uncertainty. One may say that, roughly speaking, there are two views as to how it is that this condition is brought about. There is, first, the view that *the hypertrophy of the muscular fibres of the pylorus is a congenital anomaly*—just as you may have a congenital club-foot or any other inborn malformation. The other view is that the hypertrophy is not a congenital malformation at all, but is *consecutive to a spasm of the pylorus.* Those who adopt this theory maintain that the condition of which we are speaking is, in its essence, a spasm of the pylorus of great severity and of

long continuance, and that, as a consequence of the continued spasm, you get hypertrophy. When those who hold this view are asked how this spasm is brought about, they are compelled to have recourse to various hypothetical explanations. Some assume that there is an 'inco-ordination.' Now, inco-ordination is one of those expressions like 'trophic effect,' and 'toxin,' and 'reflex action,' and a few others which are often used as a cover for much ignorance; and it is no more satisfactory in this case than it usually is. Then there are some who say the spasm is brought about by an excess of acid in the stomach contents, because it is known that excess of acid leads to closure of the pylorus. No proof has been advanced that such excessive acidity actually exists. Others say that something has gone wrong with the chemical mechanism in the duodenum, which is really responsible for the normal opening and shutting of the pylorus, whilst the latest suggestion is that it is due to an excess of adrenaline in the blood. But all these explanations, I confess, are unsatisfactory. My own view is that the condition is not a congenital anomaly, but that the second view is right, explain it how you may, and that we have here primarily to do with a spasm. In favour of that I would only point to the two great clinical facts which cannot be got over: first, that the symptoms do not come on immediately after birth, but generally a fortnight or more later; and, second, that the condition is capable of being absolutely cured by purely medical treatment, leaving the stomach, as I have proved to my own satisfaction time and again, in a perfectly normal condition afterwards. I cannot see, on the assumption of a congenital anomaly of formation, how such cases should make a perfect recovery without operation and without any dilatation of the stomach being left. I believe, therefore, that we have here to do with a spasm which goes on to hypertrophy, but how that spasm is brought about I confess I do not know, and from the point of view of treatment I do not think it matters.

That brings me to speak of **treatment.** Now the first essential here is that you should begin treatment early. Failure in these cases is due to the condition being recognized too late, with the result that treatment is left until the child is in such an exhausted condition that recovery is difficult, if not impossible. That just emphasizes what I have already said about the importance of early diagnosis.

The first point you will have to settle is whether to treat the

child **medically or by operation.** As to this I would say that if you have diagnosed the case early, certainly within the first two months of life, operation is always to be advised. This does not mean that recovery is impossible under medical means—far from it—but, seeing that the spasm does not pass off spontaneously until the child is nearly five months old, medical treatment is necessarily very prolonged and troublesome and the baby runs great risks of inter-current disease whilst it is in progress. Further, if it fails, the infant may easily have reached such a state of exhaustion that operation has no longer a fair chance. Patients, also, who cannot be treated at home, are bad subjects for medical treatment owing to the great risk of infection in hospitals.

If, on the other hand, the symptoms have been present for several weeks before the diagnosis is made it will be wise to give medical treatment the preference, for such cases do not do so well with operation.

The essentials of **medical treatment** are careful feeding, gastric lavage and the use of drugs to relax the pylorus. As to the first, if the child is on the breast he should on no account be weaned, but small short feeds should be given every two hours or so. If on the bottle he should be fed on a half-cream dried milk with the addition of a little dextri-maltose, the feeds being again rather small and frequent, though the exact size and intervals will vary with the individual case.

Lavage is carried out in the usual way once or twice daily, with normal saline.

Up to recent times the use of anti-spasmodic drugs gave disappointing results, but many observers now claim good effects with eumydrine (atropine-methyl-nitrate). It is given in solution (1 in 10,000), which must be freshly prepared, about half an hour before feeds six or seven times daily. The dose varies between 2·5 c.c. (0·25 mgm.) to 5 c.c., according to the effect on the symptoms. The drug should be continued for several weeks and for at least one week after vomiting ceases.

If **operation** is decided upon Rammstedt's splitting of the pylorus is the procedure of choice (fig. 10), but the details are beyond my province.

The post-operative treatment is very important and should be carried out if possible by a nurse experienced in the management of these cases.

Feeding is started four hours after the operation, plain water or a little breast-milk being given with a spoon. Spoon-feeding should be continued for twelve hours, 1 drachm only being given every hour at first, but later the quantity is increased and the intervals lengthened.

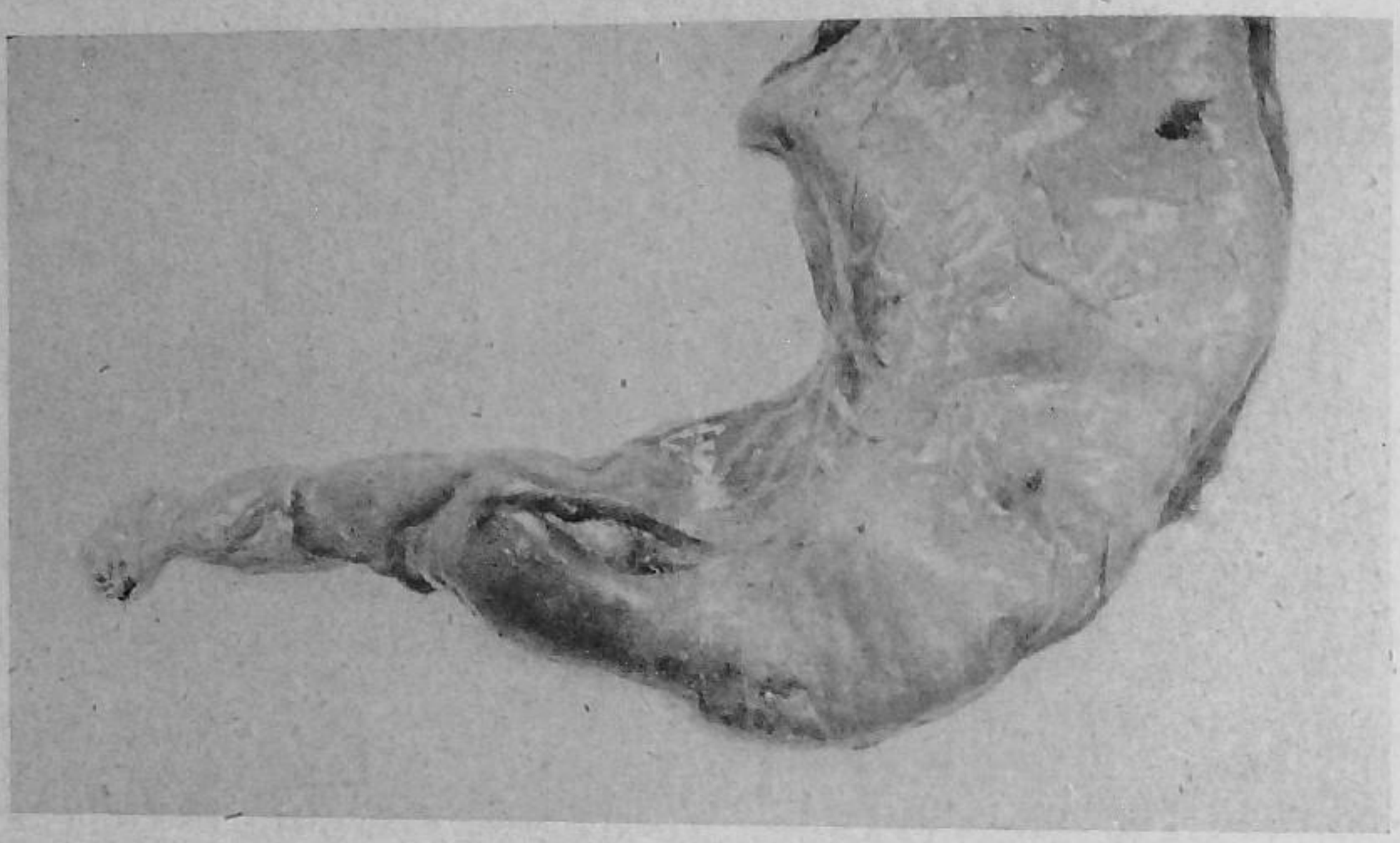

FIG. 10.—STOMACH FROM CASE OF CONGENITAL PYLORIC STENOSIS SHOWING THE INCISION IN RAMMSTEDT'S OPERATION.

Diarrhœa arising during the first few days after operation is always an anxious symptom and calls for a reduction in the size of feeds. Inter-current infection is also a real bugbear of convalescence and pyloric babies should be guarded from it most carefully. It is because the risk of this is so much less that the results of operation are better in private than in hospital. With modern facilities for isolation, however, this risk is reduced, and series with a low mortality (0 to 2 per cent.) have been reported

LECTURE IX

INFANTILE DIARRHŒA

In this lecture I wish to discuss with you the third of the symptoms of digestive disorder which I have mentioned—namely, *diarrhœa*. I think there are three causes which render infants particularly liable to suffer from diarrhœa. The first is that the sterilizing power of the stomach at this age is small; it secretes but little hydrochloric acid, and organisms which are swallowed with the food stand a good chance of running the gauntlet of the stomach and setting up irritation in the intestine. The second is that infants are fed mainly upon milk, which is often anything but a germ-free fluid; and the third is that they are particularly liable to develop diarrhœa as a complication of infection elsewhere than in the alimentary tract, such as the ears, lungs or kidneys.

One of the most remarkable changes in the incidence of disease in infancy which has occurred in the past twenty-five years is the disappearance of what used to be called '*summer diarrhœa*.' Every autumn the wards of our children's hospitals became full of infants dying of this annual epidemic disease, spread, without much doubt, by flies and dust and infected milk. That this type of diarrhœa has largely gone is attributed variously to the infant welfare movement which has educated the mothers, to the increased use of dried milk, and lastly—and in my opinion the most likely explanation—the appearance of the motor-car with the consequent banishing of the horse from the street and with that the large quantities of manure which used to be a feature of our large city thoroughfares. Diarrhœa can still occur in epidemic form, whenever infants are herded together, and it behoves us to be especially vigilant in regard to newborn babies in maternity units, infants in day and residential nurseries, and in hospital wards. But such epidemics are no longer seasonal.

Since the disappearance of the summer epidemics the importance of diarrhœa as a secondary manifestation in other infective disorders

of infancy is becoming more obvious, and since the colder weather brings its train of coughs and 'colds' and ear infections, which in infants may be complicated by diarrhœa, it has been suggested that what we have to deal with to-day is the problem of *winter diarrhœa*, and in so far as there *is* any seasonal incidence in this disease, I think this is true.

If we attempt a **classification** of diarrhœa in infants, I think we shall see more clearly what I am trying to explain, namely, the importance of the secondary types. Certainly we must still keep in our list diarrhœa which is primarily due to disorders in the alimentary system. Here we have three definite groups: first, purely *dietetic* (e.g. unsuitable food); secondly, *infective* (e.g. dysenteric); and thirdly, a *mixed* type in which after a period of dietetic troubles there comes a final episode which is often infective in origin and usually fatal. The second main type of diarrhœa is that due to *parenteral* infection, when the secret of treatment is to find the primary source of trouble. Throat infections, the ordinary 'cold,' otitis media, pneumonia, pyelitis and skin infections are some of the commonest. It is easy to exaggerate this side of the problem. For example, I am told that at one large American hospital it was the practice to puncture the ear drums of all babies admitted with diarrhœa as a routine! This is going too far, but it is equally absurd to neglect the ears in an infant with diarrhœa and allow mastoiditis to develop, as it does, making the whole picture still more complicated.

I am amazed how little parenteral infection is necessary to produce diarrhœa. I remember once seeing a dentist's baby who was having frequent loose stools. The only possible cause seemed to be a small stitch abscess in a circumcision wound. We took out the stitch and the diarrhœa ceased!

These considerations help us in the **prevention** of diarrhœa. Breast feeding up to six months of age is the greatest single measure of value because it cuts out all the alimentary causes—dietetic and infective. Next, the use of a safe milk, dried or boiled, for infants who are not breast-fed. Thirdly, we must guard the infant against what may be mild infections in adults whether these take the form of a slight diarrhœa or of upper respiratory tract infections.

In classifying the **varieties of diarrhœa** met with in infants I shall proceed upon clinical rather than upon strictly pathological lines and shall divide the cases—first, into those which are acute; and, secondly, into those which are chronic. I shall distinguish

among the acute diarrhœas three varieties—those which are simple, those which are febrile, and those which are choleraic—and I shall make no subdivision of the chronic diarrhœas at all. When we come to treatment we shall take both acute and chronic cases together.

If the diarrhœa is acute it may be simple—that is to say, it may be diarrhœa and nothing else (dyspeptic diarrhœa); or it may be accompanied by pyrexia, when we speak of it as febrile (infective diarrhœa); or it may be so severe in its symptoms, and accompanied by such profuse watery evacuations, that it is described as choleraic. The most dangerous form, what is called epidemic infective diarrhœa, the familiar 'D. and V.' of the hospital receiving-room, is febrile in type. Sometimes in very severe cases it may be choleraic. Of course, this classification is not by any means hard and fast. Diarrhœa cannot be definitely marked off into different classes; the varieties pass into one another. A case which begins as a simple diarrhœa may become febrile, and from that may become choleraic, just as it may be choleraic from the first, or may begin with fever. Or, on the other hand, it may remain simple throughout. You cannot always refer a case exactly to one or other of these classes.

What are the **symptoms** of infantile diarrhœa? In the first place, it may begin either gradually or with great suddenness. It may come like a bolt from the blue, affecting a child which was, or appeared to be, in perfect health. The motions are noticed to be more frequent, and often there is more or less vomiting. At first the stools are natural in colour—that is to say, more or less yellow—but before the diarrhœa has lasted long they become greenish, and finally they may come to contain mucus, or even blood. If the diarrhœa still persists, the motions become extremely offensive and of a watery consistence.

I wish, at this point, to say a word about the **character of the motions** in different types of diarrhœa, because I know it is a matter which will be apt to give you some trouble when you get into practice. What is the significance of the different kinds of stools which you meet with in the diarrhœas of infancy? First about the green stools, which are so common, and which resemble in appearance chopped parsley or spinach. What is the cause of their greenness? It has been supposed by some that the colour is due to the growth of a special micro-organism which produces a peculiar pigment. I do not wish to deny that that may sometimes be the case; but in the majority of instances I think it is merely due to the bile pigment being hurried on through the intestines with-

out having undergone the usual changes. What is the meaning of the motions being stinking and putrid? That means that peristalsis in the upper part of the tract is too rapid. The consequence of this is that the food is hurried on before it can be properly digested and absorbed. You get, therefore, a mass of imperfectly digested and incompletely absorbed food lodged in the colon, and putrefactive organisms grow in it; hence the putrid stools.

There is another variety of stool, which may be described as lumpy or cheesy, where you see white particles, varying in size, more or less oval or rounded in shape, scattered all through the fæcal matter. What is the significance of that? These round or oval particles are in some cases the remains of undigested milk; they consist partly of casein and partly of a soapy substance formed from the fat of milk and lime salts, and indicate that the child is getting more milk than he can digest, but in many cases they consist simply of little balls of altered mucus, showing a catarrh of the bowel. Then there is a kind of stool in children which is extremely irritating, and which scalds the skin. If a child has that form of diarrhœa he gets an erythematous eruption over the buttocks, and I have seen those stools so irritating that a drop of them falling on the dorsum of the foot produced a blister in a few minutes. To what do these stools owe their irritating properties? It is due, I think, to the development of fatty acids—butyric acid and its allies—and means that there is too much fat in the diet. Excess of sugar, on the other hand, produces frothy acid motions of natural colour. Then there is a stool which you may describe as 'slimy'; that means a stool which contains mucus in excessive quantity. Remember, however, that barley-water may produce a slimy-looking stool, which looks very like one which contains mucus. If you recognize excess of mucus, you are justified in believing that the large intestine is particularly involved; and if blood is passed in the motion, as it sometimes is, that suspicion amounts to a certainty. Such is the significance of the chief varieties of stools which you will meet with in the diarrhœas of infants.

We will now pass on to look at some of the other symptoms. The **general symptoms** may be trivial, as they usually are in simple diarrhœa; or they may be extremely severe, as they always are in the choleraic form. It must be realized that in all but the most trivial varieties of diarrhœa there are profound biochemical disturbances. The young infant readily becomes dehydrated. The body surface is relatively large, so that with a rise in temperature

there is an increase in loss by evaporation trying to increase the heat loss of the body. Added to this is the loss of fluid by the frequent stools and by the vomiting which so often accompanies diarrhœa of whatever origin. These fluid losses mean also loss of chemicals—sodium chloride through the skin, acid from the stomach, and varying amounts of alkaline bases lost in the stools. The kidney function may become impaired and lead to still more serious biochemical changes. Indeed the cause of death is often closely allied to death from uræmia or in some instances, when ketosis is a feature, to death in diabetic coma. These facts must be kept in mind when we come to discuss treatment. Moreover, there can be no doubt that the extreme collapse and depression from which those children suffer is not the consequence merely of the draining away of fluid from the body, but are a direct result of poisoning by the absorption of toxins from the alimentary canal. Those symptoms may be described as the symptoms of collapse. The fontanelle becomes depressed, the eyes sunken and staring, and by and by they are kept partially open while the child is asleep, and you can perceive muscus on the cornea. The skin becomes wrinkled, the child seeming, as it were, to shrink away from the skin, which becomes redundant; and when you pinch it up, instead of falling back again as it would normally, it remains in folds. When you get this condition, which is described as a want of elasticity in the skin, you can always be certain you are dealing with a severe case. It is one of the points always to be looked for in your cases of infantile diarrhœa. Many observers believe that when such a condition of the skin is present there is an involvement of the kidneys, and that partial suppression of urine is taking place. And unquestionably, if you examine the urine in those infants, you will usually find that it contains some albumin. The importance of recognizing this want of elasticity in the skin is due to the fact that it indicates **dehydration** and when you find it you should institute special means of treatment, which I shall describe later. If the diarrhœa goes on, the symptoms of collapse become more pronounced. The temperature, which was at first perhaps high, falls, at least on the surface of the body, though it may remain high in the rectum; the child becomes blue and livid and finally dies, death being often preceded by convulsions. That is the usual picture of a typical case of diarrhœa of severe type in a young infant. I need hardly say, however, that the picture varies a great deal. In the simple form there may be no evidence of collapse at all; in the choleraic form the signs of collapse will dominate

the whole scene; whereas in the febrile cases the depression may not at first be very great but becomes more marked at the close.

We may now pass to the **treatment of acute diarrhœa.** The first thing I want to say is that in all serious cases you are dealing with an acute emergency, analogous to the 'acute abdomen,' and I have come to believe that institutional treatment is equally essential. The infant will require expert nursing and medical care, especially the supply of fluids by the various routes, and this can only be carried out when there are trained persons as expert in this field as the surgeon who deals with the acute surgical emergencies. The general principles which must guide those who undertake the treatment of diarrhœa are *starvation*, *hydration* and *elimination*. Starvation means withholding milk for not more than forty-eight hours, i.e. until such time as you have secured some degree of elimination and restored the biochemistry towards normal. Infants, as I have already told you, bear starvation well only if they are kept warm and well supplied with fluid. Therefore give only half-strength normal saline or glucose (5 per cent.) by mouth for the first twenty-four hours and then, if the symptoms abate, begin with weak feeds of half cream, dried milk or dried protein milk.[1] Dextri-maltose may be added to increase the caloric value at this stage, as it is a non-fermentable sugar.

Hydration, that is the supply of fluid, must be begun at once. Drinks by mouth, as already mentioned, will help, provided that vomiting is not too troublesome. Rectal salines, using only a very low pressure, are often retained surprisingly well after the bowel has been washed out, as I shall describe later. But if these two routes fail you, as they often will, you must proceed without delay to other methods. Subcutaneous saline is unsatisfactory in many ways, especially as it may lead to infection, and the amount you can give is limited. Intramuscular saline by a drip apparatus is quite useful as more can be run in than under the skin. Intraperitoneal saline has its advocates, but it seems to me that in a case requiring this you will have secured transference to hospital, and there an intra-venous drip saline will be the method of choice. I do not propose to give you details of this: you will learn them best by assisting in the method for yourselves and you will learn how difficult it can be and yet how relatively easy for the expert to get into the saphenous vein at the ankle. Saline is the fluid of choice because the body will adjust its chemical loss by grabbing at the acid or the alkali radical

[1] Supplied by Mead, Johnson & Co.

as required, and it has the great advantage of being non-irritant to the veins and a stimulant to renal function.

With regard to *elimination*, if there is vomiting, begin by **washing out the stomach** in the manner I described in a previous lecture. If the diarrhœa is severe, and especially if there is reason to suppose that the large intestine is chiefly involved, **wash out the colon** also—that is to say, begin your treatment by washing out ' at both ends.' I have already described to you the technique of washing out the stomach, and I would like now to say a word about washing out the bowel. I do not think it matters much what solution you use for washing out. Warm saline is as good as anything else. It should be given through a douche-can or funnel. Care must be taken not to hold the can too high ; it should never be raised more than 2 feet. Remember it is not difficult to rupture the colon of a child in attempts to wash out the bowel. I think it is best to have the child lying on his back, with the hips slightly raised, so that the fluid can gravitate down into the colon, using a small-bore soft œsophageal tube, just as for washing out the stomach. The tube should be introduced carefully, and you will find its introduction much more easy and satisfactory if you keep the fluid flowing all the time. The reason for this is, as you will readily see, that the colon being only a potential cavity, and its walls more or less in apposition, the liquid as it runs in clears a way for the tube, and so you are less likely to get the end of it entangled in folds of mucous membrane, or to push it through the wall of the bowel. Pass the tube up about 2 inches : that is high enough. Irrigate the intestine thoroughly until the washings come away clear. By so doing you will wash away some at least of the organisms which are growing there. You also wash away a certain amount of the toxic products which these bacteria have been producing.

Now, you cannot, of course, wash out the small intestine, where the micro-organisms are chiefly growing. In order to promote elimination there you may have recourse to drugs. In other words, paradoxical though it may seem, you may treat diarrhœa, in its early stages at least, by purgatives, just as you treat dysentery in grown-up persons by aperient drugs such as sulphates. You may start with a full dose of castor oil, which may be poured down the tube after the stomach has been washed out. If there is vomiting and difficulty in getting the oil retained, give a quarter grain or so of calomel. It is only fair to say that some authorities question the rationale of giving purgatives when there is already evidence of

frequent evacuation of the bowels. Perhaps with the use of the newer sulphonamide drugs purgation may be avoided.

There are still some **special indications** to be met. If the skin is in the inelastic state I have described, it is advantageous to give the child a tepid pack. If the signs of collapse are great, you may have recourse to the mustard bath, the temperature of which is gradually raised to 110° F., this being perhaps the most powerful stimulant we possess. Niketham ide in doses of $\frac{1}{2}$ to 1 c.c. is a most valuable stimulant.

During all this time the child must be kept under the best hygienic conditions possible. He should be placed in a well-ventilated room and should be **kept very clean,** the napkins being changed as soon as they are soiled, and never allowed to stay in the room after being taken off the child. It is becoming increasingly realized that in hospitals or institutions housing a lot of infants, infectious diarrhœa is a serious menace to life. Hence babies with diarrhœa should be nursed, as far as possible, like patients with typhoid fever, so as to prevent spread from patient to patient. In every case the patient should be gently bathed with tepid water twice a day, and the mouth frequently swabbed out with cold water. In bad cases and in very hot weather the child should, if possible, be removed from the town to the country.

It has been said ' put not your faith in drugs ' in the treatment of infantile diarrhœa. But, of course, drugs can give you some help, and I now want to say a few words as to the **use and scope of drugs** in the management of such cases.

Although, as we have seen, intestinal putrefaction is an important factor in the production of diarrhœa, I do not think you will get much benefit from the so-called intestinal antiseptics. I am no believer myself in salol and all the substances of that class. I believe the best intestinal disinfectant is a dose of calomel, though whether it acts by virtue of its being calomel or because it is an aperient I do not know.

You may derive certain indications for the use of particular drugs from the character of the stools. If the motions are sour, it is well to administer alkaline remedies, carbonate of bismuth or aromatic chalk being amongst the best. If, on the other hand, the stools are slimy and alkaline, many believe in the administration of hydrochloric acid. In the severe cases, so long as the stools are green, I recommend you to give small doses of castor oil in some such form as this :

Ol. ric.	℥v.
Mucilag. tragacanth.	q.s.
Aq. menth. pip.	ad ℥i.

Give this at intervals of two or three hours for one or two days. Instead of castor oil you may give calomel and Dover's powder, $\frac{1}{8}$ grain of each. Later on, when the stools have begun to improve, give bismuth, remembering the rule to give it in large doses. A very favourite prescription, and one which is now in the hospital pharmacopœia, is carbonate of bismuth, 5 grains ; calomel, $\frac{1}{6}$ grain ; pulv. ipec. co., $\frac{1}{6}$ grain. Such a powder suspended in some albumen water, and given every four or six hours, as the case may require according to its severity, will often be of great service after the acuter stage of the illness has passed off. If the stools continue to be watery and offensive, you may have recourse to astringents of some sort. Tannic acid is helpful in such cases. Some of the new sulphonamide drugs promise to be of value in infantile diarrhœa of infective origin. Sulphaguanadine is the best, so far : give an infant $\frac{1}{2}$ tablet to start with and then $\frac{1}{4}$ tablet every four hours (this means 0·25 and 0·125 gm. respectively) for two or three days.

As **opium** plays such a large part in the treatment of diarrhœa, I want to lay down for you, as clearly as I can, some rules for its administration. You can do great harm with opium. The warnings of the textbooks in this matter are fully justified ; I have known a baby rendered dangerously comatose by 1 drop of laudanum. On the other hand, there is no one drug which is capable of rendering you such good service if properly used, and in many cases of infantile diarrhœa it is indispensable. Let me state, therefore, the following *rules for the administration of opium*, which I think you will find useful in practice. First, never give it at the outset of the illness. Begin with aperients, not with anything which will cause the contents of the bowel to be retained, as opium does. Secondly, never give it if there are signs of collapse, because then its narcotizing effect becomes extremely dangerous. Thirdly, never give it if the tongue is furred. These rules, like all rules in medicine, are subject to certain exceptions ; but when you are beginning practice let me advise you, if you want to avoid making serious mistakes, to keep them in mind.

You will now require some rules as to when you *are* to give opium. Always give it if the stools are very frequent and accompanied by much straining—that is to say, cases in which the lower part of the

large bowel is particularly involved are those in which opium is specially indicated. Secondly, if the stools are offensive and the tongue clean, you have a combination of things which cries out for opium, because, as we have seen, this is due to the hurrying on of the contents of the small intestine by too rapid peristalsis. In such a case opium calms down the peristalsis, and so prevents putrefaction and diarrhœa. Thirdly, you should always give it in those cases in which the diarrhœa is of the lienteric type—that is to say, in which a motion tends to occur immediately after food is introduced into the stomach. Diarrhœa of this sort is due to an exaggerated excitability of the whole intestinal tract, and opium is the only agent which can be depended upon to lessen that excitability.

Two practical rules remain. One is, never wake a child up to give a dose of opium. If you observe that rule, you are not likely to produce poisoning. The child will sleep off an overdose of opium if you will let him; the danger is that the mother or nurse may wake him up to give another dose when he is already in a drowsy condition. The remaining rule is that you should not add opium to a mixture, but give it by itself—that is to say, measure out a drop or half a drop of the tincture (for the liquid preparations are the most convenient), and give it separately. The reason for this is that one often wants to withhold opium while going on with other remedies.

The **treatment of chronic diarrhœa** I need hardly describe, because it is conducted on the same general principles as that of an acute case. You do not need to starve such patients, but you select a diet of dried protein milk[1] with the addition of dextri-maltose given in small three-hourly feeds, or soured milk,[2] or an ordinary half-cream dried milk, or a whey mixture (p. 75). You prevent chill, and prescribe one of the drugs I have mentioned, according to the indications furnished by the stools. Sulphaguanadine promises to be of great help in this type.

There remains a special form of diarrhœa which I have not yet mentioned—namely, **ileo-colitis.** I speak of it separately because it is different from the other forms I have described in so far as it tends to be accompanied by ulceration, and to be confined to the large bowel and the lower end of the ileum. It may start as ileo-colitis, and in that case it may be due to an organism of the

[1] Mead, Johnson & Co.

[2] A dried soured milk ('Lacidac') is supplied by the 'Cow and Gate' Co., Ltd., in full-cream, half-cream, and separated form.

dysentery group (e.g. Sonne's) or any diarrhœa such as I have been describing may end by passing into a condition of colitis with ulceration.

One reason why this disease is of importance is that it is apt to be mistaken for another important disease—namely, intussusception. How shall you recognize it ? Its chief features are that it is accompanied by fever, by straining or tenesmus, and by the passage of blood and mucus. How shall you tell it from intussusception ? In intussusception there is less tendency to fever, there is usually vomiting, and you can generally feel the tumour either through the abdominal wall or *per rectum*. In making a rectal examination in a case of intussusception the sphincter will often be found to be relaxed. The descending part of the bowel has been said to feel like the os uteri, but sometimes it is rather soft, and may cling to one side of the rectum, so that the examining finger may pass by it if care is not taken. Skilled palpation may also detect a suspicious emptiness in the right iliac fossa (Dance's sign).

The late Mr. Barnard pointed out a diagnostic sign of some value in distinguishing the two conditions. It depends on the fact that the contents of the small intestine are unable to pass the seat of obstruction in intussusception, so that if you get any bile on the napkins, or on the finger after it has been passed into the rectum, you may conclude with fair certainty that you are dealing with a case of colitis, and not of intussusception. If your minds are open to the possibility of making this mistake you are not likely to fall into it often, but I have more than once known the abdomen opened in colitis under the impression that it was a case of intussusception, and I have known one case of intussusception which was allowed to die under the idea that it was one of colitis. So there is a real danger of the mistake being made.

About the **treatment of colitis** there is little to say. I have practically described it in speaking of the treatment of diarrhœa in general. As the motions are frequent and accompanied by tenesmus, opium should be given almost invariably, combined with castor oil at first, and later with large doses of bismuth. In this form of diarrhœa you will find washing out of the bowel particularly useful, seeing that the colon is the main seat of the affection. Wash out with warm saline. Some recommend a solution of nitrate of silver, but I think it is of doubtful advantage, and is apt to produce pain. In chronic cases, however, a solution of argyrol or protargol ($\frac{1}{2}$ per cent.) is often very effective.

LECTURE X

CŒLIAC DISEASE

Cœliac disease is by no means an uncommon condition, and it is one which is very well defined. You may describe it briefly as a wasting disease beginning in childhood, which is characterized by a particular form of diarrhœa, which runs an extremely protracted chronic course with a great tendency to relapses, and which, when it lasts long enough, is apt to lead to a form of infantilism. I shall make it clear as I go along what some of the points in this definition mean.

The disease was first described by Dr. Gee in 1888. He gave a very clear and graphic description of it in St. Bartholomew's Hospital Reports for that year. A few years afterwards it was again described under the term 'acholia' by the late Dr. Cheadle. He used the word 'acholia' because he thought that in those cases there was a deficient secretion of bile. Still later it was re-discovered, so to speak, by the late Professor Herter, of America, who described these cases as examples of 'intestinal infantilism.' He believed they were due to a specific type of micro-organism. These are historical points that may not interest you much, but I bring them forward to show that the disease has been described several times by different people under different names, although it has been slow to find general recognition. And yet it is a disease which is not so very uncommon in practice. Indeed, it makes up one of those conditions which are slumped together by mothers, in the out-patient department, under the title of 'consumptive bowels,' a so-called disease of which you must all have heard mothers speak.

Passing to **clinical characters,** I have already told you that it is mainly a disease of childhood. It begins not in infancy, but very constantly about the second or third year of life. It is commoner in girls than in boys. The first thing noticed wrong with the child, usually, is that it is wasting and is dull, languid and fretful—

nothing very definite, but just the general signs of ill-health. If you make inquiry into the character of the stools, you will probably be told that they are rather more frequent than they ought to be ; that is to say, not a violent diarrhœa, but perhaps there are two or three

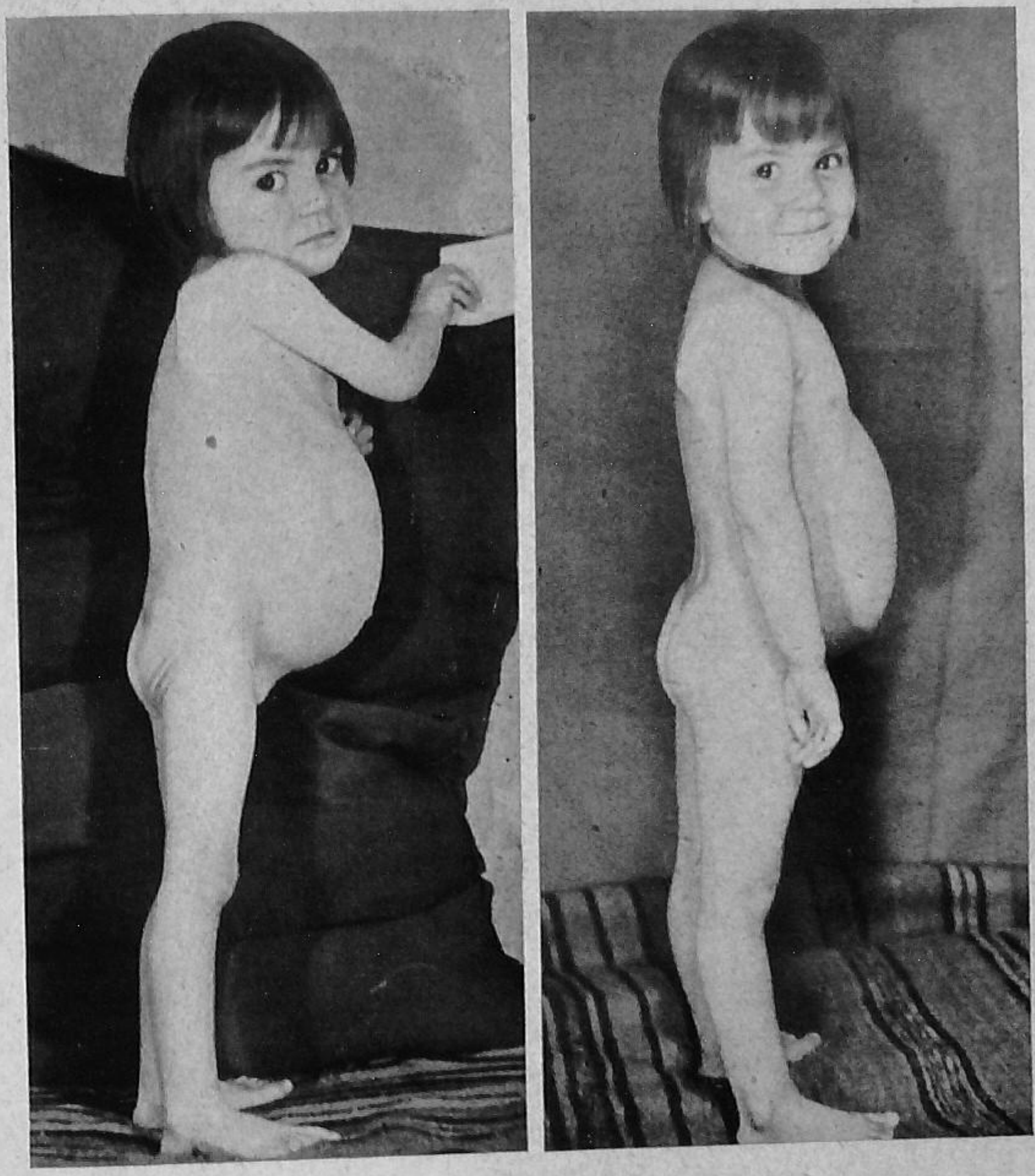

FIG. 11.—CŒLIAC DISEASE.

Child of 3 years. Note wasted buttocks and protruding abdomen. In the right-hand photograph the child is shown just over 6 months later.

or four motions a day. Further, the stools are greatly increased in bulk, and they are usually of the consistence of porridge and not unlike it in colour. They are pale, extremely offensive, and sometimes contain a little mucus or blood.

When you come to examine the child you will find that the only

symptom, in addition to the general wasting, is some distension of the abdomen. The abdomen is prominent and tympanitic, but you will not find any lumps, nor any enlarged glands or fluid. In the later stages, however, a slight degree of ascites may develop. The prominent abdomen with wasted buttocks is rather characteristic. These children, especially after the disease has lasted some time, come to exhibit quite a characteristic mental condition. They are usually rather spoilt children, and as the disease goes on they get to be remarkably precocious for their age. They acquire what is very unchildlike—an abnormal interest in their own disease and its symptoms, and may become occasionally quite hypochondriacal. I remember a well-marked example of the condition in the children's ward, about a year or so ago, in the person of a little boy three years of age. The sister in the ward said he was only a child of three, but he talked like a man of thirty—he was so precocious in his ideas and showed such abnormal interest in his own symptoms. This mental attitude has been noticed by several observers who have written about the disease. The parents also, I might say, tend to be of a certain mental type which may be described as 'fussy.' It might be supposed that this disease is one which affects neglected children chiefly, or children of the hospital class of patients. Quite the reverse is the case. You see some of the best-marked examples of it in children who have had a large amount of attention, sometimes, one might almost say, an excessive amount of attention paid to them from their infancy upwards, and who have been brought up amid the best surroundings.

As the condition progresses certain **complications** may appear, the cause of which we will consider later on. Amongst these is weakness of the legs. This may set in fairly early. I have known a case in which difficulty of walking was one of the first things the mother noticed wrong. In all cases, if they have gone on for some time, the child tends to get weakness of the legs accompanied by absence of knee-jerks—a condition which looks like one of peripheral neuritis and which may be due to a vitamin deficiency. They may get also œdema especially affecting the arms, hands, legs and feet, without any renal disease—one of the 'toxic' or 'essential' œdemas, as they are sometimes called, which you are apt to meet with in young children who have suffered from chronic intestinal disorder for some time. That you will see in a fair number of cases.

A much more severe complication is **tetany.** There may be

tetanic spasm of the hands and feet, and you get also in a few cases actual general convulsions, epileptiform convulsions. **Rickets** also may develop in some cases.

These, then, are the clinical characteristics : wasting ; diarrhœa characterized by stools that are not numerous, but large, pale and offensive ; distension of the abdomen ; a curious mental attitude ; and certain complications such as weakness of the legs with absence of knee-jerks, toxic or essential œdema, tetany and sometimes convulsions.

The disease is a very chronic one ; it runs a long course which can be measured by years. It has also this irritating peculiarity, that it exhibits a great tendency to relapse. Just when everything seems to be going well and improvement setting in, suddenly there is a set-back and all the symptoms become aggravated again. This is quite a well-marked feature of it. You must not suppose, however, that it is necessarily a fatal malady—very far from it. It may be completely recovered from even if it has lasted two or three years, and the child become perfectly sound and well to all appearance, but, of course, you may have death in the course of the disease usually from some intercurrent trouble such as broncho-pneumonia.

There is a group of these patients which is very interesting—those, namely, where the disease has lasted a long time, and has interfered so much with growth and development that **infantilism** results. This tendency for the disease to interfere with growth and development was well recognized even by those who first described it, but the late Professor Herter wrote a book on 'Intestinal Infantilism' in which great stress is laid on the arrested physical development. I think, myself, there is no great mystery about the infantilism. It is probably simply the result of chronic starvation. Any disease which interferes with nutrition will finally lead to a condition of retarded growth, and any condition of retarded growth and development one may speak of as infantilism. The child, as regards its physical condition, appears two or three years younger than it really is.

As regards **diagnosis,** the only thing that cœliac disease simulates is abdominal tuberculosis. The distinction between the two is made all the more difficult by the fact that cases which begin as cœliac disease or intestinal catarrh are apt to become secondarily infected and pass on to abdominal tuberculosis, and it may be difficult to say when this has taken place. But you are never justified in diagnosing tuberculosis unless you can feel enlarged

glands in the abdomen, or a thickened omentum, or make out the presence of free fluid. A tuberculin test, Mantoux or patch test, will help you in some instances. A negative reaction, I think, will justify you in the case of a young child in excluding tuberculosis.

The **pathology** of cœliac disease is still disputed. One thing everybody is agreed upon is that if you make a post-mortem examination in one of these cases you will not find anything beyond the fact that the intestinal wall is rather thin and atrophied-looking, but there is nothing to be made out on careful inspection of all the organs. In the absence of a definite naked-eye pathology, various theories have been put forward as to the cause of the symptoms. It has been suggested, for instance, that they are due to functional deficiency on the part of the liver; that the liver does not secrete enough bile, or that it does not perform its duty adequately. This was the theory of Cheadle. A second theory is that the pancreas is not sufficiently active. A third theory is that the condition is simply due to catarrh of the intestine, possibly set up by a specific type of micro-organism. That is the theory which Professor Herter adopted.

In deciding between these theories, careful examination of the stools might be expected to be of help. If you examine chemically one of the large pale offensive motions, in a case of cœliac disease, you will always find a great increase in the amount of fat. About that there is no doubt. The pale colour of the stools is mainly due to fatty acid crystals. I have never seen a case where the bile pigment was entirely absent from the stools. There is also no other evidence of inactivity of the liver in these cases. There is no defective secretion of urea, for instance, so that one can only say there is insufficient evidence in support of the view that the liver is inactive. It may not be forming bile acids properly, but there is certainly no complete arrest of the bile-secreting function. In favour of the pancreatic theory is the imperfect absorption of fat. But you will find that most of the fat that occurs in the stools in these cases is fat already split-up into fatty acids and soaps. When the pancreas is inactive, the form in which the fat is met with in the stools is unsplit fat. But still, in some cases of the disease there is a great deal of unsplit-fat present, looking as if there were perhaps a deficiency of pancreatic secretion. In favour of that view, also, is the fact that these patients cannot stand starch. If you give them much starchy food, you will find unaltered starch in the stools, so that there is a certain amount of evidence

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in favour of the pancreatic view. To that one might add that, undoubtedly, some patients are much benefited by giving them active pancreatic ferments by the mouth.

There is, finally, the view that the whole clinical picture is simply the result of intestinal catarrh. In favour of that is the fact that many of the cases begin with acute intestinal catarrh. A child, for instance, gets an ordinary acute attack of diarrhœa from which it does not make a complete recovery, but goes on by degrees to exhibit the full-blown picture of cœliac disease. Then, of course, the intestinal catarrh would explain the character of the stools; because, if the contents of the intestine are hurried on too fast, there is no time for the absorption of fat, so that a large excess of fat in the stools is quite compatible with the view that one is dealing with an intestinal catarrh, and the very offensive character of the motions also may result from that: the contents of the small intestine being hurried on rapidly into the large intestine, where they putrefy. So that on the basis of intestinal catarrh you can explain all the characters of the stools fairly well. Professor Herter went farther, and adopted the view that the catarrh was set up by a specific type of micro-organisms. He pointed out that in the stools in these cases you get a great excess of gram-positive organisms. In the normal stools you get an excess of gram-negative ones. I think it is an open question whether the organisms present in these cases are not the result of the state of the intestine rather than its cause. Abnormal flora you undoubtedly get, but I think they are quite as likely to be consequences of the catarrh as the cause of it.

Recently, an ingenious theory has sought to link cœliac disease, tropical sprue and idiopathic steatorrhœa by claiming a common cause—chronic jejunal insufficiency. The detailed explanation claims that there is some interference with the neuro-muscular control of the intestinal wall, so that the villi fail to absorb fat: the cause of this, it is suggested, may be a deficiency of some part of the vitamin B complex.

We now come to the important question of **treatment.** The first and most important thing in the treatment is to find a suitable diet. Seeing that fat is so badly absorbed in these cases, one must greatly lessen the amount of fat in the food. I told you also that most of these children do not stand starch well. If you give them starchy food, you will find unaltered starch in the motions, and with that you get fermentation and great abdominal distension, so

that you have also to limit the amount of starch or give a substitute for the starch in the form of dextrinized foods. Proteins the patient can stand quite well, and gelatin is also a particularly useful food constituent, so that it comes to this: the constituents of the diet must be proteins, gelatin, and dextrins. It is not quite easy to construct a diet on these lines, and yet successful treatment depends entirely upon your doing so.

I should recommend that you always make raw or under-done meat the basis of the diet. Children two years old, or even less, will take 6 ounces of raw meat daily quite well. It is made by scraping down the meat, so as to remove the fibre from the connective tissue, and may be given with a little broth. The only objection to it is the possibility of producing tape-worm. That happened in one patient that I had, but it is rare, and in any case it is easy enough to deal with the tape-worm afterwards; but there is no question at all about the immense value in these cases of raw meat. In addition to the raw meat itself, you can also give pure raw-meat juice. I do not mean the bottle juices, I mean the actual juice of the meat squeezed out by means of a press. You can buy now a small press (the Hercules Meat Juice Press), and by turning a screw you can squeeze from the meat all the juice part, which looks just like blood, and which forms a liquid of high nutritive value. Several ounces of this can be given in the course of a day. The raw meat and meat juice should be, so to speak, the *pièce de résistance* of the diet. Dextrins you can get in the form of Mellin's food biscuits, grape-nuts, breakfast biscuits, and malted rusks, which contain little or no starch, and can be given freely. Over-ripe bananas also suit well. Herter was a great believer in gelatin; he thought it does not form a basis for the growth of the particular organism which he supposed to be at work in these cases. Certainly children stand it well, and it is nourishing. You can give it in the form of calves'-foot jelly or any form of food into which gelatin can be introduced. Milk, these patients do not stand well, mainly because it contains fat, but partly because the curd is not easily digested; but you can give machine-skimmed or malted milk, and, used cautiously, you will find it of value. A dried skimmed milk powder is also available. Fortunately there is not usually much difficulty in getting the child to take this diet. The improvement in the stools under it is immediate.

One other very important point about diet I should mention—

that is, that it is advisable to give these patients some fruit juice regularly, as otherwise they are apt to develop scurvy. I have known this happen on at least three occasions. If there is any intolerance of fruit juice we can give vitamin C as tablets of ascorbic acid. It is also *most* important to supply them with vitamins A and D and seeing that fat upsets them it must be given in the form of one of the artificial preparations such as radiostoleum.

Following upon the suggestions of a vitamin B deficiency as an essential cause of cœliac disease, have come reports of successful treatment by dealing with this defect. Indeed, the results claimed for restoring fat and carbohydrate absorption are very remarkable and have been obtained without any alteration in the diet allowed. Children with cœliac disease have been given normal food with this form of treatment, which consists in alternate daily injections of 2 c.cm. of crude liver extract and 2 to 4 c.cm. of a whole vitamin B complex, also by injection. The whole course is said to be from three to six weeks, followed by a period when vitamin B complex is given by mouth.

Next in importance is the hygienic treatment, and one of the chief things to attend to here is the avoidance of chill. These children have a great tendency to get cold extremities, and even the least exposure to cold may bring on a relapse and a return of the diarrhœa. Hence you have to guard them very particularly against chill. The legs have to be kept warm ; sometimes one may have to wrap them in cotton-wool and bandage them ; these patients should also wear a flannel binder in order to keep the abdomen warm. Be careful also that they do not get chills out of doors or when they are being bathed. Further, you will often find a great advantage from change of air. If a patient is not making progress, a change of air to the seaside may help matters very greatly. In a protracted sort of illness like this, it may be necessary several times in the year to send the child away for a change.

Last we come to the treatment by medicines. Medicines help you a good deal in the treatment, but they are secondary in importance to the diet and general management. First I would put opium. It is particularly serviceable : it lessens the motions by delaying the passage of food through the small intestine, so allowing of better absorption, and, by preventing the food being hurried on into the large intestine before it has been absorbed, it lessens putrefaction, because by the time the contents of the small intestine reach

the colon there is less unabsorbed material to putrefy. Hence, under the use of opium the stools become less frequent, copious and offensive; the nutrition also tends to improve as the child gets more value out of its diet. You may give 1 minim of tincture of opium three times a day, for every year of the child's age, with a carminative mixture or with an astringent such as bismuth.

Sometimes opium and astringents tend to increase the abdominal distension, and may even induce a partial obstruction. In these circumstances a small nightly dose of castor oil is of great service.

Under the heading of medicines one may consider the question of ferments. Where there is much unsplit-fat and unaltered starch in the stools it is well worth while to try an active preparation of pancreas. I prefer holadin with bile salts, one capsule three or four times daily.

LECTURE XI

CHRONIC CONSTIPATION IN INFANCY AND CHILDHOOD

I have to lecture to-day on Chronic Constipation in Infancy and Childhood. I am afraid it is a dull subject but there is no doubt that it is an important one in practice, because constipation is not by any means an uncommon trouble in childhood, as it is also in adult life, and it must be admitted that a certain number of cases of constipation in adults have their origin in neglected constipation in childhood.

It will be convenient, for purposes of description, to speak separately of constipation in infants, and constipation in older children.

CONSTIPATION IN INFANCY

Taking infants first, you will remember that the normal infant has three or four stools in the twenty-four hours; but it would be a mistake to suppose, because a child has fewer motions than that, that it necessarily has constipation, provided always that the motions are normal in character and consistency. There are infants who have only one stool in twenty-four hours, and yet are well and gain weight. This fewness of motions—the consistency remaining normal—is particularly apt to occur in breast-fed children. You will see in books the statement that constipation is a vice of bottle-fed babies. I have not found that to be true; this scarcity of motions is particularly apt to occur in the breast-fed child. One reason for that probably is that breast-milk is so easily digested and so completely absorbed that it leaves little residue; and where it is not too abundant there may be so little residue left that one motion in the twenty-four hours is sufficient. Others of the cases, in which the motions occur at too long intervals without being otherwise abnormal, are due, I think, to what you may speak of as a sluggishness of the rectal reflex; I think there is no doubt

that some little babies have a 'lazy rectum,' that is to say, it requires a larger quantity of material to stimulate an evacuation than should be the case. But these are not, in the strict meaning of the term, cases of constipation.

Now, true constipation, that is to say when the motions are not only infrequent but too hard in consistence, departing from the 'yolk of egg' character of the healthy motion in the baby, and 'formed' like an adult motion, may be due to several causes. It is often said—and there is some truth in it—that **defective training** is a cause of constipation; if the child has not been properly educated up to regular evacuation of the bowels, constipation may result. It is easy to attach too much importance to this as a primary cause; it is more an aggravating condition in a child who is disposed to constipation than a cause of it in itself.

Second, and far more important, is **defective feeding.** This may take several forms. I think that the commonest and one most likely to be overlooked, is simple *under-feeding.* Naturally, if a child does not get enough food there is very little residue, little to stimulate the bowel, and constipation results. This can be checked by the fact that not only is the child not gaining but is losing weight, or you can give test-feeds, and weigh the child before and after.

The next form of wrong feeding in bottle-fed babies is giving an *excess of casein* in the milk mixture. Usually this is associated with too little sugar. Along with the excess of casein, or instead of it, there may be an *excess of fat.* This sometimes produces diarrhœa, sometimes constipation by the formation of soaps. You find in such a case a pale, solid, greasy-looking sort of stool, forming a large mass, which the bowel has difficulty in moving along. Lastly, the diet may be *deficient in water.* I do not think the latter is a common cause of constipation in little babies, but it may be in an older child, particularly if it perspires freely.

The next cause of constipation in infancy is *atony* not only of the bowel but also of the abdominal wall. That is apt to occur in flabby, particularly rickety, children who have large abdomens, and difficulty in expelling the bowel contents.

Opposed to that is the reverse condition, *spastic constipation*, where the bowel is the seat of cramp, so that instead of peristalsis going along smoothly there are spasmodic contractions, and the contents are held up. That, of course, will be associated with

colicky pain, and the accompanying screaming due to this is often a more prominent feature in the picture than the constipation itself. Possibly an excess of curd has caused the spasm but I spoke of this in my lecture on Colic.

There is no doubt, again, that *defective secretion* is sometimes a cause of constipation ; I mean a defective secretion of bile and of intestinal juice. That is shown by a characteristic hard, dry and friable motion, usually pale, almost to chalkiness. It is not common but when it occurs you should be able to recognize it.

Occasionally there are local causes in the rectum : I have already mentioned deficiency of the rectal reflex, but I am now referring to organic conditions such as anal fissure. In cases of apparent constipation I have sometimes found a small fissure in the anus which has made defæcation so painful that the motions are held back. Or, without actual fissure, there may be a very tight sphincter and anal canal. In some babies the anal canal is congenitally rather narrow, and that, of course, will oppose a mechanical obstacle to evacuation.

The last cause I shall mention in little babies is *mental defect*. Usually the constipation here is not the ' presenting symptom ' ; the child is brought because it is not ' getting on ' mentally. I have, however, known cases in which the child was brought because of constipation and the cause was mental defect which had not been recognized. That is particularly apt to occur in the milder degrees of cretinism.

The importance of recognizing these different causes is that effective treatment in any case will depend on the cause ; there is no routine treatment. I would particularly deprecate the indiscriminate giving of aperients to an infant because it is believed to be constipated, without as far as possible determining the main causal factor.

Defective training is a matter for the nurse ; the child must be put on the chamber at regular intervals ; a child, even a very young one, quickly learns what is required of it, and it is astonishing what a difference a capable nurse will make.

In regard to feeding, you must first think of the possibility of under-feeding. If that is the cause, the remedy is obvious : you must give more food. If the child is on the breast, it should have complementary feeds ; if it is bottle-fed, the strength or amount must be increased. If the trouble is due to excess of casein, you

must take steps to prevent the formation of masses of curd. There are many ways of doing that. Greater dilution of the milk may suffice, or you may citrate the milk, or you may partially peptonize it. The addition of more sugar is often all that is required. If the trouble is an excess of fat and formation of soaps, the amount of fat in the food is easily reduced. Deficiency of water is overcome by giving drinks of water between feeds; indeed, it is good to do that always in the case of bottle-fed babies, certainly in hot weather, whether they are constipated or not.

Atonic constipation must be met by treatment of the underlying condition which is causing the atony. Often that is rickets. The latter is treated in the usual way by regulating the diet, giving vitamin D in some form, by attending to hygienic requirements, such as exercise, open air, sunlight, and massage of the abdomen, applied not only to the abdominal muscles, but deeply so as to benefit the bowel itself. It is a mistake to suppose you require the help of a skilled masseuse; any intelligent mother can be taught how to do it. This is carried out twice a day for a few minutes at a time.

Spastic cases are to be met by regulating the diet and by drugs. Make sure there is nothing in the diet which is setting up spasm—that it contains no undigested material. The most suitable drugs are tincture of belladonna and the ordinary carminatives. In these spastic cases anything like massage would do harm rather than good, as it would still further stimulate contraction of the bowel.

In the cases associated with defective secretion your aim must be to stimulate secretion, particularly of the bile and intestinal juices. The most suitable drugs for that purpose are grey powder and podophyllin; $\frac{1}{2}$ grain of grey powder once or twice a day, or a drop of tincture of podophyllin three or four times a day.

For local causes the treatment is obvious. If there is fissure, it must be dealt with in the usual way; if the anal canal seems unduly tight, it must be stretched a little, under an anæsthetic. In young babies with a 'lazy rectum' insertion of the little finger lubricated with vaseline is quickly effective.

In the mental defect cases the only special treatment is by thyroid; in mild cases of cretinism thyroid will often have an extraordinarily laxative effect.

I have spoken as if it were easy to arrive at the cause of the constipation in a given case in infancy, but in practice you will not find it so. You will meet with a considerable number in which you

cannot be sure which, if any, of the causes I have mentioned, is at work. These may be spoken of as cases of *simple constipation*, with nothing else obviously wrong. In these circumstances it is always a good thing to introduce a more fermentable carbohydrate into the diet, such as malted foods (Mellin's food, for instance), or a little malt extract, or certain forms of sugar, especially the brown Demerara variety. Failing relief from that, you may have to give some aperient. But avoid the drastic ones. The least harmful is paraffin, which is preferably given in the form of emulsion. A teaspoonful of this given once or twice a day may be sufficient to act as a lubricant. If paraffin does not suffice, milk of magnesia is a popular and harmless aperient; it has the advantage that, introduced into the food, children take it readily. Another easily administered and successful laxative is phenol-phthalein, which can be given with a chocolate base and is almost tasteless. When the motions are very hard and are only passed after much straining I like to use the confection of sulphur, either with or without the confection of senna. Children take it well, and the sulphur has the property of softening the motion, so that hard straining is obviated.

You may ask, What about the use of local means, treatment by enemata, or treatment of constipation by material administered by the rectum? I think rectal administration has its sphere in some cases. In those I have referred to as having a deficient rectal reflex, where the rectum needs a more powerful stimulus before it will expel its contents, the old-fashioned method of inserting an oiled paper cone is valuable and harmless as an educator of the reflex. Or you may use a plain small warm-water enema. Otherwise local measures should be reserved for emergencies, for acute constipation, in cases in which the bowels have not acted for several days. In some cases of spastic constipation a warm enema, of water or oil, will give immediate relief from colic and will help to relax the spasm.

CONSTIPATION IN CHILDHOOD

Turning now to constipation in older children I would first remind you not to refuse to diagnose constipation because the mother tells you the child has a daily action of the bowels; it is always possible that what comes away to-day ought to have been evacuated yesterday or the day before and that the whole intestinal movement

is too slow. This may be spoken of as 'masked constipation.' The subjects of it are usually pale, lacking in appetite, with dark rings under their eyes, they are apt to have occasional attacks of fever with no obvious cause, and they may be subject to worms, especially threadworms. That type of child is not uncommon and always benefits from aperients, even though there seems to be a regular action of the bowels.

As to the causes of constipation in older children, there may here also be defective training, but in them **training** embraces more than in the infant. It means provision of suitable opportunities for the bowels to act. Late rising in the morning, hurrying over breakfast to get to school in time, and so on, leave no time for attention to the calls of Nature. In boarding schools I have known cases in which the trouble was due to lack of sufficient lavatory accommodation, a subject to which those who run those schools do not always pay sufficient attention.

An increasingly important factor nowadays is what is called **negativism,** a refusal by the child to allow the bowels to act for reasons largely psychological. It is very common in the solitary child of a family. This negativism may extend to the taking of food, and to sleep; there is a general refusal by these children to do what is expected of them, and that may lead to extreme constipation. I remember one case so bad that the mother sent a telegram to the effect that her child's bowels had not acted for ten days, it being suggested that an operation for acute intestinal obstruction was necessary. I took the child into a nursing home, and from the moment of entering the home the bowels acted well. The removal of the child from its mother caused negativism to disappear; she was an impossible sort of woman as a mother. These cases are getting increasingly frequent.

In these older children too, the **diet** may be wrong, but I do not believe that diet plays so important a part in constipation as is popularly supposed, and not many cases will be cured by attention to it alone. There may be several dietary defects. There may be a deficiency of water in the diet, or a deficiency of so-called 'rough-age'; or, on the other hand, this coarse food may be in excess. A common fault nowadays is excess of milk in the diet; for milk is a constipating food, largely because of its lime content. I always inquire carefully about the amount of milk taken, and often by cutting it down can cause the constipation to disappear. Older

children may have spastic constipation, showing itself, in them, in 'umbilical colic,' associated with irregular action of the bowel, constipation alternating with diarrhœa.

Lastly, I think there may be constipation as a result of excessive secretion of **mucus** in the bowel, in cases of what was formerly called 'mucous disease.' It is met with from the second dentition onwards. In those cases an excess of mucus seems to be produced, comparable to, though in less degree than, that in muco-membranous colitis in grown-up people. The mucus, by coating the fæcal contents, prevents the bowel getting a proper 'purchase' or grip on them, in other words, the contents are too slippery to be easily expelled.

And now as to the **treatment** of constipation in older children. Again it will depend on the cause. For defective training the treatment is obvious. I would only emphasize once more the importance of providing the child with regular times and suitable opportunities for action of the bowel to take place. That may mean a rearrangement of the whole life: it may mean getting up half an hour earlier in the morning than has been the custom, so that the child does not have to dash off to school after a hurried breakfast. The treatment of negativism is sometimes difficult so long as the child remains in its usual home surroundings: but it usually disappears as soon as the child is put into different surroundings. Your duty in cases of negativism is to treat the parent even more than the child. Hitherto the parents have been making a fuss about the action of the child's bowels, and the news has been eagerly awaited as to whether there has been an action or not. That is wrong: you need to inculcate into the parents an attitude of complete indifference; then, as soon as the child realizes that nobody is interested as to whether it has an action of the bowels or not, matters will begin to improve. This education of the parents, however, is not always easy.

With regard to defective diet, the treatment is obvious. In some cases there may be a need to increase the quantity of food, whether you call it 'roughage' or not. There is a popular belief that raw fruit is a good laxative: that, I think, is a mistake, but it is correct in regard to stewed fruit, especially apples and prunes; raw fruit often increases constipation. Give plenty of cooked fruit and vegetables, the latter preferably sieved. If there is an excess of milk, cut it down. If there is a deficiency of water, give drinks of water between meals.

symptoms of intestinal toxæmia and attacks of vomiting may ensue and be followed by exhaustion and death. On the other hand, in the less severe cases life may be prolonged even to adult age, although with greatly impaired health.

Treatment of megacolon by aperients and enemas is merely palliative. Hurst claims great success from dilatation of the anus and anal canal with a special conical bougie [1] which is passed into the bowel daily and kept in position for half an hour. After a week it is only kept in for a quarter of an hour and after a month is only passed on alternate days and then gradually discontinued. Such is the *medical* treatment of megacolon.

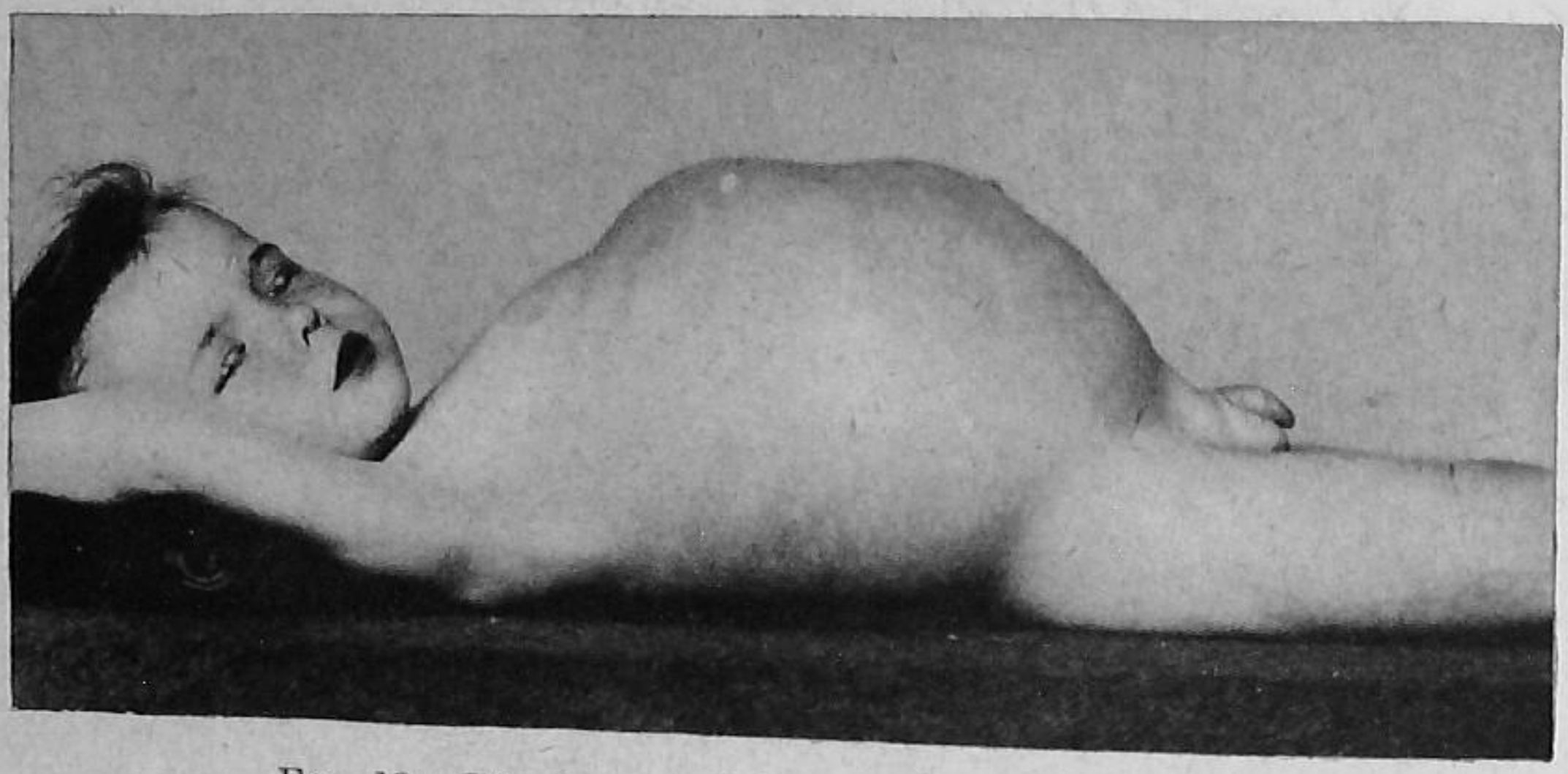

FIG. 13.—MEGACOLON (HIRSCHSPRUNG'S DISEASE).

Surgical operations on the bowel have now been given up in favour of division of the pre-sacral and inferior mesenteric nerves. This acts by abolishing the muscular spasm which is preventing defæcation. The operation is not a dangerous one and is said to give excellent results. More recently good results have been claimed from the administration of a spinal anæsthetic on *one* occasion: this seems almost incredible but the theory is advanced that it blocks certain nervous impulses and knocks out, as it were, the inco-ordination once and for all.

[1] Obtainable from Messrs. Allen and Hanbury.

LECTURE XII

WASTING

The terms malnutrition, dystrophy, atrophy, athrepsia, 'food disorders of infancy,' and marasmus, are used more or less indiscriminately in textbooks to designate that condition which is familiar to all of you under the simple name of 'wasting.' There is no need to describe in detail the clinical characteristics of the

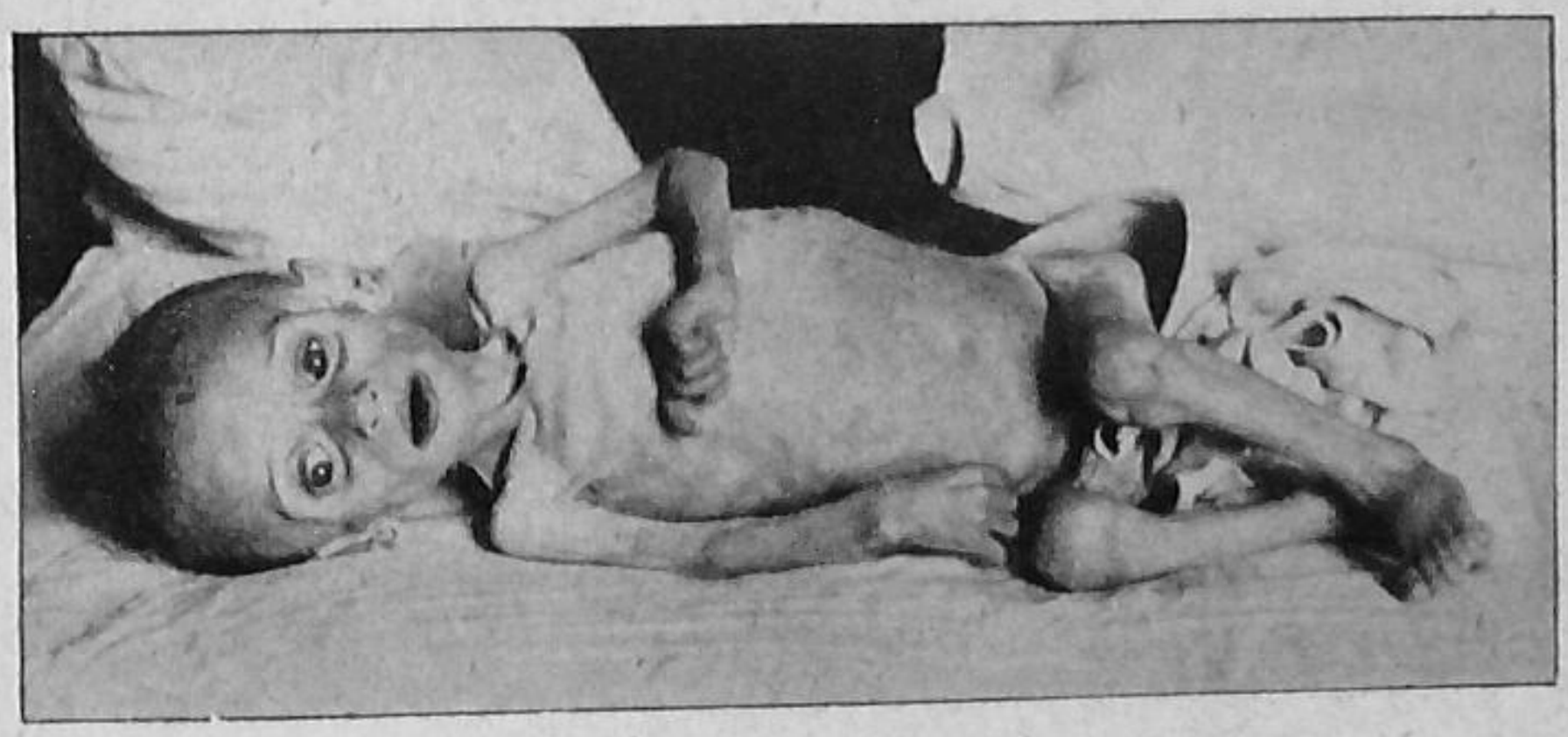

FIG. 14.—MARASMUS.

wasting infant; they are, unfortunately, only too familiar, although I think one may truthfully say for our comfort that the severe degrees of wasting are not so often met with as they used to be. None the less, the condition in greater or less degree is still common enough, and is a source of great trouble and anxiety in practice.

CAUSES OF WASTING

In order to have any success in the treatment of wasting it is important to realize that it may have many different causes, which may be classified as follows:

1. **Organic disease.**—Any organic disease, of course, is apt to be attended by wasting as one of its consequences, but we are now concerned only with those cases in which the wasting is what is sometimes called the 'presenting symptom,' and the signs of organic disease are inconspicuous or even altogether absent.

Naturally one thinks first of that great wasting disease of childhood—tuberculosis. It must be remembered, however, that tuberculosis is, as a matter of fact, very rare during the first six months of infancy, when wasting usually occurs. Nor is it likely to occur without producing some evidence of its presence in the shape of physical signs in the chest, abdomen, or glands, and X-ray examination together with a tuberculin test should enable the diagnosis to be made.

Congenital syphilis has also to be thought of, but here again I would remind you that, compared with the number of cases of wasting you will meet with, congenital syphilis is a rarity. Apart from the more florid cases of the disease in which wasting will not be the presenting symptom, it must be admitted that a syphilitic 'taint' may interfere with nutrition, and it is possible that some of the wasting infants who improve so remarkably under the administration of grey powder, as some of them undoubtedly do, may be affected in this way. If in doubt, a blood test should settle the matter.

Diseases of the lungs, such as chronic broncho-pneumonia and 'latent' empyema, may be unsuspected causes. The former is often curiously unobtrusive in young infants, and may be entirely afebrile so that its presence is only revealed by a few moist sounds in the lungs of doubtful significance. On the other hand, careful percussion ought to detect an empyema, provided it has been looked for. Chronic infection of the urinary tract, usually associated with some congenital abnormality, may be present, unsuspected unless the urine is carefully examined. I have here a specimen (fig. 15) from a baby aged only 5 weeks who lost weight despite an adequate intake of breast-milk. It was successfully removed at operation.

The only remaining organic cause to be borne in mind is congenital disease of the heart. Here again there will usually be signs on auscultation, but it must be remembered that every now and then congenital disease of the heart occurs without a murmur, and such cases are apt to be overlooked, although they may, in some obscure fashion, interfere seriously with nutrition. An X-ray may reveal an abnormal heart in such a case.

2. **Starvation.**—It is apt to be forgotten that wasting may be due to nothing more recondite than mere insufficient intake of food. This may be the result of pure *underfeeding*, either from ignorance or carelessness, or sometimes with the best intentions on the part

of the mother. Thus I once saw twins who had been fed for some weeks on barley-water *only*, and yet, curiously enough, they were not as wasted as might have been expected. A common cause is the use of condensed milk in too high a degree of dilution, or the giving of too small feeds.

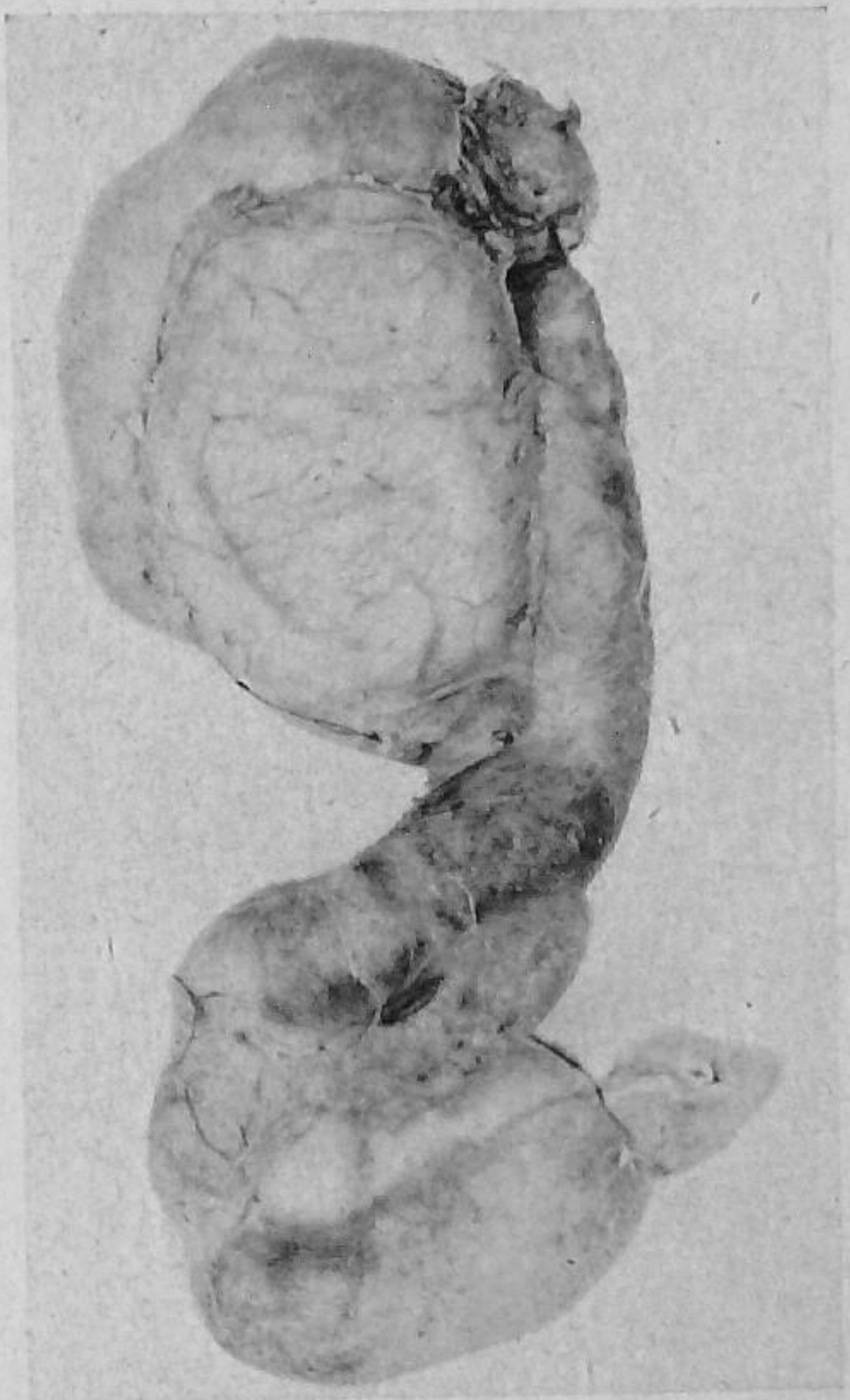

FIG. 15.—HYDRONEPHROTIC KIDNEY AND DILATED URETER REMOVED AT OPERATION FROM BABY AGED 5 WEEKS SUFFERING FROM SEVERE WASTING.

Inability to suck from some mechanical difficulty, such as hare-lip, facial paralysis, nose-blocking, or mere weakness, may obviously and naturally lead to insufficient nutrition. The same result ensues if sucking is painful, as in some cases of stomatitis, or if the hole in the teat of the bottle is too small.

A child for instance, was once brought to me a month old and weighing only 6 pounds 5 ounces. At first I could find no cause for wasting. A slight modification of the feeding was made, but at the end of a

week the weight was an ounce down. Still I could find no cause. Next week another 2 ounces were lost. I then looked at the teat, and found that the hole in it was very small. A new teat was ordered, and in a fortnight the weight had gone up 1 pound 8 ounces.

3. **Prematurity.**—Premature infants are very apt to become the subjects of wasting. There are probably several reasons for this. In the first place, the child is often too weak to suck properly, so that it gets an insufficient quantity of food. In the second place, it is probable that the premature infant is, metabolically speaking, an extravagant machine, and requires a large amount of fuel to maintain its body temperature, which leaves no surplus for the purposes of growth. In the third place, such infants have often but a feeble power of digestion and possibly also of assimilation. It is not to be wondered at, therefore, that with all these difficulties to contend with they often waste.

4. **Improper feeding.**—This is such a common cause of wasting that some people are apt to speak of wasting as if it were synonymous with the 'food disorders of infancy.' This, however, as we have already seen, is by no means always the case. None the less, unsuitable feeding is undoubtedly the starting-point of the majority of cases of wasting.

The error may be a *quantitative* one—as in the case of giving too small or too weak feeds, which we have already considered. Paradoxically enough, however, too large or too strong feeds may have a similar result, and a child will therefore sometimes start to gain weight when the amount or strength of the feeds is reduced. Far oftener, however, the error is a *qualitative* one, and consists in giving an ill-balanced mixture which is too rich in one of the food constituents, a relative excess of fat being the commonest fault. It should be noted that the want of 'balance' in the diet need not be an absolute one, but only relative to the digestive capacity of the particular infant. Some babies, for example, appear to have a congenital inability to deal with a normal proportion of fat in the diet, especially the fat of cow's milk, and in them a fat dyspepsia is easily set up. In other and less common cases there is a similar disability as regards casein or sugar.

Although a lack of balance in the diet may result merely in a failure to gain weight (the so-called 'disturbance of equilibrium'), it is very apt, if persisted in, to lead to the next great cause of wasting—

5. **Dyspepsia.**—This is so common, either as a cause or as a concomitant of wasting, that there are few marasmic infants who are not also dyspeptic.

The symptoms of dyspepsia may arise suddenly, as in cases of summer diarrhœa, and become persistent, leading eventually to chronic wasting; or, more frequently, the dyspepsia comes on gradually as the result of the continued use of an unsuitable or ill-balanced diet. Finkelstein, Czerny and Keller, and others have attempted to make a classification of the different varieties of dyspepsia according to the food constituent responsible for the digestive derangement, and Finkelstein has described their culmination in a condition to which he applies the ill-omened term 'decomposition'—which appears to be very much marasmus as we usually know it—and finally in a state of 'intoxication,' the symptoms of which appear to be indistinguishable from those of a severe case of infective diarrhœa.

His views have obtained a considerable amount of support both here and in America, but, although they are undoubtedly suggestive, I must confess for my own part that I am unable to reconcile them satisfactorily with the actual conditions seen at the bedside, and they appear to me to be an attempt to effect a rather arbitrary simplification of a very complicated subject. Be that as it may, there is no doubt that some guide to the nature of the feeding error can be derived from a study of the stools. Excess of fat in the diet is the commonest starting-point of a dyspepsia, and is indicated by constipated, pale, crumbly, or soapy stools which do not adhere to the napkin. Excess of casein—contrary to what was long supposed—is much less likely to be a source of difficulty, and when it occurs leads to vomiting of curd and the passage of tough white particles in the stools, which are insoluble in a mixture of alcohol and ether. Sugar is the most easily digested of all the food constituents, and least likely to lead to trouble; but if present in the food in an amount which is beyond the infant's digestive capacity the stools are apt to be loose, watery, and acid, and their passage attended by much flatulence. It is rare, however, to meet with pure examples of any of these different forms of dyspepsia. Usually the digestion of all the food constituents is impaired, and in severe cases it always is so.

The matter is further complicated by the fact that a secondary bowel infection may be grafted upon a simple dyspepsia, and the

green slimy stools of a true enteritis make their appearance. It must be remembered, too, that wasting of itself impairs digestive capacity, so that a case of marasmus which originated in, say, underfeeding may easily pass into a state of dyspepsia which accelerates the downward progress.

6. **Primary or idiopathic wasting.**—It is for primary or idiopathic wasting—that is to say, wasting of unknown cause—that the term 'atrophy' should be specially reserved. In cases of this sort there does not seem to be anything wrong with the food, and there are no signs of dyspepsia; but the child simply wastes, and after death no cause can be found for the failure of nutrition. Such cases are commonest in very young babies, and often the patient is the youngest child of a large family, or is a twin or illegitimate. We appear to be dealing here with a true failure of assimilation, but why it fails is quite unknown. I need hardly say that some 'endocrine' disturbance has not failed to be invoked as an explanation, but such a hypothesis is purely speculative. It is best to admit frankly that the true nature of such cases has so far baffled us.

Complications

However wasting may have come about, it is apt to become attended by complications of one sort or another. Thus such babies are—understandably enough—prone to infections by pyogenic organisms which cause boils and abscesses, by the tubercle bacillus, and by the *B. coli*. They are also apt to contract an infective enteritis or a broncho-pneumonia, or to develop otitis media. The clinical picture may further become blurred by the supervention of symptoms of acidosis or nephritis, or by the appearance of an 'idiopathic' œdema on the one hand, or of the signs of 'dehydration' on the other. It will thus be readily understood how protean the signs and symptoms exhibited by a wasting infant may be, and how they may vary greatly almost from day to day.

Prognosis

The prognosis in a case of wasting is always doubtful. Even when things seem to be going well at the moment, there is always the possibility of an abrupt and apparently inexplicable collapse which proves fatal. On the other hand, a sudden and seemingly spontaneous improvement may occur at any moment even in the worst cases.

More frequently, in the cases which ultimately do well, one finds a very gradual improvement checkered by many relapses. Ground is often gained more rapidly after teeth are cut. In fatal cases death is usually the result either of gradual inanition or of the supervention of an infection, a sudden food 'intoxication,' or of an attack of diarrhœa. In all cases the outlook is decidedly more rosy in private than in hospital practice. It is also better, curiously enough, in the cases in which there seems to be most wrong. If there is dyspepsia it may be possible to correct it, but the worst cases are those in which everything seems to be right except the power of gaining weight.

Treatment

The treatment of a case of wasting demands the highest degree of patience, optimism, and resource on the part of both doctor and nurse. At the outset it must be realized that the treatment is not merely dietetic—the general hygiene of the infant is of the first importance. Fresh air, sunshine (if obtainable), cleanliness, and *warmth* (especially keeping the feet and legs warm) are all great aids to success.

Nothing, probably, makes more difference than good 'mothering.' To look after a wasting infant is one woman's job, and he should not be left to lie alone in his cot, but should spend a good deal of his time in the mother's or nurse's arms; he wants plenty of 'cuddling' and amusing. Great care should be taken that he does not get chilled when being washed. The impossibility of providing all these desiderata in institutions is one of the main reasons, I believe, why these cases often do better even in a poor home. As to the all-important question of feeding, it is only possible to indicate some general principles.

You will do wisely, in the first place, to go slowly and play for safety, remembering that overfeeding is more perilous than underfeeding, and that one must not be too ambitious in the matter of increasing the weight. There has sometimes been a tendency to increase the strength of the feeds to a point which is beyond the digestive capacity of the infant, with the consequence that, though all goes well at first, a dyspepsia is soon set up which rapidly leads to wasting. My next advice is always to change the feeding reluctantly, cautiously, and never without good reason, bearing in mind that fat is the ingredient most likely to cause trouble. Before starting a new food it is well to clear out the bowels with a small

dose of castor oil, and if acute symptoms of 'intoxication' have supervened to suspend all food (except weak saline) for a few hours, but not for long, remembering that wasted infants stand starvation badly. If the food has been too rich in sugar, do not reduce the proportion of the latter too rapidly, or collapse may ensue.

Breast-milk is the best food in most cases, but is unfortunately rarely obtainable. In selecting an artificial food, begin with one which is rather poor in fat and relatively rich in carbohydrate, and whose protein is in a digestible form. Sweetened condensed milk (2 drachms to 3 ounces) or a half-cream dried milk (1 drachm to 1 ounce) with the addition of dextri-maltose fulfils these conditions. Some indications for feeding will also be furnished by the stools. If they point to severe fat indigestion, it may be necessary to feed on a mixture of whey and Mellin's food (dextri-maltose), or whey with the addition of a modified starchy food [1]; if casein is the difficulty, the milk should be fully peptonized or citrated, or a dried milk may meet the case.

In those—fortunately less common—cases in which there is sugar indigestion and 'intoxication' so-called 'protein milk' is often very useful.[2]

As to the size or intervals of the feed, you must feel your way in each case, and not be bound by too rigid rules, but the more wasted and exhausted the child is the smaller and more frequent must the feeds be.

Drugs are of little use in cases of wasting except to meet special indications such as flatulence, colic, etc. I have already pointed out, however, that grey powder sometimes does good even in cases in which there is no reason to suspect a syphilitic taint—possibly by correcting constipation or by stimulating the digestive secretions—and alcohol is also helpful, especially if there is much exhaustion with a subnormal temperature. It should not be added to the feeds, but given as a medicine and not too dilute. The administra-

[1] A useful formula is as follows: Take 2 flat dessertspoonfuls of Benger's or Savory and Moore's food and mix in a basin with 5 ounces of milk. Bring 15 ounces of whey (prepared with rennet) to the boil in a double saucepan and pour slowly on to the cold mixture, stirring meanwhile. Allow to stand covered up for thirty minutes, then stir and bring slowly to the boil; strain through a linen handkerchief, and the food is ready for use. Such a mixture is poor in fat and casein, but relatively rich in lactalbumin and carbohydrate.

[2] A dried form of protein milk is supplied by Mead, Johnson & Co. It is prepared for use by mixing 1 ounce of the powder with 11 ounces of water. The mixture contains about 3 per cent. protein, $2\frac{1}{2}$ of fat and $1\frac{1}{2}$ of sugar.

tion of thyroid, which is sometimes advocated, appears to me to be irrational. Injections of an extract of the suprarenal cortex have been claimed to be of value, but it is difficult to see why. A blood transfusion often starts the period of recovery on its way.

It is a common custom to anoint wasting babies with cod-liver oil. This is a dirty practice, the utility of which is in the highest degree doubtful. It is inconceivable that the child can obtain any appreciable amount of nutriment by such a method, and at the most it can only help to a slight degree in the conservation of body heat. Almond oil would do this as effectually and much less offensively than cod-liver oil, while adequate clothing is far more effective than either.

When all is said and done, you will lose a good many of your marasmic babies in spite of all your care and skill. Do not be cast down over it; the fault is not yours. It is a delusion to suppose that there is for every one of these cases some magic plan of feeding which, if one could only hit upon it, would immediately change the whole situation. On the contrary, the fault in many, perhaps most, cases is in the child and not in the food, and once the disorder has progressed to a certain length no treatment makes any dramatic difference; all one can do is to keep patiently 'pegging away,' but remembering always the first rule of therapeutics—not to do harm.

LECTURE XIII

CONGENITAL SYPHILIS

The first remark I have to make about congenital syphilis is that it is nowadays relatively a rare disease. The effect of war may lead to an increase, but official measures to encourage treatment of adults are bound to have some degree of success to offset this. Those of you who work in rural districts may never see a case of it. Even in the out-patient department of the Children's Hospital I saw it but seldom, and in private practice I have hardly met it at all. It is well for you to grasp this fact about the disease clearly at the outset, in order that you may have a proper sense of proportion in regard to it.

Congenital syphilis is also spoken of, as you know, as hereditary syphilis, and, more recently, as heredo-syphilis. There are some who would have you confine the term 'congenital' to cases in which the disease is acquired *in utero*, reserving the term 'hereditary' for those in which it is transmitted through the germ. But I do not think it is worth while to split hairs about these matters. The term 'congenital' has been in use for many years, and I propose in this lecture to continue to speak of 'congenital syphilis,' meaning by it, in general terms, cases in which the disease is derived from the parents, and not acquired by the individual in the usual way; the term 'heredo-syphilis' presents no advantages that I can see.

Mode of Transmission

I said 'derived from the parents,' but it is probable that the child always derives the infection from the mother, either by transmission through the placenta, which has been proved to take place when the latter is diseased, or, very rarely, by infection at the moment of birth during the passage through the genital tract. That, however, is very unlikely to take place, because the vernix caseosa, which covers the newly-born child all over, acts as a very efficient protective against infection.

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Now, it has long been known that the mother of a syphilitic child, although showing no signs of the disease in herself, yet has acquired an immunity. The statement of that fact was crystallized into what is known as Colles' law, meaning, as Colles put it, that the mother of a syphilitic child can suckle her infant without risk of infecting herself, whereas a wet-nurse would probably become infected. There is no doubt about the truth of Colles' law; it is confirmed by universal experience, but the curious thing is that the mother should show no evidence of what one can speak of clinically as syphilis.

Then there came the Wassermann test, and it has shown that the blood of mothers of syphilitic children reacts positively. That also, I think, you must accept as a fact, but how we are to reconcile the facts that the mother has an immunity to the disease and a positive blood reaction, and yet may show no clinical signs of syphilis, has not yet been explained.

Manifestations of the Disease

No matter how a child has become infected—whether the disease has been transmitted to it conceptionally, or via the placenta *in utero*—it may be born with all the evidences of syphilis full-blown upon it; but not often. Children who are as badly infected as that are not often born alive. More commonly, you will find that the syphilitic child is born in apparently good health, and then, after a space of two, four, perhaps six weeks—anything up to two months—things begin to go wrong, and the signs of the disease appear. Here, again, I shall not discuss with you now what is probably going on during that latent period when the infant seems to be in good health. If there was infection nine months before birth, or if infection occurred from the placenta, why such latent period at all? These questions will have to be gone into thoroughly in the light of our recent knowledge, and their solution will no doubt throw light on the pathology of syphilis.

Meanwhile I would lay stress on the fact that congenital syphilis shows one great difference from the acquired disease, in that there is no primary stage. In the acquired disease you see a primary sore and later there are the secondary and tertiary stages. But in the congenital form there is no primary stage, and the secondary and tertiary stages are mixed up; you will see a child presenting secondary and tertiary lesions simultaneously; a skin eruption, condy-

lomata, and gummata, all flourishing side by side—a thing which you very rarely see in acquired syphilis. For that reason it is not possible to describe congenital syphilis in stages in the same way as you can the acquired disease. It is more convenient, for purposes of description, to deal with the manifestations in the different structures of the body; for instance, as it affects the skin and mucous membranes, the bones, the viscera, and so on.

Marasmus.

Proceeding in this way, one of the first manifestations of the disease is **wasting,** or marasmus. The wasting, we may suppose, is simply the result of the action of the toxins on the tissues as a whole. It is not to be wondered at that the child wastes, and this wasting may be the only obvious sign of congenital syphilis. Sometimes you will find wasted babies who show no marked evidence of syphilis, but who put on flesh in a surprising manner when they are given grey powder; in such cases there may have been a slight syphilitic taint interfering with nutrition, but not enough to cause definite local lesions. The general nutrition, or general metabolism, is a very sensitive index of the presence of the syphilitic poison, perhaps a more sensitive one than any individual structure.

Skin eruptions.

Secondly, the disease may manifest itself in the skin and mucous membranes. Let us take first the skin. The **skin manifestations** are extremely important diagnostically; and one may speak of two varieties—(1) Superficial lesions; (2) infiltrative lesions. I will describe immediately what I mean by infiltrative. The superficial lesions are those which are most conspicuous to the eye, and these, again, you may divide into three classes—(*a*) Maculo-papular; (*b*) erythematous; (*c*) bullous. By maculo-papular I mean an eruption which is composed of little well-defined round spots or macules; sometimes, if the inflammation is sufficiently severe, these spots become slightly raised, and then they are known as papules. The *maculo-papular* eruption is one of the first to come out, and there are certain areas which it particularly affects, and where you should look for it. It affects particularly the eyebrows, the central part of the face round the mouth and chin, to some extent the neck, the flexor aspects of the arms, and the perineal region. The characteristics of this rash are very much those of the secondary rash in the acquired disease—that is to say, the spots are of a brownish-pink or coppery tint. At first the spots are separate from one another, but

later they may run into areas, and often you will find a slight branny desquamation on the surface of them. Such a rash, I say, is one of the earliest to appear, and usually it is seen within two months of the birth of the child.

Secondly, you may find an *erythematous* rash, and that is one which specially affects the ' napkin area,' as it is called, where there is almost constant moisture as there is under the napkin in a young baby. These napkin rashes will give you trouble in the matter of diagnosis, but let me give you one word of warning, and that is not to diagnose as congenital syphilis every case in which a child has got an eruption on the buttocks, because thereby you will be libelling numbers of innocent parents. Such an eruption may be an irritative intertrigo, the cause being the scalding of the sensitive skin by the urine or fæces. It is sometimes difficult to distinguish an intertrigo of this sort from the erythematous eruption of congenital syphilis. Here are the points to look out for : in ordinary intertrigo the rash is confined to the napkin area and those parts which touch it ; it does not wander up over the abdomen, but it may extend to the backs of the child's calves and to its heels, because these touch the napkin as it lies with its legs drawn up. On the other hand, a syphilitic rash may extend beyond those regions. Secondly, an intertrigo rash only affects the convexities in a fold of skin, but a syphilitic rash extends right down into the cavities or depressions. Further, a syphilitic rash tends to have a coppery colour, and it occurs early. If you see a rash on the buttocks of a child which is more than six months old, it is almost certainly not due to syphilis. Of course, I do not say that intertrigo may not occur at a younger age ; either may occur before six months.

Now, it is all very well to lay down rules like these, but I confess to you that I have difficulty in some of these cases where a child has an eruption on its nates in saying whether it is syphilitic or not. Sometimes you are compelled to give mercury and see what happens, or to do a Wassermann test.

We now come to the third type of skin eruption in congenital syphilis, the **bullous.** Here there are big blebs on the skin filled with watery or, it may be, slightly blood-stained fluid. The bullous is the most severe type of rash, and, fortunately, it is not often met with, except in the very worst cases, and then it is usually present at birth, and the child is otherwise in a very miserable condition.

A large proportion of such patients die, in spite of all you may do for them, because they are so badly affected with the disease. The only condition you may mistake the bullous form for is *pemphigus neonatorum*. The latter is not due to syphilis, but is a pyogenic or coccal infection of the skin. The differences between the two are these: that the child with pemphigus neonatorum is otherwise in good condition, not wasted, like the syphilitic child, and pemphigus neonatorum does not tend to affect the hands and feet, whilst syphilitic pemphigus does very markedly. The presence or absence of other signs of syphilis will also help.

By the infiltrative skin lesion of congenital syphilis I mean a degree of thickening of the whole skin, which sets in along with the wasting, and often along with the appearance of the maculo-papular rash, when the child is six weeks or two months old: and with this general thickening the colour of the skin changes, producing what is known as the *café au lait* tint, which is so characteristic. On the palms of the hands and the soles of the feet the tint is more often purple, with a glossy, polished, silky look; and by and by there is a tendency to peeling of the epidermis from the palms and soles. Remember, the infiltration goes on at the same time as the other lesions; there may be macules or papules, or erythematous areas, or bullæ, and, in addition to these, infiltration of the skin.

Affections of the hair.

At the same time the appendages of the skin are very apt to be involved, particularly the hair. In syphilitic children the eyebrows often disappear. When the eyebrows are gone and there is a coppery rash, the patient presents a very characteristic appearance. The hair of the head also is often peculiar. There is a tendency for it to be long and fine—more like fur than hair—and standing up from the surface of the head. To this the term 'syphilitic wig' has been applied (see fig. 16). You must not, however, suppose that every child with hair like this is syphilitic; there are some nationalities,

FIG. 16.—THE 'SYPHILITIC WIG.'

and perhaps families, which show a tendency to the same sort of hair without having congenital syphilis.

Rhinitis and laryngitis.

The mucous membranes are affected sometimes even earlier than the skin. The one which is most conspicuously attacked is the mucous membrane of the nose, producing that form of syphilitic rhinitis which expresses itself clinically in 'snuffles.' It is an inflammation of the membrane covering the turbinates and the

FIG. 17.—CONGENITAL SYPHILIS, SHOWING SCARRING ABOUT THE MOUTH.

septum, and is accompanied by the pouring out of a quantity of sero-purulent secretion, which sometimes may be blood-stained. The swelling of the nasal mucous membrane causes an obstruction to the entry of air, so that these children make a curious snuffling noise when breathing, and are difficult to feed, because they cannot suck comfortably. If the inflammation is sufficiently severe and extensive, there may be destruction of cartilage, and then there may be falling-in of the nose, leading to that pug-nosed appearance which is characteristic of bad cases of syphilis of the congenital form in adult life.

The rhinitis sets in early, about the second or third week after birth, and what you are most apt to mistake it for is either an ordinary cold or congenital adenoids. If adenoids are truly congenital, the snuffling is noticed actually at birth, but it is rarely so with congenital syphilis, and in the latter event it is unusual for other evidences of syphilis to be absent. Once again it is necessary to warn you not to diagnose syphilis because a young infant is 'snuffly.' The commonest cause of this is the common cold.

Frequently the mucous membrane of the larynx is also to some extent involved, and then you get the hoarse cry which, with the snuffling, is so characteristic of syphilis. Ulceration about the angles of the mouth is also common, and sometimes the scarring from it persists into adult life.

Condylomata.

Condylomata (fig. 18) do not as a rule occur until later, when the child is about two years old, and then they are usually an indication of relapse, the result of neglect of treatment. The condylomata may be very florid and marked, and the mother may say that the child has got 'piles,' but remember that children hardly ever suffer from true piles.

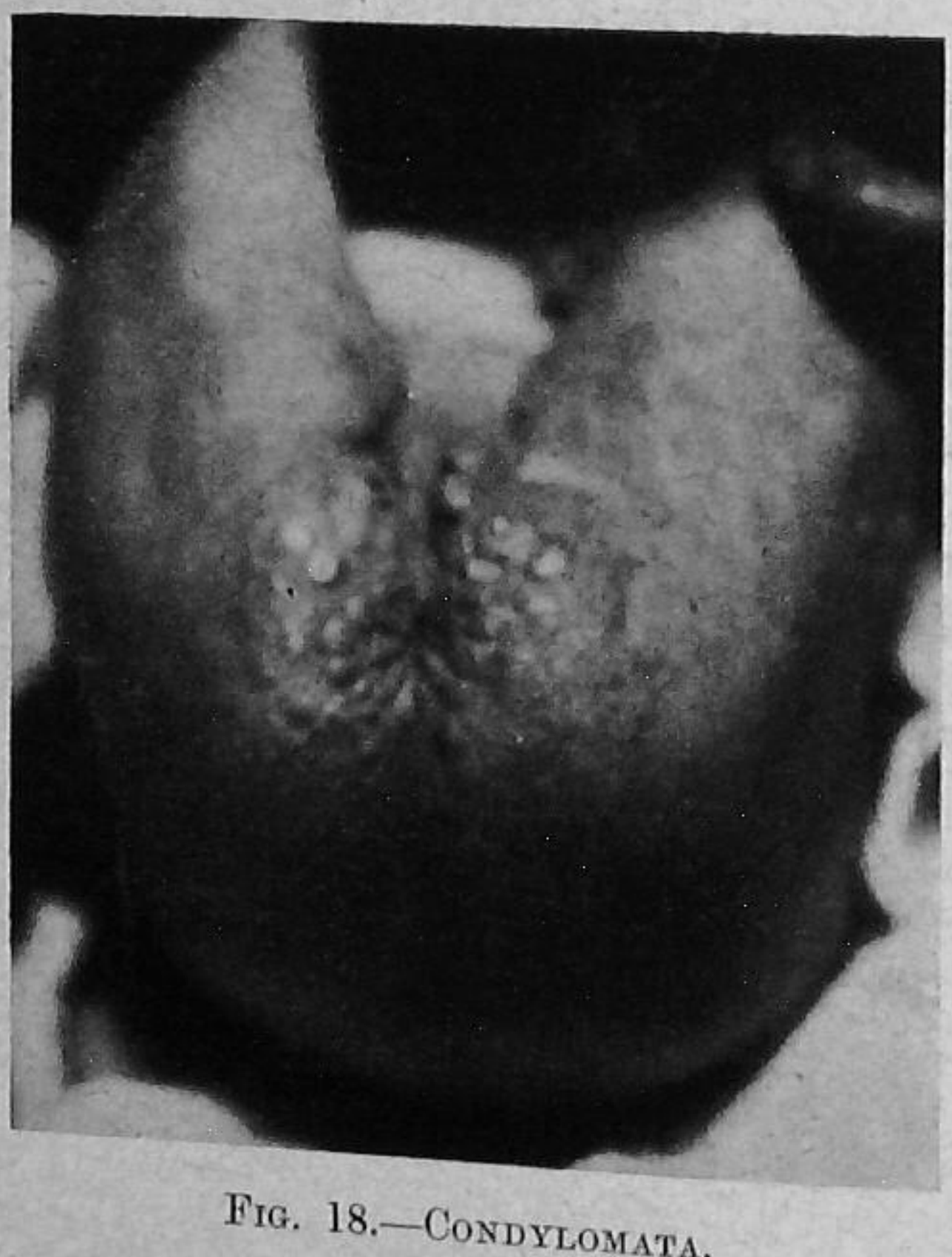

FIG. 18.—CONDYLOMATA.

Signs in the skull.

Next we come to a set of manifestations which are often very conspicuous in children—namely, the bony lesions. First let me speak of these as they affect that skull. There is apt, in the first place, to be a slight degree of hydrocephaly, the head being somewhat too large. But please do not regard congenital syphilis as a cause of severe degrees of hydrocephalus ; that is certainly not true. Along with this the fontanelle is somewhat open and bulging, and you will often find some ' lipping ' or thickening of its edge. There may also be what is known as *cranio-tabes*—little areas of the skull

FIG. 19.—ENLARGEMENT OF VEINS OF THE SCALP IN CONGENITAL SYPHILIS.

where the bones are not hard, but yielding like parchment, especially behind the mastoid. Cranio-tabes, however, is not pathognomonic of syphilis ; it occurs also in the subjects of rickets. Further, the skull may show what are called Parrot's nodes. Parrot described bosses over the frontal and parietal regions, which he said were due to congenital syphilis ; and if these are very marked, they give rise to what has been called the ' hot-cross bun head.' It is certain that these are not always due to congenital syphilis ; there is no doubt that head bosses occur in children who have got rickets only. So like cranio-tabes, head-bossing must not be regarded as conclusive evidence of syphilis. Finally, you will often notice large

veins coursing upwards on the scalp which are undoubtedly of diagnostic value (fig. 19). How they come to be there I shall not discuss with you because it has not yet been clearly explained.

Affections of the bones and teeth.

Passing on, we may look next at the manifestations in the long bones, and first at so-called '*syphilitic epiphysitis.*' The term 'epiphysitis,' as applied to this condition, is not quite accurate; it would be better to call it 'osteo-chondritis,' because the whole length of the bone is affected; there is a periostitis involving the entire shaft. The inflammation starts, however, at about the line

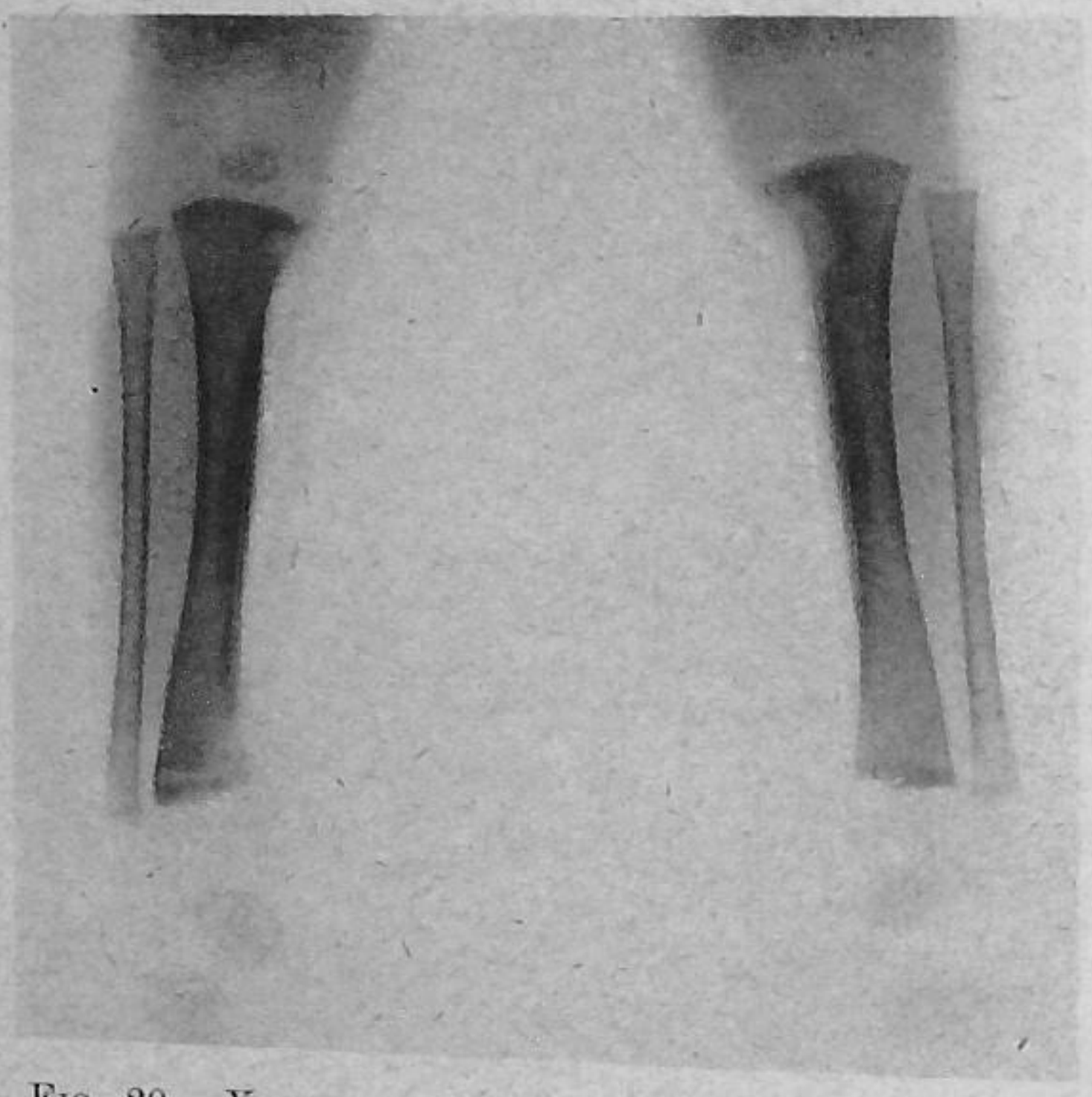

FIG. 20.—X-RAY OF LEGS IN CONGENITAL SYPHILIS.
Note periostitis of tibia and characteristic involvement of upper end on the inner side of the shaft of the tibia.

of the epiphysis, and seeing that the muscles are attached to the bone there, and pull upon the periosteum, the swelling is most marked at that point: hence the term 'epiphysitis.' Syphilitic epiphysitis occurs early, about the sixth week, and I do not think it ever occurs after the sixth month. It tends to affect the upper limbs far more than the lower and usually both limbs symmetrically. It often shows itself in an apparent paralysis, the child being unable or unwilling owing to pain, to move the limb; hence the term 'syphilitic pseudo-paralysis' has been applied to it (see p. 303). It rarely goes on to suppuration. The X-ray appearances are diagnostic (fig. 20).

Another affection of long bones which you may find is **periostitis.** This occurs more nearly at the sixth year than the sixth week; and it may either take the form of localized nodes on the bone, or the whole length of it may be involved, and then you find a general thickening, often best marked in the tibia (sabre-shaped tibia).

Another affection of the long bones is **dactylitis,** which is met with usually about the second or third year. It generally affects several phalanges at once. It closely resembles tuberculous dactylitis, but the latter more often affects the metacarpals as well. It can be distinguished by X-ray examination because syphilitic dactylitis is an affection of the outside of the bone, it is a thickening under the periosteum, whereas tuberculosis affects the interior of the bone.

There is also a condition called **syphilitic osteo-myelitis,** which it is necessary to mention for the sake of completeness. This is a gummatous process involving the whole thickness of the bone, and is more a surgical disease than a medical, because what it most resembles is sarcoma. The bone round the central mass may be brittle, and sarcoma may in this way be closely simulated.

Relating to the bony lesions of congenital syphilis is the curious symmetrical *synovitis* of the knee joint, producing what is termed 'Clutton's joints.' This usually happens when the child is six or over and is remarkable for being painless.

Along with the bones one has to consider the **teeth.** The teeth are often affected in syphilis, but it is only the permanent teeth which are involved. There is the notched tooth (Hutchinson's tooth), the dome shaped tooth, and so on. I need not describe them in detail, because no doubt you are familiar with them already. Remember the milk teeth are not involved. One has known students identify Hutchinsonian teeth among the temporary incisors!

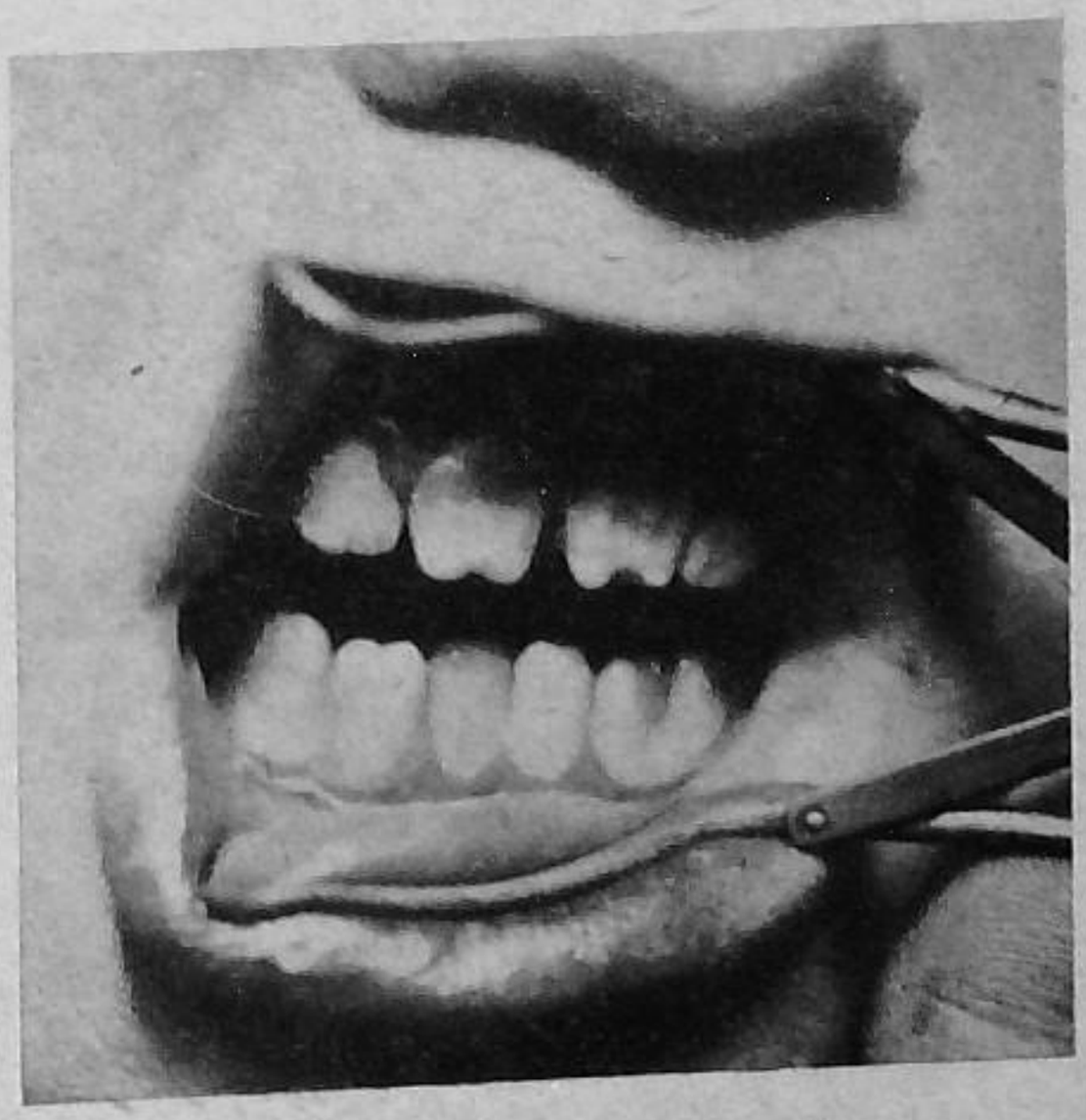

FIG. 21.—HUTCHINSONIAN TEETH.

Visceral manifestations.

The viscera chiefly affected by syphilis are the spleen, the liver, and the kidney.

The **spleen** may be enlarged quite early, the enlargement being due mainly to fibrous thickening of the trabeculæ. A palpable spleen is thus confirmatory evidence of syphilis in the early stages. At an early stage, too, there may be an infiltration of the liver by

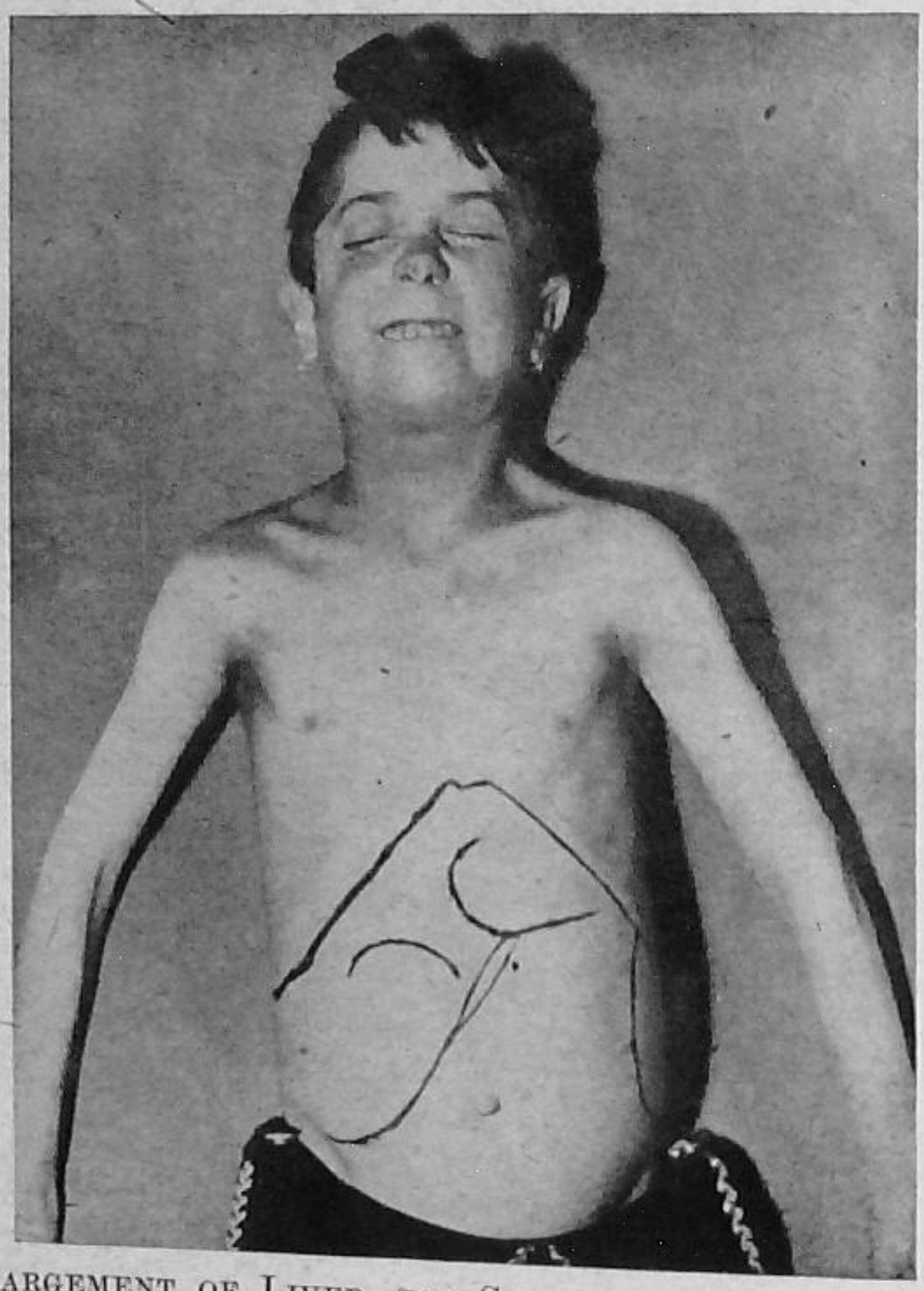

FIG. 22.—ENLARGEMENT OF LIVER AND SPLEEN, THE RESULT OF CONGENITAL SYPHILIS.
Note also the notched incisors.

small round cells—a diffuse **gummatous hepatitis**—causing the liver to be enlarged and producing a slight jaundice, rarely causing ascites, though I have known it do so, and tending to disappear under treatment. In later childhood there may be a true **cirrhosis** of the liver, of a coarse kind, the spleen being enlarged at the same time (fig. 22). That is the true syphilitic cirrhosis (see p. 399).

In regard to the kidney there is no doubt that in the early stages of syphilis an **acute nephritis** may occur which is probably often overlooked; it occurs at the same stage as the skin eruptions. In young babies the urine is difficult to collect, but if it is examined

one finds more frequently than would be expected the presence of albumin and a few casts, indicating a slight degree of nephritis. In the later stages, although it is rare, you may find a **cirrhosis of the kidney,** just as there is cirrhosis of the liver, and giving very much the same symptoms as cirrhotic kidney in the adult.

Paroxysmal hæmoglobinuria is sometimes regarded as a renal manifestation of congenital syphilis, though perhaps not quite correctly, because it is not strictly an affair of the kidney. There is no doubt, however, that paroxysmal hæmoglobinuria is often associated with an hereditary syphilitic taint, and since the Wassermann test came in it is surprising how many of the subjects of paroxysmal hæmoglobinuria have been found to give a positive reaction.

Amongst the affections of viscera one should mention **orchitis,** a swelling of the testicle which is apt to be overlooked because it is painless. This may occur fairly early, although, like condylomata, it is often a sign of a relapse from insufficient treatment.

Affections of the nervous system.

Compared with those seen in the adult who has acquired syphilis, the nervous affections in congenitally syphilitic children are infrequent. For some reason you do not often find, in the latter, gumma of the brain. Syphilitic meningitis has been described, but it is rare. Still more rare are the late syphilitic affections of the nervous system. You know how common tabes and general paralysis are in the adult, but these are very uncommon in the child. There is, however, a juvenile form of general paralysis of the insane which is one of the late manifestations of inherited syphilis, but it is very seldom met with.

Is mental deficiency often the result of syphilis? I should have said a few years ago, with confidence, that mental deficiency was a very rare consequence of inherited syphilis. But recent investigations in imbecile institutions with the Wassermann test have shown that a surprising number of the mentally defective give a positive reaction. One must not, however, in view of this jump to the conclusion that congenital syphilis is a frequent cause of mental deficiency, for, of course, evidence of that sort is of no value until we know what percentage of the children picked at hazard off the street give a positive reaction to the Wassermann test; we want, in other words, to know what is the percentage of congenital syphilitics among the population at large, and particularly in those classes

from which imbeciles are largely drawn. If we find among imbeciles a higher percentage of positive Wassermann reactions than in an equal number of children taken from the streets, we may begin to say that syphilis produces mental deficiency; but, meanwhile, I should say, from clinical experience, that congenital syphilis does not tend to cause mental deficiency to any extent. That is the consensus of opinion among clinicians, and I entirely agree with it.

Late syphilis.

Lastly, one has to mention among the late manifestations of congenital syphilis what is known as **syphilis tarda,** manifestations which come on in children who may have hitherto shown no signs of the disease at all, or where the signs have only been very slight. They are, chiefly, affections of the eye (interstitial keratitis), of the ear (deafness), of the throat and nose (ulceration). These fall into the spheres of different specialists, so I shall not deal with them further, but merely remind you of their occurrence.

Treatment

The best form of treatment is *prevention*, and in no disease in childhood is prevention so effective. It consists in treating the mother during pregnancy. A full course, along modern lines, of anti-syphilitic treatment should be carried out as early as possible, but even if the diagnosis in the mother is made late it is worth giving as much treatment as time permits.

As regards the treatment of the child suffering from syphilitic infection, there have been changes in the course of years. Mercury used to be the sheet anchor, and even with modern methods I am reluctant to omit it. You may use it either in the form of grey powder ('hyd. c. crete,' half a grain three times a day) by the mouth, or by inunction. Children stand grey powder very well. You may find it produces a little diarrhœa, but in that case you should add to each dose half a grain of Dover's powder. When giving mercury by inunction, rub in a piece of blue ointment about the size of a pea night and morning, choosing a different site each time so as to prevent irritation of the skin. Probably mercury should be given for at least a year whether the manifestations of the disease have disappeared or not. Nowadays it is considered doubtful if mercury alone can really eradicate the syphilitic infection, so you will have to consider *arsenic* in addition. The value of the organic arsenicals in this disease (e.g. neo-salvarsan) is well established, but you are at once faced by

the technical difficulty of giving it to babies and even to older children by the customary intravenous route. Hence it became the practice to use a suitable preparation for deep intramuscular injection. Even this was an unsatisfactory method, and I think the time has come to replace it by the oral route, using the preparation called acetarsone (spirocid, stovarsol). I say the time has come, because in recent years this method has been extensively used in different parts of the world and the reports show conclusively to me, at any rate, that it is most effective. Acetarsone is made in 4-grain tablets. Begin cautiously with $\frac{1}{2}$ to 1 tablet daily and work up in a week to 4 tablets daily. Give this for ten days, then miss four days, and give another short course for ten days at the full dosage. Go on after another four days giving seven short courses in all to make a full course. Examine the urine at intervals and at the end of the course for albumin. If this occurs, or any other symptoms of a toxic action (fever, vomiting, diarrhœa or a toxic rash), stop for a few days and start again cautiously. A second full course is necessary after an interval of four to six weeks. Give mercury as well all the time, as already indicated ; by inunction will be the better way. The acetarsone tablets can be given crushed up in a little water before or after meals.

For the eruptions you may use a calomel dusting-powder (equal parts of starch and calomel), and for the rhinitis wash out the nose with an alkaline lotion, and afterwards instil into it, by means of a dropper, a few drops of black wash (lotio nigra). Other local manifestations respond promptly to the general treatment employed.

There are only three questions remaining to be discussed :

(1) Is the congenitally syphilitic child a cause of the spread of the disease ? I do not think it is to any extent. I cannot recall an instance in my own experience where I was sure that syphilis had been conveyed to another (healthy) person by a congenitally syphilitic child. I do not say it cannot happen : it does happen sometimes ; but congenitally syphilitic children are not nearly so dangerous to the community at large as you might suppose. Why that should be, seeing that the disease appears in a florid form in these infants, it is difficult to say ; but it is a fact. At the same time, it is wise to take reasonable precautions, such as insisting that the feeding utensils shall be reserved for the affected child alone, that indiscriminate kissing is not indulged in, and so on. But do not regard the congenitally syphilitic child as a sort of leper, who

will convey the disease to everyone who comes near it. That is an exaggerated view.

(2) The second question is, Is an individual who has had congenital syphilis protected from the acquired form ? In other words, Can a congenital syphilitic acquire syphilis ? There appears to be no reason to doubt that the congenital form of the disease does protect up to a certain age, probably up to puberty, but not throughout life. It is possible for the congenital syphilitic to acquire the disease later on, but during the early years of life, at all events, he is immune. That appears to be the general view.

(3) Thirdly, Does the disease ever descend to the third generation ? To this the reply is that such an occurrence is so rare that it need not be reckoned with in practice. I have myself known only one clear instance of it.

LECTURE XIV

TUBERCULOSIS IN CHILDHOOD

Tuberculosis is still one of the great killing diseases of childhood although less so than it was even a few years ago. The mortality from it in the earliest months of life is small, so much so that you should be particularly cautious about diagnosing tuberculosis in a child in the first three months after its birth, but as life goes on the incidence of the disease rises very rapidly, reaching its maximum at about the second year. After that age it declines. The great frequency of tuberculosis in early life is shown by the post-mortem statistics of children's hospitals, but also by the result of the Mantoux test which has brought out the fact that by the twelfth year of life nearly 50 per cent. of children have had to deal with the tubercle bacilli.

You may take it that the susceptibility is at its greatest when the child is about a year old, and a child who becomes infected at or below that age is almost certain to develop the disease. As age goes on, infection is less apt to mean the development of active tuberculosis, either because the very susceptible individuals have been killed off or, what is also possible, there is some degree of immunity established as the result of repeated slight infections, so that it requires a massive dose, or exceptional conditions, such as diminished vitality on the part of the child, to give the bacillus a chance of establishing itself and producing the disease.

PATHS OF INFECTION

The next point we have to consider is the **path of entry**—i.e. how a child becomes infected with tuberculosis. Putting aside infection through the skin or the placenta, both of which do occasionally occur, infection always takes place through a mucous membrane: either the mucous membrane of the respiratory tract —which is the commonest mode of entry—or of the alimentary

tract, which is the next commonest route, or of the naso-pharynx, including the middle ear. I would further point out to you that the entry of the bacillus through a mucous membrane is rendered much easier if that mucous membrane is, or has been, the seat of catarrh. Chronic catarrhs of the mucous membranes predispose markedly to infection by tuberculosis; chronic bronchitis, for instance, or chronic diarrhœa. That is a very important point in regard to prevention, because one of the chief things to do if you wish to prevent a child developing tuberculosis is to guard carefully against catarrhs of mucous membranes; or if catarrh has been established, as, for instance, after measles, whooping-cough, and so on, to make sure that convalescence is as complete as possible. This is best done by getting the child away to the seaside until its health is thoroughly re-established.

The next question concerning the mode of infection which we have to consider is whether it is the **human or the bovine type** of tuberculosis which is most to be feared in childhood. There was at one time an impression that the bovine type of the bacillus was particularly apt to attack children, the theory being that they got the infection through drinking cow's milk. Much work has been done on that subject, and, although the evidence is, to some extent conflicting, I think it is now pretty generally agreed that quite a minority of cases of tuberculosis in childhood are due to the bovine bacillus. In other words, by far the greater number you will have to deal with are cases of infection of the ordinary human type. Further, I think it is also generally agreed that the frequency of infection with the bovine bacillus varies very greatly in different localities. There is reason to suppose, for instance, that in Edinburgh and its neighbourhood this form is unusually common. It is also pretty generally agreed that the bovine type of infection tends to produce what is spoken of as surgical tuberculosis—that is to say, affections of glands, bones, and joints rather than the form of the disease which particularly concerns us just now, that affecting the internal organs. In addition, it may be said that the bovine infection is milder in character, as a rule, than infection with the human bacillus. Nevertheless, it is calculated that there are 2,000 deaths annually in this country from the bovine type of tuberculosis. This danger could be entirely eliminated if all milk were pasteurized, as has been done in other parts of the world. In any case, as has been said in an earlier lecture, milk should be boiled

for children under two years of age. The argument that small doses of tubercle bacilli in mildly infected milk might be a good thing as possibly setting up immunity is very dangerous. Possibly dead tubercle bacilli can do this without any risk and there will be plenty of these in boiled milk!

I would again emphasize the fact, however, that the vast majority of cases of tuberculosis in children which you will have to deal with are of the ordinary human type, and it follows that one of the chief means of preventing the infection of children is to guard them from contact with phthisical adults. The disease in the adult may be unsuspected until a child in the family circle dies of tuberculous meningitis. The old person with an apparently simple bronchitis may have tubercle bacilli in the sputum. Some authorities go so far as to say that tuberculosis is as infectious to young children as measles is, and that if a child is brought for any length of time into contact with a phthisical adult it is as certain to develop tuberculosis as a child brought alongside a case of measles is to take that disease. Whether that be true or not there can be no question as to the great risk which children run when brought into close contact with persons suffering from tuberculosis, and attention to this is one of the chief parts of your duty in the matter of prophylaxis.

SYMPTOMS OF TUBERCULOSIS

First, I want you to distinguish between infection by the tubercle bacillus and the actual development of tuberculosis. The larger proportion of people—probably most of us—have been infected with the tubercle bacillus, but all do not develop tuberculosis; in the majority the disease is arrested at the outset. It may even be that many people successfully deal with a small dose of infection without ever developing even a 'scar' at the seat of the conflict. Such people will, however, react to tuberculin as I will describe shortly. The symptoms of infection are not definite; they may even be entirely overlooked. Often all that happens is that the child is a little 'off colour'; it refuses its food, and is fretful; the weight either falls or fails to increase, and if tested it will probably be found that there is a slight rise of temperature at night. In the majority of cases the local defences of the body overcome the onslaught, and the process goes no farther; the disease is arrested, usually in the lymphatic glands. It would seem that the bacilli

are sometimes able to pass through a mucous membrane without producing a local lesion, and that they go straight to the adjoining lymph-glands. The infection is there either completely overcome and goes no farther, the tubercle bacilli being killed off, or, what sometimes happens, they lie latent in the lymphatic glands, awaiting a future opportunity to become active. Such opportunity is afforded by any decline in the child's health, no matter how brought about. That is perhaps why tuberculosis so often follows upon some other debilitating disease. It is not that the child has become freshly infected with the bacilli, but the bacilli which have been lying latent have seized the opportunity afforded by the diminished resistance of the body to become active again.

Tuberculin tests.—As already indicated, one effect of invasion of any part of the body by the tubercle bacillus is the development of heightened sensitivity to tuberculin. This has a certain value in the diagnosis of the disease and I propose to discuss briefly the tests available. *Subcutaneous* injection is not much used in human subjects nor is the *ophthalmic* reaction. Both of these tests are too dangerous. On the other hand *intracutaneous* injection of tuberculin is of definite value and is free from danger. The commonly-used Mantoux reaction consists of injecting 0·1 c.c. of a freshly prepared solution (1 in 1,000) of old tuberculin. A positive reaction becomes maximum in about 48 hours and forms a red, raised swelling about 1 inch in diameter. In general there is no need to use a control test and there is no redness or swelling in negative cases except for a transient small area in some patients. The *Moro* test is employed by rubbing in an ointment containing tuberculin: the positive result in this test is not so clear cut as with the Mantoux test. The *patch* test is coming into favour and consists in fixing to the skin of the chest or back a small piece of adhesive plaster on which are three pieces of gauze, two soaked in tuberculin and one serving as control. After 48 hours the plaster is removed and 48 hours later the test read. To sum up the value of these tests it may be said that as a child gets older there is increasing cause to expect a positive result for obvious reasons. Hence a positive result is of most value in the young child, whereas a negative result, unless the child is suffering from an acute form of the disease, is always of value in excluding tuberculosis.

Allergic phenomena.—It is not altogether clear what value the heightened susceptibility has as a part of the body's resistance

to tuberculosis. Whatever is the explanation, however, there are certain by-products, so to speak, which must be noted. For example, an eruption in the legs, *erythema nodosum*, consisting of raised, red, painful areas, about 1 inch in diameter, may occur as allergy to tuberculin develops. Not all cases of erythema nodosum are due to tuberculosis, however, but the disease always calls for careful investigation as to the possibility of tuberculous infection. Another similar lesion modified, as it were, by local conditions is found in the eye—*phlyctenular conjunctivitis or keratitis* (fig. 23). Occasionally one finds a similar exaggerated reaction in the lung itself, when all the clinical and radiological appearances of a lobar

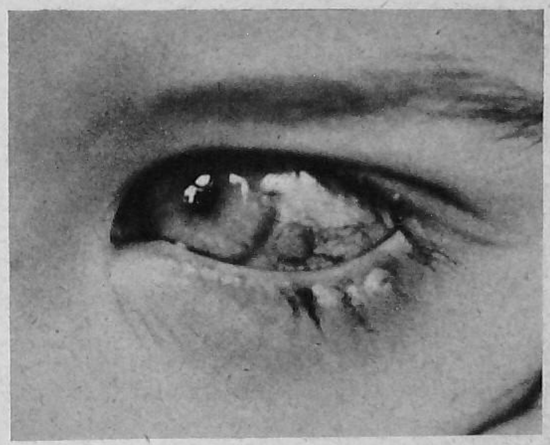

Fig. 23.—Phlyctenule in Left Eye in Child of 3 years.

Both parents have pulmonary tuberculosis. Child's tuberculin reaction strongly positive. Suspicious area in right lung on X-ray examination.

pneumonia (but with no fever) are found, and this has been termed *epituberculosis*. It is possible that pleural effusion may also be an allergic phenomenon. The importance of the occurrence of many of these allergic disturbances is that they often lead to the discovery of an unsuspected case of tuberculosis in the child's family circle and further damage to other children may be avoided.

General diagnosis.—Before coming to the localized forms of tuberculosis in children I should like to point out that the disease may affect many parts of the body and a careful search must always be made. For example, if the case begins with erythema nodosum on the legs it is necessary to X-ray the lungs and if this is negative also the abdomen and even the neck (fig. 24). Evidence of calcified

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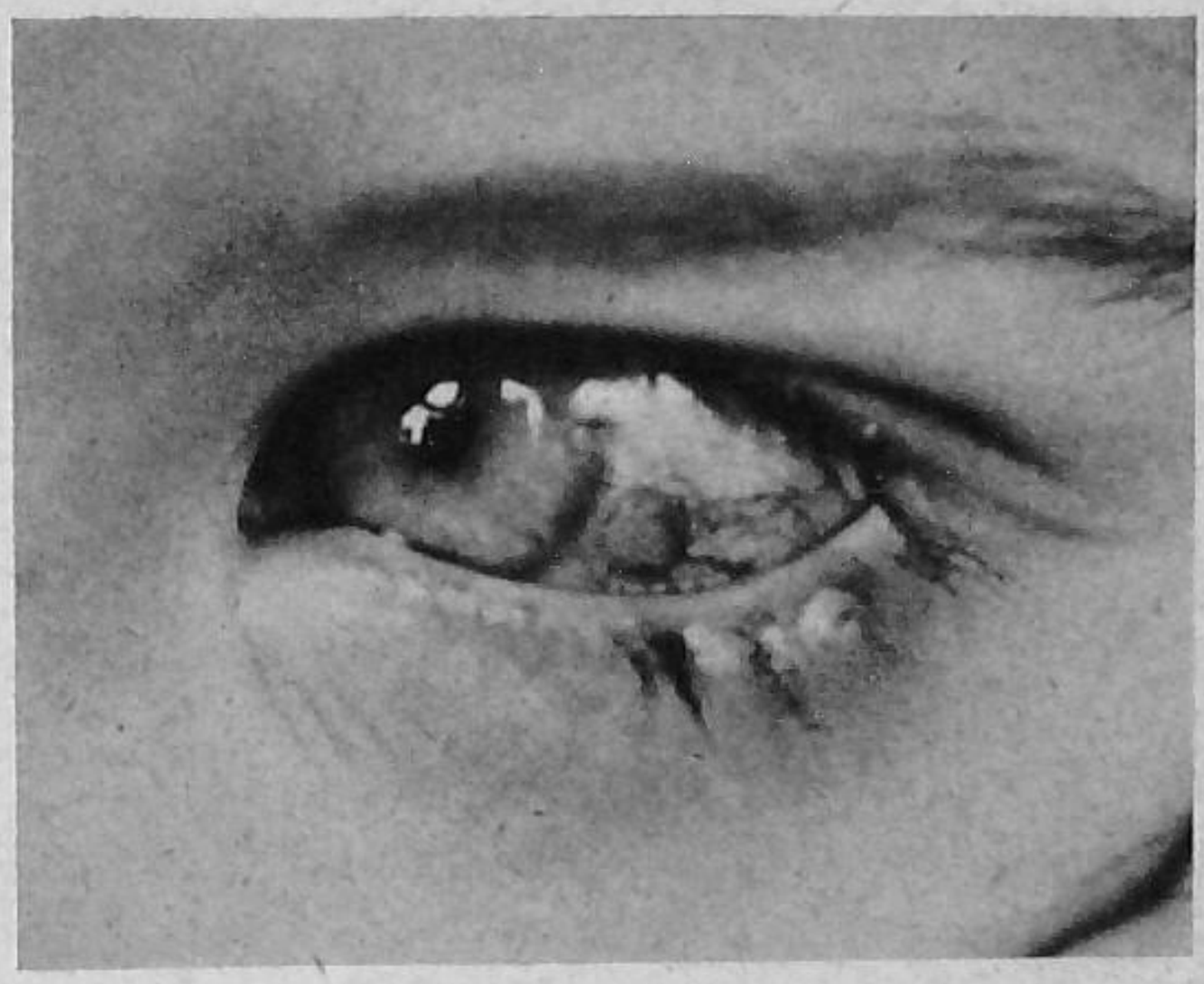

FIG. 23.—PHLYCTENULE IN LEFT EYE IN CHILD OF 3 YEARS.

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glands in these areas may be of great value in establishing a definite diagnosis since so often in children tuberculosis is a ' closed ' disease and the finding of tubercle bacilli in excreta—the final test after all in this disease—is not so likely as it is in adults. In a doubtful case of meningitis, X-ray of the chest often shows miliary tuberculosis and establishes the diagnosis even when tubercle bacilli are not found in the cerebrospinal fluid.

When the disease has become active, one can recognize various types of it, and what these are depends on the site of the chief

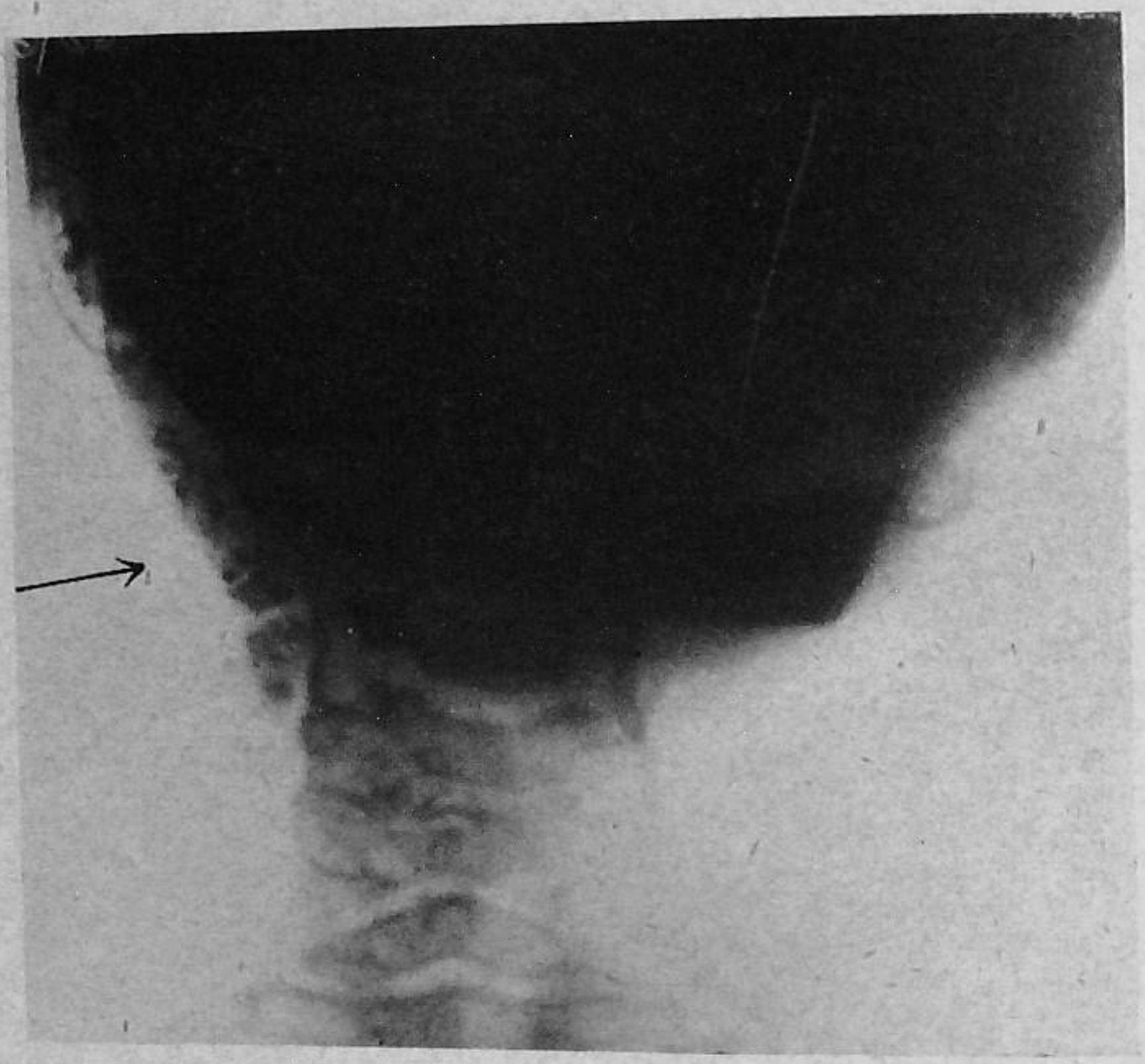

FIG. 24.—CALCIFIED GLANDS IN NECK.
Chain of glands indicated by arrow.

incidence of the process. Proceeding in that way, you may distinguish (1) general or miliary tuberculosis; (2) tuberculosis of the bronchial glands; (3) pulmonary tuberculosis; (4) abdominal tuberculosis; and (5) meningeal tuberculosis. These types are convenient for clinical purposes, but do not regard them as sharply demarcated. Thus, along with tubercle of bronchial glands there may be pulmonary tuberculosis, or a case of pulmonary tuberculosis may proceed almost unnoticed for some time until it ends up in meningeal tuberculosis, and so on.

Let us now consider, briefly, the symptoms and signs presented by each of these different types.

GENERAL TUBERCULOSIS

In many cases it is extremely difficult to diagnose this form, because there are no physical signs which are absolutely characteristic of it. You find the usual evidences of tuberculous infection: wasting, more or less marked; a rise of temperature, sometimes to quite a high level; fretfulness and general malaise; but you do not usually get marked physical signs. Indeed, miliary tuberculosis in a child may run its whole course and prove fatal without having produced any definite physical signs at all, the reason being that so long as the tubercles are not broken down they give no evidence of their presence. The lungs, for instance, may be stuffed with miliary tubercles without there being any physical signs by which you can detect them. They are too small to produce dullness on percussion, and until they break down there are no moist sounds on auscultation. Still, there is often something in the appearance of the child which is suspicious of tuberculosis. Many of them have a pink-and-white hectic complexion, with long eyelashes and a tendency to develop a lot of downy hair, particularly on the back.

Another suspicious sign is the presence of small shotty glands, especially on the inner wall of the axillæ, for which you can find no adequate explanation. I would also point out that a marked rapidity of pulse, out of proportion to the temperature, should be regarded as suspicious of tuberculosis. In some cases, again, more than one would believe, you may find visible evidence of miliary tuberculosis by examining the fundus of the eye, tubercles of the choroid being found fairly commonly in this form. I would repeat, however, that a case may go on until the very end without there being anything very definite, beyond fever and wasting, to point to the infection being tuberculosis rather than anything else. The younger the child, the more apt is the disease to be generalized and to assume this form.

TUBERCULOUS BRONCHIAL GLANDS

Intrathoracic tuberculosis in the child differs profoundly from the disease in the adult, for in the child it is the *glands* which are

affected mainly, the lung lesion (the site of infection) being very small. More extensive pulmonary lesions may develop at a later stage.

The results of careful pathological and X-ray studies show that at the site of entry of the tuberculous infection there develops a small focus, perhaps the size of a cherry, which eventually goes on to calcification (fig. 25). It is from here that infection spreads to the glands. Now the primary focus (called the Ghon focus) gives rise to no definite signs and hence it is often infection of the glands

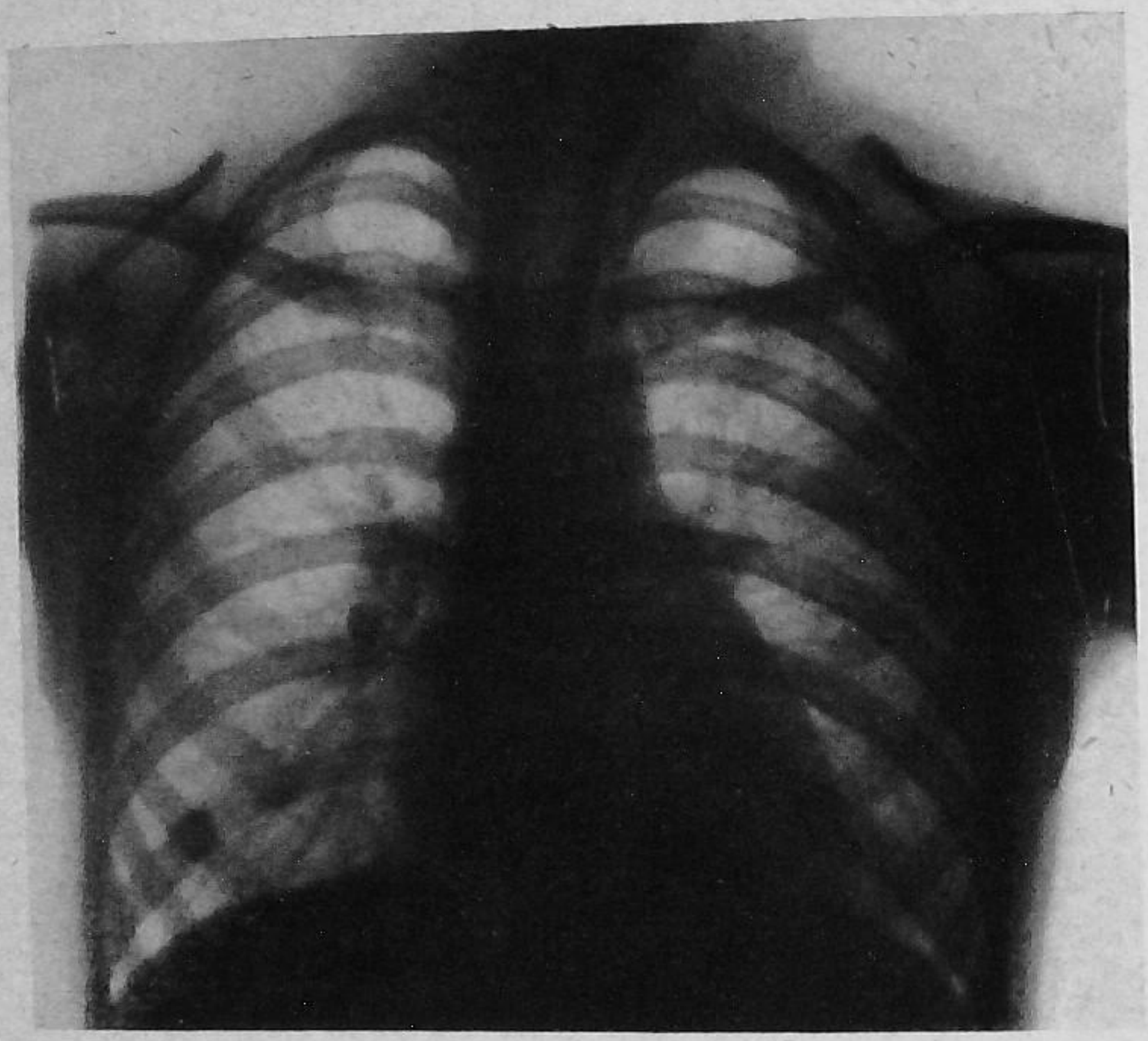

FIG. 25.—GHON FOCUS (CALCIFIED) IN RIGHT LOWER LOBE.
Calcified glands in right hilum.

or a subsequent retrograde spread to the lungs which produces obvious signs of tuberculosis.

In tuberculosis of bronchial glands you get the general symptoms of tuberculosis—slight fever, wasting, loss of appetite, fretfulness, and so on—but, in addition, there are certain special symptoms. One is the development of a peculiar brassy sort of cough, spasmodic in character. Sometimes it closely resembles whooping-cough, and it is due, no doubt, to pressure upon the nerves or upon the bronchial tubes exerted by the enlarged glands.

There are also physical signs of enlargement of the bronchial glands, which are characteristic. On gentle percussion you may find decided dullness over the upper part of the sternum, which very often spreads out under the right clavicle, more rarely under the left, and there may be dullness also over the interscapular region behind with whispering pectoriloquy heard as low as the 6th spine (D'Espine's sign). On inspection you will frequently find little enlarged veins coursing over the upper part of the chest, especially on the right side. On pushing the child's head back, so as to extend the neck, you will hear sometimes a murmur on the right

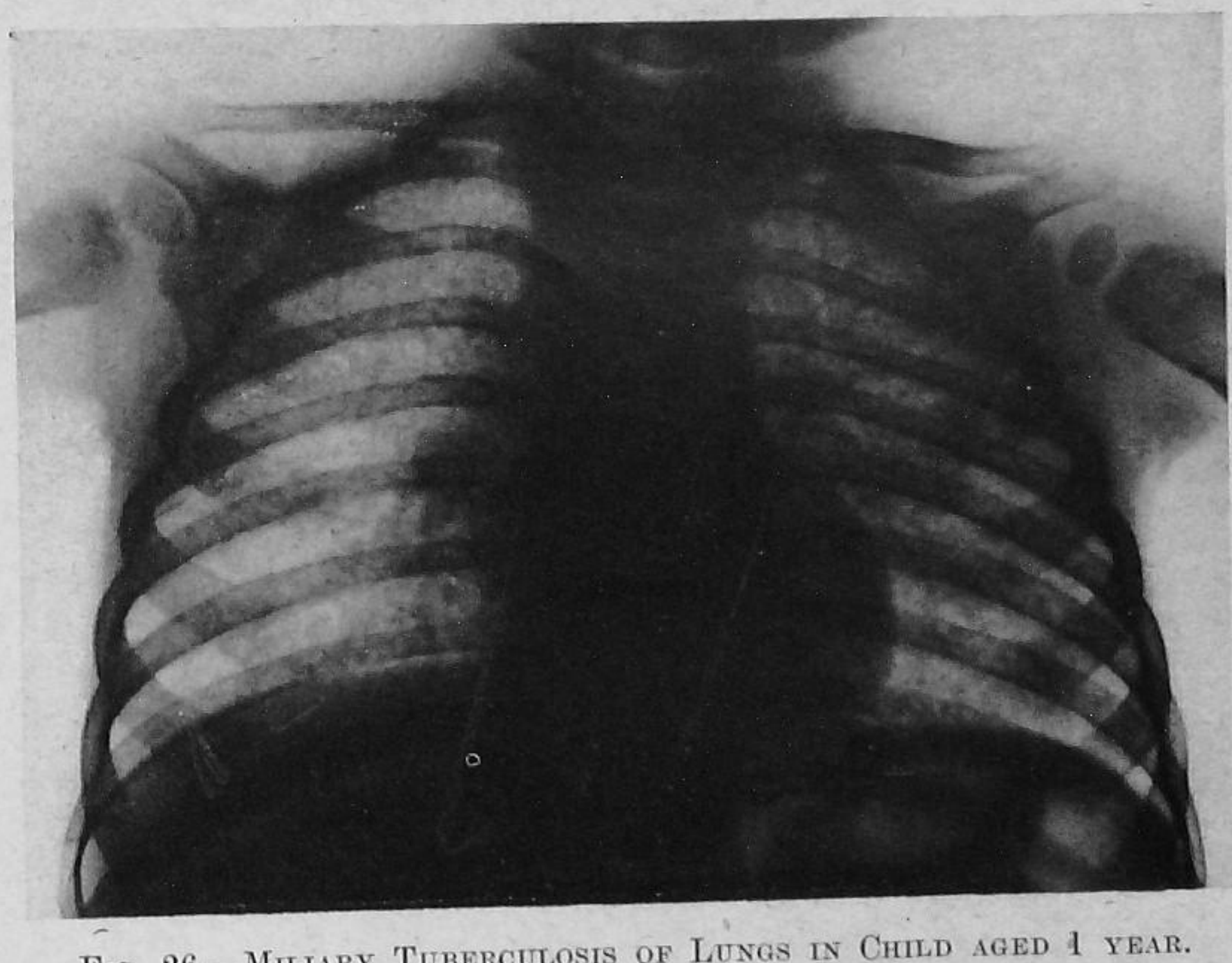

FIG. 26.—MILIARY TUBERCULOSIS OF LUNGS IN CHILD AGED 1 YEAR.

side below the inner end of the clavicle, systolic in time, which is regarded by many as a characteristic sign, although personally I don't attach much value to it. And lastly, you will frequently get help in your diagnosis by examination with the X-rays, especially by taking an 'oblique' view of the mediastinum and not merely an antero-posterior one.

It is rendered probable by post-mortem evidence that a greater or less degree of tuberculous infection of bronchial glands is an extremely common thing, but, fortunately, in most cases it does not go far; it localizes itself, and the glands become calcified, and

no permanent harm ensues. So it is only in the advanced cases that you will find it possible to make an accurate diagnosis; slight degrees of enlargement are bound to be overlooked, and in many cases you can only suspect.

PULMONARY TUBERCULOSIS

I want to lay stress here on the fact—an extremely important one—that pulmonary tuberculosis, or phthisis, in the sense in which

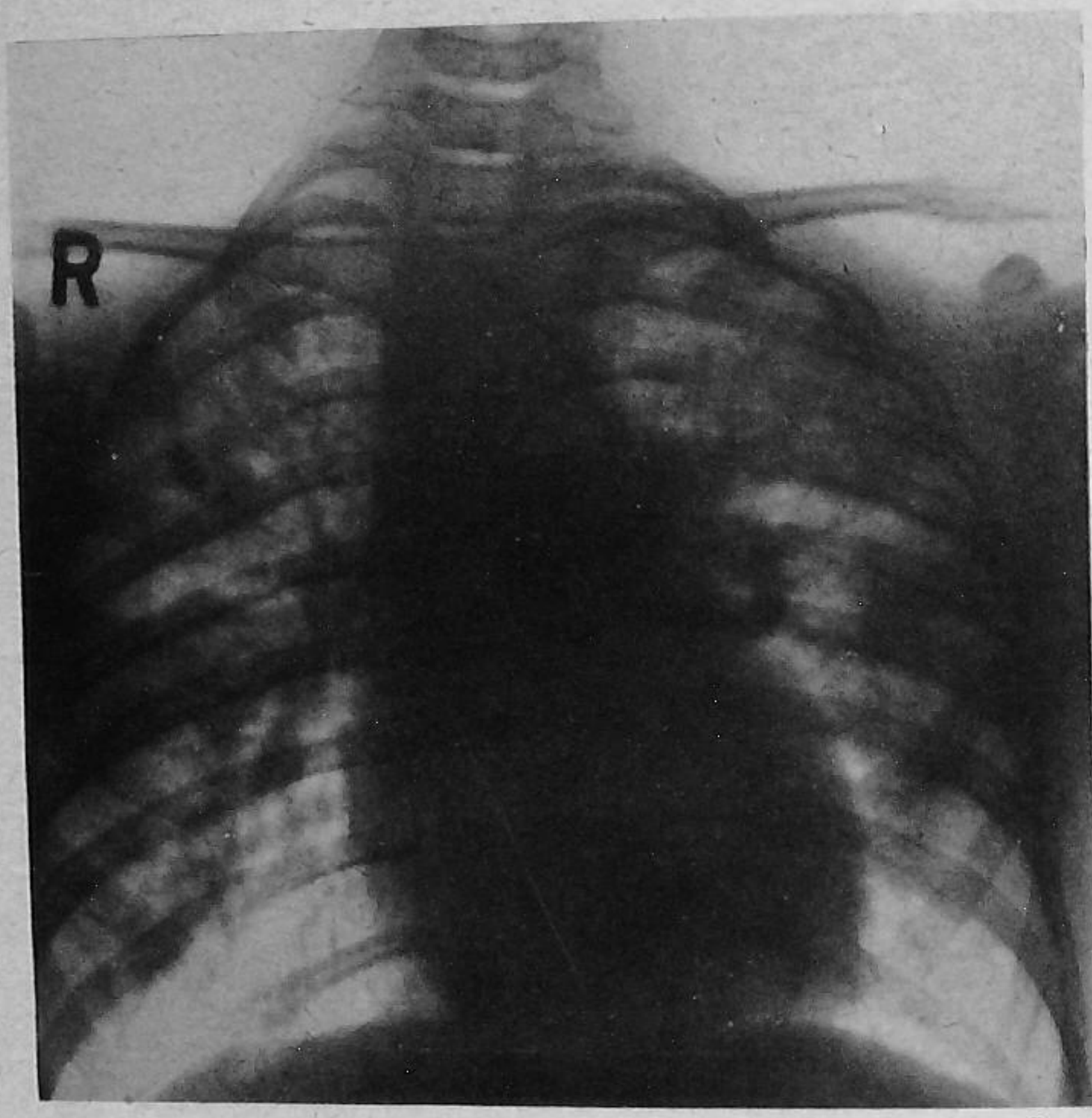

FIG. 27.—ADVANCED BILATERAL PULMONARY TUBERCULOSIS IN CHILD OF 12 YEARS.

This patient lived for three years after this picture was taken and put on 16 pounds in weight before her final illness.

it occurs in the adult, is very rare in the child. I want you carefully to remember that, because failure to do so is constantly leading to mistakes in diagnosis. You will often find 'phthisis' diagnosed in children who get out of sorts, lose flesh, have a little irritating cough, and perhaps perspire at night. The majority of such patients have not got 'consumption' at all, but are cases of dyspepsia with throat cough. Night sweating occurs in many debilitating con-

ditions in children, and is of no diagnostic value ; it is also common in children with adenoids. Similarly, children hardly ever have hæmoptysis from tuberculosis ; if a child develops hæmoptysis, it is usually due either to mitral disease or to adenoids, which in some cases bleed readily when they are soft. Of course, you may say that children who present that group of symptoms really have a tuberculous infection, which never goes farther, and shows no physical signs, but a careful use of the tuberculin tests will show this is not the case and many an anxious mother can have her fears removed by a negative Mantoux reaction. X-ray examination of the chest, if carried out by an expert, is, of course, of great value and should never be omitted in a doubtful case.

Experience shows that most of the cases diagnosed as phthisis in childhood are either cases of dyspepsia or of fibroid lung, due, not to the tubercle bacillus, but to a chronic pneumococcal or other infection, usually following upon broncho-pneumonia.

If, then, ordinary phthisis is so rare in childhood, what forms does pulmonary tuberculosis take at this age ? There are several forms of tuberculosis affecting the lungs in early life. The *primary focus* has already been mentioned. I have also described what is called *epituberculosis*. The next is *miliary tuberculosis*. This, when it affects the lungs, may present no local signs before death, because, as I have already said, the tubercles have not broken down. But if the child lives long enough for this to happen, there are much the same physical signs as would be produced by bronchitis ; there are a number of moist sounds, fine in character, scattered throughout the lungs. Indeed, the only way in which you can tell that it is not mere bronchitis you are dealing with is that the child runs a very high temperature, frequently exhibits considerable cyanosis, and has some enlargement of the spleen. The condition, in fact, looks much like typhoid fever. In such a case an X-ray of the chest may be decisive by showing the so-called 'snow-storm' picture. Sometimes, but much more rarely, miliary tuberculosis assumes a *chronic* form in which there is no cyanosis and little or no fever and in which the X-rays show coarser and less widely diffused shadows. There may be nothing to be heard on auscultation. Such cases may end in recovery or in the development of chronic phthisis.

Tuberculous broncho-pneumonia is the commonest form of acute pulmonary tuberculosis in childhood. Like the miliary form, it

also runs a rapid or, at best, a subacute course, and just as miliary tuberculosis is apt to be mistaken for an ordinary bronchitis, so this form simulates very closely an ordinary broncho-pneumonia. It is only, indeed, by its steady downward course and by the more rapid wasting that your suspicions will be aroused. The detection of signs of tuberculosis elsewhere or, best of all, the demonstration of tubercle bacilli in the sputum will settle the matter. It should always be suspected if an apparently simple broncho-pneumonia drags on for more than two or three weeks.

The next form in which tuberculosis affects the lungs is where the disease spreads out from the bronchial glands, so-called *hilum tuberculosis*. In such a case the right lung is much more often affected than the left, because there is a gland situated close to the right bronchus which is very apt, when it becomes infected, to ulcerate into the bronchus and lead to exudation, which spreads into the right lung and thence upwards and downwards, producing a dullness below the inner end of the clavicle and in the interscapular region with feeble breath sounds. In the more massive type of spread (so-called massive pulmonary tuberculosis) the dullness and absence of breath sound may be so marked as to simulate the presence of fluid. These types are not uncommon, and in their diagnosis an X-ray examination is most helpful. In a typical case of hilum tuberculosis a triangular shadow based on the hilum and tapering towards the axilla will be seen (so-called ‘ hilum flare ’).

Sometimes, though rarely, you do meet with a *fibroid form* of pulmonary tuberculosis in children which can only be diagnosed with certainty by examining the sputum. Otherwise it resembles, in its physical signs, fibroid lung due to chronic pneumococcal infection. The difficulty of diagnosis is increased by the fact that one is very rarely able to get sputum from children for examination owing to their swallowing it. In these circumstances you should introduce a swab on your finger into the throat and wipe round the top of the larynx, and so remove some of the secretion for examination. Such a manœuvre is sometimes successful, and enables a diagnosis to be made. Another plan, seeing that children swallow their sputum, is to wash out the stomach before breakfast, centrifugalize the product and either examine a film for the bacilli or inoculate a guinea-pig. This, however, is difficult to do in private practice. Examination of the fæces for acid-fast bacilli is more feasible and sometimes succeeds.

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ABDOMINAL TUBERCULOSIS

There are two varieties of abdominal tuberculosis in children. First, it may be purely **glandular.** In this form the infection is confined to the mesenteric glands, and it is to it that the term 'tabes mesenterica' should be restricted, if it is employed at all. Tabes mesenterica is not a common condition clinically. I do not say that you may not find slight tuberculous infection pretty commonly in the mesenteric glands of a child who has died of something else, but it is rare to find a degree of glandular enlargement which permits of recognition during life. The way to search for it is to feel on the right side of the abdomen, and try to compress the mesentery against the bodies of the vertebræ; you may then be able to feel the enlarged glands. Sometimes they can be made out by examination *per rectum*, but in a child it is hardly possible to do that satisfactorily without an anæsthetic. X-ray examination will show calcification of these glands (fig. 28) but, of course, when this has occurred we may assume that the disease has healed.

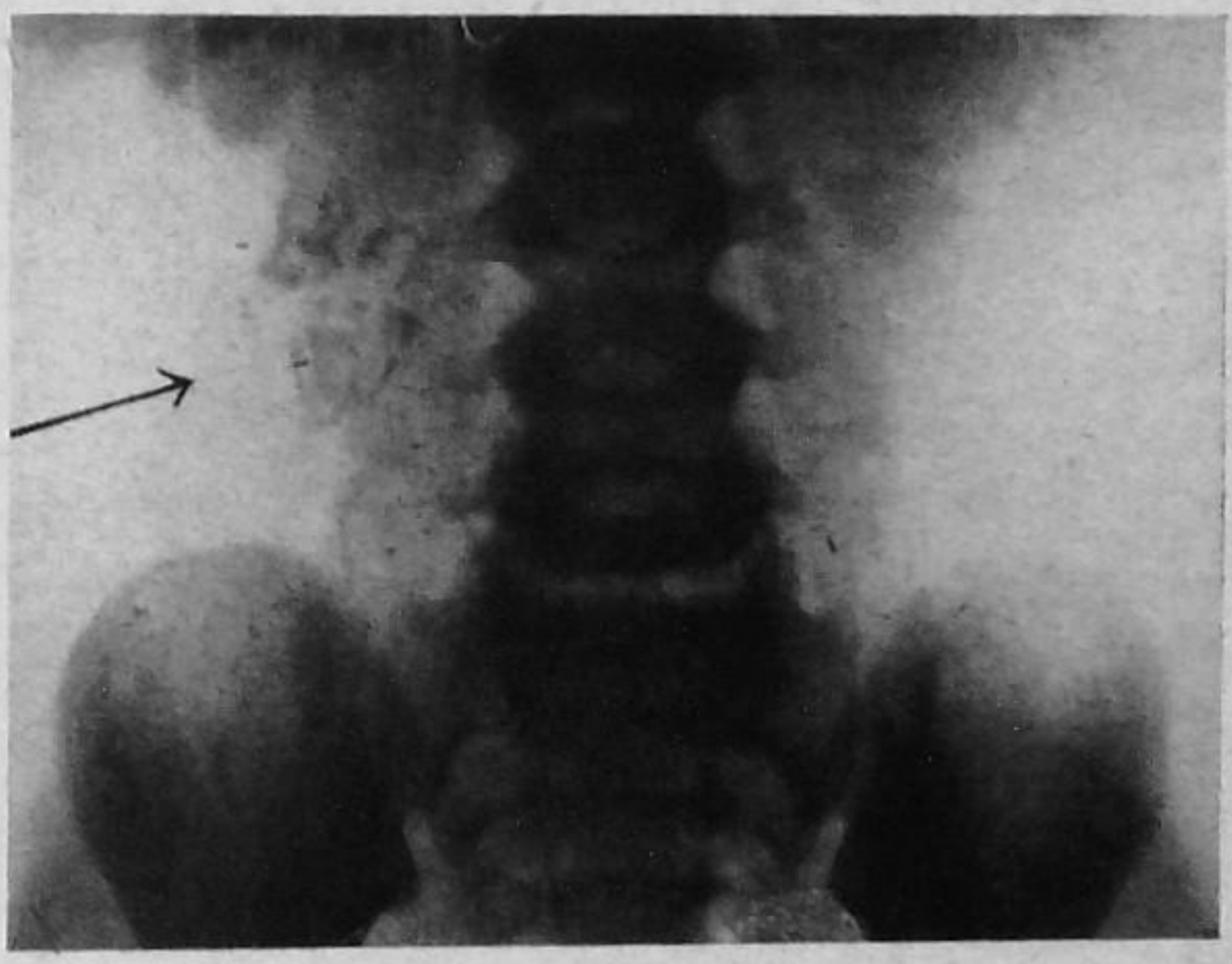


FIG. 28.—CALCIFIED ABDOMINAL TUBERCULOUS GLANDS.

The other variety of abdominal tuberculosis is much commoner, and in it the disease is not confined to the glands, but invades the peritoneum. It is therefore termed **tuberculous peritonitis,** and you have to distinguish two forms—(1) the ascitic, and (2) what we may speak of as the plastic form.

In the **ascitic** form, as the name implies, the most striking

evidence of the disease is the presence of ascites. It is not difficult to diagnose, for the simple reason that if you recognize free fluid in the abdomen of a child at all, it is almost certain that it is due to tuberculosis, because in early life other varieties of ascites are so rare. Indeed, the only other condition which occurs with any frequency and produces ascites in childhood is cirrhosis of the liver.

The **plastic** form, on the other hand, is characterized not so much by effusion, although there may be slight effusion present with it, as by adhesion of coils of intestine to one another, by great thickening of the omentum, by the pouring out of lymph, and the formation of general adhesions inside the peritoneal cavity. It is recognized clinically by a general doughy feeling of the abdomen, and frequently also one can feel the thickened omentum crossing the abdomen at or above the level of the umbilicus as a sausage-like tumour. (It is apt to be mistaken for the lower edge of the liver, although careful investigation will show that the edge of the liver is decidedly higher.) You will often be able, also, to feel actual lumps in the abdomen produced by the matting together of coils of intestine with encysted fluid, and by the presence of enlarged glands. Here, again, tuberculosis in the child is so common that if you find a lump in the abdomen the odds are in favour of it being due to tubercle; tuberculosis is more common than all the other causes of tumour in the abdomen in early years combined. In some of the plastic cases you will find the umbilicus become prominent; it opens out, becomes red and excoriated, and looks like an abscess pointing; indeed, a discharge of thin sero-purulent fluid often takes place. You must not regard this as necessarily a very serious matter; I have known many patients get quite well after such an occurrence. Indeed, it often seems to mark the turning-point towards a favourable issue.

I would again remind you that these varieties of abdominal tuberculosis are not sharply marked off from each other. Thus, where ascites is the most prominent sign, there may also be a few large glands. In the plastic variety there is always more or less glandular enlargement, if you can only feel it, and, sooner or later, there is apt to develop considerable ascites.

The last type of tuberculosis in childhood, the meningeal, I shall deal with in another lecture.

TREATMENT

And now a word about the general treatment of tuberculosis in childhood. Here I would say that the treatment of fully-developed tuberculosis is not satisfactory. Tuberculosis is a condition which should be prevented rather than cured. And let me repeat that the most essential points in the matter of prevention are—(1) To guard children from contact with tuberculous adults; (2) by the boiling of milk to protect them from infection of the bovine type; (3) to keep the bodily health at a high level, so that they can deal with an initial infection; and (4) after a catarrhal illness to take great care to make convalescence complete, which is best done by ensuring a sufficiently long stay at the seaside.

With regard to the treatment of the disease once it has developed, I would say that the key to the situation is the alimentary canal, for if you are to treat tuberculosis in the child successfully you must do so through the digestive organs. Your first care should therefore be to restore the child's appetite and digestion—if that is impaired, as it often is. Having done this, feed the child up to the level of its digestive capacity, especially with fatty foods, of which cod-liver oil, although not now so much used as formerly, is one of the best. Medicines are of little use, except for special indications, for we know of no drug which has any specific influence on tuberculosis. With regard to treatment by tuberculin, I can only say that it is an agent which it is best for the practitioner to leave alone. So that it comes to this, that when once the disease has developed, we have still to rely, as we have had in the past, upon fresh air, sunlight, good feeding, and improvement of the general health, so as to increase the child's own natural powers of overcoming the disease.

In a case of **abdominal tuberculosis** you can do more by treatment; indeed, there are no cases of tuberculosis which you approach with more hopefulness. The prognosis, however, will be better in older children than in younger, for the reason that in infants tuberculosis generalizes so fast that you do not often have to deal with the local disease only; in older children generalization occurs more slowly. The first thing to do is to attend to the general surroundings of the child. Such children should be sent away, if possible, out of towns and spend their time by the seashore in a wheeled carriage, lying down. The abdomen should be supported by a broad and firm binder.

Above all, make it your first care to see that the stomach and bowels are in good order, so that the child can take and digest a suitable quantity of food. You must correct gastric disorder if it exists, and do all you can to increase the appetite. If there is diarrhœa, you must make a point of checking it before you do anything else. Having done that, you can proceed to feed the child up, seeing in particular that he has plenty of fatty foods. With regard to medicines, you will find help from cod-liver oil, iodide of iron, and creosote.

You see there is nothing in this line of treatment other than what your common sense would dictate. But you have often to consider in those cases whether you are to continue medical treatment, or whether you are to have recourse to **operation.** I do not think it is often advisable to operate in those cases right away. Purely medical treatment leads to recovery in a great many. By recovery I mean that, at the end of some years, they are apparently in perfect health. You should speak cautiously, however, of recovery from abdominal tuberculosis. I do not think one has a right to say that a patient is completely cured simply because after a few months the symptoms have not recurred. The disease is apt to remain latent for a considerable time. Such a child may, perhaps, get an attack of diarrhœa, or other slight illness, which pulls him down, and his resistance being lowered for the time the process flares up again.

To return to the question of operation it is only in the ascitic cases that it is ever indicated; in the plastic form of the disease it is both useless and dangerous. Where there is much effusion, however, evacuation of the fluid by laparotomy—without subsequent drainage—may greatly accelerate recovery although sometimes the fluid returns in a few weeks. Curiously enough, mere tapping is not nearly so effective.

LECTURE XV

RICKETS

I shall ask you to-day to study with me a nutritional disorder, which has the peculiarity that it is found in children only—I mean rickets. On the Continent rickets goes by the name of the ' English disease.' That is not because it is commoner in this country than abroad, but because the first accurate description of rickets as we know it now was given by an English physician, Dr. Glisson, whose name will be familiar to you in association with Glisson's capsule in the liver. In the year 1681 Glisson wrote a treatise on rickets, which was then apparently a new disease. He described it as having first appeared in the south-western divisions of England, and having spread thence over the country. He thought it began in the south-western districts because these were mostly inhabited by the more luxurious part of the community.

The word ' rickets ' is derived from the Norman-French term ' riquets,' which meant deformities, particularly deformities of the spine, or persons who were suffering from such deformities. And it was owing to the fact that one of the main symptoms of rickets is the existence of deformities of bone that this name was first applied to the disease. Glisson, or one of those who wrote with him, proposed as a substitute a sort of pseudo-Greek term, ' rachitis.' That, however, presents no advantages over the name commonly used, which has at least the merit that it conveys to you one of the main signs that cases of rickets present—namely, deformities of the bones. It would be quite wrong, however, to suppose that that is the whole of the trouble. The series of events which produces rickets is likely also to cause anæmia and affections of the mucous membranes. These are now regarded as associated disorders rather than directly due to the rickets itself.

As the bone deformities, however, are the most prominent and striking change, we shall consider them first.

The **changes in the bones in rickets** are of two sorts. There

is, in the first place, an alteration at the point of ossification, the nature of which has been summed up by saying that there is an exaggerated preparation for ossification and a diminished accomplishment of it. That is to say, the preliminary stages of multiplication of cartilage cells go on in an exaggerated degree, and the cells cease to be disposed in those regular rows which are characteristic of normal ossifying cartilage, and become arranged irregularly. The result of this is that the line of ossification becomes thickened, and you can observe, clinically, enlargement of the epiphyses. Such thickening is usually first seen in the ribs. It begins there, or can be earlier recognized there than anywhere else. In consequence there is produced a row of knobs down the sides of the

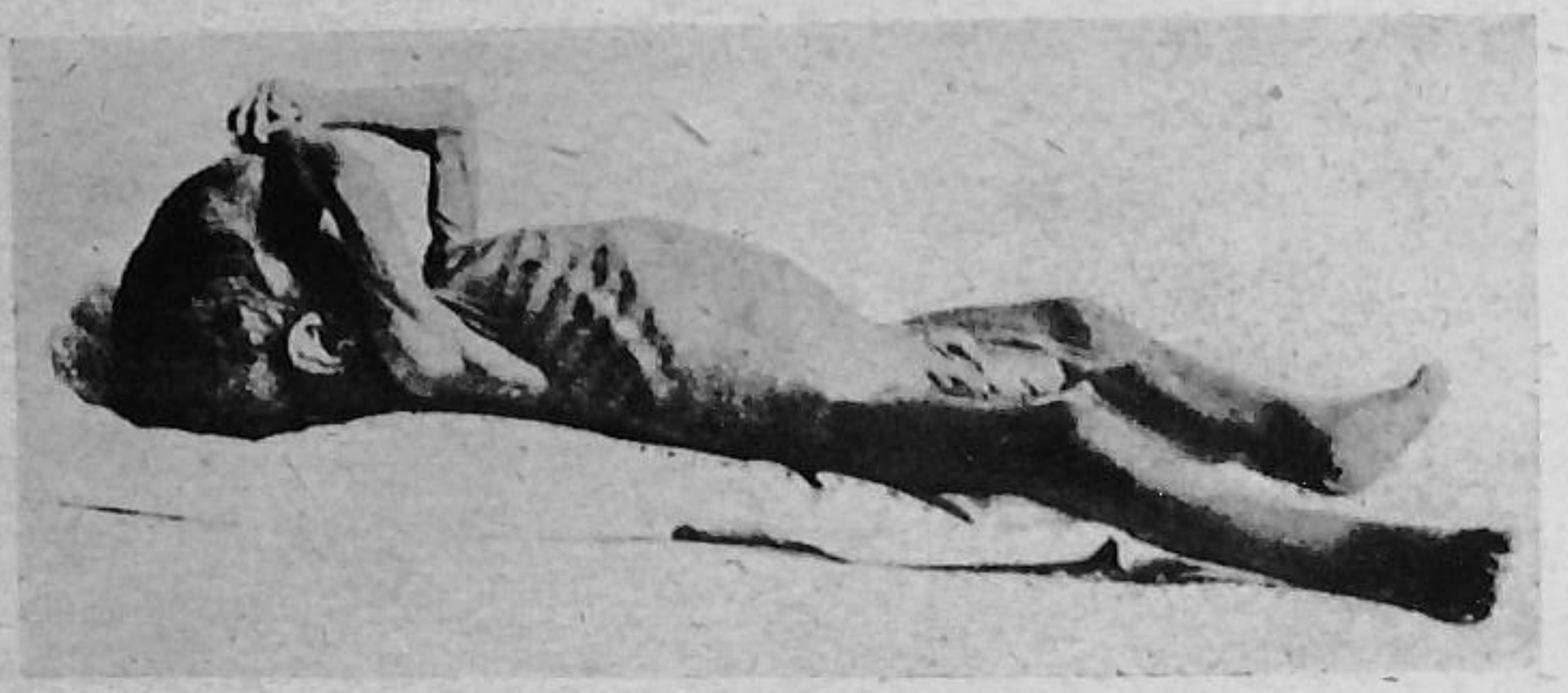


FIG. 29.—'RICKETY ROSARY.'

chest (fig. 29) to which the term 'rickety rosary' is applied, because the appearance is like a row of beads. Thickening takes place not only on the outside of the rib, but even more markedly on its inner aspect; and it may so press upon the lung that there is a groove of compressed and solidified pulmonary tissue corresponding to the enlarged epiphyses. The specimen I have here shows extremely well such an enlargement at the ends of the ribs just where the cartilage and the osseous part join. Not only are the bones which are developed from cartilage affected in this way, but a similar process takes place in those bones which develop in membrane. Hence the skull bones undergo considerable thickening at the point where ossification is most active.

Here I show you the skull of a child which was the subject of rickets, and in it you will be able to feel that in the centre of the frontal and parietal bones there is a considerable degree of thickening. That is one change which the bones show—enlargement at

the point where ossification is going on. But they show in addition another general change—namely, a process of softening. The softening is due to the absorption of the mineral constituents of the bone. If you analyse the bones from a case of rickets, and compare them with normal bones, you will find that whereas in health there is 37 per cent. of organic and 63 per cent. of inorganic matter, a bone in rickets shows quite the reverse—namely, 79 per cent. of organic matter, and 21 per cent. of inorganic. There has been an absorption or removal from the bone of its mineral constituents, chiefly phosphate of lime. I shall point out to you the significance of this more fully later on, when we come to the theories which have been advanced to explain rickets.

When phosphate of lime is removed from the bone, you can easily see what the consequence must be. The bone bends, and the direction in which it will bend is determined solely by the line of chief pressure. Now, practically the lines of pressure depend upon the position which the child habitually assumes. In the leg bones, for example, one usually finds that in the femur there is a bend forwards. In the tibia there is a sharp kink in the lower third, such as you see in the specimen before you; or another common deformity is for the tibia to be curved outwards, producing bow-leg. These deformities are largely due to the way in which the child sits with his feet tucked under him. It is that which causes the femur to bend forwards and outwards, and causes the tibia to kink forwards in its lower third. In the same way the humerus tends to bend outwards, and so also do the radius and ulna, for a rickety child tends to sit up and lean forward, supporting his weight upon the arms. In this way it is possible to give a mechanical explanation of the direction of bending, and the variations in them depend simply upon the variations in the habitual attitude of the child.

The other groups of associated changes—the visceral—are of even greater importance than the osseous. The **visceral changes** consist first and chiefly of catarrhs affecting the lungs, stomach, and intestines. Secondly, they consist in a tendency to fibroid change in some of the internal organs, particularly the spleen, with enlargement of that organ, and an increase in the fibrous stroma. The frequency with which enlargement of the spleen is met with in rickets has been considerably disputed. Probably, however, it is not appreciably enlarged in more than 5 per cent. of the cases—that

is to say, not enlarged to a degree sufficient to enable it to be felt. These visceral changes have this great importance, that they cause, or tend to cause, death much more than the osseous changes. It is largely a matter of indifference to a child whether the bones are hard or soft, unless he happens to be walking about. But it is a matter of serious importance if he is constantly exposed to attacks of bronchitis or of catarrh in the intestines, because these repeated attacks of catarrh subject the child to a great risk of the development of tuberculosis.

Types of Rickets

Now, to pass to the more clinical side of rickets, it is well that you should recognize certain distinct **types of the disease.** First of all, there is the acute type, which some people call '**acute rickets.**' This term, however, is one which it is better to avoid, because many cases of infantile scurvy have been described under it. But in spite of that there *is* a group of cases which one can class together as showing a rapid onset of the disease and a great tendency to the development of visceral rather than osseous symptoms, and as being characterized by considerable tenderness of the bones, as well as a great tendency to sweating. Such children sweat profusely about the head, particularly when they are asleep, and also about the trunk—so much so that you will often be able to recognize them in a children's ward by the way in which they kick off the bedclothes. In such cases I think you will find that the disease has come on rather rapidly. There is a second group of cases, in which the **osseous symptoms predominate,** and in which the bending of the bones is the most characteristic and striking feature. These are the cases which are likely to come under the charge of the surgeon for deformities of different sorts. Such patients may suffer from time to time from visceral symptoms also, but throughout the course of the disease the bone lesions are those which most arrest your attention. Then there is a third group of cases, characterized by a special tendency to catarrhs. These may be called the **catarrhal cases.** They are children in whom rickets is not very marked, but who tend to have constant attacks of bronchitis or diarrhœa, and in them, the rickety element is apt to be overlooked. There is a fourth group which one can profitably recognize—namely, those in which there is great **laxity of ligaments** and muscles. I saw recently a child who was brought to

the hospital with the complaint that he was unable to walk. When a child of three years of age has not yet passed that ' walking milestone ' which I spoke of early in these lectures, you may take it that he is suffering from one of three conditions : either he has rickets, or he has mental deficiency, or he is the subject of paralysis. This child I am speaking of was not the subject of paralysis, and there was no reason to suppose him mentally deficient ; but he suffered from rickets of the type in which there is extreme laxity of ligaments and great feebleness and flaccidity of the muscles. This is sometimes described as the ' acrobatic ' form of rickets, and

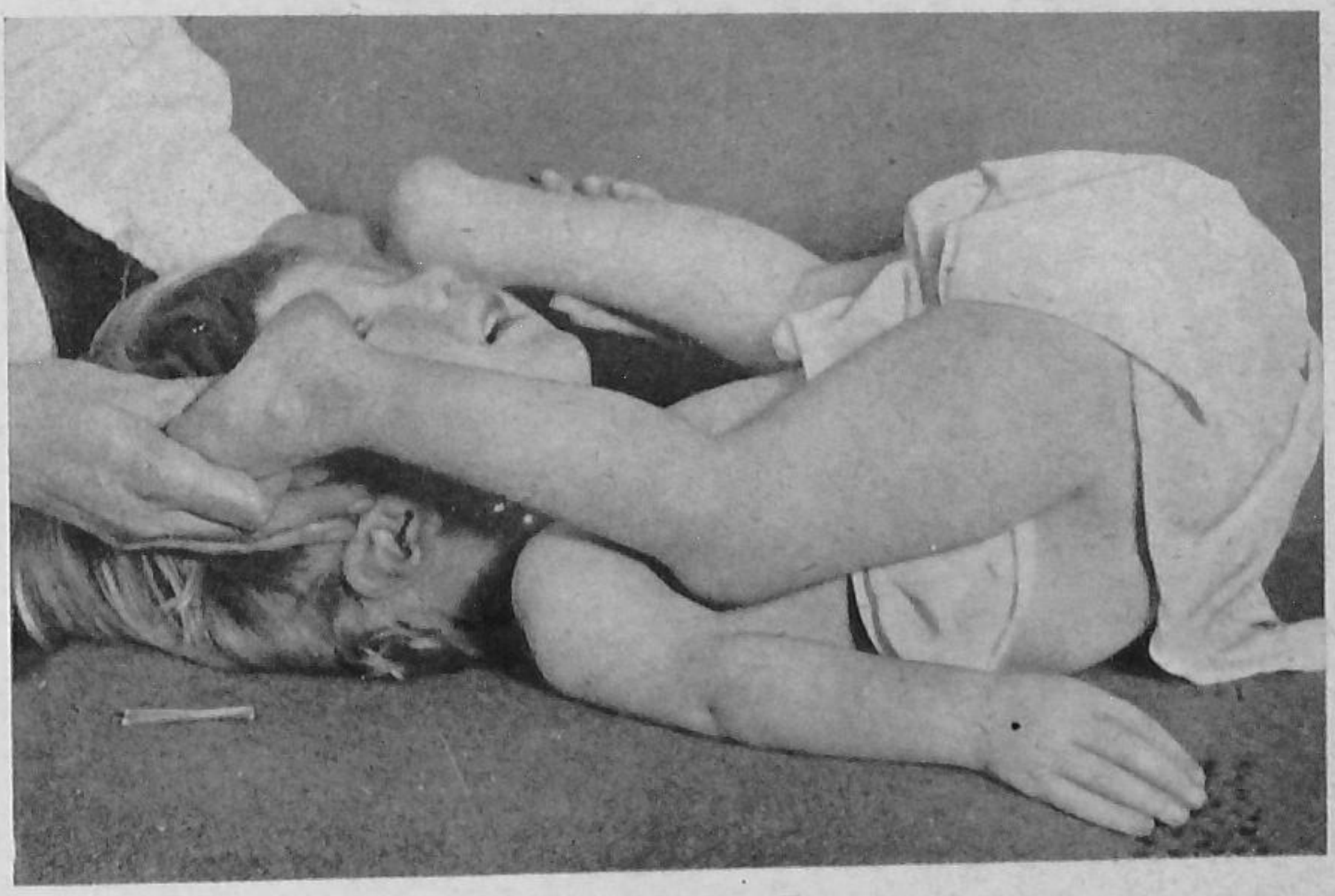


FIG. 30.—ACROBATIC RICKETS.

I shall show you at the end of the lecture some lantern slides which illustrate it (fig. 30).

All these symptoms, no matter which are most pronounced, tend to come on pretty uniformly at a particular period of life, and I want to impress this upon you, because there is nothing more important than to know what diseases are most likely to happen at a particular age. This is a touchstone by which you can invariably recognize a man who has not seen much of practice from the man who has. The beginner is always diagnosing conditions which the more experienced man is aware do not occur at that age. Thus, I constantly find that young infants are sent down from the receiving

room with the statement that they are suffering from rickets. Now, anyone who has seen much of disease in children knows that definite signs of rickets are hardly ever observed below the age of three months and rarely before six months ; the most likely time for it to come under observation is at about twelve months of age. By that time the disease usually becomes distinctly marked. It is perfectly true that it *begins* earlier than that. Many people assert that rickets is always diagnosable in the second six months of life ; but even if it were, it is only by the time the child is a year or eighteen months old that the symptoms are likely to impress themselves upon the mother.

Cause of Rickets

It is now generally agreed that rickets is brought about by a deficient supply of a specific vitamin (vitamin D). This vitamin is present in most animal fats but is particularly abundant in fish oil. Hence it is that rickets can be caused by a diet poor in animal fats and over-rich in starch, and can be cured by giving fish oil. But the specific vitamin can also be developed in the body by exposure to sunlight or to ultra-violet rays and this explains how bad hygienic conditions favour the appearance of rickets and why the disease is always more prevalent in the winter months. The two rival theories as to the production of rickets—the dietetic and the hygienic—are thus reconciled.

How it is that the specific vitamin prevents the development of the disease is still not fully understood, but apparently it promotes the absorption of calcium and phosphorus and their deposition in the bones. The catarrhal troubles occurring in rickets are due to a deficiency of vitamin A which is almost always associated with defect of vitamin D in the diet, and if anæmia is present we must blame lack of iron in the sort of diet that a rickety baby will have been getting.

Diagnosis

The **diagnosis of rickets** is not usually a difficult matter in a straightforward case. You will be struck first of all by the fact that rickety children are slow in their development, slow in passing some, at least, of those ' milestones ' which I drew attention to in the first lecture. They get their teeth late, their anterior fontanelle closes late, they sit up unaided late, and they walk late, and all these things are due to the bad development and softening of

bones. Then you will recognize, usually without any difficulty, the enlargement of the epiphyses, particularly of the ribs, that being the place where the enlargement comes first. It will also be a little marked at the wrists and ankles, perhaps, and there may be some bending of the bones. These are the points to look for in the diagnosis of a straightforward case.

But there are certain pitfalls in diagnosis to which I want to direct your attention. One of these refers to **the rickety head.** Rickety children will be brought to you under the impression that they are hydrocephalic. How shall you distinguish the rickety from the hydrocephalic head ? The head of rickets has characteristics of its own. It is a long, square head, which looks as if it had been developed in a box. It is not globular, like the head of hydrocephalus, and if you compare the foreheads of the two you will find that in rickets the forehead goes up more or less vertically in front and at the sides, whereas in hydrocephalus it overhangs the eyes and bulges out above the temporal regions ; the top of the rachitic head tends to be flat, in hydrocephalus it is convex and globular. By attention to these points you ought not to have any difficulty in distinguishing a rickety head from a hydrocephalic one. In the young infant (3 to 6 months) the softened bones in the occipital region can often be felt. This is called craniotabes.

There are certain mistakes which are likely in connection with the spine of rickety children, particularly those who belong to the ' acrobatic ' group. There is a tendency in such cases towards the production of a prominence in the lower dorsal spine, and they will be brought to you under the impression that they are the subjects of Pott's disease or tuberculous caries. But you will be able to tell a **rickety spine** (fig. 31) from a tuberculous one if you remember that one is due to the actual crumbling in of the bone, and therefore cannot be rectified, whereas the other is a mere kinking due to laxity of ligaments and so can be straightened out. If you hold the child up by the armpits, if the projection is due to kinking it will straighten out, whereas if it is due to tuberculous disease it will persist. Bowing of the legs represents another snare for the unwary. Real bowing of the bones means rickets, but in some children with chubby calves a false appearance of bowing is given by the soft parts whereas the bones are in reality quite straight. The X-ray shown in fig. 32 illustrates this point.

Another mistake into which you are likely to fall is due to the large development of the abdomen in rickety children. Such patients will be brought to you by mothers under the impression that they are the subjects of tuberculous disease, and many of them are diagnosed as such and labelled with the term 'tabes mesenterica,' against which I have already warned you. There are several reasons why a rickety child tends to develop a **large abdomen** (fig. 33). You know, in the first place, that *all* children tend to have relatively prominent and well-developed abdomens. The reason is that they have a large liver and a small pelvis. In rickets both these factors are exaggerated. The liver of the rachitic child tends to be even larger than in health, mainly from fatty infiltration; and the pelvis at the same time tends to be unusually small and to collapse upon itself, so that there is less room in it for the viscera to sink down into. Moreover, the badly developed and flabby muscles of the rickety child's abdomen allow distension to take place easily; and such distension is rendered more likely because rickety children constantly suffer from gastric and intestinal catarrh, which leads to the development of flatulence.

FIG. 31.—RICKETY SPINE, SIMULATING POTT'S CURVATURE.

There is still another error which you are likely to make, and that is attributing to disease of the nervous system symptoms which are really due to rickets. I mentioned such a case a moment ago, that of a child which could not walk when he was three years of age. The diagnosis in such a case, however, should not be difficult. You will find that a rickety child, though he cannot walk, is still able to use his limbs. As he sits in his mother's lap he will kick his legs about; or if you tickle the soles of his feet he will draw up the legs, which, of course, he could not do if they were paralysed. In rickets the trouble is merely an inability to walk, whereas in

paralysis there is inability to move a particular group of muscles in any way whatever.

Lastly, I would emphasize the importance of your being able to recognize **the rickety element in other diseases.** For instance, a child may be brought to you who is mentally deficient, but who

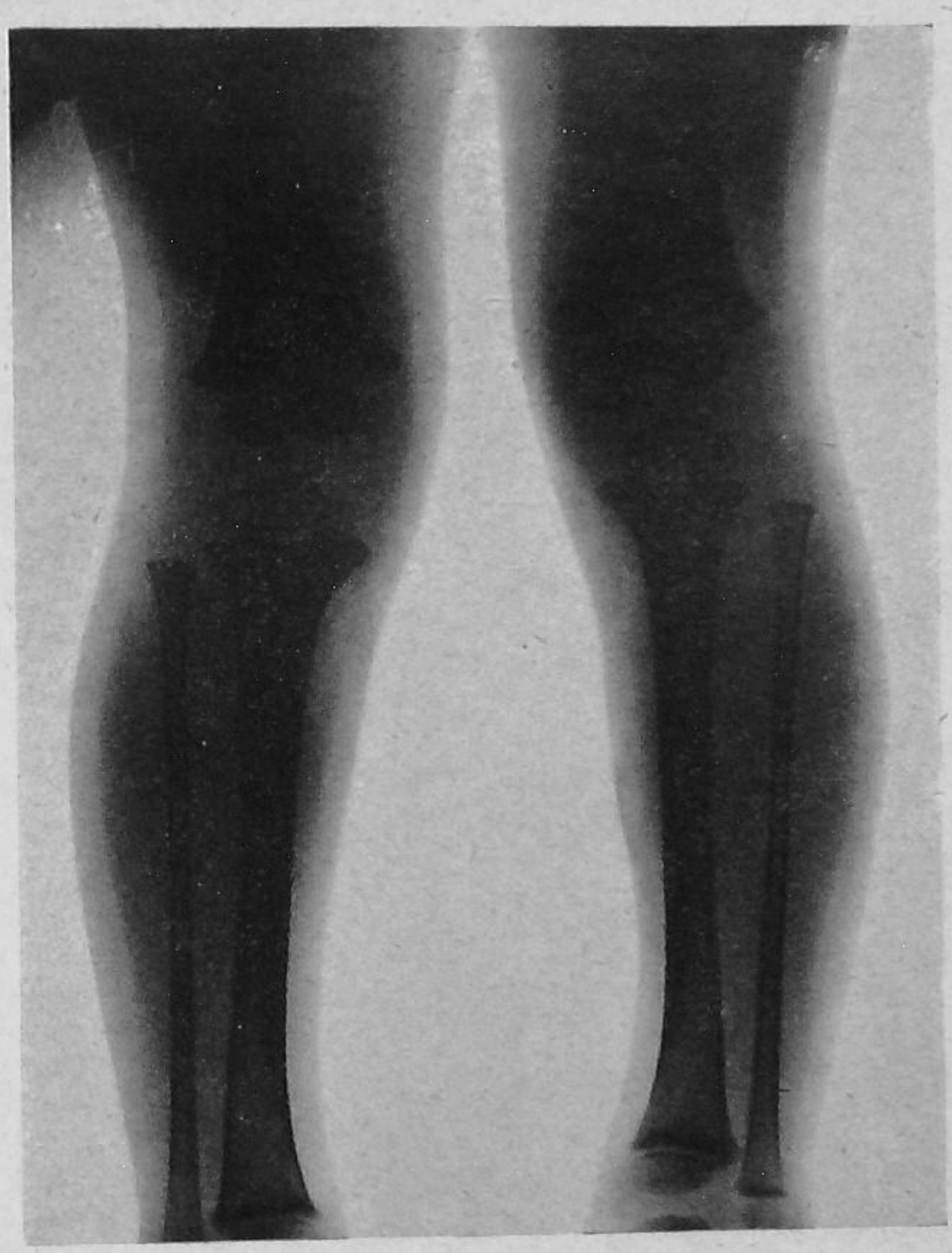


FIG. 32.—PSEUDO-BOWING OF LEGS.

This print has been made specially dense to show that although soft parts of legs indicate a bowed condition the bones are essentially straight. This is from a child of $2\frac{1}{2}$ years and is a very typical appearance in the toddler.

suffers from rickets as well; and you can do great good in such a case by treating the rickets, although it is impossible for you to improve the mental condition. Again, when we come to speak of the nervous diseases of children we shall see that rickets is a great predisposing cause in many of the functional nervous disorders, such as convulsions, tetany, and laryngismus; and you cannot

hope to treat the nervous disorder with success unless you treat the rickets, which is the basis of it. Further, in the case of a child who is the subject of repeated catarrhs, bronchitis, or broncho-pneumonia, or it may be diarrhœa, it may be of the first importance to recognize that these are simply the symptoms of an underlying vitamin deficiency.

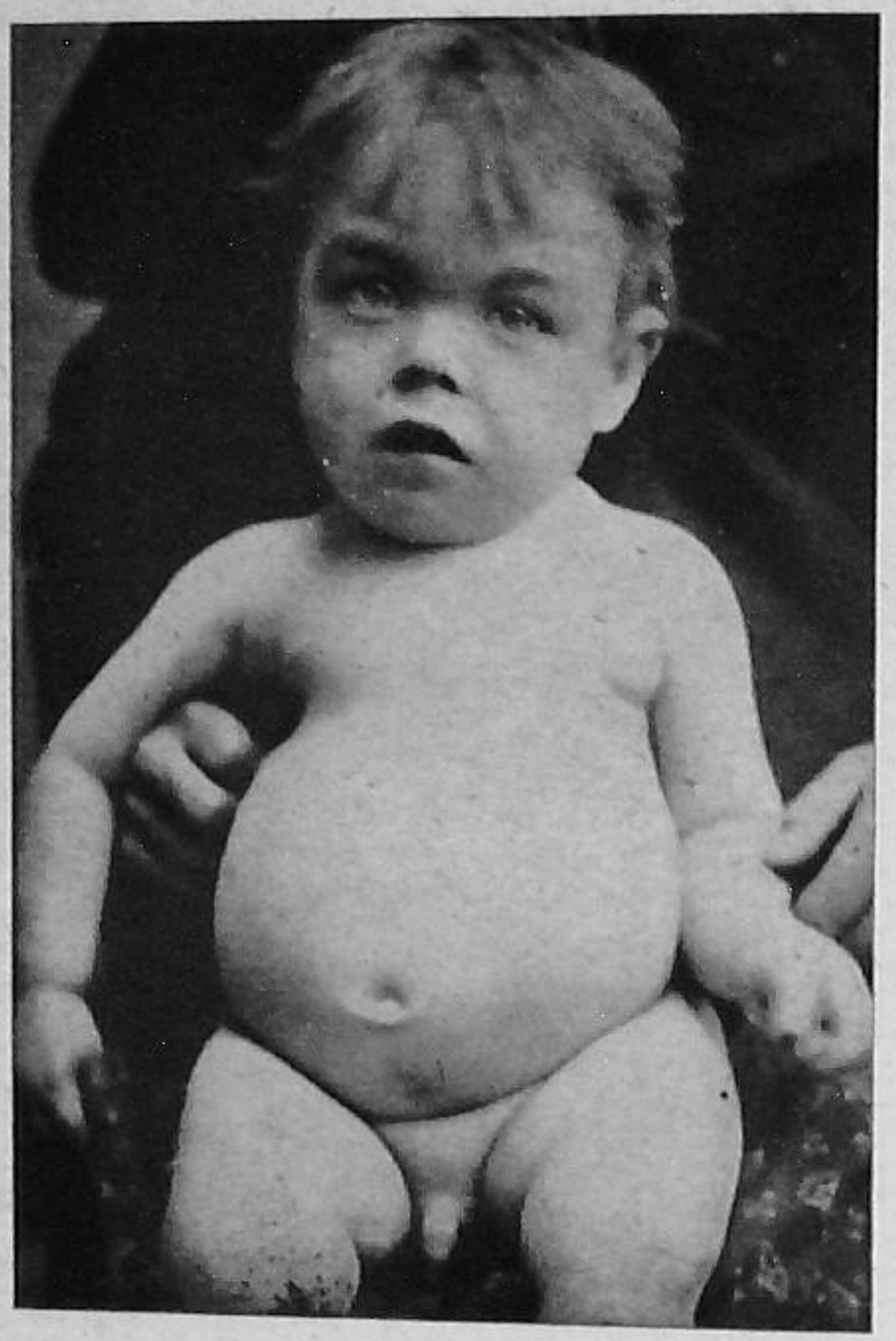


FIG. 33.—RICKETS, SHOWING THE LARGE ABDOMEN, CONSTRICTED CHEST, ETC.

In a doubtful case a **skiagram** is of great help. If rickets is present there is a tendency to cupping of the diaphysis, the epiphyseal line is irregular and fluffy and the epiphysis itself poorly ossified ; there is some degree of osteo-porosis also throughout the whole bone.

TREATMENT

We now come to the consideration of the **treatment of rickets.** Granted that a faulty diet plays a large part in its causation, your first care must be to alter the child's food. But you cannot hope to do that successfully until you have first put the stomach and intestines into a healthy condition, so that he can digest the food when you give it. So in many cases your first task must be to

correct any digestive disturbance which exists. You may have to treat diarrhœa, or gastric catarrh, or want of appetite ; and having done that you change the diet. And you will change it in this direction : diminish the amount of starchy things, and increase the amount of proteins and fat. Practically that resolves itself into increasing the proportion of cow's milk and administering cod-liver oil or halibut oil. A child of one and a half years should be getting at least one pint of milk a day, whereas he may perhaps have been getting only half a pint. The yolk of egg is also very useful in these cases, as it contains many things which the child needs. It contains phosphorus, fat, and organic compounds of iron, in addition to protein, and I like to give it early in cases of rickets. In very young children it may produce vomiting ; but in most cases it is taken quite well. It should be lightly boiled, so that it is still liquid when given ; or you may shake it up with the milk and give it in that way.

Many of these patients also do well with the addition of raw meat juice to their food ; or you may even give them underdone meat scraped down. A rickety child must be mainly carnivorous. Rickets might be said to be due to premature vegetarianism, and you have therefore to increase the proportion of animal food because animal food is characterized by richness in proteins and fat.

About the lime salts you need not trouble ; there is no occasion to administer them artificially, for milk contains them in abundance. Nor will drugs help you much, unless you call cod-liver oil a drug. In severe cases *irradiated ergosterol* may be administered in a dose of 1 mg. daily or even more. This quantity contains as much vitamin as 14 teaspoonfuls of cod-liver oil. Other preparations containing concentrated forms of vitamins A and D, usually in doses of 5 minims three times a day may be used. Bear in mind that if you use cod-liver oil as an emulsion that this is also a dilution, so be sure that you give enough oil : for the cure of rickets you must give the equivalent of 2 or 3 teaspoonfuls daily at least. If the child is anæmic, give iron in addition.

Attention to the diet and improving the child's hygienic surroundings must be your chief lines of attack, because although it may be true that bad diet is the main cause, yet there are cooperating factors. Want of sunlight, as I have said, is one of these. The child should have the benefit of the sun if there is any, or

exposure to the ultra-violet rays of the mercury-vapour lamp may be used as an artificial substitute for sunlight. Attention should be paid to ventilation, bathing, and clothing. For the deformities you may require to have recourse to surgical aid; but it is surprising how they tend to disappear after a time. Bones which have been very much bent may still, without any particular effort on your part, but simply as the disease passes off, become straightened out again in a wonderful way.

In order to prevent the deformities from becoming greater, it is sometimes well to see that the child is kept off his legs. To accomplish this you may tie the legs together, or put on light splints which project beyond the foot. And you may have to keep them on for many months, at all events during the day, until the bones have become consolidated again. But in the very severe cases, when the child is five or six years of age, and where there is permanent deformity, surgical means are alone likely to be of much assistance.

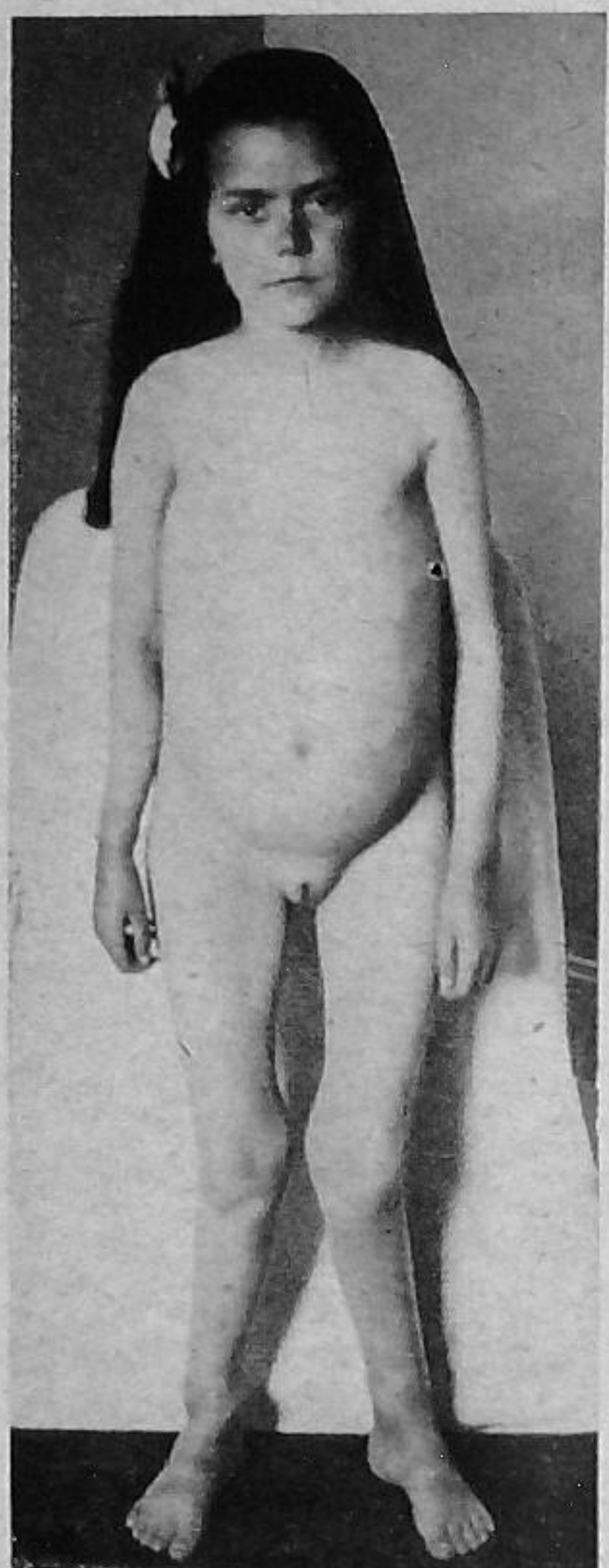


FIG. 34.—LATE RICKETS, SHOWING KNOCK-KNEE AND ENLARGEMENT OF EPIPHYSES

Before concluding this lecture I should like to say a word or two about **late** or **adolescent rickets,** and about achondroplasia.

There can be little doubt that a condition very similar in its effects upon the bones to ordinary rickets sometimes appears in later childhood, or even at the period of adolescence. To this the term 'late rickets' has been applied. Seeing that many of these patients have already suffered from ordinary rickets during infancy, some have concluded that so-called late rickets is merely a continuance or recrudescence of the ordinary infantile form of the disease. This, however, is certainly not true of all the cases, and there seems to be no doubt that late rickets is a genuine disease, although it usually occurs in association with renal infantilism or in

consequence of cœliac disease. Clinically late rickets differs from infantile rickets by showing a greater tendency to affect the long bones, the epiphyses of which show notable enlargement, especially, perhaps, about the knees, so that genu valgum and bending of the shafts of the bones are specially apt to result from it. This is very well shown in fig. 34 which is a photograph of a girl eleven years of age, in whom the disease had been present for four years. On the other hand, the skull and the viscera do not tend to be affected in late rickets. Skiagrams of the bones in these cases show that ossification is very imperfect.

The treatment of late rickets is unsatisfactory in the renal cases because a profound biochemical disorder is present. In the cœliac cases, treatment of the primary condition and the use of vitamin D concentrate will produce a cure. Surgical treatment may be required, but beware of anæsthetics in the renal cases.

ACHONDROPLASIA

Achondroplasia is a condition which is sometimes mistaken for rickets, although quite unjustifiably, for the two diseases have nothing to do with each other. Achondroplasia is really a disease of about the third to the sixth month of intra-uterine life, and consists in a partial failure of ossification in the cartilaginous bones. Why this failure of ossification occurs we do not in the least know, but the consequences of it are very striking. A child affected with achondroplasia has a very characteristic appearance (figs. 35, 36, 37, 38). The limbs are extremely short in proportion to the length of the trunk, producing an appearance which has sometimes been spoken of as a 'human dachshund.' The soft parts of the limbs also hang in folds or creases, like the sleeves of a coat which is too big for the wearer. The hands are short and stumpy, the fingers being approximately of equal length, and, instead of being parallel, radiate out like the spokes of a wheel—the so-called 'trident hand.' As the child grows the normal curves of the long bones become exaggerated, and as the flat bones are unaffected the vault of the skull grows out of proportion to the base, so that the head becomes dome-shaped, with a 'beetling brow,' and a depression at the root of the nose. The face also has a triangular shape, being broad at the forehead, and tapering to a pointed and projecting chin. Lordosis is well marked, and the patient walks with a

FIG. 35.—TWINS: THE GIRL AN ACHONDROPLASIC, THE BOY NORMAL.

FIG. 36.—ACHONDROPLASIA, SHOWING LORDOSIS.

FIG. 37. — ACHONDROPLASIA, SHOWING SHORT LIMBS AND CHARACTERISTIC FACIES.

FIG. 38. — ACHONDROPLASIA, SHOWING 'TRIDENT' HAND.

waddling gait, like that seen in cases of congenital dislocation of the hip. With all these peculiarities the viscera are unaffected, the general health is sound, and the intelligence normal. The prognosis is therefore perfectly good as far as life is concerned, which is fortunate, as the condition is entirely insusceptible of treatment.

LECTURE XVI

INFANTILE SCURVY AND PINK DISEASE

INFANTILE SCURVY

Our first subject of study in this lecture is that nutritional disease of infancy known as scurvy. This disease used to be called, and is still called by some people, ' scurvy rickets.' That is an unfortunate term, because it is now quite well recognized that scurvy in infants has nothing directly to do with rickets, but that the coexistence of rickets in many of the cases is a mere accident, and due to the fact that the kind of diet which is prone to produce scurvy is apt also to produce rickets. Beyond that I believe there is no direct connection between the two diseases, and it is better to use the term ' infantile scurvy ' to designate this complaint. The prefix ' infantile ' is used because it is not clinically identical with scurvy in grown-up persons.

Infantile scurvy seems to be a disease peculiar to infants, and I am glad to be able to say that, just as in the case of rickets, it has been chiefly made known by the labours of English physicians, and particularly, one is proud to add, by men attached to Great Ormond Street Hospital. The first cases were published by Dr. Cheadle in the year 1878, and it was more fully described by Sir Thomas Barlow in the year 1883. His work on the subject was so exhaustive and complete that very little has been added to our knowledge of the disease since that time, and infantile scurvy is now known on the Continent by the name ' Barlow's disease.' Before I pass on to describe it, I should also say that in past years this disease used often to be described under the term ' acute rickets.' When I was dealing with rickets you will remember I told you that there were some cases of that disease which you might call acute, because the symptoms come on rapidly. But that has nothing to do with scurvy, and the term ' acute rickets ' should either be abolished altogether, or, if used at all, it should be applied to genuine rickets in which the symptoms come on with unusual rapidity.

What are the **clinical characters** of a case of infantile scurvy ? The child will usually be brought to you by the mother with the complaint that he is unable to use one or both of the legs, or that the legs are swollen, or that the child cries when he is touched. These are the commonest things that the mother notices wrong first, and the majority of cases of scurvy which are brought to you will be brought with such complaints as those. When you look at

FIG. 39. — SCURVY, SHOWING CHARACTERISTIC POSITION OF LEFT LEG, THE RESULT OF PERIOSTEAL HÆMORRHAGE, AND SOME SPONGINESS OF THE GUMS.

FIG. 40.—SCURVY, SHOWING CHARACTERISTIC SWELLING IN LOWER PART OF RIGHT FEMUR.

the child you will usually observe that he appears healthy. These children are generally well nourished, they are often of good colour, or, at all events, not conspicuously anæmic ; but when the child is being stripped you will notice that he screams a great deal, and is extremely apprehensive. One has been able to recognize cases of scurvy in the out-patient room by the fact that the child starts screaming as soon as he sees the doctor, because he is so afraid he will be handled.

When you come to examine the child more closely you will find perhaps that one of the legs is kept immobile, that it is slightly flexed at the knee, and that the thigh is rotated outwards. On further examination you will probably find signs of rickets—that is to say, slight enlargement of the epiphyses. If you run your hand over the sternum you will get the impression that it has been pushed in, so to speak, for the costochondral junctions stick out and the sternum appears depressed. On opening the mouth you will observe usually that one or two teeth have come through, because infantile scurvy occurs with quite an extraordinary degree of frequency when the first teeth are being cut; by far the larger number are children of eight to ten months old. You will commonly find, then, that one or two of the lower incisors are through, and that the gum around them is swollen and of a vivid purple colour. In the upper jaw you may observe that there are teeth about to be cut, and that the gum over these is in a similarly swollen and discoloured condition.

Fig. 41.—Orbital Hæmorrhage in Scurvy.

When you come to examine the limbs you will probably find on one or both of the lower extremities, usually towards the end of the femur or the tibia, a thickening which is extremely tender when it is touched, and which presents a sort of boggy feeling on palpation. You may find, further, as minor symptoms of the disease, a tendency to hæmorrhages from the mucous membranes, and particularly the occurrence of hæmaturia. In rare cases the hæmorrhages may take place also into the looser subcutaneous tissues, and particularly into the orbit (fig. 41). These are the clinical signs which you will find when you examine a typical case; and if you inquire into the history you will find almost invariably that the child has been fed in a particular way—namely, on a tinned food, often only with condensed milk. Also the child had been given no orange or other fruit juice or only negligible quantities. It is only extremely rarely that the infant is found to have been fed on the breast. I have never seen

such a case myself, but it may happen if the mother's diet has been deficient in anti-scorbutic vitamin. Once or twice I have seen the disease produced in children who were fed simply on boiled cow's milk, but by far the greater number of the patients have been fed upon a patent food alone or combined with condensed milk.

The pathology or **morbid anatomy** of infantile scurvy is not difficult to explain. It is characterized by a tendency to hæmorrhages, and the seat of the hæmorrhages seems to be determined by the degree of vascularity of the parts. For instance, swelling of the bones is due to hæmorrhage occurring in the deep vascular layer of the periosteum; the hæmorrhage raises the periosteum, producing the swelling, and the pressure of the effused blood on the nerves produces the tenderness. Sometimes hæmorrhage takes place between the epiphysis and diaphysis, and causes a complete separation of the former, so that it may come to lie quite loose. Very rarely the hæmorrhage goes on to suppuration. Similarly, the hæmorrhage round the teeth is due to the fact that the neighbourhood of the teeth is a place which is unusually vascular. That is all one need say about the morbid anatomy. I shall not discuss the chemical pathology of scurvy except to say that it is now agreed that the disease is due to the absence of a special 'vitamin,' but shall pass at once to the question of **diagnosis**.

Diagnosis

In the first place, you will have to diagnose infantile scurvy from *rheumatism*. I say that because I have had several cases of infantile scurvy sent to me as rheumatism, in one of which the sufferings of the unfortunate child had been aggravated by the affected limb being painted with iodine. I am certain that this is one of the commonest mistakes made with regard to this disease, that the swelling and tenderness of the bones are regarded as rheumatic in origin. But that is a mistake which you ought never to make, because at the age at which infantile scurvy occurs rheumatism is unknown. So you see this point of differential diagnosis is merely another example of that principle of reasonable probability which I tried to bring before you in a previous lecture —namely, the principle that at certain ages certain diseases are more likely to occur than others. At this particular age rheumatism is not only unlikely to occur, but it does not occur at all.

Another thing which this condition has been mistaken for, and

more excusably, is *syphilitic periostitis*. Syphilitic periostitis, however, usually occurs either in children who are much younger than the age at which scurvy occurs, in the form of syphilitic epiphysitis which I described when dealing with the subject of congenital syphilis, and is a disease seen during the first few weeks of life; or it occurs considerably later in the form of periostitis affecting the long bones, very commonly the tibia, and then the children are distinctly over the age at which infantile scurvy is usually seen. You will find, too, that other signs of congenital syphilis are absent in a case of pure scurvy, and also that in congenital syphilis there is no sponginess of the gums.

Scurvy may also be mistaken for ordinary *epiphysitis*, and that is a fairly excusable mistake which I have known to be made by people who are perfectly familiar with the usual symptoms of both diseases. The only cases in which this error may be made are those in which there is no affection of the gums. But there are other points which will help you. One of these is that the temperature is always raised in epiphysitis, whereas in scurvy it is not usually so to any extent. Another test is to observe the result of treatment. Scurvy properly treated gets well almost at once, so that the therapeutic test is of very great value in diagnosis. If you are in doubt, then, whether a case is one of scurvy or not, give the child vitamin C (ascorbic acid) and see what happens. If the child has scurvy you will have done right, and improvement will set in at once; if he has not got scurvy you will not have done any harm. In early cases the therapeutic test may be the only one upon which you can depend.

I have lately seen a case of infantile scurvy which had been regarded as one of fractured femur, but the history here should prevent mistake, and a radiograph would make the matter certain.

There is a fourth condition which affects the limbs, and which it is well to be on the look out for, so that you do not mistake it for scurvy, and that is *infantile paralysis beginning with hyperæsthesia*. Some cases of infantile paralysis are accompanied at their outset by extreme tenderness of the limbs, so that whenever they are touched the child cries out. But in such cases the age of the child ought to keep you right. Infantile paralysis is a very badly-named disease, because it is not a disease of infants; it is a condition which is almost unknown below one year of age. It occurs most commonly between the ages of two and four. In infantile

paralysis, too, there will be atrophy or wasting of the limb, and no swelling of it.

The lesions in the mouth and swelling of the gums may be mistaken for other conditions. They may be mistaken, first, for *stomatitis*, and sometimes there will be no means of telling whether it is a case of mere stomatitis or of scurvy except by trying the effect of treatment, but the mouth lesions are much more likely to be mistaken for the ulcerations which occur in connection with *leukæmia*. I remember seeing a child about six years of age lying in the ward of a hospital, and by its bed was a bottle of lime-juice. The child's breath was exceedingly offensive, and on examining the mouth there was a large excavating ulcer of one tonsil and ulceration and lividity of the gums; the child was believed to be suffering from scurvy, and was being treated by lime-juice. But the child did not benefit by the lime-juice treatment, and on examining the blood 200,000 white corpuscles were found, 98 per cent. of which were lymphocytes; in other words, it was a case of acute leukæmia. It is well for you to remember, as I shall have occasion to point out when I speak of the anæmias of infancy that acute leukæmia, although very rare in grown-up persons, is not very uncommon in children; and it is often accompanied by ulceration of the mouth. But here, again, the examination of the blood will at once settle the matter.

Cases which are characterized by hæmorrhage into the orbit may be mistaken for two conditions; for *sarcoma* of the orbit, or for that very rare disease called *chloroma*. In the case of sarcoma there will usually be signs of a tumour elsewhere, and the therapeutic test—change of diet—will be conclusive. The differential diagnosis from chloroma I must defer until I come to speak of it under the anæmias.

Lastly, I would mention that renal hæmorrhage due to scurvy is apt to be mistaken for *renal hæmorrhage* from other causes. I have known a surgeon cut down upon a kidney in such a case, expecting to find a stone, because the child had been suffering from profuse hæmaturia. He found no stone, and the hæmaturia persisted until it occurred to those in charge of the patient to make a change in the diet. They did so, with the result that the hæmaturia disappeared in the course of two days. So hæmaturia from scurvy is a thing which you may overlook. Some people believe that it is a much commoner symptom of scurvy than is

generally supposed, and that you will commonly find traces of blood in the urine in a case of scurvy if you look for them.

Before leaving the question of diagnosis, I would remind you that you must be prepared to recognize the earliest beginnings of scurvy before the full-blown symptoms, such as swelling of the limbs, tenderness, and spongy gums, are present. At first the child simply ceases to thrive, his weight no longer rises, he ‘ goes

FIG. 42.—HÆMORRHAGE INTO ORBIT, THE RESULT OF SARCOMA OF SKULL, SIMULATING SCURVY.

off his feed,’ and is fretful and unhappy. If in such a case the diet is of a kind which is apt to produce scurvy, you should not wait for further developments, but start treatment immediately. Disappearance of the symptoms will often follow at once.

TREATMENT

We may now discuss the **treatment of infantile scurvy.** It was found very early in the investigation of this disease that it could be cured almost at once by ensuring the presence in the diet of fresh constituents, such as fruit juice and vegetables. And the routine treatment for you to recommend in a case which comes before you is that the child should be given liquid rather than dried or condensed milk. Also, any patent food which the child is taking should be at once stopped. You should give every day orange juice, black currant juice or tomato juice. Begin with a few teaspoons and

work up to ½ ounce. The juice scraped from the surface of raw swedes and the juice of tinned tomatoes are also good and inexpensive antiscorbutics. Nowadays the antiscorbutic vitamin can be obtained in synthetic form and tablets of 'ascorbic acid' in a dose of about 50 mgm. a day can be given for 2 or 3 days, so that the new diet can be introduced gradually.

The child should also be given baked potato. The best way is to bake the potato in its skin, and scrape away the floury part just beneath the skin and shake that up in the milk. If you put a child who is suffering from scurvy on such a diet, the child will, in the immense majority of cases, get better straight away. There is absolutely nothing in the whole range of therapeutics more striking than the effects which you get from the correct treatment of infantile scurvy, and there is nothing in regard to which you will deserve or obtain more credit in practice. But, on the other hand, there is nothing in regard to which, if you fail to recognize the condition, you will deservedly get more blame, because it is a disease which it is quite within the compass of medicine absolutely to cure if it be only recognized.

PINK DISEASE

In the remainder of this lecture I should like to refer briefly to another disorder, possibly also nutritional in origin, which goes by the name of 'pink disease.' The nutritional diseases we have just considered—rickets and scurvy—are both now generally admitted to be due to vitamin deficiency, but although pink disease has also been ascribed to this cause there is no real proof of it and the true nature of the disease must for the present be regarded as quite unknown.

Pink disease was first described by Swift, of Adelaide, in 1914. It is, however, almost certainly not a new disease, but only newly discovered, for I cannot help thinking that we used to see examples of it at Great Ormond Street years ago, although we were not able to put a name to them. But so soon as Swift's description appeared, others began to recognize the disease all over the world and various names were given to it. Thus it is often spoken of as '*erythrœdema*,' which is not quite accurate, for there is not any real œdema present. Others have called it 'erythrœdema *polyneuritis*' (surely it should be *polyneuritica* ?), but again it is doubtful whether polyneuritis is really a feature of the malady. In

America it is often referred to as *acrodynia*—another unfortunate term, for it has been used to describe other conditions associated with pain in the extremities; whilst on the Continent it is usually denominated '*Feer's disease*,' after Professor Emil Feer, of Zurich, who has given a great deal of attention to it. It is best, however, I think, to use the non-committal term 'pink disease,' first applied to it by Clubbe of Sydney, as that has at least the merit of describing one of its most characteristic peculiarities.

Clinical Features

Onset.—It is essentially a disease of the 'toddling' age, although it may be met with as early as six months or as late as four years, and boys are more often affected than girls. Odd cases may crop up sporadically here and there, or little outbreaks—'islands'—of the disease may be met with from time to time in certain districts. There have been such 'islands' in this country in the neighbourhood of Birmingham and of Newcastle-upon-Tyne; I know of none near London. The disease usually begins in the winter, either gradually or, sometimes, more abruptly, following an infection of the upper respiratory tract or an attack of diarrhœa.

Mental symptoms.—The first symptoms are often mental rather than physical. There is a disturbance of the sense of well-being, the child becomes miserable and depressed and, as someone has said, 'in no other disease of children is the expression so unchild-like, unhappy and life-weary.' Indeed, an experienced observer can often make the diagnosis from the facial expression alone. Along with this there is great restlessness and irritability and a dislike of light, so that the face is often buried in the pillows or the eyes kept screwed up. Sleeplessness is almost constant, and sometimes there is an inverted sleep rhythm—wakefulness by night and drowsiness by day. Meanwhile the child wastes rapidly.

Digestive symptoms.—The appetite is bad and may be almost completely absent, but thirst is usually prominent. The bowels are not specially deranged, but prolapse of the rectum often occurs from want of muscular tone.

In the *circulatory system* tachycardia (120–160) is a very constant feature, and the blood-pressure tends to be raised, sometimes up to 140. These signs are a valuable measure of the severity of the attack. There is a polynuclear leucocytosis of from 15 to 45,000.

The skin shows striking appearances. There is generally a miliary rash, composed of fine papules, which comes and goes and is attended by great itching. Perspiration is often profuse; in no disease of childhood is it more constant. The most conspicuous change, however, is in the hands, feet and nose, which are swollen, podgy but not œdematous, cyanosed if the child is cold and bright, pink if it is warm—hence the name of the disease. Later the hands and feet look as if macerated, like the hands of a washer-woman, and the cuticle peels off in large shreds.

The last component of the clinical picture to be described is *muscular weakness* and hypotonia, which may be so extreme that the child cannot stand or hold up the head, and which is sometimes attended by loss of the tendon reflexes.

Prognosis

The disease reaches its maximum development in about one to three months, after which gradual recovery usually sets in, and is complete in another four or six months. There is, however, a considerable mortality, variously estimated at from 5 to 15 per cent. Death, when it occurs, is usually the result of an intercurrent infection—respiratory, pyogenic or tuberculous. Sudden death without apparent reason, however, is always a possibility.

Pathogenesis

What is the real nature of this curious and interesting disease? We really do not know. Post-mortem investigation usually reveals little, although changes in the peripheral nerves have been described in some cases, which was the excuse for the description 'erythrœdema *polyneuritis*.' These, however, do not seem to be constant, and the most accepted view is that the disease is due to an affection of the vegetative nervous system, but whether functional or organic is uncertain. When one asks further what causes the affection of the vegetative system there is again no clear reply. Like every other obscure disease nowadays, the tendency is to put it down to some 'deficiency of diet'—and indeed it has some interesting resemblances to pellagra—but as to what the deficiency is there is no unanimity. Certainly in my own cases the diet has usually seemed normal enough prior to the onset. Another view is that it is due to an 'infection,' but here again no

one can say what infection, and the whole course of the malady is hardly like that of an infective disease. Anyhow there is no reason to suppose that it is infectious.

Diagnosis

The disease is still often missed, but its recognition is easy if one has once seen a case, the state of the hands and feet being usually diagnostic. The difficult cases are those in which the mental symptoms are alone present, but when in doubt one should look for tachycardia and high blood-pressure. These, if present are strongly confirmatory.

Treatment

There is no specific remedy; general management is the most important line of attack. The child should be carefully nursed and be kept a good deal out of doors, and trouble taken in getting down enough food—often a difficult task. The clothing should be of silk or cotton to avoid irritation of the skin. The diet should be mixed, and it is well to give vitamins freely, although there is no proof of any vitamin deficiency. Yeast has therefore been tried, and, of course, liver, but too much must not be expected from them. Modern concentrates of vitamin B can be given by mouth or injected but the effect varies.

Drugs do not help much, but Feer claims good results from atropine (5–10 drops of 1 in 1,000 up to four times daily); I think I have seen it do good. Sedatives (chloral or bromides) may be required to produce sleep. To allay the itching a dusting powder composed of 10 parts each of camphor and boric acid and 40 each of zinc oxide and talc is useful. High claims have been made for the curative value of artificial sunlight—about nine exposures at three-day intervals—but in this remedy also I have been disappointed. A tepid bath night and morning is soothing and helps to keep the skin in good condition.

On the whole one must depend on careful nursing in good hygienic surroundings and an abundant mixed diet as our chief therapeutic resources. Care must be taken to avoid intercurrent infections, and therefore a hospital ward is perhaps not the best place for these patients.

LECTURE XVII

THE DYSPEPSIAS OF CHILDHOOD

I purpose in this lecture to describe the dyspepsias met with in children beyond the period of infancy. It is not, of course, surprising that disorders of digestion are common in childhood, for when one remembers the importance of alimentation in early life and the enormous demands for food made by growth, it is very natural that the digestive apparatus should sometimes get overworked. Especially is this apt to happen in the city-bred child subject to the strain and confinement of school life. Dyspepsia, then, using the term in its widest sense, is even commoner in the child than in the adult, but the form it takes is very different. The serious organic forms are almost unknown in childhood, for children do not suffer from peptic ulcer or cancer. Further, disorders of the liver and intestine play a much greater part, and those of the stomach a smaller part, in the production of dyspepsia in the child than in the grown-up person. Again, in the adult other things—gall-stones, for example—simulate dyspepsia, in the child dyspepsia simulates other things such as tuberculosis. This is due to the fact that the constitutional symptoms of dyspepsia, such as wasting, tend in the child to overshadow the local complaints of discomfort in the abdomen. Hence it is that the child who is really suffering from dyspepsia is often spoken of simply as ' debilitated ' or is labelled ' neurasthenic ' or ' pre-tuberculous.' It must be admitted, too, that we are still very ignorant of the real nature of the functional disorders of digestion and assimilation which produce the dyspeptic child and this makes a clear account of the subject very difficult, but for purposes of description I would classify the dyspepsias of childhood as follows:

1. Gastric, (*a*) acute.
   (*b*) chronic.
2. Hepatic.
3. Intestinal.

GASTRIC DYSPEPSIA

(*a*) **Acute gastric dyspepsia** is usually spoken of as a ' stomach upset ' or ' bilious attack.' It is probably of the nature of an acute if transient, gastritis. It may result from irritation of the mucous membrane by unsuitable food or from chill. The first of these causes is well recognized, but the second is apt to be overlooked. Sometimes both causes act together. A boy, for instance, ate a hot mutton pie, and then hung about in a cold wind watching a football match till he was thoroughly chilled. He went home and was immediately seized with an acute gastric attack. The symptoms in such a case may be severe—more severe than is commonly recognized. They consist in abdominal pain, vomiting and fever. The temperature may go quite high and even be accompanied by a rigor. I remember, for instance, the case of a little boy who was convalescing from whooping-cough. One afternoon he complained of pain in the abdomen and his temperature ran up to 105° F. Shortly afterwards he vomited a quantity of undigested carrot and in a few hours the fever had gone. Nervous manifestations, such as twitching or convulsions, may also occur. Take another case. A little boy whilst out in his pram in the park was noticed to go pale and to have slight twitchings. He was hastily taken home where it was found that his temperature was nearly 103° and he was rather collapsed ; his father, who was a doctor, administered an emetic, a quantity of badly digested food was thrown up and in a few hours he was practically well.

It can readily be understood that such cases are easily mistaken for the onset of an acute specific fever or even for meningitis. Pneumonia, also, may produce very similar symptoms in its early stages. In the less severe cases one has to think of the possibility of an acute abdominal crisis—especially appendicitis—the usual local signs of which must be looked for.

The treatment of acute gastric dyspepsia is simple. If vomiting has not already occurred an emetic should be administered, warmth applied to the abdomen and only sips of water or soda water containing bicarbonate of soda (one drachm to the pint) given by the mouth. The bowel should be emptied by an enema. When the acute phase is over a bismuth mixture will help to bring about complete recovery.

(*b*) **Chronic gastric dyspepsia.** There are two causes of this

in children: (1) chronic gastritis, (2) functional disorders of the stomach.

(1) Chronic gastritis may arise out of an acute attack which has not completely cleared up or it may be the result of chronic irritation of the mucous membrane by food which is chemically or mechanically unsuitable. Cane-sugar in high concentration is an irritant of the gastric mucous membrane and is a cause of gastritis in some children. Fats, especially hot fats, are another cause. Ill-chewed and bolted food acts mechanically. Chill also is a factor and some children seem to get a chronic gastritis when exposed to cold just as others get a bronchitis.

The symptoms of chronic gastritis consists in poor appetite, nausea, occasionally morning retching or vomiting and some flatulence. The tongue is usually coated, but otherwise there are no physical signs. Treatment consists in a bland simple diet, attention to chewing and to the teeth, protection from cold and the administration of bismuth, rhubarb, and soda before meals with mercurials and laxatives if necessary.

(2) Functional disorders of the stomach as a cause of dyspepsia in children are more obscure even than in the adult. Children are not able to give an accurate account of their sensations and cannot describe such symptoms as heartburn, acidity, or even flatulence. Comparatively few investigations also have been made in children by means of test-meals and these have yielded contradictory results. Hence, it may be said that of disorders of secretion as a cause of dyspepsia in childhood we know practically nothing. Even of disorders of motility we are very ignorant, but in older children one meets not infrequently with the splashy stomach which probably indicates a lack of muscular tone. It is to be treated in the usual way with a dry diet and a mixture of soda, nux vomica, and bitters before meals.

I would repeat that chronic gastric dyspepsia is not common in childhood and that when it is met with a chronic catarrh is most likely to be the underlying cause.

HEPATIC DYSPEPSIA

By hepatic dyspepsia I mean that form in which a functional disorder of the liver appears to be the cause of the symptoms. Such cases are quite common and are probably getting commoner.

They are met with in children from the age of three upwards. The child will usually be brought to you with the complaint that he is thin or does not gain weight and has a poor appetite. He is usually nervous and irritable by day and sleeps badly at night. The bowels are generally constipated, but there is no complaint of pain or other abdominal symptoms. Two features are very characteristic and clinch the diagnosis. One is that the child often has a ' bad breath ' and the other is that the motions are at times pale in colour. Such is a general account of these cases, but this chronic picture is chequered by ' episodes ' which may take the form of vomiting of a cyclical character (p. 412), of recurring rises of temperature (' food fever,' p. 434), of acidosis or of headache of the migraine type.

What is the cause of hepatic dyspepsia ? I believe it is due to two causes. First, to an inborn inefficiency of the liver (so-called ' hepatic inadequacy ') which may be a family peculiarity and, secondly, to an over-use of the ' liver foods.' The chief of these foods are milk, cream, and eggs—the dairy products in fact—but such accessory articles as chocolates (owing to their cocoa fat) and oranges are also ' bilious.' The attempt to fatten these children by giving them quantities of milk and cream is the main cause of their dyspepsia ; the liver seems to get glutted with imperfectly metabolized material. The episodes of vomiting or fever appear to serve the purpose of clearing out the liver and enabling a fresh start to be made. I believe also that the present craze for giving children large quantities of orange juice is responsible for a good deal of hepatic dyspepsia, although it is difficult to say how it produces this effect. I shall deal with the treatment of hepatic dyspepsia later on.

INTESTINAL DYSPEPSIA

The third variety of dyspepsia is the intestinal and, like the hepatic, it is very common. It is sometimes known in this country by the name of ' mucous disease ' which was first given to it, I believe, by the late Dr. Eustace Smith.

I want to describe to you what those cases of mucous disease are like, and to try to draw for you a **clinical picture of the condition.** The child will usually be brought to you by the mother with the complaint that he is wasting. She may tell you in addition that

he is languid and peevish, and that he does nothing but 'lie about' all day. Very frequently you will hear the complaint that the child has a cough, and the combination of wasting and cough is often the thing chiefly noticed by the mother, and it is apt to throw you off the scent, and lead you to believe that you are dealing with a case of pulmonary tuberculosis. Over and over again such cases are sent to hospital with the diagnosis of phthisis. When you come to inquire further into the symptoms you will usually be told that the appetite is very poor. Sometimes, on the other hand, you find quite the opposite condition of things, for there is a voracious appetite. The mother will say that, in spite of the fact that the child eats a great deal, he does not seem to get any fatter. In those cases where the appetite is voracious, you will sometimes get a history that the child has a liking for quite abnormal things. You may find that he has been eating coal and cinders, or has been going to the wall and picking off pieces of plaster and eating them. To that condition of abnormal appetite the term 'pica' has been applied.

When you inquire into the condition of the bowels, you will generally be told that there is constipation. You will hear that the motions occur at irregular, and sometimes prolonged, intervals; that the stools consist of more or less hard lumps, surrounded often by material which the mother compares with white jelly—that is to say, by mucus, and that they contain undigested food. Very commonly you will get a history that the child is troubled with worms. Proceeding further in your investigation of the case, you will find that these children usually sleep badly at night, that their slumbers are restless and disturbed by unpleasant dreams, and there may even be a tendency to somnambulism. Many of them have nocturnal incontinence of urine. The mother will frequently tell you also that the child becomes very pale at intervals; she will say that he goes 'deathly white' for a few minutes, maybe repeatedly throughout the day. These fits of pallor are a little difficult to explain. Some people attribute them to colic. Dr. Angel Money described them as cases of 'vaso-motor epilepsy,' because he supposed they were due to spasmodic discharges from the vaso-motor centre analogous to the spasmodic discharge from the brain cortex in epilepsy. I need hardly say that that is a purely hypothetical explanation.

When you come to inspect the child for yourself, you will usually

find that he is at or about the period of second dentition—the majority of the cases occur in children between the ages of five and eight. The child usually looks poorly nourished; the complexion is pale, and frequently there are dark rings under the eyes. On examining the tongue you will find that it presents certain well-marked features. As a rule, it is covered by a thin fur, through which you can see projecting red points, which are the enlarged fungiform papillæ. Or, in other cases, and these perhaps the most typical, the tongue has a glazed appearance, and is of a slightly yellowish or fawn-coloured tint, looking, as it has been said, as if it had been brushed over with a solution of gum. In yet a third group you will find that the tongue shows peculiar mapped-out areas, curious irregular patches where the epithelium seems to be denuded, which have been fancifully compared to the continents in a map of the world; hence it is called the 'geographical tongue.'

On proceeding to examine the child further, you will find that the skin is dry and harsh. Usually you cannot make out any physical signs of disease in any of the great viscera; the heart, lungs, and abdominal organs appear to be healthy. But when you come to inspect the throat, you will almost invariably find that it is in a more or less unhealthy condition, that there is chronic pharyngitis, more or less enlargement of the tonsils, and very frequently adenoids in the naso-pharynx. It is really to the unhealthy condition of throat that the cough in these cases is due, and that fact I cannot too strongly impress upon you. The majority of cases of cough which are brought before you in children are cases of throat-cough, and not lung-cough at all. When you examine the urine you will often find that it is turbid, owing to a deposit of urates. Sometimes in such a case the mother will describe the urine as being 'milky' when passed, that appearance being due to pale urates. On chemical examination you may find that it contains albumin, probably in many cases nucleo-albumin. Such children make up a large proportion of the so-called 'cyclic albuminurias,' which are, I think, better termed simply functional or postural albuminuria—that is to say, cases in which there is no organic disease of the kidney, but in which albumin or protein appears in the urine at particular times of the day, usually after the child has got out of bed and is going about.

Such are the chief symptoms of a typical case of intestinal dyspepsia. But you will often meet with cases which are not so typical

as that, but in which one or other group of symptoms tends to overshadow the rest. And I think you can distinguish several such special groups. First, there are those in which the **constitutional symptoms predominate.** By that I mean that the chief symptom is the wasting, accompanied by mental depression and languor, and perhaps by some degree of mental irritability. Then there is a group in which the **abdominal symptoms predominate.** The chief complaint in those cases is that there is pain after food—a pain which you will frequently have difficulty in referring to the stomach or colon, but which may probably have its seat in both, owing to flatulent distension of the stomach and large bowel. Other patients who certainly suffer from this kind of intestinal disorder will be brought to you simply for **worms,** and I want to impress upon you at this point that you should always regard worms as a symptom and not as a disease. They are a symptom of an unhealthy state of the bowel, and not a disease in themselves. Others will be brought to you for **restless and disturbed nights,** the so-called pavor nocturnus, or night terrors.

Just as in the hepatic variety of dyspepsia the chronic course of the disorder may be broken by episodes of vomiting or fever, so in the intestinal form there may be episodes which take the form of 'lienteric diarrhœa,' 'umbilical colic' or 'faints.'

By **lienteric diarrhœa** is meant a looseness of the bowels which occurs immediately on the taking of food so that the child may have to rush from the table to relieve himself. It is probably due to an exaggerated reflex irritability of the colon. The so-called '**umbilical colic**' has probably got a similar cause, but I shall deal with it more fully in another lecture (p. 423). The '**faints**' I have also described elsewhere (p. 292), they are probably only an exaggerated form of the attacks of 'pallor' which I have already spoken of.

We will pass on now to glance at the **pathology** of this condition so far as we know it. It is difficult to speak at all dogmatically in this matter, because there is an almost complete absence of post-mortem evidence in regard to it. It is not a fatal disease; it leads to much ill-health, and it predisposes the child to suffer from more serious disorders, such as tuberculosis, but it is not in itself inimical to life, and thus there is no opportunity for investigating its morbid anatomy. Dr. Eustace Smith had a very neat theory to explain all the symptoms detailed above. He said that mucous disease is due

to an over-secretion of mucus throughout the whole alimentary canal; that the excess of mucus produces in the stomach sluggish and painful digestion; in the intestine it wraps round the intestinal contents, the consequence of which is that the digestive juices get imperfect access to the food, and hence absorption is interfered with, and as a result the child wastes. Moreover, the scybalous masses in the large intestine get coated over with mucus, so that the wall of the gut has no purchase over them to squeeze them along, but glides over the masses; hence the frequency of constipation. In addition, the mucus forms a good nidus for the worms to live and feed and lay eggs in. One might go further, and say that in the cases in which there is albumin in the urine it is very often a nucleo-albumin, and that this is due to analogous over-secretion from the cells lining the urinary passages. And one might also point to the throat, and say that there, too, one finds chronic catarrh and the production of an excessive quantity of mucus. This theory, then, as you will see, is very neat and ingenious, and it does explain fairly satisfactorily most, at least, of the symptoms; but it is one which it is very difficult to be sure about.

There are many who do not accept the mucus theory, but who consider that these cases are more obscure than any such explanation as that would lead you to believe. They think that one is here dealing with a disorder of metabolism—defective power of assimilation which they compare to the gouty condition in grown-up people. They say—and especially French authors—that those children have the 'arthritic diathesis,' whatever exactly that may mean.[1] Others again attribute a large part in the production of the symptoms to intestinal auto-intoxication—a view as to which I must confess myself rather sceptical.

Whatever the exact pathology of intestinal dyspepsia may be there is general agreement that it is chiefly brought about by unsuitable diet and particularly by a diet containing an excess of sugar and starch, although nervous over-strain, want of fresh air and exercise, and the swallowing of infected material from an unhealthy throat may also play a part in its production.

Before passing on to the treatment of these forms of dyspepsia I should point out that you will not always meet with pure-bred cases but must expect in many patients to find features of the gastric,

[1] The latest fashion is to speak of them as having the 'exudative diathesis'—whatever *that* may mean.

hepatic and intestinal types, in combination, although one type usually predominates in the clinical picture.

Treatment

I have already described the treatment of gastric dyspepsia and shall deal now with that of the hepatic and intestinal forms. The first thing to be done, especially in the intestinal variety, is to regulate the child's life as a whole. It may be necessary to reduce the hours of school or to arrange for more time out of doors or more sleep. Change of air is of the greatest benefit. In the intestinal cases a bracing seaside place usually suits well, but in the hepatic type a high inland resort is preferable. Chill must be avoided and to this end the wearing of an abdominal belt or binder is helpful. The feet and legs must also be kept warm. Attention should be given to the state of the teeth and if the tonsils are unhealthy they should be removed. Meals must be properly spaced and should be taken leisurely with no rushing off to school or play immediately afterwards, and the habit of thorough chewing must be insisted upon.

Having dealt with all these points, we come next to the question of diet. In hepatic dyspepsia the ' liver ' foods already mentioned must be greatly reduced. The child should have no cream, little milk and few eggs. Chocolates, oranges and all rich foods should be forbidden. Butter must only be allowed in moderation.

In the intestinal form the starchy and sugary foods must be cut down. No sweets and but little sugar should be allowed, potatoes should be restricted and crisp toast and rusks substituted for bread. Wholemeal and brown breads are specially harmful in such cases. Raw fruits and raw vegetables must also be forbidden as well as such things as raisins and currants which pass through the bowel undigested. Green vegetables must be well cooked and, if necessary, sieved. Eating between meals must be avoided. All these points should be impressed upon the mother in detail.

And now as to drugs. In the hepatic cases the most useful drugs are rhubarb and grey powder, and 2 or 3 grains of the former with $\frac{1}{2}$ to 1 grain of the latter may be given every night with advantage for weeks on end. For intestinal dyspepsia I usually give alkalis and bitters before meals—the old soda, nux vomica, and gentian mixture does very well—along with a laxative at night. Rhubarb is the best aperient as it has the power of lessening the production of mucus but cascara, aloes and senna are also suitable. These

children are often irritable and sleep badly and a very useful remedy is to add a little bromide to the standard 'rhubarb and soda' mixtures which exists in the pharmacopœia of most hospitals:

Pot. brom.	gr. i
Pulv. rhei.	gr. i
Sod. bicarb.	gr. i
Syr. zingib.	℥v
Aq. menth. pep.	ad. ℥i t.d.s., a.c.

You may be tempted to give iron to these children as they are usually pale and often even anæmic, but this should not be done until the tongue is clean and the bowels acting regularly. Cod-liver oil and malt—another favourite prescription—should also be postponed to the stage of convalescence.

Finally, a word as to the treatment of some of the 'episodes.' Proper treatment of the underlying dyspepsia will usually abolish these altogether, but at times they may need to be dealt with for themselves. During an attack of **cyclical vomiting** the great thing is to give bicarbonate of soda and glucose freely, but I shall describe the treatment of this condition in another lecture.

Lienteric diarrhœa is easily controlled by a drop or two of tinct. opii given before each meal; this may be added to the alkaline and bitter mixture if that is being given.

Umbilical colic is sometimes difficult to control, although it usually disappears when the diet and bowels have been regulated. In obstinate cases the best plan is to give tinct. opii as in lienteric diarrhœa, but with the addition of twice as much tincture of belladonna before meals and a laxative at bedtime. The treatment of **faints** I shall deal with in another lecture (p. 292) and I may conclude what I have to say to-day with an account of the methods of dealing with **intestinal worms.**

INTESTINAL WORMS

Three varieties of worms may inhabit the alimentary canal of children, the tape-worm, the round-worm, and the thread-worm. On rare occasions other forms are met with, but for practical purposes these are the only worms we need discuss as affecting children in this country.

Tape-worms.

Two varieties of tape-worms are found in children, *Tænia cucumerina* and *Tænia mediocanellata*. The first of these is much rarer

than the second, and is, in fact, not very well known. It is a comparatively short tape-worm, and its presence is recognized by the passage of small oval segments. Both the cases I have hitherto met with were in infants at the breast, as is usual with this worm ; this extraordinary point in the distribution of *Tænia cucumerina* naturally causes difficulty in diagnosis. You may ask, How in the world does a child nursed at the breast get a tape-worm ? It appears that this particular worm is always acquired from cats, and in every case it is found that the sufferer has been in close association with one of these animals.

Tænia mediocanellata is the common tape-worm with which you are all familiar, both in children and in adults. It seldom occurs in young children, but is frequent at and after the second dentition.

Children are often brought by their mothers for advice with regard to worms. Various symptoms are supposed to be caused by their presence, such as wasting in spite of good appetite, abdominal pain, and so on. Let me say at once that, except possibly in the case of thread-worms, I know of no symptom that points definitely to the presence of worms. The only method by which you can diagnose the existence of worms in the alimentary canal is by actually seeing segments or entire individuals in the fæces.

Treatment of tape-worms.—This is not such an easy matter as one might suppose. You will often fail to get away the head, especially if the condition is not taken seriously enough, and treatment is half-hearted.

The patient should remain at least twenty-four hours in bed, and for some hours before the anthelmintic is given and until the worm is passed, no solid food at all must be allowed. Indeed those with a great experience of treating tape-worms in adults lay great stress on this preliminary starvation phase and say it should last a full forty-eight hours. Begin in the evening by giving a purge so as to clear the alimentary canal as much as possible, and thus expose the worm to the direct action of the drug. A full dose of castor oil, or of liquorice powder, or of calomel, should be administered ; it does not very much matter which, provided the dose is a sufficient one.

Of the anthelmintics none surpasses male fern, which, on account of its nauseous taste, is best given in capsule in 15-minim doses every quarter of an hour for four doses. A child of the age at which tape-

worm is common will stand such a dose quite well. Having given four such capsules it is best to wait for an hour or so, and then to give another purge to clear out the worm. As a quickly-acting aperient is wanted, an effervescing saline, followed by a hot drink, is useful. Castor oil is not suitable.

It is very important to attend to details in the passage of the resulting motion. It should be passed into warm water to diminish the risk of breaking the worm. There should be a sheet of black crêpe on the bottom of the vessel, which assists the search for the head of the worm; this can often, with a little practice, be recognized by the naked eye. It is only when the worm is completely evacuated and the head seen that one can be sure of success.

If the treatment fails it is not advisable to repeat it until after an interval to allow of growth. As a rule this takes about two months. The essentials are rest, starvation, purgation, full doses of an anthelmintic, and care in the way in which the worm is received when passed.

About the treatment of *Tænia cucumerina* I cannot tell you much. Personally I should be rather afraid of giving male fern to a little baby; I should prefer calomel, and perhaps small doses of santonin as well. Both the cases to which I have referred had calomel only, and soon ceased to pass segments, though I had not the opportunity of searching for the head.

Round-worms.

As in the case of the tape-worms, there are no symptoms characteristic of this worm, the *Ascaris lumbricoides*. There is, however, no doubt that it may cause reflex nervous disturbances of various kinds. I have myself seen cases of convulsions which ceased after the passage of a round-worm. These worms are probably derived from certain kinds of food, particularly raw vegetables, such as salads. I once had to do with a family of strict vegetarians, in which the father, mother, and both children were suffering from round-worms, and I do not doubt that it was from their vegetables that they had contracted the parasites. With a view to prevention it is well to see that anyone who has had a round-worm should avoid such foods, or at least take the most careful precautions about washing and cleaning them.

The round-worm is the only kind of common worm to which any danger attaches. The danger arises from the fact that they are always prone to wander. When the worm wanders upwards into

the stomach, it is generally promptly vomited; but it may obtain entrance into the larynx and cause suffocation, or into a bronchus and set up bronchiectasis. If a child passes a round-worm it should be at once put through a course of treatment, for these animals often hunt in couples. Fortunately round-worms are more easily got rid of than tape-worms.

Santonin is the best remedy, for it is effective and safe; it should be exhibited as a powder in this combination:

Santonin	gr. $\frac{1}{6}$
Calomel	gr. $\frac{1}{6}$
Sugar	q.s.

Give one such dose for every year of the child's age every night for three or four nights. So much, then, for round-worms: their treatment is simple, and when once the worm is seen there is no anxiety as to any of it having been left behind.

Thread-worms.

The third and commonest variety of worm that attacks children is the *Oxyuris vermicularis*. It is very important to realize that thread-worms are not a disease in themselves, but a symptom of an unhealthy state of the large bowel. I doubt very much whether a thread-worm can exist in a bowel that is healthy. The condition required is one of catarrh associated with the presence of mucus. It is in the folds of the swollen mucous membrane that the worms lodge, and for success in treatment this condition must be remedied.

Another difficulty is that the patient, if a child, is constantly reinfecting himself. Further, the ova are often deposited in the vermiform appendix, which forms a regular breeding-place for oxyuris. When once one has seen such an appendix full of young worms, it can be realized how difficult it is to stop the constant reinfection.

This brings us to the symptoms caused by thread-worms. There are certain purely local effects, such as irritation, scratching, and so on. Beyond these, it is quite possible that incontinence is sometimes due to worms, but there is much less ground for the belief common amongst mothers that picking at the nose is evidence of them. As regards pallor, abdominal pain, wasting, headache, and other vague symptoms, I am convinced that these are really due to the condition which permits the worms to flourish, described in the earlier

part of this lecture, and to get rid of the worms permanently it is necessary to deal with this underlying condition by the methods detailed above.

Recent work in America, especially in the elaboration of efficient methods of diagnosis by means of an anal swab technique, has shown how ineffective most accepted methods of treatment really are. The best results, carefully checked by anal swabs have been obtained by gentian violet. I have used this now quite a lot and it certainly is the best remedy I know. You give $\frac{1}{6}$ grain per day, per year of age ('apparent' age is what the Americans say) in enteric-coated capsules or tablets. Work out the total dose and give the drug three times a day after meals for five days. Then wait for five days and give a second five-day course. The maximum dose is 2 grains a day. No enemas are required but local treatment is an additional safeguard.

A small portion of ung. hydrarg. or white precipitate or ointment, inserted into the rectum every night for a few nights is also a very useful local remedy, to relieve itching and to prevent wandering of the worms. The child should also wear sleeping garments so constructed as to make access of the fingers to the anus impossible. The child's finger-nails should be kept short and the hands and nails should be frequently scrubbed. Gloves should be worn at night.

Removal of the appendix has been suggested—and sometimes carried out—in the relapsing cases with the object of getting rid of the breeding-ground of the worms. Such a proceeding, however, must always be rather speculative.

Thread-worms are often a familial complaint and if the condition recurs in a child I advise you to make inquiries about other members of the family.

VIRAR MADRID OFFICE

LECTURE XVIII

RHEUMATISM IN CHILDHOOD

There are few diseases commoner than rheumatism in one or other of its many forms in childhood, and one may say also that there are few more serious, or in relation to which your responsibility is greater ; for rheumatism in early life is often the parent of cardiac disease in later life, and many people suffer from disease of the heart for a large part of their adult existence because they had an attack of rheumatism which was overlooked by the medical attendant in their childhood. That is why I want to impress upon you your responsibility in relation to this disease.

By rheumatism in children I mean something quite specific and definite. You know that the term in relation to adults is one which is used in a very vague sense. One talks of ' chronic rheumatism ' of a joint, for instance, without having any very clear idea before one's mind of the exact pathological state of that joint. Now, in the case of children one does not talk in that loose way. One means by rheumatism **an acute specific disease,** which is almost certainly due to a specific micro-organism. Indeed, I think the modern view that acute rheumatism is due to a micro-organism receives strong support from the study of the disease in childhood. All micro-organismal diseases, all acute specific fevers, as you know, tend to be commoner in early life, and the great frequency of rheumatism in childhood is an *a priori* argument in favour of the view that it also is due to a specific micro-organism. Most people have now come round to that opinion, and they regard rheumatism as an infective disease, due probably to a streptococcus. Now, though rheumatism may certainly be regarded as being due to a micro-organism, you require for its production not merely the organism but a suitable soil. Clinical observation shows that all children are not equally subject to attacks of rheumatism. There are few diseases, indeed, in which heredity plays a more definite part. You constantly find, when you inquire into the family history of

children who are suffering from one or other manifestation of acute rheumatism, that other members of their families have been similarly affected; and I think you can recognize when you meet them children who are so predisposed by heredity.

I have often pointed out to some of you in the wards and in the out-patient room the **typical rheumatic child,** from whose facies you can say that he is prone to become the subject of this disease. These children are usually past the second dentition, for young children rarely suffer from rheumatism. They are dark rather than fair; their hair is dark, the eyes are dark, and they have long, dark eyelashes. At the same time, they have a peculiarly white skin and a very good complexion; they have a clear, bluish-white sclerotic, and they have often very well-formed, massive teeth, and particularly large, square, central upper incisors—the so-called 'paving-stone' teeth. They also exhibit very constantly what is termed a neurotic temperament—that is to say, more than other children they are subject to minor nervous disorders. I think you will find it an advantage to have such a picture of the typical rheumatic child before you. It has occurred to me several times by recognizing these characters to suspect rheumatism, or to be specially on the look out for it, or to be able to say of a particular child that he would easily become the subject of rheumatism, and so to advise the taking of special precautions against it.

Unfortunately, we do not know how those children become infected. We do not know definitely the path by which the organism gains access to the body. But I think there is at least a reasonable amount of evidence to show that infection takes place in the majority of cases through the throat. Now, **unhealthy throats** are so common in children that one need never be surprised that the local resistance there is lowered; and I believe that a great many diseases could be prevented—and among them rheumatism—by a greater attention to the state of children's throats. The organism, having gained access by the throat or by some other route of which we are ignorant, tends to manifest itself in certain tissues more than in others, and in particular the fibrous tissues and membranes are subject to its attack; I mean such membranes as the valves of the heart, the tendons, the sheaths of muscles, and the fibrous aponeuroses. These are the **tissues which are most vulnerable,** it would appear, to the attacks of the rheumatic virus. Now, in this respect rheumatism, as it shows itself in children, differs considerably from

the same disease as you see it in grown-up people, and it is much to be regretted that the ordinary textbook description of rheumatism is drawn from a study of the disease as it occurs in adults. Rheumatism ought to be described as it occurs in children, because it is in children that you see it in by far its most virulent manifestation.

No one who has seen much of the disease in children will fail to recognize that the joint manifestations are often very trivial or altogether absent, so that, had it been studied primarily in children it would never have been called acute *articular* rheumatism, which is the name often given to it in textbooks. That title is correct as regards grown-up people—in them the joint manifestations dominate the whole picture—but in children it is not so. In them rheumatism may invade the body, and almost pass over the joints altogether; or, at all events, the joint affection may be so trivial and transitory that it may be completely overlooked.

On the other hand, it is an unfortunate fact that it is the heart more, perhaps, than any other part which tends to be the chief point of attack of the rheumatic poison in children, and for that reason the disease assumes in them a degree of danger and importance which is wanting in the case of the adult. Not only is the joint affection often exceedingly trivial in children, but you will often find that the degree of fever is extremely slight. Children suffering from acute rheumatism may have a rise of temperature amounting to only one or two degrees, and they hardly ever exhibit that dangerous rise of temperature which is spoken of as rheumatic hyperpyrexia. For some reason, the whole febrile side of the disease is much less marked in the young. Indeed, some people go the length of saying that it would be more true to describe the carditis as the essential part of acute rheumatism in children, and the joint manifestations as mere complications, and that it is quite wrong to assume the attitude which one is apt to do, and speak of the joint lesion as the essential thing and the carditis as the complication.

The next point I want to emphasize is that the manifestations of rheumatism in early life are far more protean than in grown-up people. There are all sorts of forms of rheumatism in children which you may be apt to think are not rheumatic at all unless the fact is first pointed out to you. One may classify the **chief manifestations** of the disease as follows: (1) Articular inflammation,

the ordinary synovitis of joints; (2) muscular or fascial rheumatism; (3) rheumatic nodules; (4) endocarditis, myocarditis, and pericarditis; (5) erythematous eruptions of various sorts; (6) chorea; (7) pleurisy; and (8) tonsillitis. I have not mentioned these rheumatic manifestations in the order of their importance. If one had done this, one would have placed the heart manifestations first. I have merely put them in series to show you how varied are the forms assumed by rheumatism in early life.

Now, these different manifestations of the disease may coexist, or they may succeed each other in any order. Given a child predisposed to rheumatism, he may show at one time a few articular pains. These pains may pass off, and a few months later the child may have a little muscular and fascial rheumatism somewhere; later on, perhaps, he gets an attack of chorea, and that may be complicated by an eruption of rheumatic nodules and by some inflammation of the pericardium; or the child may have repeated attacks of tonsillitis, and occasionally fugitive erythematous eruptions, or even, perhaps, an attack of pleurisy; and in that way all these events may be spread out over childhood, one occurring at one time and one at another, but all being simply different manifestations of the one infection—namely, that of acute rheumatism.

I now want to look briefly with you at each of these members of the rheumatic series.

The **joint pains** I have already referred to. I have pointed out that they are often so slight as to be overlooked. But if you watch your cases of rheumatism carefully you will usually find *some* involvement of the joints at least at one or other period of the illness. It is very rare for a case to run anything like a prolonged course without there being at any rate some articular inflammation but its degree may be extremely slight. Further, the inflammation of the joints is not so intense in its local manifestations as in the grown-up person; there is much less swelling and heat, hardly ever any redness, and there is comparatively little pain.

The **muscular and fascial rheumatism** of childhood deserves your special attention, not because it is of importance in itself, but because it should serve as a danger-signal. The pains of muscular and fascial rheumatism are commonly called, in the nursery, 'growing pains.' That is a phrase which has crippled a great many hearts. Mothers and nurses are apt to regard growing pains as something physiological, something to be expected in growth.

I need hardly say to you that that is an absolutely mistaken view. Those so-called growing pains are, in the great majority of cases, nothing more than the local manifestations of rheumatism in the muscles and fascia, and if they are neglected and the child is allowed to go about with them, he may very easily slide into endocarditis. These pains are particularly apt to affect certain parts of the body. I think you will find that the tendons of the hamstring muscles are more commonly involved than any other part, and that may lead you into errors of diagnosis. For instance, you may think that you have to do with a case of spastic paraplegia because the child walks on his toes, with the knees slightly bent, in order to spare the tendons ; or you may suspect some bone trouble or some affection of the knee-joint.

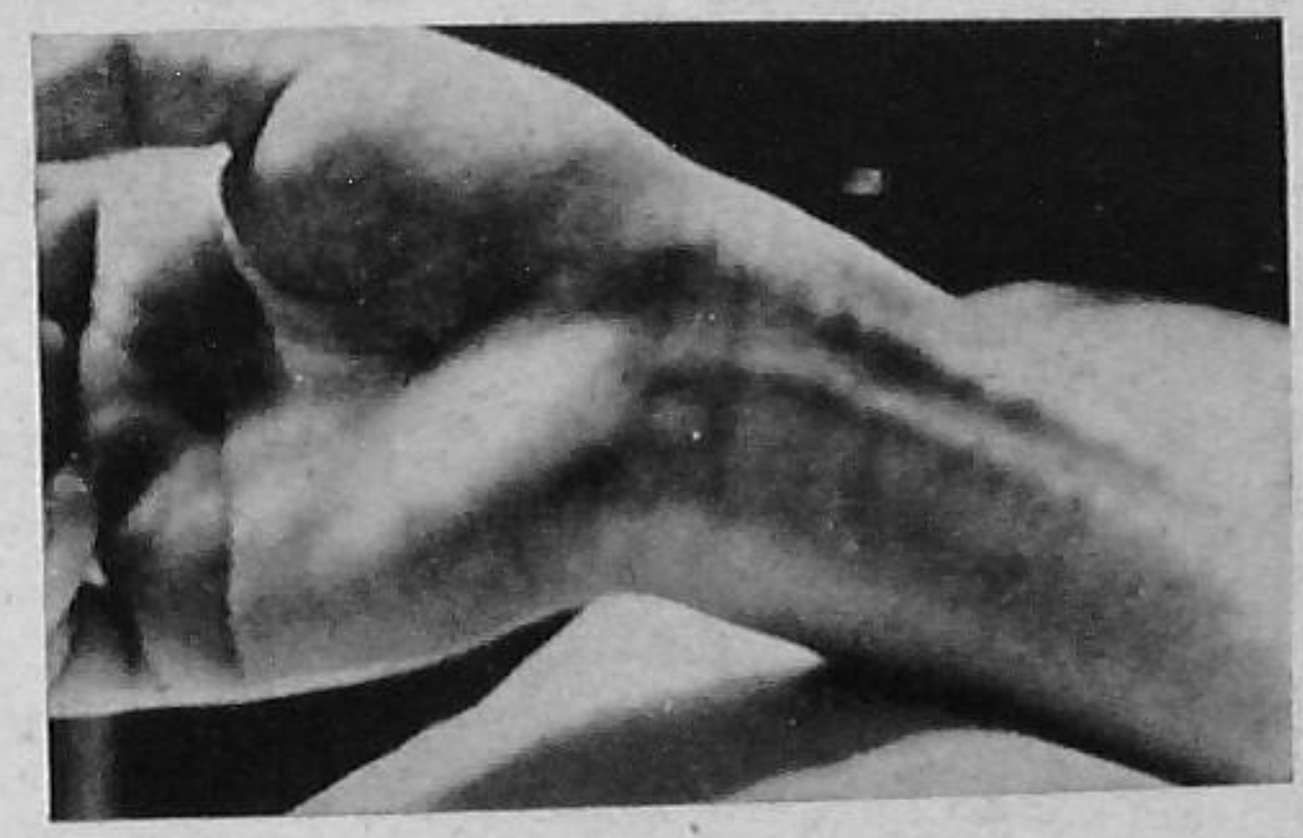


FIG. 43.—RHEUMATIC NODULES ON TENDONS OF THE WRIST.

The appearance of **nodules,** the third manifestation I have mentioned, is almost peculiar to early life. I think I have only twice seen rheumatic nodules in grown-up people, whereas in children they are fairly frequent. Probably many of you are familiar with the appearance of these nodules ; they are little things, varying in size from a pin's head to the size of a pea or bean. They tend to occur under the skin where the bones come near the surface. Thus you find them over the olecranon and the patella, or on the spines of the vertebræ, down the centre of the back. You may discover them also forming a festoon around the edge of the scapula, or dotted along the occipital suture at the back of the skull. You may also sometimes find them over the tendons. Presently I shall show you by means of lantern slides some nodules in one or two of these situations (figs. 43, 44, 45, 46). But I want to impress upon you that they require to be carefully looked for. They are

often more easily felt than seen, and they are apt to escape your observation. They are characterized by being perfectly painless

FIG. 44.—RHEUMATIC NODULES ON ELBOWS.

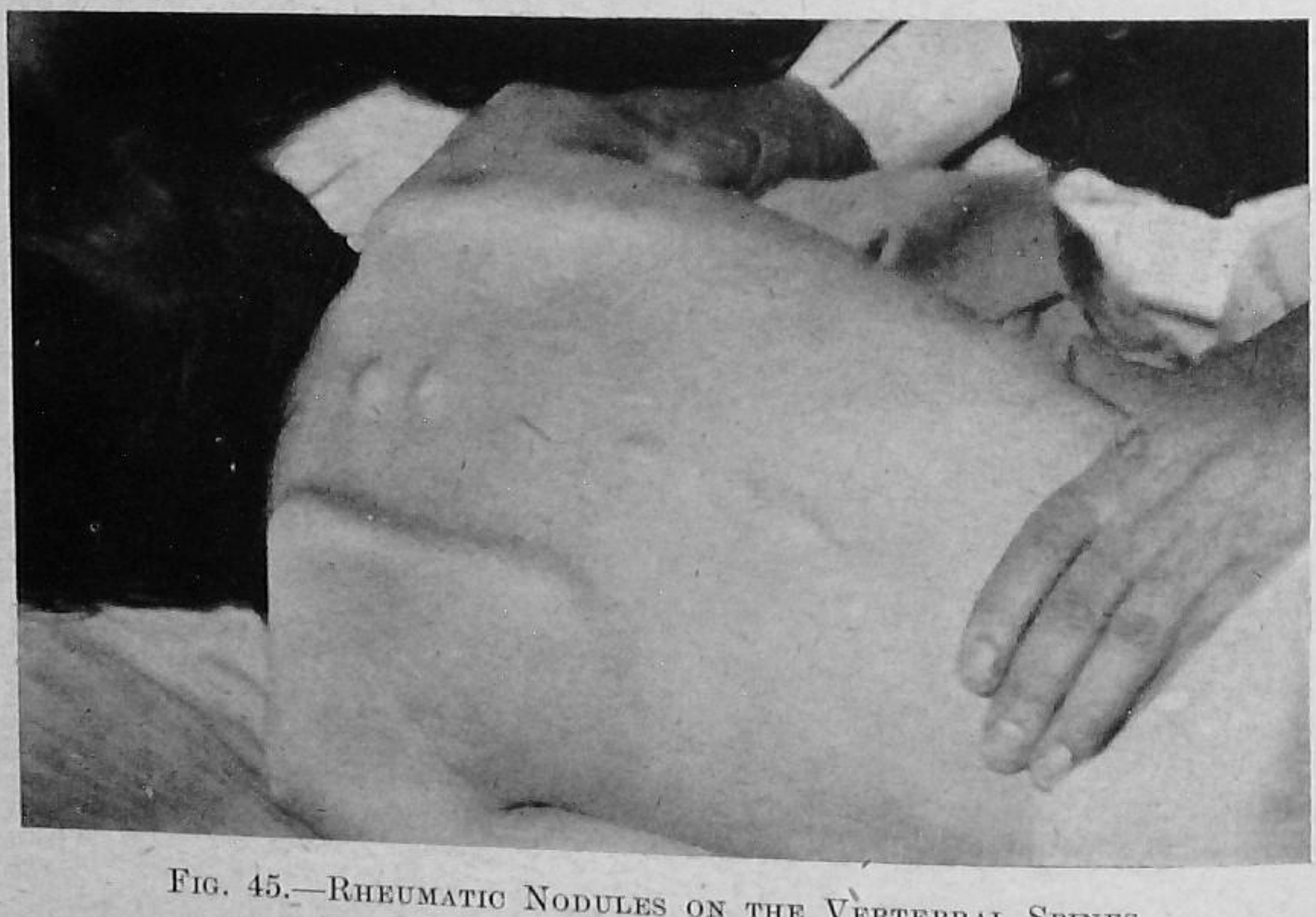


FIG. 45.—RHEUMATIC NODULES ON THE VERTEBRAL SPINES.

which may surprise you, and by being extremely mobile. When you cut into them you find they consist of little nodules of fibrous tissue, the fibres being arranged concentrically round a core of

fibrin. Those nodules are not in themselves of any importance whatever, but, like the rheumatic pains which we have been considering, they are of the greatest importance as danger-signals, because if you find rheumatic nodules coming out you may be fairly certain that there is progressive heart mischief going on. You may compare them, if you like, to the vegetations which form on the edge of the valve in the heart. Just as in endocarditis of the mitral valve vegetations form along its edge, so in the case of nodules there are analogous fibrous deposits under the skin; but in the one case you speak of a vegetation and in the other case you speak of a

FIG. 46.—RHEUMATIC NODULES OF THE SCALP.

nodule. Pathologically they are very much the same thing in the two cases. In the case of the nodules you may say they are vegetations which you can see and feel, while you may call the vegetations on the valves of the heart nodules if you like.

The fourth group of manifestations are those which occur in the **heart.** These, I need hardly say, are the most important of all, and for that reason I shall reserve their consideration for another lecture.

I now pass on to consider **erythemata** as a sign of rheumatism. The commonest manifestation is extensive areas of erythema over the trunk, a condition which is often spoken of as *erythema marginatum.* You will find that it tends to occur in circular patches with

crescentic outlines. It may involve almost the whole body at one time, and may be, and not uncommonly is, accompanied by rheumatic manifestations in the heart (fig. 47).

Of **chorea** I shall have something to say presently. It is a disease which you have plenty of opportunity of seeing in the wards, and it is hardly necessary for me to take up your time with a description of it. I shall only say that as regards its relation to rheumatism most people believe that in about 75 per cent. of the cases of chorea there is an undoubted rheumatic association. Some say it is invariably rheumatic, because you must remember that

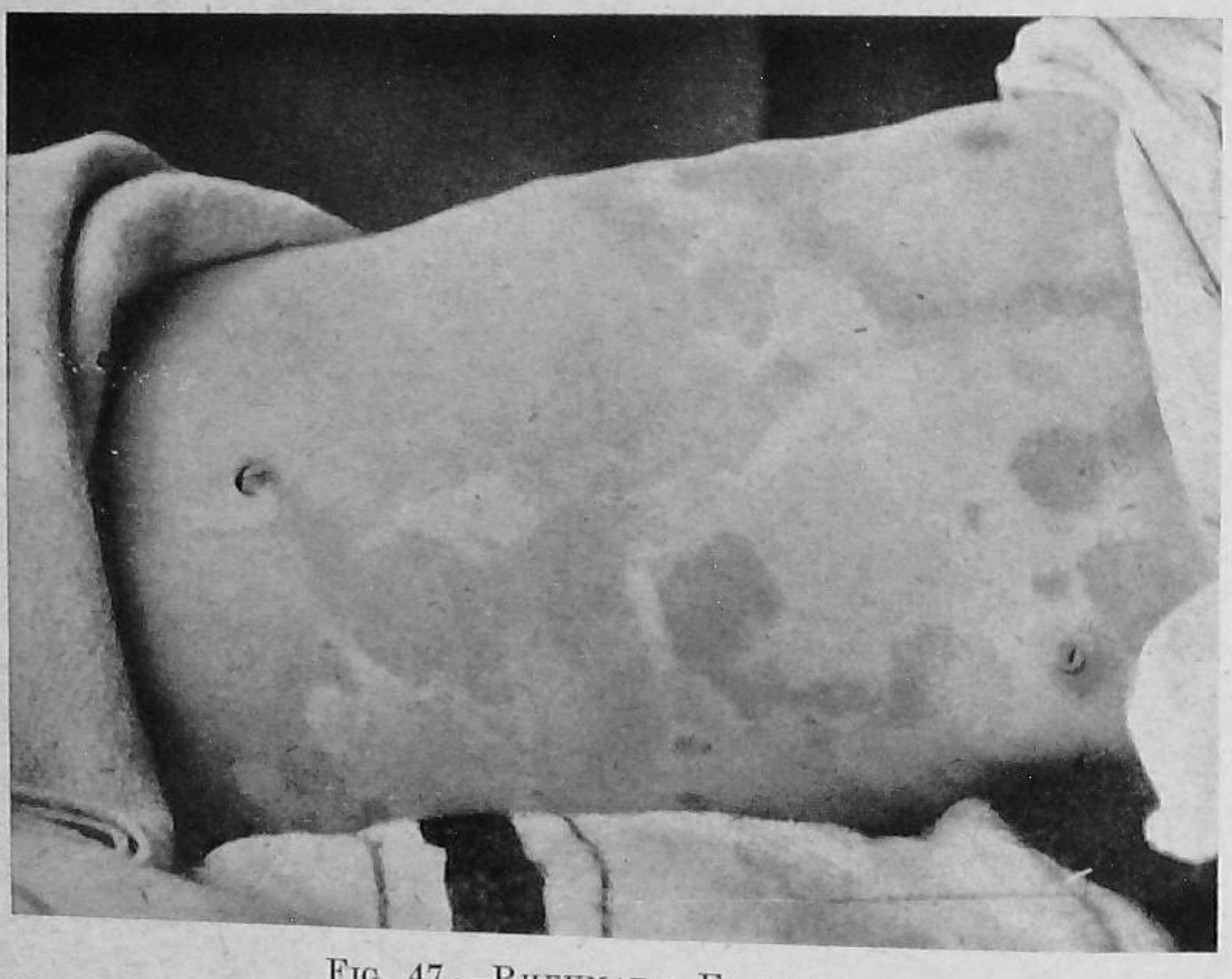


FIG. 47.—RHEUMATIC ERYTHEMA.

chorea is only one event in the series. It may be the first event, and if you follow the course of the child out you may easily find that later on he develops so-called growing pains, or joint inflammation, or endocarditis. **Pleurisy** also I do not need particularly to describe, because we shall come to it in another lecture; but it is not a common manifestation of rheumatism, and when it is rheumatic in its origin it is usually a dry pleurisy, or one without much effusion. Seeing that the pleura is a large fibrous membrane, one would expect it to be more commonly attacked by the rheumatic virus than it is. But it is not nearly so susceptible as many other fibrous membranes. The reason for that I do not know.

Tonsillitis in its relation to rheumatism is a subject about which it is very difficult to speak definitely, because I do not think one can say that there is any given type of inflammation of the tonsil of which you can assert, simply by looking at it, that it is rheumatic in origin. On the other hand, no doubt, if you go into the history of children who have suffered from other manifestations of rheumatism you will often get a report of repeated sore throats. It may be that these are simply children with unhealthy throats, that there is follicular tonsillitis, and that the rheumatic poison gets access to a throat previously diseased. In purely rheumatic cases I think you will find that the inflammation is not confined to the tonsils, but tends to spread to the soft palate, and that it particularly tends to involve the tendons of the muscles, the tensor palati, and the levator palati, and to produce pain in upward and downward movement. At all events, one expects, here as elsewhere, to get the fibrous tissues involved rather than adenoid tissue, which is, as you know, the main constituent of the tonsil.

No matter which one of those manifestations has appeared, after it has lasted for even a comparatively short time you will find that the child gets into a cachectic state—what one may call the **rheumatic cachexia.** It is recognized largely by anæmia. The rheumatic poison tends more even than most toxins to destroy the red blood-corpuscles; and if you trouble to examine the blood of a child during an attack of endocarditis or pericarditis you will find a great loss of blood-corpuscles in a short time; in a few days their number may drop by a million. One has sometimes been able to recognize the development of complications in an attack of rheumatism by the sudden pallor which the **anæmia** produces. The fresh outbreak of the rheumatic poison seems to produce a fresh destruction of blood.

Lastly, we shall consider what the **treatment of rheumatism** should be. I need hardly say you should do all you can to prevent the disease developing in those children who are predisposed to it by heredity and by their diathesis. I say that because, although rheumatism is certainly due to a micro-organism, there can be no doubt that such agents as chill have something to do with its production, probably acting by diminishing the resistance of the tissues for the time being. See, therefore, that these children are properly protected from cold, that they are suitably clothed, and that in particular they are not allowed to get wet feet and sit in school

in damp boots, which is, I believe, a very common cause of rheumatism in childhood.

Be careful of the rheumatic child also in the matter of bathing. Over and over again one has known rheumatism develop after sea-bathing, and after the injudicious use of cold baths. Cold baths are very excellent things, but you must not make a fetish of them ; and many cases of rheumatism in early life are set up by the tendency to ' harden ' the child by making him take a cold bath in the morning. See also that the throat is kept healthy and if the tonsils are much enlarged or infected they should be enucleated. The next point in the prevention of rheumatism is that you must take all the minor manifestations of the disease seriously. You have to insist upon the child going to bed at the first sign of the disease, no matter how slight and trivial it may seem to be. You may be inclined to think that a little aching in one joint or a little rheumatic stiffness about the muscles of the neck or the back is not worth paying serious attention to, but if you know the child to be predisposed to rheumatism you must insist upon his being kept in bed. If that were done regularly it is certain that a great deal of serious cardiac disease in later life would be prevented.

In regard to the treatment of rheumatism once it is actually developed, your chief care must be to protect the heart. The rheumatic pains are inconvenient and uncomfortable, and so is pyrexia, but they are not in themselves dangerous ; whereas the least degree of endocarditis or pericarditis involves a serious risk. Further, thanks to salicylates, you have the arthritis under your control and they should be given freely. Sulphonamides should not be used in the treatment of rheumatism in childhood. They seem to do more harm than good, but curiously enough it is claimed that they have a place in the prevention of relapses in a rheumatic subject. It is unfortunate that salicylates seem hardly to touch the cardiac manifestations ; in fact, some think their introduction has increased the amount of cardiac disease rather than diminished it by enabling the subjects of rheumatism to get up and go about sooner than they would have been allowed to do in the old days. When the pains are gone, you are apt to think that the disease is over, and to overlook the fact that there is a slight endocarditis going on, which, if it becomes established, ends in permanent damage in the valves. The red cell sedimentation test should help us here to keep an eye, as it were, on such smouldering activity. What one has to preach about

the treatment of rheumatism is the necessity of *rest*. I am sorry to say that of necessity in a general hospital one has to set a bad example in this matter, for we cannot keep these patients in long enough; but you have to impress upon the parents that they absolutely must allow prolonged rest so as to prevent the heart becoming permanently damaged. Nowadays several excellent 'heart homes' exist where such children can receive the prolonged and carefully supervised convalescence they require.

CHOREA

I must now say a few words about chorea—not that I regard it as an active manifestation of rheumatism, but because of its undoubted rheumatic relationship.

I need not waste time in describing full-blown chorea, as all of you must be abundantly familiar with its clinical characters, but I would only point out that you should be prepared to recognize cases in their earlier and slighter degrees. Whenever a child is said to be unusually 'fidgety' or restless suspect chorea. Such cases are often overlooked, and, indeed, the child may be punished for its troublesome behaviour, which, of course, is not only very unjust, but extremely bad treatment of the disease into the bargain. You must remember also that, although some degree of muscular weakness is present in all cases of chorea, there are a few in which the disease manifests itself almost exclusively in this way, so that a form of paralysis is suspected. These may be spoken of as cases of 'paralytic chorea,' and the weakness, as in all cases of functional loss of power, is commoner on the left side of the body than on the right. You must be careful also not to mistake mere tics or habit-spasms for chorea, but the characteristic features of these I shall deal with on another occasion (p. 291). It is sufficient to say here that, although the two diseases do undoubtedly tend to affect the same type of child, yet they have really nothing in common.

You should remember also that there is a mental as well as a physical side to chorea, and that, whilst in all cases there is more or less mental dullness during the course of the disease, in some this reaches a very severe degree, and may be attended by complete loss of speech—not a mere aphasia, but an actual dumbness, which may persist for several days. This, however, although alarming to the friends, need cause you no anxiety, as it always passes off in time.

Complications in chorea are not really very common, and when they occur are due to the occurrence of active rheumatic manifestations. Rheumatic affections of the joints in the course of the disease are, fortunately, almost unknown, for the condition of a child who had both acutely inflamed joints and at the same time uncontrollable movements of the limbs would indeed be far from enviable. The usual complication is some form of carditis, which differs in no respect from the primary rheumatic carditis described in the next lecture. This may be accompanied by the appearance of rheumatic nodules, and, in fact, in one or two cases I have known such nodules appear in chorea without there being any evidence of affection of the heart.

The prognosis in chorea is quite good. Even the severest cases, if uncomplicated, almost never die. Nor does the occurrence of rheumatic carditis affect the immediate prognosis much ; notwithstanding the supervention of severe pericarditis or endocarditis, the patient has a wonderful way of getting well, though, of course, with a damaged heart.

The average duration of the disease when uncomplicated is about five or six weeks, although one meets occasionally with troublesome chronic or relapsing cases which run on, with intermissions of more or less complete recovery, for months and sometimes even for years, but in these, too, the ultimate outlook is favourable.

Treatment.—I have long given up any very active or heroic treatment of this disease. I remember Sir Thomas Barlow saying that he would like to write a paper on 'The Diseases produced in the Treatment of Chorea,' and I am sure quite a good paper could be written on the subject. I have myself seen children poisoned and pigmented by large doses of arsenic, rendered comatose by chloretone, temporarily insane by nirvanol, and killed by acid intoxication induced by the massive use of salicylates, all in the attempt to treat a disease which gets well of itself in a few weeks. What I would advise you to do is to put the child to bed on a full diet, preferably under the control of a sensible nurse, and keep it there for six weeks. The treatment, in fact, is very much like a rest cure, and in severe cases isolation from fussy relatives is just as important as it is in hysteria.

If the movements are very violent and exhausting, of course, you may be obliged to use sedatives, of which chloral or chloretone are perhaps the best, although much can be done without drugs

by the use of tepid packs. Heart complications must be treated on the lines I have already indicated when speaking of rheumatism.

After the child is convalescent it should be sent away for a change of air, and for a long time after an attack care should be taken to avoid any occasions of excitement and any overstrain at school.

LECTURE XIX

ACUTE RHEUMATIC CARDITIS

It is my intention in this lecture to give a general description of the most serious manifestation of acute rheumatism in childhood, and that to which all the menacing character of the disease is due —namely, acute carditis.

The term 'carditis' was introduced by the late Dr. Sturges in his Lumleian Lectures in 1894 as a convenient one to cover all the different ways in which acute rheumatism may affect the heart, and it is made up of endocarditis, myocarditis, and pericarditis in varying degree. Endocarditis may, and often does, occur alone; pericarditis is usually, according to some always, accompanied by endocarditis; whilst myocarditis is probably never met with except in association with inflammation of the endo- or pericardium. In all severe cases of carditis all three structures are involved to a greater or less degree.

We have now to consider the manifestations of each of these components of carditis separately.

Endocarditis is the commonest and simplest manifestation of a rheumatic affection of the heart. Pathologically it consists, as is well known, of an inflammation of the valves, generally of the mitral, with the deposition of vegetations on the surface a short way from the margins. The clinical recognition of such an inflammation is not easy, and, indeed, it is perhaps impossible to detect endocarditis at its very outset. The indications given by different writers are very various, but on the whole the signs to look for are an increased rate with tumultuous action of the heart, reduplication of the second sound at the apex with a blurring of the first sound going on to a definite systolic murmur conducted outwards. It has been denied by some, however, that an early stage of endocarditis is capable of producing a systolic murmur, seeing that the small vegetations cannot appreciably interfere with the mechanical action of the valves, and such writers would attribute the incompetence

of the valve which the murmur indicates to a myocarditis affecting the tone of the muscle sphincter which surrounds the mitral orifice. The point is perhaps rather an academic one, inasmuch as a myocarditis in this situation is pretty sure to be accompanied by some endocarditis as well. If the heart has already been damaged by a previous attack of rheumatism, the recognition of fresh endocarditis is even more difficult. It is here, I think, that a study of the pulse-rate is of great help. If a chart is made of the pulse-rate, it will be found, if there is no fresh endocarditis, that the pulse and the temperature charts remain roughly parallel, and fall together under the influence of salicylates. If, on the other hand, fresh endocarditis has supervened, the pulse chart will remain high after the temperature has reached normal. I believe this to be true in all except the mildest and most insidious cases of endocarditis.

Myocarditis.—Sturges, in the lectures already referred to, drew special attention to the affection of the myocardium in carditis, which had up till that time been overlooked. It has since been the subject of study in this country by the late Dr. Poynton, and the late Dr. Carey Coombes. These investigators demonstrated the presence in the myocardium from cases of acute carditis of inflammatory foci of characteristic histological appearance comparable in many respects to rheumatic nodules. Their observations leave no room for doubt that an actual inflammation of the myocardium occurs. Apart from inflammation, however, there is another element in the affection of the muscle of the heart in carditis which is of equal and perhaps greater importance, and that is the toxic. It would seem that the organism of acute rheumatism produces a toxin which affects the heart in much the same way as the diphtheria toxin does, and some writers regard this poisoning of the muscle as even more important and more disabling than its inflammation. The term 'myocarditis,' then, must be taken to include the results not only of inflammation, but of toxic action upon the heart muscle, but which of these predominates in any given case it is impossible to say at the bedside.

The **physical signs** of myocarditis are as indeterminate as those of endocarditis, and, as we have seen, there are some who would attribute an apical systolic murmur when it occurs in carditis to involvement of the muscle rather than of the mitral valve. Apart from this, it seems reasonable to suppose that the greater the enlargement of the heart and the increase in the rate the more is

the myocardium involved. The results of percussion, therefore, or determination of position of the apex beat along with a study of the pulse chart must be our chief guides in diagnosis.

Pericarditis is the most dramatic manifestation of carditis, and usually appears later in the case than endocarditis. It is probable, as has already been pointed out, that it never occurs alone. The chief physical sign of pericarditis is, of course, the well-known to-and-fro rub heard at some point over the precordium. It is a very evanescent and elusive physical sign—'here to-day and gone to-morrow'—and may easily be missed. Rapid enlargement of the area of heart dullness usually accompanies pericarditis, but is not due—as is often supposed—to effusion into the sac, but to hypertrophy and dilatation of the heart. It is probable, as has been mentioned already, that it is really more the result of a coexisting myocarditis than of the pericarditis itself. An interesting phenomenon met with in many, but not in all, cases of rheumatic pericarditis is what is known as Bamberger's sign. It consists in an area of dullness about the size of a crown-piece appearing first near the angle of the left scapula, and extending thence gradually over the whole left lower lobe. The breath sounds over it are intensely tubular. It is commonly stated that these signs indicate a consolidation of the lower lobe of the left lung, the result of pressure upon it by the dilated heart. I am bound to say that this seems to me an unlikely explanation. If it were true, one would expect to meet with the sign in many cases of heart disease as well as in pericarditis—in the enormously hypertrophied heart of aortic incompetence, for example. Yet I have never myself met with it except in pericarditis. It seems more likely that there is a direct extension of inflammation from the pericarditis to the left lung, and that the consolidation is due to a pneumonic process rather than to mere compression. Post-mortem observations made by Dr. Salaman when he was pathologist to the London Hospital some years ago seemed to bear out this view, and he described the histological appearances of the consolidated lung in these cases as those of a 'pneumonic subacute consolidation.' I have also seen a typical croupous pneumonia of the left lower lobe complicate rheumatic pericarditis, and end by crisis in the usual way. Some degree of serous pleural effusion is also occasionally met with.

Whatever the true explanation of these lung complications of pericarditis—and it would not be right to dogmatize about them—

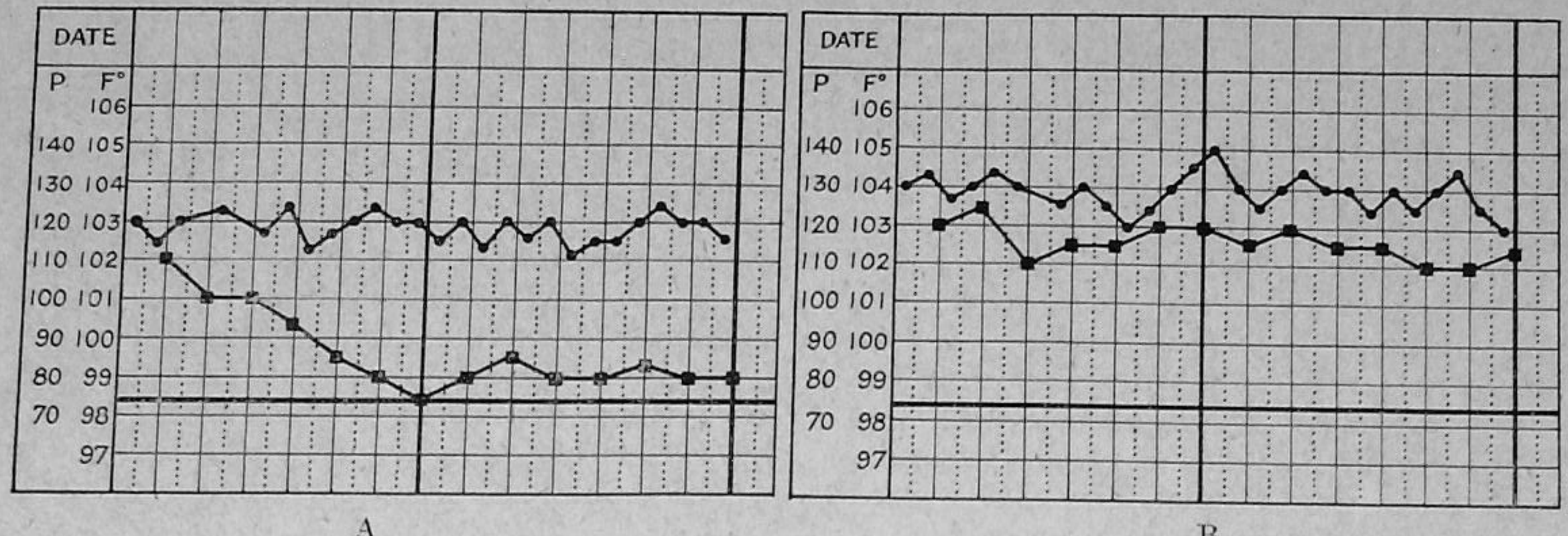
they are of great clinical interest and importance, and indicate always a severe though not necessarily fatal degree of carditis.

Apart from physical signs in the heart, the occurrence of carditis is often attended by general symptoms such as restlessness, anxiety, and delirium. The sudden appearance of pallor and anæmia is also very suggestive, especially of pericarditis. It was shown by Sir Archibald Garrod a good many years ago that acute rheumatism is a great blood destroyer, and that under its influence the red corpuscles may fall by as much as one million per cubic millimetre in a day. Such rapid destruction of blood seems specially to occur in those cases in which there is active carditis, and one can sometimes tell that carditis has supervened in a child suffering from acute rheumatism by noticing that the patient has suddenly become anæmic.

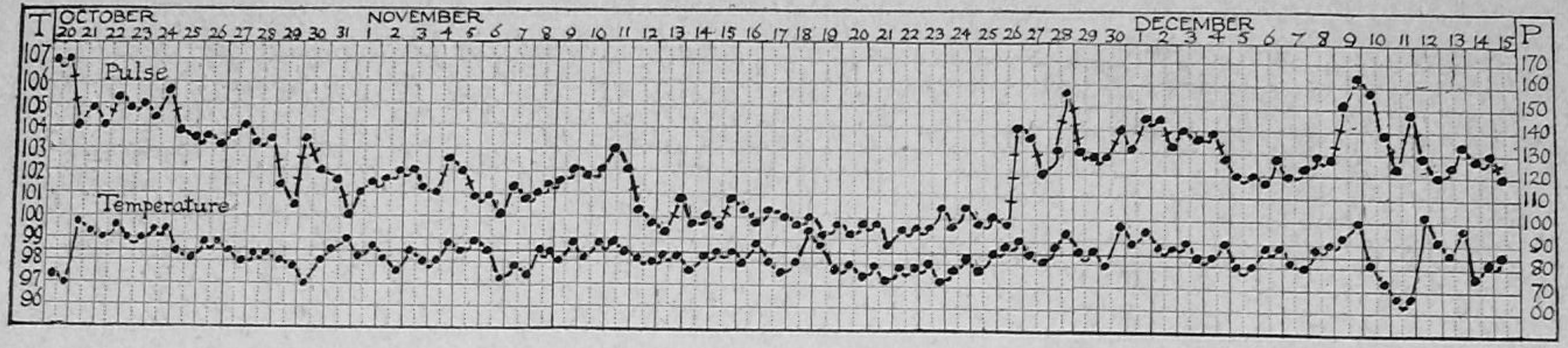
Another change in the blood is an increase in the red cell sedimentation rate—a definite indication of active carditis in a rheumatic child.

Rheumatic nodules have long been recognized as accompanying carditis. It is true, of course, that many patients with carditis never have nodules, but it is extremely rare to find nodules without carditis, and so long as fresh crops of nodules continue to appear the carditis must be regarded as active. There is an impression in some quarters that the appearance of nodules is of very grave omen. This is probably the result of a misinterpretation of a statement of the late Dr. Cheadle, who said that the appearance of *massive* nodules (see fig. 44) was 'the equivalent of a sentence of death.' The ordinary small nodules, however, have not this serious significance.

In following the course of a case of acute carditis the pulse chart is again our best guide. To Dr. Poynton belongs the credit of pointing out that rheumatic carditis is an afebrile condition, and that the temperature chart may be normal throughout its course. On the other hand so long as the inflammation is active the pulse-rate is accelerated, and only falls very gradually as the disease subsides, any recrudescence being at once shown by a fresh rise of the pulse. These ups and downs of the disease can be followed much more easily on the pulse chart than in any other way, and since taking to charting the pulse in all these cases I find that one has a much better idea of how the disease is going on (fig. 48). Most helpful of all is a chart of the sleeping pulse for the pulse-



A B



C

FIG. 48.—PULSE CHARTS IN ACUTE CARDITIS.

A. Showing sleeping pulse in nervous tachycardia—fast by day, slower by night.

B. Showing sleeping pulse chart as rapid as day-time pulse in patient with active carditis.

●—●—● = day pulse. ■—■—■ = night pulse.

C. Showing fall in pulse-rate followed by a rise due to a relapse. Note the absence of a rise of temperature.

rate during sleep is not affected by nervous influence as the day pulse is.

Treatment

It must be admitted that the treatment of acute carditis is very disappointing. Salicylates, which so effectively control the temperature and joint manifestations of acute rheumatism, seem to exert little or no influence on the heart inflammation. None the less, it is as well to administer them in order to keep the rheumatic process as a whole in check. For the restlessness and anxiety, especially of pericarditis, small repeated doses of opium are certainly useful. In order to limit dilatation of the heart and to control the pulse-rate digitalis has often been employed but opinions as to its usefulness are very diverse. My own experience has been that its effects are very variable. In some cases it slows the heart, more often it seems to be without effect especially whilst the inflammation is still active. It therefore finds its chief use during convalescence.

In cases of pericarditis various external applications to the heart have been tried. The late Dr. Lees was a great advocate of the ice-bag, but although it may quieten a tumultuously acting heart it is difficult to believe that it has any other effect. Nor does blistering appear to be of much value except perhaps in those cases, and they are rare, in which precordial pain is pronounced. Leeching also has its supporters, but it must be remembered that children stand loss of blood badly, and particularly must this be the case if anæmia is present, as we have seen that it usually is. Blood-letting should therefore be reserved for those cases in which sudden engorgement of the right side of the heart comes on with great dyspnœa. In these circumstances it may be of capital value. What we want, of course, is some form of specific treatment which will attack the microbe of acute rheumatism or its toxin directly, but so far no such treatment is forthcoming. It seems clear that the sulphonamide drugs do definite harm despite the fact that the hæmolytic streptococcus is often found in the sore throat that precedes a relapse of carditis.

When all is said and done, prolonged rest is the best means we have at our command for controlling acute carditis. Here, again, the pulse chart is of great value in enabling us to judge how long rest should be continued. So long as the pulse curve is slowly

falling, so long should strict rest in the recumbent position be enforced. If the pulse appears to have touched bottom and has remained stationary for a few days, one may begin cautiously to relax the strictness of the regime of rest, but always being prepared to reinforce it if the pulse-rate is unfavourably affected. It is astonishing how unstable the heart is in many of these cases. Even the introduction of an extra pillow or the slightest excitement or emotional disturbance may produce a notable and not evanescent acceleration of rate.

The treatment of carditis, then, offers few opportunities for very active intervention ; rest and patience are what are chiefly required from patient and doctor, and too active therapeutics may do more harm than good. In this connection it is well to recall the story, told by Dr. Sturges of a practitioner who was expressing his regret to a physician that he had not recognized the presence of pericarditis in a case of rheumatism that they were seeing together. 'Don't apologise,' said the physician ; 'if you had recognized it you might have treated it !'

LECTURE XX

SOME DISORDERS OF THE HEART IN CHILDHOOD

In the present lecture I propose to consider with you some of the commoner disorders of the heart met with in childhood, apart from those acute rheumatic affections which were dealt with on a previous occasion.

At the outset it may be well to remind you again of some of the **anatomical** and **physiological peculiarities of the heart** in children to which I referred in an earlier lecture (p. 8). You will remember that the heart lies naturally at a higher level in early life than it does in the adult, the apex-beat being often in the fourth space instead of the fifth. The apex-beat is also farther out than it is in the adult, being beyond the mammary line up to the third year, and in it from the third till about the tenth year, after which it assumes the adult position.

You will remember also that **irregularity** of the heart is very common in early life, especially during sleep, and has not necessarily any pathological significance. It is of the nature of what is known as 'sinus arrhythmia,' and may be distinguished from the irregularity of organic disease by the fact that it disappears on exertion. The greater rapidity of action of the heart in early life should also be borne in mind, the pulse-rate being—

At birth, about 130.
In the second year, about 110.
In the third year, about 100.
In the eighth year, about 90.
In the twelfth year, about 80.

It is slightly more rapid in girls than it is in boys.

As regards the **heart sounds** in childhood, I would remind you again of the relatively greater intensity of the pulmonary than of the aortic second sound up till about the fourth year.

In early life, too, so-called **cardio-pulmonary murmurs** are

not uncommon, and are apt to lead to confusion. They may be distinguished from the systolic murmurs of organic disease by the fact that they are much more variable, being audible at one time and not at another, or heard only in one position of the body, or at all events varying greatly in loudness according as the patient is erect or recumbent. Frequently, too, they can only be heard during one phase of respiration, and tend to disappear when the breath is held in expiration.

Displacement of the heart is not uncommon in childhood. It may be due to (1) pleuritic effusion (usually left-sided) pushing it over; (2) to fibrosis of the lung pulling it over; (3) to atelectasis which causes displacement towards the affected side; (4) to transposition of the viscera; true dextrocardia, without displacement of the viscera, also occurs, but is very rare. You will rarely have any difficulty in determining the cause of the displacement in any case if you examine the patient carefully, but slight degrees of fibrosis of the lung causing displacement are apt to be overlooked. X-ray examination is of great help in determining the cause in a doubtful case.

I do not intend to say much about what is really the commonest form of heart-trouble in childhood—namely, **chronic valvular disease,** for the reason that it is in all essential respects the same as valvular disease in the adult, and calls for the same management. I would, however, give you one emphatic piece of advice, and that is, *Don't treat murmurs!* By this I mean that you are not to suppose that treatment is called for merely because you have detected a heart murmur on routine examination. So long as the lesion is fully compensated—i.e. is producing no symptoms—it may safely be let alone. This simple rule, however, is constantly disregarded, and I often have children sent up to hospital to be treated 'for heart disease' because a school doctor has found a murmur in the course of his official inspection. Great harm may be done by such excess of zeal, for not only are the parents unduly frightened, but the child is apt to be condemned to a semi-invalid life quite unnecessarily, and greatly to the impairment of his happiness and often of his education. In such a case all that is necessary is to warn the parents of the danger of fresh attacks of rheumatism, and to advise them to send the child to bed and call in medical advice as soon as even the slightest pain in the joints or limbs is complained of. At the same time, they should be told

that the heart has been 'damaged,' but that this need not necessarily interfere with the child's health or prospects in life, although it will be wise to see that any over-exertion is avoided, and that in deciding upon the patient's future career strenuous occupations and those which entail much exposure are avoided.

As regards prognosis in valvular disease it is well to remember that, other things being equal, it is worse the younger the child. Puberty is the period to be dreaded, for it makes great demands upon the heart, and if the lesion is a severe one there may not be sufficient reserve to meet these. Hence the outlook is better if the damage is not inflicted until the period of puberty is passed.

CONGENITAL AFFECTIONS OF THE HEART

Congenital heart disease is the result in most—perhaps in all—cases of a malformation which originates early in intra-uterine life, but it may possibly, in a few instances, be the consequence of a fœtal endocarditis. Into these vexed pathological questions I do not propose to enter further, for the mode of origin of the lesion is really of very little clinical importance except for this, that if fœtal endocarditis *should* be the cause in any case, there is a certain risk of fresh endocarditis being grafted on the top of it during childhood. The bare possibility of this happening should be an inducement to one to guard children with congenital heart disease from infections of all sorts—rheumatic and other—as carefully as possible. Indeed, this is a wise rule whatever view one holds as to the mode of origin of the congenital defect.

A practical question which often arises at the bedside is this: Is the murmur which is present due to **a congenital or an acquired lesion ?** To this question it is often difficult—sometimes impossible—to give a satisfactory reply. In deciding it the following points have to be considered :

1. *The history.*—A clear history of previous acute rheumatism or chorea is, of course, strongly in favour of an acquired lesion; even a history of sore throats or 'growing pains' should make one suspicious.

2. *The age of the patient.*—The younger the child the more likely is it that one is dealing with a congenital affection. Rheumatic endocarditis is almost unknown below the age of two, and is rare below five.

3. *Site and character of the murmur.*—Congenital murmurs are usually best heard *internal* to the apex or in the pulmonary area. Acquired murmurs are usually loudest at the apex-beat, and are conducted out from it. Diastolic and presystolic murmurs, wherever heard, are far oftener acquired than congenital. A very loud murmur with no subjective symptoms is usually congenital.

4. *Enlargement of the left side of the heart* is in favour of acquired, of the right side of congenital, disease.

5. Marked *cyanosis* and the presence of *clubbing* point to a congenital lesion (fig. 49).

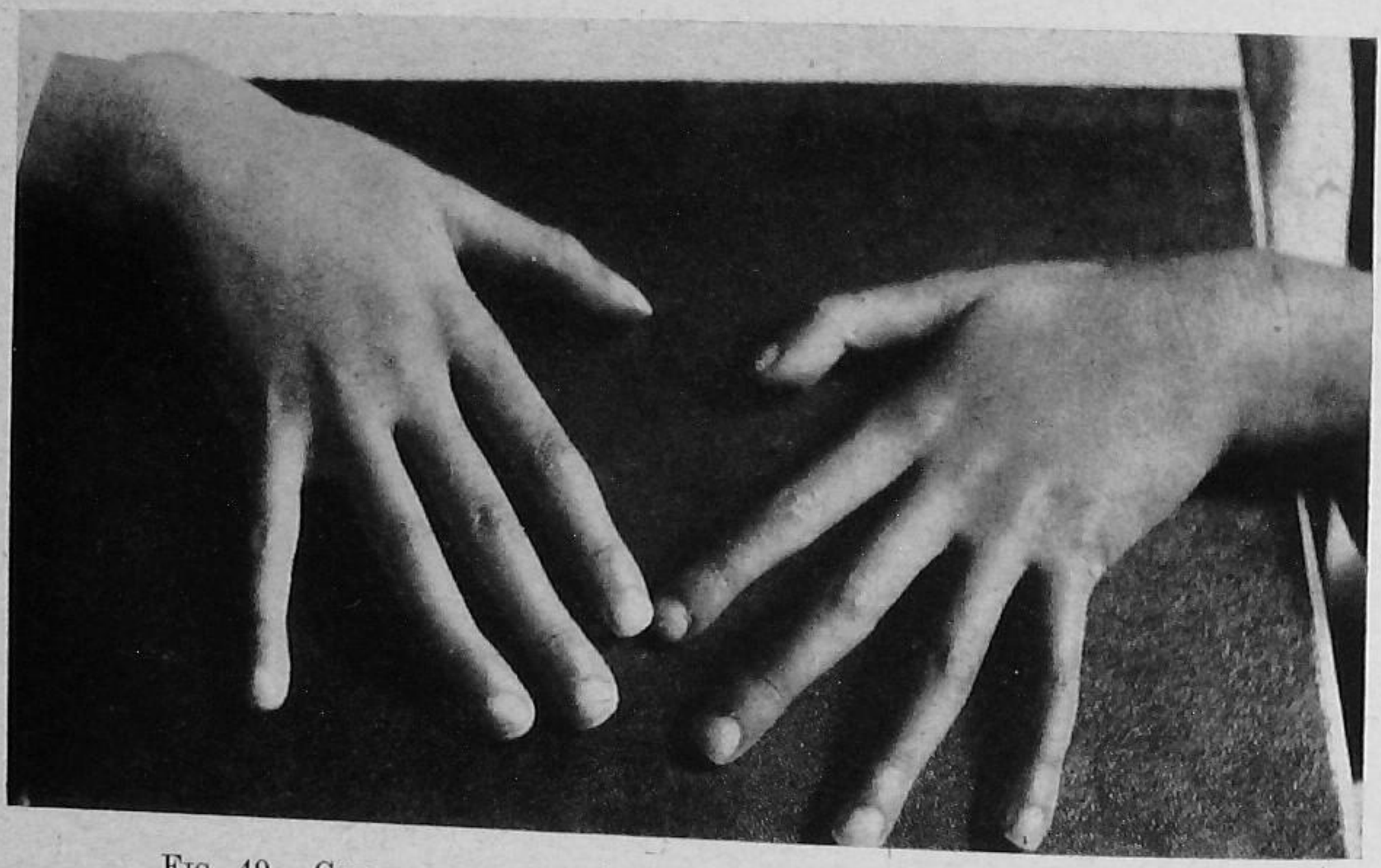


FIG. 49.—CLUBBING OF FINGERS IN CONGENITAL HEART DISEASE.

6. The *coexistence of other malformations*—e.g. harelip, cleft-palate, etc., is to some extent in favour of the lesion being congenital.

Even when due consideration has been given to all these points, you will sometimes be in doubt. This is specially apt to happen in the case of a child of about five years of age whose heart is being examined for the first time and whose past history is indefinite. Hæmic and cardio-pulmonary murmurs are also a fertile source of error. X-ray examination sometimes helps, for certain varieties of congenital heart disease show a characteristic shadow (fig. 50).

Supposing that you have decided that the lesion is congenital, the next point to determine if you can, is its exact nature. You may find it useful to remember that there are three broad groups according to the presence or otherwise of cyanosis. The first of these consists of children with congenital heart lesions who are never cyanosed. For example there may be a complete *dextrocardia* sometimes associated with transposition of other viscera, or there may be some abnormality of the valves, and in this group also must

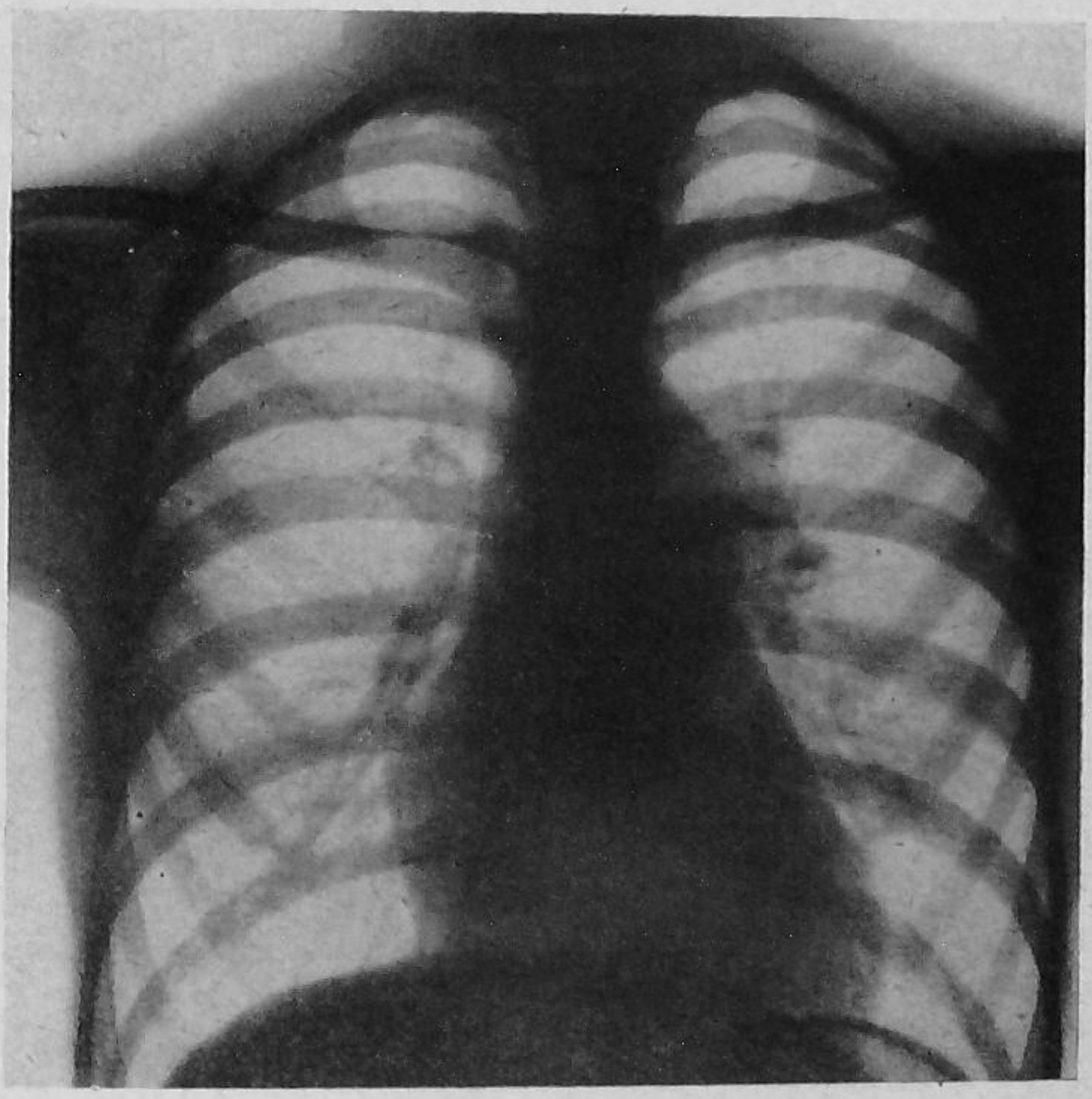
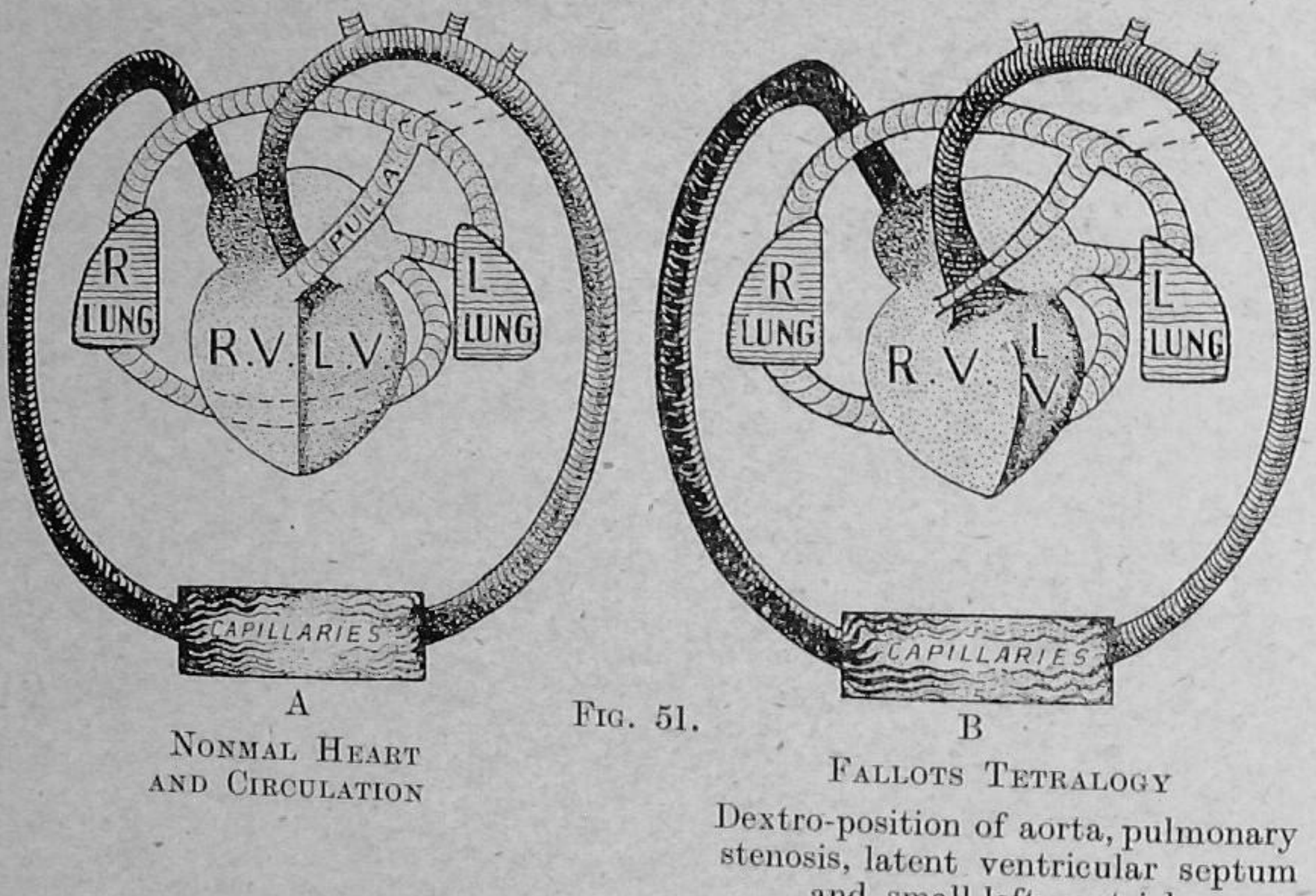


FIG. 50.—X-RAY OF HEART IN CASE OF PATENT DUCTUS ARTERIOSUS.
Characteristic bulge in upper left part of cardiac outline.

be put *coarctation of the aorta* which, however, is seldom diagnosed in childhood. The second group consists of those children who although normally not changed in colour, tend to develop cyanosis on extreme exertion or at a late stage when the heart is failing. In this group we can place *patent ventricular septum* with a systolic murmur over the body of the heart. Also in this group is *patent ductus arteriosus* with a long murmur like a 'humming top' beginning with systole and running right through into the diastole, best heard

in the second left interspace close to the sternum. In early life the murmur is often purely systolic. The third group are always cyanosed and for the most part have variations to a greater or less degree of what is termed *Fallot's tetralogy*. I have here (fig. 51) two simple diagrams to try and explain this condition. You will see that there has been a faulty division of the main arterial trunk so that the systemic aorta sits astride of the septum, so to speak, and receives blood from both ventricles. There is usually a loud systolic murmur, generally with a thrill, with maximum intensity in the pulmonary area. The murmur is often to be heard all over the chest, back and front. Clinically, it may be possible to detect that the right ventricle is considerably enlarged, a point which can be confirmed by X-ray



A

NONMAL HEART AND CIRCULATION

FIG. 51.

B

FALLOTS TETRALOGY

Dextro-position of aorta, pulmonary stenosis, latent ventricular septum and small left ventricle.

examination. Certain variations of the full-blown malformation, so to speak, not infrequently occur, and, in fact, the diagnosis of the exact lesion in a case of congenital heart disease is often a matter of guess-work. The first step is to decide whether there is a congenital malformation or not, and it is obvious that the greater the disturbance in normal circulation, the more serious the condition is going to be.

One other point in diagnosis must be mentioned. You must not suppose that all cyanosed babies suffer from congenital heart disease. Every now and then you will meet with a case—usually within the first few weeks of life—in which the child is noticed to 'go black' at times, especially about the lips and in the hands and feet.

This is particularly apt to happen after the bath or on exposure to cold, and is commonest in premature infants. It is, I think, a **vaso-motor disorder**, analogous to Raynaud's disease, and tends to pass off after a few months as the child gets stronger.

Having diagnosed the existence of a congenital lesion, the next point you have to consider is the **prognosis**, and you are sure to be asked: Can the condition disappear? Is it serious? What are the child's chances of life?

As regards the first of these questions—the **possibility of recovery** from the lesion—you are justified in the vast majority of cases in replying in the negative. Notwithstanding this, however, I have met one or two cases in which a congenital systolic murmur, not accompanied by cyanosis, finally disappeared. Presumably these were examples of a slight defect of the septum, which gradually closed, but you can rarely count on this happening. Your reply to the second question will depend upon the degree of cyanosis present: roughly speaking, the more marked the cyanosis the more serious the lesion.

As regards the chance of life in congenital heart disease, you may say that the majority of all patients with symptoms—cyanosed or not—die before the fifth year, and that, of the cyanosed ones especially, few reach adult life. They die either of inter-current disease—especially pulmonary affections, which they stand badly—of hæmoptysis, or of more or less sudden syncope (often attended by convulsions). In a considerable number, too, the fatal result is brought about by the supervention of an infective endocarditis as the consequence of some infection such as tonsillitis. Should they survive, they are apt to be stunted in development, and, in cases of pulmonary stenosis especially, pulmonary tuberculosis is prone to develop in adult life. Much, of course, will depend upon the amount of care which can be taken of the child, but even in the most favourable circumstances survival to middle life is exceptional. Some lesions, however, such as aortic stenosis, uncomplicated patent ductus, and patent septum, offer a much better chance than others.

Congenital heart disease offers little scope for **treatment**. The lesion is in its nature incurable, and all you can do is to try to mitigate its effects. The surgical treatment of a patent ductus arteriosus is an interesting exception to this—but it is still of limited application. The patient should lead a regulated life, all over-exertion, physical and mental, being avoided. You will judge of what it is

safe to allow the child to do by its effect on the cyanosis ; anything which increases the latter is harmful. Cold also is injurious in these cases, and all exposure to infections should be guarded against with the utmost care. The diet should be so arranged as to lessen as far as possible the occurrence of flatulence, which is apt to be present in these cases, and adds materially to the embarrassment of the heart. Drugs are of little help, and the ordinary heart tonics, such as digitalis, of none at all. For the attacks of palpitation, collapse, and cardiac distress which are apt to occur from time to time, the occasional use of a stimulating carminative mixture—e.g. ammonia and ether—is of service, and when the right ventricle becomes seriously overdistended, cautious blood-letting by means of leeches may be of temporary utility.

FUNCTIONAL DISORDERS

Considering the highly sensitive condition of the nervous system in children, it is perhaps surprising that they are not more subject to functional disorders of the heart of nervous origin than experience shows to be the case. Functional **palpitation,** however, is not uncommon in children of school age, and is usually due to flatulent indigestion. It resembles in its characters the same condition as seen in the adult, and its treatment consists in attention to the digestion and the administration of bromides and carminatives.

Arrhythmia, as I pointed out when speaking of the physiological peculiarities of the heart in childhood, occurs very readily in early life, and is of no significance. It has the characters of a **sinus arrhythmia**—i.e. the variation is in rate, but not in force, and often follows the phases of respiration. One also meets not infrequently with cases of irregularity due to the occurrence of so-called '**extra**' **systoles,** and in this sort of case attacks of palpitation are prone to appear at intervals as well. Flatulence is no doubt at the root of both. It is characteristic of these—and indeed, of all functional arrhythmias—that the heart tends to become steadier on exertion—indeed, it often becomes quite regular —and whenever you find this happen, you need have no real anxiety about the condition. The treatment of this form of irregularity is the same as that of palpitation.

Cases of extreme irregularity of the heart occasionally turn up in which it is very difficult to say whether one is dealing with a

functional or an organic condition. I remember, for instance, a little girl, six years of age, being brought to me for attacks of pallor and palpitation which were said to come on upon exertion. I found that she was slightly anæmic, and had an extremely irregular heart, but without any sign of enlargement and showing no murmur. Treatment by bromides had little effect, but as soon as she was put on digitalis, her heart became perfectly regular.

From the character of the irregularity in this case and the promptitude with which it subsided under digitalis, I think there can be no doubt that one was dealing with a case of **auricular fibrillation**; but if so it seemed to be of nervous origin, for there was no sign of any organic disease in the heart, and no history of rheumatism nor overstrain, nor of anything which could have damaged the myocardium. I believe, however, that the heart experts are not yet quite sure whether auricular fibrillation is ever of purely functional nature. Be that as it may, there can be no doubt that you will occasionally meet with cases of arrhythmia in childhood in which there is no sign of organic disease, and which yet respond in the most dramatic manner to the administration of digitalis.

SUSPECTED HEARTS [1]

You will meet occasionally in practice with a type of heart which will cause you a good deal of uncertainty and anxiety both in diagnosis and prognosis. I refer to cases in which a child suffers from symptoms which raise a suspicion of cardiac disease, but in which the physical signs are equivocal. These we may speak of for convenience as 'suspected hearts,' and this is the sort of case I mean. A child of school age (for these cases are not met with in infancy) complains of undue fatigue on exertion, perhaps with slight breathlessness, and possibly also of some discomfort or actual pain about the precordia. It is noticed that he looks pale or 'grey' at times, and occasionally may seem to be actually going to faint. At the same time he is languid, depressed, uninterested in work or play, and his weight tends to go down.

In the course of the routine physical examination of such a patient it is found that the apex-beat is a little farther out than it should be, and that the first sound in the mitral area is unusually short, and may show some degree of impurity, or even the presence of a

[1] These may also be spoken of as 'asthenic hearts' (Sutherland).

short systolic murmur, whilst the pulmonary second sound tends to be accentuated. The pulse also is rather rapid, and abnormally responsive to effort or to change of position. The artery wall also may be more easily felt than it should be at this age.

Now, what does this mean? Is there, as the parents will ask, any disease of the heart, and is it serious?

It is really rather difficult to be sure of the exact state of things in such a case as I have described, but I think the probability is that one is dealing with some loss of tone in the heart-wall which has resulted in slight dilatation of the left ventricle. Some children appear to have by nature a heart which is deficient in tonicity, and if any strain is thrown upon it—either in consequence of the demands made upon the organ during rapid growth, or as the result of over-exertion in games—it yields. Or, on the other hand, the tonicity may have been normal to start with, but it becomes impaired as the result of poisoning of the myocardium by the toxins of certain acute specific fevers—influenza and pneumonia appear to be the commonest—or the heart muscle is damaged by high fever, with the result in either case that slight dilatation takes place.

Such cases, of course, are not in any real sense serious in that there is no immediate danger to life, and with care the heart may recover completely, but, on the other hand, the health and activity of the child may be considerably interfered with for some time, and the heart may even be left permanently in a state of impaired efficiency and subject to more acute dilatation on moderate provocation. They will, therefore, cause a good deal of anxiety.

In the management of these cases it is difficult to do enough without doing too much. One wants to give the heart every chance of recovering itself without at the same time interfering more than is absolutely necessary with the child's education; and in the case of a boy at a public school, particularly, the reconcilement of these conflicting objects is often a difficult task. In the most severe cases, and where the damage to the heart is of recent origin, a spell of bed is no doubt the best plan, but how long the patient must remain there you can only determine by experiment, noting carefully the effect upon the heart and pulse of the resumption of the vertical position and of any degree of activity.

In the milder and more chronic cases it will be sufficient to restrict the amount of exertion undertaken to walking on the level, and perhaps to the quieter forms of games, and to insist

upon the patient lying down for an hour or two in the middle of each day, whilst in the slightest cases of all the latter rule may safely be relaxed. It will readily be understood, however, that even such slight restrictions as these may make it impossible for a boy to remain at a boarding school.

As regards the rest of the treatment I believe ample hours of sleep are very important in such cases—a point which is not always attended to at schools. Drugs are of secondary importance, but tonics such as iron and strychnine are useful, and, in the severer cases at least, digitalis may be of service.

Complete recovery may be a slow process, requiring months, or even years, of care to bring about, but throughout the whole period of treatment you must keep before yourself the object of throwing no undue strain upon the heart, whilst avoiding so far as possible making the patient an invalid.

PERICARDIAL EFFUSION

Acute rheumatic pericarditis is rarely attended by much effusion, and the extension of the area of heart dullness which so often goes along with it is not mainly due, as is often supposed, to effusion, but to dilatation of the heart itself. The real effusions into the pericardium in childhood are nearly always **purulent,** and the great majority are met with in children of less than four years—an age at which, as I have already told you, acute rheumatism is almost unknown. The pneumococcus is the commonest exciting cause of the effusion, and the pericarditis is usually only an incident in a general pneumococcal septicæmia which has followed upon an attack of pneumonia; an empyema or pneumococcal peritonitis commonly occurs along with the pericarditis. Very rarely has one to deal with a **primary pyo-pericardium** in which there is no suppuration elsewhere. The following is an example of such a case:

A female child, eight months old, was admitted to hospital with a history of having been taken acutely ill ten days previously with vomiting and general distress. She was a well-nourished baby, but looked ill, with laboured and rapid respiration, a very rapid pulse, and slight cyanosis. The temperature was normal. On examination, the area of superficial heart dullness was found to extend up as high as the second left costal cartilage, and outwards beyond the mammary line. *Resistance on percussion over the heart was markedly increased.* The sounds were very feebly heard, and there was no friction sound. The lungs

were clear. On examination of the abdomen, the left lobe of the liver was found to be pushed down to a considerable extent.

A diagnosis of pericardial effusion was made, and a needle, inserted just outside the apex-beat, drew off pus. Under an anæsthetic the pericardium was incised close to the left edge of the sternum, and 12 ounces of pus evacuated, which contained numerous Gram-positive lanceolate diplococci.

The patient was greatly relieved at first, and the wound discharged freely, but on the fourth day the temperature rose, and the child suddenly collapsed without apparent reason. At the autopsy some masses of purulent lymph were found in the pericardium, but all the other organs were healthy.

The case I have just quoted illustrates very well the chief signs by which you will recognize the existence of pericardial effusion—viz., extension of the area of heart dullness in every direction, but especially upwards, with increased resistance on percussion; feeble or absent heart sounds and displacement downwards of the left lobe of the liver. If there be—as unfortunately there often is—some consolidation of the left lung or a left-sided empyema, the diagnosis is much more difficult, and may, indeed, be almost impossible. In such a case the use of the X-rays may help. Except for purposes of prognosis, however, the establishment of the diagnosis is of doubtful advantage to your patient except in those very rare cases, such as the one described, where the pyo-pericardium is the sole lesion, and where, if the diagnosis is made in time, there is at least a chance of saving the child by operation. In those unfortunately far more numerous instances in which there are foci of suppuration elsewhere, and in which there has been a general pneumococcal infection, or in which there is an empyéma or extensive pneumonia, you will rarely be able to do much to help the patient. It is too early yet to know whether or not the sulphonamides will be of value for such patients.

LECTURE XXI

THE RESPIRATORY DISEASES OF CHILDREN

In this lecture we come to the consideration of the respiratory diseases of childhood. Of course, I need hardly remind you that this is a large subject, and so in dealing with it it will be well for us to concentrate our attention chiefly upon those diseases of the respiratory system which are commonest in early life, or peculiar to it, and, for the rest, to glance simply at those points in which the respiratory diseases of children differ from the same affections as exhibited in grown-up persons. And in taking this course I must run the risk of sometimes appearing to be rather fragmentary.

I must remind you, in the first place, of certain **peculiarities of the respiratory organs** in children to which I directed your attention in the first lecture. You remember the peculiar shape of the infant's chest, how it is circular instead of being oval, as it is in the adult, one result of which is that the child's lungs can only expand easily in a vertical direction, and not nearly so much from side to side as do the lungs of adults. It is, perhaps, in consequence of this that collapse of the lower lobes of the lungs in children is apt to be rather common. I would remind you also of certain changes in shape which the chest may undergo in consequence of disease. These changes are rendered possible by the fact that the wall of the child's chest is soft and its cartilages yielding, the result of which is that they get driven in easily under ordinary atmospheric pressure when there are abnormal conditions present in the lungs. I would remind you of the rickety-shaped chest. You will remember that a falling in takes place at each side along the line of attachment of the ribs to the cartilages; and I would again direct your attention to the pigeon chest, which you must distinguish from the rickety chest, and in which the chest wall comes to a peak or keel, as it were, in front. That condition is commonest in diseases in which there has been a great strain thrown upon the chest for a long time, and in which there has been some consolidation of the lower lobes

of the lungs, particularly, perhaps, after whooping-cough. There is a third type of chest which you should be familiar with, and that is the adenoid chest, which occurs in older children. Its chief peculiarity is a sinking in the xiphisternum and lower costal cartilages (see fig. 52). Then there are certain peculiarities of the physiology of respiration in children. There is the great ease with which respiration in children becomes irregular, and in which the rhythm becomes altered on the advent of any acute disease, with the appearance of the inverted rhythm which I described to you before. I would remind you also that the breath-sounds are relatively harsh, that puerile breathing is the normal, and that on account of the great conductivity of the child's chest you may get accompaniments produced on the one side heard on the other side, and in consequence of which the breath-sounds are apt to penetrate to the surface of the chest, even although there may be a layer of fluid of some thickness between the lung and the end of the stethoscope. All these points should be borne in mind when you are studying respiratory disease in young children.

Fig. 52.—Funnel-shaped Depression of Sternum and Adenoid Facies.

DISEASES OF THE LARYNX

We will now look at the respiratory diseases in detail, and I shall begin with the diseases of the larynx, because in young children they are very important and often very fatal. You will meet in practice with a group of cases, usually in children between the ages of one and five, in which the most striking symptom is **stridulous**

or 'croupy' breathing. These cases are much commoner than you might suppose from your observation here, for but few of them find their way to hospitals. You will find, too, that most of these cases are spoken of by parents as 'croup.' That is a very bad term, and one which I would advise you to avoid the use of as far as you can. Few terms in medical nomenclature have been the cause of greater confusion in teaching and reading than this word 'croup,' for it has been used without discrimination by different writers to designate totally different diseases. What I wish you to grasp quite clearly is that croupy breathing means simply laryngeal obstruction, and that such obstruction may be due to one of two distinct pathological processes:

1. To mere catarrh, with or without transient exacerbations of the obstruction from muscular spasm.

2. To membranous exudation in the larynx, which is practically always diphtheritic.

We shall devote our attention chiefly to cases of the first group, for you have opportunity of becoming familiar with diphtheria elsewhere.

One can distinguish two varieties in the **catarrhal cases**, which, however, are by no means sharply marked off from each other. These are (1) mild cases of laryngeal catarrh, in which there occur temporary exacerbations of the obstruction by muscular spasms; (2) more severe cases, in which the swelling of the laryngeal mucous membrane is sufficient to oppose a more or less permanent obstacle to the entrance of air.

It will be convenient to have separate names for those groups, and so we may speak of the former as cases of *catarrhal laryngeal spasm*, and reserve for the more severe cases the term *catarrhal laryngitis*.

The history of a case of **catarrhal laryngeal spasm** is usually this: A child of about three years old has a slight cold, and, perhaps, a rather metallic cough. He goes to bed, however, as usual, without much being thought of it. He falls asleep, but an hour or two later wakes up in an attack of dyspnœa, accompanied by crowing inspiration, and in a state of considerable fright. These symptoms last for from a half to three hours, when they subside almost as quickly as they began, and the child falls asleep again. Next day there is probably still some metallic cough, and the croupy attacks are apt to recur for two or three nights. Such a case is probably

never fatal. **Catarrhal laryngitis** also begins with a cold, but the croupy breathing and dyspnœa steadily increase, and there are no intermissions as in the milder variety of the disease. At the same time there may be a considerable degree of fever. Such a case will probably last for a week or longer, and you must also remember that laryngeal catarrh may occur at the outset of other inflammatory diseases, such as measles, or, as in a case we had in the ward recently, pneumonia ; and you must take care not to overlook the signs of these if they are present. You should always look upon laryngitis in children seriously. It is by no means uncommonly fatal, and I have had the misfortune to see two patients die on the table whilst tracheotomy was being performed.

We come now to the important matter of **diagnosis.** Catarrhal laryngeal spasm is usually easy to recognize. In most of your cases there will be a history of previous attacks, and the sudden onset in the middle of the night is very characteristic. Catarrhal laryngitis in its severe forms is much more difficult to be sure of. Such cases will often cause you great anxiety at the outset, for you may have great difficulty in excluding **laryngeal diphtheria.** I believe, indeed, that the diagnosis between the two may sometimes be impossible, for the signs of obstruction are the same whether it be due to mere swelling of the mucous membrane or to membraneous exudation, and there is nothing definite in the general symptoms to guide you. The only conclusive test would be a laryngoscopic examination, which it is, of course, almost impossible to perform. The chief points to be attended to, however, are these :

1. The more rapidly the symptoms set in, the more likely it is that you are dealing with simple and not membranous inflammation. As Trousseau has said, ' simple laryngitis makes a great fracas, but diphtheria insidiously installs itself.'

2. The voice is more apt to be lost in diphtheria than in laryngitis, and the cough to be less, for the membrane protects the mucous surface from irritation. Diphtheria, in short, is the more *silent* disease of the two.

3. The constitutional disturbance, but not the temperature, is usually greater in diphtheria, and the child looks more *ill* ; but this, of course, is an indication which it requires some experience to appreciate.

Enlargement of the glands in the neck is of doubtful value, for although that occurs in diphtheria, yet children who are most pre-

disposed to laryngitis from an unhealthy condition of the throat are apt to have it, too. This practical rule you will do well to lay to heart: *if in doubt regard the case as one of diphtheria, and treat it accordingly.*

Let us pass now to **treatment.** In dealing with a case of catarrhal laryngeal spasm you have to consider (1) what you should do when summoned during an attack; and (2) what steps you are to take in order to prevent recurrence.

When confronted with a case of catarrhal laryngeal spasm you will remember that, no matter how alarming the symptoms may appear, such cases are never fatal, and so you will keep calm and endeavour to allay the natural anxiety of the friends. Your next step should be to administer an emetic. You will find powdered ipecacuanha the best for your purpose. Give a 10-grain dose and repeat it in a quarter of an hour if necessary. Tinct. ipecacuanha is often recommended, but it is a much less trustworthy preparation. I have given as much as an ounce of it in teaspoonful doses every quarter of an hour without producing emesis, but if you get a fairly fresh preparation of the powdered root you will rarely fail with it. The next thing to do is to apply hot fomentations over the larynx and to arrange some method of inhalation. The best plan is to rig up a tent over the child's cot and to let the steam from a bronchitis kettle play into it. You may use a little creosote with the kettle if you like, but the great thing is to secure the local internal application of moist heat. These measures will usually be speedily successful, but if you are dealing with a severe case it may be useful to inject into the rectum by means of a glass syringe and a soft catheter an ounce of warm water containing 20 grains of ammonium bromide and 5 grains of chloral. In order to prevent the recurrence of an attack the following night, you should continue the administration of small doses of ipecacuanha throughout the next day, and at bedtime give a dose of antipyrin, 1 grain for every year of the child's age.

As most of these children are predisposed to their attacks by having enlarged tonsils or adenoids, it is well to attend to the condition of the throat in order to prevent the reappearance of the disease. Cold also should be guarded against, for it is a potent exciting cause. In Scotland attacks of 'croup' (as it was called) used to occur with special frequency on Saturday nights, the

probable explanation being that nursery floors were usually washed on that afternoon in preparation for Sunday, and the attacks were brought on by children being put to bed in a damp room.

In the treatment of **acute laryngitis** I would remind you again, that if you have the least suspicion that the case may be one of diphtheria it is your duty to give an injection of antitoxin, and to take all the precautions usual in dealing with the more serious disease. The rest of your treatment must proceed much on the lines already laid down for catarrhal laryngeal spasm. Inasmuch, however, as the obstruction here is organic and not spasmodic, you will expect less benefit from emetics. Should the case be a very 'florid' one—i.e. the temperature high and the distress great—the old so-called 'antiphlogistic' treatment often gives very gratifying results. It consists in blood-letting either from the jugular vein or by the application of a couple of leeches above the sternum. A full dose of calomel should also be given and antimonial wine administered in 5-minim doses every two hours. If the dyspnœa increases you must be prepared to perform intubation or tracheotomy, and it is well to remember that it is safer to operate too early than too late.

Other laryngeal diseases in children about which you may make mistakes are **congenital laryngeal stridor** and **laryngismus stridulus.** These are totally different diseases, and I shall have occasion to describe them when I come to speak of the neuroses of children, so I only mention them here for the sake of completeness. There is another condition which you should bear in mind when a child has dyspnœa of laryngeal origin, and that is the possibility that you are dealing with **papilloma** of the larynx. That occurs not very uncommonly in children; more commonly, I think, than in grown-up people, and oftener in boys than in girls. The child is usually about three or four years of age, and the symptoms simulate those of chronic laryngitis, for which papilloma is apt to be mistaken. So if you have a child brought to you with a husky voice and occasional slight dyspnœa, and a history that the symptoms have lasted for weeks or months, you should think of laryngeal papilloma. I think you can make an exact and certain diagnosis only by the use of the laryngoscope, and that is a matter of such difficulty that I advise you to send those cases to specialists; in any case you will require a specialist to treat them, because the modern method is to employ radium to cause the 'warts' to disappear.

Finally, you should not forget the possibility that you may be dealing with a case of **foreign body** in the larynx, although the history in that event will usually be quite clear.

TRACHEAL STRIDOR

Tracheal stridor closely resembles laryngeal stridor, but is less purely inspiratory in character ; indeed, the difficulty may be even more evident during expiration. The voice also is not lost, and the head is kept bent instead of thrown back. It may be due to pressure on the trachea by an **enlarged thymus** or enlarged **bronchial glands,** and in both these cases an X-ray examination may establish the diagnosis. It may also be due to a **retro-pharyngeal abscess,** the result, usually, of suppuration in the retro-pharyngeal lymph glands, although in older children it is sometimes the consequence of cervical caries. The symptoms are threefold : (1) pain and stiffness in moving the neck ; (2) difficulty in swallowing ; (3) stridor. The swelling can be felt by a finger passed down the pharynx. If fluctuation is felt, the abscess should be opened by a guarded bistoury under very slight anæsthesia.

BRONCHITIS

I now pass to the consideration of a disease which is extremely common in children—namely, *bronchitis*. Bronchitis is certainly commoner in childhood than at any other period of life, and the reason for that may be the tendency of children to get chilled owing to their large body surface ; contributing causes certainly are rickets and the occurrence of teething.

You constantly find bronchitis as one of those catarrhal manifestations of rickets of which I have already spoken, and you will find children who get an attack of bronchitis every time they cut a tooth. Why that should be I do not know. Some people say it is produced in some mysterious reflex fashion, but I think it is more likely that the teething simply lowers the child's resistance, like any other agency, and so renders it specially liable to suffer from chill. In some cases it may be due to the clothes over the chest getting wet from the ' dribbling ' which accompanies teething.

Now, ' bronchitis ' in itself is never a sufficient diagnosis. That is true not only of children, but of grown-up people. But it is

particularly true of children, because they are apt to suffer more than grown-up people from capillary bronchitis—that is to say, inflammation of the small tubes, which may be a very serious matter. And so, having established the fact that the disease exists, you have to ask yourselves two questions: first, Which set of tubes is involved—the large tubes or the small ones? and, secondly, What stage has the bronchitis reached—is it early or is it late? With regard to the first of these questions, you will settle whether you are dealing with bronchitis of the large or of the small tubes by the character of the accompaniments. If the **large tubes** are involved you will hear low-pitched accompaniments—i.e. sonorous rhonchi—if it is in the dry stage, or more or less coarse bubbling sounds in the moist stage; whereas if the **small tubes** are involved the accompaniments will be high-pitched rhonchi if the bronchitis is dry, or fine, crackling sounds if it is moist. Again, in a case in which the fine tubes are involved the accompaniments are far more numerous than in those cases where it is the large tubes which are chiefly affected. The obvious reason for this is that there are far more small tubes than big tubes in the lung, so inflammation of the small tubes will result in your being able to hear many accompaniments; and if you hear rhonchi coming from every quarter you can be certain you are dealing with inflammation of the small tubes.

The other question is, What **stage** has the disease arrived at?

That is settled by the character of the accompaniments—whether they are dry or moist. In the early stage of the disease you find dry sounds, and in the middle stage the sounds become both dry and moist, whereas in the latest stage the moist sounds predominate.

The question of whether the large or the small tubes are involved in the inflammation is one of great importance in the prognosis, and that is chiefly why you wish to determine that point. You take a more serious view if the small tubes are chiefly involved, because the great thing you have to fear in a case of bronchitis in a child is that the condition may pass on to broncho-pneumonia—that is to say, to inflammation of the lung substance—instead of being merely an inflammation of the lung tubes.

The remote danger in bronchitis, you will remember, is that it leaves behind an unhealthy condition of the bronchial glands, which renders them liable to become the seat of tuberculosis.

With regard to bronchitis, I would further say that you should always take a more serious view in a case in which the chest wall is

very soft and yielding. Practically it amounts to this, that bronchitis in a rickety child is a far more dangerous affection than a similar degree of bronchitis in a child who is otherwise in good health. The rickety chest is so soft that it readily allows collapse of the lung, and the extension of the process to the collapsed lung, with the production of pneumonia.

Treatment

The **treatment of bronchitis** must depend upon the stage at which the disease has arrived. If you are dealing with an early case where there is fever, more or less dyspnœa, rapid breathing, and high-pitched dry sounds, then you ought to put the child in a tent, and let it have medicated inhalations—creosote, menthol or Friar's balsam. This is probably far more valuable than the administration of any amount of expectorants by the mouth. Here at this hospital, in the out-patient room, you get a wrong idea of how to treat these cases ; you cannot get out-patients to use bronchitis-kettles, and so one has to fall back on the second-best treatment—namely, the internal administration of expectorants. But when you have complete control of the case the best plan is to put the child in a tent, and fit up a bronchitis-kettle, and get the child breathing in an atmosphere of medicated moist, *warm* air. Don't get it too hot in the tent or you can scald the child. The temperature ought not to exceed 70° F. It is well also that those patients should have either a cotton-wool jacket or a poultice over the back. It is a mistake to put poultices over the front of the chest, because they are too heavy ; they increase the labour of breathing, and the child ought not to have anything to restrict the breathing. But a poultice over the back is often very valuable. You may use for the poultice linseed-meal and mustard. There is no harm in giving, in the early stage, small doses of ipecacuanha by the mouth along with alkalis, such as citrate of potash or acetate of ammonia. And such patients should be encouraged to drink large quantities of hot fluids, because there is nothing better for promoting the secretion from the bronchial tubes than the administration of hot fluids of all sorts. That is the meaning of the old domestic teas which used to be brewed. They act, not in virtue of the material in them, such as camomile, but simply in virtue of the fact that you are giving large quantities of hot liquid.

In the later stage, when you have got to deal with much liquid

mucus in the tubes, you will find it an advantage to administer an emetic, particularly where the child has difficulty in getting rid of the mucus, and where there is much choking and a furred tongue. Tinct. ipecac. is usually used, but is rather uncertain in its action. Pulv. ipecac. (gr. v.) is better.

There are, however, two conditions which contra-indicate the giving of emetics. One of these is the presence of much cyanosis. The reason is that in that condition the nervous system is, as it were, partially narcotized; it does not respond to the irritation of an emetic, and you are apt to have the ipecacuanha or other emetic retained, and great depression result without emesis. Great prostration is the other condition which contra-indicates the giving of an emetic in the second stage.

For attacks of collapse with failure of circulation and respiration you should use hot mustard baths, oxygen inhalations and alcohol. Injections of adrenalin and strychnine are also useful. Atropine is advocated by many when the bronchial secretion is abundant and liquid and threatens suffocation, but beware of getting the mucus too sticky! Collapse attacks are apt to come on suddenly, especially in the night, so the nurse should be carefully instructed how to deal with them, and all the remedies should be at hand. In the most severe cases, especially in young children, when the whole pharynx, larynx, trachea and bronchi are full of secretion, the only hope is bronchoscopic aspiration by an expert.

In the last stage—that is to say, when the patient is convalescent—you should administer such tonics as iron and cod-liver oil, and apply stimulating liniments to the chest until all accompaniments have disappeared. You must make convalescence complete because of the great danger of tuberculosis starting in bronchial glands which have become chronically inflamed.

LECTURE XXII

RESPIRATORY DISEASES—*Continued*

ASTHMA

Asthma is always an interesting disease, but it is specially interesting in childhood, not only because we meet it then in its earliest and simplest forms, but also because it is then that we have the best chance of treating it. Relatively, too, it is commoner in the child than in the adult, for about one-third of all cases begin below the age of ten and may start during infancy. Like all the diseases of childhood in which nervous influences play a large part, it is more frequently met with amongst boys than amongst girls.

There are four varieties or types of 'asthma' in childhood:

1. The **spasmodic** type, which presents the classical picture of recurring episodes of the characteristic asthmatic dyspnœa. This type is commonest in older children.

2. The **hay fever** type, in which nasal catarrh is the most prominent feature. Such cases are apt to be regarded as simply recurrent 'colds in the head.'

3. **Bronchial asthma,** in which there is, at the outset, a good deal of bronchitis, to which spasm of the tubes becomes superadded. Cases of this type are very common, and the attacks are often prolonged.

4. The **bronchitic** type, which is peculiar to children, especially babies, and in which the asthma masquerades as a pure bronchitis, so that the real nature of the condition is often overlooked. It is distinguished from a simple bronchitis by the suddenness of the onset and subsidence, by the fact that dyspnœa precedes the appearance of physical signs in the lungs, by the comparative absence of fever, and by the presence of an eosinophilia.

Thymic asthma.—In infancy asthma may be simulated by the rare condition called 'thymic asthma,' or 'para-asthma,' as some

term it, a spasmodic form of dyspnœa brought about by the pressure on the trachea of an hypertrophied thymus or enlarged bronchial glands. Thymic asthma always begins before the age of eighteen months, and is distinguished by the presence of the physical signs of enlargement of the thymus or of the bronchial glands. A screen examination is of great help in the diagnosis.

The Nature of Asthma

The dyspnœa of asthma is attributed, as is well known, to obstruction of the bronchi either from spasm of the muscle or from swelling of the mucous membrane, or from both these acting together. The obstruction, whichever form it takes, may be brought about either (1) through the respiratory centre, or (2) by direct action on the bronchial mucous membrane. We must consider each of these modes separately.

1. The respiratory centre may be affected (*a*) psychologically, (*b*) reflexly, (*c*) by the blood.

(*a*) Amongst psychological influences may be mentioned fright and excitement (both of which are specially potent in determining an attack in children), and suggestion, the part played by which in early life is difficult to determine.

(*b*) The chief sites of peripheral irritation which may affect the respiratory centre reflexly in children are the nose and throat and the alimentary canal. Phimosis would also appear to be an occasional cause

(*c*) The centre may be affected directly by the blood circulating through it in the way to be described immediately.

2. The changes in the bronchi which lead to obstruction may also be brought about directly and without the intervention of the respiratory centre, by the presence in the blood of a specific substance which acts as an irritant. The substance in question is believed to be of the nature of a foreign protein or 'allergen' to which the asthmatic subject is 'sensitive.' Some seem to be born with this sensitiveness, others acquire it, others have it 'thrust upon them' by disease or accident.

It may be a constitutional peculiarity, often inherited. Such an inborn metabolic irritability may reveal itself by producing, not asthma alone, but other episodic disturbances such as migraine and cyclical vomiting, as well as skin disorders like eczema and urticaria. Such patients are usually sensitive to several proteins,

but the degree and character of the sensitiveness may vary from time to time and may come and go suddenly. Sensitiveness may be acquired as the result of an overdose of a foreign protein. It is said, for instance, that an infant may sometimes become sensitive to egg protein in consequence of being given large quantities of albumen water, and sensitization after the injection of serum is, of course, well known. Sensitiveness may also be 'thrust upon' a patient by an acute illness—whooping-cough seems specially prone to act in this way in children—or by an operation, particularly, perhaps, an operation on the throat.

An asthmatic child, therefore, may be born with an unstable metabolism, or it may have been thrown off its metabolic balance in one of the ways described. Our experience at the Hospital for Sick Children (Great Ormond Street), however, has been that sensitiveness is a factor in less than half of all cases of asthma in childhood, and that even when it is present it is only in a small number that the specific excitant of the asthma can be determined. It would seem, indeed, as if sensitiveness is more often a predisposing condition as regards asthma in children than the actual cause of it.

Where sensitiveness is the cause the protein to which the patient reacts may reach the blood in various ways.

Ingestion.—First, it may be ingested in the form of food. Almost any food may produce a reaction, even—perhaps especially—such common ones as the proteins of eggs and milk. Sensitiveness to food protein is usually inborn, and tends to disappear with time. It is therefore more likely to be a cause of asthma in early childhood than in later years.

Inhalation.—Secondly, the protein may be inhaled in the form of dust of various kinds—pollen and so forth. This form of sensitiveness is oftener acquired than inborn—though even when acquired there has probably been an inborn tendency to metabolic instability—and is more persistent than sensitiveness to food protein, having less tendency to disappear of itself. It is of more importance in the causation of asthma than sensitiveness to the protein of foods.

Bacteria.—Thirdly, there is some reason to believe that the protein to which an asthmatic patient is sensitized may be of bacterial origin and derived from organisms growing at some seat of chronic infection, of which the commonest is probably the naso-

pharynx. So-called bronchial asthma is the form of the disease most likely to arise in this way.

The fact that a patient is 'sensitized' can often be inferred from his history. Thus one of my patients had attacks only when living on a farm in close contact with animals, whilst another was only asthmatic when living in the neighbourhood of trees. Sensitization to common articles of food or to dust is more difficult to detect, but by the use of the cutaneous test with watery extracts of different proteins the question whether and to what the patient is sensitive can be definitely determined. I think it is advisable to use such tests in all asthmatic children, but at the same time I am bound to say that I have been disappointed in the results of treatment based upon them. This matter, however, will be referred to immediately.

Treatment

Treatment of the attack.—Treatment of the attack of asthma is much the same as in the adult. Subcutaneous injection of adrenalin (2 to 3 minims) is the best remedy, and acts by antagonizing the action of the vagus and stimulating that of the sympathetic. Ephedrine or ephetonin ($\frac{1}{4}$ to $\frac{1}{2}$ grain) by mouth sometimes act well. Morphine, though effective, has to be used cautiously in childhood, and children do not like 'fumes.'

Treatment of the tendency. More important is the treatment of the asthmatic tendency. Here the chief thing to realize is that 'asthma' is not a disease of the respiratory system, but one of the child as a whole, and that treatment must aim at breaking the asthmatic habit, for, as someone has put it, 'the best cure for asthma is not to have it.'

In the general management, therefore, the patient's whole life must be regulated, and nervous excitement and overstrain particularly guarded against. At the outset the question of 'coddling' *versus* 'hardening' will obtrude itself, and is not always easy to answer. Whilst we must carefully avoid making the asthmatic child a 'hot-house plant' invalid, it must also be admitted that needless exposure to weather, especially fog and damp, is injurious particularly in the bronchial type, and we must not make a fetish of fresh air. The question of climate also arises at this stage, and we shall be asked whether a permanent change is desirable. As to this it is unwise to be too dogmatic. All one can say is that a

warm dry climate is theoretically the best—again especially in the bronchial type—but such a climate is not to be had in these islands. Mere change of locality is highly speculative, and is apt to be only an escape from the frying-pan into the fire. It is true that some patients seem to be much freer from attacks in one place than another, but this may be due to some factor other than the climate—removal, for example, from the immediate neighbourhood of animals. Certainly we should never advise the parents to change their place of residence unless they have satisfied themselves by a prolonged trial that the child is free from the disease in the place to which they propose to move. My usual advice is that they should endure the evils they know rather than fly to others that they know not of. Immunity from attacks is usually obtained at an altitude of 5,000 feet, but this is impracticable as a permanent place of abode, and the attacks recur so soon as the patient returns to a lower level.

Diet is of importance, for experience has shown that improvement often results from the avoidance of 'indigestible' things, and the provision of small dry meals with no supper. Carbohydrates should be avoided rather than fats.[1] In children, however, it is not wise to cut down the diet so rigidly as can often be done with advantage in the adult, for the needs of growth have to be provided for, and the child must not be sacrificed to the disease. If tests have shown that the child is 'sensitive' to any particular article this should, of course, be banished from the diet, but my experience has been that it is only in exceptional cases that this has much effect on the disease.

Drugs.—The drugs which might be expected to be of use in asthma are those which lessen vagus action and those which diminish the excitability of the respiratory centre. Belladonna and its allies act in the former way, arsenic is said to act in the latter, and there is no doubt at all of the great benefit often derived from them in cases of asthma in childhood. I am in the habit of giving small doses of potassium iodide (especially where there is a bronchial element) along with full doses of belladonna or stramonium and arsenic as a routine. The mixture should be given regularly after meals for prolonged periods, and, so used, often effects great improvement. I have seen this happen too often to have any

[1] It has been found, however, that in some asthmatic children great benefit results from the administration of glucose (3 drachms of powdered glucose in lemonade between meals three times daily).

doubts of the real curative value of such a combination, and, indeed, I regard it as on the whole the best treatment for asthma in children. For the purpose of warding off a threatened attack a full dose of a sedative such as chloral at bedtime is often very helpful. Antipyrine sometimes acts equally well.

Dilute hydrochloric acid is sometimes prescribed for asthmatic children as most of them appear to have a hypochlorhydria. I am not convinced, however, of its utility.

Operations.—The question of removing enlarged tonsils and adenoids in asthmatic children will often arise, and if the enlargement is causing obstruction, or if the tonsils are infected, it is best to operate. It is unwise, however, to make any promise as to the effect on the asthma, but benefit is most likely in cases of the bronchial type. On the other hand, I have, like everyone, known several cases in which asthma appeared for the first time after the removal of tonsils and adenoids. Of the effects of cauterization of the nose for asthma in children I have had very little experience.

Vaccines.—A course of vaccines is worth trying in cases of the bronchial type, using for choice an autogenous vaccine prepared (if possible) from sputum obtained during an attack. The results, however, are extremely uncertain, though at least it may be said that the treatment can do no harm. When it is successful—and I have seen cases where it certainly seemed to be—it is always doubtful whether the benefit obtained is due to a raising of the resistance to infection or whether the vaccine has really acted as a desensitizing agent. That, however, is an academic question.

Specific treatment. If the child is known to be a 'sensitive,' removal of the offending agent from his environment is, when possible, always to be advised, though here again definite cure should not be promised. In all such cases, too, it is wise to forbid the use of feather pillows, hair mattresses, eiderdowns, and fur coats, as well as horse-riding and the keeping of pets, for even if the patient is not 'sensitive' to any of these already he may easily become so. These, however, are elementary precautions, and the ideal to be aimed at is a general desensitization which would, so to speak, cut the ground from under the feet of the asthmatic process. Various substances have been suggested as desensitizing agents—peptone, milk, some of the child's own blood, tuberculin, and cultures of organisms prepared from the nasopharynx or from the bowel. These are **non-specific desensitizers** and of them I have

had most experience with peptone, usually given intravenously. It is necessary to proceed cautiously, and it is difficult to regulate the dose, for when an asthmatic child is admitted to hospital the asthma usually disappears for a time at least, and so there is nothing by which to gauge the effect of the treatment. On the whole, I think I may say that our experience at the Hospital of Sick Children has not borne out the high opinion of the treatment which has sometimes been expressed.

Specific desensitization may be used when the causative allergen is known, but as it is rather troublesome to carry out it is best left to the expert.

Prognosis

Finally, a word as to the outlook for the asthmatic child. The immediate attack is, of course, rarely dangerous except in young or delicate infants. Assuming that preventive treatment fails, what chance is there of the attacks being 'outgrown'? A very fair chance indeed. I have been agreeably surprised, when following up cases of asthma, seen often several years ago, to find in how large a proportion the attacks have either ceased altogether or have become few and slight, although when the disease has lasted for several years there may be some resulting emphysema and some stunting of growth and development. On the whole, however, the outlook for the asthmatic child is much brighter than the inexperienced would suppose. I think at the best we can teach the child to manage his asthma so that it ceases to be much trouble. I doubt if we ever really 'cure' the classical type of primary spasmodic asthma, but we can reduce it to the level of a minor nuisance!

LECTURE XXIII

RESPIRATORY DISEASES—*Continued*

PRIMARY OR CROUPOUS PNEUMONIA IN CHILDHOOD

Attempts to classify cases of pneumonia in children have given rise to a considerable amount of confusion, and, indeed one may admit frankly that it is often a very difficult matter at the bedside to refer any given case to a particular division of the disease. Hence for practical purposes I think it is best to keep one's divisions as broad as possible, and I therefore propose to divide cases of pneumonia into those which are primary on the one hand, and those which are secondary on the other. By **primary pneumonia** I mean a case in which the inflammation starts in the lung substance; by **secondary pneumonia** I mean that form of pneumonia in which the inflammation of the lung-substance is consecutive to disease of the air-passages, usually to bronchitis. Hence the term 'broncho-pneumonia' usually applied to it. Further, you may contrast primary and secondary pneumonia in this way: that primary pneumonia is usually, perhaps always, lobar; whereas secondary pneumonia is lobular. In the second place primary pneumonias are practically always due to the pneumococcus, whereas secondary pneumonias may be due to many organisms, perhaps most commonly to a streptococcus.

When you come to ask yourselves which form of pneumonia is the most frequent, it is very difficult to answer, because it depends very much on the type or class of case which you see. In this hospital, where we do not treat cases of the acute infectious fevers, there can be no doubt that primary pneumonia is far commonest; whereas if you were to go to a Fever Hospital, where there are cases of measles and whooping-cough, you would probably find a large number of cases of secondary pneumonia, because secondary pneumonia is a common complication of many of the acute infective fevers. But in ordinary general practice, you will meet with very many cases of

primary pneumonia in children of all ages. I say it is met with in children of all ages, but it is most frequent, as statistics show, between one and two years of age. That is the period of maximum incidence of primary pneumonia in childhood, for about that age children seem to be peculiarly susceptible to the attack of the pneumococcus.

I have recently gone through the statistics of seventy cases of primary pneumonia which I have had under my care in children below seven years of age, and I found that half of these were below two years of age.

Mode of onset. As regards the onset and symptoms of primary pneumonia—and it is only with primary pneumonia I shall deal this afternoon—it may begin sometimes in a very deceptive way. It is not uncommon, for instance, for it to begin with **vomiting.** A child may be taken with severe vomiting, along with rise of temperature, and for the first few hours these may be the only prominent symptoms. A mode of onset like that is apt to deceive you. It makes you think perhaps of brain trouble, such as meningitis. Or it makes you think of some of the acute fevers, particularly scarlet fever, which you know often begins with vomiting.

Another deceptive mode of onset is with **drowsiness.** I have known cases in which, even for a day or two preceding the actual onset, there was great drowsiness. I remember, for example, the case of a little boy of whom it was said that a day or two before he was ill he was always falling asleep at school; apparently before the disease had made a proper beginning that symptom had arisen. As a rule, however, the disease sets in fairly abruptly, with a rise of temperature and a certain amount of shivering. A true rigor is not so common as it is in grown-up persons; but some shivering is not rare. And sometimes in sensitive nervous children these shivering fits take the form of a slight attack of convulsions. Very quickly you will notice an alteration in the character of the respiration, and that is one of the most important things for you to observe, because it is upon the altered character of the respiration that you will sometimes have to depend for making your diagnosis in the early stages. And the alteration is this: In the first place the respirations are greatly increased in frequency out of proportion to the increase of the pulse-rate. Secondly, the respiration takes on that character which I have often demonstrated to you in the out-patient department as 'inversion of the respiratory rhythm.' In the normal state

of things the respiratory rhythm goes thus: inspiration, expiration, pause; but in pneumonia, and indeed in all grave respiratory affections in young children, there tends to be an inversion of the rhythm, so that one gets expiration, inspiration, pause; and you will remember that the expiration is attended often by a curious grunt, and during the grunting expiration the alæ nasi are blown out, which is again an inversion of what ordinarily happens in an adult with dyspnœa, in whom, if the alæ nasi are acting, they open out during *inspiration*. Upon this altered character of the breathing, as I have said, you may have to depend in the early stages for making your diagnosis, and therefore it is extremely important that you should be familiar with these respiratory changes. Quite early in the disease you will notice that the child is flushed, and not uncommonly you find a certain amount of herpes. But in a large number herpes is absent so you must not depend upon it at all as a diagnostic sign; it is too often absent to be of use in that respect.

Physical signs. The unfortunate thing about the physical signs of pneumonia in children is that they are often absent at the outset and for some time after the disease has fairly begun, and, indeed, you may find them only becoming marked when the crisis is at hand. I have notes of cases in which they even became evident only *after* the crisis. This is no doubt due to the inflammation starting deep in the lung and only by degrees reaching the surface. An X-ray examination of the chest will sometimes show definite areas of pneumonia which present no definite physical signs (fig. 53).

The most important physical sign is an impaired note on percussion, and I am constantly pointing out in the wards that if you wish to elicit this impaired note you must percuss properly and especially lightly. Too much stress cannot be laid on the word 'lightly.' Repeatedly one sees areas of pneumonia missed on physical examination because the observer percusses so forcibly that he elicits resonance underneath the patch of pneumonia altogether. As Oliver Wendell Holmes wrote:

> 'If the poor victim needs must be percuss'd,
> Don't make an anvil of his shrinking bust!'

You cannot, therefore, be too delicate in your percussion if you are to make out the signs in an early case. It is important to know

where to percuss, and that will depend on the sites at which pneumonia is commonest.

Pneumonia in children differs from pneumonia in the adult in this, that the apex is relatively much more often affected than it is in grown-up people. I find that of my own cases, for instance, one-third were apical. That is a far higher proportion than in the adult. Apical pneumonia in the adult is the exception, but you cannot say that of pneumonia in the child. Further—and this is a curious and unexplained fact—of the apical cases two-thirds occurred in the right

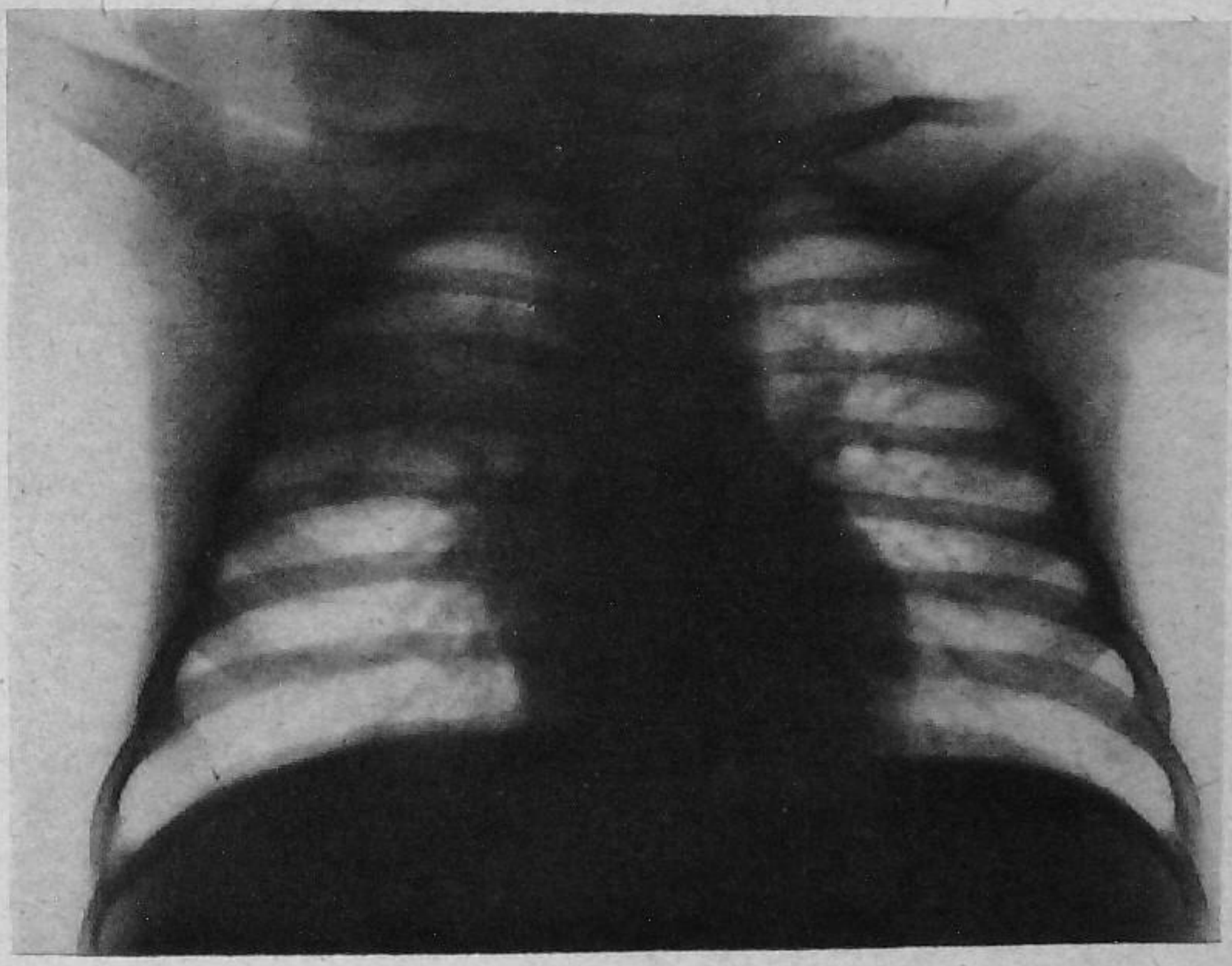


FIG. 53.—X-RAY SHADOW OF PNEUMONIA IN RIGHT UPPER LOBE. When this was taken there were no definite physical signs in the chest.

lung. I think you may say that the most common situations for pneumonia in the child are the left lower and the right upper lobe, and therefore it is these situations in a doubtful case which you will examine with the greatest care. In particular I wish to commend to you the importance of percussion below the right clavicle. I have known any number of cases of pneumonia overlooked because the observer had failed to percuss lightly below the clavicles.

Apart from percussion, the other chief sign is an alteration in the breath-sounds, the development of what is known as tubular breathing—that high-pitched whiffing kind of breathing which occurs over

consolidated lung. That is a sign which develops sooner or later in all cases, but it does not develop quite so soon as the impairment of note, and so is of less value than percussion. These are the only methods of physical examination which you need employ—delicate percussion and auscultation to determine the presence or absence of tubular breathing.

Symptoms. Compared with the adult, the symptoms are far less pronounced. You will often see—I saw one the other day—a child with full-blown pneumonia standing up and prancing about in its crib, apparently not much the worse for the fact that it had a temperature of 103° F. and consolidation of the lobe of one lung. You do not see that in the adult. A grown-up person with lobar pneumonia is very ill indeed, whereas a child with the disease may not appear to be seriously ill at all. The reason for this is that the toxæmic symptoms which are so very serious and prominent a feature in pneumonia in the grown-up person are comparatively absent in a case of pneumonia in childhood.

For some reason, which we do not know, children do not seem to suffer from the toxins of pneumonia in the way that the grown-up person does. Delirium, for instance, which is one of the most prominent symptoms of toxæmia in the adult, is a very rare thing in the child, and, on the other hand, heart failure, one of the things which gives you the greatest anxiety in a grown-up person, is something which in the child you need hardly fear at all. So that the two great groups of symptoms, the toxæmic and those which result from heart failure, are comparatively absent in children, and for that reason the symptoms, apart from the feverishness and the hurried respiration, may be very trivial indeed.

Mode of termination. Pneumonia of the primary sort in children, whether it be lobar or lobular, terminates, as a rule, by crisis. You may put that in another way, and say that pneumococcal pneumonia in children tends to be a self-limited disease, as distinct from streptococcal and other forms of pneumonia which are not self-limited, but drag on for an indefinite time. In the majority of cases the crisis occurs on the seventh or eighth day. But in a considerable number the crisis occurs earlier, on the fifth or sixth day, and in a comparatively small number after the eighth, say on the ninth or tenth day. It is rather characteristic of pneumonia in children that the crisis is apt to be preceded by one or more sudden falls of temperature, which are spoken of as ‘ pseudo-

crises.' You can distinguish a pseudo-crisis from a real one by the fact that after a true crisis the respiration-rate falls, but in a pseudo-crisis the respiration-rate is not affected. So the respiratory chart is really a better guide to the occurrence of the crisis than the temperature chart alone. Again, sometimes after the true crisis there is a temporary rebound of fever lasting a few hours, twelve hours perhaps, which may alarm you unnecessarily, and make you think that some complication is arising. These temporary rebounds after the crisis are not very uncommon in early life. The heat-regulating centres in children seem to lose their equilibrium more easily than in the grown-up person. If you set them into violent oscillation, it takes some time before they settle down again. And if the temperature drops to subnormal at the crisis, it is apt to jump up above the normal again, just as an india-rubber ball rebounds when thrown to the ground. But in a few hours it finally settles down to the normal.

Sometimes, instead of a crisis occurring, the fever is prolonged, and it is about the causes of prolongation of fever in pneumonia that I now want to say a few words.

Prolonged Pyrexia in Pneumonia

One may divide cases in which the temperature does not fall into, first, *protracted cases*—that is to say, cases in which, for some reason, the temperature runs on for ten, fourteen, or more days—I have even known it to go on for several weeks—without the disease spreading and without any complications arising, and for no particular reason which you can find. It seems that in such cases natural resolution is extremely slow, and I think they are apt to terminate in fibrosis of the affected part of the lung. Cases of fibroid lung affecting the upper lobe, which are sometimes met with in children, and which are not tuberculous, probably have their origin in an attack of pneumonia of this sort, which has run a protracted course.

Secondly, the prolongation of the fever may be due to the *spreading of the pneumonia* or to its *relapsing*. That is to say, the disease spreads from one lobe to another, and the child may, as it were, run through two attacks of pneumonia consecutively. I have known practically every lobe of the two lungs to have been involved successively, each one giving rise to a further rise of temperature as it has become affected. One speaks of these cases sometimes as

'wandering pneumonias'—the disease wanders from one lobe to another. The prognosis in these circumstances is not necessarily bad, but one is naturally always more anxious in such a case than in a straightforward pneumonia, in which one lobe only is involved.

Thirdly, failure of the temperature to come down, or a tendency for it to rise after it has once come down, may indicate the development of *complications*. These complications are due to the starting of a focus of pneumococcal infection elsewhere, and in young children —and the younger the child the more this is true—there is rather a tendency for this generalization of the infection. Indeed, in quite young infants a condition of general *pneumococcal septicæmia*, as it is sometimes termed, is by no means uncommon. Of the possible complications, *empyema* is certainly the commonest. It is characteristic of empyema that after the crisis the temperature tends to remain down for a few days, and then gradually begins to rise again. You see it gradually rise until pus is found and evacuated, and then it comes down. This is what usually happens in children above the age of three. Below this, however, empyema often develops as the result of a general pneumococcal infection before the pneumonia has resolved and whilst the temperature due to the lung inflammation is still high. Dr. H. C. Cameron has termed this 'syn-pneumonic' empyema to distinguish it from the 'meta-pneumonic' empyema of older children, as described above. In the syn-pneumonic variety the prognosis is much more grave, and the empyema should not be opened until the pneumonia has subsided, aspiration alone being resorted to meanwhile.

The signs of empyema are the development of stony dullness, with very marked *resistance* on percussion; that is the chief point, marked resistance on percussion at the base of one lung. It may, however, be extremely difficult to tell an empyema with certainty from an unresolved pneumonia, or from one of those cases in which a thick patch of lymph forms over the pleura, and, indeed, there is only one way of diagnosing empyema with certainty, and that is by putting in an exploring needle; and if you find the temperature chart creeping up like that after the initial crisis and the dullness persisting, and still more, much resistance on percussion at the base, you should not delay exploring, because it is certain that if empyema is diagnosed early and evacuated, the prognosis is vastly better than if it is left till later. The cases of empyema which do badly are those in which

the diagnosis has not been made until two or three weeks after pus has formed.

Another common complication is *middle-ear disease*—that is to say, an infection of the middle ear by the pneumococcus. I have known a case in which the temperature ran on after an attack of pneumonia for four weeks, and we could not discover what was the cause, until finally some pus was discharged from one ear, and after that the temperature came down and everything went well. Observe this about pneumococcal middle-ear disease, that it may not give rise to any local signs which would make you suspect its existence; the child may not complain of earache, and there may be nothing to put you on the scent as to the true nature of the trouble. So where there is a prolongation of the fever, and you can find no cause for it do not omit to examine the drums. We omitted it in the case I spoke of, and that is why we did not find the cause of the temperature. The third complication I have to speak of is a very serious one, namely, *pneumococcal meningitis*. The difficulty about meningitis is not so much to diagnose it when it is there, but it is that you may think there is meningitis when it is *not* there; and you are apt to do that because several of the symptoms and signs of meningitis may occur in cases of pneumonia, especially, perhaps, in apex pneumonias, simply as a result of the pneumonia itself. They may also occur in middle-ear disease where there is no meningitis at all. I refer especially to such signs as vomiting and head-retraction, and even the development sometimes of Kernig's sign. All these may occur in cases in which there is no real meningitis, and you may often be in doubt whether you are dealing with an apex pneumonia which is simulating meningitis, or with pneumonia complicated by meningitis. The term *meningism* is applied to this condition which simulates meningitis. The only way of determining this point in a doubtful case is by lumbar puncture.

Another complication is *pericarditis*, and a very serious one it is; but I do not propose to say anything about pericarditis to-day. So I shall now pass to the next complication, and that is *pneumococcal peritonitis*. This is probably a commoner complication in pneumonia than is supposed. It is a complication which is rather apt to be overlooked, and it is only in comparatively recent times that its existence has been properly recognized. And yet pneumococcal peritonitis occurs by no means very infrequently in childhood, either as a complication or as a sequel to an attack of pneumonia. The

usual symptoms by which it is ushered in are development of abdominal pain, diarrhœa, vomiting, and delirium, with a rise of temperature if it has already fallen. Quite soon, too, retraction of the abdomen can be observed. These are the initial symptoms, and as effusion takes place you begin to find signs of fluid in the abdomen, and when that has occurred, many of the acuter symptoms subside, and the case becomes more like one of tuberculous peritonitis. The treatment of this complication is surgical.

The last complication I shall mention is *pneumococcal arthritis*. This is not a common complication, but it occurs every now and then especially in quite young children, and it is a curious thing that it does not necessarily go on to suppuration. You may find that a joint, often one of the smaller joints, becomes red and swollen, and that it is hot and painful, and after a time the whole thing subsides, and no further harm results. Every now and then, however, suppuration takes place, and if this happens, the treatment adopted must be the same as in any other form of suppurative arthritis.

These, then, are the commonest complications of primary pneumonia in childhood. Sometimes several of them occur together, and it is not uncommon to have a child who has an attack of pneumonia developing middle-ear disease, and perhaps meningitis, both the result of the pneumococcus; or he may have pneumonia followed by empyema and an attack of pericarditis; or where there is generalized pneumococcal septicæmia, practically all these things may occur together. The latter cases, I need hardly say, are almost inevitably fatal.

Diagnosis

As regards the diagnosis of primary pneumonia in children, I have told you that it is at the outset that your difficulties will arise. You are called to a child who has a sudden rise of temperature and some vomiting, and you are asked by the parents what is the cause of the attack. Naturally, you think first of the acute specific fevers, and the acute specific fever which is most apt to simulate pneumonia is *scarlet fever*, because, as I told you before, scarlet fever often begins with vomiting and a sudden rise of temperature, just as pneumonia does; but, of course, in the latter you do not have a rash or sore throat, and further, whereas in scarlatina the rate of the pulse is specially raised, in pneumonia it is the respiration-rate which is

specially affected, and attention to that point of difference will usually keep you right.

Another condition which may simulate pneumonia is *acute abdominal disease*, especially appendicitis. This is now generally recognized, and I think all surgeons are alive to the possibility that a pneumonia involving the lower lobe of the right lung may simulate very closely an attack of appendicitis. Cases in which the surface of the pleura covering the diaphragm is specially involved seem to me to be those in which the simulation is most liable to occur. You will find a like simulation in cases of pericarditis, where the pericardium covering the diaphragm is involved ; these also may simulate abdominal disease very closely. Hence, when you are dealing with a case of suspected pneumonia, with pain on the right side, and vomiting and a rise of temperature, you should always have in mind the possibility of appendicitis. Conversely, when there is doubt about an acute abdominal condition, remember pneumonia, and study carefully the character of the breathing, and examine for physical signs at the right base. Very occasionally both conditions may be present at the same time.

Lastly, pneumonia may specially simulate *meningitis*. I have already referred to that in speaking of meningitis as a complication of pneumonia, and I need not say anything further about the differential diagnosis.

Prognosis

As regards the prognosis of primary pneumonia in childhood, this may be said, that it is vastly better than it is in grown-up persons, and, indeed, I always regard an uncomplicated case of primary pneumonia in a child as a comparatively trivial matter, unless the patient is below one year of age. In early infancy even an uncomplicated case may be serious, but in children of two years of age or thereabouts uncomplicated pneumonia is almost never fatal. Of a large series of uncomplicated cases below the age of seven which I have had under my care at the London Hospital, not one died. But when there are complications the case is different, for some of these are very serious indeed. The most dangerous complications are meningitis and pericarditis, but fortunately these are rare. In the absence of complications you may usually prophesy without fear that in about a week the disease will have terminated by crisis, and that the patient will get well.

Treatment

In a case of uncomplicated croupous pneumonia there is very little you need do in the way of treatment, and, as a matter of fact, my own patients, except for nursing, which is very important, get really nothing which can be described as treatment at all, and I find that they do very well without it. If the child is restless and the temperature very high—that is to say, 104° F. or upwards—it is perhaps a good thing to reduce the fever a little by tepid sponging ; but there is no use in attempting to force the temperature down too far. Patients with high temperature do as well as, or even, I think, better, than those in which it is low. You may also give a simple diaphoretic mixture to keep the skin and kidneys active.

The question of chemotherapy has nowadays to be considered. I have already said that an uncomplicated case of primary pneumonia almost always gets well without any special measures, but the parents are almost certain to ask about ' this new drug ' and we do not as yet know whether or not its use may prevent complications. Certainly in broncho-pneumonia, which we shall be speaking of in the next lecture, chemotherapy plays an important part, so the matter had better be dealt with once and for all. At the time of writing the drug of choice is sulphapyridine, also called ' M. & B. 693 ', or sulphadiazine. The former causes much less vomiting than in adults, and since the mortality rate is so low anyway it is difficult to know if later sulphonamide preparations are any better. The basis of dosage is 1 grain per pound of the child's body weight in twenty-four hours. For small infants it is probably best to give double this for the first twenty-four hours. As with all chemotherapy, use the drug in full doses for two or three days and then reduce to half the dose or less for another day or two. If you are going to do any good you will get results quickly and there is no point in going on for weeks. The dose having been worked out according to weight, make it come to the nearest $7\frac{1}{2}$ grains because this is the amount in one tablet (each tablet is $\frac{1}{2}$ gramme) and give the total number of tablets in six doses in the twenty-four hours, that is every four hours. The tablet should be crushed and given in a little sweetened orange juice. Children do not seem to have nearly as much trouble with nausea and vomiting as adults taking this drug. The fluid intake is important and must be fixed—and recorded !

Digitalis, which many use in the treatment of pneumonia in the

adult, is never required in children, and I very rarely order alcohol. Nerve sedatives, also, are scarcely ever called for; but if want of sleep is a difficulty, a few grains of Dover's powder may be given with advantage.

As regards the general treatment, see that the room is kept well ventilated, that the child is not over-clothed or over-fed, and that he is allowed plenty to drink.

The treatment of complications I have already referred to.

LECTURE XXIV

RESPIRATORY DISEASES—*Continued*

BRONCHO-PNEUMONIA

I wish to speak first to-day of **broncho-pneumonia.** This is a disease which you often see in the wards, and it is one of great fatality—so much so that about 17 per cent. of the total infantile mortality results from it. Broncho-pneumonia may be primary or, more commonly, secondary, occurring in the course of another disease, such as measles or whooping-cough, and in such cases the broncho-pneumonia is very often the last straw, as it were; it is what causes death. You will often have difficulty in deciding when you are dealing with simple bronchitis and when the disease has passed on a stage further—namely, to broncho-pneumonia. I would like to emphasize the fact here that you cannot diagnose broncho-pneumonia with certainty from simple bronchitis by physical signs; you have always to consider the general symptoms. If there is high fever lasting more than two or three days, if there is much lividity and much depression, then you can be pretty certain, no matter what the physical signs are, that you are dealing with broncho-pneumonia and not with mere bronchitis. If there is one physical sign of assistance more than another, it is the development of fine crepitations towards the base; and if in any case of bronchitis there are fine crepitations towards the base of one lung, you should be suspicious that you have to do with patches of broncho-pneumonia. The patches may be so small, and so deep down, and so enclosed by healthy lung, that there may be no dullness on percussion but the more lightly you percuss, the less likely you will be to miss such patches as exist.

Diagnosis of Croupous from Broncho-Pneumonia

The other disease from which you will have difficulty in distinguishing it is croupous pneumonia, and it will be well for you to

note some of the points which should guide you in discriminating between the two. Croupous pneumonia is always a primary disease, but broncho-pneumonia is usually secondary. Broncho-pneumonia is commonest in quite young children, in babies, whereas croupous pneumonia is not commonest in babies, but in children about two years of age. Broncho-pneumonia usually comes on more or less gradually, and the previous health has often been bad; whereas croupous pneumonia comes on suddenly, and usually in a child who has before been perfectly well.

Broncho-pneumonia usually affects both lungs, whereas croupous pneumonia is generally unilateral, affecting one lung, or, much more likely, part only of one lung. In broncho-pneumonia the base of the lung is the part which is almost always involved, whereas in croupous pneumonia it is often the apex. The course of broncho-pneumonia is uncertain, and instead of ending by an abrupt crisis, it ends usually by lysis. Croupous pneumonia, on the other hand, usually ends by crisis on the fifth to ninth day. The dullness in broncho-pneumonia is patchy and ill-defined, whereas in the croupous form it tends to follow the outlines of the lobe. Another point of some value is that in broncho-pneumonia you get signs of general bronchitis, whereas in croupous pneumonia the rest of the lung is healthy. These are the chief points to be attended to in making your diagnosis. The reason why it is so important is from the point of view of prognosis. Broncho-pneumonia is so fatal that about half the patients in hospital die, while, on the other hand, in croupous pneumonia recovery is the rule.

In spite of what I have said, I should be sorry if you went away with the impression that you should always be able to tell a case of coupous from one of broncho-pneumonia if you only pay attention to the points indicated above. That is certainly not the case; often you will have great difficulty in distinguishing between them. For although broncho-pneumonia may begin in scattered patches, it is apt to become confluent, and to produce dullness over the whole of one lobe. Further, some cases of broncho-pneumonia present very much the same kind of temperature as one sees in true croupous pneumonia, and may even end by crisis. I have seen it stated that in such cases the pneumococcus is the exciting cause of the disease. Whether that is true or not I cannot tell you, but I think that in future we shall **classify cases of pneumonia** in children according to the particular organism which excites them rather than by their

lobular or lobar distribution. From the point of view of prognosis, that would be a great advance, for there seems no doubt that cases which are due to the pneumococcus run a more favourable course than those which are of streptococcal origin.

Treatment

In the **treatment of broncho-pneumonia** careful nursing is of the first importance. The child should be kept quiet and free from any excitement. He should be placed in an airy, well-ventilated room, and not in a tent. The bedclothes should not be too heavy, and the child's position in bed should be perpetually changed. Care should be taken not to overfeed. Even the best nurses are apt to fall into this error sometimes. The diet should consist of diluted milk and broths, and cold water may be given freely for thirst. Linseed poultices are sometimes useful if applied to the back, but they are apt to encumber respiration too much by their weight if placed on the front of the chest. Antiphlogistine is not open to this objection, and is useful if there is pain. If the temperature is running high, I have found cold compresses useful. They are prepared by wringing a towel out of water at the temperature of the room, wrapping it loosely round the chest, and then covering it with jaconet. The compress should be changed every three or four hours. Such applications are useful in several ways. They lower the temperature; by making the child draw a deep breath when first applied, they tend to prevent collapse of parts of the lung, and they afterwards act as poultices.

Drug treatment, with the exception of the sulphonamides, already discussed, is quite secondary in importance to the measures I have just described. Chemotherapy has undoubtedly revolutionized the whole picture of broncho-pneumonia in children. Avoid the routine use of expectorants; they are only useful when there is much co-existing bronchitis, and are then to be employed according to the same rules as in that disease. If cough is very teasing, small doses of paregoric are useful. Some recommend belladonna, but I have not had much experience of it. Strychnine is of value as a general stimulant. It may be given in doses of 1 minim of the liquor every four hours to a child of one year. If there is much restlessness and a poor pulse, 10 to 20 minims of good brandy may be given in addition. In sleeplessness bromide of ammonium in 5- to 10-grain doses and $2\frac{1}{2}$-grain doses of adalin are probably the best remedies,

but 1 or 2 grains of barbitone may be used instead. If there is much cyanosis, oxygen inhalation should be used. If you use oxygen give it properly: the glass funnel held over the face in a vague manner is valueless. Oxygen should be given either through a nasal catheter or by putting the child in an oxygen tent.

When the right side of the heart is overdistended and the jugular veins full and pulsating, I have often done venesection from the jugular with distinct benefit. A few leeches along the costal margin may be used instead if there is a prejudice against venesection, but they are not so effective.

During convalescence tonics and good feeding are necessary, and care should be taken to promote complete resolution of the affected parts of the lung. Change of air is the most potent means of securing this end.

In concluding this subject, I may quote to you as a good summary of things to avoid an American doctor's [1] 'prescription for killing a baby with pneumonia':

'Crib in far corner of room with canopy over it. Steam kettle; gas-stove (leaky tubing); room at 80° F. Many gas-jets burning. Friends in the room, also the pug-dog. Chest tightly enveloped in waistcoat poultice. If child's temperature is 105° F., make a poultice thick, hot, and tight. Blanket the windows; shut the doors. If these do not do it, give coal-tar antipyretics, and wait.'

PLEURAL EFFUSION

Pleural effusions are not uncommon in children, and are apt to be overlooked. Here, again, physical signs leave one in doubt. There will be dullness over the effusion, but this need not be very intense; and not uncommonly, owing probably to the great conductivity of the child's chest, which I have already spoken of, the breath-sounds are preserved over the affected area, and may even have a feebly bronchial character. Hence, as you will readily understand, pleural effusion is not always easy to diagnose from consolidated lung, and sometimes you can only make sure of its existence by exploration. You need have the less hesitation in having recourse to this aid if you remember that in any case it is practically impossible to decide between clear fluid and pus without the use of the needle. The general symptoms will not help you, for clear effusion

[1] Dr. W. P. Northrup, *Medical Record*, N.Y., 1905, lxvii, p. 253.

is often attended by a greater degree of fever than empyema ; and, indeed, the symptoms of the latter in young children are often curiously slight. Even the presence or absence of a leucocytosis is no certain guide. It may be well for you to remember, however, that below the age of three clear effusions are rare, whereas empyema is comparatively common.

The **treatment of pleural effusion** I need not describe, as it must be conducted on exactly the same lines as in grown-up people.

TUBERCULOSIS

I have already referred to pulmonary tuberculosis in childhood, but I would again emphasize the fact that *phthisis as it occurs in the adult is very rare in children below the age of puberty.* It is true that phthisis is constantly diagnosed in children, because a slight cough is present, and the child is wasting, but the vast majority of such cases are really examples of chronic dyspepsia, with a throat cough. Patches of crepitation are also met with sometimes in the lungs, which, when they occur in the upper lobes, may closely simulate phthisis ; but they clear up completely, and are apparently due to local areas of catarrh. The condition, however, which is perhaps oftenest mistaken for phthisis in the child is—

PULMONARY FIBROSIS

This is relatively common, especially in patients of the hospital class, but it is very rarely tuberculous ; it is more often found in the left lung than in the right. In the great majority the condition dates from an attack of broncho-pneumonia in very early life, which has never properly cleared up, but has led to a condition of chronic interstitial inflammation, sometimes attended by dilatation of the bronchi and the formation of bronchiectatic cavities. The physical signs consist in dullness, usually over the lower part of the lung, with weak breath-sounds and crepitation. Here and there cavernous breathing and the occurrence of whispering pectoriloquy may indicate the existence of a cavity, and if the right base is affected, the heart may be displaced towards that side.

The X-rays are of great help in diagnosis and their use after an injection of lipiodol will determine the existence and extent of any bronchiectasis.

The symptoms are often surprisingly slight. The child may look fairly well—sometimes even blooming—but there is usually more or less cough, which varies greatly in severity at different times, and which may be attended by profuse expectoration, which is occasionally, though rarely, offensive. Attacks of inter-current bronchitis or of localized pneumonia round the dilated tubes are common, and at these times more or less fever develops.

When the condition has lasted some time, the chest may become deformed and the fingers clubbed. The ultimate fate of these patients is not often happy. A few of them certainly seem to live a long time, but many succumb to inter-current bronchitis or pneumonia, or to an attack of hæmoptysis. More rarely, secondary complications set in, such as cerebral abscess, and a certain number die in adolescence from failure of the right side of the heart much as a case of emphysema in the adult does. In a small number tuberculosis becomes secondarily grafted on the original infection, and ultimately proves fatal.

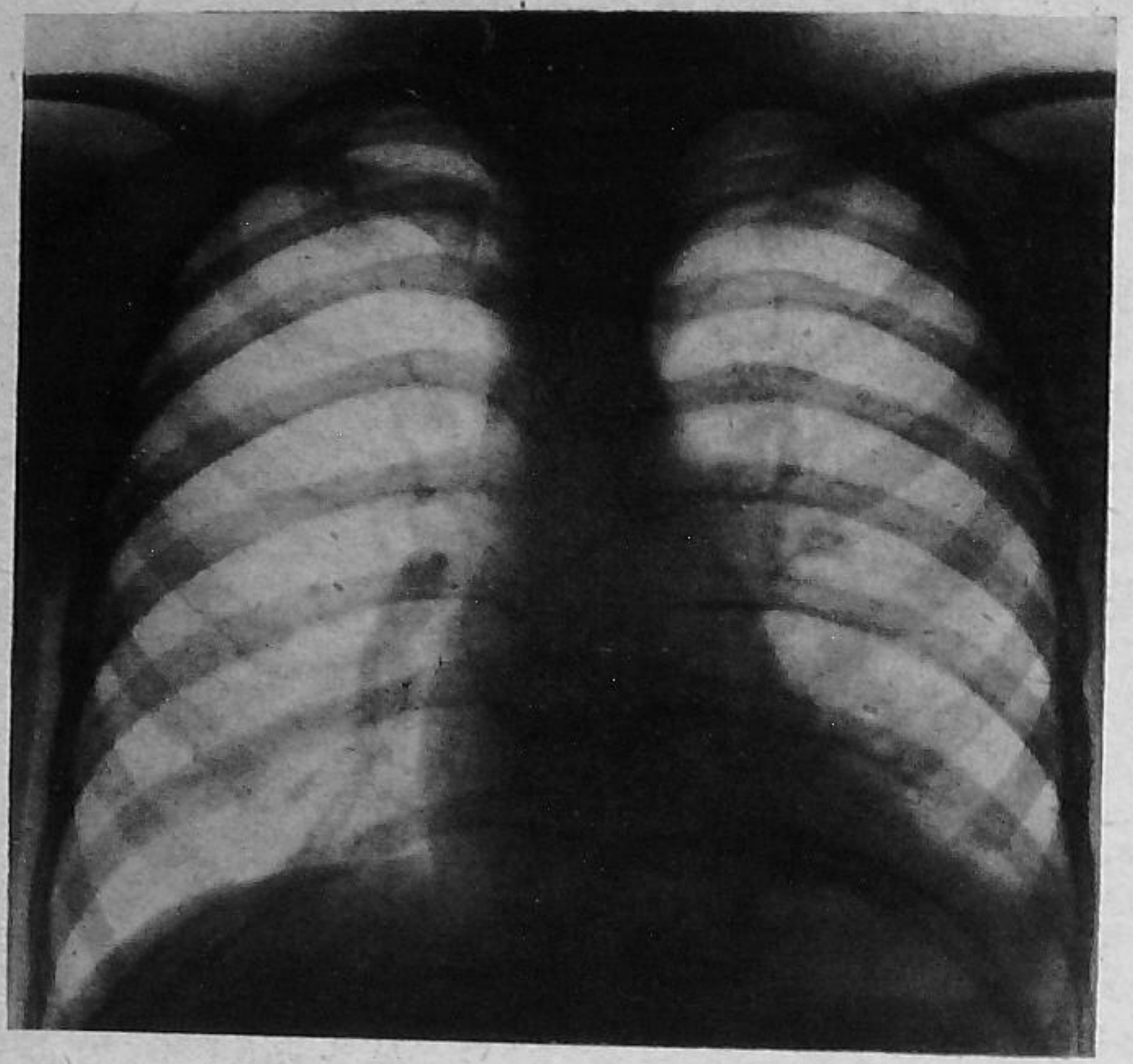
The treatment of these unfortunates is not satisfactory. If possible, one should guard them against further attacks of bronchitis by sending them to live in a warm, dry climate. When this is impossible—as it usually is—one should do all one can to maintain the general health at as high a level as possible, on much the same lines as in cases of asthma. Cod-liver oil is useful during the winter months. If there is much bronchiectasis, it is advisable to empty the tubes each morning by 'inverting' the child, and the administration of such 'pulmonary antiseptics' as creosote and guaiacol may help. Garlic, also, is a useful drug, and children usually take it readily enough. It may be given along with cod-liver oil—e.g.:

Emuls. ol. morrh. }
Syr. allii acet. } . . . . . āā ℥i.

Thrice daily after food.

I have tried vaccines in these cases without any success, the difficulty being to find the organism which is responsible for the condition. There is usually either no sputum to be obtained at all, or, if there is, it swarms with all sorts of bacteria.

The adage 'prevention is better than cure' is particularly true of this affection of the lungs, and it is of great importance to prevent the development of the condition to start with, which can best be done by ensuring complete convalescence after every attack of broncho-pneumonia, no matter how slight.



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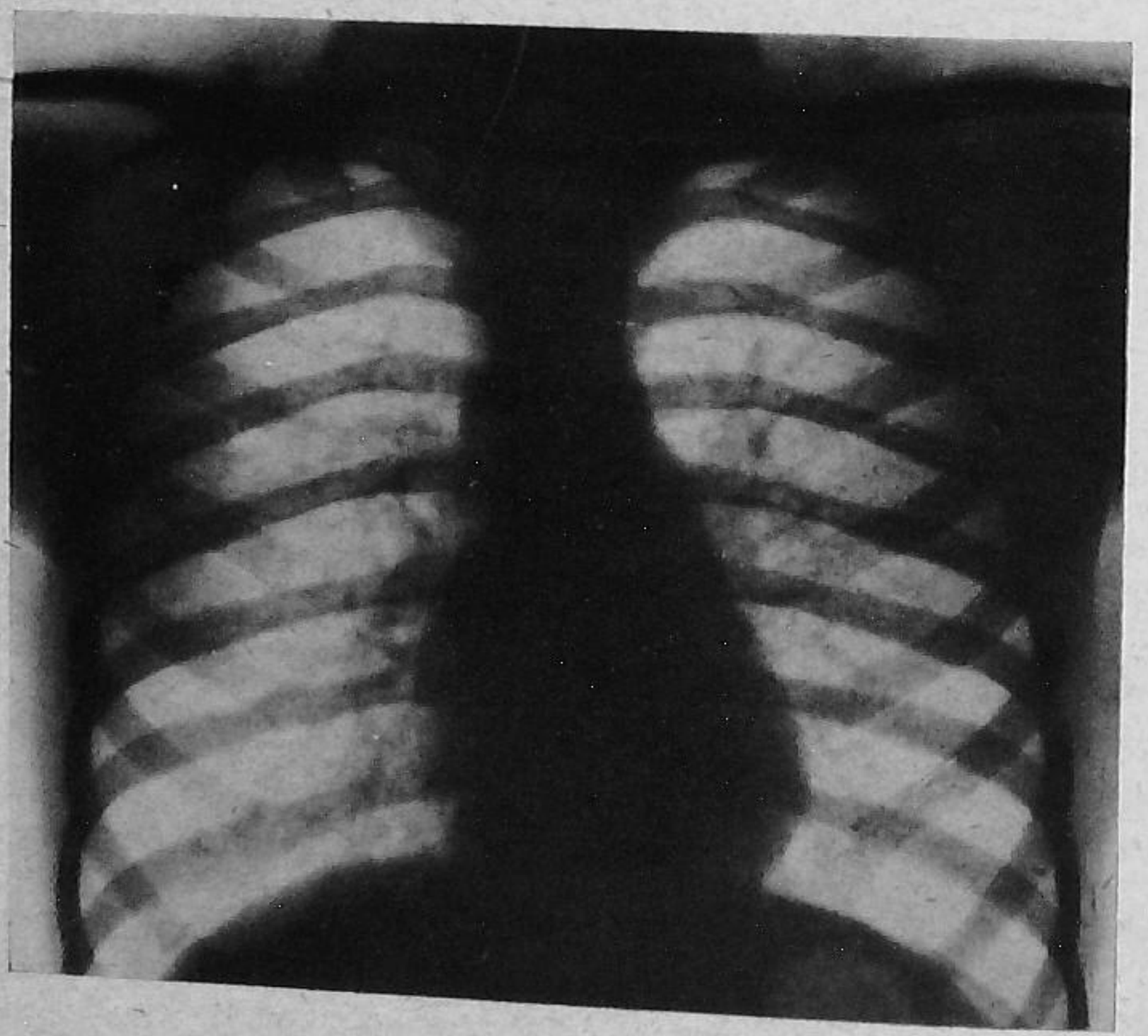

(*b*)

FIG. 54.—COLLAPSE OF LUNG.

X-ray pictures from child of 8 years with persistent cough after whooping-cough. Signs at left base and X-rays (Fig. 54*a*) show collapse there. Three weeks later signs had cleared and X-rays (Fig. 54*b*) show normal lungs.

When the affected area appears to be localized to one portion of the lung it is well worth while considering the possibility of total eradication by operation. Needless to say 'lobectomy,' complete or partial, is a serious operation, but the alternative is only too often a slow downhill course.

PULMONARY COLLAPSE

Before concluding these lectures on respiratory diseases in childhood I must say a brief word about what is termed 'collapse' of the lung. This is an important condition, not only because it may play a part in the origination of pulmonary fibrosis and bronchiectasis, but also because at times it gives rise to physical signs which are baffling. In broncho-pneumonia small areas of collapse are frequently found at post-mortem examination and they cannot be diagnosed during life. Collapse of a whole lobe or major part of a lobe also occurs. Not infrequently the child has had bronchitis and possibly a plug of thick mucus secretion is stopping up a bronchus. The cough continues and so does a slight pyrexia. Physical examination often shows very little. Occasionally there is weak air-entry at one base and the possibility of fluid is considered. X-ray examination nearly always reveals the true cause of the symptoms. Many patients with collapse of the lungs get well spontaneously as was the case in the child of whom I can show you X-ray pictures (fig. 54*a* and *b*). This can be aided by deep breathing and you can get a child to breathe deeply by making him inhale carbon dioxide from an ordinary gas bag. If the collapse persists it is wise to enlist the aid of an expert with a bronchoscope for the mucus plug can often be extracted and the lung expands at once.

LECTURE XXV

SOME BEHAVIOUR PROBLEMS IN CHILDHOOD

By behaviour problems I mean special forms of 'naughtiness,' such as excessive jealousy, obstinacy, lying, stealing, refusal to eat or refusal to sleep. I suppose that fifty years ago or even early in this century the problem of the naughty child was considered as more in the domain of the school teacher or the parish priest than in that of the medical practitioner. However, this has changed and it is nowadays no unusual thing for a child to be brought to the out-patient department of a children's hospital because of some abnormal conduct, such as stealing or excessive lying. I believe that the change in outlook dates more especially from the results of epidemic encephalitis which, as it affected children, in the early years after the war of 1914–18, was apt to result in the development of utterly unmanageable and even dangerous hooligans, the so-called 'apache' type. This brought home forcibly the fact that even juvenile criminals might be more in need of a physician than of a gaoler and nowadays all grades of abnormal behaviour may be the subject of medical consultations.

Now I believe there is a grave danger of the matter being carried too far. It is natural for children to be naughty and up to a point their characters develop and evolve through phases of natural wickedness, so to speak, just as the physical well-being of the adult emerges from a background of childish ailments. Certain schools of child psychology seem to me to have lost their sense of proportion. They even give a pseudo-scientific turn to their views and will demonstrate the biochemical and other disturbances of the angry child, for example, as facts which are expected to explain the anger. The dabblers in these matters may even be positive dangers, for to understand the behaviour problems of the child it is necessary to take a broad view and have had much experience of the ailments of the body as well as of the mind. The family doctor is far better fitted to deal with these problems than the expert psychologist in

the majority of cases. I shall therefore endeavour to set out certain aspects of the subject as are likely to occur in ordinary practice. First of all we can, I think, profitably discuss something of the

Causative Factors

I think we should first distinguish between those causes of abnormal behaviour which are fundamental and often deep-seated and those more superficial and precipitating causes which at first sight appear to be responsible. For example, under fundamental or predisposing causes I would put in the first place certain **physical factors.** A child who is naughty may be suffering from lack of sufficient sleep, from some chronic infection or even from something relatively simple such as hunger! I have seen children who seem to need more sugar than others to maintain their equilibrium and in the absence of sufficient sugar their behaviour may become abnormal—even to the extent of stealing from sweet shops! I referred in an earlier lecture to the curious mentality of the child with cœliac disease and as another example I would quote the fact that in the disorder which we shall speak of in a later lecture as cyclic vomiting the child's behaviour is often curiously abnormal for a day or so before the characteristic vomiting and ketosis begin to show themselves. Then there may be predisposing factors which are inherited. Certain families have a tradition, so to speak, of 'going off the rails' in youth and we have such proverbs as 'like father, like son.' I must confess that I find it very difficult to sort out factors of heredity from factors which are due to *parental mismanagement* from an early age and I think we must study in a little more detail the sort of mistakes that parents are likely to make. First, I think we often forget what an impressionable creature the small child is. A background of disharmony between father and mother must have a bad effect on the young child and it is not surprising that a very high proportion of delinquent children, brought before the juvenile courts, have come from what are termed 'broken homes,' where the normal father-mother-and-children sort of relationship has been missing. Next I think parents are apt to show too little or too much affection. The old saying 'love them and leave them alone' is a wise one, and children will put up with a great deal of unhappiness at school, for example, if they know that there is parental love and affection to sustain them.

Thirdly I would put defective or excessive discipline. It is absolutely impossible to get along in this world without some sort of rules and regulations and children will, for the most part, obey reasonable rules in a reasonable manner. But complete laxity, harsh discipline or injustice in matters of discipline may cause havoc to the developing mind of the child. The whole question of punishment is too big for us to discuss here but I would assert that for minor faults it has its uses and that for major faults what the child more often wants is help over some difficulty in life. Another common parental mistake is to attempt to make a perfect child; this is to miss the whole philosophy of life which should be a constant striving after something better in the future. We should help our children towards becoming healthy, balanced adults and not worry too much about the, perhaps, stormy passages of childhood which lead eventually to this end.

Next let us discuss some of the **precipitating causes** which lead to behaviour problems. Sometimes the trouble dates from a definite 'crisis' in the child's life; the arrival of a baby brother or sister, going to school or away from home, the death or removal of a parent (or even of a much adored doll or rabbit!), some shock over matters of sex or some physical illness. The child who has been 'properly brought up,' as the phrase goes, will react in a perfectly normal and balanced fashion to any or all of these events. The child who is already suffering from the effects of one of the predisposing causes we have just discussed reacts badly to a 'crisis' and develops a behaviour problem or some special form of naughtiness. When you come to deal with such a child you will have to inquire not only into the superficial and precipitating causes but also into the general background and the type of upbringing that the child has enjoyed—if that is the word.

SPECIAL TYPES OF BEHAVIOUR PROBLEMS

I think it will help us now to translate some of these generalities into more concrete facts about the type of naughty child that may be brought to you. First of all there is the common problem to-day of the **solitary child**, whose troubles are so characteristic as to lead to the diagnostic label of 'only-childism.'

I need hardly point out, however, that the victim of 'only-childism' is not of necessity an only child. Any child who is

brought up alone amongst grown-up people may suffer from it. In the majority of instances, it is true, the child is the only one in the family, but you will also find well-marked examples amongst children who are what is called 'after-thoughts'—that is, who have been born some years after their brothers and sisters—and the most pronounced examples of all, perhaps, occur amongst children who are brought up alone by grandparents. The adopted child, curiously enough, seems to be comparatively immune to the disease. Nor is it inevitable that *every* solitary child should fall a victim to only-childism. Some, possibly most, are so happily constituted by nature or are so fortunate in the character of their parents that they escape the disorder altogether and grow up just like ordinary children.

In order to understand the pathology of only-childism, let us consider for a moment the peculiarities of the environment of the solitary child. A child so situated is constantly associating with grown-up people. It is therefore, as it were, standing always intellectually on tip-toe, reaching up to ideas and conceptions which are really beyond it. The result is, on the mental side, the development of precocity and what is termed 'old-fashionedness,' and on the physical side nervous strain with all its concomitants. Almost inevitably, too, the solitary child has too much attention paid to it. It is apt to get to feel that it is the most important person in the house and the pivot around which the whole domestic world revolves. It thus acquires a liking for being in the limelight and the centre of the stage, and will resort to any devices, some of them quite pathological, rather than allow itself to be thrown even for a moment into the shade.

Further, as the parents or guardians of the solitary child have all their eggs in one basket, they naturally enough tend to fuss over the basket to an undue degree. There results from this an extreme anxiety about the child's health. Every little ailment is magnified, and the resulting atmosphere of apprehension is communicated to the child himself. Matters are made worse by the habit, so frequent with such parents, of discussing the child's health in his presence, with the result that he becomes in time a veritable little hypochondriac.

With all this it would be a complete mistake to suppose that the 'only' child and the 'spoilt' child are synonymous terms. The mother will often, and rightly, repudiate with indignation the

suggestion that her child is spoilt, and you must be careful in practice not to seem to imply that such is the view you take of the case. For the patient is probably not over-indulged; he is only over-studied, which is quite a different thing. As a matter of fact, the mother of the only child is often over-conscientious in her discharge of her maternal duties. She is the sort of mother who studies books on mothercraft, who belongs to parents' unions, and who often keeps a progress book in which every stage in the physical and mental development of the child is noted down day by day. The only thing she lacks, in fact, is a saving sense of humour. Like that rather priggish character, Wordsworth's Happy Warrior, she also makes her child's 'moral being her prime care,' and is for ever correcting, exhorting, and forbidding. The result is that she induces in the child what plain people term 'cussedness' or 'contrariness,' but what it is now the fashion to speak of as '**negativism**.' This is a state of mind in which the child has an uncontrollable tendency to do what he ought not to do, and to leave undone what he ought to do, with the result that there is no health left in him. It is this mental condition which is the explanation of the refusal to take food, the refusal to sleep, the refusal to defæcate, and all the other 'refusals' which are characteristic of the solitary child.

You will see, then, that only-childism is a complex condition made up of physical, mental, and moral ingredients. Physically the child who suffers from it is thin, restless, and nervous. He eats, digests, and sleeps badly and tires easily. Mentally he is excitable, emotional, precocious, introspective, and hypochondriacal; morally he is disobedient, troublesome, and often ill-tempered and 'impossible.'

Any one of the components of the clinical picture may be unduly prominent, and the child brought to you for that as the presenting symptom. Thus it may be the thinness that excites the mother's alarm and makes her fear tuberculosis, or it may be 'nervousness' or some disturbance of digestion or sleep that causes her to seek advice. Very often it is one of the aspects of 'negativism' which will lead to your being consulted—oftenest of all, in my experience, the refusal of food, resembling the hysterical anorexia of young women. It may begin quite early when an attempt is first made to wean the child from the bottle or breast. When solids are given, the child hawks and retches and spits them out; whilst if liquids

are given otherwise than by suction, he gargles with them and brings them back. If the child is older when the anorexia first sets in, it will be found that he begins to play with his food instead of eating it. Soon mealtimes become a perfect nightmare for the mother and nurse, and all kinds of attempts are made by coaxing, bribing, and distracting his attention to induce him to eat, but without success. Curiously enough, such a child will often take his food quite well from a stranger or if other children are present, although he will refuse it absolutely from the mother or his usual nurse.

Another curious fact with which I have been struck about these cases is that in spite of apparently eating almost nothing the child often remains in quite a fair state of nutrition. Whether this is due to their consuming food on the sly I do not know, but this explanation seems hardly likely in many of them.

Closely akin to the refusal of food is the refusal to defæcate, in which case the child will be brought to you for 'constipation.' 'Breath-holding' is another trick often met with in the solitary child, but it is difficult to say whether it is another aspect of 'negativism' or whether it is an expression of the desire to be conspicuous and interesting. **Hysteria** is another type of abnormal behaviour in childhood in which the precipitating cause is frequently some form of physical injury or some sudden fright. Hysteria may assume various forms, the commonest, perhaps, being affections of the motor system, which may show themselves as paralysis, fits, spasms, or tremors. And, first, let me speak of **hysterical paralysis.** Of the different forms of hysterical paralysis in children, monoplegias and paraplegias are the commonest. Hemiplegias are less frequent. The paralysis is often of sudden onset, and it usually assumes the flaccid or limp type. Hence the case is apt to be mistaken for one of infantile paralysis, as happened in the following example:

Case 1.—A boy, aged twelve years, was brought up from the country with a history of paralysis of the right arm, which had come on suddenly five months before. The paralysis had been regarded as due to poliomyelitis. On examination, there was complete flaccid paralysis of the right arm, but with only a moderate amount of wasting. There was anæsthesia of 'sleeve' distribution up to the shoulder. Otherwise he seemed normal. Under the application of faradism, accompanied by vigorous suggestion, he immediately recovered the use of the limb, and returned home next day cured.

I have not myself seen a case of hysterical paraplegia in a child, but those who are interested in the subject will find a very instructive example of it, which led to various errors in diagnosis, recorded by Dr. C. W. Chapman in the fifth volume of the *Transactions* of the Society for the Study of Disease in Children.

Hemiplegias are comparatively rare, and when they do occur the face usually escapes. The following is an example :

Case 2.—Susan C——, aged twelve years, got a severe fright at a fire two years ago, and has had several ' hysterical ' fits since. After one of these lately she lost the use of the right arm and leg. The arm

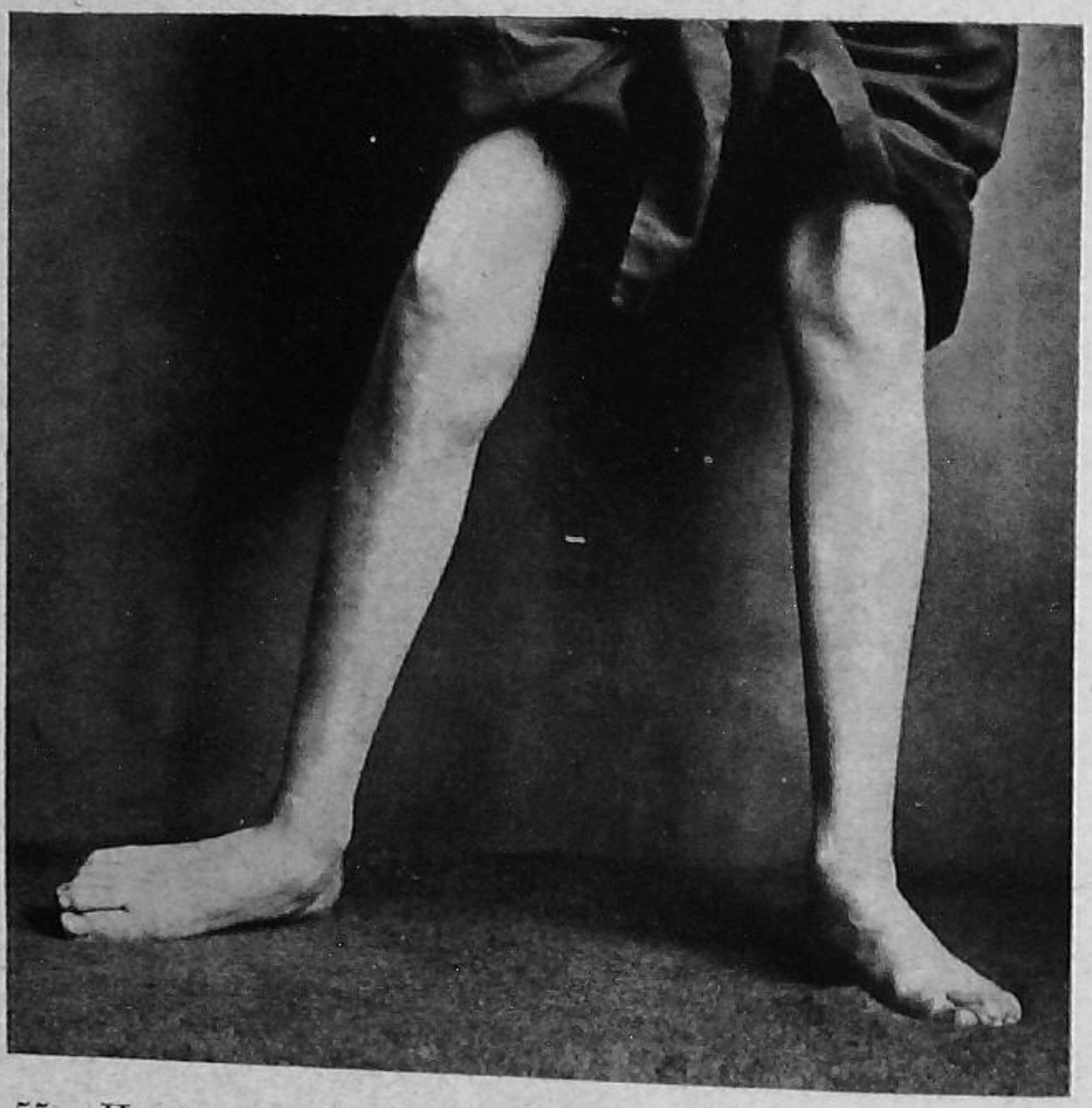

Fig. 55.—Hysterical Paralysis in Right Leg, showing how the Foot is Dragged.

quickly recovered, but the right leg remained weak, the foot being bent in the equinovarus position, and dragged along as she walked (fig. 55).

Examination showed anæsthesia over the whole of the right half of the body, with amblyopia in the right eye and considerable areas of anaglesia in the leg, and her mother said that she would sometimes refuse all food for two days on end.

She only came once or twice to the hospital, and her subsequent history is unknown.

The curious condition called ' hysterical astasia-abasia '—inability to stand or walk—seems also to occur sometimes in children, but not

commonly. Usually I think it is left behind as the result of a condition which at the outset does really make standing or walking painful, and then, after that has passed off, the astasia-abasia persists, owing to the hysterical tendency.

Passing from hysterical paralysis, we come to **hysterical fits.** They are, as you know, a common manifestation of hysteria in the adult, the typical case being that of a young woman who gets an 'hysterical attack' in which she throws herself about, cries, laughs, screams, etc. That type of hysterical fit is not common in children, although I shall have occasion later on to mention examples which might be referred to it. Much more often an hysterical fit in early life closely simulates epilepsy, and sometimes the simulation is so close that it requires great care to distinguish one condition from the other. I can give you two remarkable examples:

Case 3.—Arnold H——, aged fourteen years. About five weeks before he came under observation this boy fell down in the school playground in a 'fit,' from which he speedily recovered; but since that time he had been subject to right-sided convulsions, which were getting more frequent. For the last two days he had not spoken.

He had had an attack of rheumatic fever at the age of ten, which had left him with a mitral lesion, but otherwise he had enjoyed good health. His parents were both said to be 'nervous.' There were four other healthy children.

He was a bright, healthy-looking boy, but exhibited complete 'mutism.' The fits recurred at intervals of a few minutes, and consisted in slight twitching of the right arm, leg, and face. The attack began with a slight grunt, and ended with a sigh. He was partially unconscious in the attack, but the conjunctival reflex was not entirely abolished. The eyes showed conjugate deviation in the attack, usually to the right, but sometimes to the left. The pupils were equal. He never bit his tongue nor passed water in a fit. There was slight weakness of the right arm, leg, and face, and the knee-jerk on the right side was not so easily obtained as on the left. There was no anæsthesia, and the fundi were healthy. He readily obeyed commands, but wrote with great difficulty, and seemed to forget words and how to spell. He did not appear able to read print.

Under isolation with the use of faradism, suggestion, and cold shower-baths he quickly regained his speech, the fits stopped, and he left hospital quite well about three weeks after his admission.

Case 4.—William W——, aged fifteen years, was seen in 1906 for 'fits,' which had begun three days previously. His mother had suffered from some sort of 'fits,' but the patient himself had always been healthy. The fits in his case had begun with 'stiffness of the jaw,' after which he was said to have lost consciousness. The fits had since

been repeated nearly every quarter of an hour. He had never bitten his tongue in them, but once at least he had passed water during an attack. For the last twenty-four hours he 'had not spoken.'

On examination he seemed placid and intelligent, obeying commands readily, but quite mute. When a fit came on, the right side of the face twitched violently, and the hands were clenched, but showed no clonic spasm. There was no loss of consciousness, and the conjunctival reflex was not abolished. The pupils were dilated, the knee-jerks normal, and the fundi healthy. There was apparently no anæsthesia.

The fit lasted about a minute, and was repeated at short intervals.

He was admitted to hospital, and after about ten days recovered his speech, and the fits ceased.

In hysterical fits of this type I think mutism is a symptom of great diagnostic value. In both these cases its presence clinched the diagnosis.

Passing from fits, one comes to hysterical **contractures of joints.** These are rather common manifestations of hysteria in children, and I have seen several examples of them. They often follow injury of the joint, which has caused pain and led to some degree of flexion, and that, owing to the hysterical basis, has become perpetuated and exaggerated, so that there is extreme spasm (figs. 8, 56 and 57). It is important in cases of hysterical contracture not to overlook the fact that there may be a real organic basis for the condition. A child, for instance, may have commencing tuberculous disease of a joint, or there may be some other disease of it, but, instead of responding with the ordinary stiffness and slight flexion, there is this exaggerated response, and you must beware in such a case of attributing the whole condition to hysteria.

The last motor manifestation I have to mention is **hysterical tremor.** There are various forms of such tremor. You may have a true intention tremor, which comes on only on attempts to use the affected part. That happened in the case of a boy, aged ten years, who had tremor of the hand as soon as he tried to write, although he used it for other purposes quite well. Sometimes the tremor consists in a series of clonic movements confined to one group of muscles—a repeated clonic spasm, and I shall have occasion to describe to you immediately some cases affecting the respiratory system, which, I think, fall under this group. It is a characteristic feature of such hysterical spasms that they tend to stop during sleep.

Hysteria in childhood may also produce various **sensory** manifestations with either anæsthesia or hyperæsthesia. Other dis-

turbances include the hysterical cough, the hysterical cry, hysterical mutism, hysterical dyspnœa and even some forms of blepharospasm, ptosis and diplopia may be put in the same category.

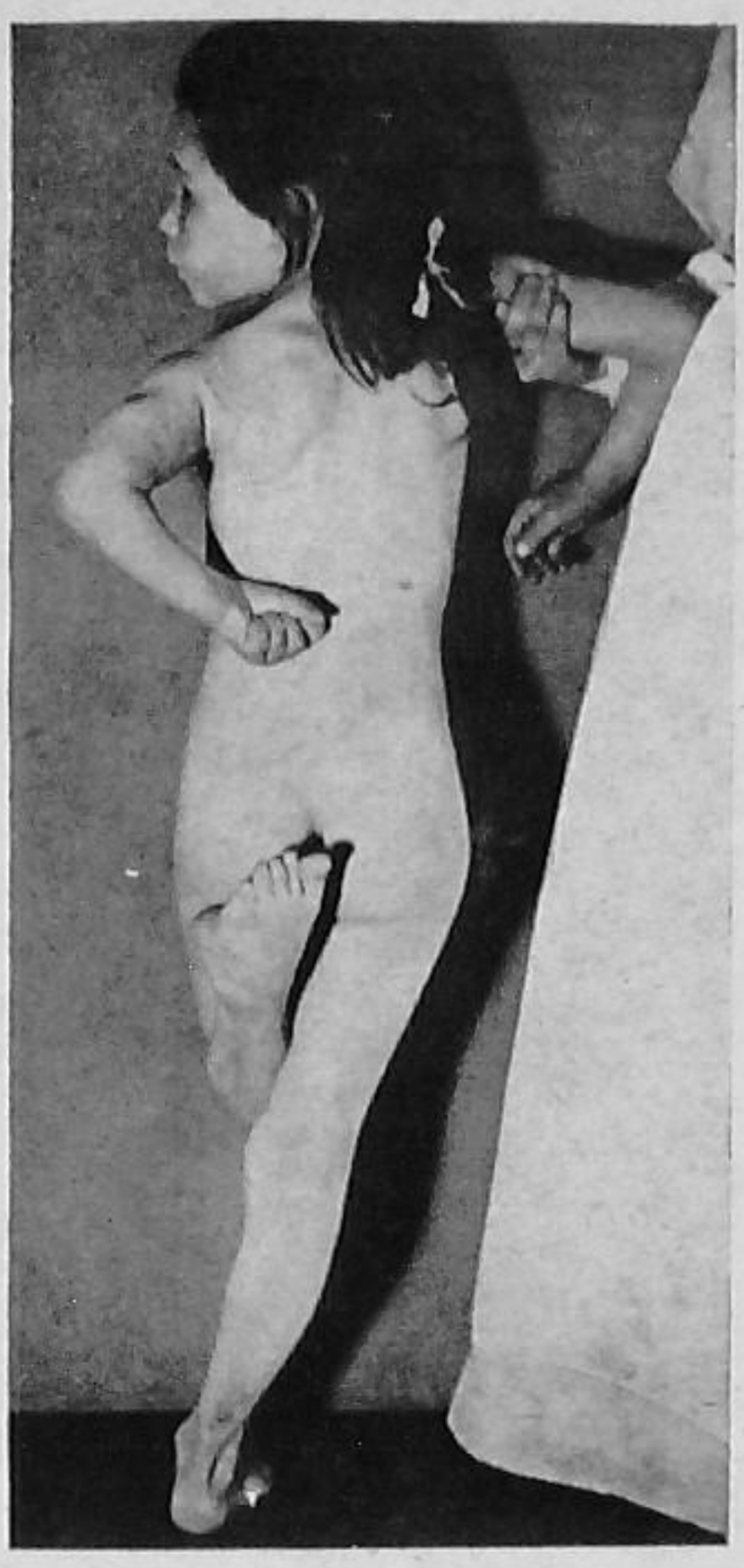

FIG. 56.—HYSTERICAL SPASM OF LEFT LEG.

FIG. 57.—HYSTERICAL SPASM OF HANDS.

Disorders of conduct represent another important group of behaviour problems which may be briefly mentioned, for we have already touched upon them in different ways. Lying, stealing, dishonesty, cruelty, disobedience, truancy may each bring the child into conflict with authority and sometimes, but by no means always, it is possible to detect the same sort of mixture of predisposing and precipitating causative factors as with the disorders exhibited by the solitary child, the negative child or the child with hysteria. There are border-line cases in which the child is found to be mentally defective or eventually turns out to be suffering from the early stages of a true psychosis.

DIAGNOSIS

The general diagnosis of behaviour problems is made by a broad survey of the situation. We must first make a careful physical

examination—often a difficult matter in a 'negative child'—and we must observe carefully the sort of relationship which exists between the mother or guardian who brings the child and the child itself. Sometimes it is helpful to interview other members of the household and you should also attempt to make friends with the child. Gradually, and by no means always at the first interview, the situation begins to become clearer. Sometimes you can see the child alone and encourage conversation. If you have doubts about the intelligence a report from the school teacher may prove helpful. The diagnosis of hysteria presents special difficulties. Organic disease of the nervous system has to be carefully excluded. Bizarre and exaggerated symptoms appear out of proportion to the superficial causative factors and there is often a curious inconsistency of the symptoms with themselves.

Treatment

In some instances a cure is effected once the mother and child can be made to appreciate what has gone wrong in their relationship, but it is seldom as simple as this. With the only child the obvious way out of your difficulties, of course, is to abolish the solitariness, but this is more easily said than done. Failing anything better, a few hours spent daily at a kindergarten, day nursery or nursery school is of some use in little children. This insures that at least for a time the patient has to take his place on an equality with other children, even though when at home he is still the centre of the picture. The outside interest which school brings into the self-centred child's life is also of curative value. For older children, and if the parents can afford it, and, what is equally important, will give their consent, a boarding school is an unfailing remedy. It at once abolishes the solitariness, and there is no longer any excuse for negativism, for discipline at school, unlike that in the home, is automatic and impersonal; all come under it alike, and it does not arouse a sense of personal grievance as it does when applied to the individual. Mothers of only children are always afraid that their child will never get on at a boarding school—'he is too nervous' or 'he will be too home-sick.' I can only say that I have hardly ever found it so, but that in the great majority of cases the child soon settles down comfortably, and his health and happiness are immeasurably improved. One term's experience is usually enough to convert the most recalcitrant parent. If the child is not old

enough for the ordinary preparatory school, a 'nursery school' in the country, of which there are now many in existence, answers the purpose equally well.

In the case of particular manifestations of 'negativism,' such as the refusal of food, of defæcation, or of sleep, even a short stay away from home or merely handing the child over for a time to the care of a strange nurse may suffice to abolish the trouble. I recall, for example, the case of a little girl, the only child of a country vicar, whose mother looked after her entirely herself, and who suffered from pronounced anorexia. I proposed to the mother that a trained nurse should be put in charge of the child, but was met with a blank refusal. Shortly afterwards the mother, worn out with vain efforts to get the child to eat, had a 'breakdown'; a nurse went to take care of her, and incidentally took on also the feeding of the child, with the result that in a fortnight the difficulty with the feeding had quite disappeared.

Should you be unable, as you often will, to get the child sent from home, you must do your best to change the whole mental environment in which it lives and moves and has its being. If the essential nature of only-childism is explained to the parents, and if it is pointed out to them that it is to some degree an inevitable result of circumstances, you will not usually have much difficulty. The doctrine of 'wholesome neglect' must be inculcated, the child must no longer be fussed over physically, nor his health and peculiarities discussed in his presence; he must cease to be the most important person in the house, and all manifestations of negativism steadily ignored—not punished, expostulated with, or wept over, but ignored. The parents must also remember the advice given to the young man by one of the characters in *Kidnapped*: 'Be souple in things immaterial'—in other words, do not fuss over and correct trifling aberrations. A steady persistence in this attitude will usually bring its reward.

When the only child is the victim of serious disease, the best place is a nursing-home or hospital. There he is usually as meek as a lamb. Failing that, a sensible trained nurse may soon work wonders in changing his disposition.

In the treatment of hysteria the first and most essential point is *isolation*. Get the patient into a home or hospital. Isolation is important, because it takes the child out of the unwholesome environment in which he has often been living, and that alone may

be sufficient to effect a cure, as in some of the instances I have mentioned. Next, ignore, as far as you can, the condition of the child. Repeated examination, demonstrations to students, and so forth, are bad for the patient. Wholesome neglect is the best attitude to adopt, and the doctor and nurse should not be over-sympathetic, though, of course, the child should be treated with the glove of silk on the iron hand. In dealing with specific symptoms, what has been called the ' method of surprise ' is good—that is to say, taking the patient unawares. In a case of astasia-abasia, for instance, put the child suddenly on its feet and tell it to walk. Sometimes in those circumstances the patient is taken aback and does walk, and then you have no trouble with it afterwards. The objection to the method is that if the child is not successfully taken by surprise you have not only failed to effect an immediate cure, but you have done much to perpetuate that symptom, and to make any subsequent treatment more difficult. But the manœuvre often comes off. ' Suggestion ' is of great help—suggesting, for instance, that the child can move the limb, or that the pain has gone away, or that it can straighten out an affected joint, etc. With regard to other aids, I still believe in the old-fashioned treatment by cold shower-baths. Drugs, such as valerian, are perhaps helpful, and in some cases tonics may be necessary—iron, arsenic, and strychnine—so as to get the general health into good condition. It is extremely important, also, to see that the convalescence is made complete, and that the child is kept away from home for a considerable time until it is well out of the hysterical habit, as otherwise there is apt to be a relapse, and that is usually more difficult to cure than the original attack.

LECTURE XXVI

SOME FUNCTIONAL NERVOUS DISEASES OF CHILDHOOD

In my last lecture I spoke to you of various disturbances of conduct and behaviour, and to-day I want to continue the subject on a minor plane, so to speak. There are functional disturbances of the nervous system in early life which arise from parental mismanagement and so on, just as we discussed last time but in addition to, or I might say in place of, these predisposing causes there are others which have to be considered. At one end of the scale we have tetany and laryngimus stridulous which some people would accept as purely biochemical in origin, and at the other such disturbances as enuresis and habit spasms which may have some sort of organic basis. Whatever the grouping, you will be struck in practice by the frequency with which functional disease of the nervous system exhibits itself in your younger patients. One reason for this, no doubt, is the comparative **instability of the nervous system** in early life. It is a nervous system which is, so to speak, still in the making; it has not settled down into the stereotyped form characteristic of mature life. The higher centres are as yet imperfectly developed, the lower incompletely controlled, and the paths of nervous impulses not clearly laid down. Hence abnormal forms of nervous action, manifesting themselves chiefly in defective co-ordination, spasmodic muscular contraction, and unchecked reflex excitability, readily become established. Several factors may come into play in rendering such abnormal action easier. First of these is a **nervous heredity.** Some babies are obviously ' neurotic ' from the moment of their birth. They are easily frightened, and start and tremble at any sudden noise or unaccustomed sight. Such children, you can readily believe, will easily become the subjects of functional disease of the nervous system. So often these children grow up into ' nervous ' individuals, thin, ' highly strung ' as the saying goes, ' live wires ' as others call them. Their excessive nervous energy often

drives them to great achievements and they are of great value to the community—but often uncomfortable to live with! Many believe them to be a product of modern civilization with its great speeding up of life. If you want to know more about this type let me recommend you to read Dr. H. C. Cameron's book on *The Nervous Child*.

Equally important in early life is the presence of rickets, for there is little doubt that a faulty calcium metabolism means an irritable nervous system, even if the full-blown picture of tetany does not appear.

Another point I want to insist upon before I proceed to speak of the different neuroses in detail is the question of **inco-ordination.** When you study those nervous disorders more closely you will find that what you have to deal with in nearly every one of them is an inco-ordinate action of the nerve-centres. It need not surprise you that a lack of co-ordination forms the basis of most of these neuroses, because, as you know, a baby is to a large extent an inco-ordinate machine; at all events, it has not yet learnt to harmonize many of its movements properly. The infant, for example, has not yet learnt to co-ordinate and control the action of the expelling and sphincter muscles of the bladder and rectum; nor has he learnt to control properly the muscles which hold up the head. And not even by the time he is a year old has he acquired sufficient co-ordinating power to enable him to walk. So you need not be surprised that a mechanism which has been but recently and imperfectly acquired should be readily thrown out of gear, and that inco-ordination should be a striking feature of most forms of functional nervous diseases in childhood.

FACIAL IRRITABILITY

I want now to pass to the study of some of those nervous disorders in detail. And first of all I wish to mention *facial irritability*, or Chvostek's sign. This is not a disease, but a symptom. It is a danger-signal by which you recognize the tendency on the part of the child to suffer from more serious forms of nervous disorder.

Facial irritability is so called because when you tap over the facial nerve, below the malar bone, you get a contraction of the facial muscles. The phenomenon is not peculiar to the face, for if you tap almost any exposed nerve you get a similar muscular contraction. In other words, there is an exaggerated excitability of

the nerves to mechanical stimuli as the basis of Chvostek's sign. If that sign is present you know that the nervous system is in an irritable and unstable condition, and will be prone to be affected by convulsive and spasmodic disorders.

LARYNGISMUS STRIDULUS

Let us take next *laryngismus stridulus*. That is a serious and sometimes fatal form of nervous disease, which consists in a spasmodic closure of the glottis, rendering the child for the time being incapable of breathing. You will almost invariably find that the child is suffering from rickets, that great predisposer to nerve disease, and you will get the history that from time to time the child stops breathing, throws back the head, becomes blue in the face, and then suddenly emits a crowing sound, after which he may cry a little as if frightened, but soon returns to his toys.

There is here, then, a period of apnœa—that is to say, cessation of breathing—followed by a crowing inspiration; these are the characteristic signs of laryngismus stridulus. The attacks come on more in the night and in the early morning than at any other time, and they may easily be excited by any external irritation, by laughing, tickling of the throat, a draught of air blowing across the child's face when asleep, or by any emotional disturbance. At first the attacks occur only at rare intervals, but by and by they become more frequent, so that the child may have a great many seizures in the course of the day.

For what may you mistake laryngismus? First of all, you might mistake it for *congenital laryngeal stridor*. I shall speak of that condition more fully soon. Meantime I will only say that congenital laryngeal stridor dates from birth, whereas laryngismus stridulus usually affects children who are about eighteen months old. So that proper attention to the history alone will keep you right in the matter of diagnosing between the two conditions. Next you may mistake it for *croup*, the catarrhal laryngeal spasm of which I have already spoken; but croup attacks last for several hours, whereas laryngismus stridulus is almost momentary. Next you might conceivably mistake it for *whooping-cough*; but whooping-cough is characterized by a series of short expirations followed by a whooping inspiration, whereas laryngismus stridulus is characterized by a period of apnœa followed by inspiration.

Laryngismus may be a serious disease, as I have said, and children sometimes die in an attack, so do not be too confident in your **prognosis.** The story is told of a consultant who was summoned to see a case of this disease. He said there need be no alarm, as these children practically always got well; but as he was going out of the house he was called back to find that the child was already dead.

We now come to **treatment.** First of all, how are you to treat the attack? What you have to do is to try to promote the taking of an inspiration, and this you can best do by dashing cold water on the child's face, or by tickling the fauces. If the spasm lasts an alarmingly long time, give a whiff of chloroform. That will generally cause the spasm to pass off and re-establish respiration. The application of a hot sponge over the larynx is also of use.

Next, how are you to ward off future attacks? A great deal can be done by keeping the child quiet. It must not be annoyed in any way; it must not, as someone has said, be 'tickled, tossed, or teased,' because emotional disturbance and physical stimulation are exciting causes of attacks. It is well, also, to calm down the nervous system. The sedatives which you employ—and I shall have occasion to speak of them repeatedly—are bromides, chloral, and antipyrin. These three drugs are very useful in all the functional nervous disorders of childhood, and children stand all of them well, particularly chloral, which you can always give boldly. Seeing that rickets is the great predisposer to those attacks, I need hardly tell you how important it is to treat the rickets. That is never to be neglected. It is absolutely necessary to recognize the rickety element and to treat it by a change of diet, by cod-liver oil, or by a vitamin D concentrate; and it is only in order to give those measures time to take effect that you give sedatives in the meantime.

CONGENITAL LARYNGEAL STRIDOR

I wish next to consider briefly the condition called *congenital laryngeal stridor*, which I told you might be mistaken for laryngismus stridulus. It is a disorder due to a sucking in of the arytænoepiglottidean folds into the narrow glottis during inspiration, but whether as the result of inco-ordination or mal-development or of an exaggeration of the natural incurving of the infantile epiglottis is uncertain.

What are its **symptoms?** The child will be brought to you

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when quite a young infant, usually only a few weeks old, with the complaint that it makes a curious noise on breathing; and you will be able to hear a slight crowing or purring going on in the throat, similar to the noise which you may hear a hen or a cat make. This sound goes on more or less continuously, but is louder if the child gets excited or agitated. During sleep it is almost inaudible. Laryngismus stridulus, on the other hand, is not congenital, and it is essentially discontinuous. One other thing which you may mistake it for is adenoids in their congenital form. It is also possible to have a similar stridor with an enlarged thymus, so have an X-ray picture made of the chest (see p. 364). Inasmuch as congenital laryngeal stridor tends to disappear towards the end of the first year it is not necessary to pursue any active **treatment,** but it is advisable to guard the infant as far as one can from any risk of respiratory infection.

TETANY

The next functional disorder of infants is the condition termed *tetany*, or *carpopedal spasm*, a name which describes the leading feature of the disease—namely, a spasmodic contraction of the flexor muscles in the front of the fore-arm and the back of the leg. You find the wrist flexed, and, as the interossei also participate in the spasm, there is flexion of the first phalanx and extension of the other two. In addition the thumb is often tucked into the palm, so that the hand assumes a conical shape, which has been described as the ' obstetric position,' because that is the position in which the obstetrician puts his hand when inserting it into the vagina. You may also find a degree of œdema on the dorsum of the hand, and that the skin looks pink or purple and somewhat glazed, and that on handling it it seems to be tender. What this is due to I do not know. It may be produced mechanically by the continuous spasmodic contraction of the muscles interfering with the proper return of the blood.

The position of the feet resembles that of the hands. The toes are flexed and curled into the soles, the heels drawn up, and the dorsum of the foot is often somewhat puffy. If you examine the electrical reaction of the affected muscles, you find that their excitability is increased, and the anodal closing contraction is greater than the cathodal.

The **predisposing conditions** of tetany are two. In children of about eighteen months old it is commonly associated with rickets as one of the manifestations of so-called spasmophilia. In younger infants you will find it coming on after an attack of diarrhœa. It is also sometimes met with in association with cœliac disease. The spasm is apparently due to deficiency of calcium in the blood, probably the consequence of an insufficient supply of vitamin D. Sometimes you meet with a similar disease in grown-up people. It is one of the curious and rare complications of dilated stomach, and results from alkalosis brought about by the excessive vomiting of

FIG. 58.—POSITION OF HAND IN TETANY.

acid. It may appear occasionally in a case of pyloric stenosis in infancy for a similar reason. Tetany is not a fatal disorder, but it is a painful one. In treatment you must first of all deal with the rickets, if there is any, and, whilst your antirachitic treatment is taking effect, you must administer nerve sedatives. Of those I would recommend the bromides and chloral in preference to antipyrin. Calcium lactate is also strongly recommended in such cases in doses of 15–30 grains every four hours. Cod-liver oil or some other source of vitamin D should be given at the same time. Hot baths are useful, for they tend to relax the spasm when it is very severe. Exposure to the ultra-violet rays has also a beneficial effect.

HEAD NODDING

I want to speak next of a disorder which is of considerable interest and rarity, and that is *head nodding* or *spasmus nutans*. In it you have to deal with slight continuous movements of the head, which occur in children who are usually about a year old or a little less. Sometimes, the movement is up and down, sometimes it is from side to side—a sort of pendulum movement—and sometimes a combination of those forms in different degrees. The essential point is a slight more or less continuous and apparently involuntary movement of the head. When you come to examine these patients more carefully you will find that a majority of them suffer from **nystagmus** also. The nystagmus may be from side to side, or it may be vertical or of the rotatary form, or it may be a combination of these varieties. And you may find in such a case a form of nystagmus which you do not find anywhere else—that is to say a form in which the eyes tend to jerk towards one another and then to fly apart. In most cases of nystagmus, as you probably know, the axes of the eyes remain parallel, but to this form the term 'convergent nystagmus' is applied.

Here, again, it is believed you have to deal with an inco-ordination of the eye and head movements. The young child has just learnt to co-ordinate the movements of the eyes and head; he is just learning to turn his head in the direction in which he wants to look, and a very slight disorder may temporarily derange that co-ordinating mechanism. It has been compared to miners' nystagmus for a reason which I shall tell you of immediately. The interesting point which connects the movements of the eyes and those of the head is that the movements of the head tend specially to come on when the child tries to fix anything with his eyes. If you persuade the child to look steadily at you, you will find that the nystagmus increases and the head begins to nod. If the nystagmus is present in one eye only, the movements sometimes cease when that eye is covered up.

Head-nodding tends to occur with marked frequency at one part of the year—namely, in mid-winter. Why is it almost confined to this particular part of the year? An explanation which has been advanced is that it is due to the absence of sunshine at this time. Some of you who saw a recent example of the disease in the out-patient room will remember that the mother admitted that the

child lived in a dark room on the ground-floor ; and it is in such conditions that the disease most commonly appears. That is why this form of nystagmus has been compared with that known as miners' nystagmus. Now, although that explanation is ingenious, it is not universally accepted, and in many cases it is impossible to find that the children who are the subjects of the condition have lived in dark houses ; some of my cases have occurred in the suburbs or in the country, where there is as much light as can be met with in this country in the winter.

That brings us to speak of the **diagnosis.** The only condition for which you are likely to mistake head nodding is *salaam convulsions*, which consist essentially of swaying or nodding movements of the body, which appear to be epileptic in their nature, one of the forms of *petit mal.* There are also certain automatic movements which idiots are apt to make which you may mistake for head nodding, such as swaying movements, banging of the head, or knocking of it against the pillow ; but all these movements are much more gross, and are larger in their range, than the typical head nodding.

The **prognosis** of this disorder is perfectly good. If you leave it alone it will always get well in time. Therefore you can reassure the mother. Sometimes, however, the nystagmus seems to persist after the head nodding has passed off ; and it is possible that some cases of nystagmus which are alleged to have begun in childhood, and for which no cause is assigned, have been cases of nystagmus with head nodding in which the eye movements have persisted.

Treatment consists in attention to the general health and in dealing with rickets, if present, on the ordinary lines. Small doses of antipyrin—1 grain for every year of life—and bromides are said to be helpful.

Somewhat akin to head nodding, but clearly to be distinguished from it, is the condition known as **head banging** which consists in a violent banging of the head against any solid object that comes handy—the floor, the wall, the side of the cot, the pillow, and so on. It usually occurs in children of about two years of age, and the violence of the bangs may be such as seriously to alarm the mother for the child's safety. I have never known one, however, do itself any real injury, though occasionally a little bruising may result. What satisfaction the child finds in this exercise it is

difficult to see, but the probability is that it is performed in order to relieve some uncomfortable sensation in the head, such as ear-ache or the pain of teething. No special treatment is required, though it is well to see that the child does not do himself any injury. A course of bromides and antipyrin may be tried in bad cases.

NIGHT TERRORS

I now pass to another extremely important group of nervous disorders, those which go by the name of *night terrors*, or *pavor nocturnus*. Night terrors of varying severity are not uncommon in children, and they are often very alarming to the friends. The history is that the child, usually about the period of the second dentition, falls to sleep, and shortly afterwards wakes up in a violent state of terror ; he sits up in bed and shouts in an incoherent manner, perhaps only partially awake, and it may be a long time before the mother or the nurse succeeds in pacifying him. Apparently the cause of the fright is sometimes an hallucination of vision ; sometimes, on the other hand, merely a nightmare. It has been aptly said, that you may divide the cases into two groups—'those that see visions, and those that dream dreams.' Those who see visions are those who have hallucinations of sight ; those who dream dreams are those who have what is equivalent to a nightmare in the adult. Although that is probably a real distinction, you will find it difficult to say to which class any given case belongs, because these children are often unable or unwilling to give an accurate description of their sensations during the attack. Sometimes they will tell you that they see frightening objects, shadows or dark shapes in the room, and in such cases, perhaps, they are the subjects of visual hallucinations. Others can give you no coherent account of their sensations. These experiences may be repeated night after night, until the nervous system is seriously upset, and the child begins to dread going to bed ; and then the friends get alarmed, and think that the child is becoming insane, and call in a doctor.

The following is an excellent account of a case of typical night terrors sent to me by a doctor with a little patient of his :

'The girl, aged five, has always been of an excitable nature, but in every way physically healthy. About one month ago she started having night terrors—every two or three days at first, but for the

last ten days every night. Her mother only consulted me three days ago, but I have been fortunate enough to see her in two of these terrors. They always come on about one and a half hours after retiring to bed—i.e. about nine o'clock. The two I have seen were very similar. The child first was heard to scream, and was found sitting up in bed (room half darkened by blind) with staring, terrified eyes, pupils widely dilated, screaming and sobbing alternately. On one occasion the jaw was dropped and she was talking gibberish, plucking at the bedclothes, and eventually cried out, "Mummy, what shall I play now?" and, "Lettice, Lettice, where is it?" (That evening she had been chalking pictures before going to sleep.) The second occasion she was shrieking out, "I want daddy!" Her mother would try to calm her, but the child could not recognize her. Her mother would then lift her up and sit her on a bedroom chamber, when she would urinate, and shortly afterwards give several yawns and finally quiet down. The whole affair would last about two minutes. Great exhaustion would follow, and she would fall asleep in spite of attempts on my part to discover what was the cause of the terror, or of what she had been dreaming. The terror is never repeated. It always occurs about one and a half hours after falling asleep, and afterwards she sleeps soundly all night. On occasions she has been found walking in her sleep in the same terrified condition.'

A very good description of the sensations of children who suffer from those attacks has been given by Charles Lamb in one of the *Essays of Elia* entitled 'Witches and other Night Fears.' Perhaps the passage is known to some of you. He says: 'I was dreadfully alive to nervous terrors. The night-time, solitude, and the dark, were my hell. The sufferings I endured in this nature would justify the expression. I never laid my head on my pillow, I suppose, from the fourth to the seventh or eighth year of my life (i.e. about the time of his second dentition), so far as memory serves in things so long ago, without an assurance, which realized its own prophecy, of seeing some frightful spectre. . . . Parents do not know what they do when they leave tender babes alone to go to sleep in the dark. The feeling about for a friendly arm, the hoping for a familiar voice when they wake screaming and find none to soothe them—what a terrible shaking it is to their poor nerves! The keeping them up till midnight through candlelight and the unwholesome hours, as they are called, would I am satisfied, in a medical point of view, prove the better caution.'

Now night terrors may always be taken as evidence of ill-health in some form or other; they do not occur in perfectly sound children. Amongst the **predisposing conditions** are a neurotic or nervous

heredity, and infection of the throat. But, far more important, is overpressure at school. Hence you will often find that children only suffer from this disorder during the school term. During the holidays they sleep undisturbed, but when they resume school they resume also their night terrors. Mothers often describe them as 'going over their sums in their heads all night,' arithmetic being apparently one of the chief things which exhaust and irritate the brain.

Of **exciting causes,** I would emphasize two which it is important for you to be familiar with. One is gastro-intestinal disorder, especially *constipation*; the other is partial asphyxia from the presence of *adenoids*. These two causes are important for you to recognize from a practical point of view.

When I have mentioned the causes of night terrors, you see what the **treatment** should be. It must not be purely sedative. You should attend to the state of the throat, and you should administer aperients. Further these children ought not to be frightened in any way, nor excited. They ought not to be put to sleep in the dark, but have a night-light in the room, and ought not to be allowed to go to bed with a full stomach, although a sweet biscuit or even a dose of glucose (1 or 2 teaspoonfuls) last thing at night is helpful in preventing a possible hypoglycæmic factor. It may be well to remove the child from school for the time being, until the nervous system has recovered its equilibrium, and it may be necessary to give sedatives, such as chloral, temporarily.

There is an analogous condition to night terrors which occurs in the day, called day terrors or **pavor diurnus,** which is much rarer. The child, in the middle of his games, may be seized with a fit of panic and rush to his mother, giving vent to inarticulate cries. The fit of terror may last for a few moments. They are probably cases where there has been an hallucination of some of the sense organs, and must be treated on the same lines as the night terrors of which I have spoken.

There are also various degrees of disturbance and restlessness at night, which cannot, perhaps, be fairly classified as night terrors. One of the mildest is **grinding of the teeth.** Such cases can be recognized by the fact that the teeth are worn down to the same level. At the other extreme is **somnambulism.** All such cases must be treated on the lines already laid down.

NOCTURNAL INCONTINENCE

The next neurosis I shall describe is enuresis, or *nocturnal incontinence.* I do not pretend to explain how it is that enuresis arises. The exact nerve mechanism of the bladder is not fully understood, but for practical purposes you may compare enuresis to the automatic action of the bladder which you find in many diseases of the spinal cord; and probably it has a very similar explanation. Just as in diseases of the spinal cord, when the brain impulse is cut off by a lesion higher up, the bladder takes to emptying itself automatically when it is full, so in children the brain cortex has sometimes such an imperfect control over the lower centres that the bladder may take on spontaneous action. You may ask at what age the child ought to be able to control the bladder. I think you may say from three years old. You should not therefore talk of enuresis before that.

I have found, too, that in some cases of enuresis there is a great increase in the amount of urine secreted during the night as compared with the normal. This nocturnal diuresis seems to cause enuresis when it occurs, but what causes the diuresis I do not know.

The **treatment** of the spontaneous passage of urine during the night must consist in the removal of the causes which lead to it. You will find it always well to inquire into the presence of threadworms. You should also inquire in the case of boys about phimosis, or preputial adhesions; these also seem to be one of the exciting causes. Further, you should see that these children do not drink much fluid before going to bed, that they sleep on a hard mattress, with few bedclothes; and it is advisable to raise the end of the bed a little, so that the head is lower than the pelvis, and the stress or pressure of the urine tends to gravitate down into the fundus of the bladder instead of pressing on the more sensitive trigone. You will find in some cases that obstructed breathing is the cause of the trouble, so that in every case of nocturnal incontinence which is brought before you you should make a point of examining the throat. Lastly, it is well to examine the urine, to see if it is unduly acid. I think hyperacidity of the urine as a cause of nocturnal incontinence has been exaggerated, but if you find uric or oxalic acid crystals, it is well to give alkalis to make the urine less irritating. I have even seen calculus in the bladder mistaken for incontinence, from the frequent micturition to which it gave rise.

If you have employed all these measures—attended to the state of the urine, removed worms and adenoids if they were present, given directions that the child should not have fluid just before going to bed, and that he should sleep on a hard mattress and with few bedclothes—and if you still fail to cure the condition, what can you do next? You have then to fall back upon drugs. There is one drug especially which is commonly used in the treatment of nocturnal incontinence, and that is belladonna: and there can be no doubt that it is a drug of extreme value if you use it in the proper way. So far as I know, it is the only drug which has a real controlling influence in the disorder. There is only one rule to be remembered in regard to the **administration of belladonna,** and that is *to give it in large doses.* Begin with 5 minims of the tincture, given after each meal; try that for a week, and you will probably reduce the frequency of the incontinence at once. You should then mount up to 10 minims each dose, then to 20 minims, and then to 30 minims, and so on, until you get the incontinence entirely suppressed. I have no hesitation in going up to a drachm of the tincture, three times a day. It is extraordinary how children will stand belladonna. It is one of the drugs which have far less influence on a child than on a grown-up person. There may possibly be a complaint of slight dimness of vision from paralysis of the eye muscles, or of dryness of the throat, during the administration; but I have not seen more than once or twice any general disturbance such as a rash or slight delirium. Now, if you have got up to 40 minims of the tincture after each meal, and then the enuresis ceases, how long should you continue the drug? I think you should do so for at least a month in the full dose, and then discontinue gradually, climbing down again in the matter of doses just as you climbed up. You will then find that in many cases when you have stopped the medicine the habit has been broken, and the child may be regarded as cured.

Other drugs are sometimes recommended as substitutes for belladonna. Ergot has some supporters, strychnine has some, and so have cantharides and thyroid. I have tried all of them, and I can only say that I have not found them of much use. Ergot is perhaps the best. It may be given in doses of 5 minims of the liquid extract flavoured with extract of liquorice in a drachm of peppermint water three times daily for a child of five, the dose being gradually pushed if necessary. Ergot seems to be specially useful in those cases of

nocturnal diuresis which I spoke of a moment ago. Of recent years ephedrine has been recommended, in doses of $\frac{1}{4}$ grain or more at bed-time.

You will find nocturnal incontinence much more easy to cure in boys than in girls. I do not know why that should be, but the most intractable cases one has to deal with are in girls. In such cases treatment by local applications to the urethra have been recommended, but have little support and obvious objections.

The psychological treatment of bed-wetters is also of the first importance. Judicious management on the part of the mother and nurse is essential. The trouble must not be allowed to get on the child's nerves, and his self-confidence should be encouraged in every way possible. He should be told to think of it as little as he can, and it should be impressed upon him that he will get quite over it in time. Stress should be laid upon the 'dry' nights and 'accidents' made light of. That is the sort of attitude to adopt, and I need hardly say that it is the very reverse of that so often taken up by those in charge of the child.

Finally, I would warn you that you must see that these children are not punished. In very few instances is the trouble due to carelessness, and therefore it is not only useless, but unfair and unkind to punish children for this unfortunate weakness. I am afraid you will fail to cure a good many cases in spite of all you do, but luckily incontinence is nearly always outgrown in time, although adult life may almost be reached before this happens.

Occasionally incontinence **occurs by day only,** though that is exceptional, and the treatment is essentially the same as in the nocturnal form. I remember being consulted, for instance, about a girl nine years old, who had suffered from diurnal incontinence since an attack of measles three years previously. She was a very nervous child, but presented no other signs, and the urine was quite normal. Under full doses of bromides and belladonna, the symptom speedily disappeared.

Incontinence of fæces is a rare condition which may occur either independently or in association with enuresis. Not long ago I had a case of it in a girl of five, who seemed otherwise in good health, but who had never been able to control the rectum. She recovered completely under belladonna and ergot.

HABIT SPASM

Habit spasm, or tic, is another of the functional neuroses of childhood, and by no means the least common. It consists in an oft-repeated and apparently purposeless movement usually affecting the face and resulting in ' blinking,' ' grimacing,' ' sniffing,' or other such act. Movements affecting the limbs are also seen, the upper limbs being more often affected than the lower. Thus, there may be a constant shrugging of one shoulder or repeated snapping of the fingers. In severe cases there may even be involuntary exclamations—often of an improper sort.

The subjects of habit spasm are neurotic children, and, as in other functional neuroses, often exhibit a tendency to rheumatism. Overstrain at school, anæmia, and feeble general health, predispose to its occurrence. Local exciting causes should always be looked for. Amongst these are errors of refraction, adenoids, and carious teeth.

The diagnosis is usually easy, but you will be apt to mistake the condition for chorea. In typical habit spasm, however, only one group of muscles is affected, whereas in chorea that is very rare. Sometimes, however, it may not be possible at first to distinguish a severe habit spasm from a case of mild or of ' residual ' chorea.

The treatment to be adopted is much the same as that of the other functional neuroses—viz. attention to the general health, and avoidance of excitement and school-work, with plenty of exercise in the open air. If any of the local exciting causes already mentioned is present, it should be dealt with. Thus it may be necessary to order spectacles, or to attend to the teeth or throat. Removal of adenoids, however, should not be lightly undertaken, as the fright of the operation may aggravate the spasm. Gentle but persistent ' moral suasion ' may do something, the child being constantly encouraged to try to control the movement. Anything like scolding, however, should be avoided. As regards drugs, I have found most benefit from a combination of bromides and arsenic but valerian is also useful occasionally.

As a rule, the condition is recovered from in time, although improvement is very often slow, and sometimes the movements become stereotyped and persist into adult life. When all else fails, a complete change of surroundings and going to live amongst strangers may have a good effect.

FAINTING ATTACKS

In hospital out-patient practice children are often brought with the complaint that they suffer from 'faints' or 'fainting attacks.' The following are brief notes of some cases of this sort which I have seen :

1. Girl, aged four and a half years. Had her first 'faint' when two years of age, but no recurrence until within the last few months, when she has had two more. She 'goes white' and 'doesn't know you,' and would fall down unless supported ; a cold sweat breaks out on the forehead ; she is not sick. The whole attack lasts about ten minutes. All the attacks have occurred in the afternoon and some time after a meal.

She has otherwise been a healthy girl and has not suffered from indigestion or constipation. On examination she appeared perfectly healthy in every respect.

2. Boy, aged eight years. For some months past 'comes over faint,' turns white, and has to lie down, but is not insensible ; is not sick, but often has a headache afterwards. The whole attack lasts some minutes. His general health has been fair, but he is 'always tired' and is backward with his lessons.

To physical examination he appeared rosy and well-nourished, and the heart perfectly healthy. He was, perhaps, rather mentally defective.

3. Girl, aged five and a half years. When two weeks old had a 'collapse' lasting some hours, and has had these on and off since, but more frequently of late. The attacks now usually occur before breakfast, but she may have as many as three in one day. She 'goes pale,' 'can't stand,' and sometimes retches. The whole attack lasts from half to three-quarters of an hour.

A nervous, rather poorly-nourished, dyspeptic child. No anæmia and no signs of organic disease.

4. Girl, aged eight and a half years. During the last twelve months has been subject to 'faints,' especially before breakfast, but also on standing for any length of time. In the attack her 'lips go white,' and she has to lie down. It passes off in a few minutes.

A thin, nervous, excitable child, but not anæmic, the heart quite healthy.

5. Girl, aged seven and a half years. For two weeks past has tended to 'faint' when getting ready for school in the morning. She 'flushes and then goes white,' and has to lie down. In a quarter of an hour the attack has passed off. Physical examination entirely negative.

6. Girl, aged ten years. For two or three months has been subject to 'fainting attacks,' especially when dressing in the morning. She goes 'right off' and is 'quite white.' May remain in this condition for about half an hour. Her general health is poor ; she has a bad appetite, and is subject to headaches and constipation.

A poorly-nourished, ill-developed child ; no anæmia ; heart normal.

7. Boy, aged twelve years. Has had 'fainting attacks' at irregular intervals for the last six years, but they are getting more frequent. Feels 'bad' when the attack is coming on, looks 'white and drawn,' 'not really insensible,' and can answer if spoken to. The attack usually passes off in less than five minutes. His health otherwise is good, but he seems 'lacking in energy.'

A healthy-looking, well-nourished boy with no signs of organic disease.

You will notice that both sexes suffer from the attacks, but girls rather more frequently than boys. Occasionally they date from quite early life, but usually they do not appear until about the fifth year or later, and are commonest during the school age. In general features the attacks are very similar in all cases. The child is observed to 'go white'; he may fall down, but does not lose consciousness entirely, although in some cases he is dazed, or even only semi-conscious. Occasionally retching or even vomiting occurs, but in no case is urine voided involuntarily. The attack lasts for a period varying from a few minutes up to half an hour or even longer, and passes off gradually. Sometimes it is followed by headache. The commonest time for the attacks to occur appear to be in the morning, often before breakfast or whilst the child is getting ready for school. Many of the children are nervous and dyspeptic, but in a considerable number the general health is quite good and the appearance of the child flourishing. There appears to be no relation between the attacks and the occurrence of any previous disease.

On only one occasion have I been fortunate enough to witness an attack. The patient in that case was a boy aged ten years, who had been subject to faints for some time. When standing up to be examined he suddenly became pale and giddy; the pulse very slow and feeble, and the heart sounds toneless. He was quite conscious, but somewhat confused. On laying him down he gradually revived, and then flushed a little.

I have not been able to discover any cause for the susceptibility to these attacks, but overstrain at school certainly seems to play a part in some cases. Dyspepsia and nervousness are so common amongst the hospital class of children that their association with the attacks may easily be a coincidence.

The diagnosis is not difficult, minor epilepsy being the only condition which might be mistaken for them. In 'faints,' however, consciousness is not completely lost, and their duration is usually much longer than that of an epileptic seizure.

As to the pathogeny of the attacks, it is difficult to speak with certainty. Mothers, and sometimes doctors, are apt to ascribe them to 'heart weakness,' and are often much alarmed in consequence. There is no reason to suppose, however, that the heart is at fault.

It seems much more probable that the condition is primarily nervous in origin, and due, perhaps, to a temporary sympathetic paralysis leading to uncontrolled action of the autonomic system, with vaso-dilatation in the splanchnic area and inhibition of the heart. Whether liability to the attacks may be the consequence of a defective suprarenal secretion resulting in a lowered sympathetic 'tone' is an interesting subject of speculation.

As regards treatment, I have always found that removal from school, change of air to the seaside, and the use of strychnine as a tonic, along with attention to the digestive organs, speedily results in cure.

LECTURE XXVII

CONVULSIONS IN CHILDHOOD

It is superfluous to insist upon the importance of convulsions in practice. They have a dramatic quality which impresses and alarms the parents of the child, and calls for instant action on the part of the doctor, whilst the uncertainty as to what they portend and what their ultimate consequences may be is a source of anxiety to all concerned.

Diagnosis

If you happen to see an attack of the 'convulsions' yourself, there is usually no difficulty in recognizing them. Indeed, the only condition you might mistake for them is **tetanus neonatorum**, though here the presence of *trismus* should prevent error. But should you only be told by the mother that the child has had a 'convulsion,' the case is rather different, for the maternal and the medical idea of what constitutes the disease are not always identical. For instance, a mother may regard a mere rigor as a convulsion, or she may describe under that term the condition known as 'breath-holding,' though it must be remembered that breath-holding may end in a true convulsion occasionally. I have known the delirium of fever mistaken for convulsions and also the shaking of a child with night terrors. The maternal phrase 'internal convulsions' is another source of confusion. It is not always easy to make out what a mother means by this, but usually it seems to refer to a slight twitching of the mouth or clenching of the hands. These may only be symptoms of colic, but, on the other hand, it is well to remember that even so they may indicate a nervous irritability which may easily lead to true convulsions. One should be careful, therefore, not to pooh-pooh such a diagnosis too readily.

The above are instances in which convulsions are diagnosed by the mother when they don't really exist, but, on the other hand, she may fail to recognize as convulsions what ought to be regarded as

such; I mean cases of *petit mal*. These, especially, when they occur in little babies are apt to be described by the mother simply as 'faints' or 'starts,' and their true nature completely overlooked. I shall have occasion to say something more about them immediately.

Causes of Convulsions

Predisposing conditions.

One must recognize in the first place a greater predisposition on the part of the child to suffer from convulsions than is present in the adult, and it seems reasonable to attribute this to an instability of the immature nervous system. The fact, also, that some children develop convulsions on much slighter provocation than others shows that the predisposition in them is greater, and this again may be ascribed to a neurotic inheritance, which, indeed, is often found to be present. Apart from such a general predisposition there seems to be a special tendency for convulsions to manifest themselves at certain age periods. The first is the earlier weeks of life—say up to the end of the third month; the second is during a period extending roughly from about the sixth month to the end of the second year. As regards the first of these periods, the special incidence of convulsions is partly due to the influence of injury at birth, to which I shall refer shortly, but independently of that there is a predisposition to the occurrence of 'idiopathic' convulsions at this age which has not yet been explained. I shall have occasion to deal more fully with this variety of convulsions later on.

As regards the second of the above periods, that between the sixth month and the end of the second year, the tendency to convulsions throughout it is due to the fact that rickets manifests itself at this time and leads to an undue irritability of the nervous system. To this state of things it is now the fashion to apply the term 'spasmophilia.' It is a term which I confess I do not like, as being an example of the intellectual vice so common in medicine of thinking you have explained something when you have stuck a label on it. I do not see myself that the word 'spasmophilia' is any more illuminating than the old phrase 'nervous irritability of rickets,' nor, indeed, so much so; but I am afraid it has found its way into medical literature, and therefore it was necessary to mention it. Call it what you will, however, it is a fact that in rickets the nervous system is unduly excitable, and the signs of this are

'facial irritability,' laryngismus, tetany, and, in extreme cases, convulsions.

Exciting causes.

Given a predisposition to convulsions, inherent or acquired, the causes which may 'fire off' the attack are various, and it will be advisable to make some sort of classification of them so as to help in diagnosis.

1. **Reflex.**—Any seat of peripheral irritation may excite a convulsion, but the alimentary canal is the first region to be thought of. The irritation there may be caused simply by indigestible food, by constipation, by colic, or by the presence of worms, especially, perhaps, round-worms. Teething is also a possible cause, though not so common a one as is popularly believed.

Or the irritation may be in the urinary tract, and set up by the passage of gravel or by infection with the *Bacillus coli*, or it may be due to phimosis, though the importance of this, like that of teething, is apt to be exaggerated. Earache is undoubtedly a cause in some cases, but indeed any source of peripheral irritation, provided it be of sufficient intensity, may excite an attack.

2. Any sudden **rise of temperature** may excite a convulsion in a predisposed child, just as in the adult it may cause a rigor. Pneumonia is perhaps the commonest disease which may begin in this way.

3. **Disease of the nervous system** is, as might be expected, specially prone to cause convulsions; hence they are common in meningitis and encephalitis. Injuries to the brain at birth are a common cause in newly-born infants, and when convulsions appear at this period signs of brain injury such as paralysis, and bulging of the fontanelle, should always be looked for. The patient is usually a boy, and there will be a history of a difficult labour and often of a breech presentation with prolonged asphyxia after birth. The diagnosis may be confirmed by the presence of blood in the cerebro-spinal fluid. Apart from injury or acute disease, congenital defect of the brain may cause convulsions. Many cases of *petit mal* in little children, especially girls, are so caused, and mental deficiency often coexists.

4. **Toxic causes,** such as uræmia, must also be thought of, but here the other evidences of nephritis are usually obvious. Cases of pyelonephritis, however, may be overlooked.

5. **Asphyxia,** however induced, may also bring about convulsions. Thus pertussis, laryngismus, and breath-holding may end in such seizures, whilst these may also occur in consequence of asphyxia as a terminal event in almost any disease. When a child is said to die of convulsions, it would usually be more correct to say that it dies *in* convulsions, the latter being only a mode of dying after asphyxia had supervened.

6. Lastly, convulsions may be **idiopathic**—that is to say, due to no discoverable cause. Many cases of convulsions in the first few weeks of life, which are not due to head injury, are of this nature. They begin often quite suddenly, when the infant, usually a boy, is two or three weeks old, starting perhaps as a slight twitching of the face or hands, but rapidly passing into generalized spasms which are repeated at short intervals, and which may go on for day after day unless they are properly treated. Such cases are usually supposed to be due to colic or 'indigestion,' but, in fact, there is generally no real evidence of anything of the sort, and their true cause is quite obscure. These 'idiopathic' convulsions of the newly-born are perhaps the most important you will meet with in practice, especially from the point of view of treatment, as I shall have occasion to point out immediately.

In older children a convulsion for which no cause can be discovered may be the beginning of epilepsy. It is only when the convulsions continue to recur that a diagnosis of epilepsy is justified, whereas everyone must have met with cases in which one apparently causeless epileptiform convulsion occurred without any successor.

In deciding, then, upon the cause of convulsions in any case one has to ask, Is there any source of reflex irritation discoverable? Has there been a sudden rise of temperature? Are there any signs of disease, injury, or defect of the brain, or is toxæmia or asphyxia present? If all these causes can be excluded, one must decide that the case is one of idiopathic convulsions.

Prognosis

Convulsions are of themselves rarely dangerous to life unless they are severe and prolonged, or unless the child is very young and feeble. As I have already said, when a child is stated to have died 'of convulsions,' it usually means that the convulsions were simply a terminal event and the consequence of an asphyxia which is in itself lethal.

Nor, probably, do convulsions often *cause* mental impairment or permanent paralysis. When these consequences follow, it is usually because some disease or injury of the brain has caused both the convulsions and the mental defect or paralysis. It is fairly safe to say that there is no risk of mental impairment unless the convulsions are due to disease, injury, or defect of the brain, or unless the seizures are severe and repeated over a long period. Whether the convulsions are likely to lead to epilepsy is a question to which it is always well to give a guarded reply. It is essentially one of those questions which can only be settled by waiting and seeing, for, as I have pointed out, it is only the fact of the convulsions continuing to recur without apparent reason that constitutes them epileptic. In any case, it is but a small minority of the children who have convulsions who end by becoming epileptic, so that the chances in any given case are always against so sinister an outcome.

Treatment

When called to a case of convulsions, you will probably find, upon your arrival, that the child has already been put in the time-honoured hot bath. There is no objection to this, and in the case of convulsions due to peripheral irritation—especially from abdominal pain—it is no doubt useful, whilst it has also the advantage of giving the mother and nurse something to do.

For the immediate suppression of convulsions you have several agents at your disposal. The inhalation of chloroform is, of course the quickest, but it cannot be continued indefinitely. An injection of morphia is also highly efficacious, but it has to be used with caution in little babies—$\frac{1}{50}$ grain is quite a sufficient dose to start with in a child of six months. A solution of sodium phenobarbitone may also be used by injection, say $\frac{1}{8}$ grain for a child of six months. Chloral is excellent, though not so rapid in its action, but, on the other hand, it has the advantage of being safe. Five grains may be given per rectum at six months or half that dose by the mouth. Where the fontanelle is tense and bulging, lumbar puncture is often of service, but, of course, it is not a method which is always advisable in private practice.

Having by one or other of these means overcome the convulsions for the moment, you have next to try to determine the cause according to the classification given above. If any source of peripheral irritation is discovered it must be removed, and in any

case it is always harmless and advisable to give a dose of castor oil. The temperature should be taken, and a careful examination made for signs of injury or disease of the nervous system. If there is any evidence of rickets, vigorous antirachitic treatment by diet, cod-liver oil and vitamin D in concentrated form should be instituted.

Meanwhile in all cases the child should be kept slightly under the influence of chloral until any danger of a recurrence of the attack is over.

I should now like to say a word as to the treatment of those cases in which no cause for the convulsions is discoverable—the so-called idiopathic group. The treatment of idiopathic convulsions in the newly-born is extremely satisfactory—if you know how to do it. Bromides in these cases are useless. The only plan is to get the infant rapidly under the influence of chloral. For this purpose 1 grain every two hours is none too large a dose to give by the mouth to a baby a fortnight old. After a very few doses the convulsions will cease and the child fall asleep. It should be kept in that condition for at least twenty-four hours, and the dose then gradually reduced. On waking up it will be found that the convulsions have disappeared, and they rarely recur. It should be added that whilst the child is under the full influence of the drug great care should be taken in feeding, as some of the milk is apt to find its way into the trachea and set up an 'aspiration' pneumonia.

Idiopathic convulsions in older children can be treated with bromides or luminal in the same way as epilepsy.

BREATH-HOLDING

Before leaving the subject of convulsions I wish to mention a condition which resembles them somewhat, and which you will not find described in your textbooks. The following extract from the letter of a doctor who wrote to consult me about it in one of his own children will give you a good idea of what I mean:

'. . . The patient is my third child, a boy of two. He has always been healthy, and there are no signs of rickets or other constitutional disease. Ever since he was quite small he has been in the habit "of holding his breath." The condition has become worse lately, and during the last few days he has had several such attacks. The child begins to cry, respiration is suspended, he gets purple in the face, and

then becomes unconscious. The spasm is then relieved, and he begins to breathe, but twice he has had very marked twitching of the face muscles, which, however, soon passed off. He remains quite inert and irresponsive for fifteen to twenty minutes, when he suddenly recovers, and, except for being upset and white-looking, is himself again. There is *no* crowing respiration.'

In another case a lady wrote to me about her little grandson as follows :

'The boy seems a thoroughly healthy little fellow, is just two, but he has a kind of choking fit. The doctor has never happened to see him in one (as he does not choke to order), and I do not think understands the cause of them. The symptoms are these : Sometimes when he is thwarted he draws in his breath to cry, but no sound comes, and he gradually turns blue about the lips ; his throat seems closed. Cold water is dashed on him, sometimes for quite a long time. At last he gasps, then goes limp and unconscious ; after a short time he revives, has a good cry, and gets better. The whole time occupied is about five minutes, but is sufficiently alarming while it lasts. Perhaps you can tell me the reason of this. Is it a common thing, and can it be cured ? The boy is being spoilt because of the fear of his holding his breath.'

I have seen many cases of this sort, all of them in boys below the age of five, but there is no scientific name for the condition, although I believe it is popularly known in some parts of the country as the **kinks.** It consists, apparently, in a spasm of the respiratory muscles, which differs from the spasm of laryngismus in that the chest is fixed in the position of inspiration instead of expiration—hence the absence of a 'crow' when the spasm passes off. The subjects of this neurosis are usually excitable, self-willed children and the spasm tends to come on whenever they get in a temper or are crossed in any way. It is, in fact, an example of 'negativism.' Although the attacks may be very alarming, I have never known them lead to any fatality.

The best treatment is to dash some cold water in the patient's face so soon as an attack is seen to be coming on ; this will usually stop it at once. In addition, such children should be guarded from all excitement, be firmly managed, and, if necessary, be put through a course of bromides.

PYKNOLEPSY

Mention should also be made of the rare condition called pyknolepsy which closely resembles *petit mal.* The attacks begin suddenly

between the fourth and tenth years and attain their maximum frequency (which may be as great as a hundred in a day) from the outset. All the attacks are exactly the same and consist in a momentary loss of consciousness with perhaps some slight twitching movement but without any falling. The attacks are quite unaffected by treatment and cease of themselves after a few years, leaving no ill-effects. In rare instances I have thought that ephedrine ($\frac{1}{4}$ grain, t.d.s.) was of some help. The following is an example :

A boy of nine had had the attacks for two years. They began suddenly during whooping-cough and had not altered since. There were about ten attacks daily, mostly in the morning, and each lasting only a second or two. In the attack his head drops or shakes, his eyes roll up slightly and there is a 'curious look in them,' but he is never convulsed, and does not drop anything he is holding and never falls. If asked a question in an attack, he will reply to it as soon as the attack is over. There was no family history of epilepsy and physical examination revealed nothing. At the end of a year he was reported to have recovered completely.

LECTURE XXVIII

THE PARALYSES OF CHILDHOOD

PSEUDO-PARALYSES

When a child is brought to you alleged to be suffering from 'paralysis,' the first thing you have to determine is if the loss of power is really due to some affection of the muscles or nerves, or if it is merely a case, not of *inability* to use the limb, but of *disinclination* to do so because any attempt at movement causes pain. Cases of the latter sort are not very infrequent in early childhood, and they are spoken of as 'pseudo-paralyses' to distinguish them from genuine nervous or muscular lesions. There are three great causes of such false paralyses; syphilitic epiphysitis is one, scurvy is another, and rheumatism is the third. I may say a word or two about each of these.

1. **Syphilitic pseudo-paralysis.**—Some of you may have seen a child in one of the cots lately who was brought to hospital because it had suddenly ceased to use the left arm. There was a vague history of injury, you may remember, and the case was at first regarded as being probably one of a lesion of the brachial plexus. On more careful examination, however, signs of congenital syphilis were detected, and I came to the conclusion that we were probably dealing with an epiphysitis of syphilitic origin. This conclusion was justified by the fact that under vigorous mercurial treatment the supposed paralysis quickly passed off. That case presented special difficulties, but usually you will make a correct diagnosis in such circumstances if you remember the existence of the condition. As a rule, syphilitic epiphysitis occurs in infants of only a few weeks old—the oldest I have seen was six months—and it almost always affects several joints, particularly the joints of the arms; indeed, I do not remember to have seen a syphilitic pseudo-paralysis of the lower limb. The proper treatment is to place the limb in a splint, and to push mercury. If this plan is adopted, a speedy cure almost always results.

2. **Scorbutic pseudo-paralysis.**—The other day a child ten months old was brought to the receiving-room for loss of power in one leg, and was sent down to out-patients with the diagnosis 'Paralysis. ? cause.' On examining the infant, one was at once struck by the great tenderness of the limbs, and on inspecting the gums they were found to show slight sponginess round the incisor teeth, whilst inquiry into the history revealed the fact that the child had been fed on condensed milk and a patent food. Anti-scorbutic treatment was adopted, and when seen a week later the child was using the limb freely.

Here, again, the apparent paralysis was really a mere disinclination to use the limb, from pain. In contrast to syphilitic pseudo-paralysis, it is the lower limbs which are most apt to be affected in scurvy. The extreme tenderness of the limb when handled should usually put you on the right track, and you will often find also some thickening round the lower end of the femur. Your diagnosis will be confirmed by finding sponginess of the gums and a history of the child having been fed on the sort of diet which is apt to produce scurvy. The diagnosis once made, the treatment is obvious, and its results usually brilliant.

3. **Rheumatic affections,** particularly of the lower limbs, may sometimes simulate paralysis. I have known this happen, especially when the tendo Achillis, or hamstrings were involved. Here it is older children who are affected, never infants, and the immobility is rarely complete, but is chiefly manifested by an inability to walk properly. You will usually find some signs pointing to rheumatism elsewhere, or a history of its occurrence in the past, and the use of salicylates and local friction with a liniment speedily bring about a cure.

Finally, it is always well to bear in mind the possibility that paralysis may be stimulated by a purely **surgical affection.** I have known a distinguished physician, for instance, be completely nonplussed by a case of double congenital dislocation of the hip, and ordinary simple epiphysitis or hip-disease or dislocations may easily lead you into error.

TRUE PARALYSES

Assuming you are satisfied that you have to deal with a case of true and not of simulated paralysis, the next thing you have to

decide is the **seat of the lesion.** In this diagram (fig. 59) there is shown the course of a motor fibre from its starting-point in the cerebral cortex to its termination in a muscle, and there is indicated also the different lesions of which it may be the subject at various points in its course. The first step to determine is whether the

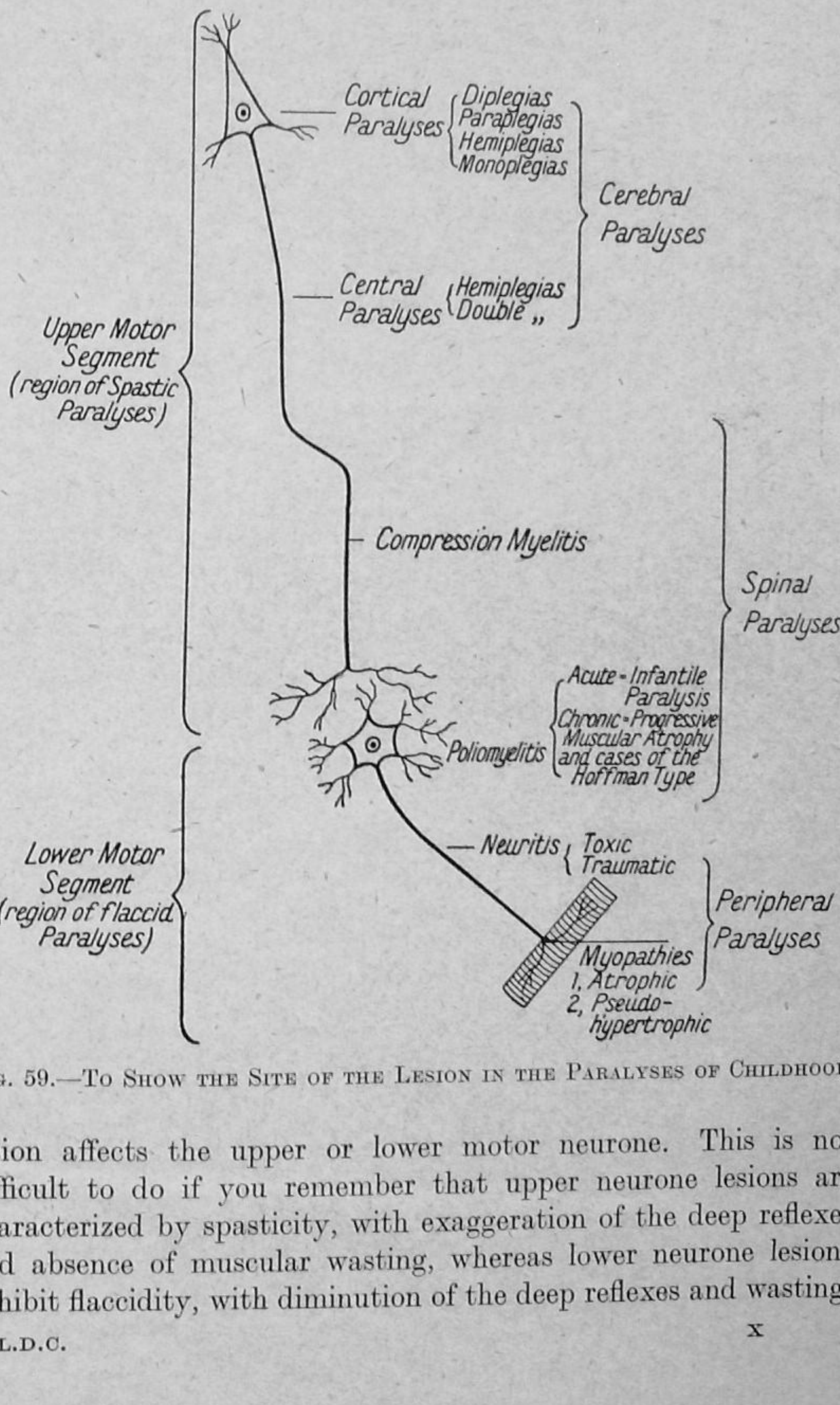

FIG. 59.—TO SHOW THE SITE OF THE LESION IN THE PARALYSES OF CHILDHOOD.

lesion affects the upper or lower motor neurone. This is not difficult to do if you remember that upper neurone lesions are characterized by spasticity, with exaggeration of the deep reflexes and absence of muscular wasting, whereas lower neurone lesions exhibit flaccidity, with diminution of the deep reflexes and wasting.

We may therefore consider first the lesions of the upper neurone, known as—

The spastic or cerebral palsies.

According to their period of onset, these may be divided into three groups:

1. Pre-natal—i.e. occurring *in utero*.
2. Natal—i.e. occurring at the moment of birth.
3. Post-natal—i.e. occurring some time after birth.

FIG. 60.—LEFT HEMIPLEGIA, DATING FROM BIRTH.

FIG. 61.—LEFT HEMIPLEGIA, SHOWING MARKED SPASM OF ARM.

Pre-natal cases are usually the result of malformation of part of the brain—a condition to which the term *porencephaly* is applied —or of an arrested development of the cortical cells (*agenesis corticalis*) or of an intra-uterine inflammatory process.

The **natal cases** are due to an injury to the brain during parturition. The nature of the injury may be obscure, but one may safely regard it, in many cases at least, as due to a cerebral anoxæmia. Often one finds that such children have been born prematurely, and

only resuscitated with great difficulty. Instrumental delivery is by no means always a factor.

Cases which come on some time after birth [1] (**post-natal**) are usually hemiplegic, and may be due to the same causes as produce hemiplegia in adults—e.g. embolism, thrombosis, or even hæmorrhage. Sometimes, at least, they are the result of surface lesions—e.g. venous thrombosis—and many are due to an acute polioencephalitis affecting the motor area analogous to the poliomyelitis which affects the cord.

FIG. 62.—CASE OF DIPLEGIA, SHOWING GENERALIZED RIGIDITY AND INTRAUTERINE POSITION.

As regards its **distribution,** the paralysis may be:

(1) Hemiplegic (involving one arm and leg);
(2) Paraplegic (involving both legs);
(3) Diplegic (involving all the limbs);
(4) Monoplegic (involving one limb only);

in that order of frequency, and depending upon the site and extent of the lesion.

The paraplegic cases are sometimes spoken of by the special name of Little's disease.

I have used the term **diplegia** to signify a paralysis affecting all the limbs. You will often find it employed, however, to signify any bilateral affection of the cortex which results in paralysis, and if used in that sense it would cover paraplegia as well. There are several varieties of diplegia. The typical form in which all the

[1] It is well to remember that the history is not always a safe guide as to whether a case is of natal or post-natal origin, for there can be little doubt that in many cases which really date from birth the paralysis is not noticed until the child tries to walk.

limbs are affected is also spoken of as *generalized rigidity*, and a good example of it is shown in fig. 62. Such cases are usually of pre-natal origin. At other times, again, it results from a progressive degeneration of the cortical cells coming on some time after birth —a condition sometimes described as one of 'premature senility' of the neurones. Such cases often occur in various members of the same family. In yet another group the progress and symptoms closely resemble those of G.P.I., and in that case the disease usually starts during adolescence, and is of syphilitic origin. To the diplegias also belong the cases of amblyopia with mental deficiency sometimes met with especially in Jewish families, to which I shall have occasion to refer in a later lecture.

You might suppose that it would be impossible to distinguish a case of diplegia from one of double hemiplegia, but it is not so. In diplegia there is always more rigidity than paralysis, in hemiplegia the reverse. In hemiplegia, again, the arm is always more affected than the leg; in diplegia this is not observed. As regards the relationship between the distribution of the spastic palsies and the time of their causation, it may be said that the hemiplegias are usually post-natal, the paraplegias natal, whilst the diplegias may be either pre-natal or post-natal. Monoplegias are so rare that I cannot tell you what is their usual time of causation.

Complications of the spastic palsies.

You will find that the spastic palsies not merely produce a greater or less degree of impairment of motor power; they also bring in their train certain remoter consequences. Two of these are specially prominent and should be remembered when you are giving a prognosis. One is mental impairment, the other is fits.

1. **Mental impairment.**—You will not be surprised to find that any extensive cerebral lesion in a young child is apt to be followed by some impairment of the mental functions, and, in accordance also with what one would expect, the earlier the lesion makes its appearance, the more apt is such impairment to be manifested. So far as I have been able to observe, diplegics are invariably imbecile; they become, indeed, practically dements, whilst paraplegics are more apt to suffer than hemiplegics. The degree of mental disturbance may vary from a slight degree of 'eccentricity' to a condition of absolute imbecility.

2. **Fits.**—If you inquire into the history of cases of spastic paralysis of post-natal appearance, you will often be told that the

paralysis arose in consequence of a fit. Whether the fit in such a case is really the cause of the paralysis, or whether both fit and paralysis are the common result of a cerebral lesion, I am not prepared to say ; but of this there can be no doubt—that in many cases the fits tend to recur in such children, or to make their appearance sooner or later, even although they were not present from the outset. The fits are typically epileptic in type, and often occur with great frequency and severity, and are of special importance, because they tend still further to impair mental development. They are probably more apt to occur in those cases which are the result of a surface lesion, especially when it happens to be both extensive and of early production.

Pott's paraplegia.

Cases of spastic paraplegia are sometimes brought to one with the history that the paralysis has come on gradually. In such cases it is always advisable to examine the back carefully for signs of spinal caries, particularly in the cervical and upper thoracic regions, for **compression myelitis,** as it is called, is not an uncommon result of caries in these situations. Here, again, it is the upper neurone which is involved—and that being so, the paralysis is, of course, spastic in type. It differs, however, in several respects from the spastic paraplegia of cerebral origin which I have already described. In the first place, the history usually makes it quite plain that the symptoms have developed *slowly*, whereas, as we have seen, in so-called Little's disease the paralysis has almost always dated from birth. In the second place, the sphincters are usually involved early in Pott's paraplegia ; indeed, over-distension of the bladder, with consequent cystitis, is one of the unpleasant results which, unless the doctor is watchful, the affection of the sphincters may bring about. The paralysis is sometimes preceded by pains referred to the chest or abdomen, and you will find it a good rule in every case in which a child complains of persistent abdominal pain to examine the back.

You should not take too gloomy a view of these cases. It is surprising how completely the paralysis may be recovered from if the child is kept for a sufficient time at perfect rest.

Lesions of the lower motor segment.

The most typical form of paralysis which results from a lesion of the lower neurone is **infantile paralysis,** which is, as you know, the consequence of poliomyelitis. Now, the first remark I have

to make about 'infantile' paralysis is that it is a rather badly-named disease.

It is not, indeed, a characteristic disease of *infancy* at all, for it is but rarely met with before the end of the first year. The great majority of cases occur in the second and third years. You will usually have little difficulty in recognizing a case when it is brought to you (figs. 63 and 64). The flaccidity of the limb, the loss of the deep reflexes, the wasting of the muscles, and the presence of

FIG. 63.—INFANTILE PARALYSIS, AFFECTING RIGHT DELTOID.

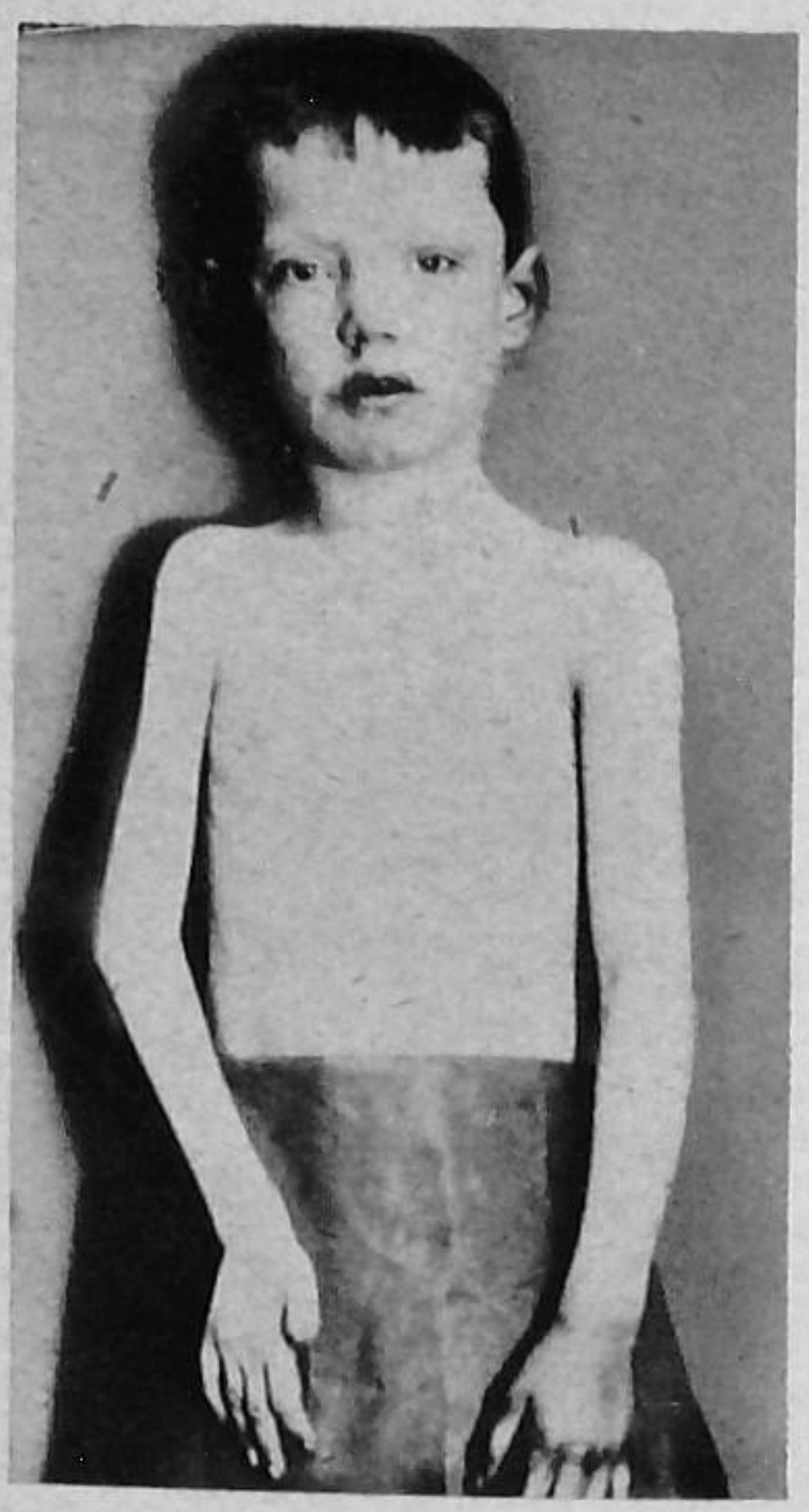

FIG. 64.—INFANTILE PARALYSIS, RESULTING IN SHRINKING OF RIGHT ARM.

the reaction of degeneration, all proclaim it at once as due to a lesion of the lower neurone, and the only thing for which you can mistake it is a paralysis due to interference with a peripheral nerve. How such an error is to be avoided I shall point out shortly, when I come to speak of the peripheral palsies of childhood.

Although a fully-developed case is not difficult to recognize, you will often be puzzled should you happen to see the patient during the active stage of the disease. It will be but rarely, however, that you will have that opportunity. The onset of poliomyelitis is often

marked by but little in the way of general disturbance of health. Slight fever, perhaps a disinclination for food, and a 'going off his legs,' may be the only indications of the serious mischief which is on foot. Sometimes, however, the disease sets in with more of a fracas. I remember being asked by one of my colleagues here to see a boy of five who had been admitted to the hospital suffering from severe fits. When I examined him he was almost in a *status epilepticus*, passing from one severe general convulsion to another, with brief intervals of only partial consciousness. A careful examination revealed no signs of disease, and one was driven to make the tentative diagnosis of convulsions, probably of reflex origin from the bowel. In a day or two, however, the mystery was cleared up for the boy emerged from his fits, but with complete paralysis of the shoulder group of muscles on one side. It had been a case of anterior poliomyelitis ushered in by convulsions.

Another mode of onset which may puzzle and mislead you is that in which the initial stage of the paralysis is attended by great **hyperæsthesia** of the affected limb. I remember being quite deceived by the first case of this sort which I saw. It was that of a little girl who was under my care whilst I was in temporary charge of one of the wards at Great Ormond Street. Her left leg seemed to be paralysed, but as soon as one attempted to examine it the child seemed in such pain, and screamed so loudly, that one was obliged to desist. Not being at that time aware that infantile paralysis might be accompanied by such great tenderness in its early stages, I was inclined to think that one had to deal with one of those pseudo-paralyses which were described at the beginning of this lecture, and discussed the possibility of mischief in the neighbourhood of the hip-joint. Examination under an anæsthetic, however, revealed nothing, and by and by the tenderness passed off, and the child was left with an ordinary infantile paralysis of some of the muscles of the thigh.

In other cases again the disease may in its early stages closely simulate **meningitis,** head retraction and other manifestations of meningism being prominent features. Lumbar puncture should settle the diagnosis in a doubtful case.

As regards the **prognosis in infantile paralysis,** one can always safely say that, after the acute stage has passed off, any change which takes place will be in the direction of improvement. The extent to which the affected muscles will be finally disabled can be

gauged better by the electrical reactions than by any other test. The more completely and extensively faradic excitability is lost, the more irreparable is the damage which has been done. It should also be remembered that acute poliomyelitis is really a specific fever and that one attack protects the patient for the future. Exceptions to this are extremely rare.

I shall discuss the **treatment** of infantile paralysis and the deformities which result from it along with that of paralyses in general.

Chronic anterior poliomyelitis (Werdnig-Hofmann's disease).

Chronic anterior poliomyelitis leading to progressive muscular atrophy is a rare disease in childhood. Hofmann described a special type of it which occurs in families, and of which the case depicted in fig. 65 was an example. This child, a boy four months old, was brought to see me with the history that he had become gradually paralysed within the last three months. The mother stated that an exactly similar fate had befallen two of her other children at the same age. On examination the infant was found to exhibit complete flaccid paralysis of all the muscles except the diaphragm, so that he lay helpless on his back, breathing with difficulty and occasionally giving vent to a feeble cry. The atrophy of all the muscles is well shown in the photograph, and a few fibrillary tremors could be observed. The child died shortly afterwards, but, unfortunately, no examination could be obtained. Such cases are exceedingly rare, and belong rather to the curiosities of pædiatrics,

FIG. 65.—HOFMANN TYPE OF PROGRESSIVE MUSCULAR ATROPHY, SHOWING GREAT WASTING OF ALL MUSCLES, ESPECIALLY OF THE INTERCOSTALS.

for you may spend a long life in busy practice and never meet one of them.

Peripheral palsies.

Paralysis from lesions of the peripheral nerves is not very uncommon in childhood, and may be divided into the two great groups of (1) traumatic and (2) toxic. The best example of the former are the 'obstetrical palsies,' which result from injury to nerves at birth; the latter are typified by post-diphtheritic paralysis. It will be well to consider each group separately.

1. **Obstetrical pasies.**—These may affect the arm, the leg, or the face. Those of the arm are the commonest and best known, and usually take the form of **Erb's paralysis,** which is the result of an injury of the fifth, sixth, and seventh cervical nerves at the root of the neck. These nerves supply, as you will remember, the deltoid, biceps, brachialis anticus, and supinator longus, as well as the external rotators of the shoulder (rhomboids, supra- and infraspinatus, and teres minor). When these muscles are paralysed, the arm assumes a very characteristic position, being held limp by the side, fully extended and pronated, and rotated inwards at the shoulder, so that the palm of the hand looks backwards and a little outwards—much in the position, as it has been remarked, in which a man places his hand when expecting a tip. (The characteristic position is well shown in fig. 66). A posterior subluxation of the shoulder-joint is very apt to supervene as a late result of the paralysis, and is often a bar to complete recovery.

FIG. 66.—ERB'S PALSY—LEFT ARM.

The injury to the nerves in these cases is probably the result of pressure or of laceration, and the labour will almost always be found to have been a difficult one. This may be the reason why the condition is commoner in boys. Of fourteen consecutive cases of which I have notes, nine were males and five females. It is commonly

said that most cases are the result of a breech presentation, the injury being inflicted in the delivery of the after-coming head, but in only four of my cases was the child born by the breech. Nor can one attribute it to injury by forceps, for in ten of my cases the child was born without the aid of instruments. Of my own cases, eight were right-sided, four left-sided, and two were bilateral; but other observers state that the preponderance of cases is on the left side.

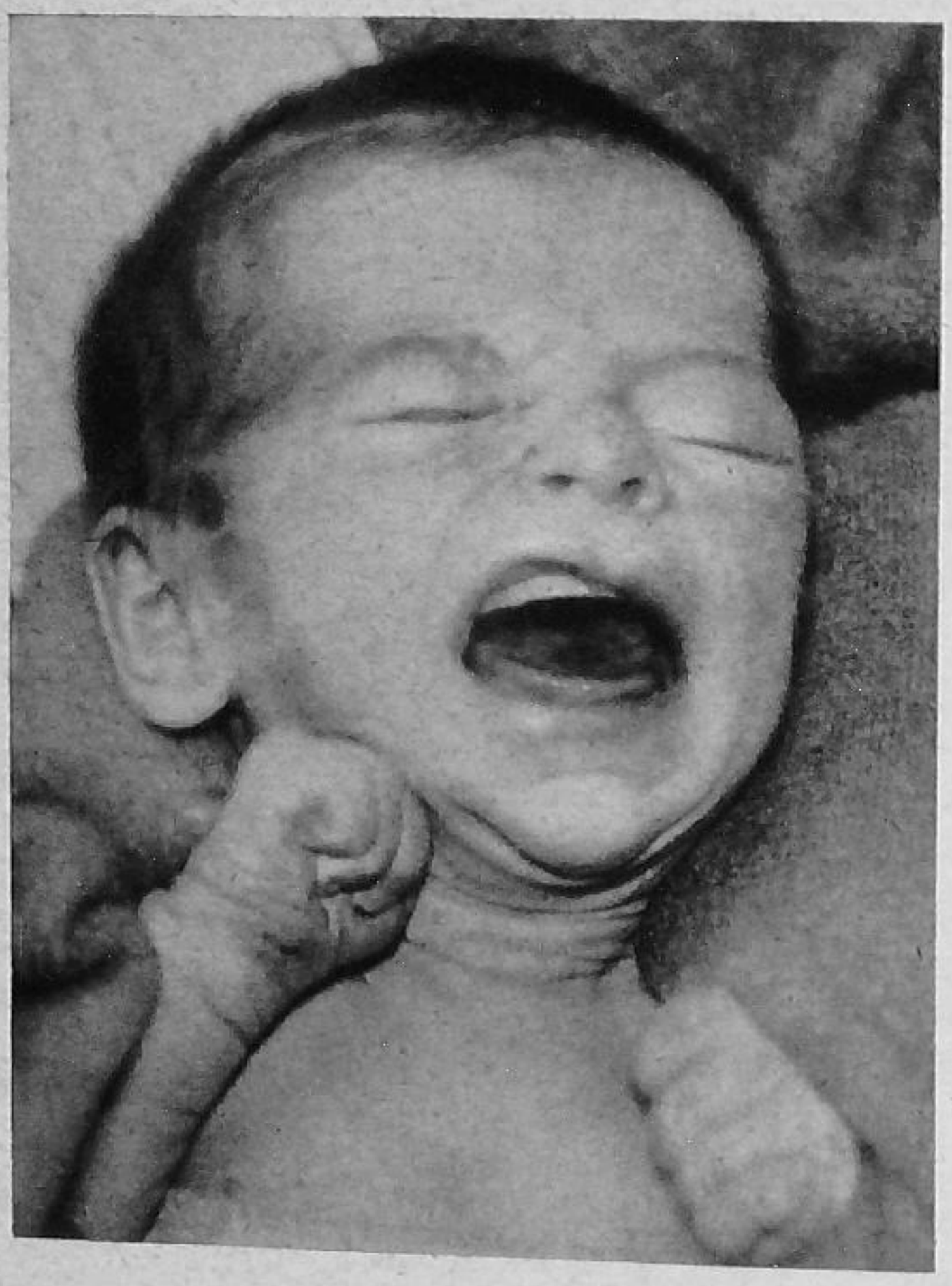

FIG. 67.—OBSTETRIC FACIAL PALSY.

In a typical case the condition can be recognized at a glance, but as all the above-mentioned muscles are not always equally affected, many atypical forms are met with. These you may have difficulty in diagnosing from infantile paralysis, and, in the absence of a history, I do not know of any way in which you could be sure which it was you were dealing with. Usually, however, you will be clearly told that the paralysis was noticed at or shortly after birth, and that should keep you right. The future of such cases

is difficult to forecast. The majority probably recover to a great extent, for typical examples of the paralysis are very rarely met with in adult life; but often, no doubt, some degree of weakness of the shoulder muscles remains. Your estimate of the chances of recovery in any given case must be based on the same data as were laid down for your guidance in the prognosis of infantile paralysis.

Obstetric paralysis affecting the **leg** is said to occur, but I have never seen an example of it. **Facial paralysis** from this cause, on the other hand, is not very uncommon, and is often the result of the pressure of the blades of the forceps (fig. 67). Usually it passes off quickly.

The treatment of these affections I shall come to later.

2. **Toxic neuritis.**—With the exception of the post-diphtheritic form, toxic neuritis is very uncommon in childhood. The forms which you are familiar with in adults as due to lead, alcohol, and arsenic you will hardly ever see in the child, although I have seen a few cases of lead palsy resulting from the licking of paint off toys.

FIG. 68.—WRIST-DROP FROM MULTIPLE NEURITIS.

Occasionally one sees cases of **multiple neuritis** in children, the cause of which is difficult to ascertain. Some of these are vaguely spoken of as being 'toxic.' They are not difficult to recognize, for, besides presenting all the ordinary characters of a lower neurone lesion (wasting, loss of deep reflexes, reaction of degeneration), they are characterized by being widespread and usually symmetrical affections, which at once distinguishes them from cord lesions. Further—at the outset, at least—they present more or less impairment of the sensory functions, as well as of the motor. They may be regarded as belonging to the same type of infection as polio-encephalo-myelitis.

Post-diphtheritic paralysis, which is by far the most important of the toxic neuritis group in childhood, I do not propose to speak of fully, for its study is always taken up along with that of diphtheria. Its chief peculiarity, you remember, is its tendency to pick out certain nerves, paralysis of the soft palate being one of its

earliest and most constant manifestations. Paralysis of the muscles of the eye is also not uncommon, nor, unfortunately, is that of the respiratory muscles and heart, for when these are affected the patient's condition is often very dangerous.

The cases of post-diphtheritic paralysis which you are most apt to overlook are those in which the child is brought to you for weakness of the legs, with inability to walk properly. In such a case there may be no clear history of diphtheria obtainable and no evidence of paralysis of the soft palate, and I have known great difficulties in diagnosis arise. The absence of the knee-jerks, however, ought to keep you right, for though this might be due to infantile paralysis, yet the latter is not likely to be symmetrical, and the wasting is usually much more pronounced. It is well to remember, too, that post-diphtheritic paralysis is rare before the age of two, whilst the majority of cases of infantile paralysis occur in the second year.

Facial paralysis in childhood.

The ordinary Bell's palsy so frequently met with in adults, and usually attributed to the effects of cold, is not at all common in childhood. When a child is brought to you with facial paralysis, you should think of two things: (1) that it may be an obstetrical palsy; (2) that it may be the result of middle-ear disease.

Of the first of these I have already spoken, and have pointed out that the paralysis in such cases always dates from birth, and is usually transitory, and you should have no difficulty in diagnosing it.

Facial paralysis which results from **disease in the middle ear** is by no means uncommon, and may sometimes even be bilateral; but its recognition is easy, for there is always a history of a discharge from the ear on the affected side. When both nerves are affected, the characteristic appearance of the face known as 'tapir-mouth' is produced.

Clearing out the mastoid in these cases often gives brilliant results, and I have known the paralysis begin to pass off within a few hours of the operation. Sometimes, however, a greater or less degree of permanent interference with the functions of the nerve results.

Myopathies.

Peripheral palsies the result of affections of the muscles themselves are almost peculiar to childhood, and may be conveniently spoken of as 'myopathies.' Such affections tend to be hereditary and familial. Whether the disease of the muscle in these cases is

truly primary, or, as some neurologists think, is really the result of an affection of the nerve cells of the cord, is a pathological question into which we need not enter. In any case, the paralyses of this group are far from common, even in childhood, and I only mention them here for the sake of completeness.

Cases of myopathy may be roughly divided into two groups—(1) Those in which the affected muscles, or some of them, are apparently enlarged and thickened; (2) those in which marked muscular atrophy is present.

1. **Pseudo-hypertrophic paralysis** (fig. 69) is the classical example of the first group, and its peculiarities are so carefully described in every textbook that I need not spend further time upon it.

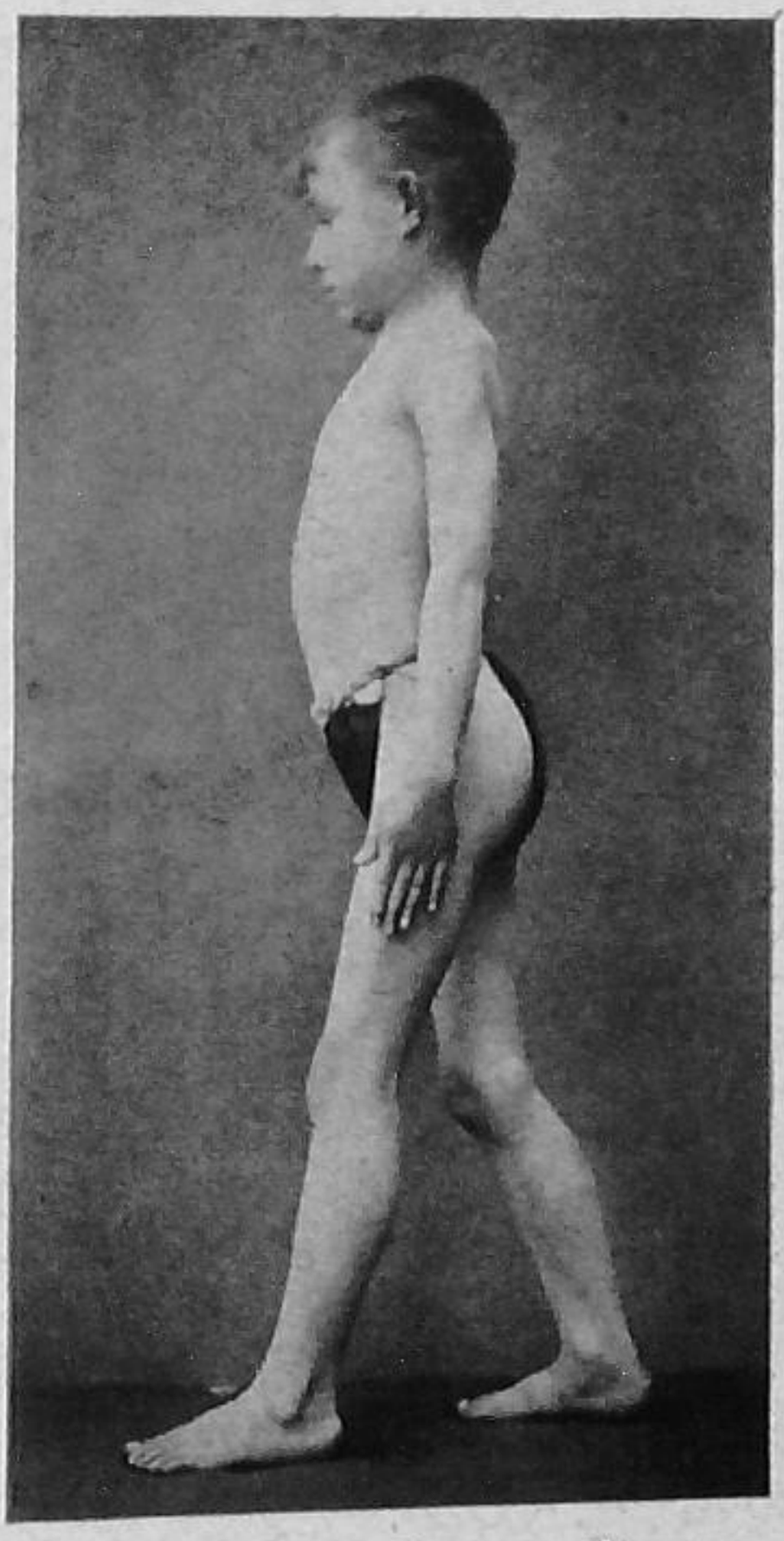

FIG. 69.—EARLY CASE OF PSEUDO-HYPERTROPHIC PARALYSIS, SHOWING ENLARGEMENT OF CALVES AND SLIGHT LORDOSIS.

2. The **atrophic cases** are divided, often rather arbitrarily, into different groups, according to the muscles affected. Thus, one speaks of the 'facio-scapulo-humeral' type, of the 'peroneal' type, and so on. In some of these cases the atrophy of the muscles and loss of power have been noted at or very soon after birth (infantile type), in others they come on gradually at some period of childhood. The degree of atrophy varies, being in some cases extreme, and various deformities of position may result, whilst the hands and feet sometimes look curiously long and thin (fig. 70). The electrical reactions may be diminished quantitatively, but usually show no qualitative alteration which marks the condition off from anterior poliomyelitis and neuritis, whilst the absence of fibrillary contraction distinguishes it from progressive muscular atrophy. Little, if anything, unfortunately, can be done to improve them, but they often exhibit but slight tendency to progress, and may survive in fair health for many years.

Apparent paralysis, the result of congenital absence of muscles or bones, may be mentioned, but if you remember the fact of its existence you are not likely to be deceived by it.

Treatment of the Paralyses of Childhood

I can promise you, I am afraid, but little satisfaction in your treatment of the paralyses which have been described in this lecture. For the most part, they depend upon organic lesions of the nervous

Fig. 70.—Myopathy of Atrophic Type.

system which it is quite beyond your power to modify or remove, and at best you can only hope in most cases to mitigate their effects.

In the treatment of the **spastic paralyses** you will find a great ally in the orthopædic surgeon, who can do much by instruments and supports of various sorts and by operation to increase the usefulness of the affected limbs. The detailed consideration of such procedures, however, would be quite beyond my province. For the fits which often ensue in these cases bromides and chloral may be given, and although I have seen it stated that bromides do no good in these cases of organic lesion of the brain, yet I can only say that I have occasionally seen great benefit from their continued use. Further, by diminishing the number and severity of the fits, one does something at least towards arresting the supervention of

mental impairment, for there can be no doubt that frequent and severe fits seriously impair the intellectual functions. Should mental deficiency be already present, it must be treated on the lines which I shall lay down in a lecture on mental deficiency in general.

For **Pott's paraplegia** I have already said that the chief treatment is rest, along with such support by a plaster jacket or otherwise as you are accustomed to see used in spinal caries. Attention to the bladder is always imperative. I have known serious cystitis set up by a neglect of this important matter.

Infantile paralysis is an infectious disease, and therefore in the acute stage the patient must be strictly isolated and a disinfectant spray used for the throat and nose, which are the probable channels of infection. Pain may be relieved by opium or aspirin, but the administration of hexamine which has been recommended to disinfect the cerebro-spinal fluid is probably useless. Convalescent serum has proved disappointing for the obvious reason that the damage to the nerve cells occurs right at the onset of the disease and it is too late then to influence the body's immunity. The limb should be placed on a light splint (e.g. of celluloid), with the affected muscles in a position of relaxation. This prevents them from becoming stretched and their opponents from becoming contracted, and thus subsequent deformity is obviated. The splint must be worn for an indefinite time both day and night, and the affected limb must never be allowed to hang down. When the acute phase is over, massage and electricity are useful. The value of the latter, however, must not be over-rated. It can certainly do no good in the long-established cases in which the paralysis has existed for some months. It has also the disadvantage of being apt to frighten the child.

Of infinitely greater importance than either massage or electricity in keeping the limb warm. This may be achieved by putting on two or more layers of stocking, fur-lined gaiters, etc., but whatever means you employ you must make a point of seeing that the temperature of the paralysed limb does not fall below that of the normal one. Any degree of voluntary movement is far more valuable than any amount of massage, and the child should therefore be encouraged to use the limb as much as possible. This can be done by putting a little collar of bells round the wrist or ankle, and getting him to ring them by moving the arm or leg; the finer movements can be developed by teaching him to prick out patterns with a pin, or

by any similar device which the intelligence of the mother can suggest.

For the club-foot and other deformities which so commonly result from infantile paralysis, you must again have recourse to surgical aid.

For **neuritis** in its early stage rest and symptomatic treatment must be your rule. Administration of vitamin B concentrates is advised nowadays. Later on, electricity, massage, and strychnine will be of help. The treatment of Erb's palsy must proceed very much on the same lines as that of infantile paralysis. *Relaxation of the affected muscles* is the most important point. This can only be done by the application of a special splint which will keep the arm abducted to about a right angle at the shoulder, and the elbow flexed to a right angle also with the forearm supinated. Pending the construction of a suitable splint, the arm in a newly-born child can be kept in nearly this position by pinning the sleeve of the vest behind the neck.

The splint should be taken off twice a day for the use of massage and passive movements. Electrical treatment is unnecessary. If there is no sign of recovery after three months, the question of operation on the plexus may be considered. Any luxation of the head of the humerus may also require surgical treatment, but these are matters for the orthopædist.

LECTURE XXIX

MENINGITIS AND ENCEPHALITIS

MENINGITIS

Meningitis is a disease which is comparatively common in childhood—much commoner than it is in the adult—but it is one which is peculiarly difficult to describe in a lecture, and I feel far from confident of my ability to give you anything like a clear description of it. This is due to the great variability in the symptoms which the disease presents—a variability which is to be ascribed to variations in the extent to which the meninges are involved in any given case, and to the variety of the agents which may excite the inflammation. Several different micro-organisms are capable of setting up meningitis. Chief of these are the meningococcus, the tubercle bacillus, the pneumococcus, the influenza bacillus, and the pyogenic organisms, and any of these may be found present alone, or 'mixed infections' may occur.

General Symptoms

No matter what its exciting cause may be, inflammation of the membranes of the brain and cord results in the production of a group of **symptoms which are common to all forms of meningitis,** though one or more of these symptoms may dominate the picture in any given case of the disease. The symptoms may be roughly divided into two groups: (1) Those which result from irritation of the brain; (2) those which are the consequence of increased intracranial pressure.

1. Amongst the **irritative symptoms** the most prominent are mental irritability—manifested in its slighter degrees by mere peevishness, and in its more pronounced forms by signs of great irritation when roused—screaming, headache, convulsions, vomiting, irregularity of the pulse and respiration, photophobia and contraction of the pupils, and sometimes optic neuritis. There is also apt to be an increase of the 'tone' of the muscles, showing itself by exaggeration of the reflexes, by muscular twitchings,

by the presence of Kernig's sign and, in extreme cases, by opisthotonos.

2. Of the '**pressure signs**' the most important are lethargy and drowsiness culminating in coma, bulging of the fontanelle, slowing of the pulse and respiration, dilatation of the pupils, and changes in the optic discs.

The special symptom known as '**head retraction**' is often one of the most prominent features of meningitis, and it is difficult to know to which of the above groups to refer it. The photograph (fig. 71) will give you a better idea of it than any verbal description. It is probably the result of irritation of the posterior cervical nerves, and is most marked in cases of posterior basic meningitis.

Diseases simulating Meningitis

Now, you may take it as a general rule in medicine that the more numerous the symptoms of any disease the more difficult is it to diagnose that disease with certainty, for the greater is the chance of its being simulated by something else. To this rule meningitis is no exception, and you will often have great difficulty in being sure that you are dealing with it, particularly in its earlier stages. But forewarned is forearmed, and if you know what the traps are which you are most likely to fall into you will have a better chance of avoiding them. The diseases, then, for which meningitis is most apt to be mistaken are these:

1. **Typhoid fever.**—By one form of typhoid fever—that known as the cerebro-spinal variety—meningitis may be closely simulated. All the signs of cerebral irritation—mental irritability, head retraction, photophobia, and the rest—may be present in a marked degree, and yet the case may prove to be one of typhoid after all. Not long ago, indeed, a case occurred in which the mistake was only discovered in the post-mortem room. Pay great attention to the state of the abdomen in these doubtful cases. In typhoid it is usually full and rather tender; in meningitis it tends to be retracted and not tender. The Widal reaction also may be decisive.

2. **Pneumonia,** especially apex pneumonia, may simulate meningitis very closely, particularly if head retraction is present, as it sometimes is. Careful examination of the lungs may disclose the characteristic physical signs, but these may be absent in the first few days. The pulse-rate may help you here. In pneumonia it

follows the temperature, whereas a combination of high fever with a comparatively slow pulse is more suggestive of meningitis.

3. **Acute gastritis** may be accompanied by many meningitic symptoms, such as high temperature, vomiting, convulsions, and constipation. The tongue here, however, is usually thickly coated, and a dose or two of calomel will probably clear everything up.

4. **Middle-ear disease** has often been mistaken for meningitis. Indeed, in a doubtful case it is not a bad plan to puncture the membranes on chance. The simulation here is all the more puzzling, inasmuch as middle-ear disease may be followed by secondary meningitis as one of its complications.

5. **Acute poliomyelitis** or **polioencephalitis** may produce a clinical picture which is the exact replica of that of meningitis. I saw such a case lately in which it was impossible to be sure for some time what we were dealing with—vomiting, head retraction, swelling of the discs, hypertonicity, all were present—but final recovery, with the exception of some spasticity of the legs, made it extremely probable that it had been an encephalitis all along.

6. **Lethargic encephalitis** may simulate meningitis very closely, especially in the early stage. The comparative absence of fever and of marked head retraction, the presence of ocular paralyses and a normal lumbar-puncture fluid are all points in favour of encephalitis.

7. **Acute serous meningitis** is probably not a true inflammation at all, but the cerebro-spinal fluid is under increased pressure and the whole picture resembles a mild case of tuberculous meningitis until detailed examination of the fluid removed by lumbar puncture shows that the composition is little changed from normal. I shall deal with these problems a little more fully at the end of this lecture.

All of these conditions may exhibit more or less marked head retraction, but it is important to remember that this sign may be mimicked by more trivial affections. Quite recently, for example, a boy was admitted with so much rigidity of the neck that he was thought to be suffering from tuberculous meningitis following cervical caries. Yet it eventually proved to be a case of **rheumatism** affecting the cervical muscles or some of the vertebral joints. It is also said, although I have never seen an instance of it myself,

that a mere **fall on the head**—apart from any actual injury—may produce temporary head retraction in young children.

Should you find yourself puzzled in any of these ways, it is well to remember that in **lumbar puncture** you have a diagnostic aid which may save the situation. This little operation is easily performed, and, if carried out under the observance of strict asepsis, is quite devoid of risk. An anæsthetic is not really necessary. Place the patient on his side, with an assistant to bend the spine and open out the spaces between the laminæ of the vertebræ. Trace the last rib on the right side back to the twelfth thoracic spine, and count down from this to the third lumbar. Place your left thumb on the third interspinous space, and enter a small trocar and cannula $\frac{1}{3}$ inch to the right of it, and pass it inwards and slightly upwards for a depth of $\frac{2}{3}$ to 1 inch, depending on the age of the patient and the thickness of his subcutaneous tissues. Withdraw the trocar, and cerebro-spinal fluid will usually exude in drops, or, if there is increased pressure, in a continuous stream. Some of this should be received into a sterilized test-tube.

There is no risk of injuring the cord, for you are altogether below its level, but the appearance of a little blood need not alarm you.

Normal cerebro-spinal fluid is perfectly clear, like distilled water. Turbidity indicates meningitis, though the opacity may be so slight that it is only noticed when the tube is shaken and held up to the light. Examination by culture, or by merely making stained films of the deposit, may reveal the presence of the meningococcus which may be recognized in the films lying inside the leucocytes (fig. 72), or of the pneumococcus, or of one or other of the pyogenic organisms, or even of the tubercle bacillus, although in tuberculous cases the fluid is often sterile, just as it is in cases of tuberculous pleurisy. The ' cell-count ' in the fluid is also of help. Excess of lymphocytes points to tuberculosis, whereas in other forms of meningitis the polynuclears predominate. Estimation of the chlorides also gives assistance because the level is lowered in meningitis, especially in the tuberculous form.

Varieties of Meningitis

I may now pass on to describe very briefly the different varieties of meningitis, adopting the following provisional classification :

1. Acute { Primary { Epidemic. / Sporadic. / Secondary { Epidemic. / Sporadic.

2. Tuberculous.

3. Posterior basic (also known as ' simple basal,' and really only one form of sporadic cerebro-spinal meningitis).

Acute meningitis may occur either as a primary disease or be secondary to some other condition.

Primary meningitis may be due to the *meningococcus* and occur either in epidemics (cerebro-spinal fever) or sporadically. In either case the onset is sudden with high temperature, headache, and vomiting. Rigidity of the neck is common and convulsions may supervene. There may also be a purpuric rash—hence the term ' spotted fever '—and delirium or stupor may occur. In other cases primary meningitis is due to the *influenza bacillus* and, occasionally, to the *pneumococcus*. The symptoms in either are very much the same as those due to meningococcal infection and the exact diagnosis can only be made by lumbar puncture.

Secondary meningitis comes on in the course of some other—and often acute—disease, which may to a large extent mask its symptoms. It is therefore often difficult to recognize, and may, indeed, be only detected after death. I remember, for instance, having a child under my care for whooping-cough who suddenly developed a left-sided hemiplegia. Everyone who saw the case supposed, naturally enough, as there were no other marked symptoms, that a blood-vessel had given way in the brain during a paroxysm of coughing. Yet examination after death showed that we were wrong. There was no hæmorrhage, but the paralysis was due to an extensive exudation of semi-purulent lymph on the surface of one side of the cortex.

I have known the same sort of thing happen in the course of a pneumonia, and it may occur also in measles or in any of the acute specific fevers. Not infrequently, too, meningitis is set up by suppuration in the middle ear.

In all these cases the onset of the meningeal inflammation may be difficult to recognize, though it is perhaps oftener ushered in by convulsions than by any other sign. Its recognition is important and is made by prompt lumbar puncture in any doubtful case. The prognosis has been entirely altered by the introduction of the new sulphonamide drugs. The organisms chiefly responsible for secondary meningitis are the pneumococcus, influenza bacillus, streptococci and staphylococci.

Tuberculous meningitis.

The mistake you are most likely to make in regard to tuberculous meningitis is in diagnosing it too often. One thing I would specially desire to impress upon you in this connection is that *tuberculous meningitis is rare in the first year of life, unless as the terminal process in a general tuberculosis.* It is in the second and third years of life that primary tuberculous meningitis, just like tuberculosis in general, reaches its maximum degree of frequency.

I shall not attempt to draw for you a **clinical picture** of this disease, for the reason that it would be of no real help to you if I did. At best I could only give you a sort of composite photograph, which would be very unlikely to correspond at all closely with any given case you might afterwards meet with. Tuberculous meningitis is essentially one of those diseases which can only be studied at the bedside, and by the careful observation of many cases. You will then be able to form for yourselves some idea of the extraordinary variability of the course of this affection, and of the extent to which the symptoms may depart from the classical type in one or other direction. This **classical type** was laid down by Whytt more than one hundred and fifty years ago. He divided in into three stages—a *prodromal* stage, marked merely by peevishness, irritability of temper, and fretfulness, along, perhaps, with slight elevation of temperature, loss of appetite, constipation, and, if the child is old enough to complain of it, headache. This stage passes after a varying number of days—or even, in very insidious cases, after two or three weeks—into the *second* stage, characterized chiefly by slowness and irregularity of pulse (the typical 'cerebral pulse'), the other symptoms meanwhile becoming more pronounced. After a short duration, this is succeeded by the *third* stage, in which the pulse becomes very rapid, the temperature continues to rise, and coma supervenes.

These three stages may often be roughly traced in cases of tuberculous meningitis, but not infrequently one or more of them may be omitted, or be so transitory as to be overlooked. The individual symptoms of the disease will be considered immediately, when I come to describe its differential diagnosis from posterior basic meningitis.

Your difficulty will be to say in an early case whether the child is suffering from meningitis or not. It is sometimes quite impossible to be sure, and perhaps the safest course in such circumstances is

to follow the practice of an old doctor of my acquaintance and say: 'The child is suffering from cerebral irritation; it *may* pass on into inflammation (meningitis), but we shall do all we can to prevent its doing so.' This 'heads I win, tails you lose' form of diagnosis usually satisfies the parents.

Posterior basic meningitis.

Posterior basic meningitis is so called because on examination after death the inflammatory exudation is found to be most marked over the posterior part of the base of the brain. The term, it must be confessed, is an unfortunate one, for the inflammation is not really *confined* to that part of the meninges, as the name might lead you to suppose, but for purely mechanical reasons that happens to be the spot where it is most easy for the inflammatory exudate—which is rather abundant in this form—to accumulate. From a strictly pathological point of view it would probably be better to speak of it simply as a form of **sporadic cerebro-spinal meningitis,** for the exciting cause of the inflammation is the same in both cases—viz. the meningococcus. Still, the term has received general recognition by clinicians, and it is as well for us to continue to use it for the present.

Posterior basic meningitis, unlike the tuberculous form, is most common in the first year of life, though it may occur at any age. This fact is so important that I wish to impress it upon you as an aid in diagnosis. It is another example of the value of what I am in the habit of speaking of as *reasonable probability in diagnosis*, which means that in a doubtful case you should lean to that disease which is most commonly met with at that age. Given, then, a case of primary meningitis in a young infant, the probability is in favour of its being the 'posterior basic' variety.

Of the **general symptoms** common to all forms of meningeal inflammation already detailed, some tend to show in this variety a peculiar prominence. Chief of these, undoubtedly, is **head retraction,** which exhibits in posterior basic meningitis a degree of development not met with in any other form. The child depicted in fig. 71 shows this in a marked, but by no means unusual, degree, and you will recognize from the photograph the appropriateness of the description given to the attitude by French writers—viz. 'the *gun-hammer* position.' In severe cases in which **opisthotonos** is present as well, the retraction may be so extreme that the occiput almost touches the heels.

Along with head retraction, and usually proportional to it, there is **bulging of the anterior fontanelle,** which forms one of our best gauges of the increase of intracranial pressure. Whenever the intracranial pressure is high, **vomiting** is apt to set in, and it may continue with intermissions for weeks. At the same time, great emaciation is prone to occur, which is not, however, so far as one can see, the consequence of the vomiting, but is rather an essential

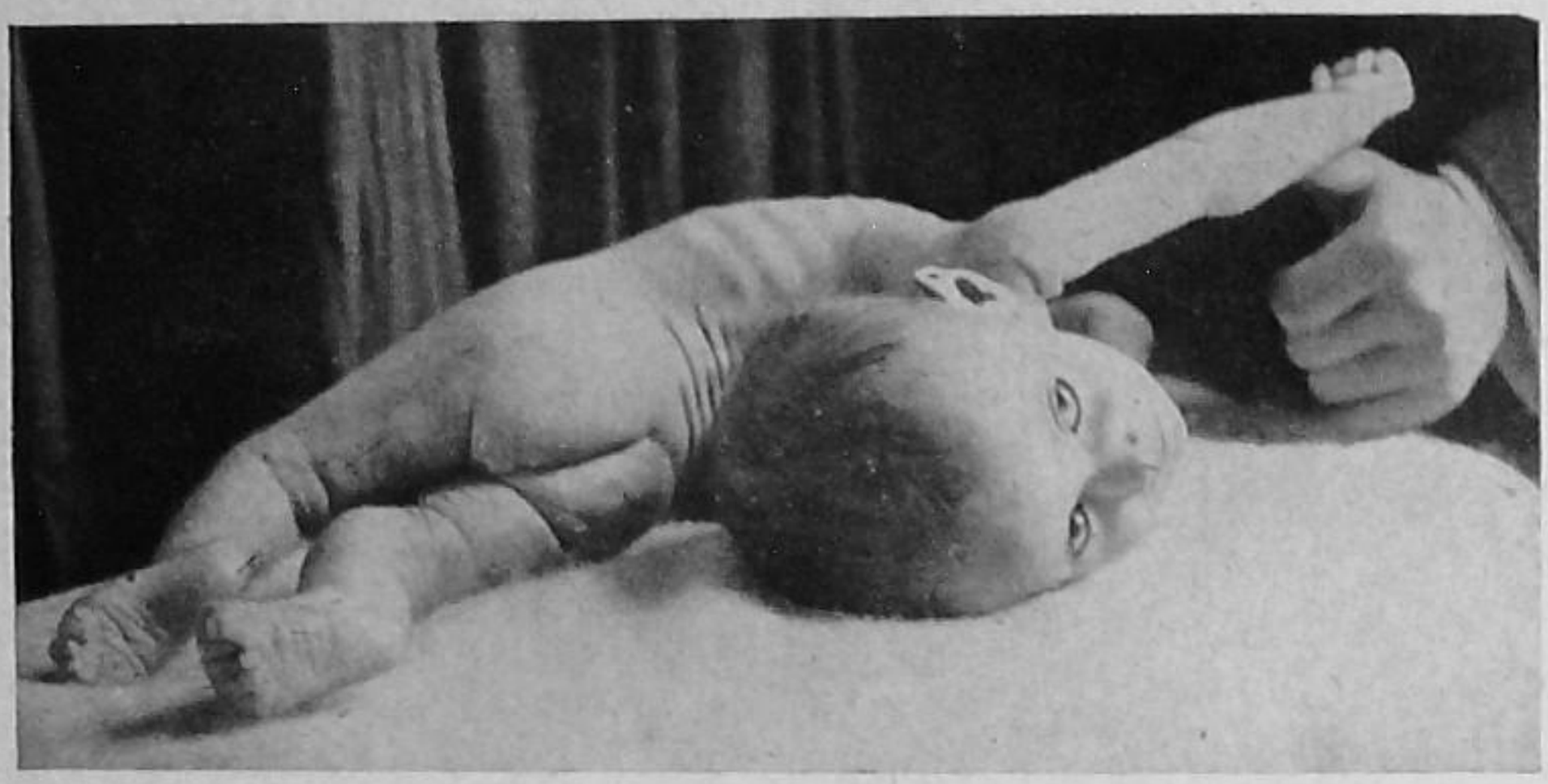

FIG. 71.—HEAD RETRACTION IN SIMPLE BASAL MENINGITIS, SHOWING 'GUN-HAMMER POSITION.'

Note also the staring eyes.

symptom of the disease. Failure to lose weight, therefore, is always a sign that the malady is taking a favourable course.

Occasionally in a young infant, say under six months, the presenting symptoms strongly suggest a digestive upset. I have more than once made an initial error in diagnosis because of the dominance of vomiting and apparent pain. Only later did the hard retraction and bulging fontanelle give real clues.

The other signs and symptoms of this form of meningitis you will gather from the following table, in which they are contrasted with the corresponding features of the tuberculous form:

DIFFERENTIAL DIAGNOSIS OF POSTERIOR BASIC AND TUBERCULOUS MENINGITIS

Posterior Basic.	*Tuberculous.*
Common in the first year.	Rare in the first year.
Temperature may be high at first, but falls later.	Temperature rises as disease progresses.
Head retraction early, and marked.	Usually much less prominent.
Wasting usually a prominent feature.	Not marked unless in consequence of general tuberculosis.

Posterior Basic.	*Tuberculous.*
Constipation usually absent.	Usually pronounced constipation.
Pulse and respiration not much affected.	Usually exhibit marked alterations, varying with the stage of the disease.
Eyes staring and lids retracted.	Tendency to photophobia and spasm of the lids.
No marked fundal changes.	Papillœdema frequent.
Cerebro-spinal fluid shows the meningococcus and excess of polynuclears (fig. 72).	Cultures sterile, or show the tubercle bacillus. Lymphocytes predominate.

The **prognosis of posterior basic meningitis** is also better than that of the tuberculous form, for whereas the latter may be regarded as almost inevitably fatal, the former is sometimes recovered from. Unfortunately, however, recovery is by no means always complete. There is apt to be left behind as the permanent legacy of the disease a greater or less degree of **chronic hydrocephalus,** with more or less spasticity of the limbs. Many cases of hydrocephalus coming on in infancy, the cause of which cannot be traced, have probably arisen in this way. Permanent **mental impairment** may also result, and the special senses be damaged. A peculiar **blindness,** without visible changes in the fundus, often lasts for quite a long time after the other symptoms have subsided, though I have never known a case in which it was permanent. On the other hand, lasting deafness may, I believe, occur.

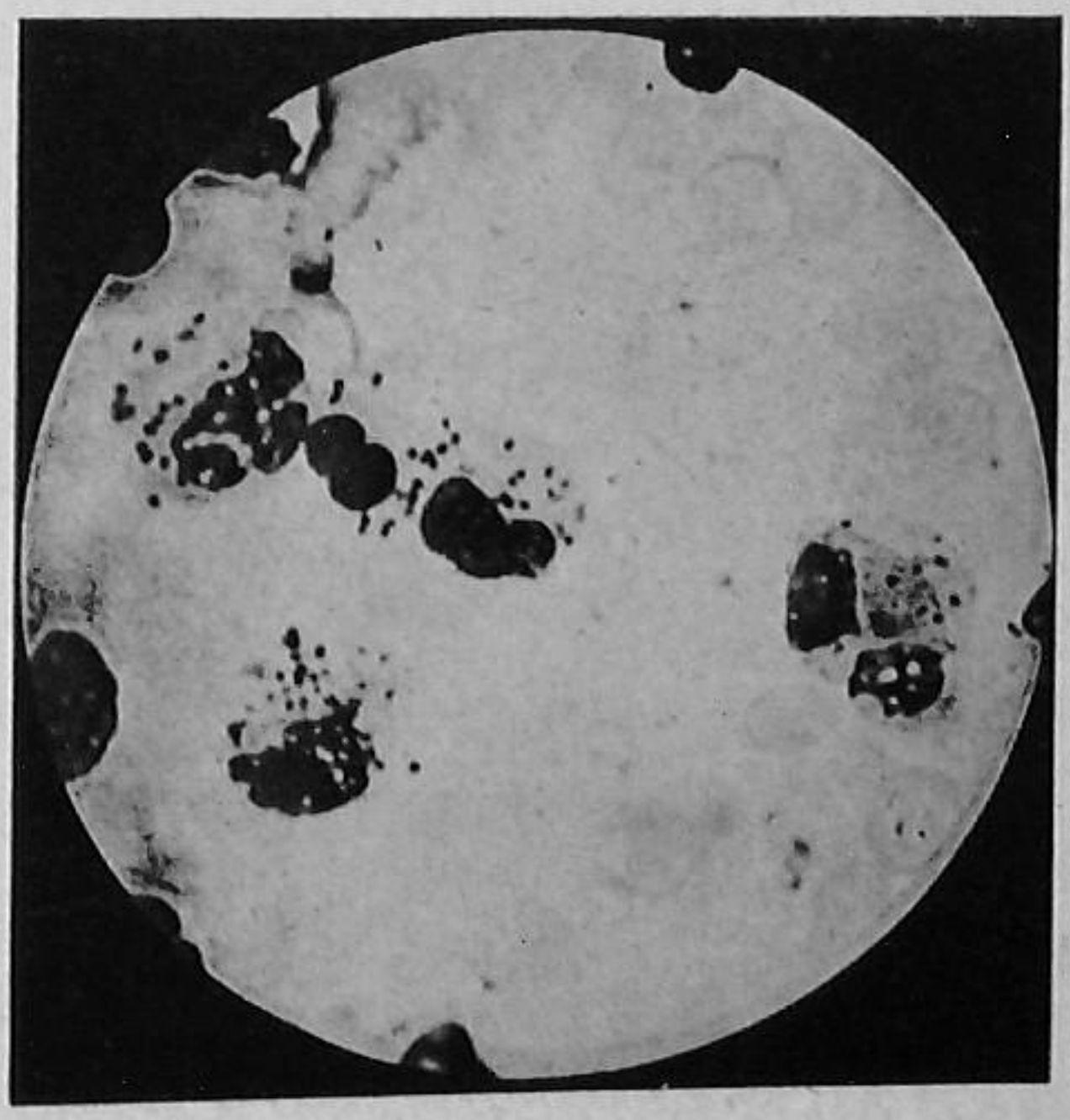

FIG. 72.—PHOTOMICROGRAPH, SHOWING THE MENINGOCOCCUS EMBEDDED IN LEUCOCYTES.
From lumbar puncture in a case of simple meningitis.

TREATMENT

Tuberculous meningitis is invariably fatal. All you can do is to see that the child is kept comfortable, with skilful nursing, and lumbar puncture to relieve excessive pressure. Meningitis due to the meningococcus and to the other pyogenic organisms producing secondary meningitis is amenable to chemo-

therapeutics. You should use the variety of sulphonamide appropriate for the causal organism and push it in full doses along the lines I have already talked to you about in the lecture on pneumonia (see p. 254). If vomiting is troublesome, a soluble form of the drug should be used. It is too early yet to be sure if this remarkable drug treatment is going to prevent some of the complications of meningitis in the past—such as a subsequent hydrocephalus. For that reason some urge that serum treatment should also be used for meningococcal cases, for example, given into the spinal canal after withdrawal of slightly more cerebro-spinal fluid than the proposed dose. Lumbar puncture and cisternal puncture should still be performed as required to relieve increased intra-cranial pressure. Skilled nursing is required to secure that infants and children receive adequate nourishment. It may be necessary to administer this by a tube (through the nose) and if the child is unconscious the sulphonamide tablets will have to be given, crushed up, in this way.

ENCEPHALITIS

After the 1914–18 war a new disease appeared which was then called *encephalitis lethargica* and now officially termed *epidemic encephalitis*. Actually since about 1925 it has ceased to be epidemic in this country and only rare sporadic examples crop up. But other forms of encephalitis have made their appearance, and it is now possible to discuss different varieties, largely according to the pathological findings. There are, broadly speaking, three main groups—those due to a virus; those associated with a demyelinization in the nervous system; and those perhaps better called the 'encephalopathies,' where there is no true inflammatory reaction in the brain.

First in the **virus** group, let me briefly describe the so-called lethargic type. Fever and headache appear suddenly and are quickly followed by ocular palsies, lethargy and a curious disturbance of the sleep rhythm, with the patient anxiously wakeful at night and comatous by day. After a week or so the acute phase begins to pass off: rarely it proves fatal, but more often nowadays it is so mild that it is called 'influenza.' But soon, or sometimes after a long interval, the chronic stage begins and you get a curious picture of generalized rigidity and an emotionless face just like what we call Parkinsonism or paralysis agitans in old people. With children this

is accompanied by a slow deterioration and often there are changes in the character producing a moral imbecile or the 'apache' type to which I have already referred (p. 264).

Polioencephalitis, as its name implies, is caused by the same virus as produces poliomyelitis when it attacks the spinal cord. I have already mentioned how closely it may mimic meningitis. Often the onset is ushered in with a severe bout of convulsions and the child recovers with a hemiplegia or monoplegia, showing that the motor cells of the cortex have suffered in the attack. Some authorities are inclined to doubt the existence of this type, but we know how the bulbar region of the brain may be attacked by the poliomyelitis virus and I see no reason to doubt that motor cells higher up are also vulnerable.

Benign lymphocytic meningitis is likely to give you most trouble. I called it 'serous meningitis' earlier on, and actually both names are misleading because a true encephalitis is probably present. Some evidence has been produced that it is caused by a virus conveyed by mice, and I well remember a doctor's son who had had a severe attack shortly after the family went to live in an old house overrun by mice. It begins acutely with all the signs of meningeal irritation—fever, vomiting and a curious wakefulness. The neck is stiff, but involvement of the cranial nerves is rare. Despite its alarming beginning the outlook is good, with complete recovery.

These three common types of virus encephalitis will suffice to show the variability of the clinical picture. But it is only fair to point out that in the past twenty years epidemics have been described in America, Australia and Japan associated with viruses thought to be different from those already mentioned. Also some think that in mumps there is a tendency for the virus to attack the nervous system early in the disease, but I have no personal experience of this association.

The **demyelinizing** type of encephalitis is of importance because of the association with vaccination and with some of the acute specific fevers such as measles, chickenpox and even rubella. It generally begins in the second week of the fever or up to fourteen days after the rash appears (or the vaccination was performed). There is a sudden onset of fever with headache, vomiting, neck stiffness, with convulsions in the younger children, giddiness and sometimes an increasing drowsiness and coma leading to a fatal termination, often as frequently as in about one-third of all cases occurring,

as they often seem to do, in small epidemics. Ocular palsies are rare but facial paralysis is common, and so is a curious trismus or stiffness of the jaw. In severe cases all varieties of hemiplegia and paraplegia occur and the sphincters become paralysed. Despite this alarming picture, however, recovery is often surprisingly complete.

I have attempted to draw a composite picture of this group, but I want to say a little more about the post-vaccinial type. The exact reason for the development of this alarming complication of vaccination is not completely understood—any more than why encephalitis sometimes follows measles or chickenpox. But we do find that it is rare in infancy: the number of *proved* cases under six months is extremely few and most cases reported at this age can be shown to be something else. The real danger appears to come with *primary* vaccination after the age of infancy, and so we have a good reason for urging infant vaccination at an age when the risk of encephalitis is slight. In other words, since no-one quite knows if a baby is one day going to meet small-pox in foreign countries and therefore *have* to be vaccinated, it is well to get vaccination over in infancy as a protection not so much against the risk of small-pox now, in this country, but against the risk of having to have a primary vaccination at an older age! Encephalitis after re-vaccination is unknown.

One final word about this demyelinizing group: there are pathological resemblances to a special type of mental deterioration coming on in later childhood called Schilder's disease, and to the findings in disseminated sclerosis in adults.

Now we come to the third group which I like to call '**acute toxic encephalopathy.**' Here, in fatal cases, we find no true encephalitis but degeneration of the ganglion cells together with congestion and œdema. It may occur with any acute infection, usually shortly after the onset, but is especially likely to occur with otitis media and apical pneumonia. You will find neck stiffness, earache and delirium, but very little else in most cases. The milder degrees of this are almost certainly, in my opinion, what we have in the past called 'meningism,' and the difference between it and 'acute benign lymphocytic meningitis' is often not to be detected in those that recover, as fortunately most patients do.

The general **diagnosis** of all those forms of encephalitis is made by lumbar puncture. The fluid will be under pressure but clear, and on chemical examination the chlorides and glucose are essentially normal, whereas the cells are usually increased to a greater or less

degree. The highest cell counts—mostly lymphocytes—are found in the benign lymphocytic meningitis group. Another point is that the fluid is sterile on culture. Clinically the onset is in all instances more abrupt than with tuberculous meningitis with which you will have the most difficulty. Lumbar puncture will give you a prompt answer with the help of the laboratory. Specific diagnosis within the group will be made by attention to the special features which I have tried to emphasize—the clinical pictures I have drawn for you.

As regards **treatment**, you will find in the mild cases that nothing is required beyond careful nursing. Lumbar puncture should be freely used if there is much headache or neck stiffness. Sedatives, such as phenobarbitone, can be given if there is much restlessness or wakefulness. Convalescent serum may become useful when we know more about some of the types: at present it is used for the post-vaccinial group because it is easy to find an adult donor who has recently been vaccinated. You can give repeated doses of 10 to 20 c.cm. subcutaneously (*never* intrathecally). In the later stages of lethargic encephalitis with Parkinsonism the drugs of the stramonium group are reputed to help.

LECTURE XXX

ON MENTAL DEFICIENCY IN CHILDHOOD

Mental deficiency in childhood is a subject which deserves your close attention, because you will find when you go into practice that it is not uncommon, and you will frequently be consulted about it. Moreover, it is a subject in regard to which if you make a mistake you are likely to get into discredit. The frequency of mental deficiency in children you will realize when I tell you that about 1 per cent. of all children of school age are the subjects of it.

Before going further I want to define for you the **use of terms** in regard to mental deficiency. You will find such words as idiocy, imbecility, feeble-mindedness, and backwardness all used with regard to children whose mental development is defective. At the present day the use of the first three of these terms has been practically discontinued, and the expression ' mental deficiency ' is used in place of them. But if you are going to adhere still to the use of the term ' idiocy,' you should reserve it for the lowest grade of case—that is to say, those in which the mental impairment is of the extremist form, and in which the child is incapable of guarding itself from physical danger. Above those, somewhat better in their mental development, but quite incapable of earning their own living will come imbeciles, and higher still, nearer to the normal children, and capable of doing simple work under favourable conditions, will come those who can be called the feeble-minded. Backwardness is a thing to be sharply distinguished from mental deficiency, and therefore we shall still retain that term, and I shall define what is meant by it later on. There is a tendency nowadays to drop these terms and to classify defective children according to their intelligence quotient (I.Q.). This is estimated by giving the child a series of tests and expressing its attainment in terms of its ' mental age ' as a percentage of its ' chronological age.' Generally speaking, a mentally defective child will have an I.Q. of 80 or less.

' Moral imbeciles ' are children in whom there is exhibited from an early age a tendency to moral obliquity (e.g. thieving, cruelty, lust, etc.), which persists in spite of correction and deterrent punishments. Such children are usually defective on the intellectual side

also, but not necessarily so, and they are often a source of great trouble and grief to their relatives. *Acquired* moral imbecility may occur as one of the most tragical sequelæ of encephalitis lethargica in childhood, as mentioned in a previous lecture.

Coming closer to our subject, it will be convenient to look, first, at the general diagnosis of mental deficiency; secondly, at its clinical classification; thirdly, at the etiology so far as we know it; fourthly, at the prognosis; and, fifthly, at its treatment. I shall begin with the diagnosis.

Diagnosis of Mental Deficiency

When you wish to diagnose a mental condition, even in an adult person, it is well to put to yourself three questions: What does the patient say? What does he do? What does he look like? In diagnosing mental deficiency in children, you will find that these three modes of attacking the problem will be of great service.

First, then, what does the patient say? Speech, of course, is the great mode of intellectual expression; it is one of the things by which you can judge best of a man's intellectual development. In the case of children who are mentally deficient, you will find often that it is not so much what the patient says as what he does *not* say which is helpful to you, and which will arouse your suspicion. You will remember that by the age of three a child ought to have learnt to talk fairly well; and if a child is brought to you at that age who is still **unable to talk,** you may conclude either that you are dealing with a condition of mental deficiency, or that the patient is deaf. There is a third possibility, but so uncommon as to be scarcely worth considering—namely, that the child may be the subject of what is called 'congenital aphasia.' But that is very rare. So practically a child of three years of age who cannot talk is either deaf or mentally defective. As regards what one may call *qualitative* alterations of speech, I would only point out that you must not conclude that such peculiarities are necessarily signs of mental deficiency. Take such a thing as *stammering*. This is by no means an indication of mental weakness; indeed, stammerers are often above the average of intellectual development. There is another condition, termed *idioglossia*, in which the child talks in a sort of gibberish of his own, which is entirely unintelligible to other people; and that also, although you may think it is an indication of mental deficiency, is not so. These children are often

otherwise intellectually sound. On the other hand, the condition of speech called *lalling* is certainly suspicious of mental deficiency, and another peculiarity certainly exhibited by some defective children is *repetition*; they have a habit of repeating after you the question which you ask them or the things which you say.

Next, let us consider what the child *does* which arouses your suspicions. I would point out that one great sign of mental deficiency is an **inability to perform** at the proper times complicated **co-ordinated movements.** The child will therefore be late in passing many of those 'milestones of development' of which I spoke in the first lecture. He will be late in holding up his head; instead of being able to co-ordinate the muscles which hold the head on the trunk by the third month, he may not be able to do so until he is a year old, or possibly more. He will be late also in sitting up; instead of being able to sit up at the end of the sixth or the ninth months, it will perhaps not be until the eighteenth month that he can do so. The child will also be backward in walking, and, as we have just seen, in performing the co-ordinated movements of speech. Further, mentally deficient children are apt to fail in the power of grasping things; they are wanting in the power of *acquisitiveness*, as it has been said, for grasping involves complicated co-ordinated movements by the muscles of the arms, and so you are not surprised to find that it is a function which is acquired unusually late by children whose cerebral development is defective. There will also be a lack of co-ordination in the muscles which move the eyeball and which enable the child to look fixedly at any object. Hence the gaze is vacant.

Amongst positive motor signs which will arouse your suspicion is the performance of rhythmic movements. These are a common indication of mental deficiency. For instance, there are swaying movements of the body, jerking of the shoulders, or grimacing movements. Such movements as those, constantly repeated, should arouse your suspicion with regard to the mental condition of your patient. There may also be certain tricks of movement which are suggestive. I refer to such things as hand-sucking and the making of meaningless gesture. Great restlessness is also a common sign. This often goes with sleeplessness at night, and that again with apparently causeless screaming or crying.

We now come to what the patient *looks like*. The **facies** or *facial expression* of the child is, after all, perhaps the most valuable

index which you have of the mental state. I told you that the face in children is a mirror of the mind in a way that it is not in grown-up people, and there is no class of case in which that is of greater service to you than in the mentally defective. So much is this the case that many forms of mental deficiency in children have an absolutely characteristic facies, so that you can say at once, 'This child is mentally deficient, and belongs to such and such a class.' We shall see immediately when we speak of classification that one makes large use of this in the sub-division of cases of mental deficiency into different clinical groups. But, apart from those which have a characteristic facies, you will find in almost all mentally deficient children *something* in the expression which will arouse your suspicion. There is a vacant look, or perhaps grimacing, or an inability to recognize the mother or the nurse. All these things should put you on your guard. You will also find that the size and shape of the *skull* is often of great service to you in enabling you to arrive at a diagnosis, because, just as in facial expression, so with regard to the skull, there are abnormalities which are absolutely characteristic.

Then you will find, in looking more carefully at such children, that you are apt to get what are spoken of as the **physical stigmata** of intellectual defect, which were described by Dr. Warner under what he termed 'the law of coincident development.' This simply means that if one part of the body is defectively developed, other parts are apt to suffer also. If, for instance, the child has a badly-developed brain, he is more likely than other children to have a congenital lesion of the heart, or supernumerary digits. Amongst the physical stigmata of mental deficiency are what are termed marked epicanthic folds—that is to say, folds of skin which come down from the upper eyelid to the lower, and cover over the caruncle. A high-arched or Gothic palate is another common physical stigma, and it frequently goes with narrowness of the nasopharynx and bad development of the jaws, and therefore also of the teeth. Then you will find adherent lobes of the ears more common in mentally deficient children than in others, the lobe being tacked on to the skin of the cheek instead of being left free. You find, too, not uncommonly, harelip, cleft-palate, and opacities of the media of the eyes. In a doubtful case you will look for such physical stigmata, and the finding of them will be in favour of the view that the child is mentally defective.

Classification of Cases

We next come to the second division of the subject—namely, the *clinical classification* of cases of mental deficiency. One would prefer to use a pathological classification if that were possible, but at present it is not. The pathology of some types is still quite obscure, and you will find it, as a practical matter, more convenient to group the cases according to the chief clinical symptoms and features which they exhibit. We shall begin with those cases which present a marked facies.

Fig. 73.—Baby Cretin.
Note protruding tongue and large umbilical hernia.

1. The first group of those are the **cretins.** It is important that you should be able to recognize cretins, because they form the group of mentally defective infants which are the most amenable to treatment; and it will depend upon your being able to make the diagnosis early what the child's future will be. The recognition of a baby cretin is not always easy, and the classical pictures of cretins which you see in books are usually taken from children several years old, by which time, of course, anyone can make the diagnosis; but if you recognize them only then, you recognize them too late. In order to do a cretin real good, you have to catch him early, and if you do so, and treat him properly, he may grow up into a child who is practically indistinguishable from a healthy and normally developed one.

What, then, are the features by which you will recognize cretinism in its early stages? Before I reply to this I would say that cretins

are not so common as is supposed. There is too great a tendency to label almost every mentally defective child a cretin. Out of many mentally deficient children of which I have notes, only 5 per cent. were examples of cretinism ; whereas Mongolism, which it is very apt to be mistaken for, occurs with much greater frequency, amounting to about 35 per cent. of my cases.

With regard to the cretin, then, the complexion is parchment-like in tint, the hair dry and scanty, and the skin redundant, so

FIG. 74.—CRETIN, SHOWING CHARACTERISTIC FACIES.

that there is a puckering of the forehead and puffiness below the eyes. There is often a squint, but by no means invariably. There is rarely, if ever, nystagmus. When you look at the shape of the head, you find it tends to be long : it is dolichocephalic. On examining the trunk you will find that the skin is dry and rough. There is frequently an umbilical hernia ; the hands are short and broad, and the finger-tips square. In manner cretins are dull and lethargic, and a feature which you will recognize in them from earliest life is that they suffer extremely from constipation. I have

said nothing of the supraclavicular pads of which you will read in books, for the reason that they are rarely developed, so far as I have seen, until a cretin is at least a few years old; they are not seen in the earliest stage, and, although it is perfectly true that you get them afterwards, yet by that time it is too late for them to be of use in diagnosis. I should, perhaps, have mentioned as a characteristic feature the tendency to protrusion of the tongue. The tongue is large, broad, and thick, and it is apt to be kept protruded. All those characteristics taken together make up a picture which, if you have once seen it, you are not likely to miss again.

2. Now I pass to consider **mongols.** The term 'mongolism' was first applied to this group of cases by Dr. Langdon Down, from their resemblance to members of the mongolian races—the Chinese and Japanese—in face, and particularly because they have the oval tilted eyes which are characteristic of these nations. When you come to compare their characteristics with those of the cretins, with which they are apt to be confused, you will find that the skin of the mongol is smooth and white, not parchment-like in tint, and that they have a good complexion. They have no puckering of the eyebrows and no puffiness of the eyes, the hair is fine and abundant instead of being dry and scanty, and they almost always have a squint and usually nystagmus as well. The head, instead of being dolichocephalic, is brachycephalic, and the occiput is almost in a direct line with the neck; whereas the cretin has a head projecting back beyond the neck. The tongue may protrude in both. The finger-tips are rounded, and they have a curious incurving of the little finger. Moreover, instead of being dull and lethargic, they are bright and lively in manner. So you see they contrast in every respect with the cretins, and if you have seen a good

FIG. 75.—BABY MONGOL.
Note protruding tongue and typical condition of the eyes (big epicanthic folds).

FIG. 76.—MONGOL IN PROFILE, SHOWING SHAPE OF HEAD.

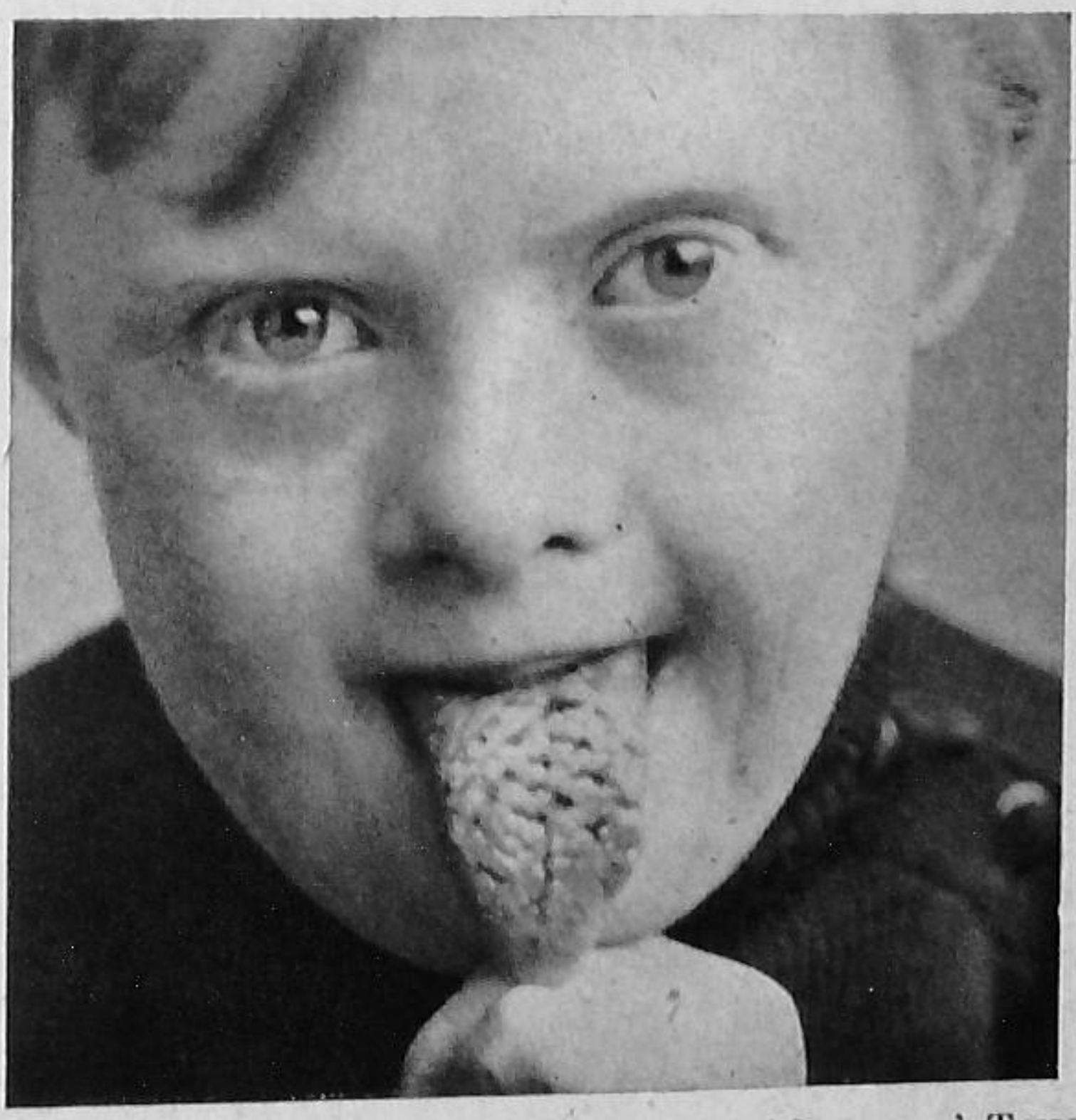

FIG. 77.—MONGOL, SHOWING FISSURED OR 'SCROTAL' TONGUE.

example of each of them you need never mistake one for the other. Of the physical stigmata which I spoke of, marked epicanthic folds are a striking feature of mongolism, and so is congenital defect of the heart. Out of eighteen cases of which I have notes, five had congenital heart disease.

3. I now pass to the third group—cases of **microcephaly**. Those patients, as the name implies, are distinguished by smallness of the head. The normal head measures 13 inches in circumference at birth, 17 inches at nine months old, 18 inches at one year, and 20 inches at five years; and heads which come below these dimensions may be described as cases of microcephaly. But I want to point out to you that the characteristic of microcephaly is not merely

FIG. 78.—MICROCEPHALUS.

deficiency in the circumference of the head, but an alteration in shape. I shall show you photographs presently indicating that the characteristic point about a microcephalic head is that the deficiency affects chiefly the frontal region and the vault. The base of the skull is apparently as well developed as in health. So a constricted frontal region with a small circumference are the characteristic points. The size of the head may be extremely small. The smallest I have seen was in a child three years old in whom the total circumference was $13\frac{3}{4}$ inches—that is to say, only $\frac{3}{4}$ inch more than it should be at birth. One sees them of all measurements, varying from what I have told you to nearly the normal size. Otherwise the child is usually well developed in face and body, but has a dull demeanour, and tends to be apathetic in manner.

4. Opposed to microcephaly is **hydrocephaly,** in which the head is unusually large. I have said so much about the diagnosis of the hydrocephalic head and the means of telling it from that of rickets that I shall not go over that again (p. 161). Hydrocephaly is not, however, a common cause of mental deficiency, and it is remarkable how large the head may be and yet the mental power well retained. I once saw a child with a head 26 inches in circumference who conversed quite intelligently. Such children are apt to be backward in their movements, because the large head upsets their balance and makes it difficult for them to walk; yet the mental power is retained in a surprising degree, even though you know that the cortex of the brain must be flattened out almost like a sheet of paper.

FIG. 79.—HYDROCEPHALUS.

5. We come next to the **paralytic group,** in which there exists some degree of paralysis in addition to the mental deficiency. Those cases belong to that group of infantile cerebral palsies of which I spoke in another lecture. The children who are most apt to suffer from mental deficiency with paralysis are those in whom there has been a lesion of the brain in intra-uterine life or at birth, and I think it is more common in those who have paraplegia than in those who have hemiplegia. You are apt to arrive at an exaggerated estimate of the mental defect in these children, because, owing to paralysis of muscles, they have a difficulty in getting about and living an active life like other children, and accordingly their education suffers, and to this, no doubt, part of their intellectual dullness is to be attributed.

6. There are two groups of cases of mental deficiency which are *associated with fits*, the eclamptic and the epileptic group. The distinction between these is that **eclamptic imbecility** includes those cases in which a fit has been the starting-point of the mental defect.

The child lives for the first two or three years of his life quite normally, and his mental development goes on in the ordinary way; and then he has a severe convulsion which appears to damage the brain, and from that time the mental development suffers, although there is no repetition of the fit. In the **epileptic group** the case may start with a fit, but the characteristic feature is that the fits recur; the child goes on having fits, and it is in consequence of the continual fits that the mental state suffers. You may take it that if epileptic fits are frequent in a child below seven years of age, that child is almost certain to have as a consequence some mental deficiency. There is nothing which apparently upsets the equilibrium of the brain more than the repeated occurrence of fits.

7. The next group is **idiocy by deprivation.** By that one means those patients who are mentally deficient because the 'avenues of knowledge' are blocked. The classical example is that of Laura Bridgman, of whom you may have heard. She was blind and deaf, and all her senses were gone, and as long as she was in that condition she was practically idiotic. Very many children suffer in their mental development because of this blocking of the avenues of information, because their hearing or their sight is bad. And whenever you are brought face to face with a case of delayed mental development you have to ask yourself, May not this be produced by the child having a difficulty in acquiring information owing to his inability to hear properly or see distinctly? The commonest causes are middle-ear disease and errors of refraction. As a consequence of these defects the child fails to benefit like other children from instruction at school, and so falls behind, and in extreme cases might be taken for a case of mental deficiency. But most of these cases belong to the group of mere *backwardness*. And that brings me to ask what is the distinction between mere backwardness and true mental deficiency. You will find it convenient to remember in this connection the dictum of Charles West, *that a mentally deficient child would be abnormal for any age, whereas a backward child is merely abnormal for its own age.* For example, a backward child of six may be at a mental stage which would be normal for a child of four. But a child who is mentally deficient would not be normal for any age. You will find that useful as a practical distinction.

8. I now come to the next group, which is one of considerable rarity—namely, **primary amaurotic mental deficiency.** This

is a group of cases which ought to be familiar to you here because it was first described by Mr. Waren Tay, I think in the year 1881. It is marked by certain definite features. First, the cases tend to occur in families, several members of a family being likely to suffer. Secondly, in most of the recorded cases, the children have been of Jewish parentage. Thirdly, they are characterized by the fact that the children may seem normal at birth and for a short time afterwards; but soon loss of vision sets in, associated with characteristic changes in the fundus of the eye and with paralysis, which leads by about the second year of life to death. Such cases are very far from being common. It is well, however, for you to have them before your minds, particularly in this part of London, where we see so many Jewish patients.

FIG. 80.—CONGENITAL PARAPLEGIA WITH MENTAL DEFICIENCY.

9. Lastly, I come to a great group which one can refer to none of these previous divisions, and which may be described as unclassifiable cases, or, if you prefer a more scientific term, cases of **simple primary amentia.** You may call them, if you like, congenital idiocy, but I do not think that helps you much. They are characterized by mental deficiency, associated, perhaps, with some 'physical stigmata,' and you can only diagnose them by giving heed to the general rules I have laid down—to the speech, general appearance, and behaviour of the patient.

As regards the relative frequency of the different varieties of mental deficiency, I find that of 150 consecutive cases seen in private practice, 45 per cent. belonged to the primary amentia group and 35 per cent. were mongols, whilst cretins and paralytic and epileptic cases and cases of microcephaly made up the remainder in almost equal numbers.

There is a modern development in **diagnosis** which may sometimes be of value, particularly in convincing the parents of the truth of your conclusions, always a difficult task. I refer to X-ray

pictures taken of the brain after air has been introduced either into the skull, merely replacing lumbar puncture fluid by air, or

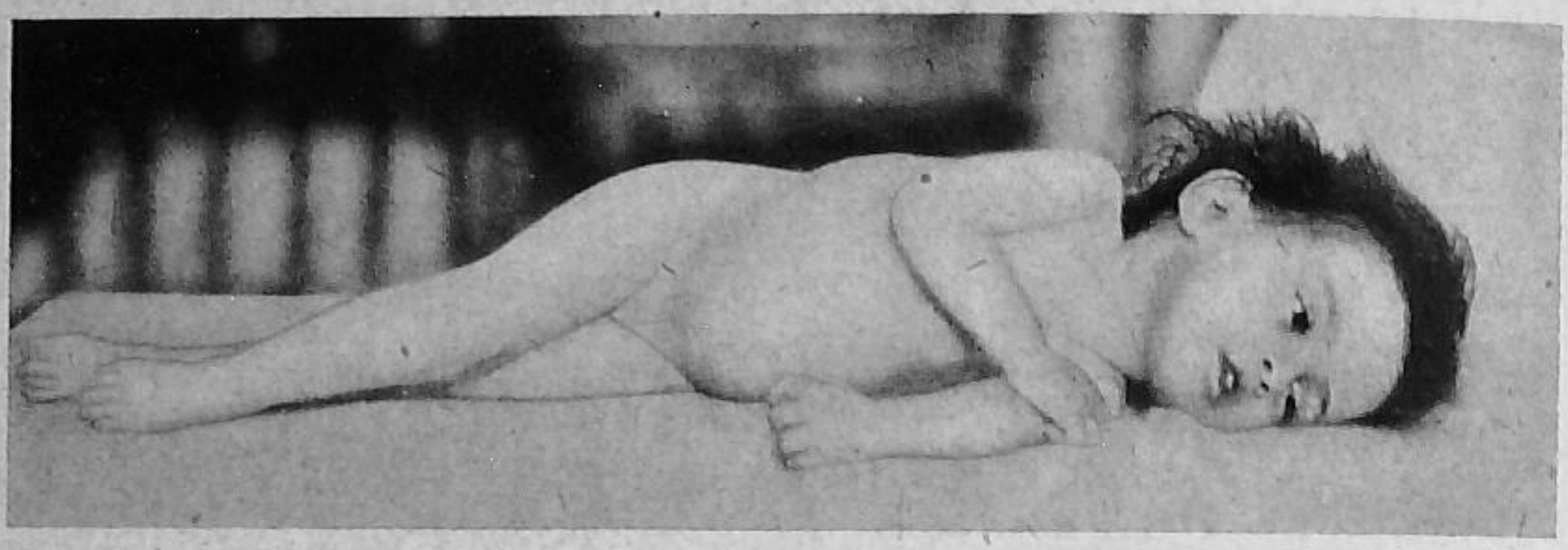

Fig. 81.—Amaurotic Idiocy.

into the ventricles after surgical intervention. The illustration I have here (fig. 82) shows a condition of porencephaly, a condition which hitherto could only be diagnosed in the post-mortem

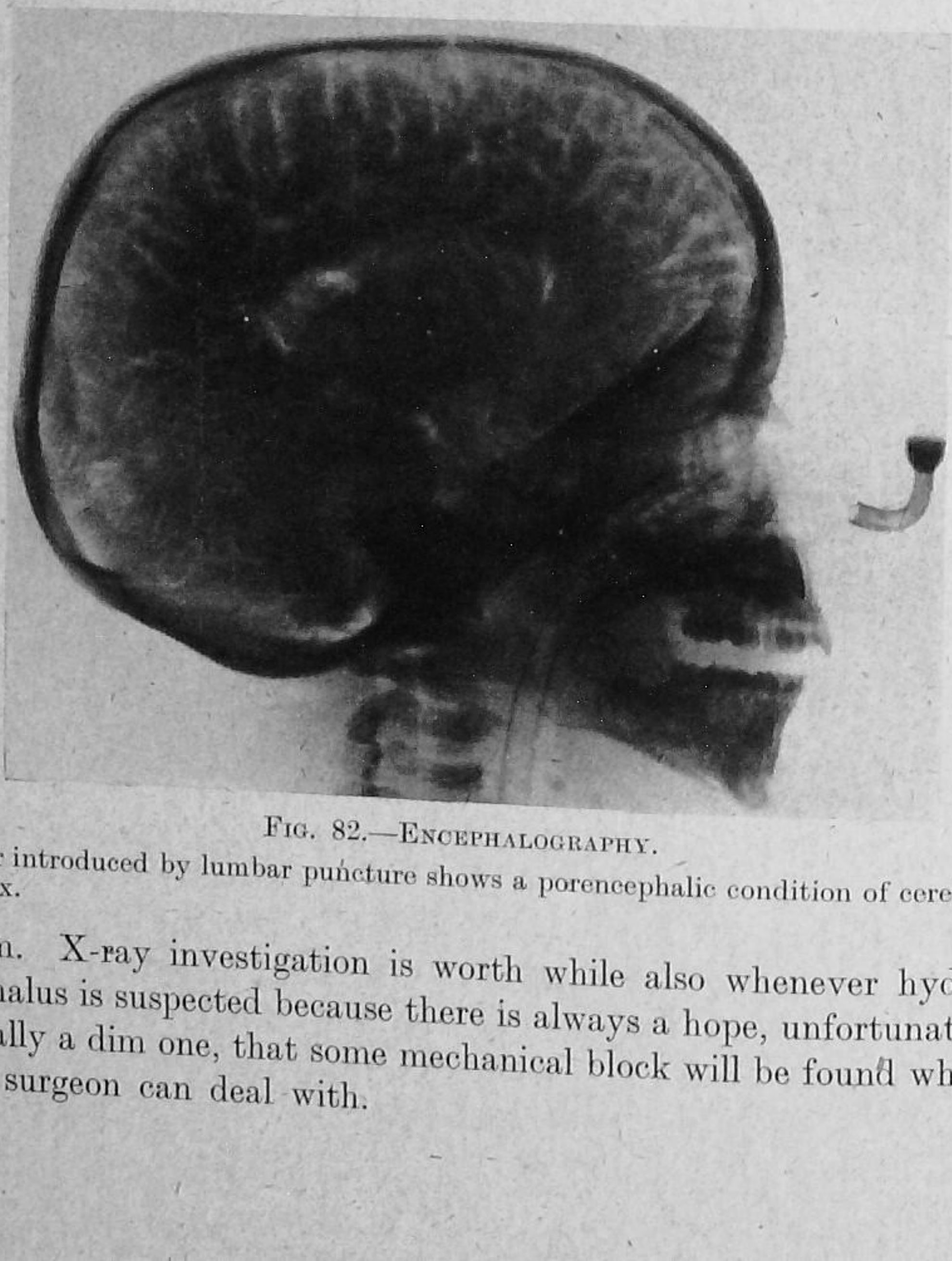

Fig. 82.—Encephalography.

Air introduced by lumbar puncture shows a porencephalic condition of cerebral cortex.

room. X-ray investigation is worth while also whenever hydrocephalus is suspected because there is always a hope, unfortunately usually a dim one, that some mechanical block will be found which the surgeon can deal with.

Causes of Mental Deficiency

I now pass to consider briefly the question of the *etiology of mental deficiency* in children. With regard to a few of the groups there is a definite causation. Cretinism, for example, is due to congenital absence of the thyroid gland, but you cannot say of many that there is such a definite causation as that. In the paralytic group, too, you have a fairly definite cause—namely, damage to the brain of some sort, occurring during the act of birth or during intra-uterine life. In the group of deprivation cases you have a definite pathology in the absence or defect of certain organs of sense. In hydrocephaly you may have a history of antecedent basal meningitis. But in regard to many of the cases you can find no definite cause at all. You will find that certain factors are commonly believed, especially by lay people, to play a part in the production of mental deficiency. You will hear it said that these children are often the offspring of intemperate parents; or that there is insanity, or syphilis, or tuberculosis in the family; or that the parents have been related to one another before marriage. As far as I have been able to make out, you cannot say definitely that these factors have any real influence. Out of seventy-five cases I only found one in which consanguinity was present, and in that case the parents were first cousins. And I cannot find that the parents of mentally defective children are addicted to intemperance more than others; whilst the coexistence of congenital syphilis is exceptional. I am, in fact, profoundly sceptical as to the influence of heredity in producing mental deficiency. It is certainly not an important factor in most cases.

A cause which might be more likely to produce mental deficiency is bad health in the mother during her pregnancy. You will frequently get a history of bad health during the pregnancy, or a history of worry; but you must remember that such occurrences are also extremely common in pregnancies which produce normal children, and you must allow for a tendency on the part of the mother to assign *some* cause for the mental defect in her child. I do not think it has been clearly proved that any of these is a common factor.

I want to speak more definitely with regard to two of the above groups—namely, microcephaly and mongolism. The pathology of microcephaly seems to be an arrest in the development of the

brain as a whole. It is not due, as has been sometimes said, to a premature closure of the cranial sutures. At one time a method of treatment was based upon that assumption—namely, craniotomy—artificial openings being made in the skull, under the impression that the brain ceased to develop because the sutures closed too soon. As a matter of fact, the reverse seems to be the case; the skull closes early because the brain is not properly developed, so that the operation was foredoomed to failure.

With regard to mongolism, I think you can trace a fairly definite cause. You will find that mongols tend to be the last children of large families, or are born when the mother is getting towards the end of the child-bearing age. It is a condition which seems to result when the reproductive powers are exhausted, especially in the mother. Out of fifteen of my cases of mongolism the mother was forty years old or upwards in nine, and in another three she was between the ages of thirty-eight and forty. Why exhaustion of the reproductive power should result in mongolism I do not know, but there seems to be an arrest at some stage, beyond which it is impossible to carry the development.

In striking contrast to this are the cases of primary amentia, in two-thirds of which (according to my own statistics) the child is the first-born of the family. I have no explanation of this fact to suggest, but it almost looks as if in some families Nature tried her prentice hand on an ament before producing a normal child.

Prognosis

You have to consider prognosis, first, in relation to the life of the child; and, secondly, in relation to its mental progress. As regards life, you will find that mentally defective children tend to be physically weak and wanting in resisting power. It is, perhaps, fortunate that it is so, and that the majority never reach the age of puberty. If they have an illness they take it badly, and are apt to go down before it. This feebleness is perhaps more marked in mongols and cretins than it is in others, and the members of these groups are particularly liable to be carried off by tuberculosis. You have also to remember in making your forecast that many will suffer from fits, particularly the paralytic cases, and the occurrence of fits makes the likelihood of mental improvement less.

With regard to mental development, there is nothing more difficult than to speak confidently about the future, and I advise you

to be always very cautious. You may say, perhaps, of cretins that if thyroid is administered from the first weeks of life they will grow up to be almost normal individuals and with regard to mongols that in the highest grade they will be fit for physical work, but that they will never be able to do much brain work of any sort, and will always lack initiative.

The prognosis of microcephaly will depend on the size of the skull; the smaller the skull the more hopelessly idiotic the child will be, and it is only in those children in whom the skull is near the normal size that you will have any appreciable mental power at all. With regard to the epileptic cases, you may assert that they tend to get worse rather than better as they go on: each successive fit leaving the brain in a more damaged condition than it was in before. But the great difficulty in the matter of prognosis is in regard to those unclassifiable cases which make up the last group. Here it will be well to remember, as has been pointed out by many observers, that the more healthy and natural-looking the child is, the worse is the prognosis as regards mental development. As Dr. Langdon Down has said, the prognosis is hopeful, contrary to what is often thought, 'inversely as the child is comely, fair to look upon, and winsome.'

Treatment

The *treatment* I can dismiss in a few words, because, I am sorry to say, in many cases you can do but little. Remember, however, that there is a physical side to mental deficiency, and although you can do little for the mental development of these children, you can do much for their physical state; you can remove obstacles out of the way of the feeble brain. You can remove adenoids; you can order spectacles; you can divide tendons; you can treat rickets. Removal of difficulties may make all the difference to the child, and put him upon a much higher plane than before.

With regard to mental improvement, careful and persistent training is the only hope. Your chief duty here will be to encourage the mother to persevere, to point out to her that improvement must necessarily be very gradual, but that all depends upon her own efforts. The child must be encouraged to take notice of his surroundings, and to do little things for himself as far as possible, whilst on the other hand, all bad habits must be steadily discouraged. Patience, especially the affectionate patience of a mother, can often

work wonders in these cases. As the child gets older, institutional treatment offers much the best chance of the most being made of his mental powers.

I would urge you to resist the temptation to play about with endocrine preparations in your cases of mental deficiency. Nowadays parents will often urge you to try some 'gland treatment' as they call it, but I would have you remember that cretinism is the only form of mental deficiency in which such treatment is of use. *There*, you should, of course, give thyroid, beginning, in the case of a young infant, with $\frac{1}{4}$ grain of the dried powder once or twice a day and cautiously increasing it. Over-dosage will show itself by too rapid loss of weight and often also by diarrhœa. The child should be kept off his feet during the early months of treatment as the legs are apt to bend. Thyroid is often given to mongols, but I have never known it do any real good and it is apt to make the child irritable and nervous.

One point more: when the first pregnancy has resulted in a case of primary amentia you may be asked, Should the mother have any more children? I see no reason why she should not provided she is not herself defective. I do not believe myself that mental deficiency is any more inherited than hare-lip. It appears to arise as a 'sport' or accidental variation, and experience shows that, as a matter of fact, it is rare to have more than one such case in a family. There is nothing in the fact of a woman having had a mentally defective child to show that she is more prone to produce such children than the majority of her sex. It appears to be mainly a question of luck.

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LECTURE XXXI

THE BLOOD DISORDERS OF EARLY LIFE

In recent years research into the diseases of the blood has been very active. It has resulted not only in a considerable increase in our knowledge as to the ways in which blood is formed and destroyed, but also in the recognition of new varieties of anæmia and, unfortunately, in changes in the older classifications and nomenclature. With many of these modern hæmatological niceties, however, I do not propose to trouble you, but shall deal mainly with the varieties of anæmia most commonly met with in practice.

FORMATION OF THE BLOOD IN INFANCY AND ITS PECULIARITIES

Before proceeding further, it is necessary that you should be made acquainted with the peculiarities of the blood in early life and its mode of formation.

The infant embarks upon his extra-uterine career possessed of a **number of red cells** considerably in excess of that of the adult, and the amount of **hæmoglobin** is proportionately high. This, of course, is an arrangement to meet the low oxygen tension in the fœtal blood and after birth it rapidly disappears, and by the end of the second week the adult figure of 5,000,000 red cells is reached, and persists throughout life. The hæmoglobin falls even more markedly, and the fall continues after that of the red cells has ceased, till by the sixth month the normal percentage for infancy (70 per cent.) is reached. This low figure persists till the second year, when a gradual rise makes its appearance, and continues till about the age of puberty. The comparative poverty of the normal breast-fed infant's blood in hæmoglobin has led some enthusiasts to advocate the giving of iron to all babies in order to raise their hæmoglobin to something nearer to the 100 per cent. found in the adult. For my own part I have never been able to see

the justification for this, for who are we that we should presume to improve upon Nature's handiwork ?

In infancy, as in the adult, the red cells are formed exclusively in

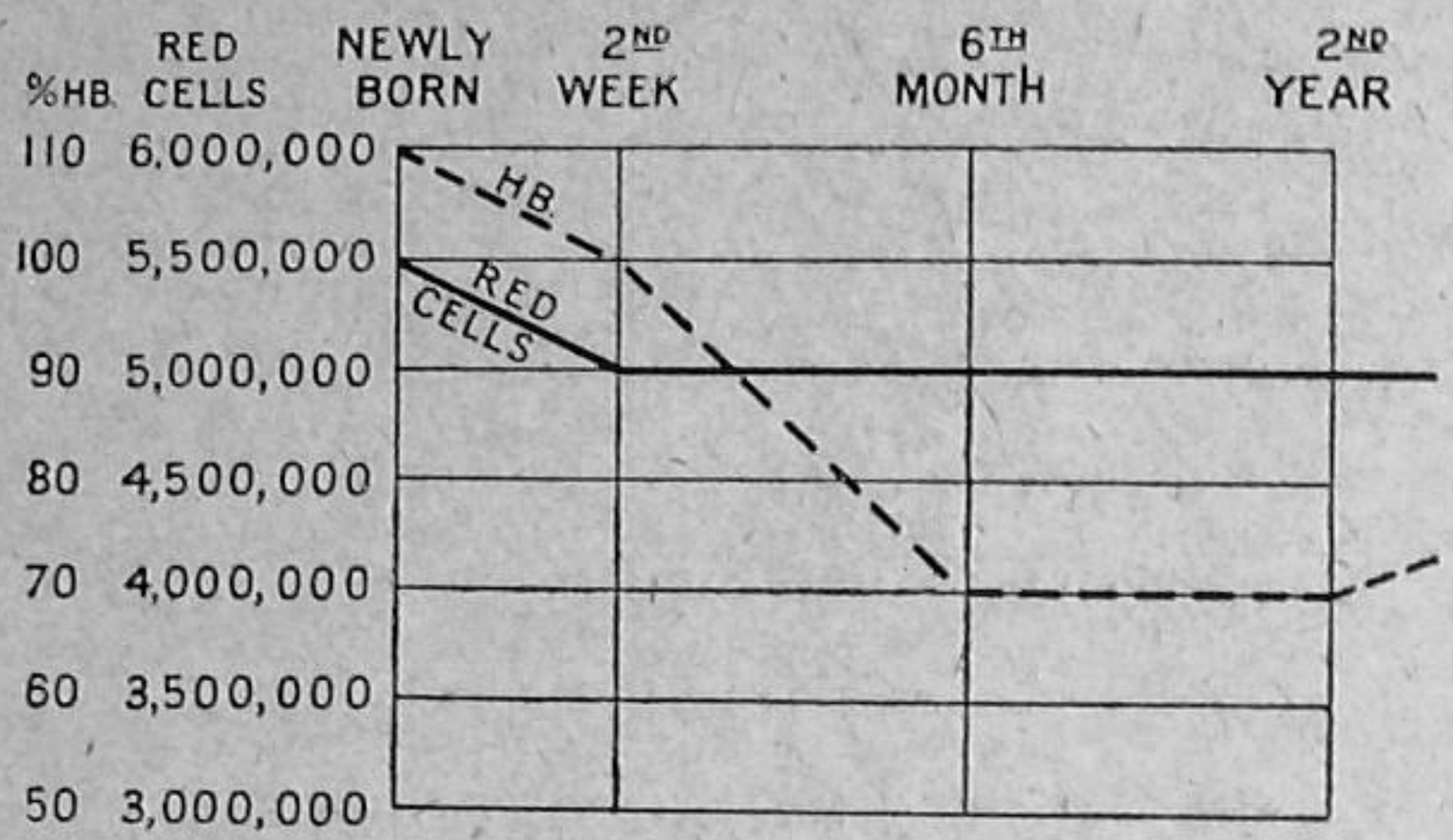

FIG. 83.—PROPORTION OF HÆMOGLOBIN AND RED CORPUSCLES THROUGHOUT INFANCY.

the red bone-marrow. The **extent of the red marrow** is very different, however, in the two cases. In the young child the marrow is red throughout the whole length of the long bones, and it is only as the growth of the bones becomes completed that it retreats to

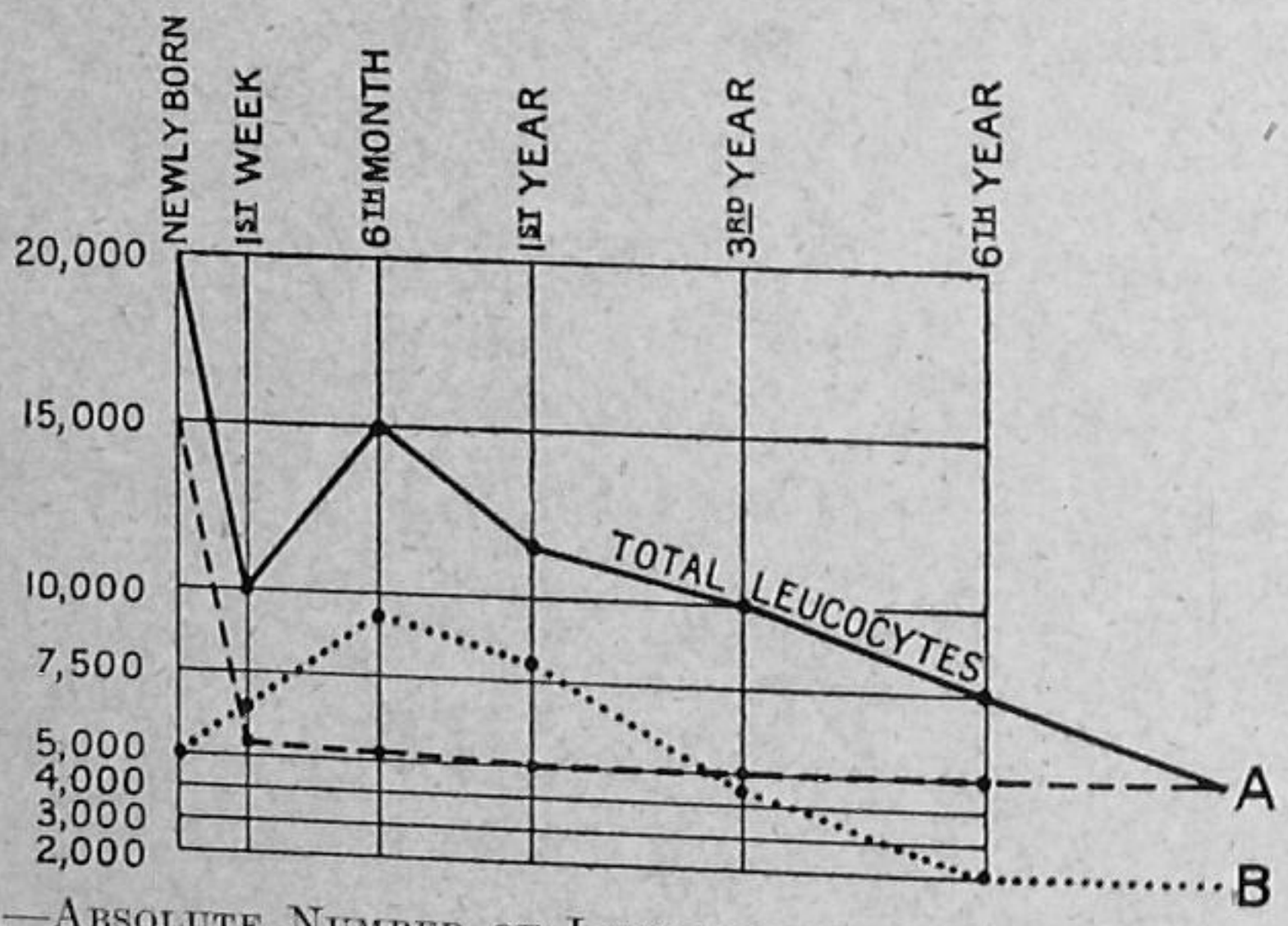

FIG. 84.—ABSOLUTE NUMBER OF LEUCOCYTES PER CUBIC MILLIMETRE AT DIFFERENT AGES. A, POLYNUCLEARS; B, LYMPHOCYTES.

their extremities, where such marrow is alone found in the adult. One consequence of this must be that the child has no great reserve capacity to form red cells as the adult has, for there is no yellow marrow for the red to encroach upon when a greater demand for coloured corpuscles arises. Some of the effects of this we shall see later.

The **white cells** in the child resemble closely in their histological characters the corresponding cells of adult life, but differ markedly in their relative and absolute numbers. The total number of white cells in the blood at birth is about 15,000 per cubic millimetre; by the end of the first year it has sunk to about 14,000, by the second year to 12,000, and by the third to 10,000, after which the decline goes on steadily till the usual adult figure of 7,500 is reached. Now, what I wish to emphasize is that, with the exception of a transient polynuclear leucocytosis which sets in just after birth, the excess of white cells throughout infancy is due to a **large absolute number of lymphocytes.** You will remember that these cells are derived from the adenoid tissue

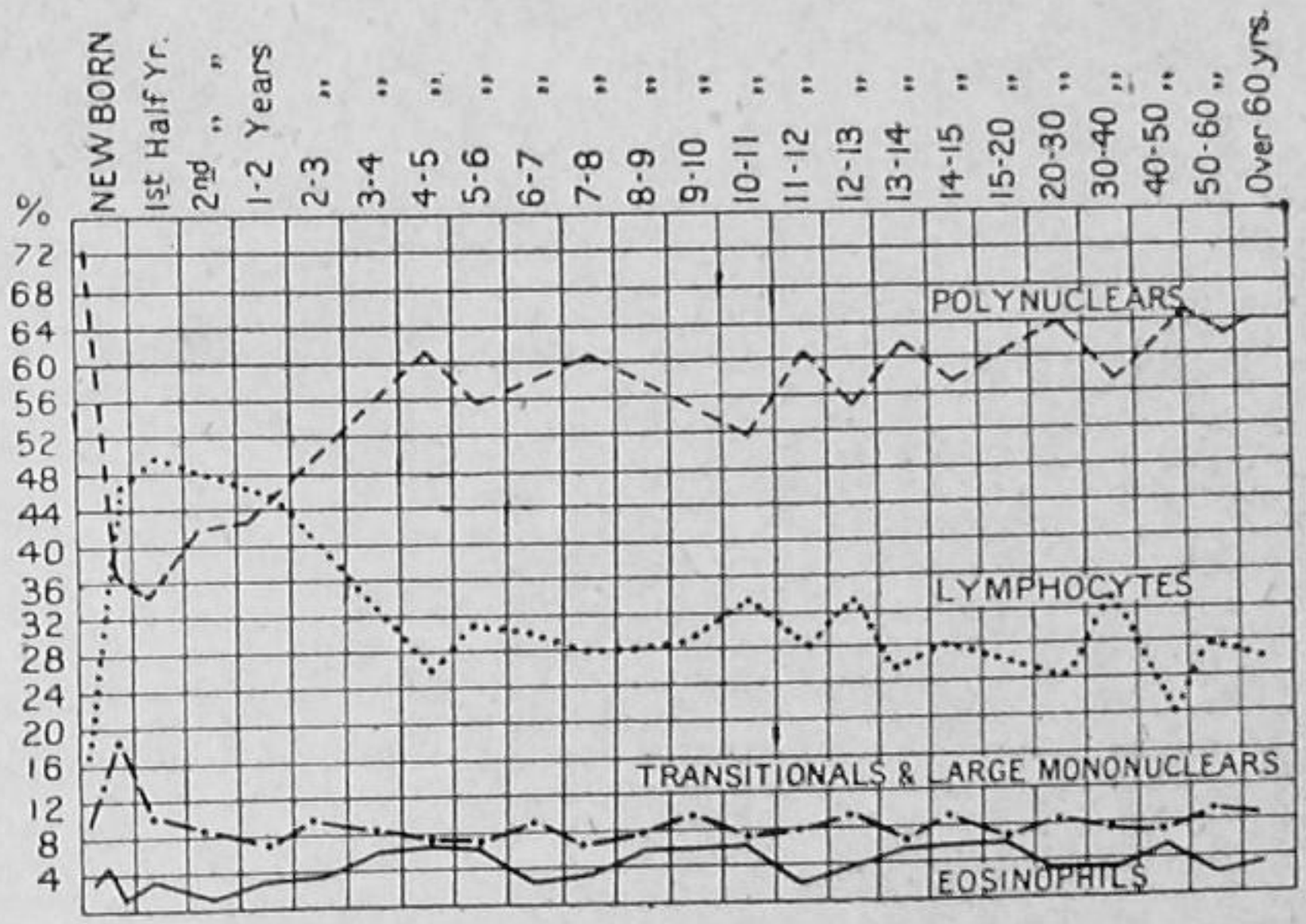

FIG. 85.—DIFFERENTIAL PERCENTAGE COUNTS THROUGHOUT LIFE.
(After Carstanjen.)

scattered throughout the body, such as the thymus, lymph glands, Peyer's patches, and Malpighian corpuscles of the spleen, and their excess in the blood indicates a high degree of activity of the adenoid tissue throughout the period of infancy. Of the meaning of this activity of the adenoid tissue it is difficult to be quite sure, but it is probably associated in some way with growth and nutrition; and anything which interferes with the nutrition of the child seems to lead to a still greater degree of activity of this tissue, and consequently to an even more pronounced lymphocytosis in the blood.

To sum up, then, the **leading characteristics of the blood in infancy** are its poverty in hæmoglobin and the large number of lymphocytes which it contains, and these two facts must always

be taken into consideration when you are trying to interpret the meaning of any 'blood picture' at this time of life.

VARIETIES OF ANÆMIA

After these preliminary remarks I may pass on to consider the clinical varieties of anæmia met with in children. At the outset we are met with the **difficulty of classification.** It is very probable—indeed, almost certain—that anæmia is always symptomatic

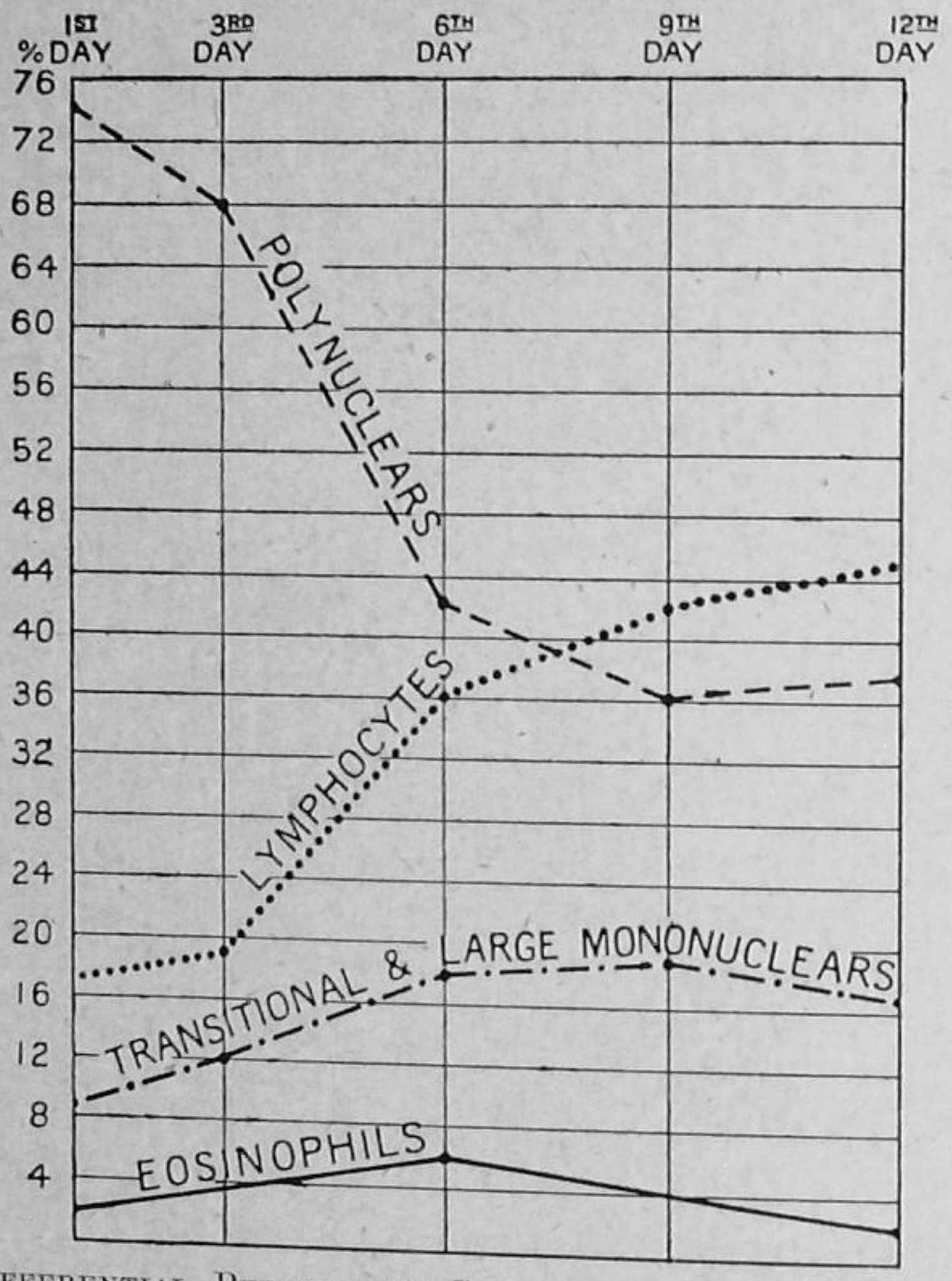

FIG 86.—DIFFERENTIAL PERCENTAGE COUNTS DURING THE FIRST FORTNIGHT. (After Carstanjen.)

—i.e. is the result of some cause which produces increased destruction of blood (hæmolytic anæmia) or interferes with its formation (anhæmopoietic anæmia)—but we do not yet know what these causes are in every case, and so, for convenience of description, I propose to deal with the blood disorders of early life under the following heads: (1) Congenital abnormalities of the blood; (2) Secondary (anhæmopoietic) anæmias; (3) aplastic anæmia; (4) hæmolytic anæmias; (5) anæmia associated with enlarged

spleen ; (6) leukæmias ; (7) affections of the adenoid tissue. You must not suppose, however, that these different varieties are sharply defined ; on the contrary, one meets with cases in which there is both an increased destruction of blood and an interference with its production whilst *some* degree of enlargement of the spleen may be met with in almost any form of anæmia in early life.

1. Congenital abnormalities of the blood.

Every now and then you will meet with children who are said to have been pale since birth, and on examination you discover nothing but a more or less definite general anæmia. I think you will find that in a considerable proportion of these cases the child is a twin, and I wish to lay some stress upon the tendency of twins to suffer from anæmia. It would seem as if the mother was sometimes unable to supply enough iron to provide for the proper formation of blood in both twins, and one or other has to go short, with the consequence that anæmia results. The administration of iron by the mouth may speedily improve matters in such a case, but sometimes it fails, and the condition may even prove fatal. When that happens there may actually be a congenital defect of the marrow present, which renders it incapable of forming blood in sufficient amount. Exactly the opposite state of things to this is found in congenital cyanosis such as occurs most conspicuously in congenital heart lesions. The reason for the excess of red cells in these cases has not been satisfactorily explained, though there are many hypotheses about it, but it may, perhaps, be an attempt to meet the difficulty of oxidation. Post-mortem examination in such cases shows that the blood-forming organs are all in a state of extreme activity, and that the fœtal method of extramedullary red-cell formation persists to a large extent. I shall deal with another rare but interesting example of congenital anæmia when we come to discuss **icterus gravis neonatorum** (p. 395).

2. The secondary or anhæmopoietic anæmias.

It would appear that the blood of the infant is peculiarly ‘ vulnerable ’—that is to say, it undergoes destruction more readily than that of the adult, and so you will not be surprised to hear that secondary anæmia is a very common occurrence in early life. Its production at this period is, no doubt, aided by the small reserve power of blood-formation which the infant possesses, owing to the complete filling of the bone marrow by red cell-forming tissue even under normal conditions. To this, however, I have already referred.

It is hardly necessary to point out that the possible **causes of secondary anæmia** in childhood are exceedingly numerous; any condition adverse to the general health of the child might, indeed, be fairly brought within the limits of such a category. For practical purposes, however, I would ask you to bear in mind that the most potent causes are these: (1) Defects of nutrition, especially a deficiency of iron in the diet; (2) loss of blood; (3) chronic gastro-intestinal disturbance; (4) the toxins of acute infective diseases; (5) 'cachexias' of various sorts, notably congenital syphilis and tuberculosis.

Nutritional anæmia from **deficiency of iron in the diet** is specially apt to occur in infants who are fed too long on the breast or bottle. You will remember that milk is naturally poor in iron and that the infant depends for a supply of that metal partly upon a store of it in the liver which he brings with him into the world. About the end of the natural period of nursing, however, this store gets exhausted and if the diet is then poor in iron anæmia results.

Loss of blood is an obvious cause of anæmia, but children do not often suffer from the chronic losses of blood which occur in adults in such conditions as hæmorrhoids and menorrhagia. The anæmia of scurvy and that which follows purpura are, however, of this origin.

That anæmia is apt to result from **chronic catarrh of the stomach and bowels** is well known. There are probably several causes at work in its production in such a case. Diarrhœa leads to deficient absorption and an inadequate supply of the materials from which the blood is built up. In addition to this it is probable that toxic substances are absorbed from the alimentary canal, possibly the products of the growth of an abnormal intestinal flora, which directly lead to blood destruction.

Of the **infective fevers** as causes of anæmia two stand out with prominence; one is diphtheria, the other acute rheumatism. The latter, indeed, leads to such rapid destruction of blood that one would almost be justified in speaking of a 'rheumatic anæmia.' It is especially, I think, when rheumatism lays hold on the heart that severe anæmia is apt to supervene. When going round the wards, for instance, one may be struck by the sudden appearance of pallor in a child with acute rheumatism, who has hitherto been going on well, and on examining the heart in such a case one will often find that endocarditis or pericarditis has developed.

The moral of this is that you should always remember to put your rheumatic patients through a thorough course of iron during convalescence.

Of the **cachectic conditions** which tend to lead to secondary anæmia I would specially mention congenital syphilis, in which blood destruction may attain a high degree. Mercury in such a case may actually produce the effects of a hæmatinic. Tuberculosis, too, is a potent destroyer of blood, especially, I think, when it affects the alimentary canal, and I have often seen cases of severe and unexplained anæmia in which extensive tuberculous ulceration of the bowel was found at the necropsy.

No matter how a secondary anæmia is produced, the **changes in the blood** are the same, and depend more upon the potency of the destructive influence than upon its nature. The hæmoglobin always seems to suffer first, and so in the mildest degrees one finds merely a hypochromic type of blood. In the severer forms the red cells are also reduced in number and in size (microcytic anæmia), and may show some irregularity in shape (poikilocytosis), whilst in the most severe degrees of all, and especially if the general nutrition suffers, an increased number of lymphocytes appears in the blood stream (lymphocytosis).

3. Aplastic anæmia.

I need only refer to this briefly, for it is very rare; but it is probable that many supposed cases of so-called 'pernicious anæmia' in children have really been aplastic. The blood picture is characteristic and shows great diminution both of red cells and of hæmoglobin as well as of the granular types of leucocyte. The cause of this variety of anæmia is obscure and it runs a steadily downhill course, beng uninfluenced by treatment except—and that only temporarily—by blood-transfusion.

4. Hæmolytic anæmia.

Hæmolytic anæmias are characterized, as you know, by a great diminution of the red cells and the appearance in the blood-stream of large and nucleated forms whilst the proportion of hæmoglobin is relatively high. It is therefore, in the terminology of the moment, spoken of as a hyperchromic macrocytic anæmia. Anæmias of this kind in children are very rare. Addisonian anæmia practically does not occur in childhood, but there is a condition called **acute hæmolytic anæmia** which is very like it only it comes on suddenly and is

often febrile. It is attended by some enlargement of the liver and spleen and rapid hæmolysis. Cases vary greatly in severity. Some respond to transfusion, but others prove rapidly fatal.

So-called **icterus gravis** (p. 395) is also really a form of hæmolytic anæmia.

5. Anæmias associated with enlargement of the spleen.

From time to time cases will be brought to you of which the following may serve as a fairly typical example:

The child is somewhere between one and two years old. At the first glance you are struck by the creamy tint of the skin and the pallor of the mucous membranes. Proceeding to your physical

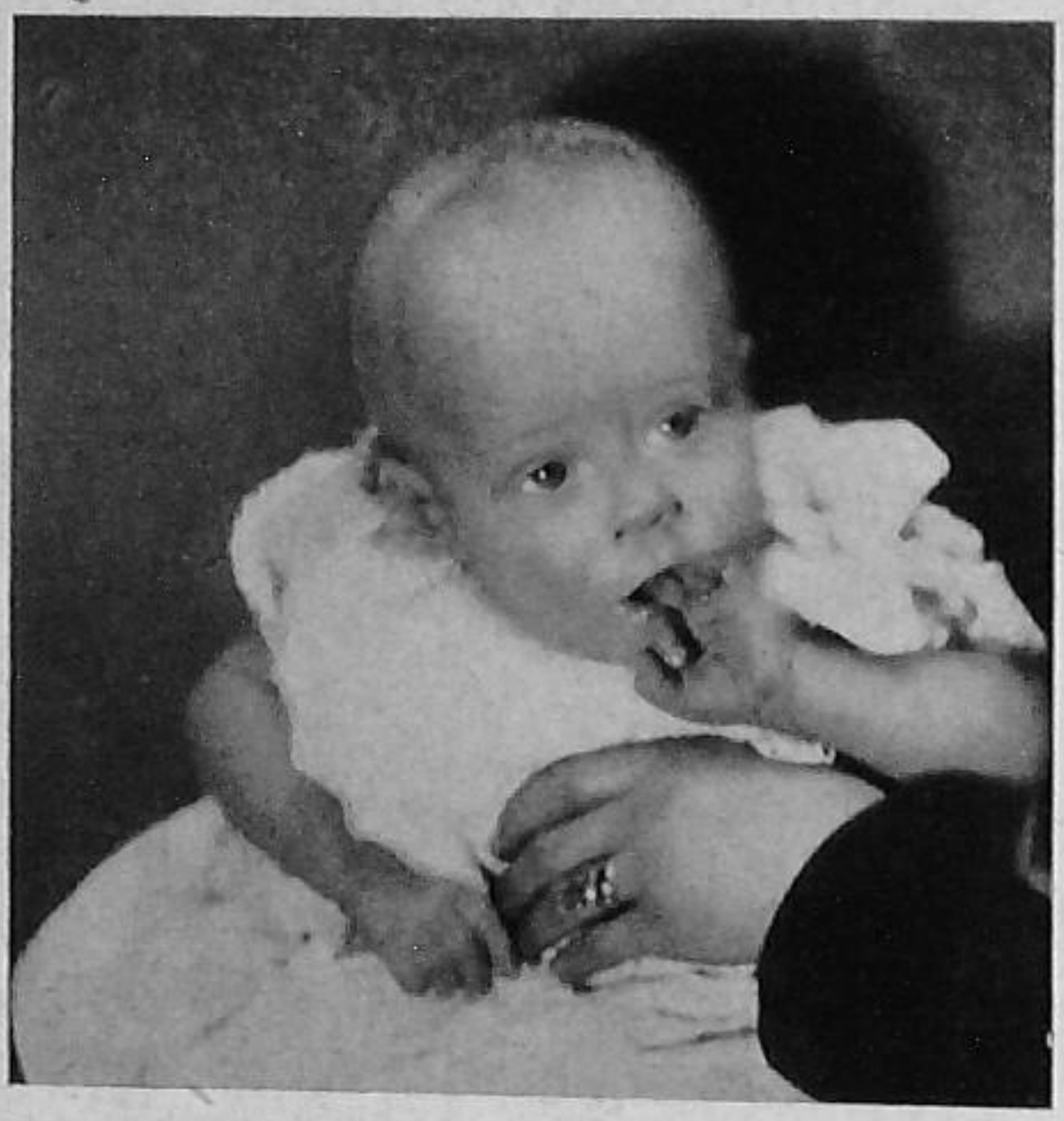

FIG. 87.—HOT-CROSS-BUN HEAD.

FIG. 88.—HEAD BOSSING.

examination, you make out the signs of more or less pronounced rickets, and on passing your hand over the head you will probably be struck by the presence of frontal and parietal bosses (the so-called hot-cross-bun head). On palpating the abdomen you come at once upon an enlarged spleen, which may extend down to or even somewhat beyond the umbilicus. The liver also you find to be notably enlarged, and the superficial lymph nodes may be palpable (fig. 89).

On auscultation of the heart you detect hæmic murmurs, and there may perhaps be some moist sounds at the bases of the lungs. The blood picture shows great poverty in hæmoglobin, along with a more or less pronounced diminution in the number of red cells. The white cells, on the other hand, are increased, often very markedly so, the increase affecting chiefly the lymphocytes. Stained films show that

the red cells are unequal in size, and more or less irregular in shape, and that a considerable number of them are nucleated, whilst on examining the white cells more minutely you are struck by the fact that ' large lymphocytes ' are numerous, and that you can often see a considerable number of typical myelocytes.

Such cases as this you will find described in the textbooks by different names. Some people speak of them as cases of ' **splenic**

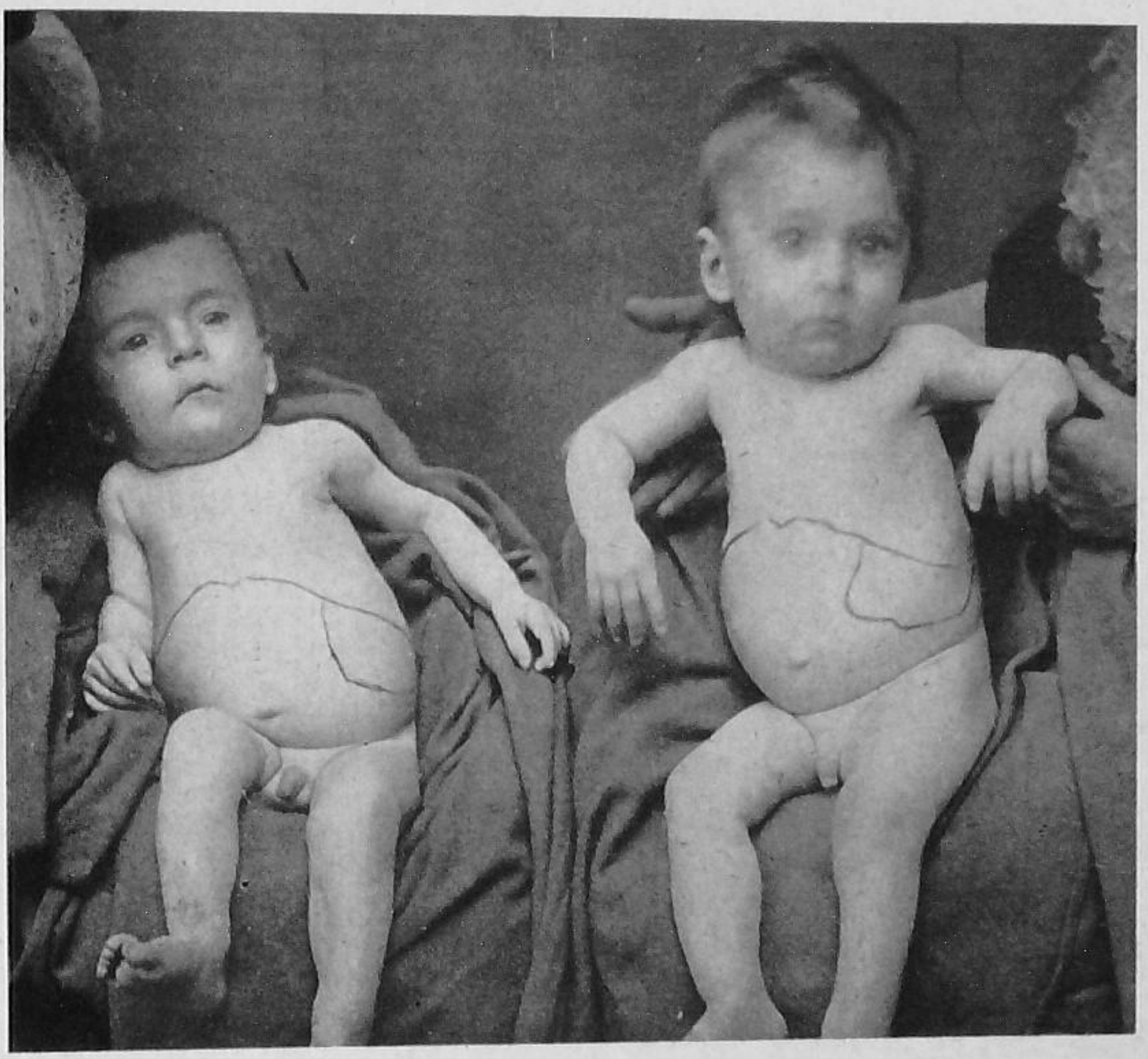

FIG. 89.—SPLENIC ANÆMIA IN TWINS.

anæmia of infancy,' others as ' **pseudo-leukæmia infantum** of Von Jaksch.' I propose to adopt the former term, for the word ' pseudo-leukæmia ' is one fraught with much possibility of confusion, but I would only remind you that you must not suppose that they have anything at all to do with the so-called splenic anæmia which you are familiar with in the case of adults. On the contrary, the two diseases are totally distinct. There has been much discussion as to the true **nature of this disease,** but there would be no use in my attempting to enumerate the different theories which have

been held in regard to it. You need only know that there are at present two chief ways of looking at it. According to one school it is merely a secondary anæmia, whilst according to the other it is a special form of anæmia peculiar to infancy—a disease, in short, *sui generis*. As to the primary disease to which splenic anæmia is supposed to be secondary there is no universal agreement. Some have attributed it to rickets, others to congenital syphilis. That the former condition is almost invariably present all observers are agreed, but to regard it as the *cause* of splenic anæmia is a totally different affair. All the evidence, indeed, points rather in the direction of both the rickets and the anæmia being the result of some common cause. The view that congenital syphilis is the causal factor is at once disposed of by the fact that in fully half the cases no evidence of such disease can be found. The opinion that the splenic anæmia of infancy is a disease *sui generis* has much to commend it, but hitherto no real light has been thrown upon its etiological factors. Some have attributed it to the absorption of a toxin of intestinal origin which exercises a destructive effect upon the blood, but against this is the fact that in quite a large number of the cases there is no evidence of any gastro-intestinal disorder. There, I fear, I must leave the matter, only saying that my own view is that although this disease is almost undoubtedly secondary in the sense of being the result of the action of some destructive influence on the blood, yet we are still quite in the dark as to what this destructive influence may be. It is probably, however, a specific agent of some sort, and in so far the disease may fairly be regarded as one *sui generis*.

You will have no difficulty in the **diagnosis** of this form of anæmia. Your troubles will arise when you come to the question of prognosis. Here I should advise you to be guided by the characters of the blood. The greater the degree of leucocytosis and, in particular, the larger the number of myelocytes present, the more grave must the case be regarded. Remember, however, that quite a number recover,[1] though the duration is always one of months, and many exhibit for years a large spleen, which is left behind, high and dry, as it were, after the anæmia has quite disappeared. Repeated blood transfusions seem to be of value in compensating for the chronic blood destruction. What you have most to dread is the

[1] The twins who figure in fig. 89 of this book both made a complete recovery, and when seen at twelve years of age no trace of an enlarged spleen could be made out in either of them.

supervention of intercurrent disease, particularly of broncho-pneumonia, by which, indeed, the fatal result, if it ensues, is usually brought about.

In older children—usually about or some time after the second dentition—you will sometimes meet with great enlargement of the spleen, with or without some degree of anæmia. I shall describe these, along with some other forms of chronic splenomegaly, in my next lecture.

6. Leukæmia in childhood.

There are, as you know, two chief forms of leukæmia : (1) the *myelocytic*, in which cells of the bone-marrow type enter the blood-stream ; (2) the *lymphocytic*, in which the blood gets flooded with an excess of lymphocytes. Now, the former of these is, like pernicious anæmia, almost unknown in early childhood in the chronic form in which it is usually met with in the adult. *Acute* myelocytic leukæmia, on the other hand, is probably commoner in childhood than has hitherto been supposed, but clinically it is identical in its symptoms and course with acute lymphocytic leukæmia, and can only be distinguished by a careful examination of stained films, which show the characteristic myeloblasts in large numbers. Why it should be I do not know, but you will scarcely find a single case of genuine chronic **myelocytic leukæmia** in childhood on record. It will be well to bear this simple fact in mind if ever you feel inclined to make a diagnosis of it in a child. Chronic lymphocytic leukæmia is equally rare.

Acute lymphocytic leukæmia, on the other hand, is by no means very uncommon in quite early life, though it is probable that it is still often overlooked. Cases will present themselves to you in four chief forms :

(1) Those which exhibit profound anæmia with general glandular enlargement and a hæmorrhagic tendency in the later stages.

(2) Those in which the tendency to hæmorrhage is exhibited from the outset, so that the case resembles one of purpura.

(3) Pseudo-scorbutic cases, in which lesions in the buccal cavity (spongy gums, ulcerative stomatitis, etc.) are the most striking feature.

(4) Cases in which pain in the neighbourhood of the joints has led to a suspicion of acute rheumatism.

The spleen is enlarged in most, but by no means in all, of these cases, and it rarely attains the size that it does in splenic anæmia.

The glands also are not invariably enlarged, and are often little more than easily palpable. In a few cases there is enlargement of the salivary glands which may make the child look as if it were suffering from mumps. The temperature is often considerably raised. You may mistake such cases for examples of purpura on the one hand, or of scurvy on the other. I remember going round the wards here on one occasion and finding a child, recently admitted, who was suffering from profuse bleeding from the gums and fœtid ulceration of the buccal cavity generally. By the bedside stood a bottle of lime-juice, and I was told that the child was suffering from scurvy. On examining a blood-film, however, there was found to be an enormous increase of the lymphocytes, which at once made it clear that the case was one of leukæmia. In any doubtful case, then, examine the blood

FIG. 90.—CHLOROMA, SHOWING PROTRUSION OF EYES.

at once, and you will avoid error. There is only one thing I would warn you of, and that is that you must not expect to find that there is invariably an *absolute* increase in the number of white corpuscles in these cases. What you will always find, however, is that the lymphocytes are relatively greatly in excess—usually numbering upwards of 90 per cent. of the total white cells present.

Closely allied to lymphocytic leukæmia is the disease termed **chloroma**, a rare condition, of which you may never meet with an example. It has the same blood-picture as lymphatic leukæmia, but differs from it in that local tumours develop, often in the periosteum of the bones of the skull and about the orbit, so that the eyes may bulge out (fig. 90). These tumours have on section a peculiar green colour the exact cause of which is still disputed, and from which the disease derives its name. If you remember the existence of the disease and take care to examine the blood, you are not likely to miss

it if ever a case should chance to come under your observation. Ordinary secondary sarcomatous deposits may produce similar tumours about the head and orbits, and I have also known hæmorrhage into the orbit from scurvy simulate it pretty closely, but neither of these exhibits the great degree of lymphocytosis which is characteristic of chloroma.

7. Affections of adenoid tissue.

The great extent and activity of the adenoid tissue in early childhood seem to render it peculiarly liable to become the seat of pathological processes. Of the local collections of the tissue the **thymus** is by far the most important. I cannot deal fully with the diseases of the thymus, nor are they, indeed, of much clinical interest, but I wish to speak briefly of its hypertrophy. We have, unfortunately, no very accurate criterion of what the normal weight of the thymus should be. All we know is that it attains its maximum weight relative to that of the body as a whole at birth, after which it slowly declines till about the second or third year, when it begins to atrophy with increasing rapidity. If, however, you take the trouble to weigh the thymus in many infants of the same age you will find that it varies very greatly, and without, apparently, any special reason. Such, at all events, has been our experience here, and the same thing has been found by all those who have given attention to the subject. Notwithstanding this normal variability, you will meet every now and then with cases in which the thymus is obviously hypertrophied, and sometimes very considerably so.

I cannot promise that you will always be able to recognize such cases clinically, for the only sign they present is an increased dullness behind the manubrium of the sternum, and that may be produced by other causes, such as enlarged bronchial glands. Examination by the X-rays, however, may reveal the condition. The symptoms exhibited by such patients include tracheal stridor (so-called **thymic asthma**), and attacks of collapse and cyanosis. Often these cases are only recognized on the post-mortem table, and you will find that almost invariably they have been ones in which death has occurred suddenly and without apparent cause, and in otherwise very healthy-looking infants. To such cases the term **status thymicus** or **status lymphaticus** is applied, or they are spoken of as instances of ' thymic ' death. Great mystery attaches to the cause of the fatal result in these children, and many theories have been propounded to account for it. According to one set of writers the

enlarged thymus acts mechanically, by direct pressure upon the trachea or upon the nerves in the chest. Others hold that the hypertrophy of the thymus in such cases is merely a part of a general overgrowth of lymphoid tissue, which is accompanied by a hypoplasia of the large vessels, which results in a general feebleness of resistance, so that death may ensue upon quite trivial provocation. Which view is right I am quite unable to tell you, but you should bear the con-

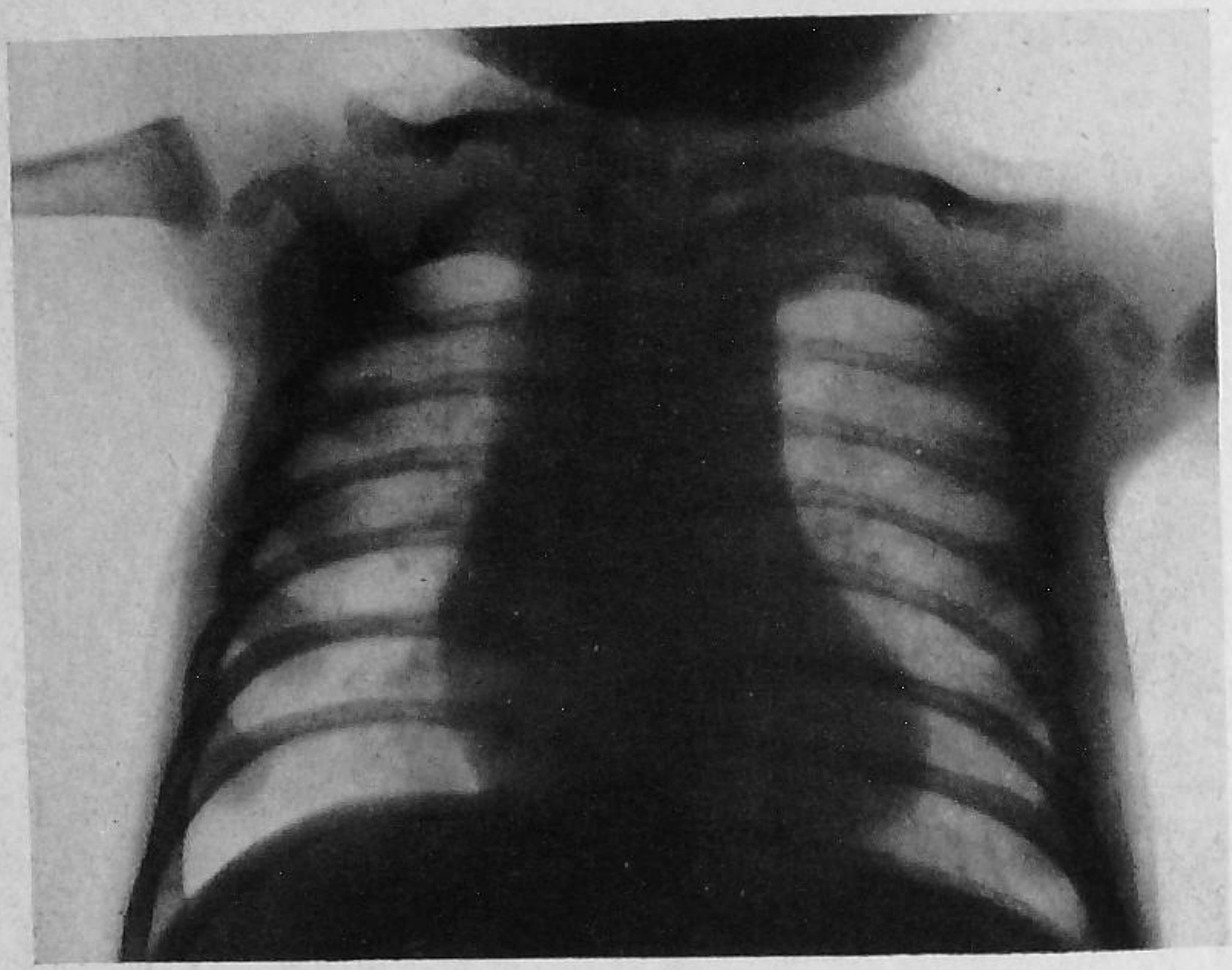

FIG. 91.—ENLARGED THYMUS IN A CHILD—BEFORE TREATMENT.

dition in mind, for it is sometimes of medico-legal importance. Some cases of supposed ' overlying,' for instance, may really be due to this cause. If you succeed in diagnosing an enlarged thymus the best treatment is by X-rays (figs. 91, 92).

Of **diseases** specially **affecting the glands** in early life I would mention infective processes, such as tuberculosis, Hodgkin's disease (which is probably also a chronic infection), and malignant processes such as lymphosarcoma. Of these I do not need to speak in detail, for they are dealt with in every textbook of medicine. This only I would say : that the distinction between **Hodgkin's disease** and chronic multiple glandular tuberculosis is, clinically speaking, a matter of the greatest difficulty, and often, I believe, an impossibility. Nor is the distinction one of much practical importance, for

the course and result of the two processes is identical. The blood affords diagnostic indications of some value. A polynuclear leucocytosis is, in the absence of any inter-current pyrexial disease, in favour of a diagnosis of tuberculosis, whereas a relative increase in the lymphocytes speaks rather for Hodgkin. But this distinction is not invariably applicable, and to settle the matter it may be necessary to examine an excised gland microscopically.

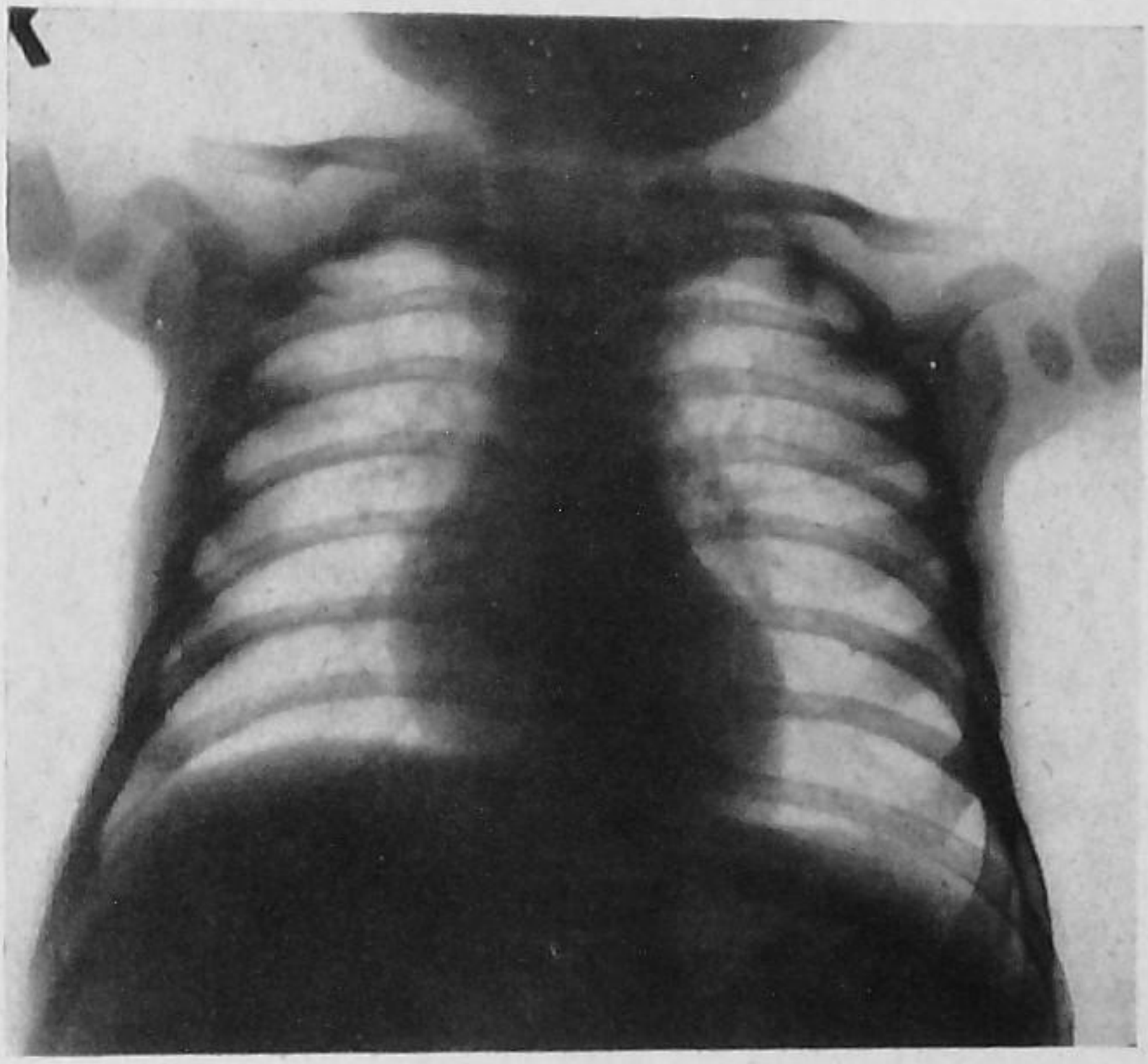

FIG. 92.—SAME CASE AS FIG. 91 THREE WEEKS AFTER X-RAY APPLICATION TO REGION OF THYMUS.

TREATMENT OF THE ANÆMIAS OF INFANCY

I have left to the last the consideration of the treatment of the anæmias of infancy because the same principles are more or less applicable in all varieties. The first thing to be done is to ascertain the cause if you can, and, having done so, to remove it. If, for instance, the anæmia is dependent upon a syphilitic cachexia, you must administer mercury; if it is due to chronic gastro-intestinal disturbance, that must be remedied by appropriate feeding and medicines. In all cases a due supply of fresh air and sunlight is a great aid in restoring the blood to a proper condition. You will often require to increase the amount of iron-containing constituents in the **diet.** Yolk of egg is one of the best foods for this purpose, for

not only does it contain organic compounds of iron, but the lecithin which is so abundantly present in it is also a constituent of the red blood-corpuscles. Oat flour, raw-meat juice and spinach are also important iron-containing foods. Of **drugs** a ferrous salt is indicated in all hypochromic anæmias. The following is a useful prescription for a child of one year :

R	Ferrous sulphate	gr. i ss
	Acid hypophosphor. dil. . .	℥ ¼
	Glucose	gr. xx.
	Aq. chlorof.	ad ℥ i.

Citrate of iron and ammonium is a popular preparation at present, but for my own part I prefer the saccharated carbonate which children take well. It should be given in large doses (5 grains three times daily for an infant). Reduced iron (gr. i with 3 grains of sugar) is another useful preparation. For the splenic anæmia of infancy there is no specific treatment—it must be managed on the lines already laid down, special attention being paid to proper feeding and hygiene. Transfusion sometimes helps, as I have already mentioned. Splenectomy is never indicated, and I have been disappointed with the results of X-ray treatment applied to the spleen. I should add that in some of the more intractable forms of anæmia in childhood you will get no benefit from iron until you combine it with an aperient, just as is the case in some of the anæmias of adults.

In the hæmolytic and aplastic forms of anæmia blood transfusion is often of the greatest help, but neither transfusion nor X-ray treatment is of any use in leukæmia.

THE PURPURAS

I wish, before concluding this lecture, to say a few words about purpura, which is commoner in children than in grown-up people. It is not, however, a disease of quite young children, the majority of cases occurring in the second ten years of life.

The essential lesion of purpura is hæmorrhage into the skin, and you will recognize purpuric spots by the fact that they do not fade on pressure. You will be apt to mistake them for flea-bites, but there is usually a darker central spot in a bite which will enable you to identify it. Sometimes, however, when the bites are fading, the distinction is by no means easy.

Several varieties of purpura are described, and you must distin-

guish, in the first place, between *secondary purpura* on the one hand and *primary purpura* on the other.

In **secondary purpura** the hæmorrhagic eruption occurs in the course of some other disease, such as one of the acute specific fevers, or in any cachectic condition. It is only of prognostic value, and indicates a severe toxæmia or advanced debility, which often precedes a fatal termination of the illness. An alarming type sometimes occurs in the new-born, about the head and neck, clearly mechanical in origin and of a good prognosis (fig. 93).

Several varieties of **primary purpura** are described, but they are probably simply different manifestations of one underlying

FIG. 93.—'MECHANICAL' PURPURA IN NEW-BORN BABY.

pathological state, although what that state is we do not in the least know, and I do not propose to take up any time with a full discussion of the various views which are held about it. It will be sufficient to say that in one variety (P. hæmorrhagica) there is a great diminution of the platelets of the blood with prolonged bleeding time and, defective retraction of the clot. This is sometimes spoken of as *thrombocytopenic purpura*. In the other varieties these features are absent, but urticaria, œdema and erythema are apt to be met with as well as the purpuric rash. These are sometimes spoken of as the *anaphylactoid purpuras*. It is doubtful, however, whether these two groups are always clearly distinct and for convenience, we may divide the clinical forms of primary purpura into the following groups :

1. *Purpura simplex*, in which hæmorrhage takes place into the skin only. The photograph (fig. 94) shows the distribution of the eruption in a typical case.

2. *Purpura hæmorrhagica* (or Werlhof's disease), which is a more severe form of simple purpura, hæmorrhage taking place from the mucous membranes as well as into the skin.

3. *Purpura rheumatica*, also known as *peliosis rheumatica* or Schönlein's disease, but better called 'arthritic' purpura, in which joint pains occur as well as hæmorrhages. It has nothing whatever to do with rheumatism.

Fig. 94.—Purpura Simplex.

4. *Purpura abdominalis*, or Henoch's purpura, in which attacks of abdominal pain, sometimes attended by vomiting and diarrhœa, are a prominent symptom. The colicky pain is probably caused by exudation taking place into the wall of the bowel, and it is of importance, because it may be so severe as to lead to a diagnosis of acute abdominal disease, such as intussusception. I have known at least one case in which laparotomy was performed on account of it. The moral is that you should always examine the skin very carefully for purpuric spots when you are called to a child who has been attacked by sudden and violent colic.

In the **diagnosis** of purpura you must first make sure that you are dealing with a real purpuric eruption. I have already stated that the characteristic point here is that the eruption does not fade on pressure. Use a microscope-slide or a glass lens to make sure. All *erythematous* eruptions fade on pressure. I have already told you how to distinguish bites.

Assuming that you are satisfied that the eruption is purpuric, the next thing you have to settle is whether you are dealing with the disease purpura, or merely with a purpuric eruption as a symptom. The other diseases which you are most likely to mistake for purpura are *acute leukæmia, scurvy, and hæmophilia.* You will distinguish the first of these by examining the blood; the second by inquiring into the diet, and, if necessary, by the therapeutic test of giving antiscorbutics; the third by the family history, and by the fact that the *coagulation* time is prolonged; in purpura it is only the *bleeding* time that is prolonged.

The **prognosis** of purpura is good, but you must be prepared for relapses, which are the rule rather than the exception. Some children, indeed, continue to have attacks every few months until after the age of puberty, when they usually cease. Always look out for nephritis in the course of purpura, especially in the abdominal type. It may materially affect the chances of recovery.

Treatment is not very satisfactory, seeing that we know nothing of the cause of the disease. The one essential is *rest in bed.* I have tried most of the medicines recommended to lessen the hæmorrhagic tendency, but cannot say that I have had much success with any of them. Salts of calcium I have found of no benefit. Epistaxis and bleeding from the gums you will treat on general principles by the use of adrenalin and other styptics.

In severe cases the injection of 5 to 10 c.c. of whole blood into the gluteal muscle is often of great value. In a boy of ten with severe purpura of the abdominal type whom I had lately under treatment it acted like a charm when all other remedies had failed. In cases with profound anæmia transfusion may be called for.

In the chronic or relapsing cases with diminution of the platelets (thrombocytopenic purpura) splenectomy is of great value. Seeing that an attack of purpura leaves the child anæmic, iron should always be given during convalescence.

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LECTURE XXXII

CHRONIC SPLENOMEGALY IN CHILDHOOD

Before proceeding to the differential diagnosis of the various splenomegalies of childhood, it is advisable to make sure that the abdominal tumour in question really *is* an enlarged spleen. Usually this is easy enough, but occasionally one is in doubt, especially as to the possibility that the tumour may be an enlarged left kidney or suprarenal. Sometimes the ordinary diagnostic criteria—that a large spleen does not extend back into the loin, that it has a sharp edge with a notch, that it crosses the middle line below the umbilicus, that the fingers cannot be passed between the tumour and the ribs, that the descending colon does not lie in front of it—all fail, and in that case it is important to remember that dullness extending above the level of the ninth rib in the mid-axillary line is in favour of the tumour being splenic. An X-ray examination, with pyelography if necessary, may help in the differentiation. I would only repeat that the difficulty of telling a splenic from a renal tumour is sometimes a real one, and that I have known the most experienced clinicians make mistakes in the matter.

Having settled that the tumour is an enlarged spleen, it is necessary, before proceeding further, to have some sort of classification of the different varieties of splenomegaly met with in childhood. The only satisfactory classification would be an etiological one, but of that our present knowledge does not allow, and I therefore propose to consider the subject under certain broad groups.

1. *Tumours* (*Cysts, New Growths, Abscesses*)

These are very rare, and for practical purposes undiagnosable. If diagnosed, the proper treatment, of course, would be surgical.

2. *Chronic Infections*

Of these we have to consider tuberculosis, syphilis, lymphadenoma, and chronic sepsis.

In chronic tuberculosis splenomegaly is not likely to be the 'presenting' sign, although in the acuter forms of tuberculosis in young infants enlargement of the spleen is very common. Cases are described, however, though I do not remember to have seen one myself, in which massive caseating tuberculosis leads to considerable splenomegaly. The spleen in such cases may be tender, and polycythæmia and some degree of cyanosis are said sometimes to be present. There will often, of course, be less conspicuous tuberculous lesions elsewhere. The proper treatment is splenectomy.

The relation of *congenital syphilis* to splenomegaly in childhood is important, but I believe that its frequency as a factor in causing the condition is exaggerated. After all, as I have pointed out to you before, inherited syphilis is a rare disease, and so we may say that, although syphilis certainly very often leads to splenomegaly, yet not many cases of splenomegaly are due to syphilis. It is in infancy that a syphilitic enlargement of the spleen is most likely to be met with, but there is reason to believe that in later childhood—say, between the ages of five and seven—splenomegaly may be the only sign of a syphilitic taint, and it appears again about the age of puberty in association with cirrhosis of the liver. In the pre-Wassermann days the tendency was to ascribe every splenomegaly of doubtful origin to syphilis, but since the introduction of that test I have been impressed by the frequency with which it is found to be negative in these circumstances. It is true that in some undoubtedly syphilitic cases—in syphilitic cirrhosis, for instance,—the Wassermann reaction may be negative, and it is a matter for serious consideration what degree of confidence we can place in it as a criterion of the presence or absence of a syphilitic taint. I would only point out that it is, after all, the only positive test we have in a case in which unmistakable objective signs of the disease are wanting, and if we are not to be able to trust it the whole question of the relation of syphilis to splenomegalies of obscure origin in childhood goes back into the melting-pot. It is all the more important that we should have some trustworthy test for the presence of syphilis, inasmuch as it has been suggested by some that an inherited taint may form a basis on which enlargement of the spleen—though not primarily due to syphilis—may develop more easily, or in a more exaggerated degree, than it does in a child whose heredity is not thus burdened.

I am bound to say that this view seems to me rather speculative.

When syphilis is believed to be the cause of a splenomegaly, vigorous antisyphilitic treatment must, of course, be adopted. In some cases, however, the changes in the spleen may have proceeded so far that they are unaffected by any form of drug treatment, and in that case splenectomy may be justifiable.

Lymphadenoma as a cause of enlargement of the spleen, without there being evidence of the disease in the glands, must be very rare. For my own part, I have never met with an instance of it, though cases have been described by others, and I do not see how it can be diagnosed with certainty. If diagnosed, the appropriate treatment would be the application of X-rays to the tumour.

Chronic sepsis has been put forward as a cause of splenomegaly, as of most other things, but in my opinion on rather insufficient grounds. That splenomegaly may be the presenting sign in cases of very chronic infective endocarditis (endocarditis lenta) is certain, but I doubt if cases of this very chronic type are ever met with in childhood; and of 'intestinal sepsis' as a cause of enlarged spleen we know nothing.

3. *Tropical Splenomegalies*

Chronic protozoal infections, as in malaria and kala-azar, may cause great enlargement of the spleen in children, but as the diagnosis and treatment of these diseases is the same as in adults I do not propose to discuss them.

4. *Splenomegaly in Metabolic Diseases*

We have here chiefly to consider the part played by rickets. This has been almost as much disputed as the rôle of syphilis in producing splenomegaly, and, I believe, fully as much exaggerated. That severe rickets tends to be accompanied by some degree of enlargement of the spleen is undeniable, though in some cases the organ is not so much enlarged as pushed down by deformity of the ribs; but I would have you believe that rickets alone is not an important factor in the production of splenomegaly, especially nowadays. The same may be said, with even greater truth, of waxy disease, which we need rarely think of now when the nature of an enlarged spleen is under discussion.

5. *Gaucher's Disease*

Gaucher's disease which you will find described in your ordinary textbooks should, I suppose, now be included under the metabolic disorders associated with splenomegaly. It is rare, though probably not so rare as is believed, and is to be diagnosed by its familial, though not hereditary, incidence, and, above all, by the demonstration of the characteristic endothelioid cells in the product of a splenic puncture. Opinions as to the value of splenectomy in the Gaucher cases are divergent. The operation cannot certainly be regarded as a cure, seeing that the disease is one which is not confined to the spleen, but affects the whole reticulo-endothelial system throughout the body. If the splenic tumour is causing local discomfort, however, its removal would appear to be justified.

Of closely similar origin to Gaucher's disease but associated with a different metabolic flaw, so to speak, are the Niemann-Pick disease, the Schuller-Christian syndrome and von Gierke's disease. In any of these the spleen may be enlarged.

6. *Splenomegaly in Diseases of the Blood*

We have here to consider the leukæmias, splenic anæmia of infancy (anæmia pseudo-leukæmica infantum of von Jaksch), acholuric jaundice, purpura, and erythræmia.

The *leukæmias* we may dismiss in a word, as I dealt with them in my last lecture. In childhood they are almost always acute, and therefore not a cause of *chronic* splenomegaly. I have only once seen a case of chronic myelocytic leukæmia in a child—a boy of twelve—and for practical purposes leukæmia as the cause of a chronically enlarged spleen in childhood need not be considered. If suspected, a blood count would at once establish the diagnosis.

The *splenic anæmia of infancy* is in an altogether different category. It used to be relatively common, and when I was giving the Goulstonian Lectures forty years ago I had no difficulty in collecting notes of a large number of cases, all of which had been under my own observation. In recent years, however, it has become rare, and I have not seen a typical case now for a long time. I do not propose to discuss again the nature of this disease, and whether it is merely the result of a hæmopoietic reaction to various debilitating influences in childhood, or whether it is—as

I at least hold—a disease *sui generis*. For our present purpose it is sufficient to point out that it should not be diagnosed after the age of three, although it is true that the enlarged spleen may persist beyond that age, when the anæmia has passed off, before gradually disappearing. In that case, however, there should be a clear history of the disease in the earlier years. The blood picture in the acute phase is characteristic, but I described it fully in my last lecture.

In considering the treatment of this form it is to be remembered that the disease tends to spontaneous recovery in the great majority of cases, given suitable hygienic measures. Transfusion will help if the hæmoglobin falls below 50 per cent. I do not myself consider that splenectomy is ever called for in it, as the operation must be highly dangerous in the severe forms and is unnecessary in the milder. Some successful cases have, however, been reported, but I think it probable that these would have recovered without operation. Whether X-ray treatment is of any use I am not sure. In the few cases in which I have tried it the results have been disappointing, but others have been more fortunate. At least, it cannot do any harm. The ultra-violet rays are also uncertain in their effects and no benefit has been reported from giving liver.

I need not say much about the splenomegaly of *acholuric jaundice*; it is now well recognized as a not uncommon occurrence, and the diagnosis can be established with certainty from the characteristic increased fragility of the red cells. There is also no doubt that splenectomy is the only effective form of treatment.

In *purpura*—even in the chronic forms—splenomegaly is not likely to be the presenting sign, and I only mention it here because in that variety of the disease in which the platelets are greatly diminished removal of the spleen is now generally agreed to be very beneficial. I referred to this in the last lecture.

I doubt whether true erythræmia (Osler-Vaquez disease) is ever a cause of chronic splenomegaly in childhood, but it is interesting to note that polycythæmia is sometimes a feature in cases of syphilitic splenomegaly.

7. *Splenomegaly Associated with Cirrhosis of the Liver*

We now come to that interesting group of cases in which an enlarged spleen is associated with cirrhosis of the liver. Three

forms of cirrhosis with splenomegaly—excluding for the moment Banti's disease—may be distinguished.

(*a*) *Idiopathic non-alcoholic progressive cirrhosis.*—This is a cirrhosis of unknown cause, which resembles the ordinary portal cirrhosis of adult, runs a fairly rapid course, and often terminates in hæmatemesis. The spleen is always enlarged to some degree, but in certain of the cases is so conspicuous and early a feature as to be the 'presenting' sign, and these have been spoken of as cases of 'splenomegalic cirrhosis,' or cirrhosis with 'splenic predominance.' Their diagnosis from Banti's disease is not easy, and will be considered later, and whether or not they are suitable cases for splenectomy is still unsettled. Some writers maintain that the operation in these cases is almost invariably fatal, but I am not at all sure that this is true. In the following case, for instance, if, as I believe, it must be included in this group, splenectomy appeared to effect a cure.

The patient was a boy aged twelve, who was admitted for bleeding from the gums with a history of always having bruised easily. His spleen was enlarged down to the umbilicus, and the liver was easily palpable. The red cells numbered $4\frac{1}{2}$ million, with 70 per cent. of hæmoglobin, and the white cells 1,600. The Wassermann reaction was negative, both in the patient and in his mother. Four months later he was readmitted with ascites. The spleen was removed, and at the operation the liver was found to be cirrhotic. Twelve years later he was in excellent health.

(*b*) *Syphilitic cirrhosis with splenomegaly.*—These cases are met with in later childhood about the time when interstitial keratitis and the other signs of 'late syphilis' manifest themselves, and I have already said that there is reason to believe that they do not always give a positive Wassermann reaction, and that, if antisyphilitic treatment fails, removal of the spleen may be justified.

(*c*) *Splenomegaly with biliary cirrhosis.*—These are the cases described by Gilbert and Fournier, and are believed to be the form which Hanot's cirrhosis takes in the child. They are said to be characterized by the presence of a much enlarged spleen, recurring jaundice, finger-clubbing, and impairment of growth, but without ascites. Now, I am very sceptical as to the existence of Hanot's cirrhosis at all, and still more sceptical as to the occurrence of such cases in the child, but I shall deal with the question more fully when I come to lecture on diseases of the liver (p. 399).

8. *Splenomegaly the Result of Splenic Thrombosis*

That thrombosis of the splenic vein could cause enlargement of the spleen was denied by Osler, and yet there seems to be no doubt that it does happen, and I have myself seen two cases in the adult in which this was proved at the necropsy to be the cause. Wallgren has drawn attention to it as a cause of splenomegaly in children, and believes that it is commoner than is supposed, but that it has hitherto been confused with Banti's disease. He describes the leading features of the syndrome as splenomegaly, anæmia with leucopenia, and slight ascites. Sooner or later hæmatemesis sets in, and after a bleeding the volume of the spleen *shrinks* —this, he believes, does not happen after hæmatemesis in Banti's disease or in cirrhosis—and at the same time a temporary leucocytosis appears. The liver in all of his cases coming to necropsy was found to be normal.

He is of opinion that the thrombosis of the splenic vein is the result of trauma or of an 'infection,' and that it leads to engorgement of the spleen and dilatation of the veins at the lower end of the œsophagus, rupture of one of which causes the hæmatemesis, with temporary subsidence of the splenic tumour. Wallgren considers that the prognosis in these cases is bad unless splenectomy is performed.

I believe that the following case may be regarded as one of splenic thrombosis:

The patient was a boy aged five. Two years before admission he vomited a teacupful of blood, and this was repeated one year later, and again one week before his admission to hospital. He had had no other illness except whooping-cough when he was four. He was a pallid, weakly, but not ill-nourished child, with a 'biscuit tinge' of skin. The spleen was palpable and the liver extended one inch below the costal margin. There were a few small palpable glands in the axillæ. A blood count showed red cells 1,160,000, white cells 5,620, hæmoglobin below 30 per cent. A few normoblasts were present. The differential count was normal. The Wassermann reaction was negative. Ten days after admission he vomited an ounce of bright-red blood, and at the same time his temperature rose to 103° F. The white cells were at this time 12,000. There is no note as to the effect of the hæmorrhage on the size of the spleen. He was transfused, but with only temporary benefit, and died two weeks after his admission.

At necropsy the spleen was four times the normal size, firm and tough. There was *ante-mortem* thrombosis of the splenic vein, and

also recent clotting in some of the mesenteric veins. The liver was large, pale, not cirrhotic. There were a few small varices at the lower end of the œsophagus.

9. *Splenic Anæmia of Adult Type and Banti's Disease*

It will be generally agreed, I think, that 'splenic anæmia' and 'Banti's disease' are to some extent diagnostic 'rag-bags' into which are thrown all the cases of enlarged spleen that cannot be placed in any other category. None the less it will be admitted that cases to which these terms are applicable have a real existence in the adult, and the question for us to-day is whether they are also met with in childhood. Now, seeing that the splenic anæmia of adults is of its essence a very prolonged disease, the final stage of which, when cirrhosis of the liver has supervened (so-called Banti's disease), is only reached after some years, it follows that Banti's disease at least is not likely to be met with whilst the patient is still a child. In spite of this I think the diagnosis 'Banti's disease' is sometimes justified in childhood, as in the following case :

A boy of twelve was known to have had an enlarged spleen for five years. The liver was not enlarged. The blood count showed 2,750,000 red cells, 45·1 per cent. of hæmoglobin, and 3,400 white cells. Wassermann test negative. Hæmatemesis supervened, after which the spleen was decidedly smaller for some days. There was no leucocytosis during or after the hæmorrhage. Splenectomy was performed, and at the operation the liver 'seemed to be the seat of early cirrhosis.' His subsequent history is unknown.

The difficulty, of course, is to distinguish such cases from those of idiopathic cirrhosis 'with splenic predominance,' already referred to. It can only be done if the history shows that the spleen was enlarged for some years (as in the above case) before signs of cirrhosis supervened, for the cases of idiopathic cirrhosis seem to run their whole course in a comparatively short space of time. There is little doubt, too, that cases of splenomegaly due to splenic thrombosis must often have been included under the term 'splenic anæmia or Banti's disease,' and so, probably, have some syphilitic cases and some of acholuric jaundice.

It is sometimes asserted that the presence of leucopenia puts a case in the category of splenic anæmia rather than of idiopathic cirrhosis, but to this I cannot agree. In the case of idiopathic cirrhosis with splenic predominance which was described earlier in

this lecture, for instance, leucopenia was present, and my own view is that leucopenia tends to be a feature of most chronic splenomegalies, no matter of what origin, and that little diagnostic value can be assigned to it.

The treatment for splenic anæmia of the adult type, everyone is agreed, is splenectomy, but whether the operation is worth doing after cirrhosis has supervened—that is, in Banti's disease—is a point on which I have not made up my mind.

In conclusion, I should like to point out that your chief difficulty when you have diagnosed a case of chronic splenomegaly will be to decide whether it is one for splenectomy. As to this the advice I would give you is, that the cases suitable for this operation are those which show a negative Wassermann reaction, no enlargement of lymphatic glands, and no characteristic leucocytic picture, but in which there is some degree of anæmia, associated with (1) leucopenia, or (2) increased fragility of the red cells, or (3) hæmatemesis. There is some difference of opinion, however, as to the advisability of removing the spleen in Gaucher's disease and in cases with cirrhosis of the liver, whether of the idiopathic or Banti variety, even although these fall within the limits of the above criteria. The operation is contra-indicated if the blood platelets are normal in number, and still more if they are increased, owing to the danger of post-operative thrombosis.

LECTURE XXXIII

SOME COMMON AFFECTIONS OF THE GENITO-URINARY SYSTEM IN CHILDHOOD

In the present lecture I propose to deal with some of the commoner affections of the genito-urinary system in childhood, but shall not refer to such features of them as are common both to children and adults.

It is usual when describing diseases of the urinary system in childhood to begin by pointing out some respects in which the urine at this age differs from that of adult life. Such difference may be summed up for clinical purposes in the statement that the child's urine is paler, of lower gravity, and relatively less acid than that of the grown-up person. It would be of some importance, however, to be able to tell you the amount of urine secreted daily at different ages, but I find that to be impossible, for if you refer to the figures given by different authorities on the subject you will discover such discrepancies as to make their estimates of little value for clinical purposes. Perhaps the estimate arrived at by Still is as good as any other, and has the advantage of being easily remembered—viz. that after the age of four the age of the child in years multiplied by two and a half gives the average amount of urine in ounces. Below the age of four, however, the secretion is relatively greater than this, because of the more fluid character of the diet.

I have occasionally had children sent to me because they were supposed to be passing too little urine, but I have always found on investigating such cases that the amount secreted was not really less than the minimum average for that age. Every now and then, however, you will meet with cases in which no urine is passed at all for some hours, and in which the bladder also remains empty. These are cases of true suppression, or **anuria.** The cause of this condition is quite obscure, but it need occasion you no alarm, for it never lasts longer than twenty-four hours, and usually a hot bath and a dose or two of spiritus ætheris nitrosi is sufficient to restart

the secretion. The opposite condition—**polyuria**—may also occur without apparent reason for brief periods, but when continued should make you think of diabetes mellitus or insipidus.

Not infrequently children pass uric acid **gravel.** I have told you in a previous lecture that the pain caused by this may closely simulate that of intestinal colic, but that the condition can usually be recognized by the detection of grains of uric acid sand on the napkins. The indication in these cases is usually to reduce the amount of food, and always to administer citrate of potash liberally.

At other times a condition is met with which one can only describe as '*uric acid storms.*' These may occur in infancy, but are perhaps commoner in children about three to four years old. They often take place at very regular intervals, exhibiting a decided periodicity. The occurrence of the 'storm' is heralded by a day or two during which the child is pale, languid, and generally 'out of sorts.' There may also be an ethereal odour in the breath, and the stools may be pale. These symptoms are followed by an attack of violent screaming, and some hours later gravel is passed, or may be noticed as a deposit in the urine without there having been any signs of pain.

The pathology of these cases is obscure, but I strongly suspect that they are in some way due to a functional disorder of the liver, and are closely allied to other periodic affections, such as migraine, 'food fever,' cyclical vomiting, and so-called 'bilious attacks.' Like these disorders, too, they are most successfully treated by cutting down the milk and carbohydrates in the food—*not* by reducing the proteins, e.g. meat, as is often done—and by the regular use of an 'hepatic' aperient, such as rhubarb and grey powder. In addition, of course, citrate of potash should be given freely during the attack.

Another disorder which may be due to the presence of uric acid crystals in the urine, or to excessive urinary acidity, is what is called **vesical spasm.** It may closely simulate vesical calculus. I remember a medical friend of mine in the country ringing me up one morning on the telephone to ask my advice about a surgeon to whom to send one of his children, because he believed the child to be suffering from stone. On asking the grounds for this opinion, I was told that there were at times great pain and strangury on micturition. I advised that before a surgeon was consulted the child should be given a course of citrate of potash and hyoscyamus. This was done, with the result that the symptoms quickly dis-

appeared. That was a case of vesical spasm. Of course, **calculus** is not very uncommon in children, too, but as its symptoms and the methods of diagnosing it are the same as in the case of adults, I need not say anything more about it, except to point out that sometimes the chief symptom is incontinence, and that when a child suddenly develops enuresis, calculus is always one of the things to think of. I recall, for instance, the case of a little girl whom I saw many years ago, who was brought to the hospital for incontinence along with some symptoms of cystitis. On sounding the bladder we found a large stone, which we were able to extract by dilating the urethra, with immediate relief to all her symptoms.

INFECTION OF THE URINARY TRACT

I wish now to turn to a totally different subject—namely, *infections of the urinary tract in childhood*, by which one means for practical purposes infection by the *Bacillus coli*. Now when the *B. coli* finds access to the urinary tract, one of four conditions may result: (1) A simple bacilluria; (2) cystitis; (3) pyelitis; (4) suppurative nephritis. Or these may occur in various combinations.

(1) *Bacilluria* alone gives rise to few symptoms. There may be some disturbance of the general health, but nothing very definite, and the condition can only be diagnosed by an examination of the urine, which is opalescent and does not clear on filtering. It usually remains acid—for the *B. coli*, as you know, does not split up urea—but has often a peculiar fishy odour. Microscopical examination shows it to be swarming with bacteria, which on culture prove to be the *Bacillus coli*.

(2) The symptoms of *cystitis* due to *B. coli* I need not describe to you in detail, as they are the same as those of any other cystitis except that the urine remains acid unless a secondary infection takes place.

(3) *B. coli pyelitis* is met with in its most striking form in young infants, and more often in girls than in boys. It usually sets in quite suddenly. The constitutional symptoms are much more pronounced than the local, and may quite overshadow the latter. They consist in high irregular fever, the temperature often running up and down in the wildest manner, and often there are rigors or, at all events, attacks of blueness and coldness. In spite of the fever the general

health may remain surprisingly good, but there may be some signs of collapse when a rigor occurs, and sometimes there are nervous symptoms which suggest a diagnosis of meningitis. Such a case may be difficult of diagnosis if your mind is not open to the possibility of pyelitis, but if you only think of that possibility there is usually no difficulty, and on inquiry you may find that there is some frequency of micturition, probably accompanied by more or less pain, and that the water has been noticed to be thick. In quite a number of cases, however, there are no urinary symptoms, and the amount of pus and albumin in the urine may be so small as to be easily overlooked unless you examine the urine in a glass vessel. Pus may even be present in only microscopical quantity. Bacteriological examination will set all doubts at rest. The moral is that in all cases of fever of doubtful origin in childhood one should examine the urine for *B. coli*. In boys a suitable specimen can be obtained after cleaning the glans with a disinfectant lotion and rejecting the first portion of the stream. With girls it is sometimes necessary to obtain a catheter specimen since contamination is frequent.

In older children the constitutional symptoms tend to be less severe and the local ones (pain, frequency, etc.) more conspicuous. In the acute cases some other acute infection such as typhoid, pneumonia or appendicitis may be simulated, whilst the chronic cases may be characterized simply by vague ill-health with recurring bouts of fever and even vomiting.

(4) *Suppurative nephritis* is fortunately a rare result of an infection, and is generally fatal. It is distinguished by the ordinary signs of an acute nephritis along with a high temperature, and is usually associated with evidences of pyelitis or cystitis as well.

The **prognosis** in cases of infection by the *B. coli* is on the whole favourable, but they are often very obstinate to treat. It is not usually difficult to get rid of the constitutional symptoms, but to banish the bacillus from the urine altogether is by no means an easy matter, and it is apt to persist off and on for long periods. Death also sometimes results. Thus Dr. John Thomson found that out of twenty-four cases of *B. coli* pyelitis in infancy three died, and out of sixty cases at all ages treated at Great Ormond Street Hospital six proved fatal (Jeffreys). In the chronic cases in older children there is always the risk of irreparable damage to the kidneys being gradually brought about.

You may now ask, *How does the B. coli get into the urinary tract at all?* This question has not been definitely settled, but there are three possibilities. (1) Infection might be by the blood-stream, the bacilli being excreted by the kidney. There is reason to believe that this rarely occurs. (2) The infection might be from below upwards, the bacilli getting up the urethra. This view is widely held, and the alleged greater frequency of cases in girls is believed to support it, the idea being that the short female urethra is more easily traversed. (3) Infection might be direct from the bowel to the kidney. Investigations made at the Children's Hospital tend to show that this is certainly a route of infection, the bacilli passing direct from the ascending colon to the right kidney, the transference being perhaps favoured by some damage to the mucous membrane of the bowel.

In some cases, at least, a diseased appendix which was adherent to the kidney has been the source of infection.

Seeing that the bowel is the primary source of the organisms, one obvious part of **treatment** must always be to avoid further irritation of the mucus membrane. Purgatives must not be given: constipation should be dealt with by enemas. Intestinal disinfectants are probably ineffective. If there is any reason to suspect disease of the appendix, that organ should be removed. The rest of the treatment must depend to some extent upon the special site of the infection. If there is cystitis, washing out of the bladder is useful. In cases of acute cystitis and pyelitis great benefit results from the use of citrate of potash in large doses. It is almost a specific in the cases of *coli* pyelitis in infants, but it must be given freely and one must go on increasing the dose until the urine is alkaline. Surprisingly large quantities are sometimes necessary to effect this, 15 grains three or four times a day being none too much in many cases even in infancy. Bicarbonate of soda may be given with it. So soon as the urine is alkaline the temperature and other constitutional signs usually subside in the most dramatic fashion, but the administration of alkalis in quantity sufficient to neutralize the urine should be kept up for some days in order to prevent a relapse. Throughout the acute phase the patient should be encouraged to drink diluents freely. In the more chronic cases the alkaline treatment is not so successful. Should it fail, you may try a combination of hexamine with acid sodium phosphate. The same measures are applicable in the cases of pure bacilluria.

In obstinate cases of all forms the use of a **vaccine,** preferably prepared from the urine of the patient is always worth trying, but I would warn you not to expect too much from it. The trial of a **ketogenic diet** has lately been recommended in the resistant cases of *B. coli* infection, but I have not been greatly impressed with its results in children and some patients cannot tolerate it. In any

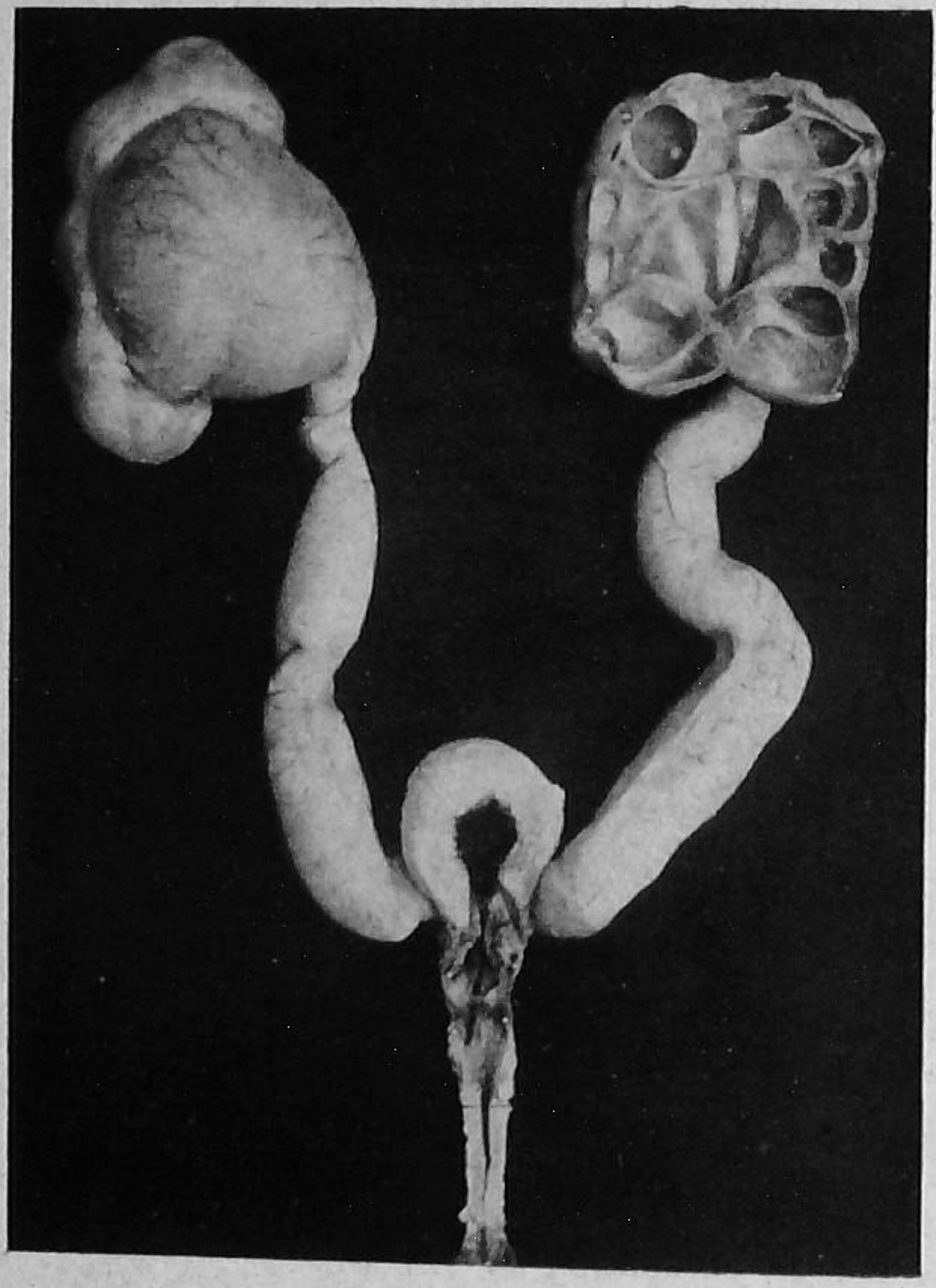

FIG. 95.—POSTERIOR URETHRAL VALVE (MARKED BY ROD) WITH EXTREME DILATATION OF BOTH URETERS AND KIDNEYS, AND HYPERTROPHY OF BLADDER.

case equally efficient results as regards raising the acidity of the urine can be achieved by the use of **mandelic acid,** but the use of **sulphonamide** has probably rendered both of these methods obsolete. A course of full dosage (as for pneumonia, see p. 254) will clear up most resistant infections.

In chronic and persistent infection of the urinary tract some **malformation** of it, such as urethral obstruction, double ureter,

and so on, should always be thought of; but for the diagnosis and treatment of such conditions you will need the help of a genito-urinary surgeon. I have brought an illustration (fig. 95) of such a condition. If a chronic infection is due to some underlying anatomical cause it is of great importance to deal with it surgically as soon as possible.

FUNCTIONAL ALBUMINURIA

I may now pass on to consider some of the commoner affections of the kidney in childhood, and first of all I may say a word about *functional albuminuria*. This condition, also known as physiological, orthostatic, cyclical, postural, or intermittent albuminuria—is, as you know, one which has given rise to much discussion. I do not propose to attempt to deal at all fully with the many interesting points which have been raised with regard to it, but I would only say that there can be no doubt (1) that such a condition really exists, (2) that it passes off usually after puberty, without apparently leaving the kidney in any way damaged, although in a few cases it persists into adult life, and (3) that it is of no serious significance. The condition is not really a disease, but merely a symptom, and is especially met with in children who suffer from general debility and poor physique with a tendency to lordosis.

The important question, of course, is how to tell this form of albuminuria from that of genuine nephritis. The chief points of distinction are (1) that functional albuminuria is not present when the child first gets up in the morning, but only comes on after he has been going about for an hour or two; (2) that blood-cells and granular casts are never present, although the hyaline variety may be; (3) the cardio-vascular signs of nephritis are absent and renal function tests are normal. I have observed, too, that in cases of functional albuminuria the urine usually gives a definite cloud on the addition of acetic acid in the cold alone, whereas this does not usually happen in cases of organic albuminuria. This point has not received much attention, but I believe it to be worth noting. It means, I suppose, that the protein present in the 'functional' cases is not all albumin or globulin, but is in part a mucin or nuclein compound. Many theories have been advanced to explain the cause of the passage of albumin into the urine in these cases, but the most convincing is that which attributes it to alterations in the vascular tension in the kidney when the erect posture is assumed.

The suggestion has been put forward that the occurrence of the albuminuria is the result of a lessened coagulability in the blood, and it is said that it can be made to disappear by the administration of calcium lactate. I cannot say, however, that I have found this happen in the few cases in which I have tried it, and I am rather sceptical about this theory. As regards the **treatment** of these cases, I believe the only thing to be done is to attend to the general health, and to leave the albuminuria to look after itself.

I may now go on to say a few words on the subject of hæmaturia in childhood.

HÆMATURIA

A child may be brought to you by the mother with the statement that he passes blood in the urine. There is here one fallacy which I would warn you against, and it is that mothers are apt to regard as blood what is really not blood at all, but a brick-dust deposit of urates. Another is that the urine may be coloured red by eosin, or other dyes contained in sweets. You must also make sure that it is blood and not hæmoglobin which is present as in paroxysmal and other forms of **hæmoglobinuria.** But assuming you have verified the fact that it is blood, there are but few possible causes of it which you have to consider. One is **hæmophilia,** which may be a cause of hæmaturia, especially in boys. Another is **scurvy,** which is often overlooked because examination of the urine of young infants is apt to be omitted. I remember a child who was under the care of a surgeon in a hospital for the passage of blood, and who was believed to be the subject of stone in the kidney. An operation was performed, but no stone was found. A physician then saw the child, and inquired into the diet; the replies given led him to think that it was a case of scurvy, and on putting the child on to fresh vegetables and fruit the hæmaturia disappeared in a few days. This, then, was a case in which failure to remember that scurvy may be a cause of hæmaturia led to the performance of an unnecessary operation. A third cause of hæmaturia is the **passage of urates and oxalates.** The hæmaturia in such a case is usually, I think, paroxysmal or intermittent, coming on and lasting for a few days and then passing off again. Careful examination will show the presence of crystals of uric acid or oxalic acid, and suitable treatment will cause the blood to disappear. A fourth cause is **sarcoma of the kidney,** and one which it is well to bear in mind.

Not long ago I saw a case where this gave rise to great difficulty. It was that of a young child who passed a trivial amount of blood in the urine, but passed it continually. Oxalates were discovered in the urine, and it was thought that they were the cause of the hæmaturia. But special treatment directed to remedying this did not affect the passage of blood, although the oxalates ceased to appear. By and by an abdominal tumour became evident, and operation showed it to be a renal sarcoma. Remember always, then, that continuous small bleedings may be a comparatively early sign of sarcoma of the kidney in a young child.

In older children, in addition to the causes already mentioned, you should think of **Raynaud's disease,** of **purpura,** and of **rheumatism** as possible causes of the appearance of blood or blood-pigment in the urine, and in the two former of these hæmaturia may be present as the only symptom. Quite recently I have seen in consultation two girls, both of whom suffered from rheumatic endocarditis and slight hæmaturia. Whether the renal hæmorrhage in these cases was due to embolism or not I cannot say, but it passed off quickly in each case.

In small male infants blood may occur because of a small **meatal ulcer,** just inside the urethra, caused by an ammoniacal irritation. It heals readily if boric acid powder is scattered freely on the napkins.

Finally, you must remember that cases of **hæmorrhagic nephritis** may present themselves under the guise of a hæmaturia, but I shall refer to these cases again in a moment.

NEPHRITIS

In childhood one has to deal, for practical purposes, with only two varieties of inflammation of the kidney: (1) acute nephritis, and (2) chronic parenchymatous nephritis. It is true that the chronic interstitial form does occur, but it is very rare, and is often associated with renal dwarfism.

Acute nephritis presents two types—(1) the acute hæmorrhagic or glomerular, and (2) the acute tubular or parenchymatous, although in this form the glomeruli are usually more or less involved too.

(1) **Acute hæmorrhagic nephritis,** due to an inflammation of the glomeruli, is the commonest form of nephritis in childhood and fortunately also the least serious. It usually follows a streptococcal

infection of the throat after an interval of ten to fourteen days, and the first thing noticed may be the alteration in the colour of the urine which may indeed in some cases look like pure blood. Œdema is often entirely absent or at most confined to a little puffiness about the eyes. There may be some rise of temperature, but the child rarely seems really ill. The blood-pressure is not raised except at the outset. The urine may be very scanty for the first few days. It contains usually relatively more blood than albumin so that pure hæmaturia is simulated. The deposit shows granular casts. Blood disappears from the urine in a few days as a rule, but a trace of albumin may be present even for weeks. If there is a focus of sepsis anywhere, as in the tonsils, ear, etc., relapses may continue to occur until the focus is removed.

(2) **Acute parenchymatous nephritis,** in which the tubules are involved as well as the glomeruli is rarer than the glomerular type and is characterized by having less blood and much more albumin in the urine with more numerous casts. Dropsy is a constant feature and a rise of blood-pressure with urea-retention more likely to occur. The child is more ill and the prognosis, both immediate and remote, not so good.

Chronic parenchymatous nephritis may follow upon an acute attack, but often seems to be chronic from the beginning. It is characterized by obstinate œdema with abundant albuminuria but no hæmaturia, a normal blood-pressure and no increase of urea in the blood. The exact pathology of this form is much disputed, and it is not certain that it is primarily or solely a disease of the kidney at all. The term 'nephrosis' was introduced to explain the type of case with œdema and heavy albuminuria without any history of an acute nephritis. It is supposed that there is a degeneration rather than an inflammation of the renal epithelium, and that is borne out by the fact that spontaneous cure sometimes occurs, suggesting that the changes are not irreversible, as they would be in any but mild inflammation. Into these recondite questions, however, I do not propose to enter.

In young infants nephritis, especially when it occurs in the course of an acute illness, such as broncho-pneumonia or infective diarrhœa, may easily be overlooked, as there may be little to direct attention to it and the urine is difficult to examine. I should warn you also that in infancy œdema may occur without nephritis in the form of the so-called *toxic* or *essential œdema*, the pathology of which is

obscure. It is specially apt to occur in cases of wasting and diarrhœa, but is not necessarily of serious omen. In syphilitic infants, on the other hand, a form of nephritis occurs associated with anæmia and œdema of the feet but without other signs of syphilis. It is usually fatal.

Causes of Nephritis

Acute nephritis in children is usually the result of the action of a bacterial toxin, particularly a streptococcal toxin, on the kidney. Scarlatina and diphtheria are both apt to cause nephritis in this way, the toxin of the former acting chiefly upon the glomeruli and that of diphtheria on the tubules, but the other acute specific fevers may also cause a nephritis occasionally and so, indeed, may any infection. It is probable, in fact, that so-called ' febrile albuminuria ' is really a sign of a mild and transient kidney inflammation. Even more important as a cause, especially of glomerular nephritis, are foci of chronic sepsis, particularly infection of the tonsils and middle-ear.

In a considerable number of cases, however, especially of the tubular variety and of nephrosis, no sources of bacterial infection can be discovered and such cases have sometimes been attributed to ' chill.' It is the fashion at present to pooh-pooh chill as a factor in the production of disease, but I am not at all sure that the old view is not right and that some cases of nephritis of obscure origin are not due to it.

Treatment

In *acute* cases the essentials are (1) complete rest in bed, (2) warmth, (3) a diet of water, fruit-juice, sugar-drinks and weak tea for the first two or three days, followed by a milk and farinaceous régime whilst the acute phase lasts, (4) the eradication of all foci of sepsis so soon as the acute stage is over, even although the operation is followed, as it often is, by a temporary fresh flare-up of nephritis.

I am a believer also in the acute stage in the benefit of applying hot poultices to the loins. Drugs, on the other hand, are of little use, although there is no harm in keeping the urine neutral or slightly alkaline by means of a vegetable salt of potash. The bowels must be kept open, but purgatives must on no account be overdone. Sulphonamides must not be given.

In the *chronic* cases rest, more or less complete, is still essential. The diet, however, must be abundant and mixed, although it is

advisable, if dropsy is prominent, to let it be as far as possible salt-free. In the nephrotic cases with much œdema a high protein diet often does best. Ordinary diuretics are not of much help, although diuretin (gr. 5 t.d.s.) is worth trying. Urea, also, in doses of 10 to 15 grains thrice daily, sometimes acts well, but it should not be used if the blood urea is already high and its use should not be persisted in after a few days if no diuresis has resulted.

In some of the badly-waterlogged cases I have carried out tapping in the same way as in cardiac dropsy and sometimes with great benefit. I have never known any ill-results from it. I need hardly say that the eradication of septic foci is just as necessary in the chronic cases as in the more acute.

A curious phenomenon which often occurs in these cases is *spontaneous diuresis*. The child may be quite waterlogged and the dropsy have failed to respond to all treatment when suddenly a great flow of urine sets in and all the fluid drains away in two or three days. Only too often, however, it returns, and such spontaneous diuresis may recur several times in the course of the case, and should it happen to set in shortly after a new diuretic mixture has been exhibited the mixture is very apt to get the credit.

For the *anæmia* which is always present in cases of chronic parenchymatous nephritis, iron is useful, and may be given either in the form of liq. ferri acetatis combined with acetate of potash, or in the old combination of sulphate of iron and sulphate of magnesia, if it is desired to act upon the bowels. The use of iron seems not only to lessen anæmia, but to favour the restitution of the kidney epithelium, and is therefore actually curative. If all the symptoms have disappeared, but some albumin is still present in the urine, change of air to a mild, dry climate is often helpful.

VULVO-VAGINITIS

I wish now to say a word or two about chronic *vulvo-vaginitis* in childhood, which is by no means an infrequent affection, especially in patients of the hospital class. There are two forms of it—the simple form in which pyogenic organisms are the cause, and the gonorrhœal form which is due to the gonococcus. The latter appears to be the commoner. The view long held that vulvo-vaginitis belongs to the ‘ strumous ’ order of diseases and is a sign of general ill-health is, I believe, erroneous. It appears to be really a purely local affection requiring local treatment. The gonorrhœal variety

is horribly contagious, and if introduced into a ward may spread round it like wild-fire in spite of stringent precautions. On the other hand, it is a peculiar fact that it rarely leads to other gonorrhœal complications, such as blepharitis, arthritis, or salpingitis.

Treatment of vulvo-vaginitis must be thorough and energetic as the disease is extremely obstinate and may last for months, whilst relapses after apparent cure are very common.

In *simple* cases the parts should be thoroughly irrigated two or three times daily with an antiseptic solution (saturated boric acid or 1 in 5,000 perchloride). The solution should be applied with a syringe or a swab, and gauze soaked in it may be left in contact with the mucous membrane. The surrounding skin should be protected by white precipitate ointment (5 grains to the ounce). In the more severe cases silver solutions (1 to 5 per cent. protargol) may be used instead of the above lotions. Suppositories containing $1\frac{1}{2}$ grains of salol in cocoa-butter may be introduced into the vagina two or three times daily. General cleanliness should be strictly observed, and the use of a sulphur bath every other night is often helpful. The presence of a foreign body must not be overlooked in resistant cases.

In the *gonorrhœal* cases treatment must be even more energetic. As I have already told you, this form of the disease is highly contagious, and the patient should therefore be strictly isolated and the utmost care taken to prevent the carrying of infection by napkins, washing utensils, sheets, etc. All such articles should be thoroughly disinfected before being washed. The local treatment is the same in principle as that employed in simple cases, but stronger antiseptics are required (e.g. perchloride 1 in 2,000, protargol 5 to 10 per cent., argyrol 5 to 25 per cent., $\frac{1}{2}$ drachm of either of the latter being injected twice a day). In obstinate cases it may be necessary to apply iodine solution to the cervix and vaginal walls through an endoscope tube two or three times weekly. A diaper or pad should be worn to prevent the child carrying infection to the eyes. Quarantine should be continued for some time after all organisms have disappeared from the discharge.

Preparations of œstrin, to change the epithelium of the vagina, are of value in obstinate cases. However, as with pyelitis, it looks as if the sulphonamide group of drugs are going to displace all other remedies. Certainly the rapidity with which bacterial tests become negative is remarkable. Full dosage should be given for a week.

LECTURE XXXIV

AFFECTIONS OF THE LIVER IN CHILDHOOD

The liver, being the great organ of metabolism, and metabolism being very active in childhood, one need not be surprised that the liver at this age is relatively larger than in adults. It can thus normally be felt in young children well below the costal margin, and owing also to the wide subcostal angle in early life, a larger area of the organ is in contact with the anterior abdominal wall than in grown-up persons. These facts of normal anatomy must be borne in mind in the clinical examination of children.

ENLARGEMENTS

Before determining that the liver is enlarged it is well to make sure that it is not merely being pushed down. This happens in cases of right pleural effusion, and also from mere constriction of the lower part of the chest—for example, in rickets. Hypertrophy and dilatation of the heart may also displace the liver downwards, and so does a large effusion into the pericardium. In the latter case it is the left lobe of the liver which is chiefly affected, and displacement downwards of this lobe is a valuable sign of pericardial effusion. Congenital dislocation of the liver—hepatoptosis—is very rare, and I have met with only one example of it. The organ in such cases is not only displaced downwards, but is rotated forwards on its transverse axis, so that the upper surface comes to lie against the abdominal wall. If the existence of such a condition is remembered the diagnosis is not difficult.

True enlargement of the liver is not uncommon. That which is most frequently met with is the enlargement which results from congestion in mitral disease. It is the only form of enlargement in which tenderness of the organ is a prominent feature.

Fatty infiltration may cause a considerable increase in size, and is relatively common in childhood. It is met with, for instance,

in many cases of chronic tuberculosis. It used to be thought that this was due to the prolonged administration of cod-liver oil in such cases, but I think it is now generally admitted that this explanation is incorrect. Amyloid disease, though it can cause great enlargement, is now rarely met with. The enlargements due to cirrhosis I shall speak of later. Apart from these, one meets occasionally with cases of enlarged liver in which the cause of the condition is quite obscure. Here are two examples:

Alice F——, aged one year and four months. Swelling of abdomen for two months, but otherwise well, except for slight bronchitis. No family or personal evidence of syphilis.

On examination a healthy-looking child with a prominent abdomen. Liver uniformly enlarged, reaching fully to umbilicus; smooth. No ascites. Spleen not felt.

Two years later the liver was found to be practically normal in size.

Eric V——, aged one and a half years. Enlargement of abdomen for three or four months; health otherwise good. On examination slight rickets, but otherwise healthy and no sign of syphilis. Liver reaches to below umbilicus; smooth, firm. Spleen not felt. No ascites.

The liver gradually got smaller, and eight years later it was only one finger's breadth below the costal margin.

In another case I was able to follow the patient for fifteen years, and watched the liver gradually recede from the umbilicus to the costal margin. What the nature of the enlargement is in these cases I am unable to say. A rare affection, called Gierke's disease, due to infiltration with glycogen which is faultily metabolized, may explain some of them.

Enlargement, the result of tumours, is, in childhood, extremely rare. Hydatids and sarcoma are the only forms of tumour ever likely to be met with.

I shall now consider those forms of hepatic disease in which jaundice is the most prominent symptom, and it will be convenient to divide them into—first, cases met with in newly-born or quite young infants; and, secondly, into cases of jaundice in older children.

JAUNDICE SHORTLY AFTER BIRTH

1. **Icterus neonatorum** is, of course, the commonest form of jaundice in newly-born babies. As it is so well known I shall not say much about it, except to remind you that it comes on two or

three days after birth, and usually passes off quite soon. In a few cases, however, it may persist for a month or even longer, and these cases are apt to give trouble in diagnosis. It is well to remember that icterus neonatorum differs from other forms of jaundice in that the pigmentation appears *first in the skin*, and only affects the conjunctiva (if at all) later. Further, bile does not disappear from the stools, and, unless in severe cases, is usually absent from the urine, although the latter often contains some urobilin. Icterus neonatorum is the result, as I told you in the lecture on diseases of the blood, of the excessive destruction of red cells which occurs after birth, and, being physiological, requires no treatment.

2. **Congenital obliteration of the bile ducts** is attended by severe jaundice. The latter is not, as one might suppose, present at birth, but comes on very soon after, and is far more profound than in icterus neonatorum. It can be diagnosed by the fact that the liver is large and irregular, and the spleen also palpable. It is entirely insusceptible of treatment, and usually proves fatal within a few months at most.

3. Jaundice is often present in **septic states** in early infancy, and is then always of grave omen. The septic infection may be derived from the umbilicus, in which case the jaundice is due to pyelephlebitis, but in some cases it appears to result from an infection of the intestines. It can be diagnosed by the presence of fever, great constitutional disturbance, and often, in the severer cases, by the appearance of a purpuric eruption. It is usually fatal. The treatment is that of any septic infection.

4. **Syphilitic hepatitis** is another cause, though a rare one, of jaundice in newly-born infants. It consists in a small round-celled infiltration of the liver—a sort of diffuse gumma—which gives rise to considerable enlargement. The jaundice in these cases is usually only slight, and other signs of congenital syphilis are pronounced. The spleen is usually palpable, and ascites may be present. Such cases are generally fatal (in fact the commonest result is a stillborn infant), but occasionally recovery takes place under vigorous anti-syphilitic treatment. The following was a fatal case of this sort:

Fred C—, aged seven weeks. Admitted for swelling of abdomen and scrotum. The second child of the family, the first having been stillborn. Healthy at birth, and weighed 9 pounds. Two weeks after birth a rash appeared on the legs, and then the abdomen and scrotum swelled. On examination a pale, slightly-jaundiced infant, with a faded

coppery rash on the body. Abdomen large, tense, and full of fluid; œdema of scrotum and legs. Veins over abdomen distended; liver considerably enlarged; spleen just felt. Eighteen ounces of fluid withdrawn from abdomen with relief to œdema, and mercury pushed, but one week later died at home in a fit.

5. There is a rare form of jaundice met with soon after birth to which the title **icterus gravis neonatorum** is given. It is a severe and, if untreated, usually fatal form of jaundice which often affects several children in the family in succession, although, curiously, the first child is often born healthy. It is associated with a severe anæmia due to an intense hæmolysis and is related to a certain type of congenital anæmia and to a rare condition of generalized œdema in the new-born, known as hydrops fœtalis. Until recently, the cause of these three conditions was obscure, but now it is possible to explain them by rather a fascinating story. It appears that 85 per cent. of white persons have in their red blood cells an antigen found also in the red cells of monkeys and therefore called Rhesus or Rh factor. Unfortunately, 15 per cent. of human subjects do not possess this factor, and since it is inherited as a Mendelian dominant, it follows that with mating between an Rh-positive man and an Rh-negative woman, there is the possibility of the offspring being Rh-positive. What appears to happen then is that the antigen from the fœtus, passing into the mother's circulation, stimulates the formation of anti-bodies which, passing back into the fœtal circulation, proceed to a serious destruction of the infant's red cells. This explains, at any rate in part, the peculiar characters of the disease, and the fact that the first baby escapes may well be because sufficient anti-body has not been formed on that occasion.

The jaundice is obvious at birth, and careful examination will show that a severe anæmia is also present. The spleen may be slightly enlarged, there is bile in the urine, and usually, but little if any, in the motions. The infant becomes drowsy and, unless treated, usually passes into coma and dies. Examination of the blood confirms the severe anæmia with a great excess of nucleated red cells, showing that the infant is trying to make up for the blood destruction. Treatment consists in giving a transfusion of Rh-negative blood of suitable group, and since this is a highly technical procedure, it follows that the child must be transferred to a hospital unless it is born in an institution where the staff are prepared to undertake the transfusion. The modern blood depots now keep a supply of this

blood available in many instances. Unfortunately, even if immediate treatment is successful, some of these infants grow up with evidence of permanent affection of the nervous system.

JAUNDICE IN OLDER CHILDREN

Catarrhal jaundice is by far the commonest form in older children. The name is probably a misnomer, because modern investigation of adult cases by liver puncture has shown that there is a hepatitis present in most, if not all, instances. As you are all no doubt familiar with its chief features, I need not describe it in detail, but I would remind you that it presents all the characters of a mild obstructive jaundice, and that it may be attended by considerable enlargement of the liver, brought about by its engorgement with bile. There may also be slight fever at the outset. You will sometimes notice in practice that the disease occurs in small epidemics, but there is no reason to suppose that it is infectious. The jaundice always passes off in less than six weeks.

Although catarrhal jaundice is never fatal, I believe that it is best to treat sufferers from it in bed, and I have the impression that the jaundice clears up more quickly if the liver is poulticed. The rest of the treatment consists in the adoption of a non-fatty, light, mixed diet, containing little milk, and the use of a stomachic mixture of rhubarb and soda, with small doses of calomel and salines as aperients. I think it is wise to keep the liver well stocked with glycogen, as it were, and hence glucose should be freely used for sweetening all drinks.

Acholuric family jaundice may sometimes be met with in childhood, but is quite distinct from the grave familial form already described. It is due, you remember, to an inherited and inborn fragility of the red blood-corpuscles, and is characterized by slight jaundice, without, however, the presence of bile in the urine, and by enlargement of the spleen, which is often of very considerable degree. There will be a history of similar cases in previous generations. Treatment is by splenectomy.

Gallstones as a cause of jaundice in childhood need not be seriously reckoned with. I have, indeed, only once met with a case of biliary colic in a child (a boy, aged six years), and in his case, though the presence of stones was proved by operation, jaundice did not occur.

CIRRHOSIS OF THE LIVER IN CHILDHOOD

Another important group of diseases of the liver is that in which some form of cirrhosis is present.

1. The first of these is ordinary **multilobar or portal** cirrhosis, progressive idiopathic cirrhosis as it is sometimes termed, which is pathologically just like the common alcoholic cirrhosis of the adult. This is usually met with after the age of five, and the child will probably be brought to you because of enlargement of the abdomen. On examination you will find ascites, and the liver may be felt to be enlarged and hard. Such cases have to be distinguished from abdominal tuberculosis, which is, of course, a far commoner cause of ascites in childhood than cirrhosis of the liver. Distension of veins on the surface of the abdomen is much in favour of cirrhosis (fig. 96) and so also is the presence of a palpable spleen. Of great value also is the recognition of the 'cirrhotic facies,' which consists in slight icterus of the conjunctivæ with dilated venules or telangiectases on the cheeks and sides of the nose.

FIG. 96.—CIRRHOSIS OF THE LIVER WITH DISTENSION OF SURFACE VEINS.

Such a facies is present in all forms of cirrhosis, and is of much help in diagnosis. The cause of this form of cirrhosis in childhood is quite obscure. It is only very rarely that one gets a history of the taking of alcohol, and certainly in children some other poison must be responsible, although its nature is entirely unknown. The disease runs a course of little more than a year's duration as a rule, and death usually results from profuse or repeated gastro-intestinal hæmorrhage, though a few cases terminate in cholæmia.

No treatment has any effect on the progress of the disease. The following are illustrative cases :

Mary W——, aged five years. Never strong since birth. About a year ago abdomen noticed to be large ; two weeks ago diarrhœa set in, and a week later abdomen swelled rapidly. No history of alcohol.

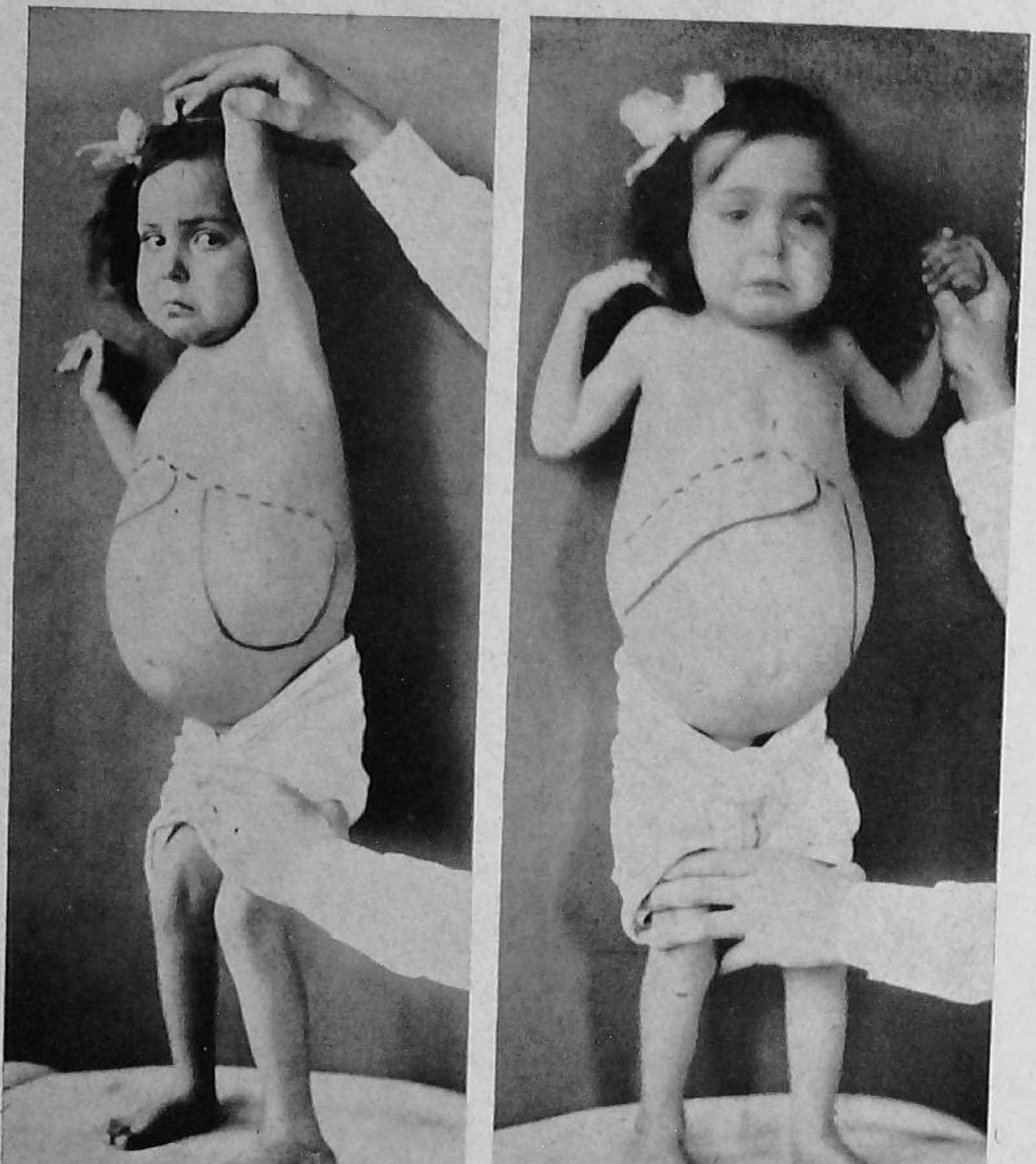

FIG. 97.—CIRRHOSIS OF LIVER WITH GREAT ENLARGEMENT OF SPLEEN DUE TO PARTIAL OBLITERATION OF THE BILE DUCTS.

On examination rather wasted; sallow and slightly icteric child, with telangiectases on face ; much ascites ; liver and spleen palpable. Examination of blood and urine negative. Continuous slight irregular fever. Tapped several times, but gradually went down-hill, and died two months after admission. Necropsy showed a fine hob-nail cirrhosis and a congested spleen.

Alice M——, aged eleven years. Two years ago an attack of jaundice, and never regained her proper colour afterwards. One year later

had another bad attack, with loss of voice. Soon after this bleeding of the gums began, and has recurred on and off ever since. Previous health always good; no history of alcohol.

On examination well nourished; slightly jaundiced; many telangiectases on face; gums soft; liver small, hard, and irregular; spleen easily palpable; slight ascites. Fragility of red cells normal; Wassermann negative. Died rather suddenly with cholæmia two months after she came under observation.

2. **Syphilitic cirrhosis** occurs as one of the later tertiary manifestations of inherited syphilis, usually about the same time as interstitial keratitis. The liver in such cases is often very large and may be extremely irregular, and there is also great enlargement of the spleen. Ascites is far less common than in the portal type, and there may be no jaundice. The 'cirrhotic facies' is present combined with that of congenital syphilis, and if there is any doubt about the diagnosis a blood test will settle it. The following is a typical case.

Ivy M——, aged nine years. Abdomen swelling for a year; no other symptoms and no previous illness of importance. Snuffled as a baby; the two preceding children stillborn.

On examination earthy complexion; telangiectases on face; teeth normal, and no other evidence of syphilis. Liver much enlarged, irregular, and hard; spleen three fingers' breadth below costal margin. No ascites; Wassermann positive.

Syphilitic cirrhosis has a far better prognosis than the portal type, and under antisyphilitic treatment by mercury and iodides the liver may become quite small again. In other cases the disease at least ceases to progress, and although the liver remains large a fair condition of health is maintained. Occasionally, however, gastro-intestinal hæmorrhage brings about a fatal issue.

3. You will find in your text-books that **unilobular or hypertrophic** cirrhosis is described as occurring in childhood. I confess that I am very sceptical about its existence. At all events I can only say that I have not yet met with a case of it. I once thought that I had, for the patient presented all the features alleged to characterize the disease—viz., jaundice with little ascites, recurring bouts of pyrexia, with a large, smooth, hard liver. At autopsy, however, it proved to be a case of partial obliteration of the bile ducts. Here are the notes:

Annie K——, aged four and a half years. Delicate from birth;

had jaundice when two weeks old, lasting a month; present illness began with jaundice three months ago.

On examination a thin child; considerably jaundiced; fingers clubbed; telangiectases on face, and some dilated veins over abdomen; slight ascites; liver enlarged and smooth; spleen almost to umbilicus; Wassermann negative; slight continuous pyrexia. Omentopexy was performed, and liver found to be cirrhotic and gall-bladder shrivelled up. Died three months later. Necropsy showed obstruction of hepatic duct (? congenital), with great dilatation of intrahepatic ducts.

Since this experience I have been more sceptical than ever about hypertrophic cirrhosis.

4. That form of hepatic cirrhosis to which the name of '**Banti's disease**' has been applied is occasionally met with in children towards the period of puberty. It is really, as you know, the terminal stage of a splenic anæmia of the adult type. This form of splenic anæmia is sometimes met with in later childhood (it must be clearly distinguished from the splenic anæmia of infancy), and after lasting for a few years terminates in cirrhosis of the portal type, which ultimately proves fatal. It can be distinguished by the fact that great enlargement of the spleen has for long preceded the signs of cirrhosis, and if splenectomy is performed before the cirrhosis has developed there is reason to believe that the disease can be arrested.

5. **Obstruction of the bile ducts**—usually congenital—gives rise to a unilobular cirrhosis, but I have already referred to this when describing congenital obliteration of the bile ducts, and I mentioned a moment ago a case of it which simulated hypertrophic cirrhosis.

6. Lastly, one meets occasionally with cases of what one may speak of as '**infective cirrhosis.**' I have seen a few of these cases in children. They begin like ordinary catarrhal jaundice, only usually with more fever, and after lasting for some time gradually go on to cirrhosis which proves fatal. The following case illustrates their features:

A schoolboy, aged fourteen years, was attacked about Christmas with diarrhœa and vomiting accompanied by jaundice. The diarrhœa and vomiting passed off in a few days, but the jaundice never quite cleared, and in the following February became more severe and he was in bed for a week. He then went to the seaside for a change, but the jaundice persisted and he had occasional epistaxis. In June he began to have fever and pain in the right side of the chest. He was

considerably jaundiced and had ascites, but the liver and spleen were not felt. He had rather profound secondary anæmia. Shortly afterwards he died, and the autopsy showed a shrunken liver, with areas of diffuse fibrosis and sero-fibrinous peritonitis and pleurisy.

It seems reasonable to suppose that we are dealing in these cases with an infection of the bile passages, which leads to cirrhosis as a secondary result. I have tried draining the gall-bladder in two of them, but in neither case did one succeed in arresting the disease.

FUNCTIONAL DISORDERS OF THE LIVER

I have now dealt with the chief organic diseases of the liver, and I wish in what remains of this lecture to speak briefly of its functional disorders. As the liver has so many and so complicated functions to perform in the economy of the body, one might expect disorders of these to be not infrequent. Nor, I believe, are they; only the existence of a functional disturbance of the liver in any case is difficult to prove. I think, however, that one can recognize a type of child in whom the liver normally functions badly—who is the victim, if you like, of an inborn 'hepatic inadequacy.' Such children are often known by their mothers to be 'livery' or 'bilious,' and may be brought to you on that account; most of the so-called 'acidosis' children are of this type. They are usually thin, highly-strung children, of capricious appetite and tend to be constipated, the motions being frequently pale. Their tongues are often coated, the breath foul, and the conjunctivæ muddy. Urates or uric acids are prone to appear in the urine, or at other times they may pass for short periods an abundant urine of low gravity. Ketone bodies may be present during exacerbations of the condition. Though intellectually such children tend to be above the average, they are subject to fits of languor, depression, and irritable temper. They are often the subjects of migraine, cyclical vomiting, or unexplained bouts of fever.

A digestive peculiarity which is, I believe, invariably present in these livery children is inability to digest milk in any quantity. You will, indeed, often get a history that even from birth the child has had difficulty in dealing with milk, especially cow's milk, yet in spite of this mothers are apt to pour milk into them in the vain hope of making them fat. This is a complete mistake. It is, I believe, impossible to fatten such children, and if you try to do so

you will only succeed in making them more bilious. The proper way to manage them is to cut down rigidly the livery foods—milk, cream, eggs and chocolates—in the diet, and to give them a regular aperient which also acts on the liver, such as a combination of rhubarb and grey powder. They should be protected from chill, from excitement, and from over-fatigue, and if they are sent away for a change it should not be to the seaside, but to a high, bracing inland place. On such lines you can effect a great improvement in health, but the functional inefficiency of the liver often persists into adult life, although it may then express itself in other ways.

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LECTURE XXXV

SOME COMMON SYMPTOMS OF DISEASE IN CHILDREN AND THEIR DIAGNOSTIC SIGNIFICANCE

In the present lecture I wish to consider some common symptoms for which children are brought to you, and their significance in diagnosis.

COUGH

Of cough as a symptom in children it would be a mistake to suppose that it is always, or indeed usually, the result of pulmonary disease; the majority of coughs are not due to any abnormal condition of the lungs or bronchi. It is all the more necessary that you should be warned of this, because the mother is apt to make the diagnosis herself, and instead of saying the child is suffering from cough she will say he has 'bronchitis,' and so you are apt to be misled. Statistics were collected from one of the children's hospitals in London on this subject, and it was found that of a large number of consecutive cases of cough only 31 per cent. were due to disease in the chest. So you see that in a great majority of cases the cough arises in the throat. The **abnormal conditions of the throat** which may give rise to cough in children are three in number. There may be simple pharyngitis, or enlargement of the tonsils (often with adenoids), or elongation of the uvula. These three conditions may occur separately, or you may have combinations of them in any one case. From the character of the cough you can often infer what is likely to be its cause. For instance, in *simple pharyngitis* it is usually dry and tickling, occurring at frequent intervals. This type of cough is sometimes due essentially to infection of the nasal sinuses. Pus trickles down the back of the throat and sets up a pharyngitis. In *adenoids* the cough is rather of a choking character, often accompanied by vomiting, and some-

times by epistaxis. On the other hand, the cough which is associated with an *enlarged uvula* is characterized by the fact that it comes on when the child lies down because the uvula falls back into the pharynx and irritates the posterior wall. This is also a feature of the cough due to sinus injection.

If the cough has anything of a croupy character you should at once suspect the **larynx,** but I referred to cough of this kind in a previous lecture when I was speaking of croup (p. 229). On the other hand, if the cough is more brassy in character it indicates pressure on the **trachea** such as may occur in enlargement of the thymus.

When you come to consider cough arising from **irritation in the bronchi,** you will often have to face the problem, Is it simple bronchitis, or is it whooping-cough? Now, that is a question which it is not always easy to answer, although it is one to which the mother expects you to be able to give a definite reply. But although you cannot always be certain about the existence of *whooping-cough*, there are some things which should make you suspicious of it. The cough of whooping-cough is essentially paroxysmal, it tends to occur specially at night, and it is often followed by vomiting. Further, when you examine the child you find that the cough is out of all proportion to the amount of physical signs. There may be only a few moist sounds in the lungs, although you are told that the cough is one of very great severity. You may find also a sublingual ulcer, which is of real value, because it may occur before the cough has begun to take on the true whooping character. There is another thing which you will notice on examining a child whom you suspect to have whooping-cough, and that is that he looks puffy about the eyes; the congestion of the head and face which the paroxysm occasions leads to puffiness, which to the experienced eye is often suspicious. The tongue also is usually furred. You will also be apt to mistake for whooping-cough the cough which results from pressure by **enlarged bronchial glands,** but I have already spoken of the symptoms and diagnosis of these in a previous lecture. Another sort of cough which may simulate whooping-cough is that of **bronchiectasis.** But bronchiectasis is not particularly common in children, and from the clubbing of fingers and signs of cavities in the lungs, and the character of the expectoration, there should be no difficulty in making the diagnosis.

The cough of a true **bronchitis** is easily recognized by the char-

acteristic physical signs in the chest and if there is a wheezing element in it **asthma** should be suspected. Lobar **pneumonia** may, in the child, be entirely unaccompanied by cough, but in the lobular variety cough results from the coexisting bronchitis.

When you have excluded the causes of cough which I have mentioned, those due to the state of the throat and those in the lungs, there remain a considerable number for which you can find no definite cause. Some of these are certainly due to gastro-intestinal disorders. There really is in children, I think, such a thing as **stomach cough** ; at all events, you find children who have a constant cough associated with a coated tongue and a red irritable state of the posterior pharyngeal wall which persists until the state of the digestive organs is attended to. And some people have even described a cough as the result of the presence of worms and the reflex irritation which they produce.

Finally, there is an uncommon form of cough, but one which it is well to be familiar with, and that is the **hysterical cough.** I remember, shortly after I started seeing out-patients some years ago, a boy being brought, who, as he sat in the waiting-room, kept on every half-minute or oftener giving out a curious kind of short cough. I examined him without finding any cause for it, and from the history of the case and the general impression one got I came to the conclusion that it was hysterical. Under the influence of suggestion and one or two doses of valerian the cough stopped absolutely in twenty-four hours. More commonly the hysterical cough is not of that sort ; it is of the typical barking character met with in older subjects. One has also to remember that cough may be a form of **habit spasm** (p. 291) which may follow upon an attack of whooping-cough or even upon enlarged tonsils, the cough persisting as a habit after its cause has been removed.

INABILITY TO TALK

I now want to take up together two symptoms which mothers frequently complain of in their children. One is *inability to talk* at the proper age, and the other is *inability to walk.* If a child is brought to you because of **inability to talk,** you should bear in mind that there are only a few possibilities you need consider. In the first place the child may be a **deaf-mute.** In such a case you will find that the child is absolutely deaf, and you will sometimes get a history of several similar cases having occurred in the same family.

These cases should not present any great difficulty in diagnosis. Secondly, some children are unable to talk at the proper time because of **mental deficiency,** or else because of backwardness short of actual deficiency. These cases are more difficult to recognize. You should examine the child for the physical stigmata of mental deficiency, for cranial abnormalities, and the other signs which you know to be the outward expression of retarded development of the brain. But in the lesser degrees of mental deficiency where such physical signs are absent or are not pronounced, you may often be in doubt as to whether that is truly the cause of the child being backward in talking.

Thirdly, pathological conditions in the throat, especially **adenoids** and **enlarged tonsils,** may be a cause of difficulty. A typical case of this kind was that of a girl two years and three months old, who was brought by her mother because she was unable to talk, although ' she made violent efforts to do so.' The mother said the child heard well, she was obviously quite intelligent, and the only thing one could make out on physical examination was the presence of an enormous mass of adenoids in the naso-pharynx. These were removed by operation, and five months later the mother wrote that the child was talking well.

A rare cause is **tongue-tie.** Mothers think it is a common affection, and most children who have difficulty in learning to talk are popularly believed to suffer from it. Now, tongue-tie does occur, but it is very uncommon. I can only recall at this moment three well-marked cases of it, and I doubt if it ever does produce true inability to talk—that is to say, real dumbness. It may cause difficulty in talking clearly and in pronouncing certain consonants for which the tongue is particularly required, but you must distinguish that from inability to talk at all. A still rarer cause is **congenital aphasia,** a condition of which I have never met with an example, but which is described as an occasional congenital defect.

INABILITY TO WALK

To turn to the kindred symptom—inability to walk, there you have to recognize two groups of cases : first, those in which the child has never walked at all ; and, second, those in which he has at one time or another been able to walk, but has gone ' off his legs,' as the mother puts it.

With regard to the first group there are three possibilities : either

the child is the subject of rickets, or of some form of paralysis, or it is mentally defective. Now, in **rickets** the difficulty is merely one of walking. Whilst the child sits on his mother's lap he can move his legs about freely, whereas in cases of paralysis the child is not only unable to walk, but he cannot move the affected limbs even when sitting. In rickets you will find also the other general signs of the disorder—enlargement of the epiphyses, deformity of the chest, and so on; the knee-jerks, moreover, are unaffected, whereas in paralysis they are exaggerated or diminished.

Of **paralysis** as a cause of inability to walk there are two chief varieties: one is infantile paralysis, the other is spastic or cerebral paralysis. Infantile paralysis comes on definitely at some period subsequent to birth, whereas spastic paralysis is often congenital. In infantile paralysis you will find the knee-jerk on the affected side diminished and the muscles atrophied. In spastic paralysis you will find the knee-jerk exaggerated and the muscles of the limb well developed. **Mental deficiency** you will recognize by the other general signs of mental impairment which I have mentioned before. Never forget, however, the possibility that children may be unable to walk for **surgical reasons** as well as medical. I have seen *congenital double dislocation* of the hip produce great delay in learning to walk, and such cases are apt to be overlooked by the physician whose mind is fixed upon medical points.

When a child who has hitherto been able to walk 'goes off his legs,' you are dealing with a different order of cases. Many children who have had an **acute illness** will go off their legs for a long time afterwards, although you can find no special cause for their doing so. Probably it is partly because of muscular weakness, and partly because the child actually forgets how to walk; having only recently acquired that rather difficult art, he gets out of practice when laid up in bed, and when he gets on to his feet once more he has to learn all over again. Such patients may go on without walking for a long time, but you may rest assured that they will walk in time. Another cause of a child going off his legs is that he may have developed **infantile paralysis,** but I have already told you how that is to be recognized.

A rarer cause which it is important to recognize is **rheumatism,** slight degrees of which affecting the hip or the muscles at the back of the knee-joint or ankle may cause the child to go off his legs on account of pain.

Remember, again, the possibility of **surgical causes.** One of the earliest signs of hip disease may be that the child goes off his feet.

HEADACHE

The common causes of headache in childhood are not the same as in adult life. Circulatory disturbances, for instance, are rare in early life, whilst 'reflex' headache—particularly from 'eye strain'—is frequent. If a child of school age suffers from headache, examine his **refraction** and see if there is any hypermetropia or astigmatism, and if you find either and get the error corrected by proper glasses you will often find that the headache disappears at once. Another group of causes of headache may be described as **toxic**; constipation and biliousness are examples, just as in grown-up people, and these are to be met by appropriate remedies. **Rheumatic headache** is not uncommon, the pain in this case being situated in the scalp. **Fatigue,** either physical or mental, may easily cause headache, especially in children who are growing fast. It yields to rest and tonics. **Cerebral tumour,** again, occurs with considerable frequency in children, and may be a cause of severe headache. Here you should inquire for other symptoms of tumour, such as vomiting, and look particularly for cranial nerve paralyses and for papillœdema. Nocturnal headache is always suggestive of an organic cause. **Adenoids** are a pretty common cause of headache in childhood. Here the headache is usually frontal. **Migraine** also is met with in older children, and sometimes with great severity. It is characterized by its periodicity and by its being accompanied by vomiting. Lastly, you should never omit to examine the urine in cases of headache, as an unsuspected **nephritis** which has perhaps followed one of the infectious fevers may be the cause.

INSOMNIA

Sleeplessness is a not uncommon trouble in early life, and one which is a great source of anxiety to parents. In some children it seems to be almost a congenital peculiarity. You will be told that the child 'has never slept well.' In such a case you will usually find that there is an inherent instability of the nervous system which shows itself in general excitability and nervousness.

Apart from such congenital predisposition, the commonest exciting

causes of insomnia in young infants are indigestion and teething. In every such case, therefore, it is your duty to inquire carefully into the feeding, into the possibility of colic, and into the state of the bowels. By rectifying anything which is wrong in these directions you will often effect an immediate cure. In other cases, again, insufficiency of food is the cause, and sleep is restored when more food is given. Much also seems to depend upon the personality of the nurse. Some nurses seem to have the knack of getting infants to sleep well, whilst others have not ; it would almost appear, indeed, as if in certain cases the nurse got on the infant's nerves, so remarkable may be the effect of a change of attendant. In such cases the insomnia is usually a form of ' negativism ' (see Lecture XXV). In persistent and intractable cases of insomnia in infancy one is always rather suspicious of mental defect, of which sleeplessness seems to be an occasional symptom.

In older children the two great causes of bad sleeping are— (1) *indigestion*, and (2) *overstrain at school*. Obstructed respiration from adenoids or large tonsils is also a potent cause. The treatment to be adopted in each of these cases is self-evident. Hypnotics should only be resorted to temporarily and in order to aid in the re-establishment of the sleep-habit. Bromural is one of the best. It may be given in 5-grain doses with a little hot milk at bedtime to a child of five. Avoidance of all excitement, an open-air holiday, and attention to the digestion and bowels are, however, the sovereign remedies.

In recent years cases have been observed of a peculiar inversion of the normal sleep rhythm in children who have suffered from encephalitis lethargica. In such a case the child will be wakeful and restless all night and sleep for a large part of the day. This condition is one of the curious sequelæ of encephalitis, but I believe it tends to disappear spontaneously with the lapse of time.

SWELLING OF THE ABDOMEN

Enlargement of the abdomen is a symptom for which children will sometimes be brought to you. The first thing to bear in mind here is that the abdomen of young infants is normally rather prominent, and an anxious and inexperienced mother is apt to mistake the natural prominence for a sign of disease. Assuming that you have satisfied yourself that the prominence is really patho-

logical, you should look next for signs of **rickets,** bearing in mind how greatly the abdomen may be enlarged in that disease. The reason for this I have already pointed out (p. 162).

Abdominal tuberculosis is another frequent cause of abdominal enlargement in children of two years and upwards, and should be examined for with great care. In the exudative form the discovery of ascites will determine the diagnosis, whilst in the adhesive variety one must look for lumps or thickened omentum. Mere **chronic intestinal dyspepsia** with flatulence may give rise to considerable and lasting abdominal distension, but I think it is a good rule to be suspicious of tuberculosis in any such case in which appropriate treatment does not effect a speedy improvement.

The distension met with in **cœliac disease** may also closely simulate that due to tuberculosis, but I have spoken of the differential diagnosis in an earlier lecture (p. 99). I would also remind you that the rare condition known as '**megacolon**' may cause a very great degree of abdominal swelling, but to this also I have already referred (p. 113).

Enlargement of the viscera, particularly of the spleen or kidney, may be a cause. I need not remind you of the main points to be attended to in distinguishing a large spleen from a large kidney, but would only say here that their differentiation is by no means always so easy as the text-books might lead you to suppose, and that mistakes in the matter are apt to be made by the most experienced and careful. The chief causes of enlargement of the kidney in early life are sarcoma and hydronephrosis; the causes of splenomegaly have already been dealt with in the lecture on that subject.

VOMITING

I have already considered chronic vomiting **as it occurs in infants** in a previous lecture, and would only remind you here that you have to distinguish between (1) 'physiological' vomiting, the automatic rejection of excess of milk, or what the nurses sometimes call 'puking,' the distinctive feature of which is that it is not attended by loss of weight; (2) the vomiting of pyloric spasm; (3) the vomiting of indigestion. The differential diagnosis of these is fully considered in Lecture VII.

After the period of infancy **acute vomiting** is much more often met with than chronic. It may be due (1) to *acute indigestion.*

The history of the taking of an excessive or indigestible meal, or of some special article of food, will usually keep you right here. The vomiting of acute indigestion is not usually accompanied by fever, but in (2) *acute gastritis* this is present. Here, again, there will usually be a history of some noxious substance having been swallowed, or of exposure to cold; but sometimes you will have difficulty in distinguishing it from (3) *symptomatic vomiting*, which sets in at the outset of some acute disease. Any of the acute specific fevers may be so ushered in, especially scarlet fever, but vomiting is also a very frequent symptom at the outset of pneumonia. *Meningitis*, again, is often preceded by vomiting for a short time before any other signs develop. A combination of vomiting with a slow pulse and constipation is very suggestive of it. (4) *Acute intestinal obstruction and appendicitis* should always be thought of in a case of sudden vomiting, especially if attended by abdominal pain. Remember, too, the comparative frequency of *cerebral tumour* in childhood, and in all cases of chronic vomiting examine the optic discs. *Uræmic vomiting* is not common in children, but there is no harm in examining the urine in an obscure case.

Bilious Attacks

Cases of frequently recurring attacks of vomiting are often puzzling. They are usually spoken of by the mother as '**bilious attacks,** but you must remember that this is a purely lay diagnosis, and you must not be satisfied with it.

As a matter of fact, there are several different conditions which may lead to so-called bilious attacks. They may, for instance, be simply frequent attacks of **gastritis,** due to habitual overfeeding, particularly, I think, to giving too much or too rich milk. Others are certainly **migraine,** but these can be distinguished by the fact that they are accompanied by headache, which *precedes* the vomiting. A very important group are those which are due to **appendicitis.** It is going too far to regard *all* bilious attacks in children as caused by the appendix, as surgeons especially are apt to do, but undoubtedly in some cases this is the real cause. Nor are these always easy to distinguish; but if you have an opportunity of examining the child during an attack you will usually be able to make out some of the usual signs which point to appendicitis, although between attacks these may be entirely absent.

A rarer cause is **hydronephrosis,** which usually results from kinking of the ureter. I remember the case of a boy of fourteen who ever since his early childhood had suffered from what were called 'bilious attacks,' characterized by vomiting with some abdominal pain, the attacks recurring every six weeks or so and lasting for three or four days at a time. On examination of the abdomen the left kidney was found to be definitely enlarged, and on further investigation this proved to be the result of hydronephrosis.

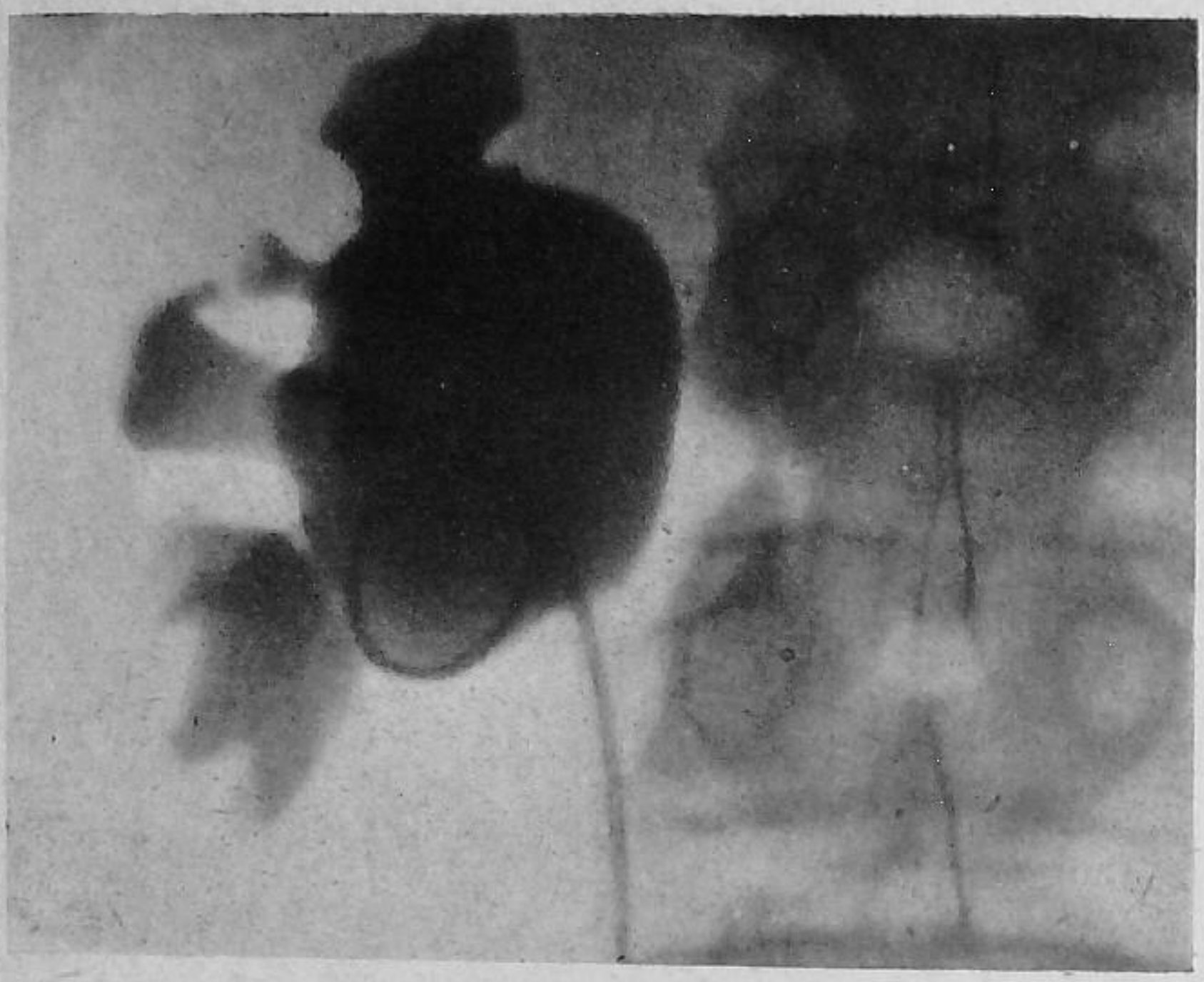

FIG. 98.—RETROGRADE PYELOGRAPHY IN CHILD OF 12.
Hydronephrosis of right kidney due to aberrant branch of renal artery. Child suffered from recurrent attacks of abdominal pain and vomiting. Lump felt in right loin, only during attack. Urine infected. Cured by removal of aberrant artery.

The kidney was removed and the 'bilious attacks' ceased. I have here an X-ray picture (fig. 98) from a similar case in a girl. At operation the surgeon found an aberrant renal artery kinking the right ureter and he was able to remove this.

The most striking, however, of all the cases of 'bilious attacks' are those which belong to the group of **recurrent** or **cyclic vomiting,** and as these are of very great importance, and as I have not had the opportunity of referring to them before, I wish to say a few words about them now.

The name 'cyclic' vomiting intimates one of the chief features of the disorder—namely, that the attacks recur in definite and often very regular cycles, separated by a few weeks of ordinary health.

The vomiting may begin suddenly, but often the child is 'out of sorts,' languid, and dull for a day or two before it begins, and during this period the motions may be pale in colour. Once it starts, the vomiting is extremely urgent, and nothing—not even water—may stay in the stomach. Obstinate constipation usually accompanies it, and the child is restless and drowsy and soon comes to look extremely ill, with sunken eyes, a retracted abdomen, and inelastic skin. Some degree of fever is usually present as well. The tongue is dry and furred, the pulse quick, and there may be an ethereal odour in the breath. On adding some perchloride of iron solution to the urine a deep port-wine reaction will be obtained, indicating the presence of aceto-acetic acid and acetone. In other words, **ketosis**—often incorrectly spoken of as acidosis—is a striking feature of the disease, but whether it is the cause of the condition or a mere accompaniment of it is disputed.

The pathology of cyclic vomiting is obscure, but personally I am convinced that the liver is the organ chiefly at fault. Apparently the normal fat metabolism in that organ is for some reason interfered with, the consequence being that fatty acids find entrance to the blood and ketosis ensues. It almost seems as if between attacks the liver gets gradually choked up, and the accumulation is got rid of during the attack. It is a state of things closely allied to migraine, and it is interesting to note that there is often a family history of sick-headaches in these cases, and that some children, when they have outgrown their attacks of cyclic vomiting, become the victims of migraine in adult life.

The **treatment** of these patients in an attack must be energetic, for if serious acid-intoxication supervenes a fatal issue may follow. The main indication is to give bicarbonate of soda freely to counteract ketosis. It may be given by the mouth *ad lib.*, dissolved in soda-water, and though much will be vomited, some is retained. It may also be administered in 5 to 10 per cent. solution by the rectum; glucose (5 per cent.) may be added to the solution with advantage, or honey, if pure glucose is unobtainable. If there is great restlessness and distress, morphine should be given subcutaneously. The dose is about one-hundredth of a grain for every year of the child's age. Morphine should not be given, however, if the urine is suppressed.

Such should be your treatment in an attack, and you have next to consider how recurrences are to be prevented. The essentials

are to cut down cream, milk, fats and chocolates in the diet, and to ensure efficient daily action of the liver and bowels, for which purpose there is nothing better than the regular use of rhubarb and grey powder.

As these children are usually highly strung, they should be protected from all excitement and overstrain, and undue fatigue and chill should be guarded against, as these are often the determining causes of an attack.

In some of these cases nasopharyngeal infection seems to play a part in bringing about the attacks, and the removal of tonsils and adenoids is often followed by the happiest results.

A word of warning, however, may not be out of place as to the danger of operations on the subjects of cyclic vomiting. Undoubtedly such children are more apt to suffer from ketosis after anæsthetics, especially chloroform, than normal children, and this should be borne in mind in the event of any operation having to be undertaken in such a patient, and all suitable precautions to prevent it observed.

Hæmatemesis is not commonly met with in children, for the usual causes of it in the adult—cirrhosis, gastric ulcer, and malignant disease—are all very rare in early life. If, therefore, a child vomits blood there is always a strong presumption that the blood has been swallowed. In the case of an infant at the breast it may be derived from a crack in the nipple, and if you are in doubt as to whether this is really the source try the effect of a breast-pump. I have also known the blood come from the sucking of an inflamed gum during teething. True hæmatemesis may occur as a symptom of the hæmorrhagic disease of the newly-born (see p. 44), and I have also known it take place during the violent vomiting associated with pyloric spasm. In such cases, however, it is generally of the grumous or coffee-grounds variety—not profuse. In older children hæmatemesis is usually a symptom of a blood-state such as purpura or leukæmia, but occasionally it results from cirrhosis of the liver, and is then a serious symptom. Very rarely is it due to ulceration of the stomach or duodenum.

ANOREXIA

The last symptom I propose to consider with you to-day is loss of appetite, or anorexia. A good appetite is so natural to a healthy child that any impairment of it at once attracts the mother's atten-

tion, and children will often be brought to you for this and nothing else. In little babies loss of appetite may occur as a temporary matter from **anything which upsets the child's health.** Vaccination and teething are examples in point. In bottle-fed babies the cause may be **monotony in the meals,** and the introduction of a little variety, such as substituting a broth feed for one of milk, or the addition of a little Mellin's Food to some of the bottles, may set matters right. During the process of **weaning,** again, there may be difficulty in getting the child to take its food, whether the weaning be from the breast or the bottle. Here a little firmness is what is chiefly needed.

During the second year of life loss of appetite may occur in association with **chronic catarrh of the bowel,** a condition which I have considered at length in an earlier lecture (p. 184), and if the child is also rickety, as it often is, may constitute a serious obstacle to treatment. The treatment is that of the bowel condition. At this age, too, profound anorexia may precede the development of **tuberculosis,** and is therefore a symptom fraught with some anxiety.

In the second year of life and onwards it is not uncommon, especially in little girls, to meet with the most profound disinclination for food, which is not unlike the anorexia nervosa of adults. In many of these cases the mother is driven almost distracted by repeated failure to get the child to take a proper meal. Such children are usually spoilt, and often solitary, and the curious thing is that they will usually take food quite well from a stranger or when other children are present. The most effective treatment is to send them away from home with a strange and sensible nurse, and to a place where they will have the society of healthy children. These cases are more fully considered in Lecture XXV.

In older children loss of appetite is a common symptom of **chronic dyspepsia,** a condition which we studied in detail in Lecture XVII. It may, however, be of nervous origin. Thus I have known it occur as a manifestation of **hysteria,** and only yield to isolation and the threat of the stomach tube. I remember too, a little boy of about four who was almost certainly somewhat of a 'moral imbecile' who flatly refused to touch any liquid out of a cup, and even preferred to be fed nasally, yet who would eat solids quite well. Such a case is difficult to classify. Occasionally, although very rarely, one meets with refusal of food from **melancholia,**

just as one does in grown-up patients. I recall such a case in the person of a little girl of six who suffered from severe valvular disease, and who declined to eat 'because she did not want to get better.' Such an unchildlike attitude, however, is fortunately very rare at this time of life.

As regards the treatment of loss of appetite *per se*, and in the absence of any organic disease, you should try the usual alkalis and bitters before meals, along with aperients and mercurials. If these fail there is nothing to equal change of air, especially to the seaside, in bringing back the natural desire for food. This is a remedy which is equally applicable at all ages.

[illegible] MEDICAL OFFICER
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LECTURE XXXVI

THE DIAGNOSTIC SIGNIFICANCE OF ABDOMINAL PAIN IN CHILDHOOD

Abdominal pain in a child must always be a source of anxiety to the doctor. It may mean so little or so much. It may be due to nothing worse than an attack of acute indigestion set up by a green apple; or it may indicate an attack of acute peritonitis set up by a gangrenous appendix. It is a matter, therefore, which craves wary walking in diagnosis, for a mistake may be fatal, and between one's anxiety not to overlook a serious cause and one's natural reluctance to alarm the parents unduly it is often difficult to steer a proper course. Our difficulty in these cases is increased by the fact that pain may be the sole symptom of abdominal disease, and the little patient may be unable to help us, as an adult can, by describing its exact situation, character, and so forth, which are often so significant for diagnosis. It seemed, therefore, that we might profitably employ our time to-day by considering together the chief causes of abdominal pain in childhood, with a view to making a diagnosis by the method of exclusion at least, if no other is available.

EXTRA-ABDOMINAL CAUSES OF PAIN

At the outset it is well to remember that pain felt in the abdomen is not necessarily being *caused* there. In children, who localize their sensations badly, these extra-abdominal causes of pain are specially apt to lead to error. We may divide them according as the cause has its seat (1) in the abdominal wall, using that term in its widest sense; or (2) in the thorax.

1. **Causes situated in the abdominal wall.**

Of causes situated in the abdominal wall, one of the commonest is **spinal caries.** The pain in such cases is due, of course, to pressure on the posterior nerve roots, and is usually referred along the lower intercostal nerves to the epigastrium. It may come on

quite early in the disease before any gross spinal deformity has shown itself, and the child will be brought to you under the belief that he is suffering from some abdominal trouble. It is well, therefore, to make it a rule carefully to examine the back in all cases of chronic epigastric pain in children. Slight prominence of one or more vertebral spines in the lower dorsal region can usually be detected, with tenderness on percussion over them and some stiffness on stooping. If in doubt one can call in the help of the radiologist. **Lateral curvature,** in its more pronounced degrees, is a cause of a similar sort of error, but is less likely to be overlooked.

Rheumatism, or perhaps one should call it fibrositis, affecting the abdominal muscles may easily simulate pain of intra-abdominal origin, and, like all rheumatic manifestations, is relatively common in childhood. It may be met with either at the insertion of the muscles into the ribs or iliac crests, or in the aponeurotic sheaths of the muscles themselves. The well-known 'stitch in the side,' which is so common in children, may perhaps be of this nature, but rheumatic pain is also met with in a chronic and protracted form. Fibrositis may be distinguished by the fact that the pain comes on when the affected muscle is thrown into action, as, for example, when the patient tries to sit up in bed without using his hands. There is often quite acute tenderness at certain parts of the abdominal wall, especially when the muscle is contracted, and if the abdomen is palpated in the ordinary way this may be mistaken for tenderness of an underlying organ. This error may be avoided by noting that tenderness is also elicited when the abdominal wall is grasped or compressed laterally. This form of pain is commonest in children of school age, and often follows hard exercise, and if the muscles of the right lower quadrant are involved, appendicitis may be simulated; one sometimes wonders whether cases of so-called 'rheumatic appendicitis' cured by salicylates are not really of this nature.

Herniæ through the abdominal wall are not such a common cause of obscure chronic abdominal pain in the child as they are in the adult, but in the form of omental herniæ especially they should always be thought of, and so should the possibility of an **undescended testis.**

Hip disease I have sometimes known to lead to error; not so much tuberculous hip disease as an acute inflammatory condition

in or near the joint. This may cause pain referred to the lower part of the abdomen; there may be fever, and the child may lie with the legs drawn up; and when the right hip is affected simulation of appendicitis may be fairly close. The limitation of movement in the joint which careful examination reveals ought, however, to keep one right.

Last of the causes in the abdominal wall one may mention **herpes zoster.** Pain in this affection may precede the appearance of the eruption by some hours or even days, and be of considerable severity; and I have known more than one instance in which it led to a diagnosis of acute abdominal disease. It can be distinguished by

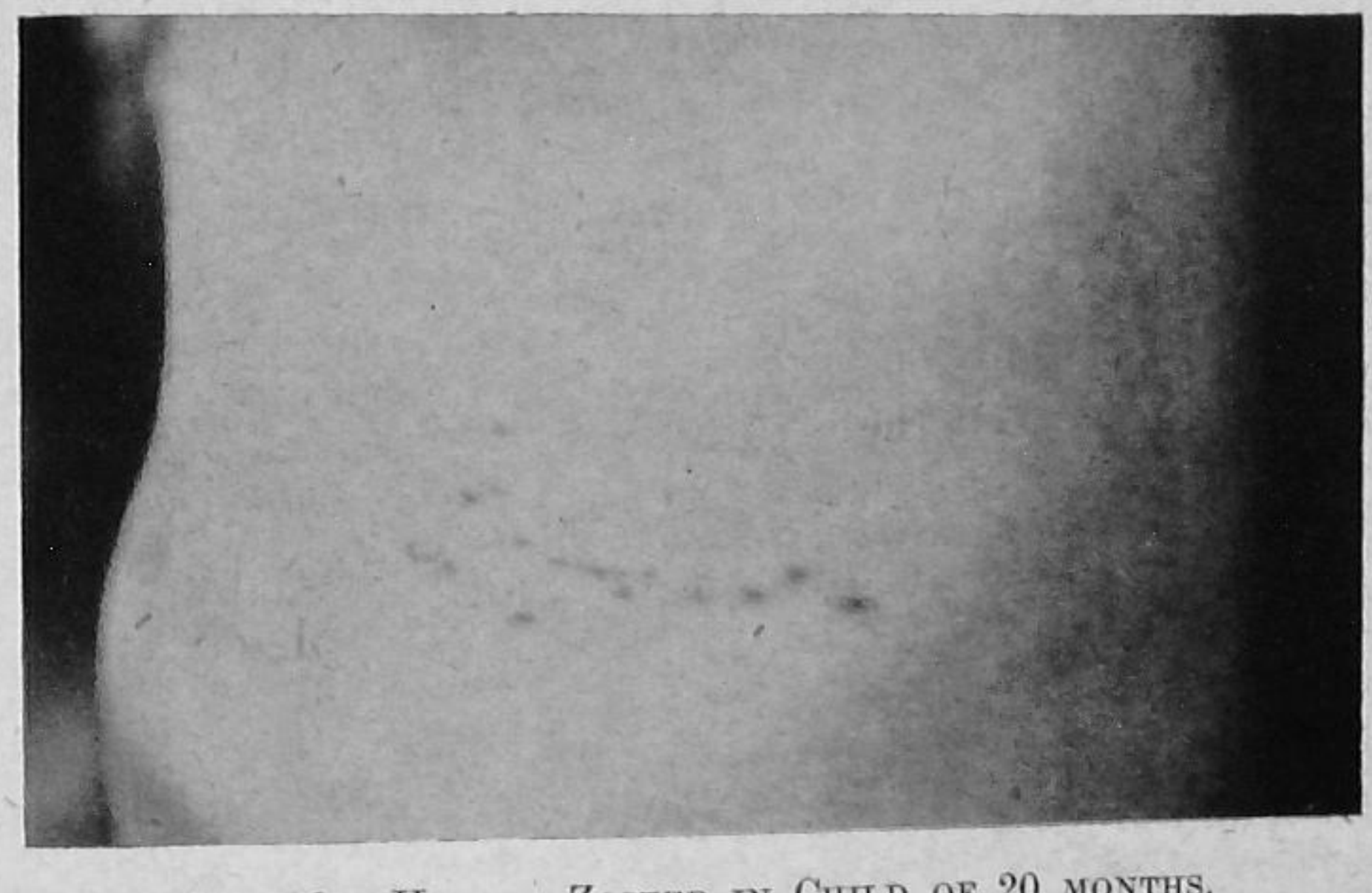

FIG. 99.—HERPES ZOSTER IN CHILD OF 20 MONTHS.

the fact that there is a band of superficial hyperæsthesia in the distribution of the affected nerve, and an absence of deep tenderness on palpation or of involuntary rigidity. All doubts are dispelled before long by the appearance of the eruption (fig. 99).

2. Thoracic causes.

The possibility of abdominal disease being simulated by acute intrathoracic conditions is now well recognized, but mistakes are still easy to make. The commonest form of error is to diagnose a case of pneumonia occurring at the right base as one of appendicitis. If the patient is a child, such an error is specially excusable, for in the child, more even than in the adult, the physical signs of a pneumonia may be entirely absent for some time after the disease has begun. Furthermore, vomiting is a constant and conspicuous

feature of early penumonia in childhood and helps still further to simulate the picture of abdominal disease. The abdominal pain in these cases is due, as you all know, to involvement of the lower intercostal nerves as they pass through the diaphragm, and in part also it may be a 'referred pain.' There is reason to believe that it only occurs when the diaphragmatic pleura is affected. Be this as it may, the combination of an acute onset with fever, vomiting, pain referred to the lower abdomen, and some abdominal rigidity coupled with an absence of the usual signs of pneumonia may be deceptive in the highest degree. In attempting to make a diagnosis in such a case you will look for an alteration in the pulse-respiration ratio, though it is well to remember that the respiration is not always hurried at the onset of pneumonia in a child. You will note that the abdominal tenderness is superficial, and that there is no sign of pain on moderately firm, deep palpation. You will also ascertain the presence or absence of tenderness in the recto-vesical pouch on examination by the rectum, although, perhaps, it is true to say that in the child this is more easily said than done. You will remember also that the higher the temperature the more likely is it that the case is one of pneumonia. In some cases you must remain in doubt, and then it is wise to stay your hand, and in a few hours, perhaps, the appearance of a pleuritic rub or a patch of dullness will make clear the nature of the disease.

Pericarditis, although apparently only when the diaphragmatic surface is involved, may also simulate abdominal disease, but it is a much less common source of error than pleurisy or pneumonia. I remember a case of the sort, which made a strong impression upon me at the time. The patient was a little boy, who was admitted to hospital under the belief that he had acute appendicitis. He looked very ill, lay with the knees drawn up and the abdomen rigid, complaining of pain which he referred to the hypogastrium. There was moderate fever, but an entire absence of other signs. I have forgotten whether he vomited. He was kept under observation for a few hours, and it was decided that if there were no more positive indications by the evening, the abdomen should be opened. Just before the time agreed upon for this, however, a loud pericardial rub became audible, and the case subsequently ran the course of an ordinary rheumatic pericarditis.

INTRA-ABDOMINAL CAUSES OF PAIN

Let me now pass on to consider the far more frequent cases in which the cause of pain is situated in the abdomen itself. We may divide these for the sake of convenience into (1) cases of sudden or catastrophic pain, and (2) cases of chronic or recurrent pain.

1. By the **'catastrophic' cases** I mean those in which the pain sets in suddenly—'out of the blue,' so to speak—and is of great intensity. They are the cases often spoken of as 'abdominal catastrophes,' and include many examples of what are known in surgical slang as 'the acute abdomen.' They are for the most part of surgical rather than medical interest and I shall not say much about them except to point out as far as one can those in which immediate laparotomy is indicated.

At the outset it may be well to remind you that the possible causes of an abdominal catastrophe are far fewer in the child than in the adult. Children do not suffer from perforated gastric or duodenal ulcer, for instance, or from acute pancreatitis, and I can recall only one case of biliary colic in a child. The possibilities which one has chiefly to consider when confronted with a case of severe abdominal pain of sudden onset at this age are acute indigestion, appendicitis, and some form of acute intestinal obstruction, of which that due to intussusception is specially frequent. Amongst the rarer causes one might perhaps mention abdominal or Henoch's purpura. Renal colic, which may also have to be considered, I shall have occasion to mention later on.

The immediate practical diagnostic problem in such a case is to eliminate acute indigestion. This is not always easy. The pain of acute indigestion may set in suddenly, may be of great severity, and may be attended by fever and considerable distress. Here is an example:

A boy of seven, whilst convalescing from a mild attack of whooping-cough, was attacked by sudden abdominal pain of considerable severity, and in an hour or two his temperature had run up to 104°. He was restless and distressed, and begged that 'something should be given him to relieve his pain.' In view of the recent whooping-cough various diagnostic possibilities presented themselves, but soon all doubts were resolved by the copious vomiting of a quantity of undigested food, shortly after which he broke into a profuse sweat, and the temperature came down with a run.

If in doubt about such a case the best course is not to pooh-pooh the whole affair as a mere stomach-ache and to give a dose of castor oil, but to hold one's hand and wait. Meanwhile there is no harm in giving an enema, and perhaps a mild emetic, and applying heat to the abdomen for relief of the pain.

The possibility of appendicitis is what will give you the most anxiety in these acute cases of pain. I shall not go into the diagnostic criteria in this matter in any detail, as they are much the same as hold good in the case of the adult, but I would only remind you that pain is the *earliest* symptom in appendicitis, that it is usually experienced first in the epigastrium, and only later settles into the right iliac fossa, and that it is rarely of great severity, except in a very nervous child. Vomiting nearly always occurs, but it follows the pain and does not *persist* as it does, for example, in cyclical vomiting; and although more or less fever is always present at some stage of the attack, it is rarely great in degree. In looking for muscular rigidity in children, *gentle* palpation is more important even than in the case of the adult.

Acute intestinal obstruction is nearly always attended by pain, which may be very severe; but I have seen cases of intussusception in which, at the outset at least, pain was entirely absent; probably they were cases in which there was an unusually long mesentery. The classical signs and symptoms of obstruction will be present in the child as in the adult, but it is well to remember that owing to the tendency of the child to develop a temperature on slight provocation, some fever may be present even in purely obstructive cases, especially, perhaps, in intussusception. In the examination of these cases an anæsthetic is often of great help. It saved the situation in the case of a little boy whom I saw not long ago, who had been seized two days previously with severe abdominal pain. Other symptoms of intussusception were absent, but the abdomen was rather rigid and difficult to examine. He did not seem at all ill, and one was inclined to regard it as a case of colic, but under a whiff of chloroform the abdominal walls immediately relaxed and a typical sausage-shaped tumour could then be felt running across the abdomen. Remember, however, that if a diagnosis of intussusception has been made on other grounds, failure to find a tumour under an anæsthetic should not alter one's opinion.

I have mentioned **abdominal purpura** as a possible cause of sudden acute pain. You will not expect to meet with this often,

but as it is usually attended by hæmorrhage from the bowel it may simulate intussusception rather closely. A mistake may be avoided by careful examination for purpuric blotches in the skin, which are often to be found in such cases.

2. **Chronic or recurrent pain.**—When an adult complains of chronic pain in the abdomen, the first organ one thinks of is the stomach. In the child, however, the stomach is rarely at fault in such circumstances. Children, I need hardly remind you, do not suffer from organic disease of the stomach, such as ulcer or carcinoma, and in them chronic or recurring abdominal pain usually has its seat of origin in the intestine. It is true that ordinary indigestion may cause some pains probably from distension of the stomach with gas; but it is rarely severe in degree—rather a mere discomfort—and its definite relation to meals and its relief after the belching up of wind will usually make the nature of the case plain.

The **intestinal** cases are, as I have said, far commoner, and we may divide them for convenience into those due to (*a*) ordinary colic; (*b*) 'umbilical' colic, the meaning of which is to be explained immediately; (*c*) enterospasm; and (*d*) chronic obstruction.

(*a*) *Ordinary colic* is, of course, the commonest cause of abdominal pain in little babies, but it is met with also in older children, and may contribute to the pain in those cases of acute indigestion of which I have already spoken. In the chronic or recurrent form it is more apt to appear as the variety known as 'umbilical' colic, which I shall speak of immediately. In whatever form it shows itself it is characteristic of colic that the pain occurs in spasms, that it is relieved by firm pressure—differing in this profoundly from the pain of appendicitis, for instance—and, what is of great diagnostic value, if it is observed, that it is relieved temporarily at least, by the expulsion of flatus from the bowel.

(*b*) The term '*umbilical*' *colic* was introduced by Moro, to designate a common group of cases met with in children of school age, in which the patient complains of frequently recurring abdominal pain which is always referred to the situation of the umbilicus. The pain may come on suddenly at any time in the day, but sometimes quite definitely after or during a meal. It may be severe whilst it lasts, even doubling the child up, and may be attended by sudden pallor. Vomiting is rare. The attack may last for only a moment or two or for a few hours, and there is sometimes a tendency

for attacks to exhibit periodicity. As I have said, these cases are by no means uncommon, and everyone who has done much out-patient work amongst children must be familiar with them, but their real nature is obscure. There are usually no objective signs of disease to be made out, and Moro was of opinion that the condition is a pure neurosis. I can hardly think this likely, and it seems more probable that we are dealing in these cases with a genuine colic in the large intestine occurring in rather constipated nervous children in whom, perhaps, the 'gastro-colic' reflex is abnormally active—a condition closely akin to lienteric diarrhœa. The treatment of these cases I spoke of when I was describing intestinal dyspepsia in a previous lecture (p. 190).

(*c*) *Enterospasm* as a cause of abdominal pain is met with in children who are the subject of muco-membranous colitis. This is not infrequent in childhood, although it appears to be rarer than it was some years ago, and when spasm of part of the colon occurs very severe pain may be produced. The contracted bowel may sometimes be felt—usually in the left iliac fossa—and the presence of mucus and membranes in the stools should make the diagnosis clear. It is worth remembering that muco-membranous colitis in children is sometimes attended by prolonged fever of considerable degree, and when attacks of enterospasm are superadded mistakes in diagnosis may easily occur.

(*d*) *Chronic intestinal obstruction* as a cause of abdominal pain is rare in childhood, and when it occurs is usually due either to compression of the bowel by a band or a persistent Meckel's diverticulum or to adhesions the result of old tuberculous peritonitis. The pain resembles that of colic in its nature, but there is usually some meteorism present and visible coils of intestine may be seen. There is not necessarily any vomiting. The use of the X-rays may help in the diagnosis if one is in doubt. The most difficult cases are those due to **chronic intussusception,** and in at least two instances of that condition I have known the true cause of the pain overlooked, even though the patient was for a long time under observation in hospital.

It should be noted that the history will always show that the pain in a case of chronic intussusception began *suddenly* at a definite day or even hour, and during the first few days of the illness there was some vomiting. The bowels in these cases may be normal in action, and blood entirely absent from the motions. Progressive

wasting is often the most prominent symptom. On abdominal palpation a sausage-shaped tumour may be felt running across the abdomen just below the liver, which, on careful palpation, can be felt to undergo alternate hardening and softening. Tuberculous peritonitis is the condition for which you are most likely to mistake it. If in doubt use the X-rays after a barium enema.

Appendicular pain.—I now come to the most anxious cases of all, those, namely, in which the question arises whether a chronic or recurrent pain has its seat in a diseased appendix. There are some, I believe, who deny the existence of chronic appendicitis in childhood altogether. I think I have heard a surgeon say that a child either has *acute* appendicitis or the symptoms are not due to the appendix at all. Most people will probably agree that this is an extreme view, but at the same time I am sure that one ought to be extremely cautious in diagnosing chronic appendicitis in children, especially when the pain is *confined* to the right side of the abdomen. On the other hand, it is probably nearer the truth to say that disease in the appendix in the child is never chronic from the beginning—there is always a preceding acute or subacute attack. In making a diagnosis, therefore, a history of such attacks should be inquired for with great care. They will usually be described as 'bilious attacks,' but if they were attended by pain, some vomiting and fever, and were bad enough on any occasion to confine the child to bed, one should be very suspicious. Physical examination in cases of chronic or relapsing appendicitis *between attacks* may be entirely negative, and I do not think that the use of the X-rays affords much help. One may be in doubt between chronic appendicitis and 'umbilical colic,' but the attacks of pain in the latter are of much shorter duration—usually only an affair of minutes—and are not attended by vomiting or any rise of temperature. If the parents, as so often happens, are worried about the state of the appendix, then I think it is best to advise exploration, although it is unwise to guarantee any result from the operation other than the relief of the parental mind; usually, however, something will be found, though it is oftener a mass of enlarged glands, a Meckel's diverticulum or some other abnormality than a really inflamed appendix.

Worms are often believed by parents to be a cause of abdominal pain in childhood, but my own experience would not bear this out. Round-worms are those which would be most likely to produce

the symptoms, for they are sometimes numerous enough even to produce intestinal obstruction. A vermifuge will soon settle the diagnosis.

Enlarged glands, on the other hand, are, I believe, a much commoner cause of abdominal pain than is generally realized. I do not mean tuberculous glands, but a simple enlargement of the glands at the lower end of the mesentery. These often become infected in cases of appendicitis in children, just as the glands at the angles of the jaw do in tonsillitis. I saw a very striking example of this not long ago in the case of a boy who was suddenly attacked with abdominal pain, vomiting, and fever. He presented the signs of appendicitis, but there was a curious tumour about the size of a walnut to be felt in the right iliac fossa which was certainly not the appendix. At operation it proved to be a mass of acutely inflamed glands, the appendix being also inflamed and its mucous membrane ulcerated. There is often no history of any acute disease in the cases of chronic enlargement of glands, though no doubt they must have been infected from the cæcum or appendix; but, once enlarged, they are apt to cause pain in the right lower quadrant, possibly by dragging upon or kinking the bowel. At all events, they are often the only thing found wrong in children on whom an exploratory operation has been performed for suspected chronic appendicitis, and their removal is usually followed by disappearance of the attacks of pain.

CAUSES IN THE URINARY TRACT

Affections of the urinary tract are a frequent and often overlooked cause of abdominal pain in early life, just as they are in the adult, although some conditions which are relatively common in grown-up persons are rare in the child. Movable kidney, for example, though it is occasionally met with in children, need hardly be thought of as a cause of pain. Attacks of true renal colic also are not often exhibited in the child, though gravel is perhaps a not uncommon cause of pain in babies. On the other hand, there are two conditions which are certainly more frequent sources of abdominal pain in the child than is generally realized—one is *kinking of the ureter* and the other is *ureteric calculus*.

Kinking of the ureter results, as you know, from a congenital abnormality in the distribution of the renal blood vessels. As a

rule it does not produce symptoms until early adult life, but I believe that it would be diagnosed oftener in the child if it were looked for. It is one of the causes of so-called 'bilious attacks.' I had once a very striking example of this in the case of a boy of fifteen who had suffered since his early childhood from attacks of abdominal pain and vomiting which recurred every few weeks with great regularity, and had been diagnosed as 'bilious attacks.' I happened to see him in an attack. He referred the pain to the left loin, and an enlarged and slightly tender kidney could easily be felt on palpation. Surgical investigation established the presence of kinking of the ureter, and a greatly disorganized kidney was subsequently removed. These cases are deceptive because of the regular periodicity of the pain and the entire absence of any urinary symptoms.

Ureteric calculus.—Just as the symptoms of a kinked ureter are apt to be diagnosed as 'bilious attacks,' so the symptoms of a ureteric calculus, when it occurs in the right side, are usually attributed to 'chronic appendix mischief,' and, indeed, the simulation may be very close. There may be a history of attacks of right-sided spasmodic pain, there may be a rise of temperature and tenderness over the ureter (which is very close to McBurney's point), and urinary symptoms may be absent. The moral is that a skiagram of the ureteric region should always be taken before an operation is performed in quest of a supposed diseased appendix.

In the male **urethral obstruction** or a narrow meatus, as a result of phimosis, may cause attacks of hypogastric pain from over-distension of the bladder, but the association between the pain and the difficulty of micturition is usually obvious, and the vesical tumour easily made out. In girls, on the other hand, one has sometimes to think of **pelvic causes** such as salpingitis, but I mention these rather for the sake of completeness than because I regard them as common causes of abdominal pain in childhood.

LECTURE XXXVII

FEVER OF OBSCURE ORIGIN

In the present lecture I propose to deal with a group of cases which, judging from the frequency with which one sees them in consultation, are very common, and which often give rise to great difficulty in diagnosis. I refer to cases in which a child exhibits a rise of temperature, but without any definite physical signs of disease—cases of *pyrexia et præterea nihil*.

Now, I need hardly point out that in *any* case of fever the diagnosis may be in doubt during the first two or three days, but the cases I shall speak of to-day are those in which the temperature has run on for a week or more and still there is nothing to show the cause of it. Such cases are apt to be labelled 'influenza,' but I would warn you against such a facile diagnosis. In the first place, influenza is not really common in young children, and you should be particularly guarded in diagnosing it unless the disease is prevailing as an epidemic at the time. In the second place, *uncomplicated* influenza does not last for more than a week, and if complications appear physical signs appear also. To pronounce a case of fever of obscure causation to be 'influenza' may satisfy parents who are clamouring for a diagnosis, but do not allow it to deceive yourself or to shut your mind to other and more likely possibilities.

Having put aside, then, the temptation to diagnose 'influenza' and have done with it, you must next proceed to arrive at the cause of the fever by a patient process of elimination. And first you have to ask yourself, Is the case one of general infection? The exanthemata are, of course, excluded by the absence of a rash, which would have appeared long before a week had elapsed, and there remain for consideration:

TYPHOID OR PARATYPHOID FEVER

Infection with an organism of the typhoid group is common even in early childhood, and, as it may appear in the most un-

accountable way and begin with any degree of suddenness, is always to be thought of as a possibility. You must not suppose that the classical signs and symptoms of the disease—diarrhœa, enlarged spleen, spots, etc.—are always present. In children, even more than in adults, they may be entirely absent, as happened in the following case:

A girl, aged eight years, began to be ill with vague pains in the legs two weeks previously, and had had continuous irregular fever since, sometimes going as high as 103° F. She had always been delicate, and there was a strong family history of tuberculosis.

She was a hectic-looking child, poorly nourished, with long eyelashes and a flushed face. The spleen was not enlarged, there were no spots, the urine was normal, and the examination of all the organs negative. Tuberculosis was suspected, but it was thought advisable to do a Widal test, and this was found to be strongly positive.

Another reason why typhoid and paratyphoid fever are apt to be overlooked in childhood is that they often run a very mild course, and may not cause the patient to appear seriously ill. It is important, therefore, to have your mind always open to the possibility of typhoid, and to have the proper bacteriological tests applied as soon as you can. Pending the carrying out of these there are some indications which may strengthen your suspicions of a typhoid infection. Among these are dullness and apathy on the part of the patient, as opposed to the irritability which is usual in tuberculosis. Headache is common if the child is old enough to complain of it, but, on the other hand, it is also common in tuberculous meningitis. The abdomen in typhoid tends to be full and distended, whereas in general tuberculosis it is often retracted. Above all, a pulse-rate which is low in proportion to the temperature is characteristic of typhoid, especially as opposed to tuberculosis.

When all is said and done, however, the final test is the bacteriological one—a positive agglutination reaction and the presence of the organism in the stools. The absence of a leucocytosis may also help in some cases.

TUBERCULOSIS

The possibility of tuberculosis will probably give you more anxiety than anything else when you are in charge of a case of fever of unknown cause. Unfortunately, also, it is a condition

which is extremely difficult to exclude, as we have no absolutely trustworthy test for it. This is the more to be regretted as general tuberculosis is almost the only cause of fever without physical signs in childhood which has a bad prognosis, and one would be glad to allay the anxiety of parents on the subject at the earliest moment. Unfortunately, that is impossible, and you will often be forced to 'hedge' on the tuberculosis question, saying 'the cause of the fever is so and so, or possibly tuberculosis,' and leaving time to settle the matter. This I would say for your comfort, that tuberculosis in these cases of obscure fever is to some extent a bogey. It is far oftener suspected and dreaded than actually present, and though it is often impossible to be sure that it is absent, yet the other causes of obscure fever are so much more numerous than tuberculosis (common though it be) that you may always cherish the hope that the case will end favourably.

I have dealt so fully with the diagnosis of tuberculosis in another lecture that I need say nothing more about it here, except to remind you that the appearance of the child is often suggestive, that irritability and fretfulness are usually pronounced, that rapid loss of weight and a pulse which is quite out of proportion to the temperature are very suspicious, and that the superficial lymph-glands, especially perhaps on the inner wall of the right axilla, may be palpable. The limitations and uses of the tuberculin test I have discussed with you before. The diagnostic problems presented by tuberculosis are illustrated by the following cases:

A girl, aged five years, was seen in March. Since the previous August she had been subject to slight feverish attacks of short duration, but for the last two weeks the temperature had gone up to 102° F. or 100° F. every evening, otherwise she had been fairly well, although rather limp when the temperature was up.

She *looked* rather tuberculous, but was not irritable, and there were no physical signs except that the spleen was just palpable. There was a slight polynuclear leucocytosis; Widal negative. The temperature continued high, and after a time a lump appeared in the right iliac fossa. Operation showed an inflamed appendix and some tuberculous glands. She went to the seaside to convalesce, but tuberculous meningitis supervened, and she died about nine weeks after she was first seen.

A girl, aged three years, had been ill about two weeks, the onset being gradual, with fretfulness and rise of temperature. High intermittent fever since. The child had always been healthy, but rather

'nervous' and inclined to constipation. The family history showed no tuberculous tendency, and the child lived a healthy life in the country.

Physical examination was difficult owing to extreme irritability, but no signs of local disease were detected. A Widal reaction was negative and the urine sterile.

The case was believed to be one of infection from the bowel, and intestinal irrigation was carried out. The temperature then gradually fell, but ten days later rose again, and the child vomited and became drowsy, with twitching of the limbs on the left side. Lumbar puncture was performed, and tubercle bacilli found in the puncture fluid. Death followed in a few days.

BACILLUS COLI INFECTIONS

Infection by the *Bacillus coli* is not uncommon in early childhood, and may easily give rise to fever, the cause of which is not obvious at first sight. The stress of the infection is specially apt to fall upon the genito-urinary system—*B. coli* pyelitis and cystitis—and in little babies this may be the cause of prolonged and high fever, often attended by rigors and without gross physical signs. As I have already described such cases and pointed out to you how they are to be recognized, I shall say nothing more about them now. Apart from infection of the urinary tract, it would appear that sometimes one meets with a general infection by the *B. coli*, comparable to infection with the *B. typhosus*. Cases like the following, for instance, are not, I think, very uncommon:

A girl, aged four years, had been ill for two weeks, with a fairly continuous temperature of 102° F. She was very irritable, and kept the legs drawn up. The tongue was dry and brown in the centre, the spleen just reached the costal margin; she was constipated and the motions offensive, but otherwise there was nothing to make out. The Widal test was negative; the urine contained a few 'coliform' bacilli. Shortly afterwards the temperature fell to normal, and she got quite well.

There were here no signs of inflammation being set up in the urinary tract. The *B. coli* seemed to be simply escaping by the kidneys.

OTHER INFECTIONS

Infection with pyogenic organisms—so-called cryptogenetic septicæmia—is not common in children, but sometimes occurs within the first few days of life. In the form of septic endocarditis it may occur in later childhood, when the heart valves have already

been damaged by a rheumatic attack, or where there is a congenital valve deformity. In any child with a heart lesion who runs an apparently causeless temperature, septic endocarditis is to be thought of. Anything which gives streptococci the chance of entering the circulation may start it. Thus I have known it follow an attack of follicular tonsillitis in children who were already the subject of mitral disease of some years' standing, and more than once have I seen it supervene upon an attack of appendicitis in similar circumstances. The coexistence of an obscure fever with heart murmurs will always arouse your suspicions in such cases, and the diagnosis will be clinched by the cultivation of pyogenic organisms from the blood.

Rheumatic infection is, of course, extremely common in childhood, but it is not a frequent cause of *obscure* temperature, because as a rule arthritis advertises the nature of the trouble. Occasionally, however, the joints may not be affected, or not, at least, until the fever has lasted for some time. In the following case we seemed to be dealing with a mild rheumatic infection, although the signs were never pronounced :

A girl, aged nine years, had a 'rheumatic attack' about a year ago, but the heart was unaffected. Ever since then the temperature had gone up to 99° or 100° F. in the evening. She had led an ordinary life, but had occasionally complained of a little headache or sore throat or pains in the joints, and had often been very irritable and peevish. Tuberculosis had been suspected. She looked a healthy child, and physical examination was entirely negative, except that the tonsils were slightly enlarged. The bowels were natural and regular.

She continued to have the temperature, with occasional joint pains and headaches, for about two years, when she suffered from a slight attack of chorea, with pains in the ankles. Shortly after this the tonsils were removed, and since then she has kept very well and the temperature has been normal.

In such obscure cases of rheumatism I have known the occurrence of typical sour perspiration lead to a correct diagnosis of the cause of the fever.

Closely allied to rheumatism is erythema nodosum, and this also is sometimes a cause of fever which may be difficult to diagnose at first. I recall, for instance, the case of a little boy, the child of a medical man, who had been running a considerable temperature for more than a week, for which there was no obvious cause, I could find nothing on ordinary physical examination, and had

almost concluded that his case was one in which physical signs were absent, when it occurred to me to look at the legs. There on the shins were some typical blotches which revealed the nature of the disease; they had not been present a few hours before.

Last, among general infections one should mention malaria, which should always be thought of as a possible cause of obscure fever in children who are living in, or who have recently come from, a malarious district. The presence of the parasite in the blood and the immediate fall of the temperature after giving quinine are here the diagnostic points.

PNEUMONIA

It might be supposed that pneumonia could hardly be overlooked as a cause of fever, and yet in my experience it frequently is, mainly because the physical signs are often so slight. This may be due either to the patch of consolidation being very small, or to its being situated very deep down in the lung. Apex pneumonias are those which are most apt to deceive. The patch in such a case is often very small, and may be situated just below the clavicle. The secret of its detection is *light* percussion; heavy percussers are prone to pass it by. In addition to this, it is well to remember that the temperature in pneumonia is more often very high (103° F. or more) than not, and the rapid respiration, and the characteristic 'grunt' which often attends it, will help to guide one aright. At all events, pneumonia, and especially an apical pneumonia, should always be thought of in cases of high temperature which have lasted some days without there being any obvious physical signs.

BLOOD DISEASES

Amongst the rarer causes of obscure fever in childhood must be mentioned acute leukæmia. Although physical signs are usually present fairly early in such cases, yet the rise of temperature may be the first thing noticed, and it may be present for some days before anything else is detected. The following are illustrative cases:

A boy, aged eleven years, became ill two days after exposure to a strong sun whilst playing cricket. When I saw him his temperature

had been irregularly high for two weeks, and he felt ill, but there had been no other symptoms except nose-bleeding on two occasions. There was nothing very striking on physical examination, except that the liver was somewhat enlarged and the spleen palpable. The superficial lymph-glands were also slightly increased in size. A Widal test gave a negative result. The white cells numbered 8,000 per c.mm., *but of these* 82 *per cent. were mononuclears.* The subsequent progress of the case showed that it was one of acute myeloid leukæmia.

A boy, aged four and a half years, had looked ' seedy ' for some time, and complained of being tired. A week ago he took to bed, and was found to have a temperature which had gone up to 101° F. every night since. There had been no local symptoms, but he had passed ' jelly ' lately. He was a pale, anæmic child, with a furred tongue, but did not appear seriously ill. The superficial lymph-glands were everywhere palpable, but the spleen was not enlarged, and there was nothing else to be made out anywhere. A blood examination, however, showed 25,000 white cells per c.mm., and 98 per cent. of these were small lymphocytes. He died a few weeks later.

Fortunately, acute leukæmia is a rare disease, and although it is well to bear the possibility of it in mind, you will not often have to diagnose it.

FEVER OF ALIMENTARY ORIGIN

We come now to a group of cases which are comparatively common, and in which the cause of the fever is in the alimentary system, using that term in its widest sense. First amongst these must be placed cases in which simple constipation appears to cause a rise in temperature. In some children this seems to happen rather easily, but the fever in such cases is rarely of long duration, and a dose of calomel soon brings it down. In a few instances, however, the pyrexia is more sustained, and only yields to irrigation of the colon, as happened in the following instance :

A girl, aged eight years, had been feverish for eight weeks, the temperature rising in the evening sometimes to as high as 104° F. Except that she was languid when the temperature was up, she seemed otherwise well. No physical signs were discovered and blood examination was negative, but tuberculosis was suspected. Seen again five months later, temperature had continued as before. Spleen now just palpable ; well-marked splash over the transverse colon ; rather constipated, and motions often pale and offensive. Was now treated by irrigation of the colon, and within three weeks temperature fell to normal, and she has remained well since.

In other cases of this group the fever is *recurrent.* In such children, a rise of temperature occurs every few weeks or months, lasts a week or more, and then subsides. In addition to the fever there is constipation, and the stools are often pale, but the child does not seem otherwise ill as a rule. The nature of this disorder is obscure. It has sometimes been spoken of as 'food fever,' under the belief that it is due to excess in the diet as a whole, or in some ingredient of it, and, as we shall see immediately, the results of treatment lend some support to this view. My own conviction is that these cases of recurrent fever are closely akin to so-called 'bilious attacks,' 'cyclical vomiting,' and 'migraine,' and that in some way or other a derangement of the liver functions is responsible for them. I have found that the best way of preventing the attacks is to cut down the intake of milk, cream, eggs, and chocolates, and to keep the bowels freely open by the prolonged administration of a laxative, of which there is none better than a combination of rhubarb and grey powder. The following is an example of recurrent fever:

A girl, aged three years, had been subject to 'feverish attacks' for several months. These varied in duration from a few days up to a couple of weeks. She had vomited in one attack, but was not specially constipated. Physical examination was entirely negative. The amount of milk she was taking (which had been excessive) was cut down, and she was ordered a regular laxative. She had no more attacks.

Quite different in character from these cases, but also of alimentary origin, are instances of *extremely prolonged fever* sometimes met with in children who are the subject of mucous colitis. Examples of these, amongst several I have seen, are furnished by the following cases:

A boy, aged eleven years, since four years of age had had a bad colour and looked 'yellow' (mother attributed this to bad drains). For four months he had passed mucus in the motions, and had a nearly continuous temperature of 99° F. The skin was rather bronzed, especially on the exposed parts, but the nutrition was good and all the organs seemed healthy. Under treatment by intestinal irrigation and calomel he lost his temperature, and the colour of the skin greatly improved.

A boy, aged four years, had had fever for ten weeks, the temperature going up every night to 102° F. General health good, but was constipated and passed quantities of mucus and shreds. Looked healthy and well nourished; no physical signs of disease detected. Finally recovered under treatment for colitis.

The fever in these cases is usually not high, but often lasts for months or even years, as witness this example :

A boy, aged five years, suddenly became ill with a temperature of 103° F. a month before he was seen. Since that time it had gone up to 100° F. every evening. As a rule he tended to be constipated, and at the outset of his illness there was some mucus in the motions ; otherwise he seemed in fairly good health. He was a well-nourished boy and looked healthy, but the cutaneous tuberculin reaction was positive. There were no physical signs. He continued to run a slight evening temperature for about four years (I was shown sixty temperature charts), but finally got quite well. The mother was convinced that keeping the bowels freely open had done more to control the temperature than anything else.

These are the cases in which tuberculosis is apt to be much feared and the intestinal origin of the fever to be overlooked. They usually yield to care in diet, the use of mild laxatives, intestinal irrigation, and change of air.

Before leaving the subject of fever of alimentary origin, one should mention that the presence of round-worms is sometimes a cause of fever in children, which may even be so pronounced as to simulate typhoid fever. To this the term ' typholumbricosis ' has been applied. I have not met with an instance of it myself.

THROAT INFECTIONS

I believe chronic septic infection from the tonsils to be a very common cause of obscure fever in childhood. Some of the prolonged cases of slight fever are of this nature. Here is an example :

A boy, aged twelve years, was first seen in June, 1910. At Easter he had had a cold with a slight bronchial attack, after which the temperature continued to go up to 100° F. every night for four weeks. He then went away for a change of air, and the temperature disappeared. When seen in June there was nothing to be made out except slight adenoid growths. He remained well until December, when he had another slight bronchial attack, after which the temperature again remained up for a month. His mother thought that excitement caused the temperature. In September, 1911, he had an attack of follicular tonsillitis. After this the tonsils were enucleated, and the temperature has remained normal since.

There need not be much visibly wrong on examination of the throat in these cases, but the tonsils are often rather ragged or

embedded, and the glands at the angles of the jaw palpable. From time to time there is apt to be an exacerbation of the infection, with increased temperature and some visible degree of tonsillitis. Certainly in all cases of prolonged or recurrent fever in childhood the tonsils should be enucleated if there is the least suspicion that they may be causing the pyrexia.

INFECTION OF THE MIDDLE EAR

I have seen several instances of obscure fever which ultimately proved to be due to infection of the middle ear. In such cases there is usually some degree of earache to indicate the seat of the mischief, but not always, and it is quite certain that the ear may be severely involved without any pain. The following case, for example, was very deceptive :

A boy, aged two and half years, had been fretful for about a month, with an evening rise of temperature. He appeared to have some abdominal pain and was wasted. Physical examination revealed nothing abnormal, but he was cutting some teeth. The following day there was some discharge of pus from one ear, after which the temperature fell to normal.

It is always worth while, therefore, to examine the ears carefully in cases of unexplained fever, but even if this is done the condition may be overlooked, as the drum changes are not necessarily pronounced, and in any case a good view of the drums is sometimes difficult to obtain in little children.

DENTITION

It is very difficult to be sure whether teething alone is ever a cause of fever, but in the case of children of unstable nervous system I think it may be. There seems little doubt, for instance, that it was the cause of a prolonged pyrexia in the following instance :

A boy, aged two years, had a slight attack of tonsillitis with a temperature five weeks before he was first seen, and there had been an evening rise to 100° F. ever since. He had become fretful and weak, but showed no other symptoms. He was rather constipated, and required aperients in considerable quantities. Physical examination revealed nothing, and a specialist reported the throat as normal. A bacteriologist also failed to throw any light on the cause of the fever. The temperature continued with variations for several weeks, but suddenly ceased coincidently with the cutting of a back tooth.

Whilst one should not be in haste, therefore, to conclude that the fever is due to the teeth, the possibility of such an origin must always be borne in mind.

INSTABILITY OF THERMOTAXIC MECHANISM

It is well known that the heat-regulating mechanism is more unstable in children than in adults, and therefore more easily upset by slight causes. In some children of nervous tendency this leads to the appearance of fever on small provocation. If in such a child the heat centres are violently disturbed the balance of heat regulation may be deranged for a long time after and the temperature take a long time to settle. It is as if a pendulum had been made to oscillate too violently, so that it only gradually gets back to a state of equilibrium. I have known this happen to a slight degree after many kinds of febrile illness in childhood but the following is a very striking example in which it took place in consequence, apparently, of a disturbance of the heat-regulating centres by an attack of pneumonia.

A boy, aged three years, was seen in April. In the October preceding he had an attack of pneumonia ending with a severe crisis ; ever since the temperature had continued to fluctuate, going up to 102° F. about 6 p.m. and falling to 96° F. about four hours later. Excitement caused the temperature to rise. The general condition had continued good, and he ran about and looked healthy and well nourished. No physical signs of disease could be detected, and blood cultures, the tuberculin test, and the agglutination reaction for *Bacillus coli*, were all negative. Two months later the temperature was reported to be gradually steadying down.

UNEXPLAINED CASES

Finally, you must be prepared to meet with cases of fever in childhood which you are unable to explain in spite of the most thorough investigation. It is possible that some of these are due to infection by organisms not yet identified, or they may indicate invasions by the tubercle bacillus which are overcome by the protecting mechanisms before local signs have had time to develop. I strongly suspected this origin in the case I am about to describe :

A boy, aged fifteen years, was admitted to hospital suffering from cough and weakness of one month's duration. He had always been

a delicate boy, and had been very underfed lately owing to his father being out of work. An uncle had died of phthisis. He was a poorly-nourished boy, but was bright and did not seem very ill. Except for tuberculous-looking scars in the neck and axilla, there were no physical signs of disease to be made out. The temperature for the first four weeks was remittent or intermittent (normal to 101° F.); it then became normal, and remained so till his discharge five weeks later. There was no leucocytosis, and the Widal reaction was persistently negative. He gained greatly in weight during his stay in hospital. The fall in temperature appeared to coincide with the administration of quinine, but he had never been abroad.

At all events, I wish to impress upon you that you will certainly meet with cases which baffle all your endeavours to elucidate them, and that you must not blame yourselves if these have to be relegated to the limbo of 'unexplained' fever. The more thorough your investigation, however, and the more you have recourse to the help of the bacteriologist and clinical pathologist, the fewer will such cases be.

LECTURE XXXVIII

SOME OF THE COMMONER SKIN DISEASES OF INFANCY AND CHILDHOOD

Diseases of the skin are, I am afraid, rather uninteresting, and in childhood, at all events, they are fortunately very rarely in any way dangerous to life or even to health; but as their treatment will undoubtedly cause you a good deal of trouble in practice, I thought it might be well to devote a lecture to them. I shall deal with the subject mainly from the point of view of treatment, for I can profess to none of the specialist's knowledge of the pathology of cutaneous diseases.

It has to be recognized at the outset that in children the skin is peculiarly apt to suffer from disease of one sort or another, and that for several reasons. In the first place, it is of a softer and more delicate texture than it is in the adult, and therefore more prone to suffer from the consequences of mechanical or chemical irritation. In the second place, owing to the relatively larger surface of the child, and the important part which the skin plays in the regulation of temperature, it is subject to great fluctuations in its blood-supply, which must predispose it to suffer from inflammatory processes. Thirdly, the vessels of the skin are apparently under less efficient nervous control in early life than they are in the adult, so that diseases of neuro-vascular origin, such as urticaria, and the erythemata, and reflex congestions (the result of digestive disorders), occur more readily than in later life. Lastly, children are more subject to the attack of parasites and to the consequences of a lack of cleanliness than grown-up people, whilst their intimate contact with each other in school and at their games, renders them specially prone to contagion. If you bear these points in mind it may help you to take measures to prevent the development of cutaneous diseases amongst any children who may be under your care.

When you are confronted with any case of skin eruption in a

child, it is well before proceeding any further to ask yourself three questions: (1) Is it the eruption of one of the specific fevers? (2) Is it the result of a drug? (3) Is it due to an insect or parasite?

SPECIFIC, DRUG, AND PARASITIC RASHES

1. In answering the first of these questions the thermometer should keep you right. Be suspicious of any eruption which is accompanied by fever.

2. To dispose of the second question you must inquire what medicines, if any, the child has been taking. Now, almost any drug may, in a child which happens to have an idiosyncrasy to it, produce some sort of cutaneous eruption, but some are met with more frequently than others. Antipyrin, for instance, produces an erythema fairly often. A copaiba rash may very closely simulate measles or scarlatina, and I have even known it to be accompanied by some pyrexia; but, fortunately, copaiba is not often administered to children. Commonest, perhaps, and certainly the most serious of all, is a bromide rash. In little babies a **bromide rash** presents quite distinctive features. It is not—as it is in the adult—pustular, but consists of curious fleshy papules, sometimes almost cauliflower-like, which are scattered discretely over the body, but with a special tendency to affect the head and face (fig. 100). They are not inclined to suppurate, but pursue an indolent course, and when they disappear leave scars behind. The true cause of such an eruption is apt to escape recognition.

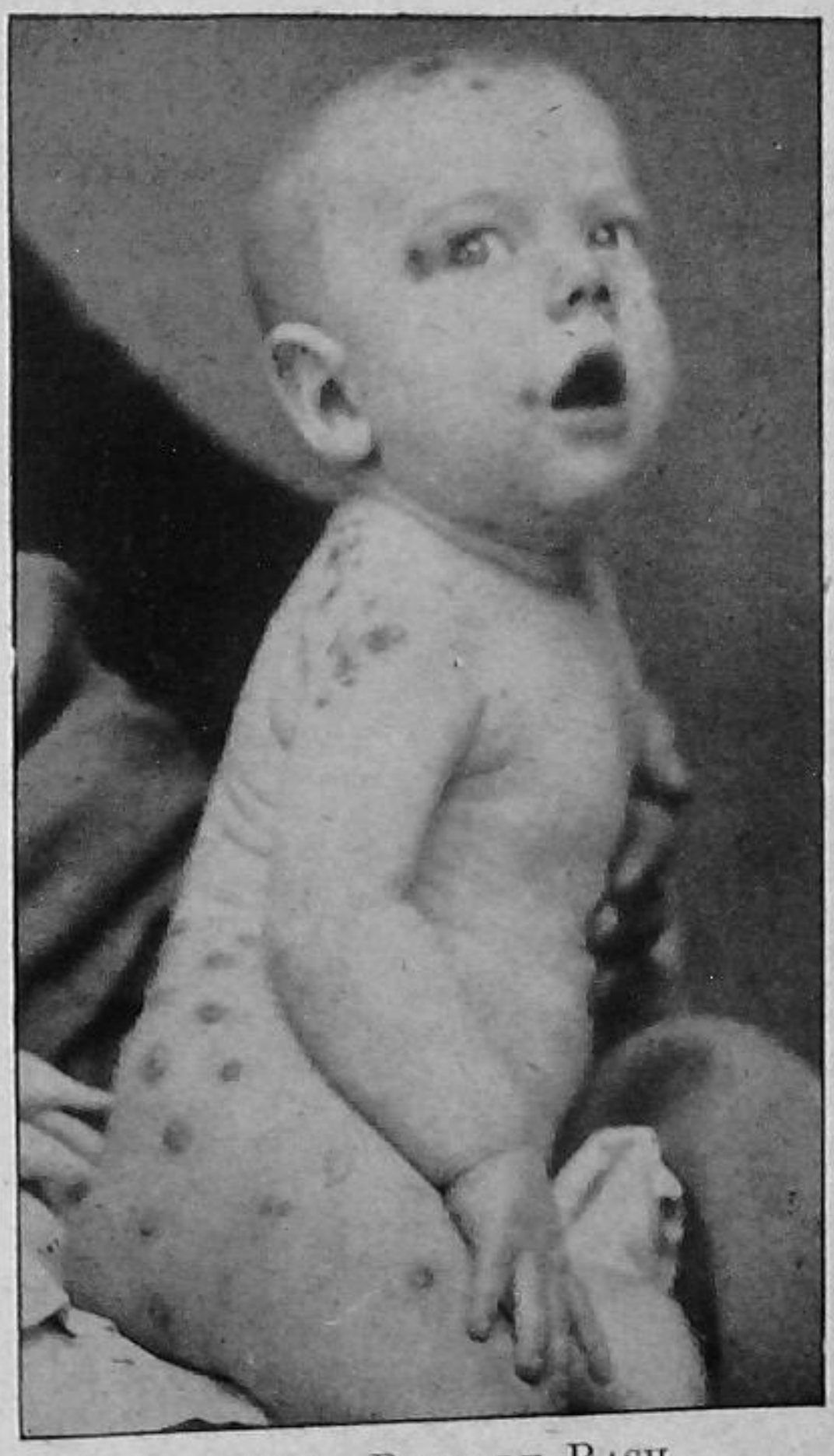

FIG. 100.—BROMIDE RASH.

3. As regards the rashes produced by insects and parasites, I

would remind you not to mistake the results of flea or other **bites** for purpura, as I have known done more than once. The presence of a darker puncture in the centre of each spot in the case of bites should prevent such an error. Mosquito or midge bites may produce rather puzzling appearances, and as I shall have occasion to point out farther on, they may closely simulate an erythema nodosum. Scabies as it affects children I shall refer to later. It is characteristic of all these rashes produced by insects and parasites that they *itch*, and in a doubtful case this may be of diagnostic value.

INTERTRIGO

Perhaps the simplest form of skin disease is that superficial form of dermatitis which we speak of as intertrigo. The appearance of this is too well known to all of you to need any special description, but, as its name implies, it is the result of a fretting or galling of the skin by some superficial irritation. Such irritation may be brought about by the excreta, in which case extensive intertrigo may occur over the parts covered by the napkins, whence it may spread to the backs of the legs and the heels, from their being drawn up towards the buttocks during attacks of colic. It is also met with, usually in fat babies, from irritation by decomposition of the secretions of the skin wherever two cutaneous surfaces are in contact—as, for example, behind the ears, in the transverse folds of the neck, or, more rarely, in the axillæ.

The **diagnosis** of simple intertrigo is not usually a difficult matter, but it is not always easy to tell it when it is confined to the buttocks and perineum from the rash of congenital syphilis. The fact, however, that it is limited to those parts which are covered by, or in contact with, the napkins, that there is no eruption on the palms or soles, that the eruption is bright red instead of being reddish-brown or ‘ coppery,’ along with the absence of other evidences of congenital syphilis should keep you right.

In the **treatment** of intertrigo cleanliness is of the first importance. The mildest soaps, however, should be used, and in some cases it may be advisable to dispense with them altogether, and to use oatmeal or bran instead. An antiseptic dusting powder, of which there are now so many excellent forms on the market, should be applied wherever two skin surfaces touch, or they may be separated by a piece of linen spread with zinc ointment. The inter-

trigo which occurs behind the ear is apt to be very resistant to treatment, and it is often a good plan in such a case to paint over the affected area a solution of nitrate of silver in sweet spirits of nitre (16 grains to the ounce). This produces a little momentary smarting, but its application is soon followed by a drying up of the oozing surface, and the formation of a protecting pellicle, under which healing quickly takes place.

In treating the intertrigo which occurs under the napkins the first step is to deprive the excreta of their irritating properties, for the stools in such cases are frequently acid and the urine ammoniacal. You will often find that the baby is being overfed, and some reduction in the amount or frequency of the feeds is necessary. The use of starchy foods, too, must be stopped, for undigested starch leads to the formation of acid stools, which are very irritating. If there is any looseness of the bowels, a few grains of aromatic chalk should be administered two or three times in the day. If the motions are curdy without being loose, small doses of rhubarb and soda may be given instead. Locally great care should be taken to keep the parts clean. The napkins should be changed as soon as they are soiled, and dusting powder, zinc ointment, or calamine liniment applied to the affected area. Boric acid in powder is especially useful, dusted freely onto the napkins, when a strong ammoniacal odour is present.

ECZEMA

Closely allied to intertrigo, and, indeed, sometimes arising out of it, is eczema. The characteristic form in which this affection manifests itself in childhood is **eczema of the face and head.** Now, I think you will find that eczema specially tends to attack babies of a particular type. There is really quite an extraordinary resemblance between eczematous babies. Briefly I may say that they are nearly always well-nourished infants, with a fine, smooth, white skin, light blue eyes, and fair hair—quite an attractive type of baby, in fact—which makes their being the victims of this disfiguring complaint all the more pathetic. I suppose the meaning of it is that it is the very fineness of their skin which makes it subject to the form of catarrh we call eczema. The disease usually begins over the malar bones and spreads upwards over the scalp, and downwards over the rest of the face and upper part of the trunk. In its full-blown form it manifests itself by redness and oozing, with much local heat, so

that when a moist dressing is removed you will often find it steaming hot. The serous fluid which exudes from the surface speedily dries into a yellowish cake or crust, which may cover the whole scalp and face, and from which the light blue eyes peer out with a clearness and brightness which is sharpened by contrast with the loathsome frame in which they are set.

I have indicated my belief that the predisposing **cause of this form of eczema** is the possession by the child of a type of skin which is peculiarly susceptible to irritation; but if you ask me what the exciting cause of the eczema in these cases is, I must confess my ignorance, although it is probable that in many cases there is an underlying protein hyper-sensitiveness such as is met with in asthma. You will hear it attributed by doctors to some fault in the diet, and by mothers to 'teething.' As regards the former of these opinions, it would appear that some babies are hypersensitive to lactalbumin or to egg and that the eczema is therefore an '**allergic**' phenomenon. As regards the question of teething, I can only say that, whilst I do not believe that by itself teething often causes eczema, yet there can be no doubt that, pending the eruption of a tooth, an already existing eczema is apt to be aggravated, and to take on a more angry and inflammatory character. I have seen this happen too often in eczematous babies who have been under treatment in the wards to have any doubt on the subject. In the same way, any disorder of digestion may intensify an eczema temporarily, and the flushing of the face after meals which is so familiar a phenomenon in dyspepsia gives a key to the understanding of how this may be.

If the views I have expressed are correct, you will not expect much from general or **internal remedies** in the treatment of the eczema of childhood. You should, however, correct any error in feeding which you may detect. The special form of dried milk known as Allergilac from which the lactalbumin has been largely removed is often helpful, and in older children one has to be on the watch for sensitiveness to special foods such as oats. If there is constipation, administer saline aperients, and if the motions are ill-digested and unhealthy, try rhubarb and soda. Such recommendations are obvious. There remain three drugs that are said to be useful. Alkalis, such as citrate or bicarbonate of potash, certainly seem to be beneficial, and in an acute case they should always be pushed until the urine is alkaline. Guaiacum was recom-

mended by Dr. Eustace Smith. It is given in the form of the tincture in doses of 10 minims three times a day. It is said to take the 'fieriness' out of an eczema, but although I have often intended to try it I have never done so yet. Quinine is said to exert a favourable effect in some cases. It is given in doses of $1\frac{1}{2}$ grains for every year of the child's age once a day. Euquinine —which is tasteless—is the best form in which to exhibit it.

When all is said and done, however, it is upon persistent **local treatment** that you must chiefly rely. The first step is to clear away all débris and get a fair field for your remedies. Crusts should be removed by the use of boracic starch poultices [1] or by soaking with oil. When softened by one of these means, they must all be picked off, so that a bare oozing surface is left. So long as the eczema continues to 'weep' there is no use in applying ointments, for they will not stick, and you must depend upon lotions. I use one of three—calamine liniment (B.P.C.), lead lotion, or black wash. By such means the inflammation can be kept at bay, and when it begins to subside, and the eczema assumes a more scaly form, you may start to use ointments, of which the best, I think, is one containing 5 grains each of resorcin and salicylic acid in an ounce of vaseline. If there is much tendency to pus formation, it may be better to use dilute white precipitate ointment first. If stronger remedies cannot be borne, $\frac{1}{2}$ drachm of oleate of zinc in an ounce of cold cream forms a good soothing application, but in the drier and less acute cases an ointment containing $\frac{1}{2}$ drachm of tar solution in the ounce is best. The ointment should be applied on strips of linen or butter-muslin covered with a mask provided with eye-holes, and fitting tightly over the scalp. If it tends to get dry, a little liquid vaseline may be dabbed on to the outer surface several times in the day. Once in the twenty-four hours the application should be changed, and any ointment which adheres to the skin wiped off with wool dipped in liquid vaseline. In order to prevent attempts at scratching, which must be absolutely avoided, it may be necessary at this or any stage of an eczema to apply light paste-board splints along the flexor aspects of the arms, so that the hands cannot reach the head.

[1] Prepared by mixing 1 teaspoonful of boric acid and 1 tablespoonful of cold-water starch to a paste with cold water, then adding 1 pint of boiling water. The paste should be spread on cotton, covered with muslin, and changed often.

Care must be taken to protect the face from cold winds or sun when the child goes out. A red veil is useful for these purposes. The skin should not be washed with soap, but bathed with water containing 1 drachm of borax to the gallon.

No matter how careful and thorough your treatment may be, you must be prepared for **relapses,** and, what is more, you must prepare the mother for them too. I always make it clear when undertaking the treatment of a case of eczema in an infant that one must not look for a rapid cure, but that the condition is likely to persist with ups and downs for a long time, possibly until all the teeth are cut. You may safely promise, however, that it will get well in time, and that it will leave no bad effects or disfigurement behind.

I should add that a few cases of sudden and rather unaccountable death in the course of acute eczema have been reported, and although this is a very rare event, it may be well to bear it in mind, and not to make too light of the complaint.

SEBORRHŒIC ECZEMA

The term ' seborrhœa ' is a bad one—one of the worst in dermatology, in fact, which is saying a good deal—for it naturally suggests an increased production of sebum, which does not really take place. It is convenient, however, to retain the term ' seborrhœic ' for that form of eczema met with on the scalp and forehead and behind the ears, and occasionally also on the upper part of the trunk, which is characterized, not by oozing, but by the production of greasy, yellowish flakes or scales, which may adhere into a brittle crust. If one removes the scales, the underlying skin is found not to be so congested as in ordinary eczema, but rather of a pinkish tint. Seborrhœic eczema is met with both in babies and in older children and in the former it often forms a greasy, yellow layer on the top of the head, which the mother is afraid to remove for fear of injuring the fontanelle. Here, again, the first step in **treatment** is to remove the scales or crust. When this has been done (in the way already described for ordinary eczema) an ointment consisting of 10 grains each of salicylic acid and sulphur, with 1 drachm of oxide of zinc, in an ounce of vaseline, should be well rubbed in. You will find that it is more amenable to treatment than ordinary eczema.

IMPETIGO

Impetigo contagiosa is a frequent affection of the skin in childhood. It is commonest in children of school age, and, as the name implies, is conveyed from one to the other by direct contact, being due, as you know, to local infection with pyogenic organisms. It most often affects the face, especially in the neighbourhood of the mouth, and you may easily recognize it by the yellow crusts, which have a curious look of having been stuck on artificially. Its **treatment** is usually as simple as it is satisfactory. You have merely to remove all the crusts in the same way as you do in eczema, and then to rub into the raw surface which is left a little weak antiseptic ointment. There is nothing better than white precipitate—5 grains to the ounce—of which a little should be well rubbed in *frequently* throughout the day. Under this plan the disease quickly disappears in most instances. However, there seems to have appeared in recent years a more resistant type of the disease. If there are only a few spots it is a good plan to cover them completely by a piece of adhesive plaster. Gentian violet solution (1 per cent.) may be dabbed on and sulphonamide ointment is said to be of value.

MULTIPLE CUTANEOUS ABSCESSES

Occasionally in infants, especially ill-nourished infants, you will meet with multiple abscesses in the skin which are apt to be rather troublesome. The abscesses vary in size from a pea to a hazel-nut or larger, and are often very numerous. They start insidiously, come out in crops, and run a slow course. They are usually unattended by any fever. Cultures from the contents of the abscesses show the staphylococcus aureus, but whether it gets to the skin from the blood-stream or as a result of a direct infection from the surface is not definitely determined.

The treatment of these cases is often difficult. One should do all one can to improve the general health of the infant by suitable feeding, fish-oil, change of air, and so on. Artificial sunlight is often of value for the same purpose. Local treatment consists in opening the abscesses and dressing them antiseptically, whilst the risk of a spread of infection to other parts of the skin may be lessened by disinfectant baths, e.g. pot. permang. 1 in 5,000.

I have an impression that small doses of quinine are helpful in these infections (euquinine gr. i t.d.s. for an infant of a year),

but I am uncertain as to the value of autogenous vaccines, although they are worth trying in small doses.

URTICARIA

Nettle-rash, in one form or another, is one of the commonest of the minor ailments met with in the nursery, especially in the warmer months, and everyone who has ever been stung by a nettle is familiar with the appearance which it presents in its simplest and uncomplicated form. Often, however, one finds that upon the top of the wheal there arises a papule which persists after the subsidence of the original eruption, and to this form the term **lichen urticatus** or **papular urticaria** is applied. The term is justified, for the papules so formed have the typical flat, polished-looking apex characteristic of the papules of lichen. Sometimes little vesicles form on them, and these may even go on to pustules. Add to these some scattered wheals and the secondary lesions and infections which result from scratching, and you will readily understand that in its fully-developed form lichen urticatus may present rather a complicated picture.

Now, there is another disease which may present an exactly similar picture, and that is **scabies.** For my own part, I confess my inability—in many cases, at least—to diagnose lichen urticatus from scabies merely by looking at it. For you must remember that scabies as it occurs in children has by no means the characteristic distribution which it manifests in the adult; on the contrary, it is often quite a generalized eruption. In addition to this, both lichen urticatus and scabies cause great itching, especially at night, so that the mimicry of the one by the other is almost complete. How, then, are you to distinguish between them? By attention to two points. Lichen urticatus is a disease of much longer duration than scabies: the life-history of the former covers months or years, that of the latter days or weeks. Secondly, scabies rarely affects but one child in a family, for before long another is almost sure to become infected. The burrows and acarus of scabies should also be looked for. Should you still be in doubt, the therapeutic test will settle the point. Under a few sulphur baths (1 ounce of potassa sulphurata to 7 gallons of warm water), or the inunction of weak sulphur ointment (equal parts of sulphur and zinc ointments), or the application of equal parts of balsam of Peru and glycerin, scabies will quickly disappear; lichen urticatus will not.

This leads me to speak of the **treatment of nettle-rash** in general, and at the outset I would remark that this is by no means an easy matter. Only too often you will find that the disease persists, with longer or shorter intermissions, for a long time, although, like eczema, it tends to disappear spontaneously about the end of the third year of life. I would advise you not to listen too readily to those who say that nettle-rash is always due to some disorder of digestion. Just as in the case of eczema, so here I have often found that there is really nothing to criticize in the food that is given or in the way in which it is digested, although I admit that in a few cases improvement will result from the use of the same general measures and internal remedies as were recommended in eczema. An attempt should be made to discover whether the patient is 'sensitive' to any particular article of food; oatmeal, raw fruits, eggs, excess of sugar, fish, sweetmeats, and cheese, are the most likely. Dr. G. W. Bray, however, is of opinion that it is the 'pig-products' (bacon, lard, etc.) which are specially harmful and that these along with cod-liver oil, should be banished from the diet. Aperients, rhubarb and soda, dilute hydrochloric acid and ichthyol, all have their place in treatment, but, on the whole, you have to depend chiefly on local measures. Nor are these at all sure to be successful; often all that you can do is to relieve itching, and it is not always possible to effect even that. Avoidance of things which irritate the skin is of the first importance. Warm baths, for instance, may have to be interdicted, and in these cases at least the generally salutary rule of 'flannel next the skin' must be broken. Silk is the best material for the under-garments and nightdresses, or, if silk is too expensive, linen. By attention to this point I have sometimes relieved cases in which all the ordinary remedies have been tried in vain. For the relief of itching many applications have been recommended. The following ointment is efficacious:

β-naphthol	gr. xvi.
Zinci oxidi	℥i.
Ung. simplicis	℥i.

This must be rubbed in all over the body at night. On the whole, however, there is nothing better than tar (equal parts of tar ointment and diachylon ointment), the only drawback to which is that it is dirty. A tar lotion (liq. picis carbonis, 1 drachm to a pint of water) is cleaner, but not, I think, quite so effective. A weak

carbolic lotion (1 in 40) sometimes answers, but I am always rather chary of applying carbolic acid over large areas in young children. It may also form an ingredient of other lotions, as, for example:

Acidi carbol.	℥ss.
Calamin. prep.	℥iii.
Cret. prep.	℥i.ss.
Glycerini	℥i.ss.
Aquam	ad ℥vi.

To be painted on at night after washing with a menthol soap.

By the use of such means as these you may at least hope to alleviate the worst symptoms of the disease, but more than this it is often not in one's power to do. When all else has failed, however, never omit to try the effect of a change of air, which will often succeed—temporarily, at least—when local applications prove of no avail.

SWEAT RASHES

Rashes from excessive sweating are met with in babies who are rather overclad, and especially, of course, in hot weather. I think their commonest distribution is over the base of the neck and upper part of the front of the chest. They consist of tiny vesicles (sudamina), often upon a basis of intertrigo. Sometimes they go on to form minute pustules, and when inflamed the eruption is spoken of by some writers as 'strophulus'; but as this term is also used by some to mean lichen urticatus, it is ambiguous, and is best avoided. The **treatment** of a sweat rash is the same as the local treatment of intertrigo.

Of psoriasis, which is certainly a fairly common skin disease in children, I do not propose to speak, for its management differs in no respect from that of the same disease as met with in adults. Nor shall I say anything of ringworm or alopecia, for I have nothing to add to what you will already find on these subjects in your text-books.

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LECTURE XXXIX

DISORDERS OF GROWTH AND DEVELOPMENT

From time to time your advice will be sought about a child simply because it does not grow properly, and you will be asked what is the cause and what are the prospects. It is not likely that the patient will be brought to you until the defect of growth is pronounced, but it is well to know the average height of normal children

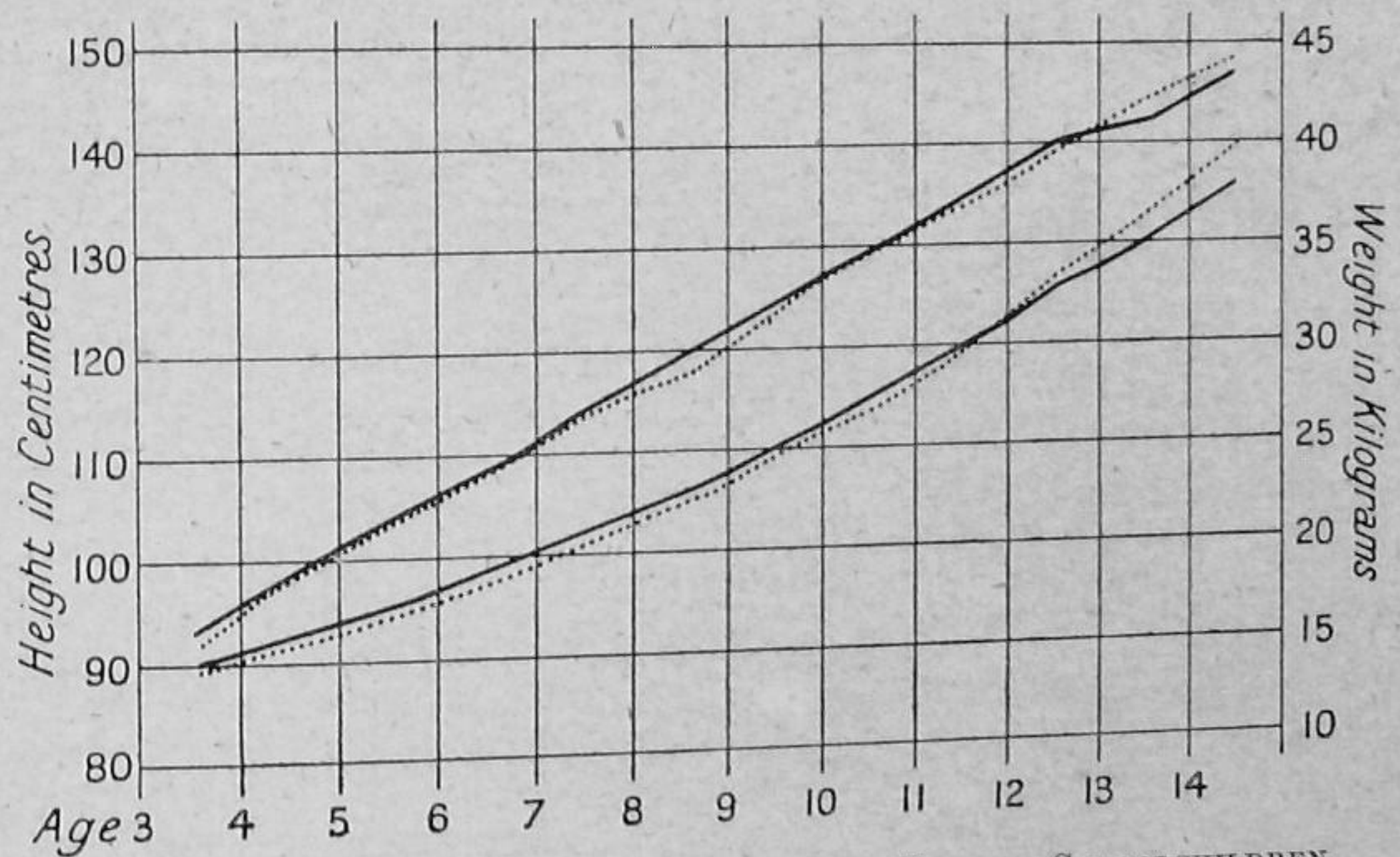

FIG. 101.—AVERAGE HEIGHT AND WEIGHT OF ENGLISH SCHOOLCHILDREN.

(After Tuxford and Glegg.)

The two upper lines represent heights, the two lower lines weights. Boys, _____; girls, ... 1 centimetre = 0·39 inch; 1 kilo = 2 pounds 3 ounces. (By permission of the *British Medical Journal*.)

at different ages for purposes of comparison. This, with the corresponding weight, is set out in the accompanying chart (fig. 101).

It must be remembered, of course, that considerable variations from the standard are quite compatible with normality, and it is also well to know that although the rate of growth is not uniform, yet between the ages of four and twelve it should average at least

2 to 3 inches yearly. In boys growth is slower between nine and fourteen (i.e. the preparatory school age) than it is before and after that period, whilst in girls the slow time is earlier and shorter —i.e. between eight and eleven. Nor is the rate of growth steady throughout the year. It tends to be greater in the spring and summer than during the colder months, and one often finds leaps forward or, on the other hand, temporary halts without obvious reason.

After these preliminaries we may pass on to consider the cases of pathological arrest or retardation of growth. We have to recognize at the outset two distinct conditions: (1) dwarfism, (2) infantilism. In dwarfism growth alone is affected, whereas infantilism consists in an arrest or retardation not of growth only, but of the whole development, including the secondary sex characters.

DWARFISM

There are two varieties of dwarfism—secondary and primary. In the former the stunting of height is due to gross changes in the skeleton, such as are met with in spinal curvature, achondroplasia, or severe rickets. In primary dwarfism, on the other hand, the child is otherwise normal, growth alone being affected, and the union of the epiphyses and the changes of puberty take place at the usual time. The patient is, in fact, a normal human being, but in miniature—a ' pocket edition ' of humanity. This form of dwarfism is rare, and nothing can be done for it.

INFANTILISM

Infantilism is much commoner than true dwarfism. Here, again, one has to recognize a secondary and a primary form.

In secondary, or symptomatic infantilism, development is checked by some general disease or disorder, and according to the nature of the cause several varieties are described.

(*a*) Any **severe constitutional disease,** if chronic and long-continued, tends to retard growth and development. Amongst such diseases are congenital syphilis, tuberculosis, cirrhosis of the liver, and heart disease, whether congenital or acquired. The degree of infantilism in this group is usually but slight, and the

clinical picture is dominated by the primary disease, on which also the prognosis depends.

(*b*) **Renal infantilism.**—This is an interesting group—rare, but probably not so rare as is supposed. It owes its name to the fact that the arrest of development is associated with cirrhosis of the kidneys. The latter condition shows itself by polyuria with great thirst, the urine being of low gravity and containing a trace of

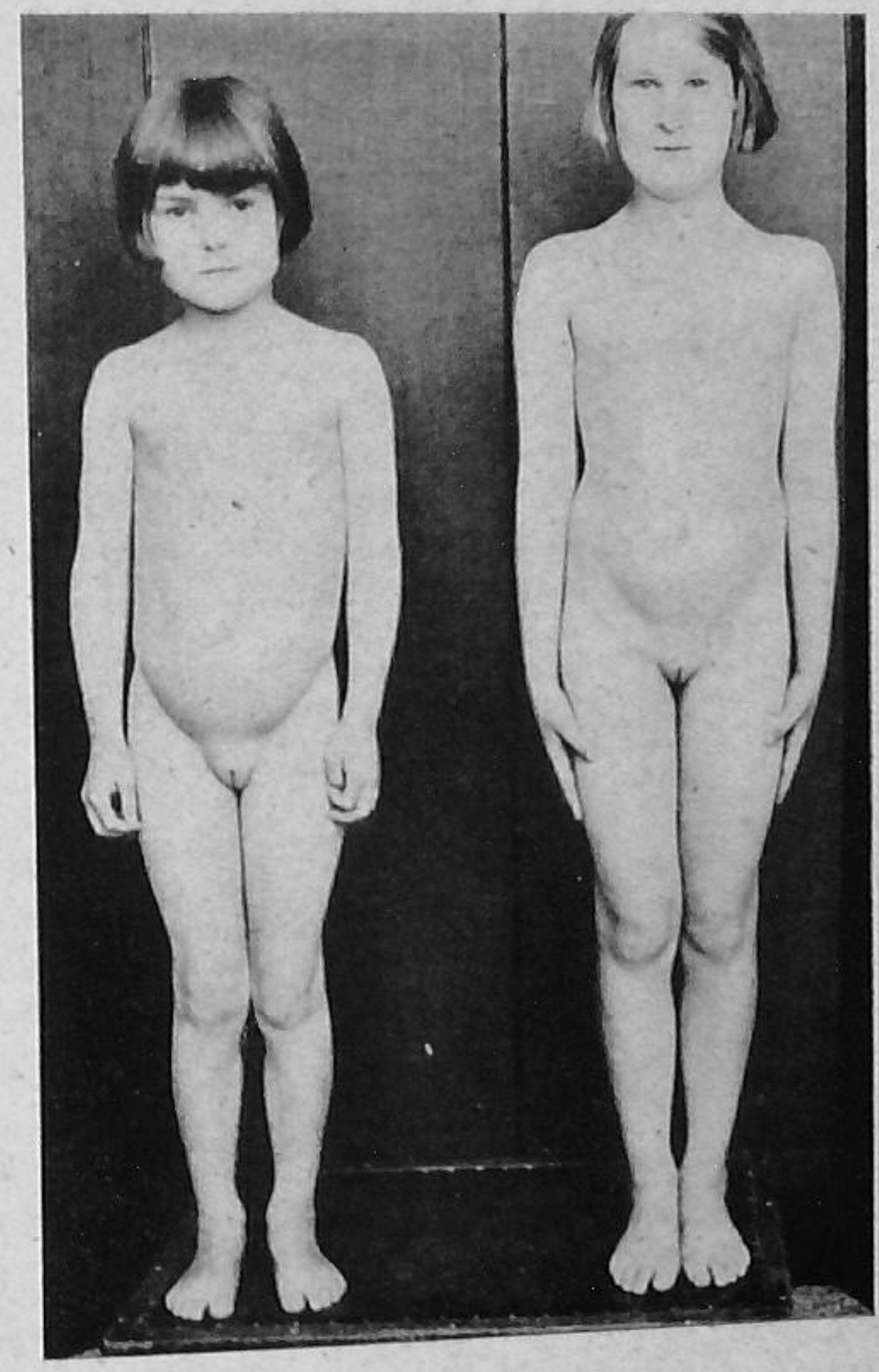

FIG. 102.—RENAL DWARFISM.
Child aged 10½ years with normal child of same age.

albumin. The degree of infantilism depends upon the age at which the renal change has supervened. It appears sometimes to be congenital, and in that event dwarfism is pronounced and there is great deformity of the bones, which resemble superficially those of rickets. In other cases the disease appears later following upon an acute glomerular nephritis which does not clear up the infantilism then being proportionately less, and the bony deformities resembling

those of so-called 'late' rickets, genu valgum being a common feature. The cause of the renal cirrhosis in renal infantilism is unknown, but in som cases it is associated with obstruction in the urethra and dilatation of the ureters; the patient usually dies of uræmia long before adult life, no treatment being of any avail.

In cases of so-called *diabetes insipidus* occurring in childhood some degree of infantilism is also met with, and as such patients also exhibit great thirst and a high grade of polyuria, they may present a superficial resemblance to the renal cases. The bony changes, however, are not characteristic, nor is the arrest of development at all conspicuous, and the cases should rather be placed in group (*a*) described above than in that of renal infantilism properly so called.

(*c*) **Intestinal infantilism.**—In this group the arrested development is secondary to that peculiar chronic intestinal disorder described as 'cœliac disease.' As cœliac disease usually begins in the second year of life, the infantilism in a protracted case may be extreme. If and when the intestinal disorder passes off growth and development are resumed, but some permanent stunting usually results. I have, however, known cases in which a normal standard of development was ultimately attained. The interference with growth is probably due to chronic starvation from imperfect assimilation of fats. It is possible, too, that a defective supply of the 'growth vitamin' which may be consequent on this also plays a part.

(*d*) **Pancreatic infantilism.**—Cases which fall within this group are much rarer than those of intestinal infantilism. They were first described by Sir Byron Bramwell, and are characterized by the passage of loose stools containing unsplit fat in large quantity. The delay in development in such cases may reach a high degree, the first of Bramwell's cases looking at the age of eighteen like a boy of eleven, with corresponding sexual development. His intelligence, however, was good. The cause of the symptoms is arrest of pancreatic secretion, and they lend themselves very well to treatment by pancreatic extract, under the administration of which the stools become normal, whilst growth and development restart and may ultimately reach normal.

(*e*) **Pituitary infantilism** (also known as Fröhlich's syndrome, dystrophia adiposo-genitalis, or dyspituitarism).—This form is distinguished from all others by the high degree of obesity (fig. 103).

Although the patients are fat all over there is a specially thick layer on the trunk, and the breasts often appear well developed, even in boys. Along with this the genital organs are infantile in development, and the patient, though intelligent enough, is usually rather childish for his age. On the whole they tend to approximate to a 'neuter' type in configuration and character. Metabolic tests show an increased tolerance for sugar.

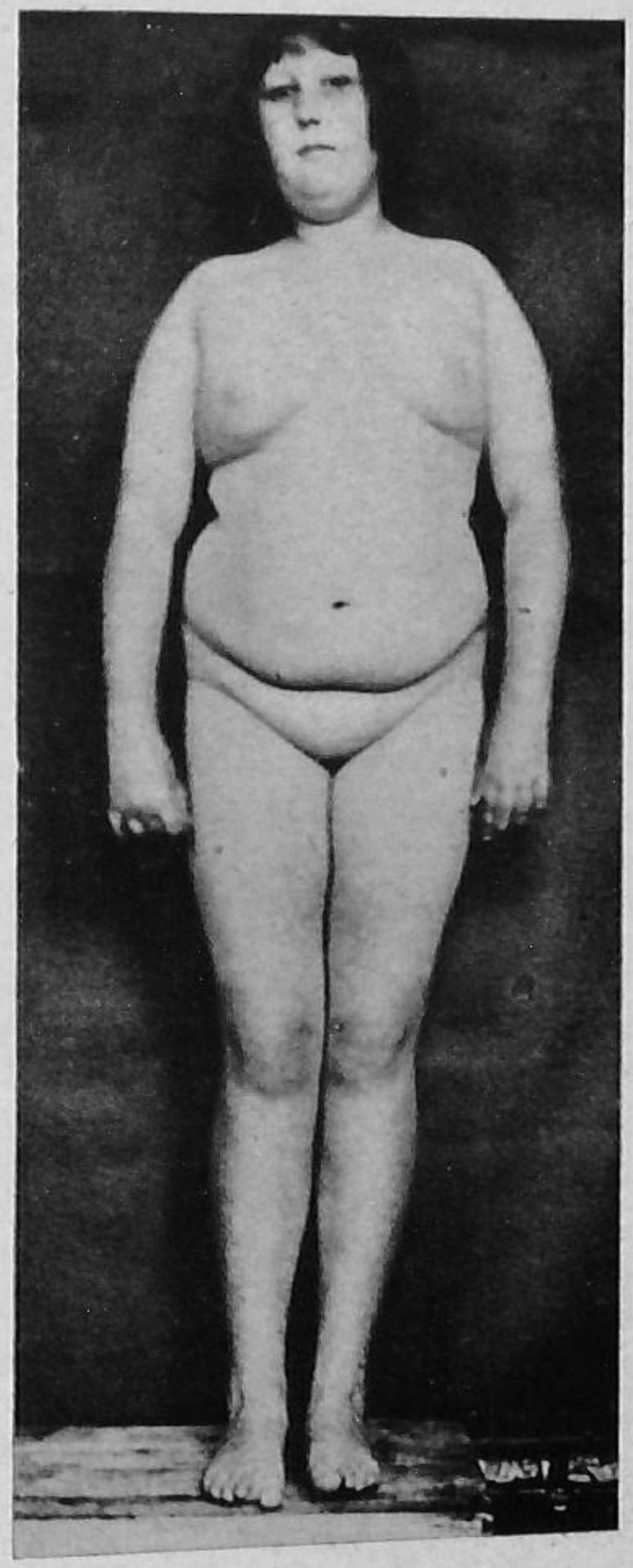

Fig. 103.—Dyspituitarism.

It is believed that this variety of infantilism is due to an underactivity of the anterior, and possibly also of the posterior lobe of the pituitary, which is often the result of its partial conversion into a cyst. It is, indeed, never wise to diagnose pituitary infantilism unless signs of a pituitary tumour are present (headache, bitemporal hemianopia, enlarged sella turcica, etc.), for it is certain that the majority of the children brought to one for obesity are *not* pituitary cases. Note especially that a true case of Fröhlich's syndrome is under the average height for the age: most cases of simple obesity are over-height as well as overweight. The prognosis as regards life is not good, and pituitary treatment, by the mouth at all events, does not help. Injections of anterior pituitary growth hormone are recommended, but seldom appear to do good. Whether in the future anything may be done by operation remains to be seen.

(*f*) **Thyroid infantilism** (infantile or adolescent myxœdema).—In this group the arrest of development is due to deficient action of the thyroid. The cases differ from those of cretinism in that

the thyroid deficiency sets in some time after birth, or, if it is congenital, is only partial. The following examples will serve to illustrate the condition :

Case 1.—*Robert W——*, aged seven and a half years, was brought to hospital because he was ' not growing.' He is the fifth child of the family, three having died in infancy, and one (a younger sister) suffers from fits and is mentally defective. The mother is a healthy woman who present no signs of myxœdema, but when pregnant with this child her health was indifferent and she was much worried. He was born at term and was bottle-fed. When about six months old he attended the hospital for some time for ' wasting,' after which he did not get on fast, and did not walk, talk, or cut his teeth until nearly two years old. He had no other illness.

His condition when first seen is shown in the photograph (fig. 104). He was very short for his age (height, 33 inches), and showed some evidence of previous rickets in the shape of his head and curvature of the legs. His facies was somewhat myxœdematous, the complexion being yellowish, the skin rather dry, the hair harsh, and the lips waxy. The milk teeth were still present and were mostly carious. The hands were ' podgy,' the thyroid could not be felt, but the viscera were healthy. His mental condition was rather quiet and dull, and his memory defective ; his speech slow and to a large extent unintelligible ; his temper good.

Under thyroid treatment he rapidly improved, and after a month he had grown 2 inches. Five months after treatment was begun he had grown 4 inches, but his weight was unaltered. After two years of treatment his height was 40 inches, he was well and bright, and, as his mother said, was now ' really like a boy ' ; he could also read and count a little.

When last seen he was fourteen years of age and measured 47½ inches. He had been taking thyroid fairly continuously, and except that he was under-sized looked quite normal. Mentally he was still decidedly backward, and had to attend a ' special ' school.

Case 2.—*Ursula P——*, aged six years. She was brought for advice ' because she did not grow.' She was the first child of a family of three, the two younger being healthy. She was born at term, the mother's health during the pregnancy being good, and she was bottle-fed. From the age of five months she had been backward. She held up her head at three months, but did not talk until she was two, or walk until she was two and a half years. Except for constipation she had always been healthy.

Her appearance when she first came under observation is shown in fig. 105. She was a small, chubby child, with fat red cheeks, thick (not coarse) hair, and a smooth skin. The epicanthic folds were rather marked and the lobes of the ears adherent. Her height was 34 inches. There was no evidence of rickets and the organs were all healthy.

The trachea could easily be felt. The hands were somewhat spade-like. Her mother did not consider her mentally backward. She was alleged to have a good memory and to know her alphabet, and to 'sit and think a lot.' She was not at school.

Under thyroid treatment ($\frac{3}{4}$ grain night and morning) she improved rapidly, and after three months had grown 2 inches and looked more normal, being brighter and less puffy-looking. Her condition one year after treatment was begun is shown in fig. 106. At ten years of age she was quite a healthy-looking girl, 4 feet $1\frac{1}{2}$ inches in height, and without any signs of myxœdema. She was, however, somewhat backward at school. She had been taking thyroid powder regularly.

FIG. 104.—THYROID INFANTILISM.

CASE 1 (ON THE LEFT) ALONGSIDE A NORMAL BOY OF THE SAME AGE.

In neither of these cases could the child be fairly described as a full-blown cretin, but they clearly belong to that group of cases of

infantilism in which the arrest of development is due to defect of an internal secretion—in this instance the thyroid. Such cases are sometimes spoken of as 'myxœdematous infantilism,' or 'benign chronic hypothyria,' and it is permissible to assume that in them the thyroid, although not completely absent, is comparatively ill-developed or inactive. The myxœdematous symptoms may appar-

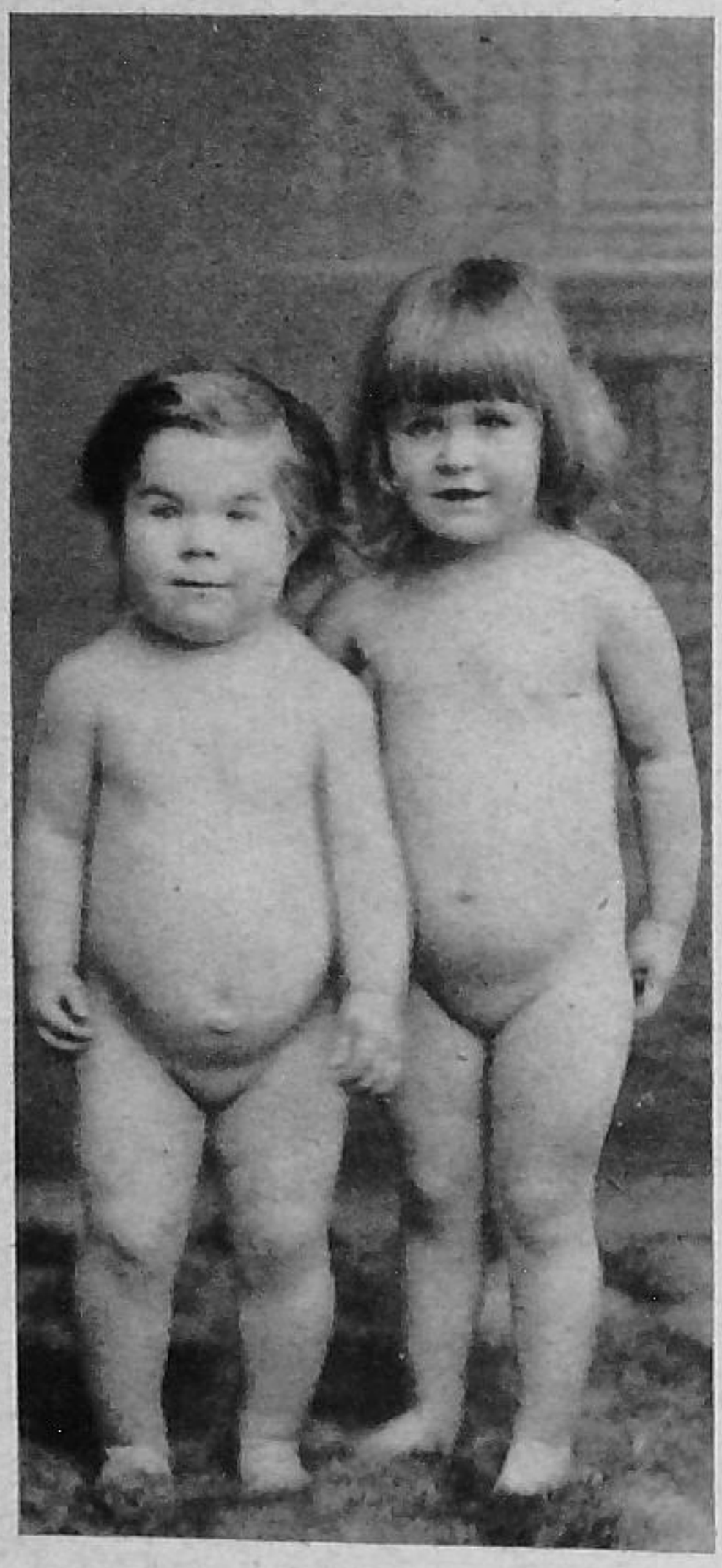

FIG. 105.—CASE 2 (ON THE LEFT) ALONGSIDE HER NORMAL SISTER, WHO IS THREE YEARS YOUNGER.

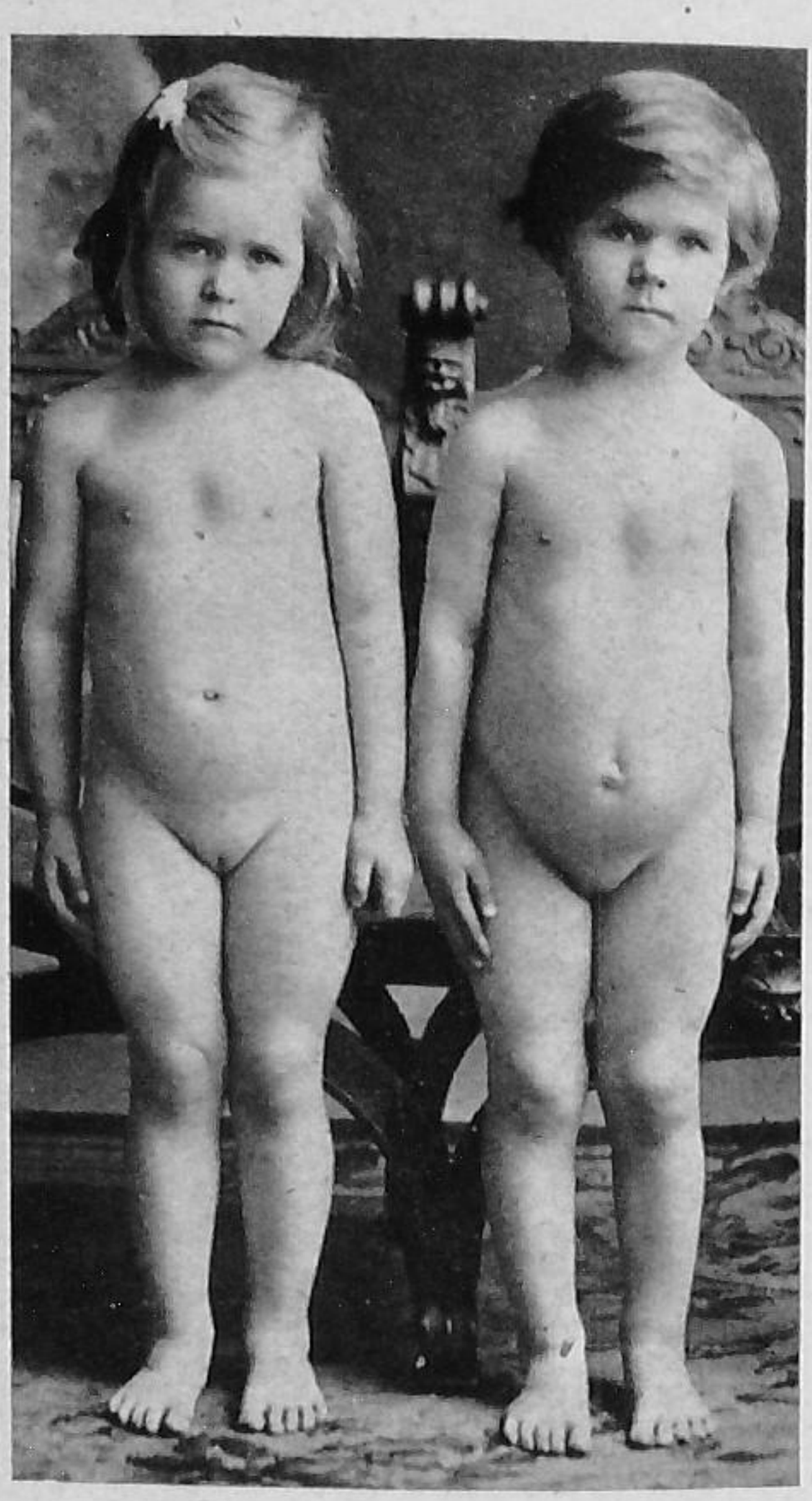

FIG. 106.—CASE 2 (ON THE RIGHT) ALONGSIDE HER SISTER AFTER ONE YEAR'S TREATMENT.

ently arise at any period of childhood, and although in the cases I have described they set in early, they sometimes do not appear until about the period of puberty (juvenile or adolescent myxœdema). In figs. 107 and 108 a case of this sort is shown in a girl, aged fifteen years, before and after treatment, but unfortunately the details of her history have been lost.

Some authors consider that many cases of so-called 'idiopathic' infantilism (Lorain type) are really instances of masked myxœdema in which the thyroid defect is very slight. The following, of which

Fig. 107.—Case of Juvenile Myxœdema.

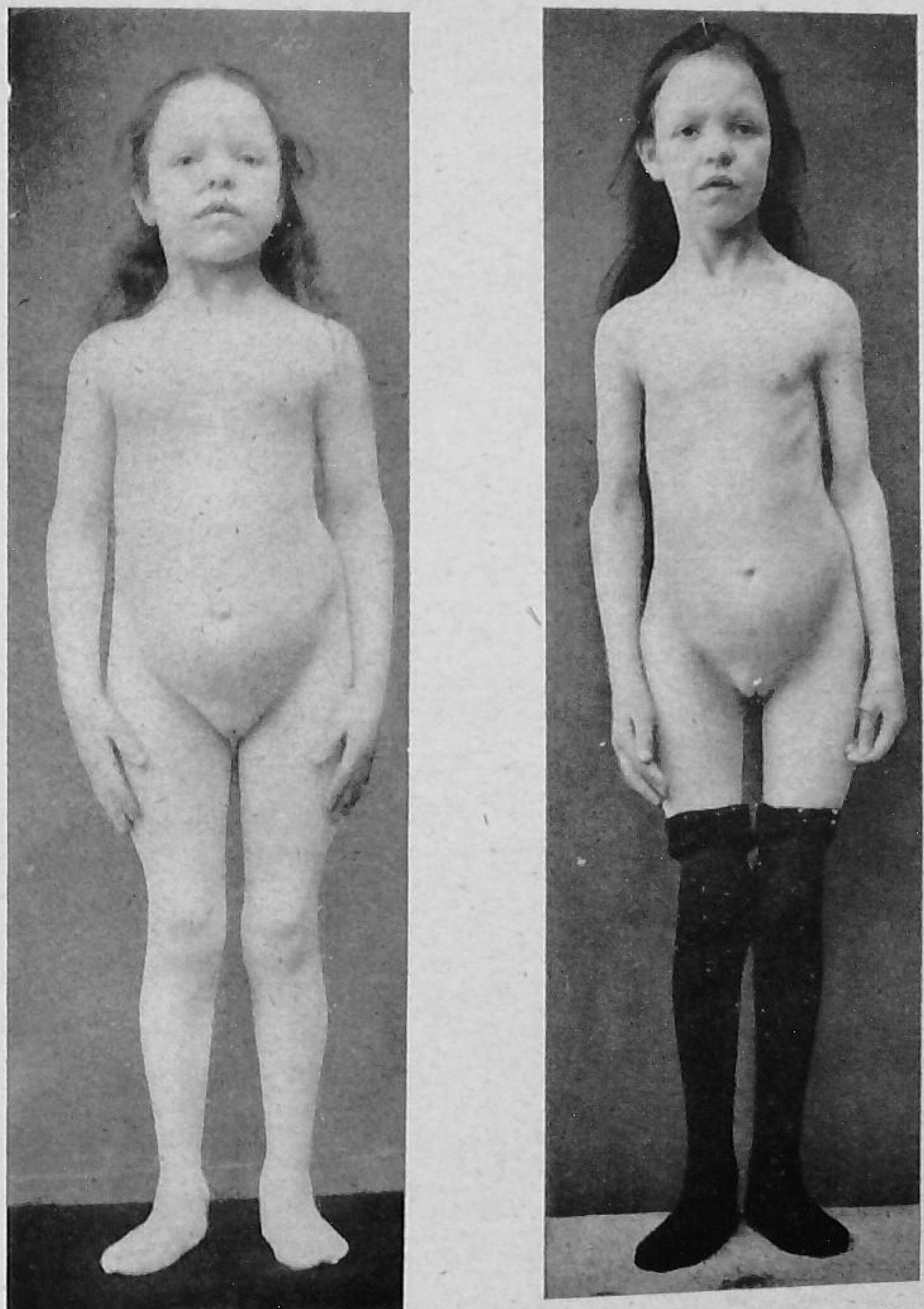

Fig. 108.—The Same after Treatment.

I regret there is no photograph, is perhaps an example of this 'masked myxœdema':

Case 3.—*Doris T*——, aged nine and a half years, was brought for advice because she did not grow. She was an only child born at term. The mother's appetite during pregnancy was very poor, but she was

quite a healthy woman. The patient looked like a fairly normal child of five. She had no appearance of myxœdema except that the hair was somewhat coarse and dry, but the skin was fine and smooth. The features were well cut and the hands normal. The voice, however, was curiously deep-toned. The milk teeth were still all present. Her height was only $3\frac{1}{2}$ feet, her weight 2 stone $10\frac{1}{2}$ pounds. Mentally she seemed bright and alert, and, her mother said, painted cleverly.

Under thyroid treatment the patient grew rapidly, quickly lost her milk teeth, and acquired a new crop of finer hair. One year after treatment was begun her height was 3 feet $9\frac{1}{2}$ inches, and her weight 2 stone $13\frac{1}{2}$ pounds. When last seen she was still taking thyroid. Her age was fourteen and a half years, her height 4 feet 10 inches, and her weight $5\frac{1}{2}$ stone. She looked quite normal, but still rather under-sized, and her voice had remained very deep. Mentally she seemed somewhat backward for her age, but she was said to be 'good at some things'—e.g. languages and music—although 'very bad at arithmetic.'

The prompt response to thyroid treatment in this case suggests defective activity of the gland, but in appearance the child was much more like a case of idiopathic infantilism than one of myxœdema.

Treatment in the cases of this group is very satisfactory, the response to thyroid medication being immediate, as the above examples show, and ultimately a normal standard of development may be reached. I believe, indeed, that this is the only form of delayed development in which thyroid treatment is of any use, and that if a case of infantilism responds immediately to thyroid it may be taken as a sign that the patient really belongs to this group. Nor is it necessary that the treatment should be continued throughout life. The patient in case 2 is now a full-grown woman, and able to do without thyroid. It would seem, indeed, as if some of these patients had enough thyroid secretion of their own to serve the purposes of adult life, though not enough for the period of active growth.

We now come to the second variety of infantilism, that which is **primary**, essential or idiopathic. It is also known as the 'Lorain' type, from the name of the French author who first drew attention to it, whilst the chief English writer on disorders of development (Hastings Gilford) has suggested for it the term 'ateleiosis' (meaning 'not arriving at perfection'). Now it may be taken as true throughout medical literature that designating a disease as 'essen-

tial ' or ' idiopathic ' simply means that we know nothing as to its causation, and that is certainly true in this instance. In primary or idiopathic infantilism the patient is organically sound. He simply does not grow and develop at the normal rate. The delay or arrest may set in at any period of childhood. Sometimes it dates from a severe illness. In one of my cases it seemed to follow upon the production of spinal anæsthesia for an operation, but often enough no cause for the cessation can be assigned. Anyhow, the physical and often also the mental characteristics of the period at which growth ceases tend to become stereotyped. The patient looks a number of years younger than his true age. He retains a child-like, rather cherubic facies, and the bodily configuration of a child, the limbs being relatively short, the head large, and the mid-point of his height being at the level of the umbilicus instead of at the pubic symphysis. The teeth tend to be backward, irregular, and crowded, and the voice high-pitched and childish. The secondary sex characters are absent, the genital organs are infantile, and cryptorchism not infrequent. The mental development is usually retarded in proportion to the physical, but not invariably. Some of the patients are fully up to the normal intellectual standard of their years. In my experience this form of infantilism is commoner in boys than in girls, and the usual degree of it is for the patient to be about two years behind the normal standard both in mind and body

An attempt has been made to subdivide cases of primary infantilism into a ' sexual ' and an ' asexual ' variety according as secondary sex characters are present or not. Clinically, however, such a subdivision is useless, for one never can tell when an ' asexual ' case may become a ' sexual ' one by the appearance of a belated puberty. This *may* not be reached until the age of thirty or more.

When you have diagnosed primary infantilism or ateleiosis the parents will have some questions to put to you :

1. *Will he live?* The answer is in the affirmative. There is nothing in the condition which tends to premature death, and in fact some of these patients have attained an advanced age.

2. *Will he grow?* Certainly ; but no one can prophesy how much. He may go on growing up to the age of thirty or more, and so long as there are ununited epiphyses there is hope. Growth

may be gradual or at any moment a leap forward may be made. The chance of growth is greater so long as the case is of the 'asexual' form, for soon after puberty sets in the epiphyses appear to unite and further growth is impossible.

3. *How about his mental development?* Here, again, you may be reasonably hopeful. Although many of these patients are backward for their age—but by no means all—and although they may be a little 'odd' or childish in some things, they are very far from being mentally deficient, and some that I have followed have done quite well in life. Thus one, after a creditable career as a student, qualified as a doctor; another became a missionary; another is doing well in business; and yet another distinguished himself in the war of 1914–18. Whether the patient will be fit for ordinary school life, however, is another question. Many of them are very sensitive about their condition and are afraid of being chaffed. Others develop a pronounced 'inferiority complex,' which makes them either morbidly shy or else—I suppose by a 'defence reaction'—rather 'cocky.' In either case the patient may be so unhappy at school that, as happened in two of my cases, he has to be removed from it and educated privately. On the other hand, some of them do not seem to mind their peculiarity in the least, and pass through the ordinary rough and tumble of school life with both pleasure and credit. One never can tell till one tries, but if possible the more such patients are treated like ordinary boys and girls the better.

4. *Is the condition transmissible?* Such a question arises only if and when the sexual stage is reached. In these circumstances it is safe to reply that, so far as we know, it is not transmissible, and that, as a fact, some extreme examples of the condition have produced normal children.

As regards the **treatment** of primary infantilism, there is, unfortunately, little to be said. It is well to *try* thyroid, as one never can be quite sure that one is not really dealing with a masked case of the myxœdematous type, but my experience is that in true primary infantilism it is useless. The same is unfortunately true of the modern anterior pituitary growth hormones in most instances. Nor have I found the least benefit from any other 'endocrine.' It must always be remembered that, as I have already pointed out, growth may set in quite suddenly in these cases of its own accord

and at any moment. Hence, any remedy which is being tried at the time may easily get undeserved credit. In one case, for instance, the ' replacement of a bone in the spine ' by an osteopath was believed to be the cause of a sudden spurt in growth which took place not long afterwards. No ; our main remedies are time and patience, and my experience is that if one trusts to these the ultimate result in cases of this kind is much better than is usually expected.

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