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LECTURES ON DISEASES OF CHILDREN

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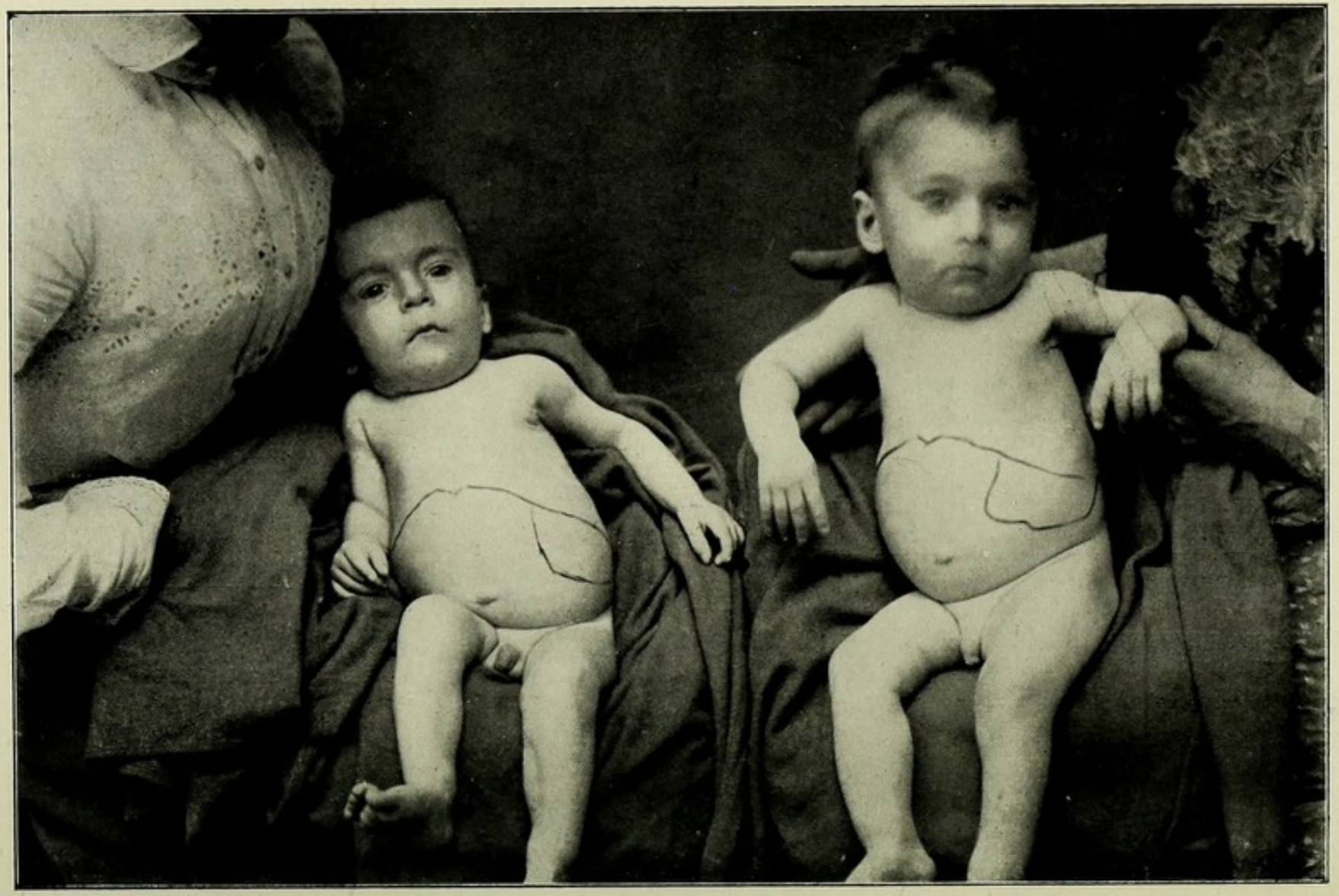


FIG. 1.
SPLENIC ANÆMIA IN TWINS.

LECTURES

ON

DISEASES OF CHILDREN



BY

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LONDON

EDWARD ARNOLD

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1904

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TO

DR. JOHN THOMSON,

MY FIRST TEACHER IN DISEASES OF CHILDREN,

I DEDICATE THIS BOOK

IN RECOGNITION OF HIS EMINENCE AS A

WORKER IN THIS FIELD

AND IN GRATITUDE FOR A LONG FRIENDSHIP.

PREFACE

THE lectures in this book were delivered in the form of a systematic course at the London Hospital two years ago, and first published serially in the *Clinical Journal*. I have gathered them together at the request of many of those who listened to them, and who have been kind enough to express a wish to have them in a more convenient form for reference.

The aim and scope of the book are sufficiently indicated in the opening paragraphs of the first lecture, but I would only say here that it is not intended in any sense to be an exhaustive treatise on the diseases of childhood, nor to compete with the many admirable text-books of that subject already in existence. My object has simply been to describe those common diseases of children which are not usually dealt with in systematic lectures on medicine, and which are apt to receive but little attention in ordinary ward teaching. The subject has been approached from a purely clinical standpoint, and with but little reference to problems of pathology ; on the other hand, questions of treatment have been dealt with in some detail. The fact

that the lectures were delivered from only a few notes and have been merely corrected from the stenographer's report must serve as an excuse for the rather colloquial character of the style in places, and for not infrequent repetitions.

The illustrations are, with one or two exceptions, original, and have been reproduced from photographs of my own cases by lantern-slides of which the lectures were illustrated.

I am indebted to my friend Mr. Robert Tanner for the care he has bestowed on the revision of the proof-sheets, and must express also my great obligation to Dr. Eliot Creasy, editor of the *Clinical Journal*, for his permission to reproduce the lectures in book form. The index is the work of Mr. Hewitt, of the Royal College of Surgeons.

R. H.

Queen Anne Street, W.
July, 1904.

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LECTURES ON DISEASES OF CHILDREN

LECTURE I

THE CLINICAL EXAMINATION OF SICK CHILDREN

Gentlemen,—It will be generally admitted, I think, 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 are 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, the consequence of which is that many men when they qualify know almost nothing of many of the commonest ailments of infant life. On going out into practice you are not much better informed in many cases

than the mother or the nurse as to the diseases which you have got to deal with. And yet you will find that in any large general practice children make up a great part, perhaps the greater part, of your patients, and the seriousness of their diseases will be brought home to you by a study of the death-rate, for whereas the adult mortality has gone down in the last fifty years by about 4 per 1,000, the infantile mortality is as high as ever it was; and, indeed, of every hundred children born fifteen never see their first birthday. Now, gentlemen, that is not creditable to our profession, and although I admit that much of it is due to social and economic causes for which you and I are not in any way directly responsible, yet I cannot doubt that if all practitioners had the opportunity as students of becoming familiar with the treatment of the common diseases of infancy, the result *would* be seen in an appreciable diminution of our infant mortality.

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 treatment in children eminently satisfactory. I am bound to confess that when you study the results of the treatment of

disease in adults in large hospitals such as this, 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 in this part of London 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.

Now, it has always been the boast of this hospital that whatever it may do or leave undone, at all events it offers students unrivalled opportunity of fitting themselves for practice. But although this is true, and although we treat here a much larger number of sick children than any other general hospital, there has not hitherto been given here, any more than at any of the other Metropolitan Schools, systematic instruction in the special diseases of childhood. That gap in your training I should consider

it a privilege to be able to fill, and I have therefore invited you to meet me here, by the consent and with the encouragement of my colleagues, in order that we may consider together, briefly and practically, the nature and treatment of those diseases which are peculiar to early life.

I may now turn, gentlemen, 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 extremely 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 to 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 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 child exhibits.

With what are called ' subjective symptoms ' in children you are not in any way troubled, because there are none. Pediatrics 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, what other children there have been, how many of those are living, and how many, if any, are dead, and the ages at which they died ; 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 the child 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 burdened. 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. 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. It is well that this should be done 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 in the face.

You should begin your inspection by noting the child's **expression.** Much has been written about the facies of disease, and you will find mysterious 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 a matter 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 infants, and you will get familiar by-and-by with the facies of abdominal disease, with the facies 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 debauchery and 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 normal, but which the child's mother may think are abnormal. One of these is that a 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 bigger 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 that 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

the 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.

Other points to notice are the character of the respiratory movements—whether they are rapid, regular, easy, 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 recognise 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 infancy.

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 far back in point of time, I remember still having been handled by a doctor with very cold hands, and I never forgot it or forgave him. I recommend you to begin palpation with the head. You do not often palpate the head of a grown-up patient, but in the case of a child it is very important to do so. First of all 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 sign of softening of the bone, such as one finds in cranio-tabes, 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 ever give you. You will also notice whether there is beading of the ribs, which is an important and early sign of rickets ; then 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, I think you should run your hands over the limbs and notice whether there is any tenderness of the legs. Many cases of obscure disease in children are to be explained by inflammatory affections of the bones, which can only be determined 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 the child is likely to have unpleasant associations with that position, and also because the pressure on the abdomen, if the child is on his face, pushes

up the abdominal organs and interferes with the expansion of the lungs. So examine the child when sitting up and leaning against the mother's or the nurse's shoulder. It is commonly recommended that you should listen to the chests of children 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. If you use a stethoscope it must be a binaural. You have to follow the movements of the child as he wriggles about, and you cannot do that with a single stethoscope. Further, do not have a stethoscope with a metal end-piece; have vulcanite, or something which is warm 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 a reversible chest-piece 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 insures 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 to me by my friend Dr. Sutherland, who has given some attention to the matter, that the result of such breathing 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. 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 encounter in grown-up persons; for example, collapse of the lung. It is one of the first things to think 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 second sound is accounted louder than the pulmonary. In the infant that is not the case. Below the age of four—and this is based upon the investigation of 1,000 cases*—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

* See a paper by Miss Sarah Creighton in the *Medical Record*, January 13, 1900.

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, 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 tension of the blood 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 of the child, which yields before the percussing finger. Be careful also when you are percussing the chest of a child 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 dulness, whereas one was only percussing one lung during inspiration and the other during expiration. Sometimes you will detect dulness over the manubrium of the sternum in a child, 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 (see p. 211).

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. Personally, I attach very little importance to it.

In examining the **fundus of the eye** in young children it is best to use the direct method, kneeling at one side of the cot with a lamp on the pillow at the opposite side of the head. 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, and in many cases you may also require to proceed to palpation of the throat ; but that I shall describe when speaking of the part played by adenoids in the diseases of children.

Finally, gentlemen, you must not expect to be very 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 it presents itself. When the child is on his back you must examine the heart, the anterior aspects of the lungs, and the abdomen, and when he is sitting up you have to 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. I cannot impress upon you too strongly the importance of weighing babies. In the children's hospital I have every baby weighed before I see it at all. The weight is often the only criterion as to whether the infant is improving or going back in health. I do not mind what weighing apparatus you use, but you must weigh all your children so far as you can. I have put into your hands a table showing what weight a child should be at different ages, and I have here a chart showing the curve of weight for

the first year (Dia. 1). There are certain turning-points of weight to which I would direct your special attention, 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 eighteen 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**

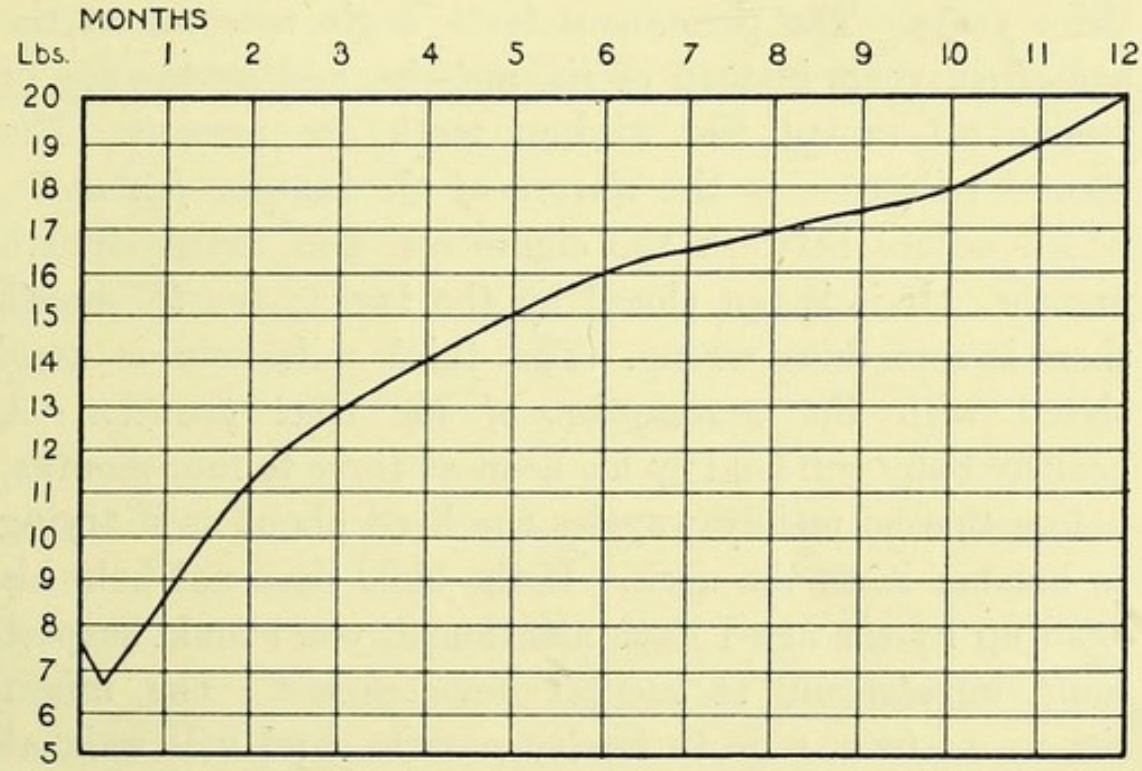


Diagram. 1.—Weight Curve of First Year. (After Holt.)

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 which you will look foolish about 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. Of course, you will look absurd if you ask whether a three-months-old baby has got any teeth. All the temporary teeth are present at the age of three years. The permanent teeth begin to come at the age of six years instead of six months, and by the age of twelve all except the wisdom teeth are present. The second milestone is the *closure of the anterior fontanelle*, 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 the head 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 well when he is two years old.

The last thing I want to mention to-day is the **character of the motions** in a child. 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. You are not to regard this number of motions at such an age as evidence of diarrhœa. Up to this period the motions resemble beaten-up eggs in colour and consistence, and

they are of somewhat sour but not feculent odour. Look specially for cheesy masses of casein in them, for if you find these you know that the child is getting more milk than he can properly digest. Up to the end of two years from about the eighth month there is an average of two motions daily. They become now more brown in colour, more porridgy in consistence, and of a slightly feculent odour. After two years of age the motions are well formed and feculent.

In my next lecture I shall deal with the subject of infant feeding.

LECTURE II

THE ARTIFICIAL FEEDING OF INFANTS

GENTLEMEN,—We approach to-day a subject which exceeds, perhaps, in importance any to which I shall have occasion to direct your attention throughout this course of lectures—I mean the subject of infant feeding. When you go into practice, the question, How is one to feed this baby? is one which will occur to you with perplexing frequency; and although it is a subject upon which a great deal has been written, I think one may say this, that the more you read about it the more confused you are likely to get, for, it must be confessed, much that has been written on the subject serves only to darken counsel, and you will find it of very little use to you in practice. I shall not deal in this lecture with all the conceivable methods by which children might be or have been fed; I want rather to describe quite plainly the methods which I have tried myself, and which I think you will find most easy and simple to carry out in your ordinary work.

Now, the first question you have to ask yourself is, Why should artificial feeding of children be necessary at all? You know that in the natural state of things every woman is the source of nourishment for her own child, but you will not have been long in practice before you



FIG. 2.

TWINS, THE ONE ON THE LEFT BREAST-FED; THE OTHER BOTTLE-FED. A COMMENTARY ON THE ADVANTAGES OF BREAST-FEEDING.

realize that under modern conditions it is the minority of women who can nurse, or do nurse, their own children. It would be unjust to the female sex generally to state that this inability to nurse their children is their own fault. I do not think that is true ; I think, in the majority of cases, one can say that the woman is unable—not unwilling—to nurse her own child. Now, that is a phenomenon of very considerable gravity, but it is one which is not confined to this country ; it has been found to hold good of the whole of Europe, and in America also.* It seems to be part of the price which we pay for civilization. It would appear that under civilized conditions of life the tendency of women to nurse their own children becomes less and less ; and I need hardly say that that is a social event of very great seriousness, because the physical advantages to the baby of being breast-fed, not to speak of the moral advantage to the mother of suckling, cannot be overestimated. This is one, indeed, of the signs of physical degeneracy which makes some observers afraid that there may arise a barbarian and more virile race which will sweep away our Western civilization altogether. Be that as it may, you will see from what I have said how important it is that you should understand how best to make good the absence of the mother's milk, and how it becomes a matter of pressing importance in practice.

If a woman is unable to nurse her own child, obviously the next best substitute is to get another woman to nurse it for her ; in other words, a wet nurse is the best substitute for the mother. But I do not think you will have occasion to employ a wet nurse often in practice.

* See Bunge, 'Die Zunehmende Unfähigkeit der Frauen ihre Kinder zu Stillen.'

There are many practical disadvantages entailed in that course, and, as a matter of fact, *artificial* feeding is synonymous in most cases with *bottle* feeding, so that what we have to consider to-day is how you can best feed a child by the bottle. There is still another question which has to be disposed of before we come to the consideration of the best way to carry out bottle feeding, and that is, Supposing a woman starts to nurse her own child, how

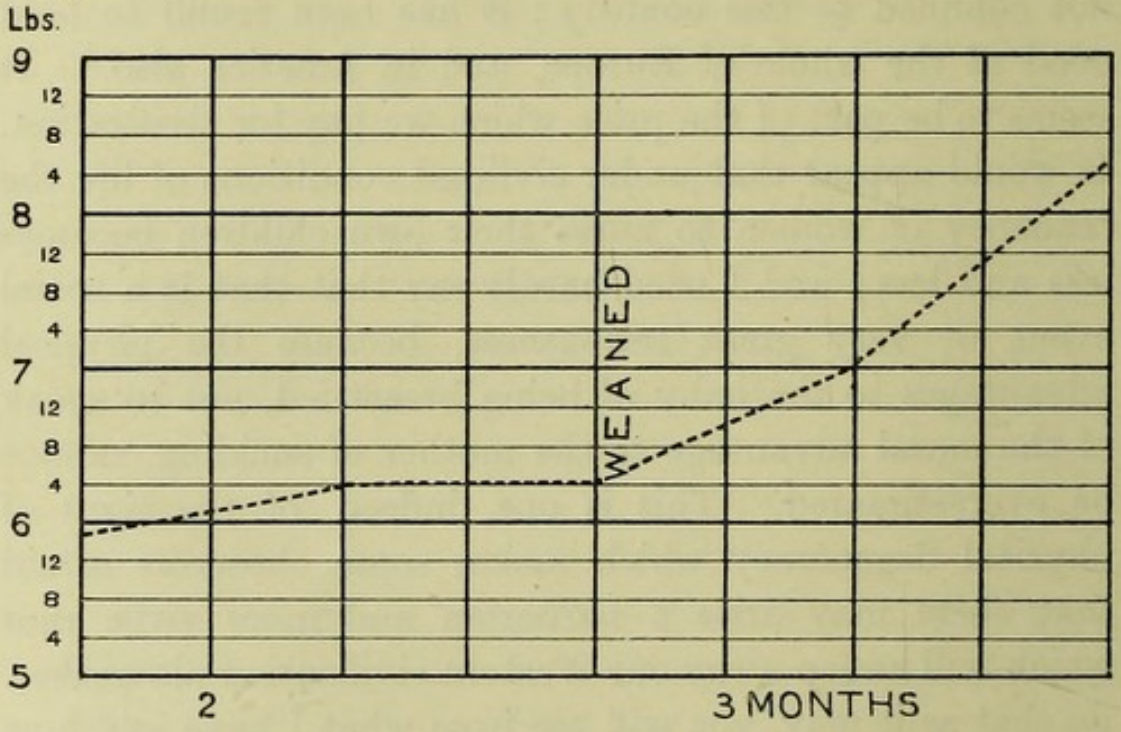


DIAGRAM 2.—INSUFFICIENCY OF BREAST MILK, SHOWING EFFECT OF WEANING.

are you to know whether the process is successful or not? In other words, what is to justify you in adopting artificial feeding in the case of a woman who has *begun* to nurse her own infant? Gentlemen, I know only one condition on the part of the child that justifies premature weaning, and that is *persistent loss of weight.* If a child is persistently losing weight when fed on the mother's breast, that is an indication, and an imperative one, for weaning,

and there is no other imperative indication that I know of. Such a state of things is illustrated in this chart (Dia. 2). It is that of a child seven weeks old which was being fed on the breast, and as we weighed it from week to week we found it increased in weight very slowly, or hardly at all. And as there was no digestive disturbance we concluded that the mother's milk was insufficient; and we accordingly weaned the child, with the result you see—namely, that the weight shot up enormously, and the child gained more in one week of bottle feeding than it had gained in two or three weeks of breast feeding. There is one caution I should give you here. Before you conclude that the breast milk is insufficient, make quite certain that you have corrected any digestive disturbance in the child; and you will find particularly that you have to correct, in breast-fed babies, constipation. This, as I shall have to point out later on, is the vice of breast-fed babies, as diarrhœa is the vice of bottle-fed babies. So you see the statement I made that progressive loss of weight on the part of the child is the only indication which justifies premature weaning has to be qualified by this, that it only holds good in the absence of digestive disturbance.

Having disposed of these preliminaries, we have next to consider **what substitute we can use for human milk.** Now, in practice, cow's milk will always be your substitute. I shall not consider in these lectures the merits of other forms of milk. Many other kinds have been recommended, but the practical conveniences of cow's milk are so great that one rarely uses any other. I cannot too strongly impress upon you, gentlemen, the fact *that it is the casein which is the source of difficulty in the artificial feeding of children with cow's milk.* There are chemical

reasons, which I shall not go into now, why the casein of cow's milk is less digestible than the casein of human milk; but whatever those reasons may be, it is a fact which you will meet in practice that you have difficulty in getting infants to digest cow's casein. For that reason you must give the cow's milk in a diluted form. How much, then, should you dilute the milk? It is best to begin first of all, as a simple practical rule, with a **dilution of half and half;** that is to say, give as much cow's milk as water. If you like, you can use barley-water or lime-water as the diluent; these make it more easily digestible than plain water. If you compare a mixture so prepared with human milk you find that from a chemical point of view it works out as follows. In human milk there is $1\frac{1}{2}$ per cent. of proteid, $6\frac{1}{2}$ per cent. of sugar, and $3\frac{1}{2}$ per cent. of fat. In cow's milk there is 3 per cent. of proteid, $4\frac{1}{2}$ per cent. of sugar, and 4 per cent. of fat. If you dilute with equal parts of water you have $1\frac{1}{2}$ per cent. of proteid, $2\frac{1}{4}$ per cent. of sugar, and 2 per cent. of fat. This makes the proportion of proteid, roughly speaking, correct, but the sugar and the fat are too poor, and you must therefore make up the deficiency of these constituents. That you can do quite simply by adding two teaspoonfuls of cream and two level teaspoonfuls of sugar to every 6 ounces of the mixture; that is assuming the cream to contain the average of fat of London centrifugal cream, which is about 45 per cent. That gives you a mixture which quantitatively is a very fair approximation to mother's milk. Qualitatively, of course, it is not the same thing as human milk, and it never can be.

The next question is, **How much** of the mixture shall you give, and **how often?** I have put into your

hands a table showing you the quantities which the child's stomach will accommodate and the proper intervals of feeding. You must not suppose that the quantities there

TIME-TABLE FOR INFANT FEEDING.

Age.	Intervals by Day.	Night Feeds.	Quantity at each Feed.
	Hours.		Ozs.
1 week - - -	2	2	1 to 1½
2 to 3 weeks - -	2	2	1½ to 3
4 to 5 ,, - -	2	1	2½ to 3½
6 to 12 ,, - -	2½	1	3 to 4½
3 to 5 months -	3	1	4 to 5½
5 to 9 ,, - -	3	—	5½ to 7
9 to 12 ,, - -	3½	—	7½ to 9

given should be quite hard and fast; you must leave it to some extent to the baby's appetite. The best plan is to tell the mother to give the child as much as he will take at each feed.

The next point is, What sort of **bottle** should you use? The only sort of bottle to be recommended is a boat-shaped one with a rubber teat attached.* The rubber tube bottles so much used probably destroy more infant life than any other single agency, and it would be a good thing if their use were illegal here, just as it is in France and in many States of America. The screw-top bottles are not much better. Impress upon the mother that the bottle must be kept very clean. With regard to the teat, many cases of failure are due to its having too small an aperture in the top. You may want to know when the aperture is big enough. It is big enough if, when you

* The Oval Hygienic Feeder and Maw's Universal Feeding Bottle are both good patterns.

hold the bottle upside down, the milk drops slowly, at the rate of about 1 drop per second. It ought not to require a great deal of sucking on the part of the baby, or it will be apt to give the child flatulence, or the infant will leave off sucking before he is satisfied, and so cease to gain weight. There is a teat in use called the 'leech-bite teat,' so named because the apertures are triangular in shape, like the shape of a leech-bite, which seems particularly liable to produce these difficulties.

The next point is, **Should the milk be boiled,** or should it be given raw? That, gentlemen, is a point which has probably been more discussed than its importance really justifies. I advise you to give it neither boiled nor raw, but scalded. That may appear to you to be a distinction without a difference, but by scalding I mean raising the milk to the boiling-point, or just short of that, and then taking it off the fire. The drawback to boiling the milk is, that there is a very slight risk of producing scurvy by it. But that can be easily remedied by adding fruit juice to the diet. I think the advocates of boiling milk have exaggerated the dangers of bacterial infection. Certainly tuberculosis is not *often* conveyed through milk. When we come to speak of tuberculosis I shall point out that in the majority of the cases children are infected viâ the bronchial glands, and that dust is the great source of infection for children, as it is for grown-up people, and the tendency to regard milk as the main source of infection is erroneous. In hot weather it is much more necessary to scald the milk, because thereby one diminishes the tendency to its going sour and causing diarrhœa.

Having given the food by the bottle I have described, and at the intervals and in the proportions pointed out, you

have to ask yourself, **How are you to know whether the results are satisfactory?** Here, again, the only test that this mixture is suitable is the balance. At what rate, then, should the child gain weight? This is not difficult to remember, because a healthy child should gain almost 1 ounce a day; he should gain 6 ounces a week. Obviously, one of two things will happen on this mixture: either the child goes on gaining weight, or he suffers from digestive disturbance, colic, possibly diarrhœa, possibly vomiting. If the child does not gain weight, or if he suffers from digestive disturbance, what are you to do? I advise you to remember the possibility, particularly if you are dealing with a very young child, that this **mixture is too strong,** and that it may be advisable to make it more dilute. In this chart (Dia. 3) I show you an instance of such a result. It is the chart of a child eleven weeks old who was fed on milk and water, equal parts of each, which resulted in the production of diarrhœa and vomiting, and only a slight increase in weight. Then we gave 1 part of milk and 2 parts of water, and immediately the weight shot up and everything went well. This shows that it is possible for a child to lose weight because it is getting too much to eat. Very often you will find that in young infants you may require to begin with 1 part of milk

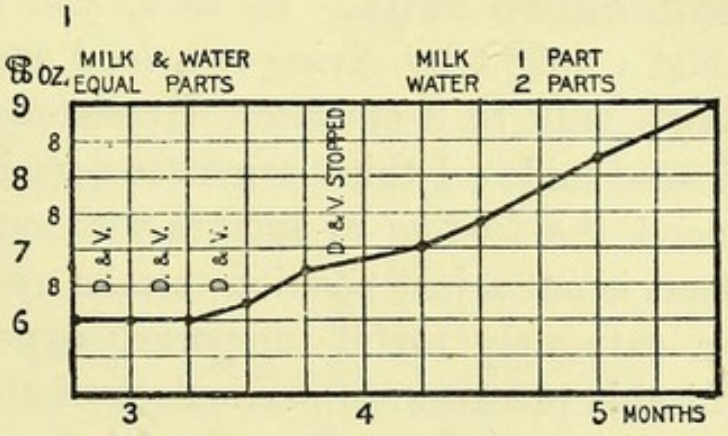


DIAGRAM 3.—FAILURE TO GAIN WEIGHT DUE TO FEEDING WITH TOO STRONG A MIXTURE.

to 2 parts of water instead of equal parts of each, sugar and cream being added in proportion.

Supposing that even in this very dilute form **the milk still disagrees,** what are you to do then? I do not think you are likely to effect much good by diluting cow's milk to a greater degree than this. One does not often see children do well on a mixture which is weaker than 1 in 3. Therefore, what you have to do is to look out for a more digestible form of milk than ordinary cow's milk. You will find that more digestible form in **condensed milk.** If, then, you fail to improve the child's weight by giving it a food consisting of 1 part of cow's milk to 2 of water, I recommend you to try condensed milk. I think some consider it a heresy to recommend the feeding of infants with condensed milk at all. That is not a just position to take up. Condensed milk is an extremely useful temporary expedient; if used with suitable precautions, particularly if the deficiency of fat is rectified, it may be quite successfully given for the first two or three, or even more, months of the child's life. It is quite true that the continued use of condensed milk is almost certain to result in the production of rickets if kept up until the child is six months old. But, fortunately, by six months the child's digestion has generally become capable of dealing with ordinary cow's milk. Well, what brand of condensed milk should you use? Never select skimmed or separated condensed milk at all. The brand selected should contain all the original cream.

There are two classes of such condensed milk, those which are sweetened and those which are unsweetened. I used to recommend unsweetened, because in theory that is the best form, but I have given it up because it is so difficult to obtain. In practice I always recommend

Nestlé's milk ; it contains 13 per cent. of fat—that is to say, all the original fat of the milk—besides considerable quantities of added sugar. The dilution I recommend to start with is one teaspoonful of condensed milk to six tablespoonfuls of water, not barley-water, but ordinary water which has been boiled and then allowed to cool. Such a mixture has the following composition :

Proteid	2·0	per cent.
Fat	1·6	,,
Sugar	8·17	,,
Ash	0·34	,,

Of the sugar, 3·5 per cent. is in the form of lactose, and 4·6 per cent. is cane sugar. The total sugar is somewhat in excess, and the fat is distinctly too low; but by the addition of a teaspoonful of centrifugal cream the total fat is brought up to 3·5 per cent., which is the same as that in human milk. I have here charts which show how successful feeding may be with condensed milk. Here is one of a child which was given two teaspoonfuls of condensed milk to eight tablespoonfuls of water, and you see how steeply the weight curve rises (Dia. 4).

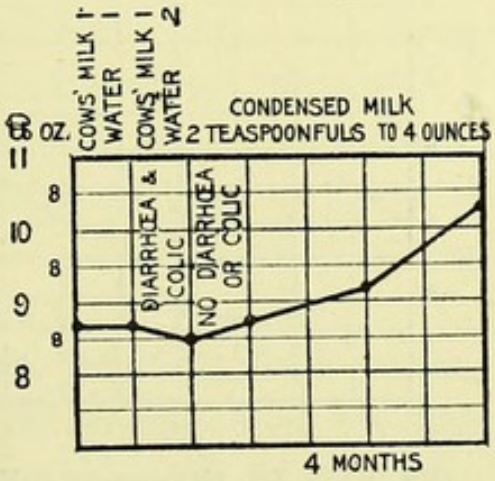


Diagram 4.—Successful Feeding with Condensed Milk.

You may ask, Why is condensed milk so much more easily digested than ordinary cow's milk ? I think it is largely because one gives it so dilute. This dilution of one in six means that the casein will be much diminished, and if you add acetic acid to such a mixture and compare

it with peptonized milk you find you get about an equal precipitate in each case.

Supposing even condensed milk fails, what are you to try next? You may then fall back upon **peptonized milk,** although it is not much more digestible than condensed milk unless it is very carefully prepared. I

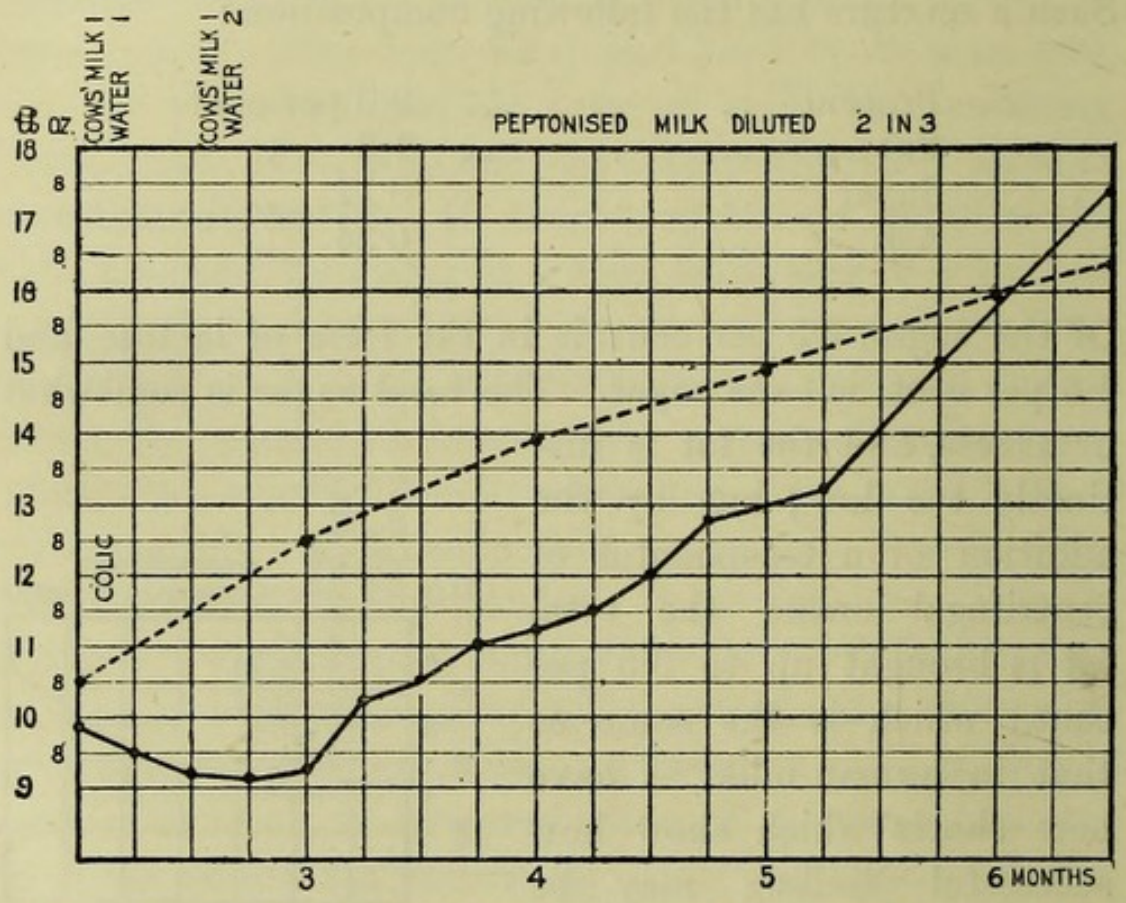


Diagram. 5.—Successful Feeding with Peptonized Milk. The Dotted Line represents the Normal Weight.

recommend you to use for making it Fairchild's peptogenic powders. Each packet contains full directions, and satisfactory results are certainly obtained by their use. But if you give peptonized milk alone it may still be too strong, and in that case you must dilute it with one-third or even one-half of lime-water. This will insure easy digestion. So, you see, one may regard diluted cow's milk,

condensed milk, and peptonized milk as the positive, comparative, and superlative of digestibility; and it is well to have in your minds some such scale of digestibility as that, so that you can adjust the diet for any particular child to his digestive power. If his digestion is very feeble, you give peptonized milk; if it is somewhat stronger, you give condensed milk; and if it is still stronger, you give cow's milk. There are many charts on the wall which show the results of successful feeding with peptonized milk. This, for instance (Dia. 5), was the case of a child which had colic and constipation when fed on ordinary milk. Then we put him on to peptonized milk and water, and you see the weight rose very rapidly. Some people raise an objection to peptonized milk; they say you will so demoralize the child's stomach by that means that it will be unable to digest ordinary milk later on. I believe, gentlemen, that this is a purely imaginary objection. I have fed children upon peptonized milk for many months, but I have never had any difficulty in getting them to digest ordinary cow's milk later on. So I think you may dismiss this bogey from your minds.

But **if you fail even with peptonized milk,** then? Well, what gentlemen, the resources of civilization are not yet exhausted; there are still some things you can do, and I should recommend you in a case like that, when you have tried peptonized milk and the child still does not gain weight, to try the effect of a little gray powder. I do not know how the gray powder acts in those cases, but I am satisfied that in many cases, even although there is no reason to suppose there is a congenital syphilitic taint, the administration of gray powder will in some mysterious way make all the difference to the child's

digestive power, and I have charts which illustrate that fact. This (Dia. 6) is the chart of a case in which we had tried the positive, comparative, and superlative of digestibility, and still we got no satisfactory increase in weight. Then we put the child on to gray powder, ½ grain twice a day, and immediately the weight went up. Gray powder seems to fatten some children, just as cod-liver oil does. Another child was fed upon milk and barley-water in equal parts, but he was not doing well. Gray powder was then given, and you see how great improvement

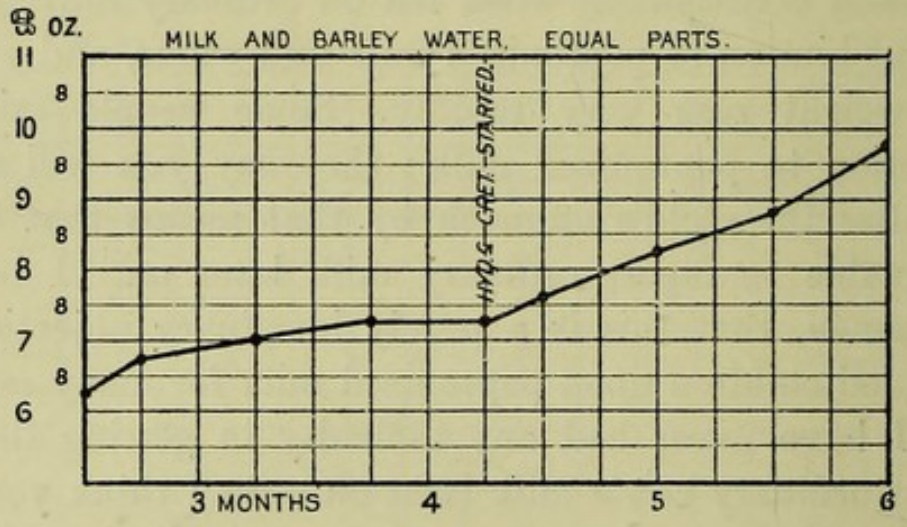


DIAGRAM 6.—SHOWING THE EFFECT OF GRAY POWDER.

resulted, although in that case also a history of syphilis was extremely doubtful.

If in spite of all you do the digestion does not improve, you are justified in thinking you have to deal with a child who is incapable of digesting cow's casein. There are such children, those who cannot digest the casein, no matter how you present it to them. And you have in such cases to take away the casein altogether, and to do that the simplest way is to prepare **whey** from the milk. Curdle the milk with rennet, and allow it to set into a solid mass ;

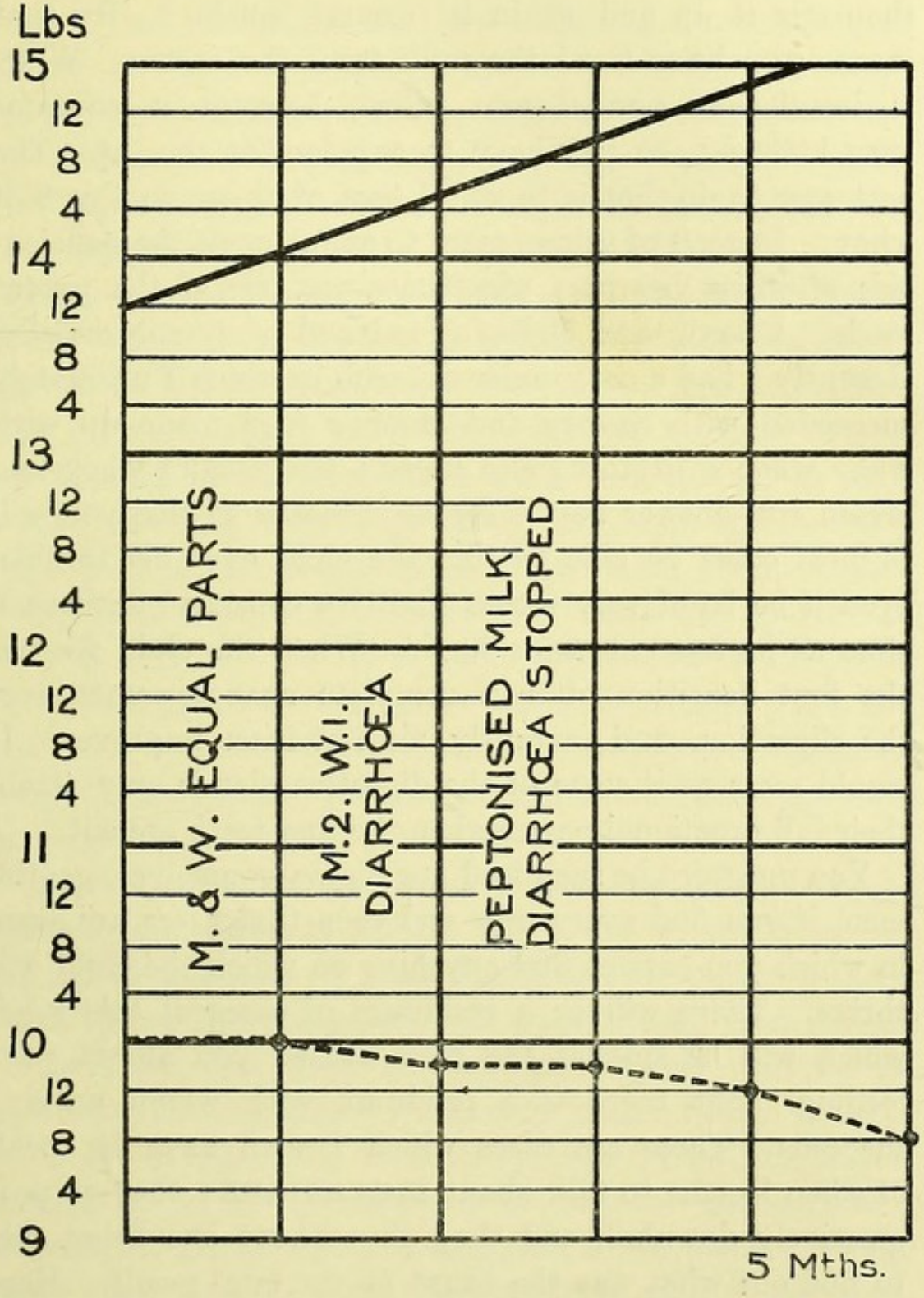


DIAGRAM 7.—WEIGHT CHART OF A CASE OF MARASMUS ENDING FATALLY THE UPPER LINE REPRESENTS THE WEIGHT OF A HEALTHY CHILD OF THE SAME AGE.

then stir it up and strain it through muslin.* By that means you have freed the milk from the casein. Whey is insufficient nourishment alone, because it contains very little fat, so you have to supplement the fat. The best way to do that is to add 1 part of cream to 4 or 8 of whey. Instead of using cream to supplement the deficiencies of whey you may sometimes use one of the patent foods. I have used Mellin's food and whey successfully. Recently I had a case under my care in which I ultimately succeeded with Savory and Moore's food made up with whey when everything else failed; but usually whey and cream will answer best. By such means as this you will in most cases be able to tide the child over his trouble, even if he be of very feeble digestive capacity, until such time as he has cut some teeth. When the child reaches the first dentition often a complete change comes over the digestion, and instantly his nutrition improves. It would seem as if some of the digestive glands only attain their full functional power when the first teeth are cut.

You must not be surprised nor blame yourselves, gentlemen, if you find every now and then that there are cases in which you cannot find anything on which the child will thrive. There will be a residuum of cases of this kind, which will be smaller the more skilled you are in your feeding. But there *is* a residuum with whom nothing succeeds. These are cases which I shall have to speak of when I come to talk about **marasmus;** they go progressively downhill, and they die without one being able to find out what was the cause of the fatal result. Here I show you a chart (Dïa. 7) which illustrates the course of

* The whey should be scalded before use, to destroy the rennet which it contains.

such a case, in which we tried milk and water in equal parts; we tried it stronger, and it produced diarrhœa; we tried peptonized milk for a time, and it was succeeded by diarrhœa and vomiting, and then the child died. Nothing which you can find after death will account for this congenital feebleness of digestive power.

I now want to impress upon you one further point about the artificial feeding of babies, and that is *that you should not lightly change from one kind of food to another.* You will constantly be having children brought to you whose mothers say they have tried everything. In such a case you may take it generally that *nothing* has really been tried in the proper sense of the term. You cannot judge from one week's experience what any particular food is going to do for a child; you have to keep the child on a diet for a reasonable time, so as to give it a fair trial, and it is only if you are finding that he is not gaining weight that you are justified in concluding that the food he is having is not suitable for him.

Another question is, What should be your attitude towards the **patent foods** which are so much in use for infant feeding? I have put into your hands a sheet which gives the composition of those foods, from which you will see that many of them are entirely unsuited to be a complete substitute for human milk. I have divided them, for convenience, into three groups: First, one which is intended to be a complete substitute for human milk; secondly, a group which is intended to be made up with cow's milk, and in which the artificial part of the food is predigested; the third class includes those which contain unaltered starch. My advice to you would be to rule out the third group at once for all children under

TABLE SHOWING THE COMPOSITION OF INFANT FOODS.

Food.	Water.	Proteid.	Fat.	Carbohydrate.	Mineral Matter.	General Description and Remarks.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	
Dried human milk ..	—	12·2	26·4	52·4	2·1	The standard of composition to which artificial substances should conform.
	GROUP I.					
Allenbury No. 1 (for children below the age of three months)	5·7	9·7	14·0	66·85	3·75	Desiccated cow's milk from which the excess of casein has been removed, and a certain proportion of soluble vegetable albumin, milk, sugar, and cream added. No starch present. Half an ounce in 3 ounces of water for a child aged three months.
Allenbury No. 2 (for children of the age of from three to six months)	3·9	9·2	12·3	72·1	3·50	Resembles the above but contains some malted flour in addition. No starch present. One ounce in 6 ounces of water for a child aged six months.
Horlick's Malted Milk	3·7	13·8	9·0	70·8	2·70	A mixture of desiccated milk (50 per cent.), wheat flour (26¼ per cent.), barley malt (23 per cent.), and bicarbonate of soda (¾ per cent.). Contains no unaltered starch when mixed. Three teaspoonfuls (equals 22 grammes) in 4 ounces of water for a child aged three months.
Carnrick's Soluble Food	5·3	13·6	2·5	76·2	2·20	A mixture of desiccated milk (37½ per cent.), malted wheat flour (37½ per cent.), and milk sugar (25 per cent.). When prepared according to directions the casein is partially digested, but a considerable amount of unchanged starch is left. One part to be mixed with 9 parts of water and boiled for a few minutes.

Nestlé's Milk Food ..	5·5	11·0	4·8	77·4	1·30	A mixture of desiccated Swiss milk, baked wheat flour, and cane sugar (30 per cent.). More than a third of the total amount of carbohydrate is in the form of starch. One ounce to be mixed with 5 ounces of water.
Manhu Infant Food	8·8	8·7	5·6	75·9	1·00	A mixture of desiccated milk and malted cereals. When prepared according to directions contains a good deal of unaltered starch. A dessertspoonful (equals 13 grammes) to be mixed with 2½ ounces of water.
GROUP II.—*Class A.*						
Mellin's Food ..	6·3	7·9	Trace	82·0	3·80	A completely malted food. All the carbohydrate in a soluble form. May be regarded as a desiccated malt extract. Half a tablespoonful (about 5 grammes), ¼ pint of milk, and ¼ pint of water for a child under the age of three months.
Cheltine Maltose Food	4·6	5·3	0·27	87·6	2·25	A fully malted food, containing no starch. To be used with milk.
Hovis Babies' Food, No. 1	3·7	7·7	0·20	86·6	1·82	A fully malted food. Carbohydrates entirely in the form of maltose and dextrins. To be made with milk.
Class B.						
Savory and Moore's Food	4·5	10·3	1·4	83·2	0·60	Composed of wheat flour with the addition of malt. When prepared according to directions, most, but not all, of the starch is converted into soluble forms (chiefly dextrins). One or two tablespoonfuls (equals from 1 ounce to 2 ounces) to be mixed with two or three tablespoonfuls of cold milk or milk and water, and ⅓ pint of boiling milk, or milk and water, to be added.
Benger's Food ..	8·3	10·2	1·2	79·5	0·80	A mixture of wheat flour and pancreatic extract. When prepared according to directions, most, but not all, of the starch is converted into soluble forms. The proteid is also partially digested as well as that of the milk used in mixing it. One tablespoonful (about 1 ounce), and four tablespoonfuls of cold milk, then add ½ pint of boiling milk and water; set aside in a warm place for fifteen minutes, then bring to the boil.

TABLE SHOWING THE COMPOSITION OF INFANT FOODS—*continued.*

Food.	Water.	Proteid.	Fat.	Carbohydrate.	Mineral Matter.	General Description and Remarks.
	Per Cent.	Per Cent.	Per Cent.	Per Cent.	Per Cent.	
Allenbury Malted Food	6·5	9·2	1·0	82·8	0·50	A mixture of wheat flour and malt. When prepared according to the directions it still contains some unaltered starch. Designed for children above the age of six months. One tablespoonful (about 1 ounce), a teaspoonful of sugar, and three tablespoonfuls of cold water; mix and add ½ pint of boiling milk and water (equal parts).
Diastased Farina ..	8·3	7·6	1·3	81·7	1·10	A malted farinaceous food. When prepared according to the directions practically all the starch is converted into soluble forms. One ounce of food, ½ pint of cold milk, and 2 ounces of water. Heat slowly till it boils; boil three minutes, and sweeten if desired.
Coombs's Malted Food	7·9	12·1	2·8	76·8	0·40	A malted farinaceous food. When prepared according to the directions it still contains much unaltered starch.
Moseley's Food ..	10·8	11·0	0·92	76·4	0·94	Complete conversion of all starch occurs during mixing. To be given with milk.
Nutroa Food ..	6·8	15·9	10·3	66·0	1·00	A mixture of cereals with the addition of a certain proportion of pea-nut flour, from which the somewhat bitter taste of the food and its high proportion of fat are derived. It is a self-digesting food, but when prepared according to the directions only part of the starch is converted. One ounce of the food to be mixed with 1 ounce of cold water, and ½ pint of boiling milk and water (equal parts) to be added.

Group III.						
Ridge's Food ..	7·9	9·2	1·0	81·2	0·70	A baked flour, containing only 3 per cent. of *soluble* carbohydrates, the remainder being starch. Recommended to be made with milk or water. Made with water alone is totally insufficient food.
Neave's Food ..	6·5	10·5	1·0	80·4	1·60	Resembles the above, but recommended to be made with milk and water.
Frame Food Diet ..	5·0	13·4	1·2	79·4	1·00	A thoroughly baked flour to which have been added cane sugar and some extract of bran. It is *not* specially rich in mineral ingredients, but nitrogenous matters are abundant, and it contains much unaltered starch. One-third of an ounce to be mixed with a breakfast-cupful of milk and water (1 part of milk to 2 parts of water).
Bananina	9·5	4·1	0·40	84·0	2·07	A highly starchy food prepared from banana flour.
Cheltine Infants' Food	7·2	16·2	3·92	71·0	1·83	Contains starch when prepared for use.
Hovis Food, No. 2	2·4	5·7	0·10	90·1	1·70	Contains about 7½ per cent. of starch. To be made up with milk.
Opmus Food ..	10·9	9·1	1·0	78·6	0·40	A granulated wheat food. One teaspoonful to ½ pint of milk. Starch unaltered.
'Falona'	7·0	8·4	3·5	79·9	1·20	A mixture of cereals (oats, barley, and wheat), with a ground fat-containing bean. The food is thoroughly baked, but contains a considerable proportion of unaltered starch. A teaspoonful to ½ pint of boiling milk *or* water, or half milk and half water.
Robinson's Groats ..	10·4	11·3	1·6	75·0	1·70	Ground oats from which the husk has been removed. Rich in proteid and mineral matter
Robinson's Patent Barley	10·1	5·1	0·9	82·0	1·90	Ground pearl barley, poor in every element except starch and mineral matter.
Chapman's Whole Flour	8·4	9·4	2·0	79·3	0·90	A finely ground whole wheat flour. Not much superior in nutritive value to ordinary 'households' flour. Starch entirely unaltered.
Scott's Oat Flour ..	5·8	9·7	5·0	78·2	1·30	A fine oat flour. Somewhat inferior in nutritive value to 'groats.' Starch unaltered.

six months old. As regards the first group, they really present very little advantage over condensed milk, and I hardly ever have recourse to them. I admit that some children may be reared successfully on certain patent infant foods, but hardly ever if condensed milk and other methods of feeding have been properly tried and have failed. They all incur the risk of producing scurvy. Therefore, if you use those foods it is well to see that some fresh elements are introduced into the diet, such as grape or orange juice.

The second group of foods is sometimes useful for children who are getting towards the period of first dentition, and who are not thriving very well because it is difficult to get them to take sufficient cow's milk. In these cases it is advisable to supplement the milk with some other food, and a dextrinized food is best. There is one class of those dextrinized foods in which the change in the starch takes place during mixing. I think it is more convenient to use a food in which the change has already taken place; and so, if one has occasion to use this group at all, one selects Mellin's food or Hovis food as being already dextrinized in preference to the others. But remember they are all deficient in fat, and their continued use is apt to result in rickets. If you use them at all it should be with that danger clearly before your minds, and they should be given only for a limited time.

One or two other questions I must discuss briefly. If a child has been on the breast, and you have not had recourse to any artificial method, and everything is going on well, **when are you to wean?** I want you to have clear ideas on this matter. It used to be said by many people that a child should not be weaned until he

has cut some of his teeth. I think even Trousseau went to that length; he was guided in his weaning of children by the dentition. But you must remember always that a child may fail to cut his teeth because he is rickety, from being fed on the breast too long; therefore, you see, the appearance of the teeth alone is no sufficient criterion of when you are to wean. You may lay it down as a general rule that the child should be weaned between the tenth and the twelfth month. Mothers, especially in the lower classes, often keep on suckling their children, partly from motives of economy and partly to prevent another pregnancy, for too long a time. I have even known children fed mainly on the breast until they were two years old, and without obvious harm. But I think in such a case there is very likely to be detriment to the *mother's* health. Between the tenth and twelfth month is the rule which you should adopt for yourselves.

Another piece of advice which I should like to give you in this matter is, never to wean a child in hot weather if you can avoid it. The reason for this is that children who are weaned in hot weather are very apt to become the subjects of epidemic diarrhœa, and to be carried off by it. It is better to allow a child to be nursed a little too long than to wean him at a time when diarrhœa is prevalent.

A kindred question to weaning is, When should you add **starch** to the diet? When is it safe to begin to allow a child to have other food than milk? Experience shows one that the individual power of digesting starch varies enormously in different cases. I have known children who got starchy food from their earliest days, and yet, apparently, did not suffer from it. I recall, for instance,

the case of twins being brought to me who had been fed with a strong decoction of barley-water *without any milk* for three months, and yet they were not so thin as you would suppose, and had no digestive disturbance. I have also known a child fed upon Robb's biscuits, which contain a good deal of starch, without any harm when one month old. On the other hand, one sees children in whom starch appears to produce fermentation, acidity, diarrhœa, and colic. So your general rule should be not to allow a starchy food in any shape until the child has cut a tooth or two, and then you can begin to give such foods very gradually and cautiously. There are some children, however, in whom it may be worth while to run the risk of giving a little starch sooner than that. Children who are not getting on well with milk, and who have a difficulty in digesting casein, should have the benefit of the trial of a little starchy food. I once got a lesson in this matter from a mother whose child I was attending for difficult digestion. We were struggling on with peptonized milk and making no headway. One day, a week after I had seen the child, he was brought back weighing a pound more than at the last visit, and it transpired that the mother had, of her own accord, added a considerable quantity of cornflour to the diet, and with complete success. The starch-digesting capacity of that child was evidently in excess of his milk-digesting capacity, and in this case the experiment was justified by the result. Anyhow, it is an experiment which cannot do much harm, because so soon as you find it disagree you can stop the food.

In my next lecture I purpose dealing with the disorders of digestion which are apt to result from the neglect of the rules which I have just brought before you.

LECTURE III

THE DIGESTIVE DISORDERS OF INFANCY—COLIC AND VOMITING

GENTLEMEN,—We come now to the consideration of the disorders of digestion in infancy—disorders which are due, in large measure, to neglect of those rules for the feeding of babies which I tried to bring before you in my last lecture.

I shall not distinguish between the digestive disorders of breast-fed and bottle-fed infants, although, for obvious reasons, such disorders are far commoner in the case of bottle feeding than in the case of breast feeding. Further, shall not attempt to describe these disorders to you from he point of view of pathology or morbid anatomy, because to do so would be to assume a knowledge which we do not possess. I shall deal with them purely from the clinical or symptomatic standpoint.

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 those 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 two causes as giving rise to the painful contraction of the intestines which we call colic. The first of these is acidity due to fermentation occurring in the milk, and producing lactic and other acids, which irritate the bowel and induce overcontraction in its wall. The other, and perhaps the commoner, cause is the existence of undigested casein or curds in the intestine. I told you that the great difficulty in the feeding of infants with cow's milk is the indigestibility of the casein, and there is no commoner consequence of that than the production of colic.

How are you to recognise colic ? Its chief symptom is pain. Of course, pain in the case of infants is expressed not in words, but by cries, and whenever you find constant and frequent **screaming** it should always make you think of the possibility of colic, for that 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 *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. Another possible cause of persistent screaming is *teething*, a cause which mothers certainly tend to exaggerate, but, neverthe-

less, it is one which you should always have in mind as a possibility. 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 fœtuses *in utero* you may find them. Another possible cause of screaming is *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 specific disease. 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 perhaps commoner than all these other causes put together.

The **signs by which you recognise 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 feel little coils of intestine standing out, coils of firmly contracted bowel. 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 recognise 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 with me 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 well 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 two and a half hours instead of every two hours, or every three hours instead of every two and a half, 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 set yourself 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 the casein of the mother's milk being either too abundant or for some reason more difficult than usual of digestion. When you meet with such a case you will sometimes find it of advantage to administer a few teaspoonfuls of lime-water to the child before each feed, the lime-water being sweetened a little to make it acceptable to the child. Further, you will remember to correct any constipation, which is so common in breast-fed children—a most important point. In breast-fed children colic usually goes

with constipation, and in bottle-fed children with diarrhœa. How are you to treat the constipation? The wrong way is to give a violent and drastic aperient at irregular intervals, which is commonly done by mothers, who wait until there has been no motion for two or three days, and then give a large dose of castor oil. Many of you, perhaps, do not realize how extreme **constipation in breast-fed children** may be. I have known such infants go a fortnight without having any action of the bowels unless something is given to produce that action. The proper way to treat these cases is, first, by massaging the abdomen. It does not matter much what you use to massage it with, so long as you prevent the skin being chafed or broken; a little sweet oil is as good as anything. The massage should be done, round and round, along the track of the colon in the morning and evening for five or ten minutes at a time. Some people recommend that you should massage with a preparation containing aloes. That is not necessary. It is analogous to the use of inunctions of cod-liver oil in wasting, a haphazard way of doing what you can do by another means with certainty—namely, by the mouth.

With regard to **drugs,** the proper aperients in these cases are those which have a tonic action on the bowel. What you want to do is to educate the child's intestine into acting spontaneously, and for that purpose you should select such a tonic aperient as aloes, with the addition of a little nux vomica. You will find some such formula as this useful: Tincture of aloes, 3 to 5 minims (the exact dose depending on the age of the child and the severity of the case); infusion or syrup of senna, 20 minims; tincture of belladonna, 2 minims (to relieve any griping which the aloes may produce, remembering that infants stand belladonna very well);

tincture of nux vomica, ½ minim (infants stand strychnine rather badly); and compound infusion of gentian to a teaspoonful. Sometimes you can add with advantage a carminative, such as peppermint-water, dill-water, cinnamon-water, or anything of that kind which you please. Such a mixture should be given at regular intervals. In a mild case it should be given only night and morning, in more severe cases every eight hours, and in still more severe cases every six hours. Go on administering this mixture, irrespective of whether there is an action of the bowels or not, until the motions become more and more regular, and then gradually leave it off. Then you will find after you have left it off that the intestines can be trusted to act by themselves. There is one other aperient which I should like to mention, if for any reason the above prescription proves unsuitable—and the only reason I know of is that children are apt to vomit it because it is not pleasant, and, containing as it does a bitter, it is very difficult to make it pleasant—and that is confection of sulphur. Half a teaspoonful of this may be given at night only, or both morning and night. It is an exceedingly efficient laxative for young children, and you will never have any difficulty in getting babies to take it; but it does not exert, in the same measure as the prescription I have just written down that tonic effect on the intestinal wall which it is your desire as far as possible to promote.

If colic persists in a breast-fed child after you have corrected constipation, the question will arise, Are you to wean the child or not? Here I would remind you of what I said in the last 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. So if, after correcting constipation, the weight still goes down, you may consider the question of weaning.

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. The great cause of **colic in bottle-fed babies** is a want of cleanliness in preparing the milk; and in particular it is the continuous use of those tube bottles of which I have already had occasion to point out to you the dangers. Such bottles produce far more colic, probably, than any other one cause. 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. Assuming that the bottle is clean, and, therefore, that fermentative action has not occurred, the colic may be put down to the indigestibility of casein. To correct that you have to give a mixture which contains less casein; in other words, you have to proceed from the positive to the comparative degree of digestibility, and put the child temporarily upon condensed milk. In very severe cases you may require to give peptonized milk. You may also require, as in the case of breast-fed children, to correct constipation. That you should do in the way I have already described. 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 **carmina-**

tive 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 (2½ 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 mixture which is also alkaline ; for instance, aromatic spirits of ammonia, 2½ minims. Secondly, to make this pleasant, you will add a little glycerine (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 glycerine. 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 very 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

opium in some shape. 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 glycerine and some carminative water, makes a very suitable opiate for a young child.

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 recognise 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 recognising 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. Many of the acute specific fevers in children 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. 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 veal broth. The liquid, whatever it may be, should be given cold with a teaspoon.

You will find, also, a 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 comparatively pleasant 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

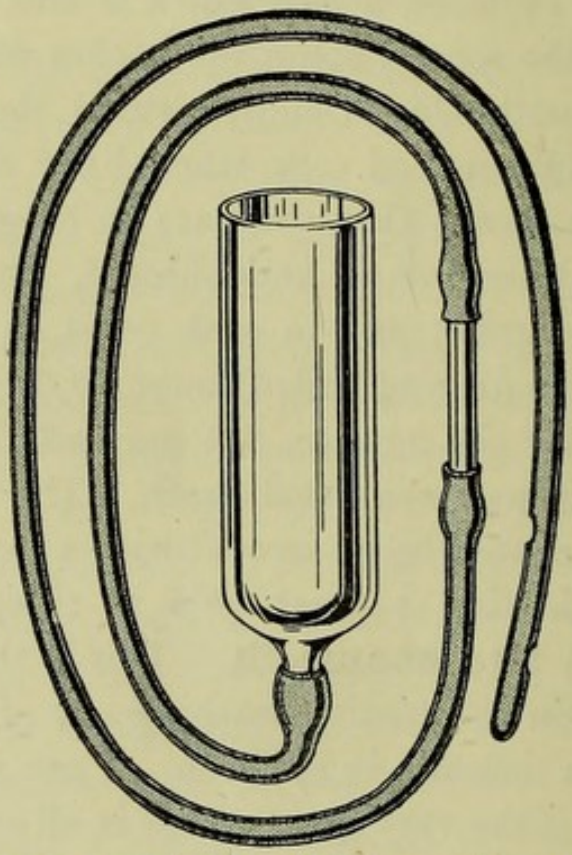


Diagram 8.—Stomach-Washing Apparatus.

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 you want to. Sometimes the eyelet of the œsophageal tube gets blocked with a mass of casein, and it is then an advantage to be able to put your mouth over the top of the funnel and displace the casein by blowing.

By the same means you can produce a sort of shower-bath in the interior of the stomach, and by projecting the fluid with some force through the tube you can wash away the mucus and masses of casein which have accumulated there. It does not matter much what fluid is used in the washing out. I usually employ plain water. You can use it quite hot, remembering that it always tends to get cooler as it passes down. But if there is a very sour odour in the breath, I recommend you to add a little bicarbonate of soda —a teaspoonful to the pint—before the final washing. Having completed the washing, you will find it an advantage to leave an ounce or two of fluid behind, for this 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.

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 $\frac{1}{6}$ 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 10 grains, and even in a young infant you can safely give more than that.

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 at all unless it be 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 of 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.** Congenital narrowing of the pylorus is a condition which is not so very infrequent, but which you will be very apt to overlook. It is characterized by the fact that the vomiting begins at or soon after birth, *and by the coexistence of constipation.* On physical examination you will be able to make out that the stomach is dilated, and if you are lucky, or examine the child often enough, you will see a wave of peristalsis pass along the stomach from left to right. Sometimes you can even feel the thickened pylorus to the right of the middle line. I shall not discuss the pathology of this condition with you—and, indeed, it is much disputed—but as soon as you recognise or suspect its presence it is your duty to call in a surgeon, for whereas medical treatment will avail you but little in dealing with it, gastro-enterostomy often gives the most brilliant results.

Now, **how should you treat** that **chronic** form of **vomiting** which is the result of purely digestive

disorder ? The first thing is to regulate the feeding. Vomiting, if it is habitual, means that the child is getting a mixture which is too strong for it, and you must dilute the milk still further, or else give it in a more digestible form. And here, again, it will be extremely important to regulate the times of feeding, taking care that the bottle is not given too often. In these cases, also, you will find periodic washing out of the stomach to be of the very greatest service. You can employ it every day, or even oftener, with the greatest benefit. With regard to drugs, I would only say again that they must take quite a subsidiary place. It is only after you have corrected the feeding and washed out the stomach thoroughly that you should think of drugs at all. You will find bismuth one of the most useful if given in large doses. In other cases, again, 2 or 3 minims of dilute hydrochloric acid, with a little glycerine and some carminative water, will be advantageously prescribed. I cannot tell you beforehand which cases will do best with bismuth and which with hydrochloric acid. I think it is more or less an experiment in every case ; but one or other of these drugs will generally be very beneficial, supposing you have failed to relieve the child by adjusting the feeding and washing out the stomach.

LECTURE IV

THE DIGESTIVE DISORDERS OF INFANCY
(*continued*)

DIARRHŒA.

GENTLEMEN,—In this lecture I wish to discuss with you the third of the symptoms of digestive disorder which I mentioned at our last meeting—namely, *diarrhœa*. I cannot exaggerate the importance of this disease as a scourge of infant life. Recent returns of the Registrar-General show that out of 22,000 deaths from diarrhœa, taking the country all over, 17,000 occurred in children under one year old. That shows you the frequency of this disease, and to what a tremendous extent it destroys life. And you will naturally ask, Why are children so liable to it? I think there are three causes which render children particularly liable to suffer from diarrhœa. The first is that the sterilizing power of the child's stomach is very 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 children are fed mainly upon milk, which is anything but a germ-free fluid; and the third is that infants are particularly liable to chill, for the reasons

I have already mentioned. These are the chief causes which render children subject to diarrhœa, and against these you must fight if you wish to prevent it.

Many of the severest forms of diarrhœa in young infants are probably due to specific micro-organisms. Take what is called *epidemic diarrhœa*, which is so common in the summer. That is almost certainly due to a specific microbe. Recent attempts have been made in America to isolate it, and it is believed that it will prove to be identical, or almost identical, with the bacillus which is the cause of epidemic dysentery. The growth of these micro-organisms is favoured by certain quite definite conditions. If you trace the admissions of acute diarrhœa in such a hospital as this in the summer, you will find that gradually as the weather gets hotter the number of such cases increases, but you will also observe the curious fact that for some time after the thermometer has fallen the admissions still continue numerous; in other words, the curve of epidemic diarrhœa follows approximately the ordinary curve of the thermometer, but not quite accurately. And if you investigate the matter still further, you will find that what it does follow pretty accurately is not the temperature of the air, but the temperature of the soil. It is when the thermometer, immersed four feet in the soil, begins to record a temperature of about 56° F. that epidemic diarrhœa becomes most common. In other words, this organism, whatever it is, grows best in the soil about that temperature. Another cause which greatly favours the development of diarrhœa is the existence of bad surroundings—exhalations of all sorts, foul drains, contaminated soil, overcrowding, dirty cesspools, dung-heaps. All these things contribute greatly to the production of diarrhœa;

and the importance of these causes is so great that often in practice you will find it an advantage to remove a child, if he is suffering from diarrhœa, right away from his surroundings altogether, and in particular to keep him out of large towns. I remember, when in Boston, going to see the Babies' Hospital there, which is kept specially for cases of summer diarrhœa. It is situated on an island down at the mouth of the harbour, and it is found that cases taken out of the slums of the city and removed to a place like that, where the air and soil are pure, often recover at once, without any additional aid. Further, you will be able to do a great deal in the prevention of diarrhœa by seeing that the milk is boiled before the child gets it, at least in hot weather. You will remember I told you that the importance of boiling milk is to some extent exaggerated. I wish now to qualify that statement by saying that the importance of it cannot be exaggerated in hot weather. All milk given to children in the summer should be boiled.

The third way in which you will be able to do something to prevent diarrhœa is by guarding against chill—seeing that the child is properly clothed, and setting your face against that foolish practice termed 'hardening,' which some mothers will try to get you to favour in the case of their offspring.

By such means as these you will be able to do a great deal in the **prevention of diarrhœa.** But in spite of all you do there is no doubt you will have a great many such cases in your practice, and therefore we may now pass on to a closer study of this disease and of the treatment appropriate to it.

In classifying the **varieties of diarrhœa** met with in infants I shall proceed upon clinical rather than upon

strictly pathological lines, and I 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 be acute it may be simple—that is to say, it may be diarrhœa and nothing else; or it may be accompanied by pyrexia, when we speak of it as febrile; 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 diarrhœa, the familiar 'D. and V.' of the hospital receiving-room, is usually 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 or two about the **character of the motions** in different cases 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 without 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 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 they indicate that the child is getting more milk than he can digest. 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. Then there is a stool which you may describe as ' slimy '; that means a stool which contains mucus in excessive quantity. If you recognise excess of mucus you are justified in believing that the large intestine is particularly involved. You will remember from your histology that the large intestine is lined mainly by mucin-forming cells, whereas in the small intestine such cells are rather scanty. Therefore, you will be justified in thinking that it is the large bowel which is at fault if there is much mucus ; 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. Where the constitutional symptoms of diarrhœa are severe, you are justified in concluding that you are dealing with bacterial poisoning. 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, although that helps in their production, but they 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 mucus on the cornea. The skin becomes wrinkled, the child seeming, as it were, to shrink away from his skin, which becomes redundant; and when you pinch it up, instead of falling back again as it would normally, it is apt to remain in folds. When you get this condition, which is described as a want of elasticity in the skin, you can be certain you are always 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 you are dealing with a case in which there is an involvement of the kidneys, and in which 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 recognising this want of elasticity in the skin is based upon the fact that when you find it you should institute special means of treatment, which I shall describe to you 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 child. I need hardly say, however, that the

picture varies a great deal. In the simple form there will be little 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.** Here, gentlemen, I would wish to give you this piece of advice: Put not your faith in drugs. Your watchwords in the treatment of acute diarrhœa in an infant must be two — one is *starvation*, the other is *elimination*. If you bear in mind these two great indications, you will never go far wrong in the treatment of your case.

A word about each of those indications. You must **starve the child** because you do not wish to furnish any further pabulum for the growth of micro-organisms; and milk in particular you must withhold, for there is reason to believe that milk in such cases is actually poisonous. In a case of acute diarrhœa, whatever you do you must stop milk. With regard to elimination, your idea should be, in the first place, to remove as far as you can those organisms which are still growing in the alimentary canal, and, in the second place, to get rid of their poisons.

How are you to carry out those indications in practice? *Starvation* is easily carried out by withholding food; but, as I have told you before, infants only bear starvation well if you keep them warm, and at the same time supply them with plenty of liquid. Withhold all food for twenty-four hours if there is severe vomiting, and give sips of boiled water, or a weak decoction of barley-water. Or, if you want to make a pretence of giving some nourishment, give a weak veal broth or egg-white dissolved in

water, one or two teaspoonfuls at a time. Even in that case, however, I think the benefit is derived from the water. I think it is a matter of indifference whether you put anything in it or not. Still, as I say, you may wish to defer to the wishes of the parent in the matter of appearing to give something nourishing. If the case is not doing well you can continue starvation for two or even three days. As the symptoms abate you may begin to give a little nourishment in the shape of whey, but the thing which you should give last, and be most careful about giving, is the casein of milk, because that is what does harm more than anything else. You may add raw egg-white to the whey, and continue this mixture until the motions are satisfactory.

With regard to *elimination*, if there is vomiting, begin by **washing out the stomach** in the manner I described in my last lecture. If the diarrhœa be 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 as high as you can—you cannot pass it too high. 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.

In order to favour elimination you should have recourse in all severe diarrhœas to **subcutaneous injections** of fluid. In cases accompanied by collapse, especially in those in which the skin is inelastic, elimination is apt to be interfered with simply from the fall of blood-pressure, and in such patients you have to make up the volume of fluid in the circulation as quickly as possible. The best way of doing this is to give a subcutaneous injection of about 5 ounces of sterilized normal salt solution under the skin of the flank. This can be injected by means of a clean brass syringe connected with a large hypodermic needle by a piece of rubber tubing. It is very quickly absorbed, raises the blood-pressure, and helps to wash out poisons through the kidneys. The injection may be repeated every six hours if necessary. Now you cannot, of course, wash out the small intestine, where the micro-organisms are chiefly growing. In order to promote

elimination there you require to have recourse to drugs. In other words, paradoxical though it may seem, you must 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 grain or two of calomel. When that has acted you have done all you can to clear away the putrefactive material from the small bowel.

There are still some **special indications** to be met. If the skin is in the inelastic state I have described, you will find it advantageous to give the child a wet pack, and you will find benefit also from the administration of diuretics, especially sweet spirits of nitre. If the signs of collapse are great, you may require to have recourse to the mustard bath, which is perhaps the most powerful stimulant which we possess. I like also to administer camphor in preference to alcohol in such cases, either in the form of a solution in olive oil (1 in 15 or 1 in 30), of which 5 minims is injected subcutaneously, or in the form of spirit of camphor, 5 to 10 drops by the mouth.

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. 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.

I said you were not to put 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 treatment 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 which are supposed to prevent intestinal putrefaction. 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 very 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 very 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 ʒi.

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, you should 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, 10 grains; calomel, $\frac{1}{6}$ grain; pulv. ipec. co., $\frac{1}{6}$ grain. Such a powder given every four or six hours, as the case may require according to its severity, will often be of great service to you after the acuter stage of the illness has passed off. If the stools continue to be watery and offensive, you may require to have recourse to astringents of some sort. You will find in nitrate of silver a very efficient aid. I regard it as my last card in the treatment of diarrhœa, and keep it up my sleeve, as it were, until the end, and there are not many diarrhœas which will withstand it—$\frac{1}{8}$ to $\frac{1}{6}$ grain of nitrate of silver is usually sufficient for a young infant. It may be given with dilute nitric acid, 1 minim; glycerine, 5 minims; and distilled water, to 2 drachms. Such a dose, given three or four times in the day when the motions are very watery and offensive in the later phases of the illness, is often very useful.

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; you can kill children with it quite easily. The warnings of the text-books in this matter are quite 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 opium 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 type is due to an exaggerated excitability of the whole intestinal tract, and opium is the only agent which can be depended upon to lessen this excitability.

Two practical rules remain. One of these 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, as far as possible, you should avoid adding opium to other mixtures, 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 the other remedies.

The **treatment of chronic diarrhœa** I need hardly describe, because broadly it is conducted on the same general principles as that of an acute case. You do not require to starve such patients, but you select a suitable diet, prevent chill, and choose one of the drugs I have mentioned, according to the indications furnished by the stools.

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, or any diarrhœa such as I have been describing may end by passing into a condition of colitis and ulceration. One reason why this disease is of importance is that it is apt to be mistaken for another important disease—namely, intussusception. You will be apt to diagnose colitis as intussusception, and *vice versâ*, and that is a mistake which is fraught with serious consequences. For this reason I bring forward the consideration of ileo-colitis separately. How shall you recognise 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*. Mr. Barnard has pointed out a diagnostic sign of some value in distinguishing these 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 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.

LECTURE V

THE WASTING DISEASES OF INFANCY—MARASMUS AND CONGENITAL SYPHILIS

WASTING.

GENTLEMEN,—In this lecture I wish to consider with you *wasting* as a symptom of disease in infancy. When you go into practice it will occur to you almost every day to have a baby brought to you with the complaint that it is **wasting.**

The mother will tell you, in the common phrase, that it was 'a beautiful baby born,' but that either from birth or at some period subsequent to birth it began to waste. And very commonly you will find on inquiry that the child began to waste when artificial feeding was adopted. I would point out to you that wasting is not a disease in itself; it is, strictly speaking, a symptom, not a disease. So when a child is brought to you with this complaint it is your duty to try to find out to what the wasting in that particular case is due; and there are many possibilities which must present themselves to your mind.

The first is that **the feeding may be insufficient.** That is the first possible cause of wasting, and, of course, a most natural one. If the case be one of a breast-fed

child you may find on analysis of the milk that it is poor in fat, or that the total quantity secreted is small. If it be a bottle-fed child you will find that by increasing the strength of the mixture on which the child is fed it will start to gain weight. So there is no great difficulty in diagnosing wasting from mere insufficiency of nourishment.

Then you may have, as a second cause of wasting, **inability on the part of the child to suck.** That is a cause which you are more likely to overlook. The child may be unable to suck the breast or the bottle because of local conditions—I mean such things as stomatitis, an inflamed and painful condition of the mouth, which makes the movements of the tongue in sucking productive of acute pain. In such a case the child will cease to suck, and whilst the local condition lasts he will waste. But that, as you will see, is not likely to be a cause of chronic or prolonged wasting, because those local conditions in the mouth are, fortunately, usually of only short duration. Then, a child may be unable to suck because he is unable to breathe. I mean that if the nose is blocked up the child has only one channel left by which both to breathe and to feed, and in that case he is unable to breathe whilst sucking; and owing to that he is probably unable to get down a sufficiency of nourishment, and so again wasting occurs. Other local causes are such conditions as hare-lip or cleft-palate. I have known children waste severely on account of their inability to suck from the presence of a cleft-palate.

In the third place, I would remind you again of the possibility, in the case of bottle-fed children, that **the teat may be unsuitable.** I saw a child not long

ago, for instance, which was brought to me when a month old, and weighed only 6 pounds 7 ounces. At first I could find no obvious cause for the wasting. I made a little modification in the feeding, and it was brought a week later weighing 6 pounds 5 ounces. Still I could find no explanation for the loss of weight. The child came back a week later weighing 6 pounds 4 ounces. Then I looked at the teat, and found it was of the 'leech-bite' form, which, as I told you before, often prevents the milk coming through easily. I recommended that the bottle should have a new teat, which was done, and the child was brought back the next week weighing 6 pounds 10 ounces, and in a fortnight it weighed 7 pounds 12 ounces. That is an example of how real this cause of wasting may be, no matter how trivial it may appear.

In the fourth place, an infant may waste on account of **persistent indigestion**—that is to say, if a child is getting a mixture which is too strong for him, a mixture which is chemically quite sufficient to supply his nutritive requirements, but which is too strong for the digestive organs, that child will waste, with the ordinary symptoms which I described to you in the last lecture—namely, colic, vomiting, and probably occasional diarrhœa. So that the treatment of this form of wasting will resolve itself practically into treatment of the disorders I described to you when we last met.

A fifth cause, and one which you must never overlook, is the possibility of **the existence of organic disease.** I refer particularly to congenital syphilis, to latent tuberculosis in some form or another, and, though this is rarer, to the presence of an undiscovered empyema. All those forms of organic disease may be the cause of serious

wasting, and I shall consider them separately in a few minutes.

We come now to the last group of cases in which one finds wasting, those, namely, in which there is **no apparent or discoverable cause.** It is to that group, a comparatively small one, that the term 'marasmus,' or, as it is sometimes called, athrepsia, is strictly applicable. I do not think one has any right to talk of marasmus when one is referring simply to a case of insufficient feeding, or where one is dealing with persistent indigestion, or where there is an organic cause, such as those I mentioned in the fifth group. I think one should reserve the term **marasmus** for those cases where there is wasting for which one can find no sufficient explanation. I saw recently in the post-mortem room an autopsy upon a baby which belonged to this group, and the house physician who brought the organs to me said he was puzzled at being unable to find any evident cause for the child's death. That is exactly what one expects in those cases. You search the body thoroughly after death, but can find no reason at all for the wasting and fatal issue. Naturally, theories have been advanced to account for such an occurrence. There is one school who state that if you examine the alimentary canal histologically in those cases you will generally find it to be the seat of microscopic alterations. They assert that you will find maldevelopment of the lymph-follicles and Peyer's patches, and that you will find also atrophy and catarrh of the glands of Lieberkühn. On the other hand, it is denied strenuously by another school that these apparent microscopic findings have any significance at all. They say you will find them as commonly in children who are not wasted, simply as

a result of post-mortem change, and if you inflate the bowel of children who have died from other diseases you will find a similar alteration brought about purely mechanically. There is perhaps more to be said for those who maintain that this condition of apparently causeless wasting is not due to anything on this side of the alimentary canal at all, but that it is a tissue vice, a failure of assimilative power on the part of the tissues generally, perhaps on the part of the liver particularly. I confess that that sounds rather vague, and I admit it is vague, but I do not know of any way in which to put it more clearly. And such a condition is not unthinkable. You can find analogies for it in certain diseases of grown-up persons. There is the failure of assimilative power with regard to carbohydrates in diabetes; you know of a similar failure as regards fat in some cases of obesity, where the fat is stored up instead of being consumed. There is also the failure of assimilative power with regard to proteids in some forms of gout. And it is possible that some children come into the world with congenital inability on the part of their cells to assimilate nutriment properly; and the conditions under which many marasmic children are born would conduce to that congenital feebleness. They are often born at the end of large families, when the reproductive power may be assumed to be on the wane, or to be partially exhausted, and they have often been brought up under very unsuitable hygienic surroundings, and probably have been always badly fed.

So much for the pathology, if one may call it so, of this condition. The symptoms I need hardly describe to you. The chief one is persistent loss of weight, and in the group of cases which I am now particularly considering—those

to which the term 'marasmus' is strictly applicable—there need be no other symptom at all; no special disorder of digestion, no diarrhœa, and no vomiting. The child simply goes on losing weight, with varying rapidity; he becomes shrunken and emaciated, and finally presents that picture of extreme feebleness and atrophy which is so commonly seen in the out-patient room.

The **treatment of marasmus** you will find by no means satisfactory. The treatment of wasting due to many of those other causes I have mentioned is quite easy. It is easy to increase the amount of food, or to alter it. It is often easy to correct indigestion, or even to deal with some forms of organic disease, but it is not easy to improve the assimilative power in one of those marasmic children. You will find that you exhaust all your ingenuity in devising mixtures of artificial foods without any success; and, indeed, these are the cases which are brought to you with the history that they have had all sorts of foods without any benefit. If you can, you will probably find that the best thing to do with those marasmic children, assuming that they are not being fed from the breast of the mother, is to get them a wet nurse. They certainly do better on human milk than on anything else, and you will save a certain proportion of them by having recourse to that means. Failing that, you often have to use the most easily digested mixtures which you can find. For instance, peptonized milk may be used, and by extreme care in feeding, and by husbanding the child's strength at the same time—that is to say, by keeping him very warm, so that his expenditure of heat is comparatively small—you may sometimes be able to tide him on until the period when the first teeth are cut. Fortunately, about that period

the digestive power of such children often appears to undergo a sudden alteration for the better, and towards the sixth month they begin to be able to deal, not perhaps with milk, which in these cases often presents a great difficulty, but they begin to be able to digest starchy foods, and thrive upon them; and after that all the difficulties which you have been combating may disappear. But you have to be prepared for frequent disappointment, inasmuch as these children often go persistently downhill and die, no matter what you do for them. One peculiar symptom which you are likely to meet with towards the termination of these cases of marasmus is that the temperature, which has been previously normal, or perhaps subnormal, rushes up to a very high point. I have known it go up to 108° F., and even higher. This development of hyperpyrexia in the last hours of life is a thing which occurs not uncommonly, and you may perhaps think, on that account, that there will be signs of tuberculosis after death, but you do not necessarily find such signs at all. I do not know what it means, but it is always a symptom of grave significance. I have never known a case which developed it survive more than a few hours at the most.

CONGENITAL SYPHILIS.

I will now pass from the subject of marasmus to consider those two organic diseases, congenital syphilis and tuberculosis, which so often manifest themselves chiefly by the symptom of wasting. We shall take **congenital syphilis** first. I shall not deal at all exhaustively with it, and I shall not describe it as it occurs in older children.

I want merely to deal with congenital syphilis as it occurs in young infants, and particularly to tell you how you are to recognise it, because upon its recognition, I need hardly say, the proper treatment of that group of cases in which the wasting is induced by the existence of the syphilitic taint must always depend.

In the first place, if you suspect that congenital syphilis may be the cause of wasting in a case with which you have to deal, you must inquire carefully into the **family history.** I have previously told you that you must inquire carefully into the family history of all infants, but you must particularly do so in those whom you suspect to be the subjects of this disease. You will ask about the health of the previous children; about the number of still-births, if any; whether there have been any miscarriages; and whether the child before you was carried to term. You may get a positive history which is extremely suspicious. You may have a narration of cases of still-birth or repeated miscarriages, or the present child may have been prematurely born. But you must be prepared every now and then to hear, in a case in which you may conclude on other grounds that the child is the subject of congenital syphilis, that the family history is good. In other words, you do not always get a suspicious family history, and you must not be led off the scent by its absence. What, then, are the **signs** by which you will recognise the existence of the syphilitic dyscrasia in a young child? You will often, by a mere casual inspection, be able to decide almost at once that the child is the subject of syphilis. There is the characteristic facies which has been often described. The child has a shrunken or wizened up appearance. The skin has a curious earthy

tint, which has been compared to that of a mixture of coffee and milk, and the nose often shows a depression of the bridge. There is commonly also an absence or a scantiness of the eyebrows. When you look more carefully at the head you will find frequently, although by no means always, a peculiarity of the hair to which I want to direct your special attention. This peculiarity of the hair has never, so far as I know, been described in books on this subject, but you will often find it of very real service in diagnosing congenital syphilis. You will observe in many of these children that the hair is unusually long, that it is straight, fine in texture, and usually dark in colour—a combination of things which one is in the habit of describing as the **'syphilitic wig.'** The existence of this as a sign of congenital syphilis in early life was pointed out to me some years ago by Dr. W. S. Colman, and I have frequently found it of the very greatest value in diagnosing early cases of congenital syphilis. I shall show you immediately lantern slides illustrating examples of the syphilitic wig, in order that you may be able to recognise it afterwards.

You will find **signs in the bones** in many cases also; a bossing of the fronto-parietal eminences is a common, but not an invariable, sign. The existence of those areas of softening behind the ears and along the occipital suture, to which the term 'cranio-tabes' is applied, is frequently to be made out. You will often find also that there is a slight degree of hydrocephalus, and that the veins on the scalp are prominent and distended. Along with that you may observe a curious ridging of the edges of the sutures, and a slight thickening round the margin of the anterior fontanelle, the bone being, as it were, slightly heaped up.



FIG. 3.
THE "SYPHILITIC WIG."

I do not say that that heaping up is invariably due to congenital syphilis; but, like head-bossing and cranio-tabes, I think it is more commonly due to that than to anything else. Then, passing down over the child, you will frequently find the existence of an **eruption;** but here, again, not necessarily always. Nor is the eruption of congenital syphilitic disease always easy to recognise. It occurs, as you probably know, more commonly about the nates than anywhere else. But you will remember that in that region also eruptions of the nature of intertrigo, due to irritation by the stools or by the urine, are not by any means uncommon, and you will not always find it easy to tell when you are dealing with a syphilitic eruption and when you are dealing with one which is due to this irritation. But there are certain points which will help you. One is the colour of the eruption. In congenital syphilis it has that coppery tint which is so characteristic of syphilitic skin lesions wherever found. You will find also that whereas an intertrigo rash appears only on the opposed surfaces, those which touch one another, a syphilitic eruption tends to wander down over the limbs, perhaps over the calves, and tends to appear on the soles of the feet or the palms of the hands. And when it appears in these situations it often has a silky or glistening look.

Snuffling is another common sign, as you know, but I would warn you not to attach too much importance to it. Snuffling and the presence of an eruption are signs which are apt to be overrated as a means of diagnosing syphilis, for those two signs in children are not by any means always due to that disease. Moreover, many syphilitic children do not have snuffling at all. Other

causes of it are the presence of a mere cold in the head, or the existence of adenoids. I shall have a good deal to say in a subsequent lecture upon the subject of adenoids in children; and I only want to point out now that adenoids do occur in infancy, and that they may give rise to snuffling and discharge from the nostrils which you may mistake for the signs of syphilis. A cold in the head you should not mistake for it, because the cold is usually of shorter duration, whereas the snuffling of congenital syphilis lasts for some time. The snuffling of syphilis does not begin immediately after birth; there is usually an interval of at least two or three weeks, and it is always accompanied, sooner or later, by the discharge of a yellowish irritating fluid from the nose.

There are certain other symptoms which you may look for. One is the existence of thickenings of the phalanges —what is called **syphilitic dactylitis.** You will find that of help in the diagnosis in a few cases; and I have also known cases recognised by the existence of gummatous deposits, particularly gummatous epididymitis, which may occur in quite young infants. You will perhaps get some help from an examination of the viscera. You may possibly find splenic enlargement. This occurs in about 50 per cent. of cases of congenital syphilis, but you must not expect to find it great in degree. A spleen which has been enlarged by congenital syphilis is not often very big; it is palpable, but usually not more than that. An affection which also occurs sometimes in this disease is an inflammation of the ends of the long bones, **syphilitic epiphysitis.** I have seen recently two cases in the out-patient room, one of which was shown at a meeting of our Medical Society. It is an affection of the ends of the



FIG. 4.
CONGENITAL SYPHILIS, SHOWING SCARRING ABOUT MOUTH.



FIG. 5.
FACIES OF CONGENITAL SYPHILIS.

bones at the point where ossification is going on, leading to enlargement and tenderness; and on account of the tenderness there is a loss of ability to use the limb, sometimes spoken of as syphilitic pseudo-paralysis, so called because it is not a genuine paralysis, but an inability to make use of the limb from the existence of pain. I shall have more to say about that when I talk about paralysis in infancy generally.

Last, I would mention a symptom which has been emphasized by Dr. Eustace Smith, and which one sometimes finds of use in diagnosis, and that is **restlessness at night.** Syphilitic children, for some reason or other, are apt to be wakeful and restless at night, and to cry a great deal at that time. Dr. Smith suggests that this may be due to the existence of periosteal pains, just as there are often pains in the bones of adults who suffer from syphilis. At all events, you will sometimes find that this symptom will put you on the right track, and enable you to diagnose the existence of a syphilitic taint.

When all is said and done, and you have all these possible signs before you, there will still remain a residuum of cases in which you must be in doubt, because there are many children who waste merely because they have a slight syphilitic taint; they are not, strictly speaking, sufferers from congenital syphilis, but there is a syphilitic strain in the family, and there is enough of the poison in them to affect their nutrition adversely; and it is in these cases that one often has got to be guided in the diagnosis by the results of treatment, by giving antisyphilitic remedies, and seeing what happens. You will remember I showed you some charts of wasting infants, in whom the administration of gray powder at once caused a gain in

weight, and in a certain proportion of these cases that result is probably due to the existence of a congenital syphilitic taint which one has overlooked. I told you that it is not invariably due to that. I think one sees gray powder cause a gain in weight in cases in which one has every reason to exclude syphilis; but in some of the cases the explanation of such a rise in weight may be that the child has been slightly affected by this disease.

If you have satisfied yourself that wasting is due to congenital syphilis, there is a definite line of **treatment** marked out. You have to treat the syphilis by mercury, and the best way of doing so is by the mouth, giving ½ grain of gray powder in this way four times a day. Begin with ½ grain in the morning and ½ grain at night, until you have got up to that dose four times a day. It is important to recognise signs of overdosage. How shall you know when you are giving too much mercury? You know that you can recognise overdosage in adults by the occurrence of salivation; but infants never salivate with mercury. I do not know why that is, but it is probably due to the fact that the salivary glands do not develop well until the teeth are cut. At all events, salivation is not to be looked for when giving mercury to young children. The great sign in infants is the appearance of diarrhœa. So if during your treatment with mercury diarrhœa develops, remember it may be due to the giving of too large doses.

How long shall you continue to give mercury? That is a question which often presents real difficulties in practice. Many people advise you to stop giving mercury when all the symptoms have disappeared. I think those who recommend you to continue

the treatment for nearly a year, in at least moderate doses, are probably better advised. There is a danger in stopping too soon. Over and over again it has been my experience to stop mercury when everything seemed to be going on well, and to have the child brought back a month or so later with definite signs of syphilis again. Of course, you often find it difficult to persuade mothers to allow treatment to be carried on. The tendency in hospital practice is for patients to cease attending when the symptoms have disappeared, so you must impress upon parents at the outset the necessity for prolonged treatment. Sometimes you will find it difficult to give mercury by the mouth ; there are some children in whom even quite small doses produce diarrhœa, and in these cases inunction into the skin is a good method of treatment. I generally use blue ointment, about ½ drachm of it being rubbed into the abdomen once, or at most twice, in the day, after which the skin is covered by a flannel binder, so that gradual absorption of the ointment goes on. You will find syphilitic children tend to suffer from a sort of cachexia and from anæmia, and if that is a prominent symptom it is advisable to give cod-liver oil and iron as well as mercury. Indeed, there is reason to suppose that the prolonged administration of mercury is in itself apt to produce anæmia, and that is probably why many people advise its early discontinuance. So when you go on giving mercury after the symptoms have disappeared, you may find it well to give iron and cod-liver oil in addition.

What about the **treatment of local lesions ?** I think you should treat the snuffles by syringing or brushing out the nose with a solution of borax or of corrosive sublimate (1 in 2,000), and putting a weak mercurial

ointment up the nostrils by means of a feather. That prevents the crusts from drying and the nose from becoming blocked up. I usually employ weak white precipitate ointment for the purpose. The other chief local symptom which one has to treat is the eruption, and that you can do either by weak corrosive sublimate baths or by dusting powders containing calomel. Sometimes you may use a combination of weak white precipitate ointment and zinc ointment.

LECTURE VI

TUBERCULOSIS IN CHILDHOOD

GENTLEMEN,—In the last lecture we were considering wasting as a sign of disease in infancy, and we saw that there were several morbid conditions which may be associated with it as one of the chief symptoms. I enumerated to you several possible causes of wasting in children which you must consider when a case comes before you, and I dwelt particularly upon congenital syphilis as one of the organic diseases which may be a cause—sometimes an unsuspected one—of that condition. Now, in this lecture I wish to direct your attention to the other great organic disease which may be a cause of wasting in children, both in infancy and in later childhood. I mean tuberculosis.

The **frequency** of this disease in children will be realized from this fact: that careful hospital statistics, taken in various parts of the world, show that about one-third of all the children who die in hospital die of tuberculosis in some form or another; and, further, in about 12 per cent. of the remaining cases tubercle is present in a latent form. If you examine the bodies of those children after death, you will find that the tuberculosis tends to attack one particular set of tissues with a very striking degree of

regularity. Those are the tissues which are concerned with the formation of the blood. Ninety per cent. of all the cases show an affection of the lymphatic system or the bones. The lymphatic system, you will remember, is engaged in the formation of one variety of white blood corpuscles, and the bones are quite as much blood-forming organs as they are supporting tissues. And so when one says that the bones are affected, one really means that a blood-forming organ is affected. And I want to impress upon you this fact, that it is this particular set of tissues which, more than any others, tends to be involved in tuberculosis in early life. If you look at the diagram on the wall, which shows the age distribution of 500 consecutive cases of tuberculosis in children, taken from the statistics of the Great Ormond Street Children's Hospital by Dr. Still, you will see that tubercle attains its maximum frequency in the second year of life. And I want you to correlate in your minds those two facts—first, that it is the blood-forming tissues that are specially affected by tubercle; and, secondly, that it is in the second year of life that tubercle attains its maximum frequency—because I think there is a relationship between them which is not an accidental one. I think it is probably owing to the blood-forming organs being in a very active state of vitality at this period that explains their liability to tuberculosis. When I come to speak of the anæmias of infant life I shall have occasion to point out that the blood of children undergoes a very important transformation in character at just about this time, towards the end of the second year. It is about that time that the tissues which form the white blood corpuscles come to attain their greatest degree of activity. After that the lymph-forming organs gradually

pass into the condition in which they are found in the adult.

Of more importance for us, however, is it to study how the tubercle bacillus most commonly finds access to the body in childhood—*i.e.*, its **path of infection.** It is all very well to know that the tissues most involved are the lymphatic or blood-forming organs ; but what we specially want to know is how the bacillus gets there at all. Of a series of 216 cases which were carefully studied from this point of view by Dr. Still, it was found that the access of the bacillus had been through the lungs in 63·8 per cent., through the intestine in 29·1 per cent., and through the middle ear in about fifteen cases. These statistics were derived from the Great Ormond Street Children's Hospital, but there are similar statistics from other children's hospitals. They all tend to show the same thing—namely, that the lung is by far the commonest path of infection. You will see at once what the significance of that is. It means that the talk about the milk of tuberculous cows being the chief cause of the infection of children with tubercle is certainly wrong, and it also means that dust must be regarded as the main source of infection in the child, just as it is in grown-up people. The tubercle bacilli are conveyed into the lungs, probably mixed up with dust, and thence they find their way into the lymphatics. When one speaks of the lung in this connection it must be remembered that the chief part affected is the bronchial glands at the root of the lung. But these are not likely to be affected if they are perfectly healthy. Unless there be a great inherited predisposition to tubercle the healthy gland may be assumed to be able to deal with such tubercle bacilli as find access to it ; otherwise all of

us would be suffering from tuberculosis. And the reason why the bronchial glands in children become so frequently affected no doubt is that they are very often not in a healthy condition; they have already been the seat of inflammation. How do they become inflamed? They become inflamed secondarily to catarrh of the bronchi or alveoli. In other words, the bronchial glands become inflamed during an attack of bronchitis or broncho-pneumonia. The bronchitis or broncho-pneumonia is recovered from, but the glands are left in an unhealthy condition, dust containing tuberculous material is inhaled, and the bacilli start to grow in the glands and set up a caseous degeneration of them, which is the starting-point of tuberculosis. So I want to impress upon you, as the great predisposing cause of tubercle in children, that group of diseases which are accompanied by catarrhal processes in the bronchi and lungs, particularly such diseases as measles and whooping-cough. And you will constantly find in your practice that children will be brought to you for wasting and some vague complaint of ill-health. You inquire into the past health, and find that they have had an attack of measles some months or weeks before, and have not been well since. Then when you come to make a physical examination, you will find that they are the subjects of tuberculosis. That is a very common history.

Now, no matter by which path the bacillus finds access, no matter whether it is through the bronchial glands or through the intestines, affecting the mesenteric glands, or through the middle ear, it sooner or later **generalizes** itself. And that is the other point I want specially to emphasize—that no matter what the primary path may be by which the child becomes infected, the disease has a

great tendency to become widespread, a much greater tendency than is the case in grown-up persons. What usually happens is that one of these caseous bronchial glands softens ; it ulcerates its way into a bronchus, and the caseous material gets aspirated into the lungs, and so pulmonary tuberculosis is set up. In other cases perhaps the infection gets conveyed by ulceration into a blood-vessel, and is disseminated in that way through the body. At all events, in whatever way dissemination occurs, it takes place very quickly. That is why it is so important for you as practitioners to diagnose tuberculosis in children as early as possible, because, unless you recognise it early and treat it promptly, it will certainly before long become generalized, and then it may be too late to treat it at all.

That leads us naturally to consider how you are to **diagnose the existence of tuberculosis** in children. I shall consider first of all the diagnosis of *general tuberculosis*. We will assume that the process has already become generalized. How are you to know, then, that the wasting in this particular case is due to general tuberculosis or not ? Often, I believe, it is not possible for you to know at all. It is not possible to know until the post-mortem in many cases. The diagnosis of general tuberculosis in children is extremely difficult, and it is so for this reason : that the process kills the child before the tubercles have had a chance of breaking down. You know that unbroken-down tubercles do not give rise to physical signs. You may have a lung stuffed with miliary tubercles, but until they soften and break down you have no means of telling that they are there, and it is on this account chiefly that the diagnosis is often so uncertain. There are, however, certain symptoms and signs which

should make you suspicious. One of these suspicious symptoms is the occurrence of irregular pyrexia along with wasting, for which you can find no other cause. Again, if there is much apparent illness and fretfulness on the part of the child, without obvious or apparent reason, you should be suspicious that it is a case of tubercle. But there is no other symptom to help you much. You will easily see that the danger you run is of attributing those symptoms to, perhaps, a slight dyspepsia, or catarrh of the stomach or intestine ; or, perhaps, to a slight bronchial catarrh, or to some trivial condition of that sort which may be present along with the tuberculosis, and not to tuberculosis itself. It is so extremely difficult to assign its proper value to each of these symptoms that you have often to suspend your judgment.

Amongst the **signs** which may give you some assistance in the diagnosis of tuberculosis are certain appearances in the child—appearances which clinical experience has shown to be often associated with tubercle. Tuberculosis is known to be commoner in children of a certain type. You know the fragile, fairy-like type which is supposed to be particularly marked out for tuberculosis. That is an extreme example of what I mean. In young babies, however, that type is not so noticeable, and you have to go by such minor indications as long eyelashes, or the presence of an excess of downy hair, especially on the back between the scapulæ. There is also, which you will find of value, a tendency to the appearance of blotches of erythema upon the skin on very slight pressure or irritation. These signs may perhaps seem to you trivial, but very often, taken together and along with the family history and the general symptoms, they may suffice to

put you on the right track. Of course, you will look also for signs indicating the starting-point of the process. First of all you will look for signs of the disease having started in the lungs. I shall describe to you immediately what the signs associated with tubercle of the bronchial glands are; but I shall only say here that in this case again you have great difficulty in coming to a right judgment, because, admitted that you get moist sounds over the lungs in a case which you suspect may be one of tuberculosis, you are still in doubt as to whether that is due to breaking-down of tubercles or not, because there is nothing commoner in marasmic children than for them to suffer from a slight attack of hypostatic pneumonia as they lie in bed, and that may easily give you moist sounds over the back. So it is only when you get signs of breaking-down on the anterior aspects of the lungs that you should attach much importance to them.

I next pass to the consideration of the physical signs associated with **tuberculosis of the bronchial glands;** in other words, the signs which you find when the tubercle finds access, as it usually does, by means of the lungs, and when it has not yet become generalized. Here, again, there is no quite conclusive symptom. There will be more or less wasting, and perhaps there will be irregular pyrexia. There will also be general fretfulness and illness. But the only local symptom of any value is the occurrence of a spasmodic cough. Children with enlarged bronchial glands often suffer from such a cough, which you will be apt to mistake for whooping-cough, which it may simulate very closely. Many cases of whooping-cough which are supposed to drag on for months are probably cases which may have begun in whooping-cough. but which are now

suffering from secondary enlargement of bronchial glands. Note, then, that one point which will help you is the existence of this spasmodic cough. When you come to the physical signs you will find that they are only present in a minority of the cases, and you have no difficulty in seeing why that should be. The bronchial glands are not very large; they lie deep down in the mediastinum, and it requires a great deal of enlargement of them to produce signs which you can recognise with certainty. So you must be prepared to find that in cases in which after death you are able to recognise a considerable degree of enlargement of the bronchial glands there were no signs of them during life. Still, in some cases, when the enlargement is very great, it will reach the surface, and the chief sign in such a case is dulness behind the manubrium sterni—dulness which extends outwards towards the apices of the lungs and downwards towards the heart. I would warn you, however, of one possible fallacy here—namely, that enlargement of the thymus in children may exactly simulate this sign. There is no way of discriminating exactly between the two. Another sign which you will find is distension of the veins in the neck. This is due to the fact that the enlarged bronchial glands tend to press upon the superior vena cava. Further, owing also to such pressure upon the veins, there may be a bruit heard on listening over the manubrium. Dr. Eustace Smith has pointed out that this is best heard if the child stretches its head back, which causes the superior vena cava to be pressed upon by the mass of glands. But I do not think you should attach too much importance to that sign. One certainly hears a bruit of that sort in many cases in which there is no reason to suppose that the bronchial glands are en-

larged. On the other hand, its absence is an indication of some value, which should make you suspicious that you are not dealing with such enlargement. I am afraid these are the only signs which one can give you as indicative of enlargement of the bronchial glands, and you will see that, taken altogether, they are not of very great value. What I wish to impress upon you by all this is the great difficulty which you will have in making up your mind about either bronchial tubercle or general tuberculosis; and you must often remain in doubt, and must not blame yourselves too readily if, after death, you find extensive tuberculosis when you have been regarding the case as one of ordinary wasting.

The next form of the disease which we come to is much more hopeful, both as to diagnosis and treatment, namely, **tuberculosis in the abdomen**—those cases in which the bacillus has gained access through the alimentary canal into the intestinal glands and peritoneum. Abdominal tubercle differs so much from tubercle elsewhere in children—and the same is true of grown-up people—that one is tempted to suppose it is a different disease; and it has recently been suggested that it may prove to be bovine tuberculosis in man, and therefore a milder form of a similar pathological process. If that were established, it would at least explain the very marked clinical differences between abdominal and other forms of tuberculosis. When we come to consider prognosis and treatment, you will find that abdominal tuberculosis is much more curable than any other form; indeed, one takes quite a cheerful view of it, because a large proportion of cases get perfectly well.

Abdominal tuberculosis resembles the other forms in

being most common in the second year of life. The statistics of a number of cases of fatal tuberculous peritonitis in children were collected by Dr. Still when he was Registrar of the Great Ormond Street Children's Hospital. He found that the maximum frequency of death occurs in the second year. That is against the milk theory. Seeing that children live almost exclusively on milk up to the first year of life, one would expect abdominal tuberculosis to be common in the first year if milk were the main source of infection. Not only does abdominal tuberculosis occur most commonly in the second year of life, but at that particular time the abdominal disease is usually only part of a general tuberculosis. In young infants it is not purely abdominal tubercle which you have to deal with. You may think when you examine the case that it is abdominal tubercle only, but you will almost certainly find that there are tubercular deposits elsewhere. That is only another way of saying that tuberculosis in young infants, no matter where it starts, tends rapidly to become generalized, and if it starts in the abdomen it ends by involving the lungs or the meninges of the brain; and what you will find constantly happen in these cases is that the case drags on until of a sudden it flares up into tuberculous meningitis, which is followed by death.

There are two **varieties of abdominal tuberculosis.** There is the plastic form and the ascitic form. Most of you must be quite familiar with those two varieties. The plastic form consists in an enlargement of the glands and a matting together of the coils of intestine with tubercular deposits in the peritoneum and mesentery. The ascitic form is that in which an effusion of serous fluid takes place into the peritoneal sac.

To the first or plastic form the term **tabes mesenterica** is sometimes applied, but I think quite erroneously. I strongly advise you to discontinue the use of that term altogether. For my own part, I have hardly ever seen a case of true tabes mesenterica. It is a disease which, if it occurs at all, must do so with extreme rarity. Rightly used, the term 'tabes mesenterica' means an enlargement of abdominal glands, with caseation, and nothing else. That may occur, and probably does occur sometimes, but it is very unusual. It will certainly conduce to clearness if we talk merely of the plastic type of abdominal tuberculosis. This type is commoner than the ascitic. Some people say that it is ten times commoner, but my own observation would not corroborate that, I think. In older children at least the ascitic form is certainly commoner than that; it is much more than one-tenth of the other. You will recognise this form by the general signs of tubercle, the wasting, pyrexia, and so on. The local signs are hardness, with nodulation, in the abdomen. You may take it as a broad general rule that if you find nodules in the abdomen of a child, if you can exclude fæcal masses, the probabilities are enormously in favour of those nodules being tuberculous, no matter where in the abdomen they are situated, and no matter what they feel like. Tubercle is so very common compared with anything else that you do wisely to regard every mass in a child's abdomen, where you can exclude a fæcal accumulation, as being a tuberculous deposit of some sort. You can easily exclude or confirm a fæcal accumulation by the administration of purgatives and enemata, and if you are dealing with a doubtful case of abdominal tuberculosis you should take that precaution,

seeing especially that the large bowel is thoroughly emptied. Then examine the abdomen again, if need be under an anæsthetic, and see if you can still discover the lumps. They are due partly to caseous deposits in the mesentery and elsewhere, partly to glandular enlargement, and partly to coils of bowel bound together by adhesions, and are the main physical sign of the plastic form of abdominal tuberculosis.

The **ascitic form** you recognise by the presence of fluid. Is the ascitic form of tuberculosis ever simulated by any other peritoneal effusion? In other words, may there be clear fluid in the abdomen of an infant which is not due to tubercle? Of course, such effusions may sometimes occur in the course of cirrhosis of the liver, and you may find a difficulty in excluding the presence of that disease. Not long ago I had the case of a little girl in whom that problem came before me in a very acute form. She looked the picture of robust health, but her abdomen was full of fluid, and the liver was about two fingers' breadths down. Its edge was also very hard. It was thought to be a case of cirrhosis, and that it would be well to treat the case from that point of view. However, we watched the patient, and she developed a temperature, and then we began to be suspicious, and thought it would be better to operate, because if it were tubercle it would be the right thing to open the abdomen; and even if it were ascites due to cirrhosis, operation would not do any harm. On the day decided upon for operation she suddenly became comatose, developed all the signs of tuberculous meningitis, and died; and, post-mortem, the abdomen was found to be the seat of very generalized tuberculosis. Such a case may cause you difficulty; but

I would again remind you of the great frequency of tuberculosis and the comparative rarity of cirrhosis, so it will be safer when in doubt to diagnose tubercle. Clear effusions into the abdomen due to simple chronic peritonitis have also been described, but they are very rare. They are said to occur after measles or scarlet fever, or after injury. Many good observers, however, are of opinion that all clear effusions of an inflammatory sort into the abdomen in children should be regarded as tuberculous.

Both forms of abdominal tubercle are commonly accompanied by **ulceration of the intestine,** according to some statistics in as many as 70 per cent. of all the cases. Ulceration does not necessarily show itself by clinical symptoms. It does not necessarily cause diarrhœa. But where ulceration becomes extreme and extensive you will find it tends to be accompanied by an extreme degree of anæmia—a degree which may almost resemble pernicious anæmia in its intensity.

We turn now to the **general treatment of tuberculosis;** but one may first say a word about prevention, which, of course, is more important than cure. I need not remind you of the general hygienic rules which should be observed in the rearing of children: attention to proper feeding, proper exercise, proper ventilation of the sleeping rooms, and all those other commonplaces which even lay people now recognise. But I would direct your special attention to the question of dust, because if dust is the chief cause of infection, as statistics show it to be, then dust must be one of the chief things to fight against. And I think there is no doubt that, just as in the last century sanitary reformers fought against a bad

water-supply and bad drains, so in the early part of this century they will have to make a fight against dust. 'Down with dust!' must be the motto of those who would prevent tuberculosis either in children or grown-up persons. There is here a boundless field for the energies of the reformer. Another point is this: seeing that dust infects through the bronchial glands, what you have to do if you want to prevent children becoming tuberculous is to keep their bronchial glands healthy. To do that you have to treat every case of catarrh of the lungs or in the bronchi seriously. You must not think that even a slight bronchitis is a trivial thing, nor yet a little attack of broncho-pneumonia. So far as you can you must see that convalescence is made thorough and complete; that there is not left behind a little wheezing, or a few crepitations at one base. You must try to restore the child to complete health. As it is certainly possible for the abdominal form of tuberculosis to be conveyed by the milk of tuberculous cows, you should, of course—particularly in the case of children who are predisposed by heredity or by surroundings to become infected with tubercle—see that the milk which they get is at all events boiled. I do not say it should necessarily be sterilized, but it should be boiled.

With regard to the **treatment of different forms** of tuberculosis, I need hardly say there is no special treatment for the generalized type. Whether general tuberculosis is ever recovered from at all it would be very hard to say. Such a high authority in the matter as Sir William Jenner was of opinion that general tuberculosis is recovered from in children much more commonly than is supposed. It depends a good deal upon what you mean

by generalized. No doubt deposits of tubercle in the lungs and abdominal organs may apparently be recovered from ; and years afterwards you may find evidence of it in calcification of the glands. But once tubercle has become really generalized, I think anything which you can do in the way of treatment is not likely to influence the process much. The same is true of tubercle of the bronchial glands, which, merely from its situation, it is difficult to treat, for you cannot get at the bronchial glands. Not even the modern surgeon has yet attempted to excise them. For practical purposes, therefore, your treatment in both general and bronchial tuberculosis must resolve itself into simply putting the child into the best possible hygienic conditions, in the hope that the natural powers of resistance may be able to bring the disease to a favourable termination. In a case of **abdominal tuberculosis,** on the other hand, you can do a great deal by treatment. There are no cases of tuberculosis which you approach with more hopefulness than those of abdominal tubercle in children ; and I should say that the prognosis will be better in older children than in younger, for the reason that in young infants tubercle generalizes so fast that you do not often have to deal with the local disease only. In older children generalization occurs more slowly, so that you have a chance of getting it in time, and treating it successfully. 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 be taken to a place like Margate, and spend their time by the seashore in a wheeled carriage, lying down. The abdomen should be supported by a broad and firm binder. You will find it an advantage to apply

iodoform ointment locally; it should be rubbed well into the skin of the abdomen. I also often use, and I think with advantage, liniment of mercury, ½ ounce or so being rubbed in night and morning all over the abdomen. 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 be 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 the question whether you are to continue medical treatment, or whether you are to have recourse to surgical aid. I do not think it is often advisable to have recourse to surgical treatment in those cases right away. I think the necessity for **surgical treatment** in abdominal tuberculosis in children is very much exaggerated. Taking the last six cases of purely abdominal tuberculosis which have been under my care simply as they turned up in the out-patient room, and all between three and nine years of age, I had the abdomen opened in only one of them, and that child recovered, although it suffered for a time from fistula of the umbilicus. But the other five children all recovered completely without operation at all. So you see medical treatment is of very considerable use. By recovery I mean that, at the end of two years, some of them now at the end of three years, they are apparently

in perfect health. You should speak cautiously of recovery from abdominal tuberculosis. I do not think one has a right to say that a case is completely cured simply because after a few months the symptoms have not recurred. The symptoms in this disease are 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 for the time being lowered, the process flares up again. Seeing, then, that medical treatment gives such good results, what should guide you in having recourse to operation? In the first place, all cases of abdominal tuberculosis should get the benefit of operation if fever and continuous wasting persist in spite of the medical treatment I have sketched out for you. The continuance of such symptoms means that medical treatment is not going to accomplish much, and it is well to give the patient the benefit of operation, although it must be confessed that many such cases, perhaps the majority, do not get well under surgical treatment either. Further, you will always be biassed in the direction of operation if you are dealing with the ascitic form as opposed to the plastic, because the ascitic cases certainly do better under operation than those in which the disease is mainly in the glands or the peritoneum. Numerous statistics bear out this statement. For instance, the records of the general hospital in Heidelberg, embracing cases at all ages, and not only in children, show that of forty-one persons operated upon for the ascitic form of tubercle, twenty were completely cured—that is to say, about 50 per cent. Of nineteen cases of the other form, the plastic, only about four could be described as cured. So you see the ascitic form certainly offers a better chance

than the other of successful treatment by means of operation. On the other hand, you must remember that the total statistical results of surgical operation are not so much better than the medical statistics if you take them all round, and so it is as well to give every case the benefit of prolonged medical treatment first, especially if there be any objection to operation on the part of the parents. Operation in the ascitic form, properly performed, is almost perfectly safe. In operation on the plastic cases there is a greater risk. Every now and again in such cases, owing to the presence of adhesions, the intestine has been opened into by the operator, and sometimes the bladder also. I have seen the intestine and the bladder both opened into at the same operation. So you will see the procedure is not devoid of danger. Still, if medical treatment fails in such a case, I think it is right that you should have recourse to surgery.

LECTURE VII

RICKETS

GENTLEMEN,—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; indeed, I suppose there is no place where rickets is commoner than in Berlin. But it is called the 'English disease' 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. Whether rickets has increased much since thar time it is a little difficult to say; but I think one can be pretty sure that it has increased, because all those factors which tend to predispose to rickets are such as are daily becoming

more pronounced. One of those factors is that inability on the part of women to nurse their own children which I have already deplored in a previous lecture. Another is the increase in women's work. With the advent of the industrial epoch in the middle of last century, and the establishment of factories, there opened up at once a great field for the labour of women. This tempted them away from their domestic duties and from the personal care of their children. That state of things persists, and in many of the large industrial towns rickets is extremely prevalent, for the reason that women go to work in the factories, and their children are left at home badly looked after and improperly fed. A third factor in the production of rickets is found in the tendency for the population to be crowded together in large cities, which is such a striking feature of social life at the present day. Now, so potent are these factors, and so prevalent is rickets in consequence of them, that in many towns more than half the children seen in the out-patient room exhibit the disease in a more or less marked degree. So that the dictum pronounced by Sir William Jenner a good many years ago, when he first began to work at Great Ormond Street, still holds good—namely, that 'rickets is the most common, and, in its indirect results, the most fatal of all the diseases which peculiarly affect children.'

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 he 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 symptoms 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. You would have a totally erroneous conception of rickets as a disease if you were to imagine that it consists solely in the production of osseous deformities. You must remember that there is a visceral side to the affection as well, which, so far as effects upon life are concerned, is more important than the mere deformities due to alterations in the bones. 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 in consequence you can observe, clinically, enlargement of the epiphyses. I show you the cast of the wrist of a child who suffered from rickets, in which you can see very well the thickening and enlargement of the epiphyses due to this exaggeration of the preliminary stage of ossification. Such thickening is usually first seen in the ribs. It begins there, or can be

earlier recognised there than anywhere else. In consequence there is produced a row of knobs down the sides of the chest, 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. In other words, 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



FIG. 6.
"HOT-CROSS-BUN" HEAD.

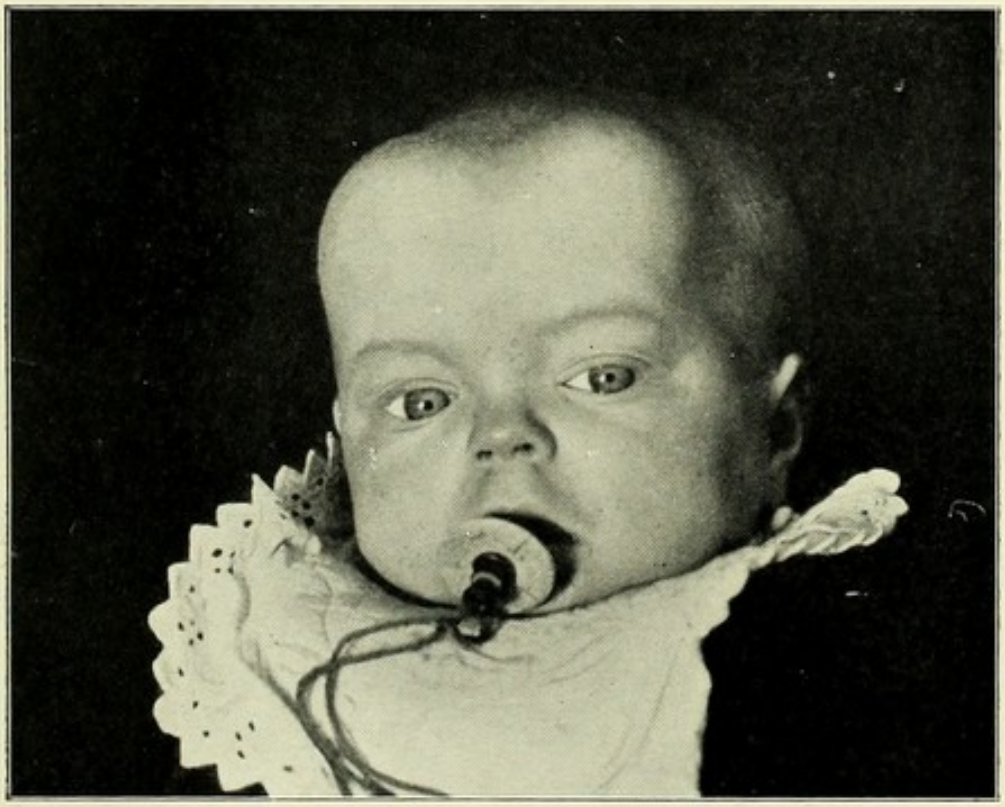


FIG. 7.
HEAD BOSSING.

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 group of changes—the visceral—are of even greater importance than the osseous. I might remind you in this connection of another saying of Jenner, that it would be as reasonable to regard rickets as a disease of bones only as it would be to regard typhoid fever as merely a disease of Peyer's patches. 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 a catarrh in the intestines, because these repeated attacks of catarrh subject the child to a great risk of the development of tuberculosis.

Now, to pass to the more clinical side of rickets, it is well that you should recognise certain distinct **types of the disease.** First of all, there is the acute type, which some people call **'acute rickets.'** But this term 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 recognise 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 recognise—namely, those cases in which there is great laxity of ligaments and muscles. I saw yesterday 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 I shall show you at the end of the lecture some lantern slides which illustrate it.

All those 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 recognise 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 six months; the most likely time for it to come under observation is at about eighteen 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.

We may now pass on to consider the **causes of rickets.** I suppose there is no disease of which it has not been asserted at one time or another that it is due to one of two causes—either to a mysterious nervous disturbance, usually described as trophic, or to the action of bacteria. To this rickets is no exception. There have been some who have asserted that it is due to a trophic influence of the nervous system, and there have been others who have asserted that the whole condition is produced by a micro-organism. I shall ask you to dismiss

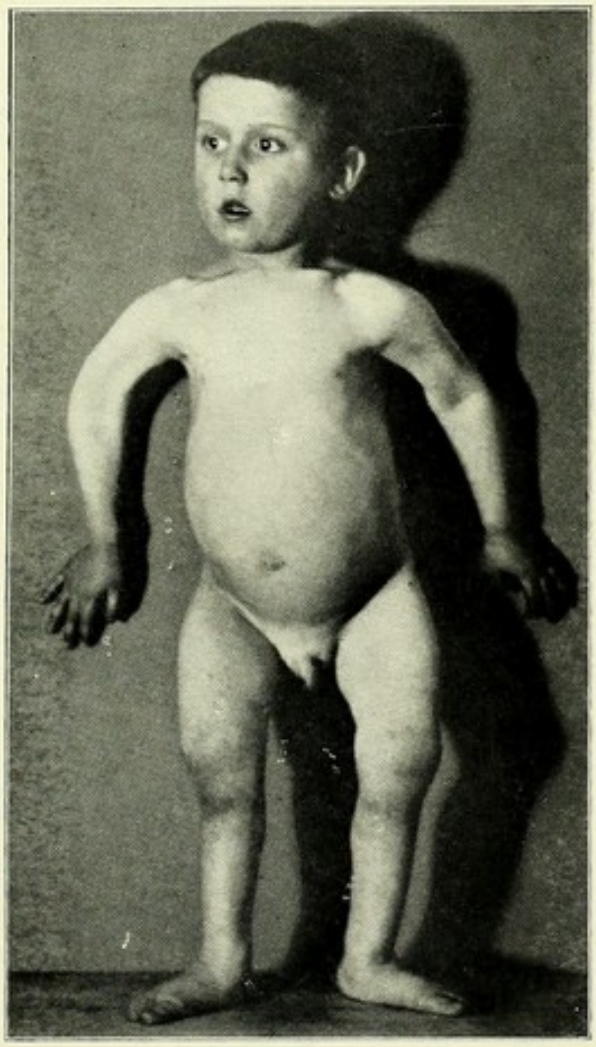


FIG. 8.

RICKETS OF "OSSEOUS TYPE," SHOWING CURVATURE OF BONES.

both those theories from your minds as being, in the present instance, quite unproven. To French authors we owe two other theories : one of them is that rickets is an expression of congenital syphilis, and the other that it is due to dilatation of the stomach. Both of those theories are equally chimerical. The first was put forward by Parrot long ago, and the other by his successor, Comby, at the infants' hospital in Paris. Neither of them has received much support, except from its author or his pupils. And, indeed, I think there are practically only two theories which have to be seriously reckoned with as likely explanations of the production of rickets. One of those is that the disease is due to defective absorption of lime salts, or to the production of lactic acid in the stomach dissolving away the lime salts out of the bones. This one might call the **'lime theory.'** The importance of considering this theory is that a line of treatment has been based upon it. Lime salts have been administered to rickety children under the vague notion that a deficient supply of them lies at the bottom of the production of the trouble. In criticism of this view it may be remarked that, in the first place, there is no reason to suppose that children who suffer from rickets are getting too little lime in their diet. On the contrary, such children are usually being fed on cow's milk, which contains far more lime salts than human milk does, because cow's milk is designed to build up the massive bones of the calf, and is therefore not likely to be deficient in calcium. Further, nobody has yet shown that the administration of lime salts is of any use in rickets. I would also point out that the inorganic matter of the bone, the calcium phosphate, is dissolved away or removed from the bones of rickets as phosphate of lime.

Were an overproduction of lactic acid the cause of the mischief, it would be dissolved away as lactate of lime. So the lime theory is falling into discredit, and people are coming more and more to adopt the other theory, which is supported by clinical observation, and also by a certain amount of experimental work—namely, that rickets is due to a **deficiency in the diet of fat and proteids.** And certainly your clinical experience will soon teach you that the main characteristic of the diet which tends to produce rickets is that it is an ill-balanced one, a diet characterized by an excess of carbohydrates and a deficiency in proteids and fat. Whether it is the absence of the fat or of the proteid which is the most important factor in producing the disease I cannot tell. One only knows that by increasing the amount of fat in the diet you do much good in rickets, and by increasing the proteids you do good also. It is probably, in most cases, the deficiency of both of these which has produced the disease. But why it is that rickets should result from such a deficiency no one has yet explained. There is no direct relation that anyone can see between a deficiency of fat and proteid in the diet and the softening of bones. Yet there is no doubt that animals which are reared upon a diet which is deficient in fat get softening of bones in some way or another.

So much for the theories which have been advanced to account for the production of rickets. I emphasize the last one, because it is by adopting that theory as a working hypothesis that you are certain to be successful in treating your cases. However that theory may stand pathologically, there is no doubt that therapeutics based upon it are justified by results.



FIG. 9.

SLIGHT DEGREE OF HYDROCEPHALUS, SHOWING GLOBULAR HEAD.

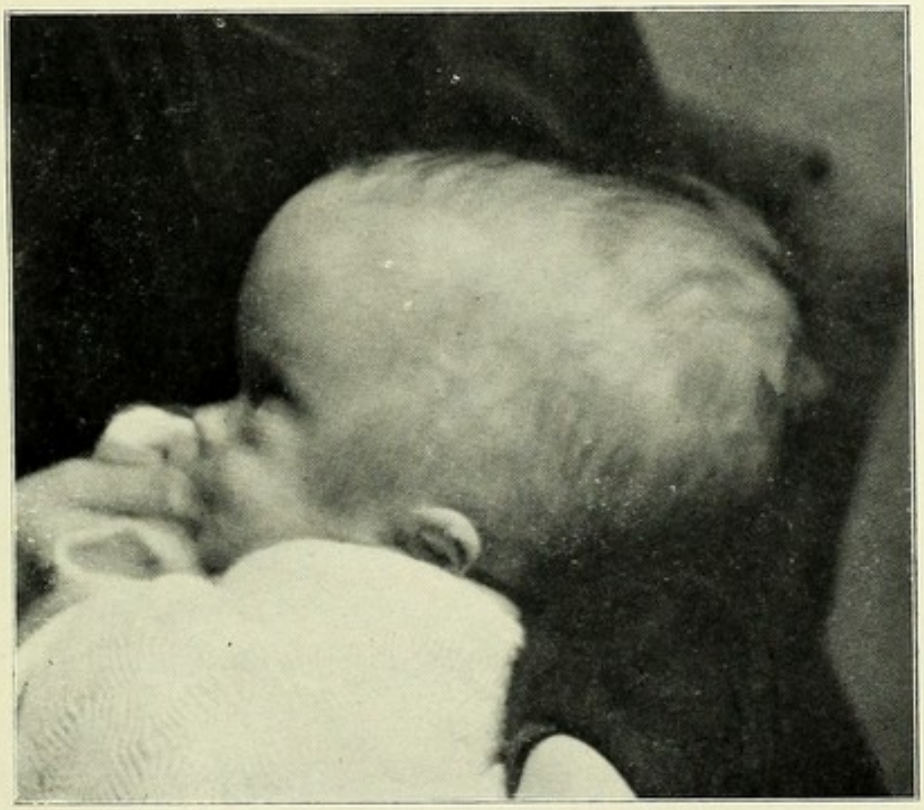


FIG. 10.

HYDROCEPHALUS, SHOWING OUTLINE OF HEAD IN PROFILE. OSSIFICATION WAS HERE VERY IMPERFECT.

Our next point is as to the **diagnosis of rickets.** That 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 at least some 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 recognise, 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 inquire into 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. That happens many times in practice. How shall you distinguish the rickety child from the child with a 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. There are certain mistakes which are likely in connection with the spine of ricktey 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 tubercular caries. But you will be able to tell a **rickety spine** 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. Another pitfall into which you are likely to fall is due to the large development of the abdomen in rickety children. Such cases 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.** 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



FIG. 11.
RICKETY SPINE, SIMULATING POTT'S CURVATURE.

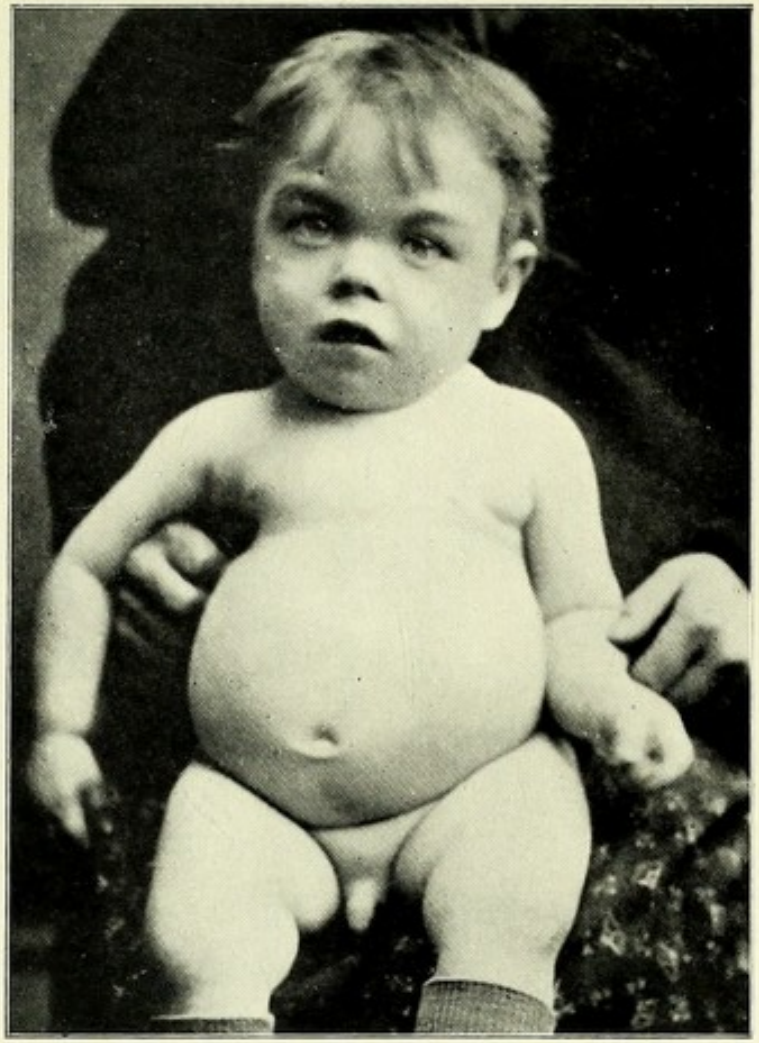


FIG. 12.
RICKETS, SHOWING THE LARGE ABDOMEN, CONSTRICTED CHEST, ETC.

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. So there are many reasons why a child should suffer from a large abdomen ; and if you recognise the rickety element you will be on your guard, and you will not mistake the enlargement for that due to tuberculous disease.

There is still another pitfall into which you are likely to tumble, and that is in 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 paralyzed. 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 at all.

Lastly, I would emphasize the importance of your being able to recognise **the rickety element in many other diseases.** For instance, a child may be brought to you who is mentally deficient, but who 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 disease 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 recognise that these are simply the symptoms of an underlying rickety condition.

That brings us to the consideration of what the **treatment of rickets** should be. Granted the theory of its causation which I tried to bring before you—namely, a faulty diet—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 proteids and fat. Practically that resolves itself into increasing the proportion of cow's milk and administering cod-liver oil. Of course, if the child can get plenty of cream you can dispense with the cod-liver oil. But always increase the amount of milk. A child of two and a half years should be getting two pints 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 proteid, 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 cases 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 proteids 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. If the child is anæmic, give iron in addition. Many people give phosphorus, but I am not convinced that it makes much difference. Attention to the diet and improving the child's hygienic surroundings must be your chief lines of attack, because although it is true that bad diet is the main cause, yet there are co-operating factors. Want of sunlight is considered by many to be one of these. The child should have the benefit of the sun if there is any, and 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 surprising 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 require to tie the legs together, or put on light splints which project beyond the foot. And you will require 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, where the child is five or six years of age, and where there is permanent deformity, surgical means are alone likely to give you much assistance.

LECTURE VIII

INFANTILE SCURVY

GENTLEMEN,—Our 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 many people, 'scurvy rickets.' That is an unfortunate term, because I think it is now quite well recognised that scurvy in infants has nothing to do with rickets at all, 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 apt 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 apparently not the same disease as scurvy in grown-up persons. It is not the same thing as the scurvy which used to be such a scourge on board merchant ships, or as the 'land scurvy' which occasionally occurs in persons who live in unhealthy surroundings. It 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 say, 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 swelled, 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 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 recognise 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, for in that way the tenderness of the bones is aggravated. 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. 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. Of the cases I have seen in the last two years, twelve or so in all, by far the larger number were 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 gums around them are swollen and of a livid 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 swelled and discoloured condition. 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. 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 patent food, usually along with condensed milk. That

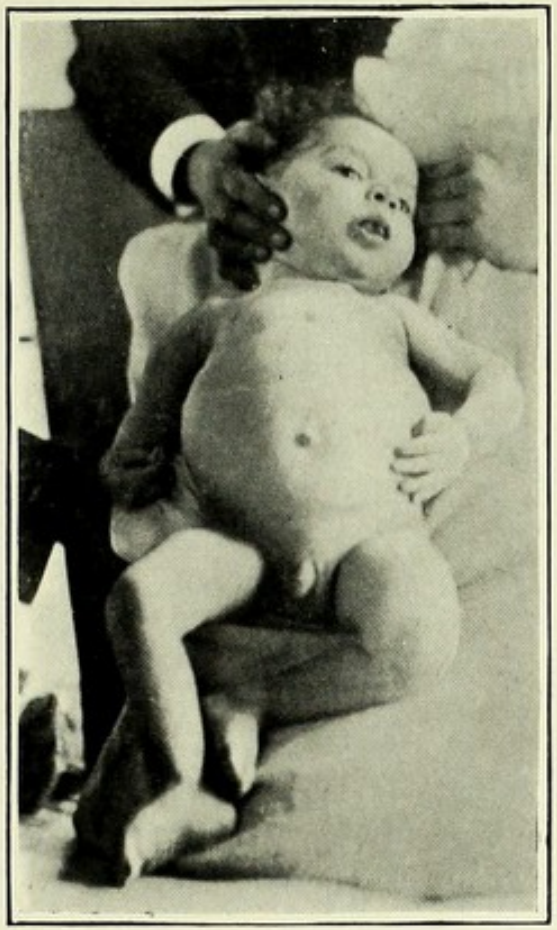


FIG. 13.

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

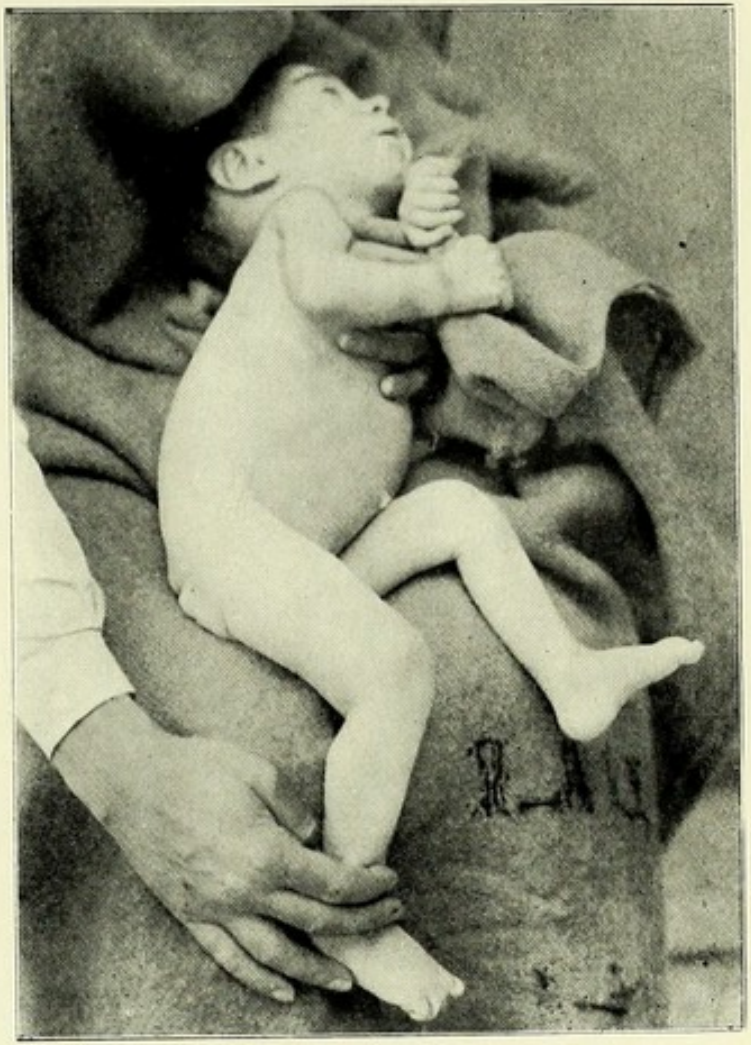


FIG. 14.

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

is why cases of infantile scurvy are as common, or perhaps even commoner, among the children of the upper classes as in the lower, because these patent foods, being expensive, are to a large extent the perquisite of the rich. 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 I believe a few are on record in which, in some mysterious way, breast milk has produced scurvy. Once or twice I have seen the disease produced in children who were fed simply on boiled cow's milk, but by far the majority of the cases of the disease have been fed upon a patent food combined with condensed milk.

The pathology or **morbid anatomy** of infantile scurvy is not a difficult matter to explain. It is characterized by a tendency to hæmorrhages, and the seat of those 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 this process of hæmorrhage goes on to the development of 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. We will discuss immediately the etiology. But I wish first of all to turn to the **diagnosis of infantile scurvy,** and to mention some pitfalls which it is well for you to be aware of.

In the first place, you will have to diagnose infantile scurvy from *rheumatism*. I say that because I have had at least two 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. Little children below one year of age do not suffer from rheumatism. I do not know why that is, but one knows it to be a fact. 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 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. But one of the best tests of all 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, put the child upon a diet which is suitable for scurvy 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 some cases the therapeutic test may be the only one upon which you can depend.*

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—namely, *infantile paralysis beginning with hyperæsthesia*. Some cases of infantile paralysis are accompanied at their outset by extreme hyperæsthesia 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

* 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.

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 firstly for *stomatitis*, and sometimes there will be no means of telling whether it is a case of mere stomatitis or scurvy except by trying the effect of diet. These 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 ; and one was told that the child was 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 we found 200,000 white corpuscles, 98 per cent. of which were lymphocytes ; in other words, it was a case of acute leukæmia. These latter cases are very often characterized by the occurrence of hæmorrhages into the gums, swelling and sponginess of them, and ulceration. And it is well for you to remember, as I shall have occasion to point out to you 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 very often accompanied by those lesions of the mouth. But here, again, the examination of the blood will at once settle the matter.

Cases which are characterized by hæmorrhages into the orbit may be mistaken for two conditions : for *sarcoma* of the orbit on the one hand, or for that very rare disease

called *chloroma*. In the case of sarcoma there will be usually signs of sarcoma 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. Here, again, I know of no very definite way of making sure of the cause of the hæmaturia except by having recourse to the therapeutic test—changing the diet and seeing what happens.

I want next to discuss with you what the **treatment of infantile scurvy** should be before we pass on to consider its etiology. It was found very early in the investigation of this disease that it could be cured almost at once by insuring 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 milk which is raw—that is to say, unboiled. Also, any patent food which the child is taking should be at once stopped.

You should give every day a few teaspoonfuls of orange or grape juice, or, if you like, a little raw meat juice. 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. You may ask why one should take that particular part and treat it in that way. The reason is that that part of the potato is richest in potash salts. 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 a radical change of diet in 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 recognise 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 is only recognised.

That leads me on to the discussion with you of the last question—namely, **What is scurvy really due to?** Why is there this hæmorrhagic tendency? As I have said, it is due in some way to there being something wrong with the diet. The results of alteration in the diet make that point quite clear. What is there, then, in a diet of patent food and condensed milk to produce this tendency to hæmorrhage in the child? That is a question to which no final answer is yet forthcoming; but I think one can get at least near to some idea of what the nature of the fault is by carrying out a series of simple observations. The first thing to determine is whether the fault in the diet is a positive or a negative one. I mean, is there something in



Fig. 15.

Orbital hæmorrhages in Scurvy.

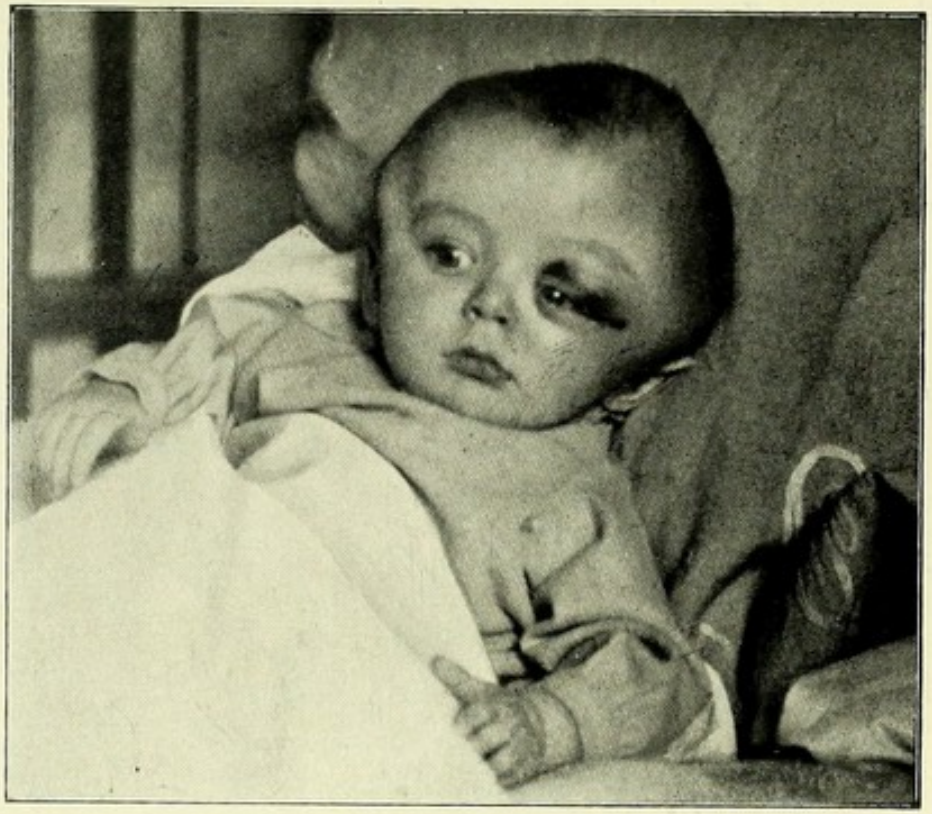


Fig. 16.

Hæmorrhage into orbit, the result of sarcoma of skull, simulating Scurvy.

the food which should not be there, or does it not contain something which should be there ? In other words, is it an error of omission or an error of commission ? You can easily settle that in this way. You can leave the diet exactly as it was, and you can add to it fresh fruit juice. If you do that you will find that the child gets well. So it is clear that there is nothing positively wrong in the diet, but that there is merely something absent from it which fruit juice contains. These cases are not due, then, as some people would have you believe, to the development of ptomaines in the food. I do not believe at all in the ptomaine theory as applied to scurvy. Certainly in the case of children any such explanation is quite insufficient. What is wrong, therefore, is that the food of the child does not contain something which it ought to contain. What is that something ? Many things have been suggested. Some people think it is a ferment. You can settle whether it is really a ferment by trying the effect of *boiled* fruit juice, for a ferment would be destroyed by boiling. Now, I find that boiled fruit juice cures these cases just as fresh fruit juice does. What constituent of the fruit juice is it, then, that brings about the benefit ? It must be a ' dead ' constituent, because it is evidently unaffected by boiling ; and it belongs either to the crystalline or colloid group of ingredients. The next step, therefore, is to dialyse the fruit juice and administer the dialysate. If one does that one finds that the dialysate has a curative effect, but the colloid constituents have not. In other words, what is absent from the diet that produces scurvy is a crystalline substance, which is present in fruit juice. One must, therefore, try the effect of the crystalline substances contained in the juice in a pure form. Of these the citrate of

potash and the citrate of lime suggest themselves for trial first. The reason one takes the citrates first is that there is presumptive evidence in favour of the view that it may be absence of citrates which produces the disease. Cow's milk contains far more citric acid than you might suppose ; a quart of it is said to contain as much citric acid as a large lemon. Citrate of lime when it is boiled passes into a less soluble form, and when milk is condensed citrate of lime is apt to separate out in the form of gritty, granular particles in the tin. There are grounds, therefore, for believing that it may be the absence of citrate of potash or citrate of lime which produces scurvy. I have not found, however, that the administration of citrate of potash or citrate of lime in pure form has a definitely curative effect. I have tried citrate of lime in large doses without producing improvement. On the other hand, from a mixture containing malate of potash, citrate of potash, and tartrate of potash, I have found great improvement, which, however, is not brought about so quickly as by the complete change of diet which I have already mentioned. Further than that I have not yet carried my observations, and I cannot speak more definitely. But I think there is ground for supposing that this mysterious alteration in the blood is due to a deficiency of salts of potash, though how it is that this deficiency tends to cause hæmorrhages I am not in a position to explain. This, however, one can say, that the hæmorrhages are not due to any defect in the coagulability of the blood, for although I have estimated the coagulation time in many cases of infantile scurvy, I have never found it materially different from the normal.

LECTURE IX

THE DYSPEPSIAS OF THE SECOND DENTITION

GENTLEMEN,—In a previous lecture you will remember we considered together the digestive disorders of infancy; and I want now to turn to the study of kindred disorders in older children, and to speak about what one may term 'the dyspepsias incidental to the second dentition.' There are probably many reasons which render children at about the period of second dentition particularly liable to suffer from digestive disorders. One of these, no doubt, is the confinement and want of physical exercise, as well as the mental overstrain, which are incidental to school life, which is usually begun at about that period. You will find that many cases of chronic dyspepsia in children are certainly attributable to that cause. In the second place, owing to the rapidity of growth at this time of life, there is a tendency for the large demand for food to outstrip the digestive power, so that the stomach is no longer equal to the demands made upon it by the tissues. This demand is apt to be met by an excessive consumption of carbohydrate foods, and that, in the third place, is one of the main causes of chronic dyspepsia in children of this age. Coming closer to this subject, one finds a difficulty in classification, just as one did in the case of the dyspepsias of infancy. That

difficulty arises, again, from the insufficiency of our pathological knowledge. We do not know what the pathology of many of these cases of dyspepsia is, but I think one may conveniently make the assumption that they are all of them essentially catarrhal in their nature, and I shall speak of them under three headings. First there is acute dyspepsia, or, if you like, acute gastro-intestinal catarrh; secondly, subacute dyspepsia, or subacute gastro-intestinal catarrh; and, in the third place, chronic dyspepsia, due to chronic gastric and intestinal catarrh.

ACUTE GASTRIC CATARRH.

Acute dyspepsia in older children resembles very closely the similar disease which I have already described in the case of infants. It begins suddenly, as its name implies, and the chief symptom is vomiting. The vomiting is accompanied by prostration, and often by a high temperature, and on examination you find that the tongue is thickly furred, but that otherwise there is very little to be made out. These cases usually arise from one of two exciting causes. One is the consumption of some irritating article of food. What that food is varies in different instances. The stomachs of some children are peculiarly susceptible to irritation by some articles which are eaten with complete impunity by others. Thus, some children have great difficulty in digesting greasy things, and these seem prone in them to produce acute gastro-intestinal catarrh. In other cases the cause may be unripe fruit; but, of course, there are many different kinds of food which may act as exciting causes of the condition. The other great exciting cause which you have to bear in mind is chill. I

have repeatedly seen cases of acute gastro-intestinal catarrh brought on in children at about the period of the second dentition by exposure to cold. Thus, I have seen it happen very suddenly after sea-bathing. These cases are apt to begin chiefly with stomach symptoms and to end up with intestinal symptoms ; in other words, the catarrhal trouble seems to start in the stomach, and to extend downwards through the stomach into the intestinal canal. That is particularly the case in those instances in which the exciting cause is some error of diet. In them the intestinal trouble ends with diarrhœa, and the diarrhœa, by carrying away the irritating material, leads to a spontaneous or natural cure. The difficulty which you will have in dealing with these cases is in being sure that they are not something else. You may have difficulty in realizing how severe an attack of acute gastro-intestinal catarrh may be ; there may be high temperature, severe prostration, and vomiting, with a furred tongue, and you will, in such a case, be apt to regard the condition as due to something more serious than a mere gastro-intestinal catarrh—for instance, you will think of one of the specific fevers, perhaps of meningitis, or some serious abdominal trouble. I have known a case of that sort in which the abdomen was nearly opened under the impression that the child was suffering from appendicitis.

There is a group of cases in which those attacks recur at intervals, in which a child will go for a few weeks and then have an attack of gastric catarrh, and then go on for another few weeks and have another attack, and the mothers will describe them as **'bilious attacks.'** Some of those cases are probably genuine examples of recurring gastritis, but others are, I think, of nervous

origin, and are closely allied to megrim. The term **'cyclic vomiting'** is sometimes applied to them.

The **treatment of acute gastric catarrh** is quite simple. In consequence of the great depression, you should put the child to bed, and withhold all food for a time, or as long as the vomiting is urgent. During that time you may administer sips of iced water in order to allay the thirst. If there is much vomiting, there is no occasion to wash out the stomach, but in cases where nausea is present you may find lavage of the stomach, as in the case of little babies, an important help. Drugs are not of much assistance to you, but the most useful are calomel and bismuth. You should begin at the outset by giving small repeated doses of calomel, $\frac{1}{6}$ to $\frac{1}{4}$ grain every two or three hours until 2 grains have been taken ; then follow that up by bismuth in large doses. Under that treatment, the attack rapidly subsides. And then you have to consider how you are to prevent recurrences of the attacks in future, particularly when you are dealing with a child who is the subject of so-called ' bilious attacks.' How you are to do that we shall consider when I come to speak of chronic dyspepsia.

SUBACUTE GASTRO-INTESTINAL CATARRH.

We may turn next to subacute cases of gastro-intestinal catarrh, a condition which I think is less common than the above, but which may be left behind after the acute attack has passed off, although it may also arise spontaneously and be subacute from the outset. In these cases the fever is not so high as in the acute attacks, but it is more prolonged, lasting perhaps a week or ten days. And as the

attacks are accompanied by a tendency to vomit, and sometimes by diarrhœa and a furred tongue, you will readily understand that these cases are very apt to be mistaken for typhoid fever, and many of them undoubtedly used to be written down as **'gastric fever.'** You know that nowadays the term 'gastric fever' has fallen into disuse, and we know that a large number of the cases so described were undoubtedly cases of enteric fever, mild in degree. But I think it is also true that some of them were really instances of subacute gastro-intestinal catarrh. Fortunately, in the agglutination test we have a means of distinguishing those cases from true enteric. Clinically, without that test there is difficulty, because the two diseases simulate one another very closely. So you must be on your guard not to mistake them for enteric, and *vice versâ*. The treatment of them is very much the same as that of acute attacks—namely, rest in bed, restricted diet, and the administration of calomel and bismuth.

MUCOUS DISEASE.

The last group of dyspepsias is the most important of all—that of **chronic cases of gastro-intestinal catarrh.** These cases are extremely common. You will meet them in some form or another in practice almost every day. Together they form a class which is fairly definite, although one can distinguish in it certain special types of the disease. They are sometimes known under the term **'mucous disease,'** that name having been first given to them, so far as I know, by Dr. Eustace Smith, for reasons which we shall see immediately.

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 of the child, 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 some 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, and it is not very uncommonly met with among children who suffer from this special form of chronic gastro-intestinal catarrh. When you inquire into the condition of the bowels, you will generally be told that there is constipation. You will hear that the child's 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. Very

commonly you will get a history that the child is troubled with worms. Less frequently there will be a tendency to diarrhœa, and one characteristic of such diarrhœa which you will often notice is that it tends to come on immediately after the taking of food. The mother will tell you that immediately the child eats a meal there is a tendency for the bowels to act. To that condition the term 'lienteric diarrhœa' is applied. 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 also 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 has 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. Certainly they are associated with vascular alterations, perhaps spasm in small arteries or sudden lowering of the blood-pressure; but what the exciting cause of those alterations in blood-pressure is we do not know.

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 six 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 described, 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 will often find that it contains proteid, 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 proteid appears in the urine at particular times of the day, usually after the child has got out of bed and is going about. The morning urine which has been secreted in the night is usually free from any trace of albumin at all.

Such are the chief symptoms of a typical case of this so-called mucous disease. But you will frequently 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, you can distinguish 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 **stomach 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 cases which certainly belong to 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 chronic intestinal catarrh, and not a disease in themselves. Other cases will be brought to you solely with the complaint of **lienteric diarrhœa**—

that is to say, the diarrhœa is so prominent a symptom as to overshadow all the rest. And, lastly, you will have cases brought to you for **restless and disturbed nights,** the so-called pavor nocturnus, or night terrors.

We will pass on now to glance at the **pathology** of this disease 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 has a very neat theory to explain all the symptoms detailed above. He says 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 gets 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 proteid in the urine it is very often a nucleo-proteid, 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. In those cases in which you get the passage of masses of mucus from the bowel there is a considerable amount of evidence in support of it. But this does not always occur, and the real importance of the theory is that the treatment which is based upon it is certainly justified by its results. That treatment I shall describe to you immediately. 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 one is here dealing with a disorder of metabolism—a 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. There are physicians in this country who also maintain that view, and believe that these cases are due in some way, not completely understood, to an imperfect elaboration of proteid material, which leads to the production of an excess of uric acid, pretty much as you know takes place in gout. Between these two theories I shall not attempt to decide, because, as I have said, there is no definite ground to go upon in the argument. I want further to point out to you that whatever the true theory of the mode of production of the symptoms is, there can be no doubt whatever about one frequent exciting cause of the disorder, and that is a matter which it is of more importance for you to know. It is the excessive consumption of sugar. That is so common and so well recognised a cause of this

disease that you might almost call it the dyspepsia of sweet-eating children. You will remember that cane-sugar is eminently a producer of mucous secretion. If you take cane-sugar into the mouth and suck it, the mouth becomes sticky, not merely from the presence of the sugar, but from the secretion of a viscid mucus by the salivary glands. So that this fact would also fit in quite well with Dr. Eustace Smith's theory.

We come now to the **diagnosis** of chronic gastro-intestinal catarrh. I have said that these cases will come before you with great frequency, and there is one condition which you are particularly apt to mistake them for, and that is tuberculosis in some form. One reason for this is that the child is so often brought for cough and wasting, a combination of symptoms which in a grown-up person you are apt to associate with the presence of pulmonary tuberculosis. But I would ask you to remember once and for all that pulmonary phthisis is a rare disease in children. Tuberculosis at this age does not tend to take the pulmonary form. If you are in doubt whether you are dealing with a case of latent tuberculosis somewhere, I advise you to give heed to the temperature; make a point of taking it night and morning for a few days. Dyspeptic cases are not accompanied by fever, or, at all events, the fever is not continuous. There may be transitory attacks of fever during the exacerbations of the condition, but these only last a short time, whereas cases of tuberculosis, no matter where situated, tend to be accompanied by nocturnal rises of temperature, which persist for several weeks.

Finally we arrive at **treatment.** If you set about this in the right way, I think I can promise you very

satisfactory results. But if you are not familiar with these cases, you will find them very troublesome and chronic, and difficult to cure. Seeing that I have just mentioned that the excessive consumption of carbohydrate food, particularly sugar, is undoubtedly one of the chief exciting causes of this form of dyspepsia, the main line of treatment must be dietetic. The first thing to do is to attend to the food. The sheet I have put into your hands describes to you the chief lines upon which the **diet** for such cases should proceed.

DIRECTIONS FOR DIET IN CHRONIC DYSPEPSIA.

No food must be taken between the regular meals.

No sugar or sweets of any sort are to be eaten.

The child must *not* eat: New bread, potatoes, peas or beans, turnips or carrots, pickles, pastry, jam, syrup or treacle, or cakes.

The diet should chiefly consist of: Stale bread or dry toast, with butter or dripping, bacon, eggs, fish, meat, milk, plain milk-pudding (except cornflour, arrowroot, or sago), and green vegetables in small quantity.

You will see that it consists essentially in the limitation of carbohydrates, and in the almost total prohibition of sugar. If you will keep that broad principle before your minds, you will have no difficulty in adjusting the details. Next to a change of diet I put in order of importance change of air. Repeatedly one has seen cases which were hanging fire and not doing well get better right away when they were sent to a convalescent home at the seaside. That is probably to be explained on the grounds which I have already mentioned, that many of these cases are due to a lack of physical exercise and confinement to school for long hours, and to unhealthy homes. Next we come to consider the question of **drugs.** You will find that the drugs chiefly

of use at the outset of the case are alkalies and aperients. I think aperients are useful no matter what the state of the bowels—whether there is constipation or not—and it is a good routine plan to begin by using them. The usefulness of those remedies may also be accounted for on the excessive-production-of-mucus theory. According to the holders of that theory, one should administer alkalies because they are an admirable solvent of mucus. And one administers aperients to get rid of the mucus which has been so dissolved. Whether the explanation is right or not, at all events you will get great success from the adoption of this line of treatment. I use some such prescription as this : Pot. bicarb., pot. cit., āā 5 grains ; tinct. nucis vom., 1 minim; compound infusion of gentian, 2 drachms, given with a little water a quarter of an hour before the meals. The action of each ingredient of this prescription is believed to be this: The bicarbonate of potash dissolves the mucus. The citrate is given largely because these children tend so often to have thick urine and the passage of urates. The nux vomica is given to increase appetite, which is usually bad. The gentian is given for the same reason, and also because it has a mild astringent action on the mucous membrane of the stomach.

The **aperients** I like to begin with consist of rhubarb and mercury in the following combination : Rhubarb powder, 8 grains ; bicarbonate of soda, 10 grains ; gray powder, 1 to 2 grains, according to the child's age. This should be given every night, or in a milder case every other night. Rhubarb is selected because it has not only an aperient action, but it has an astringent after-action, and a tonic effect upon the flabby mucous membrane. One adds bicarbonate of soda for the same reason as in the other

mixture; and many cases are undoubtedly greatly benefited by mercury in some form or another, probably because mercury seems to have a peculiar specific influence in diminishing catarrh of the alimentary canal. After a course of such treatment for a fortnight or so, you will probably find a striking degree of improvement; the child's appetite will have returned; he will begin to gain flesh; he will no longer suffer from restless nights, and you will have reached a stage at which you can begin the administration of tonics, which must include iron in some form or other. A very good form is the wine of iron, given with an aperient in the form of aloes. I want particularly to impress upon you that you should defer the administration of those tonics, particularly iron, until you have got the tongue clean and the stomach in a healthy condition. There is no commoner mistake than to treat these cases at once with cod-liver oil and Parrish's food as soon as they are seen. The doctor is apt to order them iron to make them strong and cod-liver oil to make them fat; and such treatment, if begun too early, does harm instead of good. It is a good rule in medicine not to begin the administration of iron until the stomach is in a healthy condition, because iron is almost always an irritant to the stomach, and it is not absorbed and made use of until the alimentary canal is brought into a condition somewhat approaching that of health. Although that should be your general line of treatment, you will find that special symptoms in particular cases may call for special remedies. Many patients, for instance, are troubled with gastric pain or colic, which may be extremely obstinate, and sometimes the only thing to which it will yield is opium. The best form in which to give it is Dover's powder, and 2 or 3 grains ought to be sufficient. If you

have lienteric diarrhœa to deal with, you will find the best thing to do is to give small doses of opium before meals—perhaps 2 minims of the tincture of opium may be enough—and it is well to combine with that a drop of Fowler's solution and a little nux vomica. Lienteric diarrhœa is brought about by peristalsis in the upper part of the bowel being too active and too easily excited reflexly, so that when food is introduced into the stomach, instead of merely exciting the stomach it starts a wave of peristalsis, which spreads through the intestine and leads to a motion. Opium is often the only thing which will allay that and prevent the diarrhœa. The existence of worms may be a prominent feature, and, as I have said, you should never regard these as a disease, but as a symptom of an unhealthy condition of the mucous membrane, which makes their lodgment possible ; and so in these cases, more than in any other, you should administer purgatives to clear away the mucus, and in very bad cases you can combine that, if you like, with enemata of salt solution or quassia, both of which you know act as parasiticides. It is only very rarely that you will find it necessary to resort to santonin. I almost never prescribe it. I usually find that regulation of the diet and the administration of aperients get rid of the worms, and put the mucous membrane of the bowel into a healthy condition. Should headache be a prominent symptom, as it is apt to be, I recommend you to pay particular attention to the refraction of the eyes. You will often find that there is a slight error of refraction, astigmatism, or hypermetropia, and if you correct that by means of suitable spectacles the headache disappears at once. The cough, which is so common in these children, may necessitate local treatment directed to the throat ; you may require to use nasal

douches, or washing out of the back of the throat with an alkaline and antiseptic solution. But in many instances you have to proceed sooner or later to the removal of the tonsils, and perhaps also of adenoids. But the relation of adenoids to these cases I shall discuss more fully in another lecture.

LECTURE X

RHEUMATISM IN CHILDHOOD

GENTLEMEN,—I wish to bring before you in this lecture the subject of rheumatism as it manifests itself in early life. Now, 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, you will remember, 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. It is a condition which, if properly treated, may be cut short, and, if cut short, many consequences which would otherwise blight the life and activity of the subject of it may be prevented.

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 a morbid entity, as definite and precise in its pathology as typhoid fever or diphtheria. Now, though rheumatism may certainly be regarded nowadays as being due to a specific organism, you require for its production not merely the organism, but a suitably prepared 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 recognise 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. 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 recognising 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 infection.

Now, unfortunately, we do not know how those children become infected. We do not know definitely the path by which the rheumatic organism gains access to the body. We are unable to trace it as we can trace, for instance, the path of the tubercle bacillus. But I think there is at least a reasonable amount of evidence to show that infection takes place in perhaps 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. I shall have occasion, however, when I come to talk of adenoids in infancy and in childhood, to speak a little more fully upon this subject. 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 in

children 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 bacillus. 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 text-book 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 manifestations. No one who has seen much of the disease in children will fail to recognise 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 usually given to it in text-books. 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 valves of the heart more, perhaps, than any other part which tend 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 endocarditis 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 endocarditis 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 : First, articular inflammation, the ordinary synovitis of joints ; secondly, muscular or fascial rheumatism; thirdly, rheumatic nodules; fourthly, endocarditis, myocarditis, and pericarditis ; fifthly, erythematous eruptions of various sorts ; sixthly, chorea ; seventhly, pleurisy ; and, lastly, 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 co-exist, or they may succeed each other in any order. Given a child predisposed to rheumatism, he may show at one time a few

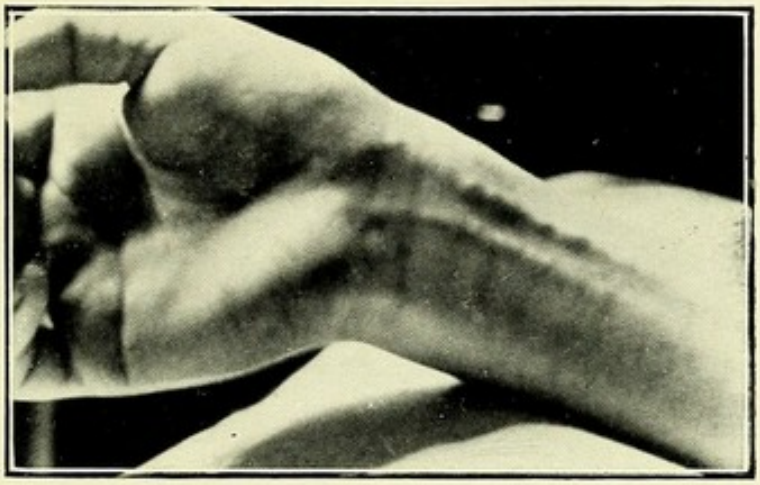


FIG. 17.
RHEUMATIC NODULES ON TENDONS OF THE WRIST.



FIG. 18.
RHEUMATIC NODULES ON ELBOWS.

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 poison—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 joint 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 act to you as a danger-signal. The pains of muscular and fascial rheumatism are commonly called, in the nursery, 'growing pains.' That, gentlemen, 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. A great many cases of heart disease come before you in later life in which you can obtain no definite history of rheumatism in childhood, and many of these, no doubt, are cases in which there have been these fugitive muscular pains, which have been so trivial that no notice has been taken of them until the heart has become seriously damaged. Those 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.

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 of extreme frequency. 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. 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, which may surprise you, and by being extremely mobile. When you cut into them you find they consist of little nodules of fibrous tissues, the fibres being arranged concentrically round a core of fibrin. Those nodules are not in themselves of any importance whatever ; it does not matter if there are little fibrous lumps under the skin here and there ; but, like those rheumatic pains which we have been considering, they are of the greatest importance as danger-signals, because they tend to go along with the heart complications, with endocarditis and pericarditis, and if you find rheumatic nodules coming out you may be fairly certain that there is progressive heart mischief going on. Dr. Cheadle says that when these nodules become really large they are practically equivalent to a sentence of death, because their significance as regards the state of the heart is so serious. 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 nodules under the skin ; but in the one case you speak of a vegetation and in the other case you speak of a 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. It is because their results are incapable of being entirely compensated for—because a valve once attacked is damaged irretrievably—that these forms of rheumatic manifestation are so serious. The commonest of them is endocarditis associated with pericarditis, and you may take it that in the majority of children who suffer from rheumatic heart affection these go together. You may not always be able to recognise pericarditis, but there is usually some degree of it present if there is endocarditis; and probably the converse is also true. You know that of recent years great attention has been paid in the pathology of the heart to the myocardium, and to the study of the changes which it undergoes in disease. Now, a study of the myocardium in acute rheumatism shows that it also is apt to be the seat of damage. You find small-cell infiltration between the muscle-fibres; you find degeneration of the latter, and you find later a development of fibrous patches.

It is because of these three things tending to occur more or less together—endocarditis, pericarditis, and myocarditis—that some people prefer to speak of the heart affection of rheumatism as simply '**carditis,**' meaning an inflammation of the heart in all its parts; and that is what you have to deal with in the majority of cases. But all the valves of the heart are not equally subject to attack in rheumatism. You may take it that in most cases the

lesion which results from rheumatic heart affection in early life is mitral stenosis ; and in the majority of cases also that is associated with more or less adhesion of the pericardium. Certainly in fatal cases these are the two changes which you find almost constantly : vegetations along the edge of the mitral valve, and more or less adhesion and fluid in the pericardium. Many children will escape those rheumatic manifestations as far as they affect the heart ; and it will depend to some extent upon you how many of your patients so escape, because the better you are at detecting rheumatism the more easily you recognise its trivial manifestations, such as growing pains, and the more seriously and energetically you treat them the less will be the tendency for the heart to be affected ; and by so doing you will prevent a great deal of subsequent cardiac disease in later life.

In the majority of cases you will find that the first attack of rheumatism does not permanently damage the heart. It is only after the second or third attack that the heart becomes the seat of lasting disease. It is true that in the first attack you may hear a whiffing systolic murmur at the apex, and you may therefore conclude that you have to do with damage of the mitral valve, which will go on to mitral stenosis or incompetence ; and it is perfectly true that in such a case you *may* have to do with permanent damage of the valve. Yet there is no doubt that in many cases the murmur disappears entirely later on, and the heart becomes, so far as you can tell by examination, perfectly healthy once more. But if there is a repetition of the attack for a second or a third time the damage to the heart is almost certain to become permanent.

I now pass on to consider **erythemata** as a sign of

rheumatism. An erythema is, as you know, a red eruption which appears like a blush over part of the skin, accompanied by more or less local heat, and which fades away again in a short time ; and there are some particular forms of erythema which are almost peculiar to rheumatism. One of them is erythema nodosum ; I think everyone will admit that it is always of rheumatic origin. It consists of an eruption of spots, slightly elevated, and usually more or less round or oval, which tend to appear particularly on the lower extremities, over the shins or calves, and are slightly tender. The skin over them is glazed, and they are often accompanied by a local inflammation of the ankle-joint. Sometimes there is a hæmorrhagic tendency in these cases, so that you get not merely erythematous patches, but actual hæmorrhages into the skin, and to that form the term *peliosis rheumatica* is applied. Then you may get large 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.

Of **chorea** I do not intend to speak in detail. 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 people say it is invariably rheumatic, because you must remember that chorea is only one event in the series. It may be the first event, and if you follow the course of that 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 require 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 probably usually of the nature of a dry pleurisy, or one without much effusion. Seeing that the pleura is in the nature of 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 recognised 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 recognise the development of complications in an attack of rheumatism by the sudden pallor which the **anæmia** produces. Constantly, when going round the wards, if you have a child who is suffering from a slight rheumatic affection with fever, you will be able to tell if pericarditis has developed from the sudden increase in the pallor of the face. The fresh outbreak of the rheumatic poison seems to produce a fresh destruction of blood.

Lastly, we will 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. 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 I am certain that a great deal of serious cardiac disease in later life would be prevented.

In regard to the treatment of rheumatism when 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 joint affections under your control. It is unfortunate that salicylates seem hardly to touch the cardiac manifestations ; in fact, some people 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 over 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 to the valves. So that what one has to preach about the treatment of rheumatism is the necessity of *rest*. One would sometimes like to put these cases in double long splints and keep them on their backs for six months! I am sorry to say that of necessity in such a hospital as this one has to set a bad example in this matter, for we cannot keep these cases 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 later on. If you insist upon prolonged and absolute rest, and at the same time use salicylates and iodides for the local affection, applying blisters over the heart when there are signs of pericarditis, you will do a great deal to prevent your patients becoming permanently crippled, and you will consequently diminish the amount of cardiac disease which one sees in grown-up persons.

LECTURE XI

THE RESPIRATORY DISEASES OF CHILDREN

GENTLEMEN,—In this lecture we come to the consideration of the respiratory diseases of childhood. Of course, I need hardly remind you that that 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 breast, 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 of the xiphisternum and lower costal cartilages. Later I shall show you illustrations of it with the aid of the lantern. 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 extremely 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

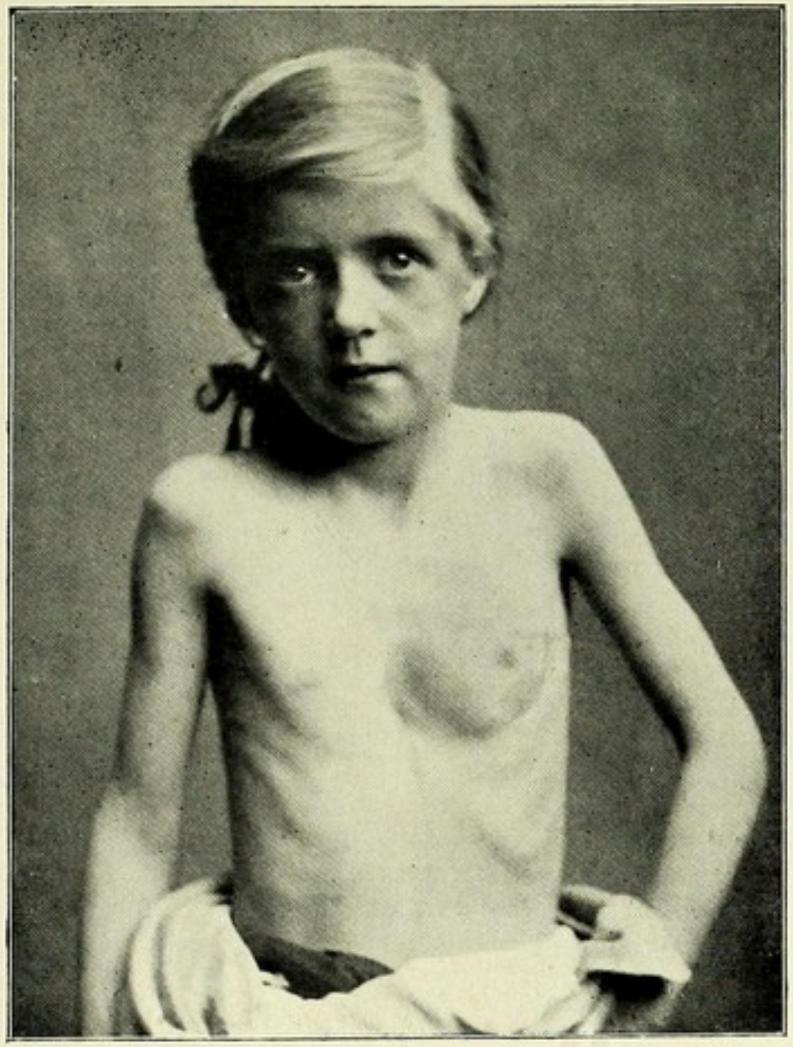


FIG. 19.
ADENOID CHEST, SHOWING DEPRESSION OF XIPHISTERNUM.

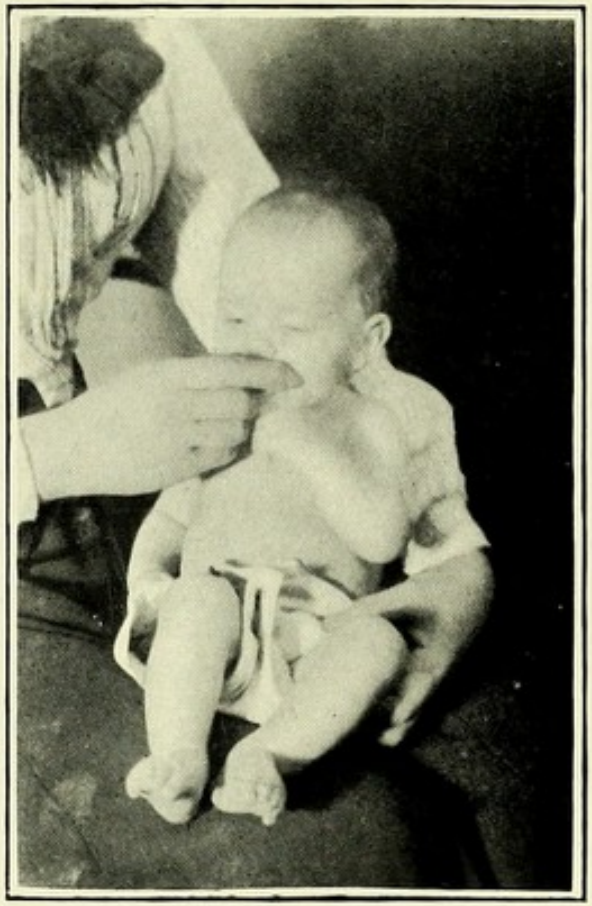


FIG. 20.
TETANY, SHOWING CHARACTERISTIC POSITION OF FEET AND TOES.

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.

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 infants 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, gentlemen, 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 from muscular spasm; (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 these groups, and so, following Goodhart, 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 cases 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 recognise. 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 membranous 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 instals 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 predisposed 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. Ipecacuanha wine 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 justifiable to administer a whiff of chloroform to allay the spasm. 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 room which was rather damp.

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. Of the relative merits of the two procedures it is scarcely my province to speak, but I would only say that I believe nearly all the advantages lie with intubation, and I should greatly like to see it more widely used in this hospital.

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 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 operate upon them, because the only successful treatment is removal of the growth with forceps through the mouth, which is a very delicate and difficult procedure.

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. 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 in your case ? That is settled by the character of the accompaniments—whether they be dry or whether they be moist. In the early stage of the disease you have dry sounds, and in the later 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.

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 creosote inhalations. That 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 use creosote as an inhalation along with the steam. It is well also that those cases 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 alkalies, such as citrate of potash or acetate of ammonia. And such children 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. There are one or 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. In the last stage—that is to say, when the patient is convalescent—you should administer such things 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 the bronchial glands, which have become chronically inflamed.

PNEUMONIAS.

I will speak next of **broncho-pneumonia.** This is a disease which you often see in the wards, one of very great fatality—so much so that about 17 per cent. of the total infantile mortality results from it. Broncho-pneumonia may be either 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 that which induces a fatal ending. Now, your difficulty will always be, when you are in contact with a case of supposed broncho-pneumonia, to say 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 that **physical signs** will here be of little assistance to you ; 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 certain, no matter what the physical signs are, that you are dealing with the more serious affection—namely, 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 of broncho-pneumonia may be so small, and so deep down, and so enclosed by healthy lung, that there may be no dulness at all, and you must not expect always to get dulness when you percuss such a chest. But the more lightly you percuss it, the less likely you will be to miss such patches as exist, for you must remember that sometimes the patches are extremely thin ; there may be only a little layer of broncho-pneumonia, and any heavy percussion will bring out the resonance of the underlying lung below that, and so you may miss the dulness.

Diagnosis of Croupous from Broncho-Pneumonia.

The other disease from which you may have a 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 the distinction between the two. Croupous pneumonia is always a primary disease, but broncho-pneumonia is very often secondary. Broncho-pneumonia is commonest in quite young children, in babies, whereas croupous pneumonia is not commonest in babies, but in children three to four 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 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 apical. I do not know what proportion apical cases bear to basal ones ; but the apical cases are very much

commoner than you would think, certainly more so than they are in grown-up people. The course of broncho-pneumonia is ill-defined, and instead of ending by an abrupt crisis, it ends usually by lysis. In croupous pneumonia, on the other hand, the course is well defined, and ends by crisis on the fifth or ninth day. The dulness in broncho-pneumonia is usually patchy and ill-defined, whereas in the croupous form it tends to follow sharply 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 seems healthy. These are the chief points to be attended to in making your diagnosis. The reason why it is so important to make the diagnosis is from the point of view of prognosis. Broncho-pneumonia is so fatal that about half the cases in hospital die, while, on the other hand, in croupous pneumonia recovery is the rule ; deaths are very exceptional. Holt, in his statistics, found something like 4 per cent. as the mortality of croupous pneumonia in young children, which is, of course, far lower than in grown-up people. There are probably two reasons for the better prognosis in croupous pneumonia in young children. One of them is the great reserve power of the heart, so that you do not easily get paralysis of the right ventricle from overdistension. The other reason probably is that children have usually very healthy kidneys, and there is nothing, I suppose, except alcoholism, which often goes with it, which makes the prognosis in pneumonia more unfavourable than damaged kidneys.

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 croupous 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 dulness 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 it is probable that in future we shall learn to **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 is reason to believe that cases which are due to the pneumococcus run a more favourable course than those which are of streptococcal origin.

I ought to say a word about the **diagnosis of croupous pneumonia** from other conditions. You will be very apt to mistake it for *acute gastritis*. If you have a child with acute vomiting and a furred tongue, do not conclude too quickly that you are dealing with a case of gastritis; you should remember the possibility that it may be croupous pneumonia, because the physical signs of croupous pneumonia, particularly when it affects the apex of the lung, are often very hard to make out. The child may be near the end of the illness before you are able to make out any dulness or bronchial breathing; you have to diagnose it from the mode of onset, the high temperature, and, above all, from the rapid respiration; the physical

signs, just as in broncho-pneumonia, take a secondary place.

Another disease for which pneumonia is apt to be mistaken is *meningitis*. All the chief symptoms of the more grave disease—vomiting, head retraction, and the rest—may be closely simulated by a pneumonia, especially, I think, when it is apical ; but to this I shall return when I come to speak of meningitis.

The **treatment of broncho-pneumonia** is exactly the same as that of acute bronchitis, *plus* the administration of respiratory stimulants such as strychnine, and of general stimulants such as alcohol, if they are indicated ; otherwise you do exactly as in acute bronchitis, keeping the child in a uniform temperature, and using a tent and steam inhalations and counter-irritation to the back, and also giving, if you like, small doses of expectorants. In addition, as I say, if there be signs of respiratory failure, you will use strychnine and alcohol, and you may require to employ inhalations of oxygen. The treatment of *croupous pneumonia* in children may be summed up in a word. There is no occasion to treat the case beyond putting the child to bed and generally nursing it. I usually treat them as out-patients, unless they are severe cases, and tell the mothers to take them home and keep them in bed, and sponge them if they are hot and restless. You may also, if you like, give a diaphoretic mixture.

I have said that croupous pneumonia in childhood is by no means a fatal disease. That, however, is only true so long as it is uncomplicated. The chief **complications** you have to dread are pericarditis, empyema, meningitis, middle-ear disease, and pneumococcal arthritis. In chil-

dren above the age of two such complications are not very frequent, but in infancy there is a distinct tendency for general pneumococcal infection to take place resembling the pneumococcal septicæmia which can be produced experimentally in rabbits, and for one or other of the above complications to make its appearance. If fever is prolonged, therefore, or if the temperature rises again after the crisis, examine the child carefully for signs of disease in the above situations.

ATELECTASIS.

Collapse of part of the lung, or 'atelectasis,' as it is sometimes termed, is a condition which you will meet with not uncommonly in children. It is sometimes *congenital*—a persistence, in part of the lung, of the fœtal condition — and is here associated with the symptoms spoken of as asphyxia neonatorum. Frequently, however, it is *acquired*, coming on in the course of other pulmonary diseases. Softness of the chest wall greatly facilitates its production ; hence atelectasis is more frequently met with in rickety children than in others. Anything which compresses the lung—*e.g.*, an effusion into the pleura or pericardium—may induce it, and it is stated to result also from blocking of the smallest bronchioles, either from the accumulation of secretion or from swelling of their lining membrane. This may explain the common occurrence of areas of collapse in the course of a broncho-pneumonia.

You will find the **physical signs of collapsed lung** rather puzzling. If extensive, the note over the affected area will be impaired and the breath-sounds feeble or even slightly bronchial in character. Occasionally, also, a few moist sounds may be heard. The general symptoms,

as you can readily imagine, will vary with the amount of pulmonary tissue involved. In slight cases they may be almost absent, but if the collapse is extensive there will be a greater or less degree of cyanosis, and you will notice that the child breathes and cries feebly, and that the extremities are apt to be cold.

In the **treatment** of this affection you must aim at promoting efforts at inspiration, so as to reinflate the collapsed areas. This can best be done by sprinkling cold water on the chest and by friction with stimulating liniments. Some writers advocate vigorous spanking, repeated, if necessary, several times in the day ; and though such treatment may sound brutal, it is certainly rational, for crying necessitates deep inspiration.

PLEURAL EFFUSION.

Pleural effusions are not uncommon in children, and are apt to be overlooked. Here, again, physical signs are of doubtful value. There will be dulness 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 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.

In a previous lecture I have already referred to **pulmonary tuberculosis** in children, but I should like to emphasize again the fact that phthisis, as one sees it in the adult, is very rare in early life. In young children, as we have already seen, pulmonary tuberculosis usually starts in the bronchial glands, and invades the lung secondarily. I would also remind you once more of the great difficulty of diagnosing between some forms of tuberculosis in the lung and chronic broncho-pneumonia ; over and over again you will be likely to make mistakes in this matter, for the physical signs of the two affections may be identical.

The **fibroid form** of tuberculosis is not uncommon even in young children, but here, again, you must remember that pulmonary fibrosis consequent upon an empyema or an imperfectly resolved broncho-pneumonia, or even upon collapse, is also frequently met with. Owing to the absence of sputum, too, your difficulties are rendered greater than in the adult, for you are deprived of that aid to diagnosis, and often you can only come to a correct conclusion by a prolonged study of the case.

LECTURE XII

SOME FUNCTIONAL NERVOUS DISEASES OF CHILDHOOD

GENTLEMEN,—In this and the succeeding lecture I propose to study with you some of the functional nervous disorders of childhood, which make up a group of diseases of great pathological interest and much practical importance. When you go into practice you will be struck by the frequency with which functional disease of the nervous system exhibits itself in your younger patients. One great 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 yet 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. But more important, because more frequent than a neuropathic heredity, are two factors which by universal consent are admitted to play a large part in the production of the group of diseases at present under consideration.

These are in young children *rickets*, and in older children *rheumatism*. **Rickets and rheumatism** are the predisposers *par excellence* to all forms of functional nervous disease. Why should rickets render the children who are the subjects of it more liable to suffer from nervous trouble ? I think it is probably to be attributed to the fact that rickets is, after all, only a form of malnutrition, and all forms of malnutrition tend to affect the highest centres first ; hence the cortex is weakened, and its control over the lower centres diminished. How rheumatism predisposes to these diseases I do not know, but one knows as a clinical fact that rheumatic children are much more subject to functional nervous disorders of all sorts than those who are not similarly affected. I would take chorea as an extreme example of that tendency. Chorea is only a functional disorder of the brain, of some part of the cerebral cortex, and it is almost invariably associated with rheumatism. Now, chorea is a functional disorder which you are not likely to forget, because it is a dramatic disease and advertises its presence ; but I want you to remember that what is true of chorea is true also of other less conspicuous nervous disturbances—namely, that they are specially prone to occur in children of rheumatic tendency.

Another point I want to insist upon before I proceed to deal with the different neuroses in detail is the question

of **inco-ordination.** When you study those nervous diseases 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 be surprising to 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. A young child 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 disease 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 recognise 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 essentially in an inco-ordination of the muscles of the larynx. It is due to a spasmodic closure of the glottis, rendering the child for the time being incapable of breathing. Here you will almost invariably find that the child is suffering from rickets, that great predisposer to nerve disease. 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 ; and these are the characteristic signs of laryngismus stridulus. These attacks will come on more in the night and in the early morning than at any other part of the twenty-four hours ; 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. What are you liable to mistake laryngismus for ? 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 in these cases will keep you right in the matter of diagnosing between those two conditions. Next you will possibly 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 an attack when it is in progress ? What you have to do is to try to promote the taking of an inspiration ; and this you can perhaps 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 if you are able to do it yourself, or if there is some responsible person to whom you can entrust the administration. That will generally cause the spasm of the laryngeal muscle to pass off and re-establish the 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, but 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 recognise the rickety element and to treat it by a change of diet, by cod-liver oil, iron, and so on ; and it is only in order to give those measures time to take effect that you meantime give bromides and chloral.

CONGENITAL LARYNGEAL STRIDOR.

I wish next to consider briefly the condition called *congenital laryngeal stridor*, which I told you might be mistaken for larnygismus stridulus. Congenital laryngeal stridor is a disease the **pathology** of which is still under dispute. It is believed by some to be due, like laryngismus stridulus, to an inco-ordination in the larynx. According to this view, it might be described as a stammer of respiration. When one makes an inspiratory effort, if the muscles are properly co-ordinated, the vocal cords fly apart to allow the air to pass in. And those who hold this theory maintain that in congenital larnygeal spasm this does not happen.

The child raises his chest, but does not at the same moment open the larynx, and it is only a second, or fraction of a second, later that the cords fly apart, and so there is a little catch as the air comes in. The other view—which is held by Dr. Lack, of this hospital, amongst others—is that you have to deal in congenital laryngeal stridor with a malformation of the larynx, consisting in a curving in of the epiglottis and flabbiness of the false vocal cords, which allows the larynx to fall in upon itself during inspiration, and that it is in that way that the symptoms are produced. I shall not attempt to decide between these two views, but would say that it is admitted by all parties that the disease is congenital, and that towards the end of the first year of life it tends to disappear quite spontaneously. So far as I know it has never proved fatal. What are its **symptoms?** The child will be brought to you 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 cat make. This sound goes on more or less continuously, but is apt to be louder if the child gets excited or agitated. During sleep the noise 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, but of this I shall have occasion to speak again (Lecture xix.). Inasmuch as congenital laryngeal stridor tends to disappear towards the end of the first year, it is not necessary to pursue any active **treatment.** I cannot satisfy myself that drugs have any effect upon it.

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 forearm 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 **causes** of this condition are two in number. In children of about eighteen months old it is very commonly associated with rickets. In younger infants you will find it coming on after an attack of diarrhœa. What the cause of the spasm is is not known. Sometimes you meet with a similar disease in grown-up people. It is one of the

curious and rare complications of dilated stomach. It is assumed that in such a case you have to do with chronic poisoning, the result of the absorption of toxic material from the decomposing contents of the stomach. Arguing from that, some people say that tetany in young infants is produced in a similar way. It may be so, and its association with diarrhœa is suggestive ; but there is no proof of it. Tetany is not a fatal disease, but it is a painful one. You must first of all treat the rickets, if there be any, and, whilst your anti-rachitic treatment is taking effect, you must administer nerve sedatives. And of those I would recommend the bromides and chloral in preference to antipyrin. Hot baths are also useful, for they tend to relax the spasm when it is very severe.

HEAD NODDING.

Lastly, I want to speak of a disease which is of considerable interest and rarity, and that is *head nodding*. This condition of head nodding has several synonyms. Sometimes it is called spasmus nutans, sometimes gyrospasm, and sometimes it is spoken of as simply rotatory head spasm, or head jerking, or head rotation. Either head nodding or spasmus nutans is the best term to use. In it you have to deal with slight continuous movements of the head, which tend to occur in young children who are usually about a year old or a little older. When the child is brought to you the mother points out that the head is kept moving. 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 cases 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 rotary 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 incoordination 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 miner's 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 steadfastly at you, you will find that the nystagmus increases and the head commences to nod. If the nystagmus is present in one eye only, the movements sometimes cease when that eye is covered up. This condition tends to occur with marked frequency at one part of the year—namely, in mid-winter. On a chart on the wall there are set out all the collected cases of head nodding which are on record,* and you will observe that the maximum

* From a paper by Dr. John Thomson in Jacobi's 'Festschrift.'

frequency is in the month of January. In the other months you will see there are hardly any cases at all. Why is head nodding 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. And some of you who saw a recent case of the disease in the out-patient room will remember that the mother admitted that the child lived in a very dark room on the ground-floor; and it is under such conditions that the disease most commonly appears. That is why people have compared this form of nystagmus with that known as miner's nystagmus, which is due to the constant strain of using the eyes in comparative darkness. Now, although that explanation is ingenious, and has much to commend it, 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*. But head nodding is associated with nystagmus, and occurs particularly at one time of the year, and tends always to pass off after a short time. There are certain automatic movements which idiots are apt to make which you may also 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 disease 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.

The **treatment** which is most likely to cut short the movements and produce improvement is the administration of small doses of antipyrin—1 grain for every year of life. This can be given two or three times in the day. I think I have seen this drug effect a distinct and decided improvement in the movements, rendering their occurrence less frequent and less marked ; but the nystagmus seems to be more difficult to control.

LECTURE XIII

SOME FUNCTIONAL NERVOUS DISEASES OF CHILDHOOD—*continued*

GENTLEMEN,—In the last lecture we began the consideration of a group of functional nervous disorders in children, the essential nature of which, we saw, was that they consist in an inco-ordination of movement. We saw, further, that there were several reasons why children are particularly subject to such inco-ordinate action of muscles, one of the chief being that, in the case of young children, at any rate, co-ordination is a recently acquired function of the nervous system, and therefore is one which is very easily lost under adverse conditions. We saw that amongst such adverse conditions must be placed many diseases of nutrition, rickets being one of the most conspicuous. In addition, it was pointed out that in older children rheumatism plays a part in the production of functional nervous disease, and that the factor of heredity can never be altogether ignored. We reviewed some examples of functional nervous diseases, taking up first the diseases of the larynx—congenital laryngeal stridor and laryngismus stridulus — and then we passed on to tetany and head nodding. I wish now to conclude this subject with you, beginning with the important question of convulsions.

CONVULSIONS.

You will meet with convulsions in children very frequently in your practice, and they are very alarming to the parents of the child. And, further, from the point of view of the medical man, their exact causation is often difficult to diagnose. I want first of all to warn you that mothers and nurses will call by the name of convulsions many things which really have no right to be so called. They will tell you, for instance, that the child is suffering from 'inward convulsions,' or 'inward fits,' or 'screaming convulsions.' And they may mislead you into thinking that you have to do with a case of genuine convulsive disease. Many children under the irritation of teething or colic, or under emotional disturbance, exhibit twitchings of muscles, particularly muscles of the face, which the parents are apt to suppose are true convulsions. But you are not to regard mere quiverings of eyelids and screwing up of the mouth, or violent fits of screaming, as genuinely convulsive, though they may be of value to you as warnings that a tendency to convulsions exists.

You will find it convenient to divide genuine convulsions into two main groups—first, those which are *idiopathic;* and, secondly, those which are *symptomatic.* By **idiopathic convulsions** one means those for which one can find no cause, which arise in a child who is apparently in fairly good health, and which may pass off after a time, or which not infrequently become established, so that the child suffers from fits from that time onwards at varying intervals. There can be no doubt that a considerable number of those cases are examples of genuine epilepsy. Epilepsy may certainly begin in early life, so

that when you are called to a child who is suffering from violent convulsions for which you can find no cause, you must bear in mind that you may be dealing with the first stage of that disease. Sir William Gowers puts the proportion of cases of epilepsy beginning as convulsions in infancy at 10 per cent., and some authors give an even higher figure.

Far more important, because more common, are cases of the symptomatic group. In the first place, you may have **symptomatic convulsions** as the equivalent of a *rigor* in a grown-up person. Many young children, instead of having a rigor at the outset of an acute illness, have actually a general convulsion. This may happen at the commencement of pneumonia, or scarlet fever, or any of the other acute specific fevers, where the temperature runs up rapidly during the first few hours of the illness. Secondly, symptomatic convulsions may be an indication of *organic disease of the brain*. They may occur in meningitis in all its forms, or they may be met with in cases of cerebral tumour, or with thrombosis of cerebral veins. Indeed, in almost any serious intracranial disease in children you may find convulsions as one of the symptoms. Thirdly, convulsions may be of *reflex* character or origin, an expression of the reflex irritation which arises from such things as the cutting of teeth or the existence of colic or phimosis. The reflex irritation which results from these causes may, as it were, 'fire off' convulsions from the cerebral cortex. There is another group which is *toxic* in origin. Uræmic convulsions are a typical example of this group; and I think it is not unlikely that some of the cases of convulsions which you meet with in the course of acute diarrhœa can also be attributed to toxic causes. Lastly, there are the

convulsions which are the result of *asphyxia*. Such are those which you may sometimes meet with in the course of pertussis. Towards the end of any illness also convulsions may come on and be, so to speak, one of the modes of dying. These also are probably due to failure of respiration or of the heart, leading to cyanosis and congestion of the brain.

Those are the chief groups into which you can divide cases of convulsions; and when you are called to a case you have to decide, as far as you can, to which one of these classes it belongs.

You will find that, just as in many of the other functional neuroses we spoke of last time, so here **rickets** is the great predisposing factor. For instance, the cutting of teeth in a child in good health may not set up convulsions, but if the child be rickety, such irritation may be a determining cause of spasmodic discharges from the cortex of the badly-nourished brain. The same is true of the other causes of which I have spoken. Many of them are insufficient by themselves to produce convulsions in an ordinary child, but where rickets has produced nervous instability they may suffice as determining causes. You can sometimes perceive that convulsions are about to come on by certain warning signs. One of these is the development of the facial irritability of which we spoke last time, and which we saw to be an indication of undue irritability of the nervous system. Another will be slight twitching movements of the face and extremities. If these twitchings are seen in a child the subject of rickets you ought to anticipate that the child may become affected by convulsions, and do what you can to ward them off. Sudden flushings or attacks of coldness are also occasional premonitory signs.

When you consider the **prognosis of convulsions** you have to take into account what the immediate chances are, and also what is likely to be the remote outcome of the seizure. Children do not often die in a convulsion, unless the convulsions are themselves merely the final scene in an already mortal complaint—that is to say, unless they are simply a symptom of the terminal asphyxia. Convulsions as a disease are not immediately fatal, unless when very severe and repeated and in quite young infants. You have to remember, however, that in a considerable proportion of the cases the convulsions may persist in the form of epilepsy ; and you have, further, to remember that nearly all, or quite a large number, of the children who suffer from convulsions manifest in later life nervous disorders of different sorts. Of about eighty-five cases followed up by Dr. Coutts and traced into adult life it was found that eleven suffered from major epilepsy, four from *petit mal*, five exhibited somnambulism, four developed melancholia, seven had chorea, and nine suffered from migraine. So you see in what a large proportion of those cases the later history of the patient is marked by further nervous disturbance. In addition to this, convulsions, especially if frequently repeated, may cause permanent mental impairment, and they often leave behind, too, traces of cerebral damage in the form of stammering or a squint.

With regard to **treatment,** your immediate line of action when called to a child who is at that moment the subject of a convulsion must be the same, no matter what the cause of the attack may be. First of all, you lay the child down, raising the head somewhat, and loosen the clothing just as you do in a case of epilepsy, so that there is no hampering of respiration. And it is a good thing, unless

you have been anticipated by the mother or nurse, to put the child into a warm bath, though this is perhaps most efficacious in those cases which are the result of colic. Next you should give sedatives by the mouth, or, if you are unable to get the child to swallow, by the rectum. Chloral is the most efficient, 3 or 5 grains by the rectum to a child six months old. If you fail, and the convulsions are very serious and still persist, and the friends are getting alarmed, there is one resource which is open to you, and which I think is practically certain to be successful—namely, the inhalation of chloroform. A few whiffs of chloroform given on a handkerchief will cause the convulsions to cease by paralyzing the brain cortex. In bad cases in which the child passes from one fit into another you may require to give morphia hypodermically, but it must be used with caution. Amyl nitrite also is sometimes of service. Such will be your immediate treatment in all cases. Next you have to consider how you are to prevent the recurrence of the attacks. In order to do that you must treat the rickets, if there be any. You must make a radical alteration in the child's diet and administer cod-liver oil. But occasionally, and until these measures have time to take effect, you may have to diminish the excitability of the nerve centres by sedatives. Bromides and chloral will be the drugs which you will use. I would remind you again here how well young children stand chloral ; it is a drug which you may give pretty freely without risk. And in those cases it is to be preferred to bromide, as being more efficient in controlling the convulsive tendency. It may be necessary, of course, to attend to the child's digestion if there be colic, and that may involve the alteration of the diet in directions I have laid down in a previous lecture.

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 occur with a considerable degree of frequency in children, and they are often extremely alarming to the friends. The usual history of a case is that a child, most commonly 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. In those cases the cause of the fright is sometimes an hallucination of vision ; sometimes, on the other hand, it is probably merely a nightmare. It has been said, I think by an American writer, 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 extremely difficult to say to which of those two classes any given case belongs, because these children are often incapable, sometimes 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. In some cases they can give you no coherent account of their sensations. These experiences may be repeated night after night, until the equilibrium of the nervous system is seriously upset,

and the child begins to dread going to bed at all; and then the friends get alarmed, and think that the child is becoming insane, and call in a doctor.

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 candle-light and the unwholesome hours, as they are called, would, I am satisfied, in a medical point of view, prove the better caution.'

Now, gentlemen, night terrors may always be taken as evidence of ill-health in some form or other. They do not occur in perfectly sound children. And you will find that amongst the **predisposing causes** particularly at work are, in the first place, a neurotic or nervous heredity; secondly, you will find a rheumatic diathesis, as I have already stated. But above all, and far more important than all, perhaps, is overpressure at school. One of the chief

predisposing causes of night terrors is school work, and you will often in many cases 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 of the very first importance for you to be familiar with. One of them is gastro-intestinal disorder, especially *constipation*. The second is partial asphyxia from the presence of *adenoids* in the throat. These two causes are by far the most important for you to recognise from a practical point of view.

When I have mentioned to you the causes of night terrors, you see what the **treatment** should be. It must not be purely sedative. There are two things above all that you should do when called to a child who suffers from this complaint; one is to attend to the state of the throat, and the other is to administer aperients. If you do these you may leave almost everything else undone. Clear out adenoids when you find them, and give an aperient no matter what the state of the bowels may be or is said to be. If you do that you will not go far wrong in the management of those cases. There are other accessory means which are of help. These children ought not to be frightened in any way, nor excited. They ought not to be allowed to sleep alone, they ought to have a night-light in the room, and ought not to be allowed to go to bed with a full stomach, because, as in nightmare in grown-up persons, so here, pressure of food in the stomach may be a cause of the condition.

It may be well to remove the child from school for the time being, until the nervous system has recovered its equilibrium. It may be necessary to give sedatives, such as bromides and chloral, temporarily; but as far as possible you should dispense with these and direct your attention to putting the child into that state of health in which night terrors are impossible, and in order to do that, as I have said, you must pay attention to the state of the throat and stomach and bowels.

There is an analogous condition to night terrors which occurs in the day, called day terrors, or **pavor diurnus,** which is very 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. These are no doubt to some extent analogous to the worst form of night terrors. They are probably cases where there has been some 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 forms is **grinding of the teeth.** Such cases can be recognised by the fact that the teeth are bevelled 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 last neurosis I shall describe is enuresis, or *nocturnal incontinence.* I do not pretend to explain how it is that enuresis arises. The exact mechanism of the bladder is not fully understood, but for practical purposes you may



FIG. 21.

TEETH-GRINDING. NOTE HOW THE UPPER TEETH ARE WORN DOWN TO A LEVEL.

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 until some time after that.

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 worms, because the irritation of worms in the rectum seems to be a cause which may give rise to enuresis. 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. Here also you will find in a great many cases that partial asphyxia is the cause of the trouble. And adenoids, again, are by far the most common cause of the partial asphyxia. 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 condition

of the urine, to see if it be acid to an undue extent. I think hyperacidity of the urine as a cause of nocturnal incontinence has been exaggerated. I do not think that it is a common cause. If, however, you find uric or oxalic acid crystals, it is well to give alkalies to make the urine less irritating. I have even seen calculus in the bladder mistaken for incontinence, from the irritation and 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 shall 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 disease. There is only one rule to be remembered in regard to the **administration of belladonna,** and that is *to give it in large doses*. If you do that, you do not need to trouble about any other points. Begin with 10 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 15 minims each dose, then to 25 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, even of the new B.P. strength, after each meal—that is to say, 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. As a rule, there are hardly any general toxic effects from large doses, whereas the local effect upon the enuresis is quite remarkable. 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 ; climb down again in the matter of doses just as you climbed up. You will then find that in the majority of 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 has cantharides. I have tried all of them, I think, and I can only say that I am by no means certain that they are of much use. I should say that if you fail with belladonna you may regard yourself as at the end of your therapeutic resources so far as drugs are concerned.

I think you will find nocturnal incontinence of urine is much more easy to cure in boys than in girls. I do not know why that should be, but I think the most intractable cases one has to deal with are in girls. In such cases treatment by local applications of nitrate of silver to the urethra has recently been recommended, a strong solution being applied on more than one occasion. I have seen one case at least where this method effected considerable

improvement, but my experience of the treatment has not been great. Still, it is one which it is well to have in reserve in the case of girls, for whom alone it is intended.

Finally, I would warn you that you must see that these children are not punished. A case which has recently excited much attention in the newspapers was an instance in which the mother punished the child because it suffered from nocturnal incontinence. It never does any good, or hardly ever, to do so. Very few cases are due to carelessness on the part of the child ; the disorder is due to a want of control of the lower centres by the higher, and the lack of control is not a voluntary one, but purely involuntary. It is a case in which the child cannot exert the will, and therefore it is not only useless, but it is extremely unfair and unkind to punish children for this unfortunate weakness. On the other hand, by carefully following out the treatment I have described, I think it will be your experience that in the majority of cases you can get the patient well.

Occasionally the 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 symptoms 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.

LECTURE XIV

THE PARALYSES OF CHILDHOOD

I. PSEUDO-PARALYSES.

GENTLEMEN,—When a child is brought to you who is alleged to be suffering from 'paralysis,' the first thing you have to determine is whether the loss of power is really due to some affection of the muscles or nerves, or whether 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-paralysis' 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. This case presented unusual difficulties, but, as a rule, you will readily 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 never 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 anti-syphilitic treatment. 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 this condition. The extreme tenderness of the limb when handled should usually put you on the right track, and often you will find also some



FIG. 22.

CASE OF DIPLEGIA, SHOWING GENERALIZED RIGIDITY AND INTRA-UTERINE POSITION.

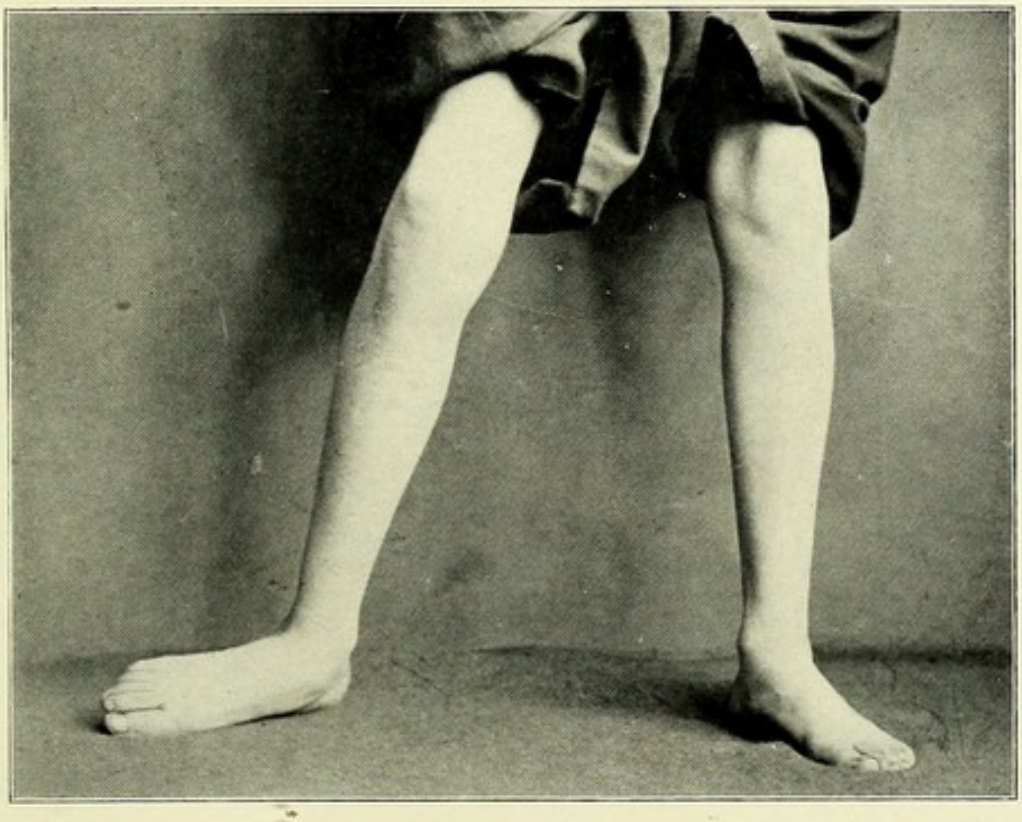


FIG. 23.

HYSTERICAL PARALYSIS IN RIGHT LEG, SHOWING HOW THE FOOT IS DRAGGED.

degree of 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's 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 also 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 simulated 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.

II. TRUE PARALYSES.

Assuming that you are satisfied that you have to deal with a case of true and not of simulated paralysis, the next thing you have to determine is the **seat of the lesion.** In this diagram (Dia. 9) 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 lesion affects the upper or lower motor segment. This is not difficult to do if you remember that upper segment lesions are characterized by spasticity, with exaggeration of the deep reflexes and absence of muscular wasting, whereas lower segment lesions exhibit flaccidity, with diminution of the deep reflexes and the presence of wasting. We may therefore consider first the lesions of the upper segment, 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.

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*).

The **natal cases** are due to an injury to the brain during parturition. The nature of the injury is often obscure, but one may safely regard it, in many cases at least, as being due to a meningeal hæmorrhage. Very commonly 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* (**post-**

* 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 till the child tries to walk.

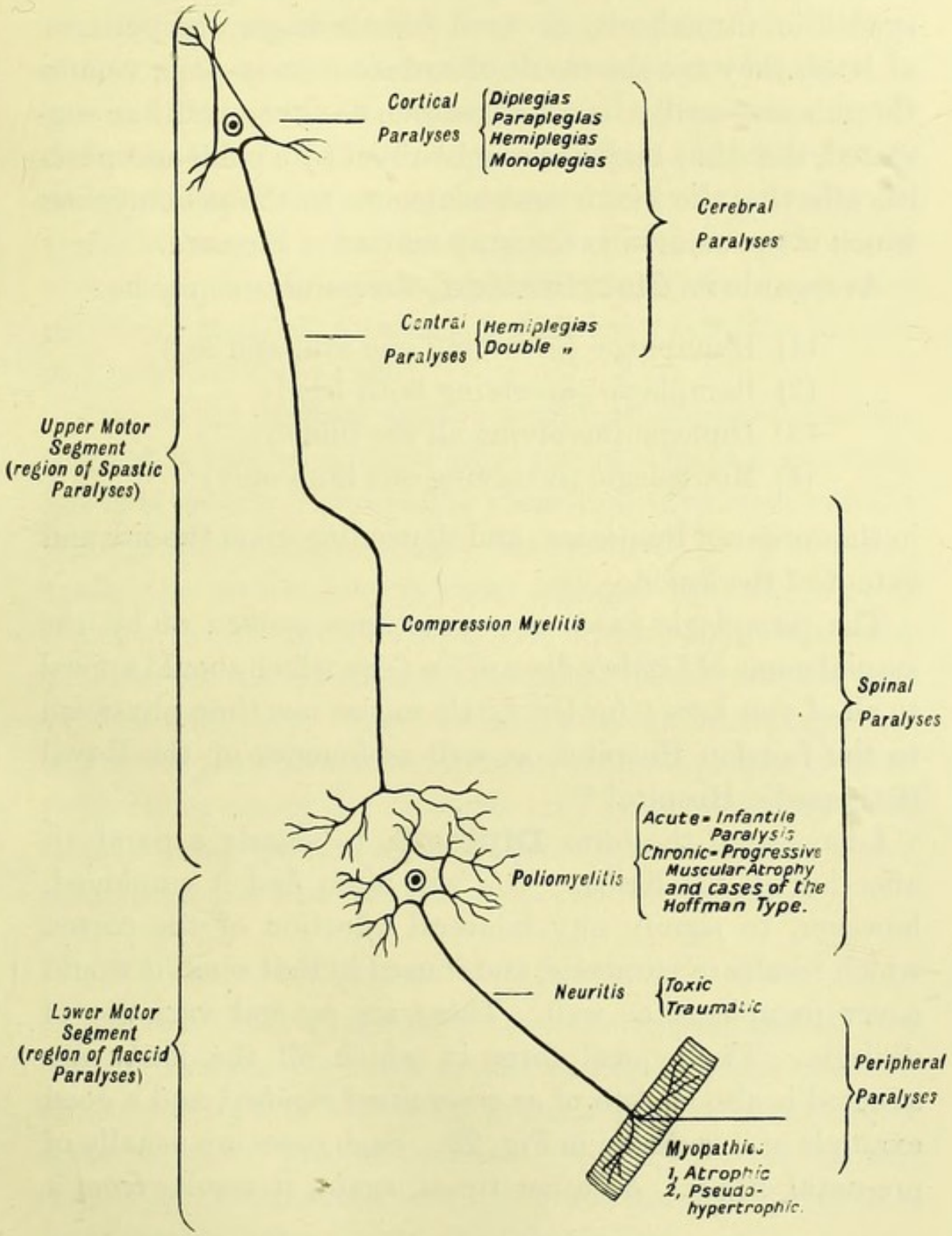


DIAGRAM 9.—TO SHOW THE SITE OF THE LESION IN THE PARALYSES OF CHILDHOOD.

natal) are usually hemiplegic, and may be due to the same causes as produce hemiplegia in adults—*e.g.*, embolism, syphilitic thrombosis, or even hæmorrhage. Sometimes, at least, they are the result of surface lesions—*e.g.*, venous thrombosis—and it is also possible, as Strümpell has suggested, that they may be brought about by a polio-encephalitis affecting the motor area analogous to the poliomyelitis which is so common in the gray matter of the cord.

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—a term which should appeal to all of you here ; for Dr. Little was at one time physician to the London Hospital, as well as founder of the Royal Orthopædic Hospital.*

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 limbs are affected is also spoken of as *generalised rigidity*, and a good example of it is shown in Fig. 22. Such cases are usually of pre-natal origin. At other times, again, it results from a

* Little's original paper was published in the Trans. of the Obstetrical Soc., vol. iii., p. 297, 1861.

progressive degeneration of the cortical cells coming on some time after birth—a condition sometimes described as one of 'premature senility' of the neurons. 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 probably of syphilitic origin. To the diplegias also belong the cases of amblyopia with mental deficiency sometimes met with in Jewish families, to which I shall have occasion to refer in a later lecture (p. 255).

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 of post-natal development, the paraplegias of natal production, whilst the diplegias may be of either pre-natal or post-natal appearance. Monoplegias are so rare that I cannot tell you what is their usual time of appearance.

Complications of the Spastic Palsies.

You will find, gentlemen, 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 sur-

prised to find that any severe and 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, one finds that 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 mere slight degree of ' eccentricity ' to a condition of absolute imbecility (see Lecture xvi.).

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 set in 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 in such cases are typically epileptic in type, and often occur with great frequency and severity, and are of special importance, because there can be no doubt that 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

very carefully for signs of spinal caries, particularly in the cervical and upper dorsal regions, for **compression myelitis,** as it is called, is not an uncommon sequence of spinal caries in these regions. Here, again, it is the upper motor segment which is involved—*i.e.*, the motor fibres on their way to the anterior coronal cells of the lower dorsal and lumbar parts of the cord—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 the 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, overdistension 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 usually 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. Rest, indeed—absolute and prolonged rest—is the keynote to their successful management.

Lesions of the Lower Motor Segment.

The most typical form of paralysis which results from a lesion of the lower motor segment is **infantile paralysis,** which is, as you know, the consequence of

anterior 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 but little difficulty in recognising a case when it is brought to you. The flaccidity of the limb, the loss of the deep reflexes, the wasting of the muscles, and the presence of the reaction of degeneration, all proclaim it at once as due to a lesion of the lower motor segment, 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 recognise, 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 anterior poliomyelitis is often marked by but little in the way of general disturbance of health. A little 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 to see a boy of five by one of my colleagues here, 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 paralyzed, but whenever 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 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.

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.

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.

Chronic anterior poliomyelitis leading to progressive muscular atrophy is a rare disease in childhood. Hofmann has described * a special type of it which occurs in families, and of which the case depicted in Fig. 24 was an example. This child, a boy four months old, was brought to see me at Great Ormond Street with the history that he had become gradually paralyzed within the last three months. The mother stated that an exactly similar fate had befallen other two of her 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 outside shortly afterwards, but, unfortunately, no examination could be obtained. Such cases are exceedingly rare, and belong rather to the curiosities of pediatrics, 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 are not very uncommon in childhood, and may be divided into the

* *Münch. Med. Woch.*, No. 43, 1900; and *Deut. Zeitsch. f. Nervenheilk.*, iii., 1893.

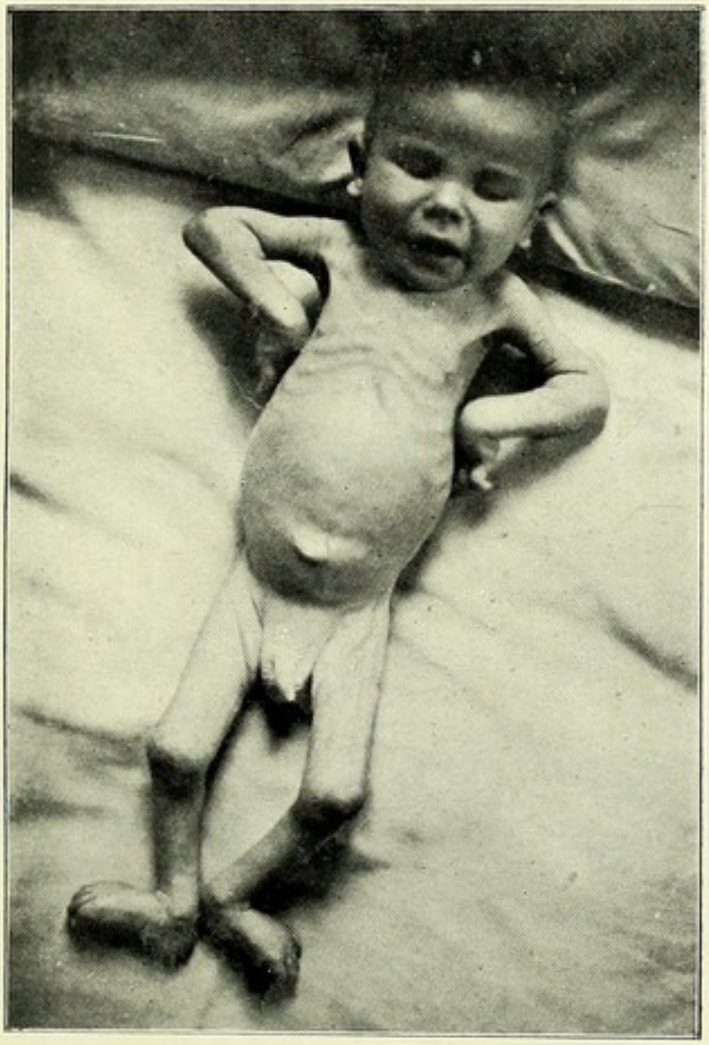


FIG. 24.

HOFFMAN TYPE OF PROGRESSIVE MUSCULAR ATROPHY, SHOWING GREAT WASTING OF ALL MUSCLES, ESPECIALLY OF THE INTERCOSTALS.



FIG. 25.
DOUBLE ERB'S PALSY.



FIG. 26.
ERB'S PALSY. NOTE CHARACTERISTIC POSITION OF RIGHT ARM.

two great groups of (1) traumatic, and (2) toxic. The best examples 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 Palsies.**—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 infra-spinatus, and teres minor). When these muscles are paralyzed, their opponents have it all their own way, with the consequence that 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 Figs. 25 and 26.)

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.

In a typical case the condition can be recognised 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 very 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. Usually it passes off very quickly, but Edgeworth has recorded * one bilateral case which was still very marked at the age of seven.

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

* *Brit. Med. Journ.* 1894.

adults as due to lead, alcohol, and arsenic you will hardly ever see in the child.

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 'rheumatic.' They are not difficult to recognise, for, besides presenting all the orindary characters of a lower segment 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.

Post-diphtheritic paralysis, which is by far the most important of the toxic neuritis group in childhood, I do not propose to speak of very fully, for its study is always taken up along with that of diphtheria. Its chief peculiarity, you remember, is its tendency to affect certain nerves more than others, 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, perhaps, 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 so 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.

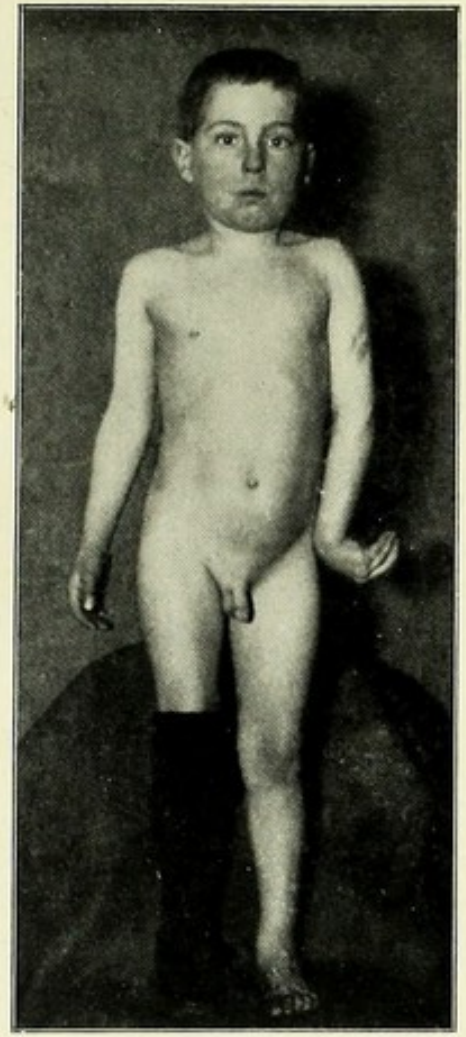


Fig. 27.
Left Hemiplegia, showing marked spasm of arm.



Fig. 28.
Left Hemiplegia, dating from birth.

Myopathies.

Peripheral palsies the result of affections of the muscles themselves are almost peculiar to childhood, and may be conveniently spoken of as 'myopathies. Whether the disease of the muscle in these cases is truly primary, or, as most neurologists are now beginning to think, is really the result of an affection of the nerve cells of the cord, is a recondite 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** is the classical example of the first group, and its peculiarities are so carefully dealt with in every text-book that I need not spend further time upon it.

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 have come on gradually at some period of childhood. The degree of atrophy varies, being in some cases extreme, and curious deformities of position may result, whilst the hands and feet sometimes look curiously long and thin. 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 them 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.

Hysterical Paralyses.

It is always rather difficult to realize that hysteria may occur in children at all. Yet it is not very uncommon in later childhood, and may occur in boys quite as easily as in girls. Although hysterical manifestations in childhood may assume the same protean form as in adults, we are concerned with them here only in their paralytic form. You will meet with two forms—**hysterical spasm** of muscles, and true **hysterical paralysis.** The former of these is really the result of an hysterical affection of joints of sensory origin, but it may closely simulate spastic paralysis from a cerebral lesion. In Figs. 23 and 29 two cases are depicted affecting the lower limb. It is the very ' exaggeration ' of the contraction in these cases which should put you on the true scent. Injuries to joints do not really lead to such extreme deformity.

True **hysterical paralysis** is much more deceptive. The following case illustrates the kind of difficulty one meets with. Some time ago a boy of twelve was sent up to this hospital from the country for an opinion. The history was that four months previously he had begun to

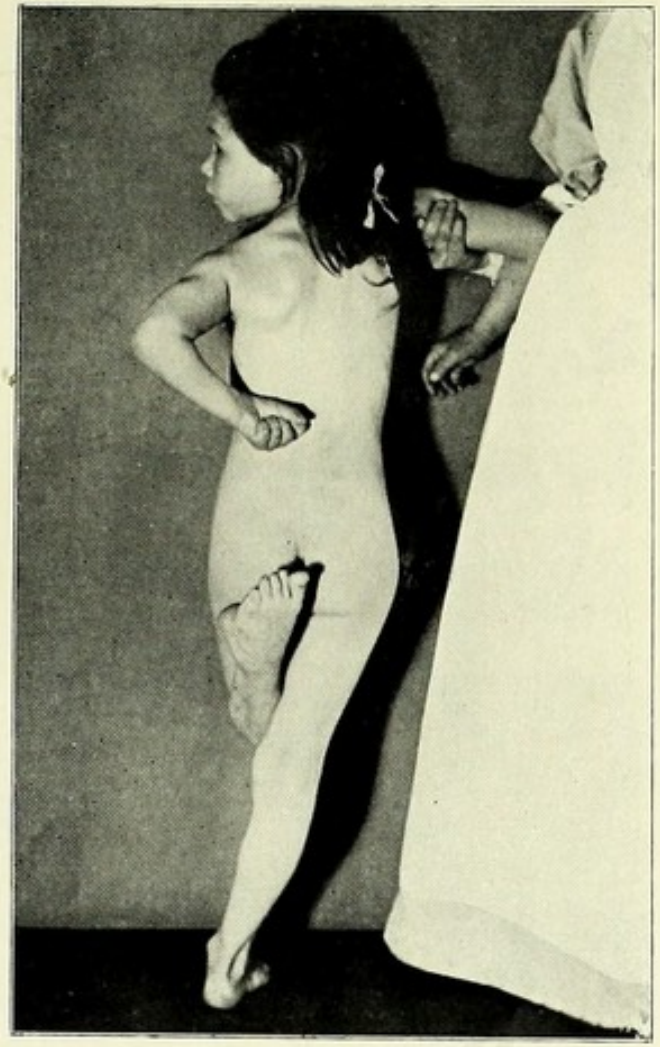


FIG. 29.
HYSTERICAL SPASM OF LEFT LEG.

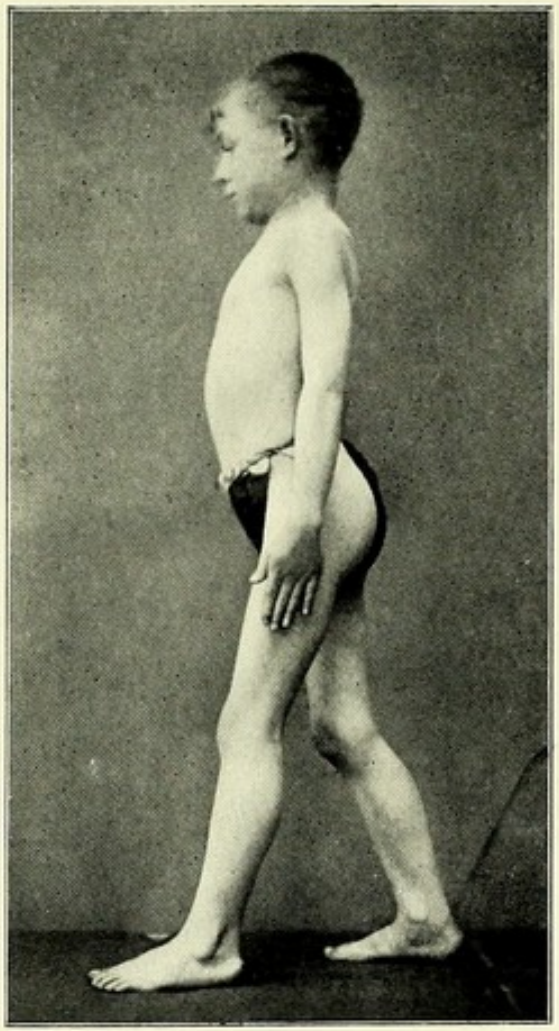


FIG. 30.
EARLY CASE OF PSEUDO-HYPERTROPHIC PARALYSIS, SHOWING ENLARGEMENT OF CALVES AND SLIGHT LORDOSIS.

suffer from prickling sensations in the fingers of the right hand, followed in three days by complete loss of power in the whole arm. He was seen by a doctor, infantile paralysis was diagnosed, and treatment by massage and faradism carried out, but without any improvement, and for several weeks he had been carrying his arm in a sling.

On examination he was found to be a healthy and intelligent-looking boy; his previous health had been good, and no cause was assigned for the onset of the paralysis. The right arm was quite motionless, and exhibited a considerable degree of atrophy, especially in the small muscles of the hand. It was blue and cold, just as in a case of infantile paralysis. On testing sensation, however, it was found that there was complete insensibility to touch, pain, and temperature, and, further, the anæsthesia *was of the sleeve type—i.e.*, it ceased abruptly at a circular line just below the shoulder. Hysteria was therefore diagnosed, and a strong faradic current applied with vigorous 'suggestion.' An emotional outburst followed, but power over the arm was instantly regained, and he went back to the country again next day perfectly well except for the atrophy from disuse.

In this case it was the existence of the anæsthesia and its characteristic distribution that enabled one to make a correct diagnosis.

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 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 splints or otherwise as you are accustomed to see used in spinal caries. Iodide of potassium in *large* doses is also strongly recommended by some writers in these cases. It perhaps acts by aiding in the absorption of the inflammatory material which is compressing the cord. Attention to the bladder is always imperative. I have known serious cystitis set up by a neglect of this important matter.

For **infantile paralysis** in its early stage you can do little, but later on, when the acute phase is over, something can be done to aid the restoration of the muscles by massage and electricity. The value of the latter, how-



FIG. 31.
INFANTILE PARALYSIS, AFFECTING RIGHT DELTOID.

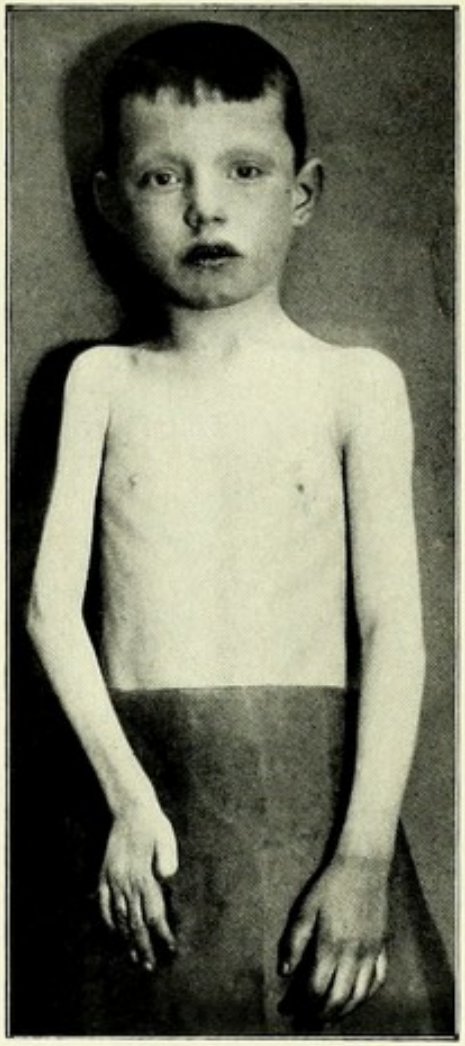


FIG. 32.
INFANTILE PARALYSIS, RESULTING IN SHRINKING OF RIGHT ARM.

ever, must not be overrated. 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 very apt to frighten the child.

The nutrition of the affected limb can be helped by warm covering and by douching with salt and water, followed by vigorous friction with a rough towel. 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 in infantile paralysis, you must again have recourse to surgical aid.

For **neuritis** in its early stage rest and symptomatic treatment must be your rule. Later on, electricity, massage, and strychnine will be of help. In the case of the special injury of nerves which causes **Erb's palsy** it has been recommended that if the lesion is recognised at birth the arm should be put up for a month in the same position as for fractured clavicle, in the hope that this may help the repair of the nerve-trunks. Later on it must be treated like any other neuritis. Some successful cases, in which the nerve-trunks were sutured in this form of paralysis, have recently been recorded,* and perhaps this method of treatment may become more widely used in the future.

* See Kennedy, *Brit. Med. Journ.*, February 7, 1903.

LECTURE XV

MENINGITIS

GENTLEMEN,—Meningitis is a disease which is comparatively common in childhood—much commoner, certainly, 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. I feel pretty confident that in the future, when our knowledge is more extensive and exact, we shall come to a bacteriological classification of the disease, though at present I have thought it advisable to abide by the recognised divisions. You must remember, however, that we already know that several different micro-organisms are capable of exciting inflammation of the membranes of the brain and cord. Chief of these are the *Diplococcus intracellularis meningitidis* of Weichselbaum (shortly known as the meningococcus), the tubercle bacillus, the pneumococcus, 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 irritability 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 (see p. 15), and, in extreme cases, by opisthotonus.

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, and dilatation of the pupils.

The special symptom known as **'head retraction'** is often one of the most prominent features in meningitis, and it is difficult to know to which of the above groups to refer it. The photograph (Fig. 34) will give you a better idea of it than any verbal description. Head retraction is probably a device which enables more fluid to be accom-

modated in the space between the medulla and cerebellum (the 'posterior arachnoid cistern'), and which prevents the inflamed arachnoid from being irritated by movement of the under-surface of the cerebellum over the top of the medulla. It is most marked in cases of so-called simple basal meningitis.

Diseases simulating Meningitis.

Now, gentlemen, you may take it as a general rule in medicine that the more numerous the possible symptoms of any disease are 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 very closely simulated indeed. 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 here 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.

2. **Pneumonia,** especially, I think, 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 even these may be absent in the first few days. The pulse-rate may here help you. In pneumonia it follows the temperature, whereas a combination of high fever with a comparatively slow pulse is much 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 (see p. 238).

5. **Acute 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, optic neuritis, 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.

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 to Hanbury

Ward 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 face, with a pillow under the abdomen so as to bend the spine a little, and open out the spaces between the laminæ of the vertebræ. Trace the last rib on the right side back to the twelfth dorsal 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

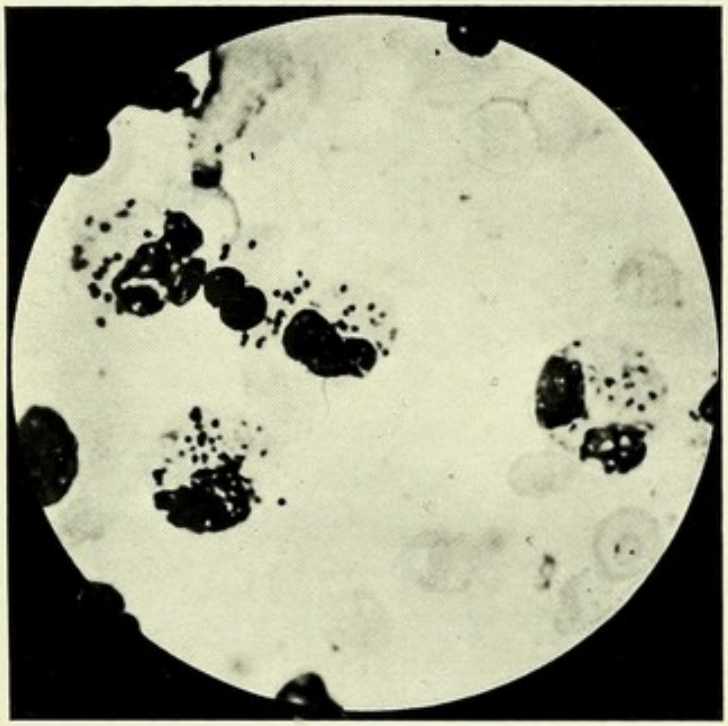


FIG. 33.

PHOTOMICROGRAPH SHOWING THE MENINGOCOCCUS EMBEDDED IN LEUCOCYTES. FROM LUMBAR PUNCTURE IN A CASE OF SIMPLE MENINGITIS.

may be so slight that it is only noticed when the tube is shaken and held up to the light. Examination by cultures, or by merely making stained films of the deposit, may reveal the presence of the meningococcus which may be recognised in films lying inside the leucocytes, (Fig. 33), 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.

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
2. Tuberculous.
3. Posterior basic (also known as 'simple basal,' and very probably only one form of sporadic cerebro-spinal meningitis).

Acute Meningitis.

Acute meningitis may either occur as a primary disease or be secondary to some other condition.

If it is **primary** it may either arise in epidemics—especially in the summer months—or may occur sporadically in isolated cases. In either case the symptoms are the same—the child is seized with high temperature, vomiting and headache, severe convulsions, and often opisthotonos supervene, and death usually results within a few hours or days. The organism responsible for the production of these cases is almost certainly the meningococcus, although it is

not quite certain that the pneumococcus may not be the cause in some cases.

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 recognise, and may, indeed, be only detected after death. I remember, for instance, having a child under my care for whooping-cough which suddenly developed a left-sided hemiplegia. Everyone who saw the case supposed, naturally enough, as there were no other marked symptoms, that a bloodvessel 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 quite often in the course of a pneumonia, and it may occur also in measles or in any of the acute specifics. 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 very difficult to recognise, though it is perhaps oftener ushered in by convulsions than by any other sign. Its recognition is fortunately not of importance from the point of view of treatment, for you can do nothing for it ; but it materially affects the prognosis, for secondary meningitis is probably always fatal, and usually within a very few days.

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 very 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 apparently 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 Whyte nearly one hundred and fifty years ago. He divided it 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 symptom of the disease will be considered immediately, when I come to describe its differential diagnosis from posterior basic meningitis.

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, however, 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 there is every reason to suppose that 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 of the ' posterior basic ' variety.

Of the **general symptoms** common to all forms of meningeal inflammation already detailed, some tend to exhibit 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. 34 shows this in a marked, but by no means unusual, degree, and you will recognise 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 symptom of the disease. Failure to lose weight, therefore, is always a sign that the malady is taking a favourable course.

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.
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.	Optic neuritis frequent.
Cerebro-spinal fluid shows the meningococcus.	Cultures sterile, or show the tubercle bacillus.

The **prognosis of posterior basic meningitis** is also very different from that of the tuberculous form, for whereas the latter may be regarded as almost inevitably fatal, the former is recovered from in about half the cases. 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** of greater or less degree 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.

Unfortunately, we know of no specific **treatment** for

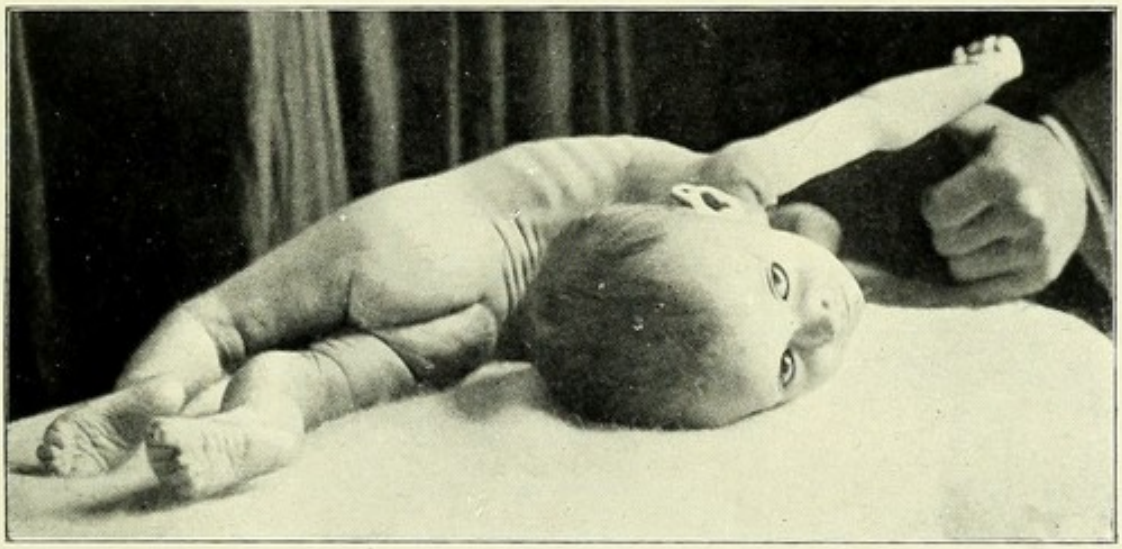


FIG. 34.

HEAD RETRACTION IN SIMPLE BASAL MENINGITIS, SHOWING "GUN-HAMMER POSITION." NOTE ALSO THE STARING EYES.

posterior basic meningitis, but you must not on that account conclude that you can do nothing for such cases. As the disease may be very protracted, careful nursing is of the first importance. If vomiting and inability to suck or swallow are persistent, nasal feeding must be carried out. Not long ago I had a case in one of the wards here in which the child had to be fed by the nose-tube for *four months*, but which finally made a perfect recovery—a result the credit for which rests mainly with the nursing-staff.

I am convinced, too, that **repeated lumbar puncture** is a therapeutic measure of great value in some of these cases, and I never hesitate to employ it wherever head retraction, bulging of the fontanelle, and repeated vomiting are prominent features. The relief to the intracranial pressure which the abstraction of one or two ounces of fluid produces in such cases is often very marked, and lasts as a rule for three or four days, when the operation can be repeated if need be.

From **drugs** I do not think you are likely to get any help, except in the treatment of special symptoms, but it is only right to say that some authorities believe in the free use of iodide of potash and mercury on the off-chance that congenital syphilis may be playing a part in the process. The later bacteriological studies of the disease, however, are rendering this chance always more remote.

LECTURE XVI

ON MENTAL DEFICIENCY IN CHILDHOOD

GENTLEMEN,—Our subject in this lecture is the mental deficiencies of children. This is a subject which deserves your very closest attention, because you will find when you go into practice that mental deficiency is not uncommon in children, 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 the children of school age are the subjects of it. But before I go further into this matter I want to define for you the **use of terms** in regard to mental deficiency. You will find, for instance, 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,' one would reserve it for the lowest grade of case—that is to say, those in which the mental impairment is of the extremest form. Above those, somewhat

better in their mental development, will come imbeciles, and higher still, nearer to the normal children, 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.

Coming closer to our subject, it will be convenient to look, first, at the general diagnosis of mental deficiency; secondly, at the clinical classification of cases of this affection; 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 to you.

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*. That 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 one of the early lectures. 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



Fig. 35.

Baby cretin. Note puckered forehead, puffy face. broad nose, protruding tongue, wrinkled skin and umbilical hernia.

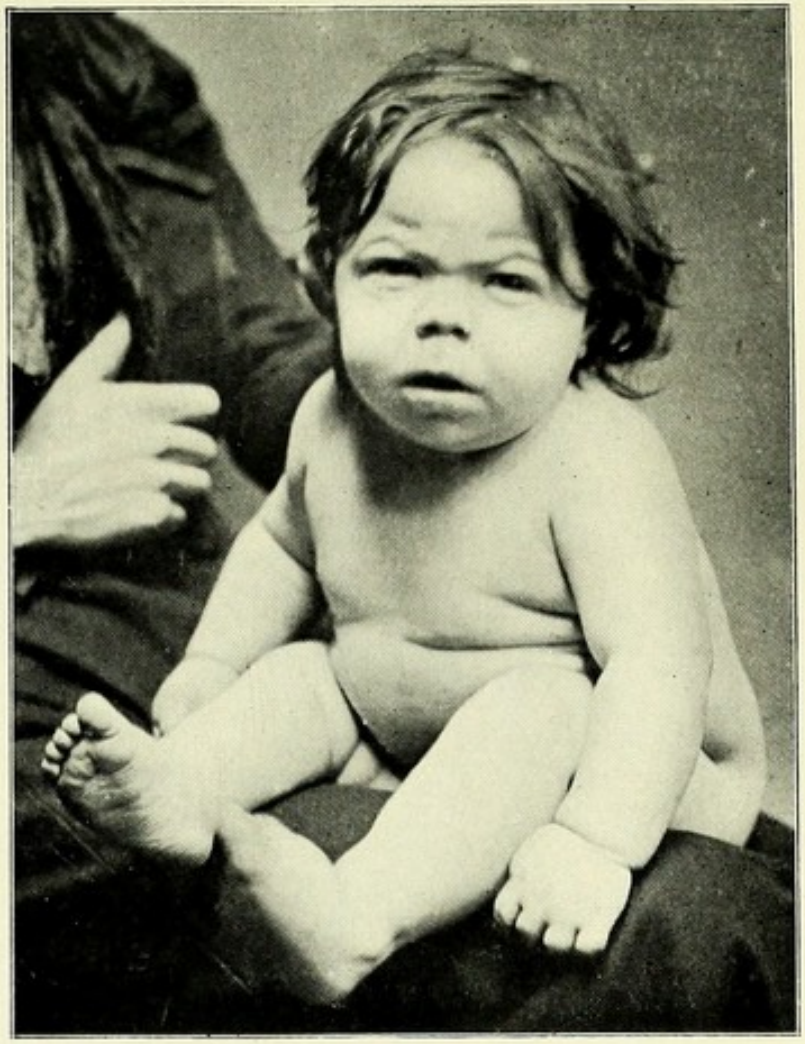


Fig. 36.

Cretin, showing characteristic facies.

able to sit up at the end of the sixth or the ninth month, 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 those co-ordinated movements which give rise to 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 suspicious. I refer to such things as hand-sucking and the making of meaningless gestures. 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 subdivision 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 recognise 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 have been described by Dr. Warner under what he terms '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 congenital lesions 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 naso-pharynx and bad development of the jaws, and therefore also of the teeth. Then you will find adherent lobes of the ears more commonly in mentally deficient children than in others, the lobe being tacked on to the skin of the cheek instead of being 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 an additional point 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 in children. One would prefer to use a pathological classification if that were possible, but at present it is not. The pathology of many cases of mental deficiency 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.

1. The first group of those are the **cretins.** It is extremely important that you should be able to recognise cretins, because they form that group of mentally defective infants which are the most amenable to treatment; and it will depend upon your being able to make the diagnosis within the first year of life whether the child will grow up mentally sound or whether it will be considerably defective. The recognition of a baby cretin is not always easy, and the classical pictures of cretins which you see in books are usually taken, unfortunately, from children several years old, by which time, of course, anyone can make the

diagnosis ; but if you recognise them only then, you recognise them too late to do anything of service for them. In order to do a cretin real good, you have to catch him early, and if you do so, and treat him properly, he will 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 recognise 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. I would impress upon you the fact that cretinism is a rare condition. Out of eighty-eight 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 28 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 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 recognise 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, and for this reason : that supraclavicular pads are not developed, so far as I

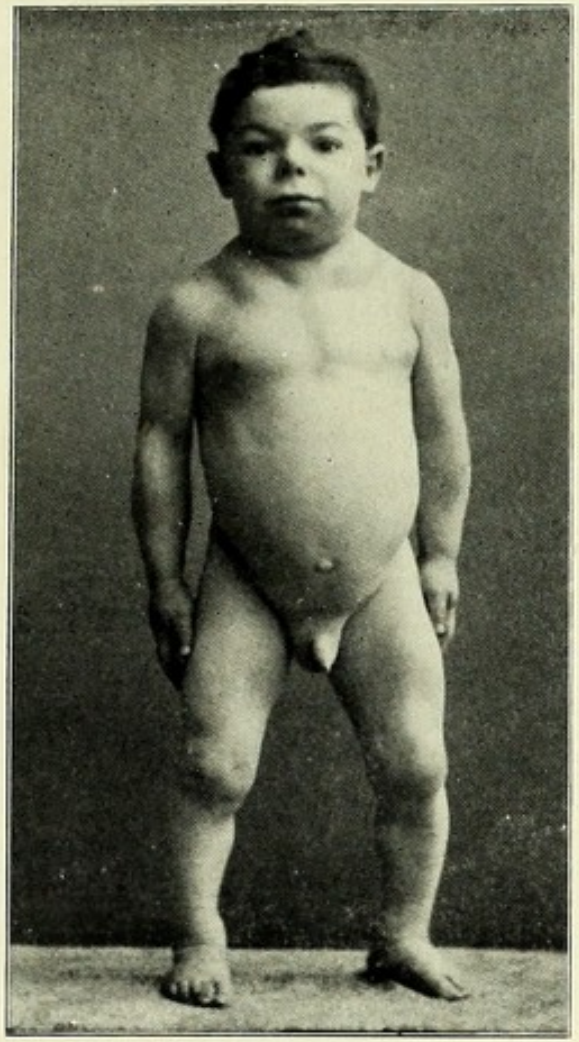


FIG. 37.

CRETIN ABOUT PERIOD OF PUBERTY, SHOWING "SUPRACLAVICULAR PADS" AND BENDING OF LEGS

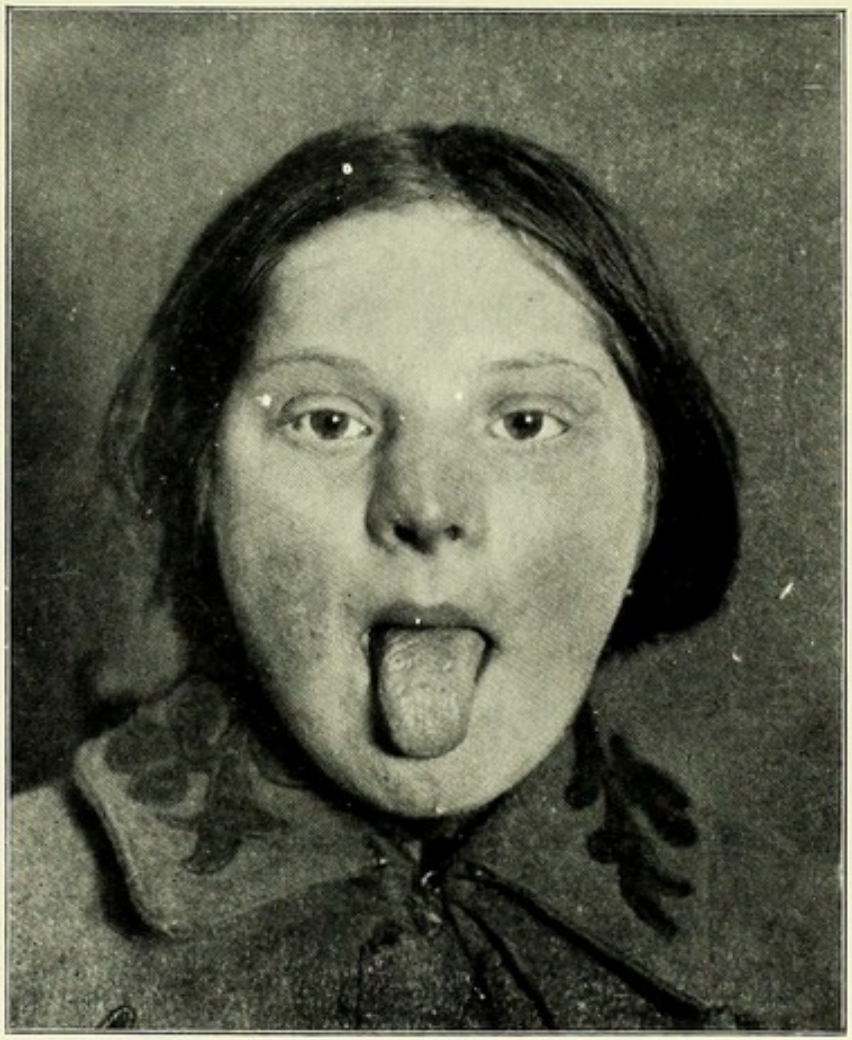


FIG. 38.

MONGOL, SHOWING FISSURING OF TONGUE WHICH APPEARS IN LATER CHILDHOOD.

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, who was a physician at this hospital, 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 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 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. Mongols tend to suffer with great frequency from defects of the septum and other lesions of a similar sort. Out of eighteen cases of which I have notes, five had marked congenital heart disease.

3. I now pass to the third group—namely, cases of **microcephaly.** Those cases, 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 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.

Cases of the microcephalic type are not at all uncommon.



FIG. 39.
MONGOL, SHOWING CHARACTERISTIC EYES.

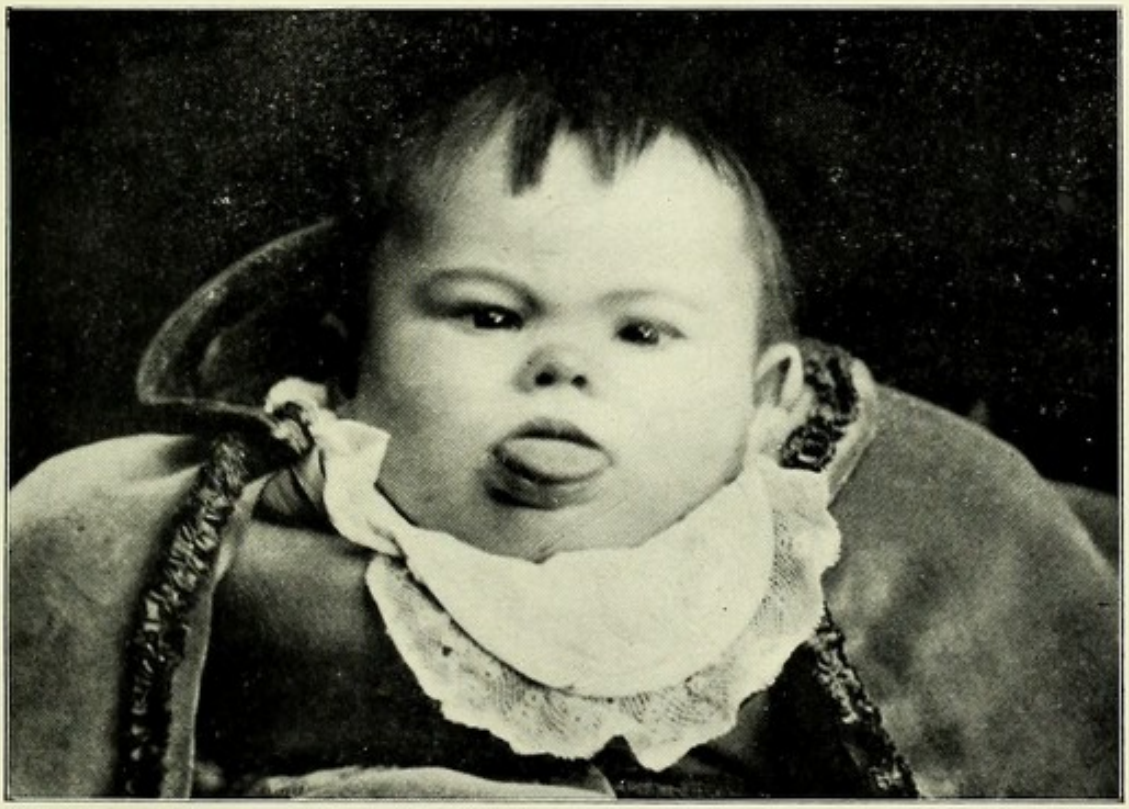


FIG. 40.
MONGOL, SHOWING PROTRUSION OF TONGUE.

They made up 25 per cent. of my own cases; and from what I have seen here and at Great Ormond Street Children's Hospital I think the condition tends to occur with unusual frequency in children of Jewish parents. That is quite the reverse of Mongolism, from which condition the Jewish race seems to have an immunity rather than otherwise.

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 rickets that I shall not go over that again (p. 117). Hydrocephaly is not, however, a common cause of mental deficiency, and it is extraordinary how large the head may be and yet the mental power be very well retained. The child shown in this photograph had a head 26 inches in circumference, and yet 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; but 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 note-paper.

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 dulness 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 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 cases which 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

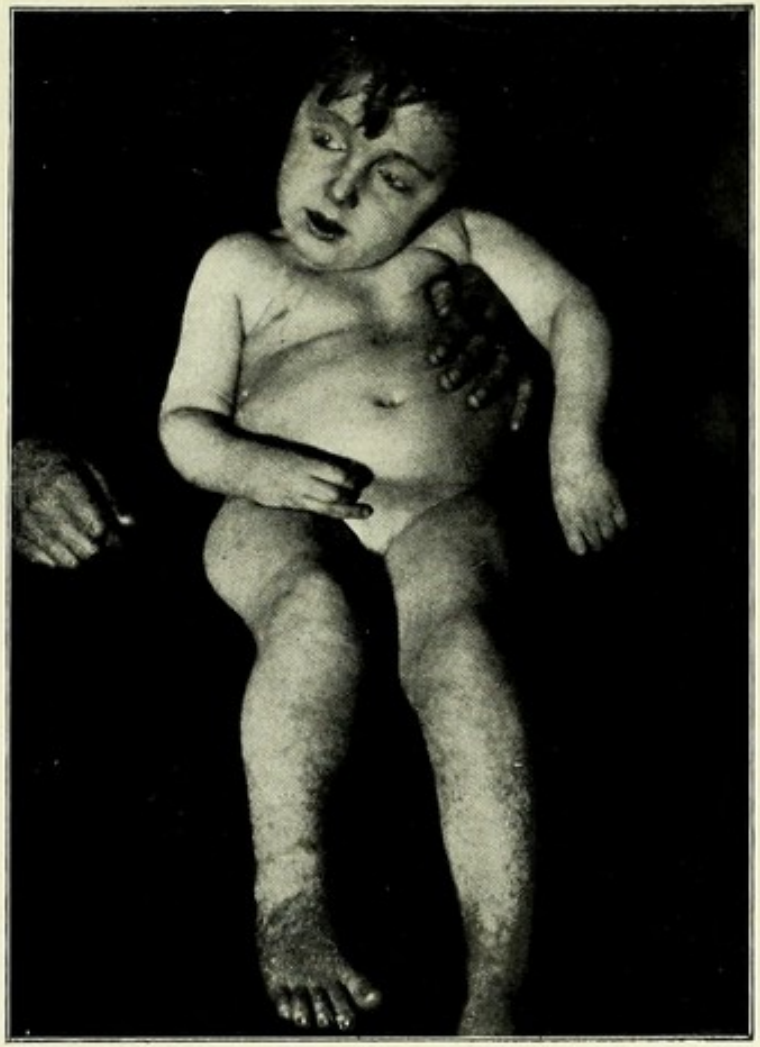


Fig. 41.
Right Hemiplegia with mental deficiency.



Fig. 42
Congenital Paraplegia with mental deficiency.

avenues of information, because their hearing or their sight is bad. And whenever you are brought face to face with mental deficiency you have to ask yourself, May not this be produced by the fact that the child has a difficulty in acquiring information owing to the fact that he cannot hear properly or see distinctly ? The commonest causes are middle-ear disease, frequently the result of adenoids ; and errors of refraction, particularly hypermetropia and astigmatism. As a consequence of these defects the child fails to benefit like other children from instruction at school, and so falls behindhand, and in extreme cases may be regarded as a case of mental deficiency. But many of these cases belong to the group of mere *backwardness*. And that brings me to ask what is to be your 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 five. But a child who is mentally deficient you cannot regard as 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 1871. It is a group which is marked by certain very definite features. First, the cases tend to occur in families, several members of a family being likely to suffer. Secondly, in the vast majority of cases the children are

of Jewish parentage. Thirdly, they are characterized by the fact that the children are normal at birth and for some time afterwards; that then 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.

9. Lastly, I come to a great group which you 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.* They make up the majority you meet with; they were 34 per cent. of all the cases I have seen. 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—and if you attend to these points you are not likely to overlook the mental defect.

CAUSES OF MENTAL DEFICIENCY.

I now pass to consider rapidly the question of the *etiology of mental deficiency* in children. With regard to a few of these 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 have often 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 very great 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 co-existence of congenital syphilis is quite exceptional.

A cause which might be more likely to produce mental deficiency is bad health in the mother during her pregnancy. There have been experiments on chicks which show that if an egg is incubated at a higher temperature than usual the chick which results is apt to be abnormal and defective in development; and you might suppose that, if a mother has an attack of fever during her pregnancy, so that the incubation of the fœtus proceeds at a temperature two or three degrees above the normal, mental defects, amongst others, might result. And I do not say that that does not sometimes happen. 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 always 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 things is a constant 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. You know that 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 soon 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. It is a condition which results 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 the mothers were between the ages of thirty-eight and forty. Dr. Still, of Great Ormond Street Hospital, reports eighteen consecutive cases, out of which ten were the last children of a large family, and in the others the child was the last but one. Why exhaustion of the reproductive power should result in Mongolism I do not



FIG. 43.
EXTREME DEGREE OF MICROCEPHALY.



FIG. 44.
MICROCEPHALY; FRONT VIEW.



FIG. 45.
MICROCEPHALY; PROFILE VIEW, SHOWING RECEDING FOREHEAD.

know, but there seems to be an arrest at some stage, beyond which it is impossible to carry the development.

PROGNOSIS.

With regard to the *prognosis* in mental deficiency, you have to consider it, 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 that is so, because the majority of them 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 this group are particularly liable to be carried off by tuberculosis. You have also to remember in giving your forecast that many of them will be apt to suffer from fits, particularly in the paralytic cases. Of course, those which are epileptic already will always have fits as one of their prominent symptoms. 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 of cretins that if thyroid gland is administered from the first year of life they will grow up to be almost normal individuals. You can say with regard to Mongols that in the highest grade they will be fit for physical work, but 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 appreci-

able 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 leaves 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. 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. Those cases which look best in physical development often give the worst outlook with regard to mental improvement. 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 OF MENTAL DEFICIENCY.

The *treatment* I can dismiss in a few words, because, I am sorry to say, for many of them you can do but little. I urge you always to remember, however, that there is a physical side to mental deficiency, and although you can do nothing 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, which make it difficult for them to hear. You can order them spectacles, and make it easy for them to see. You can divide tendons if they are paralyzed, and make it easier for them to use their limbs. If the child is rickety, you can treat the rickets. By removing these difficulties you can, especially where there is only slight mental deficiency, make all the difference to the child, and put him upon a very much higher plane than before.

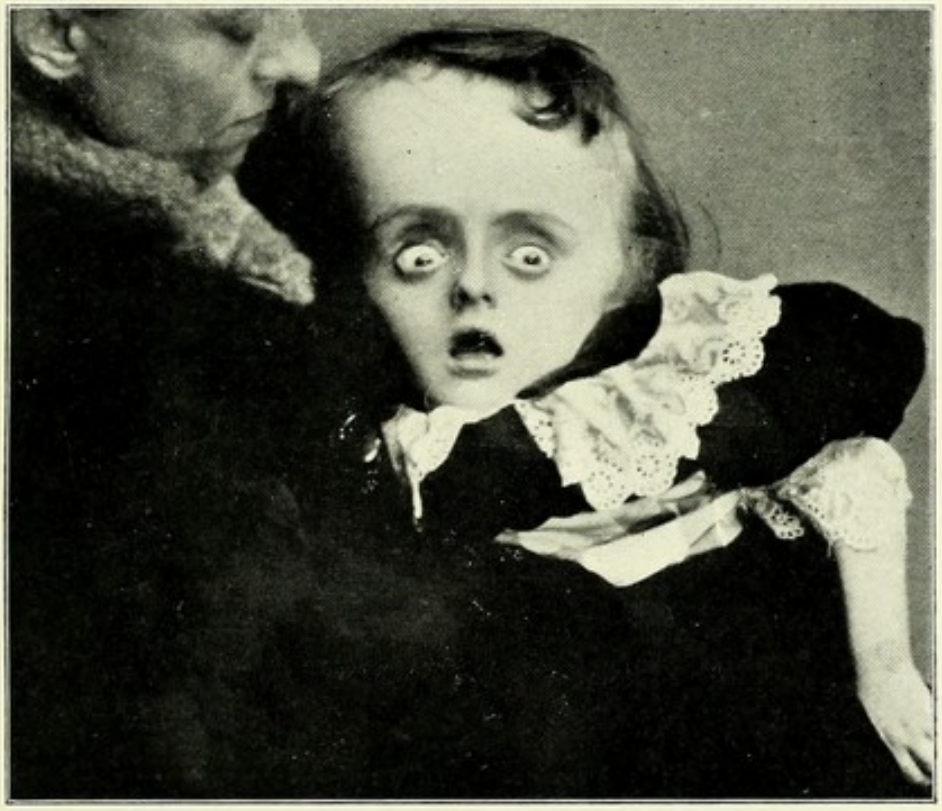


FIG. 46.

SEVERE CASE OF HYDROCEPHALUS, SHOWING CHARACTERISTIC SHAPE OF HEAD AND DEPRESSION OF EYES.



FIG. 47.
IMBECILITY OF UNCLASSIFIABLE TYPE.
NOTE GENERAL ATTITUDE AND EXPRESSION.



FIG. 48.
AN UNCLASSIFIABLE CASE OF SEVERE MENTAL DEFICIENCY WITH CONGENITAL PTOSIS.

With regard to mental improvement, all you can hope to do is by careful and persistent training. 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. In recent years much progress has been made in the training of mentally defective children, and their lot has never been so favourable as now. Under the Elementary Education Act of 1899, special classes have been instituted under the School Board in London for the training of defective children; and much has been done by the Kindergarten methods in teaching these children to use their muscles and make the best use of the feeble brain-power they have. It is only on those lines that you can hope to do good in those who are improvable. For those who are unimprovable there is no refuge but the idiot asylum, or, for people who are able to afford it, special guardianship.

LECTURE XVII

THE BLOOD DISORDERS OF EARLY LIFE

GENTLEMEN,—I need not emphasize the importance of healthy blood to a young child. The blood is the medium by which all the processes of nutrition are carried on, and if it is deficient in any respect growth and development must inevitably suffer. For it is *not* a matter of indifference whether, during the early years of existence, when the tissues are as wax, to be moulded in one direction or another, the cells are bathed in rich blood or in a poor and watery substitute for it. Throughout the whole of life the body may exhibit a bias towards health and strength or towards delicacy and feebleness, according as the blood was normal or not throughout these impressionable years. You will readily understand, therefore, how important it is that you should become acquainted with the chief causes of anæmia in infancy in order that you may be in a position to do all that you can towards warding them off.

FORMATION OF THE BLOOD IN INFANCY AND ITS PECULIARITIES.

Before proceeding further, however, 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,the total being about 5½ millions per cubic millimetre. The amount of **hæmoglobin** is proportionately high, being about 110 per cent. The meaning of this surplus of red cells and hæmoglobin I cannot discuss with you now, but I would only point out that it is very short-lived, and by the end of the second week the adult figure of 5,000,000 red cells is reached,

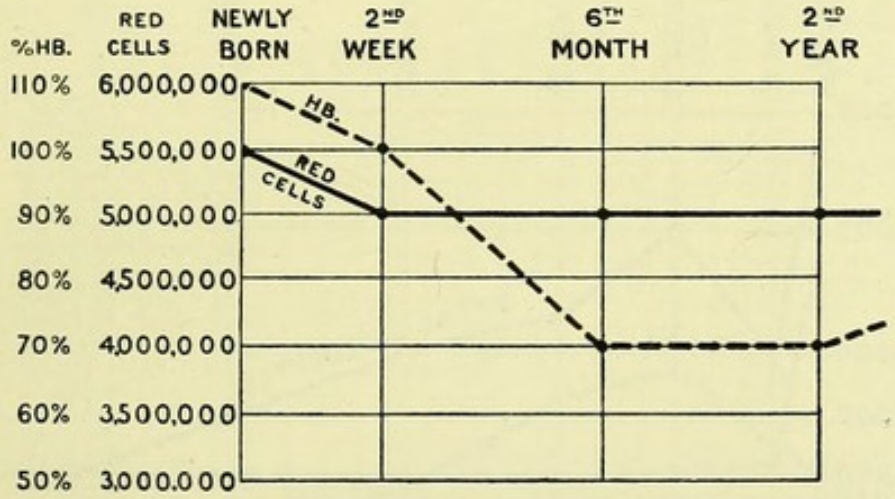


DIAGRAM 10.—PROPORTION OF HÆMOGLOBIN AND RED CORPUSCLES THROUGHOUT INFANCY.

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 rapid reduction in the red cells and hæmoglobin in the early weeks of life has this special clinical interest, that it probably is the cause of that familiar phenomenon,

icterus neonatorum, the products of destruction of the red corpuscles rendering the bile so viscid that it stagnates in the liver, and is reabsorbed with consequent jaundice.

In infancy, as in the adult, the red cells are formed exclusively in 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

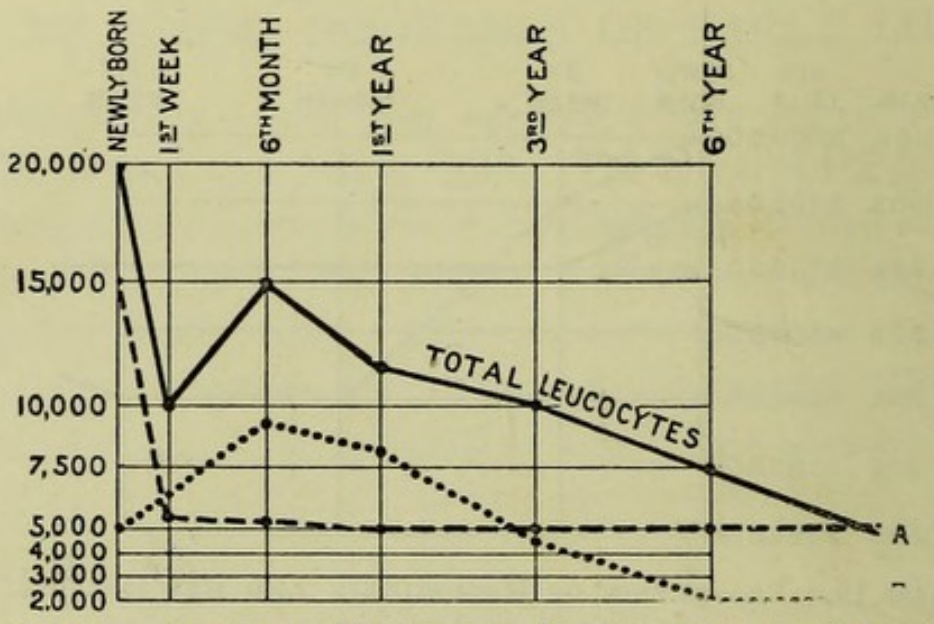


DIAGRAM 11.—ABSOLUTE NUMBER OF LEUCOCYTES PER CUBIC MILLIMETRE AT DIFFERENT AGES. A. POLYNUCLEARS. B. LYMPHOCYTES.

the whole length of the long bones, and it is only as the growth of the bones becomes completed that it retreats to their extremities, which are the only parts of the long bone in which such marrow is 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 results 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

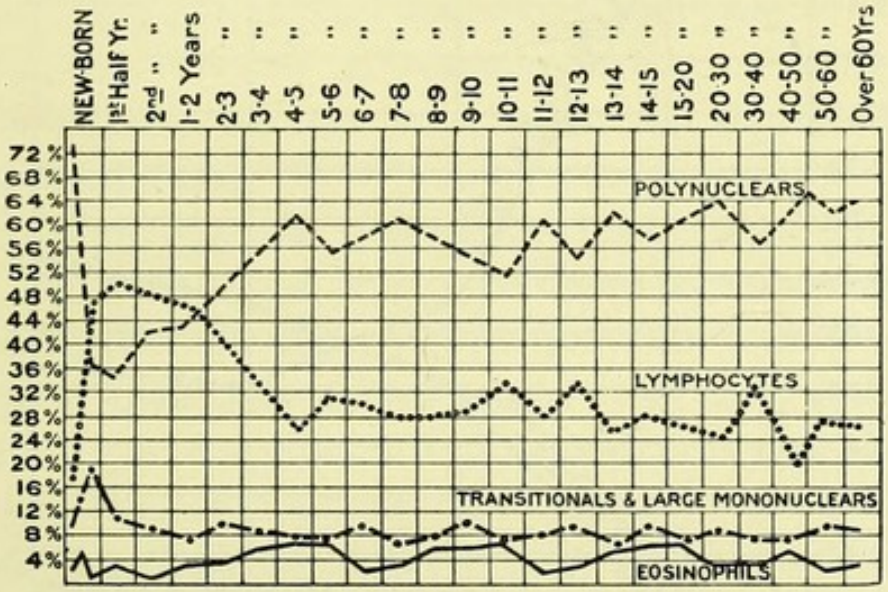


DIAGRAM 12.—DIFFERENTIAL PERCENTAGE COUNTS THROUGHOUT LIFE. (AFTER CARSTANJEN.)

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 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 rhroughout the period of infancy. What the meaning of this activity of the adenoid tissue is it is difficult to be quite sure, but it is probably associated in some way with growth and nutrition; and anything which

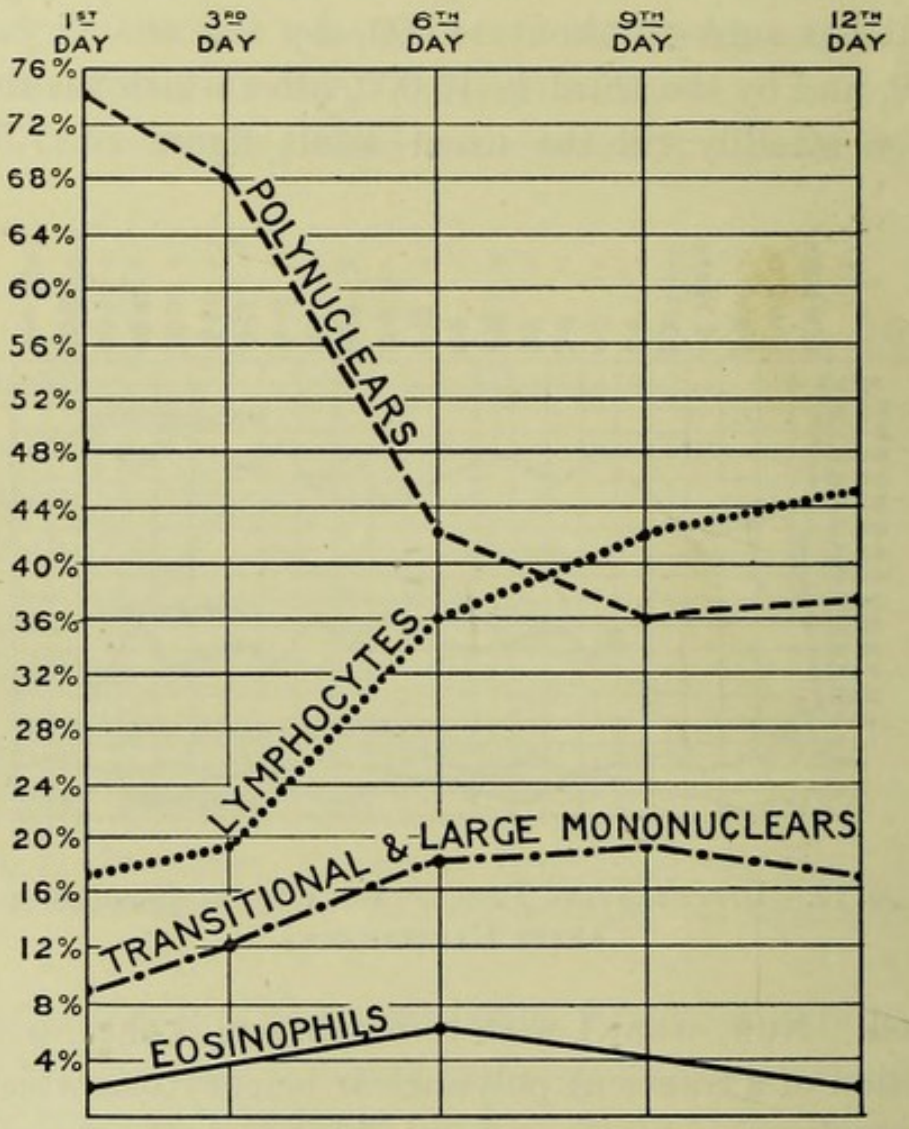


DIAGRAM 13.—DIFFERENTIAL PERCENTAGE COUNTS DURING THE FIRST FORTNIGHT. (AFTER CARSTANJEN.)

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' in anæmic conditions of childhood.

VARIETIES OF ANÆMIA.

After these preliminary remarks I may pass 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—*i.e.*, is the result of some cause which produces increased destruction of blood or interferes with its formation—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) primary anæmias —*e.g.*, pernicious anæmia and chlorosis; (3) secondary anæmias; (4) anæmia associated with enlarged spleen; (5) lukæmias; (6) affections of the adenoid tissue.

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 marked 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 I am inclined to think that 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. Some of you may have seen a baby which was shown lately at our Medical Society, and which was the subject of congenital pulmonary stenosis with extreme cyanosis. The red cells in that case numbered no less than 8,000,000 per cubic millimetre, and the hæmoglobin amounted to 110 per cent. 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 intracapillary red-cell formation persists to a large extent.

2. The Primary Anæmias.

In adults the two chief forms of so-called 'primary' anæmia are chlorosis and **pernicious anæmia.** Of the latter I would only say that it is for some reason or other extremely rare in childhood, and is practically never met with at all during infancy. That is a fact to bear in mind in diagnosis. **Chlorotic anæmias,** on the

other hand, are not uncommonly seen in early life. The best examples of them I have seen have been in rickety children who have been fed on a diet containing an insufficient quantity of proteid and iron, both of which are so essential for healthy blood-formation, or in older children who have not been supplied with a sufficient amount of animal food. Here is an example : A girl of three years had been fed on the breast till she was eighteen months old ; since then she had been rickety. She had a chlorotic complexion, a palpable spleen, and loud hæmic murmurs. The red cells numbered 5,000,000, the white 13,000, whilst the hæmoglobin was only 31 per cent. Under full diet and iron in large doses the hæmoglobin rose in six weeks to 62 per cent., which is little short of the normal at this time of life.

That too prolonged nursing leads almost inevitably to anæmia I have no doubt at all, and the explanation of the fact is probably to be found in the poverty of iron in human milk. You must never forget that the infant does not depend by any means entirely upon the iron in his diet for the supply of that metal necessary to the formation of blood. A considerable proportion of it is derived from the storehouse of iron in the liver with which every child enters the world. About the end of the natural period of nursing, however, this store gets exhausted, and if the diet is then poor in salts of iron anæmia results.

3. The Secondary Anæmias.

I have already said that it is probable that some day we shall regard all anæmias as being really secondary in origin ; meanwhile, however, it is well to reserve that term for those cases in which poverty of blood comes on as a direct

consequence of some illness or debilitating condition. Now, 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. The production of secondary anæmia 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 of secondary anæmia in childhood are these: (1) Chronic gastro-intestinal disturbance; (2) the toxins of acute infective diseases; (3) 'cachexias' of various sorts, notably congenital syphilis and tuberculosis.

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 in such a case, 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 great 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 of the heart that severe anæmia is apt to supervene. When going round the wards, for instance, one is often struck by the sudden supervention of marked 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 made its appearance. The moral of this is that you should always remember to put your rheumatic cases through a thorough course of iron during their 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 the 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 chlorotic type of blood. In the severer forms the red cells are also reduced in number, and may show some inequality of size and 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).

4. 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 pretty 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 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 proceeding to palpate 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.

On auscultation of the heart you detect hæmic murmurs, and there may perhaps be some moist sounds at the bases of the lungs. When you come to examine the blood you find that it exhibits 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 text-books by different names. Some people speak of them as cases of **'splenic 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 possibilty 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, 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. What you have most to dread is the 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. A *few* of these cases may be examples of **splenic anæmia of the adult**

type, but a greater number are the result of **congenital syphilis,** and you should therefore always make a point of inquiring carefully into the history of such cases, and of looking for other signs of inherited specific disease. Hodgkin's disease may also be attended by great splenic enlargement, but the co-existence of glandular swellings should keep you right.

Of **other causes of splenic enlargement** in childhood, such as Bovaird's 'spleno-megalie primitive,' lymphosarcoma of the spleen, primary splenic tuberculosis, and the enlargements associated with waxy disease and ulcerative endocarditis, I do not propose to speak, for they either belong to the extreme rarities of medicine, or are dealt with in the ordinary medical text-books.

5. Leukæmia in Childhood.

There are, as you know, two chief forms of leukæmia: (1) the *myelogenous*, in which cells of the bone-marrow type enter the blood-stream; (2) the *lymphatic*, 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. Why this should be I do not know, but if you search through the literature of the subject you will scarcely find a single case of genuine **myelogenous 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 myelogenous leukæmia in a child.

Lymphatic 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 three 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 infective purpura.

(3) Pseudo-scorbutic cases, in which lesions in the buccal cavity (spongy gums, ulcerative stomatitis, etc.) are the most striking feature.

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. The mistake you are most likely to make is in regarding such cases as 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 lymphatic leukæmia. In any doubtful case, then, examine the blood 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.

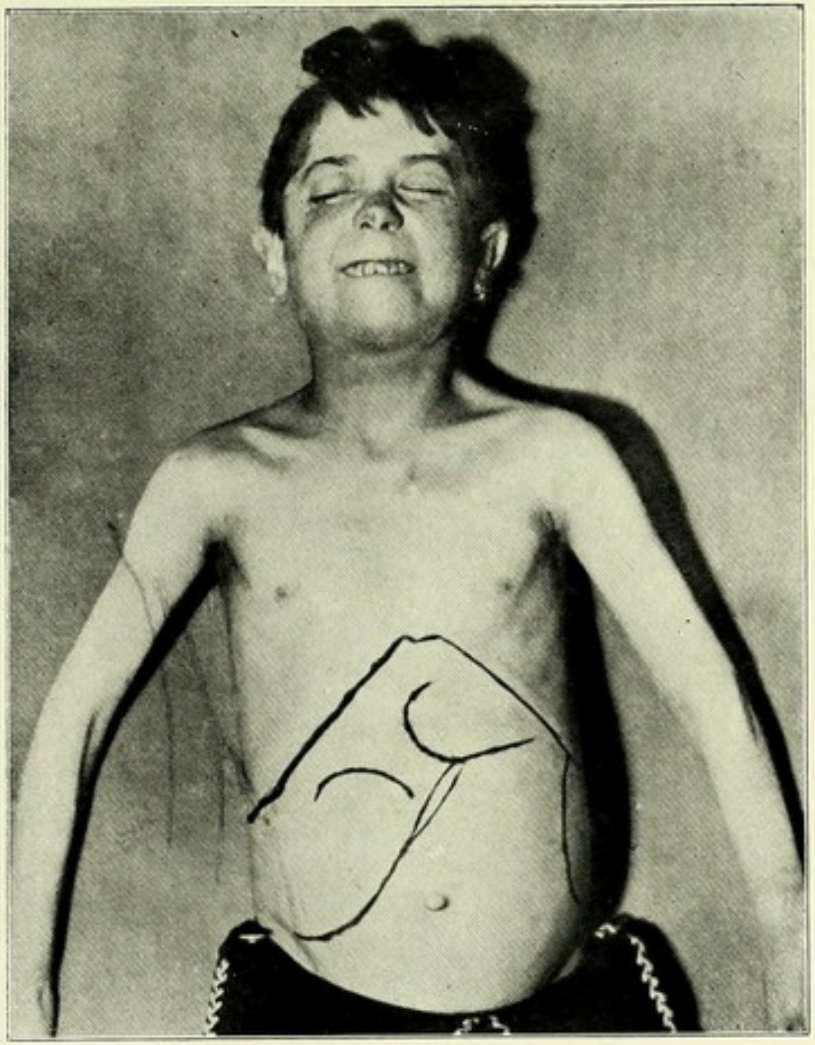


Fig 49.

Enlargement of liver and spleen, the result of Congenital Syphilis. Note also the notched incisors.



Fig. 50.

Swelling of the Abdomen, the result of enlargement of the spleen in myelogenous leukaemia.

Closely allied to lymphatic leukæmia is the disease termed **chloroma.** This is a rare condition, of which you may never meet with an example, although it does occasionally occur, even in quite little children. It has the same blood-picture as lymphatic leukæmia, but differs from it in that local humours develop, often in the periosteum of the bones of the skull and about the orbit, so that the eyes may bulge out. 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 exhibit the great degree of lymphocytosis which is characteristic of chloroma.

6. 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 this 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 markedly so. I cannot promise that you will be able to recognise such cases clinically, for the only sign they exhibit is an increased dulness behind the manubrium of the sternum, and that may be produced by other causes, such as enlarged bronchial glands. More commonly such cases are only recognised on the post-mortem table, and you will find that almost invariably they have been cases in which death has occurred suddenly and without apparent cause, and often 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 condition in mind, for it is sometimes of medico-

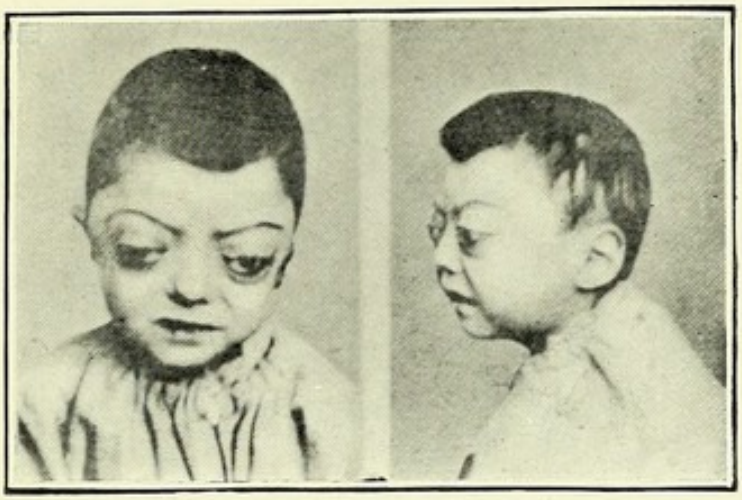


FIG. 51.
CHLOROMA, SHOWING PROTRUSION OF EYES.
(By permission of Dr. Melville Dunlop.)

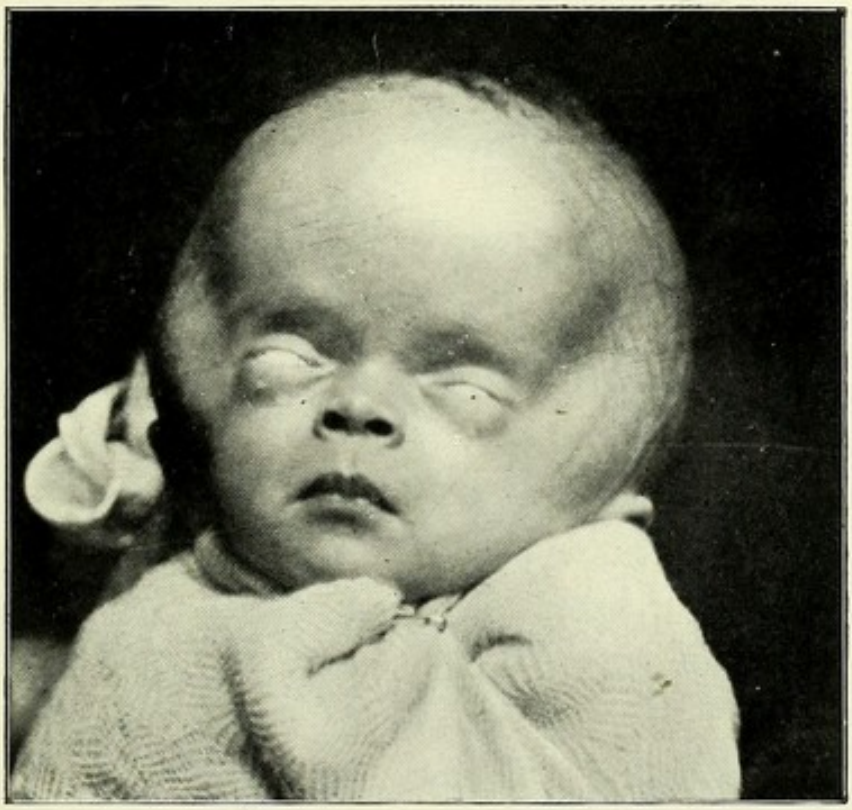


FIG. 52.
SARCOMA OF SKULL. PERIOSTEAL GROWTHS AND BULGING OF EYES SIMULATING CHLOROMA.

legal importance. Some cases of supposed 'overlying,' for instance, may really be due to this cause.

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 text-book 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 is the only diagnostic criterion of any value. A polynuclear leucocytosis is, in the absence of any intercurrent 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.

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 particularly of 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 and raw meat juice are also important iron-containing foods. These measures are of greater value in treatment than are **drugs,** but the latter can, of course, give you great help. The milder preparations of iron—of which there are so many available now—are the most suitable for children, and cod-liver oil may require to be given at the same time. I have found the combination of these in the form of the preparation known as ' Ferroleum ' very useful. I cannot recommend arsenic very strongly in the ordinary anæmias of childhood, though it seems to be the only preparation we possess which has any influence over Hodgkin's disease. The ordinary form of leukæmia in childhood (the lymphatic) does not appear to be affected by it. 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. I have tried various preparations of bone-marrow in it, but the results have not been very satisfactory. 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.

LECTURE XVIII

SOME COMMON SYMPTOMS OF DISEASE IN CHILDREN AND THEIR DIAGNOSTIC SIGNIFICANCE

GENTLEMEN,—Those of you who have attended much in the out-patient department must have been struck by the fact that the number of actual complaints for which children are brought to the hospital—that is to say, the things noticed wrong by the mother—are comparatively few in number. And that, after all, is inevitable. A young child is unable to express—even supposing he is able to experience—those multifarious abnormal sensations which make up what we call the symptoms of disease in grown-up people. And it follows from this that your diagnosis must always rest mainly upon the results of your objective examination. Now, I need hardly tell you that therein lies one of the great difficulties of children's practice—you get so little help from your patient. The more, therefore, is it necessary for you to study as carefully as you can such symptoms, few though they be, as the friends of the child have been able to observe. I thought, therefore, that we might profitably spend our time in this lecture if we took up together the study of the commoner symptoms which children present, and tried to arrive at some idea of their value in diagnosis.

WASTING.*

The first symptom that I wish to direct your attention to is one which occurs with very great frequency in the out-patient department. It is that spoken of as *wasting*. With regard to this symptom, I would say, first of all, that it may be due on the one hand to very serious organic disease, or, on the other hand, to comparatively trivial disorders of health. And what you have always to do when a child comes before you presenting wasting as one of the chief symptoms is to eliminate so far as you can the possibility of organic disease before you go further. In the case of young infants there are three **organic diseases** which are specially apt to be accompanied by wasting as their chief symptom. The first of these is *congenital syphilis*, the second is *tuberculosis*, and the third is a *latent empyema*. With regard to the first of those there should be no difficulty; you should easily be able, by inquiring into the family history, and by examining for such symptoms as snuffles, craniotabes, rashes, and other manifestations of inherited specific disease, to recognise that it is syphilitic, or exclude that disease as a cause of the wasting. If you are still in doubt, you will often find that by the therapeutic test—that is to say, by the administration of gray powder—you can tell within a very short time whether such a taint is present or not; for syphilis may cause wasting, even though present in only a small degree. With regard to *tuberculosis* in young infants it is very much more difficult to be certain; in fact, I would say that often you cannot be certain of it at all, because tuberculosis may have attained a very

* This subject was also dealt with in Lecture V., but as it is approached here from a somewhat different point of view, I have allowed it to stand.

widespread degree of development before it produces any pronounced physical signs. Often in such cases you must remain in doubt, and watch the effect of treatment. If under treatment directed to the symptoms the wasting still continues and becomes progressive, then your suspicion that you are dealing with a case of tuberculosis is, of course, greatly strengthened. But as a matter of fact it will sometimes be only on the post-mortem table that the nature of the case will be made clear. There is nothing more difficult than to diagnose accurately between a case of tuberculosis in a young infant and one of so-called simple marasmus. With regard to the third of the conditions I mentioned, *latent empyema*, you should have no difficulty. You cannot miss it if you will examine the child carefully and systematically, particularly, of course, at the bases of the lungs. Not long ago a child was sent here with the history that he had suffered from measles a month or two previously, and that he had been wasting ever since. He had been under treatment outside, but had made no progress towards recovery, rather going from bad to worse. He was sent here under the impression that he was suffering from tuberculosis, and it was thought that nothing further could be done. On examination we found marked dulness at one base, with signs of fluid. On putting in a needle pus was obtained, and after its evacuation the child at once improved, and made an uninterrupted recovery. So there is the chance that even where a child is under skilled observation an empyema may be overlooked, because it may have produced no sign other than that of continual and progressive wasting.

Of the non-organic diseases which may produce wasting as one of their chief signs in young infants, the principal

are **disorders of digestion** and unsuitable feeding. As a matter of fact, these are by far the commonest causes of this symptom; either the child does not get enough nourishment, or he gets it in an unsuitable form, with the natural consequence that in both cases continuous loss of weight is manifested. Hence in the majority of such cases your chief duty will be to inquire into the nature of the feeding and of the food given, and to rectify any errors which you find.

When you come to inquire into the causes of **wasting in older children** you will find again that there is one great organic disease which is particularly apt to produce that symptom, and I need hardly say I refer to tuberculosis. Some of you saw among my out-patients this morning a child, aged three and a half years, who was brought with a history of wasting; nothing else had been noticed, except that the child sweated at night. On examination, however, we were able to find without difficulty unequivocal signs of fairly widespread tuberculosis. The commonest functional disease which you are apt to mistake for tuberculosis is that chronic form of dyspepsia which occurs in children at about the period of second dentition, and to which I am so often drawing your attention in the out-patient room. You know the history—the child is sent up with the statement that he has wasted, that he has a cough, that he has no appetite, and perhaps that he has certain nervous disturbances, such as headache or restlessness at night. Such cases are constantly diagnosed as tuberculosis when they are not so at all. And the mistake, after all, is not an unnatural one, for there are many signs connected with them which in the adult one is accustomed to associate with phthisis; the child has been losing weight,

has lost appetite, and has a cough. Those who see little of children and much of adults are apt to conclude that these signs mean phthisis in a child, just as they would rightly make one suspicious of phthisis in a grown-up person. You should know that many such cases are simply examples of chronic gastric catarrh associated with pharyngitis, or enlargement of tonsils, or adenoid growths, and they can be perfectly cured if they are put on a suitable diet and the condition of the throat attended to.

COUGH.

I will next pass to the consideration of another symptom which is particularly common in children, and that is *cough*. Of cough as a symptom in children I would say first that it would be a mistake to suppose that it is always, or indeed usually, the result of respiratory disease. The majority of coughs in children are not due to any abnormal condition of the lungs or bronchi at all. It is all the more necessary that you should be warned of this, because frequently the mother makes 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 by Dr. Parkinson* from one of the children's hospitals in London on this subject, and he found that of a large number of consecutive cases of cough, fully 40 per cent. were due, not to disease of the respiratory organs, but to abnormal conditions in the throat, whereas only 31 per cent. were due to disease in the chest. So you see there was actually a clear majority of cases in which the cough was due to the throat. I think there is no more important point to realize

* *Brit. Med. Journ.*, August 19 1899.

about cough in childhood than that. The **abnormal conditions of the throat** which may give rise to cough in children are three in number. You may have simple pharyngitis, or enlargement of the tonsils, usually existing with adenoids, or elongation of the uvula. These three conditions may occur together, as I need hardly say, or you may have combinations of any two or all three of them in any one case. Now, from the character of the cough you can often infer what is likely to be its cause in any particular case which comes before you. For instance, in *simple pharyngitis* it is usually a dry tickling cough, occurring at frequent intervals. In *adenoids* it is rather of a choking character, often accompanied by vomiting, and frequently also 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.

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 an exact and definite reply. Although you cannot be certain always about the existence of *whooping-cough*, there are certain things which should make you suspicious of it. And first, the cough of whooping-cough is essentially paroxysmal, it tends to occur especially at night, and it is often followed by vomiting. When you examine the child you find that the cough is out of all proportion to the severity of the physical signs. There may be only a few bronchitic signs in the lungs,

although you are told that the cough is one of very great severity. You will frequently find also that there is 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 which 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. You will be apt to mistake for whooping-cough the cough which you get from pressure upon the nerves in the chest caused by **enlarged bronchial glands.** In such a case the history may be of some value, because you know that whooping-cough rarely occurs twice in the same child, and if there is a history of his having had an attack of whooping-cough, and if he develops a similar sort of cough again, the chances are that it is not true pertussis on the second occasion. The other sort of cough which may simulate whooping-cough is that which you get in *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.

When you have excluded the causes of cough which I have mentioned, those in the lungs and those due to the state of the throat, there remain a considerable number in which you can find no definite cause at all. Some of these are certainly due to gastro-intestinal disorders. There is in children, I think, such a thing as **stomach cough;** at all events, you find children who have a constant cough 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 extremely 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 here 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.

PYREXIA.

The next symptom I wish to consider with you is *pyrexia*, or fever, as an indication of disease in children. I mean by that cases in which pyrexia is the only symptom—where a child is brought to you by the mother because he is hot and feverish, and nothing else has been noticed wrong. These are often cases of great difficulty, and always of very great anxiety in practice, because pyrexia may be, and often is, a sign of serious disease coming on—disease which may be of an infectious nature ; and one always wants to make certain at the earliest possible moment what the cause of the fever is. The first thing to look for are signs of the **acute specific fevers.** You should examine the child carefully for one of these, particularly for scarlet fever

and measles. In scarlet fever examine the throat for erythema, and look for the early signs of eruption, remembering that it comes out first about the chest and neck. In measles look carefully for catarrhal signs or indications of conjunctivitis and running of the nose. Examine for a rash about the forehead and head. If the rash is not yet out, look for Koplik's spots, which are of very considerable value in the early diagnosis of measles. I need hardly say you should also inquire into the history as regards the possibility of any exposure to infection to one of the acute specific fevers. Failing the discovery of any of those, I advise you to look, especially in young infants, for signs of **pneumonia.** I saw in consultation the other day a baby who had suddenly become feverish about three or four days previously. No physical signs were discovered by careful examination, which had been repeated several times after the child was taken ill. When I saw the child there was little to be made out, but at the tip of one lung there was a small patch of tubular breathing and dulness, indicating the development of apical pneumonia. In addition the child had the characteristic grunting pneumonic breathing; and one had no hesitation in saying that the cause of the fever was a small patch of croupous pneumonia at the top of one lung. That is a frequently overlooked cause of pyrexia, as I know from my own experience. I should therefore advise you in any case of acute fever coming on in a young infant to examine the apices of the lungs with particular care, because a patch of pneumonia so small as to be easily overlooked on physical examination may yet be the cause of very high temperature. Another cause of fever which it is important you should remember is **acute gastritis.** I remember seeing a child a few years ago

of about two or three years of age who had been seized with high fever with some abdominal tenderness and vomiting, and the question arose, What was the cause of the symptoms? Various things were suggested, and finally it was thought that the child was suffering from appendicitis, and the possibility of operation was contemplated. But the whole case cleared up entirely after the administration of a few doses of calomel. This was a case of acute gastritis accompanied by high fever. Sometimes, indeed, the fever in such a case is very high, and may be accompanied by rigors. Another disease which you should never forget the possibility of, where fever is present and nothing else, is *otitis media*. Usually in such a case there will be signs of earache in addition. The child will manifest pain or discomfort in the ear; and if you suspect inflammation the best thing you can do is to examine the membranes, and if necessary puncture them, and if you do so you may often find a small quantity of sero-purulent matter escape, with the immediate relief of all the symptoms.

Another cause of fever in children which you are apt to overlook is **acute rheumatism** where the joints are not affected. Not long ago a boy was admitted to one of my beds here who had acute fever, and in whom careful physical examination revealed absolutely nothing wrong. But there was one suspicious point, and that was that, although he had a temperature of 103° F., he was sweating profusely, and, further, he looked like a child of the 'rheumatic diathesis,' as it is called. Acting upon that suspicion, we gave him salicylate of soda in full doses. His temperature came down with a run, and a few weeks later he had developed a definite cardiac bruit. This, then, was a case of acute rheumatic fever without

any joint manifestations. Such cases are not common, but when they do occur it is very important that you should be able to recognise them, because if you allow rheumatism in a child to go on untreated for a few days, you run a great risk of endocarditis developing and permanent cardiac disease being the result.

There is in young infants a cause of acute fever which you should never overlook, and it is one which is especially apt to escape the observation of a physician, and that is **osteo-myelitis.** I have seen a child lie in the medical ward of a hospital for some days with acute fever, the cause of which was not explained. It died, and, post-mortem, one radius was found entirely surrounded by a layer of pus. That is not unlikely to happen to you in practice, and the moral is, when you have a case of acute fever with no other signs in a young child, you should always examine the bones with great care for signs of tenderness and swelling, always bearing in mind the possibility that the fever may be the result of acute suppurative periostitis or osteo-myelitis. Another point of great importance is, that in every case of acute fever you should examine the **throat** for tonsillitis, which is usually of the follicular sort. I have seen several cases where it was a difficult matter to determine the cause of the fever, but where the whole case was cleared up by looking down the child's throat. It should be done as a matter of routine in every case.

In older children **enteric fever** will certainly give you a good deal of trouble. I have known several errors in diagnosis made from not recognising the possibility of enteric fever being the cause of the pyrexia. Such a thing happened to me the other day. I had a child in hospital who was admitted with a moderate degree of

pyrexia. There were some bronchitic signs in the lungs, rather pronounced, but nothing more. The child looked tuberculous, and one concluded on the whole, seeing that there was no enlargement of spleen and no spots or anything else, that it was probably tuberculosis, and we were prepared to treat it on that assumption. But as time went on one noticed that the temperature gradually mounted up, and then the spleen began to be palpable. The Widal reaction was tried, and definite clumping occurred at once. That, of course, made the diagnosis clear. Now, such a mistake as that is very easy to fall into. The recognition of enteric fever in young children is more difficult than it is in grown-up patients, and if you have fever for which you can find no other cause, even in the absence of enlargement of the spleen or other signs, bear in mind the possibility that you may have to do with a case of enteric fever, and by having the Widal reaction done, or by estimating the number of leucocytes, try to arrive at an exact diagnosis.

There is a cause of fever in infancy which is of rare occurrence, but which it is extremely important that you should recognise when it does occur, and that is **acute pyelitis.** Last summer I was called in consultation to see a baby who presented some rather puzzling features. She was a girl of a few months old who had for some weeks been running a very high temperature with occasional remissions, and—this is one of the suspicious and important points—accompanied at times by definite rigors. On inquiry into the state of the urine it was found to contain pus. One then felt certain that it was a case of pyelitis. Further examination showed that the urine contained the *Bacillus coli communis*, and on giving the child alkalies

in full doses, which is the right treatment in such cases, the temperature speedily came down.

INABILITY TO TALK.

Passing from pyrexia, I want to take up together two symptoms which mothers frequently complain of in their infants. One of these 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 which you need consider. In the first place, of course, the child may be a **deaf-mute.** In such a case you will find quite a clear history that the child is absolutely deaf, and you will sometimes find that there is a history of several similar cases having occurred in the same family. Such cases should not present any great difficulty in the matter of diagnosis. You find, again, that some children are unable to talk at the proper time because of **mental deficiency,** or else because of backwardness short of actual mental deficiency. These cases are more difficult to recognise. 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.

Pathological conditions in the throat, especially **adenoids** and **enlarged tonsils,** may be a cause of difficulty in learning to talk ; I cannot exaggerate to you the importance of this fact. I should say that the great

majority of children I have seen who were brought to me because they were unable to talk at the proper age were suffering from some abnormality of the throat. 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. That is a clear and typical instance of what I am referring to, and I could quote dozens of such cases almost which I have seen in the out-patient room here and elsewhere. It is difficult to say in such a case why it is that adenoids make it difficult for the child to learn to talk. The mother is probably correct when she says that the child does hear, but the possibility is that he does not hear quite distinctly, not well enough, at all events, for him to learn to imitate the speech of other people. Whether it is that, or whether the adenoids impose a mechanical obstacle to the movements of the soft palate I do not know, but I know if a child is brought to you with the complaint that he cannot talk, and you find adenoids present in large amount, you can promise with almost complete confidence that if the adenoids are removed the speech will become perfectly natural.

A rare cause of this lack of the ability to speak 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 rare. 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 of inability to talk is **congenital aphasia,** a condition of which I have never met with an example, but which is described as one of the occasional congenital defects of childhood.

INABILITY TO WALK.

Now, to turn to the kindred symptom—inability to walk. There you have to recognise, I think, two groups of cases : first, those in which the child has never walked at all from birth ; and, second, those in which the child has at one time or another been able to walk, but has gone 'off his legs,' as the mother puts it.

First, with regard to those who have never walked. There you have three possibilities to deal with : either the child is the subject of rickets, or of some form of paralysis, or it is a case of mental deficiency. Now, in **rickets** the difficulty is merely one of walking. Whilst the child sits in 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 also 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 usually 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 recognise 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 from **surgical reasons** as well as medical. I have seen cases of *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 after that illness, 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 the rather difficult art of walking, he gets out of practice when laid up in bed during an acute illness, and when he gets on to his feet once more he has to learn all over again. Such cases 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 recognised.

A rarer cause which it is important to recognise 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.

Another nervous symptom which children present, and which may often lead to considerable difficulty in diagnosis, is headache. Now, the first thing one has to remember about headache in children is this, that the causes of it are usually totally different from those in grown-up people. One of the commonest causes of headache in grown-up people is some vascular disturbance, some cerebral congestion, or cerebral anæmia, or high tension in the intracranial vessels; but these are not common causes in children. On the other hand, what one may call 'reflex' causes of headache are common in childhood, and particularly eye-strain. If a child of school age suffers from headache, I should recommend you to examine his **refraction** and see if there is any hypermetropia or astigmatism, and if you find either to get the error corrected by proper glasses. If you do that 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. **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 optic neuritis. **Adenoids** are a pretty common cause of

headache in childhood. Here the headache is usually frontal. **Migraine** also is a disease of comparative frequency in children, in whom it occurs quite as often as in grown-up people, and frequently with great severity. Hysterical headache, on the other hand, is a condition of very rare occurrence in early life.

HÆMATURIA.

A rarer symptom which it is important that you should be familiar with, as it often gives rise to difficulty in diagnosis, is 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. But assuming you have verified the fact that the child is passing blood, there are but few possible causes of it which you have to consider; indeed, I think there are but four causes of hæmaturia in infancy which it is necessary for you to bear in mind. One is **hæmophilia,** which may be a cause of hæmaturia, especially in boys. Another cause is **scurvy,** which I believe is commoner than one is disposed to think, and 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 entirely unnecessary operation. The 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. The 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.

CROUPY RESPIRATION.

A symptom of very great importance, and one which produces great anxiety in the minds of the friends, is that spoken of as laryngeal or croupy breathing. You are called to a child who exhibits croupy breathing, and you have to determine what the cause of it is. I need hardly say at the outset that the two things you will first think of, quite rightly, are so-called 'croup' on the one hand, and diphtheria on the other. Now, 'croup' usually means a **catarrhal laryngitis,** or it ought to mean that. Of this there are two sorts: first, that in which there is a slight degree of catarrh, but every now and then there is spasm superadded, producing croupy breathing; second, there are the more severe cases, in which the catarrh of the larynx is so great that it causes considerable swelling of the mucous membrane, and produces more or less continuous obstruction. The first sort of case you are not likely to confuse with **diphtheria,** because the history is that such a child is comparatively well during the day, but that during the night attacks of croupy breathing come on. The real difficulty arises in the second group of cases, in which the catarrh has led to so much swelling as to produce continuous obstruction. These are the cases in which it is difficult to tell whether the case is one of diphtheria or not. In some cases, indeed, the diagnosis may be impossible. But there are certain points to which you should direct your attention. First, there is the mode of onset in the two cases. In catarrhal laryngitis the onset is usually more abrupt and severe than in diphtheria, which begins more insidiously. Catarrhal laryngitis is accompanied by a higher degree of fever. In the second place, the loss of

voice and cough are less in catarrhal laryngitis than in diphtheria. In other words, diphtheria is the more *silent* disease of the two. If you find signs of a membrane on the fauces, of course you need have no difficulty at all; but the puzzling cases are those in which the diphtheritic process is confined to the larynx only, and although the above indications may be of some help, every now and then you are bound to be left in doubt. In such a case the wisest plan is to act as if it were the more serious disease—that is to say, isolate the patient and give antitoxin.

But there are other causes of croupy or laryngeal breathing which it is well to bear in mind. It may occur, for instance, as a result of **retro-pharyngeal abscess,** and I have known serious mistakes arise in this way. If in doubt, put your finger into the child's throat and explore the pharynx, and see if you can find any indication of bulging. Croupy breathing may also occur in the course of a **chronic laryngitis,** which may be simply catarrhal or the result of congenital syphilis. It may occur also from pressure on the trachea, by **enlarged bronchial glands,** or, in rare instances, by an **enlarged thymus.** It may also be simulated by certain conditions, the most notable of which are **congenital laryngeal stridor** and **laryngismus stridulus,** although there the nature of the attacks should leave you no doubt. Congenital laryngeal stridor dates from birth, whilst laryngismus stridulus is always preceded by a period of apnœa, followed by a long crowing inspiration, which does not resemble the continuous croupy breathing of laryngeal obstruction. It is always well also to remember the possibility that you may be dealing with a **papilloma of the larynx.** Two or three years ago a baby was

brought here in my absence and seen by my assistant. The child had continuous croupy breathing, and the question arose, What was the cause of it ? The symptom had been noticed since birth, and it was rightly thought that it might be a case of congenital laryngeal stridor. The child died suddenly within a week, and the post-mortem examination revealed the presence of laryngeal papilloma. I have since seen two cases where there was continuous slight croupiness of breathing, and in which removal of a papilloma caused the disappearance of the croup. Lastly, bear in mind always the possibility that croupy breathing may be the result of inhaling a *foreign body*. Careful attention to the history will generally keep you right here, because the onset in such a case is always, of course, extremely sudden.

SCREAMING.

Constant *screaming* is a distressing and not infrequent symptom in young children for which you will sometimes be consulted. It is usually, I need hardly say, a sign of pain, and it will be well for you to keep clearly before your minds the chief causes of such pain, so that you may be able to make a correct diagnosis, as you sometimes must do, by a method of exclusion alone.

Certainly the commonest cause of pain which results in screaming in an infant is **colic.** You will recognise it by the fact that the screaming is accompanied by drawing up of the thighs upon the abdomen and kicking of the feet. The motions, too, are usually abnormal in one way or another. Sometimes the mother or nurse will be able to tell you that the screaming ceases with the passage of flatus from the bowel, and if you get such a history the diagnosis cannot be in doubt.

Renal colic, which is far from infrequent in childhood, may simulate intestinal colic very closely. It is rarely, however, so persistent, and you will be able to recognise grains of uric acid on the napkins. If the screaming is specially associated with the act of micturition you should suspect **hyperacidity of the urine,** or, in boys, **phimosis.**

Earache is another pretty frequent cause of screaming, but usually it will have been noticed that the child puts his hand up to his head when the pain and screaming come on. Infants who are the subjects of **scurvy** always scream a great deal. Here the screaming tends to come on with movement, and one leg or both is kept as still as possible—quite different from the restlessness and kicking of colic. The screaming of scurvy may be simulated by that due to the **hyperæsthesia** which is often present in the early stages **of infantile paralysis** (see p. 221), but the absence of any thickening of the limb in the latter condition should keep you right. The screaming of early **meningitis** is not likely to lead to error, for it is of a peculiar and penetrating character, and is accompanied by more or less of head retraction and other signs of irritation in the meninges.

Lastly, if you can find no other cause for the screaming it is always well to remember the possibility of **mental deficiency,** for some mentally defective children exhibit persistent screaming almost from birth without there being anything one can discover to account for it.

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 (see p. 7). Assuming that you have satisfied yourself that the prominence is really pathological, you should look next for signs of **rickets,** bearing in mind how greatly the abdomen may be enlarged in that disease. The reasons for this I have already pointed out (p. 118),

Tuberculous peritonitis 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 examine carefully 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.

Enlargement of the viscera, particularly of the spleen or kidney, may cause a general abdominal prominence. 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 even by the most experienced and careful. The chief causes of enlargement of the kidney in early life are sarcoma and hydronephrosis. Great enlargement of the spleen is usually associated in infancy with anæmia (see Splenic Anæmia, p. 273), and in later childhood has probably often a syphilitic origin. Myelogenous leukæmia is an

extremely rare cause of large spleen in the child, and malaria, in this country, is also a very uncommon occurrence.

JAUNDICE.

The last of the common symptoms to which I wish to direct your attention is jaundice. This may be a sign of several different diseases of the liver, the differential diagnosis of which is often a matter of no small difficulty. It will be convenient for us to consider the case of infants and of older children separately.

The majority of infants suffer, as you know, from a slight degree of jaundice coming on a few days after birth, to which the term **icterus neonatorum** is applied. Usually this is but a trivial affair, lasting only for a week or so, but occasionally it may be prolonged for several weeks, and in such a case your diagnosis may be in doubt. What the cause of this unusually severe development of icterus neonatorum may be I cannot tell you, but it is interesting to note that in at least two cases I have known it to be followed by severe and persistent anæmia. Another, but very rare, cause of jaundice coming on at or soon after birth is **congenital obliteration of the bile-ducts.** Usually you will have no difficulty in recognising this condition, for it is characterized by a very severe degree of icterus with complete absence of bile from the stools. The liver, too, is notably enlarged, hard, and irregular, and the spleen is palpable. It is a serious condition, and usually proves fatal before the end of the second month.

Syphilitic hepatitis—a small-celled infiltration of the liver—is a third and not uncommon cause of jaundice in infancy. It is not always easy to tell it from a severe

case of icterus neonatorum, but other signs of congenital syphilis are usually present, and the liver is definitely enlarged. Whether it is ever recovered from I cannot say for certain, but I think I have seen cases clear up under mercury. In older children by far the commonest form of jaundice is the **catarrhal** variety. Do not be surprised in such cases to find the liver enlarged. It usually is so, reaching, perhaps, two or three fingers' breadths below the edge of the ribs, but rarely further. Sometimes it is slightly tender as well. **Hepatic cirrhosis** is the condition from which you have to diagnose catarrhal jaundice, and it is not always easy to do so. The icterus in such a case, however, is usually but slight, and there is a prolonged history. Enlargement of the spleen, too, is the rule in cirrhosis, whereas it is absent in the catarrhal form.

Of the less frequent forms of jaundice, such, for example, as occurs in the course of **infective processes,** of **acute yellow atrophy,** and other rare diseases, I have not time to speak in this lecture.

LECTURE XIX

SOME MEDICAL ASPECTS OF ADENOID VEGETATIONS IN INFANCY AND CHILDHOOD

GENTLEMEN,—I wish to direct your attention to-day to some medical aspects of adenoid vegetations as exhibited in the case of infants and young children. I shall assume that you are familiar with the local symptoms which are due to the presence of adenoid growths, and also with the chief surgical complications to which they may give rise—such, for example, as otitis media and other secondary suppurative processes—and shall confine my remarks more to those diseases which accompany adenoids, and which, more strictly speaking, come within the cognisance of the physician. I do not propose to deal at all with the pathology of adenoids, because that is at present very ill understood, and I can add nothing to what you already know on that aspect of the subject.

ADENOIDS IN INFANTS.

It will be convenient, I think, to divide the subject, first of all, into adenoids as occurring in infancy on the one hand, and in older children on the other. Perhaps it may be a surprise to some of you to hear that adenoids occur in

infancy at all. Everyone knows that adenoids occur in older children. But I go further, and say there is reason to believe that adenoids may be a **congenital** abnormality—that is to say, that a child may be born into the world with adenoid growths in its naso-pharynx. In order to realize the **symptoms** that adenoids may produce in young children, our best plan will be to look at the clinical history of some cases which I have had under observation. I shall begin, in the first place, with the simplest symptoms to which adenoids may give rise in infancy. For instance, a child of five months old was brought to me with a history that it had been **snuffling at the nose** and had difficulty in breathing since the time it was born. The family history was perfectly good, and there was no suspicion of congenital syphilis. On examination the respiration was laboured, and the lower interspaces were drawn in during inspiration. The child had also a markedly adenoid facies. On examining the naso-pharynx one found quite easily a mass of fibrous adenoids, and after their removal the child was brought back with almost complete relief from all its symptoms. That would be one of the simplest cases of adenoids in infancy, and you will observe that it occurred in a child as young as five months. I have seen another case in a baby of nine months, which was brought on account of discharge from the nose, accompanied by occasional **epistaxis.** This child had also a slight bronchitis, which is a very common accompaniment of adenoids and marked vegetations in the naso-pharynx. In that case, too, the epistaxis and the other symptoms disappeared after the naso-pharynx had been cleared out.

Another group of cases show, as their chief symptom, **cough.** A child only two months old was brought to me

on account of a choking cough, which came on in paroxysms. (That is worth noting ; it is the typical sort of cough which is associated with adenoids in the naso-pharynx. On account of this cough some of the patients with adenoids are supposed to be suffering from whooping-cough.) Further, the cough in this child was invariably accompanied by the involuntary passage of fæces, owing, I suppose, to the extreme spasm which it caused. Examination of the child showed the lungs to be perfectly healthy, but the naso-pharynx was full of adenoid growths.

Another baby had **snoring respiration,** and was only able to sleep in snatches, out of which he started up in a condition of partial asphyxia. During the day he also suffered from paroxysms of choking cough, and sweated profusely about the head, which is another symptom not infrequently associated with adenoid growths in infancy. In this case also great and immediate relief followed operation.

Another class of case exhibits **stridulous breathing** as the chief characteristic. A baby eighteen months old was brought to me for a ' catch in the breath,' which came on at intervals during the day and was also present during sleep. The veins of the head and scalp were distended, particularly at night, and there were moist sounds in the lungs. The child had enlarged tonsils and adenoids. I subsequently heard that he died suddenly a day or two after I saw him, and before operation was peformed. I have known that occur in another instance, but in the majority of cases one finds that the stridulous respiration is much slighter in degree, and the children after having been submitted to operation make a good recovery.

Another class of case is that in which the child has **difficulty in swallowing.** A child sixteen months

old was brought on account of difficulty in swallowing, this difficulty having been observed for a long time. The trouble was found to be greatest in the case of liquids, and on that account the child experienced difficulty in taking the bottle, and had to be fed by means of a spoon, and then the fluid tended to run down the side of the mouth unless the head was held very much back. Examination showed that the tonsils were enlarged, and there was a mass of adenoids in the naso-pharynx. The throat was operated upon, and when the child was seen two months later the mother reported that there was very great improvement. Another child, three years and six months old, had the same difficulty in swallowing, which dated from an attack of measles one year previously. The patient suffered also from deafness. The naso-pharynx was full of adenoids, and there was some enlargement of the tonsils. That child has been operated upon, but I do not yet know to what extent the symptoms have improved.

You will see from a recital of these cases that the **symptoms of adenoid growths in infancy** may be very various. They may be divided into three groups. First, there are those in which the chief complaint is snuffling and nasal discharge, occasionally accompanied by epistaxis. The second group is that in which the chief complaint is dyspnœa, often paroxysmal in character, and the breathing is definitely stridulous, frequently accompanied by attacks of choking cough. The third group comprises those in which the difficulty complained of is associated with swallowing, and especially with the swallowing of liquids.

The presence of adenoids in each of these groups **may simulate other medical diseases,** and it is on

that account that they are particularly interesting to the physician. For instance, where there is nasal discharge, snuffling, and epistaxis, one has to distinguish between adenoids and **congenital syphilis.** You know that one of the symptoms of congenital syphilis is snuffling and the discharge of ichorous fluid from the nose, and there may occasionally be a little bleeding. Under such circumstances it is often difficult to tell whether the discharge and snuffling are due to congenital syphilis or to the presence of adenoids in the naso-pharynx. The only way to settle the matter is to make careful inquiry into the family history and to look for other signs of congenital syphilis besides those in the nose. In addition, I think the discharge from the nose due to the presence of adenoids begins sooner after birth than it does from congenital syphilis. You may know that there is a distinct interval between birth and the appearance of the discharge of congenital syphilis, and sometimes that interval amounts to several weeks. But the snuffling and discharge from the presence of adenoids may begin immediately after birth. You might think that a local examination of the naso-pharynx ought to settle the matter, but, unfortunately, it is difficult to examine the naso-pharynx of children satisfactorily. If any of you have tried it you must have noticed that there is an extremely small space between the soft palate and the posterior wall of the pharynx in a young infant, so that you can hardly get your finger up behind the nose. So one can only infer the presence of adenoids in them from the symptoms ; one cannot, as is the case with older children and adults, make certain of it by digital examination. Anything like posterior rhinoscopy, I need hardly say, is quite impossible in young infants.

Another medical disease which may be simulated by these growths is the condition known as **congenital laryngeal stridor.** Some children are born with an inability to breathe properly; they have a stridulous respiration which causes a crowing sound—a sound which is sometimes compared by the mother to that made by a hen. Such stridulous respiration may be very closely simulated by the second group of cases of congenital adenoid growths. I do not know that there is any means of always making absolutely certain which kind it is that one is dealing with. I think it is probable that congenital laryngeal stridor is to be regarded as a neurosis of respiration, the child not having yet learnt to co-ordinate its respiratory muscles. In health the two vocal cords open out when the child makes an inspiratory effort. What seems to happen in congenital laryngeal stridor is that the opening out of the true vocal cords lags behind the making of the inspiratory effort, so that the child elevates its chest, but does not simultaneously open out the true vocal cords. Then when the air rushes in there is a crowing sound. How the presence of congenital adenoids leads to the stridulous respiration I am not prepared to say, but there can be no doubt whatever that congenital adenoids do lead to that, for not only is it illustrated by the cases I have mentioned, but many other observers have recorded a similar condition. There was a case reported some time ago in the *Lancet* * by Dr. Eustace Smith, in which the symptoms disappeared immediately after the removal of the adenoids.†

* March 19, 1898.

† See also Lack (*Journal of Laryngology*, 13, p. 303, 1898); Lubet Barbon (*Revue des Maladies de l'Enfance*, 9, p. 499, 1891); Huber (*Archives of Pediatrics*, xi., 38, 1894); and Coupard (abstract in *Jahrbuch f. Kinderheilk.*, 28, p. 247).

Now we come to the third group, those in which the chief difficulty is that of swallowing. These are an interesting group, as they may lead to serious malnutrition. I have seen cases of marked wasting in young infants brought about by this **difficulty in swallowing** milk. The child is unable to suck the bottle, and has to be fed by the spoon, and that is often found inadequate. The mechanism of these cases is easy to understand. When the naso-pharynx is blocked by a mass of adenoids it is impossible for the child to breathe through the nose while sucking. He has only the mouth to both breathe and suck by, and consequently, when he is using the mouth for sucking he cannot be using it for breathing, and gets short of breath, and thereupon instantly ceases to suck. Accordingly, he gives up the bottle in despair, and all attempts to feed such a child by the bottle end in failure. Even the feeding by means of a spoon in such cases may be difficult, because I think the presence of adenoids interferes mechanically with swallowing—that is to say, not merely through the respiration, but by opposing mechanical obstacles to the act of deglutition. One way in which you can imagine that happening is that the mass of adenoids may prevent the elevation of the soft palate into the naso-pharynx, which is a normal occurrence during healthy deglutition. This difficulty in swallowing may sometimes be extremely marked. I remember a case which was brought to this hospital, and was under the care of one of my colleagues, in which the difficulty was so marked that the child could only swallow when it held its head very far back, like a bird. You have, perhaps, noticed that when a bird drinks it takes a mouthful and then holds its head back in order to swallow it. In the case of this child other children had given it things to

drink so as to have the fun of watching it throw its head back in order to swallow them. That child had a considerable growth of adenoids, and the condition improved very much after they were removed.

I should add that some writers have described **reflex nervous troubles** as the result of adenoids in infancy. Amongst these are convulsions, tetany, laryngismus, and vomiting. A good example of the first of these has been described by Dun.* A baby five months old had had convulsions for three months, the attacks being at first infrequent—once or twice a week—but had increased later on in spite of treatment until they numbered four or five each day. No cause for the convulsions could be ascertained, but there had been occasional slight nasal discharge, and the baby snored when laid on his back. The naso-pharynx was scraped, and the convulsions straightway ceased. That is a very striking case which it will be well for you to bear in mind.

So much for the occurrence of adenoids in young infants. They may lead to symptoms which it may be difficult to diagnose from those due to other medical diseases, and they are symptoms which may lead to serious impairment of health.

ADENOIDS IN OLDER CHILDREN.

Now, when we pass to older children we find the condition is much easier to study, because in their case you can make certain of the existence of adenoids by digital examination. I need hardly remind you of the enormous frequency of adenoids in children up to the age of puberty. After that age they tend to disappear spontaneously. But children

* *Brit. Med. Journ.*, September 12, 1903.

at the period of the second dentition suffer from adenoid growths with great frequency, and the danger is lest one should take an exaggerated view of the presence of these growths. Some of you who have attended my out-patients may think I am inclined to overestimate the importance of adenoids in children and the part which they play in medical diseases, and after the recent pronouncement of such an eminent specialist as Sir Felix Semon, perhaps it requires some little courage to maintain that adenoids produce remote or medical effects at all. Yet I think no one can see much of disease in children without being convinced of the frequency of such a condition, and the fact that it seems to stand in causal relationship to certain remote symptoms in many instances. I can only advance the histories of certain cases in proof of this statement.

Let us consider, first of all, the relationship of adenoids in older children to *diseases of the respiratory system*, because it is upon the respiratory system that the consequences of adenoids most directly fall. Now, the first respiratory disease which it is important you should recognise as a common consequence of adenoids is simple **bronchitis.** You will frequently have children brought to you with severe cough, and on examining the lungs you will find a few moist sounds at the bases. Such children will be found to be mouth breathers and snorers at night. It is not difficult to see how adenoids lead to bronchitis. What is the normal function of the nose ? It has been calculated that the nose yields up to the inspired air as much as two quarts of water in twenty-four hours ;* it adds that quantity of water vapour to the air which we inspire besides warming it up to the body temperature. It has also been

* Mayo Collier, *Journal of Rhinology*, January, 1901.

calculated by Dr. St. Clair Thomson and Dr. Hewlett that 1,500 micro-organisms are inspired every hour, and that sometimes this quantity is enormously increased. Yet when you come to examine the mucous membrane of the trachea you find that it is practically sterile—that is to say, the micro-organisms do not get beyond the outer passages. Thus it will be seen that the function of the nose is to moisten the air and to sterilize it, and if the nose is obstructed, as it is in marked adenoids, these two functions are imperfectly performed, and the child is breathing an unduly dry air, which is, moreover, insufficiently sterilized, and the consequence of that may be that the lining membrane of the tubes gets irritated, and the child develops bronchitis. If you are going to treat a case of bronchitis of that sort successfully you must first see that the upper air-passages are rendered patent.

Another respiratory condition definitely associated with adenoids in certain cases is **asthma.** I recognise that I am here on more dangerous ground, because asthma is one of those reflex neuroses the occurrence of which is apt to be exaggerated. I shall have to mention one or two reflex neuroses which arise not uncommonly in patients who are the subject of blockage of the nose or naso-pharynx, and one of those I certainly think is asthma. Take these two cases. A girl, aged eight years, was brought to me not long ago with the complaint that she had suffered from asthma since infancy. On examination the chest was found to be flat and badly developed, and there were a few moist sounds at the base of each lung, as well as an extreme development of adenoids in the naso-pharynx. The adenoids were removed and no other treatment was adopted, and four months after the operation it was reported that she was

perfectly free from the attacks. Another patient was a boy of six years and three months, who, since three and a half years of age, had been treated for asthma, but without any real benefit. The mother stated that he had a severe attack of nocturnal dyspnœa lasting for several hours occurring once a week. He had excessive enlargement of the tonsils and a large mass of adenoids. He also was operated upon without the adoption of other treatment. Three months later the mother reported that he was almost free from attacks except when he had a cold in his head. Without mentioning other cases in which there seemed to be a definite relationship between adenoids and asthma, those that I have referred to are quite conclusive. I admit there are cases of asthma and adenoids in which removal of the adenoids does not seem to cure—that is to say, if you find adenoids and asthma co-existing you cannot promise that removal of the adenoids will invariably cure the asthma. I had a very interesting case illustrating that point. It was that of a child five or six years of age who suffered from asthma. He lived in the country, and had suffered practically all his life from the disease. Unquestionably he also had a large mass of adenoids. He was brought to the hospital here, and when I saw him I recognised the presence of these adenoid growths, and I advised that they should be removed. This was done, and he remained in London several weeks. During that time he was practically free from attacks. Of course, I naturally concluded that the operation had cured the asthma, and he went back to his home in the country. I followed the case up, and I found that immediately he got back there the attacks of asthma recurred. This, therefore, seemed to be one of those cases in which the asthma only came on

in certain localities, the child having been born in a locality which produced it in him. In London he remained free from attacks. These cases are not infrequent, and you may be easily deceived by them. The mere presence of adenoids does not insure that removal of them will cure the accompanying asthma.

The next system which may be affected by the presence of adenoids is the *alimentary*. Those of you who have seen much of children's diseases, especially in the out-patient department here, must have been struck by the fact that perhaps the majority of the cases of adenoids which one sees in children after the second dentition occur in those who are dyspeptic, and in those who suffer from that particular form of **dyspepsia** to which Dr. Eustace Smith has given the name of 'mucous disease.' In some ways that is an unfortunate term, but there is no doubt that it does represent a definite group of cases, and cases which occur with great frequency. These children are usually brought to one with the complaint that they have cough, that they have no appetite, and that they are wasting, and, in addition, they are restless at night and suffer from constipation. One has often seen such patients sent up with the diagnosis of phthisis. If you examine them you will find that they have, in addition to the symptoms which Dr. Eustace Smith described in the stomach and intestines, adenoids in a more or less marked degree. It is difficult to be sure of what the relationship is between adenoids and dyspepsia. Some people go so far as to say that the constant swallowing into the stomach of the mucus secreted by the adenoids maintains a condition of dyspepsia. The only way of settling the matter would be to treat some of those cases by putting the naso-pharynx into a healthy condition, and observing

what the effect was upon the stomach. That I have not done, but I hope to carry out the treatment on these lines in other cases, with the view of seeing what the relation is between the adenoids on the one hand, and the dyspepsia on the other. But certainly in these cases it is extremely important to recognise the existence of adenoids because of the very frequent mistake which is made in regarding them as cases of pulmonary tuberculosis.

The next system which may be involved in cases in which adenoids are present is the *nervous system*, and we have a large number of cases of nervous disorders associated with, and I think in many cases causally associated with, adenoids in the naso-pharynx.

To the first group belong cases of general **mental dulness** or *hebetude*. That is a condition—the name 'aprosexia' has been applied to it—which implies inability to concentrate the attention properly. If you go into any of the special classes which are formed in the London Elementary Schools for backward children, you can pick out at once a large number of cases which suffer from adenoids. The way in which adenoids may produce this mental dulness or backwardness has been much disputed. Some people have supposed that the adenoids interfere with the proper return of lymph from the brain. They believe that the lymphatics passing from the brain are blocked, and that therefore the brain is kept bathed in the products of its own metabolism, and in that way its proper action is interfered with. Perhaps that theory is rather far-fetched. A simpler view is that the mental dulness in these cases is due to inability to hear well, that the pupil never properly hears what the teacher says. Such children are not, strictly speaking, deaf; they hear noises, but they do not

adequately distinguish words, particularly if they are at a little distance away, and hence they become backward in their class. Another theory seeking to explain an association between adenoids and this backward condition is that the brain is kept constantly in a state of partial asphyxia from the blood not being sufficiently oxygenated. Whatever the true cause is, there can be no doubt of the great frequency with which one finds this general mental dulness associated with adenoids. In all cases in which you are consulted about a child who is backward at school examine the naso-pharynx, and at the same time examine the ears. You will find in quite a large proportion of them that the interference with hearing is due to the presence of adenoids, and I can assure you, from my own experience, that many of them have been greatly improved by having the throat cleared out. I have that on the evidence of teachers in those special classes to which I have referred.

Another neurosis which may arise from the presence of adenoids is **night terrors.** It is not quite so easy in this case to explain the association, and the cases are complicated by the fact that in the majority of them there is that form of dyspepsia which I have already spoken of present as well. It is difficult to assign a proper share in producing the night terrors to the dyspepsia and to the adenoids respectively, but I have seen cases in which removal of the adenoids alone produced a great improvement in the night terrors. Rey has recorded * the case of a girl who was operated upon on five successive occasions for adenoids, and in every case this immediately relieved the night terrors, which only recurred with the recurrence of the adenoids themselves. That is a fairly conclusive case in

* *ahrbuch f. Kinderheilk.*, 45, p. 316, 1895.

regard to the association of the two. Certainly in the case of any child who comes under your observation with night terrors you should attend to the state of the naso-pharynx in addition to treating the general health. The proper way to do this is not to give nerve sedatives, such as bromides and chloral. The necessary thing is to attend to the digestive organs, and particularly to give the patients aperients.

Allied to night terrors is another neurosis, namely, **nocturnal incontinence of urine.** That is a condition which, beyond all doubt, is often *maintained* by the presence of adenoids, if, indeed, it is not actually caused by them. The first case of this sort that I saw was an instructive one, that of a boy in a Home in the east of London, who had suffered from nocturnal incontinence of urine nearly all his life. He was sent to me by the superintendent of the Home to see what could be done for him. I found he was a typical mouth breather, that he snored at night, and that he had a large mass of adenoids and enlarged tonsils. His throat was operated upon, and almost immediately the nocturnal incontinence ceased and did not recur. Similar cases have been recorded in large numbers by various other writers. The relation was first pointed out by Major in 1884, and was confirmed by other German writers subsequently; and Grönbech has recorded * 23 cases in which adenoids were operated upon, and of which twelve were fully cured, five were improved, two were somewhat benefited, three remained as they were, and one was lost sight of. Huber, in America, found † that out of 427 cases

* *Arch. f. Laryngol. und Rhinol.*, ii., p. 214, 1895.

† Abstract in *Medical Review*, iii., p. 182, 1900.

of adenoids sixty-one had incontinence, and in thirty-nine of them removal of the adenoids was followed in twenty-six by complete cure and in eleven by great improvement. So if you have a case of enuresis, certainly adenoids should be looked for, and if they are found they should be removed.

Amongst other and rarer forms of nervous disturbances to which adenoids may give rise one is **defect of speech.** I have seen several cases of adenoids associated with difficulty in speech. Usually these cases occur in young children, and in the majority of them there is reason to suppose that the adenoids have been congenital. A child of two years of age was brought to me because he was unable to use words at all. He seemed mentally quite bright, and apparently heard ordinary sounds well enough, and the only thing which could be found amiss was the presence of extensive adenoid growths. Operation produced partial improvement, but one found some time afterwards that the growths had recurred. That child has since been operated upon again, and it will be interesting to follow the case up and see what improvement of speech follows the operation. I do not know what the difficulty in the speech is due to in those cases ; it may be that the child does not hear words spoken by other people sufficiently clearly to enable him to imitate them.

Another minor neurosis which may accompany adenoids is **headache.** It is usually frontal, and is probably due to interference with the proper ventilation of the air sinuses. Such headache is frequent in adenoids. Dr. Crowley* went through a large number of cases, amounting to some hundreds, which had been operated upon here, and in 55 per

* *Pediatrics*, iii., No. 9, 1897.

cent. of them he found there was a history of headache, which had been cured by operation in about 50 per cent. **Vomiting** has been recorded as one of the rarer forms of neurosis, particularly by Breton.* He describes recurrent vomiting in a child which was cured by removal of adenoids. I have not seen such a case, but the peculiar spasmodic cough which adenoids give rise to frequently ends in retching, if not in actual vomiting. **Torticollis** is another and rarer condition which has been reported to be cured by the removal of adenoids. Gillette records three cases.† A still rarer form of neurosis of which I have seen one example only, which seemed to be due to adenoids, is **ataxy** or *staggering*. I remember a child, aged three years, who was brought to me with the complaint that he was wasting, had attacks of nose bleeding, and was very backward of speech, and apparently did not hear well. The child had excessive adenoids, and the typical adenoid facies, mouth breathing, and other symptoms. I did not have the throat operated upon at once. Soon afterwards the child developed ataxy, and staggered about as if he were intoxicated. This ataxy became more marked after the lapse of a few days, until eventually he was hardly able to stand at all. He was then taken into the hospital, and on examining him more carefully one found that he had marked indrawing of the tympanic membranes. There was no sign of cerebellar disease—that is to say, no optic neuritis and no vomiting, neither was there any complaint of headache. The case was watched for some time, but did not improve. It was rather puzzling to know what the ataxy could be due to, and one thought it worth while to remove the adenoids

* *Rev. mensuelle des Maladies de l'Enfance*, 18, p. 235.

† Abstract in *Pediatrics*, ii., p. 540, 1896.

on the theory that the indrawing of the tympanic membrane was affecting the semicircular canals. You know that a person with a large quantity of wax in the ear may suffer from acute staggering, and I thought it might be the same with this child. The adenoids were completely removed, and then the ears were inflated. Improvement began almost at once and continued steadily. The operation was performed nearly a year ago, and when I saw him quite recently he was in practically a perfect state of health, and I was told that there had been no tendency to ataxy since he left the hospital. I do not wish to claim that this was a case of ataxy due to adenoids, but I think it is very suggestive, as the improvement began immediately after operation.

Such, gentlemen, are some of the medical diseases which may be simulated or caused by adenoids, and the question is what degree of importance one should attach to the presence of adenoids in any given case. And here again I warn you against the dangers of exaggeration. I do not want to say for a moment that all those cases which I have mentioned were directly *caused* by adenoids, but they were all, as such cases very frequently are, *associated with* adenoids ; and in many of them when you remove the adenoids the other symptoms disappear. It is perfectly true that you can, in the majority of cases, find a predisposing cause for the bronchitis, or night terrors, or whatever it is, as well, yet in many of them one can fairly regard post-nasal growths as an exciting cause, and if you remove the growths great improvement follows.

TREATMENT.

Into the technique of the operation for the removal of adenoids I do not propose to enter. But one may say a word as to **when operation is indicated.** I do not wish you to understand that in all cases such as I have described if you find adenoids you should proceed to removal at once. I think it is safer, unless the symptoms are extreme, to try simpler measures first. Mr. Arbuthnot Lane has recommended **respiratory exercises** in the treatment of these conditions, and has said that if one has recourse to them systematically and persistently operation is in many cases unnecessary. Of such a line of treatment I have had little experience, because in the out-patient department of a hospital it is almost impossible to get it carried out with sufficient thoroughness and persistency to afford a reasonable chance of success. In ordinary out-patient practice I think the best plan is to try, first of all, ordinary cleansing of the nose. Very often one finds that symptoms are not due merely to the adenoids, but to their producing an excessive secretion of mucus, which becomes dry and causes blocking. If you can remove that, the adenoids of themselves may not be sufficient to produce serious symptoms. One of the best things for this is the **nasal douche.** A solution of borax (1 drachm to the pint) is a mild and fairly good solvent of mucus, and this is injected up the nostril and allowed to run out by the mouth, so as to wash out the nasal cavity. In many cases this has resulted in a large degree of improvement. In any case, if you intend to proceed to operation it is well to submit the patient to such treatment before the operation is undertaken. One is less likely to get sepsis

afterwards if one has first taken the trouble to see that the nasal passages are reasonably clean.

With regard to the **operation** itself, you must be prepared occasionally for failure—that is to say, you must be prepared for the possible recurrence of symptoms. When first I directed my attention to this subject I used to think that in many of the cases in which the operation seemed to fail the removal of the adenoids had been incompletely carried out. But now I think that is probably not correct. In many cases I am certain that the adenoids recurred, and I think one may go further and say that the younger the child the more likely such recurrence is to take place, and it is often well to prepare the parents for that, and to tell them that you may require to perform two or three operations before you get a satisfactory result. It was found by Dr. Crowley that of the cases operated upon here, quite a considerable proportion showed recurrence, although there was no reason to suppose that the operation had not been completely performed. So if one finds that the symptoms are not relieved by the first operation it is not fair to blame the surgeon and allege that he has not done his work properly.

Another cause for apparent failure from operation may be that the nasal passages are abnormal. There is nothing commoner than for adenoids to be associated with some other cause of blocking in the nose, such as enlargement of the inferior turbinated bones, or a disordered condition of the septum, and so on. And, of course, you cannot expect in these cases that mere removal of obstruction behind will result in much improvement so long as the anterior obstruction still exists. So in some of these cases it may be necessary to attend to the state of the anterior nares as well as to remove the adenoids.

Lastly, though in many cases operation may be expected to cure those cases by itself, you must never forget to **treat the underlying condition** which is so often present. Adenoids may certainly be regarded as a symptom of ill-health, and whilst treating the local disease, you must attend also to the general condition. These children frequently want tonics of different sorts, particularly iron, and they also require attention to the stomach. Many of them, too, are much benefited by a change of air. In other words, whilst you are attending to the exciting cause of their symptoms you must never forget to give attention also to the predisposing cause which is usually a delicate condition in the child.

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