COMMON DISORDERS AND DISEASES OF CHILDHOOD

G. F. STILL

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THIRD EDITION

THIRD IMPRESSION

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COMMON DISORDERS AND DISEASES OF CHILDHOOD

BY

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TO

MY FRIEND AND FORMER TEACHER

SIR JAMES FREDERIC GOODHART, BART.

M.D., HON. LL.D. (ABERD.), F.R.C.P. (LOND.)

THIS VOLUME

IS DEDICATED IN TOKEN OF HIGH ESTEEM

AND AFFECTIONATE REGARD

Thoughts that have tarried in my mind and peopled its inner chambers,
Sober children of reason, or the desultory train of fancy,
Clear running wine of conviction with the scum and lees of speculation,
Corn from the sheaves of science with stubble from mine own garner.

TUPPER.

PREFACE TO THE THIRD EDITION

The preparation of the Third Edition of this work has been under difficulties ; one's pen, never too ready, laboured 'invitâ Minervâ', for was not Mars in the ascendant ? Nevertheless, I have striven by addition and correction to increase the usefulness of the book. Dosage has required some modification in conformity with official alterations in the strength of various preparations in the new (1914) edition of the British Pharmacopœia. A chapter has been added on that curious and little-understood disorder of children, 'cœliac disease', and one on tuberculous glands in the neck, and by numerous additions to other chapters I have endeavoured to keep abreast of recent advances in medical knowledge. For Medicine there is no abiding in one stay ; like the Athenians, we are always seeking after some new thing ; but we seek it, not because it is new, but in the hope that it is better : only we must beware lest the glamour of novelty put a spurious value upon methods, especially of diagnosis and treatment, which can only be appraised at their true value when time and experience shall have shown their fallacies and limitations.

G. F. S.

July 1915.

PREFACE TO THE SECOND EDITION

There is perhaps less of trepidation in a second appearance than in the first introductory bow. Judgement has been passed, and if some have noted only faults, others have found, with Cervantes, that 'there is no book so bad but something good may be found in it'.

But indeed it would be churlish not to acknowledge the many kindly words of appreciation as well as of criticism which have come to me from many lands, from friends known and unknown. These have lightened in no small degree the burden of responsibility which one must needs feel, who sends forth a book intended to be a guide and help in the every-day problems of disease.

One cannot but realize that some of the faults and omissions of the earlier edition remain, and must perforce remain, owing to the purpose and character of the book. In particular I must crave indulgence for its apparent egoism—a fault it is true, but one which was inseparable from the object I had in view, which was less to present a formal and impersonal treatise on diseases of children than to extract from my own experience such facts and conclusions as might be helpful to others.

In the present edition I have followed this same plan in dealing with several disorders, amongst which may be mentioned enlarged tonsils and adenoid hypertrophy, epilepsy, asthma and hydrocephalus, which were not included in the previous edition.

The rapid march of scientific investigation has necessitated,

even since this book was first published barely two years ago, considerable modification of views on some of the diseases of children, for instance, infantile paralysis and congenital syphilis. Other alterations and additions have been made wherever such changes seemed likely to be of value, and if the revision and extension which the work has undergone add to its usefulness I shall be well content.

G. F. S.

February 1912.

PREFACE TO THE FIRST EDITION

THE original plan of this work was the putting together of lectures delivered at King's College Hospital and at the Hospital for Sick Children, Great Ormond Street; and in accordance therewith I had intended to call the volume 'Lectures on Diseases of Children'. But as the patchwork grew it became evident that, if it was to be in any sense a connected whole, it would be well to combine with the lectures other clinical studies, which had been written at various times for other purposes. Some of these studies, as also of the lectures, have appeared already in medical journals or in hospital reports, but only very few in the form in which they now appear, for larger experience has called for modifications and additions, and I have not scrupled to mutilate and transform my progeny until they bear scarce the semblance of their former selves.

For the most part, however, these chapters appear now for the first time, and having abandoned my original scheme, I was at a loss to find a name for my 'farrago libelli'. It is no systematic treatise, it has no claim to vie with the many textbooks which deal with the whole subject of disease in childhood, nor indeed is it written on the lines which a systematic work demands; I have chosen rather to be selective and discursive as it suited my bent; I have disregarded altogether that sense of proportion and perspective which is of the very essence of a systematic textbook.

My theme is mainly the everyday and the commonplace, the disorders which bulk most largely in the out-patient and in-patient clinics of a children's hospital, and in the

routine of private practice. On this ground I have adopted the title 'Common Disorders and Diseases of Childhood', which at any rate conveys the main purpose of the book. But I have not debarred myself from including some disorders which cannot by any stretch of the imagination be regarded as common; for instance, infantile scurvy and congenital hypertrophy of the pylorus—conditions which, when they do occur, are apt to be mistaken for much more common disorders.

Throughout I have had in view chiefly the practical and clinical aspects of disease, particularly diagnosis and treatment; but where pathology or morbid anatomy had a direct bearing upon any practical point I have not hesitated to consider it in detail.

Coming fresh to the perusal of my work at its finish, I am only too conscious of its many faults and failings. For one thing I notice a monotonous repetition of such phrases as 'I think', and 'in my experience'; these, on consideration, I have left unaltered, for they may serve to remind the reader that the views expressed are for the most part personal opinions based on personal experience, and although, as Oliver Wendell Holmes remarks, 'a man's opinions are generally worth more than his arguments', yet, in things scientific, opinions are only to be accepted in so far as they are found to tally with the observations, not of one man, but of many.

For the cases and statistics which I have recorded wherever they seemed to emphasize or illustrate points of practical importance I need make no excuse. In medicine, every addition to the sum of observed facts, though it be but a drop in the ocean of clinical experience, has a value of its own; and for this reason I have endeavoured as far as possible to compile figures and observations from my own case-books rather than to borrow from other writers.

But if my direct debt to others can be measured, it is impossible to estimate what I owe indirectly to the writings of far better observers than myself; there is scarcely a writer on diseases of childhood in Great Britain, America, or elsewhere from whom I have not at some time drawn valuable aid in the study of disease in children.

Whatever I have consciously borrowed I have tried to acknowledge in the text; but, as I have already implied, the source from which I have chiefly drawn has been my own clinical records, and if I have made no reference to this or that writer's work it is not for lack of appreciation, but because it would have been inconsistent with the purpose of this book to make it a *résumé* of other people's observations.

To the Editors of the *Lancet*, *British Medical Journal*, *Practitioner*, and *Clinical Journal*, I owe thanks for permission to make use of articles or lectures which I had contributed to their pages, and which I have now used in their original or in modified form. My indebtedness for some of the illustrations is acknowledged in the text: others are inserted by the kind permission of the *Lancet*, of Dr. L. E. Creasy and of Messrs. Power and Murphy.

In conclusion, I must thank the Publishers and Printers for much care and patience in the production of this book, and also Mr. J. K. Murphy, F.R.C.S., for kindly advice on several points. To my sister, Miss A. Still, I am indebted for invaluable assistance in the preparation of charts, the compilation of the index, and other laborious details of publication.

G. F. STILL.

HARLEY STREET, W.

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CHAPTER I

MEDICAL ASPECTS OF GROWTH AND DEVELOPMENT IN CHILDHOOD

Infancy and childhood differ from all other periods of life in being pre-eminently the time of rapid development both physical and mental. It is this peculiarity which gives to many of the diseases of childhood special characters so that both in their clinical course and in their morbid anatomy they differ from disease in later life. Delay in the acquirement of functions and perversions of development, for instance of the bones in rickets, constitute no unimportant part of the morbid manifestations in early life. For the recognition of departures from the normal, it is necessary to know what constitutes normal physical and mental development: the medical man, especially, who is frequently confronted with parents anxious to know whether their child is up to the normal standard in this respect or that, must needs have some knowledge of the facts and figures of development in infancy and childhood; he must be familiar with the normal course of events, as well as with the variations which occur as the result of ill health. No man can carry in his memory all the weights and measures, dates and numbers which might be or have been compiled in connexion with this subject of growth and development; but it is possible with very little effort to carry in the mind a sufficient outline of statistics, to enable us to fill in the particular figure for any intervening age with sufficient accuracy for practical purposes. I shall not attempt to reproduce here any exhaustive tables, but only to give such facts and figures as can be stored in the memory and so kept readily available for use.

Let it be remembered that Nature is no stickler for uniformity: we say that the first tooth should appear at six months, but one infant will have it at four months, another will not have it till nine months, but both may be perfectly healthy; and so with the weight, how much unnecessary anxiety there often is because

an infant weighs a pound—nay, even a few ounces—less than the tabulated weight for the age! a figure which has been obtained merely by average from widely differing weights. Similarly in mental development, there is a certain range of variation which is consistent with mental health; one child will say several words at twelve months, another who proves subsequently to be equally intelligent will say nothing until he is nearly eighteen months old. Let it be recognized that our standards of development represent averages only, and that there is a variation which belongs to health as well as one which belongs to ill health.

Weight. There is no more delicate index of the health of an infant than the progress of the weight, which should be recorded weekly until the end of the first year, and once a month during the second year. Such a record is often of great value when illness occurs and particularly if there is any digestive disorder. A history of the different modes of feeding which have been tried tells us very little when we have no other information beyond vague statements that this or that food did not agree, but when there is in addition a definite record of the weight from week to week, a comparison of this with the dates of changes in feeding often gives important guidance.

There are times when weighing more often may be necessary; in severe cases of marasmus, where success in treatment depends upon frequent adjustment of the feeding to the capacity of the infant, it may be advisable to weigh twice a week or even on alternate days: if a whole week is allowed to elapse before the effect of the feeding is determined by the infant's weight several ounces may be lost in the meantime for lack of some needful change in feeding, and every ounce lost may seriously diminish the infant's chance of recovery.

But in health, there is no need to weigh more than once a week, and indeed more frequent weighing gives rise to frequent and groundless alarm, for the irregularities which are normally present in the weekly rate of progress are still more in evidence when the infant is weighed at shorter intervals, and it is difficult for parents to realize that Nature's line of progress in an infant's weight is not the regular curve which is misleadingly drawn on some weight-charts as the normal line, but is often rather a zigzag than a curve. No infant gains the same amount every week, sometimes it is less, sometimes more, and not very rarely in health there is little or even no gain one week and perhaps a larger gain than usual the following week.

With these cautions, the following data make a useful method of carrying in one's mind the average weight in health :

Birth	7 lb.
At 5 months	14 lb.
At 12 months	21 lb.
At 2 years	28 lb.
At 7 years	49 lb.

The weight, as will be seen, is doubled at five months, trebled at twelve months, quadrupled at two years ; all these weights may be remembered as multiples of seven.

A child should gain about 4 lb. a year between the end of the second year and the age of seven years ; and 6 lb. a year from the age of seven years to the age of thirteen years.

During infancy the progress of the weight is arrested by the most trivial disturbance of health, a little bronchial catarrh, even a simple coryza, is cause enough, and constipation, especially chronic constipation, is a common cause of failure to gain weight. The effect of dentition also is very striking ; often the weight ceases to rise or rises very little so long as a coming tooth is worrying the child, whereas directly the tooth is cut the weight begins to rise more rapidly again. In part, no doubt, this is due to the loss of appetite which so often accompanies difficult dentition, but I think not entirely, for the arrest of weight occurs sometimes where the infant continues to take food well, and it seems possible that it may be due in part to some influence on the nervous control of metabolism. Whatever the explanation may be, the effect of such slight causes on the weight is worth remembering, for the feeding is often changed quite unnecessarily and unwisely, where the fault lies not in the food but in some merely transient disturbance such as I have mentioned.

Length. The length of the new-born infant is 19 inches ; at one year it is 27 inches : and during each subsequent year up to five years of age there is a gain of $3\frac{1}{2}$ inches ; and each subsequent year a gain of 2 inches up to the age of fifteen years, when growth becomes much less rapid.

Size of head. The measurement of the head is of importance chiefly in connexion with mental deficiency and where the possibility of hydrocephalus is in question.

The most useful measurement is the circumference ; this is often taken through fixed points such as the frontal eminence and the occipital protuberance, but such points are difficult to locate exactly so as to be sure that any subsequent measurement is taken through precisely the same points ; it is therefore better to take the largest circumference which can be obtained.

The maximum circumference at various ages is, according to my own observations :

Birth	13 inches.
3 months	15 inches.
5 months	16 inches.
9 months	17 inches.
12 months	18 inches.
3 years	19 inches.
7 years	20 inches.
13 years	21 inches.

In the mentally defective the head is usually below the average in size. In a series of one hundred children of feeble intellect the head showed a circumference decidedly below the average in 60 per cent., while in 6 per cent. the circumference was considerably above the average. Apart from the few forms of mental defect which are regularly associated with abnormally large size of the head (cretinism, hydrocephalic idiocy, and the so-called hypertrophy of the brain), an unusually large circumference is met with in rickets, achondroplasia, and hydrocephalus.

Fontanelles. The anterior fontanelle varies greatly in size at birth ; in some infants it measures scarcely half an inch in either direction, in others it measures about two and a half inches laterally and fully three inches antero-posteriorly. In some infants, again, it diminishes steadily in size from the time of birth ; in others, and probably in the majority, it enlarges until about the age of nine months and then gradually diminishes. At one year it should measure not more than one inch in either direction. It should close about the age of eighteen months ; but in this also there is considerable variation, I have found the fontanelle completely closed in a healthy infant at thirteen months, I have also observed it to be open at two and a half years without any evidence of disease.

The commonest cause of delay in closure is rickets, in which the fontanelle is often open as late as two and a half or three years: I have seen it open as late as four and a half years in this disease. There are other causes : the fontanelle may be kept open by the tension of hydrocephalus : in the Mongol type of imbecility there is often considerable delay in closure, I have known the fontanelle to be open in these children as late as six years of age. There are also infants in whom, without any evidence of disease, the ossification of the skull is unusually slow, so that not only does the anterior fontanelle remain open unusually late, but sometimes the parietal bones remain ununited at their upper borders, and the posterior fontanelle,

which is usually closed within six weeks after birth, remains open for three or four months, and sometimes the lateral fontanelles, anterior and posterior, which should be closed or almost closed at birth, are open for several weeks. In these cases there is often some diffuse yielding on pressure near the edges of the parietal and occipital bones, but such a condition is not to be confused with craniotabes, which, as its name implies, is an acquired condition, a loss of already formed bone, whereas the diffuse yielding to which I refer is due only to delay in the formation of bone.

Dentition

The cutting of the milk-teeth begins with the appearance of the lower central incisors, which takes place usually between the ages of six and eight months. But there is considerable variation in this: some healthy infants show their first tooth at four or five months, whilst others, apparently equally healthy, show no teeth till nine or ten months, occasionally even until twelve or thirteen months old. Both unusually early and unusually late dentition seem to be a family trait in some cases; in one family under my observation one boy showed the first lower incisor at fourteen days, and his second at twenty-one days old; his brother was born with one lower incisor: in another family one girl had two lower incisors at six weeks old, her sister had her first tooth at four months.

Such unusually early commencement of dentition unfortunately does not mean an unusually early completion of dentition; there is often a long interval between the earliest eruption and the next tooth; for instance, the child already mentioned who had her two lower incisors at six weeks, had no more by the time she was seven months old. The presence of teeth at birth is seldom followed by the cutting of other teeth before the usual time; these congenital teeth also are apt to be small and ill-placed in the gum; this, however, is not always so; I have known them to be as good as those acquired at the usual time.

Whilst the effect of rickets is to delay dentition, syphilis, unless complicated by rickets, as it often is, seems not to interfere with the eruption of teeth, indeed it has been thought rather to conduce to early dentition; this, however, I cannot affirm from my own observations.

The date of appearance of the individual milk-teeth varies greatly. At twelve months eight teeth should be present, and the twenty teeth should have been cut by the end of the second

year. But the intervals between the cutting of teeth are most irregular : teeth may be cut in quick succession for a time and then a long pause may occur ; for example, Rose H. cut two teeth at the age of four months, and at twelve months she had twelve teeth, but during the next eight months, without any apparent cause, no more teeth appeared. In other cases where dentition appears to have proceeded very slowly for several months it is completed with unexpected rapidity by the appearance of several of the later teeth in quick succession.

The *order* of eruption is more constant, and is as follows :

Lower central incisors.
Upper central incisors.
Upper lateral incisors.
Lower lateral incisors.
First upper and lower molars.
Canines.
Second upper and lower molars.

Irregularity in the order of dentition is, however, not very rare in healthy infants. In some forms of mental deficiency dependent on developmental faults in the brain, abnormalities both in the time and in the order of dentition are particularly frequent : in Mongolian imbecility, for instance, there is often no eruption of teeth until some time in the second year, and the first to appear is very commonly a molar : a Mongol child, Eva C., at the age of twenty-one months, had only two teeth, and these were molars ; at the age of two years and eleven months six molars had appeared and had been followed by two lower central incisors ; a microcephalic idiot who showed her first tooth at seventeen months had only ten at twenty-two months, which had appeared in this order : two upper central incisors, one lower lateral incisor, four molars, one upper lateral incisor, two lower central incisors.

Disorders associated with dentition. Teething as an explanation of symptoms is too often a cloak for ignorance, but I would protest against the tendency nowadays to assume that because dentition is a physiological process therefore it is incapable of causing disturbance of health: pregnancy is a physiological process, but I suppose no one would deny that pregnancy may disturb the health in many ways. It has been said that teething produces nothing but teeth. I venture to think that there is more wit than truth in this: and, at the risk of being considered old-fashioned and unscientific, I shall mention some of the disorders to which in my opinion teething may give rise. I admit the difficulty of proof ; we all know that

coincidences are apt to be mistaken for cause and effect, but I am not inclined to disregard the accumulated experience of generations of intelligent parents, and still less the observations of skilled observers, who affirm without hesitation that dentition may cause certain disturbances of health. How it causes these disturbances may be doubtful: knowing how profoundly nervous influence can modify the functions of most organs in the body, I see nothing improbable in the supposition that some of the disorders produced by dentition may be due to reflex nervous disturbance; and I am the more inclined to think so when I see, as I sometimes do, an infant who has become 'nervous', with occasional twitching, rolling his eyes up, and giving a short sharp cry, and clearly being on the verge of convulsions, whilst the gum is swollen and tense over a coming tooth, whereas no sooner has the tooth come through than all these symptoms rapidly subside. If such excitability of the nervous system can be produced, as I think it undoubtedly can, by dentition, I see no reason why the transient bronchial catarrh, or the slight looseness of the bowels which recurs in some infants with the eruption of the teeth, may not be due to nervous influence.

Certainly there is nothing more improbable in this view than in the supposition that an infant who has been sucking a 'dummy' or living on a particular food for weeks or months without disturbance of health, should suddenly develop symptoms of disorder from these sources, coincidently with the eruption of a tooth, and then, after losing these symptoms directly the tooth is cut, should develop them again when another tooth is cut, and all this without dentition playing any part in the production of the symptoms. I am well aware that there are infants who cut their teeth without the slightest disturbance of health, but when I am told that dentition cannot disturb the health, and that even local worry is never due to teething but to a septic condition of the gum from dirty 'comforters' or from contamination by the various aids to teething which are in common use, I confess that I am not convinced.

It is quite possible that an inflammation of the gums may be induced in this way and may make them tender, especially when distended by a coming tooth; but as a matter of observation there is no vestige of any such inflammation to be seen in many cases where it is obvious to any one who is familiar with baby-language that there is considerable discomfort around the coming tooth. It seems reasonable to suppose that the discomfort may be due, as the appearances suggest, to tension in the gum; certainly, unless the appearances wholly belie the facts, there is

often considerable tension before the tooth is cut, and when one considers that tension from any cause is one of the most potent producers of pain one may well suppose that even slight tension can cause discomfort. I suspect that long before the gum has a distended appearance there may be some tension in the deeper parts : I would not even reject with scorn the view put forward by Mr. Tomes that there are two stages at which a coming tooth may cause irritation ; the first when it is forcing its way through the bony ring at the margin of the alveolus, the other when it is making its way through the gum. I am satisfied from my own observations that dentition sometimes begins to cause definite and characteristic symptoms, particularly the slight nervous excitability which I have mentioned, several weeks before any teeth are cut and before there is any visible distension of the gum.

I suppose the first indication of the approach of dentition is usually the increase of saliva : the infant begins to drivel. Unfortunately this is not all in many cases. The infant becomes fretful, sleeps badly, awaking often with a cry, and as a result of disturbed sleep soon comes to look pale and ' out of sorts '. Not only does he cram his fingers into his mouth and attempt to bite any object within reach, but often he plucks at his ears or at the hair over the temple, and beats his head with his fists, or bangs it against the pillow or the side of the cot. Head-rolling also, the monotonous movement of the head from side to side as the child lies in its cot, is probably due in some cases solely to the irritation of teething ; I say ' in some cases ' because it is important to remember that both head-rolling and head-banging may be due to the much more serious irritation of middle-ear catarrh.

A very common result of the worry of dentition is loss of appetite : sometimes for the few days immediately preceding the eruption of a tooth, sometimes for a much longer period of many weeks during the dentition period, the infant will refuse some feeds entirely, and take others only in small quantity, and though the appetite may improve when a troublesome tooth has been cut, it soon fails again with the worry of another tooth. This loss of appetite during dentition would be of no importance were it not for the anxiety which it causes to parents ; it may retard the gain of weight, but otherwise has no ill effect, and the medical man can assure the parents that it is a common and harmless result of teething.

Failure to gain weight, or diminution in the rate of gain is, as

I have already stated, another very common result of dentition : occasionally even there may be a loss of an ounce or two in the week whilst a tooth is worrying, but it is exceptional for any actual loss of weight to occur ; as a rule the effect is only a reduction in the rate of gain, so that an infant who has been gaining perhaps 6 to 8 ounces per week gains only one or two ounces during a week when the gum happens to be tense and worried. In this way the progress of the weight is apt to be very irregular in some infants during the period of dentition, but the rapid gain of weight in the intervals between the cutting of teeth is generally sufficient to compensate for the delay.

Dentition alone seems to be sufficient cause for a rise of temperature in some infants ; indeed it would be strange if this were not so. A slight dyspepsia or a little constipation will certainly cause a considerable degree of fever for a few hours in some older children, and in nervous children an evening rise of temperature to about 100° for many weeks may be due to very slight causes, dietetic or otherwise : it might be expected, therefore, that during the first few months of life, when nervous instability is even greater than in later childhood, the peripheral irritation which is obvious in the hot tense gums and in the fretfulness of the infant should express itself sometimes in a rise of temperature. The fever is sometimes transient, reaching 101° or 102°, and subsiding after a few hours ; sometimes, and I think more rarely, there is an evening rise to about 100° for several weeks during the progress of dentition.

Two disorders which are often attributed to the influence of teething are bronchitis and diarrhœa. How often one hears the tale that a baby 'cuts its teeth with the bronchitis.' Now I have no doubt that in many cases the association is merely fortuitous ; infancy is a time of life which is particularly prone to bronchitis, as it is also to diarrhœa, and both must therefore be coincident often with dentition. But I think there is more than coincidence in some cases : both bronchitis and diarrhœa are clearly traceable to nervous influences in certain disorders of later childhood, the bronchial catarrh of the asthmatic child, and the lienteric diarrhœa to which Trousseau gave the name of 'nervous diarrhœa' are admittedly dependent upon nervous influences, and sometimes upon influences very remote from the organs affected ; the asthmatic bronchitis, for instance, may be started by too heavy a meal, or by some particular smell. It is at least possible, therefore, that the worry of dentition may, by

its effect upon the nervous system, cause a transient diarrhœa or a bronchial catarrh. It seems to me more reasonable to admit some such hypothesis as this, than to ignore the very general experience both of the laity and of more competent observers that some infants suffer with a recurrence of bronchial catarrh, or with looseness or green coloration of stools, whenever the gums become swollen, tense, and irritated by a particularly troublesome tooth.

I hold no special brief for the nervous origin of the catarrh in these cases : it may be as some have suggested that the explanation is to be sought in some disordered state of the mucous membrane of the mouth, with alteration of saliva interfering with digestion, and that the respiratory mucosa undergoes a catarrhal change by extension from some catarrhal condition of the oral mucosa, but against such an explanation is the fact that the mucous membrane of the mouth in general, or even of the gums themselves, only exceptionally shows any inflammatory change with dentition. Catarrhal stomatitis is occasionally seen over the gums as a greyish-white discoloration, as if the gum had been rubbed with silver nitrate, but this is uncommon in my experience and its appearance does not suggest that there is any sufficient alteration in the mucosa or secretions to account either for diarrhœa or bronchitis.

I have repeatedly seen children who throughout the first dentition had occasional attacks of vomiting coming on without apparent cause, and with little or no disturbance of the bowels, and passing off again after a day or two. Each attack occurred just as a tooth was coming through the gum, and the recurrence of this association has been so striking as to leave no doubt whatever in my mind that the vomiting was excited by the worry of dentition.

Dentition certainly exercises a very marked effect in increasing the instability of the nervous system : some infants when much worried by a coming tooth show a tendency to convulsive twitching, sometimes with upward rolling of the eyes or occasional slight strabismus, and although they may never lose consciousness or fall into a general convulsion, they are evidently, so to speak, on the brink of such an attack. In infants already predisposed by neuropathic heredity or by rickets to ordinary infantile convulsions or to epilepsy, the period of dentition is undoubtedly a time of peril. I shall have occasion hereafter to consider the distinction between infantile convulsions and epilepsy ; here it need only be said that attacks like petit mal

are sometimes much aggravated by dentition, and I have known them to cease when the last tooth had been cut.

A common effect of dentition, and a very troublesome one, is sleeplessness ; it does not seem to be caused by any pain, but rather to be part of the general nervous excitability of this period in some infants.

Various skin eruptions are commonly attributed by parents to teething, and if lichen urticatus be related to nervous influences, as urticaria certainly is, it seems conceivable that dentition may play some part in its production, as is often supposed ; but I have never been able to satisfy myself that either lichen urticatus or eczema bore any special relation other than coincidence to teething. Both are particularly frequent during the teething period ; lichen urticatus is closely related to digestive disturbance, and may in this way be indirectly connected with the eruption of teeth, which, as I have already mentioned, is apt to be associated with disorder of the bowels, but both these skin eruptions are common also before and after the dentition period, and show on the whole so little tendency to recurrence specially at the times when the teeth are appearing, that there does not seem to be sufficient ground for asserting that they are due to teething.

A rare association with dentition is violent screaming without apparent cause : this is quite independent of any digestive disorder and not obviously due to any pain. It occurs in some cases several times in the day and night, and this continues with remissions for many weeks during the most troublesome stages of dentition. This screaming has seemed to me comparable to the violent paroxysms of screaming without apparent cause or upon the slightest thwarting which occur occasionally in children of markedly nervous temperament. The paroxysms in both cases are so violent and so obviously unnatural that medical advice is sought ; unfortunately the condition is sometimes very intractable and is particularly distressing, as the screams of the child can be heard at a considerable distance, and might give neighbours the idea that the child was being ill-treated.

Photophobia is another rare result of the irritation of dentition. It is so pronounced that the child will cover his eyes with his hands or bury his face in the pillow or against his mother's breast, when exposed even to the moderate light of a sunless day. In a brighter light, he will screw up his eyelids tightly like a child with a corneal ulcer, but examination shows that there is nothing abnormal in the eyes or eyelids, though in some of these cases the eyes will ' water ' when exposed to light. In one

child, aged fifteen months, the photophobia persisted for nine weeks; in another, aged ten months, it began six days before the eruption of the first tooth, and lasted at least four months. In some cases the photophobia is so extreme that the child cries or even screams violently as if in pain when exposed to any bright light, or even to ordinary daylight.

I have seen cases in which, during the eruption of a tooth, meningitis has been simulated by head retraction, which passed off soon and which seemed to be due entirely to some reflex irritation from the teeth. Increased tension in the middle ear sometimes produces head retraction in infancy, as has been proved by the rapid disappearance of head retraction after puncture of the membrana tympani; and therefore it seems only natural that the irritation of a tense gum might have a similar effect. Of course it is possible that in such cases there is actually some middle ear catarrh, to which the head retraction is due rather than to any irritation of the gums, but if so there has been nothing else to suggest ear-irritation in the cases to which I refer.

Parents very commonly attribute discharge from the ear to the influence of dentition, but although the two are often coincident I know of no special tendency of otorrhœa to recur with recurring stress of teething, nor of any reason for supposing that there is any causal connexion: but if the bronchial catarrh which occurs with dentition spreads by extension from the oral mucosa, there may be some catarrhal condition of the pharynx and naso-pharynx also which would account for the otitis media.

A rare disorder which is closely related to dentition is spasmus nutans or head-nodding with nystagmus. This disorder is so distinctly coincident with dentition in its onset and cessation, and is so definitely aggravated by the eruption of a fresh tooth, that I am disposed to share Henoch's view that there is a causal relation.

With regard to all these dentition disorders it is clear that the causal relation of dentition to the disorder is an assumption resting upon no more solid basis than the observation of many cases in which the occurrence of the disorder has not only coincided with the period of dentition but has shown a special tendency to coincide repeatedly with times of special irritation when a tooth was nearing eruption. This, however, seems to me a reasonable and warrantable assumption in many instances, and with increasing experience I am inclined rather to enlarge than to restrict my own conception of the rôle of dentition in producing disturbance of various kinds in infancy. At the same

time one cannot fail to recognize the danger of attributing too readily to dentition disorders which may really have some entirely different origin and which are apt to be lightly esteemed when they are considered as 'only due to teething': many a case of diarrhœa, dependent upon faulty feeding, or of otitis media due to some remediable condition of the naso-pharynx, is neglected with disastrous result because it happens to occur during the teething period and is regarded as a harmless phenomenon incidental to the process of dentition.

The second dentition occurs at a much less perilous age than the first: the extreme nervous instability of infancy has passed away and there is not the special liability to catarrh of mucous membranes which is so striking a feature in the first two years of life; moreover, most of the second teeth are not obliged to force their way through unbroken gums as must the milk teeth. And here I would point out to those who assert that the first dentition is a natural process and therefore cannot be a painful process or produce any irritation sufficient to cause symptoms, that the eruption of the wisdom teeth, which does involve the cutting of a way through the unbroken gum, and which occurs at an age (eighteenth to twenty-fifth year) when subjective symptoms can be accurately described by the sufferer, produces not merely local discomfort but neuralgic pain and also high temperature in some cases.

The order of the second dentition is subject to much variation: it begins usually with the appearance of the first molars or the central incisors at the age of six years, the sequence being commonly thus:

Permanent teeth.	*Date of eruption.*
First molars ('Six-year-old molars')	6 years
Central incisors	7 ,,
Lateral incisors	8 ,,
First bicuspid	9 ,,
Second bicuspid	10 ,,
Canine	11 ,,
Second molars ('Twelve-year-old molars')	12 ,,
Wisdom-teeth.	

In the large majority of children the second dentition is an entirely uneventful process, scarcely attracting the notice of the children or its parents; but there are children, particularly those of unusually nervous temperament, in whom it does, I think, occasionally produce some disturbance. I have known children

to become fretful and grind their teeth at night when the second dentition was just beginning and, after the tooth which was apparently causing worry had come through, these symptoms passed off. Two of the common nervous disorders of childhood, habit spasm and acquired enuresis—acquired, I mean, as distinct from the enuresis which is a persistence of the infantile condition —have their onset most frequently between the fifth and the eighth year, the time when the second dentition is commencing : it seems possible that their occurrence at this time is due at least in part to increased nervous instability produced by the second dentition.

Mental Development

The mental development of the child is subject to even greater variation than the physical growth. Immediately after birth the healthy infant shows evidence of ordinary tactile and temperature sensation, and taste is certainly present within the first twelve hours : an infant, aged ten hours, made a grimace of displeasure and then refused altogether to suck when I placed a camel-hair brush moistened with a solution of quinine between its lips, whereas, immediately after, when I tried it similarly with the same brush moistened with a solution of sugar, the infant sucked vigorously and smacked his lips with evident satisfaction.

Sight is present within a few hours after birth, at any rate a bright light causes blinking just before the infant is twenty-four hours old ; this of course may mean nothing more than the power of distinguishing light from darkness ; it is very difficult to ascertain at what age objects are first distinguished.

At four weeks a healthy infant follows objects with his eyes, but with very little constancy, and even at four months I have found that some healthy infants will take no notice whatever of a piece of white paper about three inches square passed in front of their eyes at a distance of about three inches. This is noteworthy in connexion with the examination of infants for supposed blindness or mental deficiency. I have sometimes seen the sight tested by threatening the eye suddenly with the finger-tip, but up to the age of six months many perfectly healthy infants take no notice whatever of this movement, and the sudden closure of the eyes, which is expected to result from it, was very inconstant even at the age of eight months. A bright metal object or a moderately bright light is more reliable as a test for the sight ; often the feeding-bottle is still better, for the infant's eagerness on seeing it leaves no doubt as to the sight.

Hearing is usually stated to be absent during the first two or three days, but I doubt if this is so. An infant, aged ten hours, showed no sign whatever of hearing the loud noise of beating a tin tray ; but another, aged twenty hours, showed some facial contraction suggesting that a sudden shrill whistle was heard, and more clear evidence of hearing was obtained on the second day. By the age of four weeks the infant appreciates sound sufficiently to be easily disturbed by noise. At four months old there should be no difficulty in attracting the attention by noises.

The question of deafness is one which often arises in cases of delayed development of speech. Parents' statements as to their child's hearing are apt to be misleading : a mother will speak to her child who is sitting on her lap, to try whether he hears : she forgets that the vibration of her chest and even her breath when she speaks are readily felt by the child, and concludes that the attraction of the child's attention is evidence of hearing : or, again, in asking the child some simple question, such as ' Where is Daddy ? ', she unconsciously attracts the child's attention by some movement of her face or eyes, or even of her hands. It is very necessary to eliminate such influences in examining a young child for deafness, the sound used should be made behind the child or out of his sight, and at such a distance that the influence of tactile sensation can be eliminated : it is well also to engage the child's interest with some toy or other object so that the child is not likely to look round in the direction of the noise by mere coincidence. A familiar voice, especially the mother's, calling the child from an adjoining room, is often a satisfactory test for a child's hearing.

Mental deficiency may simulate deafness ; the mentally defective child is often curiously stolid, taking no notice whatever of calls or commands, and yet perhaps a few minutes later showing that he hears readily when he chooses to do so.

I have occasionally met with a curious condition in which the child's behaviour was very suggestive of complete deafness, although the history showed that the child was neither deaf nor imbecile. In these cases the child, when addressed by a stranger, remained absolutely blank so far as any sign of hearing or interest went, so that, were it not for the history that when he was with members of his own family or familiar friends he both heard and talked, one might easily have supposed the child to be deaf and dumb. Such children usually show other evidence of mental peculiarity, either in backwardness of speech development, or in unnatural violence of temper, or in some other trait which marks them as ' odd ' children.

There are cases also in which deafness is simulated by a failure of interpretation of speech, although it is heard apparently perfectly: these are the cases of so-called congenital word-deafness: the child can hear and can be taught to repeat words, but the words convey no meaning to him; such children tested by speaking to them appear absolutely deaf, moreover speech is absent until special training has been given, so that the child might easily pass for a deaf-mute, were it not clear on testing him in other ways that even slight sounds are well heard.

The age at which a child sits, stands, and walks is a point of some importance in the recognition of imbecility, and also in relation to physical disorders such as rickets.

The normal infant during the first three months of life when supported in the sitting position is unable to hold his head up steadily, it tends to loll in one direction or another. At the age of four months many infants can support the head firmly in the erect position; if this cannot be done at six months, some morbid condition is present, either imbecility or physical disease.

During the first few months an infant makes no attempt to reach with his hand objects shown to him; at three to four months old some effort is made to reach objects, but the co-ordination is very defective so that the efforts are at first quite unsuccessful, but gradually the hand becomes steadier, and by the age of four months a healthy infant can often reach accurately for what it wants, failure to do this at six months of age should suggest some mental defect.

At the age of nine months most healthy infants can sit up without support: inability to sit up alone at the age of twelve months in an infant otherwise in good general health usually indicates mental deficiency. At ten months old many infants can just stand with slight assistance, at twelve months walking is possible with such slight support as having one hand held; if standing is delayed after fifteen months there is probably some disease physical or mental.

The development of speech will be considered more fully in connexion with speech disorders (Chapter LI); here it may be said that about the age of six months the infant begins to associate names with persons and things, so that the child will look towards the object named; at ten months many infants use one or two single words such as 'man' or 'Dad', and at fourteen months several single words, such as 'up', 'go,' 'puss,' &c., should be used; and simple sentences such as 'go ta-ta', 'dear dolly,' at eighteen months.

CHAPTER II

BREAST-FEEDING AND ITS LIMITATIONS

No one, I suppose, will deny that breast-feeding has its limitations and even its failures, and it is with these rather than with its wonted uneventful success that the medical man is concerned. But lest I should seem to pass over the value of breast-feeding too lightly, I will insist here upon its enormous importance to the welfare of an infant. Hand-feeding, even with the utmost care, involves risks which are reduced to a minimum by breast-feeding. The Registrar-General's reports show that the chief causes of death in the first six months of life are diarrhœa in its various forms and wasting conditions comprised under the heading 'Debility, Atrophy, Inanition'. Some statistics which I collected at the Hospital for Sick Children, Great Ormond Street, showed that 96 per cent. of the deaths from infantile diarrhœa occurred in infants who were being hand-fed entirely or partially (92 per cent. entirely): and the severe degrees of marasmus or wasting in infancy which end fatally are seen almost exclusively in hand-fed children. But apart from risk to life there are many disorders which beset the hand-fed child far more often than the baby at the breast: the sleepless infant who screams with flatulence and colic is nearly always the hand-fed infant; the infant who fails to gain weight and drives doctor and parents to their wits' end in the effort to find some food that will suit, is the hand-fed infant; rickets is almost entirely a disease of hand-fed infants: convulsions are also, I think, much commoner in these infants than in those who are suckled. Taking haphazard from my notes thirty cases of convulsions in infants under nine months of age, I find that twenty were entirely hand-fed at the time of the convulsion, only eight were entirely breast-fed. Some figures reported by Dr. Sykes illustrate well how much an infant's chance of life is diminished by hand-feeding; in the Borough of St. Pancras, London, in 1905, it was found that in a mortality of 127 per 1,000 during infancy, 102·6 were hand-fed entirely or partially (92·4 per cent. entirely), while only 24·4 were infants who had been entirely breast-fed.

Let us beware then how we counsel a mother not to suckle her child; it is a heavy responsibility and one which I ven-

ture to think is often taken too lightly. There are reasons enough which may make it advisable to stop breast-feeding, but these must be weighed carefully before such a step is taken.

How often one hears the statement, ' I never could suckle my children ' put forward as a reason for not attempting to suckle a subsequent infant. Undoubtedly there are many women who cannot suckle any of their children, but it would be well if medical men would impress upon mothers that failure in a previous effort to breast-feed is no reason whatever for assuming that there will necessarily be failure also in suckling a later child ; a mother whose milk has failed either in quantity or in quality in the feeding of earlier children, may yet suckle, and suckle successfully for several months, a subsequent infant.

And here I would emphasize particularly the importance of breast-feeding during the first few weeks of life. It is in these early days when an infant has so feeble a hold on life that any disorder of digestion, such as is apt to occur with hand-feeding, is specially dangerous, and it is at this time that the digestion is most easily disturbed. Breast-feeding at this time, even if it can be continued only for six weeks or two months, seems to give the infant a start in life, which may be of vital importance when the difficulties and dangers of hand-feeding have to be faced.

Examination of breast-milk. Breast-milk is often judged to be unsuitable on quite insufficient grounds. Perchance the baby has cried and seemed uncomfortable after the breast-feeds ; or he does not seem satisfied. The weight is rising, but perhaps not very regularly, 7 ounces one week and only 2 or 3 ounces the next week, and the over-anxious mother has appealed to the doctor. Now what happens ? The doctor very rightly says that he would like to see the milk and tells the mother to draw some off for him to examine. The mother, having no special directions given to her, naturally draws off some milk at the time when her breast is most full, just before she feeds her infant : and at his next visit the doctor sees a poor watery-looking specimen of milk, which he unhesitatingly condemns as unfit for the infant. Now this is no imaginary occurrence ; it has happened again and again in my own experience ; and it involves a serious mistake. The milk which is drawn first from the breast, the ' foremilk ' as it is sometimes called, is normally much thinner and poorer than the later milk ; there is a gradual increase in the richness of the milk from the beginning of a feed until the end ; the last milk is much richer than the foremilk. The fact, therefore, that a woman's milk, drawn off with no special

precautions, looks thin and watery proves nothing whatever as to the suitability or unsuitability of her milk. The only way in which a reliable opinion can be formed as to the quality of breast-milk is by examination either of a mixed sample of the whole contents of the breast, or by examination of the middle third. The former, of course, would be the more accurate, but any one who has attempted to pump off the contents of a breast will know that this is often very difficult and sometimes impossible; so that in practice it is more convenient to take the middle third, which perhaps owing to the stimulus of sucking, where the infant has first been allowed to suck a small quantity, is much more easy to obtain. The directions to be given are these : the mother is first to time several feeds and so ascertain how long the infant usually takes to empty the breast : then the infant is to be put to the breast for exactly one-third of this time, and immediately afterwards about 1 ounce of milk is to be withdrawn (with breast-pump if possible, otherwise by gently squeezing) for examination ; the infant can then take the milk that remains in the breast. In this way a sample is obtained which shows approximately the average quality of the milk. But even so it is seldom possible by mere inspection to form any reliable judgement as to the suitability of breast-milk for an infant ; occasionally, but, I think, very rarely, the middle milk will be found to be so thin that its unsuitability may be reasonably inferred; far more often it is only by analysis that an opinion of any value can be formed.

Composition of breast-milk. This brings me to the composition of breast-milk, and let me say at once that for a scientific understanding of the everyday difficulties of infant feeding, an accurate knowledge of the percentage composition of the common foods, particularly human milk and cow's milk, is absolutely essential ; no mortal man can carry in his memory the percentage composition of every food that is used for infants, but every medical man ought to know the average proportions of the various constituents of human milk and cow's milk ; for upon the composition of these two milks turn most of the practical problems of infant-feeding.

Human milk has an average specific gravity of 1030–1032 and is composed thus :

Proteid . . .	2·0 per cent.	Casein 0·6 per cent. Lactalbumen 1·4 per cent.
Fat	3·5 per cent.	
Lactose . . .	7·0 per cent.	
Salts . . .	0·2 per cent.	
Water . . .	87·3 per cent.	

The striking feature in its composition is the low proportion of curd-forming proteid, casein, as compared with the soluble and much more easily digested proteid lactalbumen, which, unlike the casein, is not precipitated by acids or by rennet. The constituents which vary most in quantity are the fat and the proteid, especially the fat ; the sugar is much more constant.

The following observations will show how fallacious examination of the milk may be when a chance specimen is taken without special precaution to obtain the middle third : in one case the foremilk showed only 0·4 per cent. of fat, while the middle-milk showed 3·2 per cent. ; in one specimen of foremilk there was 1·4 per cent. of fat, in one of after-milk 10 per cent. of fat.

Another fallacy I must mention : it might be concluded hastily that the specific gravity would afford some guidance, but this is not so, unless either the proportion of fat or of proteid in the milk is known. If the sugar and salts may be assumed, as they fairly may, to be constant in their proportions, then the variation of specific gravity will depend on the proteids and the fat. The effect of a high percentage of fat is to lower the specific gravity : the effect of a high proportion of proteid is to raise the specific gravity. Therefore, if the proportion of neither is known, a high specific gravity might be due either to a low fat percentage or to a high proteid percentage : and conversely a low specific gravity might be due to a high fat percentage or to a low proteid percentage. But if the percentage of one is known, then we can form a reliable opinion as to whether the percentage of the other is above or below the normal. It is usual to ascertain the fat percentage as this is more easily determined than that of the proteid.

If it is known that the fat percentage is unduly low, then a low specific gravity must mean that the proteids also are low, while if the fat percentage is above the average then a normal specific gravity must mean that the proteid percentage is higher than normal. The estimation of fat with a high-speed centrifuge is a very simple matter. But if no centrifuge is at hand it can be done very fairly accurately by the following method. A cylindrical five cubic centimetre measure, having a diameter of three-eighths of an inch, and graduated in cubic centimetres and millimetres, is filled with the breast-milk exactly up to the five cubic centimetre mark, it is then corked and allowed to stand for exactly twenty-four hours. The cream rises to the top and is read off in cubic millimetres. Empirically the cream readings

are found to correspond with fat-percentages approximately thus :

Cream.		*Fat.*
2 cubic millimetres	=	2·8 per cent.
3 cubic millimetres	=	3·5 per cent.
4 cubic millimetres	=	4·4 per cent.
5 cubic millimetres	=	5·6 per cent.

For taking the specific gravity if only a small quantity of milk can be obtained, so that even a small urinometer is not available, I use specially made glass beads which any scientific glass-blower will make for the purpose, each bead being made so as just to float in fluids of a specific gravity which is marked on the bead.

Nowadays there are many laboratories where a complete analysis of breast-milk can be obtained; and when this is practicable it is well worth while to have such an analysis done ; but it is to be remembered that it is useless sending a sample for analysis unless it be either the middle portion of the milk, or the entire contents of a full breast. In this way information may sometimes be obtained which may be of great practical value, for to some extent it is possible to modify the composition of breast-milk ; and I would insist upon the duty of attempting to overcome any fault that is detected in the breast-milk before concluding that it is necessary to wean the child. Even if it is necessary to wean the child, the information gained from an analysis of the breast-milk may still be of value in showing what particular modification or dilution of cow's milk is likely to suit the infant.

Having said thus much of the value of analysis in cases of difficulty in digestion of breast-milk, I must add that it has been my own experience that only in a minority of cases does analysis, however full and accurate, give any practical help. I have analysed breast-milk in many cases where an infant was doing badly, and sometimes where the infant was wasting considerably in spite of an ample supply of milk, and I have often found that, so far as ordinary analysis could show, the milk was perfectly good : nevertheless, the substitution of hand-feeding partly or entirely has been followed by immediate improvement. I have no explanation to offer for this occurrence ; but it suggests that there are subtler constituents in milk than proteid, fat, and sugar. For instance, it is known that there are several ferments normally present in milk, and who shall say but that these may vary in quality or quantity and affect its digestibility ? and again, if the addition of one to two grains of sodium citrate to a three-ounce feed can, as it certainly does, affect the digestibility of the

milk, it seems possible that variations in the salts of the milk may be more important than we know. However this may be, it is noteworthy that an exact analysis of the three chief constituents of the milk may not throw any light upon the infant's failure to thrive.

Faults in breast-milk. I suppose the commonest complaint is, ' My milk does not satisfy the baby ' ; a state of things which may depend either upon deficiency in quantity or deficiency in quality of the milk. In the former case the infant cries angrily after he has taken the breast for a few minutes, and then perhaps goes to sleep and wakes again crying for the breast some time before it is due : in the latter where the milk is thin and watery, there may be an abundant flow of milk and the infant may seem satisfied and fall asleep for a short time after a feed, but then begins to cry with flatulence and discomfort. What is to be done ? certainly not to wean the child off-hand. If the milk is deficient in quantity it may be possible to increase it. The effect of rest in promoting the flow of milk is often very striking, many mothers have noticed it themselves. A day or two spent chiefly on the sofa or in bed may restore the flow, and a little care in avoiding all unnecessary exertion may enable a mother to continue the suckling. Then in the way of diet something can be done : more milk, or gruel, or perhaps a glass of stout with the mid-day meal may promote the flow of milk. But here I must insert a caution : whilst I do not think there is the least doubt that a small amount of alcohol in this form sometimes makes suckling possible where it would otherwise have to be stopped, I think it is a measure never to be advised unless other aids have failed. Apart from the possibility of inducing in the mother the alcohol habit, it seems that even a small amount of alcohol taken by some women produces some change in their milk whereby it disagrees with the infant. A more useful means in some cases is the administration of malt extract to the mother three times a day after her meals; this may work only indirectly by improving her digestion, but, however this may be, it seems to improve not only the flow of the milk but also its quality in some cases. Recently it has been found that powdered Cotton Seed extract, which can be obtained under the trade name ' Lactagol ', is often very effective in improving both the quantity and the quality of the breast milk. A teaspoonful of this preparation is given three times daily mixed with milk or cocoa.

If the milk is thin and watery but abundant, a liberal diet of proteid food, chops, steaks, joints, fish, poultry, eggs, is to be advised ; it is a curious fact that a diet rich in proteid seems to increase not only the proteid in the milk but also the fat.

But supposing that with all our efforts we fail to improve the milk in quantity or quality, there may still be no justification for weaning. Some women can continue suckling if the strain is reduced by substituting hand-feeding two or three times daily, other women have milk enough to suckle their infant at night when they are resting in bed although they are unable to provide enough for the day also : not only is the flow of milk usually more copious at night but its quality is richer owing, no doubt, to the rest, so that even if the failure to satisfy be due to deficiency in quality, it may still be possible to suckle at night.

Here, however, a difficulty may arise which may make it necessary to wean the child. Breast-milk which is scanty often seems to act as an irritant, sometimes making the infant sick, often making him cry with colic, and occasionally starting a greenness and looseness of the stools. Moreover, it is remarkable how an infant seems to detect some change in the milk when it is diminishing in quantity ; sometimes before the mother or the doctor is aware that the supply is failing, the infant will take a dislike to the breast-milk, and may refuse it altogether.

And this brings me to another common complaint from mothers : ' My milk does not agree with the baby.' Now as I have already mentioned incidentally, a scanty milk is apt to disagree, and there are, as I shall show, other causes for the disagreement ; but before assuming that there is any fault in the milk, it is very necessary to make sure that the fault is not rather in the feeding ; a mother's milk may be perfectly sound, but if the baby is fed haphazard—' when he wants it,' or ' when he cries '—the result is likely to be unsatisfactory. There must be regularity in the feeding and, as a general rule, I am strongly in favour of waking an infant for its feeds at the proper time. If this is done from the beginning, the infant usually very soon gets into the habit of waking just as the feed is due, and goes off to sleep again quite easily after it.

An infant should be fed every two hours during the first two months, except at night, when the intervals may be three hours : during the next month it should be fed every two and a half hours by day, and three hours by night, and from the end of the third month onwards it should be fed every three hours by day, and may miss one feed at night, until the age of six months, when it may miss two feeds at night.

Where the mother's milk is very profuse, trouble arises sometimes from the very ease with which the milk is sucked ; the infant gulps the milk down in haste and repents at leisure, screaming with flatulence and colic. This is difficult to prevent. To some extent the mother may regulate the flow by compressing

the nipple between her fingers, and it may assist if the baby is taken from the breast at short intervals during the feed ; or the difficulty may be overcome by using a nipple-shield with a teat, which will not allow too easy a flow. I have often thought that digestive disturbances in breast-fed babies resulted from excessive feeds. Certainly, if a mother has an average quantity of milk, one breast should be sufficient for an infant, and trouble is likely to result from allowing the baby to empty both breasts at a feed : but even with the milk of one breast only, I think that some infants take too much and scream in consequence with wind and discomfort. Undoubtedly an infant can tolerate and digest a larger bulk of breast-milk than of cow's milk or of any artificial food, but if one may judge from the analogy of hand-feeding, I think it is quite clear that too large a bulk of food, quite apart from its quality, is a cause of much gastric disturbance in infancy.

Apart from these causes, however, there are cases—and by no means infrequent—in which the milk obviously disagrees ; the stools are green, sometimes of a bright grass-green colour throughout, sometimes of a dark olive-green colour ; slimy, perhaps, but not too frequent ; the infant is evidently uncomfortable after the feeds ; the weight is rising, but erratically. In other cases there is frequency of the stools, which are green and watery; I have known as many as twelve and thirteen stools in the twenty-four hours ; the buttocks become red and excoriated, the weight ceases to rise or actually falls, the infant takes the feeds well, but seems uncomfortable, passes wind often by mouth and bowel, and clearly is not thriving. What is the meaning of all this ? Analysis of the milk explains it in some cases, but not in all ; I have found an abnormally high proteid percentage alone as the apparent explanation of green stools where there was no diarrhœa. For instance, a female infant aged three weeks was brought to me for screaming and some vomiting after feed ; analysis of the breast-milk (middle third) showed fat 3·0 per cent., proteid 2·5 per cent., sugar 6·0 per cent. Where, as more often happens, the stools are not only green, but very frequent and loose, so that there is actual diarrhœa, I have several times found that the fat was in excess, more rarely the proteid also. For instance, in one such case the middle-milk showed fat 6·5 per cent., proteid 1·7 per cent., sugar 7·0 per cent. ; in another, fat 4·4 per cent., proteid 1·8 per cent., sugar (approximately) 6·5 per cent.

But I am not at all sure that in every case where in spite of properly managed breast-feeding the milk continues to disagree,

the trouble is due to abnormal character of the milk; I suspect that in some of these cases the explanation is better expressed as a foreign mother put it, when she held out her wasted baby to me and said: 'De baby se not agree wid de milk.' In one instance I had three wet-nurses in succession for an infant shortly after birth; with each and all the result was the same, frequent loose green watery stools; whereas directly a simple whey mixture or peptonized cow's milk was used, in the intervals while a fresh wet-nurse was being obtained, the stools at once improved, so that ultimately the attempt to feed on the breast was abandoned and the infant did perfectly well with hand-feeding.

There is no doubt that some infants are remarkably intolerant of fat, as others are of carbo-hydrate, and it may be that for such the proportion of fat or sugar normally present in mothers' milk is too high. If, however, it is found that the milk is abnormally rich, this fault can be overcome to a certain degree. As proteid is increased by shortening the intervals between feeding, so an undue proportion of proteid may be reduced by lengthening the intervals between feeds, if feeding has been too frequent.

The effect of exercise is also to be remembered, both the proteid and the fat may be reduced by increasing the amount of exercise. In some of these cases I think that too much is being done in the way of stuffing the mother with meat and milk. In one instance where a suckling woman was having two quarts of milk a day in addition to liberal feeding with beefsteaks and mutton-chops, the breast-milk which disagreed with the infant contained 3 per cent. of proteid; a reduction of the diet reduced the percentage of proteid within three days to 1·7 per cent. But if the milk still remains excessively rich, there are three ways in which the difficulty may sometimes be surmounted; the infant may be allowed to suck for about 4–5 minutes at both breasts, so that he gets only the weaker foremilk, instead of taking the whole contents of one breast at each feed. If this method is used the infant must, of course, be strictly timed, otherwise he will take too much; another procedure is to give ½–1 ounce of plain water to which a quarter of a teaspoonful of milk sugar may be added immediately before each breast-feed, in the hope that the breast-milk may be diluted in the infant's stomach; the third is to give some drug which may aid digestion. Sodium citrate may be given thus: Sodii Citratis gr. ij. Aq. Anethi ad ℥j, immediately before the infant is put to the breast. Sodium Bicarb. gr. ij, Papain gr. j with 2 drachms of water just before each feed certainly helps in some cases.

A possibility always to be remembered where the breast-milk appears to be disagreeing with an infant is that the mother may be taking some drug which is either excreted in the milk or affects the milk indirectly; all the saline aperients are excreted to some extent in the milk, as also potassium iodide, mercury, sodium salicylate, arsenic, and antimony (and even some of the vegetable aperients in common use, senna, and castor oil), and possibly the alkaloids of opium.

It would be interesting to know whether any of the hypnotics in common use, especially the newer ones, have any deleterious influence on the milk, for they are used not uncommonly for mothers soon after delivery. One thing is quite clear, that the less drugs a nursing woman takes, the safer for the infant.

Wet-nursing

I cannot leave the subject of breast-feeding without some reference to wet-nursing, which in my opinion might with advantage be used much more often than it is in this country. There is nothing more striking in the whole range of therapeutics than the effect of wet-nursing upon an infant who is steadily emaciating upon this and that modification of cow's milk or other artificial food ; often from the day the breast-milk is begun there is a rapid gain of weight, and the infant becomes contented and restful instead of whining and miserable. Unfortunately, wet-nursing is beset with difficulties ; in the first place, it is often very difficult to secure a wet-nurse ; in the second place, when a woman is found who is willing to suckle, she may be unsuitable for various reasons ; in the third place, from one cause or another, her milk may not suit the infant ; and last, but not least, the infant may absolutely refuse to take the breast.

My own experience leads me to think that it matters little whether the wet-nurse's infant is of the same age as the foster-child, even approximately ; certainly it does not matter in the least what may be the complexion of the wet-nurse, or her character, so far as the milk is concerned ; although the latter consideration may be a weighty one, when an unmarried woman is to be introduced into a household as wet-nurse.

The first point to be determined is the health of the wet-nurse, and in this matter the utmost care is needful. In one instance under my own observation, a wet-nurse who had undergone medical examination, was approved as healthy and suckled an infant for some months before it became evident that the

woman was very ill, she died with acute miliary tuberculosis after a few weeks' illness; it transpired that she had had hæmoptysis before she was engaged as wet-nurse, and diarrhœa on and off most of the time she was suckling, but she had apparently deliberately concealed her illness. I saw the infant for a year or more after the suckling was discontinued, but fortunately it did not show any sign of tuberculosis. In another instance, after much trouble, I had secured as I thought a suitable wet-nurse, when it was found that her head was swarming with pediculi. In a third case an apparently healthy woman whom I was about to engage brought her infant to be examined; it looked so clean and healthy in the face that I was almost tempted to dispense with a complete examination, but on undressing it I found a very characteristic syphilitic rash over the buttocks and thighs. This last possibility, syphilis in the mother, is the most difficult to exclude by ordinary clinical examination; as is well known, it is common for the mother to show no signs whatever of this disease, although her infant shows indubitable evidence; it may be doubtful whether in such cases a woman is liable to transmit the disease to a foster-child, but certainly it is not justifiable to run the risk; moreover, if her infant be syphilitic the mother may at any time develop manifestations of syphilis, for instance condylomata, from which infection might be conveyed to a foster-child; for these reasons it is important to exclude as far as possible syphilis in her offspring as well as in the wet-nurse herself. Now as I have shown elsewhere, the first manifestations of syphilis in an infant may be delayed at least as late as the third month, and occasionally they are delayed much longer; clearly every week of freedom from evidence of syphilis in the infant adds to the security against syphilis in the wet-nurse, and, therefore, if the milk is equally suitable in quality when her own infant is two or three months old, it is better to engage a wet-nurse whose infant has reached that age, than to engage one whose infant is only two or three weeks old.

In view of the evidence which has accumulated now as to the value of the Wassermann test, this should certainly be done as part of the examination of the wet-nurse if there is the slightest ground for supposing that syphilis may be present. There are those indeed who hold that it should be done in every case before a wet-nurse is engaged, but often it is impracticable, and in many cases, for instance where the wet-nurse is a married woman with several healthy children, it is unnecessary.

It is usually taught that much attention should be paid to the

appearance of the woman's breasts, their size and fullness, and to her own healthy appearance; as a matter of experience I think these are most fallacious guides; it is of much more importance to know how her milk has suited her own infant. I have seen fine healthy-looking girls with excellent breasts fail completely as wet-nurses, whilst a sickly, pale girl, with flabby empty-looking breasts, has nursed an infant for many months with excellent result. But even the effect of her milk on her own child is not an absolutely reliable guide, for it does not always follow that one who has successfully nursed her own infant can equally successfully nurse another infant; nor indeed does it follow that if she has failed with one infant she will fail with another. It has been shown that breast-milk which disagrees entirely with one infant may suit another admirably, and I have seen the converse, a milk which had suited one infant well for several weeks producing green and loose stools in another infant. In fine, it comes to this, a wet-nurse is always an experiment.

I am much inclined, in the rare cases where it is practicable, to have a wet-nurse's own infant with her; for I have so often seen a good wet-nurse become homesick, or fretting as to her own infant who is away from her, with the result that her milk has either begun to disagree with the foster-child, or has fallen off in quantity. There is a great tendency to treat these women, especially if they be unmarried, as mere machines for the supply of milk; no sooner is the baby satisfied than he is taken away from the wet-nurse and she is hustled out of the room, perchance to mope or to idle away her time in some distant part of the house, until the next feed is due. This is bad from every point of view; a wet-nurse, even though she be unmarried—possibly even more on that account—needs kindness and consideration. It may be that if she were allowed to do more for the infant than merely feed it, the satisfaction of her maternal instinct would go far to keep her milk in good condition; employment of some kind must be found for a wet-nurse if her milk is to continue good, and I know of no better way of employing her than in looking after the needs of the infant beyond its meals. Of course, strict supervision is necessary, but if those under whom the wet-nurse is placed, the domestic nurse and the trained nurse, are tactful persons, this can be done without making the wet-nurse feel, as is too often the case, that she is a prisoner watched night and day and never trusted to handle the infant except at its meals. If there is accommodation for the wet-nurse's own child, it

must, of course, in most cases be fed by hand entirely, and supervision will be necessary to prevent surreptitious suckling of the infant; but it will sometimes be good policy to allow breast-feeding of this infant once or twice a day, for, compared with the small wants of the wasted feeble foster-child, not only may its stronger sucking and large appetite encourage the flow of milk, but the mental satisfaction of the wet-nurse may be well purchased at the price of depriving the foster-child of one or two breast-feeds daily, and substituting hand-feeds therefor. In any case there must be no suckling of the wet-nurse's offspring in the two- or three-hourly intervals between the breast-feeding of the foster-child, for this would entail shortening of the intervals between suckling and so probably alter the composition of the milk by increasing the curd: any suckling of the wet-nurse's child must take place at the time when a feed would have been due for the foster-child.

A wet-nurse usually requires supervision outdoors even more than indoors; some trustworthy person should accompany her whenever she goes out; otherwise if her child or her home be near enough, she will certainly go thither and perchance bring back infection or livestock, or she may obtain drink or eatables which will disturb the character of her milk. Lastly, it is very necessary to make quite sure that the wet-nurse's bowels act regularly; an aversion to taking aperients leads a wet-nurse sometimes to conceal the fact that she is constipated, and for this reason it may be necessary even to arrange for inspection of the daily evacuation.

With all these difficulties it is hardly to be wondered at that the use of wet-nurses is generally recognized as an extreme resource; no one can be more cognizant of these obstacles than I am, but, none the less, I have seen so many wasting infants saved from what appeared to be certain death by the use of a wet-nurse, that I feel sure it is a resource which should always be recommended if necessary.

Lastly, it is worth remembering that if a wet-nurse cannot be obtained there may be some neighbouring mother who cannot leave her house, but has an abundant supply of milk from which she would be willing to spare some, if it were drawn off once or twice a day to be given by bottle to the wasting infant. Even a couple of feeds of breast-milk daily for a week or two may be of vital importance to such an infant. Valuable help has even been obtained by making a collection daily of small quantities of breast-milk from several mothers, and so obtaining enough for a feeble infant.

CHAPTER III

THE MODIFICATION OF COW'S MILK FOR INFANT-FEEDING

I SUPPOSE that most of us when we first entered upon the practice of our profession cherished a faith that, failing the mother's milk, the only proper food for an infant was cow's milk, and that it was the simplest thing in the world to feed an infant successfully if only this golden rule were remembered. A very little experience, alas ! and any such belief in the simplicity of infant-feeding has been rudely shaken ; often, indeed, if cow's milk is ordered at all, it is ordered with fear and trembling, and on the first sign of its disagreeing, recourse is had to some patent food or condensed milk.

Far be it from me to pass any sweeping condemnation on such methods of feeding ; it is the abuse, not the use of these foods and condensed milks which is so disastrous ; used with an intelligent appreciation of their composition, and of the ages and conditions for which they are suitable, they undoubtedly have their value. But none the less it is true as a general statement that there is not a single patent food or condensed milk in the market which can adequately replace fresh cow's milk in the feeding of an infant who is deprived of the mother's milk ; and, moreover, that a vast amount of infantile suffering and misery, and no small sacrifice of infant life, are due solely to the indiscriminate feeding of infants with these artificial preparations.

Even when the unsatisfactory result of such resources is evident in gastro-intestinal trouble of one kind or another, or in failure to gain weight as the infant should, any proposal to give cow's milk is commonly met with the objection, ' It is no use ordering cow's milk ; we have tried that, and the baby can't take it.' In such cases the question always arises in my mind, ' Has sufficient care been taken in the modification of the milk ? ' Too often one finds that a mixture has been used which differs so widely from human milk that it is little wonder if the infant fails to thrive upon it. Judging from my own experience, I am inclined to think that there is a certain number of infants who

cannot take cow's milk, modify it as you will; but I am convinced that this number is *extremely* small, and that the vast majority of infants will thrive excellently on cow's milk, provided this has been properly modified.

The first and absolute essential for the intelligent modification of cow's milk for infant-feeding is a knowledge—I mean a practical working knowledge—of the exact average composition of human milk and cow's milk. Undoubtedly mere rule of thumb will serve for the feeding of many a vigorous infant; but if our sole idea of modification of milk is to dilute it once, twice, or thrice, and throw in 'a little sugar' and, perchance, 'some cream,' trusting to luck or to the mother, or, worse still, to the monthly or domestic nurse, to adjust the proportions, we shall certainly have many failures in our efforts to feed even healthy infants, and many more in the case of weakly infants or those whose digestion has already been impaired by disease or unsuitable diet. It is especially these more difficult cases—and are they not common enough in every one's practice ?—in which accurate and intelligent modification of cow's milk is all-important.

Now, as in this chapter I shall have to refer again and again to the percentage composition of milk and of various modifications of milk, let me say at the outset that such percentages are only a convenient method of calculating approximately the composition of the food; and for clinical purposes a strictly exact percentage of any one constituent is neither necessary nor practicable. We have heard a great deal recently about 'percentage-feeding', as if it were some great discovery. There is nothing new in 'percentage-feeding'; like Monsieur Jourdain with his 'prose', we have used it all our medical lives, whether we knew it or not; the only innovation—and a very important one—is that the medical man should *know* approximately what percentage he is giving. It makes no difference whatever, whether the medical man prescribes equal parts of milk and water, or prefers to order casein, 1·625 per cent.; lactalbumen, 0·375 per cent.; fat, 1·75 per cent., &c. (assuming the average composition of cow's milk to be as stated below); but it does make a very great difference whether he realizes that the difficulty in digesting cow's milk lies in the large proportion of casein [1] which it contains, and whether he understands how to get over this difficulty without reducing

[1] The terms casein and caseinogen, strictly used, refer respectively to the formed curd and the potential curd, i.e. the curd-forming proteid before it is coagulated; for brevity' sake the term casein has been used sometimes where caseinogen would have been more strictly accurate.

the other important constituents of milk to a harmful degree, or falling into the not less disastrous mistake of making the food too rich in cream or sugar.

Below is shown the average composition of human milk and cow's milk. For convenience' sake I shall leave the salts out of consideration; they undoubtedly have their importance, but we know so little about them, and they probably play such a comparatively insignificant part in nutrition, that it is hardly worth while to burden our memories with their percentage. The proportion of water can also be left unmentioned; it forms, of course, the residue of the percentage.

Human milk.		*Cow's milk.*	
Proteid, 2 per cent.	Casein, 0·6 per cent. Lactalbumen, 1·4 per cent.	Proteid, 4 per cent.	Casein, 3·25 per cent. Lactalbumen, 0·75 per cent.
Fat, 3·5 per cent.		Fat, 3·5 per cent.	
Sugar, 7 per cent.		Sugar, 4 per cent.	

Proteids. The one great obstacle to the digestion of cow's milk is the large quantity of curd which is formed when it enters the stomach, and this is due to the heavy percentage of caseinogen which it contains. This caseinogen forms a curd when mixed either with acid or with rennet, whereas the remaining proteid, lactalbumen, is only coagulated by heating above 160° F.

Lactalbumen is so easy of digestion that so far as this portion of the proteid is concerned no dilution of cow's milk is necessary; the only object of dilution is the reduction of the casein to a proportion which the infant can digest.

If human milk is to be taken as the measure of a healthy infant's power of digestion, it is clear that in order to reduce the proportion of casein in cow's milk to this standard, considerable dilution is necessary. Fortunately nature has provided even the youngest infant with some range of accommodation in its powers of digestion, and many an infant will digest a larger proportion of casein in cow's milk than the average composition of mother's milk might suggest.

But, notwithstanding this power of accommodation, I think I shall be correct in saying that a large majority of the failures in infant-feeding with cow's milk are due to insufficient dilution.

For an infant two or three weeks old it is a common practice to order a mixture of one part of milk with two parts of water—that is, 1 in 3. This, of course, means division of each percentage

by 3; and a comparison with human milk shows how widely such a mixture differs therefrom.

Human milk.	*Cow's milk and water*, 1 *in* 3.
Proteid, 2 per cent. { Casein, 0·6 per cent. Lactalbumen, 1·4 per cent	Proteid, 1·3 per cent. { Casein, 1·08 per cent. Lactalbumen, 0·25 per cent.
Fat, 3·5 per cent.	Fat, 1·16 per cent.
Sugar, 7 per cent.	Sugar, 1·3 per cent.

The casein is still nearly double the proportion which occurs in human milk. (I am not at present considering the other constituents; my object now is only to show how far the ordinary dilution fails in its sole purpose—namely, the reduction of the casein, the curd proteid.)

Even if three parts of water are used with one of milk, there is still a slight excess of casein, as can be seen from the following comparison (the percentages will, of course, be divided by 4):

Human milk.	*Cow's milk and water*, 1 *in* 4.
Proteid, 2 per cent. { Casein, 0·6 per cent. Lactalbumen, 1·4 per cent.	Proteid, 1 per cent. { Casein, 0·81 per cent. Lactalbumen, 0·18 per cent.
Fat, 3·5 per cent.	Fat, 0·87 per cent.
Sugar, 7 per cent.	Sugar, 1 per cent.

It is evident that in order to reduce the curd-forming proteid to the same proportion as is present in the mother's milk, four parts of diluent must be added to one part of milk (1 in 5). The percentages will thus be divided by 5, and the result will be as shown here:

Human milk.	*Cow's milk and water*, 1 *in* 5.
Proteid, 2 per cent. { Casein, 0·6 per cent. Lactalbumen, 1·4 per cent.	Proteid, 0·8 per cent. { Casein, 0·65 per cent. Lactalbumen, 0·15 per cent.
Fat, 3·5 per cent.	Fat, 0·7 per cent.
Sugar, 7 per cent.	Sugar, 0·8 per cent.

But how often is milk diluted to this extent? Hardly ever, even during the first few weeks of the infant's life; and yet it is considered a matter of surprise—or at any rate an evidence of some peculiar weakness in the infant's digestion—that an infant suffers with colic or vomiting or other gastro-intestinal trouble, and fails to thrive when—say at four weeks old—it is having milk diluted with only two parts of water or barley-water. Surely

the wonder is that so many infants manage to digest their food in spite of the excess of curd which the usual dilution entails.

Now I do not for a moment wish to advocate any extreme dilution for the vigorous infant who can digest the larger proportion of curd—the more of this that the child can take and digest the better—but I do wish to insist very strongly on the necessity for more accurate modification of cow's milk, if only in respect of the faulty excess of curd, before rejecting this food in favour of any of the far less satisfactory substitutes with which the market is flooded.

In a very large number of cases where milk-feeding is tried and found wanting, the failure is due simply to insufficient dilution. Even at the age of four or five months some infants cannot digest a mixture of equal parts of cow's milk and water (casein, 1·6 per cent.); and when it is remembered that the proportion of curd in mother's milk scarcely increases at all during the later months of lactation, we might expect that even at such an age it may be necessary to dilute cow's milk, if not to so great an extent as in the earlier months, at any rate much further than is usually done.

So far I have considered only the curd-forming proteid in cow's milk, but obviously any such dilution as I have mentioned involves serious alteration in the proportion of the other constituents also; and no doubt it is this fact which accounts for the common practice of insufficient dilution.

The proportion of lactalbumen, which at the outset is little more than half that in human milk, becomes still more deficient if the ordinary diluents, such as water, barley-water, lime-water, &c., are used.

On this point I shall have more to say later; here I will only point out that whilst it is the proportion of casein, not the proportion of total proteids, which has to be corrected by dilution, it is, nevertheless, important that the resulting total of casein plus lactalbumen should not be far below that present in human milk. The right course in determining the degree of dilution in any case where there is difficulty of curd-indigestion, is to determine it solely with reference to the proportion of curd-forming proteid which the infant can digest, and then in one way or another to rectify as far as possible the deficiency of lactalbumen, fat, and sugar. It is the curd-forming part of the proteids—the casein alone—not the lactalbumen, which has to be considered in deciding what degree of dilution is necessary.

Having said thus much as to the theoretical grounds which determine the dilution of cow's milk, I hope I shall not appear inconsistent when I say that there is no need whatever to work out mathematical calculations or percentage problems in every case of infant-feeding that comes before us. Common experience has determined what dilution is sufficient to overcome the curd difficulty for a healthy infant with ordinarily good digestion, and as a matter of routine it is more convenient to remember these data of experience in simple terms of dilution than in any formulæ of percentage composition.

Only let the medical man carry in his mind the exact composition of milk so that at any time, when it may be necessary, he can calculate readily the exact composition of a particular mixture. The ability to do this sometimes means all the difference between success and failure in infant-feeding, especially in those difficult cases where a minute variation in the proportion of this or that constituent of the feed is sufficient to disturb digestion.

For the healthy infant, experience shows that, as a general rule, the following dilutions are successful :

Age.	*Dilution.*
3 months	Equal parts of milk and diluent.
6 months	Twice as much milk as diluent.
9 months	Three times as much milk as diluent.

The advance from one dilution to another is, of course, to be made gradually, the more gradually the better ; and it is always to be remembered that healthy infants vary in their requirements, so that a mixture which may be strong enough to nourish one infant perfectly, may be insufficient for another ; some infants will do well on equal parts of milk and water as early as eight weeks old, nay there are medical men who advocate for infants undiluted milk from a few weeks old, but, none the less, I think that the proportions mentioned above may be taken as good working standards, and that trouble arises far more often from diluting too little than from diluting too much.

The dilution given at the age of three months is reached by a gradual advance from birth thus :

Age.	*Milk.*	*Diluent.*
At birth . . .	1	4
One week . . .	1	3
One month . . .	1	2
Two months . . .	1	1½
Three months . .	1	1

As will be seen, the proportions mentioned above, if expressed in terms of percentage composition, would be as follows :

Age.	*Proteid.*	*Approximate Composition of Proteid.*
3 months	2·0 per cent.	Casein 1·6 Lactalbumen 0·4
6 months	2·6 per cent.	Casein 2·1 Lactalbumen 0·5
9 months	3·0 per cent.	Casein 2·4 Lactalbumen 0·6

It seems likely that the proportion of proteid in these mixtures is more than is absolutely necessary ; in human milk the total proteid is often below 2 per cent. at any period of lactation: and experience seems to show that, so long as the deficiency of fat and sugar in diluted cow's milk is corrected, a proportion of about 2 per cent. of proteid or very slightly above, is sufficient. But in this very correction lies a practical difficulty ; the sugar is easily enough arranged, but the fat cannot always be adjusted conveniently ; cream is not always easily obtained, and if it is, may be of doubtful quality, so that we are constrained sometimes, especially in dealing with the poor, to use mixtures in which nothing further is attempted than simple dilution and addition of sugar. Under these circumstances it is important that the dilution should be as little as is consistent with digestion, lest the deficiency of fat give trouble by inducing rickets or otherwise interfering with nutrition. As a matter of experience, I think it may be said that given a fairly good milk supply, dilution in the proportions mentioned above gives just a sufficiently high proportion of fat to avoid the occurrence of rickets.

There are, of course, other factors in the production of rickets besides deficiency of fat in the diet ; the presence of starch, especially of much starch, may prevent the assimilation of the fat that is present and so aggravate the evil of a low proportion of fat : but assuming that a milk mixture is given which contains no starch, the above proportions can safely be used without addition of cream. Any further dilution makes some compensatory addition of fat necessary, if the risk of rickets is to be avoided.

Cream and fat. The methods of correcting deficiency of fat in the food are worthy of careful study.

However little cow's milk is diluted, there is necessarily some deficiency of fat in the mixture ; compare, for instance, the following mixtures of cow's milk and water, with human milk :

Human milk.	*Cow's milk and water Equal parts.*	*Cow's milk, 2 parts; Water, 1 part.*
Proteid 2	Proteid 2	Proteid 2·6 per cent.
Fat 3·5	Fat 1·75	Fat 2·2 per cent.
Sugar 7·0	Sugar 2·0	Sugar 2·6 per cent.

Our aim should be to give a proportion of fat corresponding as closely as possible to that present in human milk, say 3–3·5 per cent. of fat.

If we had a standardized cream, it would be perfectly simple to correct the percentage of fat in our diluted mixture by addition of the required proportion of cream. Such a cream is much needed ; at the present time, so far as I know, there is only one dairy in London which regularly supplies a cream warranted to contain a constant and declared percentage of fat. But in the absence of such cream what is to be done ? There are several ways of getting out of the difficulty. It is sometimes convenient to use 'top-milk', by which is meant the upper portion of milk which has been allowed to stand in a vessel until the cream (i.e. the fat) has risen to the top; if this upper part be used for dilution instead of the lower, it has the advantage of containing the additional fat which has risen from the lower part, whilst the proportion of proteids is scarcely altered, so that the result of subsequent dilution is to diminish the proteid without causing as great a deficiency of fat as occurs when ordinary milk is diluted.

With average London milk (i.e. milk which contained 3·2 to 3·5 per cent. of fat) which had been allowed to stand three hours, I obtained the following results :

Quantity of top-milk taken from one quart of milk after three hours.	*Proportion of fat.*
1 pint	5·5 per cent.
15 ounces	6·5 per cent.
10 ounces	8·0 per cent.
5 ounces	11·0 per cent.

But such a method is unsatisfactory ; the resulting percentages of fat are subject to considerable variation, and as this variation occurs in a comparatively small percentage, it makes the use of such cream unreliable where accurate modification of milk is necessary. The richer gravity cream—that is, cream made by allowing milk to stand several hours, and then skimming off the cream which has risen without taking with it any of the milk below the level of the cream—is open not only to the objection of variability of richness, but also to a more serious one, namely,

that its preparation involves prolonged standing of the milk, which at any time, but especially in warm weather, is accompanied with risk of chemical change from bacterial contamination. I have known cream to be prepared in this way by allowing ordinary London milk to stand as long as twelve hours after it had been delivered to the retail purchaser, and as such milk was probably at least twelve hours stale when it was delivered, and the cream from it would be used for infant-feeding several hours after its preparation was completed, the practical risk of bacterial contamination must be a very real one, however much it may be possible in theory to reduce it by special precautions.

In the country, where milk is available for home use almost directly it is drawn from the cow, this objection is less serious ; but even so it cannot be desirable that milk should be left standing fifteen to seventeen hours, as it sometimes is, for the cream to rise. Where, however, only such cream is available, it may be useful to know that a quart of milk allowed to stand twelve to fifteen hours at the ordinary temperature of a cool dairy will yield a cream containing 17 to 25 per cent. of fat ; and, as a working hypothesis, it is safe to assume that simple gravity cream (not clotted cream) contains 24 per cent. of fat ; some samples will contain less, very few will contain more.

Far better than any such methods is the use of centrifugal cream, such as is supplied by any of the large dairy companies in London and is commonly sold in the shops of London and other large towns. In the preparation of this cream there is no standing of the milk for many hours ; the cream can be separated from the milk in a few minutes by a centrifugal machine, and with such a machine working at a fairly constant rate the cream made at any particular dairy shows only slight variations in strength.

Out of twenty-three samples bought from twelve shops in various parts of London, sixteen showed a proportion of fat between 40 and 50 per cent. ; only three contained less than 40 per cent. (36, 36·8, and 38·4 per cent. respectively) ; five showed proportions varying from 50 to 54 per cent. ; the average fat percentage of the whole twenty-three samples was 46·3.

Now the addition of fat which is required in any mixture for infant-feeding is practically never more than 3 per cent., and much more often only 2 or 2·5 per cent. is required. In order to obtain this with a cream containing 40 to 50 per cent. of fat, a dilution of 1 in 16 at least is necessary ; and it is evident that an error even of 16 per cent. in the assumed fat percentage of the cream would involve only an error of 1 per cent. in the fat of the

mixture ordered. For practical purposes 48 per cent. may be taken as the average proportion of fat in centrifugal cream—this is, I think, a safer average than 46·3, which was obtained only when the few exceptionally low percentages were included—and on this assumption the error either way cannot be more than 10 per cent. at the utmost, and will very rarely be more than 6 per cent. in either direction ; so that if we ordered, as is commonly necessary, a dilution of 1 in 24—that is, 2 per cent. of fat (the cream must be assumed to contain 48 per cent. of fat),—the utmost possible error is less than 0·5 per cent. in either direction. and it will rarely be as much as 0·25 per cent.

It is therefore a perfectly simple matter to correct the fat deficiency in any mixture. For example, suppose the infant is to have one part of milk with three of water, this will reduce the fat in the milk from 3·5 to 0·8 per cent. approximately ; it is necessary to add sufficient cream to raise the percentage from 0·8 to 3—3·5 per cent. The addition of 1 drachm of cream (assumed to contain 48 per cent. fat) to 2½ ounces of the mixture, i.e. 1 to 20, will be the addition of 2·4 per cent. fat approximately, raising the total fat percentage of the mixture to 3·2 per cent.

A further advantage of this high-percentage centrifugal cream is the very small proportion which it is necessary to add to any milk mixture ; this makes it possible to neglect all the other constituents of the cream, except the fat, in calculating the resulting percentages in the mixture. With any weaker cream of which a larger quantity must be added, it becomes necessary to consider the proteid and sugar in the cream as well as in the milk, and this makes the matter somewhat complicated.

I have sometimes used butter dissolved in the food, and have generally found that infants tolerated it well. It is more easily obtainable by the poor than cream, and is sometimes available when cod-liver oil is not at hand or is disliked. By the addition of a piece of butter about three-quarters of an inch square to a four-ounce feed of milk and water the fat was raised from 1·5 to 3·5 per cent. But so large a quantity of butter may disagree, and for an infant three or four months old, I have usually ordered a piece the size of a large pea, to be put into a feed of three or four ounces of diluted milk.

I do not think this method is to be recommended where other means are available for supplying the deficiency ; even with vigorous shaking it is difficult to mix the butter thoroughly with the rest of the mixture ; the taste of the butter is very obvious, and may be distasteful ; and lastly, butter is very commonly

adulterated with other substances, especially preservatives, which may prove harmful to the infant.

Sugar. I come now to the correction of the deficiency of sugar, which is easy enough.

But here, again, I must point out that Nature is not the hard taskmaster which some writers would make her, and it is not necessary to adhere rigidly to any exact percentage. Some infants thrive excellently on 4 to 5 per cent. of sugar, while others will do equally well on 8 to 10 per cent. But, none the less, I think that Nature has shown us the most generally suitable proportion in the milk which she has provided—namely, the mother's milk, which contains 6 to 7 per cent. of sugar ; some infants are troubled with flatulence or colic or diarrhœa directly this proportion is exceeded even by as much as 1 per cent. It is evident that in using cow's milk for infant-feeding, even if it were used undiluted, some addition of sugar would be desirable, for it contains only 4 to 5 per cent. of milk sugar. Dilution involves reduction of this proportion ; for example, if milk be diluted with three parts of water, the percentage of sugar will be only 1 per cent.

Elaborate methods have been devised for adding the exact quantity of sugar necessary to yield a proportion of 7 per cent., and with this object some writers have recommended the use of a solution of sugar as a diluent for cow's milk. This seems to me far less satisfactory than the use of the dry sugar itself ; the solution is much less convenient for storage, and what is much more important, it adds an unnecessary risk to infant-feeding by affording an excellent medium for the growth of micro-organisms while it is waiting to be used. The argument in favour of it is the facility it offers for exactly modifying the percentage of sugar when a standardized solution is at hand, and this, of course, is easily prepared.

But I would venture to object that in the first place no such extreme exactitude is necessary ; and in the second, a quite sufficiently exact percentage is obtainable by simpler methods.

A level teaspoonful (using a teaspoon which holds 2 drachms of water, i.e. rather a large domestic teaspoon) of milk sugar in 3 ounces of any fluid gives a 5 per cent. solution of sugar. Therefore if this quantity be added to a three-ounce feed of any milk mixture, 5 per cent. of sugar has been added ; and as the original proportion of sugar in the mixture is readily calculated by simple division, according to the number of times the milk has been diluted, the total percentage, after addition of the milk sugar, is known without any difficulty. Similarly a lump of ordinary

white cane sugar, half an inch square, in 3 ounces of fluid, gives a 5 per cent. solution.

With these two data alone it is easy to regulate the percentage of sugar accurately enough for the feeding of an infant with the most delicate digestion. The quantity of sugar which must be added to a feed of any amount, or of any proportions, can easily be calculated, and also easily measured with sufficient accuracy to ensure a proportion of 5 to 7 per cent. of sugar. As a matter of fact, with any of the ordinary milk mixtures made with such diluents as water, barley-water, or lime-water, the addition of 5 per cent. of sugar will give a total percentage which is quite near enough to Nature's average for practical purposes. For instance, if the milk has been diluted with two parts of water, the sugar will have been reduced to 1·3 per cent., and the addition of sugar in the proportion of one teaspoonful of milk sugar, or one lump, half an inch square, of cane sugar, to 3 ounces of the milk mixture, will raise the total sugar percentage to 6·3 ; while if two parts of milk to one of water are being given, the mixture will contain 2·6 per cent. of sugar, and addition of sugar in the proportion suggested will raise the percentage to 7·6. By making the added teaspoonful of sugar slightly less full, or reducing the size of the lump of the sugar added, it is easy to keep the resulting percentage within the 7 per cent. limit, if desired.

The choice between milk sugar and cane sugar is not, I think, one of very great importance, but I prefer the former if expense is no difficulty, for it has seemed to me less liable to cause flatulence. The result of modifying milk as I have suggested can be seen from the following example, in which a 3-ounce mixture has been prepared, containing one part of milk with three parts of water. The cream, as I have said, may be considered merely as an addition of fat ; its fluid bulk and also its proteid and sugar constituents bear so minute a proportion to the whole mixture that they may be left out of account for the sake of convenience.

Mixture.		*Resulting percentages.*	
Milk . . .	6 drachms.	Casein . .	0·81 per cent.
Water . .	18 drachms.	Lactalbumen .	0·18 per cent.
Cream (48 per cent. fat) . . .	1½ drachms.	Fat . . .	3·87 per cent.
Milk sugar . .	1 level teaspoonful.	Sugar . .	6·00 per cent.

The obvious defect in such a mixture, as compared with human milk, is the low percentage of lactalbumen—a defect which is present to some extent in cow's milk, even before

dilution, but which is increased when any of the ordinary diluents (water, barley-water, lime-water) are used to dilute the milk.

Diluents. What diluent is best in the modification of milk? In theory there is one, and only one ideal diluent, and that is whey. Whey is, in fact, Nature's diluent for milk, and milk may be regarded as whey containing in suspension curd-forming proteid and fat. When the curd is strained off it carries with it the greater part of the fat, and there is left a clear or slightly turbid fluid, the whey, which contains all the original constituents of milk except the casein and fat (a varying proportion of the fat, but rarely more than 1 per cent., according to my own analyses, can be retained in the whey, if the curd is thoroughly broken up before straining); in particular, the whey contains practically the same proportion of lactalbumen, salts, and sugar, as was originally present in the milk.

If, therefore, whey be used as a diluent, however far the dilution be carried, in order to reduce the proportion of curd-forming proteid, there will be no dilution whatever of the lactalbumen.

This makes an appreciable difference in the nutritive value of the mixture. In the example given above, the substitution of whey for water would raise the proportion of lactalbumen from 0·18 per cent. to 0·75 per cent., and the proportion of total proteids, therefore, from about 1 per cent. to 1·5 per cent.; and this increase in the proteids is obtained without increasing the difficulty of digestion.

The use of whey involves some care in its preparation, if the whey is to be of the greatest possible value. The rennet or curdling ferment which is used to prepare the whey must be destroyed by heat before the whey is used as a diluent, otherwise the ferment, being still active, will curdle the milk to which the whey is added; but if a temperature above 160° F. is used, the lactalbumen is coagulated. To avoid this, a temperature of 150° to 155° may be used, which destroys the ferment but does not coagulate the lactalbumen.

This is, I think, desirable, but not absolutely necessary; the change in the lactalbumen, which results from heating even to boiling-point, seems to interfere little, if at all, with its digestibility, and probably equally little with its nutritive value. Of course, if whey is used as a diluent, it must be remembered that the percentage of sugar in the milk is not reduced at all, and therefore less addition is necessary to raise the 4 per cent. present to the required 6 to 7 per cent. The fat in the whey must also be considered. If the whey has been made without breaking up

the curd at all, the amount of fat present (0·2 to 0·5 per cent.) is so small that it can be disregarded; but as some breaking up of the curd has usually taken place before the whey is strained off, it is generally more accurate and certainly safer for practical purposes to assume that the whey contains 1 per cent. of fat, and that therefore the resulting deficiency of fat, when milk is diluted with whey, is 1 per cent. less than it would be if water were used as diluent.

The following modification may be compared with that shown above:

Mixture.		*Resulting percentages.*	
Milk	6 drachms.	Casein	0·81 per cent.
Whey	18 drachms.	Lactalbumen	0·75 per cent.
Cream (48 per cent. fat)	1¼ drachms.	Fat	3·3 per cent.
Milk	½ level teaspoonful.	Sugar	6·50 per cent.

Here the proportion of curd-forming proteid has been reduced without reducing the proportion of lactalbumen; the mixture approximates more nearly to human milk in respect of its lactalbumen than is possible with any other diluent.

But the preparation of whey entails a certain amount of trouble and makes infant-feeding more expensive than when plain water or barley-water is used as diluent, so that its use, though excellent in theory, is not generally practicable. In many cases, however, it has proved useful, and I have found even hospital out-patients willing to use it for several months.

If whey cannot be obtained, barley-water has no doubt some advantages over plain water in rendering the curd more flocculent. This has been disputed, but in some experimental observations which I made with cow's milk and acetic acid it seemed that dilution with barley-water resulted in a slightly less tough curd than when plain water was used as diluent: and it is, I think, indisputable that some infants can digest cow's milk better when it is diluted with barley-water than when diluted with plain water. It may be granted also that for an infant who can digest starch, barley-water may increase to a very small degree the intrinsic value of the mixture. But in spite of these possible advantages I have no great liking for its use in infant-feeding.

Barley-water consists practically of nothing more than water with a little starch: prepared by any of the ordinary methods whether from pearl-barley or from prepared barley, it contains 1 to 2 per cent. of starch. It is quite certain that an infant's

power of digesting starch is very slight during the first few months of life ; although there is probably some amylolytic power both in the salivary and in the pancreatic secretion even at birth ; indeed in an extract from the salivary glands of a stillborn infant I found distinct amylolytic power. But clinical experience shows that starch digestion remains very feeble in most infants for at least six or seven months after birth ; and even 0·5 to 1 per cent. of starch seems to be more than some infants can digest.

I have several times seen slight degrees of rickets where barley-water had been used from early infancy, and there is no doubt that it is not uncommonly a cause of flatulence and discomfort, and sometimes of looseness of the bowels and of redness about the buttocks. These objections apply, of course, specially to infants under the age at which starch becomes a suitable element in the food, a point which I shall consider later : here I will only say that in my opinion starch is best avoided until the age of nine months.

I have sometimes used rice-water as a diluent, prepared by soaking two heaped tablespoonfuls of rice, previously washed, for three hours in a quart of warm water, and then allowing the mixture to simmer for an hour before straining. For infants of nine months or more with looseness of the bowels, this is certainly preferable to barley-water as being less laxative, and I have seen good results with it ; but nowadays we have a less troublesome resource in simple dilution with water and the addition of sodium citrate, which is at once a more effective method of assisting digestion of the curd and at the same time of diminishing looseness of the bowels ; the nutritive value of any of the cereal decoctions which have been used—barley-water, rice-water or oatmeal-water—is so small as to be hardly worth considering.

My own bias is in favour of plain water as a routine diluent for milk in infant-feeding ; in the large majority of cases the infant does perfectly well with such dilution, if the proportions are properly arranged ; moreover, it has the great advantage of simplicity. The addition herewith of lime-water has a certain value : in some experiments which I made a few years ago, lime-water seemed to have some definite though slight effect in diminishing the firmness of the curd, and I think that there can be no doubt from clinical experience that it assists digestion to some extent ; but in my opinion its chief value is in counteracting any tendency to looseness of the bowels. The proportion of lime-water to be added is $\frac{1}{2}$ to 1 ounce to every 3 ounces of milk.

mixture, or if the more concentrated liquor calcis saccharatus be used, 15 to 30 drops to every 3 ounces.

For a healthy infant who is able to digest the milk simply diluted with water there is nothing to be gained by the addition of lime-water. It has been customary to contrast the acidity of cow's milk with the neutral or alkaline reaction of human milk and to advise the addition of some alkali, whether lime-water or bicarbonate of soda (gr. iv added to each feed of 3 ounces) to counteract the acidity: but is this necessary? The success of buttermilk, or milk acidified with lactobacillin, and of whey prepared by curdling milk with acids, such as sherry or tartaric acid, clearly proves that acidity *per se* is not necessarily harmful to the infant; and clinical experience abundantly proves that healthy infants thrive on mixtures of fresh cow's milk in which no attempt has been made to neutralize the acidity.

But there is another reason which I often hear assigned for the addition of lime-water to milk: 'We are giving lime-water to prevent rickets.' Now how or when this idea arose, that lime-water is of any value in the prevention of rickets, I do not know, but that it is an entirely mistaken idea is shown by many facts, one of which is that cow's milk, even if it were diluted with two parts of water—a degree of dilution which, if continued for many months, would almost certainly result in rickets—would still contain a very much larger proportion of lime salts than does human milk. Lime-water is neither preventive nor curative of rickets.

Laboratory modification. Having said this much about the home modification of cow's milk, I must mention also the 'laboratory modification' of milk and 'percentage feeding', of which we have heard so much within the past few years. How far is any such exact modification practicable? A very interesting paper has been written by Dr. Wentworth, of Boston, U.S.A., showing that of twenty-six milk prescriptions made up at a milk laboratory in that city, not a single one corresponded exactly with what was ordered; there was as much as 0·2 per cent. variation in the proteids, and as much as 1 per cent. error in the fat and in the sugar. I have made a few control analyses of the same kind myself, and have found small variations (as much as 0·5 per cent. in the fat and rather less in the proteids) from the formula ordered; but I confess I am more inclined to wonder at the degree of accuracy which was attained than to criticize these slight deviations. At the same time these observations show that even at a special

laboratory the percentages obtained are, if more accurate than those obtained with home modification, still only approximate; and to expend scrupulous care over the second place of decimals —as I have seen done—in writing a ' milk prescription ' is either to humbug ourselves or our patients.

But is any such strict accuracy either necessary or desirable ? Surely not, if Nature is to be our guide. Human milk varies not only with the health of the mother and her diet, but even with the time of day and with the portion of breast-milk taken. I have found a difference of 1 per cent. in the fat in two samples of a woman's milk taken at 11 a.m. and 11 p.m. respectively, and I have repeatedly found the morning milk of women to be richer in fat than the evening milk. In the milk first drawn from the breast I have found as little as 0·4 per cent. of fat when the middle-milk at the same feed contained 3·2 per cent. of fat, so that obviously the infant will get a food varying in its average composition according to the completeness with which it empties the breast. A rigidly fixed percentage of each constituent is not only unnecessary, but is also unnatural.

Nevertheless it is clear that there is a certain range of variation in the proportion of each constituent which is permissible for any particular infant, and this range has no very wide limits. The value of a knowledge of the percentage composition of milk, and the methods by which the percentage of each constituent can be regulated approximately, lies in its enabling us to determine with some degree of accuracy what these limits are, and when we have determined them, to regulate the proportion of each constituent so that it falls within this range. Where expense is no object, it is a very great convenience to have milk mixtures supplied with the proportions already regulated according to the medical man's instructions; but let it be realized that this regulation of percentages is carried out in exactly the same way as is done in the home modification of milk, and that any medical man who possesses a working knowledge of the average composition of milk and cream (or, still better, can obtain analyses of the particular milk and cream which are to be used), can regulate the percentages of proteids, fat, and sugar, with sufficient accuracy for any practical need of infant-feeding.

But here I wish to insist upon an extremely important point in the preparation of an infant's food, whatever its nature may be, namely the use of accurate measures of quantities: the doctor orders one part of milk to two parts of water, and the nurse thinks she can calculate the amounts by rough measure-

ment in a jug, or a domestic tablespoon is used which may give correct proportions but probably makes the total amount of the feed much larger than was intended. No constituent is more often inaccurately measured than the cream, and accuracy is specially needful in the measurement of this, for a very small excess of cream is likely to give rise to trouble : the nurse has perhaps a measure graduated in tablespoonfuls, but often she has none showing drachms, so a domestic teaspoon is used with the result that twice the quantity of cream ordered is given.

A domestic teaspoon very commonly holds 2 drachms, and sometimes $2\frac{1}{2}$ or 3 drachms : and it is a serious matter if 2 drachms of the ordinary centrifugal cream, containing 48 per cent. fat, are given instead of 1 drachm in a 3-ounce mixture, for the result will be a mixture containing altogether about 5 per cent. of fat, or even more, and it is no wonder if the infant is sick, or has loose stools. Cream should be measured in a minim measure, and the other constituents of the food in an accurately graduated glass measure.

Boiling of milk. Lastly I come to the important question, should the milk be boiled? Now on this point I wish to speak with no uncertain voice, and the more so, as I have often been responsible in former years for the usage of raw milk, that is, milk unheated except to the feeding temperature of 100° F. Not only throughout infancy, but throughout the whole period of childhood, the use of raw milk is attended with very serious danger, a danger which, in my opinion, altogether outweighs the disadvantages of heating the milk by pasteurization, boiling or sterilization. As is well known, the tubercle bacillus is present in cow's milk not very rarely; and even if cows are tested with tuberculin at intervals, it is still not certain that they will remain free from tuberculosis in the intervals; so that even where cows are kept by private individuals specially for their children and examined with every expert care, I hold that nevertheless the milk should not be given raw.

According to my own observations, 29 per cent. of the cases of tuberculosis in childhood are due to alimentary infection ; and I have been even more impressed with the clinical sequence which I have repeatedly observed : a child of good family history, who has always had boiled or pasteurized or sterilized milk, usually because he has been living somewhere where hygienic conditions were unsatisfactory, is brought to London or to some place where the parents suppose that milk is sufficiently pure to be given raw, and within a few months after he begins the use of

raw milk he develops tuberculosis. I have seen pitiful cases of this sort where it seemed as if life were thrown away by the giving of raw milk.

There are, of course, other risks besides tuberculosis; for instance especially in the summer an acute infective diarrhœa may result from some milk-borne bacteria, and occasionally scarlet fever, typhoid and diphtheria, may be conveyed by raw milk. If the heating of milk will avoid these risks, what objection can be raised to so simple a precaution ?

Amongst the laity one finds two objections very often raised when one suggests heating the milk ; the first is that any considerable heating of the milk makes it less nourishing; the other is, that boiled milk is a cause of rickets. It cannot be denied that marked changes occur in milk as the result of heating it to the boiling-point or even nearly to the boiling-point : certain ferments usually present in the fresh milk are killed : the lactalbumen undergoes some change by which it probably becomes slightly less digestible : the casein is somehow altered so that it is less completely curdled by rennet: and some of the salts, particularly the calcium salts, are said to become less soluble; but has all this any practical importance? Experience shows that boiled milk is digested usually just as easily as unboiled milk, and there is, so far as I know, no evidence that its nutritive value is impaired in any way except in one special direction, namely as an antiscorbutic.

It is quite certain that heating milk, especially if the heating is to the boiling-point or higher, and is prolonged for many minutes, renders it scorbutic ; but if one may judge from the rarity of scurvy in infants fed on milk which has merely been heated just to the boiling-point without prolonging the boiling, it is only scorbutic to a very slight degree, and certainly less so than is sterilized milk. Pasteurized milk, that is milk heated to 155°–160° F., for twenty minutes, is even less open to this objection, for although cases are on record in which it has apparently produced scurvy, they are excessively rare. So far as its nutritive value is concerned, I think it may be taken as proved by experience that milk, whether pasteurized or heated to just below the boiling-point, loses so little, that this disadvantage is not worth considering in comparison with the safety gained thereby from tuberculous infection.

The idea that rickets is produced by boiled milk has, so far as I know, no foundation whatever : in fact, I suspect it arose from the confusion produced by the unfortunate term 'scurvy rickets',

which was in use before it was recognized that infantile scurvy is not a form of rickets and has no essential connexion with rickets.

A far more practical objection to the use of boiled milk is the dislike which many children show to its taste and to the 'skin' which forms on the top of it. The latter is easily removed with a spoon, but if the milk has been allowed to stand many minutes before it is removed some of the cream will be removed with it, and the milk rendered by so much the poorer. The taste is a difficulty which with some children is insuperable. On every ground, I think, pasteurization is to be preferred to boiling if it is practicable: it necessitates some special apparatus—various so-called 'sterilizers', such as Aymard's or Hawkesley's, which can be obtained through any instrument-maker or chemist, will serve the purpose—and it requires care. If on grounds of expense and trouble pasteurization is impracticable, I think the next best procedure—which has the great advantage of being perfectly simple—is to heat the milk until bubbles just begin to rise, but not so that it 'rises to the boil'.

Goat's Milk. When boiled or pasteurized milk is so distasteful to the child that it cannot be given, the difficulty may be overcome by substituting goat's milk, which does not require boiling or pasteurizing, as the goat is almost entirely immune from tuberculosis.

There is a popular idea that goat's milk has necessarily an unpleasant flavour of its own, but obtained from cleanly and properly-kept goats, the milk is quite indistinguishable in taste from cow's milk.

It must be remembered, however, that goat's milk is fully as rich as cow's milk, and therefore in any particular case may require dilution upon the same lines. This will be evident from its composition:

Proteid . . .	3·7	Casein 3·0 per cent. Lactalbumen 0·7 per cent.
Fat . . .	4·2 per cent.	
Sugar . . .	4·0 per cent.	
Salts . . .	0·5 per cent.	

So far as digestibility is concerned, goat's milk offers no advantage over cow's milk, but its comparative safety as regards tuberculosis is a very real gain, and in the country the small cost of keeping a goat brings it within the reach of most. In towns, goat's milk is less readily available, but in London at any rate it can be obtained from various suburban sources.

Humanized Milk. To obviate the necessity for careful modification of milk at home, many of the large dairy companies sell what they call humanized milk, and on this subject I must say a few words, for I frequently find that one of these preparations has been given under the impression that the milk has undergone some mysterious change whereby it is rendered more digestible than an ordinary milk mixture prepared at home. The term 'Humanized' has an attractive, not to say alluring, sound which is apt to be misleading: humanized milk is nothing more than an ordinary mixture of diluted milk with cream and sugar, and differs in no way from a similar mixture made at home, except that the shop-sold mixture has usually been sterilized at a very high temperature. The reason of its success in some cases is simply that a careful accuracy is maintained in the proportions of the mixture—a point in which home modification, especially when left to an untrained domestic nurse, too often fails.

As examples of such preparations from well-known London dairies, the following analyses, for which I am indebted to the Companies named, may be quoted:

Aylesbury Dairy Company. Humanized Milk.

	No. 1.	No. 2.	No. 3.
Proteid	1·16	2 21	2 94
Fat	3 88	3·79	3·82
Sugar	5 38	5·42	4 93

Welford's Dairy Company. Humanized Milk.

	'Modified.'	'Facsimile.'	'Enriched.'
Proteid	0 93	1·35	2·00
Fat	3·10	3·60	4·00
Sugar	6 40	6·60	6 50

Friern Manor Dairy Company. Humanized Milk.

	No. 1.	No. 2.	No. 3.	Special.
Proteid	1·5	1·8	2·0	0·6
Fat	3 5	3 5	3 5	3 5
Sugar	7·0	7·0	7 0	7 0

The Express Dairy Company, under the name of 'Infants' Milk', supplies milk modifications of three different strengths, in bottle, containing sufficient for each feed according to the age of the child. The composition of the three preparations is: A, proteid 1·00 per cent., fat 3·00 per cent., sugar 6·00 per cent.; B, proteid 1·5 per cent., fat 3·75 per cent., sugar 6·75 per cent.; and C, proteid 2·00 per cent., fat 4·00 per cent., sugar 6·50 per cent.

Humanized milk is sometimes valuable where the home conditions make accurate milk modification impracticable, only let

it be remembered that there is no special digestibility of the curd in these preparations ; they are neither more nor less digestible than any home-made milk mixture containing the same proportion of curd. It is therefore a mistake to order humanized milk where a home-made mixture, containing the same or a smaller amount of casein, or where a peptonized milk, has already failed.

The complete sterilization of most of these commercial humanized milks is an important point to be considered in their use ; for a short time, say three or four weeks, this may constitute no objection ; it will, indeed, be an advantage where they are used upon a journey, but for ordinary purposes it is certainly a grave fault, for experience shows that infantile scurvy is far more likely to result from a fully sterilized milk than from one that has only been pasteurized or heated just to the boiling point.

CHAPTER IV

CURD-INDIGESTION

I SUPPOSE there is no commoner difficulty in infant-feeding than curd-indigestion. To some extent this is a preventable disorder, in the sense that if cow's milk is properly diluted from the beginning, curd-indigestion does not arise at all in the large majority of infants. It is induced in many cases by insufficient dilution of the milk during the first few weeks of life ; the infant who is given equal parts of milk and water at a month old is likely enough to fall into a troublesome condition of curd-indigestion; once upset the digestion of an infant by injudicious feeding and the power of digesting curd is often so impaired that it seems to be wellnigh lost altogether for a time and it may be weeks or even months before cow's milk can be given at the proper strength for the age. I have considered the subject of dilution already; here I will only repeat the opinion I have already expressed, that insufficient dilution of milk is a much commoner source of trouble than is too great dilution.

But curd-indigestion is certainly not always due to faulty dilution ; there are infants who from the very beginning show an extraordinary intolerance of the curd of cow's milk, so that even when dilution is carried to quite an extreme degree they are still unable to digest the curd. For these, as well as for the cases in which this trouble arises from improper feeding, we must have some method of securing assimilation of a sufficient proportion of proteid. As I have shown in the previous chapter, there are reasons why dilution should not be carried to an extreme degree : indeed, where circumstances make the addition of cream a difficulty, it is most important that dilution should be as little as is consistent with satisfactory digestion. In some way or other therefore we must assist the digestion of curd in these cases where moderate dilution is not sufficient.

Sodium citrate. Where the difficulty is slight, I think that by far the best means is the addition of sodium citrate to the feed. How this acts is uncertain; it has been stated that whereas under ordinary circumstances curd consists of tough calcium paracasein,

when sodium citrate is given the calcium combines with the citrate, and a much less tough sodium paracasein is formed. But this is mere theory; the clinical fact is certain, that many infants who have previously had difficulty with curd, can digest cow's milk well when sodium citrate is added. The amount to be given is gr. ij–gr. iv in each feed, according to the amount of milk contained in the mixture: for milk up to 1 ounce 2 grains should be used; for any larger amount of milk, 3–4 grains are required. Sodium citrate is easily soluble in water, and is most conveniently prescribed with the required dose dissolved in a drachm of water, 1 drachm to be added to each feed.

This has the great advantage over barley-water that it is not liable to cause flatulence, nor has it any tendency to cause rickets, while it is superior to peptonizing inasmuch as it does not render the food scorbutic; but it must be remembered that it does not assist digestion to the same degree as peptonization does, and therefore fails entirely in the severe cases of curd-indigestion in which peptonized milk may succeed.

Sodium citrate has, moreover, one very marked effect which, whilst usually a drawback, is sometimes an advantage; namely, that it is constipating. Almost invariably one finds that after sodium citrate has been used for a few days there is complaint that the bowels are costive, and if the addition of cream to the milk is not sufficient to prevent this, it is often necessary to give some drug regularly two or three times a day such as manna, grey powder, or senna, to counteract the effect. From my own observations I do not think that anything is to be gained by giving sodium citrate in larger doses than those I have mentioned. Indeed, I think that harm may possibly be done by very large doses; in the case of a wasted infant to whom 10 grains of sodium citrate had been given in each feed, general œdema appeared within a few hours after these doses were begun and the doctor, suspecting it might be due to the sodium citrate, stopped this drug, whereupon the œdema subsided; he again added the sodium citrate, and again the œdema appeared.

Peptonization. If there is still curd-indigestion in spite of careful dilution of the milk and the addition of sodium citrate, I think the wisest plan generally is to proceed at once to peptonization of the milk. For this purpose Fairchild's Peptogenic Milk Powder is useful; one cap-full (the screw-cap of the bottle in which it is sold is to be used as a measure) is sufficient to peptonize a pint of milk mixture, whatever the strength of the mixture may be, and no sugar is to be added, for there is

sufficient in the powder. Dr. B. Myers kindly estimated the sugar in this powder for me, and found that the addition of one cap-full to a pint of diluted milk raised the proportion of sugar by 3·4 per cent. The milk and water and cream, if any is to be used, are mixed cold, and the powder then added, and the whole warmed very slowly either over a flame which is small and capable of regulation so that the fluid comes to boiling-point in not less than ten minutes, or else by standing the mixture in a vessel of hot water as hot as the hand can bear for twenty to forty minutes, and then pouring into a saucepan and heating just to the boiling-point. This latter method is best for the severe cases in which full peptonization is required. The proportion of water and milk must be adjusted to the capacity of the infant ; in the case of a feeble wasted infant, 6 ounces of milk to 14 ounces of water will do for a beginning, and a slow advance can be made by increasing the milk by ½ ounce and diminishing the water by ½ ounce once every week or ten days. It is often wise to withhold cream altogether at first, and if it is to be added at all it must in these cases be added in much smaller proportion than the directions sold with the Peptogenic Powder advise. It will often be found that 2 drachms to the pint is as much as an infant will tolerate and at the most 6 drachms of the ordinary town-sold centrifugal or ' separated ' cream will be sufficient.

In ordering peptonized milk for an infant it should always be impressed upon the parents that it is only to be used for a limited time, the shorter the better, and that if it is necessary to continue its use for many weeks some antiscorbutic, either raw meat juice or fruit juice, must be given in adequate quantities, as the risk of scurvy from peptonized milk is a very real one ; this, however, hardly applies to the first four months of life, in which scurvy is almost if not quite unknown ; it is after the end of the fourth month and especially in those cases in which patent foods have been used for many weeks before peptonized milk is begun, as is often the case, that scurvy is likely to occur.

Except for the possibility of scurvy I know of no objection to the use of peptonized milk ; it has been feared that to pre-digest the food artificially would prevent proper development of the digestive function of the infant ; this objection I think has no foundation whatever ; in fact, as a matter of experience, infants who are fed thus even for several months ultimately digest as well as any other infant, and generally much sooner acquire normal digestion than those who are allowed to struggle along

beset with frequent digestive disturbance from the attempt to live on food which is ill adapted to their feeble digestive capacity.

A more weighty objection, if it be proved, is the possibility of impairing growth by the use of peptonized milk. Certainly some of the infants who are fed for a long time on peptonized milk are for some years fragile-looking and under the proper weight, but it must be remembered that the reason for using peptonized food was great and continued feebleness of digestion, and the probability is that any infant with similar digestive difficulty whether fed on peptonized milk or otherwise would be below the average in growth and development; in short, it is not clear that the poor development is a result of the peptonized milk, and I can assert from a very considerable experience of its use that in the large majority of cases where pre-digested milk is used for several months the child is subsequently fully up to the average in growth and weight.

The return to unpeptonized milk must be made very gradually: using the Peptogenic Milk Powder, I am in the habit of ordering a gradual replacement of the contents of the measure by milk sugar; one fourth part at first, then a half, and then three-quarters is to be milk sugar, and so on till the whole measure contains only milk sugar; these changes are made at intervals of a week or ten days, while the time and manner of peptonization is otherwise unchanged: in this way it is usually easy by degrees to dispense with peptonizing altogether. Another mode is gradually to diminish the time of peptonization. Whatever method is used I think that where there has been much difficulty of curd-digestion it is wise to continue the peptonization until the proportions of milk and water used are up to the proper strength for the age, or at any rate, not far short of it.

But in spite of full peptonization it will occasionally be found that an infant cannot digest the curd of cow's milk. In theory the next step should be the removal of the curd altogether as is done in the preparation of whey.

Whey. Whey is usually made by the addition of a teaspoonful of some preparation of rennet such as Benger's or Lazenby's Rennet, or Fairchild's Pepsencia to a pint of milk warmed to 100° F. After addition of the rennet, and stirring it thoroughly into the milk, the milk is allowed to stand until a solid mass of curd has formed, and the whey has so far separated that it can be strained off as a slightly turbid fluid. Whey made in this manner consists of lactalbumen 0·75 per cent., fat 0·2 per cent., sugar 4 per cent., from which analysis it is clear that not only

has the casein or curd disappeared, but entangled in its meshes the fat globules also have been almost entirely removed, so that whey is but poor food if used alone. To some extent the whey can be made less deficient in fat by breaking up the curd very thoroughly with a fork before straining; in this manner I have had prepared from ordinary London milk (3·2 per cent. fat) a whey containing 0·8 per cent. fat, and with richer milk it is possible to obtain a whey containing about 1·5 per cent. of fat.

Now in theory such a food as this should suit admirably in cases where there is difficulty of curd-digestion, and sometimes it does. When this is so, by the addition of cream in suitable amount (1 measured drachm of the ordinary 48 per cent. 'separated' cream to every 3 ounces of whey) and of sugar (half a level teaspoonful of milk sugar to the 3 ounces) we may make a food which may be continued for many weeks with excellent result. The low proportion of proteid may be rectified by adding raw beef juice,[1] 1 drachm to every 3 ounces.

Where expense is a serious consideration, as in the case of hospital out-patients, I have sometimes used with excellent results a whey prepared with tartaric acid after a method devised by Dr. Bernard Myers and myself. My own observations on this tartarated whey were as follows: tartaric acid gr. viij dissolved in half a drachm of water was added to half a pint of milk which had been heated till it just began to bubble. After stirring the tartaric solution enough just to mix it well with the boiling milk, the mixture was kept simmering for five minutes; it curdled and was then strained through butter muslin. This gave a whey, somewhat turbid, with no appreciable acid taste, but faintly acid to litmus; its specific gravity was 1030; it contained fat 1·2 per cent., total proteid 0·58 per cent. On experimental addition of about 15 drops of pure acetic acid (which would coagulate casein if any were present), no further curd was formed; but on warming the whey to 100° F., and adding 15 drops of rennet to 1 ounce of whey, a light flakey curd appeared and floated on the top, but only forming a thin layer which disappeared on shaking the whey. I found that milk could be added to this tartarated whey without any curd being formed in spite of its slight acidity. Whey prepared thus costs $\frac{1}{64}$ penny per half pint (above the cost of the milk) whereas if any

[1] Quarter of a pound of raw beef is minced finely and placed in a cup with just enough water to cover it, let it stand two hours covered in a cool place, then squeeze through muslin.

form of rennet is used, the cost is ½–¾ penny per half pint. It has also the advantage that it is distinctly richer in fat than is rennet-made whey, owing to the fact that the curd does not form *en masse* but only in small clots, and so does not entangle so many of the fat globules, and evidently there is also some proteid left in the whey which is coagulable by rennet, and which would therefore be absent from rennet-made whey.

I have used the tartarated whey, as I said, with excellent results, I have also seen it fail several times : but where any food may fail, and where the more expensive methods of feeding are not available, this cheap method of preparing whey is certainly worthy of trial. Sugar in the proportion of one lump of white sugar half an inch square, or one level teaspoonful of milk sugar to every 6 ounces of this tartarated whey, will bring the proportion of sugar to 6–7 per cent., a suitable proportion in most cases. It will seldom be necessary to increase the proportion of fat by adding cream, for the cases in which such whey is necessary are just those in which it is usually advisable to give also a low proportion of fat, and for the two or three weeks during which it may be found necessary to use this whey, the fat present in it will often be sufficient.

Yet another form of whey and one which is extremely useful under certain conditions is white wine whey or sherry whey. Where curd-indigestion has already reduced an infant to a state of emaciation and exhaustion, especially where there is much vomiting, and where indigestion is associated with much screaming soon after feeds, I have often found that improvement at once followed the use of sherry whey. From some experiments which we made upon this food, Dr. B. Myers and I concluded that the best method of preparing sherry whey is as follows : 10 ounces of milk are heated until just boiling, then 2½ ounces (accurately measured) of cooking sherry are added and heat is applied until the mixture begins actually to ' boil up ', when it is removed from the fire and allowed to stand three minutes ; the curd is then strained off through a two-fold layer of butter muslin. It is best to use a ' cooking sherry ' (costing about a shilling per bottle) not a ' drinking sherry ' (costing about 2*s.* 1*d.* per bottle or more), for the cheaper cooking sherry is more acid and therefore a less quantity is required to curdle the milk ; we found that 3–4 ounces of ' drinking sherry ' were required where 2½ ounces of the cheaper sherry was sufficient. This is a point of practical importance, for, however prepared, sherry whey contains a considerable proportion of alcohol, and it is desirable to keep this proportion low, otherwise

the quantity of the food which can be given at each feed is necessarily very limited.

'Drinking sherry' we found to contain 16·46 per cent. of alcohol by weight, whereas 'cooking sherry' contained 14 per cent. of alcohol by weight; clearly, as more of the drinking sherry would be required, and it contains more alcohol, the whey prepared with it would contain much more alcohol than that prepared with the cooking sherry. Taking brandy as a convenient standard of comparison in considering the alcoholic value of sherry whey, we found that prepared according to the directions given above, this whey contained 2·3 per cent. of alcohol by weight and, as average brandy contains 42·5 per cent. of alcohol, 1 ounce of sherry whey is equivalent in respect of alcohol to 25·8 minims of brandy.

The effect of sherry whey, however, like that of the various alcoholic liquors, does not depend merely upon the amount of alcohol present; there are other constituents which take a part in determining the effect of each particular wine or spirit, and there can, I think, be no doubt that the essential oils or volatile ethers present in sherry, contribute to the carminative effect which is one of the virtues of this food. On account of the alcohol which it contains, sherry whey is quite unsuitable for prolonged use. I have sometimes used it as the only food for several days, but it is best used to replace some other food only at the alternate feeds or two or three times a day. Its use should be discontinued as soon as the digestive trouble has subsided. The cases for which sherry whey is most suited are those in which, on account of intolerance of larger feeds, a very small amount has to be given at each feed; at any period of infancy I think 2–2½ ounces of sherry whey should be considered the largest amount allowable, although I have in exceptional cases given larger quantities for two or three days with decided benefit, for instance, 3 ounces every three hours to an infant of six months, and even 4 ounces every three hours to an infant of twelve months. Sherry whey is, in fact, to be regarded only as an emergency food which may be of the greatest value in carrying an infant round a dangerous corner, but is to be discarded directly the emergency is past.

In practice, however, it is surprising how often whey of any kind fails in cases where there is curd-indigestion, or perhaps one should rather say where there is intolerance of milk, for it really seems in some cases as if the infant's difficulty were not merely with the curd but with cow's milk, however modified.

These are the cases in which every now and then some patent food or condensed milk succeeds after all other feeding has failed, and gains thereby a reputation for general suitability which is wholly unwarranted, inasmuch as the prolonged use of the very food which seemed, when used only for a few weeks, to save the infant's life, is likely to produce disastrous results such as rickets and scurvy. Before, however, considering these foods I must mention another and more valuable means of overcoming the difficulty of curd-digestion, namely, the use of asses' milk.

Asses' milk. The composition of this is shown by the accompanying analysis:

Proteid	1·8 per cent.	Casein	1·0 per cent.
		Lactalbumen	0·8 per cent.
Fat			1·0 per cent.
Lactose			5·5 per cent.
Salts			0·4 per cent.
Water			91·3 per cent.

Asses' milk should not be heated to the boiling-point; it is only to be warmed to the ordinary feeding temperature (100° F.) and is given undiluted, unless it be necessary to add some lime-water or sodium citrate to counteract the slightly laxative effect which is a peculiarity of asses' milk. If either of these must be added, as small a bulk of water as possible should be used with them, otherwise the extremely weak asses' milk will be made still weaker; for this reason the liquor calcis saccharatus is preferable to lime-water for this purpose (20–30 drops may be used to every 3 ounces), or sodium citrate, gr. iij may be used dissolved in half a drachm of water; this is more convenient for measurement than the plain powder, but tabloids are prepared by Messrs. Burroughs & Wellcome which might be used for this purpose.

Asses' milk is very weak both in proteid and in fat, and it is partly on this account that it is so easy of digestion, but not only so, for the curd formed is finer and more like the curd of human milk than would be that of cow's milk diluted down to the same strength. Upon asses' milk the stools often rapidly lose their undigested appearance, the infant ceases to whine and cry, and there may be a rise in weight for two or three weeks, but, as might be supposed, so weak a food is not sufficient to secure much continued progress in weight, and my own experience has been that it is almost always necessary after a short time to abandon the asses' milk on this account; nevertheless, as a

temporary measure, I have again and again found it of great value.

There is, however, another reason why asses' milk can seldom be continued long ; in London it costs six shillings per quart, and in most parts of the country it is obtained from London, so that then there is the added cost of conveyance, making the cost prohibitive except for the wealthy.

Wet-nurse. But there is a better resource than asses' milk, in these cases where fresh cow's milk cannot be digested, namely, a wet-nurse ; I have already referred to the wonderful improvement which usually follows the use of a wet-nurse ; here I will only say that I believe there are some infants whose feebleness of digestion is such that no preparation of cow's milk, and no patent food will save them, the one and only resource which will succeed is a wet-nurse.

In such cases the change which often follows at once upon the use of a wet-nurse is truly remarkable ; the infant who has been wasting for weeks and crying continually in fretful misery will often gain weight rapidly, sometimes at an almost incredible pace ; I believe I am right in saying that I have known nearly a pound gained in three days when a marasmic infant began being suckled by a wet-nurse, and hardly less gratifying is the placid contentment which takes the place of the former fretfulness.

Dried milk. But if asses' milk and a wet-nurse are impracticable luxuries as they must needs be for the majority, what is to be done if peptonized milk has failed ? The recent introduction of desiccated milk has, I think, furnished us with one useful resource in such cases ; there have long been upon the market preparations of desiccated milk combined with malted cereals ; such are the Allenbury Foods, and Horlick's Malted Milk, but these have the great disadvantage that they contain an excessive proportion of carbo-hydrate (not starch, but soluble carbo-hydrates, the products of malting), and this excess, though it serves to increase the fattening property of these foods, is liable to set up digestive trouble at the time, and probably eventually to favour the occurrence of rickets.

The plain desiccated milk is free from this objection while it shows the same fine division of curd when an acid is added to the solution of the milk powder, and for this reason is sometimes digested well where even peptonized milk has failed. It can be obtained in various strengths ; for instance, the 'Cow and Gate' Brand is prepared in three strengths, one consisting of full milk, dried into a powder, another consisting of milk from which half

its original fat has been removed, called 'Half-cream Milk', and another called 'Dried Separated Milk', made from skim milk, and said to contain about 1 per cent. of fat. Another firm prepares a dried milk to which cream and milk sugar have been added before desiccation so that a sufficient amount of water can be added to reduce the proportion of proteid as low as 2·5 per cent. without reducing the fat below 3 per cent., and at the same time there is no excess of sugar. This preparation, sold under the name of Glaxo, has the following composition :

Proteid . .	21·7 per cent.	Mineral salts .	5·2 per cent.
Fat . . .	25·4 per cent.	Water . .	4·8 per cent.
Milk Sugar . .	42·9 per cent.		

After dilution according to their directions, it contains one tenth of these proportions, so that there is fat 2·5 per cent., proteid 2·1 per cent., milk sugar 4·29 per cent. Upon one or other of these preparations of desiccated milk an infant will often thrive excellently for a time, but I think it is very advisable that parents as well as medical men should know that any drying of milk must render it liable to cause scurvy if used too long, and therefore these desiccated milks, though safe enough for an infant under four months of age when scurvy rarely if ever occurs, should not be used for older infants for more than a few weeks unless some antiscorbutic such as raw meat juice or orange juice is added to the diet.

CHAPTER V

ON THE USE AND ABUSE OF CONDENSED MILK AND PATENT FOODS

One outcome of the common difficulty in curd digestion is the widespread and too often indiscriminate use of condensed milk and patent foods. How often one hears that an infant 'couldn't take fresh cow's milk', so recourse was had forthwith to some patent food! Now I venture to say that in the large majority of such cases, the infant could have taken fresh cow's milk perfectly well, had it been properly adapted from the beginning to the child's digestive powers: often it has been given insufficiently diluted or in too large bulk or no attempt has been made to overcome the difficulty by such methods as I have described in the preceding chapter, and perhaps the most useful of all measures in such cases, partial peptonization, has not been tried at all.

'But,' says one, 'surely peptonized milk is scorbutic, and therefore just as objectionable as any patent food.' To this I would reply that peptonized milk is undoubtedly scorbutic, though when only peptonized for a short time, say ten or twelve minutes, it seems to have very little tendency to produce scurvy, certainly much less than any of the patent foods most commonly used; but even when more fully peptonized, such milk has still the very great advantage over patent foods, that the composition of the mixture can be adjusted by gradual increase or decrease of this or that constituent to the requirements of the particular infant, deficiency of fat and excess of sugar and presence of starch can be avoided, and the transition from the peptonized milk to unpeptonized fresh milk can be graduated so finely that it is rarely difficult by this means to coax a feeble digestion into tolerating a plain mixture of fresh milk. It cannot be questioned that the digestive disturbance and failure of nutrition which results from curd indigestion are sometimes overcome by substituting for fresh cow's milk condensed milk or some patent food in which milk is present in a desiccated form and therefore gives a fine loose curd which is much easier of digestion than the large tough clot of fresh cow's milk; but if at the same time we are compelled by the composition of the food, to give a proportion of sugar

which is much higher than an infant should have, or a proportion of fat which is far too low, we shall sometimes merely substitute one form of indigestion for another, and in place of curd indigestion we shall produce in the infant the flatulence, colic, disturbed sleep and looseness of the bowels which are the evidences of carbo-hydrate indigestion ; or if the infant is not so readily upset by excess of sugar or starch, we may find after some months that although the infant has appeared to thrive, the low proportion of fat in the food has led to some degree of rickets, so slight it may be that only a trained observer would detect it, but sometimes so evident that none can overlook it.

But the mischief does not end here, for the lay public seeing that Dr. So-and-so ordered Somebody's Patent Food with good result, conclude that what suits one infant must suit another, and that even if that particular food does not suit, Somebody Else's Infant Food may equally well be tried, and knowing nothing about the particular properties of particular foods, and the conditions under which a particular patent food or condensed milk may sometimes be advantageous, they use these various 'foods' with disastrous result, inflicting upon infants a vast amount of needless misery and suffering, sometimes even with fatal ending.

Amongst the out-patients of a children's hospital, one of the commonest sights is the miserable screaming emaciated infant who has been fed upon some patent food or condensed milk, and although amongst the well-to-do the infants are rarely allowed to fall into such a condition, because a doctor is usually consulted before the mischief has gone so far, yet I can say, from my own observation, that the minor disturbances from this cause are common enough in all classes, and I have sometimes seen in the infants of the well-to-do advanced marasmus, and many times very severe scurvy from the use of patent foods.

One is tempted, indeed, to condemn all such preparations unconditionally and without exception ; but such condemnation would be too sweeping ; there are conditions and circumstances which may not only justify the use of condensed milk or a patent food but may even make it advisable if—and it is a very important if—these preparations be used with an intelligent appreciation of their composition and properties, and therefore of the particular age and conditions for which each is suited.

The exact composition of each of these preparations, whose name is legion, it is impossible to retain in the memory ; but it is certainly possible for any medical man to know the chief

features of all the preparations which are in common use. These various foods fall into two groups: (I) those intended as substitutes for fresh cow's milk, and (II) those intended as additions to cow's milk. These are usually further classified in some such way as this :

I. Foods intended as *substitutes* for fresh cow's milk.
   (1) Condensed milk: desiccated milk (Glaxo).
   (2) Dried milk with cereal,—starch completely converted : Allenbury, Nos. 1 and 2 ; Horlick's Malted Milk.
   (3) Dried milk with cereal,—starch partially converted : Carnrick's Soluble Food ; Milo Food.

II. Foods intended as *additions* to fresh cow's milk.
   (4) Cereal,—starch completely converted : Mellin; Hovis Baby Food, No. 1 ; Moseley's Food.
   (5) Cereal,—starch partially converted : Allenbury Food No. 3 ; Benger's Food ; Savory & Moore's Food ; Theinhardt's Soluble Food.
   (6) Cereal,—starch mostly unconverted : Ridge's Food ; Neave's Food ; Frame Food ; Robb's Biscuits ; Chapman's Wheat Flour.

Upon the relative value of these preparations, their individual faults and virtues, some light may be thrown by certain general considerations. In the first place, I would emphasize the fact that the presence or absence of starch in a food is by no means the only point which determines its suitability for infant-feeding : a food may not contain a particle of starch and yet may be extremely faulty as an infant food.

It is clear, however, that a large proportion of patent foods do contain starch : in the above classification it will be seen that only those foods mentioned under groups (1), (2), and (4) are free from starch, and the question arises whether any starch is permissible to an infant, and if so, at what age. My own experience leads me to think that even such a small amount of starch (1 to 2 per cent.) as is present in barley-water is often harmful, and that all foods containing any starch whatever should be avoided until the infant is eight or nine months old, and even then starch-containing foods should only gradually be introduced, so that by the end of the tenth month an infant may be having two meals of starch-containing food in the day. Such preparations as Benger's Food, and Savory & Moore's Food, both of which contain a small proportion of starch after preparation in accordance with the makers' directions, are therefore unsuitable for the feeding of an infant during the first eight or nine months of

life, although after this age there is no objection on this score to their use once or twice a day; indeed, most of them may be useful in this way as a step in the introduction of starch-feeding.

But this, as I have said, is only one of the several points which have to be considered in deciding whether a food is suitable for infant-feeding: the question whether the carbo-hydrate present is soluble or insoluble, that is to say, whether it is all in the form of sugar or the products of malting, or is partly in the form of the insoluble starch, is of great importance; but whether soluble or insoluble there is yet another point to be considered, and one which is too often overlooked with reference to the carbo-hydrate contents of an infant's food, namely, the quantity. Many of the proprietary foods as given to the infant, contain a proportion of carbo-hydrate which is greatly in excess of that present in human milk. In human milk carbo-hydrate is of course present only as the soluble sugar, and very rarely exceeds 7 per cent. under any conditions; usually it is between 6 and 7 per cent., while the proportion of fat is about 3·5 per cent.: so that the proportion of fat to carbo-hydrate is approximately 1 to 2.

It seems hardly credible in the face of Nature's standard that the profession should be informed, as it is by one manufacturer's advertisement, that the 'normal standard for infants' foods prepared from milk and dextrinized cereals', is, fat, 5 per cent., carbo-hydrate, 71·5 per cent.! If such a food is to be used with addition of water only, it is clear that by no possible adjustment can it be made a suitable food for any period of infancy: if it is to be used as an addition to milk it is equally clear that, in order to prevent excess of carbo-hydrate, so very small a proportion of the food would have to be added, that the fat in the food becomes a negligible quantity, and the food can only be considered as an addition of a small quantity of carbo-hydrate to the milk mixture, the fat in which is just as much or as little as there would be in the plain milk-and-water mixture without the addition of this 'food'. But, according to the makers' directions, the foods are usually to be given in considerably larger proportion than would bring the proportion of sugar even approximately to that present in human milk, and this constitutes a grave defect in nearly all these patent foods.

In the Allenbury Foods, No. 1 and No. 2, for instance, the proportion of carbo-hydrate, when the food is diluted according to the maker's directions, is 10 to 11 per cent., that is half as much again as the percentage present in human milk; while Horlick's Malted Milk contains nearly 12 per cent. when four

teaspoonfuls of the food are diluted with eight tablespoonfuls of water as directed. Mellin's Food, again, which is intended to be used as an addition to fresh milk, is altogether free from starch, and on this ground commendable as an infant's food, but when one comes to investigate the proportion of carbo-hydrate which it would necessitate in the milk mixture, if given in accordance with the maker's directions, it can be understood why it sometimes gives the infant flatulence and looseness of the bowels.

Mellin's Food . . .	7 heaped teaspoonfuls.
Fresh milk . . .	15 tablespoonfuls (7½ ounces)
Cold water . . .	5 tablespoonfuls (2½ ounces)

These are the directions for preparing two meals for an infant of six months and over. Using an ordinary teaspoon, of 2 drachms fluid capacity, I found that a heaped teaspoonful of Mellin's Food weighed 85 grains; of this 70 per cent. is carbo-hydrate. Calculation on this basis shows that the mixture, as directed, would contain about 12 per cent. of carbo-hydrate. If Mellin is to be used—and there is no doubt that it sometimes assists the digestion of milk, and moreover acts as a gentle laxative—it should be used only in such proportions as to avoid excess of carbo-hydrate. It is convenient to know that, with a teaspoon of two-drachm fluid capacity, a heaped teaspoonful of Mellin's Food in 3 ounces of any milk mixture, means the addition of about 5 per cent. of carbo-hydrate to that already present in the diluted milk, so that at no period of infancy should this food be added in a higher proportion, and no sugar should be allowed with it.

Undoubtedly there are infants who will tolerate and even thrive upon, a food containing more than the breast-milk standard, 7 per cent., of carbo-hydrate; but it is equally certain that there are many infants who suffer from flatulence, discomfort, and looseness of bowels whenever this proportion is exceeded, even to a very slight degree.

But here I wish to emphasize a general principle of the greatest importance in infant-feeding, namely, that the suitability of a particular food for prolonged use is not proved by the mere fact that it is taken well, and produces no immediate ill effects. The evil results of unsuitable food may not appear until the food has been continued several weeks or months; indeed, in the case of rickets or scurvy the food which has produced the disease has often appeared to suit excellently, so far as freedom from obvious digestive disturbance is concerned. It seems likely that excess of carbo-hydrate, when continued for several months, has some such slowly produced effect, favouring the ultimate onset of

rickets, probably by interfering in some way with the assimilation of fat.

And this brings me to another very serious fault which is common to many of these trade preparations as given to the infant, namely, deficiency of fat. My own observations support the views put forward by the late Dr. Cheadle, that the chief dietetic factor in the production of rickets is deficiency of fat-assimilation, a deficiency usually dependent upon deficiency of fat in the food, but occasionally—so it appears—due to defective assimilation of the fat which is present in the food. The whole process of fat-assimilation, and the part played in metabolism by fat taken as such in the food, is still very imperfectly known, but there is experimental evidence to show that the assimilation of one food-constituent is influenced by the proportion of other food constituents present in the diet, and there are clinical facts which suggest strongly that excess of carbo-hydrate, whether soluble or insoluble, interferes with the assimilation of fat. Whilst therefore deficiency of fat is in itself a serious fault, the deficiency probably becomes even more harmful when associated with excess of carbo-hydrate, and it seems likely that a proportion of fat, which is just sufficient to prevent disorder of nutrition, so long as no excess of carbo-hydrate is present, may become insufficient, when its assimilation is hindered by an associated excess of carbo-hydrate.

But what constitutes deficiency of fat ? Obviously no hard-and-fast rule can be laid down, but if the occurrence of rickets may be taken as an indication, in most cases, of deficiency of fat in the diet—an assumption which has yet to be proved, but which affords, I think, the most useful working hypothesis—then it would appear that, at three months old, 1·75 per cent. of fat, at six months old 2·25 per cent., and at nine months old 2·5 per cent. of fat, is the minimum which will protect from rickets, and that even these proportions are not sufficient for the purpose if there is associated therewith an excess of sugar or starch.

These proportions I give as the result of clinical observation upon the occurrence of rickets in infants fed upon plain milk-and-water mixtures of various dilutions at different ages : if an infant is fed with equal parts of milk and water at three months old, gradually increased to two parts of milk to one of water at six months, and three parts of milk to one of water at nine months, there is very little risk of rickets, whereas feeding with weaker mixtures often does result in rickets. I have assumed on grounds which I shall explain in a later chapter, that the proportion of

fat in the food is the cause which determines the occurrence or non-occurrence of rickets, and in this way have arrived at these figures as a useful working standard.

These proportions, it must be understood, are not those to be recommended, they are simply the minimum which can be used without harmful results. The proportion of cow's milk fat upon which a healthy baby will usually do best, is about 2·5 per cent. during the first few weeks of life, gradually increased up to about 3 per cent. at three months old : I say advisedly ' cow's milk fat ' because very many infants cannot take as large a proportion of this as they can of the fat which is present in human milk. It is never necessary, and seldom wise, to exceed 3·5 per cent. of fat, more often, I think, a proportion of 3 per cent. will be found sufficient throughout the whole period of bottle-feeding.

But how far do the patent foods fulfil these conditions ? A glance at the published analyses of the various foods which are advertised as substitutes for fresh cow's milk, and which are intended to be used simply diluted with water, will show that not one of them is either in accordance with the standard of human milk, or fulfils the requirements which are necessary if the risk of rickets is to be avoided.

	Fat.	*Carbo-hydrate.*
Condensed Milk (Nestlé's) . .	13·1	54·2
Allenbury No. 1 . . .	16·7	66·6
Allenbury No. 2 . . .	14·9	68·7
Horlick's Malted Milk . .	8·4	63·5
Milo Food	4·4	77·7
Carnrick's Soluble Food . .	2·5	76·2

It is evident that no dilution can so adjust these proportions as to make the percentage both of fat and carbo-hydrate correspond with the standard of human milk: as a matter of fact in nearly all cases as the food is used the fat is deficient, often extremely deficient, and the carbo-hydrate is considerably in excess. Such foods are therefore unsuitable for prolonged use in infant-feeding, but they may nevertheless serve a useful purpose in the cases of severe milk-indigestion, where it is necessary to feed for a short time with some food which yields little or no curd, and which contains a low proportion of fat.

There are cases where, owing to extreme intolerance of fresh milk, especially during the first few weeks of life, a weak solution of carbo-hydrate, even if there is associated therewith a small proportion of starch, seems to be assimilated better than anything else : and in such cases one of these foods diluted rather beyond the maker's directions so as to bring the soluble carbo-

hydrates in it down to a more suitable proportion, will sometimes tide the infant over a period of danger : but it is to be remembered that such feeding amounts to little more than a diet of sugar and water.

There is yet another point which must be remembered in comparing these manufactured products with human milk or fresh cow's milk, namely, that a statement of percentage composition, although it affords some guidance, gives but an incomplete and sometimes misleading description of a food. For instance, two foods may contain a precisely similar percentage of fat and yet differ in their fat-value. Compare a dried milk food with a dilution of fresh cow's milk containing the same percentage of fat : in the latter, the emulsion of fat is extremely fine and the fat is easily assimilable ; in the former, the emulsion is so imperfect that, even before the infant has time to finish his feed, the fat has risen in yellow droplets floating on the surface, a fault which, in some cases, seems to interfere considerably with the assimilation of the fat, part of which indeed is apt to remain coating the sides of the bottle. On the other hand, compare a dried milk food with a dilution of fresh cow's milk in which the percentage of proteid is the same ; the proteid in the former produces so little curd that an infant may assimilate it easily when he is unable to digest the curd of the fresh cow's milk.

A statement of percentage composition, moreover, tells us nothing of the subtle changes, whatever they may be, by which the nutritive value of a food is so impaired in the process of manufacture that if its use be continued for some months the food is liable to set up all those serious symptoms which constitute scurvy and which sometimes end in death. It might be thought that this risk would be limited to those patent foods which are used only with plain water with no addition of fresh milk : but so far from this being the case, my own statistics show that a large proportion of cases of infantile scurvy arise where a patent food is being added to fresh milk, and I believe that in this respect those foods which contain an active digestive ferment, such as Savory & Moore's and Benger's Foods are more risky than others in which there is no such ferment. Even the addition of completely malted cereal, such as Mellin's Food, to milk seems to involve some risk of scurvy, and I have repeatedly seen infantile scurvy, when the chief or only article of diet was one of the starchy foods, such as Ridge's, Neave's, or Frame Food, made with fresh milk, which, however, in most if not in all cases, was boiled.

Condensed milk is liable to cause infantile scurvy, whether used alone or in combination with some patent food. I have notes of at least fifteen cases in which condensed milk with or without some patent food had produced this disease, and one of these cases proved fatal.

There is every reason to suppose that the dried milks which have recently been introduced, will prove to be at least as dangerous in this respect as condensed milk.

Now I am well aware that infantile scurvy is not a common disease, but it is such a cruelly painful affection when it does occur that we may well hesitate to give what might seem to be an indiscriminate sanction to the use of foods which are responsible not only for grievous suffering but actually for death in some cases. Any physician attached to a children's hospital necessarily sees a specially large proportion of the less common diseases of childhood but, making allowance for this, I think that my own experience shows that infantile scurvy, though uncommon, is unfortunately no great rarity. I have notes of sixty-four cases which have been under my own personal observation: five of which died after much suffering. Out of these sixty-four cases no less than fifty-eight were being fed upon patent foods or condensed milk (see Chap. VIII).

Condensed Milk

I turn now to the consideration of condensed milk: one hears sometimes that an infant could not take fresh cow's milk, so it was put on condensed milk, and straightway its digestive troubles mended, and if only the condensed milk had been stopped, after two or three weeks, no harm would have been done; but it has been continued for several months, and now the child has rickets. What has happened? The infant was probably tried at first with a mixture of fresh milk and water, 1 to 2, perhaps, even, equal parts; and he was sick or had colic, so resort was had to condensed milk; a teaspoonful in 4 ounces, or even in 6 or 8 ounces of water. The following figures will show how such mixtures compare with the milk previously tried:

	Condensed Milk.		*Fresh Milk.*
	One teaspoonful to 4 ozs.	One teaspoonful to 6 ozs.	With two-thirds water.
Proteid . .	0·9 per cent.	0·6 per cent.	1·3 per cent.
Fat . . .	1·0 per cent.	0·75 per cent.	1·2 per cent.
Sugar . .	4·5 per cent.	3·2 per cent.	1·3 per cent. (+ added sugar).

It is evident that the condensed milk in these mixtures, which are in common use, is diluted, especially as to the proteid, which

has nearly always been the cause of the difficulty with fresh milk, to a degree which was never tried when the cow's milk was given fresh. I have seen cases in which condensed milk has been given in the strength of a teaspoonful to 8 ounces of water (proteid, 0·45 per cent., fat, 0·5 per cent.) because 'fresh milk would not agree': the fresh milk had never been diluted with more than an equal quantity of water, whereas the dilution of the condensed milk was equivalent, so far as proteid was concerned, to one part of fresh milk with seven parts of water! It would probably be correct to say that in a large proportion of the cases, in which an infant is supposed to be unable to digest fresh cow's milk whereas it can digest condensed milk, the difference depends far less upon any special digestibility of the curd of condensed milk than upon the simple fact that it is diluted to a degree which was never tried with the fresh milk.

In calculating the strength of a condensed milk mixture, it must of course be remembered that a domestic teaspoonful is not a drachm, and that, if the spoon be dipped in the can, there is further allowance to be made for the viscid milk which clings to the undersurface of the spoon. For purposes of calculation, I have ascertained by experiment that if an average teaspoon (capacity when brimful about 2 drachms) be used, by dipping it in the can and taking up as much as can be withdrawn without special care, the condensed milk may be reckoned as approximately ʒiij; with a larger teaspoon (capacity ʒiij) the condensed milk should be reckoned as fully ʒiv; whereas, if the milk is poured from the can into the teaspoon, so that none adheres below, the amount held by the average teaspoon should be considered ʒij. The undiluted condensed milk contains approximately:

Proteid	11 per cent.
Fat	11 per cent.
Sugar	55 per cent.

(In the 'Milkmaid' Brand there is 9 per cent. of fat, in Nestlé's 'Nest' Brand 13·1 per cent., according to my own analyses.)

so that, for instance, with an average teaspoonful to 3 ounces of water, the mixture is 3 drachms to 24 drachms, = 1 to 8 or 1 in 9, that is, proteid 1·2 per cent., fat 1–1·5 per cent., sugar 6 per cent. As a ready way of calculating approximately the strength of a condensed milk mixture, this is, I think, sufficiently accurate to be of practical value.

It is evident that this dilution, one teaspoonful of the condensed milk to 3 ounces of water, reduces the proportion of sugar approximately to that present in human milk, and that any less

dilution would make the sugar excessive. On the other hand any further dilution would make the deficiency of fat even more marked; so that if condensed milk is to be used at all this is the proper degree of dilution of the ordinary sweetened variety.

Such a mixture sometimes proves useful as a temporary food for two or three weeks, where there is much difficulty in digesting fresh milk; the resulting curd from condensed milk is somewhat less firm than that from fresh cow's milk, and intolerance of fat is very common in infants with disordered digestion, so that the low proportion of fat may be no objection to this food for a short period. The prolonged use of the ordinary sweetened condensed milk is a common cause of severe rickets; sometimes, no doubt, owing to excessive dilution of the milk with correspondingly great deficiency of fat; but, as I have shown, however it may be diluted, it is impossible to avoid one or other of the two faults which play an important part in the production of rickets, deficiency of fat and excess of carbohydrate.

Very possibly it might be used for longer periods without harm if cream in proper proportion were added to the mixture, but the very circumstances which most commonly lead to the use of condensed milk, poverty and inability to prepare milk mixtures which require careful measurement of the several constituents, are just those which usually make the use of cream impracticable. If the ordinary fresh 'separated', that is centrifugal cream, which is sold in the shops of London and other large cities in the country, is to be used for this purpose, a carefully measured drachm of this 48 per cent. cream to every 3 ounces of the mixture of condensed milk and water will raise the proportion of fat from 1–1·5 up to 3–3·5 per cent.

Much less open to objection are the various brands of unsweetened condensed milk. In these the milk after sterilization has been evaporated *in vacuo* down to about a third of its volume; no cane sugar has been added and in some cases the proportion of fat in the milk has been slightly increased by the addition of cream before condensation.

The 'Ideal' Brand is stated by the makers to contain:

Proteid	8·3 per cent
Fat	12·4 per cent.
Milk Sugar . . .	16·0 per cent.

The addition of three parts of water to one part of the unsweetened condensed milk will give a mixture containing:

Proteid	2·08 per cent.
Fat	3·1 per cent.
Milk Sugar . . .	4·0 per cent.

If to 3 ounces of such a mixture half an average-sized teaspoonful of milk sugar be added the proportion of sugar will be raised to 6–7 per cent., and so far as percentage composition goes the resulting food would do very well for an infant of three to six months of age. For a younger infant dilution with four or five parts of water would be required, making the proportions as follows :

Unsweetened Condensed Milk. ½ ounce with water 2½ ounces, i.e. 1 to 5.		*Unsweetened Condensed Milk.* ½ ounce to 2 ounces water, i.e. 1 to 4.	
Proteid . .	1·4 per cent.	Proteid . .	1·6 per cent.
Fat . . .	2·0 per cent.	Fat . . .	2·4 per cent.
Milk Sugar . .	2·6 per cent.	Milk Sugar . .	3·0 per cent.

The weaker mixture with the addition of a teaspoonful of milk sugar to every 3 ounces is suitable for an infant in the first month or six weeks, the other with the addition of half a teaspoonful of milk sugar to every 3 ounces is suitable for an infant between the ages of six weeks and three months.

A similar preparation is that made by the Aylesbury Dairy Company under the name of ' Humanoid ', the composition of which is stated to be:

Proteid	4·2 per cent.
Fat	10·2 per cent.
Milk Sugar . . .	17·8 per cent.

In this the proportion of proteid has been made lower, so that without any great deficiency of fat it is possible to give a low proportion of curd and so to overcome the digestive difficulty of some infants. Diluted with water, the resulting mixture will be :

Humanoid 1 part, Water 2 parts.		Humanoid 1 part, Water 3 parts.	
Proteid . .	1·4 per cent.	Proteid . .	1·0 per cent.
Fat . . .	3·4 per cent.	Fat . . .	2·5 per cent.
Milk Sugar . .	5·9 per cent.	Milk Sugar . .	4·4 per cent.

It is evident that such a preparation, if it can be used with the less dilution, one part of the ' Humanoid ' to two of water, is not even open to the objection that there is any deficiency of fat or excess of sugar, the proportions in fact of proteid, fat, and sugar are theoretically excellent. In this respect it is superior to the ordinary brands of unsweetened condensed milk; its cost however is greater, and it is therefore hardly within the reach of the poorer classes, who indeed comparatively rarely use any preparation of unsweetened condensed milk owing to

its being slightly more costly than the sweetened brands. The difference in cost depends not only on the actual price per tin, but on the fact that, 'owing to the greater dilution required,' less of the sweetened condensed milk is used, and when each tin is opened the milk in it will remain fit for use much longer, owing to the preservative effect of the cane sugar, than will the unsweetened milk, which must be used within thirty-six hours.

The curd of condensed milk, whether sweetened or unsweetened, appears to be just as large as that of fresh cow's milk when an acid is added, but it is certainly digested more easily by an infant; and I have seen nutrition improved by the use of this unsweetened condensed milk, where other much weaker modifications of fresh milk had failed. But none the less I am averse to its continued use, for there is clear evidence that condensed milk, whether from the boiling or sterilization which forms part of the process of manufacture or whether from the condensation *in vacuo*, is capable of producing infantile scurvy.

One reason assigned for the use of condensed milk and patent foods is their freedom from bacterial contamination, and therefore supposed virtue in the prevention of summer diarrhœa. Now I venture to doubt whether this argument is really sound: with regard to the ordinary sweetened condensed milk my own observations seemed to show that exactly the opposite was the case, for whilst it was found that 12 per cent. of infants attending hospital for ailments of all sorts, were being fed on condensed milk, it was shown that 25·8 per cent. of infants dying of diarrhœa had been fed on condensed milk. It does not seem reasonable to suppose that this association is the result of any special infection from condensed milk; it is far more likely that an unhealthy condition of intestine induced by excess of sugar in the food, or by the fat starvation which the use of condensed milk so often involves, makes a favourable soil for the multiplication of bacteria reaching the intestine from other sources. In the same way, I believe that other patent foods, partly by the excess of carbohydrate which they contain, and partly by their deficiency in fat, actually pre-dispose to those infective conditions which are known as 'Summer Diarrhœa'.

It may be doubted whether, apart from breast-feeding, there is any safer food in this respect than ordinary fresh milk, properly modified and pasteurized or scalded; I suspect that in the prophylaxis of infantile diarrhœa we are apt to lay too much stress upon the possible introduction of the bacteria of diarrhœa by fresh milk and too little stress upon the danger of diminishing

the resisting power of the intestine by feeding with unsuitable food which by its excess of carbohydrate supplies the material for fermentation, and by its deficiency of fat may favour the general tendency to catarrhal conditions of mucous membrane which we know to be one feature of rickets.

Lastly, I would point out that upon the medical profession there rests no small responsibility in the use and abuse of condensed milk and patent foods for infants. Useful as most of these may occasionally be, the lay public, even if not misled by advertisements, has not the requisite knowledge to discriminate between the different conditions under which different foods may safely be used in infant-feeding. The doctor's orders, unless most carefully guarded and hedged about with cautions for each particular case, are apt to be taken as a general sanction for the particular food ordered. We may well, therefore, be careful in our advice, lest we should seem to countenance the indiscriminate use of these trade-preparations, and thereby add to the deplorable suffering which such ignorant use brings upon infants. Of this I feel sure, that the more the medical man knows of the many simple methods of adapting fresh milk to the needs of the infant, the less use he will find for condensed milk or patent foods.

CHAPTER VI

COMMON FAULTS AND FALLACIES IN INFANT-FEEDING

The first fault to which I would draw attention is neither in the food nor in the manner of feeding, but in the personnel which directs the feeding: why is it that one hears so often from the present-day monthly nurse, such expressions as this: 'I always feed my babies with such and such a mixture,' or 'with such and such amounts at each feed'? or again, why do we hear the modern domestic nurse, especially in the upper classes, talking of the feeding of an infant as if she forsooth with less than a smattering of knowledge of the very rudiments of infant-feeding were a competent person to direct the feeding of a baby? I am afraid that in many such instances the fault lies with the medical man: he has attended the confinement, he has taken admirable care of the mother; but has left the details of feeding the infant to the monthly nurse, or perhaps if artificial feeding is inevitable he has said, 'Oh, try him with some milk and water'; leaving the nurse to decide on the proportions, and to add sugar and possibly cream on her own responsibility. Naturally enough when the food disagrees, the nurse, seeing that the doctor apparently attaches so little importance to details, begins to modify the feeding on her own account, and what is the result? First and most important, that the infant suffers; secondly, that after the nurse has already done mischief by her 'experienced' inexperience, off go the parents to a 'specialist', probably without even mentioning it to their family doctor; and it is not much to be wondered at, if they say, 'Doctor So-and-so is very good for ladies, but he takes no interest in babies.' If the medical man who attends the confinement would give detailed directions as to the feeding of the infant, and see to it that as long as he was attending no nurse, whether trained or untrained, presumed to alter the feeding except in accordance with his directions, he would do good service to the infant and at the same time would prevent such very natural criticism as that to which I have referred.

And here let me emphasize the importance of detail in

direction ; to tell a mother to give her child equal parts of milk and water may be admirable advice as far as it goes ; but if the mother or the nurse is to guess what quantity is to be given, and how often, and whether sugar is to be added and if so how much, the result of the doctor's advice or rather of the defectiveness of his advice may be, and often is, disastrous.

Next I wish to mention a fallacy which I venture to think misleads not only the laity but also many medical men in infant-feeding : namely that because an infant is gaining weight and has neither sickness nor diarrhœa on a particular food, therefore this food is a good one. The proof of the pudding is not only in the eating, it is also in the after-results, and in the case of infant-feeding this result may not be apparent for months. I may mention as a well-marked instance of this, the fact recently pointed out by Dr. Holt that an infant fed with a mixture of milk and water and cream, containing too high a proportion of fat, say 5–7 per cent., may appear to thrive upon it almost extravagantly, and may be for a time fat and jolly, but sooner or later, perhaps not till the child is more than six months old, the evil effects of the excess of fat in the food begin to appear, and marasmus, rickets or some troublesome digestive disorder is the outcome of what seemed to suit so excellently : take again as a much less-marked instance the common mixtures of milk and barley-water; everything goes smoothly enough apparently, but how often one finds when the infant has reached nine or ten months old, that there is very slight but definite rickets, the proportion of fat has been lower than it should be, and starch has been given in the form of barley-water long before a child should have any. I mention these particular instances because they are faults which are more easily overlooked and are more often ignored than such generally recognized faults as occur in the condensed milk, which may be excellently digested and may increase the infant's weight steadily but ultimately produces a more or less marked degree of rickets, or the patent food—Allen & Hanbury's, Benger's, or Savory & Moore's, or what you will—which agrees so well but ultimately may produce scurvy.

And this brings me to the mention of one particular fault which like those to which I have already referred may not produce any apparent ill effect until it has been continued for many weeks or even months,—I mean feeding with too large a bulk of fluid. This is I believe quite one of the commonest faults in infant-feeding in all classes.

I see many infants with some such history as this : at the age of six weeks the infant was having 3 ounces at a feed, at two months $3\frac{1}{2}$ or 4 ounces, and by the time he is three months old he is having 6 or even 7 ounces.

For a time the infant appears to be doing perfectly well : he is gaining weight not only steadily but rapidly and perhaps the most punctilious of theorists could not find a flaw in the proportions of the milk-mixture ; there may perhaps be some slight posseting, but what of that ? and so it goes on until the infant is some three or four months old : and then the trouble begins, the child ceases to gain weight, then begins to lose definitely, he begins to vomit, not severely but enough to make the marasmus worse ; and soon the infant is a wasted child with no organic disease to be found but with a stomach so intolerant that it rejects this food and that ; and the wasting continues until at last in despair the doctor does what ought to have been done months before, cuts down the size of the feed : but unfortunately the stomach long abused is not to be appeased by this tardy diminution of the feed to the proper amount for the age, it is often necessary to give much smaller and weaker feeds than would be suitable for a healthy infant of the same age, and even then there are often weeks and weeks of trouble before the child begins to assimilate food.

If one inquires why such large feeds were given, one is told that the infant seemed hungry ; and that as the stools were well digested and the weight was progressing so rapidly, the feeds were increased in size.

It is obvious that Nature's method is to allow an infant to suck until he is satisfied, but the hand-fed infant is not fed on Nature's food, and experience shows that with substitute feeding, however carefully we may strive to imitate human milk, it is necessary to restrict the quantity of each feed within certain limits. But says the mother, ' the infant is not satisfied, surely we must give a larger feed ? ' Yes, but only within those limits ; if the proper bulk of feed for the age has been reached and the infant is still not satisfied, increase the strength of the feed, not its size. By increasing the proportion of milk or cream it is often possible to satisfy the appetite of the infant without increasing the amount of the feed at all, and by so avoiding excessive bulk of feed, one may avoid troublesome digestive disturbance.

Of course the amount of the feed has to be increased gradually as the infant grows older, and some big infants will require more

than a smaller infant of the same age; but on the whole it is possible to lay down simple rules as to the amounts of the feeds. During the first week the feeds should be 1½ ounces, at the second week 2 ounces; at the end of the sixth week 2½ ounces; at three months 3 ounces; and from this time up to the end of the eighth month the food should be at the rate of 1 ounce for every month of age: i.e. 4 ounces at four months, 5 ounces at five months, and so on. For an exceptionally large infant an additional ounce may be allowed, i.e. 3 + 1 ounces at three months, 4 + 1 ounces at four months, 5 + 1 ounces at five months, and so on. In giving these directions as to the amounts I am assuming that the proper intervals of feeding are observed, namely, two-hourly up to the age of two months, and three-hourly from the age of three months up to the end of the ninth month. But here I would add a caution: 'Nature does nothing by leaps,' and he will be most successful in infant-feeding who makes every change whether of amount or interval gradually.

Connected with this fault of excessive bulk is a fallacy which I have sometimes heard seriously propounded, that it makes no difference whether one gives a food containing a lower or higher proportion of proteid or fat, provided the infant takes more, the result is the same; for instance, if an infant has 4 ounces of a mixture containing 1·5 per cent. of fat, this is just as good as 2 ounces containing 3 per cent. To this I would reply that undoubtedly to a certain extent such compensation is possible, but only to a very limited extent; directly the bulk of the food is increased beyond what experience shows to be the proper amount for the age, there is a risk of slowly producing the chronic feebleness of digestion which I have already mentioned as arising from excessive bulk of feeds.

The next point to which I turn is the so-called 'nursery milk'. Parents often tell me that they are having nursery milk for their infants, and they say it with an air of satisfaction, as if 'nursery milk' were the *ultima Thule* of perfection. Now I would warn doctors and parents to beware of 'nursery milk'. The milkman apparently imagines that a milk which from his point of view is an exceptionally good one, namely, an exceptionally rich one, is therefore particularly suited to the infant, who, as he knows, requires 'good milk', so he either puts aside to be sold at a special price the milk of a cow which has recently calved, and which therefore is specially rich, or having some Jersey cows he reserves their milk to be sold for nursery use while that of the ordinary Shorthorn

is supplied to the ordinary customer; in some cases he sells as 'nursery milk' strippings, that is to say, the milk obtained from a cow by re-milking a few minutes after the ordinary milking has been completed. However it is obtained, some of these so-called nursery milks contain so high a percentage of fat that they are practically a weak cream. Some time ago I had an infant brought to me because he was doing badly and the food did not agree. I found he was on nursery milk, and I ascertained by analysis that this milk contained 12 per cent. of fat: in another similar case, the nursery milk contained about 6 per cent. of fat. The most suitable milk for infant-feeding is the milk of a mixed herd of Shorthorn cattle, such as is supplied for ordinary uses; nursery milk is best avoided altogether, unless an analysis can be obtained and the particular dairyman's interpretation of 'nursery milk' is known. Obviously, when the doctor is having such rich milk diluted as if it were ordinary milk, it is likely enough that trouble will ensue.

But nursery milk does not always mean a specially rich milk; sometimes, indeed, it is difficult to guess on what grounds it is supposed to be particularly suitable for the nursery; the only appreciable difference in some instances being that it is particularly poor milk for which a particularly high price is charged; for example, in 'nursery milk' for which an extra charge was made to a poor woman who could ill afford it, the proportion of fat was 2·1 per cent. One dairyman who supplied 'nursery milk' for which he charged a specially high price admitted that the only special feature in it was that it had been pasteurized, whereas the milk sold at the ordinary price was fresh milk. In the case of one infant who was under my care, and was being fed on special 'nursery milk' the ingenuous dairyman explained to the mother that the difference in the nursery milk and the ordinary milk was that he put colouring matter in ordinary milk whereas 'nursery milk' was not coloured. Some recent observations on 'nursery milk', obtained from some of the best-known dairy companies in London, have shown that so far from being specially pure and clean, it was dirtier and more contaminated than the ordinary milk. In some instances it contained from two to five times as many bacteria as the ordinary milk, obtained at the same time from the same dairy company.

I have repeatedly known difficulty to arise where the milk of Jersey cows was being used, without due allowance for its extra richness. A comparison between this milk and that of the ordinary Shorthorn, which is the cow in common use for dairy

purposes in England, will show how considerably this may affect the composition of the food

Ordinary Shorthorn's milk.		*Jersey cow's milk.*	
Proteid . .	4·0 per cent.	Proteid . .	4·5 per cent.
Fat . . .	3·5 per cent.	Fat . . .	5·2 per cent.
Sugar . . .	4·0 per cent.	Sugar . . .	4·0 per cent.

Now if this Jersey milk be treated as if it were of the same composition as the ordinary Shorthorn milk, and a drachm of ordinary cream (48 per cent.) is added to a three-ounce feed of equal parts of milk and water, the resulting fat percentage, instead of being approximately 3·75 per cent., will be 4·6 per cent., a proportion which is more than many infants can tolerate. If cream is not available and the only modification of milk which is practicable is simple dilution and addition of sugar, undoubtedly there is some advantage in using a milk in which the fat is relatively more abundant than the proteid, as in that from Jersey cows, for the dilution required will result in less deficiency of fat than it would with other milk; but if cream can be added there is no advantage in using a specially rich milk; the only point of practical importance, so far as the composition of the milk is concerned, is that we should know the strength of the milk, and this we can know just as well with ordinary Shorthorn milk as with a Jersey cow's milk: if we must add cream, it is just as easy to rectify a small deficiency as a large deficiency of fat in the diluted milk.

The last point of which I wish to speak is the vagueness with which cream is often ordered. I ask, 'What is being given?' and I am told, 'Milk and water with some cream, a couple of teaspoonfuls of cream in a three-ounce feed.'

'But what strength cream is being used?' 'It is very good cream from a very reliable dairy,' I am told, and when I point out that this is not sufficient, we must have some idea of its strength, people are sometimes quite vexed that I should insist upon such minutiæ; as who should say, we bought this arsenic mixture from a good chemist, he supplies excellent drugs, why worry about the quantity of Liq. Arsenicalis in each drachm? Such an attitude with reference to a potent drug would be absurd; but cream, be it remembered, if given in excess is a potent irritant, and may set up diarrhœa or vomiting, or if given in slighter excess for a long period may produce more chronic disturbance of digestion as I have already mentioned. It is very necessary, therefore, that the strength of the cream should be taken into account if cream is to be used at all. I have considered the composition of cream according to the method of its preparation

in a previous chapter, here I will only reiterate the practical conclusions to which my own observations have led me : first, that gravity cream, or skimmed cream, may safely be regarded as containing on the average 24 per cent. of fat, and varies so much in its strength that it is very unsatisfactory for infant-feeding; secondly, that ' separated ' cream, that is, cream made by a centrifugal machine or ' Separator ' (as almost all the cream sold in London and other large towns is) should be reckoned as containing 48 per cent. of fat.

On several occasions when the need for ordering cream according to its strength has been realized, and I have asked what cream is being used, I have been told, ' The usual 20 per cent. cream.' Now, if there is one thing which does not exist in this country as a regular article of commerce it is a 20 per cent. cream, but there is a widespread notion that this is the usual strength of cream, and this notion has arisen, I fancy, entirely from the writing of American authors, who are able to buy a 20 per cent. cream. In London, certainly, unless a standardized cream of that strength is made to special order, it may be taken for granted that the cream is not a 20 per cent. cream, and if cream is bought as fresh cream from any milk-shop, the strength may safely be assumed as approximately 48 per cent.

In ordering cream I would advise every medical man to think in percentages, and always to keep the fat percentage of the feeds rather below the standard of human milk, in which the fat is 3·5 per cent. It is easy to exceed the proportion which an infant will tolerate ; I think it will be found that 3 per cent. is quite as much as many an infant can bear, and some will do better with slightly less. When this limit is exceeded some infants will suffer with vomiting or looseness of bowels.

' But,' says one, ' the mother's milk contains 3·5 to 4 per cent. of fat, surely the child ought to be able to stand more fat.' Yes ; perhaps he should, on theoretical grounds, if only fat 3·5 to 4 per cent. always meant the same thing ; but here comes in one of the fallacies not only of the so-called ' percentage-feeding ', but also of those specious advertisements which prove conclusively that the percentage composition of some patent food corresponds exactly with that of mother's milk. It is not merely the percentage or proportion of fat that has to be considered ; the fat in two different foods may be identical in percentage, but may differ widely in its digestibility and nutritive value, the difference being due sometimes to subtle physical differences, sometimes

to equally subtle chemical differences. And these remarks apply even to the ordinary milk or cream and water mixtures: the fat globules of cow's milk, even before it is altered by heating and watering, differ in size from those of human milk, the cow's milk fat has also a higher melting point than the fat of mother's milk; moreover, in diluting cow's milk with water, we disturb the emulsion of the fat to such a degree that its digestibility may well be affected. But however this may be, it is to be remembered that in the making of cream the fat of milk is thrown out of its emulsion almost altogether; and although we may mix it with water or milk subsequently it is very unlikely that we can restore the fat to a state of emulsion anything like so perfect as exists in natural milk; and this is, I think, one reason why infants will only tolerate a very moderate proportion of fat given in the form of cream.

But there may be other reasons: a very large proportion of commercial cream contains boric acid used as a preservative, and this has been thought to disagree in some cases; moreover, commercial cream sold as fresh cream, is often so stale that the ordinary preservatives may not suffice to prevent the growth of harmful bacteria in it; in a recent case in which a dairyman was being prosecuted for adding starch to thicken cream, a witness admitted that it was very usual in the trade to keep cream four days in stock.

Lastly, it must be pointed out that cream may be just as dangerous as milk in conveying tuberculous infection. One often finds that a child is having all milk carefully boiled whilst cream is being given freely without any precautions.

To some extent this risk depends upon the method of preparation. It has been shown by experiment that cream prepared by a separator contains a small number of bacteria of any sort, compared with milk, but if the milk from which it was made was tuberculous, some tubercle bacilli remain in the cream. Cream made by gravity, i. e. as top milk, will show a much larger number of bacteria. It is only when cream has been subjected to a high temperature, either by pasteurizing or boiling, as can easily be done in any milk mixture to which it is added, or when it has been prepared by the old-fashioned method of scalding in pans, which has been customary in Devon and Cornwall, that it may be considered free from this risk.

CHAPTER VII

RICKETS

Rickets is one of the commonest disorders of early life amongst the poorer population of large cities, nor is it very rare in its slighter manifestations amongst the well-to-do, and yet there are few diseases which are more easily preventable. Undoubtedly there are problems in its causation which we cannot solve at present, but enough is known to enable us to reduce its occurrence almost if not quite to a vanishing-point, if only this knowledge could be diffused and carried into effect amongst the poorer classes.

My own statistics showed that of children in London under the age of three years who were brought to hospital for various diseases, 44·6 per cent. showed rickets, while Dr. John Thomson in Edinburgh, amongst children of the same age, found that 'rather more than 50 per cent.' were affected by this disease. In Manchester, Dr. Ritchie found that 30·3 per cent. of 'sick children' (he does not state their ages) had rickets: figures taken in several large cities in America and in various parts of Europe, show that the proportion of rickety children amongst hospital out-patients is, generally speaking, from 30 to 80 per cent.

It is commonly supposed that rickets is less frequent in rural districts than in large towns; this seems probable, but I know of no statistics that prove it; it is quite certain that rickets is by no means uncommon in the country, and I would lay stress upon this fact, for some have pointed to such causes as overcrowding and lack of sunlight and defective sanitation as causes of rickets, whereas clinical evidence seems to show that rickets has no necessary connexion with environment.

An infant whose feeding is suitable does not get rickets, though he live in the worst of London slums, and rickets is treated daily amongst the out-patients of a children's hospital with no change whatever in the child's environment, and yet correction of the diet and the addition of cod-liver oil produces rapid improvement.

For practical purposes I think it cannot be too emphatically laid down that rickets is a food disorder; it means faulty feeding or faulty assimilation; and wherever poverty or ignorance leads to the use of improper food for infants, there rickets will be pre-

valent. In crowded cities and factory towns where the struggle for existence is great, and where poverty abounds, and the mother as well as the father must go out to work, the infant is likely to be weaned too early, and to be fed on improper food, and on cheap substitutes for fresh milk such as condensed milk; it is in this way that rickets becomes a disease of the cities rather than of the country. Even amongst the well-to-do, the stress of town life exercises an injurious influence on infant-feeding, for it makes women less capable of suckling their infants and so necessitates hand-feeding with its attendant risks, one of which is rickets.

Dietetic causes. Is it possible to determine the particular fault in diet which underlies the occurrence of rickets? A comparison of the various diets upon which rickets arises gives, I think, some guidance; they may be grouped as follows, in the order of their frequency in the causation of this disease:

(1) Starch-containing foods, e.g. potato, biscuits, bread and butter, sopped bread, cornflour, with more or less milk.

(2) Condensed milk, usually excessively diluted.

(3) Patent foods, whether containing starch or not, and whether made with or without fresh milk.

(4) Cow's milk excessively diluted without addition of cream.

(5) Breast-milk with addition of starch-containing foods.

(6) Breast-milk alone; when the milk is thin.

Upon every one of these diets rickets is known to occur; and clearly if there is one particular fault which induces rickets, it must be common to all these methods of feeding.

Starch-containing food has often been blamed, but it cannot be the essential factor, for in groups (2), (4), and (6) there is no starch, and some of the worst degrees of rickets occur on excessively diluted condensed milk.

Deficiency of proteid has been suggested, but the child who becomes rickety, as many infants do, because cow's milk is being given diluted with an equal quantity of water when the child is eight or nine months old, is having an amount of proteid fully up to the normal if the proportion in human milk may be taken as a standard.

Excess of sugar is certainly not common to all the diets; indeed in the very one in which it is usually supposed to give trouble, namely, where condensed milk is used, the occurrence of rickets almost always goes with excessive dilution of the condensed milk so that the sugar is actually below the normal proportion, sometimes only 3 to 4 per cent. instead of the normal 7 per cent.

Deficiency of lime salts cannot account for rickets where cow's milk is used as the only food, diluted say half and half at eight months, for the proportion of lime salts in cow's milk is, according to some observers, about three times, according to others about six times, as large as in human milk.

The occurrence of rickets upon diets (2), (4), and (6), suggests that the fault is one of defect; it is a negative rather than a positive fault, and as neither deficiency of proteid nor of lime salts, nor of sugar seems to be in question, we are almost driven to the conclusion that deficiency of fat is the fault in point.

Deficiency of fat is certainly common to diets (2), (4), and (6); it is usual in diets (1) and (3), but what about diet (5), in which breast-milk, often of good quality, is given with addition of starch foods? this appears at first sight opposed to the fat deficiency, but here I would point out that wherever assimilation is hindered by unsuitable feeding, a child may be starved of this or that food constituent just as effectually as if it were withheld altogether; the fact that a diet contains a sufficiency of fat will not prevent fat-starvation if at the same time the child's power of assimilation is diminished by unsuitability of the food in other respects.

If a child is given starch at an age at which starch cannot be properly digested, or is given starch at any age in excess, the power of assimilation for food in general, including fat, may be diminished, and in this way rickets may arise from fat-starvation on any of the diets in which starch is given in addition to milk, even though the milk given may contain a sufficiency of fat.

As a matter of fact where potato, &c., or a patent food is being given in addition to milk, one usually finds amongst the poorer classes that the milk is also being diluted too much for the age; and where a patent food is used without milk, it is almost always deficient in fat, though some of the least objectionable of the patent foods contain just enough fat to prevent rickets if the child's digestion is good enough to ensure assimilation of the fat that is present.

The fault which disturbs digestion, and so prevents assimilation of fat need not necessarily be starch, or excess of starch; it may be excess of soluble carbo-hydrates, sugar, dextrin, &c., which are particularly liable to upset the digestion in some infants. Where the proportion of fat should be just sufficient to protect from rickets, the addition of an excess of soluble carbo-hydrate, such as is present in many of the starch-free patent foods, may be sufficient in this way to cause fat-starvation, and so to induce rickets.

In rare cases the fault which disturbs digestion and causes fat-starvation is excess of fat itself; if cream is added to a milk mixture so that it contains 6 or 7 per cent. of fat, an infant may gain weight well for a time, but soon the digestion may suffer, and rickets may occur for exactly the same reason as in cases where starch is given, namely, because the resulting disorder of digestion prevents assimilation of the fat in the food.

Fat-starvation as the cause of rickets accords well with the clinical facts so far as the diet preceding the onset of rickets is concerned; but there is other evidence which gives strong confirmation to this view. Mr. Bland-Sutton's observations upon animals in the Zoological Gardens showed that young monkeys, bears, and lions suffered severely with rickets when fed upon diets in which fat was deficient, but directly milk, cod-liver oil, and pounded bone were added to the diet, they recovered from their rickets. It might be objected that the recovery was due to the lime salts of the pounded bone, but when this treatment of the animals is compared with the routine treatment of rickets in children by the administration of cod-liver oil, it seems almost certain that the effective element in these therapeutics was the fat of the milk and cod-liver oil.

There can hardly be stronger evidence of the correctness of the fat-starvation theory than the treatment which prevails at most English hospitals, of administering cod-liver oil for rickets: moreover, experience at the Children's Hospital, Great Ormond Street, has shown that any oil is equally effective provided it can be taken without disturbing digestion. It is noteworthy also that where phosphorus is used for the treatment of rickets it is usually given in oil.

I am well aware that occasionally cases occur in which it is difficult to explain the occurrence of rickets upon the theory of fat-starvation, or indeed upon any theory, but after inquiring carefully into the methods of feeding in a very large number of cases of rickets, I am satisfied that no other theory is so generally applicable.

No doubt there may be other contributing factors; for instance, the syphilitic infant is, I think, specially prone to develop rickets, and it may be that any condition which diminishes the vigour of assimilation—whether it be ill health from lack of fresh air, or a feeble constitution because the infant was born of a very young or weakly mother—may favour the onset of rickets, but as a working hypothesis I think it may safely be held that *rickets*

means deficiency of fat-assimilation, whether the defect be in the food or in the assimilation.

If this were more generally realized, some of the common mistakes in infant-feeding might be avoided. There is one which I must mention here, for I have met with it frequently : I find that an infant is having lime-water in his milk, and that this is being given to prevent or to cure the rickets. There is no evidence that lime-water has any effect whatever either in preventing or in curing rickets ; indeed, I think that both on clinical and on experimental grounds it may be confidently stated that it has no value for either purpose.

Age-incidence. There is one other important practical point which I wish to emphasize in connexion with the etiology of rickets, namely, its age-incidence : I quote the following figures from the article on Rickets in Osler's *Modern Medicine*. They were taken from consecutive patients under the age of three years in my out-patient clinic :

	Birth to 3 *months.*	*Months* 3–6.	*Months* 6–12.	*Years* 1–2.	*Years* 2–3.
Rickets	10	10	24	38	18
No Rickets . .	31	24	22	27	20

These statistics do not show the age at which the disease began, for this it is impossible to determine in a disease of such insidious onset, but they show that the proportion of children who have rickets is larger in the second year of life than at any time in the first year, a fact which suggests, what I believe to be true, that the onset of rickets is often not until the end of the first year, if not in the second year. The bearing of this upon the feeding in infancy (infancy I use here, as elsewhere, to mean the first two years of life) is important ; the fact that the infant has been having nothing but breast-milk for nine or ten months, may not safeguard him from rickets if the feeding after this age is faulty either in excess of starch, or in deficiency of fat. Mothers are apt to think that when an infant has been weaned at nine months, he can have starchy food, bread and butter, sops, &c., at every meal, and that it matters little how much milk is taken ; whereas, for the prevention of rickets it is most important that up to the end of the second year, milk should form a large part of the diet, and that the fat should be increased by giving the yolk of an egg daily ; of course, at this age, much more starch can be taken than in the first year, for it can be digested, and therefore, in moderation, does not interfere with assimilation of fats.

Symptoms

The characteristic symptoms of rickets are familiar enough: the fretfulness at onset, with disturbed sleep, in which the child likes to throw the bedclothes off and lie uncovered, sweating about the head during sleep, the lateness of dentition, the ' beading ' of the ribs, the epiphysial thickening at the ends of the long bones, especially the radius, the large protuberant abdomen, the reluctance or inability to stand which makes the child late in learning to walk, or if he has already learnt makes him 'go off his legs ', the late closure of the fontanelle, the softness of bones and ligaments which makes the bones bend and the joints yield, with resulting ' bandy-legs', knock-knee, or other deformity, the large size of the head with its square shape, the stooping curve of the spine, which is only one manifestation of the general muscular weakness, and with all these the tendency to convulsive disorders and to catarrh of the respiratory and alimentary tract.

Amongst these symptoms no doubt those which affect the bones are the most prominent, but let it be realized that rickets is not merely a disease of the bony structures. One of the greatest obstacles to the right understanding of rickets is the conception of this disease as merely an affection of bone and cartilage. Rickets is a general disturbance of metabolism, and its effects are not limited to any one tissue of the organism.

Such apparently disconnected phenomena as a tendency to catarrh of mucous membranes, muscular weakness, and increased nervous instability must be recognized no less than the characteristic bone change, as manifestations of rickets. This recognition becomes a matter of considerable practical importance in some cases, for it happens not very rarely that one or other of these manifestations altogether overshadows the bone changes, and if we fail to recognize rickets as the cause of the trouble in these cases, we fail to recognize an important indication for treatment. I recall cases in which a child of eighteen months or two years has been brought to me with limbs so feeble and weak that even to a medical man the possibility of infantile paralysis had suggested itself, but the key to the condition was to be found in other indications of rickets, such as delayed dentition and delayed closure of fontanelle and slight beading of the ribs, symptoms which were so slight that the possibility of so much muscular weakness being due to rickets, where the osseous changes were so slight, had hardly been considered.

In some cases, similarly, severe nervous symptoms such as convulsions, and laryngismus stridulus, are the result of rickets where bony manifestations are so slight as only to be detected by careful examination.

It would seem indeed that not only may rickets affect several tissues but the stress of rickets may fall upon particular tissues in particular cases; just as tuberculosis in one case attacks especially serous membranes, in another bones or joints, and in a third lymphatic glands, while other tissues escape, or are only slightly infected. Tissue proclivity is a vague and shadowy subject at present; but there is good reason for believing that not only individuals but also families may manifest a proclivity of certain tissues to disease, so that not only in one child, but in several children of a family the stress of a discase falls on a certain tissue. A child was brought to my out-patient clinic with tuberculous meningitis, making the fourth child in that family who died of that particular form of tuberculosis; Dr. J. Thomson has published instances in which congenital syphilis in the same way showed a selective incidence on certain tissues in certain families, and the late Dr. Finlayson of Glasgow drew attention to the occurrence of hypertrophic cirrhosis in several members of the same family. I have seen instances which suggested strongly that the same holds good of rickets, and that tissue proclivity both in the individual and in the family may determine that in one case the muscular, in another case the osseous, and in a third perhaps the nervous symptoms may predominate.

Passing now from theory to fact, I shall consider some of the symptoms of rickets: an infant with this disease is often fretful; but is rickets ever painful? The question is one of practical importance in reference to the detection of infantile scurvy which usually occurs in an infant who already has rickets; the pain and tenderness of scurvy is too often attributed to rickets; as far as my own observation goes, rickets never produces any acute pain or tenderness such as is seen in scurvy; I fancy that it does rarely produce some very slight degree of tenderness, so that the child cries if the limbs are handled roughly; but certainly the presence of any considerable degree of tenderness should suggest, not rickets, but scurvy.

Delay of dentition is a very frequent symptom, and one of considerable value in diagnosis; it was noted in 76 per cent. of my cases. The first tooth may not make its appearance until the age of eighteen months, but it is exceptional to be as late as this. The normal variability in the date of dentition is to be remem-

bered (see Chapter I) ; the laity nowadays have often a smattering of knowledge of the indications of rickets, and if there be no teeth at the age of nine or ten months, an anxious mother is very ready to conclude that her child has rickets ; whereas late dentition alone is quite insufficient evidence.

Whether rickets plays any part in the production of dental decay is disputed ; it is pointed out that the calcification of the temporary teeth is so far advanced at birth that it is unlikely rickets could exert any influence upon their formation; but the permanent teeth whose calcification takes place almost entirely during the first year might suffer; it would be interesting to know

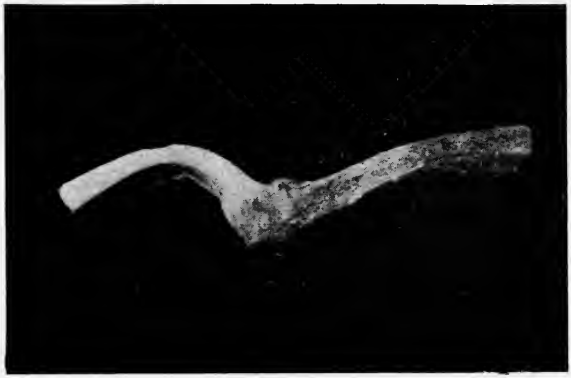

Fig. 1. Costochondral displacement in severe rickets. Section through costochondral junction, showing formation of 'internal bead' (the lower edge in the figure is the internal surface) by dislocation of the bony rib so that it joins the costal cartilage almost at a right angle instead of end to end. The specimen shows also rachitic thickening and irregularity of epiphysial line. From patient in Fig. 5.

what proportion of the very large number of children with caries of the permanent teeth have suffered with rickets in the first year of life.

Even if the formation of the milk teeth cannot be influenced by rickets it is still possible that the unhealthy condition of the saliva from the improper and indigestible food which causes rickets may favour the onset of caries. Some such explanation seems to me necessary to explain the early decay of the teeth which is common in rickets.

Beading of the ribs, the rickety rosary, is one of the most constant symptoms of rickets, though, as I shall point out, a just palpable bossing at the juncture of rib and cartilage is not neces-

sarily rachitic. The beading is best marked in the fifth, sixth, and seventh ribs, and at post mortem examination is seen to be more marked on the inner than on the outer surface.

There is also a spurious beading on the internal surface which is entirely different in its formation, as can be seen on cross sections. It is produced by a partial or complete dislocation backwards of the bony rib, so that instead of joining the cartilage end to end, it meets it at an angle (see Fig. 1). In these cases there is often no beading to be felt externally, although there is a very large ' spurious bead ' internally and although the rachitic change is present in extreme degree.

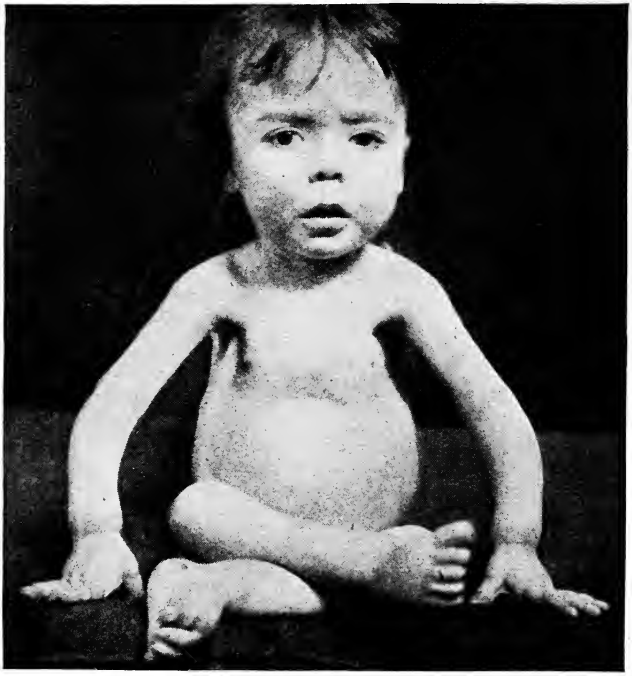

FIG. 2. Tailor position in rickets: forearms bent by partially supporting weight of trunk.

The so-called ' posterior beads ' are only found in severe cases, and are really projections due to green-stick fractures which take place near the posterior angles of the ribs.

The thickening of the epiphysial junctions, which is most noticeable usually at the lower end of the radius and the tibia is due, like the beading of the ribs, to excessive and irregular proliferation of cartilage cells and irregular ossification with increased vascularity producing a lengthening and widening of the epiphy-

sial line, which is felt clinically as the epiphysial enlargement near the ends of the bones. This is not limited to the long bones; occasionally it is well marked on the scapula at the epiphysial line just above the lower angle.

It is remarkable that a change so pronounced at the site where the growth in length of the bone mainly occurs, should affect the stature so little as it does; but stunted growth is certainly to be reckoned among the results of rickets. Actual dwarfing, which fortunately is of rare occurrence, is usually associated with bending or distortion of the bones; but I have often seen ill-grown children whose stunted growth I believed to be attributable to rickets, although no bending of the bones was present; and I have little doubt that a slight degree of rickets, which in other respects may have little effect upon the child's future, may, by the stunting of growth which it causes, be a lifelong handicap.

The gross deformities of rickets are in large measure preventable if the disease is recognized early and proper precautions are taken; the two chief changes are bending of bones and green-stick fractures. There are two factors in their production: (1) the softness of the bones, which is due partly to the relative poorness of the newly formed bone in lime salts, and partly to the rarefaction of the older bone; (2) mechanical influences, particularly pressure and traction, the former in supporting the weight of the body, the latter from the action of muscles or the dragging of an unsupported limb.

Bending of the bones of the upper limb, especially of the forearms, occurs chiefly when the child uses its arms to support its weight in crawling or in sitting tailorwise. This posture, shown in Fig. 2, is often assumed by children with severe rickets, and the forearms are used to assist the weak spinal muscles in supporting the trunk in the erect position.

The common deformity of the clavicle, which is a sharp projection upwards and forwards at the junction of the inner and middle third, is in part due to muscular traction, but in part to the weight of the arm dragging downwards the outer part; similarly the forward curving of the femur is due largely to the weight of the leg dragging upon the lower end of the femur as the child sits on its mother's arm or lap. The common curve of the tibia outwards and forwards in the lower third, and the general outward curve of the femur, and the occasional deformity in the neck of the femur, coxa vara, are all the results of the superimposed weight of the trunk acting upon the softened bones of the lower limbs.

Nor is it only the bones which yield to mechanical influences in rickets; the ligaments are unduly soft and lax, as can be seen in some rickety children in the extraordinary contortions of which their joints are capable; some rickety children can easily put their toes behind their ears, and the knee-joint can be felt to be altogether abnormally lax. In part the yielding of ligaments is, no doubt, due to the feebleness and laxity of the muscles which afford but little support for the joints, but it is probably due also to some change at present unknown in the ligaments

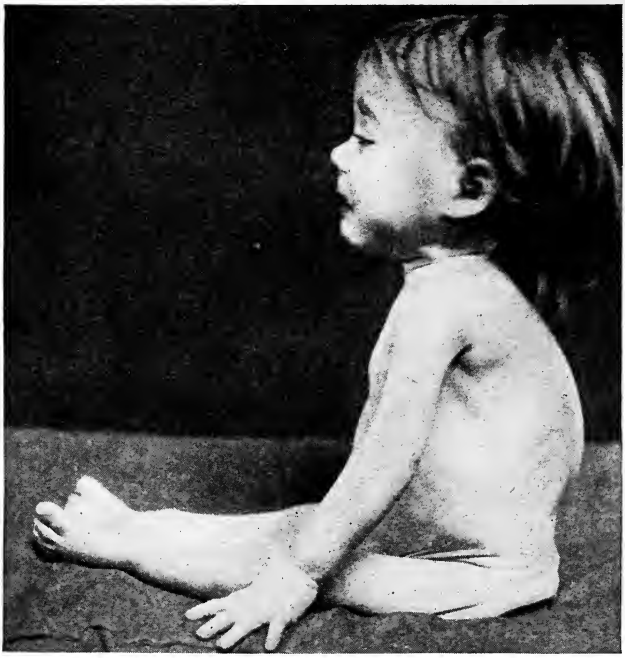

Fig. 3. Kyphosis due to weakness of back-muscles in rickets.

themselves. Consequently any stress upon joints is likely to result in deformity in addition to that resulting from bent or fractured bones.

The prevention of these deformities consists in relieving the bones as far as is practicable of weight or traction by keeping the child as much as possible off his legs, if necessary by the use of splints, and securing recumbency as much as possible: this is one of the many conditions in which the excellent habit of a midday sleep gives invaluable help; a 'pram' in which the child can lie down at other times will assist in securing fresh air as well as recumbency. If only parents would realize the

importance of keeping the child who has rickets off his legs until the softness of the bones has been rectified by proper dieting and the administration of cod-liver oil, we should not see the deplorable deformities which are so often inflicted upon the rickety child by parental carelessness or ignorance.

The general rounding of the spine which constitutes the rickety kyphosis (Fig. 3) and which is due to muscular weakness, in which the muscles of the spine partake, is sometimes a striking symptom: the child seems to 'sink down in a heap', when he is put in the sitting position. It is curious that this symptom, which is by no means a constant one, should have attracted the attention of Glisson and his contemporaries so much that they devised the name 'rachitis' (ραχις) as an alternative for the popular name 'rickets'.

The head of the rachitic child is often characteristic, not only in the late closure of the anterior fontanelle, but in shape and size. Large, square, and flat on the top, is an accurate description of the head in many cases; in addition, the rickety skull can often be felt to be so deeply grooved by the vessels of the scalp, that their course can be traced by one's finger-nail. If it be asked why the head is large in rickets, the answer is not clear: some observers, amongst whom is Stoeltzner, have described a dilatation of the ventricles in rickets, but I have never found such a condition myself: the calvarium is undoubtedly thickened in some cases, especially in the position of the frontal and parietal eminences, so that a rachitic bossing is produced, and I am much inclined to think that thickening may occur nearer to the fontanelle, producing a condition like Parrot's nodes without congenital syphilis, but I am not aware that there is ever sufficient thickening to account for the large size of the skull in some cases of rickets; moreover, if the large size of the head were due to an exuberance of bone formation one would expect the fontanelle to be closed rather earlier than is normal in these cases, whereas the reverse is true: e.g. Arthur C., aged $2\frac{1}{2}$ years, came to me with a marked degree of rickets, the tibiæ were bowed, the radial epiphyses considerably enlarged, and the head, which was of the characteristic square shape of rickets, measured $20\frac{1}{4}$ inches (the normal average circumference at this age is about $18\frac{3}{4}$ inches), the fontanelle, which should be closed at about eighteen months, measured $1\frac{1}{2}$ inches in both directions.

The large size of the head is not so far as my own observations go associated with any special mental characteristics; the child

with severe rickets, like any other invalid child who is perforce much in the company of adults and unable to join fully in other children's games, is apt to become quaint and precocious in his ways of thought and speech, but I am as little able to confirm the 'great acuteness of mind' which Underwood [1] described as 'observed in this and some other chronical complaints', as Sir W. Jenner's assertion [2] that children with extreme rickets are almost always deficient in intellectual capacity and power.

FIG. 4. Craniotabes. Skull showing characteristic shape and distribution of patches of true craniotabes.

I turn now to a vexed question, the relation of craniotabes to rickets; craniotabes consists of a thinning of the bones of the skull: usually of the posterior part of the parietals and the upper part of the occipital, rarely of the upper part of the temporal. It occurs usually in round or oval patches, ¼–1 inch in diameter, situated near but not actually reaching the suture. Sometimes there is a more diffuse thinning of the bone adjoining the sutures, but the nature of the thinning must then be regarded as doubtful, in very young infants certainly it may indicate nothing more than a physiological variation in the rate of ossification.

[1] *Treatise on Diseases of Children.* Lond., 1784.
[2] *Lectures on Rickets,* vol. iii, p. 416.

The round or oval patches are most characteristic, with the peculiar crackling sensation which they give when they yield like parchment under the pressure of one's finger.

My own clinical investigations, so far from inclining me to accept the view that true craniotabes is always of syphilitic origin, have led me to regard this connexion as quite exceptional. Of twenty-five consecutive cases of craniotabes only two were noted to be syphilitic and one possibly syphilitic; there was definite rickets in twenty of the remaining cases, in two rickets was doubtful; but in two out of the three syphilitic cases there was rickets also, and in the third case it was not noted whether rickets was present. So that in at least twenty-two out of the twenty-five cases rickets was present.

But even when craniotabes appears to be associated with syphilis without rickets, there may be a fallacy: I have used the term 'true craniotabes' above, because there is such a thing as delay of bone formation, in contrast to absorption of already formed bone; the latter alone in my opinion should be described as craniotabes. I have already mentioned (Chapter I, p. 5) that there are infants whose skulls remain thin, and whose sutures and fontanelle remain open longer than usual without any evidence of disease of any kind; in these children the edges of the parietal and occipital bones may show at two or three months of age a diffuse thinness, and may yield on pressure just like the softened bone in craniotabes.

In some infants syphilis seems to retard bone formation in this way, and a soft-yielding bone at the age of seven or eight months may really be simply a persistence of the unossified condition which is found in very young infants.

In one of the syphilitic infants I have mentioned, I watched the child from the age of eight weeks until it was seven months old; during the whole of this time there was diffuse thinness of the bones adjoining the parieto-occipital sutures, and I suspect that, although I have included this case amongst my series of craniotabes, it should rather be classified as delayed ossification due to syphilis.

But delay of ossification is certainly quite an exceptional result from syphilis; indeed the reverse is usual: I have frequently observed the skull of a syphilitic infant to be noticeably firmer than that of a healthy infant of the same age; a fact which makes it improbable that syphilis should be the usual cause of craniotabes.

There is another reason that makes me chary of attributing

craniotabes to syphilis: namely the effect of antirachitic treatment. One of the most striking illustrations of the value of fat in rickets is the remarkable rapidity with which craniotabes disappears in rickety children under the administration of cod-liver oil. I have noticed this in several cases; in some it has been much diminished and in others it has completely disappeared in seven to ten days ; in these cases no mercury was given.

Another argument, and, I think, a very strong one in favour of the rachitic nature of craniotabes is its close association with laryngismus stridulus. Out of twenty-two cases of craniotabes in which the point was noted seventeen had well-marked laryngismus stridulus. This spasmodic affection of the larynx is generally acknowledged to be one of the convulsive manifestations of rickets ; out of sixty-seven cases of laryngismus stridulus sixty-three were certainly rickety, three probably had rickets, only one was thought to show none of the bone affections of rickets ; no one has ever suggested as far as I know that laryngismus stridulus has any connexion with syphilis.

Lastly I would point out that it has long been recognized that syphilis aggravates rickets; some of the most extreme degrees of the characteristic changes of rickets are seen where the disease is associated with syphilis, and it is likely enough therefore that if craniotabes is a manifestation of severe rickets, it should occur where these two diseases are associated.

A striking feature of rickets is the protuberant abdomen, the ' pot-belly ', which is in part an indication of the cause, and in part a result of the disease. Disordered digestion, usually from excessive sugar or starch in the food, causes fermentation, flatulence, and catarrh in the intestine so that assimilation is hindered and rickets results; rickets in its turn predisposes to catarrh of mucous membrane so that flatulent distension easily occurs. But there are other factors producing the large abdomen of rickets: the pelvis is unduly small, the eversion of the lower ribs displaces the liver and spleen downwards, and the lax muscles of the abdominal wall afford insufficient support to the bulging contents. The recti muscles are often separated in the midline as much as half an inch or more by the chronic distension, but this 'diastasis of the recti', as it has been called, is not peculiar to rickets, it is seen with any chronic enlargement of the abdomen.

Chronic intestinal catarrh, to whatever cause it is due, is not necessarily associated with diarrhœa; it may show itself in chronic constipation ; and so it comes about that the rickety

child is often troubled with constipation, which no doubt is also favoured by the general lack of muscular 'tone' in the muscles of the abdominal wall as well as in those of the intestine. There is also a special liability to acute intestinal catarrh with diarrhœa: a tendency which exposes the rickety child to special danger in the summer months.

More dangerous still is the special tendency of rachitic children to catarrh of the respiratory tract. My own experience has abundantly taught me never to regard as trivial even the slightest bronchial catarrh in a child with rickets; the danger is greatest where there is much rachitic distortion of the chest-wall; a child, such as that shown in Fig. 5, with a chest pinched in laterally, so that there is a depression at the anterior part of the axilla, and a transverse furrow (Harrison's sulcus) just above the everted costal margin, is a bad subject for bronchitis; there is already but poor expansion of the alveoli and if the catarrh spreads down to the alveoli, as it is very apt to do in rickety children, the resulting broncho-pneumonia is apt to prove fatal.

Another manifestation of rickets which may be altogether out of proportion to the bone changes, and which therefore is sometimes not recognized as rachitic, is anæmia. This is of all degrees and is sometimes very profound: the child's face is I think generally whiter than in the anæmia of congenital syphilis, where it is usually of a markedly yellowish tinge. The spleen is little if at all enlarged, and herein the simple secondary anæmia of rickets differs from the so-called 'splenic anæmia of infants' where the spleen is greatly enlarged, reaching often below the level of the umbilicus. There is nothing characteristic about the blood changes in the rickety anæmia: the hæmoglobin is diminished in proportion to the diminution of red corpuscles; a differential count of the white corpuscles shows no disproportion of polymorphonuclears or of lymphocytes.

The recognition of the cause of the anæmia is of great practical importance, for the dietetic treatment is hardly less important than drugs; and cod-liver oil is more likely to do good than iron. I shall have more to say of splenic anæmia under the head of syphilis. It is quite certain that splenic anæmia is associated with rickets in a large proportion of the cases, but in many there is also some evidence of syphilis: the remarkable frequency of Parrot's nodes with splenic anæmia is also noteworthy as suggesting a syphilitic origin. But at present the status of splenic anæmia remains undetermined, it can only be said that both syphilis and rickets appear to play some part in its causation.

Diagnosis. One might imagine that a disease with so many characteristic symptoms must always be easy of recognition: as a matter of fact it is often extremely difficult to be sure when rickets is present: this is sufficiently obvious from the discrepancies between various observers as to the frequency of rickets: in my own clinic one observer found that 95 per cent. of the children in the second year of life had rickets, whereas my own

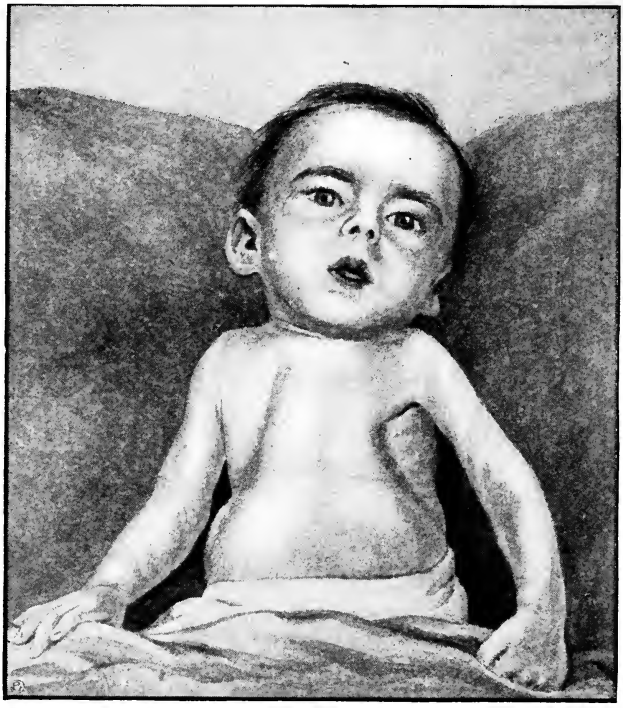

Fig 5. Characteristic deformity of chest in severe rickets.

figures showed 58 per cent.: some continental writers have asserted that 70 to 80 per cent. of new-born children have rickets! whereas Fédé and Cacace of Naples found that only 0·4 per cent. had rickets at birth, and for my own part I am very doubtful whether rickets ever occurs at birth.

Any one who has attempted to collect statistics as to the frequency of rickets will very soon realize the difficulty of deciding what constitutes rickets: in its extreme degrees any

one can recognize it, in its slightest degrees the most experienced observers may be doubtful of it.

Sweating about the head is a symptom to which very little diagnostic significance can be attached : as supposed evidence of rickets in infants and of tuberculosis in older children it often causes quite a needless amount of alarm : children who are out of sorts from any cause are particularly prone to sweat about the head during sleep ; certainly apart from other much more distinctive evidence head-sweating must not be taken as evidence of rickets, or of tubercle.

Beading of the ribs is another symptom to which I think it is possible to attach too much significance : the junction of the bony rib and the costal cartilage is often distinctly palpable as a small boss-like elevation within a few weeks after birth, and if this alone were sufficient evidence of rickets it would be true that almost all children are rickety, but recent observations on the histology of this slight beading of the ribs have shown that it may exist with no rachitic change whatever. I need not describe here the characteristic microscopic appearances at the epiphysial line in rickets : suffice it to say that these are entirely lacking in some of the supposed ' beadings ' found during earliest infancy. Undoubtedly, taken in conjunction with other symptoms, even a very slight beading is of importance, but by itself it does not necessarily indicate rickets.

The size of the fontanelle is so variable in healthy infants during the first six months of life that I do not think any significance as evidence of rickets can safely be attached to the size of the fontanelle at this stage : it is only after the age of six months and especially in the second and third year that its size comes to have an importance in this respect : if the fontanelle measures much more than 1 inch in each direction at one year, or is open after the age of two years this is suggestive of rickets.

Late eruption of the teeth is certainly important ; taken in conjunction with other characteristic signs it is valuable evidence of rickets.

In connexion with the diagnosis of rickets I would again emphasize the special incidence of the disease upon particular tissues in particular cases : one case may be brought to the medical man for convulsions, while the osseous changes of rickets are present only in the slightest degree, another case is brought for the large abdomen, another for its bronchitis, and another for its profound anæmia, another for pronounced muscular weakness, and in all these the bone manifestations may be

limited to slight beading of the ribs, lateness in dentition and delay in closure of the fontanelle but none the less the symptoms for which the child is brought are rachitic.

Treatment

The prevention of rickets lies in the proper feeding of the infant. The surest safeguard against rickets is breast-milk, and if a mother is unable to perform her bounden duty in this respect to her child and a wet-nurse is impracticable, the next best preventive of rickets is fresh cow's milk. Now there are certain fallacies with regard to the use of fresh cow's milk which I wish to mention in this connexion: when I advise that all milk for infants should be either pasteurized or boiled I am often met with the objection 'but surely boiled milk causes rickets!'. I know of no evidence that pasteurizing or boiling milk has any connexion whatever with the production of rickets: I suspect that this mistake has arisen from the long-standing confusion introduced by the unfortunate and misleading term 'scurvy rickets'; there is no doubt that the boiling of milk tends to diminish or destroy its antiscorbutic power, but rickets and scurvy are entirely independent diseases and clinical experience shows, I think, that the heating of milk, at any rate within the limit of pasteurization or of just reaching the boiling-point, does not render it liable to produce rickets.

Again, I find an infant with pronounced rickets and I ask how it has been fed; I am told that it has always had fresh milk, but when I inquire into the proportions of milk and water I find that at the age of six months the infant is having equal parts, in other words, the child is only having 1·75 per cent. at most of fat. Let it be remembered that if fresh milk is too much diluted, it may cause rickets just as much as any other food which involves fat-starvation. It is not sufficient to instruct a mother to give cow's milk to her child, the proportions of milk and water, and the amount of cream, if any is available, to be added, must all be carefully adjusted according to the age of the infant.

Again, I am told that an infant is having fresh milk and nevertheless has developed rickets: but on inquiry it turns out that the infant is having in the milk some starch-containing food, patent or otherwise; or perhaps once or twice a day is having bread-sop or mashed potato when the child is seven or eight months old. As I have already pointed out, anything, be it food or disease, which interferes with the child's power of assimilation may cause fat-starvation and rickets: and even

though a milk mixture contain enough fat to prevent rickets if it were given without the addition of starch or of too much sugar, either of these additions may so interfere with assimilation that the child becomes rickety. It is not enough to instruct a mother to give fresh milk, we must warn her that the addition of starch-containing foods or of foods containing excess of carbo-hydrate, whether sugar or the products of malting, may prevent the good results of the fresh milk.

Here I shall venture to reiterate what I have said elsewhere as to the doubtful expediency of using barley-water as a diluent for milk in infant-feeding. I have often noticed slight degrees of rickets where this has been done, and I suspect that even the small proportion of starch (1 to 2 per cent.) present in barley-water may be responsible for the rickets in some of these cases: especially where the barley-water has been used habitually for an infant less than six months of age, and where the milk has at the same time been diluted so much that the slightest interference with assimilation would make its proportion of fat insufficient.

Now I turn to another error which has somehow gained currency, the belief that in adding lime-water to milk we are providing a valuable prophylactic against rickets. As far as I know there is not the least evidence that lime-water has any value whatever either for the cure or for the prevention of rickets. Cow's milk contains so much larger a proportion of lime salts than human milk that even if it were diluted excessively it would still be richer in these salts than is breast-milk. Moreover experience shows that rickets arises in spite of free use of lime-water in infant-feeding.

The proper food during the first eight or nine months for an infant who for some inevitable reason is deprived of breast-milk, is fresh cow's milk: and the most important item in the prevention of rickets is to secure a sufficient proportion of fat; as a matter of clinical observation I think it may be said that for an infant over three months of age a proportion of fat below 2 per cent. involves some risk of rickets, and that at any time during the first year safety from rickets is better assured by a proportion of fat reaching about 3 per cent. The addition of starch in any form to the diet of an infant under eight months of age, is liable to interfere with assimilation and in this way involves danger of rickets, especially if the milk mixture is already poor in fat. Similarly, excess of sugar or of the products of malting favours the onset of rickets and, as I have shown in

a previous chapter, the much-advertised malted foods which are prepared by the addition of water without milk are faulty both in their excess of the soluble carbo-hydrates and in their low proportion of fat, and for this reason are liable to produce rickets.

The next point of importance upon which I would insist very strongly is this : the risk of rickets is not limited to the first nine months of life. It seems quite clear in some cases that rickets has begun later, sometimes in the second year, and in most of these cases one finds that the mother, instead of introducing starch-feeding gradually at eight or nine months of age by giving one starch-containing feed a day for a month and then two feeds daily, has given starch at every feed or almost every feed from this age onwards, with the result that not only has the child satisfied its appetite with bread and butter or farinaceous food to the neglect of milk, but the digestion which only gradually acquires the power of dealing satisfactorily with starch has been disordered, so that even what milk is taken is but poorly assimilated.

From nine months old up to the end of the tenth month an infant should not have more than one meal a day of starch-containing food : I like Robb's Biscuits, or ' Malted Rusks ' for a start, but at this time there is no objection to any of the partially malted cereal foods or to the unconverted cereal foods, only let it be quite clear that such ' foods ' are to be given only once a day.

At the age of eleven months two starch-containing meals can be given in the day, one of which I think may advantageously be a milk-pudding either of pearl sago or ground rice. The rest of the feeds are to be milk, not less than $1\frac{1}{2}$ pints of milk should be taken daily. In addition the yolk of an egg very lightly boiled should be given daily from the age of nine months. Next to milk I know of no food which is of more value in the prevention of rickets and in the cure of it, than yolk of egg : it contains about 20 per cent. of fat, which is of a particularly easily assimilable kind.

After the age of twelve months a dessertspoonful of gravy of fried bacon with a little crumbled bread soaked in it makes a valuable food. Throughout the second year it is most important that a child should have abundance of milk, and that the diet should not be too largely starch-containing. Chicken or veal broth, fish, boiled brains, red gravy, custard, blancmange, all these are useful as variations which avoid the starchy element. The child who at fourteen or fifteen months lives mainly on bread

and butter and potatoes is hardly less likely to develop rickets than the infant who at six months is fed upon some starchy patent food.

When rickets is already present the first point which requires attention is the diet; it must be corrected in accordance with the child's age upon the lines which I have just laid down.

The drug treatment which in this country is generally admitted to be of most value is the administration of cod-liver oil: and here I shall draw attention to what I believe to be an error in its administration in some cases. I find an infant of eighteen months or two years having a domestic teaspoonful of plain cod-liver oil as a dose, with the result that the child's digestion is upset, a large part of the oil is passed in the stool, and probably more harm is done than good. Cod-liver oil is best given in doses of 20 to 40 minims in an emulsion or mixed with malt: I am rather in favour of the latter method, especially where the infant is old enough to be having some farinaceous food in his diet, for the diastasic effect of the malt helps to prevent starch-indigestion.

There seems to be no specific virtue in cod-liver oil, any other oil will do equally well provided that it can be taken without disturbing digestion or causing nausea by its taste. At one time we used at the Children's Hospital, Great Ormond Street, olive oil, and pilchard oil and cotton-seed oil, made into as palatable emulsions as possible, and these seemed to be as useful as cod-liver oil except that they were more apt to cause nausea or digestive disturbance.

In some parts of Europe phosphorus is the drug most used for rickets ($\frac{1}{200}$ grain may be given three times a day) but as it is usually given in oil one cannot but suspect that the vehicle may be of more importance than the drug. It is difficult to believe that deficiency in the intake of phosphorus can be the cause of rickets, for according to Bunge, cow's milk contains nearly six times as much phosphorus as human milk, so that even with the excessive dilutions of cow's milk which produce rickets, there would be no deficiency of phosphorus. It is, however, possible to reconcile the apparent discrepancies in treatment: for according to some recent observations by Freund, the urine of children taking an increased amount of fat in the food, shows an increased amount of phosphates; which presumably indicates that the absorption of phosphates from the food is in some way facilitated by the larger intake of fat; according to Ritter phosphates are present in abnormally high

proportion in the fæces of children with rickets: in other words, phosphates are deficiently absorbed from the food : this deficient absorption of phosphates would be explicable by the theory of fat-starvation if Freund's observation be correct.

Whilst therefore an explanation is given of the value of fat in the dietetic and in the drug treatment of rickets, it is quite conceivable that when the possibility of absorbing phosphates is increased by increasing the proportion of fat in the diet, as it probably is in most cases where phosphorus is relied upon, the increased intake of phosphorus may be of value.

Possibly a similar explanation may apply to the use of the various preparations of iron- or calcium-phosphate and of hypophosphites which some have thought useful in rickets ; I confess that so far as my own experience of them has gone—and it has chiefly consisted in observing the effects of this treatment in the hands of others—they have seemed to be of little value : they may assist treatment by improving the appetite, but apparently have no specific virtue in rickets.

I have discussed the treatment of rickets so far, as if it consisted solely in dieting and drugs : but I would not have it supposed that no importance is to be attached to general hygiene in the prevention and cure of this disease. It seems to me certain that rickets cannot be produced by lack of sunlight or of fresh air or by any climatic conditions, it is a diet disorder and is curable by dieting, but this does not make it any the less probable that such factors may play some part by interfering with the child's general health and vigour and so influencing its assimilation and nutrition and predisposing to rickets. The rickety child is undoubtedly the better for plenty of sunlight and open air, and I would send him, if possible, to a bracing seaside place, such as Herne Bay, Broadstairs, or Folkestone. I like also cold douches—the infant should sit in a tepid bath while some colder water is poured over his shoulders and back : and after the bath gentle massage of the limbs helps to restore firmness and ' tone ' to the flabby muscles.

I have already referred to the importance of keeping the child's weight off his limbs as much as possible during the acute stage of rickets ; for this purpose splints are to be used if necessary to prevent the child from standing and in severe cases even from sitting. Where deformity has already occurred, it is remarkable how much it may be diminished if effectual means are taken to keep the child from carrying the weight of his trunk upon the limbs for several months : during this time the

nutrition of the muscles should be maintained by massage twice or even thrice daily.

The nervous affections of rickets I shall consider under the heading Convulsive Disorders; suffice it here to say that the nervous excitability of rickets subsides very rapidly under the administration of cod-liver oil, but where there are already indications of a convulsive tendency whether in the form of an ordinary convulsion, or of laryngeal spasm, or of 'facial irritability' or tetany, it is safest to give bromide in combination with the cod-liver oil: a formula I often use for this purpose is as follows: Potassii Bromid. gr. ij, Liquor Calcis ℥xv, Glycerin ℥v, Spirit. Menth. Pip. ℥1½, Ol. Morrhuae ad ℥j, ter die. Under the influence of bromide the convulsive tendency disappears even more rapidly than with the oil alone, and as there is always some danger to life attaching to these convulsive manifestations it is only right to adopt the speediest method of escape from the danger.

CHAPTER VIII

INFANTILE SCURVY

INFANTILE scurvy is often spoken of as if it were a disease of recent discovery and of modern development. Its recognition all over the scientific world is justly associated with the names of two distinguished physicians both connected with the Great Ormond Street Children's Hospital, the late Dr. Cheadle and Sir Thomas Barlow, to whose investigations is due almost all that is known of the pathology of the disease. But to suppose that infantile scurvy was unknown, or even that its true nature as a scorbutic condition complicating rickets was unrecognized until the nineteenth century is an error, and one that has crept into many textbooks.

As long ago as the middle of the seventeenth century, Glisson, in his *Treatise on the Rickets*, had not only described the characteristic symptoms of infantile scurvy, but, with more accuracy than some of his successors, had recognized that it was scurvy complicated with the rickets and not an acute form of rickets. In the English translation, published in 1651 (p. 249), he says: 'The Scurvy complicated with this affect (rickets) hath these signs: (1) They that labour under this affect do impatiently endure Purgations; but they who are only affected with the Rachites do easily tolerate the same. (2) They are much offended with violent exercises, neither can they at all endure them. But although in this affect (rickets) alone there be a kind of slothfulness and aversation from exercise, yet exercise doth not so manifestly, at least not altogether so manifestly, hurt them as when the Scurvy is conjoyned with the rachites. (3) Upon any concitated and vehement motion they draw not breath without much difficulty, they are vexed with divers pains running through their Joynts, and these they give warning of by their crying. . . . (4) Tumors do very commonly appear in the Gums. (5) The urin upon the absence of the accustomed Feaver is much more intens and encreased.'

Glisson (ibid., p. 227) actually emphasizes what is not yet sufficiently recognized, that there is no essential connexion

between infantile scurvy and rickets; he says, 'the Scurvy is sometimes conjoyned with this affect,' and after mentioning as causes heredity, infection, and the possibility that it may be 'produced from the indiscreet and erroneous regiment of the infant', though he attributed it chiefly to climatic influences, he adds, 'for it (scurvy) scarce holdeth any greater commerce with this Diseas (rickets) than with other diseases of longer continuance.'

Such terms as 'scurvy rickets', 'acute rickets,' and 'hæmorrhagic rickets'—names which would imply that scurvy is a variety of rickets—have been responsible for much confusion. Obscure though the exact ætiology of infantile scurvy may be, it is clear from clinical facts that this affection arises from causes entirely distinct from those which produce rickets; severity of rickets raises no special probability of scurvy; indeed the worst cases of scurvy are often associated with very slight rickets, and in some cases with none. Both diseases are due to faults of diet, and it is likely enough that a diet which is faulty in one respect may be faulty in another; the particular fault which produces rickets may be, and no doubt often is, present when the diet shows also the particular fault which produces scurvy. But the fault is not one and the same; witness the many infants who suffer with severe rickets after being fed from an early age with potato, which would seem when given at that age to favour if not actually to cause the occurrence of rickets, whereas there is probably no more powerful agent than potato in the prevention or cure of infantile scurvy.

The clinical picture of the fully-developed disease is striking enough; an infant who has been fed upon one of the patent foods, with or without milk, or on milk which has been condensed, sterilized, or otherwise altered, has been ailing for some weeks, has taken food badly and probably lost weight. Moreover the mother says it cries whenever it is touched, and, as she puts it, 'has lost the use of its limbs.' The infant is pale, it lies quiet perhaps until it is approached, when it cries out in obvious dread of being touched; the legs lie motionless usually with the thighs slightly abducted and everted and the knees slightly flexed; the arms are less often affected. There may be some swelling of part of one or other of the limbs, obliterating the natural curves. Any handling of the affected limbs causes a piteous cry, evidently of acute pain. If teeth are present the gums around them are swollen and purple, occasionally projecting like a mass of granulations almost completely hiding the teeth,

and bleeding readily when touched. The urine is perhaps smoky, if not red with blood.

Such in outline is the characteristic picture of infantile scurvy, which I shall now consider in more detail, chiefly with reference to diagnosis and treatment.

Age-incidence. The age at which infantile scurvy begins is a point of considerable importance in diagnosis. As the accompanying chart of sixty-four cases under my own observation shows, its onset is almost limited to the later half of the first year of life, and in nearly 80 per cent. of the cases the disease begins between the ages of six months and ten months.

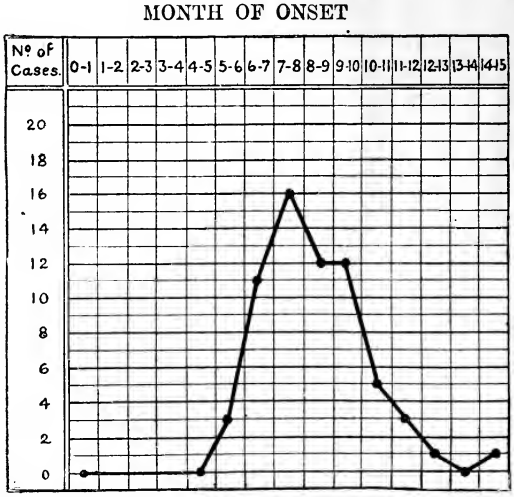

FIG. 6. Age-incidence of infantile scurvy (sixty-four cases).

So marked is this special age-incidence, that given an infant of six to twelve months and a history of tenderness in the limbs, the diagnosis of scurvy should at once suggest itself as a possibility. In several of the cases in this series the disease had been diagnosed as 'rheumatism'—a mistake which ought never to occur, for rheumatism is practically an unknown disease under the age of eighteen months, and indeed is exceedingly rare under the age of three years. This chart shows also that in no case did scurvy begin before the age of five months—a point which distinguishes this disease from many cases of syphilitic epiphysitis which begins most often under the age of three months.

Mode of onset. So much stress has been laid upon the

affection of the gums and upon the tender swellings in the limbs in infantile scurvy that there is danger of forgetting that in many cases there are other more insidious symptoms which may appear some weeks before these obvious manifestations. An infant who has thriven hitherto upon some patent food begins to ail, the weight ceases to rise or actually falls, the feeds are taken badly, and the child is fretful and miserable; there is no swelling of limbs, perhaps no definite tenderness anywhere, and the gums are perfectly normal; but that these early symptoms indicate scurvy may be shown not only by the rapid improvement within two or three days on antiscorbutic treatment, but also in some cases by the presence of blood in the urine even in this early stage.

I doubt whether the onset of scurvy is ever really sudden, but undoubtedly the more pronounced symptoms may appear quite suddenly, and are then apt to be mistaken for the results of traumatism; for example, an infant, aged eight months, was brought to me because he was 'not getting on'; there had been some tenderness in the legs for at least four weeks, but the nature of this had not been recognized; one morning the child had his usual bath and then went to sleep for a short time: he awoke with the left eye partly closed by great swelling of the lids, which were discoloured and soon looked as if bruised; it was concluded that during the bathing the child must have struck his eye against the side of the bath. The history, however, of feeding with Allenbury and Benger's foods, the tenderness of the legs, and the presence, as examination showed, of blood in the urine, all pointed to scorbutic hæmorrhage, and the rapid disappearance of the swelling and hæmorrhage from the lids under antiscorbutic diet confirmed the diagnosis.

It would, I think, be rash to assert that the supposition of traumatism is wrong in all such cases. The hæmorrhagic tendency of scurvy may well favour the occurrence of hæmorrhage from slight traumatism, which would have no such effect in a healthy infant, and this view is supported by the situation of superficial hæmorrhages; for example, in one case the only subcutaneous hæmorrhage was over the bony prominence of the sacrum, a part obviously exposed to pressure and jarring; in another which was sent to me as a case of infantile paralysis the efforts of the doctor to obtain a knee-jerk had resulted in a subcutaneous hæmorrhage over the patella.

Limb affection. Perhaps the most striking feature of infantile scurvy is the tenderness of the limbs and loss of movement. These

symptoms occur much more often in the lower limbs than in the upper. In the sixty-four cases tenderness or swelling was present as follows: in forty-seven in the legs only, in ten in legs and arms, in one in the arm only. In six out of the sixty-four cases there was no tenderness in any of the limbs: the diagnosis in these cases rested on the association of a scorbutic diet with hæmaturia, confirmed in two, where teeth were present, by affection of the gums, and in all by the rapid cessation of hæmaturia on antiscorbutic diet.

With the tenderness there is sometimes associated some visible or palpable swelling of the affected limb, but in many cases this is so slight that it is easily overlooked; and as it is the result chiefly of subperiosteal hæmorrhage it may be detected only by careful—and, I would add, most gentle—palpation of the bone as a vague, deep thickening. The situation of this thickening is of some importance; it is not limited to the epiphysial region like an epiphysitis, but extends some distance along the shaft of the bone—a point which should distinguish it also from any joint affection. In my own cases the swelling has been most often about the lower third of the tibia or the femur (Fig. 7), but any part, or the whole, of the shaft may be surrounded by subperiosteal hæmorrhage, and occasionally, though much more rarely, similar deep hæmorrhage may occur over the flat bones of the skull, producing large bluish swellings on the head which may simulate sarcoma, or over the scapula, as in a specimen in the museum of the Children's Hospital, Great Ormond Street.

The loss of movement in the affected limbs is so marked that the disease had been mistaken more than once in my series for infantile paralysis—a mistake which hardly ought to occur, for the acute tenderness, the severe pain on passive movement, the swelling and local thickening, and the associated conditions of the gums and urine are all features quite foreign to infantile paralysis. The loss of movement is no doubt due partly to fear of the acute pain which movement causes; but it may also be due partly to mechanical disability, for not only is the periosteum stripped off the bone by the underlying hæmorrhage, but, as I have seen in some autopsies on infantile scurvy, the muscles also in severe cases are infiltrated with serum and extravasated blood. Occasionally there is further disablement from separation of epiphyses by extensive hæmorrhage at the epiphysial line; in one case which I examined epiphyses had been separated in all four limbs. This danger of separation of epiphyses, which is due to effusion of blood at the epiphysial junction, is a point to be

remembered in the handling of these infants with scurvy. Cases have been recorded also in which fracture occurred in the shaft of the bone.

Œdema. The occasional occurrence of œdema over the thickened part of the limbs in scurvy is worthy of notice. In the diagnosis between scurvy and suppurative periostitis it might have been thought that œdema pointed to the presence of pus. This is certainly not so; I have seen well-marked pitting of the skin on gentle pressure in cases of infantile scurvy.

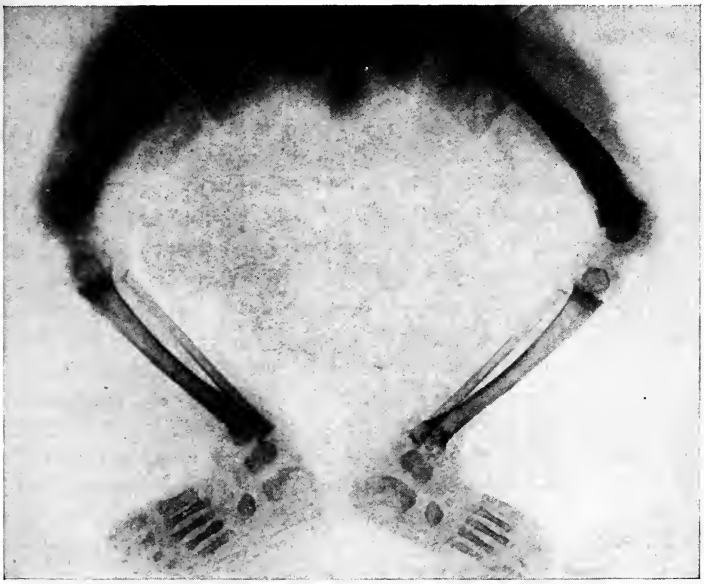

FIG. 7. Skiagram of legs in infantile scurvy. Showing hæmorrhage surrounding lower two-thirds of femora.

Gums. The characteristic appearance of the gums has already been mentioned, but it is to be remembered that any great swelling or discoloration of the gums is by no means a constant feature; often it requires careful observation to detect the little affection that exists. There may be only a thin purple line or minute patch of red discoloration at the free edge of the gum; and I have specially noted that in some cases it was only on the posterior edge of the gum behind the tooth, and therefore very easily overlooked.

It is generally stated that where there are no teeth there is no

gum affection ; this is not strictly accurate, for where the teeth are close up to the surface of the gum, but not actually through, I have seen hæmorrhage in the gum over them. When teeth are present the gums are not necessarily affected ; in seven out of thirty-nine cases in which teeth were present in my series the gums showed nothing abnormal.

Very rarely the gums are markedly affected when there is no other obvious symptom of scurvy. In one of my cases there was no tenderness whatever in the limbs, although the gums showed very marked swelling and purple discoloration. In another some tenderness had been noticed in the legs, but had passed off when I saw the child, whose gums were swollen and purple. In both these cases the existence of scurvy, suggested by the diet and the gums, was proved by the finding of blood in the urine.

Palatal hæmorrhage. I wish to call attention here particularly to a manifestation of scurvy which has not, I think, attracted much notice, namely hæmorrhage in the mucous membrane of the hard palate. The discoloration occupies the middle part of the vault of the hard palate ; it has not in my cases extended forward to the alveolar portion, nor backward to the soft palate. The colour varies from a bright blood red to a deep almost black purple. As in the gums, the affected area may become secondarily inflamed and the mucous membrane over it roughened. Such were the appearances in four of my cases, but as I had not looked for this manifestation in the earlier cases I cannot tell in what proportion it is to be found. This palatal hæmorrhage is certainly noteworthy, as it may assist diagnosis when it occurs—as in three out of my four cases—in infants who, owing to absence of teeth, show no gum affection.

Orbital hæmorrhage. 'Black eye' in an infant six to twelve months old, without obvious traumatism, should suggest the possibility of scurvy ; but it is not a very frequent symptom. It was present in six out of the sixty-four cases, and in five of these was sufficient to cause protopsis of the eye. Curiously enough, in all six it was the left eye that was affected. The discoloration appears usually chiefly or only in the upper lid.

Urinary symptoms. The diagnostic importance of the urine in infantile scurvy is not sufficiently appreciated. Urinary changes are actually more constant than the gum affection, and an examination of the urine will usually clinch the diagnosis if there be any doubt. During the last few years

I have examined the urine of all cases under my notice as far as possible, and out of 38 cases only 4 showed nothing abnormal. In 12 there was blood only, in 9 blood and casts, in 2 blood and pus, in 2 pus only, and in 9 albumen only ; so that the proportion of cases showing some urinary change was 89 per cent., and the proportion showing hæmaturia was 60 per cent.

But it must not be supposed that blood is to be detected by the naked-eye appearance of the urine in this proportion of cases. In many the amount of blood was so small that it was seen only by careful microscopic examination ; and when pus was present, although sufficient to attract attention at once under the microscope, it was not sufficient in any of my cases to be detected by the naked eye, although in one there was enough to make the urine slightly turbid.

It is generally stated that hæmaturia may be the only symptom of infantile scurvy ; and if the hæmorrhagic manifestations are to be taken as the only evidence of scurvy, this is certainly so. In three of the cases mentioned above there was no affection of the limbs or gums—in fact, no characteristic symptom except hæmaturia ; but in all three there had been wasting recently, and one was fretful and miserable, and another would not take his food ; there were, in fact—as I suspect there usually are in these cases where hæmaturia is stated to be the only symptom of scurvy—those vague symptoms of disturbed nutrition which I have described as marking the onset of scurvy. The presence of casts in the urine in association with the blood may indicate a nephritis in some cases. As I have pointed out [1] the casts are sometimes numerous, and easily found without centrifuging, and the albuminuria occasionally persists for many weeks or months. The occurrence of pyuria with acid or neutral urine, and without any special evidence of irritability of the bladder, would seem to point to a pyelitis, and this was the clinical condition in three of my cases. The recognition of this pyuria is of some practical importance, for, like the nephritis, it is apt to persist after all the acute symptoms of scurvy have passed away. In one case where the tenderness and loss of movement in the legs had begun to improve within forty-eight hours, and were as usual cured in a few days by anti-scorbutic diet, the pyuria persisted nearly five weeks, and during this time the child remained pale, and failed to gain weight until the pyelitis began to subside. Not only may the pyelitis retard the infant's recovery, but it seems likely that this

[1] *Lancet*, Aug. 13, 1904.

condition, which when it occurs as a primary affection in infants causes severe pyrexia, may account in some cases for the high temperature which sometimes accompanies infantile scurvy. In a case in which the urine showed pus without blood the temperature was 104·4°; and in one of the cases in which the pus was associated with blood, a slighter degree of pyrexia persisted for nine days after antiscorbutic treatment began, although the gum symptoms and tenderness of limbs had rapidly subsided; the pyuria in this case lasted twelve days.

Temperature. Pyrexia in infantile scurvy, although rather the exception than the rule, is by no means a rarity; the temperature may be raised to 101° to 102° F. when the infant comes under treatment, but it subsides generally within a few days as the acute symptoms of scurvy disappear. In some cases, however, the temperature rises to 103° to 105° F., and, as I have pointed out, this may depend upon the presence of pyelitis. The occurrence of fever in infantile scurvy is worth remembering, for this symptom, like the œdema to which I have referred, has been regarded as pointing to suppurative periostitis—a very serious mistake if it leads to incision, a measure which would only increase the risks of infantile scurvy. If the possibility of scurvy is borne in mind, and the history of feeding and the gums and the urine are investigated, this error is not likely to arise.

Other hæmorrhages. Hæmorrhage into the skin is quite the exception in infantile scurvy; it was present in six out of the sixty-four cases, excluding those in which orbital hæmorrhage showed as 'black eye'; in two cases it showed as a linear bruise running downwards and outwards for nearly an inch from just below the inner canthus; in one the situation was the scar of vaccination.

Mucous membranes. I have already mentioned hæmorrhage in the mucosa of the hard palate. The visceral mucous membranes may be similarly affected; in a specimen figured in the museum of the Children's Hospital, Great Ormond Street, hæmorrhage had occurred into the mucous membrane of the bladder. Bleeding from mucous membranes is not very common; in three of my cases slight epistaxis had occurred, and in one of these there was bleeding also from one ear. Less rare is the passage of streaks of blood with the stools (noted in eight cases).

Visceral hæmorrhage. Post mortem examination showed in one of my cases that extensive hæmorrhage may occur in connexion with viscera, though such an occurrence is probably

rare. A boy, aged thirteen months, who had been fed on Savory & Moore's food mixed with condensed milk since the age of four months, had much swelling and tenderness in the left thigh and leg, and purple swelling of the gums. He was admitted under my care at King's College Hospital, and at once put upon antiscorbutic diet, but developed diarrhœa and died within a few days. Autopsy showed, in addition to the subperiosteal hæmorrhage in the leg, that there were strands of old adhesions over the right lung, and in the meshes thus formed there was a layer of fresh blood-clot about one-eighth of an inch thick over a large portion of the lung. Another case in which visceral hæmorrhage raised an important practical point was one seen in consultation with Dr. H. J. Spon. The child, aged eight months, had been fed on the Allenbury foods and had tender swelling about the lower third of the right femur, and some scorbutic affection of the gums; the infant was extremely ill, and was passing blood and mucus frequently from the bowel; it had vomited twice on the day when I saw it. The abdomen was flaccid and hollow, but in the direction of the transverse colon it showed an elongated sausage-like tumour which on palpation was firm, and seemed to vary in hardness at intervals during palpation. Had there been no scurvy I think no one would have doubted that there was an intussusception. The child continued to vomit, but the bowels were open, and after consultation with my surgical colleague, Mr. F. J. Steward, and an unsuccessful attempt to reduce the tumour by injecting fluid into the bowel, it was decided to postpone operation, as the possibility of the tumour being hæmorrhagic was in our minds; moreover, the child's general condition seemed almost desperate. Under antiscorbutic treatment the child improved, and I heard subsequently from Dr. Spon that the abdominal tumour became less, and gradually disappeared after several weeks. I suppose there can be no doubt that in this case there was a mass of blood-clot surrounding the bowel, perhaps in the wall of the bowel.

Joints. As already mentioned, infantile scurvy is sometimes mistaken for some joint affection; careful examination, however, shows that the tenderness and swelling is not connected with the joints, but with the shaft of the bone. Whilst, however, I know of no clinical evidence that arthritis ever forms part of infantile scurvy, hæmorrhage into joints certainly may occur; in one of my cases there was found at autopsy a fresh blood-clot about the size of a shilling, lying free in the intercondylar part of the knee-joint.

In a drawing preserved at the Children's Hospital, Great Ormond Street, a hæmorrhage is shown under the synovial membrane of the acetabulum in a case of infantile scurvy.

Prognosis

There are few diseases in which the effect of treatment is so striking as in infantile scurvy ; under *efficient* antiscorbutic diet

Fig. 8. Subperiosteal ossification in infantile scurvy. Section of femur showing at upper end a layer of bone enclosing the decolorized blood-clot between the periosteum and the shaft of the femur.

the tenderness and pain on movement is usually appreciably less in forty-eight hours, and I would lay it down as a rule that if proper antiscorbutic dieting has produced no definite improvement within four days the diagnosis of scurvy should be questioned.

But although such rapid diminution of the tenderness and pain may be expected, other symptoms persist longer ; the subperiosteal hæmorrhage, if large, may cause some deep thickening

to remain for two or three weeks. Occasionally the thickening remains for many weeks or months, in spite of the complete disappearance of all other symptoms of scurvy. This is explained by a specimen in the museum of the Children's Hospital, Great Ormond Street, which shows a layer of bone deposited by the separated periosteum and forming a bony sheath enclosing the subperiosteal hæmorrhage (Fig. 8). I am indebted to Dr. Lees for kind permission to mention this case, which was under his care.

But apart from the mere persistence of symptoms, there are other more serious risks in infantile scurvy. I have several times heard medical men make light of the danger of scurvy from patent foods on the ground that not only is scurvy an uncommon result, but that when it occurs it is speedily cured by simple changes in the diet. No doubt the risks of scurvy would be minimized if the disease were detected at its earliest onset; as a matter of fact, it is hardly ever recognized until the infant has already passed through weeks of needless and intense suffering. This, surely, is serious enough; but there is also a very real risk to life; in five out of the sixty-four cases the disease proved fatal by diarrhœa and exhaustion, and even when death does not result there is sometimes prolonged disturbance of nutrition, so that the infant fails to gain weight and remains ill-nourished and frail for weeks or months after all other symptoms of scurvy have disappeared. The renal symptoms may influence the prognosis—in one of my cases of nephritis with scurvy the child showed a trace of albumen in the urine, and was frail in appearance two years after the occurrence of scurvy—and pyelitis may persist for several weeks and nutrition may suffer therewith. The mere presence, however, of blood in the urine does not seem to be of serious import; it usually ceases in a week or ten days under antiscorbutic treatment.

Prevention and Treatment

Scurvy is an entirely preventable disease, and it is important, therefore, to know the foods upon which it is liable to arise. In the subjoined table are stated the kinds of food in use for several weeks or months before the appearance of scurvy in this series of cases:

Patent foods containing dried milk, prepared according to directions with water only	18 cases
Patent foods prepared with fresh milk	22 cases
„ „ „ „ condensed milk	11 cases
„ „ „ „ sterilized milk	1 case
„ „ „ „ fresh (unboiled) milk	1 case

Condensed milk diluted with plain water, barley-water, or lime-water	5 cases
Sterilized milk	3 cases
Peptonized milk	1 case
Boiled milk	2 cases

In the above table, where ' fresh milk ' was used in mixing the food, it was usually, if not always, heated to boiling during the process.

It is clear that the mere fact that cow's milk is mixed with a patent food is not sufficient to prevent scurvy. Nor can it be shown that the occurrence of scurvy when fresh milk is thus used is due to the degree of dilution rather than to the patent food, for in some of my cases the milk had been used for many weeks in the proportion of not less than two parts of milk to one part of water, and in others with equal parts of milk and water. The extreme rarity of scurvy from plain boiled milk when used even more diluted than this seems to prove also that the scurvy in these cases is not due to the boiling of the milk but to the addition of the patent food. In one of my cases in which scurvy resulted from the use of diluted milk with Mellin's Food the milk had been heated only to about 167° F. for some months, and had been used fresh without even pasteurization for three weeks before the onset of scurvy (the milk was examined chemically and bacteriologically, and certainly had not been sterilized, and there seemed no ground for disbelieving the statement that it had not been heated, except to the usual temperature of the feeds). The antiscorbutic power of fresh unboiled milk is evidently slight, and is diminished or destroyed by heating, but from the rarity of scurvy on boiled milk compared with the fact that boiled milk is probably the commonest of all infant foods, it would seem that milk has but little tendency to produce scurvy if merely heated to the boiling-point for a few seconds, as is commonly done.

In order to prevent the occurrence of scurvy in an infant fed upon some scurvy-producing food, it is advised to add some antiscorbutic food to the diet, and for this purpose raw meat juice, orange, or grape juice are commonly used. It might seem hardly necessary to point out that the meat juice must be from fresh meat, and probably from uncooked meat ; but I have found in at least three instances that some patent meat juice, or one of the concentrated meat preparations which are so widely advertised, was being given with the idea that it would prevent or cure scurvy. As far as I have seen, such preparations are entirely worthless as antiscorbutics. The red gravy from cooked meat

was shown to be ineffectual in one of my cases where the child was having one tablespoonful of this red gravy daily as an addition to its diet of Allenbury Food No. 2, with one feed of baked flour with water and a little cream. In another case occasional feeds of beef-tea had not prevented the onset of scurvy. Even raw meat juice, unless given sufficiently freely and regularly, may not prevent scurvy. In one of the most severe cases raw meat juice had been given for five months before the disease began; and in the only case in my series which arose on peptonized milk the infant was having a teaspoonful of raw meat juice three times a day on every alternate day, then, owing to some looseness of bowels, it was omitted fourteen days, by the end of which time the infant showed well-marked scurvy.

Fruit juice, though probably a more powerful antiscorbutic than raw meat juice, is not an unconditional safeguard. In the case already mentioned where red gravy of cooked meat was being given with the Allenbury Food, the juice of two black grapes had also been given daily for four and a half months before the scurvy began; in another case where scurvy had been recognized the juice of four grapes had been given daily for fourteen days with no apparent improvement (on potato there was definite improvement in two days).

No doubt this failure of fruit juice depends upon insufficiency of dose; but it may be that this insufficiency is relative, not absolute—that it requires a larger amount of fruit juice to prevent the scorbutic tendency of some foods than of others. The superior potency of potato as an antiscorbutic seems to me quite certain, but as I have almost always given also raw meat juice or orange juice my own results do not illustrate this point, and some of my cases have shown very rapid improvement on orange juice alone. While, however, potato seems specially valuable in the treatment of scurvy, it is less suitable for prophylaxis, for although it is usually tolerated well for a few weeks during treatment it is more apt to cause digestive disturbance than is fruit juice, and is therefore less suitable for prolonged use as a regular addition to the diet.

The potato is prepared by boiling or steaming in the ordinary way with care to obtain a floury potato; the outer floury portion is then scraped off and beaten up thoroughly with enough milk to make a smooth cream, sufficiently thick to pour out of a jug rather heavily—potato 2 heaped teaspoonfuls (with average 2 drachm teaspoon) to 1 ounce of milk; 1½ to 2 teaspoonfuls of this potato cream are given three or four times daily; after two

or three weeks the dose of this should be gradually reduced, and omitted altogether within four weeks from the commencement of treatment. The mode of giving the potato cream is of some importance ; most infants take it best mixed up with the ordinary feed, but if this is done it should be mixed with a portion only of the feed to ensure the whole of the potato cream being taken, otherwise, if part of the food is left, the child does not get the full dose of potato. Occasionally it is taken more readily given separately ; in either case it is often disliked at first, but I have rarely had any serious difficulty in getting it taken. In addition 2 teaspoonfuls of raw meat juice may be given three or four times in the twenty-four hours, and sometimes ½ teaspoonful of orange juice two or three times a day, but any looseness of the bowels should make us cautious in adding this to the potato cream, for diarrhœa in infantile scurvy is a serious complication, as Glisson seems to have observed. At the same time, the child is placed on a diet of milk which has been heated just short of boiling-point and diluted with water ; the scurvy-producing 'food' is of course stopped.

The necessity for moving and handling the infant as little as possible is obvious ; the pain caused by pulling on socks, shoes, and tight sleeves, and by bathing is so evident and so piteous to behold that it is only humane to have the infant wrapped in loose clothing and let him lie undisturbed as much as possible ; the duration of the tenderness and pain is only a few days after proper treatment is begun, so that all ordinary dressing and bathing and outing may well be in abeyance until all tenderness and pain have gone.

CHAPTER IX

FLATULENCE AND COLIC IN INFANCY

How often we hear that a baby is ' always crying ', or has ' the screaming convulsions ', a complaint which at that age usually means colic from some cause or other. Colic in an infant is never to be regarded as a trivial symptom ; it is easy enough for us to assure the parents that it is of no serious significance, and that it means simply flatulence, or a little curd in the stool, but those who have to be continually with the infant know best how extremely distressing is the uncontrollable and violent screaming which may last for an hour or more in spite of every effort to soothe the child. Moreover, colic in an infant is of consequence quite apart from the distress which it causes to the parents, for it will be found, as a rule, that the infant who screams much with colic is not gaining weight as he should do; moreover, in those who are predisposed by neurotic inheritance to convulsions, the abdominal discomfort which is evidenced by colic is very apt to give rise to a convulsive attack, and in very young and feeble infants colic may produce serious collapse. The attack of screaming is followed by grey pallor of the face with a pinched appearance of the nose, the extremities become blue and cold, the pulse becomes very feeble, and the infant sinks back exhausted.

There are three common causes of colic in infants—flatulence, constipation, and undigested food in the intestine ; and even though it may be obvious from the history that one or other of these is present, it is well to examine the abdomen in every case, for valuable information is often to be obtained in this way. If the colic be due to flatulence, the treatment required will vary according to the position of the flatulence, it may be gastric, it may be intestinal ; and a glance at the abdomen may show at once where the trouble is, the distended stomach may stand up in bold relief, or the colon or small intestine may be seen and felt to be distended with gas. Obviously it is useless to give enemata if the flatulence is of gastric origin ; and equally useless to give gastric carminatives or to wash out the stomach if the colic is due to gas in the intestine. For instance, I saw in consultation

a feeble infant aged eighteen days, who had apparently abdominal distress leading to convulsions and leaving the child blue and collapsed. One might have ordered some bromide as an enema off-hand, but this would have been bad treatment, for inspection of the abdomen showed that the stomach was quite ballooned and tense with gas, whereas the intestine showed little or no distension; gastric lavage, with bicarbonate of soda solution (gr. iv to the ounce) was advised, and the infant made a good recovery.

It is remarkable how much distress and disturbance is produced in infants by flatulence. Gastric flatulence often leads to vomiting after every feed; the infant belches up the gas and food together, and in this way nutrition may suffer. It is the usual cause of hiccough in infants, and hiccough, although usually of little importance, may become serious if it occurs often and lasts long in feeble infants, for it exhausts them considerably. When the gastric distension has been considerable, I have seen it so hamper the respiration that the infant grunted with the difficulty of breathing and lay with the head retracted like a case of meningitis, evidently to give the muscles of respiration as free play as possible.

As in older children, gastric flatulence is also one of the causes of sleeplessness, and I think not an uncommon one in infants.

Again, on palpation of the abdomen, scybala may be distinctly felt in the colon, indicating the need for immediate use of an enema. In such cases an injection of warm olive oil, say half an ounce, followed by 3 or 4 ounces of warm soapy water, will often give immediate relief.

But palpation of the abdomen may reveal something more serious than flatulence. The possible presence of intussusception is never to be forgotten in the case of an infant who has suddenly started screaming without obvious cause, and it is no easy matter to exclude the possibility at once in some cases. More than once I have been called to an infant who had been screaming violently for an hour or more, and had evident pain in the abdomen, which with the presence of mucus and some vomiting, and even a trace of blood in the stool suggested that there might be an intussusception, and it was only after the most careful watching and examination that one could make a diagnosis.

I was called to see a female infant aged four months: she was being fed on Savory & Moore's Food: two days previously at 10 p.m. she had suddenly begun to scream, and continued screaming for two hours. A hot bath had been given and an enema administered: the child was then

sick, and at 8 a.m. the next morning, after vomiting green material, she passed some blood and mucus with only a trace of fæces. Vomiting continued throughout the day and at 5 p.m. she again passed mucus stained with red blood, with little or no fæcal matter. During the next night she was restless, waking frequently and crying out with pain in the abdomen. I saw her about midday of the second day, i. e. about thirty-eight hours after the onset of the attack, the bowels had been open twice that day, the first stool consisted only of mucus with a trace of blood, the second of similar blood-stained mucus, and a trace of green fæces. There had been only slight vomiting in the previous eighteen hours. The infant was small and pale, but did not look extremely ill; she lay for a few moments quiet and then began to scream in evident pain. Palpation of the abdomen was impossible, the infant held it rigid and screamed continuously as long as any attempt was made to feel the abdomen and at most other times. One was loathe to give an anæsthetic, for the cessation of vomiting, and the appearance of fæcal material in the last stool, and the duration of the attack in so young an infant without more evidence of extreme collapse, all seemed to tell against a diagnosis of intussusception, so I waited an hour or more to see if the infant would fall asleep, which she did eventually, when I was able to palpate the abdomen and failed to find any tumour. An enema of warm olive oil with 4 ounces of soap and water brought away a large quantity of mucus, no fæcal material: white wine whey was ordered for feeding, to alternate with very weak peptonized milk: the symptoms steadily diminished and the child made a good recovery.

I suspect that occasionally, perhaps more often than we imagine, there really is an intussusception which undergoes spontaneous reduction. When one sees the slight intussusceptions which are so frequently found at autopsies on children, sometimes several in various parts of the small intestine, one can well suppose that occasionally a similar condition might occur during life; it is true that these intussusceptions of the dying are only in the small intestine and nearly always in the reverse direction to those met with clinically, but sometimes they are in the same direction: and it is noteworthy that like the ordinary clinical intussusception, they are much commoner in infants than in older children. It seems likely enough that in the small intestine with its smooth and even wall, an intussusception may occur more easily and reduce itself more easily than when it is of the ordinary ileo-cæcal or ileo-colic variety. The sacculated and thicker wall of the large intestine may tend to prevent the occurrence and spontaneous reduction of an intussusception, but even here there is no doubt that spontaneous reduction does occur sometimes.

In a child aged $2\frac{4}{12}$ under my care at King's College Hospital, the typical symptoms of intussusception, including an obvious sausage-shaped tumour, had occurred three times at intervals of eight months, and ten days. On the first two

occasions the severe symptoms of vomiting, colic, constipation, and passage of blood and mucus, all passed off and the tumour disappeared without surgical interference, although on the second occasion the child was so extremely bad that the surgeon decided against operation only because the intussusception had been present two days, and there seemed no hope that the child would stand the severe shock of a bowel resection. On the third occasion acute peritonitis occurred with the intussusception, and the child died ; a large intussusception was found with its layers only slightly adherent, so that it had probably only occurred quite recently. The case was evidently one of relapsing intussusception.

My colleague, Mr. Kellock, tells me of an infant upon whom he operated at Great Ormond Street Hospital for the same affection. The usual symptoms had been present, including the tumour, but on opening the abdomen, the lower part of the ileum was found to be much congested, as if it had recently been constricted by an intussusception, which had already disappeared.

I do not quote these cases as any argument in favour of postponing operation where there is clear evidence of intussusception, as a general rule delay in such cases is most dangerous, but I mention them because they support, I think, the view that slighter intussusceptions perhaps of the ileic variety may occasionally be the explanation of severe screaming in an infant who is vomiting and passing blood and mucus with little or no fæcal matter, and yet gradually recovers.

Any screaming in an infant which suggests abdominal pain calls for examination of the stools. A stool with lumps of white curd in it or a green stool with much mucus, may point to indigestion as the cause of colic ; there is a particularly close relation between abdominal pain and the presence of much mucus in the stools ; I think the pain in such cases is probably due to the mechanical difficulty of driving the tenacious viscid mucus along the intestine, which consequently makes violent efforts at peristalsis, and so produces the abdominal pain.

Occasionally the stools may reveal some gross fault of diet : I was puzzled by an infant of twelve months with symptoms of intestinal disturbance without apparently sufficient cause, but a few hours later examination of the stools cleared up the mystery by demonstrating the pips of red currants which the nursery maid had been giving surreptitiously, in addition to further evidence of nursery carelessness in the shape of a collar stud, two solid knobs out of the baby's rattle, and a long hair !

Again the presence of gaseous fermentation with resulting flatulence and colic in the intestine, may sometimes be evident in the frothy character of the stools. The hardness and dryness of the stools which, as I have sometimes been told, 'rattle like marbles in the chamber,' may also account sufficiently for colic.

Investigation of the diet will be necessary in every case; abdominal pain in infants is usually the result of some fault in the method of feeding, or in the food itself; *too large feeds* are often being given, with the result that the persistently over-distended stomach falls into a state of chronic catarrh, if indeed this be the correct interpretation of the flatulence which so often marks the dyspepsia of these cases. *Too frequent feeding* is another common cause for colic: and this applies to breast-feeding quite as much as to hand-feeding. Indeed, apart from the overtaxing of digestion and resulting flatulence and dyspepsia which result from too frequent feeding, whether with substitute feeding or with breast-milk, there is in the case of the latter a special reason why this fault should produce colic: any excessive shortening of the intervals between milking whether in the cow or in the woman is found to increase the proportion of curd in the milk.

Too much carbo-hydrate is a fruitful source of flatulence and colic. There is not sufficient attention paid to the proportion of sugar in infant-feeding: 'put some sugar in the feeds,' is not sufficient direction to a mother or nurse, who very naturally puts in as much as she thinks will sweeten the food sufficiently to gratify the infant's taste; as if it mattered not the least whether the food contained 5 per cent. or 10 per cent. of sugar. Fortunately, on the whole, the tendency is to add less sugar than Nature allows: human milk contains 7 per cent. of sugar. Cow's milk contains 4 per cent. before it is diluted; a very ordinary addition to a mixture of cow's milk and water is a teaspoonful of sugar, a level teaspoonful of which, in 3 ounces of fluid, means the addition of 5 per cent. of sugar to the fluid, so that with any dilution of cow's milk, there is not likely to be much excess of sugar, if only one teaspoonful is added; but if a heaped teaspoonful or more is added, the allowable maximum of 7 per cent. will be exceeded.

Far more often the excess of carbo-hydrate is being given in the form of the products of malting. Many of the best known patent foods, which contain no starch whatever, are open to objection on this account: the Allenbury Foods, Nos. 1 and 2, for instance, contain when diluted according to the maker's directions

10 to 11 per cent. of carbo-hydrate; Horlick's Malted Milk, as given, often contains about 12 per cent.; Mellin's Food, which is intended to be an addition to milk, consists almost entirely of soluble carbo-hydrates (about 70 per cent.) and therefore, although starch-free, too much of it may cause flatulence and colic; used in accordance with the maker's directions, seven heaped teaspoonfuls to 7½ ounces of milk, and 2½ ounces of water, for an infant of six months and over, the total proportion of carbo-hydrate in the mixture, if the heaped spoon contains, as I found it to do, 85 grains, would be 12·5 per cent.

If a carbo-hydrate food be used, whether it consists of soluble carbo-hydrate like Mellin's, or of insoluble carbo-hydrate or starch, as in so many patent foods, allowance ought to be made for this in calculating the amount of sugar, if any, to be added. In the case of Mellin's food, it is safer to add no sugar, for even with one level teaspoonful of Mellin's in a three-ounce feed the percentage of carbo-hydrate will be so nearly correct that any further addition is almost certain to mean excess.

Experience shows that there are some infants who will not only tolerate but thrive upon a food containing sugar or other carbo-hydrate in a proportion somewhat above that present in human milk: but, none the less, to give an infant a food which contains more than 7 per cent. of carbo-hydrate is always to run a risk of slimy loose stools with flatulence and colic; certainly one of the common causes of pain in the abdomen in infants is the use of starch-containing foods, which set up fermentation and so produce either flatulence or a mucous catarrh in the intestines. I have already considered these foods in Chapter V, here I will only say that in some infants the merest trace of starch in the food seems sufficient to cause abdominal discomfort, and for this reason even barley-water must be looked upon with suspicion when the cause of colic is in question.

Too much fat in the food is no doubt a much less common cause of abdominal pain in infancy than excess of carbo-hydrate, but I have seen cases which strongly suggested that the pain was due to excessive addition of cream, and where breast-milk has caused screaming, the proportion of fat in it has sometimes been found unusually high. However this may be, there is no doubt that cream, even when carefully proportioned, is very apt to cause digestive disturbance, and when colic in infants is due to indigestion it is often wise to diminish cream or omit it altogether for a time.

Insufficient dilution of milk is a common fault in infant-

feeding, and the undigested curd which results therefrom is one of the causes of abdominal pain and screaming. Even with breast-feeding, the proportion of curd may be higher than the infant can digest; I have found, when breast-milk was disagreeing, the proteid of human milk as high as 3 per cent. instead of the usual bare 2 per cent., and it can be understood therefore why the administration of some plain water just before suckling sometimes relieves colic due to this cause.

Gross faults in the diet may require correction ; the infant who at six months old is having odd scraps of ' unconsidered trifles ' at its parents' meals, is likely to have abdominal pain. I sometimes find that bananas are being given to quite young infants, no doubt in consequence of the popular fallacy that bananas are easily digestible : whatever theory may suggest, experience shows that bananas are very badly digested by infants and young children, and it is no matter for surprise if an infant cries with abdominal pains when such food is allowed. As we all know there are infants who will take and apparently suffer no ill effects from food which causes serious digestive disturbance in most infants, but this is no argument for sanctioning such food for routine use.

Lastly, I must point out that although colic in infants is a gastro-intestinal disorder in the very large majority of cases, it is possible that occasionally it may be of quite different causation. In examining the bodies of infants I have occasionally found small concretions in the pelvis of the kidney, the size of millet seed or slightly larger, and any one who frequently examines the diapers of infants must be familiar with the deposits of uric acid which are sometimes to be found on them.

Treatment

In considering the causes of abdominal pain in infants I have indicated the lines upon which treatment must proceed ; in the majority of cases some fault in feeding will require correction, but here I must emphasize a point of some practical importance ; a baby's digestion, once upset by feeding unsuitable for its age, is often not to be won back to better ways by simply substituting such feeding as would be correct for a healthy infant at the same age : it is usually necessary to give food which is considerably weaker in strength and less in quantity than would naturally be given to an infant of that age ; and if milk is being used the screaming will generally be stopped most

quickly by giving a very dilute peptonized milk without added cream for a few days. I have often found that the mere discontinuance of barley-water causes rapid diminution of the flatulence and colic : and in the slighter cases the addition of sodium citrate (3 grains in a drachm of water) to each feed is sometimes sufficient.

I must not repeat here what I have said elsewhere on the methods of overcoming curd-indigestion, and on the proper adaptation of milk to the needs of an infant (vide Chapters III and IV) but it must be remembered that the proper application of the general principles of infant-feeding constitutes the most important part in the prevention and treatment of colic in infants.

There can be no doubt of the value of the ordinary carminatives for abdominal pain due to gastric flatulence. I am fond of the Tinct. Carminativa, of which one or two minims can be given in an alkaline mixture; for instance, Sodium Bicarb. gr. ij, Tinct. Carminativa (B. P. C.) ℥j, Glycerin ℥v, Aq. ad ʒj, three or four times a day, ten minutes before a feed: or one minim of sal volatile, or a minim of Spirit. Ammon. Fœtidus, or five minims of Tinct. Cardamomi Co. with one minim. of Spirit. Chloroformi may be used instead of the Tinct. Carminativa. The carbonate of magnesium may do more good than the bicarbonate of soda in these cases, 2 grains of either making a suitable dose for an infant up to a year old: or the sulpho-carbolate of soda, one grain for an infant under one year, may be used in the hope that it may exercise some antiseptic effect in preventing fermentation. Creosote also in doses of one-eighth of a minim is sometimes very effective for this purpose. Bismuth is, I think, distinctly valuable if gastric flatulence is very constant ; three or four grains of the carbonate with two grains of bicarbonate of soda suspended by a couple of grains of compound tragacanth powder in a drachm of dill-water makes a suitable prescription, but bismuth is only to be used for a few days to relieve the abdominal pain when very frequent ; its continued administration is only likely to aggravate the constipation which so often accompanies and increases the distress.

Papain gr. j administered before every alternate feed is sometimes useful, and I have known Taka-diastase in small doses to give decided relief.

Nux vomica is a rational therapeutic in cases where the stomach seems to become so distended with gas that it has difficulty in expelling it, and as a matter of experience I think

it is distinctly useful in doses of quarter of a minim for an infant up to six months, and half a minim from six months to one year.

There is a small point which may be worth mentioning in connexion with prescribing for these cases of flatulent colic in infancy; it applies also, only perhaps in less degree, to children past the age of infancy, namely the choice of a sweetening agent. It is, I think, well to avoid syrups as much as possible; the amount of sugar contained in the syrup, though small, may be quite enough to favour flatulence in an infant who already has flatulence and colic from inability to digest the carbo-hydrate in the food; glycerine is not open to this objection, and chloroform-water or the spirit of chloroform (ɑ)i–ij), used with or without glycerine, has the advantage that it not only sweetens but also exerts some anti-fermentative action in the stomach.

But now, what is to be done when the attack of colic is on, and the baby screaming with pain and drawing his legs up in evident abdominal distress? By frequently changing the infant's position, letting him be for a minute or two on his stomach, over the nurse's shoulder, then on his back upon her lap, then in the sitting position, and so on, the eructation of wind may be assisted, or the application of hot flannels or a piece of spongiopiline, wrung out of hot water, to the abdomen may give temporary relief. For gastric flatulence a drop of sal volatile, or 5 to 10 drops of brandy, according to the age, in a teaspoonful of warm water, or, better, dill-water, may ease the distress. I have often found salad oil useful in these cases, about 20 drops with half a teaspoonful of warm water and 1 drop of sal volatile. If a feed is to be given, sherry whey is worth trying; it suits some of these cases excellently, having a very definite carminative effect. Whether there is constipation or not, a warm enema of plain water or of warm olive oil, followed by 4 to 6 ounces of water, has often a remarkable effect in rapidly soothing the infant. Lastly, and I put it last because I think it is best avoided if possible, opium in some form may be not only permissible but advisable, when the infant has been screaming for a long time, perhaps an hour or more, for the resulting exhaustion may be serious in a feeble infant. Dover's Powder, in doses of $\frac{1}{8}$ grain at three months, $\frac{1}{4}$ grain at six months, $\frac{1}{2}$ grain at one year, is perhaps the most convenient and effectual form.

CHAPTER X

INFANTILE MARASMUS

Marasmus, or wasting, is a symptom rather than a disease, and like most symptoms it may result from many different causes.

I begin with this platitude because I think there is a tendency to jump to the conclusion that whenever an infant is wasting there must be something wrong in the food, and that all that is required is to change the food and keep on changing it until the weight begins to rise. Undoubtedly digestive trouble from unsuitable feeding is by far the commonest cause of wasting in infancy, but even so it does not follow that the food requires changing ; it may be that the fault is not in the food, but in the manner of feeding, particularly in the frequency or the size of feeds ; moreover, there are other cases by no means infrequent in which the wasting is not due to any fault in the food or in the manner of feeding, but to some cause in the infant such as chronic constipation or congenital syphilis.

The causes of wasting in infancy—and under the term wasting I shall include failure to gain weight as well as actual loss of weight—fall into one or other of these two categories, faults in the food or feeding, and faults in the infant.

The commonest of all causes is unsuitable food, but I would emphasize the fact that in the large majority of cases marasmus is due not to any gross impropriety in the food, but to some slight excess or deficiency in one or other of the constituents of an ordinary milk mixture.

Occasionally it is true the fault is gross : for instance, a female infant, aged ten months, was brought to me at hospital for wasting : at the age of about eight months she had been fed on fish, greens, and potatoes ; then, owing to symptoms of indigestion, she was fed for a whole month on nothing but barley-water, a pint and a half with half an ounce of brandy daily : what wonder that she was emaciated ! Fortunately such gross mal-feeding is quite uncommon.

Far more often the fault is insufficient dilution of cow's milk in the early months of life. The infant who is given equal parts

of milk and water at a month old is likely to waste : and a mixture of two parts of milk to one of water at three months may have the same result. I am well aware that there are infants who will tolerate and thrive on undiluted cow's milk almost from birth, and I have used this method of feeding with good results, but in my experience its success is the exception, not the rule ; and I have no hesitation in saying that marasmus is more often due to insufficient than to excessive dilution of milk. As a general rule the proper dilution is equal parts of milk and water at three months, two parts of milk to one part of water at six months, and three parts of milk to one of water at nine months ; the transition from one strength to another is to be made of course gradually.

Another common fault which is responsible for wasting is an excessive proportion of cream. An infant may tolerate an average proportion of 4 per cent. of fat in human milk, but the cream from cow's milk is less easily borne ; there are many infants who cannot digest a mixture containing more than 3 per cent. of this fat. Often I find that a wasted infant has been given two teaspoonfuls of cream in a feed of 3 ounces, consisting of equal parts of milk and water : no attempt has been made to ascertain the strength of the cream, and this proves to be the ordinary 48 per cent. cream, the percentage of fat in the mixture therefore is 5 to 6 per cent. ; and after gaining weight rapidly perhaps on this mixture for a time, the infant has begun to waste.

Herein lies the importance of a practical working knowledge of the composition of milk and of the ordinary foods in common use for infant-feeding. Percentage analysis may be and often is a very incomplete guide to the choice of food, but it gives at any rate one means by which we may compare any food with the ideal standard, human milk, and with the standards of artificial feeding which experience has shown to be most generally successful. Nature is lenient in her requirements, exacting no slavish adherence to decimal points of percentage as some would have us believe, yet there are fairly well-defined limits of variation which must be observed if an infant is to thrive. To give an infant a food which contains less than 2 per cent. of fat after the first three months of life is to incur the risk of rickets, on the other hand to give a food containing over 5 per cent. of fat is to expose the infant to risk of gradual failure of assimilation, if not to acute digestive disorder. So, too, with the sugar ; any excess of sugar is likely, sooner or later, to bring flatulence and discomfort, with loose or slimy stools and wasting. It is remark-

able how little sugar seems to be necessary, but nutrition is likely to be poor where the sugar percentage falls much below 5 per cent., while, on the other hand, sugar above 8 or 9 per cent. is very apt to cause digestive disturbance, especially flatulence and colic. In the proportion of sugar is to be included any other carbo-hydrate, whether soluble, as in the products of malting, or insoluble, as starch. But starch, in my opinion, should not enter at all into the diet of an infant under nine months of age ; it is quite certain that the small proportion of starch in an ordinary mixture of milk diluted with barley-water, that is to say, 0·5 to 1 per cent. of starch at most, is commonly taken without ill effects, but as I have already pointed out, even this small proportion sometimes causes disturbance, and certainly in large proportion, as it is present in the majority of starch-containing patent foods, starch is a potent factor in exciting a chronic dyspepsia which may result in considerable wasting. These are the cases that every medical man is familiar with, the infants whose big tumid abdomen, contrasting strangely with the thinly covered chest and wasted limbs, suggests the possibility of some tuberculous disease of mesenteric glands or peritoneum.

In hospital practice feeding with condensed milk is often responsible for marasmus ; in these cases the fault is usually in the excessive dilution. An extreme example of this was the feeding of an infant aged three months, who was brought to me for wasting : condensed milk was being given in the proportion of 4 teaspoonfuls to 2 pints of water, which would yield a mixture containing at most, proteid, 0·5 per cent.; fat, 0·7 per cent.; sugar, 2·75 per cent. : practically nothing more than a weak solution of sugar in water. Even when diluted most carefully (an average teaspoonful to 3 ounces of water), so that the proportions become.

Proteid	1·2 per cent.
Fat	1·5 per cent.
Sugar	6·0 per cent.

it is clearly not suitable for infant-feeding beyond the first few weeks of life. If excess of sugar is to be avoided in the ordinary sweetened condensed milk it must be diluted so that the proportion of proteid and fat becomes quite insufficient.

Fresh cow's milk is sometimes given too much diluted : I see infants who, at the age of six months and sometimes older, are having equal parts of milk and water, or, as more often happens, equal parts of milk and barley-water; for most parents cherish a mistaken idea that in giving barley-water they are supplying

a valuable food which makes the proportion of milk quite a secondary matter, and the result is an ill-nourished infant with rickets. If an infant cannot tolerate the curd of a milk mixture stronger than this at six months, either cream must be added to the mixture, or the use of a stronger mixture must be made possible by adding sodium citrate or by peptonizing the milk.

The manner of feeding is sometimes more responsible than the food for wasting in infancy. Even with breast-feeding irregularity in the intervals of suckling may be sufficient to disturb digestion and produce marasmus, so, too, does feeding 'whenever he cries', which generally means that the infant is taking milk almost every hour or even oftener. It is always a wonder to me how mothers—who know well enough that if they themselves took meals haphazard, sometimes at short intervals sometimes at long intervals, they would suffer very quickly with indigestion—expect their babies, whose digestion is so much more easily upset, to digest and thrive when they are fed in this irregular manner.

Even with regularity the intervals may be wrong; too infre quent feeding is very rare, occasionally I see infants who are being fed only three times a day when they should be having food every three hours; but this is quite unusual, more often one sees marasmic infants who are being fed with the full amount for the age, at intervals of two hours or less, when the interval ought to be three hours: to feed an infant of three months with 3 or 4 ounces every two hours is to overtax digestion, and induce marasmus. Even when it is necessary, as it often is, in various diseases to reduce the size of feeds considerably below the normal amount for the age, and when on this account the feeds must be given more frequently than to a healthy infant, I think that it is always desirable to lengthen the intervals of feeding as soon as it is possible.

Another cause of marasmus which I have mentioned elsewhere but shall mention again here, for it is not, I think, generally realized, is the use of feeds too large for the age. An infant is given 2 ounces when he is a fortnight old, at a month or six weeks old he is getting 3 ounces, and by the time he is three months old his feeds are perhaps 6 ounces. Now what happens? At first the weight rises rapidly; at three or four months old the infant is a theme of admiration, his weight is considerably above the average and everything seems *couleur de rose*; then comes a pause, the weight is not

rising; then it begins to fall; there is perhaps no marked evidence of digestive disturbance, the stools are normal or almost normal, there is no vomiting, but clearly the infant is not thriving, and by degrees he falls into a state of marasmus, from which it is extremely difficult to extricate him; indeed, I regard these cases of marasmus from too large feeds as some of the most difficult to treat.

Of course there must be some elasticity in the size of feeds, for a large infant will require more than a small infant of the same age, but it is seldom wise to exceed the amounts which I have already mentioned (p. 79) as suitable at each month.

And here I will insist again upon a point which I think is sometimes forgotten, that an infant may gain weight steadily and even unusually rapidly for a time upon feeding which is nevertheless disastrous in its subsequent results: witness the rapid gain in weight which infants often make upon foods containing starch or an unduly high proportion of sugar; the result of which, after months of apparent thriving, is rickets, or failure of digestion with consequent marasmus.

Excess of cream may have a similar effect: at first it causes a large gain in weight and the infant seems to be thriving admirably, but after a few weeks or months, with or without obvious signs of digestive disturbance, the power of assimilation is exhausted and the weight begins to fall.

The question whether a particular food or mode of feeding is suitable for prolonged use, must be determined not merely by the immediate results but by its subsequent effect as proved by the evidence of experience in a large number of cases.

The cause of marasmus, as I have already said, may lie rather in the infant than in the feeding, and if the term marasmus could ever be properly used to signify a disease rather than a symptom it would, I think, be in certain cases, which seem to form a group by themselves, as lacking any sufficient explanation for their wasting. These are infants who either from birth or from a few weeks after birth begin to lose weight in spite it may be even of breast-feeding, and steadily go downhill or pass through many vicissitudes in the way of feeding, before they slowly emerge from a parlous state of feebleness and marasmus into a healthy state of nutrition. Such a condition sometimes occurs in successive children in the same family about the same age: each infant, after it reaches the age of, perhaps, two or three months,

begins to waste without any other evidence of digestive disturbance, and without any apparent fault in the feeding.

For these cases, as I say, we have no satisfactory explanation. I have sometimes been tempted to apply to them the theory of Sir William Gower's ' abiotrophy ', which supposes some tissues to be endowed in certain individuals with less than the ordinary share of durability ; it would seem, in fact, as if certain infants have their function of assimilation endowed with so little staying power that after a few weeks' use it falls gradually into abeyance, and the result is intractable marasmus. Some of these infants are feeble from the time of birth, and it is evident that the power of assimilation, like the other functions, suffers from the general feebleness and lack of vitality.

A clinical fact which, perhaps, throws light upon some of these cases is the remarkable difficulty which certain infants show in digesting particular constituents of food : this idiosyncrasy may apply to fat, to sugar, or to proteid, and is seen in connexion with breast-feeding as well as in artificial feeding. One infant will thrive perfectly on the ordinary proportion of proteid and sugar, but directly the fat in the food exceeds a very low proportion (perhaps 1·5 or 2 per cent.) there is digestive trouble ; another infant will thrive on the ordinary proportion of proteid, but seems unable to digest the ordinary proportion of sugar. It seems reasonable to suppose that in some cases the idiosyncrasy may apply to more than one of these constituents, so that nutrition is difficult, perhaps even impossible. However this may be, it is I think clear that there is a group of cases in which the marasmus is due to a primary failure of the processes of assimilation, whether for particular food-stuffs or for food in general.

A very common group of cases is that in which marasmus follows upon a more or less acute gastro-intestinal disturbance, in which there has been diarrhœa with or without vomiting. Every autumn many such cases are seen after the prevalence of summer diarrhœa. The stools are no longer watery or particularly frequent, but they are green and slimy, and contain white pellets of undigested fat or curd, or are simply unduly pale or almost white throughout. The infant has gradually become emaciated and miserable, and food after food has been tried without arresting the gradual decrease in weight. Some of these cases resist all treatment, and it is at least a plausible suggestion that in them there is some profound alteration in the lining membrane of the intestine which interferes with absorption. Various observers have described such changes : a small cell infiltration of the deeper parts of the mucous membrane is said to give rise to fibrous replacement of the glandular parts of the

mucosa, so that a cirrhosis of the absorptive tissues results, and if such a change were extensive, nutrition would be impossible; as a rule, however, it is said to occur only over small areas of intestines, so that there is enough absorptive surface remaining to support nutrition. But even if such a change does really occur in some cases—and some observers have disputed its occurrence in the belief that the appearances described might have been due to post mortem change—it is not necessary to suppose that it occurs in all; a simple disturbance of function, an intestinal dyspepsia, may be the explanation of the marasmus in these cases.

A cause of marasmus which is much more easily overlooked than might be supposed is congenital syphilis, for there may be no other manifestation of the disease at the time, and it may be only by a careful inquiry into family history, or by the statements of the parents, that the syphilitic origin of the marasmus can be ascertained. Usually the marasmus in these cases dates from birth; the infant was a 'fine baby born', but has steadily wasted since. In most cases other symptoms of syphilis show themselves sooner or later, but certainly it is not right to wait for these before administering mercury, even if there be only ground for suspecting syphilis. Unfortunately even free administration of mercury seems to have no good effect in some undoubted cases of syphilitic marasmus; there are, however, cases in which nutrition improves rapidly when this drug is given; so that the diagnosis is a matter of great importance. The occurrence of severe marasmus as a result of congenital syphilis is strong reason for remembering Colles's law that the mother of an infant with inherited syphilis, even though she has shown no signs of the disease herself, does not contract the disease from her infant. There is, therefore, no justification for stopping the mother from suckling her infant because he has congenital syphilis; on the contrary, it is highly important that the infant should not be weaned, for if to the syphilitic marasmus there is added the marasmus which arises so often from digestive difficulties with hand-feeding, the child's chance of survival will be by so much the less.

Where the possibility of syphilis is in question, a Wassermann test may give help, but, as a rule, it is unnecessary, for, so far as treatment is concerned, the simplest and most satisfactory course is to give mercury and see whether any good results.

The marasmus which results from congenital syphilis is always of grave prognosis: Dr. Coutts says, in his Hunterian lectures, 'Syphilitic infants with atrophy generally die, whether such atrophy dates from birth or many months later, and whether

treated with mercury or not : ' but ' generally ' is not always ; I have notes of several syphilitic infants who, in spite of great wasting, recovered on mercurial treatment, and although my own experience agrees with that of Dr. Coutts, as to the intractable character of the marasmus in many of these cases, I am satisfied that the infant can sometimes be saved by mercurial treatment. Why an infant with congenital syphilis should waste is not clearly understood ; it seems probable that in some cases there may be gross changes in the intestinal mucosa : Dr. Coutts mentions changes in the mucosa, like those mentioned above as resulting from gastro-enteritis ; changes have also been described in the vessels of the mucosa, apparently a syphilitic endarteritis, which might well lead to degenerative changes in the mucous membrane ; moreover, the production of cirrhosis in the liver and pancreas by congenital syphilis suggests that, short of such a gross lesion, syphilis might affect these organs sufficiently to alter their secretion in quantity or quality, and so affect digestion. The pathology of the syphilitic marasmus must remain *sub judice* : the importance of recognizing its occurrence is beyond question.

There is one cause of marasmus upon which I wish to lay special stress, for it is so often ignored, namely, chronic constipation. The result of this is more often failure to gain weight, or diminution of the rate of progress, than actual loss in weight, but sometimes there is actual wasting from this cause. Where marasmus is due to other causes, chronic constipation may be a contributing factor, and all efforts to improve nutrition may fail until the constipation is remedied. It may be quite useless—nay worse than useless—to try food after food in the hope of hitting upon one which will ' suit the child ', when the one thing needful is regular administration of some laxative drug : and in the minor trouble of unsatisfactory progress in weight it is often the greatest mistake to change the food which may be suiting the child perfectly when chronic constipation is really the cause of the slow rate of progress. The infant whose bowels are open once a day regularly may still be constipated, if the stool is small and dry, and this may be sufficient to prevent the weight from increasing ; but when, as often happens, the bowels open only once in two or three days there is still more likely to be some disturbance of nutrition. And here I will insist upon a practical point which I have mentioned elsewhere, that in these chronic cases of constipation to open the bowels by a glycerine or soap suppository, or by an enema, seems to be less satisfactory in its

results than to open them by some drug administered by the mouth. Most of the drugs in common use for this purpose do more than merely stimulate peristalsis of the colon, as an enema or suppository does; many of them act by promoting the flow of secretions into the small intestine, and thus may serve a valuable purpose as an emunctory, with a much wider influence than is exerted by mere mechanical stimulation of the rectum. I doubt not that some of the successes which follow the use of grey powder in non-syphilitic cases of marasmus are due in part at least to the laxative effect of the drug; and I know of no drug which is more generally useful in combating chronic constipation in infants. If the ordinary powder of hydr. cum cret. gr. ½, sod. bicarb. gr. j, pulv. cretæ. aromat. gr. j, ter die, is not sufficient, a combination of hydr. cum cret. gr. ½–j, pulv. rhei. co. gr. ij–iij, given regularly three times a day, seldom fails to keep the bowels regular, and this can be continued for several weeks, or even months if necessary; but whatever is used it must be given every day regularly in sufficient dose to keep the bowels just working well. The good effect is lost if aperients are only given occasionally when the bowels have already become constipated for twenty-four hours or more.

Another condition which profoundly affects the nutrition of an infant apart from the feeding is congenital heart disease. The 'blue child', with its grey livid skin, blue-black lips, and cyanosed tongue, congested conjunctival vessels, and blue fingers and toes, excites no surprise when it fails to thrive and remains a feeble puny infant. Every medical man recognizes it at once as a case of congenital heart disease, and rightly attributes its poor nourishment to this cause, but the cases to which I wish to direct attention are the much less rare ones in which congenital heart disease is not associated with any cyanosis whatever, and in which the abnormal heart condition is usually discovered only more or less accidentally in the course of routine examination of the chest. I have seen many such cases in which the infant had been under treatment for wasting: there was nothing to call attention to the heart, and it had been assumed that the food was at fault. First one food was tried and then another, but still the infant failed to thrive: examination of the heart showed signs of congenital heart disease. Such cases were the following:

Emily H., aged seven weeks, was brought for wasting; she had been fed on the breast for four weeks, but as she was evidently not gaining weight the mother was advised by a nurse to wean the child: this was done and

the infant was given a mixture of milk and barley-water, but still failing to make progress she was seen by a medical man, who finding the infant much wasted did not examine the chest, but concluded that it was 'an ordinary marasmus' due to digestive trouble, and treated it as such. Subsequent examination shewed that although there was not the least cyanosis, and nothing in the child's aspect to suggest cardiac disease, there was nevertheless 'a loud systolic blowing bruit heard all over mid-portion of heart, loudest in third space and over fourth rib, about one finger's breadth from sternum'.

Ellen T., aged fourteen months, was a fine child at birth but had wasted much since one month old. She had been under medical care and various methods of feeding had been tried, without success; she had become much emaciated, weighing 6 lb. 5 ozs. at fourteen months. Examination of the chest shewed a 'roaring systolic bruit all over præcordium'; its maximum was just to the left of the lower end of the sternum. There was no cyanosis, and no clubbing of fingers: on inquiry it was stated that at times the child had been short of breath.

I could quote several similar cases, but these may suffice to show how easy it is to overlook the underlying cause of marasmus, where congenital heart disease produces little or no symptom beyond the wasting. Why the abnormal condition of the heart in these cases should affect nutrition in this way is not very obvious, but there is, at any rate, a strict analogy in the wasting which is so often a striking feature in the acquired heart disease of older children; the child with chronic rheumatic endocarditis, whether there be any failure of compensation or not, often wastes considerably; indeed, it is no uncommon thing for a child at the school age to be brought to hospital solely for wasting, when auscultation shows well-marked endocarditis, the existence of which had never been suspected as the child had had no symptom of rheumatism beyond occasional 'growing pains'.

Unfortunately the recognition of congenital heart disease in an infant does not give us much help in the treatment of the resulting marasmus, but it does, I think, assist us to some extent. In the first place, it is no small thing to know the cause of any symptom which we are called upon to treat; such knowledge gives confidence to the doctor, and the knowledge that the doctor knows what he is dealing with gives confidence to the parents; it is far more satisfactory to be able to say to the parents, 'the wasting in your infant is not due merely to indigestion or to lack of a suitable food but depends upon the deformity of the heart,' than to try food after food with unexplained failure whilst the parents are disappointed and dissatisfied at what they suppose to be the doctor's lack of skill in 'hitting off' the particular food required. In the second place, recognition of congenital heart disease as a cause of marasmus will prevent the hasty assumption

that because the infant is not thriving the food must at once be changed; in the first case mentioned above the weaning which was done upon the unskilled advice of a nurse was probably the worst possible step which could have been taken. Undoubtedly something may be done for these cases by very careful dieting, particularly in the direction of such easily assimilable food as asses' milk, or peptonized milk, or whey with raw meat juice, or a temporary use of desiccated milk, if human milk is not available; but the results are likely to be much less satisfactory than in the infant whose marasmus is due only to digestive disorder.

Another cause for chronic wasting in infancy, and indeed also in later childhood, which is apt to be entirely overlooked, is chronic pyelitis. I have seen cases in which no suspicion had been entertained that there was anything beyond impaired digestion, when examination of the urine revealed pus-cells and bacillus coli, and suitable treatment of the urinary affection was followed by rapid improvement in nutrition.

I have not mentioned tuberculosis hitherto amongst the causes of infantile marasmus, because it should occupy a place in the background rather than in the foreground of the clinical picture of infantile marasmus. It is one of the rarest of causes of chronic wasting in infants under three months of age, and under six months of age is still so uncommon that it should never be diagnosed lightly. One hears again and again in hospital practice of 'consumptive bowels' as a cause of infantile marasmus, but almost always the condition to which it is applied is one of faulty feeding, and is not related in any way to tuberculosis; the sooner this term 'consumptive bowels' is dropped the better, for it too often confers upon the results of improper and sometimes careless feeding the dignity of a dispensation of Providence, and the mother is led to regard as unalterable and unavoidable the sufferings of an infant which might easily have been prevented, and can still be remedied by proper feeding.

Again, one sees cases in which 'tuberculous peritonitis' has been diagnosed on the grounds of wasting with a distended abdomen; when the condition is entirely due to bad feeding, generally with starch-containing food at too early an age. All degrees of distension of the abdomen may result from this cause, but the characteristic doughy feeling and the firmer masses of caseous infiltration, particularly the transverse band just above the umbilicus, which are found by palpation in matted tuberculous peritonitis, and the shifting dullness of fluid in the peritoneal cavity characteristic of the ascitic form of tuberculous peritonitis,

are entirely lacking in the cases to which I refer, and in their absence we should be very chary of diagnosing 'tuberculous peritonitis', unless indeed there be strong evidence of tubercle in the lungs or elsewhere. In this connexion it may be mentioned that when tubercle does affect infants under six months of age, it very rarely presents the clinical aspect of tuberculous peritonitis; it far more often appears as an acute pulmonary tuberculosis, or a tuberculous meningitis or a tuberculous disease of the ear or of the bones. Amongst 100 consecutive cases of tuberculous peritonitis proved by autopsy at the Children's Hospital, I found not a single case under the age of six months, and only eight under the age of one year.

Whilst, however, tuberculosis is only a very rare cause of chronic wasting in an infant under six months, it must certainly not be taken for granted in any particular case of marasmus that tubercle can be excluded merely on account of age: there are cases in our post mortem records at the Children's Hospital where extensive tuberculosis was present in infants as young as eight weeks, and this emphasizes the need for very careful examination of the chest as well as the abdomen in every case of infantile marasmus. And indeed the examination needs to be careful, for it is remarkable how much gross disease may be present in an infant's chest without producing signs sufficient to suggest any serious affection of the lungs: I well remember a case of infantile marasmus which I treated without a suspicion that it was other than due to simple digestive failure; the infant died, and autopsy revealed extensive tuberculous caseation in the lungs.

Complications. To whatever causes marasmus is due there are certain symptoms and complications which are incidental to it, and which are of practical importance as they may call for special treatment. A feeble wasted infant falls an easy prey to local infections, whether it be in the mouth where thrush (oidium albicans) quickly gains a hold and may set up sufficient inflammation to make an infant unwilling to suck, and so may interfere seriously with the already difficult nutrition; or in the ear, where an acute otitis media, leading to perforation of the drum and discharge of pus externally, is a not uncommon complication of marasmus; or in the skin, where angry-looking 'boils' or small superficial abscesses, the result of a local staphylococcal infection, are apt to occur when wasting is much prolonged and severe. Probably these are to some extent preventable by good nursing; if the mouth be wiped out three or four times daily with some glycerine of borax,

and the teats used on the feeding-bottle are kept in a weak solution of borax, thrush might be prevented, and it seems likely that frequent cleansing of the mouth may also reduce the liability to infection of the middle ear from the naso-pharynx. The skin infection occurs especially on parts where, in addition to sweat or to the contamination of incessant urine, there is local pressure, so that infective material is rubbed into the skin ; in this way superficial abscesses are specially apt to occur on the back of the head, and about the buttocks and thighs ; these are to be prevented by scrupulous cleanliness, by frequent changes of bed-linen and diapers, and free use of the ordinary antiseptic dusting powder (boric acid 1 part, zinc oxide 2 parts, starch powder 2 parts). Where, in spite of all precautions, the abscesses continue to recur, as they sometimes do, for several weeks, an autogenous vaccine is sometimes very effectual.

The temperature in an emaciated infant often remains subnormal for days together, and this I always regard as an unsatisfactory sign ; it means considerable exhaustion, and should quicken our anxiety to improve nutrition, for a very slight additional disturbance, such as a 'little looseness of the bowels', may then endanger the infant's life. I think it is sometimes an indication of the need for stimulants in small doses three or four times a day, either brandy in doses of 5 or 10 minims for an infant under nine months old, or tincture of nux vomica $\frac{1}{2}$ or one minim.

Another symptom of grave significance is the appearance of œdema ; this is by no means uncommon in prolonged marasmus ; it usually becomes definite first in the feet and on the back of the hands, but I have often noticed as its earliest manifestation a watery translucent appearance of the skin of the face, which may soon develop into definite puffiness of the eyelids just as occurs in the dropsy of renal disease ; with this œdema of infantile marasmus the urine shows no albumen whatever. There are certain practical points which are worthy of attention in connexion with the œdema. A sudden rise in weight in a marasmic infant may be due to the occurrence of this dropsy, and so, instead of meaning improvement, may be of evil significance ; dropsy is not necessarily of fatal omen ; I have seen infants with very marked œdema ultimately make a good recovery. Its pathology is very uncertain, but on the view that one factor in its causation is retention of chlorides in the system, it is unwise to use ordinary saline solution for irrigation of the bowel where œdema is present.

I am chary also of using 'citrated milk', i.e. milk to which

sodium citrate has been added to facilitate curd digestion, in any case of severe marasmus, especially if the skin of the face already shows the translucent appearance mentioned above, for I have known its use followed by the rapid onset of œdema.

In one instance a marasmic infant, aged about four months, the child of a medical man, was given an unduly large quantity of sodium citrate, 10 grains in each feed, with the result that after a day or two the limbs became œdematous ; the citrate was stopped, and the œdema disappeared ; the citrate was then tried again, and again the infant became œdematous and lost its œdema when the citrate was stopped. In another instance, the father, a medical man, informed me that after giving sodium citrate in the usual dose, the infant's face became puffy, whereupon he stopped the sodium citrate and the puffiness subsided. Such an occurrence must, I think, be very exceptional, for I have used sodium citrate very extensively, and have only come across these two instances ; but it must be added that I have used citrated milk chiefly for infants with only slight difficulty of curd digestion,—it has not seemed to me to be suitable for the severer cases of digestive difficulty with much marasmus.

Purpura, like œdema, is a symptom of very grave significance in an infant with marasmus ; these two symptoms are sometimes found together. It has always seemed to me probable that both purpura and œdema are dependent upon degenerative change in the vessel walls, which in its turn may result partly from the impoverished character of the blood and partly from feebleness of circulation ; the latter seems evident in many such cases from the blue and cold condition of the extremities which is associated with the œdema and purpura. The purpura occurs most often on the trunk and usually in the form of small petechial spots which might easily be mistaken for flea-bites, from which they differ in lacking the central dark point of puncture which is characteristic of the flea-bites ; but sometimes it occurs as large bruise-like patches which might, as I have known, raise the question of ill-treatment or carelessness in nursing, when the condition was purely due to the marasmus, and no blame whatever attached to the attendants.

Prognosis. The outlook in any case of infantile marasmus depends to a large extent upon its cause. I have already mentioned the intractable nature of some cases of syphilitic marasmus; the cases with congenital heart disease are necessarily serious, inasmuch as the cause cannot be removed, but if the earliest months of infancy can be passed in safety, these cases may live on for

years, frail at first, but gaining strength and nutrition as they grow older. I always regard as highly unsatisfactory the infant who is losing weight with no apparent cause ; these are infants whose feeding is in every way perfectly correct, the stools are of normal appearance, and there is no evidence of gross disease of any sort; such cases will often fail to respond to any treatment for many weeks, or even months, and sometimes, baffling every attempt to improve nutrition, will slowly emaciate and die ; whereas the infant whose feeding has been obviously unsuitable, and whose stools show clear evidence of disturbed digestion, even though he be extremely emaciated, will generally improve when treated with some weak and easily assimilable food.

There are certain risks to which a marasmic infant is specially liable ; as might be expected in cases where there is much difficulty in digestion, there is a special proneness to diarrhœa, and especially during the summer months a marasmic infant is always in danger of an attack of diarrhœa, which may speedily terminate in fatal collapse : another danger is bronchitis or broncho-pneumonia ; it is very noticeable how slight may be the physical signs of pulmonary disease, and how little may be the effect upon the temperature in a much-wasted infant. For instance, Henry C., aged five months, was under treatment for marasmus ; during the fortnight before death the temperature was only once above 100°, and during the six days before death it was subnormal : there had been no sign in the lungs at any time except occasional crepitation, and yet post mortem examination showed in addition to the usual patches of pulmonary collapse definite patches of broncho-pneumonia in both lower lobes.

There is another possibility of which it is well to be forewarned, namely, sudden death. I do not mean by this instantaneous death, but rather a sudden collapse which ends fatally, perhaps in half an hour or in a few minutes. In some cases this sudden termination comes with a convulsion, but in others the infant's condition has appeared to be no worse than usual, and the end comes so quietly and unexpectedly that the infant may be found dead in his cot where he was left but a few minutes before in no apparent immediate danger of death. This has happened several times in my experience, usually in cases of long standing and considerable marasmus, where it was evident that the infant was very feeble, although there was no special reason for expecting a fatal termination at that particular time.

Treatment

The treatment of infantile marasmus embraces the whole subject of infant-feeding in addition to the treatment of the various non-dietetic causes which I have mentioned above. I cannot repeat here all that I have already said as to infant-feeding, but I will emphasize some broad principles of practical importance. When marasmus is dependent upon digestive difficulty, a weak food is commonly the right food ; it is often wise to start afresh with veal broth only or chicken broth, to which milk sugar may be added in the proportion of a teaspoonful in every 3 ounces ; after a day or two milk freely diluted and peptonized may be substituted for the broth at alternate feeds. A more readily assimilable food where even the weak peptonized cow's milk cannot be assimilated is ass's milk, and it is often very remarkable how well a feeble marasmic infant will thrive for a time on ass's milk (given undiluted, for its extreme weakness wherein lies a large part of its virtue calls for no further dilution). Where ass's milk is impracticable on account of expense, one of the various brands of plain desiccated milk is sometimes successful, and in any case may be useful as a temporary food when it is decided to try something a little stronger than ass's milk or much diluted cow's milk. If fresh milk in various dilutions has already failed, a much diluted peptonized milk is often the quickest method of overcoming the difficulty.

Fats are very commonly ill-tolerated by marasmic infants ; the addition of cream, therefore, is very often best avoided altogether, and if any is used it should be given in very small quantities : for the same reason cod-liver oil is often quite unsuitable for the marasmic infant ; it retards digestion and aggravates the trouble.

Lastly, in connexion with diet I would insist upon the need for slowness and deliberateness in increasing the strength of the food. I know only too well how difficult it is to resist the temptation to make the milk a little stronger as the infant seems to be assimilating better and the weight is rising so slowly ; but again and again I have had to repent of yielding to this temptation ; the parents have urged it, the nurse has urged it, and in the hope of hastening the slow but definite progress, we have increased the strength or the amount of the milk mixture, or added some cream, or given some additional food, and what was the result ? Instead of a greater gain, the next weighing has shown a loss, and perchance before we could show any further

progress, it has been necessary to go back to some mixture even weaker than the original. So long as an infant whose weight has previously been stationary or falling is showing even a small progress in weight on some particular feeding, it is usually poor economy to make any change ; when the weight no longer rises, if the stools show that the food is being digested, it may be wise to increase the quantity or the strength of the food ; or it may be necessary to make some other change ; but so long as the weight is rising and the infant seems to be improving, I am convinced that the soundest principle is to ' leave well alone '.

I turn now to another item in the treatment of these cases, and one which I believe to be of no small importance, namely, warmth. In these days of open-air treatment, excellent under proper conditions, but carried, as it often seems to me, to a dangerous absurdity, people are apt to forget how vitally essential warmth is to feeble infants. No one appreciates more than I do the value of fresh air for infants ; there can, I think, be no doubt that in some cases of marasmus the infant's digestion and nutrition improve if he can be taken out daily in a suitable climate ; but to take a feeble marasmic infant out on a cold raw day, so that the child comes in with its hands and feet cold and blue, or to keep him lying in a room with the window wide open, and the room warmed with difficulty to 60° F., is seriously to diminish the infant's chance of recovery. If any one doubts that warmth is of more importance to a puny wasted infant than open air let him gain some experience of the effects of the incubator. I suppose next to breast-feeding there is nothing that contributes more to save the life of an infant born very feeble or premature, than the incubator, and for marasmic infants also it has been found of the greatest value. The supply of fresh air in an incubator is reduced to a minimum, while the temperature is kept at 80° to 90° F. No one doubts that the value of the incubator lies in its warmth, which saves the energy and so maintains the vitality of the infant.

Infants stand cold badly, and much worse when they are already enfeebled by illness ; I have never forgotten the reproof which an aged practitioner administered to me in the days when I was newly qualified, and on a cold day had a sick infant stripped while I examined it all over ; he told me in effect that more harm would result from the exposure of the infant than good from my examination : and though I would advise no one to omit careful examination of any patient he is called upon to treat, I recognize that there are conditions in which all needful examination can be

made and ought to be made with a minimum of exposure, and that one of these is infantile marasmus. There is a great tendency at all ages to coldness of extremities where there is digestive disorder, but in the infant especially this is a very striking feature, and the marasmic infant, unless the utmost care is taken, has often both hands and feet livid with cold, showing that the circulation is feeble, and indicating a condition of depressed vitality which aggravates the difficulty of assimilation. In the case of severe infantile marasmus, I would have the temperature of the room kept at 65° F., and there should be hot water-bottles in its cot (with proper precaution, of course, to prevent the hot water-bottle touching and scalding the infant), and particular care should be taken that the arms and legs are covered with some warm clothing down to the wrists and ankles, indoors as well as outdoors, for it is much easier to keep the hands and feet warm if there is some covering in the form of a woollen sleeve and gaiter for the limbs. In warm summer weather by all means let the marasmic infant go out daily, but in winter there are times when, in my opinion, it is wiser to keep the infant indoors, and to secure fresh air by change of rooms, one room being thoroughly ventilated by widely opening the window and then re-warmed, while another warm room is in use. If the extremities still remain cold, they should be rubbed with gentle massage twice a day.

I have already referred to the important part played by constipation in preventing gain of weight; and have described how this may be remedied: here I will only reiterate what I have already said, that an essential part of the treatment of infantile marasmus may be, and often is, the treatment of chronic constipation.

Lastly, a word with regard to drugs, which may directly affect nutrition. Cod-liver oil in my experience is very rarely useful in the treatment of infantile marasmus; more useful is malt extract, which may be used as a sweetening agent instead of cane sugar or milk sugar, and seems sometimes to exert a distinctly beneficial effect in promoting nutrition; but malt is not to be given where there is looseness of the bowels or much flatulence, and in any case is to be used in small quantity, a very small half-tea-spoonful to a three-ounce feed three or four times a day.

Some very interesting observations on the use of thyroid in infantile marasmus have been recorded by Dr. J. W. Simpson [1] of Edinburgh, who found that in some cases where there was

[1] *Scot. Med. and Surg. Journ.*, Dec., 1906, p. 504.

failure to thrive in spite of careful feeding, the administration to infants of three and four months old of $\frac{1}{3}$ to $\frac{1}{2}$ grain of thyroid extract given three times daily (Burroughs & Wellcome's tabloids were used), without any alteration of the diet, was followed by a more or less steady rise in weight. In some cases in which I have tried this method of treatment there has been a gain of weight where hitherto little or no progress had been made ; but bearing in mind the occasional ill effects of thyroid, I have contented myself with using the doses mentioned only once or twice a day, so that the good results which I have obtained may not fairly represent the full extent of its usefulness.

The old-fashioned practice, but I think a good one apart from its 'messiness', is to rub the infant with oil, preferably neat's-foot oil, which is less odoriferous than cod-liver oil. I am very doubtful how much, if any, of the oil can be absorbed this way ; but it is certain that oiling the skin serves to maintain the warmth of the infant, and if it does nothing else, it is good on this account.

CHAPTER XI

HYPERTROPHY OF THE PYLORUS IN INFANTS

THE importance of early diagnosis and of a clear understanding of the lines of treatment in the so-called 'congenital hypertrophy' or 'hypertrophic stenosis' of the pylorus in infants has become increasingly evident within the past few years, for it has been shown that what was formerly regarded as an incurable and inevitably fatal condition is, sometimes at least, amenable to treatment. Judging from my own experience I should say that many, perhaps even a majority of these cases, recover completely if only they are recognized sufficiently early and subjected to suitable treatment. I propose to deal chiefly with these two points, diagnosis and treatment, and my remarks will be based upon forty-two cases which have been under my own personal observation either in hospital practice or in private consultation.

The salient features of the disorder are briefly these: an infant under the age of four months has been vomiting his food. The vomiting began at the age of three or four weeks and for the first week or so was thought to be no more than a little indigestion might account for. But it persisted and soon began to attract notice more by its persistency than by its frequency, for it occurred perhaps only two or three times in the twenty-four hours. Then the food was thought to be at fault and change after change was made in the feeding, each time perhaps with temporary diminution of the vomiting. But still the vomiting persisted and at times was noticed to be so sudden, copious, and forcible that the vomit was shot out a foot or more from the mouth and perhaps through the nostrils as well.

Since the vomiting began the bowels have been costive, perhaps only opened with enemata. And now the infant is wasting to a marked degree and perhaps it is this wasting rather than any alarm at the vomiting which leads the parents to seek medical advice. Such is the history which leads one to examine specially for the two characteristic signs—visible and very marked peristalsis of the stomach and a palpable thickening of the pylorus—upon which the diagnosis rests.

Diagnosis. Having said thus much by way of general description I shall proceed to consider in detail the various points which bear upon diagnosis. But first let me emphasize the fact that no symptoms or combination of symptoms, however suggestive they may be, are sufficient for the diagnosis of congenital hypertrophy of the pylorus in the absence of the two characteristic signs which I have already mentioned. No doubt such a history as I have outlined would be strong ground for suspecting the existence of this disease and the suspicion might or might not prove correct, but where treatment, especially operative treatment, is so vitally serious a matter, we have no right to be satisfied with anything short of certainty and this depends upon the presence of well-marked visible peristalsis of the stomach associated with palpable thickening of the pylorus.

Sex. No great stress is to be laid on sex in the diagnosis of this disease, but none the less the remarkable predominance of males is to be remembered. Out of forty-two cases in which the point was noted thirty-five were males. Not only is this disorder very much commoner in boys than in girls, but when it does occur in girls it is, I think, usually of milder degree.

Place in family. Hardly less remarkable than the sex incidence is the tendency for this disorder to affect first children in a family rather than later ones. Out of thirty-eight cases in which I noted the position in family no less than eighteen were firstborn.

Family incidence. The question is often asked, when one case has occurred in a family, especially in the first born, whether the subsequent children are likely to suffer from the same condition. Formerly I had always replied in the negative, but in recent years, amongst a large number of cases subsequent to the forty-two mentioned above, I have seen three instances of two children in a family showing congenital hypertrophy of the pylorus. In two of these the children affected were the first and second of the family; in one they were the third and fourth. The rarity of this family incidence is evident, from the fact that only these three instances were observed in ninety-one families in which the condition occurred.

Age. The age incidence is of importance. On this point some misapprehension has arisen out of the nomenclature of the disease. More than once when I have seen these cases in consultation and have spoken of the condition as 'congenital hypertrophy of the pylorus' I have been met with the objection, 'But this is impossible, for the baby was perfectly well until it was four or five weeks old. How can the condition therefore be

congenital ?' Let me say at once that whatever may be the correct view as to the pathogeny of the disease, whether it be a congenital hyperplasia of the muscular tissue of the pylorus or a congenital lack of co-ordination with resulting spasm and thence hypertrophy as Dr. John Thomson has suggested, or whether the hypertrophy be entirely of post-natal development, the symptoms are very rarely congenital.

Of thirty-eight cases only one began to vomit within twenty-four hours after birth, and six others within the first week.

The term 'congenital', therefore, is misleading, and although I retain it as perhaps representing a correct theory there is no proof that all cases are congenital in origin. That the obstruction is primarily due to spasm of the pylorus I regard as certain, and to me it seems most probable that the hypertrophy also is the result of spasm; how long this hypertrophy may take to develop we cannot tell; but so far as the spasm is concerned I see no reason why it may not, like the disturbance of co-ordination which is evident in the hiccough which babies so easily get, or in the stuttering which is almost physiological in the young child who is learning to talk, be due in some cases to irregularity of function beginning after birth; may not the nervous mechanism of the stomach be as easily disturbed as the nervous mechanism of speech, particularly in the early days before function has become stereotyped by usage? Perhaps, indeed, the analogy may be closer than this and the tonic spasms of the muscles of speech find a counterpart in a stuttering stomach. But, however this may be, the point I would emphasize is this, that the symptoms very rarely date from birth; of my own cases, twenty-eight certainly and one other probably, dated symptoms (vomiting) from before the end of the fourth week; in the remaining cases vomiting began between the fourth and the end of the seventh week. It is not always possible to be certain of the date of onset, for slight regurgitation which the parents have regarded as normal and which may of course be so in early infancy has sometimes been present before the definitely abnormal vomiting began. I have never seen a case in which vomiting began later than the seventh week, but onset at least as late as the ninth week has been recorded.

Obviously these facts have an important bearing on diagnosis. For example, in the case of an infant aged nine months who had been vomiting persistently for three weeks a diagnosis of congenital hypertrophy of the pylorus was suggested, but in such a case the age alone, apart from various other indications, was

almost if not quite sufficient to exclude this condition. I am aware that the name 'congenital hypertrophy of the pylorus' has been applied to a condition supposed by some to be identical in the adult, and if the disease is primarily spasmodic it is conceivable that it might begin at any period of life, but so far as children are concerned the onset of the disease is invariably before the age of three months, and unless vigorous measures are adopted death usually results before the end of the fourth month.

Vomiting. I would insist, first, with regard to vomiting that however persistent and chronic it may be, it is never *per se* proof that an infant has congenital hypertrophy of the pylorus. No doubt it is the symptom which in most of the cases has first suggested the presence of this condition, but it is only when it is associated with the characteristic signs, the visible peristalsis of the stomach and the palpable thickening of the pylorus, that it becomes diagnostic.

Apart from its persistency the vomiting of congenital hypertrophy of the pylorus has certain features which are suggestive of the disease. 1. Its forcible character. A mother informed me recently that her child with this condition 'pumped up' his food suddenly with such force that it shot more than two feet from the mouth. Her account may have been exaggerated but it may serve to illustrate my point. She also mentioned another frequent result of this forcible expulsion that the vomit comes through the nostrils also. 2. Its occurrence in an infant who has been carefully fed; particularly when, as happened in at least sixteen out of my forty-two cases, the infant was being fed by the breast only. 3. Its persistence in spite of such alterations of feeding and such general treatment as will usually control the vomiting of dyspeptic conditions. 4. Its large amount showing that the vomit represents more than one feed, perhaps the accumulation of several feeds in the dilated stomach. When vomiting in an infant under four months has these features there is very strong ground for suspecting the presence of congenital hypertrophy of the pylorus, but each of the four, including the forcing of vomit through the nose, has occurred within my own experience also in cases in which there was no reason to suppose the presence of pyloric obstruction.

I have referred incidentally to the fact that the vomiting is sometimes distinctly influenced by diet. In most cases the improvement is only short-lived, perhaps for a day or two, but I have known this to cause error in diagnosis; the temporary

improvement with each change of diet was taken to indicate that the vomiting must be dependent simply on some fault in the feeding. The observation was correct, the inference was wrong.

Naturally much stress has been laid upon vomiting as a prominent symptom of the disease, but it must be remembered that the vomiting is not necessarily frequent; it may be only once or twice in the twenty-four hours. There may even be an interval of twenty-four hours in which there is no vomiting. No doubt this depends to some extent upon the degree of dilatation of the stomach which has occurred, but I suspect that it means more and points to variation in the degree of obstruction a variation which seems most naturally explained by varying degrees of spasm of the pylorus at different times.

As the result, no doubt, of the infrequency of vomiting in some cases this symptom has sometimes attracted much less attention than the wasting which accompanies it, and in two of my cases at least the vomiting was thought scarcely worth mentioning, the condition being regarded as simply obstinate infantile marasmus. In one of these both the medical attendant and the nurse stated that the vomiting was ' nothing particular ', but on cross-questioning one found that the vomiting, although it occurred sometimes only once in the day, was often more in amount than one or even two feeds and that it was shot out with unusual force through the mouth and nose.

Constipation. The association of chronic constipation with this persistent vomiting is of some importance in diagnosis, for where chronic vomiting in an infant at this age is dependent upon faulty diet it much more often keeps company with loose or at least unhealthy slimy stools and frequent action of the bowels. But there are exceptions in both ways. I have seen congenital hypertrophy of the pylorus with loose and frequent stools and I have notes of chronic vomiting with constipation in infants who showed no evidence of congenital hypertrophy of the pylorus. Nevertheless, I think it is a useful rule to examine carefully for congenital hypertrophy of the pylorus in all cases where chronic constipation is associated with persistent vomiting in an infant under the age of four months.

Wasting. This, as already mentioned, may be the symptom which has attracted most attention and I only mention it again to emphasize the possibility of mistaking the case for one of marasmus from faulty feeding. I suppose there can be no doubt that until recently these cases of pyloric obstruction have mostly

passed for the common marasmus of infancy or have been attributed to unsuitable feeding; and the mistake is easy enough to make, for the history may differ but little if at all from that of many a commonplace case of infantile wasting.

The two characteristic signs. My own experience leads me to think that the condition is overlooked largely because the method of examination for the two characteristic signs is not generally understood. In several of my cases the presence of this disease had been suspected, but as the characteristic signs were supposed to be absent no diagnosis had been made, whereas when the case was examined under the requisite conditions the signs were shown to be extremely marked. In the first place it must be recognized that these two signs are only present at intervals, so that cursory inspection and palpation of the abdomen may and almost always do result in overlooking the condition altogether.

Patient examination occupying perhaps ten or fifteen minutes may be necessary before the signs can be elicited, and occasionally, though I think rarely if proper precautions are taken, examination on a second occasion may be required. The reason of this is simple: both signs depend upon active contraction of the muscular wall of the stomach, and just as happens with the uterus during labour, at one time it is easily stimulated to activity and at another it remains inactive in spite of all efforts to excite contraction. The second point to be considered is the time at which the examination is to be made. I have almost always found that the abdomen has been examined without any reference to the time at which the infant was last fed, and the result has been failure to detect the characteristic signs. The abdomen should be examined immediately after the infant has been fed. It is at this time, and apparently in some cases hardly at all except at this time, that the abnormal peristalsis of the stomach is to be seen, and it is during peristalsis that the thickened pylorus is to be felt. I think it is well also to watch the abdomen during the taking of the feed; sometimes after part of the feed has been taken vigorous peristalsis of the stomach commences. I would add as a third counsel, and one particularly applicable to these cases, that warm hands are no unimportant factor in the palpation of an infant's abdomen.

Visible peristalsis of the stomach. Sometimes spontaneously, sometimes only after repeated stroking or gentle kneading of the epigastrium, a rounded lump varying in size from half a large walnut to half a Tangerine orange rises at the left costal margin

and passes very slowly across the epigastrium slightly downwards towards the right hypochondrium. Before this lump has yet reached the mid-line a second similar lump is already rising at the left costal margin, and sometimes before these two have yet disappeared in the right hypochondrium a third is already appearing on the left side, so that at one time three bulging eminences are seen like a chain of hills extending across the epigastrium. They move so slowly that at times they may even be seen to pause altogether, but each in turn fades away in the right hypochondrium, and a succession of these bulgings may continue to appear for one or two minutes. This peristaltic wave is so gross that it could easily be seen at a distance of two or three yards, and in two of my cases the mother or nurse had already noticed it. Upon this grossness I lay some stress for it is of importance in the diagnosis.

Two questions obviously arise with regard to the diagnostic significance of the sign : (1) Can any other movement be mistaken for this peristalsis of the stomach ? (2) Does visible peristalsis of the stomach occur in infants apart from this disease ?

There are two movements which I have known to cause doubt in diagnosis. One is the voluntary irregular contraction of the muscles of the abdominal wall which in an infant 'squirming' about as an infant often will, especially when the abdomen is exposed for observation, may cause local bulgings in the hypochondrium or epigastrium, but these although they may deceive the unwary ought not to cause any real difficulty, for the bulging produced thus does not travel transversely across the epigastrium, nor does it appear and disappear with the slow, even regularity of the gastric peristalsis ; moreover, it is definitely coincident with voluntary muscular effort. The other movement is much more difficult to distinguish ; it is peristalsis of the transverse colon in certain cases where, owing to an unusual arrangement of parts, peristalsis is from left to right in a portion of the colon. In an infant who was taken to King's College Hospital with vomiting at the age of five weeks I found definite but very slight peristalsis passing from left to right in the epigastrium ; the pylorus could not be felt and the peristalsis was so slight that I doubted its significance. The infant looked ill and was admitted and died with broncho-pneumonia and pleurisy. It was found that the transverse colon passed across from the hepatic flexure towards the left hypochondrium and then turned back upon itself into the right hypochondrium before passing

finally across the epigastrium to the splenic flexure ; it formed, therefore, an S-shaped loop in one part of which, of course, the peristalsis would travel from left to right. In this case the feeble character of the peristalsis had suggested during life that it was not due to hypertrophy of the pylorus. With regard to the second question I think it may be said that visible gastric peristalsis of such marked degree as that to which I have referred is during the first few months of life almost, perhaps quite, peculiar to hypertrophy of the pylorus. So far as my own experience goes I have never known it to occur apart from this condition.

But I have purposely emphasized the degree of peristalsis, for I have seen in infants with chronic vomiting and constipation, where there was no other evidence of pyloric hypertrophy and where simple treatment by careful feeding and aperients caused rapid cessation of the vomiting, a very feeble peristaltic wave passing over the stomach just after feeding. I suspect that this might be found more often if specially sought. The peristalsis in these cases was so slight that it could only just be detected by inspection of the abdomen obliquely in a good light ; the pylorus could not be felt. Of course it may be argued that such cases were very slight degrees of pyloric hypertrophy and this I cannot disprove. The peristalsis to which I have referred as a characteristic sign of hypertrophy of the pylorus and which was seen in all my cases except the first (which occurred some years ago before the methods of examination were known to me) is something altogether more striking than this, and if it happens to occur whilst the abdomen is uncovered is as obvious to the layman as to the medical man.

Palpable thickening of the pylorus. On deep palpation, usually just outside the right nipple line and about a third of the distance from the umbilical level to the costal margin, a hard lump, barrel-shaped and seeming perhaps about three-quarters of an inch long by half an inch wide, is to be felt *at intervals.* This tumour, the hypertrophied pylorus, behaves exactly like an intussusception in its variations of palpability. We are all familiar with the intussusception tumour which at one moment may not be felt at all and the next moment is so hard that it is obvious to anyone. So it is with the pyloric tumour ; it is during peristalsis that it is to be felt ; at other times the muscle is so soft that no amount of *tactus eruditus* will detect it. For this reason palpation should be made during the visible peristalsis of the stomach, and at these times I suspect that with

experience the pylorus could be felt in every case, although perhaps more than one examination may be necessary.

In forty-one out of forty-two cases I have noted the hard pylorus as palpable: in almost all these cases the palpability was confirmed by others and in most of the hospital cases by several people.

Occasionally, even when there is no visible peristalsis in progress at the time, the pylorus can be felt evidently undergoing contractions, being palpable and hard one moment and completely lost the next. Such contraction may be excited by gentle kneading with the tips of the fingers pressed deeply into the abdomen in the situation of the pylorus.

It may be doubted whether any assistance is to be gained from an anæsthetic in this examination, indeed it seems probable that it might actually prevent detection of the pyloric thickening by hindering indirectly the occurrence of peristalsis; certainly any anæsthetic is quite unnecessary; tact, patience and warm hands are much more likely to be successful. No doubt the diagnosis of pyloric hypertrophy can be made with all but certainty from the visible well-marked peristalsis of the stomach alone, but it is my opinion that in every case the evidence should be clinched by feeling the thickened pylorus.

Treatment

I shall preface my remarks by some general considerations on the treatment of this disease.

Within the past few years successful results have been obtained by operative relief of the pyloric obstruction—a method of relief which seemed at first to promise but little hope at so frail an age. But now it would seem that there is a danger lest the very brilliance of this success should lead us into adopting as routine a mode of treatment which, however greatly methods may improve, can never be otherwise than dangerous in an infant a few weeks old. If the disease were incurable by other and far less dangerous measures we might well accept the chance which surgery offers; but, as I shall show, it is now quite certain that there are cases in which no operation whatever is necessary, and there are other cases in which the child's chance of surviving operation may be greatly increased by the use of simpler measures for a time until the infant is older and stronger and therefore in a better position for operative treatment. If, as I hold, the hypertrophy be merely a secondary phenomenon, the result of spasm, then we have a rational basis for adopting such treatment

as may tend to allay the spasm, and if this can be done one may suppose that just as the hypertrophied muscles of the athlete degenerate when he ceases his exercises so the hypertrophied pylorus loses its hypertrophy and, partly from the diminution in the thickness of its wall, but much more from the cessation of spasm, the obstruction ceases to exist.

This seems to me the most natural explanation of the complete recovery which occurs in some well-marked typical cases under such simple treatment as repeated stomach-washing and sometimes even with simple care in feeding. How these measures allay the spasm I know not, but that they do is I think actually demonstrable in the gradual disappearance not only of the vomiting but also of the two characteristic signs which I have mentioned.

Passing now from theory to more practical considerations I would point out that by whatever method this disease is treated, whether by medical or by operative measures, the medical man must be prepared to give much time and much care to the case, not for a few days but for many weeks if the infant's life is to be saved. It might be imagined that operation, inasmuch as it relieves all symptoms of pyloric obstruction forthwith, would solve all difficulties at once, and that after the few days immediately succeeding the operation but little care would be required. As a matter of fact it is often far otherwise and the infant's life hangs in the balance for several weeks after operation.

One factor in success I will mention here, for it is equally important whatever method of treatment is adopted—namely, an accurate weighing-machine. Variations in weight, so helpful a gauge in most infantile diseases, are of the utmost importance in this condition, where the requisite changes in the feeding may be many and may be of vital necessity and often have to be determined by the loss or gain of an ounce or two.

Treatment by dieting only. As already mentioned many cases are improved temporarily by change of diet, but it is probably very rarely that a cure can be effected by such treatment alone. Dr. H. Willoughby Gardner,[1] of Shrewsbury, has published the case of a male infant whose vomiting began at the age of 8½ weeks and who showed a peristaltic wave 'almost the size of a small Tangerine orange' and had also a palpable thickening of the pylorus. The infant was fed with whey alone for a time, then malt was added, then barley-water and maltine were

[1] *Lancet*, 1903, i, p. 100.

given occasionally, and gradually raw meat juice and white of egg were given also. For a long time all residue-leaving food was avoided and the amounts at each feed were only slowly increased from a teaspoonful every twenty minutes to two, three and four teaspoonfuls at longer intervals. The vomiting gradually subsided, all symptoms disappeared, and the child at the age of 10½ months weighed 20 pounds.

Drs. J. R. and W. J. Harper,[1] of Barnstaple, by whose kindness the accompanying photograph (Fig. 9) is reproduced here, have published a case with very typical symptoms and signs of congenital hypertrophy of the pylorus: the vomiting began when the infant, a male, was 3½ weeks old; there was constipation and gastric peristalsis sufficiently obvious to be photographed and the pyloric tumour could be felt.

He was fed with teaspoonful doses of hot water by the mouth

Fig. 9. Congenital hypertrophy of the pylorus. Case under Drs. W. J. and J. R. Harper; showing characteristic wave of gastric peristalsis: complete recovery without operation.

and rectal injection of nearly half a pint of normal saline solution twice a day at first, then teaspoonful doses of peptonized milk by mouth with Valentine's meat juice and white of egg were gradually introduced. Enemata either saline or nutrient were continued for over a month, and gradually larger quantities of peptonized milk and later of Benger's food were taken by mouth, and the child made a complete recovery.

The photograph of this case is the more interesting as it proves conclusively that well-marked gastric peristalsis does not indicate necessity for operation; the fact also that the pyloric tumour was felt in this case makes it certain as far as is possible that this was a typical case of hypertrophy of the pylorus. The clinical course shows that simple dieting may be sufficient to cure these cases.

[1] Ibid., Aug. 19, 1905.

I have had one case in which the effect of even simpler dietetic measures was equally striking. A female infant, aged six weeks, was brought for persistent vomiting which had begun at the age of three weeks; the vomit was said to be 'shot right out', coming through the nostrils as well as the mouth. The infant had been carefully fed with milk and barley-water. A well-marked wave of gastric peristalsis was seen, but on the two occasions on which I saw the child at this stage I was unable to feel the pylorus. The food was changed to peptonized milk and some bismuth was given. Four days later gastric peristalsis was still very obvious and vomiting continued; gradually, however, the vomiting became less, and after about four weeks it ceased. The gastric peristalsis also gradually disappeared, and when I saw the infant again at the age of $7\frac{1}{2}$ months she weighed $15\frac{3}{4}$ pounds and was perfectly healthy. In this case the failure to detect palpable hardening of the pylorus may be considered to make the diagnosis less than absolutely certain, but it does not alter the importance of the clinical fact that an infant with chronic, very forcible vomiting and with well-marked visible peristalsis of the stomach recovered gradually with simple alteration of food.

Treatment by nasal feeding. My colleague, Dr. F. E. Batten, has published a case which I also had the opportunity of watching, in which an infant, aged eleven weeks, with typical symptoms and with the two characteristic signs pronounced, recovered completely with nasal feeding. All feeds (consisting of 2 ounces of milk and 1 ounce of barley-water) were given by the nasal tube for twenty-seven days, except on one day when bottle-feeding was unsuccessfully tried. I have not used this method myself; it has the disadvantage that it is liable to cause troublesome excoriation of the nostrils.

Treatment by stomach-washing. This is the method which, apart from operation, promises, I think, to be of most value, and it is my own opinion that when careful feeding with some thin fluid, such as whey and raw meat juice, fails after a trial of a few days to arrest the loss of weight, which is the chief indication of the gravity of the vomiting, stomach-washing should be given a trial in every case, the duration of its trial to be determined by its effect upon the weight. It is certain that some cases which, so far as can be judged clinically, are in every way typical instances of pyloric hypertrophy and which show just as marked signs, as cases in which operation is deemed necessary, namely a peristaltic wave travelling in bulging prominences across the

epigastrium and a hard palpable pylorus, recover completely and so far as my present experience goes show no tendency to recurrence after treatment with stomach-washing alone.

The stomach is washed out twice daily for several weeks just before a feed and then once daily for some weeks longer with a solution of sodium bicarbonate (2 grains to 1 ounce) through a Jacques's soft rubber catheter, No. 12 or 14. The frequency of the washings and the duration of treatment depend upon the improvement in the vomiting and especially upon the weight, which should be taken every alternate day. How soon should improvement be expected? Not necessarily at once. In three of my cases which subsequently gained weight steadily under this treatment the weight had remained almost stationary, or even fallen 2 or 3 ounces at one weighing and regained its former level at the next, for two or three weeks, in one case for five weeks, before continuous gain began. So long as the infant is not definitely losing ground there is no need to rush to operation. Of course, with wasted infants at this early age every ounce is of importance and the utmost care and judgement must be exercised in deciding for or against the continuance of medical treatment; certainly so long as there is any gain in weight operation may and in my opinion should be deferred. The food during this treatment has generally consisted of whey with raw meat juice or a very weak peptonized milk mixture perhaps with a very small quantity of cream.

Such cases were the following:

A male infant, aged 8½ weeks, seen with Dr. A. Reeves, of Streatham Hill. In addition to chronic forcible vomiting the infant showed very marked visible peristalsis of the stomach and at times the pylorus could be felt. He had lost 2 pounds ¾ ounce in weight since birth. The stomach was washed out first twice and then once daily for nineteen weeks. All vomiting gradually ceased, the weight after 2½ weeks began to rise steadily, the two characteristic signs disappeared, and when he was seen at fourteen months old he had been quite well for more than six months and weighed 23½ pounds.

A male infant, aged eight weeks, seen with Dr. C. F. Wakefield, of Horley. The child had been vomiting very forcibly since 6½ weeks old, and the vomiting consisted sometimes of more than the last feed; he showed the wave of gastric peristalsis well marked and the pylorus could be felt distinctly. The stomach was washed out at first twice and then once daily, then two or three times per week, for sixteen weeks. During this time all symptoms and signs had gradually disappeared and the patient seen again at the age of six years was a bonny healthy child.

Albert D., aged three weeks, came under my care in King's College Hospital at the age of three weeks with the characteristic symptoms and signs of congenital hypertrophy of the pylorus: they had begun on the fifth day after birth; Dr. G. P. Young, of Hinckley, then my house-physician, washed the

stomach out twice daily for ten weeks and subsequently it was washed out once daily; the lavage was continued altogether for six months. For the first five weeks the weight was almost stationary and some vomiting continued; the weight then began to rise and the child, whose weight on admission was 5 pounds 14 ounces and who was so feeble and puny that laparotomy would have been a desperate measure, gained weight slowly and at the end of six months weighed 12 pounds 8 ounces and left the hospital in a thriving condition.

Walter C., aged thirteen weeks, was sent to me with the usual symptoms of congenital hypertrophy of the pylorus. They had appeared at the age of four weeks whilst he was being fed on breast-milk only. The gastric peristalsis was so marked that the mother herself had noticed it, and the thickened pylorus which was palpable at that time could still be felt several weeks later. The child weighed 8 pounds 3 ounces at thirteen weeks, and as the mother was able to suckle it I advised Dr. Tremlett Wills, under whose care the infant was, to wash out the stomach daily but not to have the infant weaned until the proper time. The stomach-washing was continued until the child was 11¼ months old. I heard two years later that he was 'now a fine sturdy little boy'.

In another case an emaciated infant with the usual signs and symptoms of pyloric hypertrophy, aged seven weeks, and already 'almost too feeble to cry', was treated by stomach-washing for six weeks; at first she gained weight slowly, if at all, and after about a month was only half an ounce heavier than when washing began; then she began to gain weight steadily and in thirteen days had gained 13½ ounces. The child being now stronger it was decided to operate. From the day of the operation, although the vomiting was stopped thereby, continuous marasmus occurred, and the child died in convulsions twenty-five days after the operation. It is easy to be wise after the event, but here the steady gain in weight which had commenced before the operation makes it probable in the light of the cases already mentioned that had the stomach-washing alone been continued the child would have recovered.

In one case after washing out the stomach for fourteen weeks there had been a gain of 22 ounces, but progress was so slow that operation was done and the child recovered. In two other cases where stomach-washing was done for a few days only there had already been slight gain in weight, but it was decided to operate.

Whether the weight rises at once or not there is usually a marked diminution of the vomiting after the stomach has been washed out for a few days; but this must not be taken as an indication for stopping the stomach-washing until the infant has been making steady and considerable progress in weight for several weeks.

Having said this much of the success of stomach-washing

in this condition I must mention its failures and possible risks ; in four cases where stomach-washing was tried for a few days only there was no improvement ; in one case death occurred with some pyrexia about forty-eight hours after stomach-washing was begun and, although the necropsy showed no evidence of vomited material in the respiratory passages, death may have been due to matter drawn into the lungs in vomiting during the stomach-washing ; if so it must be an exceedingly rare accident, for I have had infants' stomachs washed out many hundreds of times without such an occurrence.

In another case a dry rotten tube was inadvertently used and the end broke off in the stomach necessitating immediate resort to the operative treatment which we had intended to defer ; the pylorus was stretched, but an accidental rupture of its wall was overlooked (the operation was done in great haste owing to extreme feebleness of the infant), and death occurred about twenty-seven hours later. Such exceedingly rare accidents must, however, weigh but little, and I think it remains true that stomach-washing is a procedure which should certainly be tried in the pyloric hypertrophy of infants before resorting to operation ; in some cases it is sufficient alone to secure complete recovery, in others by increasing the nutrition and the strength of the infant and by enabling us to postpone operation till the infant is a few weeks older it increases the chances of success from operation ; where, however, the weight is steadily diminishing for several days in spite of stomach-washing the question of immediate operation must be considered.

Lastly, I would insist upon an important point—namely, that the degree of gastric peristalsis (I mean the size of the bulging wave which is seen) and the palpability of the hard pylorus are no criteria of the possibility or even probability of recovery or improvement by stomach-washing or of the likelihood of necessity for operation ; the most pronounced signs and symptoms may occur in cases which respond well to stomach-washing alone.

Operative Treatment. The actual operation to be done, if any be necessary, is a question which falls outside my province ; forcible dilatation of the pylorus, pyloroplasty, and gastro-enterostomy have each their advocates. Fourteen cases out of forty-two included in my series (one of which was only seen by me once in consultation and was subsequently operated upon successfully by Mr. H. J. Stiles, of Edinburgh), were treated by forcible dilatation of the pylorus—all except two by my colleague,

Mr. F. Burghard—and eight of these fourteen recovered. One of these died several months later from broncho-pneumonia.

Operation has risks apart from those connected with shock and with the operation itself. There is a tendency to looseness of the bowels after it and also a difficulty of nutrition which in some of my cases seemed to make death from marasmus almost inevitable for some weeks after the operation although they ultimately recovered; an important point this which increases the need for careful consideration before embarking on operative measures and which increases also the importance of previous stomach-washing if thereby even a few ounces of weight can be gained.

Results of Treatment. It is difficult to compare the results of medical treatment with those of operation: for in several cases which proved fatal where no operation was done the infant was already so bad when the diagnosis was first made that he died within three or four days and was not likely to have been saved by any treatment. In one case where no operation was done death was due to a purely accidental cause, the infant appeared to be doing fairly well but three weeks after the onset of the symptoms of congenital hypertrophy of the pylorus he suddenly became very ill and was found at autopsy to have died of acute intussusception.

Of the forty-two cases in my series three were seen in the days before any special treatment was known for this disorder, and in three the ultimate course of the case could not be ascertained. Of the remaining thirty-six cases nineteen recovered (including the case mentioned above which died subsequently of broncho-pneumonia).

Of the nineteen which recovered eight were treated by operation (in three of these the infant had been gaining weight with stomach-washing before the operation was done) eleven were treated medically (nine by stomach-washing, two by dietetic measures only).

Of the seventeen cases which died six were treated by operation, eleven by lavage or dieting, including four in which death occurred within a few days after I first saw the infant who was already in such an extreme condition that any treatment was hopeless and also the case in which death was due to acute intussusception.

I have given these statistics in detail not as supporting any routine practice of this or that method of treatment; but to show that a considerable proportion of these cases can be cured

without operation. Each case must be carefully considered on its own merits : there is no doubt of the curability of this disorder in some cases by stomach-washing or even by dietetic measures alone : but I think there is equally no doubt that in some the only resource which will save life is operation.

CHAPTER XII

ABDOMINAL PAINS IN CHILDREN BEYOND THE AGE OF INFANCY

I KNOW no symptom which may be more obscure in its causation than colicky abdominal pain in childhood. In this chapter I shall consider the various conditions to which it may be due.

The commonest cause is, as in infancy, some form of indigestion, most frequently, I think, the disorder to which Dr. Eustace Smith has given the name of mucous disease. I shall refer to this more fully in the chapter on indigestion, here I will only recapitulate its chief symptoms: the child is usually 'going thin', his lower eyelids are puffy, or he is 'dark under the eyes', he sleeps badly, is restless, grinds his teeth, or talks in his sleep, or has definite night-terrors; in the daytime he is unnaturally nervous, his stools, although perhaps costive, show an excess of mucus, which is not necessarily present in every stool, but will generally be noticeable in some of the stools if these are observed for several days; and lastly, he complains of frequent slight pains in the abdomen. The pain is usually quite transient, lasting less than a minute; it is generally not severe enough to make the child cry out; it is associated sometimes with a sudden pallor of the face, the child 'turns white'; this, however, would seem to be a reflex vasomotor effect which does not depend upon the severity of the pain, for, as I have pointed out elsewhere, it is not an uncommon symptom of indigestion in children without any pain whatever.

A common source of abdominal pain is undigested vegetable matter in the intestines, the stools are large and of 'porridgy' consistence, and in them are to be seen large pieces of scarcely altered fruit or vegetable, such as banana, potato, or cabbage. Difficulty in digesting fruit and vegetables is so common in children that I am always inclined to suspect this cause where colicky pains of obscure causation are present. It is to be remembered also that children are very apt to bolt their food without mastication, and vegetable substance, if not thoroughly broken up in the mouth, undergoes but little change during its passage through the body, and is likely to act as an irritant: sometimes examination of the stools from these children with abdominal pains at once furnishes a clue to the proper line of

treatment ; I have seen large masses of shreddy pulp of banana, blocks of unbroken potato, and pieces of cabbage-leaf nearly an inch square in the stools ; little wonder that the child suffered with colic!

In some cases the failure to masticate food which underlies the abdominal pain is no mere faulty habit, but is due entirely to the condition of the teeth, which are either extremely defective, or are so tender that the child is afraid to chew its food ; I have several times seen speedy relief of colic by extraction or stopping of carious teeth.

In considering indigestion I have referred to the case of a girl aged $3\frac{1}{2}$ years, whose recurring abdominal pains were so intractable, that the presence of some organic disease was suspected, and exploratory laparotomy was proposed: the administration of a pancreatic extract proved effectual in stopping the pains after a variety of other remedies had failed. In this case there was nothing to suggest 'mucous disease', but I suppose the cessation of pain when pancreatic extract was given, showed that the colic was due to digestive disorder. Certainly chronic indigestion of any variety is very apt to cause recurring colicky pain in children, and I think much more often in children in the earlier part of childhood, that is, before the age of six years, than between the ages of six years and puberty.

Next to digestive disorder, perhaps the commonest cause of occasional slight pains in the abdomen is the presence of threadworms. It can hardly be supposed that the mechanical irritation caused by such small parasites is sufficient to excite the irregular or unduly vigorous peristalsis of the bowel, which no doubt underlies colicky pains, but post mortem examination shows that the presence of many threadworms in the intestine is generally associated with an excessive quantity of mucus, and it seems likely that it is this rather than the presence of worms, which is the actual exciting cause of the colic in these cases, for excess of mucus in the intestine from any cause seems to be very liable to set up colic in children.

In the earliest years of childhood, perhaps up to the age of four or five years, simple constipation is sufficient cause for frequent complaint of pains in the abdomen, but in later childhood constipation is less and less apt to cause pain. I mention this difference because it illustrates, I think, a physiological point which may be of some practical importance. It would seem that the sensitiveness of the bowel to stimuli decreases as the child grows older. In the first few weeks of life, constipation is an exceedingly

common cause of colic and screaming, it is less so in the infant of nine months or a year, and the tendency to colic from this cause seems to diminish steadily as the child grows older. In other words, the bowel shares in the general nervous instability of childhood, which, as we know, tends to diminish spontaneously. Whether this nervous irritability produces only an exaggeration of normal peristalsis, or whether the contractions produced are also irregular and ineffective, we cannot tell, but it is worthy of note that during the earliest years of childhood, the period when colic is most apt to occur, many of the functions of the body have not yet acquired the fixity of later age, the heart, for example, easily becomes irregular, the excretion of urine may be suspended for many hours, muscular co-ordination is easily disturbed, stuttering is almost physiological in the very young child, the rhythm of the diaphragm is easily disturbed with resulting hiccough, and so on : it seems therefore likely enough that the colic to which infants and young children are so specially liable may mean irregularity as well as excess of peristalsis.

But if the bowel shares in the nervous instability which is peculiar to early life, it seems more than probable that it shares also in the individual peculiarities of children in this respect : so that there may be a ' nervous bowel ', if I may use such a term, a bowel which goes off into colic upon the slightest provocation by stimuli which would have no such effect in a less nervous child.

There can be no doubt that such a condition obtains in the case of so-called lienteric diarrhœa, the ' diarrhée nerveuse ' of Trousseau, in which the taking of food is almost immediately followed by colicky pain in the abdomen and an urgent desire to defæcate, although the stools may not be loose and the child may be quite free from any such symptom between meals ; this condition which in my experience is much more common in boys than in girls (thirteen out of sixteen cases were boys) is closely associated with nervous excitability ; almost always the child is said to be ' very nervous ', and other nervous symptoms, such as night-terrors, sleep-talking, and enuresis, are present ; it occurs also specially in the child of rheumatic inheritance, which is perhaps another indication of its nervous connexion, for the rheumatic child and the child of rheumatic parentage show a very marked tendency to the nervous temperament. In these cases of nervous diarrhœa the colicky pain which is started by food is followed, as a rule, by evacuation of the bowels ; but this is not always so, sometimes at one meal the child complains only of the abdominal pain, while at another there is also urgency of defæcation. Pos-

sibly in the one case the part of the bowel thrown into spasmodic contractions is higher up than in the other case, so that defæcation does not result; but, however this may be, this occurrence of colic without evacuation of the bowels in cases of so-called 'nervous diarrhœa' supports the idea that in some children the main factor in the tendency to colicky pain is what I have called a nervous bowel.

There is another disorder in which one is tempted to suspect that pains in the abdomen may possibly be of nervous origin, namely, cyclic albuminuria. This is probably less rare than is generally supposed, it is easily overlooked, for there is little to suggest the necessity for examining the urine, and even when the urine is examined, the nature of the albuminuria may be mistaken unless a morning as well as a midday specimen is examined. The vague symptoms for which children with this disorder are brought to the medical man are certainly often of a nervous character, headaches, night-terrors, 'always so tired,' flushing of the face, these are some of the symptoms which I have noted particularly in these cases, and with these are associated often colicky pains in the abdomen. Dr. L. Guthrie goes so far as to include cyclic albuminuria amongst the functional nervous disorders of childhood, and says in his admirable work on these disorders, that cyclic albuminuria is evidence of nervous instability affecting chiefly the vasomotor system. I doubt whether enough is known of the pathology of this curious disorder to justify any dogmatic statement on this point; and so far as the abdominal pains associated with it are concerned, it must be remembered that the children with cyclic albuminuria often show distinct evidence of digestive difficulty and, as Dr. Guthrie points out, they are often the subjects of 'mucous disease' in which colicky pain is a common symptom.

The urine should be examined in every case of obscure abdominal pain, children like adults are subject to renal calculus and colic therewith, but in my own experience this has been rare; Leslie S., aged eleven years, was under my care for several months with attacks of vomiting which were thought to be characteristic attacks of so-called 'cyclic vomiting'; the mother mentioned that the boy had some pain in the abdomen during the attacks, but laid so little stress on the pain that its significance was entirely overlooked; until the boy was suddenly seized one day with severe pain and difficulty in passing urine, and after about twenty-four hours of severe pain in the hypogastric region, passed a rough calculus the size of a cherry-stone. Inquiry then elicited

the fact that with each attack of vomiting, there had been pains in the right lumbar region.

There is another cause for colicky pain in connexion with the kidney which is worth remembering, though it may be rare, namely, movable kidney. I once saw two children in one day, with abdominal pain due to this cause; both were boys, one aged three and a quarter years, the other aged ten years. In the former there were attacks of colic which were sometimes severe enough to double him up, but more often apparently slight, and these were specially apt to occur when he was walking. Palpation of the abdomen showed that the left kidney was freely movable; the urine was normal. In the other case attention had been drawn to the movable kidney by pain on the right side of the abdomen simulating appendicitis. Dr. Jules Comby has drawn attention to this possibility of confusing the pain due to movable kidney with that due to appendicitis in young children. I have found a freely movable kidney on the right side in an infant under twelve months old, and there can be little doubt that the condition, though seldom detected in infancy, is sometimes congenital, so that even in infancy it is a possible cause for abdominal pain.

Biliary colic is practically unknown in childhood; I have elsewhere referred to the very rare occurrence of minute biliary calculi in early infancy, but after that period I have never in my own experience known biliary calculi to occur under the age of puberty, and I was only able to find eight cases on record in children between two and fourteen years of age; in two of these it was stated that there was biliary colic.

Frequent complaint of pain in the abdomen in a child usually suggests, amongst other possibilities, the presence of tabes mesenterica, caseation of the mesenteric glands; and in many cases it is no easy matter to exclude this, but if the glands are already firmer as well as larger than normal, careful palpation may detect them without difficulty. The parts at which they are usually to be felt most easily are, I think, on each side of the midline, just below or just above the level of the umbilicus, where they may be felt by palpating deeply so as to press the fingers towards the sides of the bodies of the vertebræ; and also in the right iliac fossa.

Frequent recurrence of pain with enlarged glands in the abdomen generally indicates some adhesions between the glands and surrounding parts. There is a great liability to the formation of bands of adhesion between caseous glands and adjoining coils of intestine or the parietal peritoneum, and the pain may be due

to dragging of these adhesions, but it may be due to a more serious cause, the catching of a loop of intestine under one of these bands ; this accident I have seen more than once ending in acute strangulation. No doubt the colicky pain which sometimes accompanies tuberculous peritonitis is due chiefly to dragging on adhesions, and a similar pain due to adhesions sometimes follows recovery from appendicitis, whether the appendix has been removed by operation or not.

Lastly, it is always to be remembered that the earliest evidence of appendicitis may be vague pains in the abdomen, which may not be sufficiently severe to excite any alarm in the minds of parents or even of medical men until the supposed 'stomach-ache', which with a little vomiting, was thought to indicate 'bilious attacks', declares its nature by a sudden outburst of acute symptoms of appendicitis. I have seen such recurring attacks of slight pain mistaken for indigestion or bilious attacks even when the child was under frequent medical observation, and I know of no way to avoid this mistake but by repeated careful examination of the abdomen which should never be omitted when a child complains of colicky pains.

Treatment

I have grouped together in this chapter the conditions which are to be thought of when a child is brought to the medical man for the very common complaint of 'pains in the abdomen', for even if it is not always possible to arrive at a certain diagnosis of the exact cause of the pains, we can, at any rate, by bearing in mind the various possibilities exclude some of them in the particular case, and so avoid some serious errors in treatment. Obviously the treatment will vary according to the probable cause, and if no evidence of organic disease is to be found, and there is no constipation to treat, the proper course will usually be to diet along the lines laid down in the chapter on indigestion, especially avoiding those foods which leave a coarse indigestible residue, such as brown bread, coarse oatmeal, fruit of all sorts fresh and dried, and such vegetables as onions, celery, carrot, turnips, tomatoes, and of course all pickles. Potatoes also are best avoided in these cases. Sometimes even more important for these children than dieting is dentistry ; I have repeatedly seen recurring colicky pain in children rapidly relieved by the removal or stopping of carious teeth. If the teeth are so few or so bad that proper mastication

is impossible, the only thing to be done is to feed the child on sopped bread, milk broths, lightly-boiled eggs, boiled brains, pounded fish, custard, blanc-mange, and sloppy food in general, or at any rate such food as can be sufficiently broken up without the aid of the teeth.

In all such cases it will be wise to keep the bowels acting freely, but to use for the purpose laxatives which are not likely to gripe and which are not likely to retard digestion. Paraffin is particularly useful in such cases, and most children take it well. A formula especially useful where it is desirable to combine an antifermentative with a laxative is the following, which is in use at the Children's Hospital, Great Ormond Street :

R. Paraffin Liq. . . .	℥xl	Pulv. Tragacanth. . .	gr. $1\frac{1}{3}$
Sod. Benzoat. . . .	gr. $2\frac{2}{3}$	Ol. Amygdal. Essent. . .	℥$\frac{1}{8}$
Calc. Hypophosph. . .	gr. $2\frac{2}{3}$	Aq. Chloroform. ad . .	℥ii
Pulv. Acaciae . . .	gr. xx		

ter die, ante cib.

This is suitable for a child of five years ; half this dose may be given to a child of two years. Senna in small doses two or three times a day is good, either a decoction of the senna pods, four pods soaked in a wine-glassful of hot water for three or four hours, half of this to be taken twice a day, or half a drachm of syrup of senna in a teaspoonful of dill-water, to be taken two or three times a day regularly ; or one of the preparations of malt with cascara may do good by combining the diastasic effect of malt with the laxative action of cascara ; this, however, must be used in small doses, say $\frac{1}{2}$–1 drachm two or three times a day, rather than in one big dose, which is apt to gripe.

I have mentioned above the striking effect of pancreatin in an otherwise intractable case of abdominal pains in a young child ; the glycerine of pepsine ℥ xx ter die, or Fairchild's diazyme in doses of $\frac{1}{2}$–1 drachm may prove useful, and I have found the preparations of malt with pepsine and pancreatin very beneficial. The pains which occur with cyclic albuminuria can only be combated by reducing the irritating residue of food in the intestine to a minimum by care in diet as suggested above ; it is certain that when the bowel shows that excessive nervous irritability which I have described as characterizing the so-called lienteric or nervous diarrhœa, some foods excite the pain and evacuation more readily than others ; I have noted particularly that hot drinks of any kind and dried fruit, such as currants, were particularly active in starting the colicky pains. So that although the irritability of the bowel may be regarded as a nervous disorder,

it is to be treated partly by dietetic measures, particularly by excluding all foods which are likely to leave an irritating residue in the bowel. The nervous excitability of the bowel in such cases is rapidly relieved by Dover's powder, which not only stops the frequency of evacuation of the bowels, but also causes speedy cessation of the pains ; it should be given in doses of $1\frac{1}{2}$–2 grains three times a day to a child of five years old, and 2–3 grains ter die to a child of ten years. Arsenic has also a markedly beneficial effect in these cases, and is worthy of trial also for the colicky pains of cyclic albuminuria.

CHAPTER XIII

INDIGESTION IN CHILDREN PAST THE AGE OF INFANCY

Amongst the common disorders of childhood perhaps none is commoner than indigestion, but it appears under so many guises that it is little wonder if it is often unrecognized ; certain it is that the varied symptoms to which it gives rise are often attributed to any cause but the right one, and a child is dosed with this and that medicine when the one thing needful is a revision of diet.

I shall not discuss here the chemistry or the physiology of indigestion, I shall consider the disorder purely from the clinical point of view and as it affects children.

The child, unlike the adult, is still in the stage of active construction ; he is or should be steadily building, and therefore requires an income sufficient not merely for the upkeep of a structure already completed, but also for the carrying on of further building ; and this building must needs be continued even if the income is insufficient for both purposes ; so that in this case the upkeep of the already completed structure will suffer. So it is that many disorders which in the adult interfere only to a slight degree with the general nutrition produce a much more noticeable degree of wasting in the child ; and one such disorder is indigestion. How often one hears that a child is ' going so thin ' : the appetite is poor, there is perhaps occasional pain in the abdomen ; the child is languid and complains of feeling tired after very little exertion ; his hands and feet are ' always cold ', he is so dark under the eyes, or perhaps is said to be ' puffy under the eyes ', and it is noticed that sometimes his face turns very white suddenly as if he were going to faint. Such a history as this usually means that the digestion is at fault ; but the symptom that troubles the mother most is the wasting which, though usually slight in degree, is sufficiently obvious to a mother's eye.

I lay stress upon this wasting, because not only does it arouse the parent's anxiety, but even to the medical man it may suggest possibilities of latent tubercular mischief, and in those cases to which Dr. Eustace Smith gave the name of mucous disease, where there is with chronic indigestion passage of excess of

mucus with the stools (not necessarily with all), the wasting is sometimes very marked, the skin becomes dry and harsh, and there may even be a short dry cough which may add to the suspicion of tuberculosis, although the effect of careful dieting subsequently shows that the whole trouble is indigestion.

Next to the wasting I suppose there is no symptom which distresses a mother so much as the loss of appetite, which is a very common feature of indigestion in children. I would direct attention especially to this point because anorexia in young children is often a most puzzling and baffling symptom; a child three or four years old will sit down to its meal and absolutely refuse to eat or will eat so little all day that the parents become alarmed, and often if an attempt is made to coax the child by feeding it with a spoon the child will actually retch as if the very idea of food nauseated him. Such a history as this is very typical of indigestion in a young child: Rose B., aged $4\frac{4}{12}$, was brought for 'pains in the stomach'. These pains were referred to the epigastric area, and were only occasional; the mother mentioned also that the child 'goes white' suddenly sometimes, but not apparently in relation to the pains, her appetite was said to be very bad, after taking 'just a mouthful she says she has had enough'. Many times I have been told that a child complained of 'feeling full' after eating only a few mouthfuls, a symptom very characteristic of indigestion.

Pain in the abdomen, sometimes in the epigastrium, sometimes at the umbilicus or over the lower part of the abdomen, is also a common complaint; the pain is usually quite short and slight, but sufficient to make the child complain, and occasionally it is sharp enough to make the child cry out; it is not necessarily just after meals. As in the case mentioned above, this is often the reason for bringing the child to the doctor, and it is sometimes very difficult to be sure of the exact cause of the pain. The association of 'going thin' with 'pains in the abdomen' naturally suggests the possibility of some tubercular disease, caseous mesenteric glands, or early tuberculous peritonitis, and not very rarely such a possibility seems the more likely, as we are told that the child's stomach 'is so large' owing, as is found on examination, to the chronic flatulent distension of indigestion. One little girl aged $3\frac{1}{2}$ was sent to me for pains in the lower part of the abdomen, which she had had for about six months; she was said to turn white with the pain, though she did not cry out; she had become more nervous lately, and did not sleep well. She had lost one pound in weight in two months. The bowels

were regular, santonin had been given, but no worms could be found. The tongue was only slightly furred. Nothing abnormal was to be felt in the abdomen, but this was said to be distended at times. Whilst in my consulting-room, the child had an attack of the pain, in which she bent forwards and pressed her hand over her abdomen in evident pain, but after a few seconds she was quite bright again. No tuberculous glands could be felt, and I was of the same opinion as her medical attendant, that the pains were due to digestive difficulty; in spite of dieting, however, and various drugs, the pains were so persistent that the child was sent to a surgeon who proposed an exploratory laparotomy; it was decided, however, to give a further trial to medical measures before resorting to any surgical procedure, and upon administration of a pancreatic extract, the pains entirely disappeared, and the child made a good recovery.

In this particular case the stools contained no mucus, but such colicky pains are especially common where the child frequently passes mucus in the stools. Both in infancy and in early childhood it is very noticeable how often the passage of mucus from the bowel has been preceded by colicky pain in the abdomen, and this whether the mucus be due to indigestion or to the presence of worms; and indeed there seems to be a sufficient explanation to any one who has made many autopsies on children, in the tenacious viscid layer of mucus, which so often coats a great part of the intestine, where for instance there are threadworms; the difficulty of driving such viscid material along the intestine must be great, and it is no wonder if in the effort to do so the bowel undergoes a more forcible and perhaps irregular peristalsis than usual, giving rise to the sensation of colic.

The case to which I have just referred illustrates another of the common symptoms of indigestion in children, an increase of nervous instability. Naturally this will be most marked in the child who is of specially nervous temperament, but even in those who have hitherto shown no such tendency the nervous symptoms of indigestion are sometimes very marked. Sometimes these take the form only of fretfulness and tearfulness, 'the child cries almost if you look at her': Audrey B., aged three years, was brought to me with the history that for nearly six months her appetite had been bad and she had suffered with pains in the abdomen; a fortnight ago she had vomited one day; frequently she would suddenly turn a greyish-white colour; she was sometimes restless, and cried out in her sleep; her bowels were open regularly once a day, but there was mucus in

the stools at times; there were no worms; lately her breath had smelt unpleasant; she had some nocturnal enuresis; the symptom upon which the mother laid special stress was the extreme fretfulness of the child, and this had become worse as the symptoms of digestive disturbance increased. Often sleep is disturbed, the child either lies awake long after going to bed, or wakes several times in the night and has difficulty in going to sleep again,—a symptom which to my mind is always suggestive of flatulence in stomach or bowel. This relation of insomnia to indigestion is worth remembering; sleeplessness is often an exceedingly troublesome symptom both in infancy and in childhood and its causation may be as obscure as its treatment is difficult; but if the possibility that indigestion may underlie it is borne in mind, a revision of the dietary may be successful where other measures have failed.

A common symptom of indigestion is the occurrence of night-terrors. The child generally about an hour or two after he has fallen asleep starts up in a half-waking condition with the eyes open but recognizing no one, and screams in terror, and perhaps speaks of some imaginary object or person at which he is frightened; with difficulty the child is thoroughly awakened, and perhaps being soothed by those about him he goes off to sleep again, and next day has no recollection of the night-terror. These attacks are particularly common with that form of indigestion in which the stools contain much mucus, the 'mucous disease' already mentioned, but they are also a frequent result of the presence of threadworms in the intestine, whether from the direct irritation of the worms, or from the mucous catarrh which is associated with their presence. Night-terrors in children are, I think, in the very large majority of cases of intestinal origin, and the irritation of undigested food is an important factor in their production. The following case illustrates the association of disturbed sleep with indigestion. Louis G., $3\frac{10}{12}$, was brought to me because his appetite was very poor and he suffered with sleeplessness. On going to bed he quickly fell asleep, but would wake about 10 p.m. and lie awake till 4 a.m.; on several occasions he had started up screaming in fright, with the usual symptoms of night-terror; on other nights he would sob for hours in his sleep. He was pale and puffy under the eyes, his abdomen was very large, and he had occasional colicky pains; his motions contained some mucus but no worms.

The complaint that a child 'changes colour', that is, goes very pale frequently without obvious cause, is exceedingly common,

and points I think almost invariably to gastro-intestinal disorder, whether it be indigestion as it usually is, or the mucous catarrh which is associated with the presence of threadworms. The pallor comes on quite suddenly and passes off after some minutes. At one time I supposed that it was never accompanied by loss of consciousness, but I have found that rarely the child does actually lose consciousness, and may even sink down as in a faint. The term ' vasomotor epilepsy ' has been applied to this symptom, but it seems to me in every way a most undesirable name, for it suggests a relationship to ordinary epilepsy for the existence of which we have no warrant whatever ; indeed, the clinical fact that there seems to be no tendency for such attacks to be replaced by any other epileptic manifestations, is strongly opposed to any such relationship ; it is not even proved that the pallor is due to any primary vascular disturbance ; it is quite conceivable, indeed it is probable, that the pallor is due to cardiac inhibition. It is evident that the heart's action may be considerably affected by indigestion, for the coldness and the blueness of the hands and feet, which is a very common symptom of indigestion at all ages from infancy upwards, is most reasonably attributed to feebleness of cardiac action.

' My child is always so tired, he never used to be so, but now he complains of feeling tired after he has walked ever so little ' ; this is a tale that one hears again and again, and the subject of it is generally a child with just such symptoms as I have already described—a pale or sallow child, probably ' getting thin ' lately, with a poor appetite, and occasional slight pain in the abdomen, with cold hands and feet, and perhaps a furred tongue and costive bowels ; in fact, the symptoms which go with the tiredness are those of chronic indigestion.

The appearance of the child is often in itself sufficient to suggest some feebleness of digestion : the complexion lacks the clear red tinge of health, it is of a pale white or sallow opaque colour, not necessarily with pallor of mucous membranes ; there may indeed be no actual anæmia ; but much more characteristic is the appearance about the eyes, which is well described by the mother as ' dark under the eyes ', or ' dark rings around the eyes '. In other cases there is a translucent bluish puffiness of the eyelids, most often I think in the lower eyelid. The dependence of these appearances about the eyes upon indigestion is extremely frequent ; both of them, however, are also associated with the presence of threadworms in the intestine. It is curious how many symptoms are common to indigestion in children and to the presence of these

worms in the bowel. I have often thought that the explanation may lie in the fact that both conditions produce a mucous catarrh in the bowel, and that it is this common factor rather than the particular form of irritation of the intestinal tract which determines the symptoms. Puffiness under the eyes is, of course, not peculiar to these conditions: I have often noticed it in children with cyclic albuminuria, but the doubt has arisen in my mind whether in these cases also there may not be some digestive difficulty, a possibility which is also suggested by another, not very rare, association with cyclic albuminuria, colicky pains in the abdomen. The puffiness which accompanies whooping-cough and nephritis is usually more marked, and less limited to the eyelids than is that of indigestion; but occasionally with indigestion only, and perhaps particularly where the child is passing excess of mucus in the stools, the puffiness of the eyes is so marked as to suggest one or other of these diseases.

Enlargement of the abdomen is by no means always present with indigestion, but it is very frequent; I frequently see children concerning whom the mother's chief anxiety is that 'the stomach is so large'; and well it may be, for the child is usually being fed on a diet in which bread-and-butter and porridge and potatoes and fruit play far too large a part. As in the infant in the first two years of life, the abdominal enlargement which results from chronic flatulent distension in these older children sometimes leads diagnosis astray, and abdominal tuberculosis is suspected where only digestion is at fault. The distinction lies chiefly in the evidence from palpation, the characteristic doughy or infiltrated feeling of tuberculous peritonitis is lacking, no abnormal masses can be felt, and inquiry almost always elicits a history of other symptoms such as I have described pointing to indigestion.

The child with chronic indigestion sometimes complains of nausea, and there may be vomiting, perhaps once or twice at intervals of a week or two: sometimes the vomiting seems to coincide with a definite exacerbation of digestive disorder, the breath becomes offensive, the tongue furred, and the complexion sallow, and therewith the child vomits two or three times within a day or two, and perhaps the temperature rises to 100° or 101°; probably the mother calls this a 'bilious attack', but the history and the result of dieting shows that although the acute attack lasts only a couple of days or so, it is part of a much more chronic disturbance of digestion.

The stools in such an exacerbation are very apt to be pale in

colour, or even white ; and here I would point out how common is paleness or actual whiteness of stools in the dyspepsias both of infancy and of early childhood. No doubt this absence of pigment from the stools means some deficiency in the function of the liver. The late Dr. Cheadle [1] pointed out that in the cases of wasting with white stools, to which he gave the name of acholia, bile pigment and bile acids may be completely absent from the fæces; but it would be a pity if this absence of bile from the stools were regarded as always constituting a disease *sui generis*, it is rather a symptom, and a very common one, of indigestion, especially in infancy and early childhood. It seems certain that both the liver and the pancreas sometimes fail to contribute their share to the digestive secretions in the bowel, and it may be that, as Dr. Cheadle suggested, the irritation of dentition exercises some reflex inhibitory influence upon these organs; but I think that more often the inhibiting influence is from irritating material in the bowel, and that the pale or fatty stools are the indication of deficiency in hepatic or pancreatic function, and are usually a secondary phenomenon; certainly they often do not make their appearance until there has been for days or even weeks obvious evidence of indigestion.

The offensive odour of the breath which I have mentioned incidentally is another complaint for which children with indigestion are brought to the doctor; of course there are other reasons which may account for this trouble, foul and carious teeth, decomposing secretion in the crypts of chronically enlarged tonsils, not to mention the much more pungent offensiveness of ozæna, or of bronchiectasis, but the heavy unpleasant odour of the breath in indigestion is usually less persistent than in these conditions, and certainly has none of the metallic nauseating smell of ozæna or bronchiectasis ; it is usually diminished or abolished for a time by a dose of calomel, but to prevent its recurrence careful modification of diet may be necessary.

There is another curious symptom of which children sometimes complain, and I have several times had them brought to me on account of it, namely, 'shortness of breath after meals': the child has a sensation of being unable to get air enough, in fact suffers with a kind of air hunger, so that he takes a deep inspiration at intervals, and may even, as in one case under my observation, want the windows opened, so that he may get more air. As an isolated symptom this might puzzle any one who is not familiar with it ; but it is so characteristic of indigestion that

[1] *Lancet*, May 30, 1903.

once known it should always suggest this cause. Children also, like adults with dyspepsia, sometimes complain of palpitation or rather of distress and discomfort, which are found to be associated with violent beating of the heart. Lionel C., aged 3¾ years, was brought to me for such attacks : ten days previously just after his dinner he had seemed ailing, and it was found that the heart was palpitating violently, and continued to do so for a few minutes, then the attack ceased ; there was no recurrence until five days ago, when about two hours after dinner a similar attack occurred again ; a bismuth and soda mixture prevented a recurrence of attacks.

In the early years of childhood, up to five or six years of age, as in the first two years of life, one indication of digestive disorder is lichen urticatus ; it is, however, far less common in these older children than in infants. Peggy D., aged 8½ years, was brought to me because she had become so nervous lately ; she was afraid to go upstairs in broad daylight alone, and at night she was terrified if left by herself ; lately also she was ' always tired ', and it was noticed that she turned pale suddenly at times ; her appetite was very variable, her bowels costive, and she complained at times of pain about the navel. Spots (of lichen urticatus) some of which were present when I saw her, came out occasionally. This was in every way a characteristic history of the child with chronic indigestion, but the lichen urticatus is, in my experience, not a very common feature at this age.

A much more rare result of chronic indigestion is a curious dryness of the hair, which looks ' dead ', loses its natural gloss, and easily comes out, so that the hair becomes sparse ; this is probably not peculiar to chronic indigestion, but I have notes of cases in which the child's ill health was apparently the result only of chronic indigestion, e.g. Grace R., aged four years, was brought for occasional attacks of vomiting and offensiveness of stools ; she turned pale often suddenly ; her breath often smelt unpleasant : her hair lately had become very dry and dull-looking, and came out easily. There was no physical sign ; the child was fairly nourished, and apart from her indigestion, the child seemed healthy.

Treatment

I turn now to the treatment of indigestion in childhood, and first I would emphasize the fact that faulty digestion does not necessarily mean faulty diet. In every case it is right and necessary to investigate the diet carefully, but there are other

points to be considered. Again and again I have seen children suffering with severe symptoms of indigestion whose diet was excellent but whose teeth were atrocious, and a few visits to the dentist have cured the dyspeptic symptoms which would have, and sometimes had already, been treated in vain with dieting or drug. In some of these cases the trouble arises from actual deficiency of teeth, the child has lost some of the upper or lower molars on one or both sides, so that the remaining molar is little or no use, having no corresponding tooth in the lower or upper jaw, as the case may be, to bite upon ; in others not only is the bite of some of the teeth prevented by the broken-away carious condition of the corresponding tooth, but the tenderness of the teeth and fear of pain prevents the child from masticating its food, which is therefore bolted without proper chewing.

Even without decayed teeth, children are only too apt to bolt their food without proper mastication ; with many this is simply faulty habit, and to some extent means faulty training, and may be remedied by admonition, but it is often a habit very difficult to break, and it may be necessary for a time to keep the child upon 'sloppy' foods, to overcome the digestive disturbance ; certainly, if there is any tendency to bolt food, it should all be reduced as far as possible to a thoroughly broken up condition before it is given ; fish, meat, or poultry should be shredded, or pounded, or very finely minced, and potato should be mashed so thoroughly as to be reduced to a powdery or flakey consistence, and similarly other foods should be given as finely divided as possible.

But food-bolting is not always the result of mere faulty habit or of decayed teeth, it is the result in some cases of faulty domestic or school arrangements ; in our top-speed twentieth century life even a child suffers from the all-pervading rush, and ten minutes have to serve to dispatch a meal in time to catch the train or electric car to school, and it is little wonder if the hastily swallowed meal is ill digested : often a different arrangement of the meal-time could be made so as to leave the children ample time for a leisurely meal before it is time for school. Nor is it only the exigencies of school which are responsible for too hasty meals ; a child even in its earliest years often takes more interest in games than in meals, and if he be interrupted in the midst of some interesting game to come to a meal, will hurry over it, if allowed to do so, in order to return as soon as possible to his play. This should not be permitted either at school or at home : by insisting upon the child's remaining at table until his

less-hurrying companions have finished their meal, or until a proper time has been spent at table, this inducement to food-bolting might be avoided. Mothers and nurses are sometimes responsible for this habit by actually hurrying a child, and telling him to 'be quick and finish his meal'. Of course there are many children who if not corrected will dilly-dally over food, paying so little attention to it, that quite an inordinate time may be spent; but this is no justification for making a child swallow its meals hurriedly. In the case of little children about three or four years of age, who 'play with their food', as the mother says, I have seen a mother or nurse hasten the completion of the meal by feeding the child with a spoon at such a pace that the child had scarce time to take breath between the spoonfuls, and in one case the mother herself noticed that the child, owing to this hasty feeding, suffered with indigestion and vomited after these lightning meals.

One other point in domestic arrangement; the time of the midday sleep in relation to food is not, I think, altogether unimportant. It goes without saying that this excellent institution should be kept up as long as it is practicable. I like it continued, if possible, until the child is fully seven years old, but I do not think that it is wise that a child should be put to sleep immediately after his dinner, nor that he should take his dinner immediately after awakening; either practice is apt, so it has seemed to me, to favour indigestion: the best time for the sleep is before dinner, but so that the child is up again and running about for half an hour before the mid-day meal. Such details may seem trivial, but nothing that plays a part in the production of ill health should be beneath the notice of a physician; the whole train of symptoms which are associated with indigestion may arise from causes such as these.

The diet has now to be considered, and I shall preface my remarks on this subject by two pieces of practical advice, which are of wide application in dietetics: the first is this, in recommending this or that article of food, never forget that what is one person's meat is another person's poison: the second has been oft quoted, but will bear repetition; it is this, never advise what a patient should eat until you know what he does eat. We cannot always know from personal observation the idiosyncrasies of a particular child whom we are called upon to diet, but it is well to recognize that some children have such idiosyncrasies; the more I see of children the less I am inclined to pooh-pooh the mother's tale that the child 'cannot take this

or that food'. It may be perfectly true as a generalism that a particular food is an excellent one, and an easily digestible one, but it may, nevertheless, be anything but a suitable food for a particular child; eggs, for instance, are most valuable in the feeding of children, and are often the best food to replace some of the starchy foods which so commonly cause indigestion, but there are healthy children who, in spite of a liking for eggs, are made sick by the smallest quantity of egg, even when it is given without their knowledge in their food. As an unusual instance of intolerance of a particular food, I may quote the case of a boy about fourteen years of age who was said to be unable to take porridge because it was alleged the face swelled after he had eaten this food. Whilst he was under my observation a plate of porridge was given one day for breakfast; before the meal was finished the left half of the face was beginning to swell, and by the time I saw the boy about twenty minutes later the swelling was so considerable that the left eye was almost completely closed by œdema of the lids; after a few hours the swelling disappeared. These cases of so-called angeioneurotic œdema are, of course, very exceptional, but they serve to emphasize the importance of the personal equation in matters of diet.

The ascertaining what a child does eat, requires some care; a mother is very apt to give the doctor an admirable outline of an ideal diet, which represents the truth, but not the whole truth; she mentions an egg or some bacon fat with bread and butter for breakfast, a midday meal of chicken or joint followed by milk pudding, and tea consisting of bread and butter with sponge cake and milk, but she omits to mention that the child has a raw apple before breakfast, that the bread eaten is whole-meal bread, that the child has a special liking for potatoes and makes the midday meal chiefly of potato, and that sweets and biscuits are eaten at odd times between meals, and that he sometimes stays up at night to dinner with his parents, and then has just a taste of whatever is going, including perhaps some pastry and a few nuts at dessert. The diet must be ascertained in detail; it is commonly the details which are at fault rather than the general plan.

Now, as to food for children past the age of infancy, I think it may be laid down as a useful general rule that the dyspepsia of childhood is most commonly a carbo-hydrate dyspepsia: certainly in the large majority of cases treatment upon this hypothesis gives the best result. There are children who prefer bread and butter to almost any other food, and by cramming them-

selves with what Shakespeare aptly describes as 'distressful bread', they set up a condition of chronic flatulent distension of the abdomen, so that the child is brought to the doctor because 'the stomach is so big', and 'the child is getting thin'. For the child with indigestion it is generally advisable to cut down the amount of bread to a minimum: unfortunately it is difficult to find anything to replace bread satisfactorily, but by adding something of a different nature to the meals, for instance, giving some bacon or fish for breakfast, and an egg for tea, we can satisfy the child's appetite with less bread, and I think that even plain light biscuits, such as 'cream crackers' or Marie biscuits, or, still better, one of the various babies' biscuits, such as Robb's Biscuits, or Hill's Malted Rusks, which may be eaten dry with some butter, are less harmful for these children than bread. I think also that bread soaked in milk is less indigestible than dry bread, and a teacupful of this sopped bread may form part of breakfast or tea. Plain Madeira cake, of course only to the extent of a small slice, may also form part of the tea, and is, I think, generally more suitable in these cases than sponge cake, which is apt to be tough and dry. I often find that a child with chronic indigestion is having brown bread, which the mother supposes to be more nourishing than white bread. I am convinced, from my own observation, that brown bread is often responsible for digestive trouble in children. It is very noticeable how easily the mucous membrane of the intestinal tract is irritated in children so that excess of mucus is passed in the stools, or the child has colicky pains and wastes, when foods are habitually given which leave much coarse residue in the bowel; such a food is brown bread, and the coarser the brown bread the worse the result. Whole-meal bread, for instance, is particularly bad for such cases, indeed I doubt if it is ever wise to give a coarse brown bread to children. Let any one who doubts the irritating capabilities of such bread give it for a couple of days to a child of four or five years old, and then observe the stools, which are seen even with the naked eye to be studded with coarse husk-like particles of entirely undigested grain. There is one more point to which I would draw attention in connexion with bread, namely, the erroneous idea that bread becomes more digestible when it is toasted: so far from this being the case, I venture to assert that as a rule toast is far more likely to cause indigestion than untoasted bread, especially when, as usually happens, only the outer surfaces of the bread are toasted, leaving the middle portion tougher or less easily broken up by the teeth and mixed

with the saliva than is plain untoasted bread. Hot buttered toast is even more objectionable in this respect.

Amongst breakfast dishes there is one which is a fruitful source of trouble in children, namely, oatmeal porridge. Let me not be misunderstood; every one knows that porridge suits many children excellently, but none the less it is true that many children with symptoms of indigestion will begin to improve directly the breakfast porridge is discontinued. One symptom in particular I have repeatedly noticed to clear up when the porridge was stopped, namely, lack of appetite for the rest of the meals. Oatmeal porridge, when made with a coarse or medium oatmeal, gives much the same appearance in the fæces as does brown bread; the undigested fragments of oatmeal can be seen scattered throughout the stool, and may well keep up that chronic mucous catarrh of the intestine which is so common a feature with dyspepsia in childhood.

In connexion with the child's dinner, potatoes are one of the most troublesome foods: most children are very fond of potato, and many children suffer much from it. For the child who is passing much mucus with the stools, potato and fruit are particularly harmful. It is generally wisest in such cases to prohibit potato altogether for a few months, and then to have it all thoroughly mashed with a proper potato-squeezer, not with a fork, which allows unbroken pieces to pass between the prongs. In theory potato should be an easily digestible form of starch, but where a child masticates badly I have seen pieces almost as large as a filbert passed unaltered in the stool, and even when the child does not apparently bolt it unmasticated it still seems to cause irritation in some children.

Of other vegetables I like best for children with weak digestion green vegetables without stalk, such as spinach, the head of asparagus, the leaf of 'greens', thoroughly mashed Brussels sprouts, and the head of cauliflower and seakale, also well mashed; vegetable marrow, too, generally agrees well; but artichokes, carrots, turnips, and onions, are to be avoided.

In the way of meats, roast beef (not salt beef) or mutton are generally suitable; there are some children who seem to digest mutton better than beef; either should be given underdone if the child will take it so: breast of chicken or turkey, boiled sole, plaice, turbot or hake, any of these are good: sweetbreads and tripe, the latter boiled in milk and served with bread sauce without onion, are easy of digestion: brains also boiled in milk and mashed up with bread sauce make an excellent food, but one

which only a few children will take. For breakfast, the gravy of fried bacon, and for children past the age of three years a little well-minced fat of the bacon is a valuable food ; most children like it, especially with bread crumbled and soaked or fried in the gravy. Eggs, as I have already suggested, may be given for tea if not for breakfast. I often order eggs both for breakfast and tea for children who take them well. As a useful addition to tea, also, sardines can be given occasionally, most children like them and take them without any digestive difficulty.

One of the chief offenders in the dyspepsia of childhood, is fruit. As a rule this is being given with the idea of preventing constipation, and the result is anything but satisfactory. In the first place the bowels gradually become accustomed to the irritating indigestible residue of the fruit upon which its laxative effect depends, so that more and more fruit is required to fulfil the purpose : in the second place this same indigestible residue sets up a chronic mucous catarrh of the intestine which may interfere considerably with nutrition. To give a child a raw apple half an hour before breakfast and a banana after breakfast is quite sufficient in many cases to keep up a troublesome chronic indigestion. I mention these two fruits in particular as being specially productive of digestive trouble : there is a popular misconception as to the banana ; it is supposed to be easily digestible : the fibres which run lengthwise down the centre of the banana are so indigestible that they are often passed almost unaltered in the stools. I have more than once had children brought to me by their parents on account of the appearance of extraordinary worms in the stool, the ' worms ', as it turned out on examination of the fæces, were nothing but undigested banana fibres. At any time of day a raw apple is likely to aggravate indigestion in a child, but one can hardly imagine a worse time to give it, or any other fruit, than an hour or half an hour before breakfast, thereby taxing the digestive powers heavily just before they should be applied unimpaired to the task of breakfast.

If fruits are to be given at all they should either form part of one of the three main meals of the day, or should be given at a time which is not less than two hours distant from the preceding and coming meal, for instance, at 11 a.m., when breakfast is at 8 a.m. and dinner at 1.30 p.m. But purely as a matter of experience I would strongly urge those who have to deal with children's indigestion to forbid all raw fruit, except possibly the juice of grapes and of orange, though even these will have to be stopped in some cases. Any food that contains much indigestible pip

or fibre is bad, and for this reason pears and figs, whether cooked or uncooked, are harmful; and the dried fruits, currants, sultanas, and raisins are bad. Of cooked fruits the juice of any stewed fruit without the substance of the fruit can be given; in this way prune juice may be allowed, but the prune itself is to be forbidden on account of its skin. A soft-baked apple seldom does any harm.

On similar lines the choice of jams is limited; it is generally wise to forbid all the ordinary jams which contain the substance of the fruit (including its pips or skins) and to replace these by the clear fruit jellies which can now be obtained almost anywhere. For children who like very sweet things honey or black treacle or golden syrup is allowable, and Frame Food Jelly is good, having the advantage that it has some diastasic value.

So far I have dealt only with eatables, but I would not have it thought that indigestion comes only from what a child eats, it may come from what the child drinks. A common fault is excess of milk in a child's diet; I see many children of seven or eight years old who, in addition to a substantial dietary, are made to drink a quart of milk a day, in which perhaps milk sops and milk puddings are not included, and then the parents wonder that the tongue is furred, and the breath heavy-smelling, and the child seems languid and evidently has indigestion. By all means let a child have as much milk as it can easily digest, but remember that it is quite possible to overdo this important item in the diet. It is often advisable to dilute milk slightly for children with indigestion, and in some cases the child will be better if the amount of milk is greatly reduced. Tea and coffee, of course, should not be allowed at all to the child with indigestion, and to any child should be given only extremely weak: cocoa is usually taken well.

The time when drink is taken has also some importance; to drink at the beginning of a meal, or to wash mouthful after mouthful down with frequent drinks is to favour flatulence and indigestion; the child who suffers from indigestion should be taught to postpone drinking until the end of the meal, and, if he be a thirsty child, it may be well to let him have a drink of water one hour before his meals.

Before leaving the subject of diet there is one other point I must mention, for it is one which I believe is often overlooked in the regimen of children beyond the age of infancy, namely, the intervals between food. I have thought that in many cases indigestion was due in no small degree to the giving of food too frequently.

Surely if an infant suffers, as it very frequently does, by feeding at intervals of two hours, say, at the age of six months, it is only reasonable to suppose that other children too are likely to suffer from a similar cause. This will apply particularly to feeding before breakfast: if a child wakes at 6 a.m. and does not have breakfast until 8.30 a.m. or 9 a.m. it is wise to give a biscuit or a little diluted warm milk: but if the child does not wake till 7 a.m., and has his breakfast at 8 a.m. or 8.30 a.m., the child is better without any food before the breakfast. Similarly in the middle of the morning, if a child has breakfast at 8 a.m. and does not dine till 1.30 p.m., it is advisable to allow some light refreshment such as milk and plain cake or biscuit at 11 a.m., but if breakfast is at 8.30 a.m. and dinner at 1 p.m. a child will often be better without any mid-morning food, the appetite comes uncloyed to dinner, which is from the physiological standpoint an important meal.

In this connexion must be mentioned a fault of regimen, which is responsible for continued ill health in some children, namely late dinner. Not infrequently I see boys or girls at the school age, sometimes even not more than twelve years old, who are having dinner at 7 p.m. or 8 p.m. with their parents, with the result that they are pasty-faced, unwholesome-looking children, suffering with lassitude or headaches in the morning, and showing in various ways the symptoms of indigestion. Even at fifteen or sixteen years many children are much better without any meal after 5 or 5.30 p.m., except a light supper consisting of such things as biscuits, Madeira cake, cocoa, Benger's Food, or milk.

Lastly, I must say a few words about the drug treatment of indigestion in children. 'Going so thin', as I have already said, is one of the commonest complaints from the mother in these cases: there is a tendency, I think, with the medical profession as well as with the laity to fly to cod-liver oil, as the sovereign remedy for 'going thin'; but not wisely, for we can do better: indeed, in some cases cod-liver oil not only does no good, it seems actually to increase the trouble by retarding digestion. Far more useful in most of these cases is a good preparation of malt; by 'good' I mean one of those made by reliable firms, so as to contain a large proportion of active diastase. The value of these malt preparations resides more in their diastasic value than in their worth as containing sugar. The chief difficulty for the child's digestion as a rule is the starch-containing food, and it is upon this that malt exercises its digestive power as a diastasic. I attach considerable value also to those preparations of malt

which are combined with pepsin or a pancreatic extract; there are several such in the market.

In ordering malt for a child it is easy to give too much, with the result that the large quantity of malt sugar in it sets up some gastric disturbance; this I have seen happen many times, and for this reason I prefer the liquid preparations, which allow of accurate measurement in a medicine glass which is impossible with the thick viscid preparations; 1 to 2 drachms three times a day is a proper dose for a child up to ten or twelve years of age.

But even malt, though it gratifies a mother's heart as being of a 'feeding nature', is not always advisable: often a mixture of rhubarb and soda, with some nux vomica to aid the gastric peristalsis, will do more good than anything; and if there is not much flatulence and the appetite is poor, I have often found a mixture of Acid Phosph. Dil., ℥x, Tinc. Nucis Vomicae ℥iij, Glycerin ℥xv, Aq. Anethi ad ℥ij, three times a day, very useful. A very important point in the treatment of indigestion is to keep the bowels working regularly: it is remarkable how very frequently chronic constipation is associated with chronic indigestion when one might have expected that the bowels would rather have been loose owing to the undigested food. I have considered the treatment of constipation elsewhere, here I will only add that in these cases it is not sufficient to open the bowels by mechanical stimulation of the rectum by suppositories or enemata; the greatest benefit will be obtained only by giving some drug regularly every day by the mouth until the bowel is educated to a better habit; and, above all, the attempt to overcome the constipation in these cases by fruit and other irritating food must not be allowed.

CHAPTER XIV

THE MEDICAL ASPECT OF DENTAL CARIES IN CHILDHOOD

CARIES of the teeth in children is a subject which must interest every medical man who has to deal with diseases of childhood, for dental caries is an important cause of ill health in children, and, I venture to think, a cause which is too often overlooked. Moreover, there is a converse fact which is not less important, that ill health in the child is responsible for many cases of dental decay.

Childhood is the period at which growth and development, physical and mental, are at their greatest activity, and when, therefore, it is most essential that the tissues should be properly nourished. If it be true, as it certainly is, that dental caries interferes with the proper assimilation of food, then its occurrence in childhood is likely to be specially mischievous.

As to the frequency of dental caries in this country amongst children at the school age, there is very general agreement; the statistics of most observers show that 80 to 90 per cent. of school children have some decayed teeth. I have made a small series of obervations on this point myself with the kind assistance of Dr. C. S. Singer and Dr. R. Todd, amongst children under twelve years of age brought to hospital for treatment of other conditions.

In all 354 children were examined; out of 226 between the ages of five and twelve years, 187—that is 82·7 per cent.—showed carious teeth, while out of 128 between two and five years of age, 50—that is 39·8 per cent.—showed caries. Of the 226 children between the ages of five and twelve years, 55 had not less than six teeth carious or missing, many had seven or eight decayed teeth, some ten to fifteen; and amongst the children less than five years old four or five carious teeth were not uncommon, and some had nine or ten.

It is clear that dental decay is extremely prevalent amongst children, and the question arises, How far does dental caries at this age cause any mischief beyond local pain and discomfort? It were much to be desired that parents as well as medical men should realize how much interference with the health of a child

may, and too often does, result from dental caries, and this although there may never have been the slightest toothache. The commonest evil from decayed teeth in children is disorder of digestion, and digestive disorder in childhood means far more interference with nutrition than it does in adult life, for in childhood nourishment is required not only for the maintenance of structure already built, but also for the building of new structure, and failure of digestion means failure of nourishment. Hence it comes about that wasting is one of the commonest symptoms of digestive disorders in childhood, and this disorder may be, and often is, dependent upon caries of the teeth preventing thorough mastication. Common enough is such a case as this :

M. A., aged 6 years, has been 'getting thin' for several months: she has lost her appetite, and 'her stomach is getting large', a history suggestive enough of abdominal tuberculosis; but examination shows no sign of tubercle anywhere; there is only one tooth, a very carious molar, remaining more or less whole in the upper jaw, where all the rest of the teeth are only decayed stumps; in the lower jaw there are also several decayed teeth. The child has, in fact, no wherewithal to carry out that mastication which is so important for the digestion of solid food.

It is unnecessary to multiply instances of this kind—they are only too common; I will only repeat what I wish specially to emphasize, that interference with nutrition is one of the commonest results of caries of the teeth in children, and one perhaps which is most likely to mislead both parents and medical men; the child who is wasting may be dosed in vain with this drug and that when the one thing needful is treatment of decayed teeth.

It has been said that the child is an inveterate food bolter : very often he is, but not always from sheer perversity; the reason may be caries and tenderness of teeth, and the result of swallowing food unchewed may be not only impairment of nutrition but also colicky pain in the abdomen. A boy of eleven years was brought to me for pains in the abdomen; his appetite was said to be very bad; there was nothing abnormal to be found in the abdomen, but examination of the mouth showed that thirteen of his teeth were carious. The dental condition was improved as far as possible by a dental surgeon and some alkaline mixture was given, after which the pain in the abdomen rapidly diminished.

Failure of appetite I have already mentioned incidentally as one result of the disturbance of digestion due to carious teeth; it is a common symptom of dyspepsia in childhood and one which causes no little anxiety to parents, and I think often puzzles the medical man by its obstinacy. There are many causes for failure

of appetite in childhood, but certainly amongst them must be reckoned caries of the teeth. In connexion with disordered digestion from dental decay I must mention also the anæmia which some children show when they have extensive caries of teeth. Is this also the result of impaired nutrition from disturbed digestion ? or does it mean some chronic poisoning ? Excellent work has been done on the bacteriology of dental caries by Mr. Goadby and others, but I think more investigation is wanted of the chemistry of the mouth in dental caries. Dr. William Hunter has drawn attention to the effects of oral sepsis on the blood ; certainly the foully offensive smell of the breath in children with extensive decay of teeth makes it difficult to believe that they can escape septic absorption altogether, and it may be that the anæmia to which I have referred is one result of such absorption. I have spoken of the digestive disorder which is associated with dental caries as if it were due only to mechanical disability of mastication, but it seems at least possible that there may be other factors. Any one who has tested the secretions about carious teeth knows that they are often distinctly acid. It may be that products of acid fermentation mixing with the food retard digestion, or it may be that other products of oral decomposition which are constantly being swallowed by these children set up some gastro-intestinal disturbance. However this may be, there is no doubt that decayed teeth afford a nidus for bacteria which may interfere with the child's health either by their local effect—for instance, by producing some form of stomatitis or gingivitis, and the latter is very common amongst children with decayed teeth—or by their passage into neighbouring lymph glands, or even by producing some general blood infection.

This brings me to a very important question—the relation of caries of the teeth to tuberculous infection of the lymphatic glands in the neck. Tubercle bacilli have been demonstrated in carious teeth ; carious teeth are often associated with enlarged, sometimes with undoubtedly tuberculous, glands in the neck ; it has been assumed, therefore, that decayed teeth are a source of tuberculous infection of glands in the neck. Now, this is a proposition of extreme practical importance ; and whilst I would not for a moment deny the existence of such a relation, I think there is a great need for careful investigation to ascertain how often such infection occurs. Smale and Colyer, in their work on *Diseases of the Teeth*, quote Odenthal's observation that of 987 children examined, 70·7 per cent. (697) showed glandular

enlargement, and more than half of these had carious teeth, whereas of nearly 29 per cent. (267) who had no glandular enlargement only five showed carious teeth.

Caries of the teeth is, as I have shown, extremely common in children, and enlarged glands in the neck are also extremely common, but it must be remembered that pharyngeal conditions, adenoid hypertrophy, and tonsillar enlargement, are probably responsible for much of the glandular enlargement, and it is no easy matter to distinguish in all cases between these various sources of irritation. We want more information as to the proportion of cases in which glandular enlargement is directly due to the teeth, and also as to how often the glandular infection from decayed teeth is of tuberculous nature. Even if it could be proved that tubercle bacilli never make their way directly from a carious tooth to a lymphatic gland, it would still, I think, be almost certain that decayed teeth in children are indirectly responsible in no small number of cases for tuberculous infection of glands. Whatever causes swelling of glands in a child, whether it be the catarrh of whooping-cough or measles causing the mediastinal glands to swell, or a catarrh in the naso-pharynx causing the cervical glands to swell—whatever the cause may be, it converts those glands into a *locus minoris resistentiæ*, and, especially in the child of tuberculous family, is a powerful predisposing cause of tuberculous infection of those particular glands. For this reason I hold that decayed teeth are a real danger to the child whose family history or whose own past history shows a tuberculous tendency.

There is another direction in which caries of the teeth seems to be responsible occasionally for more or less serious trouble in childhood, namely, as a cause of certain nervous disorders. I refer particularly to habit-spasm and epilepsy. With regard to the former, there is no doubt that some slight local irritation, for instance, blepharitis, a sore nostril, or eyestrain, is often the starting-point of habit-spasm in a child ; it might, therefore, be expected that dental worry, whether it be the eruption of the second teeth or the discomfort of a decayed tooth, might also start a habit-spasm. I cannot consider here how this comes about, whether by increase of general nervous excitability, or by initiating muscular contractions which, at first voluntary and purposive, soon become habitual and involuntary, but I will only say that I have known very rapid improvement to occur in facial habit-spasm after the dentist had dealt with carious teeth, and that the onset of habit-spasm occurs much more

often during the second dentition than at any other time in childhood.

As to the relation of dental worry to epilepsy, I cannot do better than refer to Ramskill's case (quoted by Smale and Colyer): A boy of thirteen was having fits at intervals of one to three weeks. Just before each attack he put his hand up to his face and complained of face-ache. Extraction of a carious molar was followed by complete cessation of the fits up to the time of the report, which was four months after removal of the tooth. Sir William Gowers also mentions two cases, in which epilepsy in childhood seemed to be related to toothache. Mr. J. G. Turner has also mentioned the case of a child in whom epileptic attacks were greatly reduced in frequency by extraction of dirty and decayed teeth.

There is one more nervous disorder to which I must refer, for it is one in which the part played by dental caries is too often forgotten—namely, headache. My own experience leads me to think that dental caries is by no means an uncommon cause of headache in children. Whenever a child is brought to me complaining of very frequent or daily headache, especially if the headache is very erratic in its onset and disappearance, coming and going quickly, perhaps more than once a day, I suspect the possibility of its being due to teeth, and sometimes the complete success of dental treatment has justified my suspicion. The headache in these cases is not necessarily localized like a neuralgia: it may be general, or referred to the whole frontal or vertical or occipital region, so that so far as the child's complaint goes there may be nothing to suggest the need for examination of the teeth; moreover, if there has been toothache at all, there may be none at the time when the headaches occur, so that toothache may not be mentioned.

With regard to the causes of dental caries in childhood, there are several important practical questions to be considered: Is it due to neglect of the toothbrush? How far is it dependent upon diet? How far upon the general health of the child?

For my own part, I believe that all three causes p ay their part, and that in one case one factor, in another another, plays the chief part. Of the importance of daily cleansing of the teeth I have not the least doubt, but I am sometimes impressed with the utter failure of most scrupulous cleanliness to prevent dental caries in children. I see children who from their earliest years have had their teeth cleansed twice daily, and yet have several decayed teeth; on the other hand, I see children at hospital

who have never used a toothbrush in their lives, with a perfect set of teeth. I do not know what proportion of boys in our upper-class public schools clean their teeth daily, but presumably a far larger proportion than amongst the poorer children, of whom, according to some inquiries which I have made, considerably more than 50 per cent. of those at the school age have never cleaned their teeth. From this point of view it is noteworthy that in one of our great public schools, according to Smale and Colyer, 87 per cent. of the boys, with an average age of $13\frac{7}{12}$ years, had carious teeth—a proportion fully as great as among the children of the poor. There is also a curious fact which decomposing food débris hardly seems to account for—namely, the difference in the distribution of caries at different ages. In children from two to five years old, in my own series of cases, the incisors were affected in three-fourths of the cases; in children from five to twelve years old the incisors were affected in less than one-fourth of the cases.

I mention these facts only to emphasize the point I wish to make—that lack of cleanliness is not the only factor in dental decay, and, indeed, there is, I think, proof of this, if any were needed, in the black enamel-lacking condition of the primary teeth in some children before they have emerged completely from the gum. This happens sometimes in rickety children, and, no doubt, means very imperfect formation of enamel, so that the dentine is very rapidly exposed to bacterial invasion; this may result from malnutrition in very early infancy, for it seems probable that the enamel of the temporary teeth does not reach its *full* development until some time after birth. But I strongly suspect that there are also congenital differences in potentiality of tooth development, just as there are in potentiality of growth in stature—so that it is natural to one child to have thick enamel and to another to have thin, just as one child has fine hair and another coarse; and if this be so it is at least conceivable that there may be an inherited tendency to form thin or imperfect enamel, so that in certain families there may be a special liability to early caries, as has been thought by some dental authorities.

The occurrence of dental caries specially with rickets suggests that the nutrition of a child during the first year of life may have a potent influence in determining the occurrence of dental decay in the permanent teeth. When one sees how profoundly the growth and development of a child may be influenced by improper feeding and by the gastro-intestinal disorders which result from it, one can hardly doubt that the development of the teeth,

and so their power of resistance to caries, must also be influenced thereby.

It is obvious that in the rickety child caries sometimes occurs too early to be due to any local effect of the food; but when caries occurs later, whether in rickety or non-rickety children, the possibility of direct or indirect effect of diet has to be considered. This important practical point calls for more consideration than I can give it here; but I would suggest that the influence of diet is chiefly indirect, that whatever causes digestive disturbance favours the onset of dental caries. Look at the furred tongue, notice the heavy odour of the teeth, see the aphthæ which sometimes occur in the mouth of the child with disturbed digestion—surely all these are indications of a state of mouth which must favour the growth of bacteria and the occurrence of acid fermentation which may erode the enamel.

There is no doubt that the acid fermentation of sugar has a destructive effect upon enamel, but I very much doubt whether sugar taken in the form of sweets has so directly a harmful effect as is commonly supposed, for the sugar is very quickly washed away by the saliva. I found that fourteen minutes after two large chocolate-with-sugar sweets had been dissolved in the mouth the saliva did not show a trace of sugar with Pavy's solution. Starchy food, on the contrary—such as biscuits, bread, and potato—remains in every crevice, and may cause acid fermentation in prolonged contact with the teeth. Excess of sugar and starch and the taking of either between proper meal times are common sources of indigestion in children, and may thus indirectly favour the onset of caries.

Prevention and Treatment. Lastly, the all-important question of prevention and treatment has to be considered; and here the physician must needs go delicately, lest he be hewn in pieces by the dental expert. We have heard much recently of dental inspection in schools and the need for dental supervision of school children; but I would emphasize the fact that the mischief is present in a large proportion of children *before* the school age. My own observations showed that 39·8 per cent. of children between two and five years of age had decayed teeth, and it is not at all uncommon to find dental caries in children between two and three years old. Evidently the prevention of dental caries must date from earliest infancy. The most important step in this direction is proper feeding in infancy. Moreover, if, as seems almost certain, any disturbance of nutrition occurring during the first two or three

years of life may, according to the time at which it occurs, influence the development of the primary or the permanent teeth and render them specially liable to decay, then the importance of proper feeding and prevention of digestive disorders at this age becomes still more apparent.

I would have every mother see that her child's teeth are cleaned morning and evening, especially the latter, from the time of their eruption, doing it first with a soft rag and some borax solution, and later teaching the child to use the toothbrush for himself.

With regard to treatment let me say this much: I am frequently seeing children with extensive caries of teeth, whose health is suffering partly from indigestion, partly no doubt from chronic poisoning from the foulness of their teeth. I send them to a dentist, and I am told that he advises leaving the teeth alone as they are first teeth. Now, I am well aware that there are reasons which make it desirable not to remove primary teeth if it can be avoided; but I have wondered sometimes whether the dentist realized how much the child's health and nutrition were suffering at this important period of life owing to the presence of these teeth. I must leave it to the dental expert to decide whether it is possible to save the tooth by stopping it, or whether he can only get rid of the caries by extraction, but I am convinced that one or other procedure ought to be adopted far more often than it is in connexion with the temporary teeth. Moreover, I think that if parents would take their children regularly two or three times a year to a dentist for inspection the primary teeth could often be saved by timely stopping before the extent of the caries makes this procedure impracticable. There is an unfortunate idea current amongst the laity that there is no need to take much notice of decay of the first teeth, whereas in reality decayed teeth at that period are perhaps even more harmful than at a later age, for nutrition is more easily disturbed, and its disturbance means more interference with general development, glandular enlargement is much more likely to result, and probably decay in a temporary tooth not only destroys the present tooth, but favours the occurrence of caries in the coming permanent tooth.

CHAPTER XV

CONSTIPATION IN INFANCY AND CHILDHOOD

In infants, during the breast- or bottle-feeding period, constipation is a very common trouble ; indeed it is so frequent that one is almost driven to suppose some special cause peculiar to the age. It might be suggested that the reason lies in the peculiarity of the diet at this time, a fluid food leaving but little solid residue to stimulate the intestine to active peristalsis ; but this can hardly be the whole explanation, for were it so one would expect that an infant fed upon cow's milk, with its large tough curd, would be less liable to constipation than the infant fed upon human milk with its fine flaky curd : as a matter of fact exactly the reverse is the case, constipation is common enough in breast-fed infants, but it is not nearly so frequent as in those fed on cow's milk.

The feeble musculature of the bowel has been held responsible for the frequency of this trouble in infancy, a view which is supported by the fact that feeble and premature infants are even more prone than other infants to constipation. One can hardly doubt that this lack of muscular strength in the bowel must be one factor, but it cannot be the only factor, for the feeblest infant will sometimes prove the most regular in its bowel actions, whilst a big, strong baby, fed similarly, may suffer much with constipation.

The possibility that an anatomical peculiarity may account for this tendency was suggested by some observations upon the relative length of the large intestine and the body at different ages ; these measurements were made without any reference to the subject of constipation, but they may have an interesting bearing upon the special frequency of this disorder in infancy. The function of the large intestine is chiefly the absorption of the fluid part of the food : Nature has provided for infants a food capable of rapid assimilation, and to secure abundant absorption special provision is also made of a relatively long colon. Without quoting here the tables of figures worked out from my series of observations I may say that although the

measurements varied much in individual children at the same age, they show on the whole very clearly that while in fœtal life the large intestine is very short relatively to the length of the body—at 3½ months, for instance, with a body length of 6 inches, the large intestine measured 2·5 inches—this shortness gradually becomes less marked until at full term the large intestine is about the same length as the body.

After birth the large intestine rapidly becomes longer relatively to the body, and up to the end of the second year is usually several inches, often as much as 4 or 5 inches, longer than the body. Towards the end of the second year this difference becomes less marked, and by the time the child is three or four years old the relation is reversed, the large intestine is 5 or 6 inches *shorter* than the body, and in later years this relative shortness is even more marked; for instance, at nine years I found the body length 45 inches, the large intestine only 35 inches ; at 11¼ years, with a body length of 56 inches, the large intestine measured 43 inches. From these observations it might be expected that there would be some difference in the anatomical arrangement of the large intestine at these different ages, and that if there were, owing to its relatively excessive length, some folding and kinking of this part of the bowel in infancy, these folds and kinks would be more or less straightened out in later childhood by the enlargement, especially in length, of the abdominal cavity. It was pointed out by Jacobi that this is so : the colon of an infant is very commonly tortuous and folded on itself, especially in the sigmoid region, where it often forms a large ' omega ' loop passing over to the right side of the abdomen, or even up into the left hypochondrium before finally turning down to the rectum : whereas in later childhood this tendency, though persisting in some children, is less common.

There can be little doubt that the sharp turns upon itself which the colon of an infant often makes in the sigmoid region must offer considerable obstruction to the course of the fæces ; the presence of formed fæces must still further accentuate the kinking, and if with these obstacles there is also excessive feebleness of musculature it is easy to understand why an infant should be especially prone to chronic constipation. These observations seem to explain also the clinical fact that the constipation of infancy tends to right itself as the child gets older ; the folds and kinks are diminished as the colon is straightened out by the rapid growth in length of the abdomen, and thus it may be truly said that the child ' grows out of ' the constipa-

tion. It may be that a contributing factor in some cases of infantile constipation is deficiency of bile secretion: I have elsewhere pointed out that the bile in early infancy is apt to be specially viscid, and as we all know the stools in infancy and in early childhood are extremely apt to become pale or even white at times with very slight disturbance of digestion.

I have dwelt on these inherent causes of constipation in infancy somewhat fully because, even though they afford but little guidance in treatment, they at any rate afford a reasonable hypothesis to explain the difficulty which is often found in overcoming constipation at this age: and they give us good ground for assuring the parents that there is every probability that the trouble will disappear as the child grows older.

Both in infancy and in early childhood, and even to some extent in older children, there is a negative factor which may play an important part in the production of chronic constipation; I mean the absence of voluntary effort to cultivate regularity; the child, conscious only of a weak desire to defæcate and of a much stronger desire to go and play with its toys, neglects the former in favour of the toys, and what is wilful neglect at the time very soon becomes real inability. A good nurse can do much by regularly 'holding out' an infant at certain times, or by insisting upon an older child's 'sitting down' directly after breakfast, to encourage regular action of the bowels, whilst a nurse careless in this respect may be responsible in large measure for chronic constipation in the child.

In some children a sluggish habit of mind and body seems to go together; certainly there are phlegmatic children with poor circulation who are habitually constipated, and I am the more inclined to think that the constipation is the result and not the cause of the general sluggishness in these cases, because chronic constipation is so often a marked feature with actual mental deficiency, particularly with the stolid dullness of untreated cretinism.

It is customary in textbooks to mention fissure of the anus as a cause of constipation in infancy; no doubt it is so occasionally, but in my own experience it has been very rare. I can only recall one or two such cases amongst the many thousands of infants I must have treated for constipation, and I should suppose that when fissure does occur, although it undoubtedly aggravates the constipation owing to the child's fear of pain on defæcation, yet the fissure is not originally the cause but the result of the constipation; and any treatment, therefore, must

be adapted not merely to the cure of the fissure, but also to the cure of the constipation independently of the fissure.

Amongst local causes a very important one, though fortunately a very rare one, is anal stenosis. I have twice seen children past the age of infancy with severe constipation which had been treated with unsatisfactory result with all sorts of remedies, until it was discovered by rectal examination that the anus was so small that it was difficult to get even one's little finger into the rectum : the anus was enlarged by incision with great relief in both cases. I have seen also one child in whom troublesome constipation was at length explained by a stenosis of the rectum : the child had undergone operation for imperforate anus in infancy, and after the correction of this deformity the bowels had acted, but always with difficulty : rectal examination showed a constriction about an inch above the anus. I mention these rarities because it is well to be aware of their occurrence, inasmuch as no treatment but operative interference is likely to be of any permanent use.

Results of Constipation

Foremost amongst the evil results of habitual constipation in infancy I shall put marasmus, meaning by this rather failure to gain weight, or very slow progress in nutrition, than active wasting. I lay stress upon this effect because I know from experience how little importance is often attached to constipation when a baby is not gaining weight : all sorts and varieties of feeding are tried, but the constipation is regarded as quite a secondary consideration and comes in for very scanty treatment. In many such cases the constipation is the one thing that needs attention, and if regular and thorough action of the bowels is ensured by daily administration of some suitable aperient, the weight will begin to rise without any alteration whatever in the feeding ; and even where marasmus is dependent in part upon difficulty of digestion, the correction of constipation is often as important as suitable feeding.

There is another result of constipation in infancy which I think is not very generally recognized as such, namely chronic vomiting. I do not refer to severe vomiting, such as occurs with congenital hypertrophy of the pylorus, but to the cases in which an infant brings up perhaps half an ounce or an ounce, and does this after so many of the feeds each day that the mother becomes alarmed ; it is clearly more than the usual slight 'posseting' of

a drachm or two, and the weight is not increasing as it should do. Here, again, the treatment that is required is often no change in the food or in the size of the feeds, but simply regular administration of grey powder, or some such aperient, twice or three times daily to keep the bowels working well.

I suppose that one of the commonest results of chronic constipation in infancy is screaming : the infant who is said to be 'always screaming' is very commonly suffering from habitual constipation : the bowel, unable to pass its contents along, makes vigorous but ineffectual efforts, and the result is colicky pain, which is aggravated often by the presence of flatulence, a very frequent accompaniment of constipation.

A more serious outcome of constipation in some infants is a convulsion ; no doubt there is a predisposition in such cases to convulsions, but it is the intestinal worry of constipation which acts as the exciting cause ; and I would point out that this may happen where the infant has shown no evidence of colic or pain from the loaded bowel.

The straining of a constipated infant is liable not only to cause passage of streaks of blood with the stools, but also to bring down the bowel itself in the form of a prolapse, or to produce some variety of hernia. Even in infancy constipation sometimes produces piles as in later life. I have seen them in three cases under the age of one year : one case, a girl aged seven months, after not having the bowels open for three days, showed an external pile the size of a small pea ; the two others were boys, one of whom, at the age of $8\frac{1}{2}$ months, had three large external piles, and the other, at the age of about nine months, had also external piles, being habitually very constipated. I saw one boy, at the age of $3\frac{3}{4}$ years, in whom it was stated that piles had been detected when he was taken to a hospital at the age of one month.

In older children, as in infants, habitual constipation interferes in some way with general nutrition, and although this effect is naturally less striking at an age when the gain in weight should normally be slow compared with that in infancy, still it is often quite perceptible to the medical man, as well as to the mother. Moreover, the general health is impaired in other ways ; the child is sallow or of pasty complexion ; above all, he is languid and easily tires on exertion. I always regard this complaint that a child 'gets tired so quickly' as highly characteristic of two common disorders in childhood, indigestion and constipation. The child who is constipated is apt to suffer from headaches :

the appetite is often very poor, the tongue is furred, sometimes the breath is offensive, and the child is ' not getting on '.

On the other hand, it is surprising how little acute disturbance results in some cases from prolonged complete constipation; a fact which makes it the more necessary to exercise constant supervision of children in this respect, for they will pay little attention to this matter themselves. I have notes of an infant, aged nine weeks, who was brought to me when its bowels had not been opened for eight days : of another, aged $1\frac{8}{12}$, whose bowels had not been open for fourteen days, and another, aged $2\frac{1}{2}$ years, whose bowels had not been open for five weeks. In this last case I had to break up the hard pieces in the rectum with a metal instrument before I could get the bowels to work. A boy, aged $5\frac{1}{2}$ years, was brought to me with a history that he had always been very costive ; his bowels were usually open only once a week, and he had been as long as nearly three weeks without an evacuation : his abdomen was distended and a large mass of fæces could be felt in the colon. This was not a case of idiopathic dilatation of the colon, but simply neglected constipation, which was easily set right eventually by the regular administration of a simple mixture of senna and aloes.

Treatment

Some at least of the constipation in childhood might be prevented by the encouragement of regular habit. I have already referred to the nurse's duty in regard to infants and young children : I wish to lay stress upon the mischief often done to older children by lack of care in this matter. The boy who goes off to school daily is allowed to take his breakfast at an hour which leaves him barely time to snatch a hasty meal, much less to attend to the evacuation of his bowels, before he starts for school, when by insisting upon his having his meal half an hour earlier, good digestion and the daily evacuation might both be ensured. A grave fault at some boarding-schools is insufficient provision of water-closets, so that in the short time allowed between breakfast and morning school it is practically impossible for all the boys to get their bowels open. School masters and mistresses would do well to pay more attention to this matter : there should be both ample accommodation and ample time allowed, both are altogether insufficient in some schools. The practice of forbidding children to leave the room during school hours is one which cannot be too strongly condemned : I should hardly have thought that a school teacher could be found

ignorant or cruel enough to do such a thing, but I have repeatedly come across instances in which a child had been unable to satisfy Nature's needs, or had passed urine or fæces in school because permission to leave the room had been refused : children at the school age often enough suffer from false modesty in this respect, but when a child does ask to leave the room no teacher ought to withhold permission.

Now as to the treatment of constipation, I must first consider the dietetic measures which may be of value : but let me say at the outset that in my opinion the treatment of constipation in infancy and in childhood by diet is apt to be most unsatisfactory : the digestion at this age is very easily upset, and the foods which exercise a laxative action upon the bowels are just those which are apt to disturb digestion : the dietetic treatment takes us in fact between the Scylla of constipation on the one hand and the Charybdis of indigestion on the other.

In the case of the breast-fed infant such measures are seldom practicable : it is true that occasionally the trouble is due to poorness of the breast-milk, and we may try by increasing the proteid in the mother's diet, by giving her more eggs, fish, poultry, or meat, or by insisting on the mother's resting much in bed or on the sofa, or by administering to her some preparation of malt, to increase the richness of her milk : but as a rule if any dietetic measures are to be tried it must be by direct addition to the breast-feeding.

Some have recommended that one or two feeds of a cream mixture should be given daily : but this is not altogether a satisfactory method, for it will probably be necessary for this purpose to use an excessive proportion of cream, and the result may be digestive disturbance. The administration of one or two teaspoonfuls of a solution of sugar, or of some starchy food such as oatmeal mixed with milk, may purchase an action of the bowels at the cost of flatulent dyspepsia which may be worse than the constipation. One of the foods which contain no starch but a large proportion of completely malted cereal may be given once or twice a day : such are the Allenbury Foods No. 1 and No. 2 and Horlick's Malted Milk, or Mellin's Food mixed with diluted milk according to the maker's directions ; any of these will sometimes be sufficient to keep the bowels regular, and used thus only once or twice a day to replace breast-feeds I know of no objection to them if the infant is able to tolerate the excess of soluble carbo-hydrate which they contain and upon which their laxative effect depends.

For the hand-fed infant also these completely malted foods may be a useful addition to one or two feeds in the day, if this is sufficient for the purpose, but if more of the feeds must be given to ensure regularity of action it becomes a question whether it is not better to give drugs than to risk the likelihood of flatulent dyspepsia, which occurs very readily in some children with this class of food. The substitution of brown Demerara sugar for milk sugar is sometimes successful, and I have used treacle in the same way: but to my mind any of these methods are objectionable if they involve giving more soluble carbo-hydrate than an infant should take. The limit which should not be exceeded is 7 per cent.; if brown sugar or treacle, in the proportion of a *small* teaspoonful to a three-ounce feed is sufficient, there is little or no objection to their use, but if more must be used such a method is, I think, to be avoided: I hesitate to say that there is absolutely no objection to the use of these forms of sugar when given even in proper proportion, because I suspect that cane sugar is a little more liable to set up flatulence than an equal quantity of milk sugar.

I sometimes find that an infant is having a food containing far too much cream under the impression that the constipation is due to deficiency of fat in the food, and that therefore the fat should be increased until a laxative effect is obtained. Here, again, I would caution against using a proportion of fat which exceeds what one may call Nature's limit, the 3 to 4 per cent. which is present in human milk. I am fully in agreement with Dr. Holt as to the harmful effects of excessively high percentages, such as 6 per cent. and 7 per cent., which are often being given for this particular purpose. Dr. Holt points out that not only may serious disturbance of nutrition result, but the very purpose of the added fat may be defeated, for excess of fat will sometimes cause constipation. If by raising the fat percentage, by addition of cream, to 3 or 4 per cent., we can procure regular action of the bowels, I know of no better way: but, as I have already pointed out, in order to do this we must know approximately the strength of the cream used, and even when we have calculated out the proportion of fat to a nicety, we shall still find that there are many infants who are extraordinarily intolerant of cream and in whom the effect of even a very modest addition of cream is to produce vomiting or diarrhœa. It is very rarely wise to exceed one measured drachm of average London cream in a three-ounce feed, however much the milk may be diluted: this will mean the addition of 2 per cent. of

fat to the mixture; the use of 1¼ drachms of cream (48 per cent.) in a three-ounce mixture of equal parts of milk and water would raise the proportion of fat to 4·25 per cent.

In children past the age of infancy dieting for constipation is one of the commonest causes of indigestion. The average mother has a profound conviction that it is bad to give drugs for chronic constipation: it sounds like Nature's ideal to give plenty of fruit, and surely brown bread and porridge must be so nourishing! So she plies her child with fruit and more fruit, and whole-meal bread and porridge, and at first the constipation is less troublesome, but soon it is as obstinate as ever, and now the mother is distressed to find that her child is getting thin, he is pale and puffy under the eyes, has pains in the abdomen, is so tired and has no appetite. Now what does all this mean? The food has set up a condition of chronic indigestion, which very likely has taken the form of that mucous catarrh of the bowel which has been so admirably described by Dr. Eustace Smith in his treatise on the *Wasting Diseases of Children*. This disorder is accompanied usually by constipation rather than by looseness, and the one essential in its treatment is the exclusion of such articles as fruit, brown bread, and porridge from the diet. 'But,' says the mother, 'surely drugs must irritate the coats of the bowel, if given again and again.' I venture to say that few laxative drugs in the doses required for the treatment of chronic constipation are so irritating to the gastro-intestinal mucous membrane as the foods which I have just mentioned; they owe their effect indeed almost entirely to the mechanical irritation caused by the undigested woody fibres, husks, and grains, which are often passed almost unaltered in the stools on such a diet. The stools of some children who are being fed on brown bread or porridge are a mass of undigested material, recognizable even by the most unskilled as the coarse particles of the brown bread or porridge; one can only wonder that ill results from such food are not more common. To give a child an apple before breakfast, a bowl of porridge at breakfast, banana in the middle of the morning, and brown bread at most of the meals, is to court trouble: it would be absurd to say that any one of these articles of food was intrinsically bad, we all know they are excellent as occasional foods for some children: but none the less I am sure from experience that there are very many children who stand raw fruit very badly, and that to ply a child daily with such foods as I have mentioned in order to overcome constipation is often merely to add the

troubles of indigestion to those of constipation. The proper time to give raw fruit, if it is to be given at all, is as part of one of the meals, for instance, at the end of breakfast or dinner, and if the juice of an orange, or some grapes, or an occasional banana, given thus is sufficient to keep the bowels acting regularly no harm may be done by such treatment. One of the least harmful of fruit aperients is, I think, the juice of stewed prunes, which may be given at tea-time or at supper.

Coming now to the use of drugs for habitual constipation in infancy and childhood, I shall first lay down one general principle upon the recognition or non-recognition of which rests the success or failure of our treatment; it is this: the object of treatment is not merely to open the bowels when constipated, but to prevent constipation. The great mistake which is constantly made in the treatment of this disorder is to prescribe some drug to be taken 'when required': let me lay it down as a rule that to order a drug to be given 'when the child is constipated' is a sure way to fail in the cure of habitual constipation. Whatever drug is used should be given regularly, once, twice, or three times a day, in such a dose as will keep the bowels open without purging: and its administration should be continued for weeks or months until the habit of regularity in the action of the bowels has become a fixed one: the time for omission of the drug is to be found by attempting to drop one of the daily doses or to reduce the size of the doses after several weeks of treatment, and if this is found not to alter the regularity of the bowels, the other doses can be gradually diminished or omitted. As a matter of fact it generally becomes evident after two or three months that the bowels are working so easily and well, that there is little risk in reducing and gradually omitting the medicine.

The choice of drugs will differ according to the age of the child. For infants there is one drug which is sanctioned and sanctified by tradition from time immemorial, castor oil: and I know no drug which is responsible for more chronic constipation in infancy than castor oil. Let it be remembered that this drug has not only an aperient action but a markedly constipating effect, which follows as an after effect when a large aperient dose is given, but which is seen more evidently when a small dose, say 4 or 5 minims, is given regularly three times a day, when it has no aperient action whatever, but makes the infant very costive. If castor oil is given, as it often is, as an occasional aperient to an infant with habitual constipation, the effect each time is to open the bowels, and then to make the child costive

again, and so the bowel is encouraged in its habit of constipation. There is no better nor safer aperient than castor oil where it is desired merely to evacuate the bowels once, especially if subsequent constipation will be rather an advantage than otherwise, as for instance in the case of an infant who has diarrhœa from the presence of irritating food in the bowel; but where there is habitual constipation there could hardly be worse treatment than an occasional dose of castor oil. For the mildest cases I like manna; a piece roughly the size of a hazel-nut may be put in three or four of the feeds daily; but this is no use for the more severe cases. For these I think that probably the most reliable drug is grey powder: I am in the habit of ordering Hyd. c. Cret. gr. ½, Sod. Bicarb. gr. j, Pulv. Cretæ Aromat. gr. j ter die, and if this is not sufficiently strong I replace it by Pulv. Rhei Co. gr. ij–iij, Hyd. c. Cret. gr. ½–¾ ter die (the stronger doses for infants over three months if required). I have been asked sometimes whether the use of such powders for several months would not be likely to injure the teeth; I have paid special attention to this point and have satisfied myself that so far from injuring the teeth the mercurial seemed, if it had any influence upon them, to act as a preservative, for the teeth of children treated with this drug were often exceptionally good. Rarely the stronger of these two powders is not sufficient; under these circumstances I have generally given a simple senna mixture twice or three times a day in addition: viz. Syrup. Sennæ ℥ x–xx, Aq. Anethi ad ʒj, telling the mother to increase the dose of this to 1½ or 2 drachms if necessary. This mixture is sometimes quite sufficient alone.

My colleague, Dr. Hutchison, introduced a formula which I have often found useful: Tinct. Nuc. Vom. ℥ j, Tinct. Hyoscyami ℥ v, Tinct. Aloes ℥ iv, Syrup. Sennæ ℥ xv, Aq. Anethi ad ʒj, which may be given three times a day to an infant of nine months.

Chronic constipation in an infant, as in an older child, is sometimes amenable to liquid paraffin, which may be given in doses of 15–30 minims, twice or three times daily (see p. 213), and I think this is less likely to upset digestion than the administration of olive oil, which is sometimes given in doses of half a teaspoonful or more.

For infants over six months of age if the stools are pale as well as costive podophyllin is sometimes useful; I have generally given it thus: Tinct. Podophylli ℥ j, Tinct. Nucis Vomicæ ℥ j, Glycerini ℥ x, Aq. Anethi ad ʒj ter die. I have thought, however, that in some cases it griped and therefore was less satisfactory than those I have already mentioned.

Where the stools are dry and hard, especially if there is much flatulent colic with the constipation, the saline aperients are good, such as sodium phosphate, of which 10 or 15 grains may be given in two or three of the feeds daily. Dinneford's Fluid Magnesia or Phillip's Cream of Magnesia are both excellent in such cases. I have used also the synthetic purgative purgen, or phenol-phthalein, both for infants and for older children. Oppenheimer's preparation of this, sold under the name of Laxoin, is in convenient form, but on the whole it has not seemed to me to be so reliable a drug as the older aperients already mentioned; in cases, however, where a fluid stool is desirable, it may be useful.

For older children I think that the preparations of cascara with malt make an excellent drug for habitual constipation; and I prefer those made in liquid form as capable of accurate measurement, which is very desirable, so that the mother may ascertain exactly what dose is necessary. One of these preparations, given in a dose sufficiently small to allow of its being given three times daily immediately after meals, has the great advantage that it not only acts as an aperient, but also by its diastasic value assists the digestion of starch which is often at fault in these children. The plain preparations of cascara without malt have seemed to me more apt to gripe, and also less reliable in their action.

A mixture like that mentioned above for infants, containing nux vomica and senna and aloes, but with larger doses of these drugs according to the age, often acts very satisfactorily.

The preparations sold as Syrup of Figs, which consist chiefly of senna, form a useful laxative for chronic constipation, and can be given in doses of 1–2 drachms every night if necessary for many weeks or months without the slightest harm. For children who object to the taste of medicine of any kind as some will, simply because it is medicine, an infusion of senna pods makes a useful aperient, as it is practically tasteless, and if necessary can be concealed in milk or any liquid food. Three to six pods should be soaked in a wine-glassful of cold water for several hours, and the resulting infusion should be given at night. A pleasant and effectual method of administering senna is to give it in the form of a fruit paste made as follows: Take 1 lb. of French plums, 1 lb. of Demerara sugar, $1\frac{1}{2}$ oz. of pounded senna, and $\frac{1}{2}$ oz. of ground ginger. Stew the plums in a little water until very tender, while hot remove the stones, then add the rest, mix and beat to a paste. One teaspoonful of this paste may be

given to a child of four years every night. Recently Agar-agar has come into use for these cases of habitual constipation; a particular preparation of this is sold under the name of Regulin. This is given in doses of one to four teaspoonfuls mixed with cream or any moist food, such as jam or mashed vegetables.

Paraffin is very successful in some cases. It is given in doses of twenty to forty minims in an emulsion, a useful formula for which is given on p. 174. When larger doses are required, it is more convenient to give the plain liquid paraffin, of which 1–4 drachms may be given two or three times daily before meals. For those who dislike the plain paraffin there are now preparations sold in which it is mixed with malt, or supplied in a jelly-like form, pleasantly flavoured and coloured. But, with all this assistance, paraffin does not always agree; there are children who are made sick, and others whose digestion is impaired by it, and it is not very uncommon for children to pass the liquid paraffin speedily through the bowels without obtaining any proper evacuation of fæces; as the mother puts it, the paraffin simply runs through the child.

For children with 'mucous disease', I think the decoction of aloes recommended by the late Dr. Eustace Smith is particularly useful: Potassii Citrat. gr. iv, Spirit. Chloroformi ℥ $1\frac{1}{2}$, Glycerin. ℥ xv, Decoct. Aloes Co. ad ʒ ij ter die.

In mild cases of constipation sulphur is sometimes very useful: it may be given either as the sulphur lozenges of the British Pharmacopœia, of which two may be taken every night by a child aged five or six years, or as the confection of sulphur, which may be combined with the confection of senna, half a teaspoonful of each.

A very useful powder to be given every night is Powdered Rhubarb 5 gr., Potassium Sulphate 5 gr.

I have seen excellent results even in severe cases of chronic constipation from Apenta Water given every morning, $\frac{1}{2}$–1 wineglassful, before breakfast.

Whichever of these various drugs are used it is to be impressed upon the parents that regularity of the bowels must be procured and maintained, if not by one drug then by another, and that the aim of treatment is to establish a regular habit, and that although it may require several weeks or months of patient perseverance in treatment to do this, it can generally be done; after a time drugs can be reduced in frequency and then in dose, and so by degrees discontinued.

There is yet the use of enemata and suppositories to be con-

sidered. On several grounds I think that these are less satisfactory for chronic constipation than treatment by mouth: the action of the bowel is made to depend on local mechanical stimulation in excess of the normal stimulus, and when this has to be repeated frequently for long periods, the bowel is certainly not encouraged to respond to the less powerful normal stimulus, but rather habituated to the tolerance of accumulated fæces, and children I think even more easily than adults come to rely upon such artificial assistance. Another, and I feel sure a very real objection, is the dilatation of the rectum and perhaps of the sigmoid which may result from continual use of enemata. In a little boy I examined who had been treated habitually by enemata, I found the rectum ballooned and evidently to some degree atonic: such a condition must favour the accumulation of fæces, especially if the sensitiveness to the normal stimulus is dulled by the continued use of enemata; and lastly I suspect, though I cannot prove, that the emunctory function of the whole bowel is less thoroughly performed when action is induced simply by rectal stimulation than when it is caused by drugs which promote the secretory activity of intestinal glands or promote the flow of bile, or stimulate the whole of the neuromuscular apparatus of the intestine.

If enemata are to be used at all—and in some cases they are inevitable, for in no other way can the bowel be made to work, while in others they may be necessary occasionally when the daily action from aperients fails—the smallest sufficient bulk of fluid should be used in order to avoid dilatation of the rectum and sigmoid; for this reason olive oil is much to be preferred to plain soap and water; for whereas 6 or 8 ounces or more of the latter may be required, generally half an ounce of olive oil with 2 or 3 ounces of warm soap and water is quite sufficient; a much smaller quantity of glycerine is effective, but this is, I think, to be avoided for it is a much more powerful stimulus, and is therefore more likely to establish tolerance of lesser stimuli; for the same reason glycerine suppositories are objectionable, but the use of a small piece of soap cut into the shape of a small suppository half an inch long is, I think, less open to objection.

I have said nothing about massage; occasionally I have found it of value for these older children, especially in combination with a morning dose of Apenta Water or some such draught.

CHAPTER XVI

INFANTILE DIARRHŒA

One of the commonest and most troublesome of the disorders of infancy is diarrhœa, and there is no disease which is responsible for so large a number of deaths as are due to diarrhœa at this age. The following statistics compiled from the Registrar-General's reports give some idea of its fatality:

	Deaths from all Causes in London.			Deaths from Diarrhœa in London.		
	Total under 1 year old.	*Under 3 months.*	*3 months to 6 months.*	*Total under 1 year.*	*Under 3 months.*	*3 months to 6 months.*
1891	20,776	9,662	4,286	2,272	608	742
1892	20,441	9,614	4,162	2,340	745	845
1893	21,814	10,282	4,752	3,265	953	1,144
1894	18,812	9,083	3,680	1,866	518	620
1895	22,252	10,091	4,755	3,803	1,024	1,282
1901	19,678	9,565	4,113	4,029	1,071	1,383
1902	18,307	8,927	3,545	2,542	590	859
1903	17,223	8,594	3,463	2,818	729	981
1904	19,012	9,050	3,948	4,408	1,076	1,474
1905	16,603	8,253	3,224	3,347	869	1,092

From these figures it can be seen that on the average 2,000–4,000 infants under one year of age die annually in London alone from diarrhœa, and that in spite of improved sanitation the proportion of deaths due to diarrhœa in the first year of life has rather increased than diminished during the past ten years. Another interesting fact also appears that a much smaller proportion of the deaths from diarrhœa during the first year occur within the first three months than in the subsequent months of the first year, a fact which no doubt has its explanation, not in any special resistance to diarrhœa at this period, for, as every clinician knows, diarrhœa is more dangerous to an infant under three months than to an older infant, but in the fact that breast-feeding is much commoner during these first three months than in later months. One might even venture to draw from these figures the conclusion that improved sanitation is a far less powerful means for reducing the death rate due to infantile

diarrhœa than is the inculcation and encouragement of breast-feeding.

At the Hospital for Sick Children, Great Ormond Street, statistics showed that 96 per cent. of the infants up to the age of nine months who died of diarrhœa were being hand-fed (4 per cent. partially, 92 per cent. entirely); only 4 per cent. were entirely breast-fed at the time of onset of diarrhœa. From the same source it was found that 90 per cent. of the deaths from diarrhœa in children occurred during the first year of life; it is evident, therefore, that to suckle an infant for nine or ten months is to save it from a very serious risk.

Here I would emphasize an important point, namely, the special liability to severe diarrhœa in infants fed upon condensed milk. It has been argued that one of the merits of condensed milk is sterility, and that therefore it is specially useful in avoiding the risk of diarrhœa in infancy. Experience proves that this is not so; on the contrary, feeding with condensed milk seems to render an infant particularly liable to severe diarrhœa. Details of the feeding of 100 infants in consecutive series, without regard to the ailment for which they were brought to hospital, showed that 12 per cent. had been fed on condensed milk, whereas similar details in a series of 112 cases of fatal diarrhœa in infants showed that at least 25·8 per cent. of these cases had been fed on condensed milk.

Among the factors which play a part in the causation of diarrhœa none is more prominent than a high mean temperature of the atmosphere. Each year in London the diarrhœal mortality begins to rise early in July, reaches its maximum in August or September, and subsides to what may be called its normal level about the middle of October; moreover, in a general way it may be said that the hotter the summer the greater is the diarrhœal mortality. The chart shown here (Fig. 10) illustrates these points How this high atmospheric temperature acts is entirely unknown. It can hardly be by direct effect, for if so the breast-fed should suffer equally with the hand-fed; it seems more likely that it favours the growth of bacteria, which are easily carried by food, especially by milk, and perhaps grow with special ease, as so many micro-organisms do, in milk. Whence the organism comes or how it reaches the milk is also mere matter of conjecture; possibly in some cases, as Dr. Nash of Norwich has suggested, it may be conveyed by the common house-fly. Dr. Ballard's observation that the temperature curve shown by the four-foot earth thermometer corresponds

more closely than that of the atmospheric temperature with the curve of prevalence of infantile diarrhœa, favours the idea of some soil-inhabiting micro-organism, but none of the various bacteria which have been suspected as possible causes of infantile diarrhœa have been shown to be specially connected with the soil.

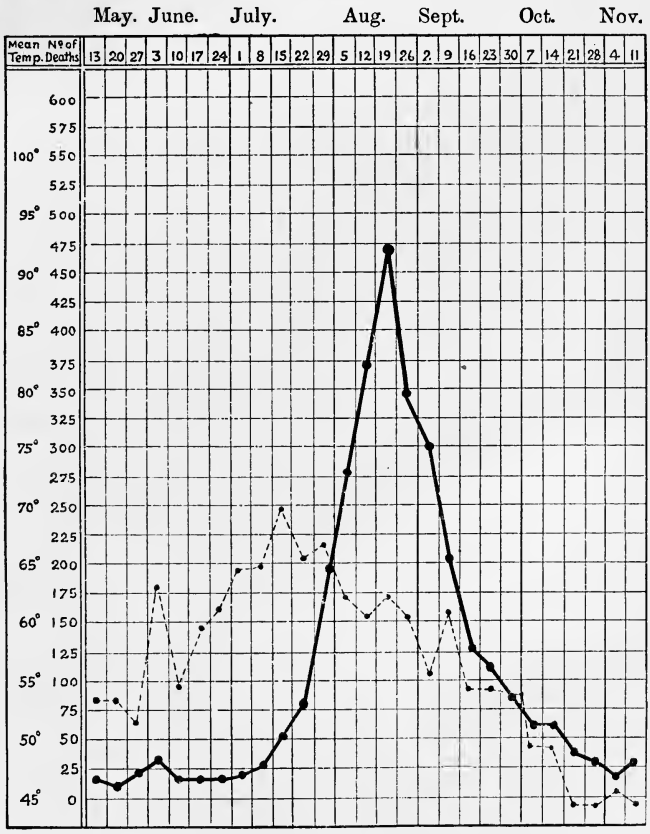

FIG. 10. Chart showing seasonal incidence of infantile diarrhœa. The continuous line shows the mortality from diarrhœa in children under two years of age; the dotted line shows the mean atmospheric temperature.

Hitherto it cannot be said that any micro-organism of the many which have been described in recent years as causal agents in infantile diarrhœa has been proved to bear a specific relation to the disease; it seems possible that infantile diarrhœa, even in its most epidemic form, may be due to many different micro-organisms. In some cases the bacillus enteritidis sporogenes

described by Dr. Klein may be the cause, in others the diarrhœa may be due to streptococci, but according to the recent observations of Dr. Flexner and others at the Rockefeller Institute in New York, the Bacillus of Dysentery (Shiga's Bacillus or allied forms) would seem to be present in so large a proportion of the cases of 'summer diarrhœa' as strongly to suggest a causal relation. This last micro-organism has been found occasionally in small numbers in the stools of healthy infants: it may be that bacteria which normally inhabit the intestinal tract may under certain conditions multiply excessively and assume pathogenic properties.

But whilst in the majority of cases infantile diarrhœa, especially as it occurs in the summer months, is due to some micro-organism, and is in this sense 'infective', there is no reason to suppose that it is always so. Indigestible food, certain drugs, perhaps exposure of the abdomen to chill, as is so apt to happen in the infant whose nether parts are too often left uncovered, and probably the irritation of dentition, may all be causes of diarrhœa. Arising from these conditions the diarrhœa is usually a mild and transient affair, but whatever the cause may be, if it persists so that the catarrhal condition of the intestine is repeatedly induced, the repetition of 'insults to the intestinal mucosa', as Dr. Flexner expressively calls them, renders the intestine specially liable to bacterial invasion; in this way what began as a simple gastro-intestinal catarrh from indigestible food may pass into a severe infective gastro-enteritis or ileocolitis.

Diarrhœa in infants as in older persons may be secondary to many diseases, to most of the specific fevers, to pneumonia and to empyema, or it may be a symptom of tuberculous ulceration of the bowel; but here I am not concerned with these secondary diarrhœas, and would only point out that sometimes most unexpectedly what has been regarded as an ordinary gastro-enteritis proves at autopsy to be due to tuberculous disease of the intestine.

Infantile diarrhœa falls perhaps most naturally into three groups: (1) Gastro-intestinal catarrh, (2) Gastro-enteritis and Ileocolitis, (3) Cholera Infantum.

The morbid anatomist may go further, and classify in accordance with the lesions found; he may distinguish a follicular enteritis from a gastro-enteritis with no enlargement of follicles; he may differentiate a simple ileocolitis from an ulcerative or a membranous colitis; but for the clinician to draw such distinctions is usually to assume more than he knows.

Even the classification adopted here assumes distinctions which are not always possible; who shall say where gastro-intestinal catarrh, or, as some would call it, intestinal dyspepsia, passes into gastro-enteritis? And certainly it is often quite impossible on any clinical grounds to determine whether a case is one of gastro-enteritis or of ileocolitis. The recent researches on the dysentery bacillus raise hopes that at some time a clinical classification in accordance with bacteriological differences may be practicable, but the time is not yet. Even the distinction of infective from non-infective diarrhœa is not altogether reliable in the present state of our knowledge. Until we know which micro-organisms cause diarrhœa, we can only surmise that most of the severer cases of diarrhœa in infancy are due to some infection; it is quite conceivable that toxic substances in the food—for instance, products of bacterial action on milk before it has been swallowed by the infant—may set up acute catarrhal change in the intestine without assistance from bacteria in the bowel. Recognizing these limitations, we may adopt the grouping suggested as being of some practical value in indicating lines of treatment.

Gastro-intestinal catarrh, the simple diarrhœa, or intestinal dyspepsia of some writers, is the mildest form of diarrhœa. It begins often with some evidence of colic, the infant cries fretfully and draws up his legs as if in pain, there is often some vomiting at this stage, and within a few hours there is diarrhœa. The bowels are open perhaps six or seven times a day, the stools are loose, at first yellow and then greenish and slimy, and often studded with pieces of white curd. The parts about the anus are apt to become reddened and excoriated. The infant remains fretful and sleeps badly; the temperature is often raised to 101°–102°, sometimes much higher, for a few hours, but soon falls to normal. If the attack lasts more than a few hours the limbs become flabby, and the weight is found to have fallen.

Such attacks are often self-terminated when the irritating substance, which is commonly some undigested food, has been evacuated by the diarrhœa; in others a dose of castor oil ejects the offending material and then the diarrhœa ceases.

If the cause is allowed to remain, for instance, by the continuance of unsuitable feeding, the catarrh becomes chronic and gradually the infant falls into a condition of marasmus; occasionally an attack, which is apparently of this mild type and due to some unsuitable food, passes into one of the severer forms, and becomes gastro-enteritis or ileocolitis; this danger is specially

to be remembered in the summer when no looseness of the bowels in an infant, however slight it may appear, is to be regarded lightly.

Gastro-enteritis and Ileocolitis ('Inflammatory Diarrhœa': 'Infective Diarrhœa'). There can be no doubt that in some cases the stomach and upper part of the intestine are much more affected than the lower part, whereas in others the stress of the inflammatory process falls on the lower end of the ileum and colon; moreover, it is possible in some cases to recognize clinically that the colon is the seat of some acute inflammation. In accordance with these facts we may speak of a gastro-enteritis in the one case and an ileocolitis in the other; only let it be recognized that in many cases clinically no such distinction is possible, and pathologically it is probable that an ileocolitis very rarely occurs apart from some enteritis or gastro-enteritis. Ileocolitis, then, may be taken to indicate those cases in which from certain characters of the stool and the local distension or tenderness of the colon it is probable that this is the part which is chiefly affected.

In most cases of acute gastro-enteritis the onset is more or less sudden: the infant begins to vomit, passes a few loose, perhaps green and slimy stools, and then the stools become watery, often of a dark brownish colour and very offensive, and the bowels are open perhaps ten or twelve times a day. Vomiting varies much in severity in these cases: sometimes the child vomits only two or three times a day, although the diarrhœa continues very frequent; in other cases the vomiting continues for several days, severe and frequent. The appearance of the infant is affected much more speedily by severe vomiting than by diarrhœa: the eyes become sunken, the face has a pinched appearance, and the fontanelle is depressed much sooner in these cases with severe vomiting than in those in which the fluid taken reaches the intestine, for although diarrhœa may hurry the intestinal contents along very rapidly there is opportunity for some absorption to take place. In any case, after even a few hours of diarrhœa the limbs lose their firmness, the fontanelle becomes depressed and the infant begins to look ill. The tongue at first is somewhat furred, but after a few days is often particularly clean and almost unnaturally red. The abdomen is often somewhat tumid with coils of small intestine visibly distended, but it is supple and free from tenderness. The temperature, even if it is raised for the first few days, soon becomes normal, and if the diarrhœa is prolonged a subnormal temperature is common.

In most cases the vomiting ceases after a few days, but the diarrhœa continues and after fluctuating for a week or two may slowly subside; too often, however, it becomes worse rather than better, and as a fatal ending approaches the vomiting reappears and the temperature rises rapidly to 104° or higher.

In other cases the diarrhœa continues several weeks but with less severity than at first: the bowels are open perhaps five or six times a day, and any attempt to advance in feeding beyond some very dilute and weak food causes an alarming increase of the diarrhœa; and so between diarrhœa and starvation the infant goes slowly down hill, becoming more and more wasted and feeble until exhaustion with some exacerbation of diarrhœa, and perhaps a sudden hyperpyrexia, ends the scene.

Ileocolitis differs from gastro-enteritis in several points: the temperature is usually higher and the pyrexia, which is irregular and of intermittent or remittent type, lasts longer; the abdomen is full and distension of the colon is sometimes obvious, and there may be tenderness of the abdomen, especially along the region of the colon; tenesmus and prolapse are apt to occur; but the most constant feature is the character of the stools, which in addition to loose brown or yellow fæcal matter, contain much mucus often mixed with streaks of blood.

Cholera Infantum is much rarer than either of the preceding, but it may be doubted whether it differs in kind. The symptoms differ in their extreme acuteness and intensity; the infant in twelve hours may pass from perfect health to a dying condition. The face becomes grey, with pinched nostrils, the eyes sunken with a dull vacant appearance, the fontanelle deeply depressed, the tongue is dry and brown, the extremities are cold and blue albeit the temperature may be 105° or 106° in the rectum, the skin is dry and loose over the sunken abdomen and the infant lies in quiet apathy, or flings its arms restlessly from side to side as it licks its dry lips in the misery of thirst; with these symptoms there is sometimes associated complete absence of sleep; a symptom of most sinister significance—indeed, I think, almost always presaging death.

Vomiting in these cases is almost always extremely severe; even teaspoonful doses of water are vomited; the stools at first yellow and loose quickly become completely liquid with a characteristic colourless 'rice-water' appearance, the resemblance of which to that seen in Asiatic cholera (with which, of course, the infantile disease has no connexion) has suggested the name of cholera infantum.

The temperature is usually subnormal, but in some cases, as already mentioned, it is much raised; the respiration is rapid and shallow, sometimes almost panting; the pulse becomes extremely rapid and barely to be felt at the wrist, and with increasing exhaustion the child dies within a day or two after the onset of the diarrhœa. But cholera infantum is not always fatal, the case which looks most desperate will sometimes recover, the stools begin to show some trace of colour, they become less watery, and slowly the diarrhœa ceases.

Such are the symptoms in the three groups of infantile diarrhœa: their differences are chiefly differences of severity; indeed, beyond the points which I have mentioned as indicating affection of the colon, I doubt whether there is any valid point of distinction other than that of severity.

The so-called cholera infantum has nothing whatever to distinguish it from a very severe case of gastro-enteritis but the colourless appearance of the stools, and at present we have no proof that this indicates more than an extreme degree of severity of the irritating process in the bowel, and, as I have already pointed out, it is impossible to draw any sharp line of distinction between gastro-intestinal catarrh and gastro-enteritis. I emphasize this point because I think there is a tendency nowadays to force infantile diarrhœa into various groupings with no sufficient clinical grounds, and with no certain knowledge yet of differences in pathogeny.

Complications. In all forms of infantile diarrhœa complications are common. If the diarrhœa is at all prolonged there is often *thrush* in the mouth, and the tradition which still lingers amongst the laity that this is an occurrence of fatal significance probably arises from the fact that thrush is specially liable to occur in any condition of extreme malnutrition, but there is no reason to suppose that the prognosis is made either better or worse by the presence or absence of thrush, unless indeed the stomatitis which accompanies thrush in some cases be severe enough to make the infant unwilling to take food and so increase the difficulty of maintaining the infant's strength. But it is not only in the prolonged cases of diarrhœa with marasmus that thrush occurs, it is seen often enough in transient gastric or gastro-intestinal disturbances in infancy, where the secretions of the mouth are in some way altered so that the normal alkalinity is reduced or gives way to the actual acidity which is often found with thrush and which seems to favour the growth of the oidium albicans. I have found thrush coating the œsophagus almost down to the cardiac orifice in infantile diarrhœa with marasmus; and cases have been recorded in which

thrush has been demonstrated on the skin about the anus, so that although thrush probably never really traverses the intestinal tract there is some excuse for the popular idea that the 'thrush has gone through the child', a statement which usually means nothing more than that the anus has become reddened and excoriated by irritating stools.

Respiratory complications are particularly common; in any severe case of diarrhœa if it lasts more than a few days bronchitis is apt to occur and passes very easily and insidiously into a broncho-pneumonia. Such a condition is sometimes found quite unexpectedly at autopsies on cases of infantile diarrhœa. The pulmonary collapse, which is a noticeable feature in many cases of diarrhœa as in other wasting diseases of infancy at autopsy, is seldom recognizable during life.

Otitis media is to be remembered as a complication of diarrhœa; any exhausting disease, be it acute or chronic, seems to bring with it a special liability to catarrh of the middle-ear in infancy, sometimes with external discharge, sometimes without, but in either case adding its own symptoms to those of the primary disease. A sudden rise of temperature may be due to catarrh in the middle-ear; I have often wondered whether the *head-retraction*, which is not very rare in cases of infantile diarrhœa where there is much exhaustion, may not be due, at least in some cases, to the same cause; for it is quite certain that middle-ear catarrh is responsible for marked head-retraction in some cases apart from diarrhœa; the proof is the disappearance of the head-retraction after puncture of the membrana tympani. I have several times found pus in one middle-ear or both after death in cases of infantile diarrhœa where there had been head-retraction during life, but this, I admit, rather suggests than proves a causal relation, for pus is very commonly found in the middle-ear in infants who have died of any disease which has lasted more than a few days whether there have been symptoms of ear disease during life or not. In seventy-nine consecutive autopsies on infants who died from any cause under two years of age, I found pus in one or both ears in thirty-two.

Screaming as with pain in an infant with diarrhœa should always suggest the possibility that the pain may be in the ear rather than in the abdomen.

Otitis media, when it occurs apart from diarrhœa, sometimes produces symptoms closely simulating meningitis; it seems quite possible, therefore, that the symptoms seen in some severe cases of diarrhœa and described as 'spurious hydrocephalus' (the resemblance is to tuberculous meningitis, which was formerly known as 'acute hydrocephalus', not to the chronic condition

to which the term hydrocephalus is now applied) may sometimes be due rather to some middle-ear inflammation than to any toxæmia, as is assumed.

The cerebral symptoms known as ' spurious hydrocephalus ' are illustrated by the following case. Geraldine W., aged nine months, had had diarrhœa and vomiting fourteen days; she was admitted to hospital about thirty-six hours before death; the infant lay on its back quite apathetic and apparently only semi-conscious; the fontanelle was depressed; the eyes were sunken, rolled somewhat upwards, with divergent strabismus; occasionally the infant gave a piercing scream, which was noted as ' very like meningitis '; the mother said that there had been some rolling of the head from side to side. Autopsy showed nothing abnormal in the brain or its vessels. There was pus in the left middle-ear. The bowel showed enlargement of solitary follicles and five small shallow ulcers in the middle part of the ileum; all other organs were normal.

Charles D., aged ten weeks; diarrhœa began fourteen days before death; on admission, nine days before death, the eyes were sunken, the fontanelle depressed, there was occasional divergent squint; the head was rigidly retracted, and there was some opisthotonos of the dorsal and lumbar spine; the limbs were rigid, especially the left arm; there was no optic neuritis. The head-retraction continued with remissions, and there was some rolling of the head from side to side; rigidity of the limbs, especially of the left arm, persisted. Autopsy showed nothing abnormal in the brain, meninges, or vessels; the colon showed extensive ulceration, with very acute colitis.

Cerebral symptoms in infantile diarrhœa are occasionally due to *thrombosis of sinuses*. In my experience this has been very rare; it was found only once in eighty-two autopsies on infantile diarrhœa at the Children's Hospital, Great Ormond Street; this was in an infant aged one year, who died after severe gastro-enteritis of $3\frac{1}{2}$ weeks' duration; during the illness some facial paralysis had been noticed; at autopsy thrombosis of the longitudinal sinus was found. Even when thrombosis has been found there have not always been cerebral symptoms during life; sometimes convulsions have occurred, but these are not very uncommon with infantile diarrhœa without thrombosis of sinuses. *Convulsions* are always to be regarded as a grave complication of diarrhœa, but they do not by any means necessarily indicate a fatal ending; they are less serious when they occur at the onset of the diarrhœa than when they occur in the stage of exhaustion.

Œdema, especially of the extremities, is not an uncommon complication of infantile diarrhœa; it occurs usually when the diarrhœa has lasted long enough for the infant to become much wasted, and is no doubt similar in its pathology to the œdema which is often seen with severe marasmus in infancy; what this pathology may be is uncertain, but it is probable that some altered state of the blood, and perhaps of the walls of the blood-vessels together with some feebleness of circulation is sufficient cause for the œdema. Certainly it is very rarely that œdema is due to any nephritis in infantile diarrhœa; it is common enough to find a trace of albumen in the urine in cases of infantile diarrhœa; in some cases this may be explained by the presence of uric acid granules in the pelvis of the kidney, for with the concentration of the urine during diarrhœa these granules are apt to be deposited, as I have often seen in the renal pelvis. They are associated sometimes with the so-called 'uric-acid infarcts', i.e. deposits of uric acid in the straight tubules of the kidney, which may be not merely a post mortem phenomenon but an actual cause of irritation during life.

Occasionally, it is true, the kidney is found to be pale and swollen with hæmorrhagic points under the capsule and in the cortex, and well-marked nephritic changes are seen under the microscope, but more often the occurrence of œdema or of slight albuminuria clinically is not explained by any naked-eye change in the kidneys, and microscopic examination shows only some granular appearance and feebleness in staining of the cells of the tubules which can hardly be taken as unequivocal evidence of nephritis.

Purpura is not very uncommon where diarrhœa has already produced much wasting; it is always to be regarded as of grave significance, but I have several times seen recovery occur in spite of the presence of purpura.

There is another complication which, although unrecognizable during life, may be of some practical importance, for it may make the infant unwilling to swallow, namely, an *acute inflammation of the œsophagus*. It is a rare occurrence, but has been observed several times at the Children's Hospital, Great Ormond Street; the œsophagus, chiefly near the cardiac orifice, shows deep congestion with hæmorrhagic points in the mucous membrane. It occurs apart from any obvious cause such as the passage of a stomach-tube or severe vomiting.

Treatment

In spite of all that has been written on the treatment of infantile diarrhœa, those who have largest experience will be most ready to admit that there are few diseases of infancy in which the choice of treatment is more perplexing. There is no lack of methods; the difficulty is to decide what mode of treatment is likely to suit the particular case; certain general principles we can lay down, and certain drugs and modes of feeding can be mentioned as generally useful, but in the individual case drug after drug, and food after food, has often to be tried before success is obtained. And here I venture to point out what has sometimes seemed to me to be a shortcoming in the treatment of severe infantile diarrhœa; the doctor sees the infant perhaps in the morning, gives his orders, and says he will come again next day. Now in many a case of acute 'summer diarrhœa' the infant passes so rapidly from bad to worse that if a food or drug which is doing no good is continued for several hours, the chance of saving the infant's life is lost; it may be necessary for the doctor to see the infant twice or even thrice in the day, to adapt his measures to the changing and pressing needs of the infant.

In the so-called gastro-intestinal catarrh or simple diarrhœa, if the infant comes under treatment within a day or two after the onset of the disorder, it is often well to give a mild purge to clear out the cause of the trouble, and for this purpose I think nothing is better than the old-fashioned remedy castor oil, in a dose of ½ to 1 drachm for an infant up to one year old, for it not only acts as a purge, but has a subsequent constipating effect, which is just what is needed in these cases. But often medical advice is not sought until the diarrhœa has already lasted several days, and Nature has evacuated the bowel so thoroughly that any further purging would do more harm than good. In these cases a combination of opium with *small* doses of castor oil is a very effectual remedy. And here let me point out the very marked difference in effect of castor oil according to the dose given. Castor oil in doses of 5 minims given, say, three times daily to an infant, has only a constipating effect, it has no aperient action whatever; it is indeed almost as constipating as opium. For infants under two months old, it is wise to use only 4 minims for this purpose, for at this tender age 5 minims has occasionally, though rarely, an aperient effect; at any age up to the end of infancy (two years old) the dose of 5 minims must not be exceeded if the castor oil is used to constipate; any larger dose may act

as an aperient; a 10-minim dose usually has this effect. A useful mixture is the following: ℞. Ol. Ricini ℥v, Spirit. Chloroformi ℥j, Mucilag. Acaciæ ℥xv, Aq. Anethi ad ℥j; to which for an infant two months old, Tinct. Camph. Co. ℥ij may be added, at three months Tinct. Opii ℥ $\frac{1}{6}$, at six months Tinct. Opii ℥ $\frac{1}{3}$, and at one year Tinct. Opii ℥ $\frac{2}{3}$.

The feeding will require some modification. If the infant is being hand-fed, it will usually be well to dilute the milk considerably, and the addition of lime-water or of sodium citrate to the milk may be useful, the former being used in the proportion of at least 1 ounce to every 3 ounces, the latter in the proportion of 1 grain to every ounce of milk up to 4 grains; both these drugs have a distinctly constipating effect in addition to their value in assisting the digestion of milk. If the infant is past the age of one year, a thin arrowroot gruel made with milk and water, equal parts, and given only lukewarm, makes a useful food.

In the severer forms of diarrhœa, gastro-enteritis and ileocolitis, and even more in the severest cases of all, the so-called cholera infantum, dietetic measures are of vital importance. First and foremost in the acute stage is the prohibition of all milk; it is poor economy to temporize with various dilutions of milk in the hope that so nutrition may be maintained; far less is lost by substituting at once some much weaker form of nutriment, which may relieve the bowel from irritating residue, and so favour the subsidence of the inflammation. But what is to be given instead of milk? My own bias as the result of experience is against all modifications of milk, even the most carefully prepared whey will sometimes perpetuate vomiting and diarrhœa in these cases if given during this early stage of the disorder, and peptonized milk, however much diluted, does only harm. The choice lies, I think, between albumen-water, barley-water (or, better, rice-water), weak veal or chicken broth, ordinary tea freshly infused and made very weak, a food much favoured for severe infantile diarrhœa in some parts of Germany as having perhaps some value. I shall have something to say about this later on.

Broth is by no means always successful; sometimes it is vomited, and sometimes it excites diarrhœa where albumen-water may be tolerated well; but except in the very severe cases it is worthy of trial, and an infant can be kept on broth alone for two or three days if necessary; if the diarrhœa is abating after twenty-four or thirty-six hours, half a teaspoonful of milk sugar may be added to every 3 ounces of broth, and then feeds of whey may alternate with feeds of broth.

Barley-water is to be used with caution; the starch which it contains may set up fermentation processes in the bowel which may aggravate the diarrhœa; sometimes, however, it seems to suit excellently. Barley-water in these cases must be made thin.

Rice-water is perhaps preferable to barley-water on account of its being less laxative; I have used it in these severe diarrhœa cases with good result. (For method of preparation, see p. 44.)

Albumen-water[1] is, I think, the most useful food in severe cases, for not only is it retained easily, but it seems to be less irritating to the bowels than either broth or cereal decoctions. It should be remembered, however, that the white of an egg is not necessarily sterile; the utmost care should be taken to procure fresh eggs for this purpose.

But there are cases in which even albumen-water is vomited; in these plain cold boiled water should be tried. The urgent need of the infant with severe diarrhœa and vomiting is for fluid; it matters little whether it contains this or that amount of nourishment, plain water will serve the immediate purpose at least as well as any other fluid.

The commonest fault in dealing with these cases is to use too large feeds. Many an infant who vomits all that he takes, so long as two- or three-ounce feeds are given every hour, will retain perhaps a drachm, or it may be 2 or 3 drachms, given every half hour; it is to be remembered that the retention of the fluid is not only a gain in itself by supplying the need of the infant, but it means that the stomach is not irritated as it was by the larger feed, and so there is more opportunity for subsidence of the inflammatory or catarrhal condition. There can, I think, be no doubt that, *mutatis mutandis*, the same is true of the intestine; where there is no vomiting but severe diarrhœa and the bowel ejects all food rapidly, it is wise to use small feeds often, say ounce feeds every hour or 2 ounces every 1½ hours rather than three- or four-ounce feeds every two or three hours; it seems as if the smaller amount coming into the intestine at one time excites peristalsis less than does a larger amount.

But whilst I would insist upon the importance of small feeds during the very acute stage of vomiting and diarrhœa, I would also deprecate the prolonged use of very frequent feeding. So long as only a teaspoonful or a dessertspoonful can be retained it is necessary to feed every half hour or every three-quarters of

[1] To prepare albumen-water, take the white of one fresh egg, divide it in several directions with a pair of clean scissors, then mix it with 6 ounces of cold water in a bottle, and shake vigorously; strain through muslin.

an hour, but after four or five hours an attempt should be made to increase the feed to half an ounce every hour, and then to 6 drachms every 1¼ hours, and so on.

In cases where even teaspoonful feeds are vomited, and sometimes where the stomach will just, but only just, tolerate these small amounts of water or albumen-water, the best result may be obtained by stopping all attempts at feeding by mouth for some hours, perhaps four or five hours, and in the meantime a large rectal injection of saline solution (a drachm of sodium chloride to the pint), 5 to 6 ounces to an infant of three months, may be given slowly with the idea of procuring absorption of some of the fluid if it is retained long enough, and of washing out the bowel if the fluid is returned.

If the infant is very collapsed, the eyes sunken, and the fontanelle depressed, and the vomiting and diarrhœa extremely severe, so that it is evident that there is urgent and immediate need for some absorption of fluid, the best procedure and the only one which will ensure the necessary absorption of fluid, is subcutaneous infusion. This is really a very simple matter if a funnel, a piece of rubber tubing, and an exploring needle of medium size are available.

The barrel of an ordinary glass ear syringe without the piston makes an excellent funnel, being narrow enough to allow the upper open end to be easily stoppered with aseptic cotton-wool, a precaution which should not be neglected, for the fluid may take nearly an hour to run in, and during this time serious contamination may occur if the surface of the fluid is left exposed. The needle, barrel, and rubber tubing must be carefully sterilized by boiling water, the skin over the lower ribs in the axilla is thoroughly cleansed by rubbing first with ether, then soap and water, and then with some antiseptic solution, e.g. 1 in 1,000 hydrarg. perchlor. solution; the barrel and tube are then filled with the saline solution, which must be prepared with boiling water, and cooled down to 105° F. (if no suitable thermometer is available the temperature can be guessed with sufficient accuracy), and the lower end of the tube must then be clamped or pinched to prevent escape of the fluid whilst the needle is inserted. The skin over the lower part of the axilla is then pinched up between the finger and thumb of the left hand, while with the right hand the needle is pushed through the skin into the subcutaneous tissue, in which it should be pushed along for about three-quarters of an inch.

The infusion can be done more rapidly and a larger quantity can be introduced if the tube from the funnel be attached to a Y-shaped connecting piece, so that the fluid can pass by two separate tubes and needles into two different parts, e.g. into the two axillae simultaneously.

The funnel must, of course, be refilled frequently so that it does not empty itself completely, and to facilitate the flow it is often necessary to push the needle slightly onwards into a fresh area of subcutaneous tissue from time to time as the tension becomes great, or to move the needle slightly so as to disengage the point from any fat or fibrous tissue which may be blocking the lumen ; in this way the fluid runs in slowly, and in about three-quarters of an hour as much as 6–8 ounces may be introduced into the subcutaneous tissue. The large bulging swelling at the site of infusion disappears completely in an hour or two, and it is remarkable how little pain beyond the first prick of the needle seems to be caused by infusion. Various fluids have been used for infusion. A five per cent. solution of glucose which can be prepared readily with special sealed tubes containing sufficient glucose to make one pint (Martindale) has the advantage that it supplies nourishment as well as fluid. Sea water has been in fashion recently and can be obtained in sterile sealed tubes, but so far as I have seen has no advantage over ordinary 'normal saline '. The most readily available is this ordinary saline made by adding one drachm of sodium chloride to the pint of water; of course it is necessary that the solution should be thoroughly sterilized by boiling.

The effect of subcutaneous infusion is generally very striking, the eyes become less sunken, the fontanelle less depressed, the infant is less apathetic, and looks less distressed ; often he falls into a calm sleep, which considerably assists recovery from the previous exhaustion.

But before infusion is done, if the exhaustion and collapse is extreme, it is advisable to use some more rapid stimulant ; the process of infusion must necessarily occupy at least half an hour, and this may be half an hour too late in the case of a collapsed infant ; moreover, if infusion is done when an infant is already moribund, there may not be sufficient circulatory power to ensure its rapid absorption. In the case of extreme exhaustion, therefore, strychnine should be injected subcutaneously first ; very young infants are particularly susceptible to strychnine, and I have seen convulsive symptoms follow the injection of one minim of the B.P. Liquor Strychninæ ; to an infant under three months half a minim of this solution should be given, to an older infant one minim.

Here I would dissuade from the subcutaneous injection of ether or brandy unless the infant is either *in articulo mortis* or unconscious ; the effect of either of these is probably more rapid than that of strychnine, indeed, it is almost instantaneous, but it is also more evanescent and therefore less valuable, except in

the most extremely urgent cases ; but apart from their evanescent effect, both these drugs when injected hypodermically cause considerable pain, whereas strychnine, apart from the mere prick of the skin, causes none.

An excellent stimulant in cases of collapse from diarrhœa or vomiting is the hot mustard bath (a tablespoonful of mustard to a gallon of water) at a temperature of 100°–105°. The infant should be undressed and supported on the nurse's hands in the bath, so that the whole body with the exception of the head is immersed ; often I have seen in the administering of a mustard bath the greater part of the body uncovered by the water, so that the infant was being chilled by the exposure rather than stimulated by warmth. After remaining about four minutes in the bath the infant should be quickly dried with a warm towel, wrapped in a hot blanket, and hot water-bottles should be placed outside the blanket. If infusion is to be done immediately after the hot bath, great care must be taken to keep the infant as far as possible warmly covered, for the dilatation of cutaneous vessels makes even the slight exposure which may be necessary during infusion a possible depressant.

The value of alcohol in the early stage of severe diarrhœa when there is extreme exhaustion and collapse, is, I think, quite undeniable ; 10 or 20 drops of brandy in a teaspoonful of cold water may be retained when all else is vomited, and this amount of brandy acts as a powerful stimulant to an infant, the small dose for an infant under six months, the large for an infant from six to twelve months old. This dose can be repeated three or four times at intervals of two hours or even of an hour if necessary ; but if it is to be repeated after this smaller doses should be used, 5 minims for an infant under three months, 10 minims for an older infant. It is not only as a stimulant that these small doses of brandy are valuable, but also in the prevention of vomiting; formerly I was sceptical of the possibility of, say, 5 drops of brandy in a three-ounce feed having any appreciable effect ; I have satisfied myself that this small dose has a very valuable effect both in preventing vomiting, and also in assisting the digestion of milk in some cases of curd-indigestion ; in this way small doses of brandy are sometimes useful both in the acute stage of diarrhœa and vomiting, and also a little later when an attempt is being made to return to milk-feeding. Having said this much in favour of the use of alcohol, I must also express my conviction that great harm is done by too large doses of alcohol in this disease ; I have seen babies who were being dosed with brandy every hour and even oftener in amounts far exceeding

those I have mentioned, with the result that vomiting was actually aggravated, and sometimes it has seemed to me that the drowsiness or the supposed delirium of the infant was largely if not entirely due to the brandy. If brandy is ordered for an infant with any disease, the exact dose and frequency should be specified by the doctor, and I think it is very rarely that the doses I have mentioned above should be exceeded, and last, but not least, the doctor should specify how long the alcohol is to be continued; I have known brandy and other alcoholic stimulants continued for many weeks or months simply because the doctor had omitted to mention that it should be used only for a few days, and the fewer the better.

With regard to choice of alcoholic stimulants I doubt whether whisky has any advantage over brandy; some, however, use it, and circumstances may sometimes make it convenient to do so; similar doses should be used.

Sherry makes a convenient stimulant in the form of sherry whey[1]; this, however, is usually not suitable in the earliest stage if the vomiting and diarrhœa are very severe; but later, when both have become less acute, sherry whey often makes a useful step towards the return to milk-feeding, it seems to have a carminative effect, so that not only is it retained itself, but other food given at alternate feeds is also retained. If necessary, sherry whey can be used alone for a day or two where feeds of not more than 2 ounces are required, but as soon as possible it should be used only as an alternate or occasional feed, the other feeds being either broth, or plain rennet whey, or later, weak peptonized milk with some lime-water to counteract its laxative effect.

Drugs must next be considered, and I place opium first, for there is, I think, no drug which is so generally valuable in this disorder. This statement, however, needs qualification, for there are cases in which the administration of opium seems to do harm: when the stools are extremely offensive and the diarrhœa is checked by opium, nervous symptoms have sometimes supervened; the infant falls into a semi-comatose condition, and symptoms occur such as have been described as those of spurious hydrocephalus (vide p. 224); the sequence suggests that the longer retention of the fæcal material in the bowel consequent upon the administration of opium has allowed more absorption of toxic material to take place. This, however, is rare in my experience, and it is, I think, true that there are very few cases of infantile diarrhœa in which the use of opium is not beneficial.

[1] For method of making sherry whey, vide p. 57.

As to the form of opium, it is often more convenient to use the liquid preparations than the solid, so that it may be given in a mixture with other drugs; but in the solid form as Pulv. Ipecac. Co. (Dover's Powder), it is, I think, sometimes more effectual. For infants a few weeks old, the Tinct. Camph. Co. in doses of ℥j during the first month, and ℥ij during the second month, makes a convenient mode of administration; for older infants at three months, Tinct. Opii ℥$\frac{1}{6}$, at six months Tinct. Opii ℥$\frac{1}{3}$, at one year Tinct. Opii ℥$\frac{2}{3}$ can be given every three or four hours. Dover's Powder can be given in doses of $\frac{1}{8}$ grain at three months, $\frac{1}{4}$ grain at six months, and $\frac{1}{2}$ grain at one year, every six hours.

Where the vomiting is severe and the infant is in a restless semi-delirious condition as sometimes happens, the hypodermic injection of morphia in a very minute dose, $\frac{1}{100}$ grain for an infant three months old, has a valuable effect in quieting the child and husbanding his strength, apart from its good effect both upon the gastric and intestinal irritability.

Of other drugs there are two which are of paramount value in infantile diarrhœa, namely, bismuth and minute doses of castor oil. Bismuth is particularly valuable where there is much vomiting associated with the diarrhœa, but to obtain good results from bismuth it must be given in much larger doses than are often used. An infant a few weeks old should have gr. v every three or four hours, and to an infant of nine months or more 10 grains should be given at each dose. A useful prescription for ordinary use is Bismuth Carb. gr. v, Sod. Bicarb. gr. ijss, Spirit. Chloroformi ℥j, Pulv. Trag. Co. gr. j, Aq. Anethi ad ℥j, to which Tinct. Opii can be added according to the age. I do not think that anything is gained in effectiveness by administering the bismuth in powder form. I sometimes use a formula such as this: Bismuth Carb. gr. iv, Sod. Bicarb. gr. j, Pulv. Cretæ Aromat. gr. j, or Bismuth Carb. gr. v, Pulv. Ipecac. Co. gr. $\frac{1}{8}$, for an infant of three months, but it is difficult to give a sufficient dose in this way without making the powder inconveniently bulky, and there are many infants who will spit out powders, whereas there are few who will not swallow a fluid medicine without hesitation when suffering with the thirst which accompanies diarrhœa and vomiting.

Castor oil, as I have already said, when used in doses not exceeding 5 minims has no aperient action whatever, and has a powerful effect in checking diarrhœa. The mixture in use at the Hospital for Sick Children, Great Ormond Street, is ℞.

Ol. Ricini ɑ)v, Mucilag. Acaciæ ɑ)xv, Aq. Menth. Pip. ad ℥j. This should not be given more often than every six hours, otherwise the purgative instead of the constipating effect may be obtained. For cases in which the lower part of the bowel seems chiefly to be affected, as may be shown by the presence of much mucus with or without blood, and the slightness or absence of vomiting, these small doses of castor oil are particularly valuable; the addition of Tinct. Opii, according to the age, makes it still more effectual. If there is much distension of the bowel, or the stools are particularly offensive or frothy as if fermenting, the addition of Creosot. ɑ)$\frac{1}{4}$ in each dose is often helpful for an infant over six months of age, or 1–3 minims of Liq. Hydrarg. Perchlor., or a minim of Glycerin. Acidi Carbolici may be added.

An excellent mode of treatment is to give the castor oil mixture as mentioned three times a day without addition of opium, and to give also three or four times a day a powder of Hydr. cum Cret. gr. $\frac{1}{4}$, Pulv. Ipecac. Co. gr. $\frac{1}{4}$–$\frac{1}{2}$, according to the age.

Of the value of purely antiseptic treatment in infantile diarrhœa I cannot speak highly; I have seen cases in which recovery occurred where only small doses of grey powder (gr. $\frac{1}{6}$–$\frac{1}{4}$) or calomel gr. $\frac{1}{12}$ had been given three or four times a day. I have seen the stools made less offensive by the administration of Salol (gr. ii–iij) every four hours; Glycerine of Carbolic acid (ɑ)i–ij) B. Naphthol gr. i–iij, and Resorcin gr. ii–iv (which has the advantage of being soluble in water and not unpleasant in taste); all these are occasionally used, but generally speaking, I am sure that such methods are much less effectual in checking diarrhœa than those I have already mentioned.

In the acute stage of diarrhœa most of the astringents are of no value, but when the diarrhœa has become chronic these are often useful. Tincture of Catechu ɑ)v with Tinc. Opii $\frac{1}{6}$–1 minim, according to the age, in Mist. Cretæ ℥j may then be successful. I have found Tincture of Coto very effectual at this stage. It may be given either with bismuth or with five-minim doses of castor oil in a mucilage emulsion; the dose of the tincture of coto is 5 minims for a child of one year.

Silver nitrate also in doses of $\frac{1}{12}$–$\frac{1}{6}$ grain dissolved in distilled water is sometimes very useful, especially where the presence of much slime and streaks of blood, and perhaps tenesmus and prolapse, make it probable that the colon is chiefly affected. Tannalbin, which has recently been introduced, is sometimes very useful at this stage in doses of 5–10 grains every four or six hours in a teaspoonful of milk and water.

Tannigen in my hands has proved quite ineffective, but good results have been reported from its use; the dose is 2–3 grains every six hours for an infant under six months.

At this stage also a combination of sodium salicylate with bismuth is sometimes useful; Sod. Salicylate gr. i–ij can be added to the bismuth mixture mentioned above, or the combination bismuth salicylate can be given in doses of gr. ij to gr. iij. For infants under a year old it is safest to avoid salicylate altogether, and if used for older children it should always be combined with double as much bicarbonate of soda to avoid the toxic effects which I have known to prove very nearly fatal where sodium salicylate was being given to an infant in a dose of about 3 grains every two hours.

Sometimes a mixture of Acid Sulph. Dil. ♏ijss, Tinct. Opii ♏$\frac{1}{6}$–j, Syrup ♏x, Aq. Anethi ad ℥j ter die, seems more effectual than any other astringent.

Irrigation of the colon is of great value under certain conditions; where the stools are unusually offensive, and there is a tumid abdomen, or where from the frequent passage of much mucus and streaks of blood, it is clear that the colon is greatly affected. I think that irrigation per rectum with plain warm water or a weak solution of boracic acid, or with salt solution, i.e. Sodium Chloride ℥j to the pint, is valuable. The irrigation should be done once or twice in the twenty-four hours; if the infant's temperature is very high, the irrigation may be done with fluid at a temperature of 70°, some even recommend 60°–65° F., but under the more usual condition of feebleness and low or subnormal temperature, the fluid should be used at a temperature of 100°–105° F. About 6–14 ounces, according to the size of the infant, should be allowed to run in slowly through a funnel and soft rubber catheter.

Rectal injections of 4–5 ounces of tannic acid (gr. j to the ℥j) and of protargol (gr. $\frac{1}{2}$ to the ℥j) have also been recommended.

Where there are frequent small stools, or tenesmus occurs, showing irritability of the rectum, great benefit sometimes results from washing out the bowel with normal saline solution, and after this has been returned, injecting $\frac{1}{2}$–1 oz. of a very thin mucilage of starch.

CHAPTER XVII

ON SO-CALLED CŒLIAC DISEASE

THERE is, amongst the abdominal diseases of early childhood one which has hitherto received but little recognition, the affection known as 'cœliac disease'. Although by no means an everyday occurrence, its importance is out of proportion to its frequency, for it is very apt to be mistaken for other and more common diseases, especially tuberculous peritonitis. Its treatment also is worthy of study, for although it is too often an intractable condition, there are certain definite lines of treatment upon which the child's chance of recovery very largely depends.

This affection was described by the late Dr. Gee [1] under the name of 'cœliac disease': a vague term, inasmuch as it indicates nothing more than disease of the bowel. More descriptive is the name 'recurrent diarrhœa', but this exalts too much a symptom which is by no means invariable; nor, again, is 'chronic colitis' altogether satisfactory, for although it describes the pathological condition which has been found in some fatal cases, in others no such inflammatory lesion has been demonstrated; moreover, there are forms of 'chronic colitis' which certainly do not present the clinical features of the disease under consideration. The late Dr. Cheadle, attaching much importance to the pallor of the stools, included cases of this disease in the category of 'acholia'; but even in the most typical cases the stools are not always white, and it is evident that bile is not necessarily deficient. On the whole, therefore, it seems advisable at present to retain Dr. Gee's term 'cœliac disease'.

Put briefly, the story of these cases is usually as follows: A child of two years or thereabouts is wasting, the abdomen is enlarged, and though there is no constant diarrhœa, the bowels are loose. The stools are large, pale, and offensive, often shreddy or full of mucus. The child, though not emaciated, has grown thinner and flabby, and the association of wasting with enlargement of the abdomen has raised in the doctor's mind the idea of tuberculous peritonitis. As will be seen, however, from a more

[1] *St. Bartholomew's Hosp. Rept.*, 1888, vol. xxiv, p. 17.

detailed consideration of the disease, this has special characteristics of its own which are usually sufficient to mark it off as something 'sui generis'.

My description will be based upon twenty-four cases which have come under my own observation.

Etiology. Cœliac disease would appear to be much commoner in girls than in boys; of my 24 cases, 18 were girls, 6 were boys. It must be mentioned, however, that in a recently published series of cases,[1] which included 2 boys who figure in my own statistics, there were 7 boys and 2 girls; so that, if the two series be combined, the numbers are 20 girls and 11 boys.

There is some difficulty in determining the age at onset, for although the affection may have followed directly upon an acute attack of diarrhœa, sometimes it seems to have begun altogether insidiously, so that no exact date can be assigned. The commonest time of onset would seem to be the second year. The earliest age in my series was 8 months, the latest 3 years and 2 months.

Sometimes, but by no means always, an acute diarrhœal illness preceded the onset of cœliac disease. One might have expected, in view of the lesions found in some cases, that this preceding illness would have been an ileocolitis or colitis, but the history in some at least is rather that of a simple gastro-enteritis, which passed off leaving the child apparently well; the stools, however, remained somewhat irregular, and gradually assumed the characteristics of cœliac disease.

In three of my cases infantile scurvy preceded or accompanied the onset of the cœliac disease. Larger figures would be needed to prove that this was more than a coincidence, but it has seemed to me that children fed upon scurvy-producing foods are particularly liable to bacterial infection in connexion with the intestinal tract; thus I have noticed that pyelitis, presumably due to coli infection from the intestine, is apt to occur in children who have been fed for several months upon such foods, and I have elsewhere pointed out that pyelitis is sometimes a complication of scurvy. Moreover, there is a special tendency to diarrhœa in infantile scurvy, which may also indicate a diminished resistance to bacterial infection. One might add that the large intestine is often directly involved in scurvy, as is shown by the passage of bright red blood in the stools. It would seem, therefore, that as a predisposing cause there may be a real

[1] Poynton, Armstrong, and Nabarro. *Proc. Roy. Soc. Med.*, 1913, vol. vii, p. 10.

connexion between the preceding scurvy and the occurrence of cœliac disease, if we may assume that the latter is of bacterial origin.

At present we have no certain evidence of any specific micro-organism in this disease, but it is tempting to believe that a grouping of symptoms so constant in character and consistent in course is due to one particular organism. Dr. Nabarro has examined the stools bacteriologically in four cases, and in three of these he found the dysentery bacillus (Flexner), in the remaining one only Bacillus coli, streptococci, and pneumo-bacilli. It must be left to further and more extended observations, to determine whether the dysentery bacillus is the specific cause, or whether in different cases different organisms may produce the symptoms.

In connexion with the bacteriology of this disease, I would recall Dr. Gee's observation that 'sometimes from India Englishmen return sick with the cœliac affection', apparently referring to the condition now known as 'sprue'. In one of the cases under my own observation the mother had acquired 'sprue' whilst in India, and was still bad with the disease when her child was brought to me, at the age of $5\frac{3}{4}$ years, with very typical and severe cœliac disease, which had begun in India when the child was about nine months old. In another case possibly of this nature a boy aged 2 years had suffered with looseness of the bowels seven months, the abdomen was large, but the stools, though pale at times, had more colour than is usual in cœliac disease. His mother had 'sprue' when pregnant with this child. There are many points of similarity between the 'sprue' of adults and the cœliac disease of children. Both are characterized by wasting with looseness of the bowels, and pale, slimy, or shreddy stools. Both show the same prolonged course extending over years, and the same resistance to treatment. In 'sprue', ulceration of the mouth has been described as a frequent symptom. I cannot say that it is frequent in cœliac disease, but I have known it to occur.

I would not assert that the two affections are identical, but the clinical resemblances are worth bearing in mind, as bacteriological research on the one may possibly throw light on the other. At present neither can claim a specific organism, though in both the dysentery bacillus is thought to play a part.

The uncertainty as to the etiology of cœliac disease is increased by the scantiness of observation on its morbid anatomy. Thickening of the mucosa of both large and small intestine has been recorded with enlargement of solitary follicles and Peyer's

patches, but no ulceration. The mesenteric glands have been found swollen and the liver fatty.[1]

Symptoms. The first and most characteristic symptom of cœliac disease is chronic looseness of the bowels. There is no watery diarrhœa, nor indeed any constant frequency of action, for the bowels may not be opened more than once or twice daily, but there is a tendency to periodic increases of frequency, when the bowels will be opened perhaps four or five times a day for two or three days ; but even then the stool is only rather looser or contains more mucous ; it rarely becomes watery.

Perhaps the most characteristic feature of the stool is its colour, which is pale, greyish, like oatmeal, or even actually white ; in some cases, however, it is pale yellow, or even dark brown, and the colour varies from day to day. The stool is almost always unformed, and of porridgy consistence ; there is considerable excess of mucus, and therewith may be seen numerous grey shreds or flakes of what looks like thin membrane, but proves, on microscopic examination, to be mucoid material containing leucocytes and oval or elongated cells. Occasionally the stools contain streaks of blood, but this is exceptional.

With this unhealthy condition of the stools, there is more or less enlargement of the abdomen, usually not of any extreme degree, but sufficient to attract the mother's attention. The abdomen is seldom tense, and there is usually no peristalsis of particular coils of intestine to be seen. On palpation there is no localized resistance, no sense of infiltration or doughiness, such as characterizes tuberculous peritonitis, nor is there any tenderness. Occasionally slight colicky pain is present, a symptom common to most intestinal disorders of childhood where there is much mucus in the stools.

Vomiting is not a common symptom, but it occurred in some cases, especially in the early stage of the disease.

Thirst is quite a noticeable symptom, and the appetite is usually large, or perhaps, rather, one should say that the child is constantly hungry, as well it may be, for any attempt to satisfy its appetite results in frequent and bad stools and a corresponding rapid loss of weight, so that the child is necessarily kept upon a meagre and unsatisfying dietary.

Perhaps no feature is more striking in cœliac disease than the rapid fluctuations in weight ; a very slight error in a most carefully regulated diet will result in a loss of as much as ½lb. in forty-eight hours, whilst an improvement in the colour and con-

[1] *Proc. Roy. Soc. Med.*, loc. cit.

sistency of the stool for a few days may be associated with a gain of a pound or more, which, in turn, is as rapidly lost; and so, with disappointing fluctuations, the net result may be little or no gain after a year or more of constant rigid care and dieting.

The wasting which results from this disease is, at first at any rate, rather a passive than an active failure. The child grows older, but does not grow heavier ; the limbs are flabby and thin, although the face, as Dr. Gee pointed out, may still look round and full ; but if the disease progresses unchecked there is apt to be more active wasting, for with the erratic fluctuation of weight, the losses on the whole predominate over the gains.

A prominent feature of cœliac disease is weakness ; it is so marked that walking and even standing may be delayed long past the usual age. In some cases standing is still impossible at $2\frac{1}{2}$ or 3 years old ; one child was only beginning to walk at $6\frac{1}{4}$ years.

The child is usually peevish and fretful, and as it grows older, reaching the age of five or six years, shows a quaint precocity which is rather apparent than real, for whilst the physical growth is remarkably hindered, so that the child of six or seven years looks barely three years old, its mental development is in some respects in advance of its years, owing to the constant association with adults which results from inability to share in normal nursery life.

The remarkable hindrance of growth is one of the most serious results of this disease ; a girl aged $6\frac{1}{4}$ years measured $36\frac{1}{4}$ inches, the height of a child aged $3\frac{1}{2}$ years ; another at six years measured $37\frac{1}{8}$ inches.

The weight similarly is far below the normal ; a girl who at 6 years measured $39\frac{1}{2}$ inches in height weighed 30 lb. 13 oz., the weight of a child of $2\frac{3}{4}$ years ; another at $5\frac{3}{4}$ years weighed 21 lb., which is barely the weight of an infant aged 12 months.

This delay of development has been described as ' infantilism ', a term which, I think, has been considerably misused. We hear of ' intestinal infantilism ', ' renal infantilism ', &c., as if the ' infantilism ' constituted a disease in itself, whereas arrest of development, greater or less in degree, is merely a symptom produced by many different causes and common to many different diseases. If the term ' infantilism ' is to be retained at all, it should surely be used only for conditions in which the characteristics of adolescence, the sexual changes of puberty, fail to appear, but this is not so in some of the diseases where ' infantilism ' is said to occur, and I know of no evidence that it is so in ' cœliac disease '.

In spite of the interference with weight and growth, teething is usually not delayed ; a curious fact when one considers how greatly dentition is delayed by rachitic disturbance of nutrition.

The temperature in cœliac disease is usually normal.

The liver and spleen show no enlargement. In long-standing cases there is more or less anæmia, and this may reach a severe degree.

I have occasionally seen itching papules (' lichen urticatus ') on the limbs, an association which one might expect to be more frequent than it is when one considers how commonly lichen urticatus is seen with intestinal disturbances in childhood.

Prognosis. When once the diagnosis of cœliac disease is certain, it is well to explain to the parents that there is no question of cure in a few weeks or even months, and that they may consider it a happy result if, after a year or two of tedious dieting and treatment with varying success and failure, the child slowly regains its health.

There is undoubtedly risk to life. It is difficult to give actual figures, for, owing to the chronic character of the disease, these cases are apt to pass out of observation, or rather, as the parents become discontented with the slowness of progress, they drift from one doctor to another ; it seems probable, however, that few survive beyond childhood : if increasing debility and exhaustion from the disease do not prove fatal, sooner or later the child is liable to fall a victim to some complication or intercurrent disease. I have known several cases die thus, rather from the indirect than the direct effects of the disease.

On the other hand, I would particularly insist—and in so tedious a disease one can hardly insist too strongly—upon the possibility of a complete recovery. I have seen cases where after four or five years of rigorous dieting, and long arrest of growth, the child has at last struggled back to health and normal conditions of nutrition.

Complications. I have already mentioned anæmia amongst the symptoms of the disease. In some cases, it becomes very profound, and then, as in anæmia from other causes, purpura may appear. But apart from anæmia there seems to be a liability in cœliac disease to purpura, usually of mild degree, and affecting only the skin, not the mucus membranes. Dropsy also is not uncommon, and varies in degree from slight puffiness of the hands and feet to a general dropsy associated even with ascites. I would particularly draw attention to this occurrence of fluid in the abdomen, which may be associated with only very

slight œdema of the extremities. It is apt to be taken as evidence of tuberculous peritonitis. The occurrence of dropsy in these cases is not easy to explain ; it is not necessarily associated with any severe degree of anæmia. One might compare it with the curious cases of dropsy without albuminuria, in which a prominent association is the offensive unhealthy character of the stools.

In 4 out of my 24 cases, one aged 7 years, and the others aged 25 months, 15 months, and 18 months respectively, tetany occurred associated with facial and other nerve irritability. In the series [1] of nine cases mentioned above, one case also is noted as having had tetany at the age of eighteen months, but in this child there was also rickets, which was absent in two out of the four in my own series. This association is interesting in relation to the occurrence of tetany with chronic dilatation of the colon, and to the fact that occasionally apart from any such disease, irrigation of the bowel, where the stools are unhealthy, is followed, as I have seen, by tetany. It seems a reasonable inference that absorption of some toxic material from the colon is capable of producing tetany, and that this is the explanation of its occurrence in cœliac disease.

As in most chronic exhausting diseases, bronchitis and bronchopneumonia may supervene and prove fatal.

A complication which is specially to be remembered where diet is so restricted, as it necessarily is in this affection, is scurvy ; two out of my twenty-four cases developed scurvy, one at about six years of age, and the other at 4½ years. In both, the gradual onset of pains in the limbs was at first very puzzling, and one, seen by a surgeon who had not followed the dieting of the child, was mistaken for a condition needing surgical treatment.

In one of my cases a very remarkable complication occurred, namely, so-called late rickets. After the child had been dieted four years for cœliac disease, and had gradually improved, she began, at the age of eight years, to show rapid bending of the bones of the legs, so that they became severely distorted. The cause of this very rare condition of softening of the bones, which probably bears no relation to true rickets, but should rather be classified as osteomalacia, has never been determined ; its occurrence in this case suggests either a dietetic origin, or nutritional disturbance by toxic absorption from the bowel.

Treatment. *Diet.* Dieting is by far the most essential part of treatment in this disease, and it is necessary to lay down very exact rules as to what may and what may not be given. Unfor-

[1] Poynton, Armstrong, and Nabarro, loc. cit.

tunately, the menu which is permissible is extremely meagre, and experience shows that the slightest departure from it is likely to be attended with ill results. First and foremost in importance is the prohibition of all fresh cow's milk. I lay great stress upon this point, for one has often found that the mother has been specially advised to 'give the child nothing but milk'. No surer method could be adopted for perpetuating the trouble, and the omission of all fresh milk is generally sufficient alone to cause improvement in the character of the stools, and therewith more or less gain in general well-being.

Why exactly fresh cow's milk should be so harmful in these cases is not clear, but possibly it may be explained by the success which sometimes follows the use of the fat-free diet to which I shall refer later.

Whilst, however, fresh cow's milk is to be entirely forbidden, the same ruling does not apply to dried milk. There are various preparations of dried milk on the market, and some of these are made to contain a very low proportion of fat; these are invaluable as a substitute for fresh milk in coeliac disease; indeed, many a case which has been unable to tolerate the smallest quantity of fresh milk without increased looseness of bowels and rapid loss of weight, has done well on dried milk as the main article of diet for several years. Naturally, where very little else can be allowed, the child is apt to grow weary of the dried milk: when this is so, the addition of a very small quantity of tea, or of cocoa, may get over the difficulty for a time. I have found a preparation of cocoa mixed with acorn ('acorn cocoa') suit well in some of these cases.

The dried milk should be used not only for drinking, but also in the preparation of any foods that are allowed, such as custard or milk pudding, or milk jelly.

If dried milk does not suit, or is refused, by far the best alternative is asses' milk, on which I have known great improvement to take place. It has also the important advantage that it is not liable to produce scurvy as dried milk is. This, however, is a resource open only to the wealthy, especially if, as is usual in this disease, it has to be continued for a long time.

For people of ordinary means, the most practicable alternative, if dried milk fails or is refused, is a condensed milk. I have found the 'humanoid milk' prepared by the Aylesbury Dairy Company useful in these cases, but usually a condensed milk is less successful than the dried preparations. It might have been thought that whey, being deprived of nearly all fat, would

suit these cases well, but although it is sometimes tolerated, it is less satisfactory usually than the substitutes already mentioned.

Bread is usually to be avoided altogether, and a substitute must be found in some kind of biscuit or rusk. The biscuits which are made of dried milk with a very small addition of starch, such as 'Nurso Biscuits' or 'Malac Biscuits', have proved particularly useful. I have also given 'Mellin's Biscuits' in several cases without harm.

Broths and jellies are allowable; red gravy in small quantity from underdone meat seems to suit these cases better than raw meat juice; pounded chicken and boiled brains are also permissible for the older children. Farinaceous pudding must be used with caution, if at all; Revalenta is more suitable, but its taste is disliked by some children.

In spite of its high fat content, yolk of egg is sometimes tolerated well, and then makes a very important addition to the diet. When this is so, custard made, as already suggested, with the dried milk, can also be given.

Fruit and vegetables are usually to be avoided altogether, as they tend to aggravate the symptoms, but their omission constitutes a danger in another direction, for the restricted diet, particularly the absence of fresh milk, entails considerable risk of scurvy. I am not speaking now of the scurvy which occasionally accompanies or precedes the onset of cœliac disease, but of its occurrence after months of dietetic treatment, as has happened more than once in my own experience. Fortunately, although fruit and vegetables are ill tolerated, many of the children with cœliac disease can take two or three teaspoonfuls of grape juice twice daily without making the stools more loose, and in this way scurvy may be avoided.

Out of the foods already mentioned, a diet which is passable enough for a time can be constructed, but, as might be expected, the monotony of it soon palls on the child, and it is difficult to satisfy the child's constant craving for something new.

More monotonous still, but at times undoubtedly useful, is the fat-free diet, which includes rusks, Robb's biscuits, jellies, chicken or veal broth, potato, Revalenta, rice boiled in water, pounded chicken or fish, barley water or weak tea with sugar.

It must not be supposed that all these foods can be tolerated by every child with cœliac disease, but experience shows that the range of choice usually lies within these limits.

I would only add that owing to the unsatisfying nature of the

diet, it is usually necessary to feed at shorter intervals than a healthy child requires.

Drugs. Of drugs, the most useful are the astringents and antiseptics, and I know of no mixture which suits better than the one I have mentioned elsewhere of salol with castor oil, viz., Salol. gr. iss, Ol. Ricini ℥ v, Spirit. Chloroformi ℥ i, Mucilag. Acaciae ℥ xv, Aq. Anethi ad ℥ i ter die. In many cases, after trial of various drugs, it has been so evident that this gave the best result, that the parents have asked that this mixture might be repeated, and in some cases it was given almost continuously for months, and even years, as the child seemed unable to do without it.

Occasionally, when the stools were particularly loose and slimy, I have found Silver Nitrate valuable in doses of one-sixth of a grain. It may be prescribed thus: Argenti Nitratis gr. $\frac{1}{6}$, Glycerini ℥ v, Aq. Distillat. ad ℥ i ter die. Given for a week or two, it sometimes improves the character of the stool. Tannalbin, of which at least 8 grains ter die should be given to a child of two or three years, is also useful. Bismuth I have found of very little value in this disease.

With wasting as a prominent symptom, there is a temptation in cœliac disease to give malt or cod liver oil, but, as a rule, not only does no gain in nutrition result, but there is considerable risk of aggravating the looseness of the bowels and producing further loss of weight.

On the assumption that some defect of pancreatic secretion underlies cœliac disease, a view put forward by the late Dr. Cheadle and others, various pancreatic extracts have been tried. I can only say that in my experience such drugs as Liquor Pancreaticus, Trypsogen, 'Pankreon', &c., have had very little, if any, effect, certainly none comparable to the effect of the castor-oil mixture. The pale colour or whiteness of the stools has suggested an attempt to supply biliary constituents in the form of drugs: in one of my cases Holadin, in another Oxgall was administered, but no good resulted. In one case I gave Radium water in large quantity by mouth for several weeks, but without the slightest advantage.

Lastly must be mentioned the Vaccine treatment, which was suggested by the finding of the dysentery bacillus in the stools. Dr. Nabarro prepared and administered autogenous vaccines for me in some cases, with temporary improvement in the weight, but with no constant success.

CHAPTER XVIII

BILIOUS ATTACKS SO CALLED, IN CHILDREN

'My child is subject to bilious attacks.' One hears this complaint so often that, although the term 'bilious attack' belongs rather to popular than to scientific phraseology, we shall do well to consider what is its real significance. And indeed it is a complaint which always calls for a very careful consideration, for while in some cases it refers merely to passing digestive disturbances, there are others in which it serves as a cloak for much more serious conditions. Herein lies the danger of a vague term like this; it has a misleading sound of innocence which may inspire an altogether unjustified feeling of security.

Now what are the symptoms to which parents give this name 'bilious attack'? As a rule the child has attacks of vomiting, perhaps being sick only once or twice, perhaps many times, and these bouts recur at intervals of weeks or months. With the vomiting there is perhaps some abdominal pain, and, it may be, some headache and a rise of temperature: the tongue is furred, the bowels are costive. After a few hours or a day or longer the symptoms subside, and the parents say, 'It was only a bilious attack'.

Appendicitis. There is one disease which should always be in the medical man's mind, when he is told that a child has 'bilious attacks', and I shall mention it first and foremost, not because it is the disease which is most often called by this name, but because it is so dangerous if unrecognized, namely, appendicitis.

A surgeon once told me that he thought 'bilious attacks' in children nearly always meant appendicitis. His opinion was the result of a large experience of the cases of this kind which come under a surgeon's notice; the physician sees many in which the attacks have other significance, but my own experience has convinced me that slight attacks of appendicitis are not uncommonly supposed to be 'bilious attacks', and consequently made light of, until a more severe attack occurs, and a child's life is sacrificed, which might have been saved if only a careful examination of the abdomen had been made during the supposed 'bilious attacks'. We are all familiar with the classical symptoms of appendicitis, the pain in the right iliac fossa, the constipation

and vomiting, the pain often on micturition, the high temperature, the tenderness especially at a spot two-thirds of the distance from the anterior superior spine to the umbilicus (MacBurney's spot), the dullness on percussion, and the infiltration and resistance in the pelvis on rectal examination; but I want to emphasize here the slightness of the symptoms in many attacks. A girl, aged eight years, had had attacks of slight pain in the abdomen four or five times in the past year, with some constipation; there had been little or no vomiting, and the attacks were regarded as so trivial as hardly to call for attention. Then another occurred which was supposed to be 'one of her bilious attacks', but this time there was severe pain and vomiting; within a few hours general peritonitis supervened, and in spite of operation and removal of an inflamed appendix the child died.

In another case a girl, aged nine years, had been 'subject to liver attacks' for four years; there was vomiting and some slight pain in the abdomen, but the attacks were so slight that the mother had been told by her doctor 'just to give the child a dose of calomel whenever an attack occurred'; then a more severe attack than usual occurred; at first it was called, as before, a 'bilious attack', but it soon became evident that it was serious. I saw the child a few days after the onset of this attack; there was then evident appendicitis, the child's condition appeared desperate, and immediate operation seemed to give the only chance; laparotomy showed not only a perforated appendix but an encapsuled abscess with dense fibrous matting which must have been the result of previous attacks of appendicitis.

In the majority of cases appendicitis in childhood is a disease of the school age, but it occurs even in infancy occasionally. In a boy whom I saw at the age of 2 years and 4 months with appendicitis and general peritonitis, which proved fatal, there had been a supposed 'bilious attack', which was almost certainly an attack of appendicitis, at the age of 19 months. In three other infants, all boys, aged respectively 19 months, 16 months, and 14 months, I have seen very acute appendicitis, which at operation revealed in two cases a gangrenous appendix, and in the third case also the appendix was very acutely inflamed. With operation all three cases made a good recovery, but in all of them the diagnosis was less easy than it usually is in older children. Appendicitis in the infant is very apt to be mistaken for simple digestive disturbance; it is only by carefully observing the greater rigidity

of the abdomen on the right side than on the left, and by watching for evidence of local tenderness on pressure in the right iliac region, that the distinction can be made, and this may require no small amount of tact and resourcefulness when the infant cries seemingly just as much when he is touched in one part as in another, or even when he is disturbed at all. There are times when it may be advisable to use an anæsthetic, but, both in infants and in older children, I feel sure, from experience, that an anæsthetic, so far from being always a help, is sometimes a hindrance to diagnosis, for whilst in some cases there is a local thickening which it may be very difficult to feel until the muscles are relaxed by an anæsthetic, in others, especially the most acute cases, there is no such thickening, and the anæsthetic only serves to obliterate the rigidity and tenderness which give the best indication of the diagnosis. A warm hand and a light touch will often obtain information which even an anæsthetic will not give.

Let me emphasize the risk of overlooking appendicitis by referring to the slightness of symptoms in some cases. In a child, whose appendix when removed showed considerable inflammation and ulceration of its wall, there had been no vomiting and the temperature had not been above 100° F.; in some children tenderness and increase of resistance on palpation in the right iliac fossa are the only distinctive features of an attack of appendicitis, which otherwise might pass for any of the other much less serious conditions, which are described by parents as 'bilious attacks'. A history of bilious attacks always calls for careful examination of the abdomen, if possible during the attack, for it may be impossible to detect anything abnormal locally during the intervals between these slight manifestations, and if the child is seen first during an interval, the parents should be advised to let the doctor examine the child when an attack occurs; many a child's life which has been sacrificed to appendicitis would have been saved if this precaution had been adopted.

Digestive Disturbance, 'Gastric Catarrh'. I pass now from this most serious significance of a 'bilious attack' to the most ordinary and perhaps least serious. In some children indigestion takes the form of an attack of vomiting, the temperature rises to 101° or 102°; the tongue is furred, and there may be some 'stomach-ache' or epigastric pain, but there is no tenderness in the right iliac fossa. There are children—usually, I think, very excitable nervous children—who get such attacks every few weeks; they have usually shown some evidence of feeble digestion at other times. Such a case is the following.

Rose R., aged $9\frac{11}{12}$ years, was said to have had two 'bilious attacks'; in each vomiting persisted for about thirty-six hours; in one there was some pain in the epigastrium: the child had been under observation for pain in the abdomen and flushing of the face after meals. Care in diet and a mixture of rhubarb and soda prevented any recurrence of the attacks so long as she was under observation.

Sometimes there is no vomiting, the child simply becomes sallow and seems languid, the breath is offensive, and there is some headache, the stools are costive and pale, the temperature is raised; a smart purge sets the child right in a few hours.

Arthur D., aged $3\frac{1}{2}$, was said to have frequent 'bilious attacks', in which he became dark under the eyes; there was no jaundice, but the sclerotics lost their clearness and were said to look sallow: sometimes there was more or less vomiting in the attack, but sometimes there was none; the stools were white, and the appetite very poor.

It would be easy to multiply illustrations of such attacks, but it is unnecessary, they are familiar to every medical man. The pale colour of the stools which so often accompanies this condition suggests some deficiency of bile secretion, and there are those who talk glibly enough about 'liver attacks', and 'chill on the liver', but I think it is a pity to use terms which imply more than we know: it is possible, perhaps even probable, there may be some disturbance of the function of the liver in these attacks, but if so it is probably secondary to disturbance of digestion, or to deficient elimination from the bowel owing to constipation. Certainly dyspepsia or chronic constipation is the only disorder that can be affirmed with any certainty in the large majority of cases, and the successful treatment is the correction of dyspepsia or constipation or of both. It would be out of place to enter on any full description of the treatment of either of these disorders here; I will only say that in the dyspepsia of childhood carbo-hydrates, that is to say, the starchy and saccharine elements of food, but especially the former, are by far the commonest cause of indigestion. This is a point worth remembering in the treatment of these so-called 'bilious attacks', when they are really manifestations of dyspepsia; the amount of farinaceous food is to be regulated; for instance, the child is not to make a breakfast of a large bowl of porridge followed by several pieces of bread-and-butter; the porridge is better omitted, an egg or a little fat bacon, or some fish, with a little bread-and-butter, is a better breakfast; and again at dinner some blanc-mange or junket or custard may take the place of any cereal milk-puddings; fruits also, apparently on account of their indigestible residue of fibres and seeds, are common causes of indigestion in childhood and may require strict limitation.

With the same purpose of preventing starch dyspepsia the administration of malt is valuable in these cases, and its diastasic action may be increased by the addition of some pancreatic extract; there are several excellent preparations in the market which fulfil these intentions, such as Bynopancreatin (Allen & Hanbury's), Maltine with Pepsin and Pancreatin (Maltine Manufacturing Co.). A teaspoonful of any of these given immediately after meals has a very marked effect in aiding digestion.

At the same time it is often advisable to give a rhubarb and soda mixture just before meals; in some cases this alone, if given for several weeks, is sufficient to prevent any recurrence of the attacks, or at least to reduce their frequency.

In most of these cases there is more or less chronic constipation, and if this is so it is not sufficient to treat any indigestion which may be present; regular and sufficient action of the bowels must be ensured. In these cases I think the best method is to use one of the combinations of cascara with malt, which are now made by several chemists, and to give this after each meal in sufficient doses to keep the bowels regular; or a dose of fluid magnesia or some apenta water may be given regularly every morning: whatever laxative is used it must be given regularly for many weeks or months so as to establish in the bowels a regular habit of action. With such treatment there are very few cases of this kind which cannot be freed altogether from the 'bilious attacks'. If the child comes under treatment during an attack it is best to give a dose of calomel at once, one grain for a child under three, $1\frac{1}{2}$ grains for a child between three and six years, and 2 grains for an older child; after this a bismuth and soda mixture, containing 10 grains of the carbonate of bismuth and 10 grains of sodium bicarbonate should be given every four hours.

Cyclic vomiting. There is another disorder which very commonly goes by the name of 'bilious attacks', the condition first described by Dr. Gee as 'fitful or recurrent vomiting', and now often called cyclic or periodic vomiting. This disorder seems to be almost, if not quite, peculiar to childhood. It is characterized by sudden onset of vomiting without apparent cause, the child is often very drowsy, there is sometimes headache, and sometimes, but not always, a rise of temperature which may be 102° or more. I have noted in several cases pain in the epigastrium, the bowels are costive, the vomiting is usually severe, even teaspoonful doses of water may be vomited, and the vomit is sometimes coloured bright green with bile, and in a severe

case may even be tinged with blood. During the attack the child is often drowsy and apathetic ; sometimes restless, waving the arms about and unable to sleep. Thirst is often distressing, and in a bad attack the lips are dry and covered with sordes. After one to three days usually the vomiting subsides, and the child recovers its ordinary health within a few days ; but the attacks are occasionally much longer ; I have twice known the attack to last fourteen days, and, as may be supposed, the degree of exhaustion may then become very serious.

I have never myself seen an attack prove fatal, but in one of my cases life seemed to be in danger from the severity of the exhaustion. Dr. Crozer Griffiths, however, mentions two fatal cases within his own experience, and there was in the Children's Hospital, Great Ormond Street, under the care of Dr. Lees, a girl aged $4\frac{1}{12}$ years, who was admitted with a history of recurrent attacks of vomiting; for the fourth and fifth of these the child was taken into hospital, and the fifth proved fatal. The child, whose breath smelt of acetone and whose urine showed acetone and diacetic acid, vomited severely and fell into a comatose condition with gasping respiration like the 'air hunger of diabetes' ; after some terminal convulsions she died, and the post mortem revealed some fatty change in the liver and kidneys, but otherwise nothing except some œdema of the lungs.

This case was recorded by Dr. Langmead,[1] who has also put on record two other fatal cases in girls aged respectively 4 and $4\frac{11}{12}$ years; in both there was found some fatty change in the liver.

Of fifty cases, which I supposed to be of this nature, twenty-four were boys and twenty-six were girls : in one family two children were subject to severe attacks of cyclic vomiting, and an elder brother had had attacks probably of the same nature ; in another family two sisters were affected. In thirty-four out of the fifty cases the attacks began during the first five years of life, in six the attacks began before the end of the first year, one just under the age of three months and one during the second week of life. The frequency of the attacks varied from three in two years to once a fortnight.

The association of this disorder with a nervous temperament is, I think, a fact which cannot be ignored in considering its etiology. The children affected are nearly always unduly excitable, nervous children, or show more definite nervous symptoms, such as habit spasm or somnambulism; in two cases the attacks of

[1] *Brit. Med. Journ.*, Feb. 5, 1905; ibid., Sept. 28, 1907.

vomiting were sometimes associated with convulsions. Dr. Gee noted a history of migraine in one or other of the parents in some of his cases, and my own notes show a similar occurrence in at least four of the series (in a fifth case there had been 'sick-headaches' in the mother). Like migraine, the attack of cyclic vomiting is sometimes preceded by premonitory nervous symptoms: in one of my cases a boy, $10\frac{1}{2}$ years old, was said to become very excited and to have headache just before the attack began; in another a girl, aged $11\frac{1}{2}$ years, was said to be specially irritable for two days before the onset of each attack; in others more general warnings were noticed, in sallowness of complexion, lassitude, and dark colour under the eyes.

It seems probable that these cases are quite distinct from the recurring attacks of vomiting and malaise which I have already described as associated with dyspepsia and chronic constipation, but I think we must remember that no proof of this has yet been brought forward: for aught we can prove to the contrary, the difference may be merely one of degree not of kind; certainly differentiation at present is little more than an arbitrary distinction between mild cases, in which we think we can recognize sufficient cause for the attacks, and those more severe cases in which there seems to be no adequate cause. Both may be and probably are due to some auto-intoxication.

Within the past few years it has been observed that acetone and diacetic acid can be detected in the urine, and sometimes the smell of acetone, a sweetish apple-like odour, in the breath in these cases of cyclic vomiting. At first it was supposed that this feature indicated some special form of auto-intoxication which differentiated them from cases of recurring vomiting due to other causes; this, however, has proved fallacious, for with severe vomiting due to any cause it is not uncommon to find acetonuria, and occasionally the acetone smell of the breath. There is, however, some ground for supposing that the acetonuria, or rather the acetonæmia which presumably underlies it in these cases, does bear a direct causal relation to the vomiting, and is not a result or coincidence: acetonæmia implies a diminished alkalinity of the blood, an acidosis as it has been called, and Dr. Edsall of Philadelphia, arguing that if this were the cause of the symptoms, the disorder should be amenable to treatment by the administration of alkali, suggested the use of large doses of bicarbonate of soda in the treatment of this disorder. To the excellent results of this treatment I can testify: it seems to act almost as a specific in the prevention of attacks, and I think undoubtedly curtails

attacks if given during the first few hours after the onset. If it can be retained, a solution of bicarbonate of soda (15 grains in 2 drachms of water, with five or ten drops of brandy) should be given every hour until six doses have been given, and then every two hours. If this is not retained (and it should not be assumed that it will not be retained because all else is vomited, for sometimes the alkaline solution is kept down where other fluids have been rejected at once) then the bicarbonate of soda must be given per rectum, 2 drachms may be injected in half a pint of water.

Small doses of morphia injected subcutaneously have been found useful in some cases, $\frac{1}{30}$ grain for a child aged two years, $\frac{1}{20}$ grain for a child aged three to four years, $\frac{1}{12}$ grain at six years, $\frac{1}{10}$ grain at ten years.

The ordinary treatment of vomiting in other conditions, such as bismuth, hydrocyanic acid, tincture of iodine, and so forth, seems to have little effect in these attacks ; usually no food is retained for several hours, and then only watery fluid, broth, whey, or perhaps only soda-water in small quantities. If the inability to retain food persists beyond twenty-four hours it will be wise to give nutrient enemata of peptonized milk ; 3 ounces may be given every six hours, being allowed to run in slowly from a funnel, through a soft tube, which should be introduced quite 4 to 6 inches. Where exhaustion is extreme it may be necessary to administer saline infusion subcutaneously.

I have thought that as a matter of experience glucose is of value in the severe attacks of cyclic vomiting, and recently on theoretical grounds this form of sugar has been recommended [1] for the treatment of these cases. I have given it usually as a rectal injection, using a five or ten per cent. solution. I have also used it by mouth, when it could be retained, in doses of one or two drachms of the ten per cent. solution at short intervals. It can also be used for subcutaneous infusion (vide p. 230).

The preventive treatment of this recurrent vomiting is most satisfactory since the introduction of the sodium bicarbonate treatment. A dose of 10 grains of the sodium bicarbonate is to be given three times a day, and the mother can be instructed to give the medicine more often if an attack threatens, and to give it every hour if vomiting begins. The result of this treatment in the prevention of these attacks has been most gratifying in my experience. Before the introduction of this method I had been in the habit of ordering liq. arsenicalis for them, and I think

[1] Mellanby, *Lancet*, July 1911, p. 11.

with benefit, but I do not think it is so effectual as the sodium bicarbonate.

I have mentioned the occurrence of high temperature in some of these attacks of cyclic vomiting, and I have pointed out that the symptoms vary considerably in severity : now I wish to refer to a disorder which I believe to be very closely allied to this so-called cyclic vomiting, and its mention here will not be out of place, for it is sometimes called ' bilious attack ' by the parents, namely, recurrent pyrexia. I am not aware that this term is in general use, but it seems to me a good one for the disorder to which I apply it, especially if the relation to recurrent vomiting is as close as I imagine. Some children are subject to bouts of fever lasting sometimes two or three days, sometimes a week or more, without any apparent cause. The onset is sudden, but there are often vague premonitory symptoms like those preceding recurrent vomiting ; there is usually some headache and the stools are often pale and almost always constipated. After two or three days or longer the temperature, which has been almost continuously 102° to 104°, sometimes even 105°, falls within twenty-four or thirty-six hours to normal, and the child quickly recovers his normal health. Now in some cases these attacks are sometimes associated with vomiting, sometimes not ; when vomiting is present the attacks may so closely resemble those of recurrent or 'cyclic' vomiting as to suggest that the two conditions of recurrent pyrexia without vomiting and recurrent vomiting with pyrexia are interchangeable. I do not assert that this is so, but I think it is worthy to be considered whether this recurrent pyrexia, which has been described as ' carbo-hydrate pyrexia ' and 'food fever', may not be the manifestation of some auto-intoxication, allied to, if not identical with, that which produces recurrent vomiting.

Migraine. The occurrence of headache is an accompaniment which might be expected in the febrile condition which I have already mentioned as passing for ' bilious attacks ' ; but there is another condition which I must now mention in which the headache is, so to speak, a more integral part of the disorder, namely migraine.

This is not uncommonly spoken of as ' a bilious attack ', and though in my experience not very frequent in childhood, occurs in its most typical form occasionally during the later half of childhood between five and twelve years of age. Sir William Gowers notes a point in which the migraine of children differs from that of the adult, namely, in a tendency to pyrexia with

the attack. He quotes in illustration the case of a child who, from the age of two years, had attacks of severe pain on one side of the head associated with a temperature of 102° to 103° : after vomiting the child fell asleep and woke up well. The headache of migraine is not necessarily a hemicrania, there are not necessarily any visual changes, the vomiting may be repeated several times, though usually the child vomits only once or twice in each attack ; so that it is easy to see how with headache, vomiting, and rise of temperature, it is sometimes very difficult to say whether the so-called ' bilious attacks ' are really the result of some dyspepsia or of constipation, whether they are short attacks of the so-called cyclic vomiting, or whether they are migraine. And, indeed, these conditions may be very close akin. I have already mentioned the occurrence of migraine in the parents of children who suffer with recurrent vomiting, and Dr. Rachford of Cincinnati has observed an additional relation of great interest, namely, the replacement of cyclic vomiting by attacks of migraine when the patient reached adult life ; this he observed in four cases.

In the treatment of these attacks of migraine in children, regular action of the bowels and good digestion must be ensured if prevention of the attacks is to be successful. I would lay great stress upon this point, for I think there is a tendency to rely too much upon sedative drugs in the prophylaxis of migraine : the regular administration of some laxative, for instance one of the preparations of cascara and malt, or of some fluid magnesia, may be the most successful line to adopt if at the same time the diet is regulated, especially in the direction of preventing over-eating and the bolting of food with deficient mastication. The children who suffer with these attacks are usually at the school age, and in those who attend our city schools, the hurrying off to school after a rapid breakfast, very likely without waiting to relieve the bowels for fear of being late at school, and the hurried and sometimes quite unsuitable midday meal at school, perchance of sandwiches or even some pastry or a roll and butter, may be a fruitful source of chronic constipation, dyspepsia, and so-called 'bilious attacks', whether migranous or otherwise. In some schools also the atmosphere of the class-rooms, especially in the winter, when windows are apt to be closed and ventilation very deficient, is a sufficient determining cause of migraine. If the attacks recur frequently, I think that phenazone given twice or three times a day regularly in a mixture of rhubarb and soda is likely to be useful,

and to this 1 or 2 minims of liq. arsenicalis, according to the age, may be added. In the attacks phenazone or phenacetin with caffeine citrate gives perhaps the speediest relief to the headache; but often, and I speak from the fullness of personal experience in boyhood, nothing seems to give relief until vomiting has occurred and sleep has followed.

Renal vomiting. Lastly, there are more remote possibilities which must be borne in mind as possible sources of a supposed 'bilious attack'. Walter F., aged six years, was stated to have had 'bilious attacks' for the last three years. Every three or four weeks he had an attack of vomiting usually lasting only a few hours, but occasionally lasting several days; in a recent attack he had been sick for ten days, not able to keep down even a drop of water; he was feverish during this time, constipated, and had some pain in his abdomen. The pain in the abdomen was always on the left side, and on palpating the abdomen in the routine of examination one found a rounded smooth tumour almost the size of a man's fist in the left lumbar region, overlaid by bowel, which could be rolled under the finger over the underlying tumour. He was admitted into the children's ward at King's College Hospital, where he seemed quite well except for the presence of the tumour, and after a few days the tumour suddenly disappeared completely. The amount of urine passed on that day was about 4 to 5 ounces in excess of what he had passed on other days. The tumour did not reappear while he was in hospital; so after remaining there for about a fortnight he was sent home. After a short time he reappeared with a history that another attack of vomiting had begun five days previously, he had had some pain on the left side of the abdomen, and the vomiting had been very continuous. On palpation the tumour was now to be felt as previously, but not so large. He was re-admitted and the tumour increased slowly in size, but the boy had no pain and no vomiting and seemed well; eight days after admission the tumour completely disappeared again, and as before it was noticed that he had been passing about this time a larger quantity of urine than usual.

It seemed clear that there was a hydronephrosis, and my colleague, Mr. Carless, operated and found a dilated pelvis with a valvular kink of the ureter at its entry into the pelvis: for this he did a plastic operation, with excellent result. Here, then, was a case of hydronephrosis which gave rise to bouts of vomiting with some abdominal pain, which were described as 'bilious attacks'.

A girl of seven years was brought for 'bilious attacks', which had recurred for two years: she was feverish and vomited in the attacks, which lasted one to three days. I saw her in an attack which had lasted five days with severe vomiting; she could keep down nothing except soda-water: the temperature was 103° to 104°; there was some acetone in the urine, which was acid. So far the case might have passed for one of recurrent vomiting, but on examining the urine further it was found to contain many pus corpuscles, and on palpation there was tenderness over the right kidney. There was evident pyelitis, and it seemed probable that there might be a renal calculus. I lost sight of the case before the diagnosis had been cleared up, but these facts are sufficient to illustrate my subject—pyelitis producing a so-called 'bilious attack'.

The last case I will mention of renal sources for such attacks was an instructive one, for it gave rise to a diagnosis of 'cyclic vomiting'. Leslie S., aged ten years, had had attacks of vomiting lasting one to two days, every two or three weeks for eleven months; in the attacks there was no headache, but he had some pain in the right hypochondrium on which no stress was laid. He was treated with bicarbonate of soda for about eighteen months, which seemed to diminish the frequency of the attacks, and I had no doubt whatever that it was a case of cyclic vomiting until a severe attack occurred with great pain in the abdomen followed by sharp pain in the urethra, and the boy came to the hospital showing with great satisfaction a renal calculus the size and shape of a date-stone which he had passed with his urine.

Meningitis. There is one more condition which must be mentioned here, for it comes so often in the guise of an innocent 'stomach attack', or, as the parents say, a 'bilious attack', namely, tuberculous meningitis. Face to face with a case of vomiting, headache and constipation, who shall say during the first day or two whether the symptoms are simply the result of some gastric catarrh, or whether the attack is a first bout of cyclic vomiting, or the first indication of a fatal meningitis? I have notes of cases in which mistakes were made owing to complaint of pain in the abdomen during the early stage of tuberculous meningitis; I know not what the explanation of this may be, hard scybala, the result of the constipation, may account for it, or possibly the tuberculous mesenteric glands which are so often present may be causing pain; but any way the fact is worthy of note, for it should put us on our guard against tuberculous men-

ingitis, even where complaint of abdominal pains seems to localize the trouble to the abdomen. An additional caution I would give is this : headache is occasionally absent or extremely slight in the early stages of tuberculous meningitis, although vomiting and constipation may be very marked. For instance, George B., aged two years and ten months, the child of a doctor, had vomited twice within two days, ten days before I saw him ; about a week later he vomited again two or three times within two days, the bowels were costive, but there was no headache nor pain anywhere apparently ; the attack was regarded as due to some digestive disturbance. Four days later there was squint, with optic neuritis and other signs pointing to tuberculous meningitis, of which the child died a few days later. I have seen cases in which the child repeatedly declared that there was no headache, although the course of the illness and subsequent autopsy proved that there was tuberculous meningitis. Of course, both abdominal pains and absence of headache are quite unusual in this disease, but I mention them to emphasize my point, that it is only too easy to mistake the early symptoms for some slight gastric disturbance, or, as the parents call it, 'a bilious attack,' more especially when our wishes are all in favour of the trivial ailment.

The conclusion of the whole matter is this, that the parent's tale of a 'bilious attack' needs careful sifting : it may mean the most innocent of digestive upsets, it may on the other hand mean cyclic vomiting, or serious diseases such as I have mentioned.

CHAPTER XIX

FEVER OF OBSCURE CAUSATION

I PROPOSE in this chapter to consider a problem which must be only too familiar to most medical men, the child with fever for which we can find no explanation. One of the most striking features of infancy and early childhood is nervous instability, and this may show itself, not only in what we commonly recognize as nervous disorder, for instance, the tendency to convulsions upon provocation which would have no such effect in later life, but also in irregularity of almost every function in the body: how irregular, for instance, the respiration of an infant is apt to be, how easily the rhythm of the diaphragm is disturbed, so that hiccough occurs many times a day during the first few months of life; how easily the cardiac rate and sometimes the rhythm is disturbed in a child, and how erratic is the secretion of urine in early life, giving rise to scares of suppression or retention, when there is really nothing amiss; even the co-ordination of the gastric movements is so easily disturbed that the pylorus may contract when it should dilate, and the result may be troublesome vomiting, and hypertrophy of the stomach. The nervous control of all these functions only becomes stereotyped, so to speak, as the child grows older; with increasing years comes increasing steadiness of function.

So it is with the temperature, in the regulation of which, as we all know, the nervous system plays an important part: in the infant the temperature rises on the most trivial cause; it may be lumpy fæces in the intestine, it may be a slight coryza. I have seen cases where even some irritation of the skin seemed sufficient cause; as the child grows older, similarly but less often, constipation, some slight deviation from a customary diet, or even some unwonted excitement may be sufficient cause for a rise of temperature.

The next point that I wish to lay stress upon is this: that the degree of nervous instability and therewith the tendency to pyrexia, varies in different children. One child will get a temperature of 101° with simple constipation, where another child at the same age will get no rise of temperature at all: moreover,

this instability of temperature lasts to a much later age in some children than in others; one will have lost it at four years old, another will show it at ten years: there are, indeed, cases on record which show that occasionally it persists even into adult life. Further, any cause which produces fever in the stable child is likely to cause a much higher temperature in the child with this instability. This peculiar tendency to fever is seen chiefly in the children of neurotic parents, or in families with some neuropathic taint, and generally the child whose temperature is easily disturbed is recognizable as in other ways a nervous or excitable child.

In his knowledge of the tendencies of a particular family and of the behaviour of the temperature in previous disturbances in the particular child, the family doctor has a piece of information which is not to be despised, and which is included in that mysterious 'knowing their constitution' to which the laity very rightly attach much importance.

But whilst it is important to realize this peculiarity of early life and its accentuation in the children of certain families, it is easy in a particular case to be led astray by these seductive generalisms, and I think that we shall do well to lay it down as a rule that nervous instability is to be regarded rather as a contributing or modifying factor, than as the 'fons et origo' in any given case of obscure fever in a child.

Recurrent pyrexia. Turning now from the general to the particular, I wish to consider first a group of cases which must be familiar to most medical men, cases which I would label 'recurrent pyrexia'. The history is this: that at intervals of weeks or months, perhaps five or six times a year, the child, usually, I fancy, a boy at any age up to ten or twelve years old, seems out of sorts, languid, sometimes markedly drowsy, and feverish, his temperature is found to be raised, perhaps 102° or 103°, sometimes 105°, in one of my cases 106·2°. The onset of the fever is generally quite acute: in one of my cases the temperature was stated to have risen from normal to 103° within an hour. The fever usually subsides within three or four days, but its duration is variable; it may last only twenty-four hours; occasionally it lasts a week or ten days with slight remissions. During the attack the bowels are generally costive and the stools pale. In some cases there is more or less vomiting. There is no pain usually; occasionally the child complains of slight headache. Physical signs there are none whatever, as a rule, though occasionally a few râles in the chest may raise

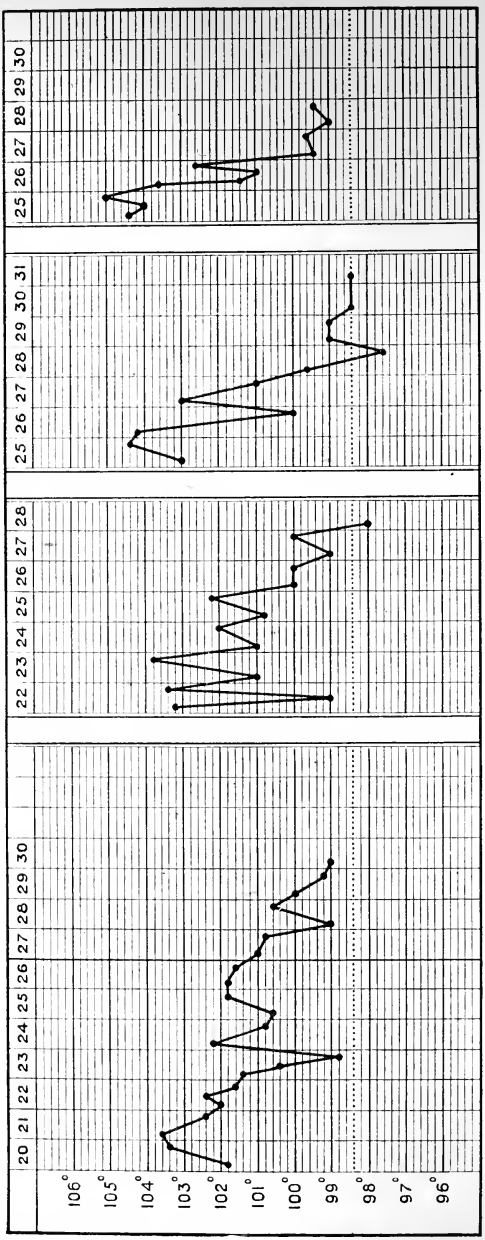

FIG. 11. Chart I. Recurrent pyrexia. Charts of four attacks of fever which occurred within twelve months, in a boy aged $2\frac{1}{2}$. Characteristic 'recurrent pyrexia' without obvious cause.

suspicions of some commencing acute pulmonary attack, which, however, fails to appear.

Chart I shows the temperature in four such attacks in a boy aged two years and one month: these all occurred within twelve months; he was a bad sleeper, often started up in his sleep and cried out, his bowels were sometimes costive, and after the febrile attacks he passed 'slime' in his stools; but there were no other symptoms associated with the fever beyond the usual accompaniments of any feverish condition,—the pulse became quick, 160 per minute on some occasions, and his respirations as much as 52 per minute.

Seeing these cases as the consultant generally does after several attacks have already occurred, he has an easier task in recognizing their nature than has the family doctor who is called in at the first attack, when a diagnosis is impossible, or at best can only be a matter of conjecture. Judging from the histories given to me, I feel sure these cases give much trouble in diagnosis, even when the attacks have recurred several times. 'Influenza' they are called until the most implicit confidence is shaken by an influenza which recurs once every two or three months, or even oftener, for several years. In the case of one boy who had had about four attacks each year from infancy until he was nearly eleven years old, various doctors had seen him in various attacks: one had diagnosed influenza, another pneumonia, and another peritonitis, and another had seen him in an unusually prolonged attack, and had, very prudently, treated him as a typhoid, although the mother's experience of her son's attacks made her very positive that the fever was not typhoid, and she proved to be right.

The association of constipation with fever and perhaps vomiting has raised the question of appendicitis in some of the attacks, but the absence of abdominal pains and tenderness, and of any resistance on palpation in the right iliac fossa has negatived this; then again the drowsiness which may be a marked feature, and its association with constipation and perhaps vomiting, has raised fears of meningitis, a possibility which may be difficult to exclude, although the history of similar previous attacks is opposed to it, and the subsequent speedy recovery clears up the doubt. The term 'bilious attacks' is sometimes applied to these bouts of pyrexia, undesirably, I think, because it is applied to several entirely different conditions, and is often the cloak which conceals such a serious disease as appendicitis.

The significance of these attacks is by no means clear. I doubt

if they have the same significance in all cases, but certainly some of the symptoms seem to point to a gastro-intestinal origin: the costiveness of the bowels, the pallor of the stools, the vomiting in some cases, the striking value of calomel in stopping the attacks, and the apparent influence of diet all seem to point this way; in one case the mother told me that she could always recognize when attacks were coming by the passage of much mucus in the stools, in another case mucus was passed in the stools just after the attacks, in a third the onset of the attack was usually preceded by hiccough. Simple constipation is not, I think, sufficient to account for the attacks; indeed, in some cases the bowels are open thoroughly though the pyrexia continues; there would seem rather to be some auto-intoxication from the bowel, but how this arises there is not sufficient evidence to show. Whatever may be the determining cause, one thing, I think, is clear—and herein lies the application of my opening generalisms—these attacks of recurrent pyrexia occur chiefly in children of nervous excitable tendency, a point to be remembered in their management.

The outlook in these cases is good: the tendency to the attacks becomes less as the child grows older; they recur sometimes for several years, but I should say rarely later than ten or eleven years old. I have never seen any serious mischief resulting from them, even when the temperature reached 105° or 106°.

Now what is to be done for them? When the attack has come, the child is languid and out of sorts, and no doubt bed is the best place for him, and until the bowel has been cleared out, the lightest of food, broth or diluted milk, will be sufficient; a dose of calomel will hasten the disappearance of the fever, but can anything be done to prevent another attack?

The value of special dieting in some of these cases of recurrent pyrexia was pointed out by the late Dr. Eustace Smith,[1] who applied the term 'Food Fever' to the attacks. The efficiency of the regimen laid down by him is illustrated by a case recorded by Dr. Davy,[2] of Exeter. From the age of $2\frac{1}{2}$ up to 11 years, his patient, a boy, had frequent attacks like those I have described. The boy eventually lost them entirely after careful dieting, the chief point in which was the exclusion of starchy puddings, jam, marmalade, fruit raw and cooked, potato, turnips, carrots, sweet cakes, and oatmeal porridge. He referred to other cases cured

[1] *Brit. Med. Journ.*, Feb. 10, 1906.

[2] Fever in children caused by indigestion of certain kinds of carbo-hydrate food. *Lancet*, Sept. 24, 1904.

by similar care in diet. My own experience would not justify me in attaching a specific value to the dieting, but I think that it is worthy of trial, and in some cases does good ; I have generally dieted these cases upon similar lines, and given a mixture of arsenic and citrate of potash, the arsenic being of value in reducing the nervous excitability of these children.

There is a group of cases which ought perhaps to be mentioned here, inasmuch as it resembles those described above in the recurrence of seemingly unexplained fever, beginning suddenly and lasting several days, with a temperature rising sometimes even to 105°. The point of difference is the presence, as shown on careful inquiry, of some unhealthy condition of the throat. This may have attracted little if any notice, but when once attention is drawn to it, perhaps by the child's complaining of slight sore throat, or by a little puffiness of glands at the angle of the jaw in one of the attacks, it is found to occur on each occasion, and is evidently the underlying cause. It would seem that there is a recurring infection of the nasopharynx, but there is sometimes a perplexing association, namely, an unhealthy condition of the stool, which contains much mucus and is extremely offensive. Whether the throat or the bowel disorder is to be regarded as primary is not clear, but it seems most probable that the nasopharyngeal infection comes first, and that the swallowing of infected mucus is responsible for the bowel disturbance ; a sequence which, I think, is by no means uncommon in the throat affections of childhood.

Prolonged slight fever. The next group of cases to which I would draw attention is one in which, for weeks or months, the child has a slight evening rise of temperature to 100° or thereabouts, or, as less commonly happens, a continuous slight elevation of the temperature ; in either case the rise of temperature may persist for many months, without signs or symptoms to explain it. Most of the children I have seen with this condition have been children in the first half of childhood, that is, under the age of six years. The child has looked perfectly well, and seemed so in every way, except, perhaps, that the weight has not increased as it should ; but, like those with recurrent pyrexia, they are almost always markedly nervous children, and usually children of neurotic parentage.

Such a temperature is shown in Chart II. The patient was a boy aged 5 years, the elder of two brothers, both of extremely nervous temperament, both subject to night-terrors, and the elder one to habit-spasm also. Both children successively showed in early childhood for many months a rise of temperature

to 100° or 100·2° at night; the morning temperature in both cases was seldom below 99°. The father, a medical man, was anxious lest tuberculosis should underlie this slight fever, but there was never any sign to suggest tuberculosis; at times the temperature would keep within a perfectly normal range for a week or two, then it would fluctuate at the higher level again for several weeks without any apparent alteration in the health of the child. After many months the temperature became normal in both children, both, apart from their nervous temperament, remained perfectly healthy, and lapse of several years has brought to light no evidence of organic disease in either.

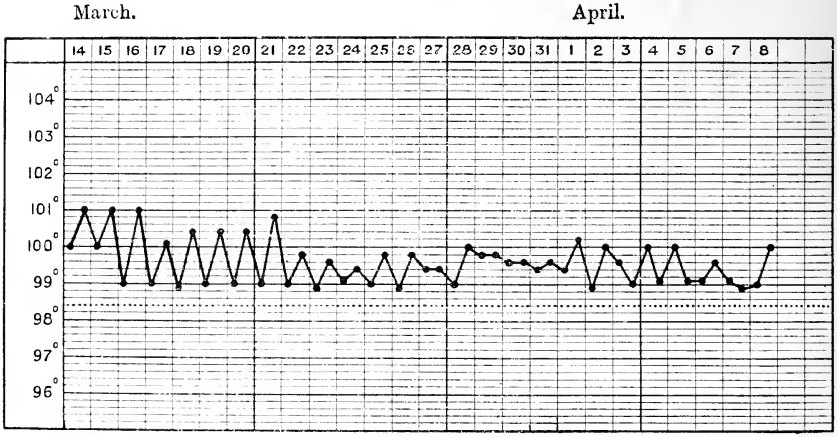

Fig. 12. Chart II. Prolonged slight fever in nervous child without apparent cause.

I had under my care a girl, aged 9¼ years, whose temperature for many months was exactly similar to that shown above: it was usually about 100° at night. The child showed no symptoms except that she was morbidly nervous, and during part of the time that the rise of temperature persisted, she suffered with a habit-spasm consisting of a lateral twitch of the head. In this particular case the slight elevation of the temperature was discovered almost accidentally; the habit-spasm was mistaken for chorea, and the temperature was taken as a precaution in watching for evidence of rheumatism; when it was found to be above normal it was taken daily, and showed the slight evening elevation, on account of which I was consulted. I suspect that many such temperatures go undetected; I have noticed that in my own experience they have occurred much more often in the chil-

dren of medical men than in those of other people, which probably indicates a more frequent resort to the thermometer in doctors' families, and so more ready discovery of such abnormalities.

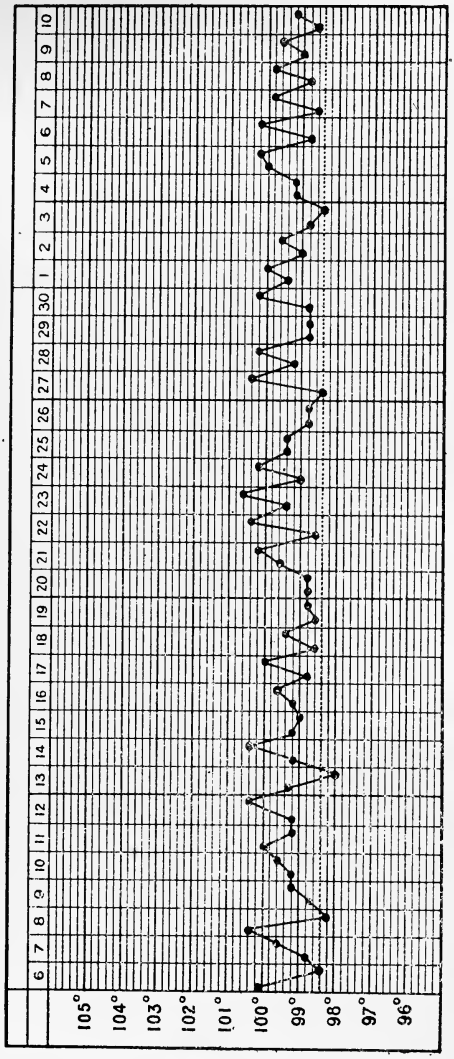

FIG. 13. Chart III. Prolonged slight fever without apparent cause in girl aged 3½ years. Fever lasted six months: no indication of disease.

It is at least tempting to consider the temperature in such a case as purely neurotic; it is clear from the occurrence of habit-spasm that there was a morbid instability of the nervous

system, what more natural than that the thermotaxic function of the brain should also be unstable ? That these slight daily rises of temperature over prolonged periods without apparent cause may be of no sinister significance I can assert from having had a considerable number of such cases under observation at intervals for several years. This particular child was said to be quite healthy when I heard of her four years later ; the daily rise of temperature ceased after some months.

The temperature in a similar case is shown in Chart III from a girl aged $3\frac{1}{2}$ years who had suffered at various times with vomiting and pale stools and other symptoms of indigestion. She was said to be an extremely nervous, excitable child. Careful examination failed to show any other cause for the fever, which persisted for six months.

I have notes of many similar cases, and if I were to sum up my impression of them as a whole, it would be this : that the nervous temperament plays a very large part in determining the fever; but that in a majority of the cases the exciting cause is in the gastro-intestinal tract. In several of the cases under my observation, there has been some chronic difficulty of digestion ; the child has shown more or less of the usual symptoms of chronic indigestion which I have described in a previous chapter. In some cases it has seemed to me that without producing any obvious symptom of indigestion, the feeding was excessive, and a reduction of the large quantity of milk or other food taken, and an increased allowance of exercise where it had been restricted because tuberculosis was suspected, have been followed by subsidence of the temperature.

A clinical feature which may perhaps point in the same direction is the occurrence in some of these children of bouts of much higher fever at intervals of several weeks or months, resembling the ' recurrent pyrexia ' which I have already described as being probably related to faulty digestion of certain foods. It is easy to talk glibly of auto-intoxication, it is difficult to prove it ; nevertheless I think there is much to be said for the view that this prolonged slight fever, as well as the recurrent attacks of severe fever, may be due to an auto-intoxication from the alimentary tract ; in the one case the absorption may be conceived as occurring daily in small doses over long periods ; in the other case the child absorbs a much larger dose at long intervals ; in both cases the effect of the toxic material in producing fever is determined by the nervous instability of the child.

It may be said that if this were so it should be easily proved

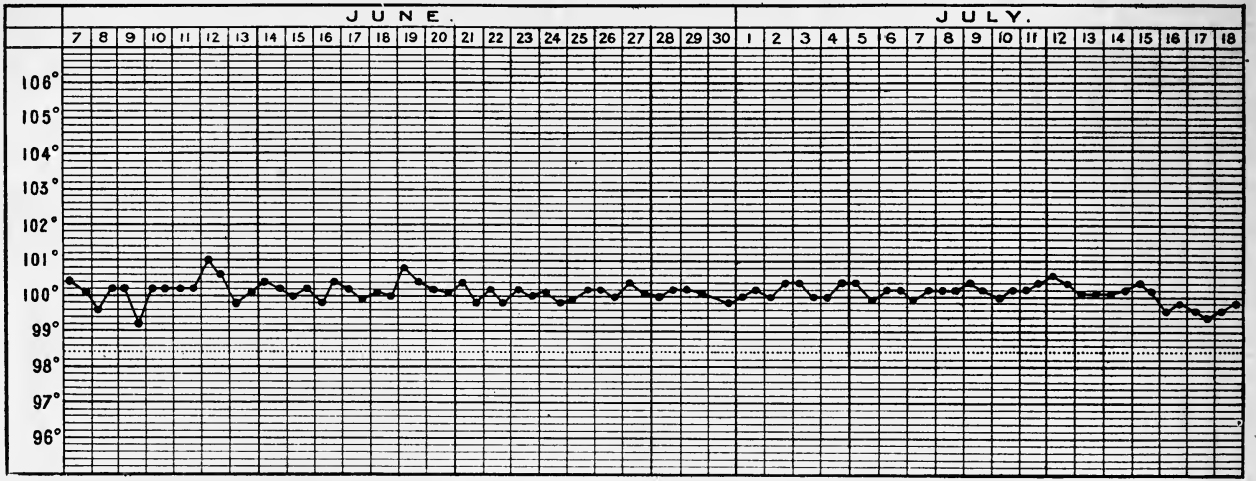

FIG. 14. Chart IV. Prolonged fever due to middle-ear catarrh. Chart showing slight fever which persisted for ten months at least, and probably for nearly a year, after otitis media, although ear symptoms had lasted only three weeks after the acute onset.

by the effect of dieting. I have already pointed out that dietetic measures sometimes seem to have a marked effect, but I must add that there are many more cases in which they seem to have none ; yet knowing how protracted digestive difficulty often is in childhood in spite of most careful dieting, I do not think that the failure of such measures to stop the fever necessarily disproves an intestinal origin.

However this may be, these cases always present a difficult problem ; one may feel almost confident that the yards of chart which are exhibited by an anxious parent, showing a regular evening rise to about 100° or 100·4°, have no serious significance and depend on nothing worse than some slight disturbance of function, perhaps digestive, in a nervous child ; and yet one hesitates to be too positive, for there are always present to one's mind possibilities of more serious disease.

I have long believed what the chart shown on p. 269 seems to demonstrate, that a latent catarrh in the middle-ear, without pain and without external discharge, is capable of producing slight daily rise of temperature for many weeks or months. It is difficult perhaps to differentiate in these cases between the part played by the naso-pharyngeal catarrh, with or without adenoids, which is so often associated with middle-ear disorder, and that played by the middle-ear affection itself ; but in the case from which Chart IV was taken there seemed to be no reason to doubt that the temperature was due to the ear.

The patient was a girl aged 8 months ; on March 13 she began to ail ; next day the temperature rose to 103° and the child was sick, the bowels were costive, the fontanelle was unduly full ; meningitis was feared, but no definite diagnosis could be made, although the temperature continued to rise daily to 101°–102°. On March 21 the mystery of the temperature was solved by a discharge from one ear, although the membranes had been examined and no sign of otitis had been detected. The drum of the other ear was then incised, and a little pus escaped. After two or three weeks all discharge from the ears ceased, the child seemed perfectly well in every way, but for ten months the temperature rose almost every day to 100°–101°. After this the record of the temperature was discontinued, for the child was in excellent health. But when the thermometer was used again, about the end of the child's second year, the irregularity of the temperature was found to continue. Gradually it settled down, and when I heard of the child some years later she was quite healthy. The chart shows a few weeks of the temperature about $2\frac{1}{2}$ months after the otitis media.

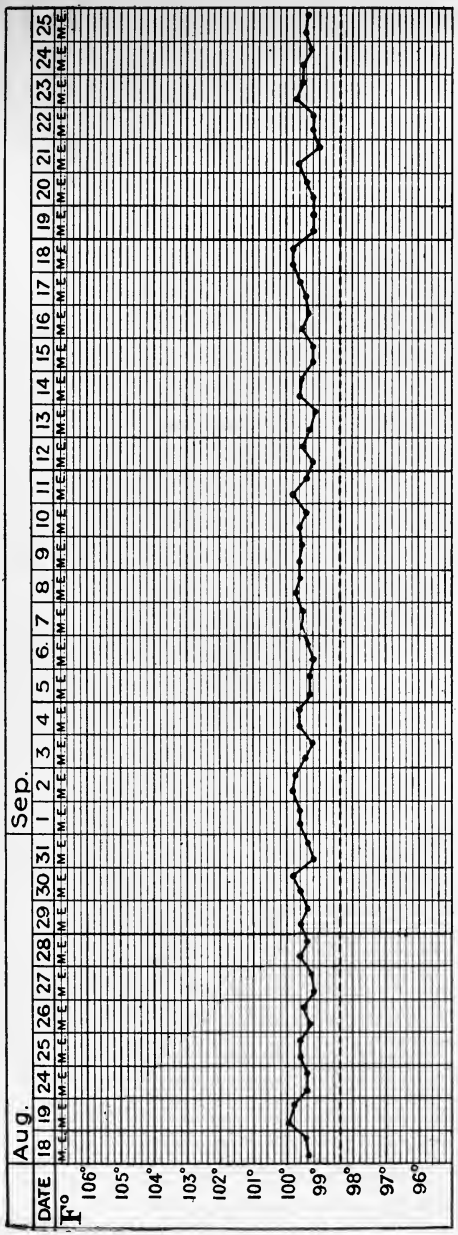

FIG. 15. Chart V. Prolonged slight fever which was still continuing after twelve months in a boy aged 7½ years with a palpable, hard, tuberculous gland in the abdomen.

I have seen several cases in which slight daily rise of temperature for many weeks or months was associated with obvious naso-pharyngeal catarrh, or had first been detected when the child had a slight sore throat ; the initial trouble had passed off, leaving the irregularity of temperature, which probably would never have been noticed had it not been for the sore throat at onset. In some of these cases there has been a complaint of transient pain in the ear at some time, which, although it was not followed by any evidence of acute otitis media, strongly suggested that there was a slight catarrh in the middle-ear, sufficient perhaps to explain the daily elevation of temperature.

In almost all these cases of prolonged fever the disquieting thought arises, Is there tubercle lurking somewhere ? No doubt many such a case finds its explanation in the old tubercular focus, which perhaps is only discovered in the post-mortem room years after the obscure ailment of childhood has been completely forgotten. Careful and repeated examination of the child, including perhaps the use of X-rays, is the only means of excluding this possibility, and even so we cannot arrive at certainty.

I remember a boy who came under my care at the age of about 6 years, a highly nervous boy with a stutter, whose temperature for months showed a daily slight rise for which no explanation could be found ; he was seen by several medical men, and one of them seemed for the time to have hit upon the explanation of the fever : he forbade milk, whereupon for a few days, but only for a few days, the temperature remained normal ; the intestinal origin of the trouble seemed the more probable as the boy suffered at various times with obvious symptoms of indigestion. I examined him thoroughly on many occasions without detecting any other disease, except that at one time I discerned, I thought, vague signs of enlargement of mediastinal glands ; after a time some glands became palpable in the abdomen, and for many months the enlargement of mesenteric glands remained evident. With long residence at the seaside the boy recovered, but it seemed clear that in this case there was tuberculosis.

The type of fever which commonly results from a caseous gland in the abdomen is well shown in the accompanying chart (Fig. 15, Chart V) from a boy aged 7½ years. During an attack of whooping cough at the age of six years it was discovered that his temperature was usually about 99° in the morning and 100° at night. At first the whooping cough seemed a sufficient explanation, but after the cessation of the whooping cough the temperature continued as before, and a few months later some pains

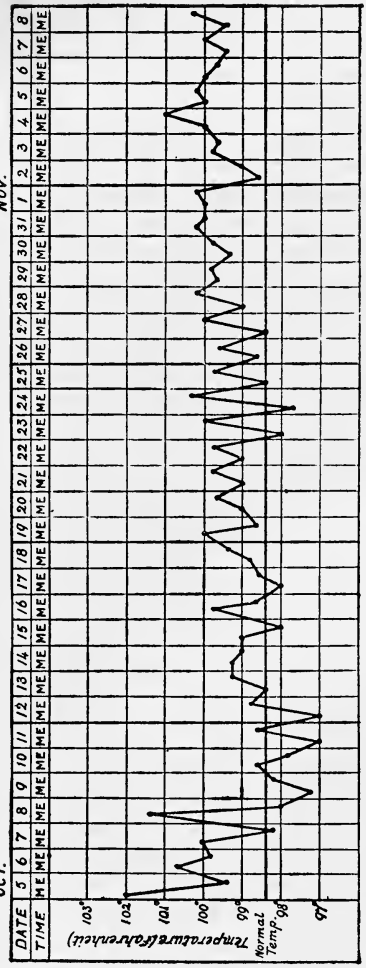

FIG. 16. Chart VI. Prolonged slight fever following an attack of acute rheumatism in a girl aged 6 years.

in the abdomen drew attention to a palpable gland about the size of a walnut just to right of the umbilicus : it was hard, and slightly tender on pressure. The slight elevation of temperature continued unaltered for more than a year, during which time also the gland remained palpable and hard, although the boy improved steadily in his general condition.

There is another cause for prolonged slight fever which is familiar to every one who sees much of acute rheumatism in children. The raised temperature is usually a sequel of active rheumatism in heart or joints.

Chart VI, taken from a case of acute rheumatism in a girl aged 6 years, illustrates this residual fever of rheumatism. The child was admitted to hospital on October 2 with acute pain and tenderness in the joints. Under salicylates the pain disappeared completely in about three days, but a systolic bruit appeared at the apex of the heart, and proved persistent. Apart from this the child seemed perfectly well, but for several weeks the temperature showed the slight elevation which is seen in the chart, and this continued in spite of free administration of salicylates.

Treatment. I am not considering now the treatment of cases in which there is some organic disease underlying the fever; these, of course, require such measures as are called for by the particular disease : sea air and cod-liver oil for tubercle, salicylate for rheumatism, and so on; but in the cases where no such explanation is forthcoming, and we are driven to conclude that the temperature is due to functional disturbance only, what should be done ? I have seen a great variety of methods of treatment tried, and I must say, with remarkably little effect; antipyretics usually have no effect whatever, or if they lower the temperature for a day or two, it rises again directly they are discontinued. I have tried, or seen tried, all sorts of antipyretics ; I know of none that is of any permanent benefit in these cases ; quinine, given in a dose of 2 grains once a day to a child about 5 or 6 years old, has seemed to have some effect in reducing the temperature. I am much more inclined to use such measures as will ensure as far as possible a healthy condition in the intestinal tract : keeping the bowels working regularly, and especially adjusting the diet so as to eliminate those foods which one knows from experience are most apt to cause intestinal irritation in children (see Chapter on Indigestion).

I have tried creosote on the principle of intestinal antisepsis, but with no definite success, and, as in other conditions, one has had to discontinue it before long on account of its interference

with appetite. In view of the nervous excitability of these children, small doses of arsenic may be useful given with potassium citrate, which often seems to do good where there is mucous catarrh in the intestine, or with an ordinary stomachic mixture of bicarbonate of soda with spirit of chloroform. The possibility that there may be some latent catarrh in the middle-ear, or an unhealthy condition of the naso-pharynx must specially be borne in mind in the treatment of these cases. I am fond of a nasal spray for such cases, half a drachm of oil of eucalyptus in an ounce of paroleine makes a useful spray; or an alkaline lotion of Bicarbonate of Soda gr. v, Sod. Chloride gr. iv, Borax gr. x, Glycerin ℥j, Aq. ad ℥j, may be sniffed up the nostrils and spat out through the mouth twice a day; half an ounce of this to be placed in a little two-ounce medicine tumbler, which is filled nearly to the brim with warm water; the child is directed to try to drink it through his nose, and spit it out through his mouth; this is managed well by some children about 7 or 8 years old; for younger children the spray is easier, and is not dreaded as is syringing. Possibly politzerising may be advisable when there has been complaint of pain in the ear.

A question which has to be faced is the advisability or otherwise of keeping the child in bed. In some cases the temperature is certainly higher when the child gets up, but I have known exactly the reverse. In one case it seemed clear that the fever was kept up by too much recumbency with a liberal diet, for it was reduced at once and kept down by allowing more exercise and curtailing the excessive feeding. Probably the wisest plan is to decide this question by experiment, and not to be guided only by the temperature but also by the general condition of the child; if getting up while the temperature is raised leads to complaint of feeling tired, of headache, or of general unfitness, no doubt it is better to keep the child in bed or let the child rise late and go to bed early; but, as a rule, I think these cases can be allowed up without harm; and sometimes I have advised disregard of the temperature altogether and taking the children to the seaside; this may be specially advisable where there is reason to believe that the irregularity of temperature arises from some catarrh in the naso-pharynx or ear, a condition which is very apt to be perpetuated by a cold, foggy atmosphere such as that of London in the winter.

High fever due to latent otitis media. It is, I think, not sufficiently recognized that very high fever may be produced in a child by acute inflammation of the middle-ear without any

symptom beyond the temperature to suggest ear mischief, and particularly *without pain*; a purulent discharge may be the first indication of the cause of the fever. I have repeatedly seen this happen where the suggestion of otitis media had been met at once with the objection, 'But there has been no pain.' It is worth remembering that the absence of pain by no means disproves the presence of acute suppurative otitis media; in an infant it is often difficult to be sure whether there is pain or whether his crying is due only to the fretfulness of illness, and even if the cry seems to indicate pain it may still be difficult to know whether it is in the ear or in some other part; but whether

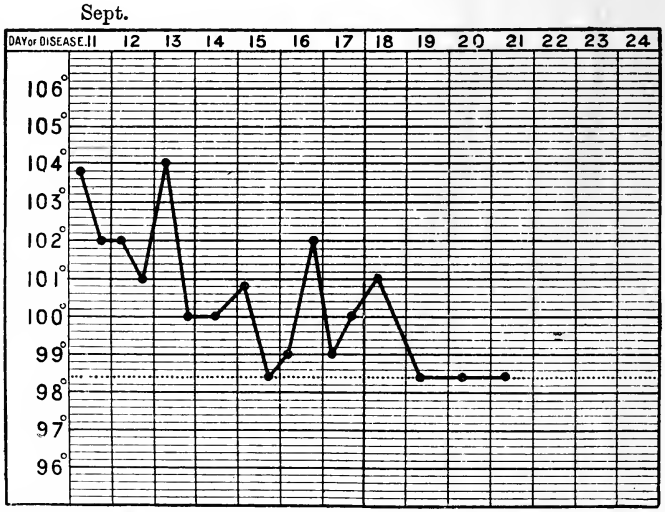

FIG. 17. Chart VII. Fever due to acute otitis media. Boy aged 15 months; no pain or tenderness; fever unexplained until discharge from ear.

we can be sure of pain or tenderness over the ear or not, the possibility of ear mischief as a source of fever is to be borne in mind.

The following case, from which Chart VII is taken, illustrates the difficulty of diagnosis in these cases.

A boy, aged 15 months, had been sick on August 28 and feverish. After a dose of castor oil he improved but soon became feverish again; he was under the care of Dr. Cyril Cheatle, with whom I saw the boy on September 13. There was some enlargement of glands at the angles of the jaw on both sides, but with no definite tenderness; the tonsils were considerably enlarged,

but there was no sign of recent inflammation. There was no tenderness on pressure over or behind the ears. The possibility of otitis media was suggested, but no positive diagnosis could be made. On September 18 I heard that the child had improved considerably in general condition, being brighter and sleeping well, but the fever remained a puzzle. On September 19 a purulent discharge appeared from the left ear and the temperature became normal. Throughout the fever the child appeared to have had no pain in spite of the acute otitis media.

In older children who can describe their own symptoms, the certainty of absence of pain in the ear does not exclude this source of fever; for instance, I had in my ward a very intelligent little girl aged 5 years who had been convalescent from pneumonia, and whose temperature had been normal for several days, then the temperature suddenly rose to 103·6° without any apparent reason; within a few hours a discharge appeared from the left ear, and the temperature became normal the same day; the child stated that she had had no pain whatever.

One might think that the diagnosis could be settled easily enough by examining the ear; I have no doubt that it could be in some cases, but I am satisfied that the aural speculum even in the hands of expert observers may fail to discover the cause of the temperature in such cases. I have more than once had the ears examined by an aural expert, who has found no evidence of otitis media, and within twenty-four hours after the examination there has been profuse discharge from the ear. It would seem, therefore, that the absence of visible alteration in the membrana tympani does not necessarily exclude otitis media as a cause of fever.

Acute primary pyelitis. In infants especially, but occasionally also at any period of childhood, there is another source of high fever which is very apt to be overlooked, namely, acute primary pyelitis. I shall not enter into any detailed account of this disease here, as I shall describe it fully in a later chapter (chap. xl).

Briefly stated the clinical picture is this: a female child acutely ill and in considerable distress, although with no definite evidence of localized tenderness or pain anywhere, is found to have a high temperature, perhaps 105° or even higher, and nothing whatever to explain the fever until the urine is examined. The onset is sudden, and often there has been some shivering or an attack of blueness and collapse during the first day or two of the illness.

In the case from which Chart VIII is taken, a female infant, aged 14 months, whom I saw with Dr. Owen Lankester, the illness began on September 5:

the child had been quite well up to that day, but whilst being dressed in the morning 'she went blue, and seemed cold and faint'; the temperature was found to be 104°; but nothing was found to explain it. During this early stage of the illness, the nurse noticed once or twice that the child's 'chin worked as if with shivering'. The fever continued, but beyond some fullness of the abdomen, and fretfulness of the child, no symptoms were found and the condition remained a puzzle until the child, who had been taken ill while away at the seaside, was brought home on September 21, and placed under Dr. Lankester's care; the urine was then obtained and found to be acid, with sufficient pus to make it just turbid, it had the peculiar heavy odour which is noticeable when the urine is infected with *Bacillus coli*, and a bacillus was found in it which appeared to be this micro-organism. On September 24, treatment with potassium citrate was begun, the temperature became lower, and after a few irregular rises, remained normal. Urotropin seemed to improve the condition of the urine in this case, but the potassium citrate, for which it was substituted for some days, had more effect in reducing the temperature.

The fever is continuous and lasts for many days unless the cause is recognized and properly treated; recognition is only possible if the urine be carefully examined, and hence it is that the fever is so often unexplained. It is not always easy to obtain the urine of an infant for examination at once, especially if the infant be a girl, as is almost always the case in this disease; but a trained nurse, if the importance of the urinary examination is realized, will usually succeed in obtaining a specimen. It should be pointed out that even a few drops of urine are sufficient for the examination, for it is microscopic not macroscopic examination which is necessary.

As might be expected with a very high temperature, nervous symptoms may be prominent, so that these cases of acute pyelitis are often mistaken for cases of meningitis. There are many infants who with any considerable rise of temperature readily develop slight twitching of the face and eyes, or limbs, which may pass into general convulsions with loss of consciousness, but with acute pyelitis the nervous symptoms are occasionally so severe as to simulate gross cerebral disease very closely. An infant, Marjory S., aged 11½ months, whose temperature is shown in Chart IX, had been ailing on September 14, when the bowels were costive; on September 16 the temperature was found to be raised and fever continued as shown in the adjoining chart. On September 17 and 18 the child was drowsy and twitching slightly; vomiting occurred once on September 17, but it was only after a calomel powder. I saw the child in consultation on September 20; the child had fallen into a semi-comatose condition that day, she took no notice of my hand passing close to her eyes; there was thought to be a very slight internal squint of the left eye,

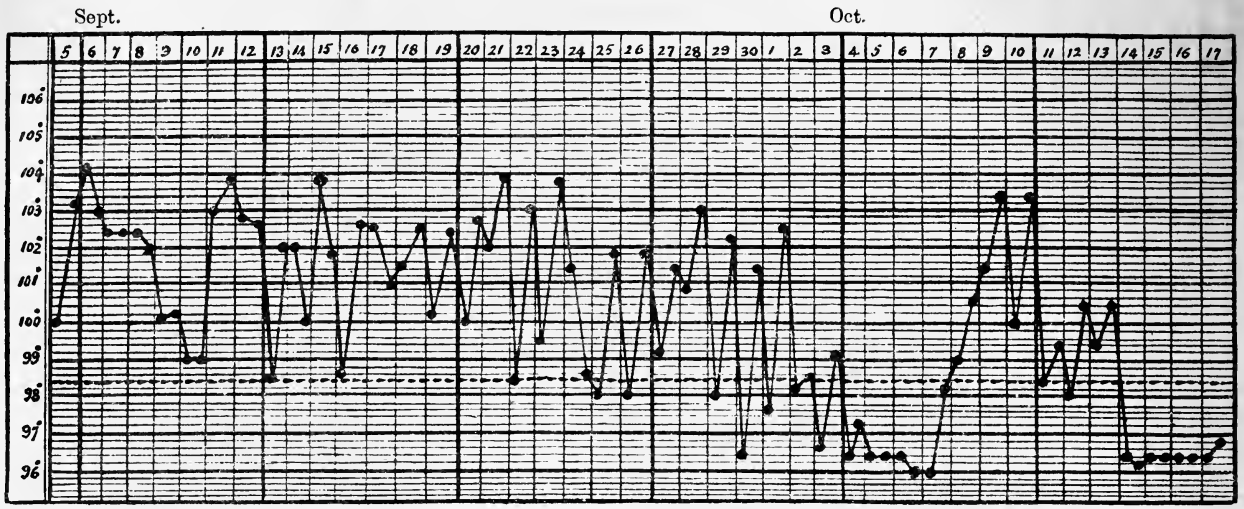

FIG. 18. Chart VIII. Acute primary pyelitis in infancy, showing prolonged high fever of remittent and intermittent type: unexplained until discovery of pus in urine due to *Bacillus coli* infection in female infant aged 14 months.

the neck was slightly stiff, and there was a question whether the left leg was moved quite as well as the right; whilst being observed the child gave a short cry and threw its body into a position of opisthotonos. The child was evidently extremely ill, and the advisability of lumbar puncture was discussed as a diagnosis of tuberculous meningitis had been suggested. But there was much against this view: the onset had been too sudden, the temperature was too high, and the child's stuporous semi-convulsive condition made it difficult to be sure whether

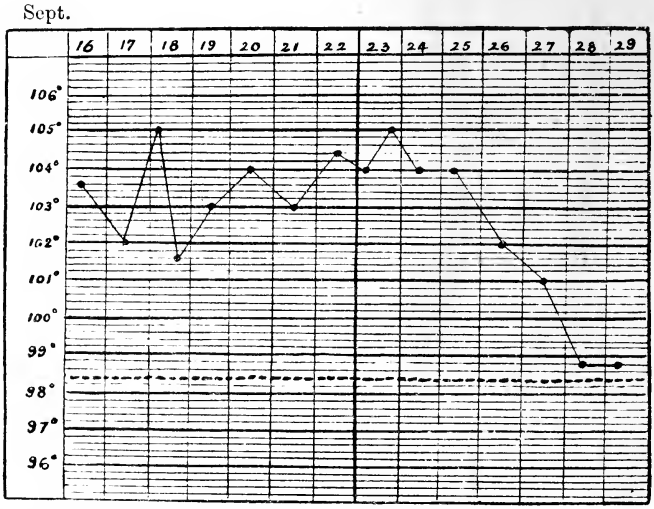

FIG. 19. Chart IX. Acute primary pyelitis in infancy, showing prolonged high temperature which was associated with nervous symptoms due to pyelitis in a female infant aged 11½ months.

the nervous symptoms, which were all ill-marked, really indicated any gross lesion of the nervous system: the possibility of acute pyelitis was therefore suggested, and it was decided not to do lumbar puncture, but to get the urine as soon as possible. The next day I heard that there was some vomiting, respiration was irregular, there was conjugate deviation of the eyes to the left, the left arm and leg showed some rigidity, and the doctor added, 'Can't get urine,' and the following day there was thought to be slight ptosis of the left eye and a transient weakness of the right side of the face. On September 24 the urine was at length obtained, and was found to contain about twelve pus corpuscles per field under a high power. The child was treated with potas-

sium citrate, the temperature speedily fell to normal, and the child made a good recovery.

The importance of recognizing acute pyelitis will be appreciated by those who have seen the effect of treatment. It is a condition which, when undiagnosed, drags on for days and weeks with high fever and severe constitutional disturbance. It is a grave danger to the child, for life may be sacrificed through failure of adoption of the proper treatment, whereas a timely recognition of the disease and a skilful administration of the requisite drugs is followed by speedy subsidence of the fever and by complete recovery. There is hardly anything more striking in the field of therapeutics than the effect of proper treatment upon acute pyelitis in infancy. This will be described in detail in chapter XL.

Dentition, gastro-intestinal disturbance, constipation, as causes of fever. I have already expressed my opinion that the worry of dentition is sometimes the sole cause of fever in infants : I know only too well that this is dangerous doctrine, for it is liable to be abused ; but none the less it is, I think, a fact, as well proved by experience as most scientific observations in clinical medicine, that in some infants the temperature will rise to 102° or 103° when a tooth is nearing eruption, and will fall to normal within a few hours after the tooth is cut, and this without any other ascertainable cause for the fever. Lancing the gums is out of fashion nowadays, and probably deservedly so, for no doubt far more mischief and pain was produced by indiscriminate gum-lancing than was caused by the dentition which it was supposed to assist, but although I have never personally had the courage of my conviction to proceed to gum-lancing, I strongly suspect that there are cases in which the fever could be stopped speedily by a judicious lancing of the gum when the tooth is at the point of eruption, and unless most of the writers of half a century ago were extremely bad observers, their experience proves that this is so.

Since these remarks were written, Dr. Lovett Morse,[1] of Boston, has recorded two cases which illustrate well the production of fever by teething and the relief of symptoms by gum-lancing. The first I quote in Dr. Morse's own words :

A boy, 17 months old, began to be fussy and a little feverish the night of April 14. The next evening the temperature was 103° F., the next day 100° F. in the morning, and 104·8° F. in the evening. On the morning of the 17th the temperature was 100·8° F., the pulse 140, and the respiration 28. There

[1] *Boston Med. and Surg. Journ.*, July 9, 1908.

had been no symptoms beyond the fever except restlessness and sleeplessness. Careful physical examination, including the ears, showed nothing abnormal beyond marked swelling of the gums over the first four molars. The gums were lanced over all. The temperature dropped to normal during the day and did not rise again. No other treatment was given.

The other was a girl of 3 years, who had always had some disturbance with dentition. Whilst cutting one of her second molars she had a temperature of 100° to 102·5° F. for twelve days with no other symptom except occasional vomiting. Beyond the swollen gum over the tooth nothing was found, the Widal test was negative, the urine normal, there were no physical signs. Gum-lancing produced a temporary fall of the temperature, and was done several times; finally a large piece of the gum over the tooth was removed and the tooth came through, the temperature immediately fell to normal and remained so. With each of the other second molars and in less degree with the six-year-old molars there were similar symptoms.

In the absence of serious symptoms, such as convulsions, it has not seemed to me necessary to resort to gum-lancing, inasmuch as the fever does little or no harm in itself, being of quite short duration; moreover, it may be amenable, as other dentition symptoms often are, to treatment with small doses of phenazone, to which may be added usefully an aperient dose, a drachm or more, of fluid magnesia or a grain of grey powder.

The presence of undigested food in the intestine is enough to raise the temperature considerably in some infants and also in some older children: so also is a bout of constipation in the child who is not habituated to it. It is in such cases that a dose of castor oil $\frac{1}{2}$–1 drachm for an infant and 2–4 drachms for an older child, produces rapid improvement; calomel also is good in these cases, $\frac{1}{2}$–1 grain at night according to the age of the child.

In illustration of the production of high fever by an irritant in the intestine I may mention here a curious case which was under my care in the Children's Hospital, Great Ormond Street. The temperature is shown in Chart X. Rose O., aged $3\frac{3}{4}$, was brought with a history that for three weeks she had waked every night screaming for about four hours, and put her hands to her head as if in pain. She was kept in hospital thirty-eight days and, as the chart shows, there was a considerable rise of temperature nearly every night; the child screamed for one to three hours while the temperature was raised, and usually vomited when the temperature was at its highest. The child during the daytime was rather pale and quiet, otherwise nothing

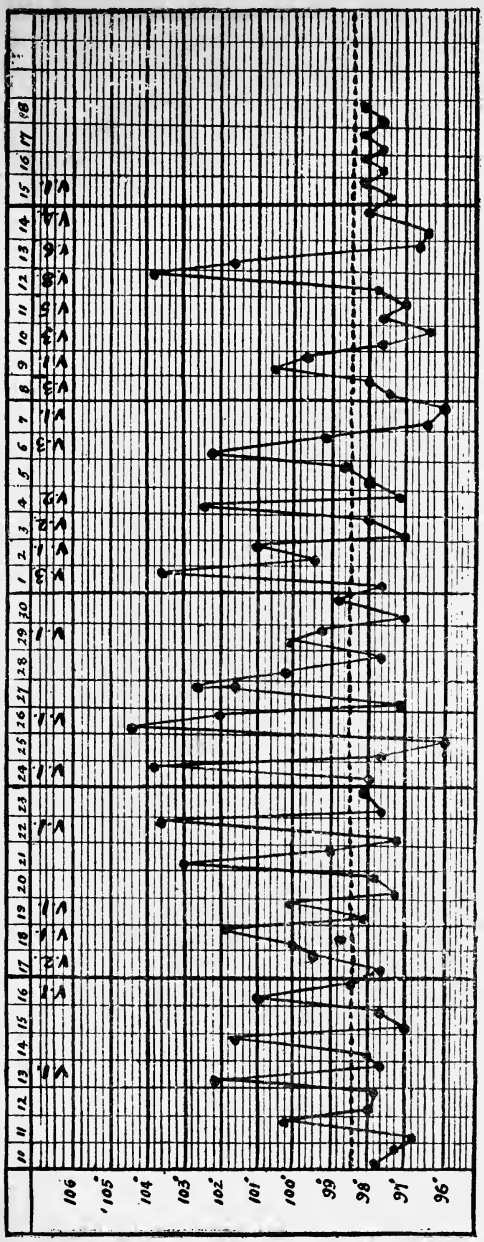

FIG. 20. Chart X. Prolonged fever due to Roundworm (*Ascaris lumbricoides*). Intermittent type of fever, probably of eight weeks' duration, with nightly vomiting and screaming: fever unexplained until one roundworm passed, when all symptoms ceased.

abnormal could be found except that on several occasions there was acetone in the urine ; the child sat up and took some interest in her surroundings. She was seen by several of my colleagues and others, and various diagnoses were proposed, mostly leaning to gross cerebral disease of some sort. Various lines of treatment were tried without apparently the least effect on the temperature, the vomiting became worse and the child began to look more ill. On the thirty-fifth day of her stay in hospital the child's temperature became normal and steady, the next day the vomiting ceased, and two days later the child passed a dead roundworm and remained perfectly well.

Inflammatory conditions in the throat. It is a good rule that in every case of unexplained fever in childhood the throat should be examined. In infancy and early childhood there may be nothing whatever to direct attention to the throat, which may nevertheless show an acute follicular tonsillitis, or patches of diphtheria, accounting for an acute onset of fever.

I have already mentioned the probable relation of naso-pharyngeal catarrh to the prolonged slight fever which is often seen in nervous children, and I strongly suspect that recrudescence of pharyngeal catarrh with or without adenoids is responsible for some attacks of 'recurrent fever' in children (vide p. 265). I have seen several cases which seemed to support this view.

Blood Diseases. In any case of prolonged fever without apparent cause, the possibility of some blood disease must be borne in mind. In the early stage of some of the most severe blood diseases—for instance, acute lymphatic leukæmia—there may be no noticeable degree of anæmia, and beyond some malaise and the seemingly inexplicable fever there may be nothing to be found until the blood is examined. Such a case as the following is likely to cause difficulty in diagnosis, and, as actually happened, the difficulty may remain unsolved for weeks :

Gerald Z., aged 10 years, five months ago had a septic adenitis in the left axilla. This subsided, and the boy apparently recovered completely within a fortnight, and remained well until two months ago, when urticarial rash occurred, with constipation and a temperature of 103°. The rash passed off, but the temperature continued. A consultant was called in, and the condition was thought to be ' food fever '. In spite of dieting the high temperature continued. Another consultant saw the child, and whilst not approving the dietetic theory was unable to find any cause for the temperature. With continued high fever the gums now became swollen and ulcerated, and a third consultant suspected some scorbutic affection, and fruit juices were given. The fever, however, persisted, being often 103°, and only falling to 100° in the mornings, and by this time, after eight weeks' illness, the child was becoming wasted and markedly anæmic; moreover, slight enlargement of superficial

glands in several parts of the body suggested the advisability of a blood count. The red cells were found to be 3,312,500 per cub. mm., and the white, 103,300 per cub. mm.; the differential count showed polymorphonuclears 2·8 per cent., lymphocytes 92·6 per cent. The case was in fact one of acute lymphatic leukæmia.

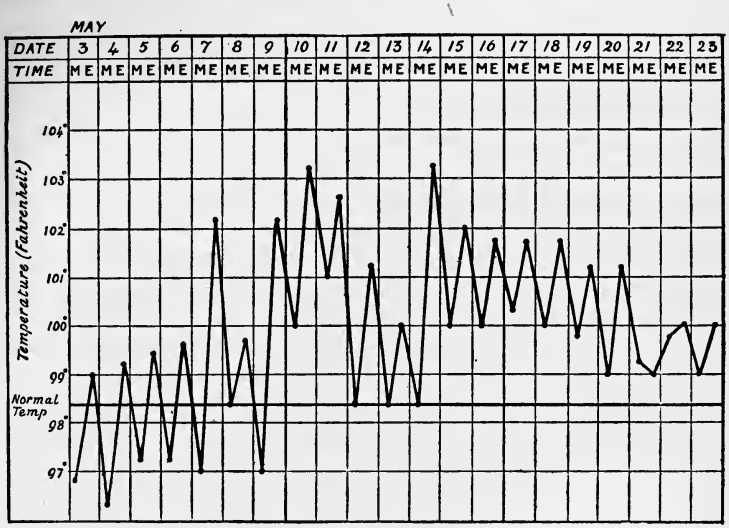

FIG. 21. Chart XI. From a case of acute leukanæmia in a child aged 5 years.

The temperature (Chart XI) in the case of a child aged 5 years and 10 months would have been hardly less puzzling than in the case just described had it not been for the prompt examination of the blood made by the doctor in charge, who found a condition corresponding with that which has been described as acute leukanæmia.

CHAPTER XX

INTESTINAL WORMS

The presence of threadworms in the intestine is a common trouble in childhood, and often very difficult to cure. In 200 necropsies, as far as possible consecutive, on children under the age of twelve years at the Children's Hospital, Great Ormond Street, the intestine was carefully examined for the presence of threadworms, and these were found to be present in thirty-eight cases, that is, in 19 per cent. When only children over the age of two years were included, the percentage was much higher; in a series of 100 necropsies on children between the ages of two and twelve years, threadworms were found in thirty-two cases, that is, in 32 per cent.

The point, however, on which I wish to lay special stress is not the frequency of threadworms, which is familiar enough, but their situation. It is often stated that the threadworm inhabits chiefly the rectum and lower part of the colon and occasionally the cæcum, but the fact that the vermiform appendix is a favourite habitat of the threadworm has received but little attention.

In no fewer than twenty-five out of the thirty-eight cases in which the oxyuris vermicularis was present the worm was found in the appendix vermiformis; in other words, the threadworm was found in the appendix in two-thirds of the cases.

Moreover, the appendix is in some cases the only part in which the threadworm is found; in six of the thirty-eight cases mentioned no threadworms were found either in the cæcum or elsewhere in the large intestine, but in each of these six cases threadworms were found in the appendix, and sometimes at its extreme tip.

The number of threadworms in the appendix is sometimes very large, thus in one case described below, 111 were present here; and it is noteworthy that these large numbers may be found in the appendix, when only very few or none are present in the large intestine.

A further point which may be of significance, is the immature character of the worms found in the vermiform appendix. In some cases only one or two of the worms found here were

full grown, the rest were extremely minute, measuring 1·5 to 3 mm. in length, and requiring the use of a lens for accurate counting. These minute worms had already acquired the characteristic differences of sex, from which it could be determined that many, probably the majority, were immature females, though some certainly were males. It has been stated by Blanchard [1] that the embryo when first hatched measures in the case of the female 1·7 to 1·97 mm., and in the male 1·1 to 1·8 mm., whereas the full-grown female measures 9 to 12 mm., and the male 3 to 5 mm., in length. Many therefore of the minute threadworms found in the vermiform appendix must have been hatched quite recently.

This observation seems to throw considerable doubt upon the view held by Leuckart and others, and now current in the text-books, that the oxyuris vermicularis never multiplies in the intestine; it is absolutely necessary, they say, that the ova should be passed and reintroduced into the mouth, and so into the stomach, in order that they may be hatched; in other words, every single threadworm passed from the bowel has been introduced as an ovum from without into the alimentary tract.

Were such a view correct, it would be difficult to understand why threadworms are so rarely found in the small intestine. The embryo is said to go through the earliest stages of growth here after being hatched in the stomach, and by the time it comes to the large intestine it has reached, or almost reached, maturity. If this be so, then considering the enormous number of threadworms which are passed by some children for months or even years, and how variable must be the rate of passage of each of these worms through the small intestine, one would surely expect that the young threadworms would frequently be found at necropsies still in the small intestine, whereas, as a matter of fact, this very rarely happens. I have found young threadworms once as high as 45 inches above the ileo-cæcal valve, and two or three times I have seen mature worms either just above the valve or in the last few inches of the ileum, but such a finding has been very exceptional in my experience.

Further, this hypothesis does not explain the immaturity in some cases of almost all the threadworms found in the appendix, for even supposing that owing to the variability of rate of transit through the small intestine the degree of maturity varies in those

[1] *Zoologie Médicale*, Paris, 1889, i, p. 713.

that have reached the large intestine, it is nevertheless hardly conceivable that a large number of very immature threadworms, hardly larger than those just hatched, should happen to congregate in the vermiform appendix, whilst the great majority of those in the cæcum are almost or quite full grown.

The presence, therefore, of numerous very young threadworms in the vermiform appendix, associated with only a few full-grown ones, seems to show that at least in some cases the vermiform appendix serves as a breeding-place for the oxyuris vermicularis. It might be added that although there is experimental evidence to prove that the swallowing of the ova is followed after a few weeks by the appearance of threadworms in the fæces, the proof that every threadworm that is passed from the bowel has been introduced in this way is yet to seek. According to the accepted view we are asked to believe that a child who for months, and sometimes years, passes scores of threadworms daily in its stools, has introduced into its mouth as an ovum every single one of these worms. To support this theory it is necessary to suppose either that the ova are so widely distributed from the fæces that they are re-introduced in various articles of food, or else that they are introduced by the fingers directly from the anus. Both these sources are supposed to contribute to the supply.

But if the ova were distributed in such enormous numbers that one child could swallow hundreds or possibly thousands of them in articles of food it would be difficult to see how any person in an infected house could possibly escape being infected also. It happens indeed not very rarely that more than one child in a family is suffering from threadworms ; but this does not happen with sufficient constancy to justify the assumption that such a source is the usual or even a common one.

If again the fingers be the source, then considering the number and persistency of threadworms in the fæces one can only suppose that the number of ova on the fingers must also be very large and easily demonstrable. With the object of investigating this point I selected five children who were passing large numbers of threadworms and gave orders to the mother that the hands were to be left unwashed after the night's rest. The dirt on the finger ends and under the nails of all the fingers was then scraped off and examined microscopically, in three of the cases on two occasions, in two of them once.

The result of the examination was that one solitary ovum was found in the dirt under the nail of one child out of the five.

Further investigation would of course be necessary before one could make any definite statement on this point, but this observation so far as it goes seems to show that the dirt on the fingers is extremely unlikely to supply ova in sufficient numbers and with sufficient constancy to keep up the continual discharge of enormous numbers of threadworms from the bowel. In the one ovum found under the nail a fully-formed embryo in the tadpole stage was present, and one cannot doubt that if this had been swallowed—and it is to be remembered that even when the ova do happen to be present on the fingers it is by no means a certainty that they will be swallowed—the embryo would in due time have become a mature threadworm in the intestine.

That a certain number, possibly even a large number, of threadworms are introduced as ova into the mouth from one or other of the sources mentioned is not improbable, but the observations which are here recorded suggest, I think, that the oxyuris vermicularis also multiplies in the intestine and that the appendix vermiformis is in some cases its breeding-ground.

On this view, the extreme obstinacy of those cases in which, in spite of all treatment, threadworms are discharged in the fæces for months or years becomes at once intelligible. The difficulty of dislodging threadworms from the tip of the appendix, where they are not uncommonly found, is sufficiently obvious, and one can well imagine that a few female worms maturing here would keep up a continual supply of young threadworms, many of which would gradually find their way out into the cæcum and so pass down with the fæces.

So many of the ailments of childhood have been attributed to 'worms', some with good reason and some with none, that one hesitates to add another to the list. I wish, however, to draw attention to a condition which might almost have been foretold in the light of the previous observations on the presence of large numbers of threadworms in the vermiform appendix.

A boy, aged nine, was admitted into the Hospital for Sick Children under the care of Dr. Lees for acute rheumatism and pericarditis. Four weeks before death he complained of pain in the right iliac fossa. Pericarditis and probably pleurisy being present, the boy was questioned carefully as to the situation of the pain in view of the possibility of the pain being from diaphragmatic pleurisy, and being described, as so often happens with children, with no strict accuracy. The boy, however, who was very intelligent, persisted in localizing the pain definitely in the right iliac fossa. This part was, therefore, particularly examined, but no abnormal resistance and no tenderness could be detected. The pain passed off without further evidence of appendicitis.

Post mortem acute pericarditis and pleurisy were present. The whole of

the colon was minutely examined with the naked eye, and the mucus in the cæcum scraped off with a knife and examined microscopically, but only in the latter situation were two threadworms present. The vermiform appendix appeared thicker than normal, as if swollen, and this was due to actual thickening of its wall, not to distension of its lumen. The mucus here also was examined microscopically. In the proximal inch not a single threadworm was found, but in the remaining $2\frac{1}{4}$ inches at the distal end no fewer than 111 threadworms were counted, nearly all of which were very small ones, many of them measuring scarcely one-sixteenth of an inch (about 1·5 mm.), while some of the mature worms present contained large numbers of ova.

This case may, I think, be taken as evidence that pain in the right iliac fossa may be associated with a catarrhal condition of the appendix as a result of threadworms.

The discovery of this association seemed to throw some light on another case which was of very uncertain nature.

A boy, aged nearly six years, was admitted to the Hospital for Sick Children, under the care of Dr. Lees, for abdominal pain and vomiting. The pains in the abdomen had been present several months; the vomiting had only been present four days. The bowels were regular. The child did not look particularly ill. He complained of some pain in the right iliac fossa; the abdomen was quite lax. In the right iliac fossa there was some vague thickening, the nature of which was uncertain; it was thought to be due either to inflammatory adhesions or to enlarged glands, but the child allowed free palpation here, and the presence of any tenderness was doubtful; the hip was extended freely; the temperature was 100·2°. I thought at the time that the case was one of appendicitis, but was puzzled by the extreme mildness of the symptoms. These disappeared in a few days, and the boy was discharged apparently well. Unfortunately, no inquiry was made at that time as to the presence of threadworms in the stools. Within the next few months the boy was found to be passing large numbers of threadworms continually, and one or two roundworms also, and on inquiry there was reason to believe that he had been suffering from worms before his stay in the hospital. Frequent vomiting, night terrors, and fits occurred, and the relation of these symptoms to the passage of threadworms was very striking. The mother, an intelligent observer, herself noticed that each time the threadworms diminished as the result of treatment the symptoms diminished, and the boy was better in every way, only to relapse again as the threadworms reappeared. The threadworms persisted in spite of all treatment for two years.

It seems probable that in this case, as in the preceding, the symptoms may have been due to the presence of threadworms in the appendix, which would also account for the obstinacy of the case if the view I have suggested be correct. That pain in the right iliac fossa was due to threadworms in the appendix has seemed possible in other cases, but it can only rarely happen that actual evidence can be obtained as in the first case mentioned. There was, however, ample evidence in the series of 200 cases examined post mortem that the presence of threadworms may

cause swelling and thickening of the vermiform appendix, especially when the worms are present there in large numbers. Such a condition was found several times, and, indeed, it was sometimes possible to predict the presence of threadworms in the appendix from its swollen appearance before opening it. The following cases may serve as typical instances.

W. M., aged 2½ years; empyema, suppurative pericarditis, and commencing peritonitis. The vermiform appendix had a markedly swollen and thickened appearance; the rest of the intestine appeared quite normal. The appendix was found to contain twenty-nine threadworms, nearly all of these being in the distal inch; of this number only five were full-grown females containing ova; the rest were very minute, measuring from about 3 mm. upwards; many of them were females, but some were males. Only four threadworms, all full grown, were found in the large intestine; no young ones were seen with the naked eye, nor in portion of the mucus examined with the microscope. One full-grown female was found 4 inches above the ileo-cæcal valve.

J. L., aged 3¾ years; tuberculous meningitis. The vermiform appendix had the typical swollen appearance suggesting at once the presence of threadworms; it was, therefore, examined carefully. In the distal ½ inch, close to the tip, twenty-six were found; in the rest of the appendix thirty-three, or forty-three including ten at the mouth of the appendix—that is, sixty-nine in all. Most of those in the appendix were very young females. The mucus of the lower part of the ileum near the ileo-cæcal valve was examined microscopically, but showed no ova and no embryos. There were numerous threadworms in the colon and rectum.

A. A., aged 4 years; spinal caries, tuberculosis. The vermiform appendix was much swollen—'nearly as thick as one's little finger'. The appearance suggested the presence of threadworms. The mucus from the appendix was examined with a low power of the microscope, and showed forty-three threadworms, chiefly very minute young females. There were many threadworms in the cæcum, but only a few in the rest of the colon.

The colicky pains which are associated with the presence of threadworms are not limited to the cæcal region, often they are vaguely referred to the lower part of the abdomen or the umbilical region. The occurrence of colic is not surprising, for where there are threadworms there is usually more or less mucous catarrh of the intestine; in some cases I have found the colon coated with a thick layer of jelly-like mucus; no doubt the intestine in its efforts to pass onward this tenacious material is excited to the excessive peristaltic movements which are felt as colic.

There is another result of this mucous flux in children with threadworms, namely, wasting. Many a child who is suffering only with these worms is brought to the medical man for wasting which perhaps is sufficient to arouse fears of some tuberculous disease. If the function of the large intestine, especially at its

lower part, is largely absorptive as physiologists tell us, it is natural enough that a thick layer of viscid mucus over the surface of the mucous membrane should interfere with nutrition by diminishing absorption.

Next to the wasting and colicky pains which are the most frequent symptoms of threadworms, nervousness is probably the commonest result of their presence. There is nothing specially characteristic in the nervous symptoms due to threadworms, they are just those which a child who is specially nervous from any cause may show, but the degree of nervous instability is often very striking. The child becomes unnaturally timid, sleeps badly, grinds his teeth, has night terrors and wakes with a headache in the morning or with nausea. I have seen fidgety, almost choreiform movements, which subsided almost completely when the worms were expelled; I have also seen habit-spasm arise in association with threadworms.

The occurrence of squint as the result of threadworms is, I think, a fact. I was consulted about a girl of $3\frac{1}{2}$ years, whom I had seen several times previously and knew to have been free from squint. She had now become extraordinarily excitable, and within five weeks had developed a very marked convergent squint, sometimes in one eye, sometimes in the other. No explanation of the symptoms was forthcoming until, a few days after I saw her, threadworms began to appear in the stools.

I have mentioned above a case in which fits occurred and were markedly influenced by treatment of the threadworms; I have seen other cases in which convulsions seemed to be due to threadworms.

Enuresis is certainly related to the presence of these worms in some cases, possibly owing to direct irritation when they reach the rectum as they sometimes do, especially at night, but probably far more often through the general increase of nervous instability which worms somehow produce and which would specially favour the occurrence of enuresis.

There is a popular idea that nose-picking is specially indicative of worms, but I know of no evidence that this is so: the nervous child is undoubtedly more prone to fall into abnormal habits or 'tricks' than other children, and in this way the child with worms may be rather more likely to form such a habit than another child; but certainly nose-picking is of no value for diagnosis.

Threadworms sometimes cause much itching at the anus, which is very distressing to the child at night—and not at night only.

I have seen numerous threadworms wriggling at the anus of a child in the daytime. I have also seen them in the vulva, where they may cause much irritation.

It is sometimes thought that there is a particular facies from which the presence of worms can be diagnosed : it is a pale face, puffy under the eyes. There is no doubt of the association, but the same appearance is seen with chronic intestinal indigestion, 'mucous disease,' which has so many other symptoms in common with worms ; indeed, I doubt if there is any symptom produced by threadworms which may not also be produced by intestinal dyspepsia with mucous catarrh of the intestine without the presence of these worms.

Threadworms when present only in small numbers are easily overlooked in the stool by those who are not familiar with their appearance ; on the other hand, various fibres are mistaken for worms. A child was brought to me who was said to be passing 'hundreds of worms' ; I saw the stools, which contained a very large number of fibres 1–1½ inches long, some whitish, some brown, but all clearly of vegetable origin. I was told that no fruit was being given, but on pressing the question I discovered that the child ate bananas freely, and comparison with the fibres from a banana showed that this was the source of the supposed worms. In another case a child was said to be passing many worms, and the mother showed me white stemlike pieces ½–1 inch long, which proved to be the stem of water-cress, of which the child was fond.

Roundworm. The roundworm (ascaris lumbricoides) can hardly be overlooked or mistaken, it is very like the ordinary earthworm, both in appearance and in size ; on the average it measures 5 inches (male) to 10 inches (female), but I have seen it 13 inches long. It is very much less frequent than the threadworm in children : and when it is present is seldom found in large numbers, usually only three or four and sometimes only one.

One child was brought to me who had passed twenty from the bowel within a fortnight ; another boy of ten years had vomited two and passed twenty-four per rectum within three weeks, his sister had passed roundworms three months previously ; cases have been recorded in which many scores of these worms were passed.

Several children in a family are liable to be affected : in some cases this may mean direct transmission of the ova from child to child ; in the following case the intervals between the appear-

ance of the worm in different children suggests rather coincidence or some common source of infection.

Lilian M., aged three years, was brought for roundworms, of which she had passed altogether about two dozen within the past twelve months. She had frequent colicky pains in the abdomen, and generally became feverish and ill just before passing the worm. One sister had a roundworm five years ago; it crawled up into the pharynx, and she pulled it out of her mouth without vomiting. Another sister, aged fourteen years, complained when five years old of pain in her nose, and some hours later pulled a roundworm out of one nostril; a younger sister, aged seven years, passed a roundworm from the bowel one month ago.

A child aged $1\frac{5}{12}$ was brought to me with a history of having coughed up a roundworm a few hours previously; he had passed threadworms, but never any roundworms by rectum. The roundworm shown to me was below the average size and would easily have slipped into the larynx, but I suspect that in this case it was really coughed up from the pharynx. There are, however, cases on record in which these worms have found their way into the air-passages; they have even produced suffocation, but I have never known any serious harm from them myself.

I saw one child with jaundice associated with the presence of roundworms, which suggested the possibility of a worm in the bile-duct, as has happened, but there was no means of proving it, and the jaundice passed off like an ordinary catarrhal attack.

In addition to the colicky pains I have noted the presence of ravenous appetite and of excessive thirst in children with roundworms and the cessation of these symptoms when the worms were expelled. Nervous disturbances similar to those seen with threadworms are also seen with roundworms; disturbances of sleep and general 'nervousness' are common.

Occasionally convulsions have been produced. Sigaud [1] recorded the case of a child aged six years, who whilst eating his supper suddenly became unconscious, the right arm and leg were paralysed and when consciousness returned and the administration of santonin had resulted in the passing of twenty roundworms, the paralysis and impairment of speech which had accompanied it passed off in a few days.

I have already mentioned the case of a girl aged $3\frac{3}{4}$ years, who had a mysterious fever for at least five weeks with vomiting and screaming for hours almost every night (the chart is shown on page 283); the symptoms all subsided suddenly with the death of a roundworm which was passed dead and slightly shrivelled two days after the symptoms had ceased.

[1] *Gaz. des Hôpitaux*, June 30, 1904.

Trichocephalus dispar. Next to the roundworm the whipworm or trichocephalus dispar is probably the commonest intestinal parasite in children in this country, and yet I doubt if most medical men have ever seen it. In its white colour it resembles the threadworm, but it differs altogether in its length and shape. The terminal half-inch is nearly twice as thick as the threadworm, and the rest of the worm up to the head consists of a fine hair-like filament about 1¼ inches long. I have found this worm curled up in a spiral manner in the intestine with the minute head fixed in the mucous membrane so firmly that I have had to pull to detach it. I observed the trichocephalus dispar in 8 per cent. of children examined post mortem at the Children's Hospital, and I think invariably with threadworms; I have rarely found more than three or four whipworms and these have been almost always in the cæcum.

I have not seen a single case myself in which the worm was detected in the stools during life: apparently it causes no symptoms which can be distinguished from those of the threadworm with which it is associated.

Tapeworms are so rare in children in this country that I shall not consider them in detail here. The usual variety in this country is the tænia mediocanellata (from beef) distinguished chiefly by the four suckers on its head from the tænia solium (from pork) whose head shows a row of hooklets. The distinction is very difficult if only the ripe proglottides or segments are seen, as is usually the case; fortunately the distinction is of no practical importance, for the treatment is the same.

I have seen tapeworm at the age of twelve months, but it is rarely seen until after the age of three years. There are no characteristic symptoms except the passage of the segments, but I have noted nervous symptoms in some cases, e.g. Rita M., aged seven years, had been passing segments of tapeworm (which proved to be tænia mediocanellata) for two months; she was 'very nervous' and lately had been suffering with night-terrors. I gave extract of male fern ɑ)xl, and a considerable length of tapeworm was passed, but not the head. Eleven and a half weeks later, segments again appeared in the stools and the child was brought with a history that she had again become nervous and had lately complained of pains in the abdomen. With 80 minims of extract of male fern the worm was expelled entire with the head.

Treatment

Threadworms. The observations which I have described above have a practical bearing on treatment; as the ordinary habitat of the threadworm is the cæcum, and it is often in the appendix, and sometimes even in the small intestine, any injection which is only large enough to reach the top of the rectum or the lower part of the colon is not likely to do much good. Some have even gone so far as to say that injections are useless, on the ground that it is not possible to reach the cæcum by this means. Such a statement is no doubt perfectly true if the injection consist of only 3 or 4 ounces of fluid. With such a quantity no part higher than the descending colon is likely to be reached. But with larger quantities—12 to 18 ounces or more—it is quite possible to fill not only the cæcum, as is apparent in the treatment of intussusception by injection, but also the vermiform appendix, if one may judge from post mortem experiments in which I have repeatedly demonstrated this. Injections, therefore, are of value, but it is essential that large quantities of fluid should be used—a child of six to twelve years will often tolerate as much as 16 to 20 ounces—and experience has shown that large injections of salt and water or of infusion of quassia are often effective.

I have occasionally used a decoction of garlic for injection: three or four roots of garlic are allowed to simmer in a quart of water for about two hours, ½–1 pint of the resulting decoction is used as an enema and is sometimes very effectual, but I have occasionally had complaints that the child turned faint when this was given. It would be wise therefore to make the decoction weaker by diluting it with an equal quantity of water after it is prepared.

Treatment by injection alone, however, often fails, and this is easily intelligible, for the large intestine varies so much in length, and therefore in capacity, in children of about the same age, that it is practically impossible to know what amount of fluid is required in any given case. Thus, in one child, aged fifteen months, the large intestine was 28 inches long, in another, aged fifteen months, it was 34 inches, while in another, aged twenty-three months, it was 44½ inches long, and similar differences were found in older children.

Moreover, as the above observations have shown, threadworms may be present, though not very commonly, in the small intestine; and the chance of reaching these by any injection is

very slight, even when they are situated, as is usually the case, only just above the ileo-cæcal valve. In the post mortem experiments alluded to it was found that occasionally the fluid did pass up into the small intestine, but it is obvious that the conditions after death are so different from those during life that any conclusions drawn therefrom must be of very doubtful value. There is, however, some evidence that this occasionally happens during life, and in one case where an enema of olive oil, followed by a large enema of soap and water, was given as an aperient to a boy, aged 4½ years, twenty-four hours before death, I found the oil as high up as 6 inches above the ileo-cæcal valve. This, however, is probably quite exceptional, and for practical purposes one may say that the small intestine cannot be reached by injections.

It is necessary, therefore, to give drugs by the mouth also, and no drug has proved more effectual in my experience than santonin.

This drug is best given in combination with an aperient thus: Santonin gr. ij, Pulv. Scammonii Co. gr. ij, Calomel gr. ½; 4 grains of this powder can be given every alternate night to a child of about eight years and 3 grains to a child of four years (see below).

Podophyllin is said to be of value against threadworms. I have used the tincture in doses of 1–2 minims according to the age of the child and have seen good results apparently from it, but even in these small doses it has the disadvantage that it is apt to cause griping pains.

Naphthalin has been recommended; it can be given twice a day in doses of 2–3 grains suspended in mucilage, to a child of four or five years; it should be given only for a few days, and every alternate night a purgative such as calomel should be administered.

Liquid paraffin has been thought to be effectual in some cases, and as many children with threadworms are inclined to constipation paraffin is doubly useful. It should be given in doses of at least 1–2 drachms three times a day half an hour before meals.

In addition to the use of drugs the prevention of any possible conveyance of ova to the mouth must be attempted, for, as shown above, this is probably one mode of keeping up the supply of worms, and the cutting short of finger-nails and the prevention —by mechanical restraint if necessary—of scratching the anus are no unimportant adjuncts to treatment.

Roundworms. If a child can take santonin the treatment of roundworms is simple enough, and they are generally much more speedily got rid of than the threadworms with which they are associated. But there are many children who are made very sick by the smallest dose of santonin, and occasionally yellow vision (xanthopsia) and reddish-yellow or dark reddish-brown urine and even delirium show how profoundly the child is affected by this drug.

In some cases it may be rather the purgative which is given with it, than the santonin itself, which causes the vomiting ; for instance, in the powder which I have mentioned above, the compound scammony powder or the calomel may be responsible ; it is not very uncommon for calomel alone to make children vomit, and there are children who vomit with powders of any sort. To these santonin can be given in lozenge form as the trochisus santonini (B.P.), which contains one grain ; a child of three years old can have two of these at night followed by an aperient in the morning. One of the chocolate ' cocoids ' (Oppenheimer) containing santonin grs. i–ij with calomel gr. $\frac{1}{2}$–j can be ordered to be given every alternate night three or four times. Santonin can also be given with an aperient dose of castor oil emulsified in mucilage.

Naphthalin is useful for roundworms as well as for threadworms ; its odour, however, makes it an unpleasant drug to take.

Tapeworms. I have generally used the liquid extract of male fern for tapeworms in children ; an aperient such as calomel or castor oil is given on the previous day to ensure a free action of the bowels in the evening, and the following morning at an early hour a draught of male fern is given. To a child of five years a drachm of the liquid extract should be given ; as in the case mentioned above, I have used as much as 80 minims for a child of seven years. The drug may be given in emulsion thus : Extract. Filicis Maris liq. ℥j, Spirit. Chloroformi ♏ij, Syrup ℥ij, Mucilag. Acaciæ ℥ij, Aq. Menth. Pip. ad ℥j. About two hours later a saline purgative such as magnesium sulphate 30 grains, half a Seidlitz powder or $\frac{1}{2}$–1 ounce of castor oil should be given.

I have also used kamala with male fern, giving it in the manner described by Dr. Eustace Smith. To a boy of four years a dose of Pulv. Kamala gr. xx, Extract. Filicis Maris Liquid. ♏xx, was given suspended in mucilage with water at 5.30 a.m. and again at 8 a.m. This dose caused some slight pain in the abdomen, and the passage of a large quantity of mucus and

7 feet of tapeworm, but not the head. Usually no aperient is necessary with this treatment as the kamala itself has a purgative effect. Thymol has been used with success; but on the only occasion when I have tried it, giving $1\frac{1}{2}$ grains to a child of four years, it was a failure, probably from insufficiency of dose.

CHAPTER XXI

JAUNDICE IN CHILDREN

JAUNDICE is a symptom which in childhood, as in later life, has a very varied significance : I shall consider it here chiefly with reference to prognosis. It is a matter of importance to recognize the different conditions under which jaundice may occur, for while in many cases it is merely a symptom of some slight and transient disorder, in others it has the gravest possible significance.

Icterus Neonatorum. The commonest condition in which jaundice occurs is that known as icterus neonatorum. This occurs in a large proportion, variously estimated at 30 to 80 per cent., of all infants. It is hardly necessary for me to describe this condition ; the jaundice appears almost always within the first week, usually on the third or fourth day. It is seen first on the trunk and face, then on the limbs and conjunctivæ ; the urine and fæces remain normal in the milder cases. The child's health does not suffer in any way, and after four or five days, or at most one or two weeks, the jaundice disappears. Such is the ordinary course of icterus neonatorum. Most writers on the subject mention two weeks as the limit of duration in the most severe cases, but I should like to draw attention to certain cases in which, instead of lasting a few days, the jaundice lasts as long as two months or even more, and then passes off, leaving the infant apparently none the worse for it.

A male infant was brought to the Children's Hospital for jaundice at the age of twenty-nine days, with a history that the yellow colour had been noticed first on the fourth day after birth, and that, except for occasional vomiting and screaming after food, the infant was in good health. The urine was said to be ' very dark ', but the stools were often green, and once at least when I saw them were of a good yellow colour. There was nothing either in the child or in its parents to suggest the possibility of syphilis. The infant was well nourished ; the liver was little if at all enlarged, and certainly not hard ; the spleen could not be felt. The jaundice was not very intense, but was well marked both on the skin and conjunctivæ. It persisted for 9½ weeks altogether, gradually disappearing, and leaving the child in apparently good health except for some flatulence.

A similar case was the following:

Gilbert W. was admitted to King's College Hospital, at the age of seven weeks, for jaundice: this had been present 'since birth'; there had been some bleeding from the navel at three weeks, but not since. The spleen was three finger-breadths and the liver about two finger-breadths below the costal margin; the child was somewhat wasted, the stools were yellowish but very pale; the urine was not obtained until the ninth day after admission, when no bile was detected in it.

The jaundice was still present 9½ weeks after birth, but was then diminishing and disappeared within the next few days. The child was seen again at 7½ months old, when, except for marked pallor, he seemed healthy; the spleen could not be felt and the liver was only just felt.

In this case the family history showed miscarriages and stillbirths, thus: (1) lived fourteen days only; (2) healthy, aged six years; (3) miscarriage; (4) stillborn; (5) stillbirth; (6) miscarriage; (7) the patient. There was, therefore, at least a possibility of syphilis, and perhaps the enlargement of the spleen in the patient pointed that way, but there was no other evidence, and the complete recovery seemed opposed to any gross syphilitic disease of the liver.

It is impossible to say what is the explanation of these prolonged cases of icterus neonatorum, but clinically, at any rate, one may group them provisionally with the commoner cases of one or two weeks' duration, remembering that the nature even of the common condition is uncertain. The importance of recognizing the occurrence of these prolonged cases lies chiefly in the matter of prognosis, for when the infant shows jaundice within a few days after birth, and the jaundice persists more than three weeks, one's tendency is to think of syphilitic hepatitis, or of congenital obliteration of the bile-ducts, in either of which conditions the prognosis would be extremely bad; but bearing in mind these unusual cases in which the jaundice passes off after two months or even longer without ill effect, one must examine such infants very carefully before giving prognosis.

In the first case which I mentioned there were certain points which had to be taken into account: the jaundice was not the intense yellow of the cases with congenital obliteration of ducts; moreover, there was not the enlargement of liver and spleen, nor the hardness of the liver which characterizes the obliterative cases; the apparently normal size of the liver and spleen was also opposed to a syphilitic lesion, and the presence of bile in the stools showed that at any rate there was no complete obstruction of the ducts.

I felt by no means certain of the prognosis in these cases, but

the event showed that it was altogether favourable so far as the immediate future was concerned.

I say advisedly 'so far as the immediate future is concerned', for I think more observation is needed before we can make any general statement as to the ultimate fate of such cases. My own experience leads me to regard them with suspicion. It may be that this unusual prolongation of jaundice, although it passes off apparently as innocently as in the ordinary cases of short duration, has, nevertheless, some sinister significance.

A male infant, according to the mother's statement, had been jaundiced for eight weeks after birth. When seen by me at the age of nineteen months he was intensely anæmic, the spleen was enormous, the liver not felt. The child died at the age of twenty-one months. The post mortem showed in addition to the enlargement of the spleen a very slight intercellular increase of fibrous tissue in the liver; the pancreas was extraordinarily hard and large, but unfortunately was not examined microscopically.

In this case there was no clinical evidence of syphilis in the child or his family history.

The clinical aspect of the case was that of the so-called 'splenic anæmia' of infants, a condition of extremely uncertain origin, but known to be associated in some cases with slight degrees of proliferation of fibrous tissue in the liver, and undoubtedly occurring sometimes where there is strong evidence of congenital syphilis.

In this connexion I should like to mention another case in which there was a history of prolonged icterus neonatorum. This was a boy who had jaundice lasting six weeks from the time of birth; the jaundice then passed off, leaving the boy apparently healthy. At the age of two years and three months, however, jaundice again appeared; it lasted several weeks, and ended fatally. I made a microscopical examination of the liver, which showed advanced intercellular cirrhosis, which strongly suggested congenital syphilis, although there was no other evidence of it.

Even as regards the immediate future of these cases of prolonged jaundice in the newborn, I think we may well be cautious in prognosis. I have mentioned cases in which there was apparently permanent recovery, but I have also seen a fatal ending where some complication has supervened; for instance, a male infant who had been jaundiced 'from birth' came under my care at the age of six weeks with intense jaundice and some diarrhœa. He rapidly sank from exhaustion and was still much jaundiced when he died at $6\frac{1}{2}$ weeks; post mortem, there was no cirrhosis of the liver, the bile-ducts were normal, as we had supposed, for

there was a trace of bile in the stools; the spleen was normal. In this case there was no evidence of syphilis.

Even where the jaundice has not been greatly prolonged, I have seen what appeared at first sight to be an innocent icterus neonatorum end fatally.

Ivy G. was brought to hospital at the age of two weeks with jaundice, which had been present 'since birth'; the stools had been green until a few days previously, when they had become white; neither the liver nor the spleen could be felt. The child was born at full term; had had no snuffles; she was the fifth child; there had been no miscarriages; the cord had separated on the third day, and the umbilicus was healthy.

On the sixteenth day she vomited some blood and passed some in the stools, but no further hæmorrhage occurred from these parts. Two days later, however, there was some hæmorrhage from the umbilicus, and large subcutaneous hæmorrhages appeared over the back of the hands, and the child became convulsed and died, still deeply jaundiced, on the nineteenth day after birth.

Post mortem showed a layer of recent blood-clot over the vertex and base of the brain. The spleen was very slightly enlarged and unduly soft; the liver, beyond being rather soft, perhaps slightly enlarged and stained greenish, showed nothing abnormal; all other organs were normal. No cause was found for the jaundice. I examined the liver and kidney microscopically; only a few of the liver cells were fatty, otherwise I could detect nothing abnormal in either organ.

Perhaps this should be regarded as an infective condition, or perhaps as an instance of 'Buhl's disease', the so-called 'fatty degeneration of the newborn', in which just such symptoms are described; but the point of practical importance which it may serve to emphasize is the need for regarding with suspicion jaundice which persists unusually long in a newborn child; the only point which made one hesitate in prognosis in this case when I first saw it at fourteen days old, was the intensity of the yellow colour at this time when the jaundice of icterus neonatorum has usually almost, if not entirely, vanished.

There are rare cases in which several successive children of a family succumb to a malignant type of jaundice within a few days of birth.

This family icterus gravis neonatorum begins when the child is but a few hours old, the jaundice gradually deepens, the infant is fretful and may seem to have some abdominal pain, he becomes increasingly drowsy, and with progressive feebleness death ensues within a week or thereabouts.

The liver in these cases is not apparently enlarged, nor is it particularly hard; there is no enlargement of the spleen. Autopsy has shown no gross lesion to account for the condition.

Two instances of this family affection have come under my

own notice; in one of these three successive infants had died from this cause; in the other, four had died including two twin children. As many as six deaths from this affection have occurred in one family. No cause is known, and at present we have no effective treatment for the disease when it has occurred, nor can we prevent its occurrence in subsequent infants of the same family.

I shall not enter into the vexed question of the etiology of icterus neonatorum here, except to say that, in my opinion, there is little doubt that in most of the cases, if not in all, the jaundice is not only hepatogenous, but is also obstructive.

I do not mean that there is necessarily any gross obstruction in the larger bile-ducts; but when we remember that the bile is secreted at very low pressure, it is easy to understand that a very slight increase in viscidity of the bile, especially in the smaller ducts, might cause sufficient obstruction to produce jaundice.

If the bile secreted during the later months of intra-uterine life is particularly viscid, one could well understand that with the sudden increase in the secretion of bile which presumably occurs when the child begins to take food after birth, and probably even before food is taken, the difficulty of exit, owing to viscid bile already in the ducts, may cause re-absorption of some of the newly secreted bile into the circulation. And there is some evidence that the bile, both in intra-uterine life and in very early infancy, is apt to be very viscid. This I have myself observed at autopsies, and the tendency to formation of biliary concretions which seems to be present in intra-uterine life probably points in the same direction. In some cases of icterus neonatorum extreme viscidity of the bile has been demonstrated post mortem. In one infant who died with jaundice at the age of fourteen days I found this viscidity marked, and in another case, which died at the age of four weeks, when the jaundice was disappearing, I have noted the contents of the gall-bladder as 'extremely viscid'. In an infant who died at the age of four weeks with pyæmia and jaundice I found the bile so thick that it could scarcely be squeezed through the common duct with considerable force.

Biliary concretions or calculi are extremely rare, it has even been stated that they never occur in children; but cases have been observed, and they appear to be commoner in infancy than at any other period of childhood. Of twenty-three cases which I collected,[1] including three which came under my own observation, fifteen were in infants, and fourteen of these were in infants

[1] *Path. Soc. Trans.*, vol. l, p. 151.

under ten months of age. In several of these there was intense jaundice at birth or shortly afterwards, and calculi were found in the ducts.

In one of my own cases minute calculi were found impacted in the common duct in an infant aged nine months, but there had been no jaundice.

I mention this very rare occurrence as indicating a tendency to stagnation of bile at this age, and as just worth remembering in connexion with some of those cases of icterus neonatorum which run an unusual course.

Syphilitic Jaundice. Congenital syphilis, as I have suggested, may possibly be a factor in some of the prolonged cases of jaundice in infants who recover, but even temporary recovery, with complete intermission of symptoms, is probably quite unusual in the jaundice of the newborn with congenital syphilis.

The usual course in syphilitic infants is steadily downwards; the jaundice is present at birth, it persists, and may be very intense. Hæmorrhage may occur in the skin or from mucous membranes, and after a few weeks the infant dies. The liver shows only slight enlargement, and feels only slightly firmer than normal; the surface is smooth; the change found is an interstitial hepatitis extending throughout the liver with formation of fibrous tissue between the individual liver cells, in fact, an intercellular cirrhosis.

The presence of deep jaundice at birth is, I think, a point of some value in distinguishing these cases from the simple icterus neonatorum, but I should not care to lay any great stress upon it, for I have seen jaundice almost certainly due to syphilis, and ending fatally, which did not begin until eight weeks old.

Syphilitic hepatitis, however, with its resultant intercellular cirrhosis, is not the only cause of jaundice which ends fatally in congenital syphilis; some of the cases of pyæmic jaundice in infants are indirectly due to syphilis. Acute epiphysitis, which is a very early manifestation of congenital syphilis, may become suppurative, owing to a secondary infection with pyogenic micro-organisms; in this way, a pyæmia may be started which may give rise to pyæmic jaundice. In two, probably syphilitic, infants which I examined post-mortem, this appeared to be the sequence of events.

This pyæmic jaundice, whether it is secondary to syphilis, or due directly to umbilical infection, almost invariably ends fatally.

Congenital Obliteration of Bile-ducts. There is another group of cases of jaundice in infancy in which the prognosis is always bad ; I mean those with congenital obliteration of the bile-ducts. It is by no means always easy to distinguish these cases from those due to syphilis, for in both the jaundice may be present from birth ; it becomes very intense, and is associated frequently with hæmorrhages ; the liver and spleen are enlarged and too firm ; the infant wastes and dies.

One might have expected that with such a malformation of the bile-ducts jaundice would necessarily have been present at birth, but in forty out of the series of fifty cases collected by Dr. John Thomson this point was specially mentioned, and only nine showed jaundice at birth. Four cases of this kind have come under my observation, and were examined post mortem. None of them had shown jaundice at birth ; in one of them the jaundice had not appeared until three weeks after birth, although autopsy showed that the gall-bladder and ducts were completely absent, being represented only by fibrous tissue.

In the cases which I have seen, the liver has seemed much harder on clinical examination than in the syphilitic cases, and corresponding with this, post mortem, the liver showed more advanced cirrhosis than I have found in cases of congenital syphilis. This may, however, be due to their longer duration, for whilst many of the cases with congenital syphilis die within a few weeks, the cases with congenital obliteration of ducts often live as long as four or five months. Two of the series mentioned above lived ' into the eighth month ', and one of the cases which I examined post mortem died at the age of $9\frac{1}{2}$ months.

An interesting point which I may mention here is the occurrence of severe jaundice in several infants of the same family. This has been noticed in cases of congenital obliteration of the bile-ducts. Dr. J. Thomson, in his valuable monograph on that subject, mentions two instances in which the malformation was found, post mortem, in more than one infant of the same family. But it is not only with this condition that several infants of a family may suffer with jaundice. In one autopsy which I made on an infant who died at the age of fourteen days with jaundice, there was nothing obviously abnormal in the liver or ducts, nor was there any appearance of cirrhosis (unfortunately no microscopic examination was made). Four other children in that family were said to have had jaundice lasting four to six weeks after birth, but none of them had died. There was nothing in the history to suggest syphilis in the parents ; but some such cases are certainly syphilitic.

Henoch records fatal jaundice in an infant whose mother had syphilis; three other children in the family had been jaundiced at birth, and had died soon after. Dr. Thomson (loc. cit.) also refers to cases in which several children of one family died with jaundice, perhaps the most notable being one recorded by Underwood, in which the first nine infants of the family died with jaundice under one month old, and the tenth died, aged six years, with jaundice also.

Catarrhal Jaundice. In children beyond the age of infancy jaundice is most commonly catarrhal in origin. So far as my own experience goes this catarrhal jaundice would seem to be most common between the ages of two and six years. Thirty out of fifty cases fell within these limits. Almost always the child has been ailing for some days before the jaundice appears, feeling languid, and sometimes drowsy. Often the child is cross and fretful, the appetite is bad, and almost invariably there is something abnormal in the state of the bowels; in some there has been diarrhœa, in others constipation, in others only offensive stools.

Vomiting commonly precedes or accompanies the appearance of the jaundice. The urine quickly becomes darkened, and the fæces become pale. In many cases there is pain in the epigastrium and right hypochondrium, and this may be a marked symptom. In a boy who was brought to hospital with only slight jaundice, the pain in the abdomen was so severe that I felt doubtful whether the case was really one of catarrhal jaundice only; the subsequent course of the case seemed to show that it was so. The liver was also apparently very tender in this boy and, as in many cases of catarrhal jaundice, could be felt about $1\frac{1}{4}$ inches below the costal margin; one can sometimes easily detect a gradual diminution of the size of the liver as the jaundice passes off.

The temperature in this case was raised to 101·6°; a slight rise of temperature is not uncommon at the commencement of the illness.

It is customary to mention slowness of the pulse and itching of the skin as symptoms of catarrhal jaundice, but neither is common in children. The most marked slowing of the pulse which I have noted was in a child of eleven and a half years, in whom the rate fell to 56 per minute. Itching I do not remember to have observed in any case in a child.

As a rule catarrhal jaundice passes off in about ten days or a fortnight, but I have seen cases in which it has lasted much longer; for example, in one child it lasted about seven weeks, in

another it lasted twelve weeks, so that some caution is needed in prognosis.

In this connexion also I should like to draw attention to the recurrence of jaundice at short intervals in some children. A boy, aged $8\frac{3}{4}$ years, was brought to hospital with this history. Two years ago he had jaundice; since then he has had seven or eight attacks of jaundice. For a short time before each attack he becomes very drowsy, so much so that the mother says she can tell beforehand when the jaundice is coming; the attack is associated with vomiting and pain in the abdomen. In another case an attack of jaundice, apparently catarrhal, lasted fourteen days, then the child remained well for six weeks, after which another attack of jaundice came on and lasted a fortnight.

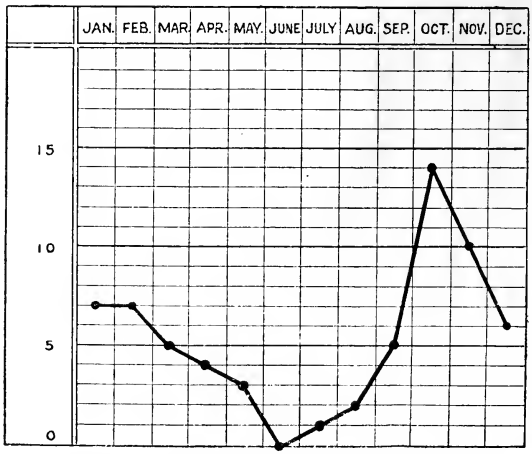

FIG. 22. Seasonal incidence of catarrhal jaundice in children.

It is, of course, possible that such cases in children may occasionally be due to biliary calculi, but, as I have already pointed out, these are extremely rare in childhood. I have elsewhere (loc. cit.) recorded the case of a boy, aged ten years, who, during an attack of severe abdominal pain with vomiting and jaundice, showed a fluctuating tumour in the position of the gall-bladder. He was then under the care of Sir Thomas Barlow, at the Children's Hospital. The tumour suddenly subsided and the jaundice passed off. I saw the boy six years later, at the age of ten years. The mother then stated that since the original illness he had suffered with repeated attacks of abdominal pain, in which he became of a 'sallow colour'. It was suggested that these were possibly

due to biliary colic, but in both the cases mentioned above I was inclined to regard the attacks as recurring catarrhal jaundice.

What may be the cause of catarrhal jaundice is at present unknown, butI think there can be little doubt that sometimes at least it is an infective condition. I have noticed at the Children's Hospital that in some years it seems to be much more prevalent than in others, and it seems to have a seasonal incidence, being most frequent in the autumn and winter, especially during October and November, as the accompanying chart of sixty-four cases shows.

But the strongest evidence of its infective nature is the occasional affection of several members of a family in succession; e.g. I had under my care four children of one family: Alfred C. was first taken ill on February 12, Wilfred C. on February 14, Elsie C. on February 16, and Maud C. on February 23; these four were the only children in the family, the parents were not affected. The jaundice in all ran a benign course, the temperature was taken only in two of them and was found to be subnormal; in each the illness lasted about one or two weeks.

Cirrhosis. One must always remember that there are other more serious causes of jaundice in children beyond the age of infancy, and the one which is perhaps most often overlooked, because unexpected in childhood, is cirrhosis. I have already referred (p. 305) to the intercellular cirrhosis which occurs in early infancy, usually where the syphilis is speedily fatal, but occasionally cirrhosis is found in older children associated with unmistakable evidence of syphilis elsewhere. In the museum of the Children's Hospital, Great Ormond Street, is the liver of a boy, aged 10 years and 4 months, who had jaundice and ascites, with Hutchinsonian teeth, syphilitic retinitis, a sunken bridge of nose, and scarring round the mouth. The liver showed extensive fibrosis with much atrophy of liver cells. Whether such a case should be regarded as a late result of the intercellular cirrhosis of infancy in a case which had survived beyond the usual age, or as the outcome of a syphilitic hepatitis beginning in later childhood, is not clear, but it seems probable that syphilis may cause an acute hepatitis in later childhood which may lead to cirrhosis.

A girl aged about eight years was admitted into King's College Hospital with severe jaundice and high fever. The child seemed very ill, the fever continued, and I suspected that the case might prove to be one of 'Subacute Yellow Atrophy'. After about three weeks of fever, the jaundice was still as intense as ever, the child showed no symptoms of syphilis, but a Wassermann proved to be distinctly positive. The child was then put upon mercury and

potassium iodide, and rapidly improved, the jaundice and temperature subsided, and the child made a complete recovery.

Less rare than intercellular cirrhosis in children beyond the age of infancy, is the interlobular variety, which seems to have no connexion with syphilis. A large, hard, irregular liver, exactly like that seen in adults with alcoholic cirrhosis, is occasionally found in children. The surface is studded with hobnail-like bosses which are coarse enough to be detected by palpation during life. The jaundice in such cases is generally very slight indeed, there may be no jaundice at all. The striking feature of these cases is rather the great enlargement of the spleen, and, given a child with slight jaundice, a very large spleen, and a tendency to epistaxis or other hæmorrhages, one has sufficient grounds for suspecting cirrhosis. If, in addition, the liver can be felt—and I would point out that the liver may not be easily felt in these cases—its hardness and irregularity of surface may clinch the diagnosis.

I have seen at least six cases of this kind, three in girls, aged respectively six and a half, seven, and eleven years; the two latter cases were fatal in hospital and verified by autopsy, the other case died at home at the age mentioned, which was about twelve months after the first onset of jaundice.

Three cases were in boys, in one the cirrhosis was verified by post mortem examination at the age of $5\frac{1}{4}$ years; the other two were aged nine years and ten years respectively when they first came under observation: one of them was seen again at the age of nearly fifteen years with a very large hard and irregular liver, and the spleen considerably enlarged.

The duration of the disease in this last case was, I think, quite exceptional; in most cases death occurs within two or three years after the first symptoms have appeared.

I must mention that a history of alcoholism is very rarely to be obtained in these cases of cirrhosis in childhood; a few of them are due to the giving of beer and spirits, not necessarily in large doses, but it seems likely that in many, if not in most, cases there is no connexion whatever with alcohol.

Amongst the few cases I have seen I have failed on careful inquiry to obtain any evidence of prolonged or excessive use of alcohol; but the difficulty of excluding this cause is great: a mother may easily forget that she gave her infant brandy in what she considered a suitable dose, perhaps a teaspoonful two or three times a day for many weeks when it had diarrhœa or was not thriving in infancy. I remember a child under four years

of age who cried for beer when in hospital; it seemed likely enough that she was accustomed to it as a beverage; but one may hope that the giving of alcoholic drinks to such young children is very rare.

Dr. Ernest Jones[1] points out that experimentally it has been proved that young animals are more susceptible to the noxious influences of alcohol than older ones, and this fact may have a bearing upon these cases of cirrhosis in children; certainly it is always wise in prescribing alcohol as a medicine for children to use the smallest dose which will produce the necessary stimulation—and this dose is, I think, much smaller than is sometimes supposed. I see half drachm or even drachm doses of brandy given to infants of twelve months where, judging from my own observation, I should consider 10 to 15 minims a more suitable dose; but apart from dosage there is another point in which care is necessary in ordering alcohol for children, namely, to see that it is stopped when no longer required. I have myself seen instances in which brandy had been given for many weeks after all need had ceased because the mother had received no directions as to stopping it. Dr. Jones (loc. cit.) states that in some of the cases of cirrhosis in children the disease was thought to have originated in this way.

Jaundice with other diseases. Amongst my notes I find several cases of jaundice occurring in children as a complication of various conditions, such as heart disease, pneumonia, tuberculosis, &c. It is an important question in connexion with such cases whether the supervention of jaundice in any way influences the prognosis. One of the commonest associations is jaundice with heart disease, and so far as my experience goes, this occurs usually in the more advanced cases of cardiac disease, and I think one's tendency is to regard the supervention of jaundice as usually 'the beginning of the end'; but this is certainly not always so.

I had under observation a girl who was in hospital at the age of 9½ years with a very severe mitral incompetence, a greatly enlarged heart, and probably adherent pericardium. At that time there was considerable œdema, jaundice appeared, and one might very reasonably have thought that the case was near its end. She improved, however, and when seen 3½ years later was so much better that she could walk about and enjoy life in a quiet way. She lived until she was about seventeen.

I have seen other similar cases in which, in spite of temporary jaundice, the heart condition improved under treatment, and the children were relieved, at any rate temporarily.

[1] *Brit. Journ. Dis. Childr.*, Jan., 1907.

Both in pneumonia and tuberculosis jaundice is a symptom of very grave significance.

There are other rarer conditions under which jaundice is seen in childhood; but with these I am not concerned here: for instance, acute yellow atrophy, a disease rare in adults, has been seen but only very rarely in children; I have not met with an instance myself.

Family acholuric jaundice (congenital family cholæmia) is another rare affection in which jaundice is usually present from birth, becoming increased at times and lasting throughout life. The urine in these cases contains no bile pigment and the stools are of normal colour. The blood serum is found to contain bile and the red cells are abnormally fragile.

This condition seems to interfere but little with the general health and is probably dependent upon some change in the blood-forming system.

Treatment

The child with congenital obliteration of the bile-ducts, or with 'hobnail' cirrhosis of the liver, is beyond the reach of curative treatment; we can only treat any special symptom that arises.

Icterus neonatorum is usually so evanescent a phenomenon that it calls for no treatment beyond—and I fancy this may be of importance—keeping the infant warm.

I have often thought that some of the cases in which the jaundice just after birth lasts for an unusually long time may be due to some catarrhal condition consequent upon chilling of the newborn infant: he has just come from an environment where the temperature was 99° or more; he now lies naked, or in the sorry protection of a flimsy shawl, in a room with a temperature of 65° or less, while he awaits the attentions of a nurse who is perhaps none too careful to avoid chill in washing him. If cold has any share in producing catarrhal jaundice, some such origin seems at least possible in some of the cases of jaundice in the newborn.

However this may be, there is no doubt that feeble and premature infants are specially liable to icterus neonatorum, and on every ground, therefore, these are cases in which warmth is desirable. If the jaundice has not disappeared by the end of the second week, it is, I think, well to give grey powder. Hyd. cum Creta gr. $\frac{1}{4}$, Sod. Bicarb. gr. j, may be given three times a day;

and if there is any reason for suspecting that congenital syphilis may be present the dose of hyd. cum creta should be increased to half a grain, or unguentum hydrargyri, a piece the size of a large pea, may be smeared over the chest or back each night and left on till morning covered with a flannel bandage.

In the treatment of the catarrhal jaundice of older children, the diet is of first importance; for whether as cause or effect there is no doubt that gastric disturbance is commonly associated with it, and I think that I have seen recrudescence of jaundice produced by overhaste in returning to an ordinary diet after the restrictions which had been practised during the earlier stages of the disorder.

The lightest of food should be given until all jaundice has disappeared: at first veal broth or chicken tea, milk partially peptonized, or Benger's Food prepared so as to be thin, calves'-foot jelly, not for any special nutritive value, for it contains but little, but as a change from the weary monotony of milk foods and broth; then if the child is one of those who, when in health, tolerates eggs well, an egg may be given very lightly boiled or beaten up raw with the milk or in the soup; then custard, and so back to milk-puddings and rusks, and if these cause no relapse of the jaundice, bread, chicken, fish, and gradually the ordinary diet.

I think such cases are best kept in bed, but with care to see that the child is so clothed that while he sits up and plays as children will in bed, he is not exposing himself to cold.

In the way of drugs where there has been vomiting, I have usually ordered bismuth carbonate in 10 grain doses, with bicarbonate of soda in a mucilage mixture. Sometimes I have used the ordinary rhubarb and soda mixture, such as Pulv. Rhei gr. ij, Sod. Bicarb. gr. v, Syrup Zingiber. ɑ)xv, Aq. Anethi ad ʒij ter die.

Recently I have been using sodium salicylate. To a child of six, Sodium Salicylate gr. v, Sodium Bicarb. gr. v, Syrup ɑ)xv, Aq. Menth. Pip. ad ʒij ter die may be given. In some of the cases in which I have used it the jaundice has disappeared within a few days after the administration was begun; but in a disorder of such variable duration it is difficult to assign to any mode of treatment its fair share, if any, in the recovery of the patient.

CHAPTER XXII

ENLARGED TONSILS AND ADENOIDS

If in this chapter I have undertaken a subject which might seem to appertain rather to the surgeon or the throat specialist than to the physician my excuse must be that the physician is very frequently consulted with regard to the naso-pharyngeal troubles of childhood, even though the treatment of them may pass into surgical hands.

Troubles in this region have so often a close relation to disturbances of general health and to symptoms remote from the nose and throat, that it is very necessary for every medical man not only to realize that there is such a relation, but to define it as clearly as possible in his own mind. I shall restrict my remarks to two of the commonest, viz. enlargement of the tonsils and adenoid overgrowth, and I have chosen these for consideration because there are in connexion with them many points of practical and everyday importance.

In one sense it is a mistake to speak of ' adenoids ' and ' enlarged tonsils ' as if they were two different affections ; they are really both manifestations of one process, an overgrowth of normal lymphoid tissue, and are therefore likely to be associated together. And here I would point out that lymphatic gland tissue or adenoid tissue has a very wide distribution in the body. In the naso-pharynx there are not only the tonsils but patches of exactly similar tissue on the wall of the pharynx, aggregated in the middle line to form the so-called third tonsil or Luschka's tonsil, with outlying small patches on the lateral parts of the pharyngeal wall near the orifices of the Eustachian tubes. At the base of the tongue there are some small patches, and throughout the intestine there are others in the form of solitary follicles and Peyer's patches, whilst in the appendix there is much lymphoid tissue, and in early life the thymus in its cortical part consists of similar tissue. The spleen also is largely a lymphoid structure ; and lastly there are the lymphatic glands proper all over the body, the number and wide distribution of which are hardly realized until some such disease as lymphadenoma brings into prominence many which in health are not appreciable by ordinary examination.

But it is not only the wide prevalence of lymphatic or adenoid

tissue in the body which I wish to emphasize here, but also the curious variability of its development in different individuals. Any one who is familiar with the post-mortem room appearances of childhood will know how greatly the prominence of the solitary follicles and Peyer's patches varies in different cases without any apparent reason. Again, any one who has tried to determine the average size and weight of the thymus gland in children of a given age will know that the variations are so great that hardly two individuals can be found to agree as to what constitutes a normal thymus in these respects. Again, examine clinically a score of children in succession, children of about the same age and apparently free from any local source of irritation which could affect the glands, and you will find that the palpability of the lymphatic glands varies in so striking a degree that it is very difficult to say what size of glands, in the groin or in the neck or axilla, is to be considered normal. So it is with the adenoid tissue of the naso-pharynx and tonsils; in different children it varies largely in development quite apart from any variation of health.

I have drawn attention to these points with a practical purpose: in the first place I wish to insist that the so-called 'adenoids' are not new growths; they are simply a more or less excessive development of normal structures which are normally present in this situation in every child. Perhaps I ought to apologize for mentioning such an elementary fact to medical men; but I do so because I have sometimes thought that medical men might do more to prevent the mischievous conception in the popular mind of 'adenoids' as new growths, which, being of the nature of 'tumours', must inevitably require operation. Excess of adenoid tissue in the naso-pharynx may be apparent within so few days after birth that it seems probable that in some cases at least it is congenital, whilst in others the differences of amount are due to variation in the development of these tissues after birth. Childhood is the age at which the lymphoid tissue is specially abundant, and it must not be forgotten that this tissue undergoes an involution during adolescence, a fact of no small importance in connexion with treatment.

Lastly, I will mention here a point to which I shall refer again in connexion with treatment, that the adenoid tissue of the naso-pharynx, and the tonsils, being part of the general scheme of adenoid tissue in the body, may share in such a general overgrowth of this tissue as is supposed to occur in the 'status lymphaticus'.

Symptoms. Turning now to the results of adenoid hypertrophy

and enlarged tonsils, I suppose that the majority of them are directly or indirectly due to simple mechanical obstruction to the ingress of air; that some are due to the catarrhal state which seems to be favoured so greatly by the presence of adenoids and enlarged tonsils; and that a further count against these structures is the possibility of their serving as a portal of infection.

It is hardly necessary to catalogue here the results of mechanical obstruction which may be due to adenoids; the facies, with its open mouth and its 'pinched-in' nose, the heavy or snoring breathing, the nasal voice, the stooping shoulders with the head poked forward, and the contracted so-called 'funnel-shaped' chest, with its depressed lower end of sternum and often transverse constriction below the nipples (Harrison's sulcus), or, as sometimes happens, a pigeon chest; all these are only too familiar. Hardly less characteristic are the catarrhal symptoms—frequent colds, deafness, at first occurring when the child catches a fresh 'cold', but soon becoming persistent; otitis media, hoarseness indicating a spread of the catarrhal condition to the larynx, cough, especially at night soon after going to bed or in the early morning. Occasionally there is persistent catarrh with muco-purulent discharge from the nostrils; there may even be some epistaxis, and I have seen cases in which the irritation of the pharynx by the catarrh seemed to induce vomiting sometimes after food. Such are the more or less direct results, mechanical and catarrhal, of adenoids, and no one, I suppose, doubts their relation to the adenoids; but when we come to the supposed indirect results, there is much more room for scepticism.

It is natural enough that the child whose air-entry is diminished, and whose sleep is so disturbed by the difficulty of breathing that he never has a properly refreshing sleep, should be pale and lacking in vivacity, may, indeed, seem stupid, and suffer with headache, and it is even possible that the nervous system under such conditions may become unduly irritable, so that the child is predisposed to such affections as habit-spasm or stuttering, or to a combination of nervous with catarrhal conditions, as in spasmodic croup, laryngitis stridulosa, or asthma; possibly even the tendency to so remote an affection as enuresis may be increased by the deficient aeration and consequent irritability of the nervous system.

But to suppose, as some have done, that adenoids can produce nervous or any other affections by some mysterious reflex effect apart from any mechanical obstruction or catarrh is an assumption which seems to me wholly unwarranted.

In addition to the mechanical and catarrhal results of adenoids, there is another group of consequences, and by no means the least important, namely, those connected with bacterial infection.

Look at the irregularities, the pits and furrows on the surface of enlarged tonsils : see the cheesy secretion protruding from the open crypts ; or again, look at a recently removed pharyngeal tonsil, the so-called 'adenoid' mass, with its folded irregular surface ; it is hardly surprising that in the nooks and crannies of such a structure bacteria should find a nidus. Local infection must surely be favoured by this condition, and when such an infection as diphtheria has once taken hold, everything is against the rapid disappearance of the bacillus from such a throat ; it is more than probable also that under such conditions any infective disease attacking the fauces or naso-pharynx is likely to run a severe course.

Research has actually shown the tubercle bacillus both in the tonsils and in 'adenoids', and Drs. Poynton and Paine have obtained the coccus which they have found in rheumatic joints and rheumatic heart lesions from the tonsils.

So far I have referred chiefly to the results of adenoid hypertrophy ; now let me say a few words about the results of enlarged tonsils. These similarly range themselves under the three headings—mechanical, catarrhal, and infective. In the very large majority of cases enlargement of the faucial tonsils is associated, as one might expect from the similarity in the character of the structures, with enlargement of the pharyngeal tonsil that is 'adenoids' ; and the resultant evils are for the most part only an aggravation of those I have already mentioned. But enlargement of the faucial tonsils does introduce certain modifications into the picture : the voice is different, the catarrhal affection takes the form of a recurring tonsillitis, a cold always 'flies to the child's throat'. Enlargement of the faucial tonsils adds also to the infective risks. As already mentioned, the micrococcus of rheumatism has been found in the tonsils, not, so far as I am aware, in adenoids ; the tubercle bacillus has been found in both, but Dr. Hugh Walsham, in his work on the channels of tuberculous infection, says that he could not find it after many investigations on adenoids, whereas it was found repeatedly in enlarged tonsils.

In connexion with recurring tonsillitis there is one point which calls for special mention, as it is, I think, often overlooked, namely, the connexion between decayed teeth and these faucial inflammations. No one, I suppose, doubts that the recurrence of

tonsillitis depends upon fresh outbreaks of infection in the crevices and irregularities of the tonsils, and no doubt repeated attacks of inflammation are caused similarly in the adenoid tissue of the naso-pharynx; but how often one sees children who are being assiduously sprayed and painted and dosed in the hope of preventing the frequent sore throats, whilst no one pays any special attention to several decayed and foul stumps, perchance with pus actually oozing from the gums, a veritable hotbed of infection.

There are cases in which a frequently recurring acute inflammation of chronically enlarged tonsils is associated with gastro-intestinal disturbance, the stools become offensive, loose, and slimy, with much mucus. This association is so constant in these particular cases that one cannot but suppose that some recurring infection of the throat leads to an infective catarrh of the stomach or bowel. I have seen several such cases and have found them most intractable.

Treatment. The first principle that I wish to lay down very emphatically is this, the mere fact that a child has adenoids or enlarged tonsils is not necessarily an indication for treatment of any kind, much less for surgical interference; the one and only one criterion of the necessity of treatment is the presence of harmful results from them. I see children in whom some one with an excess of zeal for examination, as it seems to me, has passed his finger up into the child's naso-pharynx, and finding, as he thought, that the pharyngeal tonsil is more easily felt than normal, has announced that the child has 'adenoids' and requires an operation, although perhaps admittedly they are producing only the slightest symptoms, and these possibly of doubtful relation to the adenoids, and sometimes, indeed, they are producing no symptoms at all.

I think that such advice is wholly bad: but before stating my reasons for this view, let me, in passing, say a word on the digital examination for adenoids. Is it necessary to poke one's finger into the naso-pharynx to see whether there are adenoids? It is, to put it mildly, an extremely uncomfortable procedure. To a child it is more than uncomfortable, it is terrifying; and I know only too well from experience of children who have been examined in this way before they are brought to me, that henceforth the very sight of a doctor makes them scream with fear.

Now I very much doubt whether adenoids which so far fail to declare themselves by symptoms as to require a digital examination to determine whether there is adenoid hypertrophy or not,

require any treatment whatever: always assuming, of course, that the observer has sufficient experience to know what the symptoms of adenoids are: and therefore I would lay it down as a general rule (and every rule has its exceptions) that there is no occasion whatever to poke the finger into the naso-pharynx in order to decide whether there are 'adenoids' which require treatment. Every child has adenoid tissue in the naso-pharynx; the pharyngeal tonsil is a normal part of our anatomy; the question whether it is sufficiently enlarged to require any treatment should be determined solely by the symptoms to which it is giving rise.

Now to return to my previous point that adenoid tissue, whether that in the naso-pharynx or the faucial tonsil, even when distinctly hypertrophied, does not necessarily need removal.

It is to be remembered that both the naso-pharyngeal and faucial tonsils undergo an involution, so to speak, after puberty, and this even where there have been catarrhal or inflammatory symptoms which might be thought to entail fibrous changes in them; no doubt the presence of such fibroid inflammatory change makes the involution after puberty less than it otherwise would be; but I wish to insist upon the fact that the occurrence of two or three attacks of tonsillitis, or of repeated 'colds' as happens so often with adenoids, does not preclude the possibility of considerable diminution in the size of these structures after puberty.

If, therefore, adenoids or tonsils are producing no symptoms or are associated with symptoms which are not of serious gravity, for instance, merely with a rather greater tendency to 'catch cold' than usual, or with the occurrence on one or two occasions of a tonsillitis, without any affection of the ears or other complication, there is no occasion to rush into operation.

It may be that further enlargement of these structures may occur, and produce symptoms which may make the operation necessary; but if so, the harm of waiting until this occurs is less than the harm of doing an unnecessary operation.

But, says one, if you wait, surely there is risk of ear affection, which is perhaps the most serious of all the direct evils from adenoids and enlarged tonsils? To this I would reply that one of the admitted and immediate results of the operation, an occasional one it is true, but nevertheless an undoubted one, as I can testify from my own experience, is acute otitis media, set up no doubt by the inflammatory reaction in the naso-pharynx after the operation. This, however, is one of the lesser risks. The chief reason for avoiding the operation whenever possible is

that, like other operations, it is not free from danger to life. I am assured by throat specialists that they have done so many hundreds or thousands without a single fatal result. This no doubt proves that the risk to life is a small one ; but it does not alter the fact that death has resulted. I myself could mention one in which it occurred from hæmorrhage, another apparently from sepsis, two from 'death under the anæsthetic', and another in which it appeared to be due simply to shock, and I could quote other cases from my own experience in which life was all but lost through this operation. No doubt such accidents are rare, but the rarity is no comfort to the parents when they have lost their child.

If it be true that 'death under the anæsthetic' is to be attributed to the so-called 'status lymphaticus', and if this status lymphaticus is evidenced partly, as we are told, by such an increase of lymphoid tissue as is seen clinically in enlargement of tonsils and the presence of 'adenoids', it follows that the subjects of these affections are just those on whom operation is to be avoided as far as possible.

Let us therefore realize that although in comparison with some operations that for adenoid overgrowth and enlarged tonsils is a small one, it is not entirely free from danger to life ; and therefore unless the symptoms produced by adenoids or enlarged tonsils entail grave harm to the child, operation is not to be recommended.

But what constitutes grave harm from adenoid hypertrophy and enlarged tonsils ? Recurring earache, and still more, the slightest degree of deafness, even if it only recurs when the child has a cold, is one of the strongest reasons to my mind for surgical treatment of adenoids and tonsils ; but I doubt whether one attack of earache necessitates operation ; it is quite certain that a slight catarrh in the throat may cause earache which may never recur.

In some cases the chief harm resulting from adenoid or tonsillar obstruction is general ill health : the child is pale, or of pasty complexion, partly no doubt owing to deficient aeration of the blood, but partly, I think, from lack of refreshing sleep, for many of these children with marked adenoid or tonsillar hypertrophy sleep restlessly, wake frequently, and are dull and heavy-eyed, perhaps nervous and peevish, in consequence. One of the gratifying results of operation in such cases is the marked improvement in the sleep, and I agree with parents who have told me that they thought the improvement in the child's general health was largely due to this.

Another class of cases which sometimes calls for operative procedure is that in which large tonsils are present, and there is a frequent and harassing recurrence of acute tonsillitis which can only be stopped by removing the tonsils.

Similarly there are cases in which catarrh starting in the naso-pharynx spreads to the bronchi repeatedly, so that it may be necessary to remove adenoids to prevent the recurrence of bronchitis. I have felt tempted to advise the operation in some cases of laryngitis stridulosa and asthma, where these affections were associated with adenoids and enlarged tonsils, but the effectiveness of medicinal treatment has deterred me unless there were other reasons for the operation.

Another sufficient reason for operation is deformity of the chest resulting from throat obstruction; the child with a constantly open mouth, rounded shoulders, head poked forward, narrow chest, pigeon breast or depressed sternum, is one that requires operation.

The probability of rheumatic infection through the tonsils has been mentioned; certainly it is very common to find enlarged tonsils associated with rheumatic affections. Whether rheumatic infection can be prevented by removal of the enlarged tonsils has yet to be determined. I have kept clinical notes for some years of cases bearing upon this point, but they only prove that the ordinary partial removal of tonsils does not prevent the recurrence of rheumatism in a child who has previously had rheumatism.

One serious result to which perhaps not enough importance is attached is enlargement of the glands at the angle of the jaw. Whether the tubercle bacillus gains access to these directly through the pharyngeal or faucial tonsils, or comes to them via the blood, I think there can be no doubt that the lymphatic glands, once enlarged, are particularly liable to become a nidus for tubercle; and therefore, especially in the child of tubercular family, or of known tubercular tendency, enlargement of the gland from these throat sources should raise the question of removal of adenoids and tonsils.

But here caution is necessary; the question is often not raised until the glands are already not only tuberculous but greatly enlarged, more or less evidently caseous, and perhaps on the verge of softening. It is a difficult point to decide whether under these circumstances immediate operation should be done upon the throat, even though the condition there obviously will necessitate operation sooner or later. There is a risk that by inducing

a temporary inflammatory condition in the throat the glands may be stirred into further activity and the last chance of their subsidence be extinguished.

Probably it is best to wait and send the child to the seaside under close medical supervision. If the glands subside, all well and good; when the subsidence has reached a considerable degree the tonsils and adenoids can be removed. If the glands do not subside they can be removed, and after the wound has healed and the child has been away again to convalesce, the throat can be dealt with.

One hears of cases in which such troubles as night-terrors and enuresis have been stopped by removal of adenoids or large tonsils; I confess to a certain amount of scepticism as to the relation. I can say positively that the mere presence of adenoids or enlarged tonsils does not preclude successful treatment of night-terrors or enuresis by simple medicinal measures; I have seen it again and again. On the other hand, I have repeatedly known these operations to have no effect whatever on enuresis—sometimes apparently to aggravate it; moreover, in the few instances where I have known the enuresis to stop immediately after the operation it has generally recurred subsequently.

Epilepsy, I think, is never caused primarily by enlarged tonsils or adenoid hypertrophy, but it seems only reasonable to suppose that the ill-health which is produced by any marked degree of naso-pharyngeal obstruction may aggravate the tendency in a child already disposed to epilepsy, and this, I think, is the case. I have seen a few instances in which operation on the throat under these circumstances seemed to diminish the attacks greatly.

It will be evident from what I have said that whilst the operation for enlarged tonsils and adenoid overgrowth is never to be done unless there is very grave reason for doing it, there are many cases in which such reasons are present.

But even when operation is necessary there is still need for careful consideration of the special requirements of the individual child—there is a time to operate and a time not to operate. The child's condition, apart from its naso-pharynx, must be taken into account. It is exceedingly common in all classes, and perhaps specially in these mouth-breathing children, to find carious teeth and foul stumps; the dentist should deal with such, and if possible get the mouth into a cleaner and healthier condition before any operation on adenoids or tonsils is done. As I have already suggested (p. 317), the mere removal of carious stumps and removal of sepsis in connexion with the gums and teeth may

do much to prevent the recurrence of inflammation in the fauces and naso-pharynx.

Then, again, I often see children who have recently had an attack of acute tonsillitis ; the tonsils are greatly enlarged, and the question of their removal is raised. It is, I think, never wise to recommend operation very soon after an acute tonsillitis, for the inflammatory enlargement may gradually subside within three or four months to such a degree that operation is entirely unnecessary.

When a child is in a highly nervous state, as shown, for instance, by the presence of habit-spasm, it is better to wait unless there are any symptoms which make the operation urgently necessary, for there is no doubt that it entails a certain amount of nervous shock, especially to the timid, nervous child ; I have seen habit-spasm markedly aggravated thereby, and I have known children after this operation to fall into a state of morbid nervousness, from which they did not recover for many weeks.

Another point which should be considered is the season of the year at which the operation should be done. Of course circumstances may leave us no choice of season, but where choice is possible I think it is undoubtedly safer to have it done during the warm months than in winter, for one of the sequelæ of the operation is a bronchitis apparently due to spread of the inflammatory reaction from above downwards into the air-passages.

Lastly, with regard to the operative treatment, I feel I must say a word on the actual doing of the operation, and perhaps, being a physician, and having, therefore, no active part in the operative treatment, I may speak with the greater freedom. I am constantly—I use the word deliberately—constantly seeing children for conditions, generally catarrhal conditions, connected with the throat, who are said to have had their tonsils and adenoids removed. It is obvious, both from the symptoms and from examination of the throat, that the removal has been very incomplete ; a certain amount both of the pharyngeal and of the faucial tonsils has been removed, and the stump left behind is perhaps even more ragged and irregular in surface than the original whole tonsil would have been, with the result that bacteria are still caught in its crevices, recurring catarrh with its attendant symptoms and complications still troubles the child, and the parents are grievously disappointed.

I would lay stress upon the importance of *complete* removal of the adenoids and tonsils by whatever method is found to be most practicable : the results of enucleation seem to me the most satisfactory, but I am well aware that in the hands of an expert

tonsillotomy may be to all intents and purposes tonsillectomy; anyway, I feel sure the thing to be aimed at is complete removal of the tonsils. It is not for me to dilate upon the applicability of particular methods in particular cases; I feel sure that while no man will be equally successful in all cases, the success of this or that method depends very largely upon the individual using it.

I will even venture to insist that the operation for removal of adenoids and tonsils is not so simple an operation that anybody and everybody who happens to have a surgical qualification and a guillotine can do it as it ought to be done: it is one which, if done at all, ought to be done by the most expert skill obtainable.

So much for surgical treatment. I have said already that the mere presence of adenoid and tonsillar hypertrophy is not sufficient ground for operative interference. I have also stated that a mere slight tendency to naso-pharyngeal catarrh or an occasional attack of tonsillitis is not, *per se*, sufficient justification for operation. Is there any good to be gained from medical treatment?

The slighter catarrhal conditions associated with adenoids are undoubtedly amenable to treatment by local applications. Children of about eight years or more will use successfully a lotion to be sniffed up the nostrils night and morning; this method of application is less frightening to most children than syringing. I generally direct that a small medicine glass is to be filled with the warm lotion and the child is to 'drink the lotion through his nose and spit it out through his mouth'. A useful formula for this purpose is: sod. bicarb., borax, sod. chlorid. aa gr. xv, glycerin ℥j, aq. ad ℥j (K. C. H. Pharmac.), a table-spoonful to be used with two table-spoons of warm water; or boro-glyceride, a teaspoonful in four ounces of water, may be used. Probably an alkaline lotion is generally most useful as tending to wash away mucous secretion most readily. Dr. Eustace Smith recommends dropping a few drops of a lotion down each nostril as the child lies on his back, and for this purpose advises a 1 per cent. solution of resorcin in 'normal saline', to each ounce of which 20 drops of tinct. hamamelis may be added, if the child is subject to epistaxis. For those who are not old enough or are too timid to use a lotion in this way, a very fine spray or vaporizer can be used with one of the various apparatuses sold for the purpose. The fine sprays in which an antiseptic, such as oil of eucalyptus, is mixed with an oily base, such as liquid paraffin, are particularly useful, as the child does not feel the spray as fluid, and consequently is not frightened, and the oily material remains in contact with the parts longer than a watery solution would do.

Whether the tonsils can be diminished in size by any local application is perhaps open to doubt. It is certain that they sometimes become smaller while astringents or antiseptic paints are being applied, but it is equally certain that tonsils which have become swollen by some recent tonsillitis may slowly decrease in size without local treatment of any sort, until after several months there may be practically no enlargement remaining—a point to be remembered when the question of operation is being considered. On the other hand, there are enlarged tonsils of hard fibrous appearance, the result of repeated inflammation, which are not likely to diminish in size either with or without applications. None the less I think that where tonsillar enlargement is associated with a tendency to recurring tonsillitis, a persevering application of some antiseptic or astringent twice daily for many months reduces the liability to a recurrence of the tonsillitis, and in this way, at least, favours the subsidence of the enlargement. A useful paint is tinct. hydrastis ℥j, glycerin boracis ℥ss, glycerin ad ℥j, to be applied with a camel-hair brush to the tonsils twice or thrice daily; the application should be after meals, so that the paint may not be carried away by any food or drink taken. Glycerin and tannic acid is often used similarly. A paint consisting of iodine gr. 1½, pot. iod. gr. vi, menthol gr. ½, glycerin ad ℥j, is in use at the Children's Hospital, Great Ormond Street: a similar combination with much larger doses is often used, e.g., iodine gr. v and pot. iod. gr. x in the ounce of glycerin.

In the slighter cases of adenoid and tonsil overgrowth breathing exercises may have some remedial value, partly directly by producing a healthier condition of the mucous membrane, and partly indirectly by improving the general health and so reducing the liability to catarrh. But the chief value of breathing exercises is after removal of enlarged tonsils and adenoids: too often one sees children losing in great measure the benefit of the operation because no directions have been given as to teaching the child to breathe properly afterwards, and the child continues a mouth breather when the necessity is no longer present.

Lastly, the climatic environment may have to be considered. A cold, damp atmosphere, and especially the fogs which we have in London during the winter, are potent factors in the production of repeated naso-pharyngeal catarrh, whatever may be the bacterial element. The ideal place of residence for children with the tendency to enlargement of faucial and pharyngeal tonsils is one which stands high and has a porous soil, allowing of rapid drying after rain.

CHAPTER XXIII

LARYNGITIS STRIDULOSA, AND OTHER AFFECTIONS KNOWN AS 'CROUP'

I HAVE placed at the head of this chapter the popular term 'Croup', departing thereby from present-day scientific nomenclature, and doing so deliberately, because this term is still in common use by the laity in describing a child's ailment, and is sometimes employed by medical men with such a varying significance that it is well we should realize the several conditions to which it may refer.

In former days croup was a recognized scientific term; in the medical textbooks of less than a century ago it was regularly used to signify what was supposed to be a disease *sui generis*. In Underwood's *Treatise on the Diseases of Children*, which he dedicated to the Queen in 1784, 'The Croup or Acute Asthma' is described very fully, and as alternative names are mentioned 'Asthma infantum spasmodicus' and 'Suffocatio Stridula'; like other writers about that time, he was evidently impressed with the fact that whilst in some cases the attack was harmless, in others it was very dangerous, and he mentions two stages, the earlier of which is highly amenable to treatment, whilst in the later no treatment avails.

It is evident from his description that he did not differentiate between conditions so widely different in their etiology and pathology as Laryngismus Stridulus, Laryngitis Stridulosa, and Diphtheria. He states that in the only post mortem he had seen on a case of croup there was membrane all down the trachea.

Fifty years later (in 1836), when Sir Thomas Watson was lecturing at King's College, London, on 'croup', the title of his lecture was 'Cynanche trachealis, Tracheitis, or Croup'. He laid stress on the membrane in the trachea in cases of croup; he described how certain places—for instance, the Cow Gate and Canal Street in Edinburgh—were infamous for croup, which killed a large number of children. Curiously enough, however, he regarded it as non-contagious, although he observed that it was endemic in certain places, and even when describing two children in one family as suffering from it on the same night he attributed it to walking in a cold wind.

He considered a sudden onset at night, and a remarkable tendency to recur as characteristic of the affection. Almost incidentally he referred to a disease which he said was much more common in France than in this country, a 'diphtheritis' which resembled 'croup' in producing a membrane, but this, he said, could not be the same disease, for the membrane was on the fauces primarily and only secondarily in the trachea.

On this point it is interesting to note that, according to his own statement, his opinion was not shared by Dr. Charles West, who, a few years later, founded the Hospital for Sick Children, Great Ormond Street. West regarded diphtheritis and the membranous croup as one and the same disease.

I have referred to Sir Thomas Watson chiefly because he mentions what he calls 'a sort of bastard croup', which, he says, had already been recognized by various observers as a distinct malady. He says that one Dr. Good bestowed upon it 'the somewhat pedantic and cacophonous title Laryngismus Stridulus'; he prefers to call it 'child-crowing', whilst others, he says, have called it 'cerebral croup', and others again 'spasmodic croup'; under these titles he gives a good description of what we know now as the laryngismus stridulus associated with rickets.

West in his *Lectures on Diseases of Children* comes very near to making a further differentiation between these conditions known as croup, but failed owing to his preconceived idea that in the early stage of what he calls 'true croup', laryngeal, or tracheal diphtheria, the obstructive dyspnœa is due chiefly to spasm of the larynx, whilst later the inflammatory symptoms predominated with formation of membrane. He tells how some observers had thought that there was a group of cases which were quite distinct from diphtheria, cases in which a spasmodic laryngitis was present, but in which the inflammatory process played a much less prominent part than the spasm; he dismisses this view as untenable on the ground that the difference depends merely on the idiosyncrasy of the patient, and he mentions in this connexion almost incidentally the name 'Laryngitis Stridula' which had been used by a Monsieur Guersant in 1835 to distinguish this spasmodic condition, which we now recognize as absolutely distinct from diphtheria under the name 'Laryngitis Stridulosa'.

This brings me to our present position as regards 'Croup': when we are told by a mother that her child has the croup, what may be the significance of the term? For the mother as for the older physicians, croup is any condition in which the cough has the ringing, barking, almost brassy character of a laryngeal

cough, or in which there is stridor. There are four common conditions which are included thus in the category of 'croup': (1) Diphtheria, (2) Simple Laryngitis, (3) Laryngitis Stridulosa, (4) Laryngismus Stridulus.

With regard to diphtheria there is only one point I need mention here. When the likeness between the membrane on the fauces in ordinary faucial diphtheria and the membrane in the larynx and trachea in cases of 'croup' was first observed, it was held that although the faucial membrane might extend down the air-passages from above, there was no such thing as a diphtheritic affection starting in the larynx or trachea, and so the latter occurrence continued to be regarded as distinct from diphtheria. Undoubtedly it is quite uncommon for diphtheritic membrane to form first or only in the larynx or trachea, and even up to quite recent years it was still disputed whether such an occurrence ever took place. It seems certain now that it does, but perhaps more rarely than is supposed, for some of the statistics on this point are based on post mortem examination, and it must never be forgotten that these tell us only about terminal conditions. It is not safe to conclude from post mortem examination that because there is only membrane in the larynx or trachea, there has never been any in the fauces; it is clear from clinical evidence that the faucial inflammation in diphtheria may disappear before the laryngeal appears, and it is noteworthy—for it is a point of great clinical importance—that even when there is no faucial membrane to be seen, it may be there none the less, for sometimes at post mortem examination membrane is to be found only on the upper or posterior surface of the soft palate where it would be invisible by ordinary inspection during life; in such cases there is usually, if not always, a nasal discharge which may help in the diagnosis.

In any child with croupy cough and stridor, even if no membrane be visible on the fauces, the possibility of diphtheria is to be considered, the presence of nasal discharge, of albumen in the urine, the history of exposure to infection, and the finding of diphtheria bacilli in the swabbing from the fauces, although no membrane is present there, may point to diphtheria of the larynx or trachea. It must be admitted, however, that in the absence of membrane from the fauces it is not uncommon for the swab to prove negative, although subsequent events may prove that there is diphtheritic membrane in the larynx.

Simple acute laryngitis is sometimes the meaning of 'croup'; it is often extremely difficult to diagnose from diphtheria; in fact, one has to judge almost entirely from the absence of those

indications which I have just mentioned as pointing to diphtheria. In infancy a simple laryngitis causes much more severe symptoms than are usual at a later age: the temperature may rise to 103° or 104° F., and in addition to the hoarseness of the cry there may be definite stridor and paroxysmal attacks of dyspnœa, which are generally worse at night.

A practical point to be remembered in diagnosis when a child becomes hoarse and stridulous with a high temperature, and the mother says her child has 'croup', is the occurrence of these symptoms as part of the invasion of measles. I have repeatedly seen this acute laryngitis of measles mistaken either for ordinary catarrhal laryngitis or for diphtheria, and have seen tracheotomy done under the impression that the child had laryngeal diphtheria, whereas the appearance of the measles rash twenty-four hours later showed that the symptoms were due to the pre-measles laryngitis.

This laryngitis may be the very first symptom of the invasion of measles; it is sometimes of considerable severity, but very rarely calls for intubation or tracheotomy; the following case illustrates the difficulty of diagnosis in such a case.

Maud J., aged five years, was perfectly well until the evening of May 13, when she was suddenly seized with intense dyspnœa and loud croupy cough; she became blue and 'fought for breath'; the dyspnœa diminished after a short time; on the evening of May 14, the child was admitted to hospital; the temperature was 103°, there was no definite coryza; there was no membrane to be seen on the fauces, there was considerable inspiratory stridor, the lower part of the chest was sucked in with inspiration; the cough was of brassy laryngeal type. With a steam-kettle, the breath became easier for a few hours, but soon became more laboured with loud stridor and croupy cough, and the child was restless; it was thought that the laryngitis was diphtheritic; tracheotomy was proposed, but was postponed as the breathing again improved. On the morning of May 16, there was a well-marked measles rash, and the laryngitis was much less.

The stridor and obstruction to respiration in these cases usually subsides as soon as the rash appears; it is worth while, therefore, deferring tracheotomy as long as possible, even if the difficulty in breathing is severe; and where intubation is practicable it is certainly to be preferred to tracheotomy. The nature of the laryngitis in such cases may be quite unrecognizable until the rash appears, but, since the case I have described above occurred, a valuable aid to diagnosis has been discovered in the sign known as 'Koplik's Spots', bluish-white minute spots which are to be seen with a good light on the buccal mucosa, and sometimes on the inner surface of the lower lip near the

angles of the mouth; these appear two days, and sometimes three days, before the beginning of the measles rash, and so may be of great assistance in determining the nature of a doubtful laryngitis. Whilst referring to the laryngitis which precedes measles, I must mention also the laryngitis which sometimes occurs at a later stage when the rash is already fading. An acute simple laryngitis is not very rare, but there seems to be some special liability also to diphtheria at this time, so that a 'croupy cough' after measles must be regarded with suspicion.

Another infectious disease with which laryngitis is sometimes associated is whooping-cough. During the early catarrhal stage which precedes the appearance of the characteristic whoop, the larynx sometimes shares in the catarrh of the respiratory tract; I know of no way in which the significance of the laryngitis can be recognized in such cases, but to be forewarned of the possibility is, at any rate, to be forearmed against surprise.

Albert D., aged two years, was brought to hospital with a 'croupy cough on September 2; there was nothing abnormal to be seen in the throat; the eyes were rather suffused, and the onset of measles was suspected, but there were no 'Koplik's Spots' to be seen. The temperature was 103·2°; the cough was hoarse and laryngeal. On September 7 the child was brought again with obvious whooping-cough.

Laryngitis stridulosa. Perhaps more often than to any of the foregoing conditions, the term croup is applied to a disorder which is peculiar to childhood, and is distinctive on the one hand from diphtheria and, on the other hand, from simple laryngitis, the so-called laryngitis stridulosa. This disorder is sometimes spoken of as 'spasmodic croup' or false croup; the latter term referring to the days when true croup was identical with diphtheria.

Laryngitis stridulosa rarely occurs in children over the age of ten years. Amongst twenty-six consecutive cases under my care, only one was in his eleventh year, one in his tenth year, and three in their ninth year; the others were all between the ages of three and eight years when they were brought for treatment. The attacks date usually from some time between the end of the first year and the end of the fifth year of life; only two of my cases were said to have had attacks before they were a year old. It is generally said that boys are more liable to this affection than girls; my own figures show no great preponderance of boys: there were sixteen boys and ten girls.

The symptoms in a typical case are these: the child goes to bed apparently perfectly well; some time between 10 p.m. and

2 a.m. the child wakes with a hard brassy cough, which in several of my cases was described as being 'like a dog barking'; at the same time, or immediately afterwards, inspiration is accompanied with a loud stridor which may be heard easily in the next room; the child is in a state of urgent dyspnœa, 'fighting for his breath', with beads of perspiration standing on his forehead, and sometimes with the lips livid and blue; he may be in a state of terror at what appears to be impending suffocation; twice I have been told that the child 'felt as though his throat was closing'. After one to two hours of this distress the stridor gradually becomes less, the dyspnœa subsides, and the child falls asleep exhausted, and wakes next morning quite well. Thus ends the attack only to recur again with exactly similar symptoms after an interval of one to two months.

From this typical course there is some variation in particular cases. Usually the child has gone to bed perfectly well, and the attack comes on with no warning whatever; but this is not always so: sometimes there are premonitory symptoms. One girl was noticed to be specially excitable before each attack; some showed slight hoarseness or had coughed a little during the preceding evening. The time of attack varies remarkably little, the onset is seldom earlier than 10 p.m. or later than 2 a.m. It fell outside these limits only in five out of twenty-two cases; three of these had attacks beginning about 5 a.m., one at 8 a.m., and one began an attack at 5 p.m. The duration of the attacks varied considerably: in a boy aged four years the attacks were said to last sometimes 'only a few minutes', whilst at other times they lasted much longer; in two cases the child had the symptoms more or less marked for three days and nights continuously; in four cases the child was quite well all the following day, but had a return of symptoms each night for two or three nights in succession. The interval between attacks varies greatly in different children: in one case, for instance, it was six months, in another the attacks occurred once a week for three months; usually the interval is about four to six weeks.

During the attack the voice is sometimes hoarse, but may be quite clear in spite of the loud stridor. Examination of the chest at this time may show some râles and rhonchi; in several of my cases there was a slight bronchial catarrh persisting for a few days after the attack. The temperature is probably normal in most cases, and if raised at all, the elevation is usually slight.

Etiology. Cold is certainly one factor in the causation of

this disorder. It was noticed in several of my cases that walking against a cold wind would induce an attack; in one case it was stated that the child had the attacks only in the winter, and in another that the attacks were limited to the winter unless an exceptionally cold day occurred in the summer, when the 'croup' might occur. Another exciting cause is over-eating: one girl aged eight years was said to have these attacks whenever she ate more freely than usual.

In addition to these exciting causes I think there can be little doubt that there is an underlying nervous instability which predisposes to this disorder. I have specially noted that some of my cases showed definite evidence of such instability in stuttering, night terrors, enuresis, abnormal excitability, and general 'nervousness', and I would lay considerable stress on this element, for it may be the link connecting laryngitis stridulosa with asthma, to which I believe it to be very near akin. The parallel indeed between these two disorders is so very close that it is almost impossible to escape the conclusion that if the one is a respiratory neurosis, the other is also; the sudden onset at night, the fact that in both disorders a cold wind or an overloaded stomach seems equally capable of exciting an attack, the striking effect of climate on both disorders, and lastly, as I shall show, the similarity of treatment required, all these points suggest that asthma and laryngitis stridulosa are of very similar nature. There is, I think, further evidence of affinity in the fact that asthma occasionally replaces laryngitis stridulosa as a child grows older: in a boy aged seven years, the history seemed to show that the asthma from which he had suffered repeatedly in the past two years, had replaced attacks of laryngitis stridulosa, which occurred at intervals from the age of two years up to the age of five years.

Like asthma, this laryngeal spasm may be started by any catarrh of the respiratory passage, and in this way the presence of adenoid overgrowth in the naso-pharynx, which is so often associated with a great tendency to recurring catarrh extending sometimes down into the larynx and sometimes into the bronchi, no doubt contributes to the perpetuation of the attacks. But I have been entirely unable to satisfy myself that the presence of adenoids is in itself sufficient cause for the attacks, and I have repeatedly known removal of adenoids and the excision of large tonsils in such cases to fail completely to effect a cure.

Prognosis. Is there any danger to life in these attacks? Certainly they look alarming enough, but I know of no fatal

result, and beyond the prostration which is sometimes left for a day or two after a severe attack, I know of no harm from them. Trousseau mentions a fatal result in three cases, but it must be remembered that in his time the differentiation from diphtheria was less accurate than at the present day.

The duration of the disorder varies greatly; in some children after two or three attacks within a few months it ceases to recur; in others it lasts for several years—for example, in one of my cases for five years, in two for seven years; but I have never known it to persist beyond the age of eleven years, and it rarely lasts so late as this.

Treatment. To some degree these attacks are preventable, as may be gathered from what I have said as to their causation. The child who is prone to laryngitis stridulosa is not to be 'taken out in all weathers'; in particular, he is not to be taken out in the teeth of a bitterly cold wind. On the other hand, I believe that in this disorder, as in asthma, the liability to attacks may be increased by shielding the child 'like a hothouse plant' from every variation of temperature and from every wind that blows; he should indeed live an outdoor life as much as possible, but with the special precaution that he is not exposed to cold winds or to cold fog. The diet, too, will need care; any overloading of the stomach and overtaxing of digestion is to be avoided, as likely to bring on an attack.

The drug treatment which I have found most effectual is very similar to that used in asthma. Arsenic has a marked effect in diminishing the frequency of the attacks, one minim of the liquor arsenicalis may be given to a child of three years, and 1½–2 minims to a child of seven years three times a day for three weeks, and after an interval of ten days or a fortnight the drug may be resumed, and so on for three or four months. I have also used stramonium with arsenic or with potassium iodide when the attacks were frequent; 5 minims of the tincture of stramonium with 2 or 3 grains of potassium iodide and 10 minims of sal volatile with syrup and water make a suitable mixture for a child of six years.

When the attack occurs there are several simple measures which may give relief: moistening the atmosphere by a steam-kettle has seemed to give definite relief in some cases; one mother told me that a hot mustard bath was very successful for these attacks in her child aged eight years; hot applications such as a sponge squeezed out of hot water, or a poultice or fomentation over the larynx seemed to relieve the spasm in

others. In several cases speedy relief was given by making the child vomit with drachm doses of ipecacuanha wine.

I should have expected that suprarenal extract which in the form of adrenalin chloride has been found to give almost immediate relief in severe attacks of asthma, would prove useful also in the attacks of laryngitis stridulosa, and quite recently it has been stated that this is the case; 1 or 2 drops of the adrenalin chloride solution (1 in 1,000), as prepared by Messrs. Parke, Davis & Co., is given by mouth in a drachm of water, and is said to give ease very rapidly.

The effect of climate in this disorder is very marked, but I fancy that, as in asthma, there is no rhyme or reason by which we may foretell with any degree of certainty what place is likely to suit a particular child, we can only advise on general principles; seaside seems to suit most of these cases better than inland, and in order to avoid cold and boisterous winds it is well to choose the warmer seaside places, at any rate those which come in for as small a share as possible of the cold east winds. I have noted, for instance, Eastbourne, Worthing, and Penzance as south coast resorts at which children with this disorder remained free from attacks.

Laryngismus stridulus. I have mentioned this condition as one of those sometimes spoken of as 'croup'. It belongs more properly to the convulsive disorders, and I shall describe it more fully in a later chapter; but I must refer to it here partly because of its figuring in popular parlance as 'croup', and partly because its 'pedantic and cacophonous title' bears such an unfortunately close resemblance to laryngitis stridulosa, that there is apt to be some confusion between them.

Laryngismus stridulus or child-crowing really bears little or no resemblance to laryngitis stridulosa in its symptoms. It is limited to the first three years of life (I have seen it occasionally as late as $3\frac{1}{2}$ years) and consists in a momentary spasm of the glottis, which usually lasts only about five seconds, and recurs several times a day, especially just as the child awakes from sleep or when he begins to cry. Laryngismus stridulus is so closely associated with rickets that it may almost be regarded as one of the manifestations of rickets; it is associated also closely with convulsions and tetany, and is readily amenable to the treatment of rickets and convulsions. In all these points it differs entirely from laryngitis stridulosa, with which indeed it could hardly be confused, except in name.

These attacks are seen in rickety infants mostly between the

ages of six months and eighteen months. The mother's description is that the infant 'holds his breath and goes black in the face' and then 'makes a funny noise' which I find described in my notes as 'like a whoop' or a 'crowing noise'. This happens at any time of day, sometimes many times a day, most commonly on some extra inspiratory effort as in crying; but at times it happens with no apparent reason. In addition to the rickets, the infant with laryngismus stridulus almost always shows nerve irritability, which is easily demonstrated by tapping over the facial nerve or the musculo-spiral or the anterior tibial (see Chapter XLV), and in many cases also some craniotabes and tetany, and a great liability to convulsions.

The attacks of laryngeal spasm are not free from danger, I have known sudden fatal asphyxia to result in at least three cases under my own observation. They are, however, readily amenable to treatment in most cases; a mixture of potassium bromide gr. iij with a drachm of cod-liver oil emulsion given three times a day causes rapid subsidence of the attacks.

Other causes for stridor in Children

Lastly, I must point out that there are some rarer causes of stridor which may have to be considered in the child who is said to have 'croup'.

Laryngeal spasm in the newborn has come under my notice several times, and a most alarming disorder it is. The first case I saw was a female infant aged six days who had been having attacks occasionally during the previous twelve hours. Two attempts to see the throat brought on severe attacks: the infant made violent inspiratory efforts without getting any air in, became more and more leaden-coloured, bloody froth appeared at the mouth, and the child appeared to be dying of asphyxia. These attacks occurred sometimes without apparent cause, sometimes when the infant tried to take the breast; nine such attacks occurred within a few hours after I saw the infant, and she died apparently of exhaustion on the evening of the same day. Post mortem examination showed some congestion of the pharynx and soft palate, suggesting that there had been some degree of pharyngitis, but there was no exudation and nothing else to account for these attacks.

Another case was a boy, aged 7½ weeks, who had been subject to sudden 'crowing attacks' since birth; two of these had been very severe, the infant became cyanosed, 'unable to get his breath', and seemed likely to die in the attack. In this case

the attacks came on quite suddenly with no apparent cause. There had been some trouble with digestion, and the infant was now put upon peptonized milk, and no further attacks of the laryngeal spasm occurred.

Evidently such cases are very similar in their pathology to the laryngismus stridulus which is seen with rickets in older infants, but in these younger cases there is no suspicion of rickets. The late Dr. Eustace Smith [1] drew attention to this disorder of the newborn; he found in three cases adenoid overgrowth and post-nasal catarrh. In mild cases a weak resorcin solution should be instilled into the nostrils several times a day. In the attack respiration must be stimulated by sprinkling the child's face or chest with cold water or holding smelling-salts to the nostrils; in one attack I held some lint sprinkled with ether to the infant's nostrils, apparently with good effect; amyl nitrite capsules (1 minim) might also be useful in such a case.

Congenital laryngeal stridor. In the new-born infant there is the curious disorder known as congenital laryngeal stridor. Ten cases of this affection have come under my own observation, six were girls, four were boys. The stridor was noticed on the day of birth in all except one, in which it was first noticed on the third day, perhaps because the infant was feeble and breathing less vigorously during the first two days.

The character of the stridor is peculiar. I find that in most of my cases it was compared to the noise made by a hen in 'clucking', whilst in others it was called 'croaking', or 'crowing'. It is inspiratory, and occurs with every inspiration; during much exertion or excitement expiration also is accompanied by slight stridor in some cases. I have noted that in one case it was audible at a distance of 12 feet, in another that it was 'heard easily 23 feet away from the infant'; but it varies considerably at different times in the same case; so long as the infant is lying quiet it may be scarcely audible, while directly the infant is disturbed or excited and takes deeper respirations the crowing may be loud with every inspiration. The stridor persists during sleep in some cases; in one I have noted that it was heard during light sleep, but disappeared when the infant was soundly asleep.

The cry is normal; there is no hoarseness. The infant is not in the least distressed by the inspiratory difficulty; there is no cyanosis, but the obstruction to air entry is evident sometimes

[1] *British Medical Journal*, July 20, 1907.

in the retraction of the lower intercostal spaces, and the sucking in of the suprasternal and supraclavicular fossæ.

The affection looks a far more serious one than it is; as far as I know, only one of the ten cases proved fatal (from broncho-pneumonia). Dr. John Thomson,[1] to whose writings the recognition of this disorder is mainly due, states that in most cases the stridor passes off before the end of the second year.

There is some variation in this: in one female infant, whom I first saw at the age of three months with marked congenital laryngeal stridor, the noise with inspiration was only occasionally heard when the child was eleven months old, and at twenty months it was not heard at all during ordinary breathing, but I noted that after the exertion of crying inspiration was still rather 'heavy'; in a girl, whom I first saw at 9½ months, the stridor had become extremely slight at the age of 2½ years, but was still quite noticeable on exertion; in a third case, also a girl, the stridor which had been present since birth was still quite distinct at 3½ years.

Allowing for this variation, we may assure the parents that in all probability within two or three years the disorder will pass off without causing any harm; the one danger is some intercurrent pulmonary disease in which these infants are apt to fare badly, as might be expected where respiration is hampered not only by pulmonary disease but also by laryngeal obstruction. In the earliest observation on the morbid condition, by Dr. D. B. Lees,[2] death was due to diphtheria; Dr. Koplik[3] has recently recorded one fatal ending from broncho-pneumonia at the age of one year. In both these cases, as in some others recorded by Dr. Thomson and Dr. Logan Turner,[4] the upper opening of the larynx has shown in an exaggerated degree the narrowness of opening which is peculiar to the infant, and corresponding therewith an unusual degree of the normal infantile condition of longitudinal folding of the epiglottis.

Are these peculiarities, which are presumably the cause of the stridor, a congenital deformity? or are they the result of 'ill co-ordinated and spasmodic nature of the breathing', as Dr. Thomson and Dr. Turner have shown by experiment to be possible? I have only examined the larynx after death in one case myself, and I must say that to my mind a congenital exaggeration of the normal peculiarities of the infantile larynx seems ample explanation of the clinical features of the disorder;

[1] *Edinb. Med. Journ.*, Sept., 1892.
[2] *Pathological Society Trans.*, 1883.
[3] *Archives of Pediatrics*, Dec., 1905.
[4] *Brit. Med. Journ.*, Dec. 1, 1900.

the occurrence of the stridor on the day of birth, its continued character, varying with the depth of inspiration, and its gradual diminution as the laryngeal aperture enlarges with the growth of the child, all seem compatible with a simple congenital peculiarity of conformation in the larynx. It is likely enough that the obstruction may set up some degree of inspiratory spasm which may aggravate the deformity of the larynx, but it seems to me unnecessary to suppose that the spasm is the primary cause of the condition.

It is well to caution the parents that special care is to be taken to prevent bronchitis in these cases; the slightest 'cold' is to be treated seriously, and if any bronchitis occurs the child should be placed at once under the doctor's care. If there is very severe bronchitis, or broncho-pneumonia with great difficulty of respiration, I believe the wisest procedure may be intubation, but this is likely to be specially difficult owing to the unusual narrowness of the laryngeal opening, and should only be attempted by those who are thoroughly experienced in intubation.

Enlargement of mediastinal glands is an occasional cause of stridor in children. For instance, Catherine G., aged 5½ years, was brought to King's College Hospital for a 'croupy cough' which had been present about eight weeks, during which time she had also made a crowing noise in breathing. Examination showed in addition to loud inspiratory stridor, marked dullness at the inner end of the first and second right spaces, and bronchial breathing over the greater part of these two spaces in front, whilst just above the inner end of the clavicle an enlarged gland the size of a hazel-nut could be felt.

A skiagram showed that there was some abnormal shadow to the right of the middle line at the upper part of the chest, but it was not possible to distinguish whether this was due to consolidation of lung only, or partly to enlargement of mediastinal glands, as seemed most probable. Sir St. Clair Thomson, who kindly saw the child for me, thought there was certainly compression of the trachea by something in the mediastinum. The child was put upon potassium iodide, and, whether as a result of this or not, the stridor gradually disappeared, so that thirteen days after admission the stridor was only very slight and the dullness at the right apex had almost gone, nor was there any bronchial breathing. A month after admission the stridor had completely gone and the child remained free from any recurrence of the respiratory obstruction during the several weeks she remained under observation.

In another case a boy, aged $2\frac{3}{12}$, had had difficulty of breathing with some respiratory stridor for about a month, his voice was perfectly normal, there was markedly diminished air entry at the apex of the right lung behind; skiagrams showed what were thought to be undoubtedly enlarged mediastinal glands on the right side of the mediastinum. He was taken to the seaside, and although for a few days after I saw him the difficulty in breathing became extreme it soon diminished and gradually disappeared completely; the boy seemed quite well when I heard of him nine months later.

These two cases are interesting as showing that even when mediastinal glands are already large enough to cause compression of the trachea, they may, nevertheless, diminish under treatment, and the child may remain well, at any rate, for months, perhaps for years.

Recovery, however, from the respiratory obstruction in these cases must not make us too sanguine, for the fact that tuberculosis has occurred in the mediastinal glands—as symptoms running this course must always indicate—points to a diminished resistance to tubercle, and therefore to the possibility, if not probability, that at some future date tuberculosis will declare itself in the lungs or elsewhere.

Syphilis. Very rare cases have been described in which stridor was due to syphilitic stenosis of the larynx. I have seen one case of stridor due to cicatricial narrowing of the trachea from syphilitic disease; it occurred in a boy at the school age, and was associated with other evidences of severe syphilis in earlier life.

Papilloma of the larynx. Another rare cause for stridor, though more common in children than in adults, is papilloma in the larynx.

The following case shows how misleading the symptoms may be, resembling in their sudden onset an acute laryngitis or diphtheria, and also how dangerous the condition is.

Eliza R., aged $3\frac{1}{2}$ years on March 4, began suddenly one night to make a stridulous noise with inspiration, and at the same time her voice became a hoarse whisper; these symptoms continued, and on April 22, she was brought to me as an out-patient at hospital, seeming quite well, with no discomfort, but still with marked stridor on inspiration, and hoarse whispering speech. That same night she was suddenly seized with suffocative symptoms and died within a few minutes. Post mortem, made by Dr. O. Hildesheim, showed a large papillomatous growth in the larynx.

In some of these cases attacks of dyspnœa recur many times without proving fatal, but in all the outlook is grave, and the only hope of recovery lies in operative treatment.

Retro-pharyngeal abscess is another condition which must be borne in mind in any case of apparent 'croup' in infancy or childhood.

As an acute affection, this almost always occurs during the first two years of life, and is then generally due to some infective process in the fauces or adjoining parts, causing suppuration in the small lymphatic glands which lie behind the pharynx over the bodies of the cervical vertebræ or else in the glands at the side of the neck from which the suppuration extends inwards towards the middle line.

In later childhood a chronic retro-pharyngeal abscess may occur from tuberculous disease of the vertebræ, but this is much less likely to give rise to any symptoms which might be mistaken for 'croup'.

In the case of the acute infection the abscess is generally well above the level of the larynx, and the symptoms produced in addition to fever and constitutional disturbance are difficulty of swallowing and some alteration of the voice like that of a child with greatly swollen tonsils. Stridor is most likely to occur when the abscess is rather lower than usual, particularly when it is about the level of the upper part of the larynx. The inflammation and œdema may then extend to the larynx and produce symptoms like those of acute laryngitis, marked inspiratory stridor with recession of the chest wall, owing to the difficulty of inspiration, and sometimes repeated more acute suffocative attacks in which life appears to be threatened. I have seen such a condition exactly simulating a diphtheritic laryngitis. The diagnosis is extremely important, for if the abscess be not recognized and opened it may kill by asphyxia, either owing to the œdema of the larynx or from rupture and the sudden flooding of the air-passages with pus. Rarely it spreads downward to the mediastinum, and pulmonary troubles ensue.

Foreign body. As a possible cause of stridor, a foreign body in the air passages must be remembered; the following case illustrates this, and shows also how misleadingly slight may be the symptoms at the time when the foreign body first enters the air passages:

Sidney B., aged 10, had been holding a hobnail in his mouth, at school, when a boy suddenly ran at him, and, taken by surprise, he 'swallowed' the hobnail. He only coughed slightly at the time, and after going into class, he thought he had better tell the teacher. He put up his hand to attract the teacher's attention, but was told to put it down again; so he went on with his lessons without informing any one. Some weeks later he was brought to hospital for inspiratory stridor, which had been present since the supposed swallowing of the nail.

The stridor was distinct, but not loud; it could be heard a yard from the boy. X-rays showed a hobnail with the point upwards in the left bronchus. A tracheotomy was performed, and with the aid of the X-ray screen, my colleague, Mr. Burghard, was able to withdraw the hobnail by a long forceps. The boy made a complete recovery.

A foreign body in the œsophagus is also able to produce stridor by pressure on the adjoining trachea:

David W., aged 4 months, was brought to me with a marked stridor, which had been present for a week, and was said to be absent when the child was asleep. I was puzzled by the unexplained commencement of stridor in an infant at this age and apparently in perfect health. Subsequently, however, as the child's breathing was embarrassed, a skiagram was taken, and showed a foreign body, which was thought to be in the trachea; tracheotomy gave no relief, and a further X-ray showed that the foreign body was still present and it was found to be in the œsophagus. All efforts, however, to remove it failed, and the child died. At autopsy a coin, nearly as large as a halfpenny was found firmly impacted in the œsophagus, about the level of the cricoid cartilage.

CHAPTER XXIV

ASTHMA IN INFANCY AND CHILDHOOD

I HAVE chosen the title at the head of this chapter deliberately to emphasize the fact *in primis* that asthma is an affection of the earliest years, nay, even of the earliest months, of life. True it is but seldom recognized in infancy, but it is overlooked chiefly because its occurrence at this age is not generally realized. Nor is the recognition of asthma in early life a matter of mere academic interest; it is a matter of vital importance, for the emphysematous changes which asthma is so apt to produce in the lung at all ages, and which may render a child a chronic invalid before he is yet in his teens, are specially liable to result when asthma occurs in very early life, and is not checked by proper treatment.

The early age at which asthma begins is well shown by the following table, compiled from consecutive cases in my note-books. In 69 out of 78 the first appearance of the asthma could be dated with some degree of accuracy; in 47 out of the 69 the onset was under the age of three years:

Age at onset.	*No. of cases.*
3 weeks to 1 year	9
1–2 years	21
2–3 years	17
3–4 years	6
4–5 years	6
5–6 years	0
6–7 years	3
7–8 years	4
8–9 years	2
9–10 years	1
10–11 years	0
11–12 years	0

It is not always easy from the history to assign a date to the first appearance of asthma, especially when it has begun in infancy, for its nature has often been unrecognized at this period; the affection has been regarded as a simple bronchitis of rather wheezy character until its frequent repetition at short intervals, and the unusually sudden onset and perhaps as sudden disappearance, raise a suspicion that it may be asthmatic; often indeed it is not until the child grows older, possibly at five or six years, when the attacks have already assumed the charac-

teristic features of the full-blown disease, that the nature of the earlier attacks is recognized. Almost all the cases grouped in the above table as having begun under the age of one year were instances of this posthumous diagnosis, but if the fact of the occurrence of this immature asthma in infancy were borne in mind, there is no reason why the diagnosis should not be made much earlier than it usually is, for the bronchitis has certain features which even lay parents recognize on looking back as more or less closely resembling the more characteristic attacks of a later age; one might add that the resemblance is sometimes more obvious when the wheezing rhonchus of the infantile asthmatic bronchitis is, as it sometimes is, chiefly expiratory.

In one case seen for typical paroxysmal asthma at the age of 8½ years, the mother was confident that the first attack had occurred at the age of three weeks, and that others of the same nature had occurred throughout infancy; in another the first attack was at six weeks old, in another at three months, and in two others at four months. All these had developed more characteristic attacks of asthma as they grew older.

In view of this very early onset—which is probably commoner than my statistics show—it seems likely that congenital predisposition plays some part in the causation of asthma.

Sex. It is difficult to see why asthma should be so much commoner in boys than in girls; my own figures show 52 boys to 26 girls, a proportion of 2 to 1. This sex-incidence is interesting when compared with that of laryngitis stridulosa (spasmodic croup), which shows a similar predominance of males, and which has, as I shall show, several points of contact with paroxysmal asthma.

Predisposing Causes. Asthma and hay-fever figure so largely in the family history of the asthmatic child that one cannot doubt that heredity is an important factor in its etiology. In 21 out of 49 cases in which the family history was noted there was asthma or hay-fever in one or more near relations (parents, uncles, aunts, or grandparents), in 6 of these 21 there was asthma or hay-fever in mother or father. Such histories as these are common enough:

Gordon M., aged 10 years, has had asthma since the age of 3½ years; his maternal grandfather and a maternal aunt had asthma, another maternal aunt had hay-fever.

Marjorie M., aged 5 years, began to have asthma at the age of 20 months; her father has hay-fever, a paternal aunt and also a maternal aunt had asthma, the maternal grandfather had gout.

Gouty inheritance has been thought to play some part in the causation of asthma, but I have not been able to satisfy myself that this is so. Out of 47 cases only 4 gave a family history of gout.

Some have thought that the gouty taint is evidenced by infantile eczema, which appears with perhaps more than average frequency in the history of the asthmatic child, but this also is not shown very clearly by my own figures, in which only 7 out of 78 asthmatic children had had eczema. In all these cases the eczema occurred in infancy or early childhood, and had preceded the asthma by some months or years. These statistics probably to a slight degree understate the frequency of this association, for special inquiry was not made in all cases.

A much more evident predisposing factor is nervous instability, showing sometimes in the family history, sometimes in the child. I have noted again and again that the child brought for asthma was an unduly nervous or excitable child; in some there was stuttering, in some habit-spasm, one was under treatment for hysteria, one had epileptiform attacks, one cyclic vomiting, in others headache, night-terrors, fainting, or head-banging were noted.

What part, if any, is played by enlarged tonsils and adenoids in the causation of asthma is an important question; there is no doubt that these are present in some children with asthma, but not, I think, in larger proportion than is found amongst children who have no asthma. Out of 71, 3 showed definite enlargement of tonsils with or without 'adenoids', 3 showed only adenoid hypertrophy, 13 had already been operated upon for more or less enlarged tonsils or 'adenoids', mostly in the hope of curing the asthma thereby. Only in 1 out of these 13 cases had the operation been of the slightest value, and in this one the good effect was only temporary. It seems likely enough that enlarged tonsils and hypertrophied adenoid tissue encourage frequent outbreaks of nasopharyngeal catarrh, and may thereby serve as a starting-point for asthma in a child of asthmatic tendency, but the results of operation do not prove this.

The **exciting causes** of asthma throw perhaps as much light upon its character as anything, for such a motley group is surely consistent with nothing but a neurosis. In one child the smell of tar always starts an attack, in another the near presence of a cat or a horse, apparently by some emanation or smell, is sufficient cause, in two of my cases violent laughing would produce an attack; sitting in a hot or stuffy room, walking against the

wind, especially if it be cold or easterly, an outburst of passion, over-fatigue, any special excitement, indigestible food and over-eating, all these were amongst the causes assigned.

In some cases I have thought that asthma was induced by the fine dust or pollen carried by the air from hay and flowers, as apparently happens in the very closely allied condition hay-fever.

Almost as bizarre in its inconstancy is the causal relation of climate to asthma; the place which is the salvation of one child with asthma may be the undoing of another, and the same child may enjoy complete freedom from attacks at a place which is but a bare mile or even less from one where the asthma was most severe.

Relation to other Respiratory Affections. It is no uncommon story that the first attack of asthma began soon after a bout of 'congestion of the lungs', or 'double pneumonia', or 'severe bronchitis'. Is this a coincidence, or is there some causal connexion? It is quite conceivable that, given a congenital instability of the respiratory nervous mechanism, any acute infection of the lungs or air-passages may leave the respiratory tract in a morbidly sensitive condition, so that spasm of the bronchi—if that be the essential change in asthma—occurs on very slight provocation. But such histories must be received with caution; if it has not already been recognized that the child is asthmatic, it is easy to make much of the catarrhal symptoms, which may be very marked, and to overlook the spasmodic element, which may be much less obvious. Given a child, say of two or three years, with râles and rhonchi all over the chest, and a temperature of 102°–103°, it is natural enough to diagnose 'congestion of the lungs' or acute bronchitis, or to suspect some bronchopneumonia, although the unusually quick subsidence of the symptoms may raise doubts in the medical man's mind at the time, and subsequent attacks show more and more of the spasmodic symptoms of asthma.

It is noteworthy that whilst asthma is perhaps in the majority of cases not associated with any rise of temperature, it is sometimes accompanied by considerable fever, and may vary in this respect in different attacks in the same child, some being febrile, some not. I have thought that fever is more often present when catarrhal signs are a prominent feature.

Probably more than a chance antecedent of asthma, though much less common than one might have expected, is laryngitis stridulosa, 'spasmodic croup'.

Lillie K., aged $7\frac{1}{12}$, suffered at the age of 2 years with attacks called 'croup', with barking and loud inspiratory stridor; the attacks occurred at night, and sometimes on several successive nights. Since the age of 3 years the attacks have altered in character; she now wakes in the night with a choking dry cough and much wheezing, with signs of bronchitis; her lungs are markedly emphysematous.

Fred M., aged 7 years, had attacks of 'croup' from the age of 2 years until 5 years old; about 10 p.m. he awoke, 'making a noise like a dog barking,' and having considerable difficulty in breathing; the attack lasted several hours. At 5 years old he had an operation, apparently for adenoids, and a few months later had 'pneumonia', since then the 'croup' has been replaced by attacks which seem to be typical paroxysmal asthma, with laboured breathing and orthopnoea; these begin suddenly at night, and have lasted as long as three days continuously.

In both these cases spasmodic croup (laryngitis stridulosa) seemed to have been replaced by asthma as the child grew older; but there are cases in which the distinction is very difficult if one has to judge from history only: both affections begin suddenly at night, in both there is difficulty of breathing, so that the child 'fights for his breath'; an attack of asthma occasionally begins with barking laryngeal cough, like that of 'croup'; both affections are commonly without fever; asthma sometimes ceases during the daytime, and returns at night, just as laryngitis stridulosa does. Nor is it only in their symptoms that these two affections resemble one another; there is a striking similarity in the exciting causes: over-eating, indigestible food, the excitement of a 'party', over-exertion, running or riding in the teeth of a cold wind, all this strange medley of causes, so suggestive of a functional nervous affection, is common both to laryngitis stridulosa (spasmodic croup) and to asthma. Moreover, there is a close resemblance in their pathology; in both the essential lesion is a combination of spasm with catarrh; in croup a slight laryngeal catarrh is associated with much spasm of the laryngeal muscles; in asthma a slight bronchial catarrh is associated with much spasm of the muscles of the bronchi. Surely these affections must be close akin, if indeed they be not pathologically identical. I have often thought that the term laryngeal asthma would well describe the relation of spasmodic croup to bronchial asthma.

There is a curious symptom which calls for mention here, not only as another link in the relationship between spasmodic croup and asthma, but as having other relations which bear upon the subject, namely paroxysmal sneezing.

It is a not uncommon tale that at the onset of a bout of asthma the child has a more or less prolonged attack of sneezing.

Henry H., aged $8\frac{2}{12}$, has suffered with asthma for two years, until recently the attacks always began with a bout of sneezing; for instance, on one occasion he sneezed nineteen times, after which his respiration became laboured and wheezing, and the asthma ran its ordinary course.

Sneezing is certainly not a common symptom in spasmodic croup, but I have notes of one case in which the attacks of spasmodic croup in a child three years old began exactly similarly with a bout of sneezing.

Paroxysmal sneezing occurs also as an isolated symptom, and there is much about it then suggestive of asthma.

Mary F., aged $9\frac{1}{2}$ years, a highly sensitive child of nervous parentage. Whenever she was overtired would be seized in the evening with severe sneezing. This would sometimes continue for hours, and had lasted from 5 p.m. till midnight, so that the child was quite exhausted. In some of these attacks she became feverish. They occurred about once in three weeks, were not specially related to the hay season, and continued to recur for about six months.

Such cases are no doubt close akin to, if not identical with, hay-fever, but they lack the special causal relationship which has given to hay-fever its name.

Between hay-fever and asthma the connexion is very close. It is particularly noticeable how frequently hay-fever figures in the family history of patients with asthma, and there are cases in which with the characteristic symptoms of hay-fever there are associated some of the symptoms of bronchial asthma, e. g.

Vivien D., aged $6\frac{11}{12}$, last year, suffered with hay-fever during the early summer. This year, in the latter part of May, the attacks reappeared; about 5 p.m. she is seized with violent sneezing, there is running from the nose, and the conjunctivae become congested. She seems quite dazed during the attack, which lasts for some hours. Her chest shows wheezing rhonchi all over: and there is well-marked emphysema as in a bronchial asthma. This child's maternal grandfather and uncle both suffer from hay-fever.

I have thought that some children who were subject to asthma at all seasons, and who showed none of the special symptoms of hay-fever, nevertheless had the asthmatic attacks more frequently or more severely during the hay season, when hay-fever was prevalent.

Symptoms. Reference has already been made to the recurring bronchitis, which is usually the only manifestation of asthma in infancy; but at a later age also it sometimes takes this form. Whenever the child 'catches a cold' it 'flies to his chest'; perhaps he becomes slightly wheezy for a few hours; perhaps a more definite bronchitis occurs with mingled râle and rhonchus, but chiefly rhonchus, especially with expiration, and with more

hurry of respiration and more tendency to wheeze than one would expect with a simple bronchitis.

The attacks mostly begin at night, and if they begin in the daytime or in the evening they are usually worse at night. Often there is some premonitory symptom, such as sneezing, or a short dry cough, 'cold in the head', or some discomfort in the epigastrium. In one child, aged seven years, there was always intolerable itching of the nose at the onset.

The symptoms of asthma vary greatly in their severity, a point to be remembered, for in its mildest forms the asthmatic nature of this affection is sometimes overlooked. In the most characteristic case the child goes to bed well, and wakes about 2 a.m. with a short dry frequent cough, which is soon replaced by loud wheezing respiration, in which long laboured expiration predominates, the child is unable to lie down, he is 'gasping for air', and perhaps begs to have the windows opened; the lips are blue; in the worst cases the whole face has a leaden cyanotic colour, and the child is in a dull almost stuporous condition, his whole energies are concentrated on the struggle for breath. If the chest be examined at this time it will be found to be hyperresonant, the cardiac and hepatic dullness are more or less obliterated by pulmonary resonance, loud wheezing, almost entirely expiratory, is heard all over the lungs, there is little or no râle, the heart is beating tumultuously, and its impulse may be seen and felt in the epigastric angle. Often the attack subsides as morning comes, but the child is left prostrate, and sleeps most of the day, and with night all the symptoms return almost as severely as on the previous night.

Duration and Prognosis. In the slighter cases the whole attack may last only an hour or less, usually the respiratory distress which has begun in the night lasts until daylight, and some wheezing rhonchi are to be heard on auscultation for two or three days subsequently; sometimes there is a recurrence of severe symptoms on two or three successive nights, and sometimes the severity of the attack continues unabated for two or three days and nights continuously, after which a wheezing bronchitis may persist for a week or more.

After asthma has recurred many times the child may be left in a persistent condition of chronic bronchitis, such a state is generally accompanied by more or less emphysema.

During any severe attack of asthma some emphysema is likely to be present, but until the asthma has recurred many times and become very severe, this is only a transient result of the violent

expiratory efforts, and when the attack is over disappears within a few days or weeks. This is of prognostic importance only as an index of the severity of the bronchial spasm; so long as the emphysema is recoverable, in other words, so long as the lung has not lost its elasticity, the asthma does not threaten life.

It is when emphysema has become permanent, not disappearing in the intervals between the attacks of asthma, that the ultimate prognosis becomes bad.

The child who has already reached the stage of chronic emphysema, owing to the frequency and severity of the attacks of asthma, may drag on for years a laborious lethargic sort of existence, always a little blue in the lips, always short of breath on the slightest exertion, and always dreading the next attack of asthma; pitiful cases are these, and the more so because such a condition is preventable, if only proper treatment is given in the earliest stage of the disease. In this condition a child stands but a poor chance when an attack of acute bronchitis or of broncho-pneumonia supervenes, as it is likely to do sooner or later. Fortunately, there are many cases which run a much more favourable course, especially where circumstances make it possible to carry out treatment, and particularly climatic treatment, with a free hand from the earliest appearance of the asthma: both the typical asthma and the allied condition of paroxysmal sneezing cease entirely in some children.

Treatment. Asthma is one of those diseases in which, as in epilepsy and migraine, and perhaps nocturnal enuresis, there is much of pathological habit, and I doubt not that, in things pathological as in the affairs of life, 'habit is second nature', and the longer the habit persists the more ingrained and the more difficult of eradication it becomes. For another reason also the early recognition and proper treatment of asthma is of extreme importance. Each attack brings with it the risk of chronic emphysema, and once induced the emphysema is likely to form a vicious circle, the special liability of the emphysematous lung to bronchial catarrh favours a fresh attack of asthma, and each fresh bout of asthma tends to increase the emphysema. Now, what happens in most cases? The parents take the child to a doctor, and, recognizing, perhaps, that the condition is asthmatic, he prescribes some medicine, lobelia or stramonium, or some such drug, to be given when the attacks occur. Now I venture to say that this is not the best way to treat an asthma; the ideal to be aimed at is prevention, and even though we may not succeed in preventing the attacks entirely we can usually

make them decidedly less frequent, and also much less severe; but this can only be accomplished by *continuous* treatment. Whatever drug is used it must be given regularly for months or even years. A very effective mixture is potassium iodide gr. i–iv, tinct. stramonii ♏ iss–x, spirit ammon. aromatic ♏ ii ss–x, syrup ♏ xx, aq. anethi ad ʒii ter die. The lower doses are suitable for infants, for older children larger doses will be necessary, but it is always wise to feel one's way with these drugs. Stramonium particularly must be given with the same watchful care as is necessary with belladonna, for it is liable to produce the same toxic symptoms, and some children show a marked idiosyncrasy to it. Five minims of tinct. stramonii was sufficient to produce dilated pupils with blurring of vision in a boy of seven years; on the other hand a child of 3½ years took ten minims of tinct. stramonii with great advantage, and without toxic results. I am in the habit of ordering this mixture to be given continuously, except for one week's intermission after every third week. Sometimes it is beneficial to alternate the course of this stramonium mixture with courses of arsenic: each may be given for three weeks with or without a week's interval between the two courses. This method affords an opportunity for comparing the efficiency of these drugs in the particular case: there are asthmatic children who seem to be greatly benefited by arsenic, especially, I think, when the asthma is only of recent onset, and there is no persistent bronchial catarrh or emphysema. Tincture of lobelia (B. P. 1885) or the ethereal tincture of lobelia are often used in place of stramonium, but they have no advantage so far as I know. Atropine and belladonna are also used.

Nervous sedatives are sometimes of value; I have known the addition of phenazone to the potassium iodide and stramonium mixture to be followed by great improvement: at a year old one grain may be given, and two grains thrice daily at eight years. Some give larger doses, but phenazone is a drug which occasionally produces cardiac depression, and should be used carefully, and always in combination with some cardiac stimulant. Bromides are sometimes a useful addition; chloral also I have seen given continuously thrice daily for many months to a boy of seven years, with good results, but it is a drug which one would prefer to avoid.

During an attack of asthma relief may be given by inhalation of the fumes of burning nitre-paper (white blotting-paper soaked in 20 per cent. solution of potassium nitrate and dried) or of one of the various powders of stramonium or lobelia and

potassium nitrate, which are sold for the purpose, e.g. the pulvis lobeliae co. (Martindale). Ethyl iodide is also useful for inhalation, and is prepared in very convenient form in capsules either alone or with chloroform (Martindale). Some children are more rapidly relieved by an emetic, such as a drachm of vinum ipecacuanhae, than by anything else; in a very severe case I have seen very rapid relief by a hypodermic injection of morphia: $\frac{1}{20}$th of a grain can be given to a child of four years, $\frac{1}{12}$th of a grain at six years.

But the treatment of asthma does not consist merely in drugging; it is essential that, if possible, the exciting cause should be discovered and removed; indeed there are cases in which drugs are of very little if any value, and the one thing needful is to remove the exciting cause of the attacks. This may require a minute investigation, not only of the child, the state of his organs and their functions, but of his home surroundings and his whole regimen.

I remember a child in whom no treatment proved so effective as regular correction of his constipation by daily administration of syrup of figs; and another in which one of the chief items of treatment was the prevention of gluttony. It is poor policy to dose a child continuously with drugs, if the attacks of asthma are being excited by idiosyncrasy, such as the presence of a cat, or by some affection of the nose or throat. On this last point I must say a word: there is no doubt that there are cases of asthma which are relieved greatly, if not stopped entirely, by correction of some abnormality in the nose, be it a deflection of the septum or hypertrophy of a turbinated bone, or the presence of a polypus, but such cases are, I think, very uncommon; and in childhood the only local condition for which operation is likely to be suggested as a cure for asthma is enlarged tonsils and adenoid hypertrophy. If by removal of these we could guarantee the removal of the tendency to naso-pharyngeal catarrh which they favour, I suspect that the operation would more often do good than it does: but it is my experience that the removal of adenoids and tonsils—perhaps owing to the difficulty of *complete* removal—leaves behind it, in a very large proportion of cases, more or less of the tendency to naso-pharyngeal catarrh for which the operation was done.

Be this as it may, the removal of adenoids and tonsils is almost always a woful failure as a cure for asthma. I have already mentioned thirteen cases in which the operation was done; only in one single case was there any improvement whatever in the

asthma. This was a boy aged about 9½ years, who had been having attacks every 2–3 months; after the operation he remained entirely free from asthma for nearly eleven months, but at the end of this time the attacks returned more frequently than ever.

I have left until last the question of climate, not that I would minimize its importance, for it is the first and most essential point in the treatment of asthma, but because I can say so little that is sufficiently dogmatic to be helpful. For years I have kept notes of the places where attacks occurred and where they did not occur in asthmatic children, hoping to tabulate from my experience a useful list of suitable and unsuitable places. The result was only to illustrate the fickleness of the disease; here is a child who remained entirely free from attacks at Torquay; here is another, seen the very next day, who became so bad at Torquay that the parents were glad to remove her; one child found relief at Worthing, another was particularly bad at Worthing; so with Tunbridge Wells, Farnham, Brighton, and many other places; in one child a particular district worked like a charm, apparently stopping the asthma as soon as he went there, in another the same place seemed only to aggravate the disease. In short, I can only draw some very general conclusions from my records: (1) that inasmuch as there is no certainty as to the effect of any particular climate on asthma, it is always wise to recommend a trial of a place before deciding upon permanent residence there, (2) that some children with asthma do best at the seaside, others inland; (3) that on the whole the milder places seem to succeed more often than the colder and more bracing; (4) that a dry soil, such as sand or gravel, is generally more suitable than a cold or damp soil, such as chalk or clay; (5) that a hilly district is more likely to succeed than a flat one, and that by choice the residence should be facing towards the warmest quarter, generally the south in this country, and a few hundred feet up on the slope of a hill. With the caution that most of the places mentioned have failed entirely in some cases, or even made the child worse, I will mention the following as having suited well some children with asthma:—Near London: Redhill, Farnham, Tunbridge Wells, Sevenoaks, Norbiton. Inland places distant from London: Bath, Dartmoor, Malvern. Sea coast places: Bournemouth, Southbourne, Torquay, Penzance, Worthing, Ryde, Folkestone, Rhyl, Grange over Sands, Falmouth.

Most of these, be it noted, are hilly places, and as a rule the asthmatic child obtained benefit from living in the higher parts of these districts.

CHAPTER XXV

BRONCHITIS

THERE is hardly a commoner complaint in childhood than cough; there is also hardly a symptom which has a more varied significance. It may be the indication of transient or permanent pulmonary affection: it may have no connexion with the lungs, it may depend entirely on naso-pharyngeal conditions, or it may even be a purely nervous symptom. The medical man must have all these possibilities before him when he has to deal with the child who is brought to him for a cough; here I shall confine my remarks to bronchitis as being one of the commonest of pulmonary troubles in childhood.

It is no part of my purpose here to describe the ordinary symptoms and physical signs of bronchitis or of any other of the pulmonary diseases which are common to all ages, a description of these may be found in any textbook of general medicine; my object is only to describe those special features which characterize the occurrence of such affections in childhood, and which are of practical importance.

Bronchitis is common enough at all periods of life, but probably far commoner in the first five years of life than at any subsequent age, and this for two reasons: firstly, because the two specific fevers with which bronchitis is almost always associated, namely, measles and whooping-cough, affect a very large proportion of children during the first five years of life; and secondly, because there is a special liability to primary bronchitis during infancy, a liability which seems to diminish steadily after the end of the second year. Some statistics taken in the Children's Out-patient Department at King's College Hospital, where children are seen up to the age of ten years, showed that 52 per cent. of the cases of primary bronchitis occurred in infancy (up to the age of two years): and there were nearly twice as many cases in the first year as in the second (34 per cent. out of the 52 per cent. were under one year).

Upon what does this special tendency to bronchitis in infancy depend? There are undoubtedly certain features in which the lung of the infant differs in its histology from that of the adult,

particularly in the relative abundance of loose connective tissue in the walls of the bronchi and alveoli. It has been suggested that the blood-vessels in this connective tissue, being but loosely supported, are the more ready to become dilated. It has been stated, moreover, that the pulmonary artery is relatively larger in infancy than in later life, so that the lungs at that age are more richly supplied with blood and so more liable to hyperæmic conditions than in later life. I would add that the small size of the nostrils in infancy and the ease with which they become blocked by coryza, is a frequent cause of temporary mouth-breathing in infancy ; and may thus contribute to the frequency of bronchitis.

However this may be, there is no doubt that the infant, especially during the first few months of life, is very prone to catarrh, both of the upper air passages and of the bronchi : and in these days of open-air upbringing it is needful to remind parents that while dry warm open air may be excellent, a cold, foggy atmosphere, such as we often have in London in the winter and autumn months, is a common cause of bronchitis.

A smoking fire in the nursery is sometimes the source of trouble; one of the youngest patients I have seen with bronchitis was an infant eight days old who had an extremely severe attack, apparently due to the smoky atmosphere of its nursery, dependent on some fault in the chimney.

In later infancy dentition is, I think, certainly a cause of bronchial catarrh. How this comes about I know not, but of the causal relation between the eruption of teeth and transient attacks of bronchial catarrh I have not the slightest doubt. The bronchial affection in these cases is quite slight, and like the corresponding intestinal catarrh, which is often associated with the eruption of a tooth, it lasts only three or four days and tends to recur with fresh worry of dentition.

This affection of the bronchial mucous membrane in one case and of the intestinal in another by the same cause, namely, dentition, reminds one of the curious sympathy which apparently exists between the respiratory and the alimentary tract. There are children who are subject to attacks of digestive disturbance, in which a furred tongue, pale or loose stools, and perhaps some nausea and vomiting and more or less fever are associated almost always with a transient bronchial catarrh.

A similar connexion is seen in the close relationship between some attacks of asthma and gastro-intestinal irritation : it is no uncommon story in the case of some asthmatic children that

whenever they eat too heartily they begin to wheeze a few hours later and have an attack of asthma.

It is also noteworthy how often the gastro-intestinal disorders of infancy, especially diarrhœa, are associated with some degree of bronchitis.

After the age of six months, there comes into play another important factor in the etiology of bronchitis, namely rickets. The rickety child is prone to catarrh of the mucous membranes, particularly of the respiratory tract; but the bronchitis of rickets does not depend entirely on constitutional vice, it is a result in part of the mechanical conditions determined by rickets: the softness of the ribs and the diminished range of movement of the diaphragm which is a necessary sequence of the eversion of the lower ribs in rickets, and also the general feebleness of musculature in severe cases, all favour the occurrence of pulmonary collapse; and atelectasis or pulmonary collapse, from whatever cause it may arise, strongly favours the occurrence of bronchitis. This is a point worth remembering in cases where signs of bronchitis remain localized in one lung or in one part of a lung: such a limitation of signs suggests the possibility of tubercle, but I would emphasize the fact that even when râles remain limited to one lobe or to part of a lobe (generally near the base of a lung) for several months, they may still indicate nothing more than a chronic bronchial catarrh in a portion of lung which has been tied down by adhesions or left collapsed by some previous illness, such as whooping-cough, so that it has never properly expanded since.

I have already referred to the frequency of mouth-breathing in infancy as one reason for this special frequency of bronchitis at this age: in the infant the mouth-breathing is often dependent merely on slight coryza, but even at this age adenoids may be cause of mouth-breathing; in older children the mouth-breathing, which is usually the result of adenoid hypertrophy, is a very common cause of bronchitis. I say 'cause', but perhaps it would be safer to say that mouth-breathing is associated with bronchitis, for it may be that the bronchitis is due rather to extension from the nasal or naso-pharyngeal catarrh, which is so frequent where adenoids or enlarged tonsils are present, than to the breathing through the mouth, and it is probable that incomplete expansion of the lungs owing to naso-pharyngeal obstruction plays an important part also in producing the special liability to bronchitis.

However this may be, the presence of adenoid overgrowth or

hypertrophied tonsils is very commonly responsible for frequent attacks of bronchitis, as is shown by the cessation of the attacks after removal of the adenoids and tonsils.

It is difficult to gauge the exact part taken by constitutional tendencies in the production of bronchitis. I have often thought that very fat babies were more liable to bronchitis than thinner babies. It is quite certain that the so-called 'Mongolian' imbeciles are extraordinarily liable to bronchitis, but in this particular case, again, mouth-breathing due to the special smallness of the naso-pharynx in these children may be a predisposing cause.

The special liability of children with congenital heart disease to bronchitis is well known; no doubt it depends upon the abnormal vascular conditions in the lung, for it is seen, I think, only in those cases in which there is more or less cyanosis.

Symptoms. In infancy and early childhood, acute bronchitis is often ushered in with considerable fever, causing an amount of anxiety which, as the event shows, is quite unjustified; for instance, I saw recently the child of a medical man, a little boy, aged about eighteen months, whose temperature had been raised for two days to 102°–104° F., respiration was rapid, and the child was flushed with a slightly cyanotic tinge; there were râles all over the chest and those at the bases were fine and rather sharp; but there was no dullness on percussion. The father was confident that the child had broncho-pneumonia; but about twenty-four hours later, the temperature had fallen to normal, the signs remained those of bronchitis only, and this diagnosis seemed to be confirmed by the course of the illness.

In this case I had little doubt that the sharp character of the fine râles was due to some collapse of the lungs, which, although not sufficient to produce dullness or bronchial breathing, sufficed to give this character to the râles.

That this does actually occur, I have proved by comparing post mortem appearances with the signs observed during life. The readiness with which pulmonary collapse occurs, is one of the special features of bronchitis in infancy and early childhood. Not only with bronchitis but with severe or frequent cough from any cause, and with any obstruction to air-entry, such as coryza in infancy or adenoids or large tonsils in early childhood, collapse of lungs occurs very readily and shows itself in diminished air-entry, sometimes with slight impairment of note, and with occasional fine râles.

This special liability to collapse is to be remembered in

interpreting the physical signs at this age ; it is easy to mistake for broncho-pneumonia what is nothing more than bronchitis with some collapse of the lung.

Another feature of bronchitis during the first two or three years of life is the remarkable rapidity with which emphysema develops, if the bronchial affection is severe and causing much cough ; it would seem indeed that even a few hours may suffice for the development of well-marked emphysema. Diminution of cardiac and hepatic dullness, evidence of emphysema, will sometimes throw light upon the rapidity of respiration which otherwise might be attributed to the more serious lesion, broncho-pneumonic consolidation. The emphysema, even when very extensive, passes off quickly when the cough diminishes; but while it is present it undoubtedly increases to some extent the gravity of the pulmonary condition.

This tendency to emphysema in early life is very noticeable in whooping-cough. Whenever an infant or young child with this disease is found to be breathing rapidly or becoming livid and short of breath, the likelihood of emphysema is to be remembered. I have frequently been consulted about children with whooping-cough, who had rapidly become worse, with blue lips and dusky cheeks and very rapid respiration, and therewith only a few râles in the chest. The medical attendant, realizing that the amount of bronchitis was not sufficient to account for the child's distress, had been puzzled to explain the severity of the symptoms.

In such cases I have sometimes found the cardiac and liver dullness to be completely obliterated by the emphysematous lung ; and this condition, associated no doubt with some dilatation of the right side of the heart, has been responsible for the exacerbation of symptoms.

There is another condition in which emphysema very quickly becomes established in children, namely, asthma ; and the child, like the adult, after several attacks of asthma, is often left with chronic emphysema and bronchitis, which may be very intractable, especially if residence in a suitable climate is impracticable. I mention this affection here because it is important to recognize that bronchitis even in an infant is occasionally of asthmatic nature and amenable to the treatment of asthma. The characters which suggest such an asthmatic bronchitis are very ill-defined and may amount to nothing more than suddenness of onset and a tendency to recurrences. Sometimes, however, the respiration is decidedly more wheezy, especially

with expiration, than is usual in simple bronchitis, but even then it is not of the noisy character which is so familiar in the typical paroxysms of spasmodic asthma which are seen in later childhood.

Prognosis. If only one could be certain that bronchitis would remain bronchitis, one would feel but little hesitation in giving a good prognosis; the trouble is that, especially in infants, it may at any time extend to the alveoli and become a broncho-pneumonia, and it is this possibility which should give us pause; the younger the infant the greater this risk appears to be. Apart from the age there is another point which may affect prognosis, especially in hospital practice, namely, the presence of rickets. I think there can be no doubt that the child with active rickets, especially where there is severe rickety contraction of the thorax, stands bronchitis badly and is specially apt to drift into a broncho-pneumonia.

In both cases, in the very young and in the rickety, this tendency to do badly with bronchitis probably depends largely upon the feebleness of expulsive respiratory power, in consequence of which secretions tend to accumulate in the tubes, and the *materies morbi*, instead of being expelled from the tubes, is drawn into the alveoli, carrying infection thither. It is in these cases, with feeble respiratory muscles and soft ribs, that bronchitis occurring several times in infancy is apt to leave behind it the transverse constriction of the lower part of the chest which is called 'Harrison's sulcus', an indication of the collapsed condition of the bases of the lungs which is both the result and the predisposing cause of bronchitis. But even when the chest has become distorted in this way by bronchitis in infancy, the increasing muscular and respiratory power as the child grows older tends to expand the chest to its normal shape, so that the 'Harrison's sulcus' rarely persists in later childhood.

When signs of bronchitis persist, and even when they remain localized to one lung or to the base of one lung as they sometimes do, this persistence is not in itself sufficient justification either for a diagnosis of tubercle or for a gloomy prognosis. I have watched several such cases for years without seeing any harm come of these conditions beyond the obstinate cough and occasional exacerbations of the bronchitis: pleural adhesions preventing full expansion of the lung, failure of the lung to re-expand after collapse induced by some previous attack of bronchitis or of whooping-cough, these are the conditions which may lead to persistent and localized signs of bronchitis.

In their clinical course and in their etiology such cases are very similar to others in which the persistent signs are those of localized consolidation, namely, sharp crackles with variable tubular breathing, bronchophony and some impairment of note, usually with some local flattening of the chest on the affected side and more or less displacement of the heart towards that side; in these cases also the signs persist for years with little or no change and with no serious damage to the child's general health; and in them also the change in the lung, which is a fibrosis with some dilatation of bronchi, is often the sequel of pleurisy or of whooping-cough.

Treatment

The child who is acutely ill with bronchitis wants air, and for this reason, if for no other, it is bad to have the air in the room used up by well-meaning friends and relatives coming in to see the poor child, who already has ample attendance in the mother or the trained nurse, in addition perhaps to its ordinary nurse. If the weather is warm, by all means have the top of the window open, provided, of course, that no draught from the window strikes upon the child; but if the weather is cold, and especially if it is foggy, as it often is when bronchitis occurs, I think the less of two evils is to keep the windows closed, and secure what ventilation we can by an open door, for damp, foggy air is specially prone to aggravate bronchitis.

I have no liking for complete tents in the treatment of bronchitis; if the room be a draughty one, a half tent round the head of the bed is, I think, sometimes an advantage; but to exclude free access of air to the extent that an ordinary complete tent does, is, I think, a thing to be avoided. In some cases the complete tent is necessary, where with deficiency of secretion in the tubes the cough is dry and ineffectual and it is decided to use a steam-kettle to moisten the air and induce freer bronchial secretion; in such cases a speedy effect from a steam-kettle is only to be obtained by confining the vapour in this way; but in a small room a tent is not necessary for this purpose, or very definite relief to the frequent ineffectual cough may be obtained by merely keeping a bronchitis-kettle boiling on the fire so that the steam passes into the atmosphere of the room.

The temperature of the room is a matter of practical importance; for an infant with bronchitis the temperature should be kept as nearly as possible at 65° F. Nowadays, gas-stoves are

extensively used for warming bedrooms; for a sick-room I think they are almost wholly bad; and especially where the illness is bronchitis; in most cases a gas-stove has little or none of the valuable ventilating effect of an ordinary fire; moreover, in spite of special precautions, a gas fire of any sort is apt to make the air of the room very dry, a condition which is particularly unsuitable in some stages of acute bronchitis.

In the early stage of acute bronchitis, when there is much rhonchus in the chest but little râle, and there is reason to believe that the child's cough would be freer and easier if there were more secretion in the tubes, the bronchitis-kettle used as I have described undoubtedly gives much relief.

The addition of compound tincture of benzoin (a drachm to the pint) to the water in the kettle, has, I think, a real value in these cases; at any rate I have seen improvement occur when this drug was added, where no improvement had taken place with the unmedicated steam.

In the early stage, whilst the secretion in the tubes is still scanty and the adventitious sounds are chiefly rhonchi, such a mixture as the following is generally useful: Potassii Citratis gr. ij, Vin. Ipecac. ɑ)ijss, Spirit. Ætheris Nitrosi ɑ)ijss, Tinct. Camphoræ Co. ɑ)ijss, Syrup ɑ)xv, Aq. ad ℥j, which may be given every three hours to an infant a year old, and double this dose to a child of three years. For younger infants or to one who is feeble or has a considerable degree of rickets, it will be better to omit the opiate; a mixture of Vin.Ipecac. ɑ)ijss, Sod. Bicarb. gr. ijss, Spirit. Ætheris Nitrosi ɑ)ijss, Syrup ɑ)xv, Aq. Anethi ad ℥j, will be suitable at first.

Directly the cough has become loose, ammonium carbonate or squills should be given to assist the child in clearing the tubes of the secretion. A useful mixture is Ammon. Carb. gr. ½, Vin. Ipecac. ɑ)ijss, Tinct. Scillæ ɑ)ijss, Glycerin ɑ)v, Spirit. Chloroformi ɑ)j, Mucilag. Tragacanth. ɑ)xxx, Aq. ad ℥j.

There is a class of cases to which I have already alluded in which bronchitis even in infancy is a manifestation of asthma, although it may appear at first in the guise of a very ordinary bronchitis. The possibility is specially to be remembered where the infant is particularly wheezy and there is a history of previous attacks. For these cases ½–1 grain of potassium iodide with 1–1½ minims of tincture of stramonium and a minim or two of sal volatile in syrup and water three times a day will be the proper treatment for an infant at one year.

As the disease progresses the amount of secretion in the tubes

may threaten to suffocate the child, and it is in this stage that emetics are usually recommended. Perhaps my own experience has been exceptionally unfortunate, but I confess it has not encouraged me in the use of them. Some infants are remarkably tolerant of emetics, and it is anything but gratifying to wait in vain for the return of a large dose of some depressing drug which the infant shows no tendency to vomit. For an infant ½–1 drachm of the vinum ipecacuanhæ will sometimes be effectual, but I would rather trust to direct stimulation of the pharynx by a feather or by one's finger if necessary; even this, however, I have known to fail in a very young infant to whom an emetic dose of ipecacuanha had been given without producing vomiting.

It is very rarely that an emetic of any sort is desirable; frequent doses of ammonium carbonate are usually to be preferred in those cases where an excessive amount of fluid is accumulating in the tubes: to an infant of a year, half a grain of ammonium carbonate, with 2½ minims of tincture of squills, may be given every two hours for about twelve hours, and then every four hours.

The great objection to free dosing with ammonium carbonate is its tendency to set up diarrhœa, which in an infant may be an exhausting complication. If there is any tendency to looseness of the bowels it will be better to substitute a mixture of Tinct. Nucis Vom. ℥j, Syrup Scillæ ℥x, Aq. Anethi ad ℥j, which may be given every three or four hours to an infant of six months.

Alcohol in some form will probably be advisable at this stage; 5 minims every three hours to an infant of three months or younger, and 10–15 minims every three hours for a child of one year.

When all the acute symptoms have passed off, but the child still has occasional cough, and occasional râles and rhonchi are to be heard in the chest, I have found cubebs sometimes of great use in the form of a mixture of Tinct. Cubebæ ℥v, Tinct. Camph. Co. ℥v, Glycerini ℥v, Mucilag. Tragacanth. ℥v, Aq. ad ℥j ter die. This is suitable for an infant of one to two years; double the dose can be used for a child of five years or older.

In the more chronic cases of bronchitis when there is little secretion, I think that potassium iodide in doses of gr. i–ij for a child of five years is sometimes valuable; and where a chronic bronchitis with a good deal of secretion is the manifestation of atelectasis or of some fibroid condition in the lung with or without

evident bronchiectasis, I am in the habit of prescribing a mixture of Tinct. Benzoini Co. ℥ijss, Creosot. ℥¼, Tinct. Camph. Co. ℥v, Syrup Tolut. ℥v, Mucilag. Acaciæ ℥x, Aq. Menth. Pip. ad ʒj ter die. This is suitable for a child of three years old; double this dose may be given to a child of eight years.

CHAPTER XXVI

BRONCHO-PNEUMONIA

Broncho-pneumonia as a primary affection—if it may be called primary when it is almost always an extension to the alveoli of a catarrhal process which begins as a simple bronchitis—is seen far more often in infancy than at any other period of childhood. It is this tendency to alveolar involvement which gives to the slightest bronchial catarrh in infancy an importance which would be lacking altogether in the case of an older child. Some statistics of cases under my observation, including children up to ten years of age, showed that 84 per cent. of the cases of primary broncho-pneumonia occurred in infancy; that is, during the first two years of life.

As a secondary condition broncho-pneumonia keeps company especially with infective conditions, particularly measles, whooping-cough, and infantile diarrhœa; but it is very frequently found in the post mortem room, as a terminal affection, in children who have fallen gradually into an exhausted or stuporous condition before death from any cause. This secondary form is less limited to infancy than the primary, but is very much commoner in children under four years of age than in older children.

The signs and symptoms of broncho-pneumonia are familiar enough,—the gradual onset with a shorter or longer period of bronchitis, then the increase in severity of the symptoms, the irregular fever, the restlessness, the purplish lips, the rapid breathing, the working of the alæ nasi, the scattered areas in which there are fine sharp râles with high-pitched if not definitely bronchial breath-sounds, nowhere perhaps actual dullness on percussion, but here and there a patch over which the note is not quite so good as elsewhere.

Sometimes after three or four weeks of varying fever, with appearance of fresh patches of consolidation from time to time, the signs gradually disappear, the temperature slowly subsides, and the child recovers. Too often the child goes from bad to worse, the areas of impaired note show more definite dullness, the bronchial breathing becomes more marked, the respiration is

rattling, the child passes from restlessness to somnolence, and within two or three weeks after the onset dies of exhaustion. There are other cases in which after a week or so the signs diminish and the child seems convalescent, with a normal temperature for three or four days; then the temperature gradually rises again and for a few days continues very high with only slight daily remissions, then falls again within two or three days to normal, and so with alternating periods of fever for a week or so and then normal temperature for a few days, the illness drags on for three or four weeks. Some, certainly, I think most, of these cases of 'relapsing broncho-pneumonia' ultimately get well.

One might imagine that nothing could be easier than the recognition of broncho-pneumonia, but in practice it is often very difficult to be sure whether we have to do with an acute bronchitis, with a broncho-pneumonia, or with a lobar pneumonia.

Simple bronchitis in infants and young children often begins with an amount of fever and respiratory hurry which may easily suggest broncho-pneumonia, and who shall say where the clinical line of division comes? In one case the râles remain medium-sized or large, they are not sharp or crackling, the breath-sounds are nowhere high-pitched, and after a day or two the temperature gradually falls and we say the child had bronchitis; in another the râles are smaller and sharper in character in some parts, and the breath-sounds are a little high-pitched but not bronchial, there is no dullness, perhaps no definite impairment of note, and we suppose the child has broncho-pneumonia; sometimes post mortem examination proves we are right, sometimes the suspected broncho-pneumonia proves to be nothing more than some bronchitis with collapse of parts of the lung. It is quite certain from post mortem examination that the only indication of broncho-pneumonia may be a little sharpness in the character of the râles and perhaps a slightly higher pitch of the breath-sounds than over the rest of the lung; and these are subtle distinctions upon which to base a diagnosis between acute bronchitis and pneumonia, but sometimes they are the only ones available.

In the infant also broncho-pneumonia very rapidly becomes confluent, so that a large part of one lobe may be consolidated, and the physical signs may resemble very closely those of lobar pneumonia. In such cases the distinction must rest upon the more gradual onset, the added signs of bronchitis, the more

irregular temperature and the subsidence by lysis in the broncho-pneumonic affection, but there are cases in which some or all of these distinctions fail: even the temperature may not help if the case is first seen two or three days before the fever ends, for the pseudo-crisis of lobar pneumonia simulates the intermittent or remittent fever of broncho-pneumonia; and occasionally a lobar pneumonia ends by lysis. It is true that broncho-pneumonia usually affects both lungs, but it does not always do so, and, on the other hand, lobar pneumonia may occur on both sides.

In infants also, I fancy more often than in older children, râles or crepitations are to be heard in other parts of the chest when there is undoubted lobar pneumonia in part of one lung, so that râles heard near the area of consolidation or in the other lung would not necessarily indicate broncho-pneumonia. The distinction is, I believe, impossible in some cases during life, and in many it must remain doubtful until the duration of the disease, its termination by crisis, and perhaps the occurrence of pneumococcal empyema point to the lobar form of pneumonia. Even in its bacteriology broncho-pneumonia is not always to be distinguished, for whilst no doubt in the majority of cases it is due to other micro-organisms it is occasionally due to pneumococcus, so that the sequence of a pneumococcal empyema does not necessarily prove that the previous pneumonia was a lobar pneumonia. It must, however, be admitted that an empyema is very rare with broncho-pneumonia, and the occurrence of a pneumococcal empyema is, I think, prima facie evidence that the antecedent pneumonia was lobar.

But even when it is clear from the gradual onset of the pulmonary condition and from the signs that there is a broncho-pneumonia, the possibility that it may be secondary to whooping-cough, or may be tuberculous, must always be remembered. It is a very easy and very common mistake to regard as a primary broncho-pneumonia what is really secondary to whooping-cough: sometimes the primary catarrh of whooping-cough passes rapidly into a broncho-pneumonia before any definite whoop has been heard, and even if the child has already begun to whoop, the whoop often disappears altogether when broncho-pneumonia supervenes, so that it is only by inquiring carefully into the previous history—especially the paroxysmal character of the cough and its association with vomiting, and perhaps the coincidence of whooping-cough in other children in the house—that the significance of the broncho-pneumonia may be determined. The

value of the frænal ulcer in diagnosis is to be borne in mind in these cases; this sign may serve as evidence of the paroxysmal character of the cough, even if the child has not actually whooped: it is present in about one-fourth of cases of whooping-cough, and is so rarely found in any other condition, that it may be taken as almost pathognomonic of whooping-cough.

There is sometimes much difficulty in excluding tubercle, especially where the broncho-pneumonia runs a lingering course. With measles and whooping-cough particularly one must needs regard a broncho-pneumonia with suspicion, especially if it persists for an unusually long time; again and again post mortem examination proves that what had been thought to be a simple broncho-pneumonia complicating these diseases is really tuberculosis. In these cases where the sputum is so seldom available the Von Pirquet reaction may assist diagnosis, but it must be remembered that a negative result is frequent where tuberculosis is running an acute course.

Prognosis. The more I see of broncho-pneumonia in infants and young children the more I dread its occurrence; not only is its mortality high—much higher than that of lobar pneumonia—but it is such a treacherous disease that one can hardly point to any particular condition or signs upon which a prognosis may be based. Age is, I think, the most important element in prognosis: Dr. T. R. Whipham [1] has published a very interesting analysis of cases: the mortality in children up to twelve years was 45·9 per cent., but in infancy, that is in the first two years of life, the mortality was 64·4 per cent.; Dr. Voelcker [2] found a mortality of 50 per cent. in the broncho-pneumonia of whooping-cough. Even higher rates of mortality have been recorded, but these are sufficient to show that broncho-pneumonia is always a dangerous disease in childhood, and especially in infancy.

I do not think that much information as to outlook is to be gathered from the temperature: it is not uncommon to find broncho-pneumonia at autopsy in an infant who has died after several days of normal or even subnormal temperature, at the end of a chronic diarrhœa or other exhausting disease; on the other hand, I have seen many children get well who had had a temperature of 105° or even higher with broncho-pneumonia.

The extent of the consolidation of course affects the prognosis: if a large area of one or both lungs is involved the child's chance

[1] *British Journal of Children's Diseases*, July, 1908, p. 284.
[2] *Clinical Journal*, Nov. 5, 1902.

of recovery is not good, but a small area of involvement is no guarantee of recovery, clinical signs are deceptive, and post mortem examination often reveals much more extensive affection than was suspected ; but on the other hand I think there are few conditions in which one may more legitimately hope until the end, for however extensive the consolidation may appear to be, the child will sometimes struggle through successfully.

I am inclined to think that the combination of severe diarrhœa with broncho-pneumonia, whether the diarrhœa is primary or secondary, makes the prognosis very bad.

Treatment. Nowadays it is very necessary to point out that cold air may do harm to the child with broncho-pneumonia : the doctrine of open air—excellent when checked by common sense and experience—has taken such hold that one finds the tenderest babes lying ill of broncho-pneumonia in a room with the windows wide open and the thermometer at 55°, or even lower. By all means let the child have fresh air, the more the better, and in summer time or even in winter, when there is a clear dry cold, there is no reason why the windows should not be open if the temperature of the room is kept up to 65° with a fire if necessary ; but when the raw, cold fogs of winter are about I would rather risk the closed window and trust to ventilation through the open door of the room.

The important observations of Dr. C. B. Ker[1], at Edinburgh, on the treatment of the broncho-pneumonia of whooping-cough by open air are sufficient to prove that traditional methods of treatment are not necessarily the best. He placed even severe cases of broncho-pneumonia out in the open air for seven hours daily during the summer, and in the winter treated them in a ward which was practically open to the air on two sides : the mortality amongst these cases of broncho-pneumonia was only 31·5 per cent., whereas amongst cases treated by the usual indoor methods it was 66·9 per cent. ; the chief contra-indication to the open-air treatment he considers to be laryngitis complicating the broncho-pneumonia.

As with bronchitis (see p. 359), I think the complete tent is to be avoided as preventing free access of air, but a half tent may be advisable if the room is a draughty one.

The value of local applications is difficult to prove, but I have thought that a hot application with either a thin jacket poultice or a hot turpentine stupe is useful if—and it is a big ' if '—applied *secundum artem*, by a skilful nurse ; if no expert nursing

[1] *Scot. Med. and Surg. Journ.*, Jan., 1904.

is available, I think that a hot fomentation is perhaps the easiest application, but whichever is used the utmost care is necessary that the application is not hot enough to scald the child; it is no great rarity to see amongst the poorer classes children who are scarred extensively on the chest from severe burns due to hot poultices or fomentations in infancy. It is horrible to think of the deliberate infliction of such cruel torture upon a little child through the ignorance of a well-meaning mother or nurse.

The drugs which are of most service in broncho-pneumonia are the stimulants, particularly, Nux Vomica and Ammonium Carbonate. In the early stage a mixture of Vin. Ipecac. ℥ijss, Ammonium Carb. gr. ¼, Syrup ℥xx, Aq. Anethi ad ℥j, may be given every three hours to an infant of three months, and double this dose to a child of a year.

Belladonna was recommended by the late Dr. Coutts, if with the consolidation there is also much bronchitis, so that secretion tends to accumulate in the tubes, adding to the difficulty of respiration; the dose he advises is ¼-grain of the Extract every three or four hours, and this dose is independent of age; it is given to an infant of a few weeks or to a child of six or seven years. As he points out, it is liable to produce toxic symptoms such as delirium, flushing, &c., and is therefore less applicable to private practice than to hospital patients who are under immediate medical supervision.

When the consolidation of the lung persists, as it sometimes does, for a long time, or fresh patches of broncho-pneumonia keep appearing for two or three weeks, Potassium Iodide is sometimes helpful and may be given in the mixture of Ipecacuanha and Ammonium Carbonate mentioned above; ½ a grain may be given every three hours to an infant of 6 months and 1 grain to a child of 1–2 years.

It is in these prolonged cases also that vaccine treatment has sometimes seemed to be of considerable value. Inasmuch as many such cases are due to pneumococcal infection, a stock pneumococcus vaccine may be used for this purpose, 5–10 millions may be given at a time to an infant of twelve months, the injection to be repeated if necessary at the end of four or five days. Some have punctured the consolidated lung with an exploring needle, and from the drop of sanguineous fluid withdrawn in the needle have prepared an autogenous vaccine, whether of streptococci, staphylococci, or pneumococci; but painful methods of this kind are always to be avoided if possible in the case of a child, and it may be practicable by gently swabbing the pharynx

to excite a cough and catch sufficient sputum for the preparation of a vaccine.

If the child shows signs of exhaustion, Tinct. Nucis Vom. ℥½, Syrup Scillæ ℥v, Glycerini ℥v, Aq. ad ℥j, may be given every three hours to an infant of three months, and double the dose to an infant of nine months or a year. For a child of a year old or more I think the Tincture of Digitalis, or perhaps better, as Dr. Melville Dunlop[1] advises, Tincture of Strophanthus, in doses of 1–1½ minims, is sometimes valuable, where the lips are becoming purple and it is evident that the right side of the heart is in difficulty. But as in other conditions, where it is necessary to increase the vigour of the heart-beat by these drugs, there comes a stage when mischief may be done by their action unless the circulation is first relieved by leeching or otherwise.

I have no doubt whatever of the great value of leeching in some cases of broncho-pneumonia where the right heart is becoming considerably dilated. Two, three, or four leeches, according to the age of the child, should be applied over the sternum.

When the cheeks are livid, and the lips blue, and the child is restless from dyspnœa, nothing gives so much relief as the inhalation of oxygen: in a minute or two the colour improves and the child becomes more comfortable; the easing of respiration means the husbanding of strength, and in this way oxygen may tide the child over a time of real danger.

An important point to be remembered in the treatment of broncho-pneumonia in early life is the liability to collapse and congestion if the infant is kept lying in one position for a long time. An adult or an older child can shift his position for himself, but an infant requires to be shifted from side to side frequently, so that he may give free play to each lung in turn.

Lastly, with regard to alcohol, I think there are very few cases of broncho-pneumonia in infancy in which alcohol is not advantageous at some period of the disease; in older children also, when the heart action is becoming enfeebled and the child is becoming exhausted, alcohol is most valuable; but as I have said elsewhere, it is quite possible to overdo the alcohol: a dose of 5 to 15 minims every three hours for an infant under a year old, and of 15 to 40 minims for a child one to five years of age, will be sufficient in most cases.

[1] *British Medical Journal*, August 15, 1908, p. 368.

CHAPTER XXVII

PNEUMONIA

THE main differences between lobar and lobular pneumonia are familiar enough : in the lobar variety there are the sudden onset, the continuous and high fever, the consolidation limited to one lobe or perhaps two lobes of one lung, whilst the other side usually escapes altogether, the crisis on the seventh to the tenth day, and the specific micro-organism the pneumococcus ; whereas in the lobular or broncho-pneumonia there are the gradual onset, often with some bronchitis at first, the irregular fever, the patchy consolidation often combined with bronchitic signs, and usually affecting both lungs, the termination of the fever gradually within no fixed period, and, lastly, the variety of micro-organisms which may cause it.

It used to be held that if an infant or a young child developed pneumonia it was almost certain to be a broncho-pneumonia, whereas in an adult it was almost equally certain to be a lobar pneumonia. There is some truth in this, but more error. If we eliminate secondary broncho-pneumonia, that is to say the broncho-pneumonia which occurs as a complication of other diseases, such as measles and whooping-cough, and consider only that which starts as an extension of primary bronchitis, perhaps without an antecedent bronchitis, the so-called primary broncho-pneumonia, it is, I think, true that in early infancy, up to the age of nine months, a primary pneumonia is usually, but not always, a broncho-pneumonia, but after this age a primary pneumonia is just as likely to be lobar as it is in the adult.

Taking 50 cases of lobar pneumonia at random from my note-books of cases seen mostly at the Children's Hospital, Great Ormond Street, or in the Children's Department at King's College Hospital, I find that 8 were under the age of two years, the youngest was ten months, 29 were aged two to five years, 13 were between five and twelve years old ; but I have notes of lobar pneumonia at the age of three months, and of one case which died with pneumonia, empyema, and suppurative meningitis, from which a pure growth of pneumococcus was obtained, at the age of 9½ weeks. I fancy that these figures underestimate the relative frequency of lobar pneumonia in the first two years

of life. Dr. J. A. Coutts,[1] at the Shadwell Children's Hospital, found that 50 per cent. of the cases of lobar pneumonia were under two years of age, and Dr. Melville Dunlop, at the Children's Hospital in Edinburgh, found that 45 out of 147 cases occurred under the age of two years. It is evident, therefore, that no great stress can be laid upon age in the diagnosis between lobar and lobular pneumonia.

An interesting point in regard to the etiology of pneumonia is the possibility of conveyance by contagion. I have had several cases under my care in which lobar pneumonia attacked two members of a family within a few days. Two sisters were in my ward at King's College Hospital, one aged twenty-one months, the other aged five years, with lobar pneumonia, which began in the former on March 22, in the latter on March 27; another girl, aged seven years, was admitted for lobar pneumonia which began on May 22; her father developed lobar pneumonia on May 19; both had empyema.

Such cases undoubtedly suggest contagion, but they should be compared with others, such as these, in which the onset was simultaneous: Gladys A., aged $4\frac{3}{4}$ years, was seen by me for right lobar pneumonia, which had begun on December 21 (it ended by crisis on the 28th); her brother, aged two years, developed lobar pneumonia on the same day. Charles J., aged two years, was in my ward with pneumonia, which began on December 24 (crisis on the ninth day); another child of the family developed pneumonia also on December 24 (crisis on the third day). In these cases the onset upon the same day makes it very unlikely that the infection had spread from one child to the other; it suggests rather that both had been exposed to some common source of infection, which might be air-borne or otherwise, but was not necessarily directly from an infected person.

The point is one of some practical importance; there are other instances on record like those mentioned above which suggest that pneumonia may occasionally be conveyed from person to person, and in face of such cases it seems advisable to avoid close contact as far as possible. Presumably the risk must be lessened by large air space and free ventilation. I have never known the disease to spread from one child to another in hospital, but when a child has pneumonia in the comparatively small room of a private house, it is advisable to keep other children out of the room.

[1] *Edinburgh Medical Journal*, September, 1902, p. 210.

Symptoms. The onset of lobar pneumonia in the child is almost always accompanied by vomiting: it has been said by Dr. Donkin that there are two acute infective fevers in childhood which almost constantly begin with vomiting, one is scarlet fever, the other is lobar pneumonia. It has also been said that vomiting takes the place of the rigor of the adult, as convulsions may upon occasion. But I am by no means sure that this is so: four out of the fifty cases began with definite 'shivering'; one of these was aged twenty-one months, and in his case there was no vomiting; the others were respectively 5, 5½, and 7 years old, and all of them vomited as well as shivered at the onset.

Vomiting sometimes but rarely recurs throughout the illness, as in the case whose temperature chart is shown below.

Convulsions are quite exceptional at the onset of pneumonia: there were none in the series of fifty cases, but I have noted them occasionally in other cases. Dr. Dunlop noted them as an initial symptom in 12 out of 147 cases; my own experience agrees with his as to the absence of serious import in these initial convulsions, and the grave significance of convulsions at a later stage.

A very common feature at the onset is drowsiness, which may very early be replaced or accompanied by delirium. Delirium is, I think, often overlooked in infants and young children, for in the absence of speech it declares itself only by an unnatural, restless, and vacant manner, the significance of which may easily go unrecognized by those who have no large experience of the ways of infants.

I have several times been told that the first indication of the illness was earache, a history which may be misleading, for unless the rapidity of respiration be noticed, and a careful examination of the chest made, it would be easy to attribute the whole symptoms to acute otitis media. I doubt whether the 'earache' in these cases is always genuine; a child of three or four years is often very vague in the distinction between headache and earache, and headache, sometimes severe, is, as we know from older children, often one of the initial symptoms of pneumonia.

A common complaint at the onset is pain in the abdomen, no doubt due to diaphragmatic pleurisy: young children are at all times particularly vague in the localization of pain, so that in this instance, when asked where it hurts them, they will pass their hand impartially over most of the abdomen, and if the doctor, imbued with visions of appendicitis, points to the right

iliac fossa and asks if it hurts there, they will nod or say 'yes' in the most misleading way. What more natural than to suspect 'appendicitis' when a child is seen with pain in the abdomen, referred perhaps to the right side, with vomiting, constipation, a high temperature, all of sudden onset, and perhaps on most careful examination nothing abnormal to be detected in the lungs?

Misleading cases are these in which the appearance of signs in the lungs is delayed, not one day or two, but possibly, as I have seen, until the day of crisis. One child I remember whom I watched carefully for signs which did not appear until the day of the crisis, when bronchial breathing appeared at one base and quickly disappeared again. It is this delay of physical signs which makes mistake so easy, especially where the abdominal symptoms are so suggestive of appendicitis. The error is not an uncommon one, Dr. Crozer Griffith[1] has recorded several cases in which the child narrowly escaped laparotomy, and quotes Dr. Morse as mentioning two instances in which this operation had actually been done upon children with pneumonia.

I have seen the converse mistake made in a girl about ten years old whose high fever and abdominal pain was thought to be due to pneumonia; careful abdominal palpation showed a definite tumour in the right iliac region and operation showed appendicitis. The rapidity of respiration, which is likely to be out of proportion both to the pulse rate and to the fever in pneumonia, is I think the most reliable criterion, but the *tout ensemble*,—the flushed cheeks, the bright eyes, the hot, dry burning skin, and above all an extremely careful examination of the chest, must determine the diagnosis of pneumonia.

And here I would point out that there is one part in which signs of consolidation are very apt to be overlooked, to wit, the extreme apex of the axilla. I have several times, in doubtful cases, detected definite evidence of pneumonia by pushing the end of my stethoscope against the chest as far up in the axilla as possible, when examination of the apex in front showed no signs of consolidation. This is a more likely spot for signs of lobar pneumonia in children than in adults, because in early years pneumonia is far more often apical than in later life: my own figures showed 19 apical cases (15 right, 4 left) out of 50, that is, 38 per cent.: this proportion agrees fairly closely with that obtained by Dr. Lovett Morse,[2] of Boston, who, amongst

[1] *Journal of American Medical Association*, August 29, 1903.
[2] *Archives of Pediatrics*, September, 1904.

101 cases, found 31 per cent. apical, 17 per cent. being right apex, 14 per cent. left apex.

Dr. Melville Dunlop's figures showed 34 (23 right, 11 left) out of 147, i.e. 23 per cent.; the late Dr. Coutts stated that even 60 per cent., which was the proportion of apical cases in his series, was less than his impressions would have led him to expect.

It is clear that apical pneumonia is very common in children, and probably much commoner than in adults (Professor Osler, at the Montreal General Hospital, found, amongst cases examined post mortem, only 13 per cent. with pneumonia limited to the upper lobe). I think it is also commoner in infants than in older children. I have thought that these apical cases were more liable to profound nervous disturbance, such as delirium, tremor, and apathy, than the basal cases, but my notes do not confirm this, nor can I satisfy myself that any practical guidance as to prognosis is to be obtained from the distribution of the pneumonia: it is the extent, not the localization, which matters; if both lungs are involved the danger to life is much increased, but, as I shall point out, prognosis is not to be determined solely by extent.

Amongst the early symptoms I have several times noted a history of 'bringing up bright red blood', apparently by vomiting, but in some of these cases, certainly, and I suspect in all of them, there was epistaxis, so that the blood was probably from the nose.

Herpes on the lips figures in the pneumonia of children about as frequently as in adults; it was present in ten out of my fifty cases. Its practical value is in the distinction between lobar and lobular pneumonia; where the physical signs leave the diagnosis doubtful, herpes would point strongly to the lobar variety.

The blood shows a marked increase of leucocytes, which average 30,000 to 40,000 per c.mm., with relative increase of polymorphonuclear cells; but the range of variation is considerable. I have found as few as 14,000, and, in an infant aged 13 months, with pneumonia, which ran an uncomplicated course, the blood on the fourth day of the disease, two days before the crisis, showed 77,600 per c.mm. (polymorphonuclear 82 per cent., lymphocytes about 18 per cent.). I mention this case to show that even so high a degree of leucocytosis as this is no evidence of pus forming in the pleura or elsewhere, nor does it necessarily indicate a bad prognosis.

The temperature in children with lobar pneumonia runs much

the same course as in adults, but I fancy that a pseudo-crisis, that is to say, a sudden fall of the temperature and then another rise before the final crisis, is much commoner in children than in adults. Such a chart as the subjoined is common.

Termination by lysis is very rare in children; it occurred in two out of the fifty cases.

Diagnosis. The distinction between lobar and lobular pneumonia is no easy one in some cases, for in infancy especially the

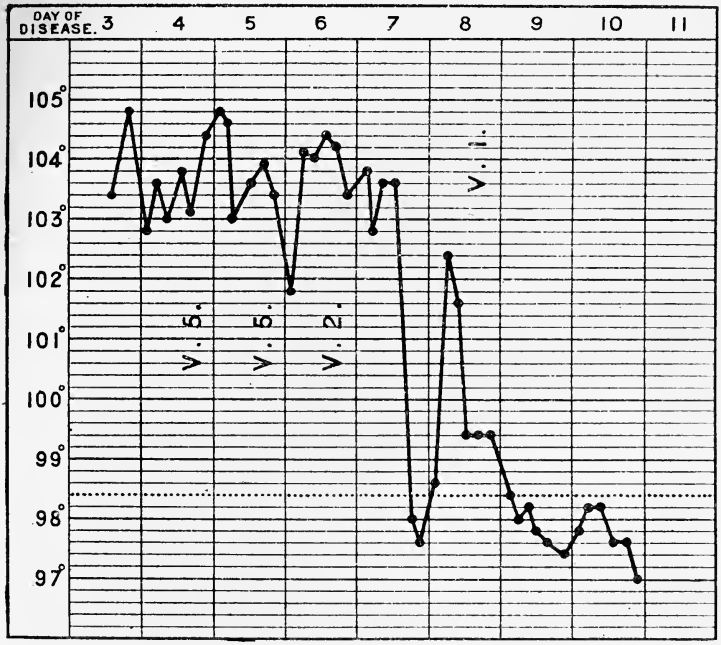

FIG. 23. Temperature chart of lobar pneumonia in a child aged 20 months, showing pseudo-crisis at termination of the attack; also vomiting continued throughout the illness, an unusual feature.

patches of consolidation in lobular pneumonia very easily become confluent, so that the distribution of the signs may agree with that of lobar pneumonia; and even when signs appear in the other lung which would suggest broncho-pneumonia, there is always the possibility of a double lobar pneumonia to be considered. I have sometimes noticed in the apparently sound lung in young children a small area of high-pitched or even definitely bronchial breathing, perhaps with very slight impairment of

note, generally about the region of the angle of the scapula. Whether this means an area of collapse or whether there is really a patch of pneumonia on this side also I know not, but when the signs in the other lung, in which there has been extensive lobar pneumonia, have cleared up on the occurrence of the crisis at the usual time, this small patch on the supposed sound side has disappeared also: I mention this only because it might suggest broncho-pneumonia, but I think it is quite clear from the course of these cases that the pneumonia is lobar.

Another feature which may confuse the diagnosis and which I think is commoner in infants and young children than in older patients, is the occurrence of râles or crepitations in the parts of the pneumonic lung which are not consolidated, or in the supposed sound lung. I have sometimes been in great doubt for this reason, but the diagnosis of lobar pneumonia, suggested by the history of sudden onset and the continuous high fever, was confirmed by the termination in crisis at the orthodox time.

In its temperature lobar pneumonia is simulated both by typhoid and by acute miliary tuberculosis: but the sudden onset is usually distinctive; moreover, lobar pneumonia lacks the enlargement of the spleen which is common to both these diseases and shows none of the general bronchitic signs which are characteristic of acute miliary tuberculosis, nor of the rose spots which are associated with typhoid; but whilst in theory the distinction should be easy enough, in practice it is sometimes exceedingly difficult.

I have notes of more than one case in which physicians of large experience were misled into a diagnosis of typhoid by a pneumonia in which signs of consolidation were delayed, and in which the delirium, with headache and drowsiness, all seemed to point to typhoid. After a few days the occurrence of a crisis or the development of physical signs makes diagnosis easy: we can all be wise after the event.

Where cerebral symptoms predominate, such as headache, drowsiness, delirium, and even retraction of the head, with vomiting and constipation, the condition sometimes simulates a cerebro-spinal meningitis, and the resemblance is increased by the occurrence of labial herpes in both diseases, although in meningitis the herpes is, I think, more often on other parts of the body. The rapid respiration and, within a few days at most, the signs of pulmonary consolidation will point to pneumonia; but it may be advisable to assist diagnosis by lumbar puncture, if further experience proves that the early use of Dr. Flexner's

serum is of value in the meningeal affection ; otherwise there is no advantage to the child from lumbar puncture, indeed there may even be risk : in one case in which lumbar puncture was done in a doubtful case of this kind under my care, death occurred almost before the needle could be withdrawn.

The misleading character of some of the symptoms in pneumonia, the earache suggesting a simple otitis media, and the pain in the abdomen simulating appendicitis I have already mentioned.

Prognosis. The danger of lobar pneumonia in childhood lies almost entirely in its complications : and it is in infancy especially that the two most serious complications occur, namely, suppurative meningitis and suppurative pericarditis. Usually these are associated with empyema, but the primary focus of pneumococcal infection is nearly always a pneumonia. I shall deal with these and other pneumococcal manifestations in a subsequent chapter.

Here I would only emphasize the need to be ever on the watch for empyema in children with pneumonia. At no time is empyema commoner than during the first two years of life, and it may be that by early detection of this complication we may save some of these infants not only from suppurative pericarditis which may possibly be due to direct extension from the pleura in some cases, but also from suppurative meningitis which must be due to blood infection, but may nevertheless be secondary to an empyema rather than to the original pneumonia, as the time relation seems to prove in some cases. I am no advocate for using the exploring needle indiscriminately, the highest skill is shown in deciding correctly when it is *not* necessary to explore : but none the less I think that where there is doubt much mischief may be done by delaying exploration, for even though the child may develop no further complication, the prolonged presence of an empyema materially diminishes the chance of complete re-expansion of the compressed lung.

Suppurative meningitis as a cause of death in pneumonia is very much commoner in infancy than at any other period, and the onset of head-retraction with squint and perhaps some rigidity of limbs may be the indication of its occurrence. But I would point out that there are other causes for head-retraction in pneumonia : infants with respiratory difficulty from any cause not infrequently assume the position of marked head-retraction, apparently in order to assist breathing by the action of the extraordinary muscles of respiration ; in this way head-

retraction may occur in pneumonia without any complication. Another cause of head-retraction is reflex irritation from the middle-ear, and as otitis media is a common complication of lobar pneumonia as of most other febrile diseases in childhood, head-retraction may also be due to this cause in pneumonia.

Another complication which I have known to mislead is laryngitis. I remember well a little girl, aged about nine years, whom I saw in consultation some years ago for acute laryngitis. The case had caused anxiety because the sudden fever which was attributed to laryngitis had continued high for several days, and because the child seemed more ill than is usual with simple laryngeal inflammation. When I saw her there was a croupy cough, and it was obvious the child had acute laryngitis, but on examining the chest it was found that there was dullness and bronchial breathing at the left base ; and it was evident from the signs, which had probably appeared late, that the condition was a lobar pneumonia.

I have only twice seen malignant endocarditis with pneumococcal infection, once at one year and eleven months, and once at three years, but never with pneumonia apart from empyema : in both the cases mentioned there was also suppurative meningitis.

A thickness of the first sound, at the apex of the heart, sometimes amounting almost if not quite to a bruit, is not uncommon during pneumonia, but this is purely functional ; it is probably caused in some way by the high temperature, for it is noticed sometimes in other conditions where there is much fever.

I have seen several cases in which a suppurative arthritis complicated pneumonia in children when there were other pneumococcal lesions, especially empyema, but I do not think the arthritis is always suppurative. In one child, aged $3\frac{1}{2}$ years, the left knee became painful and swollen with some fluid in it on the seventh day of the pneumonia ; under hot fomentations the arthritis disappeared, but five days later pus giving pure growth of pneumococci was found in the left pleural cavity.

Twice I have seen acute nephritis during the acute stage of lobar pneumonia in boys aged respectively $4\frac{1}{2}$ years and 2 years; in both cases there was a large quantity of albumen, and many casts were found in the urine ; in the latter there was also blood in the urine : in neither was there anything in the child's appearance to suggest nephritis ; both made a good recovery.

This rare complication is, of course, quite distinct from the slight albuminuria which is probably due to the high temperature and is common in children with pneumonia.

Clearly there is need for caution in the prognosis of pneumonia in children. I have very rarely seen it prove fatal, except through complications; my impression is that lobar pneumonia is much less often fatal *per se* in childhood than in adult life; of the fifty cases mentioned above only three died of pneumonia without complication (in two of them there was slight pleurisy also but no empyema). One of these three cases, however, shows that in the child, as in the adult, pneumonia may be malignant in its severity.

A boy, aged six years, went to school on July 16 apparently quite well in the morning and joined in games at school; at midday he came home complaining of headache and seemed drowsy. He was brought to King's College Hospital in the afternoon, his temperature was 105°, but on careful examination no physical signs were detected. The temperature rapidly rose to 108·6°, and the boy died the same evening; the whole illness had lasted less than twelve hours. Post mortem examination showed at the base of the left lung a circumscribed area about 2 inches in diameter of greyish red, evidently quite recent, pneumonic consolidation.

Death is far more often due to complications, particularly to empyema with suppurative meningitis, or pericarditis; both these occur chiefly in infants. In this way the danger from lobar pneumonia is much greater in the first two years of life than subsequently, and my impression is that the risk from the pneumonia itself is also greater in infancy. Dr. Melville Dunlop's figures showed a mortality of 26·6 per cent. in the first two years of life, whereas between two and twelve years of age it was only 2·9 per cent.

In the cases that do well the duration of the fever is seven or eight days as a rule.

I have always thought that the crisis was apt to occur earlier in the child than in the adult, but my own statistics do not show this; only six out of fifty had the crisis within the first five days; whereas, according to Musser and Norris[1], statistics at all ages, including adults, show a proportion of 25·8 per cent. having the crisis as early as this.

In children as in adults the crisis is occasionally delayed considerably. I had an infant, aged one year and eight months, under my care at King's College Hospital with lobar pneumonia: the crisis did not occur until the fifteenth day; the whole left lung was involved, but the right remained free throughout, and no complication occurred: the temperature ranged some days at 105° to 106° F., but the infant made a good recovery.

Treatment. Lobar pneumonia is a self-limited disease: pos-

[1] *Modern Medicine*, vol. ii, p. 588.

sibly in the days to come we may, by working along bacteriological lines, arrive at some method of curtailing the attack, but at present I cannot satisfy myself that anything we can do aborts the pneumonia. I have seen a crisis occur with rapid recovery under all sorts of treatment, and under no treatment at all; but I am rather disposed to admire the *vis medicatrix Naturæ* than to speak positively of the termination of the pneumonia by this or that method of treatment. But let me not be misunderstood; I do not for one moment suggest that no treatment is of value in pneumonia; on the contrary, I am certain that symptoms may be relieved and life saved by skilful treatment.

In the first place there is the pain and distress which often accompanies it; some children complain of pain in the affected side of the chest, others in the abdomen; for this and for the general distress caused by the high fever I think an ice-bag applied over the affected part of the lung is of great value, provided always that it is applied with proper precautions, in other words, with expert nursing and with medical supervision not once but twice or thrice daily.

Dr. Lees [1] has insisted upon the necessity of keeping the child's feet warm during the application of the ice-bag by wrapping the child's legs and feet in hot flannels or by hot water-bottle, and says that it may even be desirable to apply warm fomentations to the abdomen; if the ice-bag is used for an infant or young child he would have the temperature taken hourly and the ice-bag removed when the temperature falls to 100°, and replaced when it rises again to 102°. I have seen many cases treated by this method and have not the least doubt of its value; most children like it after the first sensation of cold is past, and I think it husbands strength by calming the child and keeping the temperature within reasonable limits; it also relieves the pain.

In infants and very young children I have a liking for warm applications for the relief of pain; for I have seen blueness and collapse from the use of the ice-bag: but I have no evidence that warm applications are of any value except for this purpose. For the reduction of high fever, if the ice-bag is not practicable, I think tepid sponging with water at a temperature of 75° to 80° is advisable if the temperature rises to 105° or higher; if the child is very restless or delirious, or is known to be liable to convulsions, tepid sponging may be advisable when the temperature reaches 104°. Children stand high temperature remarkably well as a rule, the one exception is the child who is subject to

[1] *Treatment of some Acute Visceral Inflammations*, Lond., 1904.

convulsions, and in this case I think that antipyretic measures, whether drugs, or sponging, or cold packs, or ice-bags to the head, are often advisable where the temperature might be disregarded in other children.

At the beginning of lobar pneumonia, when the child is restless, with grunting respiration and in evident distress, Dover's Powder, gr. j ter die for a child of two years, $1\frac{1}{2}$ to 2 grains for a child of five years, is useful, and this can be taken with a mixture of Liq. Ammon. Acetatis ℥xl, Spirit. Ætheris Nitrosi ℥v, Syrup ℥xx, Aq. Anethi ad ℥ij ter die, for a child of five years, or half this quantity for an infant. The Dover's Powder should be omitted after the first day or two. From the outset a careful watch must be kept upon the child's heart and pulse: if it is evident from increase of cardiac dullness to the right of the sternum and from the child's bad colour that the right side of the heart is becoming much over-distended, it may be necessary to apply two or three leeches over the sternum, and to substitute for the diaphoretic mixture a stimulant such as Tinct. Digitalis ℥v, Ammon. Carb. gr. j, Syrup ℥xxx, Aq. Menth. Pip. ad ℥ij sextis horis, half this dose for a child of two years or less.

Recently I have treated some cases of lobar pneumonia with pneumococcus vaccine; 5 to 10 millions may be given to an infant of 6–9 months, and 10 to 15 millions to a child of 1–5 years as initial dose. The vaccine is injected subcutaneously and the injection may be repeated if necessary with a larger dose, say 5 millions more, after four days. I have not been able to satisfy myself that the course of the pneumonia is appreciably affected by this treatment; but some observers have thought that it did good, and it has been recommended especially where the resolution of a lobar pneumonia is delayed.

Oxygen is valuable not only in giving temporary relief, which it certainly does, when the lips are becoming blue and the cheeks dusky, but also in tiding the child over a time of peril until the crisis arrives.

In spite of all that has been said to the contrary, I think that any unbiassed observer must admit that brandy is sometimes very valuable in the bad case of pneumonia, but it is certainly not necessary in the large majority of cases; it is only when the pulse is becoming feeble or irregular and it is evident that the child is becoming exhausted, that brandy should be given, and even then I am inclined to think that harm is sometimes done by giving large doses. I have seen cases in

which it seemed to me that vomiting was being caused, and others in which I think delirium was produced by excessive dosing with brandy; for a child of two years it is rarely advisable to order more than 30 minims of brandy, say every three hours, and for a child of five years a drachm (not a domestic teaspoonful) at most.

Judging only from clinical observation, I think that where the immediate danger is respiratory rather than cardiac failure, strychnine given hypodermically, or by the mouth if the danger is less pressing, is of more value than alcohol.

At all stages of pneumonia it is very important to keep the bowels working freely, for which purpose calomel gr. $\frac{1}{2}$–$1\frac{1}{2}$, according to the age of the child, is the drug most useful; and the dose should be sufficient to produce a loose stool once a day.

Lastly, there are some points which, although they come perhaps under the head of nursing, are worthy of the doctor's attention: the child with pneumonia wants oxygen even more than in health; the mother, only too anxious to do her best for the child, hearing that it has 'inflammation of the lungs', at once shuts every window and door and, as far as she can, blocks every approach for fresh air; at the same time she adds to the suffering of the fevered child by wrapping him up in a thick vest, a warm night-dress, a woollen hug-me-tight, and over all a dressing-gown and perchance a shawl, and then wonders that the child is unable to sleep! I doubt whether even the conventional cotton-wool pneumonia jacket is always desirable, but it has the merit of being light—and whatever form of vest is worn, it should be loose and light so as not to hamper the breathing—and when this is used a thin flannel night-dress over it is quite enough in the way of clothing if the room is kept at 62°–64°, as it should be.

It is well to point out to the parents also that a sponging over of the hands and face with cold or tepid water two or three times a day, if done without disturbing the child much, not only does no harm but may ensure sleep; and last, but not least important, that there is no reason whatever why the child should be denied the frequent small drinks of cold water for which he craves.

CHAPTER XXVIII

EMPYEMA

Empyema, though not one of the commonest diseases in childhood, has a special incidence upon the earliest years of life; moreover, there are in connexion with it questions of diagnosis and treatment which arise specially with regard to children, so that I need make no excuse for considering it here.

In five years (1899–1903) at the Hospital for Sick Children, Great Ormond Street, where children up to the age of twelve years are admitted, there were 183 cases of empyema (making 1·7 per cent. of the total admissions); of these fifty-four were under two years of age. This high proportion of cases in infancy (up to two years) is borne out by a series of fifty cases under my own observation, in which the age incidence was as follows:

0–1	1–2	2–3	3–4	4–5	5–6	6–7	7–8	8–9	9–10	10–11	11–12
4	12	13	5	4	2	5	2	2	0	1	0

These figures show that empyema is specially frequent in the first three years of life. It occurs very early in infancy: I have seen it at eight weeks with staphylococcus pyæmia, and at nine and a half weeks with a pneumococcal meningitis. It shows a curious tendency to affect boys rather than girls: amongst the 183 cases there were 112 boys and 71 girls.

Empyema in children is, in the very large majority of cases, a complication of pneumonia, and with this statement I will couple another which is of no small practical importance: that when pleural effusion occurs with pneumonia in a child it is practically invariably purulent. It is hardly possible, I think, to lay too much stress upon this latter fact; I see children with signs of effusion at one base after a typical lobar pneumonia, and the doctor hesitating as to the necessity for exploration, hoping that it may be serum which will disappear spontaneously: a delusive hope, for if the diagnosis of lobar pneumonia was correct, the diagnosis of empyema follows almost as a corollary.

Let me not be misunderstood: I know that sometimes if fluid

is drawn from the chest in such cases, within a few hours after its first appearance, it is found to be only turbid serum, but microscopic examination shows that there are many pus cells and that the fluid is swarming with pneumococci, and must be treated as an empyema ; if it is left for a day or two it rapidly becomes opaque like ordinary pus.

It is very necessary, therefore, to inquire carefully into the history of onset in a case of pleural effusion, to ascertain whether there were the sudden invasion, the herpes, the vomiting, the temperature, and the physical signs of a lobar pneumonia.

Empyema, however, is not invariably preceded by a lobar pneumonia ; rarely it seems to follow upon a broncho-pneumonia. I have notes of cases in which it occurred with the broncho-pneumonia of measles, and with that of whooping-cough ; but in some of these cases also the pus gives a pure culture of pneumococcus, so that we may suppose the broncho-pneumonia in these also to have been of pneumococcal origin.

Certainly empyema in childhood is far more often due to the pneumococcus than to any other micro-organism. Dr. J. Graham Forbes, Clinical Pathologist to the Children's Hospital, Great Ormond Street, found that amongst 250 cases of empyema in children under twelve years of age, 186 (74·4 per cent.) were due to pneumococcus alone, 21 to pneumococcus mixed with streptococcus or staphylococcus pyogenes, 17 (6·8 per cent.) to streptococcus alone, 11 to staphylococcus pyogenes aureus alone, 5 to streptococcus and staphylococcus mixed, 1 to pneumococcus with tubercle bacillus, 1 to proteus bacillus, and 1 to bacillus coli ; 7 gave no growth.

Amongst hospital cases there are many in which the early history of the illness is vague : the mother says the child was feverish and sick, had a cough and breathed quickly, was kept in bed for a week or so, perhaps was not seen by a doctor at all ; then the fever passed off, but the child has been wasting for many weeks since, and by the time the child comes to hospital, he is white and emaciated, with a hectic flush on the cheeks, and with a dry harsh skin, and looks the picture of advanced tuberculosis. And indeed the diagnosis may be no easy one ; there is dullness it is true, but perhaps this is more at the apex in front or in the axilla than at the base ; the breath sounds are diminished but there may be well-marked bronchial breathing over the dull area ; there are perhaps also a few crepitations, so that the physical signs are most misleading.

I would lay stress specially upon the frequency of well-marked,

perhaps almost loud, bronchial breathing over an empyema in children. I remember seeing a little girl about seven years old who was said to be dying of pulmonary tuberculosis: the child was much wasted, and sat propped up in a chair; her face was grey, she breathed with evident difficulty, and all over the back of one lung there was dullness and very marked resistance on percussion with loud bronchial breathing; there was also some displacement of the heart. To my mind the extreme resistance on percussion made fluid almost a certainty, and the history showed that there had probably been pneumonia at the onset; the medical man in charge, a most careful observer, had attached so much importance to the loud bronchial breathing that he thought empyema impossible. A large quantity of pus was evacuated, and the child did well.

So closely does the appearance of the child with a longstanding empyema resemble that of the tuberculous child, that even when the empyema has been found, one is sometimes asked whether it may not be tuberculous.

There is no doubt that tubercle and empyema occasionally go together; in fifty consecutive post mortem examinations which I made upon children who had died with empyema, I found six who had also pulmonary tuberculosis, and four who had tubercle only in other parts of the body, but I very much doubt whether in any single one of these cases the empyema could be called tuberculous; in some the association was clearly accidental, a pure growth of pneumococcus was obtained from the empyema; in one a tuberculous cavity near the surface of the lung had burst into the pleura, but even then the empyema was probably rather a septic infection from the many micro-organisms in the cavity than due to any tuberculous affection. I think that an empyema directly due to tuberculosis must be an extreme rarity in a child, and that when the association of pulmonary tuberculosis with empyema is observed, if there is any causal relation at all it is usually an indirect one, the ordinary pyogenic micro-organisms which swarm in the broken down tuberculous focus, have gained access to the pleura.

In early infancy an empyema and indeed an acute pleurisy of any sort often produces profound constitutional disturbance quite out of proportion to the physical signs, so that at this age the cause of the infant's sudden acute illness and almost moribund condition is sometimes extremely obscure, and indeed the pleurisy is often discovered quite unexpectedly at post mortem examination. At this age also empyema is specially likely to

be associated with pneumococcal inflammation of the heart or meninges. The following case illustrated these points.

John P., aged twelve weeks, had had a cough for five days, and been sick several times since; when brought to hospital he appeared moribund, his face was pale, the lips ashy grey; his temperature was 101·2°; the only signs were slight impairment of note at the left base with some diminution of breath sounds; the signs were not thought to indicate fluid. The child rallied slightly with mustard bath, strychnine, and oxygen, but died within three days after admission, when it was found there were 4 ounces of pus in the left pleura, and some consolidation of the left lower lobe; there was also suppurative pericarditis.

In connexion with the signs and symptoms of empyema there are certain points which are specially noteworthy. I have already referred to the vagueness of onset in some cases, and to the wasting and the dryness of the skin with empyema of long standing, symptoms which may suggest tuberculosis.

Another misleading symptom in some of these longstanding cases is a paroxysmal cough, which may culminate in vomiting, and closely resemble whooping-cough. I have known this mistake made even when the child was under continuous observation in hospital.

The temperature also in a longstanding case of empyema may mislead, for it is often normal, and even when the empyema is more recent the temperature chart seldom shows the continuously high temperature which is common with a serous effusion. I would draw attention specially to this difference between serous and purulent effusion, for I often find that the reverse is expected; the high temperature which points to serum is mistaken for an indication of pus.

I need not consider the signs of empyema here; they are to be found in any book on general medicine; but I will mention two or three points which seem to me of special importance. I have already mentioned that bronchial breathing may be well heard all over a chest which nevertheless contains an empyema.

In the child, even in health, tactile vocal fremitus is often not to be obtained, or is extremely slight, so that it is difficult to derive much information from it where fluid is in question; when comparison with the sound side shows that tactile vocal fremitus is absent only on the affected side this is strong evidence of fluid.

To an expert finger I think the sense of resistance on percussion over fluid will very rarely give a false impression; but there is one rare condition which is almost peculiar to childhood, which

gives exactly the same sense of resistance, and moreover may produce just as absolute dullness and as complete absence of breath sounds as any pleural effusion: I refer to the so-called cheesy-consolidation of the lung: that form of tubercle in which the lung or a large part of it is converted into an almost uniform mass of solid caseous material like the section of a firm caseous gland. I know of no way of distinguishing between this and empyema except by the exploring needle.

Skodaic note at the apex is a sign to which very great importance may be attached; where this has been present I have not hesitated to explore several times, when necessary, to find the fluid which I felt sure was present; it is a sign which very rarely plays false. But no sign is always to be relied upon; even displacement of viscera away from the affected side does not necessarily mean fluid. I have seen several cases of pneumothorax in little children, generally, I think, with tuberculosis, and in these the heart is sometimes considerably displaced; again, if the empyema be a small and limited one of old standing, the contraction of the lung with some fibroid change may cause the heart to be displaced towards the side with the empyema. Even in the acute stage of a pleural effusion measurement often shows that there is some contraction of the affected side; I have measured many children to ascertain this point, and frequently found that the affected side, whether with serum or pus, was half an inch smaller than the sound side; I fancy that this means that the collapse of the lung is not merely a mechanical result of pressure, it may be out of proportion to the amount of the fluid, so that in spite of the presence of fluid that side of the chest, so far from being bulged, is contracted.

In infants and young children there is confirmation of this in the great diminution of breath sounds which occurs when there is recent pleurisy, even though there be no evidence of fluid at all.

Are there any means by which we may distinguish between empyema and serous effusion without exploration? As a rule, physical signs help but little; the occurrence of a localized œdema of the chest wall, or a localized bulging, points, of course, to empyema, but no one should allow the question to remain unsettled until this occurs. Even œdema unless localized to a part of one side of the chest is no evidence of pus; occasionally when a serous effusion occurs very rapidly a generalized œdema of the chest wall occurs, as I have once or twice seen in children.

Of far more importance is the history: where a definite lobar

pneumonia has preceded the effusion, or when there is some pyæmia present, we can be practically certain that it is an empyema. If, on the contrary, the illness began with pleural effusion, and especially if the effusion increase very rapidly, so that within three or four days the signs of fluid have extended almost up to the apex of the lung, or where a gradual onset has been noticed with friction first, followed, after a few days, by effusion, the probability is strongly in favour of serum; and in the child a serous effusion almost invariably means tubercle.

Profuse sweating and diarrhœa are certainly commoner with empyema than with serous effusion. As confirmatory evidence the blood count may be of some value, a high degree of leucocytosis is in favour of empyema, but this is by no means an infallible guide.

I have kept till last the mention of the exploring needle: not because I regard it as least important—it is essential in most cases where the possibility of pleural effusion is in question—but because, especially in dealing with children, it should be our ambition in diagnosis as well as in treatment to avoid as far as possible any method which inflicts pain; and one has proved by experience that there are cases—few, I admit—in which a careful consideration of the history and the course of the illness makes it possible to do without exploration; rapid absorption of the fluid in such cases has proved that one was correct in supposing it to be serous.

If there are no sufficient grounds to justify us in assuming that only serous effusion is present we must needs explore; and here let me point out that exploration is not absolutely free from danger; it is common enough for the child to spit up a small quantity of blood just after the puncture, proving that the lung has been wounded, but this is usually of no importance; it has, however, happened that severe bleeding has taken place from the lung; I have also seen pneumothorax, extensive surgical emphysema, and hæmorrhage into the pericardium, as results of exploratory puncture.

Certain practical details in the matter of exploring are worthy of mention. I shall assume that the skin and the needle have been rendered as far as possible aseptic, and that the needle has been tested not merely by driving boiled water out of the piston through the needle but by drawing it up the needle into the piston, which is a far more important test, as one too often finds that the plunger does not fit accurately enough to draw fluid even against the slight resistance of a fine lumen in the needle.

In infants and young children the space between the ribs is so small that it is easy to drive the needle against a rib; to avoid this the child should be made to lie upon the sound side with a small pillow under that axilla, and the arm of the affected side should be drawn up over the head so as to stretch the intercostal spaces as widely as possible.

The child's trunk must, of course, be steadied to prevent his lurching forward as the needle enters. In older children the same posture may be used, or if preferred the child may be in the sitting position with the arm drawn forward and slightly upward.

I need not describe here the various causes of failure to find pus when it is present, they are the same in children as in adults; but I will mention two fallacies which I have noticed with special frequency in my ward. Often the needle is not inserted far enough; any one who has seen the large masses of shaggy lymph which come from a pneumococcal empyema, and which coat the pleura, can understand that it may be necessary to push the needle in quite an inch or more before any pus can be drawn. Secondly, I have several times been told that the result was negative because no fluid had been drawn into the barrel of the syringe, when by driving down the piston after the needle had been withdrawn from the chest it was found that there was a single drop of fluid in the needle itself, and under the microscope this drop proved to be pus.

In every case the fluid withdrawn should be examined carefully with naked eye and with the microscope, as well as bacteriologically. I have sometimes been told that the fluid was serous, when careful examination even with the naked eye showed that it was slightly turbid, and microscopic examination showed a large number of pus cells and pneumococci, which made it evident that it was not serum but seropus, and required the treatment of an empyema. The importance of examining the fluid carefully was impressed upon me some years ago when I had explored a child's chest and was about to throw away the small quantity of fluid in the syringe, having hastily concluded that it was clear serum, when the physician in charge of the case, Dr. F. G. Penrose, drew my attention to the unusually colourless appearance of the fluid; microscopic examination showed hydatid hooklets.

One other point in the fluid is worth noting: its smell; twice in the series of fifty cases under my observation, I have found the pus stinking with an almost fæcal odour where it proved to

be secondary to an appendicitis; both cases made a good recovery.

Complications. Amongst the complications of empyema, none is more important than suppurative pericarditis. Like suppurative meningitis, which is also far more frequent in the first three years of life than at any other time, and which is also most often associated with pneumonia or empyema, suppurative pericarditis is usually fatal, but unlike the meningeal affection it offers a possibility of recovery if recognized and treated.

Out of 31 cases of suppurative pericarditis which I examined post mortem, 20 were associated with empyema. Dr. Poynton [1] found that 83 out of 100 cases of suppurative pericarditis at the Children's Hospital, Great Ormond Street, occurred before the age of four years, and about 60 per cent. were associated with empyema; out of my 31 cases (some of which are included in Dr. Poynton's figures) 18 occurred within the first two years of life.

The recognition of this complication is extremely difficult; even when it is suspected the physical signs are so indefinite that it needs a bold man to advise opening the pericardium in a child who already looks desperately ill, and with no other evidence perhaps to support the diagnosis beyond the extreme illness which does not seem to be sufficiently accounted for by the pulmonary or pleural condition. Nevertheless, I think that more weight is sometimes to be attached to symptoms than to signs in diagnosing this complication; when a child who has already had an empyema opened continues to look profoundly ill, with irregular fever, and especially with extreme pallor of face and lividity of lips, hurried breathing and a rapid pulse, suppurative pericarditis should be in question.

In some cases there are sudden attacks of cyanosis. Dr. Poynton lays stress also, very rightly I think, on the importance of orthopnœa and syncopal attacks; the extreme pallor and hurried respiration are also striking symptoms in most cases. As to signs, they are of the vaguest: pericardial friction is almost never present, and the amount of pus is so small in most cases that it does not cause any considerable increase of the cardiac dullness; usually it does not exceed 2 ounces, often it is not more than 1 ounce. If there is any increase of the dullness, its extension upwards is specially significant; more often I think information is to be gained from the heart sounds which sometimes, but by no means always, become distant and muffled.

[1] *British Medical Journal*, August 15, 1908.

Where there is good reason to suspect suppurative pericarditis the diagnosis must be determined by the exploring needle, which should be inserted in the fifth left space close to the sternum or in the angle between the xiphoid and the left costal cartilages, pushing the needle upward and backward.

Suppurative meningitis I found in eight out of fifty fatal cases of empyema; it occurs usually in infants, and is fatal within a few days; its presence may be indicated by some squint or slight head-retraction or convulsion, but often it is found quite unexpectedly at post mortem examination.

Arthritis was present in two out of the fifty cases; in one it was certainly pneumococcal and affected both hips and the left shoulder in a male infant aged ten months; in the other it affected the left hip-joint, and was probably also pneumococcal.

The peritoneum shows suppuration in some cases, but this is rarely recognizable during life.

Prognosis. Empyema is a much more dangerous disease in a child under two years of age than in an older child. Out of the series of 183 cases mentioned above, 40 proved fatal, that is 21·8 per cent.; but, as may be seen from the following figures, the proportion is much higher in infancy:

	Number of Cases.	*Deaths.*
Under 2 years	54	28
2–12 years old	129	12

The very heavy mortality in infancy is due partly to the special tendency to suppurative pericarditis and meningitis at this age. In many of the cases included in these statistics, there was generalized pneumococcal infection which proved fatal; it would be difficult to say in what proportion this was secondary to the empyema, no doubt in some the infection of the serous membranes and meninges may have been of the same date, and have resulted from a blood infection during pneumonia, so that the fatal ending could hardly be attributed to the empyema.

One element in prognosis is the bacteriology of the empyema: in infants the danger of generalization of pneumococcal infection makes an empyema due to the pneumococcus probably quite as dangerous as that due to any other micro-organism; but in older children where there is much less risk of suppurative pericarditis or meningitis, the outlook is, I think, better with a pneumococcal empyema than with one due to infection with other pyogenic bacteria, whether of single variety, such as streptococcus, or of mixed kinds, for instance, streptococcus with bacillus coli.

Often one can recognize with a fair approach to accuracy the

nature of the infection from the appearance or smell of the pus when the empyema is opened; when large masses of lymph are present, the pus almost always contains pneumococci, whereas with a streptococcal empyema the fluid is thin, often only seropus with only a few small shreds of lymph in it. Stinking pus would point to infection with other micro-organisms, probably the bacillus coli.

Another point which must influence prognosis is the duration of the empyema; if pus has remained in the chest for many weeks the lung may not re-expand perfectly when the pus is evacuated; and these are the cases in which there remains a pus-discharging cavity for many weeks or months—a sequel fortunately much less common in the child than in the adult; these also are the cases in which the open cavity is apt to become infected with some extraneous bacteria so that the discharge originally 'sweet' becomes offensive in odour, and the wound unhealthy and indolent in appearance, and the child does badly.

I have laid stress on the unsatisfactory elements in prognosis, I must refer now to the brighter aspects of empyema. Of children over two years of age with empyema, according to my own figures, fully 90 per cent. recover, and if the empyema is opened within a week or two after its occurrence, the recovery is astonishingly complete. Even the gap in the rib where resection has been done is completely repaired within two or three months, and by this time the breath sounds are often so perfectly normal that except for the scar one would not know that any affection of the lung had occurred.

The ordinary pneumococcal empyema is, in fact, far more satisfactory in its outlook than a simple serous effusion; the child who has recovered from the empyema has, so to speak, 'a clean bill of health'; the child who has had a serous effusion in the pleura, however completely all traces of it have disappeared, must live the rest of his life in danger of tuberculosis, which too often declares itself in the lungs or elsewhere within a very few months or years after its first indication in the pleura.

Treatment. The treatment of an empyema falls within the scope of the surgeon rather than of the physician: but as much of the responsibility for the treatment usually rests—and should rest—upon the physician, I may perhaps be allowed to make some general remarks upon it.

In the first place I would insist upon the importance of opening the chest as soon as possible in every case of empyema; it seems

probable that in some cases the pneumococcal infection of the meninges and pericardium to which children are specially liable is the result of allowing pus or lymph, which is usually a pure culture of pneumococcus, to remain pent in the pleural cavity. No doubt, in many cases, the pneumococcal infection of the pericardium, meninges, and other parts, are only manifestations of a general blood-infection, to which the empyema also is due; but this is not always so.

I had under my care a boy aged ten months; I first saw him five weeks after an attack of pneumonia, he was ailing with vague chest symptoms which did not suggest empyema; he died nearly three months after the pneumonia with suppurative meningitis, and post mortem 1 drachm of pus was found shut up by adhesions in the right pleural cavity. The suppurative meningitis must have begun only a few days before death; it seemed clear that the source of infection was this small collection of pus which I had failed to detect in the pleura.

In twenty-four out of fifty fatal cases of empyema in children whom I examined post mortem, there was suppuration in the meninges, pericardium, peritoneum, or joints; in thirteen of these cases the empyema had not been opened (in two of these thirteen the child had been explored with a needle twice without finding the pus). I do not think that these complications were in all cases secondary to the empyema; in most cases probably they occurred simultaneously with the empyema, and were part of a general blood-infection, but none the less, the liability to these fatal complications is certainly increased by leaving an empyema unopened, and as we cannot tell at what hour the spread of infection may occur, the sooner the empyema is opened the better.

But, says one—and this is not an imaginary proposal, I have heard it more than once—may we not leave the pus alone in the hope that it may be absorbed? There is nothing absurd in this suggestion; it is more than probable that a very small loculus of pus may gradually dry up, so that the pathologist happening to examine the child's body a year or two later finds only a dry washleather-like patch of yellowish white material between the adherent layers of pleura, and if this were all that remained it would be an excellent result: but, unfortunately, it is not all. Apart from the risk of dissemination of the infection from the loculus, however small, there is a danger of chronic fibrosis of the lung when an empyema is left unopened. The history of some of the cases of fibroid lung and bronchiectasis in children

strongly suggests this origin, and in some cases the post mortem appearances confirm it.

Next I would point out that seropus is just as dangerous from the point of view of secondary infection as thick pus, and therefore it should be treated by free opening of the chest.

Many years ago aspiration alone was done in cases of empyema at the Children's Hospital, Great Ormond Street, and it is said that a certain number did just as well as with the modern treatment of open drainage; but the risk of secondary infection presumably is greater with this procedure than with free opening, for the large masses of lymph which are evacuated by the open method are swarming with micro-organisms, and the retention of these in the pleura must increase the risk. Nevertheless, there are conditions in which aspiration is useful and advisable; where the empyema appears to be very large in amount, and especially if the child's condition is very bad so that an anæsthetic seems risky, I like to have the chest aspirated and about 8 or 10 ounces drawn off one day, so that on the following day or a few hours later the chest may be opened with less risk than would be incurred if the larger quantity of fluid originally present had been rapidly removed by immediate operation.

There are instances also in which, owing to parents' refusal to allow operation, aspiration may be the only course open to us.

The choice of operation lies between simple incision in the intercostal space and resection of a rib. Nowadays the latter is in vogue: at some hospitals it is the only method in use; and undoubtedly it has advantages—it allows a more thorough examination of the empyema cavity, which sometimes gives useful information, and probably the opening is freer so that the masses of lymph escape more easily.

But I think it should be realized that resection of a rib is quite unnecessary in many cases. I have seen many cases treated by simple incision, with excellent results; it is an operation which can be performed more quickly even than resection of a rib, and this is an important point when we are dealing with a feeble infant, or a child who is extremely bad. In cases where the pus is thin, with few masses of lymph, so that no large opening is required, there is room between the ribs for a sufficiently large drainage tube even in an infant.

After the chest has been opened the temperature should fall to normal if it has been raised, and the child's condition rapidly improve; if the child continues to look ill, and there is some

fever, one must suspect the presence of pus elsewhere. There may be an empyema on the other side, or there may be another collection of pus separated by adhesions from the empyema which has been opened : or there may be, as I have found at autopsy, a collection of pus in the mediastinum.

If the child looks acutely ill, with livid lips and hurried respiration, after the empyema has been relieved, the possibility of suppuration in the pericardium must be carefully considered (see p. 390).

Sometimes recovery is delayed by failure of the wound to heal, a sinus remains and discharge persists. I remember one child in whom this was explained by the discovery on further resection of a strip about 6 inches long of green-protective which had dropped into the pleural cavity from the dressings ; a similar accident has happened with the drainage tube, where this was not properly secured either by a stitch or by a suitable flange. In enlarging the gap in the rib after the pleura has been opened the piece cut off the end of the rib may fall into the pleural cavity and cause a sinus to persist ; such mishaps are to be avoided by proper care.

More difficult to prevent is the unhealthy state into which the wound will occasionally fall when the utmost care has been taken. I suppose that in these cases there has usually been some accidental contamination from without, but it seems quite conceivable that the added infection may sometimes be from within, and that either from the blood or from the lung bacteria gain access to the wound and flourish in the damaged tissues, especially if the child is in a feeble state. Such a condition may call for the use of a vaccine, and, if possible, an autogenous vaccine, i.e. one prepared from cultures taken from the purulent discharge in the particular case, should be used in preference to any stock vaccine. It may be that a mixed vaccine of pneumococci with streptococci or staphylococci or even *Bacillus coli* may be found necessary after the bacteriological examination of the discharge. An unhealthy dry and glazed appearance of the wound together with a rise of temperature has sometimes made me suspect septic contamination, when the event has proved that those symtoms were due only to the onset of one of the specific fevers, for instance measles.

CHAPTER XXIX

TUBERCULOSIS

THERE are few diseases so common and so disastrous in their results as tuberculosis in childhood. The yearly sacrifice of childlife to tuberculosis in this country is nothing short of appalling; and it is made only the more distressing by the certainty that some of it, perhaps much of it, might be prevented if only proper precautions were taken.

It is practically impossible to ascertain the actual death rate from tuberculosis in children; the only complete statistics on this point, namely, those of the Registrar-General, are rendered valueless by the lack of post mortem verification in the majority of cases. Some idea, however, of the proportion of cases in which death is due to tuberculosis can be obtained from the post mortem statistics of children's hospitals, and a rough estimate of the mortality from tuberculosis amongst children can thus be be made.

Amongst 769 autopsies on children under twelve years of age at the Hospital for Sick Children, Great Ormond Street, I found tuberculous lesions in 269 cases, and in 223 of these—that is, in 28·9 per cent.—the fatal result was directly due to tuberculosis. In another series of 180 autopsies at the same hospital Dr. Colman [1] found tuberculosis in 59 cases, and it was the immediate cause of death in 51—that is, in 28·3 per cent. Owing to the limitation, by the hospital regulations, of the number of infants (under two years of age) which may be admitted at one time, these figures perhaps rather understate the frequency of tuberculosis; but the error is probably not large, for a very similar proportion was found by Dr. Carr [2] at the Chelsea Children's Hospital, where a series of 330 deaths from all causes showed 100 cases with tuberculous lesions—that is, 30·3 per cent.

It would seem, therefore, that at any rate amongst hospital patients in London nearly one-third of the child mortality is due to tuberculosis; and although such an estimate may for various reasons be too high when applied to the whole child mortality

[1] *Brit. Med. Journ.*, 1893, ii, p. 740.
[2] *Internat. Med. Mag.*, 1893, p. 498.

of this city, it seems almost certain that the average given by the Registrar-General's statistics is far too low. During the five years 1891–5 the average number of deaths per annum from all causes amongst children up to the age of ten years in this city was 36,349; out of these the average number of children certified each year as having died of tuberculosis was 3,335. The evidence of post mortem statistics makes it practically certain that a very much larger number than this die of tuberculous disease.

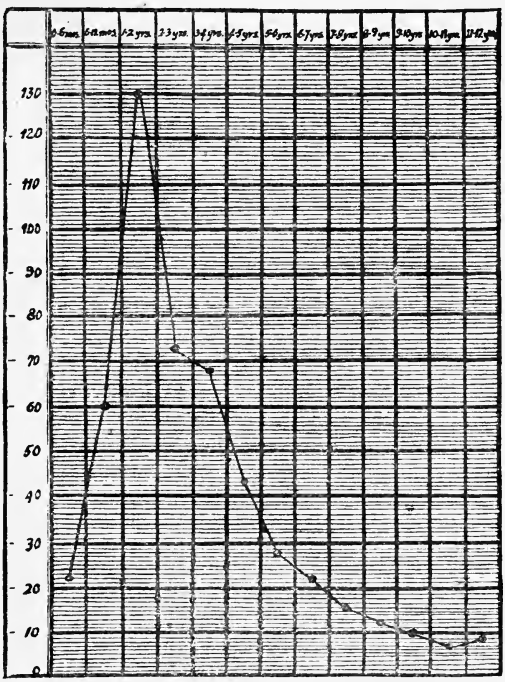

FIG. 24. Chart showing age-distribution of 500 consecutive cases of tuberculosis in childhood.

The incidence of tuberculosis is not the same at all periods of childhood. Statistics show that the special liability to tuberculosis is much more marked during the first five years of life than during the later years. A still more striking fact is illustrated by the chart given above—namely, the remarkable frequency of tuberculosis during infancy, especially in the second year. In 500 cases from the post mortem records of the Hospital for Sick Children no less than 130—that is, more than 25 per

cent.—occurred during the second year. The chart illustrates also the gradual increase of the liability to tuberculosis during the earlier months of infancy; only one case occurred below the age of three months (at ten weeks).

The significance of this very marked age-incidence is a matter of considerable practical importance, for it can hardly be doubted that it bears some relation to the mode of infection, and if prophylaxis is ever to be successful it is all-important that we should determine what this relation is.

The discovery in recent years that the tubercle bacillus is frequently present in cow's milk, as supplied commercially, affords at first sight a very simple explanation of this special incidence of tuberculosis on infants and young children. It has been proved experimentally that tuberculous material given as food may produce tuberculosis in guinea-pigs; cow's milk frequently contains the tubercle bacillus. The period of infancy is the period of milk-feeding; infants, therefore, are specially exposed to infection.

Plausible as such a theory may appear, more evidence is required before it can be regarded as proven; indeed, the facts of the post mortem room are overwhelmingly opposed to it.

I have elsewhere discussed this subject at length, but in view of its important bearing on the question of prophylaxis, I venture to refer here to some of my own conclusions, and to compare with them the entirely independent results obtained by other observers.

The difficulty of determining the channel of tuberculous infection in children is greatly increased by the marked tendency to generalization of tuberculosis at this age. We have, however, a valuable index of its path and duration in the condition of the lymphatic glands, which in childhood show a special susceptibility to the infection of tuberculosis, so that in many cases it is possible from a careful consideration of these to determine with at least a high degree of probability the primary channel of infection.

Amongst 269 tuberculous children under twelve years of age whom I examined post mortem, I found it possible in this way to determine the channel of infection with some degree of certainty in 216 cases: in 138—that is, in 63·8 per cent.—infection appeared to have entered through the lung; in 63—that is, in 29·1 per cent.—primary infection was probably through the intestine; in 15 it appeared to be through the ear.

It would seem, therefore, that if the whole period of infancy and childhood up to the age of twelve years be taken into account,

the view that milk infection is chiefly responsible for the heavy incidence of tuberculosis on childhood is untenable.

But if we take into consideration only the period of infancy, in which we should expect the most striking evidence of the frequency of milk infection, the proportion of cases showing primary intestinal infection, instead of being larger, is actually less. Amongst 100 infants (up to two years of age) in whom the primary channel of infection could be ascertained, 65 appeared to be due to pulmonary infection; only 22 to intestinal. The comparative infrequency of milk infection is even more strongly emphasized by considering only infants up to one year old; it was possible to determine the route of infection in 39 out of 50 infants up to one year old; in 27 out of these 39 infection had occurred through the respiratory tract, in only 5 through the intestine. (In the remaining 7 it was through the ear.)

These observations are confirmed by the statistics of other observers. Dr. Carr,[1] in a series of 120 autopsies on tuberculous children at the Chelsea Children's Hospital, found primary thoracic infection in 65·8 per cent., whilst only 16·7 per cent. showed evidence of primary abdominal infection. Dr. Guthrie,[2] at the Paddington Green Children's Hospital, in a series of 77 cases, found evidence of primary thoracic infection in 42 cases, whilst only 19 appeared to have been infected through the intestine.

From the records of post mortem examinations at the Royal Hospital for Sick Children, Edinburgh, Dr. Shennan[3] collected 355 cases of tuberculosis, in 331 of which he was able to determine the channel of infection: in 67·07 per cent. this was respiratory, in 28·1 per cent. it was alimentary.

Some observations from America by Dr. Northrup[4] showed an even higher proportion of cases with primary thoracic infection —namely, 70 per cent.

The above statistics show a remarkably close agreement between the results of entirely independent observers, and they tend, I think, to disprove some of the exaggerated statements which have been made as to the prominence of milk infection in the causation of tuberculosis in childhood.

The frequency of tuberculous lesions in the mesenteric glands has been put forward as evidence that infection is usually by the intestine in children. But even if merely the relative fre-

[1] *Trans. Med. Soc. Lond.*, 1894, p. 292.
[2] *Lancet*, 1899, i, p. 286.
[3] *Edin. Hosp. Rep.*, 1900.
[4] *New York Med. Journ.*, February, 1891.

quency of affection of the mesenteric glands were to be considered, the argument would fail entirely; for, as will be seen from the figures given below, the thoracic glands are affected far more frequently than the abdominal.

Such an argument, however, cannot be based on a mere study of the relative frequency of affection of the various glands without taking into account also the apparent duration of the process in these glands. In many cases of pulmonary tuberculosis in childhood there are tuberculous lesions in the mesenteric glands as well as in the mediastinal, but this is no proof whatever that there was primary infection through the intestine; indeed, in many such cases it seems quite certain that the primary infection was thoracic, for whilst the thoracic glands show evidence of longstanding disease, and perhaps some old calcareous deposit, the lesion in the intestine and mesenteric glands appears to be of quite recent date.

Moreover, there is an obvious reason why tuberculous affection of the mesenteric glands should be frequently associated with pulmonary tuberculosis in childhood: a child under the age of five years hardly ever expectorates voluntarily; indeed it is often difficult to teach even older children to expectorate. Consequently they swallow the tuberculous material coughed up from the lungs and in this way a secondary infection of the intestine and mesenteric glands might well be expected.

It would seem, then, that the special frequency of tuberculosis in the first two years of life, and to a less degree in the first five years, is *not* 'due chiefly to infection through the alimentary canal by milk from tuberculous cows', as has been stated. The evidence of the post mortem room shows that the commonest channel of infection with tuberculosis in childhood is the lung, not the intestine; and so far from there being any special frequency of intestinal infection during the milk-feeding period, it would seem that this mode of infection is relatively much less common in infancy and in early childhood than it is in older children. My own statistics showed that whilst in children under five years of age the proportion of cases showing primary intestinal infection to those showing primary lung infection was about one to three (39 to 115), in children over the age of five years it was exactly one to one.

The commonest mode of tuberculous infection in childhood, and especially in infancy, is therefore by inhalation; and if it be asked why the liability to pulmonary affection should be so much greater during the first two or three years of life than in

the later years of childhood, there is at least a striking analogy in the age-incidence of bronchitis and pneumonia. Some statistics of children (up to the age of ten years) under my care at King's College Hospital showed that 65 per cent. of the cases of primary bronchitis occurred during the first three years of life and the incidence of bronchitis rapidly diminished after the age of five years. Of cases of primary broncho-pneumonia under the age of five years—and it becomes quite infrequent after that age—80 per cent., according to Holt, occur in the first two years of life. Lobar pneumonia similarly occurs far more frequently under the age of five years than in later childhood.

There is, therefore, good reason for believing that there is some special vulnerability of the respiratory tract during the first five years of life, and hence, although exposure to infection by contaminated air may be shared equally by all ages, it is in infancy and early childhood that such exposure is most often followed by a tuberculous lesion either in the lung or in the lymphatic glands about the air-passages.

This susceptibility to pulmonary infection must be regarded as the chief cause of the special incidence of tuberculosis on infancy and early childhood, and next in importance—but *longo intervallo*—is the liability of this age to milk infection.

There is, however, a third factor which is almost peculiar to this age, namely, the frequency of ear infection. In my series of 269 tuberculous children there were fifteen cases in which it seemed most probable that the primary focus of infection was the middle-ear; all these fifteen cases occurred in children under the age of five years, and no less than thirteen of them occurred within the first two years of life. The tubercle bacillus in these cases probably passes up the Eustachian tube from the naso-pharynx, but whether from the food or from inhaled air it would be difficult to decide; perhaps inasmuch as air is continually passing through the naso-pharynx, a part which food-carried bacilli would only reach indirectly, it is most likely that the bacillus enters with the air.

It has been suggested that the tonsil and naso-pharyngeal adenoid tissues are in some cases the portals of entry for tuberculosis: Dr. Hugh Walsham found tubercle of the tonsils in 20 out of 34 cases of tuberculosis, chiefly in adults. The tubercle beginning in the tonsil infects the cervical glands and spreading downwards along the chain of cervical glands reaches the mediastinal glands and thence spreads to the lungs.

Such is the theory, but it must be remembered that the

frequency of tubercle in the tonsil is open to exactly the same interpretation as the tubercle in the intestine. Every time tuberculous sputum is coughed up there is a risk of infecting the tonsil, and it is likely enough that a secondary infection of the tonsil should occur where there is already disease in the lung.

Moreover, there is good reason for believing that direct extension of tubercle from the cervical glands to those in the mediastinum if it occurs at all is extremely rare. If this occurred one would expect to find from above downwards a less and less advanced stage of disease in the glands, so that while the glands at the upper part of the neck were much enlarged and completely caseous those of the lower part might be smaller and partially caseous, whilst those in the mediastinum would show perhaps only scattered foci of tubercle and be still less enlarged. So far as the chain of glands in the neck is concerned, post mortem observations show that this supposition is correct, the enlargement and caseation is progressively less from above downwards; but the condition of the glands in the mediastinum entirely fails to accord with this view, for, when they happen to be affected in such cases, where the affection of the glands at the lower part of the neck is extremely slight, with little or no enlargement and only a few grey points of tubercle, the glands next below in the mediastinum show enlargement and caseation quite as advanced as the topmost of the cervical glands.

It seems, therefore, most natural to suppose that the mediastinal glands have been infected in the usual way through the lung.

There is another very strong reason for supposing that any extension of tubercle directly from the neck to the mediastinum must be very exceptional. The glandular affection in the mediastinum is for some unknown reason much more frequent and more extensive on the right side than on the left. This was pointed out by Dr. F. E. Batten,[1] and my own figures corroborate his observations: in 113 out of 145 cases I found that the mediastinal glands showed more advanced tuberculosis on the right side than on the left; only in 32 was the affection greater on the left side, and, as I shall point out, perforation of the bronchus by a gland is far commoner on the right than on the left.

If the mediastinal glands were commonly affected by extension of tubercle from the cervical glands one would expect that the cervical affection also should be earlier and more frequent

[1] *St. Bart. Hosp. Rep.*, vol. xxxi.

on the right side than on the left. Statistics of operations on tuberculous cervical glands, kindly collected for me by Dr. Rowland, showed in 100 cases in which one side was chiefly affected 51 on the right side, 49 on the left; in other words, the two sides of the neck were almost equally affected.

But, although extension of tuberculosis from the cervical glands to the mediastinum is probably very rare, one can hardly doubt that unhealthy tonsils and adenoid tissue play a part in the production of glandular tuberculosis in the neck, if only by causing enlargement of the glands, and providing a nidus in which the tubercle bacillus very readily gains a hold, but the observations of Dr. Walsham and others make it almost certain that they sometimes play the more direct part by transmitting the tubercle bacillus.

Apart from the channels of infection—and I have considered here only the more frequent, and therefore more important channels—there are two predisposing causes which play no small part in determining the heavy incidence of tuberculosis on early childhood; and keeping in mind the paramount necessity for more efficient prophylaxis, I wish to lay some stress upon these two potent factors in the etiology of tuberculosis at this age: they are measles and whooping-cough.

Again and again one hears the same tale—that a child, apparently healthy, has had measles, and then, sometimes immediately, sometimes after an interval of weeks or months, has begun to lose flesh and shows insidious symptoms which, as the event proves, are the result of tuberculosis. Many a case of tuberculosis follows directly on whooping-cough; sometimes even before the whoop has ceased the wasting and the pulmonary signs which were thought to be due simply to the whooping-cough begin to assume a more serious aspect, and the child dies with tuberculosis.

This very frequent occurrence of tuberculosis as a sequel of measles and whooping-cough no doubt contributes to the special incidence of tuberculosis on infancy and early childhood, for nearly 50 per cent. of the cases of whooping-cough occur during the first two years of life, and about 80 per cent. under the age of five years, whilst in London at any rate measles also occurs much more often in early childhood than in later years.

It seems likely that the catarrhal element in these two diseases is the particular predisposing cause of the subsequent tuberculosis, and one can well imagine that the swollen condition of the lymphatic glands in the mediastinum, which is a marked

feature in some cases of whooping-cough, and is also a common result of the pulmonary complications of measles, must favour the starting of a tuberculous process in them.

The prominence of glandular infection in the tuberculosis of childhood has already been mentioned, but it is so important a feature from the clinical standpoint that I shall consider it here again with special reference to the mediastinal glands.

Tuberculosis of mediastinal glands. One of the commonest lesions found post mortem in tuberculous children is enlargement and caseation of the mediastinal lymphatic glands; in many cases this is quite out of proportion to the tuberculous lesion in the lungs, which may be so slight as to have produced no signs during life. In some cases this affection of the mediastinal glands appears to be the earliest gross lesion, for in children who have died of other diseases these glands are sometimes found to be caseous or calcareous when no other tuberculous lesion is found in the body. It seems most probable that in nearly all such cases the bacillus has entered through the lung, even although no lesion may be found there; but, however infection has entered, there is no doubt that such a focus may directly infect the adjacent pleura, lung, or bronchus, or it may be the source of infection through the blood-stream for a tuberculous meningitis or for some more widely disseminated form of tuberculosis.

The presence of calcareous foci alone in some cases proves that even after caseation has commenced in the mediastinal glands complete recovery may take place; it is, therefore, of the utmost practical importance that this condition should be detected as early as possible. My own experience leads me to believe that with careful treatment, climatic and otherwise, children who come under observation with wasting, perhaps slight cough, and well-marked signs of enlargement of the mediastinal glands, which there is every reason to regard as tuberculous, may make a good recovery if only treatment is begun sufficiently early.

Some idea of the frequency of this affection and also of the similar condition in the mesenteric glands may be obtained from the following figures: my own statistics showed that in 254 tuberculous children in which the condition of the glands was noted, there was caseation of the mediastinal glands in 209—that is, in 81 per cent.; and caseation of the mesenteric glands in 151—that is, in 59 per cent. Dr. Carr's figures agree

closely with these; he found caseous mediastinal glands in 80 per cent. and caseous mesenteric glands in 54 per cent.

It would seem, therefore, that the mediastinal glands are more often affected than the mesenteric, and it is interesting to note that when only one or other of these groups of glands is affected, the mediastinal are those which are more likely to be affected. In a series of 67 such cases there were 45 in which the mediastinal alone showed caseation, and only 22 in which the mesenteric alone were affected.

Apart from their obvious bearing upon the question as to the most frequent channel of infection, these figures have a practical value in emphasizing the necessity for including in the routine examination of the chest in children the examination for signs of enlarged mediastinal glands. I shall refer to the clinical aspect of tuberculosis of the mesenteric glands in a subsequent chapter, here I shall consider only the diagnosis of the mediastinal affection.

Diagnosis of tuberculous mediastinal glands. Is it possible to recognize the presence of enlarged glands in the mediastinum? I think that from certain symptoms and signs it is sometimes possible to diagnose this affection with at least a high degree of probability.

The symptoms which may point to it are those common to tuberculosis in other parts of the body, an ailing condition with loss of flesh, and some irregularity of temperature. There are also certain characteristic symptoms which if present are of much value in diagnosis, but they are frequently lacking; one is a curious clanging cough somewhat paroxysmal in character, so that whilst partaking to some degree of the brassy character of the cough produced in an adult by aortic aneurism, it is easily mistaken for whooping-cough, as has sometimes happened in my own experience. Another is inspiratory stridor due probably to pressure upon the trachea; I have elsewhere quoted instances of this (p. 338).

As a rule the diagnosis has to be made from physical signs which are sometimes sufficiently characteristic.

1. Impairment of note in the first and second intercostal spaces close to the sternum: this is of more value if it occur on the right side, for if it be only on the left it may be normal as there is often quite a finger's breadth of impaired note adjoining the sternum in the first left space and still more in the second, presumably due to the large vessels passing up from the heart.

2. Enlargement of veins, usually most noticeable in the second

space where one large vein is seen passing inwards from just below the coracoid process to the inner end of the space: this sign is of value chiefly when it is limited to one side of the chest; there are many thin-skinned children in whom a plexus of veins is normally visible all over the upper part of the chest, no importance can be attached to this.

3. A bruit heard just below the inner end of the clavicle on extending the head as fully as possible with the child in the sitting or standing position. The late Dr. Eustace Smith,[1] who first described this sign, regarded the sound as a venous hum, produced by the bending back of the head tilting forward the lower end of the trachea with its adjoining glands, which are thus made to compress the left innominate vein. I have examined a large number of children for this sign, and have often found it in cases where there was other evidence of enlarged tuberculous glands in the mediastinum, and I think that taken in conjunction with the other signs and symptoms it is of real value in the diagnosis of this condition; but it is certainly not always present where there are undoubtedly enlarged and even greatly enlarged mediastinal glands. I have often heard it also where there was considerable anæmia and perhaps a *bruit de diable* in the neck without anything to suggest tuberculosis or enlargement of mediastinal glands.

4. A marked deficiency of air-entry into some part of the lung. This is particularly characteristic if it involves a whole lobe. It is thought to indicate pressure upon one of the bronchi.

5. Dullness about the root of the lung in the interscapular space. This cannot be due directly to mediastinal glands, but points to consolidation of the lung near its root which is particularly likely to happen when the glands which lie in contact with the earliest divisions of the bronchi are enlarged, as they usually are where those in the mediastinum are affected.

6. Increased resistance on pressure over the manubrium is sometimes noticeable where there is much enlargement of mediastinal glands. This can be appreciated by steadying the child by one hand placed behind against his upper dorsal spine while the fingers of the other hand make an intermittent pressure upon the manubrium. In the healthy child, especially in the earlier half of childhood, there is a well-marked normal resilience, and with sufficient *tactus eruditus* one can detect a distinct diminution or loss of this resilience in some cases of enlarged mediastinal glands.

[1] *Lancet*, August 14, 1875.

7. Rarely the upper rounded edge of one of the enlarged glands can be felt by pressing the finger downward behind the top of the manubrium.

There are cases in which the enlarged glands can be demonstrated by the X-rays, but often the findings are of doubtful significance for an opacity about the root of the lung might equally well indicate some caseation in the lung itself, moreover,

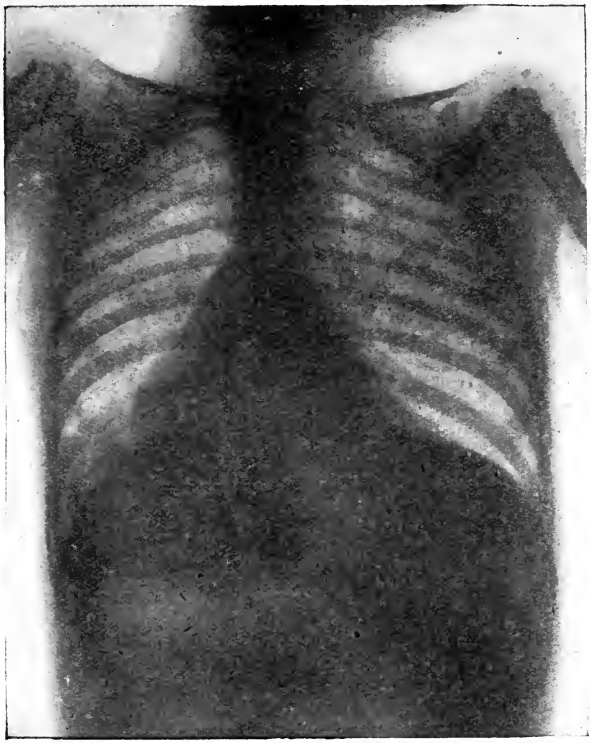

Fig. 25. Skiagram of child's chest showing opacity at root of lung on both sides, probably due to caseation of mediastinal glands.

if the glands are but slightly caseous they are not likely to show at all. In the skiagram reproduced here, taken from a child about seven years of age, the opacity about the root of the lung is probably due to caseation of enlarged glands in this position.

I have described these indications of enlargement of mediastinal glands at some length because I have several times seen children with obscure symptoms such as prolonged fever and wasting which were explained, as the event proved correctly,

by the detection of signs pointing to this affection. The outlook is not necessarily a bad one; I have seen cases in which the subsequent appearance of tuberculosis in some other part of the body confirmed the diagnosis which had been made previously of tuberculous glands in the mediastinum, but nevertheless the child made what appeared to be a complete recovery both from the earlier and from the later tuberculous affection.

On the other hand, I have seen the condition declare itself by pointing of the softened glands. In one girl, aged 9½ years, who had been wasting for two months, no physical signs had been detected, but a fluctuating tumour appeared in the second right space close to the sternum; my colleague, Mr. Burghard, incised and evacuated what was evidently the remains of a softened caseous gland which had tracked outwards from the mediastinum. In another child an area of softening appeared in the middle of the sternum and a caseous mediastinal gland discharged itself through the bone.

Dr. Voelcker[1] and others have recorded cases in which perforation of a bronchus resulted in sudden death from asphyxia, a part of the caseous gland becoming lodged in the glottis. In doing autopsies at the Children's Hospital I have seen at least four cases in which a gland ulcerated into the œsophagus, but in none of them did this accident produce any clinical symptoms.

It is not only by their local effects that tuberculous glands in the thorax or abdomen of a child are fraught with danger; their presence involves continual risk of an even more serious blood-infection. One of the commonest endings—and perhaps the most distressing—in the tuberculosis of childhood is tuberculous meningitis. In 238 children who died with tuberculosis I found tubercle of the meninges in 114 cases—that is, in 48·3 per cent., and it was the immediate cause of death in most of these cases.

One sometimes hears of a 'primary tuberculous meningitis', but I have never yet seen a case in a child where careful post mortem examination failed to reveal some tuberculous focus elsewhere which was evidently of older standing than the meningitis; and often, when the rest of the body seems to be free from tuberculosis, the mediastinal or mesenteric glands show some focus of caseation—and I fancy that an actually softened area is more mischievous in this respect than firm caseation—which has probably been the *fons et origo* of the meningeal infection.

[1] *Practitioner*, June, 1895.

Pulmonary Tuberculosis. The frequency of caseation of the mediastinal glands is to some extent the cause of the differences between the pulmonary tuberculosis of children and the phthisis of adults—I use these terms advisedly as accentuating the fact that such differences exist.

The lung affection in childhood tends to be not only a more acute process, but also more diffuse than in the adult. The slowly progressing form of phthisis which is so familiar in the adult, and which for a long time may remain limited to its starting-point just below the apex of the lung, with cavities lined perhaps by a well-defined wall and traversed by trabeculæ of fibrous tissue or blood-vessels, is quite unusual in childhood. It is common in children to find the tuberculous infection spreading in from the root of the lung apparently directly from the caseous bronchial glands, and starting thus it tends to affect the lower part of the lung almost as much as the upper. Moreover, it is not a very rare occurrence in children for one of the caseous mediastinal glands to perforate a bronchus, and if this perforation occur beyond the first division of the bronchus the caseous débris of the gland may be discharged into one lobe only of the lung and set up an acute tuberculous process, sometimes with rapid cavitation throughout the whole of that lobe, while the rest of the lung is only slightly affected.

The limitation of signs of active tuberculosis to one lobe when there are signs of enlarged mediastinal glands is highly suggestive of ulceration of a caseous gland into a bronchus; and the more so if the signs be on the right side.

In 260 tuberculous children under twelve years of age, in whom the chest was examined post mortem, I found perforation of a bronchus by one of the caseous mediastinal glands in 23 cases. This occurs much more frequently on the right side than on the left; in 21 out of these 23 cases the perforation had occurred into the right bronchus.

But without actual perforation the bronchus may be infected by direct extension from an adjacent gland, and I have often found the mucous membrane of the bronchus in such cases studded with grey or greyish-yellow tubercle, or already showing superficial ulceration, a source, no doubt, for extensive infection of the lung. Nor is it only the root of the lung which may be infected thus; there is a lymphatic gland lying in contact with the right side of the trachea just above the bifurcation, which is very frequently found to be caseous. From this gland the caseous process sometimes spreads directly to the adjacent pleura, and, after formation of local adhesions, spreads into the right lung from without inwards, just below its apex.

As I have already said, it seems probable that in the vast majority of cases these glands are primarily infected through the lung with or without the formation of an actual focus of tuberculous disease in the lung, but when once the glands are infected a vicious circle is formed, and perhaps to some extent the wide dissemination of tubercle in the lung of a child is due

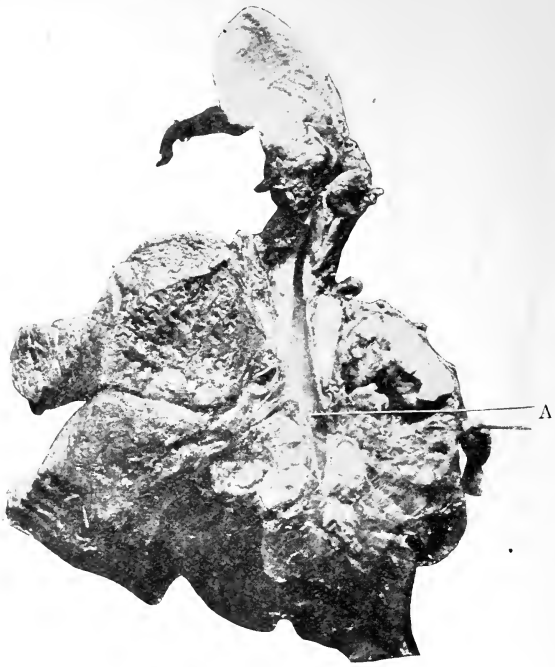

FIG. 26. Common type of pulmonary tuberculosis in an infant. Caseous mediastinal glands; perforation of right bronchus (the opening is indicated by the fine line A); caseation and cavitation of right upper lobe. The specimen shows also the enlarged and caseous right tracheal gland between the trachea and the right upper lobe, and the greatly enlarged caseous bifurcation gland between the two bronchi, and a large caseous root gland imbedded in the root of the right lung. The left lung is very little affected by tubercle. From an infant aged nine months.

to the fact that not only is there the primary infection by inhalation, as in the adult, but also this re-infection from the mediastinal glands.

Apart from this glandular infection, the early deposit of tubercle in the lung of a child shows the same tendency as in the adult to occur just below the apices of the upper and lower lobes, but it tends to run a more rapid course. The deposits of tubercle

occur as a few scattered grey tubercles which in parts are grouped together in racemose fashion and in parts have advanced to a stage of caseation forming irregular yellowish solid areas, in fact a caseous broncho-pneumonia.

Cavitation occurs by no means rarely—I found cavities in 60 out of 212 children with pulmonary tuberculosis—but it is an

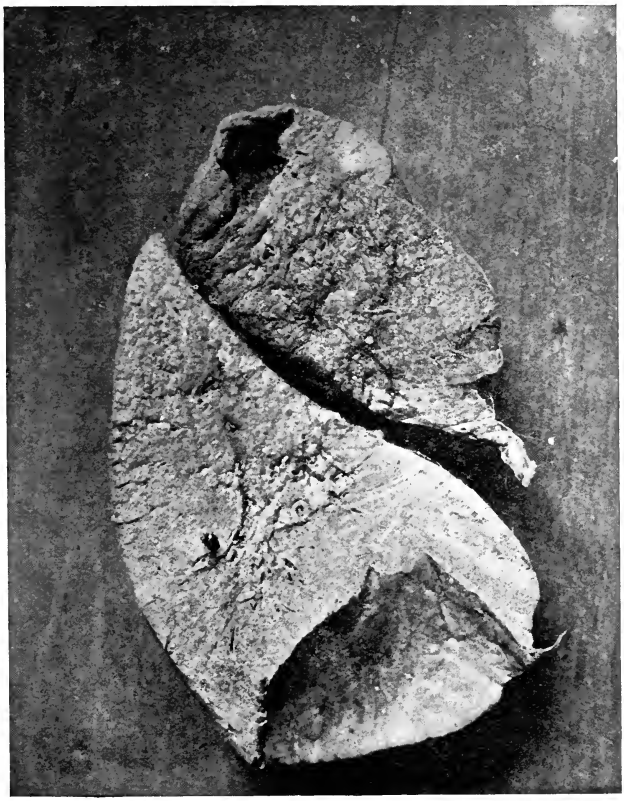

FIG. 27. Pulmonary tuberculosis in a child. Unusual type, with chronic cavitation resembling the phthisis of adults.

acute cavitation, the rapid softening of a caseous area, and anything like the fibrosis which attempts repair in the adult is completely lacking, as a rule, in the child. These different varieties of cavitation in a child's lung are seen in Figs. 26 and 27.

Occasionally the caseation is diffuse and involves a large part of one lobe, which is thus converted into a cheesy mass with

little trace of lung-tissue remaining. Such a condition is shown in the accompanying illustration (Fig. 28), from a child aged eighteen months. The clinical signs of this form are sometimes most misleading, for the breath sounds may be almost entirely abolished over the affected lobe, and the absolute dullness and very marked resistance on percussion complete the likeness to

FIG. 28. Pulmonary tuberculosis: diffuse caseous consolidation in a child aged eighteen months; lower lobe almost completely caseous.

pleural effusion. I have known exploration both by needle and by incision done in such a case, where the signs had exactly simulated those of empyema.

A variety of pulmonary tubercle which is less common than those I have mentioned is the general miliary tuberculosis which forms part of a similar acute miliary affection of almost all the organs. As is shown by the photograph reproduced

here (Fig. 29) the lung is uniformly and closely studded with grey or greyish-yellow tubercles; and one might expect from its appearance that the signs would be characteristic enough to make diagnosis easy; as a matter of fact, the signs are usually only those of bronchitis, numerous râles all over the chest which as the disease advances often become somewhat sharp and crackling in character, but even in the most advanced stage of the

Fig. 29. Acute miliary tuberculosis of the lung in a child.

disease there is usually no dullness or bronchial breathing. The diagnosis rests rather on the associated symptoms than on the physical signs; the child is acutely ill, with dusky face and purple lips, the respiration is hurried, the spleen is enlarged and extends usually two or three finger-breadths below the costal margin, the temperature is continuously elevated like that of a patient with typhoid and, lastly, the diagnosis can usually be made certain in these cases by examining the fundus oculi.

Tubercle of the choroid, which is rare enough in other forms of tubercle, is present in a large majority of the children with general acute miliary tuberculosis.

In accordance with the tendency of tuberculosis in the child to generalization, pulmonary tuberculosis at this age is so often part of a more general infection that the outlook in any case is necessarily very grave. In the acute miliary form there is no hope of recovery, the fatal termination is usually within six to eight weeks after the onset ; but in the other forms with localized signs the outlook is by no means hopeless. It is encouraging to find evidence in the post mortem examination of children who have died from other diseases, that even in quite early life complete healing of a tuberculous lesion may occur in the lung as well as in the mediastinal glands. Of course the earlier the condition is recognized and treated the better is the chance of recovery.

Diagnosis of pulmonary tubercle. The diagnosis of pulmonary tubercle in the child is often a matter of considerable difficulty: a bronchitis or a pneumonia which runs a prolonged course, as happens so often after measles or during whooping-cough, raises suspicions of tuberculosis which may or may not be correct, there are crackling râles and high-pitched breath sounds in patches, perhaps definite impairment of note and bronchial breathing at some part of the lung, but the child gradually recovers, and the diagnosis remains uncertain.

There are two symptoms upon which an emphasis is often laid which might be quite justified in the case of an adult, but has little justification with reference to children, namely sweating and hæmoptysis. Children sweat very easily, especially when the health is depressed : rickets, debility after any acute illness, anæmia from any cause, may be ample explanation of profuse sweating at night ; the presence of night sweats is, I think, of no value whatever in the diagnosis of tubercle in children.

Hæmoptysis is very rarely due to pulmonary tuberculosis in children, it usually means whooping-cough or is due to pulmonary congestion with advanced heart disease, unless, as sometimes happens, it is a spurious hæmoptysis due to bleeding from the posterior nares or from the gums. Very rarely does a child with pulmonary tubercle cough up any blood ; and a fatal hæmoptysis from this cause is an extreme rarity in childhood : amongst the many hundreds of children with pulmonary tuberculosis who have been under my observation I have only twice known death to occur from hæmoptysis ; once in a child about three years of age and once at the age of eleven months.

Until recently the bacteriological examination of the sputum has generally been regarded as impracticable in children under three years of age, for they swallow their expectoration, and even older children usually do this unless instructed to do otherwise; but Dr. Emmett Holt [1] has shown that it is possible to obtain sputum and to verify the diagnosis by its bacteriological examination even in infants. He recommends exciting a cough by irritating the pharynx by a small bit of muslin held in an artery clamp, the secretion is coughed up into the pharynx and caught on the muslin; in this way tubercle bacilli were detected in the sputum in 80 per cent. of cases examined at the Babies' Hospital, New York, where most of the cases were under two years of age.

There is one condition which must be mentioned here as sometimes mistaken for pulmonary tuberculosis in children, namely, fibroid lung. Impairment of note with tubular or perhaps cavernous breathing, sharp crackling râles, bronchophony and pectoriloquy are found over a limited area, most frequently, I think, at the left base, just below the angle of the scapula. The rest of the lung shows nothing, but perhaps, as is common in these cases, there are a few crepitations to be heard near the base or the root of the other lung. So far the signs would accord well enough with tubercular consolidation and cavitation, but on further examination it is found that the heart is displaced towards the affected side, and careful inspection detects some slight flattening or contraction of the lower part of the chest on that side; and the child's fingers just above the nails are found to be full, shiny and pigmented if there be not definite clubbing. On inquiry also the mother states that the child expectorates a considerable quantity of mucopurulent sputum, especially on awaking in the morning, and perhaps that the breath and the sputum smell very offensive at times. The signs and symptoms are in fact those of fibroid lung with bronchiectasis. In an adult such a condition might be due to chronic tuberculous affection of the lung, but in a child—conformably with the tendency to rapid caseation which I have already mentioned—an extensive fibrosis, such as is indicated by these signs, is very rarely due to tubercle; it is the result usually of a simple broncho-pneumonia of prolonged duration at some former time, especially that due to whooping-cough, or of a former pleurisy with much lymph exudation, or of a small empyema which has been left unopened and has

[1] Kelynack's *Tuberculosis in Infancy and Childhood*, p. 99.

gradually dried up. Occasionally it seems to be due to a chronic syphilitic pneumonia.

Tuberculosis is so rare as a cause of fibroid lung in children, that such signs as those I have described would be almost enough to disprove the presence of tubercle; but the sputum is generally easily available in these cases, so that more positive evidence can be obtained.

Lastly, I must mention the most recent methods of diagnosis by the tuberculin reaction. These are Calmette's ophthalmic reaction (instillation of 1 drop of a ½ or 1 per cent. solution of tuberculin into the eye, which in tuberculous subjects should show within twenty-four hours a definite congestion of the conjunctiva), Von Pirquet's cutaneous reaction, and the Moro cutaneous reaction; the opsonic index must also be mentioned, though, owing to the elaborate technique and special experience required, it is seldom available in the ordinary routine of general practice.

As to the ophthalmic test there is no doubt that in the healthy eye it may set up a severe conjunctivitis, and I have seen children in whom two or three weeks after it had been applied a fresh conjunctivitis appeared in the eye which had been tested; this in itself is not a trifling matter, but it might be outweighed by the value of the test if this were found to be specially reliable.

It is, however, now quite clear that its reliability is no greater than that of the other tuberculin tests, and the risk of causing serious mischief in the eye is a very real one, as experience has proved; the method has already fallen into well-deserved disrepute.

The choice, therefore, lies between the Von Pirquet and the Moro reactions, and, so far as my own experience goes, the Von Pirquet would appear to be the more reliable.

The Moro reaction is obtained by gently rubbing into a small area of skin (usually a patch about two inches square in the epigastric region) a specially prepared ointment, containing tuberculin; after about twenty-four hours this area shows reddening, often with some eruption of small papules if tuberculosis be present, whereas if this be absent the skin remains normal.

For the Von Pirquet reaction a fluid preparation of tuberculin is inoculated in exactly the same manner as an ordinary vaccination, except that only one site, usually about the size of a threepenny piece, is inoculated; after twenty-four to thirty-six hours this area becomes reddened and slightly raised if tubercle be present, whereas a control area scratched similarly without application of the tuberculin shows no such reddening.

The chief fault of these reactions is their extreme delicacy, they are too successful, if one may say so; they detect the smallest trace of tubercle, which may be entirely latent, and may have nothing whatever to do with the symptoms from which the child is suffering. A large proportion of children past the age of infancy have at some period become infected with tubercle, even if it be only in a single lymphatic gland in the neck or the mesentery or elsewhere, and the likelihood of such latent tubercle is greater the older the child; consequently these tuberculin tests are likely to give a positive result in many children who show no clinical symptoms whatever, and may never show any, of tuberculous disease: but in addition to this positive fallacy there is a negative failure also of these tests, for the reaction often does not appear when the tuberculosis is of acute form or is widely diffused and much advanced. The child with tuberculous meningitis, for instance, may give a negative result with the tuberculin test, so also the child with acute miliary tuberculosis. Nevertheless in infancy and early childhood, perhaps up to the age of three or four years, these tests have some value, and even in later childhood a negative result in some conditions may carry weight in determining whether particular symptoms are due to tubercular disease. How little value is to be attached to a positive result in these older children is shown by the fact that various observers have found evidence of tubercle by this test in 50 to 65 per cent. of all children between the ages of five and fourteen, although a large proportion of them showed no symptoms whatever to suggest tuberculosis.

Lastly, the opsonic index, as I have already mentioned, is but seldom available, and even when it can be obtained, its practical value for diagnosis is, I think, less than that of the tests already mentioned. It is difficult to arrive at any certainty in the interpretation of its variations. General rules can easily be laid down, but in the individual case so much doubt attaches to the significance of the unduly high or unduly low or much varying index, which may possibly indicate tuberculosis, that one is hardly justified in attaching much weight to it.

Treatment

My remarks upon the treatment of tuberculosis in children will be rather of general application than with reference to any particular form of the disease.

Prevention is better than cure: there is no period of life at

which the liability to tuberculosis is greater than during childhood, especially during the first five years of life : for this reason prophylaxis is specially needful during this period and particularly in the case of the child who comes of tuberculous stock. The two chief sources of infection are the breathing of infected air and the drinking of infected cow's milk.

In order to avoid the risk of aerial infection it is most important that a child should not be allowed to associate with persons who have active tuberculous lesions in any part of the body.

Tuberculous disease in one part indicates a tendency to tuberculosis which may be active although latent in other parts; the person with tuberculous bone disease or with tuberculous glands in the neck may be a source of danger through incipient and unsuspected disease in the lung. In the choice of domestic nurses and of the child's companions this is specially to be borne in mind. I have seen cases in which it seemed clear that a child's life was sacrificed by allowing him to stay even for a few weeks in a house where there was some person with phthisis.

No doubt the risk of such infection is diminished by thorough ventilation in the house and by keeping the child out in the open air as much as possible ; but whenever it is practicable a child should be kept altogether from association with tuberculous persons, whether they be children or adults.

The risk of infection from cow's milk is a very real one; my own figures show that rare though alimentary infection may be in comparison with respiratory, it probably accounts for nearly one-third of the fatal cases of tuberculosis in childhood ; and it can hardly be doubted that the source of the bacillus in these cases is cow's milk.

Some years ago I was opposed in general to the boiling of milk for children : with larger experience I have adopted different views; I have seen so many cases in which there was good reason to suppose that unboiled cow's milk was the source of infection, that I regard it as always advisable to pasteurize or boil cow's milk for children during the most susceptible period, the first five years, and longer if the child has shown evidence of tuberculous tendencies, for instance, tuberculous glands in the neck, or has a strong family history of tuberculosis.

There can be little doubt that properly regulated inspection of cows and cowsheds would do much to abolish milk infection altogether; until this ideal is attained safety can be ensured only by the pasteurization of milk or by heating it nearly or quite to the boiling-point.

'But,' says the parent, 'we have our own cows, and they are specially tested with tuberculin; surely there is no need to pasteurize or boil the milk.' Even under such conditions I would not relax this precaution, for the test is usually applied only at intervals of some months, and in the cow, as in man, tuberculosis is sometimes a rapid process; the tuberculin test is no guarantee whatever that the cow may not develop the disease before the next testing. No doubt affection of the udder, which is most likely to be associated with tuberculous milk, does not occur as a rule until the disease in other parts of the body is somewhat advanced, but there is ample evidence that milk may be infected where there is only disease in other parts. The conclusion of the whole matter is, I think, that under any circumstances cow's milk should be pasteurized or boiled for children at least up to the age of five years, and that this practice is a wise precaution for any period of childhood.

If for any reason there is difficulty in giving cows' milk which has undergone boiling or pasteurization—and there are children who will not drink milk when it has been treated in this way—goats' milk makes an excellent substitute; it can be given unboiled with exceedingly little risk so far as tubercle is concerned, for tuberculosis is extremely rare in goats.

The special liability of children to tuberculous affection of lymphatic glands has a practical bearing upon the prophylaxis of tubercle at this age. A swollen lymphatic gland, whatever be the cause of the swelling, seems to furnish a nidus in which tubercle bacilli thrive; an unhealthy condition of the throat with enlarged tonsils and adenoid hypertrophy is therefore a danger to the child with tuberculous tendencies, for the resulting enlargement of glands at the angle of the jaw makes them specially liable to tuberculous invasion.

So too with carious teeth, particular attention should be paid to the teeth of children who come of tuberculous family or have already shown manifestations of tubercle themselves, for whether the bacillus enters through the unhealthy dental alveolus or not, the simple enlargement of glands due to an unhealthy condition of the teeth and gums probably predisposes to tuberculous disease in these glands and from them it may spread elsewhere.

I have referred above to the frequency with which tuberculosis follows measles and whooping-cough, and one can only deplore the reckless way in which children with either of these diseases are allowed to scatter infection broadcast. There seems to be

a current idea that, in comparison with other specific fevers, these are of little consequence; but I fancy that if it were possible to add on to the direct mortality of these two diseases the large number of deaths from tuberculosis which are indirectly due to measles or whooping-cough, we should realize more fully the danger of such infection. Certainly for children who are predisposed to tuberculosis by heredity, no unimportant part of the prophylaxis against tuberculous disease is the guarding against exposure to the infection of measles or whooping-cough; and probably much may be done to avert the onset of tuberculosis after an attack of measles or whooping-cough by sending the child away for a time into the pure air of country or seaside.

The choice of climate for the child with tuberculous tendencies, or declared tuberculous disease, is an important matter. I think it may be said generally that the tuberculous child derives more benefit from seaside than from inland places, and that if the seaside is not available, high ground with a sand or gravel soil is much more suitable than a low-lying place. Children stand cold winds badly, a bleak spot wind-swept from north and east is not a suitable place for the tuberculous child however high and dry it may be. Whatever be the place chosen it must possess such a climate as regards sunshine, temperature, and rainfall that the child can live in the open air the greater part of the day, and this qualification is quite as important as the particular aspect or altitude of the place.

One more general principle is, I think, worth notice before mentioning particular places, namely, that many children seem to do better with a change of climate after a few months than if kept permanently at the place which at first seemed to suit admirably.

Amongst seaside places the east coast resorts, Cromer, Sheringham, Felixstowe, Southwold, Frinton, and Hunstanton, are all excellent in the heat of summer, but except during this hot season it has seemed to me that tuberculous children do better in places where cold winds are less prevalent: the Kent coast resorts, Westgate, Margate, and Broadstairs, have a deservedly high reputation for tuberculous diseases, but sometimes as early as the end of October and usually, I think, about the end of December or January these places are too cold and biting. Folkestone and St. Leonards may be more suitable for the winter months if the winter be not severe, but usually it is wiser to send the tuberculous child further west at this time of the year; Torquay, Bournemouth, or Sidmouth will be suitable

places. For those who are unable to go far from London, Westcliff near Southend offers a climate which is sunny and warm enough to allow the child to be in the open air great part of the day even in winter.

For those who prefer to take their children inland, a residence high up on the slopes of the Cotswold or the Chiltern Hills gives excellent results. Hindhead or Tunbridge Wells are also suitable, and in summer Crowborough or the Surrey Hills are admirable for tuberculous children. I have referred chiefly to parts of the suitability of which I have personal knowledge, many other places will occur to those whose experience lies elsewhere: there are many places on the continent which have the reputation of being specially valuable for tuberculous children, for instance in France, Berck-sur-Mer, which, according to Dr. Walter Carr[1], has a climate resembling that of Margate or Broadstairs, or Arcachon, which is said to have a climate like that of Bournemouth; while for the winter months Cannes in the French Riviera and Alassio in the Italian, are specially recommended for tuberculous children.

Lastly, I must refer to the medicinal treatment which, though far less important than the climatic, has its value. There is probably no drug so generally useful for tuberculous children as cod-liver oil, but I think it is often given in too large doses; a drachm of the oil is more than some children can tolerate at six or seven years of age and for those who can tolerate it I doubt whether there is any advantage from giving larger doses. Often I think a combination of malt extract with cod-liver oil is better tolerated than the plain oil or the ordinary emulsion of cod-liver oil; and for the child who is sick or has disturbance of digestion when given cod-liver oil, malt extract alone or in combination with iron iodide is valuable.

Both for the pulmonary and for the abdominal lesions of tubercle, creosote has seemed to me to be of value; in the former case it can be given by inhalation, 5–10 drops of a mixture of equal parts of creosote and spirits of chloroform are dropped on the sponge of a Yeo's inhaler which the child wears for twenty minutes several times a day. Dr. Lees recommends for this purpose a formula of Acid Carbol. ℥ii, Creosote ℥ii, Tinct. Iodi ℥i, Spirit. Ætheris ℥i, Spirit. Chloroform ℥ii. I have not found it practicable with children to carry out the almost continuous inhalation which he advises for adults, but I think that even the interrupted treatment by this method, amounting to

[1] *Practitioner*, July, 1908.

1½–3 hours daily, is of real value. For tabes mesenterica and tuberculous peritonitis the creosote can be given in doses of ½–1 minim in 1–2 drachms of cod-liver oil emulsion or, if the bowels are loose, in an emulsion containing 5 minims of castor oil.

I have referred elsewhere to the value of iodoform in tuberculous peritonitis.

The time is not ripe yet to speak confidently of the value of tuberculin in the treatment of tuberculosis in children: even the dosage is at present undetermined, some observers recommend doses of $\frac{1}{5000}$ milligramme or less for an infant at one year, whilst others give $\frac{1}{1500}$ at the same age; Dr. Clive Riviere[1] mentions $\frac{1}{2000}$ milligramme at five years, and $\frac{1}{6000}$—$\frac{1}{4000}$ at one year. Using injections of the new tuberculin T. R., I have usually, in the case of a child of three years or older, begun with $\frac{1}{10000}$ milligramme, and after two or three injections at intervals of five days, increased the dose to about $\frac{1}{8000}$, and after a similar interval to $\frac{1}{5000}$ and then to $\frac{1}{3000}$, and in some cases to $\frac{1}{1000}$ milligramme; and when using tuberculin by the mouth as suggested by Dr. Latham,[2] I have given $\frac{1}{3000}$ milligramme in five cubic centimetres of horse serum, or in half a wineglassful of warm milk to a child of eight years: the dose is given two hours before breakfast, so as to ensure an empty stomach. As to the frequency of administration, whether it should be every three or four days or only once a week, there is also a difference of opinion, and the necessity of determining this question by repeated estimations of the opsonic index is another moot point.

I have seen children do well who had had tuberculin subcutaneously, or by mouth, or by rectum, but as in all therapeutics one must be careful in drawing conclusions as to cause and effect; and the more so when as in this case there is much discrepancy between the dosage and the results of different observers.

Of the possibility of doing good by such therapeutic measures as I have described there is no doubt, but the evidence of morbid anatomy and the results of clinical experience seem to show that, whilst we may do much for the tuberculous child by careful treatment, it is chiefly to a more efficient prophylaxis that we must look for any considerable decrease in the terrible mortality from tuberculosis in childhood.

[1] *Kelynack's Tuberculosis in Infancy and Childhood*, p. 294.
[2] *Trans. Roy. Soc. Med.*, vol. i, No. 6, p. 195.

CHAPTER XXX

ABDOMINAL TUBERCULOSIS IN CHILDREN

Abdominal tuberculosis in children may be considered under two heads, tabes mesenterica and tuberculous peritonitis.

Looked at purely from the clinician's standpoint, neither of these is a very common condition, but from the more reliable estimate of the pathologist abdominal tuberculosis would seem to be one of the commonest of all tuberculous lesions in children.

My own statistics show that 88·3 per cent. of tuberculous children have tuberculous lesions in the abdomen. Abdominal tuberculosis, therefore, is exceedingly common in the post mortem room, but not so common in the ward ; in other words, tubercle is often present in the abdomen without producing any characteristic symptoms during life. This latent abdominal tuberculosis is only too familiar to the pathologist. The body of a child who has died, it may be with symptoms pointing only to pulmonary tuberculosis, shows numerous tubercles in the spleen, a few in the liver, and probably one or two in the kidney, the mesenteric glands show caseous foci, and there may be one or two tuberculous ulcers in the intestine. In spite, however, of all these lesions in the abdomen, there has been nothing during life to suggest abdominal tuberculosis.

Now such a condition is of interest, it is true, chiefly to the pathologist, but it has also a clinical interest, for it emphasizes a very important point in the tuberculosis of childhood, namely, the tendency to rapid generalization. Whatever may have been the clinical aspect of tuberculosis in a child, whether death has been due to tuberculous meningitis or to pulmonary tuberculosis, or to whatever form of the disease it be due, tubercle is usually found scattered more or less widely over the body, and there are likely to be some tuberculous lesions in the abdomen. Realizing this tendency to rapid generalization, one can realize also the vital importance of the early detection of tubercle in children, for if this disease is overlooked in the early stage the only chance of saving the child may be lost ; it is certain, both from clinical experience and post mortem observation, that if only treatment is begun whilst the disease is still in its early

stage, tuberculosis in many of its forms may be arrested even in very young children.

The frequency of abdominal tuberculosis, therefore, although it is a matter chiefly of pathological observation, has a practical significance as a manifestation of the tendency to rapid and wide dissemination of tubercle in a child.

Tabes Mesenterica

The term tabes mesenterica is not altogether satisfactory; it has come to be used in a slovenly way, sometimes to indicate almost any form of abdominal tuberculosis, and sometimes—worse still—to indicate no tuberculosis at all, but simply the wasting of an ill-fed infant.

Again, it is too comprehensive a term in one sense, and too narrow in another. If by tabes mesenterica we mean any case in which there is tuberculosis of the mesenteric glands, we should have to include under this head nearly 60 per cent. of the cases of tuberculosis of any sort or kind, although in many of them the lesion of the mesenteric glands is of merely secondary importance, and the clinical aspect may have been that of pulmonary tuberculosis or tuberculous meningitis.

On the other hand, the term is too narrow if we restrict it to the cases in which there is great enlargement of the mesenteric glands, for in this way we create an entirely artificial distinction between the case in which the glands happen to be the size of a walnut and the case in which, although extensively caseous, they are only slightly enlarged; and the enlargement, be it observed, is not more likely to be 'primary' in the one case than in the other, so that we cannot base any distinction of 'primary' and 'secondary' affection of the mesenteric glands on their degree of enlargement.

It is, perhaps, most satisfactory to speak only of 'tuberculosis of the mesenteric glands'; but it is convenient to use the clinical term 'tabes mesenterica' to indicate those cases in which the tuberculous enlargement of the mesenteric glands can be detected clinically, and is associated with wasting, without evidence of tuberculous peritonitis, and with little or no clinical evidence of tubercle elsewhere. In this sense I shall use the term tabes mesenterica; and I may point out at once that this condition is by no means common. Tuberculosis of the mesenteric glands is common enough—59 per cent. of tuberculous children show it—but cases in which this is a prominent feature clinically are quite uncommon.

Now what is the history of such a case ? The child is brought to the medical man because it has been wasting for some weeks or months, and has had colicky pains in the abdomen. The pain is rarely severe, it is usually not enough to make the child cry. It is colicky in character, and recurs in an erratic way from time to time. The wasting is sometimes considerable, and in many cases the bowels have been irregular ; diarrhœa has been followed by constipation, and this in turn by diarrhœa again, and so on. The appetite also varies from day to day, but is more often poor than excessive. If the temperature has been taken frequently, it is found to be irregular (see Chart, p. 271) ; but, as a rule, in the early stages the temperature has not been taken, for the onset is insidious, and the child hardly looks ill enough to be suffering from anything very serious.

The occurrence in a child of insidious wasting with colicky pain in the abdomen, especially if the symptoms have lasted several weeks, should always arouse a suspicion of tabes mesenterica. But this is not sufficient to establish a diagnosis ; the only reliable guide in the diagnosis of tabes mesenterica is actually to feel the enlarged mesenteric glands ; and this is not always easy.

The place to feel for them is just to one side of the vertebral column, especially to the left of the mid-line at or just below the level of the umbilicus ; they are also to be felt not uncommonly in the right iliac fossa. Many children will begin to cry at once if made to lie down on a couch, or even on their mother's lap, and for this reason I often find it best to palpate the abdomen with one hand as the child sits on my knee with the trunk bent slightly forwards.

One would think that when the glands were enlarged to the size of a walnut, or even larger, they would be easily felt, but they are not, partly, perhaps, because the child often throws its abdominal muscles into a state of rigidity directly one tries to palpate, and partly because the glands are movable to some extent, and slip away to one side of the vertebral column and rest on the soft tissues of the lumbar region, where they are not easy to define. Be this as it may, in many cases where post mortem examination shows them to be considerably enlarged, careful palpation has failed to detect them during life.

It is not always easy by a single examination to distinguish between enlarged glands and solid fæces, but in some cases of tabes mesenterica there is definite tenderness over the enlarged glands, which may help in the diagnosis, and fæces can some-

times be indented distinctly by the finger; but often the disappearance of the masses after an aperient or an enema is the only reliable distinction.

A much greater difficulty in diagnosis is caused by some cases of appendicitis in which the thickened appendix, or, as sometimes happens, its distended terminal portion, feels so like a swollen gland that the distinction may be impossible unless there is a history of recurrent acute symptoms indicating appendicitis. In some cases where exploratory operation has shown appendicitis there have been no severe symptoms at any time, and even in retrospect it is difficult to see how the diagnosis from tuberculous glands could have been made.

Rectal examination may be of value in such cases, for the diffuse inflammatory thickening of appendicitis is often appreciable by rectal palpation; but for the detection of mesenteric glands I have not found rectal examination of much value, the glands are usually situated beyond the reach of the finger.

To sum up, the symptoms of tabes mesenterica are: wasting with colicky pain, irregularity of the bowels and appetite, and irregular temperature, whilst on palpation of the abdomen enlarged glands can be felt, sometimes forming a nodular mass, and often slightly tender on pressure.

There is, however, a further important feature, which is not apparent clinically, but which, nevertheless, has a bearing upon the treatment and prognosis of this condition, and may therefore be mentioned here, namely, ulceration of the intestine.

Probably even in the mildest cases and in the earliest stage of tabes mesenterica, there is some tuberculous ulceration of the bowel in nearly all cases. My own figures, taken from examinations made whilst I was pathologist at the Children's Hospital, showed that out of 132 cases with tuberculous enlargement of the mesenteric glands, 107, that is, over 80 per cent., showed ulceration of the bowel.

Ulceration of the intestine is not, of course, essential to the production of enlarged caseous glands in the mesentery. It has been shown experimentally that when small doses of tuberculous material are given to animals in their food, the mesenteric glands may become caseous without ulceration of the bowel; but judging from post mortem evidence it seems probable that the majority of children who come under observation with tabes mesenterica have some ulceration in the intestine, and this makes the condition graver than it would be if we had only to deal with caseation of the mesenteric glands. I have seen cases, however,

in which there was complete scarring of the intestinal ulcers which had been associated with caseous mesenteric glands, and it is not very rare to find calcified mesenteric glands—evidence of arrested caseation—in children.

The prognosis, therefore, of tabes mesenterica, is by no means hopeless. I have seen many of these cases improve so much that it seemed probable the disease was arrested, an event which is most likely where treatment is begun early, before there are signs of tubercle in the lung, and before there is any extreme wasting. It is to be remembered, however, that even if tabes mesenterica seems to have recovered completely, there are still possibilities of trouble at a later period. A mass of caseating glands may easily be the starting-point of a localized inflammation of the peritoneum, and without any more general inflammation occurring, adhesions may form between the glands and the neighbouring structures. These local adhesions are apt to give rise to trouble long after the tabes mesenterica seems to have quite recovered.

A child, aged five years, was brought to me at King's College Hospital for vomiting. He was fairly well nourished, and walked up to the hospital with his mother. He had complained of some pain in the abdomen and had been sick two or three times in the last forty-eight hours. I examined the abdomen carefully, but could feel nothing abnormal. There was, however, a look of illness about the boy's face for which I was unable to account, so I sent the boy into the ward for observation. He continued vomiting, and the abdomen was opened by my colleague, Mr. Burghard, who found a narrow fibrous adhesion passing from a dry caseous mesenteric gland to the ascending colon; under this a loop of ileum had slipped and become strangulated.

In a similar case in the Children's Hospital, the strangulating band passed from the gland to the wall of the abdomen. In the post mortem records of that hospital there are other cases in which this accident occurred after tabes mesenterica.

Treatment. As to the treatment of tabes mesenterica, by far the most important part is the hygienic treatment, particularly by climate. A prolonged stay at the seaside will do far more for this disease than any drug, and these patients do better at the seaside than in the country inland (see p. 420).

The diet to be aimed at is such as shall yield the maximum of nourishment with the minimum of residue, for, as I have already mentioned, the intestine is ulcerated in most of these cases. The child should be fed as far as possible with eggs, custard, well-cooked milk-puddings, and gravy. The eggs are particularly valuable, for they contain a large percentage of fat in the yolk. Milk and egg are to be considered chief items in the diet, and in choosing other foods, those should be selected

which will leave little residue, and are not likely to give rise to fermentation in the intestine. These children are not to be starved, they require rather 'good feeding, and plenty of it'.

There is one objection which might be raised both to milk and eggs, an objection which has also been raised to the use of cod-liver oil in this condition, and that is, the blocking of the lacteals in some of the cases by caseous material. Fat passes *viâ* the lacteals from the intestine into the lymph stream, and if this passage is blocked it may be useless to give such food.

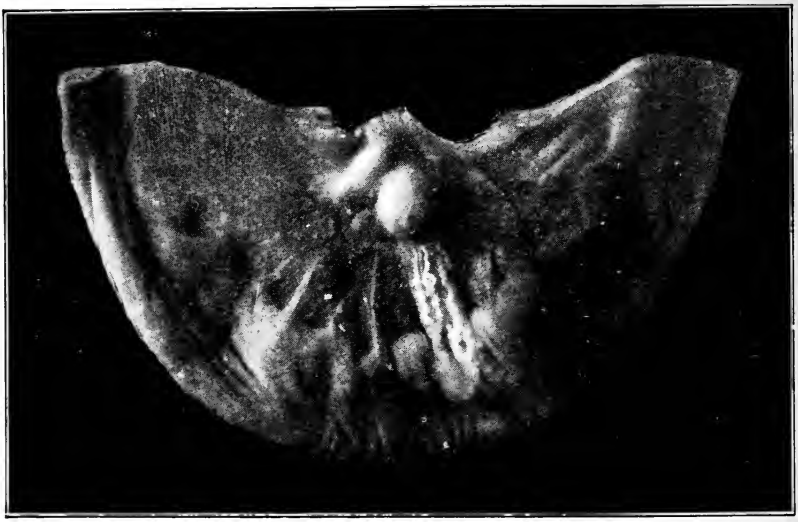

FIG. 30. Tuberculous mesenteric glands. Photograph shows the lymphatics, which pass from a tuberculous patch in the intestinal mucosa, obstructed and distended. Specimen from a child aged $11\frac{3}{4}$ years.

The photograph shown here will illustrate this point; it is a short piece of small intestine attached to its mesentery, in which is a caseous tuberculous gland; tuberculous ulceration of the bowel has occurred, and passing up from the outer surface of the bowel over the ulcer to the mesenteric gland are seen dilated lacteals filled with whitish caseous material, perhaps also with some fat.

But even in such a case probably the majority of the lacteals are free from obstruction, so that the objection to fats is perhaps more theoretical than practical; at any rate I think it will be

found that such a diet as I have mentioned is suitable for these children.

As to drugs, my own belief is that iodide of iron is particularly valuable in these cases. I should be sorry to have to explain its good effect, but certainly in some cases after its use the colicky pain ceases, and there seems to be general improvement. If there is no diarrhœa it is well to combine the iodide with a malt extract, or with a mixture of cod-liver oil and malt; there are several such combinations of iron iodide on the market. Another drug which may be useful is creosote, which is perhaps specially indicated in such a condition as this, inasmuch as not only is it an intestinal antiseptic, but it seems to have some specially beneficial influence on tuberculous lesions.

For the diarrhœa I have used castor oil in small doses (℥v) three times a day; it seems to have a particularly soothing effect on the mucous membrane of the lower part of the intestine, and creosote (℥¼ to ℥j) may usefully be combined with it and opium if necessary.

Sometimes an astringent seems more effectual, a mixture of Tinct. Opii ℥i, Tinct. Catechu ℥v, Spirit. Choloroformi ℥i½, Mist. Cretæ ad ℥j may be given to a child of two years three or four times a day; the Tincture of Coto in doses of 5–10 minims, or Tannalbin gr. x given as a powder, may be useful.

Lastly I must point out that surgery has a place in the treatment of tabes mesenterica. There are cases like those I have mentioned above in which obstructive symptoms necessitate operation, but there are others in which laparotomy may be advisable though it is not imperative. Such are those in which with the palpable presence of enlarged glands in the abdomen there is constantly recurring colicky pain. I have seen excellent results from laparotomy and removal of the affected glands in these cases; it may not be practicable to remove all the tuberculous glands, but it may be easily possible to remove those which show most enlargement and caseation, and to divide strands of adhesion which may be accountable for the pain; in a boy, aged 8½ years, who was under my care for palpable enlargement of mesenteric glands, my colleague Mr. Carless found a firm fibrous thread like a cord of fine catgut, 9 inches long, passing from one coil of intestine to another. There was a softened caseous mesenteric gland in this case without any general peritonitis and Mr. Carless was able to remove the gland as well as the strand of adhesion which might at any time have caused acute strangulation.

The operative treatment of tabes mesenterica is on an entirely different footing from that of general tuberculous peritonitis; there is no doubt of the removal of a definite focus of infection in the former, whereas in the latter the benefit of opening the abdomen is, except under certain rare conditions, quite problematical.

Tuberculous Peritonitis

As I have already said, a localized peritonitis sometimes starts from caseous mesenteric glands, and in some cases it seems likely that a generalized tuberculous peritonitis may have a similar origin. In the majority of cases, however, tubercular peritonitis probably begins independently of any tabes mesenterica; indeed the mesenteric glands are found to be very little affected in some of the cases of this disease which come to the post mortem room.

Tuberculous peritonitis may be divided for practical purposes into two varieties, the plastic and the ascitic; and this division is of considerable practical importance, as will be seen when we come to consider treatment.

The plastic variety is by far the commoner; in fact, speaking roughly, I should say the proportion of plastic to ascitic is probably about ten to one.

As to the frequency of this particular form of tuberculosis the following figures will give some idea. Out of 266 children who died with tuberculosis under twelve years of age, forty-five died with tuberculous peritonitis; so that 16·8 per cent., of the fatal cases of tuberculosis were cases of tuberculous peritonitis. Limiting my observations to children under two years of age, that is, to infants, I found twelve cases of tuberculous peritonitis in 100 tuberculous infants.

This is an interesting point in view of the teaching in former days that tuberculous peritonitis is practically unknown in infancy. All the cases which I have included here were such as are ordinarily recognized as general tuberculous peritonitis, and most of them, if not all, had been recognized as such during life. This occurrence of tuberculous peritonitis in infancy is even more strikingly shown by the accompanying chart (Fig. 31), which shows the age-incidence in each year of childhood.

The age-incidence of tuberculous peritonitis as shown by this chart—which includes only cases which were verified by autopsy—corresponds closely with that of tuberculosis in general; indeed, the chart might serve almost equally well to show the

age-incidence of tuberculosis in childhood. It will be seen that tuberculous peritonitis with a fatal result is most frequent in the second year of life, so that so far from tuberculous peritonitis being almost unknown in infancy it is actually commoner then—at any rate as a fatal disease—than at any other period of childhood.

Trusting to one's impressions, I should have said that tuberculous peritonitis was more frequent in boys than in girls, but my own statistics show that this is not so : in 100 cases there were 48 boys and 52 girls, and in further cases the proportion came out very similar. Rilliet and Barthez, however, met with

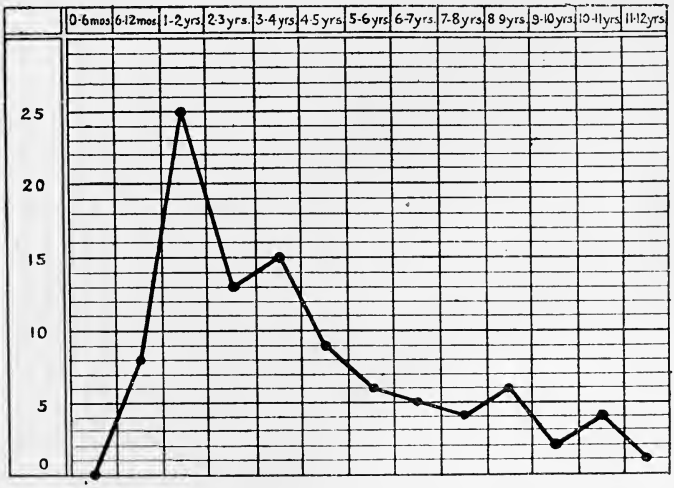

Fig. 31. Age-incidence of tuberculous peritonitis.

this disease in 53 boys and 33 girls. Probably the incidence is about equal in the two sexes.

The symptoms of tuberculous peritonitis in its earliest stage are very much the same as those of tabes mesenterica ; but later characteristic signs appear which can hardly be mistaken.

The abdomen has a tumid appearance, with a 'podgy', doughy feeling on palpation, as if it were packed full of some softish material. In many cases there is also a transverse band-like tumour lying across the epigastrium ; this I have several times seen mistaken for the lower edge of the liver, but with the exercise of a little care this mistake can be avoided, for the tumour has not only a lower edge, but also a definite upper margin—the

upper edge of the thickened and caseous omentum which forms the tumour. This band-like and often nodular mass lies across the epigastrium, generally just above the umbilicus, and slopes upwards towards the spleen. In some cases the edge of the liver can be defined, and is then found to be quite separate from the tumour. When the abdomen is opened in these cases the caseous omentum looks very like a large pancreas lying superficially just below the edge of the liver.

The appearance of the child when the disease is advanced is very characteristic: the emaciated limbs and the shrunken covering of the chest-wall, through which the ribs stand out distinct, contrast strangely with the fullness and tumidity of the abdomen.

At this stage there is another sign which is very characteristic, namely, an unfolding of the umbilicus, which looks as if it were pushed outwards from within; and in addition to the unfolding there is often induration and redness of the skin extending outwards about an inch all round the umbilicus. This reddening and induration about the umbilicus is sometimes of value in diagnosis where the general doughy resistance is not very marked. For instance, in a case in which the enlargement of the abdomen had been attributed to rickets some redness and induration about the umbilicus was the first evidence of tuberculous peritonitis.

This 'pointing' at the umbilicus is not very rare, and is one of the complications which must always be watched for in the course of tuberculous peritonitis. An actual discharge of broken-down caseous material may occur, and is not necessarily a matter for alarm; indeed, I have several times known cases to do much better after this event than before it. A case which impressed this point upon me was the following: the child was extremely ill with advanced tuberculous peritonitis; the umbilicus was reddened and shiny, with induration all round it, and it was obvious that the skin would give way very soon. I was inclined at that time to regard the prognosis as extremely bad, and when the parents insisted on taking the child away from the hospital they were strongly dissuaded from doing so on the ground that he might die on the way home. They took the child away against advice; a day or two later the skin over the umbilicus gave way, and there was a free discharge of caseous débris; when I heard of the child, about six weeks later, he was running about at home, apparently making good progress towards recovery.

A much more serious accident, and one which is usually speedily fatal, is perforation of the bowel before there is much adhesion.

In such a case the symptoms may suddenly change from those of tuberculous peritonitis to those of acute septic peritonitis.

When there is already much adhesion, perforation is not so serious an occurrence; for then a localized abscess forms, which may track amongst the coils and eventually point at the umbilicus or elsewhere, and in spite of a fæcal discharge from the abscess the child may seem little the worse. Perforation does not always take place from the inside of the bowel by rupture of the peritoneum over a tuberculous ulcer. In many cases a deposit of caseous material between two coils breaks down and discharges into the neighbouring bowel, so that the perforation occurs from without.

Ulceration of the bowel is present in many—about 70 per cent.—of the cases. I found it in fifty-four out of seventy-seven cases where it was possible to examine the bowel sufficiently thoroughly.

An unusual complication which I have seen in the early stage of tuberculous peritonitis is acute distension of the abdomen, a misleading symptom, as it suggests rather some acute septic peritonitis than a tuberculous condition; indeed, the diagnosis in such a case is hardly possible until the gradual subsidence of the distension and the increasing doughy resistance of the abdomen make the tuberculous nature of the peritonitis clear.

In the ascitic variety the symptoms are very different; here the abdomen is distended with fluid as in other forms of ascites, but it is often quite impossible at first to distinguish ascites due to tubercle from that due to any other cause. If one can have the abdomen tapped, and a bacteriological investigation made, it may be possible to determine this point, but in most cases it is necessary to wait some time, perhaps several weeks, before a positive diagnosis is possible.

Under these circumstances it is necessary to examine the child very carefully for evidence of tubercle elsewhere. In the case of a boy about five years old whose abdomen was distended with ascites of doubtful nature I had almost come to the conclusion that there was nothing to throw any light on the ascites, when it occurred to me to examine the testicles; I found the epididymis on one side considerably enlarged and hard, making it practically certain that the ascites was tuberculous.

These children rarely die with ascites. As a rule the fluid gradually disappears, and, as adhesions form, the condition becomes that of the ordinary plastic form. Upon this point I would lay some stress, for I think that in estimating the value

of operative treatment for tuberculous peritonitis some surgeons have attached undue importance to the disappearance of ascites after operation. It should be recognized that under simple medical treatment tuberculous ascites disappears spontaneously in the large majority of cases, and moreover that the mere disappearance of fluid, whether by incision or by tapping or by less active treatment, does not show that the peritonitis is cured, it may still run the course of a plastic tuberculous peritonitis, and it would be premature to boast of a cure until several months have passed, and until we can be quite sure that we have not simply converted a case of ascitic tuberculous peritonitis into one of the plastic variety.

Prognosis. The prognosis of tuberculous ascites is good so far as the immediate present is concerned; but if the ascites is gradually replaced by general matting and adhesions, as it is in many cases, the prognosis does not differ from that of cases in which the peritonitis is plastic or adhesive from the beginning.

It is difficult to determine the proportion of recoveries from tuberculous peritonitis amongst hospital patients, for the reason that so many pass out of observation after a few weeks or months, and their subsequent course is unknown; but certainly many cases leave the hospital greatly improved, and some apparently cured.

I say 'apparently cured', for I would insist very strongly on the need for caution in talking about 'cure' or 'recovery' in such cases.

A boy, aged about six years, was brought to me at King's College Hospital when he was four years old for wasting and pain in the abdomen. There was some resistance, and impairment of note on percussion over the lower part of the abdomen, and it seemed quite certain that he had tuberculous peritonitis; the diagnosis was confirmed by the scar of an old tuberculous dactylitis on the right hand.

Under medical treatment the resistance gradually disappeared, and the abdomen became as supple as a normal abdomen.

But to call such a case cured would have been misleading. It is true that during the eighteen months he was under observation after his apparent recovery, there was no return of the abdominal symptoms, unless some occasional colicky pains of which he complained towards the end of this time indicated some fresh abdominal tubercle, but more probably they were due, as they often are in such cases, to the old adhesions left by the former peritonitis His tuberculous peritonitis may have

been cured, but his tuberculosis was not cured. Eighteen months after his recovery from the peritonitis the supra-condylar gland in the right arm caseated and broke down.

Unless a case has been kept under observation for at least a year or eighteen months after the supposed recovery, it is worthless as evidence of a cure; some at least of the so-called 'recoveries', both after surgical and medical treatment, end fatally within two or three years with other manifestations of tuberculosis or a recrudescence of the abdominal disease.

Unfortunately, in many cases there is not even a temporary improvement. In a series of fatal cases at the Children's Hospital, Great Ormond Street, I found that the average duration was about five months; the shortest was four weeks.

The outlook is, I think, less hopeful in the first two years of life than in later childhood, but I have known recovery which was apparently complete to occur even in infants: one was about nine months old when the symptoms appeared with the typical hard infiltration of the omentum; six months later the abdominal signs had disappeared and the child was seemingly quite well; another at the age of eight months had fluid in the abdomen with wasting and fever, he had made an apparently perfect recovery when seen six months later. Another at the age of eighteen months had the usual signs of plastic peritonitis, and three months later a discharge of caseous material from the umbilicus, after which she steadily improved, the hard masses of infiltration were much less and the child seemed to be doing well when last seen nearly five months after the discharge at the umbilicus began.

Treatment. The question of the frequency of recovery with various methods of treatment is of great practical importance, for in recent years we have heard much of the surgical treatment of tuberculous peritonitis, and some have even gone so far as to recommend laparotomy as a routine treatment for this disease.

I imagine that no one with a well-balanced mind is likely to maintain that operative treatment is advisable where it is unnecessary; experience has abundantly proved that some cases of tuberculous peritonitis will get well without operation, and therefore that in these at least operation would have been a mistake. But what proportion of cases will recover without operation? The question is a difficult one to answer, for it is difficult to trace a sufficiently large number of cases for a sufficiently long period to afford reliable evidence.

Dr. Guthrie and Dr. Sutherland have published statistics [1]

[1] *Trans. Soc. Study Dis. Children*, vol. iii, p. 118.

showing that of 41 cases treated at the Paddington Green Children's Hospital, 14 underwent laparotomy and 7 of these died, whilst of the remaining 27 treated medically only 4 were fatal. Dr. George Carpenter recorded 10 cases as recovered and 16 as improved out of 54 cases treated medically (19 died and the result was not known in 9 cases).

Without attempting to settle this question by statistics I will state certain facts which seem to me to have a bearing upon it. As I have already pointed out, the stage of ascites, when it occurs at all in tuberculous peritonitis, is an early stage; the fluid usually disappears spontaneously and the peritonitis may then subside or pass into the plastic variety. In this early stage of ascites tuberculous peritonitis is eminently amenable to simple hygienic treatment; so far as the mere presence of ascites goes there is in the majority of cases no occasion whatever for laparotomy or even for tapping, it is only in rare cases that the mechanical distension becomes such as to require some operative relief.

If it could be shown that laparotomy had any specific influence by so altering conditions in the abdominal cavity that the power of resistance to tubercle was increased, the treatment of these ascitic cases by incision would then stand on a different footing; but I know of no clinical evidence which establishes this.

There is no doubt whatever that tubercular ascites and miliary tubercle in the peritoneum have many times recovered after laparotomy, but there is equally no doubt that recovery occurs in similar cases without any operation where the child is kept lying down, carefully fed, sent to the country or seaside, and kept much in the open air; and it must be remembered that when operation is done the child is almost always placed under just these conditions as an adjunct to the surgical procedure; and therefore one must be cautious in concluding that the recovery is due to the operation.

Next I would point out that the peritoneum has a marvellous power of resistance to tubercle; and that even where plastic tuberculous peritonitis is present with hard bands of infiltration, and forms masses which to any one unfamiliar with this disease might seem a hopeless condition, recovery without any operative interference is so complete in some cases that it would be difficult for any one seeing the child a few years later to believe that there had ever been peritonitis of any sort.

One child who came under observation at 7½ years old, when she had large masses of hard infiltration in the abdomen and was

much wasted, had two years later a discharge of broken-down caseous material from the umbilicus; she then gradually recovered, and at the age of 11½ years was apparently in perfect health, fat and jolly, and the abdomen showed no infiltration on palpation, and might easily have passed for normal except for the scar of the former umbilical opening.

I have seen other similar cases in which this plastic form of peritonitis subsided spontaneously with or without discharge from the umbilicus, and I mention this because the possibility of spontaneous recovery from so advanced a stage of tuberculous peritonitis emphasizes the need for caution in attributing good results to laparotomy. But if the good results of incision in the plastic variety of tuberculous peritonitis are doubtful, the bad results are not open to doubt: the best of surgeons cannot be sure of avoiding damage to the intestine when there is such general matting as is usual in this form. I have more than once seen a troublesome fæcal fistula as the only result of the operation, and this fistula from operation is likely to be larger and to heal less readily than the fistula which results from Nature's method of evacuation through the umbilicus—the natural fistula is sometimes fæcal, sometimes not, but in either case is usually very small and heals up after a few weeks.

In my opinion laparotomy is rarely if ever advisable in the plastic form of tuberculous peritonitis except for the relief of particular symptoms.

There are cases in which as a result of the adhesions some part of the bowel becomes strangulated or compressed so that symptoms of more or less acute obstruction occur, with severe pain and vomiting; under these circumstances obviously it is only right to attempt relief of the distress by removing the band or other cause of obstruction: the operation is a hazardous one, for it usually involves tearing asunder matted coils of bowel which are probably thinned by ulceration; but it is an operation of necessity.

Again, there are cases in which, with caseous material between the coils of bowel, softening or perforation of bowel occurs, but the resulting abscess fails to point at the umbilicus and a collection of pus occurs which must be evacuated by incision.

In the medical treatment of this condition, climate stands first and foremost; the child should be sent away to the seaside or the country as quickly as can be managed and taken out in some sort of carriage in the open air daily and almost all day if possible. A bracing atmosphere suits these children best, at any rate in the

summer, but cold is to be avoided ; and a dry gravel soil is to be preferred to sand or clay. I need not repeat here what I have said elsewhere on the climatic and general treatment of tubercle in children. Good feeding is essential, and must be upon the same lines as in tabes mesenterica ; in both conditions the ulceration of the bowel is to be remembered.

The importance of recumbency for these cases of abdominal tubercle in children is worthy of emphasis. It is not sufficient to take the child to the seaside and to feed it well, the child should be kept lying down ; a long perambulator in which the child can lie should be provided, and in this the child should be kept out in the open air the greater part of the day.

The value of recumbency has rested hitherto solely on the test of experience, but recent studies of the opsonic index have gone far to supply more exact grounds for insisting upon rest in such cases. Rest in bed combined with good feeding is probably responsible for the improvement in many cases admitted to hospital ; it is remarkable how much children with tuberculous peritonitis will often improve, even in our atmosphere in London, with these measures alone, but the best results are to be seen where similar treatment can be carried out in the open air in the country or at the seaside.

Turning now to the value of drugs for tuberculous peritonitis, I would point out that although in my opinion drugs have a very real value, they must be regarded only as an adjunct to the far more important remedies, sea-air and recumbency. Iodoform has, I think, a distinctly beneficial effect, perhaps chiefly in the cases with ascites. I have seen fluid disappear very quickly during the administration of iodoform by mouth, and I have very rarely had any difficulty in getting children to take it in the form of an emulsion with cod-liver oil. Mr. Fairweather, chief dispenser at King's College Hospital, devised the following formula, which I have found generally useful : Iodoform gr. $\frac{1}{2}$, Ol. Morrhuae ℥xx, Ol. Caryophylli ℥$\frac{1}{8}$, Tinct. Lavandulæ Co. ℥v, Pulv. Acaciæ gr. v, Aq. ad ʒi ter die.

If the child objects greatly to its taste it can be used by inunction : years ago I made some observations at the suggestion of Dr. Burney Yeo upon the absorbability of iodoform through the skin, and I found that it was absorbed with rapidity. For instance, half a drachm of the unguentum iodoformi gently rubbed over the abdomen at 9 a.m. was easily detectable by the presence of iodine in the urine at 11 a.m., and the drug continued to be excreted in the urine for at least fourteen hours

after its application. It is evident that it may have some influence on the tuberculous process in the peritoneum. Dr. Burney Yeo has recommended the use of equal parts of iodoform ointment and cod-liver oil for inunction twice daily. It is necessary to be watchful in the use of iodoform inunction; a boy of five years whom I treated thus suffered with vomiting and irregularity of the pulse after a second inunction of half a drachm of the iodoform ointment.

All treatment by inunction has the great drawback that it renders dosage vague and uncertain, and therefore I prefer when possible to treat by oral administration.

Creosote is another drug which I think undoubtedly has a therapeutic value in this, as in some other forms of tuberculosis: not only has it a very decided effect in correcting fermentative processes in the bowel—and any such condition is to be combated in tuberculous peritonitis in view of the possibly ulcerated condition of the intestinal mucosa—but creosote seems also to be in some way antagonistic to the tubercle bacillus.

Creosote can be given conveniently in a cod-liver oil emulsion; there are several combinations of this kind in the market, some of which disguise the unpleasantness of creosote as far as is possible. If there is diarrhœa the creosote is best given in a castor oil mixture with opium if necessary, thus: Creosote ♏½, Ol. Ricini ♏v, Spirit. Chloroformi ♏i, Mucilag. Acaciæ ♏xv, Aq. Anethi ad ℥i, to be given thrice daily immediately after food.

In giving creosote to children it must be remembered that even with the small doses which are appropriate for them, such as ½–1 minim, its administration continued for many weeks is very apt to cause loss of appetite.

I have also used malt extract with iron iodide, I think with advantage. Urea has been thought to exert some special influence in restraining tuberculous processes, and having seen rapid recovery follow its use in a case of tuberculous dactylitis, I have tried it also in cases of tuberculous peritonitis. In one of these apparently complete recovery followed, but other drugs had been used before, and may have had a share in promoting the recovery: I have used it in several advanced cases without any perceptible influence. The doses I have used have been 3–5 grains three times a day, given with some syrup of tolu and water.

Mercurial inunction is a very old method of treatment. I have used it in many cases but am not very certain as to benefit

from it: half a drachm of the unguentum hydrargyri, amounting in more convenient terms to a piece the size of a large pea, may be smeared over the abdomen every night daily and covered with a flannel binder; the ointment should be thoroughly cleaned off the skin each morning.

Lastly—and I put it last because it is yet in the stage of probation—the use of tuberculin must be considered. Others perhaps who have had larger experience of it can speak with more confidence than I of its beneficial effects in tuberculous peritonitis. I have used it, and seen it used, subcutaneously, by mouth, and by rectum. In advanced cases it has not seemed to me to have any effect which could be appreciated by bedside observation; in an infant of eight months, with a small amount of fluid in the abdomen and some fever and wasting, the parents took the child at my suggestion to Dr. Latham for rectal injection of tuberculin; the symptoms and signs passed off entirely under this treatment. If further experience confirms the interesting observations of Dr. Latham on the value of tuberculin given by mouth or rectum, this mode of administration would be much preferable to the subcutaneous injection for children, who are so easily distressed by the slight pain of a needle-prick.

CHAPTER XXXI

ON TUBERCULOUS GLANDS IN THE NECK

I SHALL preface what I have to say on this subject by some general remarks on affections of the lymphatic glands in children.

In the diseases of childhood, the lymphatic glands play a more prominent part than in those of the adult. At any age their function as filters for the arrest of irritating substances, bacterial or otherwise, passing along the lymph-stream, must render them specially liable to morbid processes, but in childhood this liability seems to be at its height. As a result of this special sensitiveness, enlargement of glands is particularly frequent in childhood, and the enlargement is generally to be traced to absorption of infective material from the area which the gland drains. I mention this elementary fact because I think that in dealing with tuberculous glands there is a tendency to make much of constitutional predisposition, and to forget that, as in other infections of glands, the virus has usually entered through the area which the gland drains, and that our treatment must take this into account.

The lymphatic glands have a further function in the production of white corpuscles, and they may become enlarged as part of a widespread disturbance of blood formation, as in lymphatic leukæmia.

Apart from the causes of enlargement already mentioned, there are lymphadenoma and lymphosarcoma, of which at present we know only enough to surmise that they may be due to some unknown irritant or infection.

I have implied that enlargement of lymphatic glands necessarily indicates some morbid process, but I would venture to question whether unusual palpability is necessarily a sign of disease. Other lymphoid tissues, the thymus, the spleen, the Peyer's patches, and, I think, even the tonsils, and the adenoid tissue of the naso-pharynx, vary in amount in different children at the same age without any apparent disturbance of health; it seems therefore quite conceivable that the lymphatic glands may also vary in this respect, so that without any departure from

health, they may be more palpable in one child than in another. Clinical experience seems to show that this is so, and it is worthy of note, because unnecessary alarm is sometimes caused by the discovery that a child has many lymphatic glands felt more easily than is usual.

But having ventured this suggestion, I must again insist that palpable enlargement of glands should always make us search for irritation in the area which they drain. It is difficult to get the laity to realize this. We are all familiar with the mother who brings her child to hospital for enlarged glands in the occipital region, and goes away indignant and unbelieving when it is pointed out that the one thing needful is to rid the hair of pediculi capitis. The relation of the enlargement to its source is still more apt to be overlooked where, as very commonly happens, a child with chronic nasopharyngeal catarrh has palpable and sometimes even visible swelling of small lymphatic glands in the posterior triangle of the neck.

One might multiply instances to show the special sensitiveness of the glands in childhood. With any unhealthy condition of the tonsils, how much more readily the glands just behind the angle of the jaw become swollen in the child, than in the adult. Again, how sensitive an index of catarrh of the middle ear, in a young child, is the little gland which, when swollen, becomes just palpable over the mastoid bone. I have instanced glands in these parts, rather than elsewhere in the body, because I want to insist that there are many sources of irritation and infection for the cervical glands, and the one great reason why these glands are so often enlarged is their close relation to the nasopharyngeal mucous membrane, which, like mucous membranes in other parts of the body, is peculiarly liable to morbid processes, catarrhal or otherwise, and, like them, has an extensive lymphatic drainage. I would specially emphasize this relation, because it accounts for a very considerable majority of the enlargements of cervical glands, and I might add that a large proportion of children, at any rate in the damp climate and variable temperatures of this country, suffer from some catarrhal condition of the nasopharyngeal mucosa, and consequently have more or less enlargement of glands in the neck.

It is perhaps hardly realized how few children there are in whom no glands can be felt in the neck. With this point in view, I examined a series of 100 consecutive cases, seen in private practice, children in whom there was no suspicion of tubercle in the glands or elsewhere (nor had any of them nits in the hair).

No less than 83 per cent. showed palpable glands in the neck; in 69 per cent. the glands behind the angles of the jaw were more or less enlarged.

I have said enough to indicate that there are many other causes for enlargement of glands in the neck besides tubercle; nevertheless it remains true, that of all the superficial lymphatic glands in the body, the most frequently affected by tubercle are those in the neck; even in comparison with the deep glands, this special liability of the cervical is striking, for in post mortem observations they showed more or less caseation in 81 per cent. of tuberculous children, the same proportion as in the case of the mediastinal, whilst in the mesenteric glands the proportion was 59 per cent.

The frequency of tuberculosis in these three groups is no doubt due to the fact that these particular glands are, so to speak, the outposts of defence, and therefore most exposed to attack at parts where the tuberculous infection most commonly makes its entry, namely, the fauces, the lungs, and the intestine.

It is easy to understand on these grounds also why certain lymphatic glands are very rarely affected, as they drain areas comparatively seldom attacked by tubercle, e. g. the skin. Thus, the lymphatic glands of the groin, and of the axilla, as also those along the brachial artery and in the popliteal space, and in the back of the neck, very rarely become tuberculous. I mention this because one is not infrequently consulted about glands which are unusually palpable in the back of the neck, or in the groin; the parents have noticed them, and the question of tubercle is raised. Remembering the rarity of tubercle in these glands, one can almost always assure the parents that palpability is not due to any tuberculous enlargement. On the other hand, in very rare cases some tuberculous affection of the skin of the limbs, or scalp, has caused tuberculous affection of these glands, and I have known operation necessary for tuberculous enlargement of axillary glands where no source could be traced, and one supposed that the infection might have been carried from some distant part by the blood-stream, a method of infection which is probably very uncommon in the lymphatic glands, to which infection is usually conveyed by the lymphatics, not by the blood.

Related to this rarity of infection by the blood-stream is the rarity of general tuberculosis of the superficial lymphatic glands, a condition which has only occasionally been observed. It is common enough to find in any fatal case of tuberculosis in a

child the cervical, mediastinal, and mesenteric glands all more or less infected with tubercle, but the glandular disease in these cases is no doubt due partly to infection by the lymph-stream from the areas drained by these particular glands, and partly to direct extension from gland to gland; such a condition is quite distinct from the general infection of superficial glands which has in rare instances simulated Hodgkin's Disease. I have never met with such a condition myself.

As I have already mentioned, tubercle was found at autopsy in the glands of the neck in 81 per cent. of tuberculous children, but it must not be supposed that the infection of these glands was primary, or even an early event in these cases; on the contrary, in nearly all it was obvious from the limited and early character of the tubercular lesions, that they had followed upon extensive tuberculosis elsewhere. In many cases small foci of tubercle in the cervical glands were associated with advanced pulmonary tuberculosis, and it seemed probable that the disease in the cervical glands was the result, not the cause, of the lung infection. The child coughs up tubercular sputum from the lung into the nasopharynx, where the constant presence of tubercle bacilli leads, sooner or later, to infection of glands through the adenoid tissue of the pharynx or through the tonsil. In the majority of these cases the enlargement of the cervical glands had either been so slight that it had not attracted attention during life, or the evidence of advanced disease in other parts had been so pronounced that the enlarged glands in the neck were regarded as of secondary importance, if not as secondary in order of infection.

Very different is the order of events in the common cases where treatment is sought for enlargement of cervical glands; these, it is true, may be secondary to tuberculosis in some other part of the body, particularly the lungs, but clinical experience suggests that this is quite the exception. Extensive tuberculous disease in the lungs or elsewhere is commonly associated with infection of the glands of the neck, but the converse does not hold good. Tuberculous disease of the glands in the neck, however extensive it may be, is not necessarily nor indeed usually associated with tuberculosis elsewhere, so far as can be judged from clinical evidence, and experience shows that affection of these glands but seldom leads to tuberculous disease in other parts of the body. This is a point of considerable practical importance, because the first inquiry of parents, when they learn that their child has tuberculous glands in the neck, is

'Does this mean that the child has the disease in other parts?'
'Does it mean that he will develop consumption?'

Age at onset. In about 75 per cent. of the cases of tuberculous enlargement of glands in the neck the disease begins before the age of 7 years; it is uncommon under the age of 18 months, and seldom begins after the age of 10 years.

Etiology. The age incidence has an interest in relation to the source of infection. It has recently been shown by Dr. A. P. Mitchell [1] that tuberculosis of cervical glands in children is due to the bovine type of bacillus in the large majority of cases (65 out of 72), and investigations [2] of the milk supply have confirmed the view that in a very large proportion of the cases the infection has been conveyed by unboiled milk.

These observations explain the comparative infrequency of tuberculosis of cervical glands in infants, for although infancy is the period of milk-feeding, it is also the time when milk is most often given boiled or pasteurized. But these remarks will not apply to all countries; if milk infection plays so important a part, the frequency of tuberculosis of the cervical glands in infancy, as at other ages, will depend largely upon the prevailing usage of boiled or unboiled milk in the particular district from which the statistics are taken.

Dr. Mitchell's figures, taken in Edinburgh, show an unusually high percentage of cases amongst infants aged 1–2 years—22 per cent.; but this agrees with his observation that 84 per cent. of children under 2 years of age, in his series, had been fed since birth with unsterilized cow's milk, whereas my own observations are drawn from parts of England where for at least the first year of life cow's milk is usually given pasteurized or boiled.

It is hardly possible to exaggerate the importance of the milk factor in the etiology of tuberculous glands in the neck. Dr. Mitchell concluded that 'cow's milk containing bovine tubercle bacilli is clearly the cause of 90 per cent. of the cases of tuberculous cervical glands in infants and children residing in Edinburgh and district'. If only the danger of tuberculous milk were fully recognized, and the enormous gain in safety from boiling or pasteurizing were appreciated, we should see less of these troublesome cases of tuberculous glands in the neck.

There is a predisposing cause which, perhaps, also has some bearing on the age incidence. Any inflammatory enlargement

[1] *Brit. Med. Journ.*, Jan. 27, 1914.
[2] *Trans. Internat. Cong. Med.*, 1913, Sec. X, Pt. 1, p. 47.

of the lymphatic glands renders them more susceptible to tuberculous infection, hence the chronic enlargement which is so often associated with enlarged tonsils and adenoids favours the incidence of tubercle, and this is a factor which is seldom in evidence before the age of 2 years.

Symptoms. As a rule, the enlargement of the glands is slow and insidious. The child is paler than usual, and, without seeming ill, is lacking in tone and vivacity. The temperature is not necessarily raised at all; if it is taken regularly, there may be found some slight elevation, 99°–100°, at night, occasionally. The general nutrition is hardly affected. The glands at first are rather felt than seen, and the one to be first affected is most commonly that behind the angle of the jaw, which drains the tonsil; subsequently the disease extends downwards, from gland to gland, along the anterior edge of the sternomastoid, and sometimes beneath the upper part of the sternomastoid, or along its posterior edge. Soon, the swollen glands become visible, and there may be some periadenitis, causing matting together of adjoining glands. All this may be, and usually is, a process of many months, or even two or three years, but I would particularly draw attention to the occasional acute onset of tuberculous glandular enlargement; sometimes, in a child who has previously shown no tendency to tubercle, the glands behind the angle of the jaw suddenly enlarge, so that within two or three days a bulging swelling appears, which in the acuteness of its onset seems so unlike a tuberculous condition, that some septic infection from the throat is suspected. Nevertheless, subsequent events prove that it is tuberculous; occasionally it passes forthwith into the stage of softening, and tubercle bacilli are found in the caseous pus, or it subsides into a chronic enlargement which runs the ordinary course of tuberculous glands.

A point which is worthy of note in the behaviour of the tuberculous gland is its variation in size from time to time: for a few weeks, perhaps, it seems to be growing smaller, then it increases again for a time, and then perhaps diminishes again. Such changes are very characteristic of the disease and must carry some weight in diagnosis. I have been assured by some parents that the glands varied in size even during the day, and several have told me that the glands were often larger in the evening. No doubt these observations by lay persons may be fallacious, but I am not sure that they are altogether incorrect.

Lastly, I would emphasize the rapidity with which a tuber-

culous gland may undergo softening: sometimes, within a few days, a gland which has remained for months firm or hard, suddenly becomes larger, and, at the same time, is found to be soft and fluctuating—an important point to bear in mind when sending these children to the country, perhaps out of reach of proper surgical intervention.

Diagnosis. The differentiation of tuberculous from other enlargements of cervical glands is by no means easy. The size has undoubtedly some bearing upon diagnosis; tuberculous glands tend to enlarge beyond the lesser degrees; where there is no gland in the neck larger than an ordinary almond, a simple irritative enlargement is more likely than where the glands are as large as a walnut, but, as any one knows who has seen tuberculous glands dissected out of the neck, there are usually, in addition to the larger caseous glands, many quite small, some even not larger than a hemp-seed, with yellow points of tubercle in them. In regard to size, however, some distinction must be made between acute and chronic enlargement, for a simple septic adenitis will produce an acute swelling of a gland as large as any tuberculous condition, but a chronic enlargement of the same size would point to tubercle.

As I have already mentioned, frequent variations in the size of chronically enlarged glands are strongly in favour of tubercle.

The distribution of the enlargement may throw some light on the problem; a considerable unilateral enlargement suggests tubercle, but only because any simple septic inflammation of the fauces usually affects both sides of the throat, and therefore is likely to affect the glands on both sides.

A more reliable indication is afforded by erratic distribution of enlargement amongst the glands, as if the morbid process were much more advanced in one gland than in another; for instance, where in a chain of glands moderately and for the most part evenly enlarged, one is swollen altogether out of proportion to the rest, the probability is that it is tuberculous.

The consistency of the gland is also a help; hardness suggests tuberculosis; in its early stages, however, tuberculosis produces no more induration than a simple inflammatory enlargement; it is only when there is already firm caseous or calcareous change or extensive fibrosis that the gland loses its normal feeling of elasticity, and becomes so hard that one can feel fairly confident of its tuberculous nature.

The rate of enlargement is but a poor criterion. As a rule tuberculous enlargement is of slow and insidious onset, but, as

already mentioned a gland may swell up within forty-eight hours to such an extent that simple septic inflammation is regarded as almost certain, nevertheless the glands have proved to be tuberculous.

I have often thought that the shape of an enlarged gland has some bearing on diagnosis ; a tuberculous gland, especially when the process is chronic, and caseation has occurred, tends to become round rather than oval ; a hard round gland is most likely to be tuberculous.

It is evident from the uncertainty of any of these criteria, taken alone, and indeed of them all taken together, that there is often great difficulty in pronouncing upon the character of gland enlargement, particularly in distinguishing between a simple septic adenitis and a tuberculous enlargement. Lymphadenoma is, next to simple inflammatory enlargement, the affection of glands which is most likely to be confused with tubercle ; no doubt the distinction is easy enough in a pronounced case, the discrete character of the glands and the multiple enlargement, affecting not only the cervical but other superficial glands elsewhere, and perhaps also some of the deeper palpable glands, are points sufficient for diagnosis ; but in the early stage, when the lymphadenoma has affected perhaps only two or three glands on one side of the neck—and I have known it to remain thus for many weeks—decision may be impossible until either examination of a removed gland or the gradual generalization of the enlargement shows the nature of the disease. Lymphosarcoma is much rarer, but I have seen it in the glands of the neck at the age of three years, and have known the elasticity of the very vascular growth to simulate closely the fluctuation of a broken-down caseous gland.

There is yet another condition in which enlargement of glands may raise the question of tubercle, namely, acute lymphatic leukæmia. In its early stage, before the pallor becomes so striking as in itself to suggest a blood disease, there is sometimes quite a noticeable enlargement of the glands in the neck, as well as elsewhere, but the enlargement does not reach any great degree, the glands are rather palpable than visible, or, at most, are just visible ; they are markedly discrete, and the enlargement is general, so that many glands which are not commonly affected by tubercle, such as those in the back of the neck, the axilla, and groin, are affected. If the possibility of leukæmia is borne in mind, it is of course recognized easily by a blood count.

There is a tumour in the neck which one might think could hardly be confused with swollen glands, but as I have more than once known the mistake made, I mention it here, namely, sternomastoid hæmatoma, which occurs in the new-born as the result of injury during delivery. The tumour, situated usually about the junction of the upper and middle third of the muscle, is firm, or indeed quite hard; it is often the size of a small walnut, and so far might pass as a swollen gland, but careful palpation shows that it is in the substance of the muscle, and the history that it has been noticed usually when the infant was only a few weeks old would in itself make any glandular swelling improbable, and puts tuberculous enlargement almost out of court.

Prognosis. The general outlook is favourable; experience shows that only a small proportion of children who come under treatment for tuberculous glands in the neck have, or subsequently develop, tuberculosis elsewhere.

Granted, however, that infection of other parts of the body, directly or indirectly from tuberculous glands in the neck, is uncommon, one cannot ignore the fact that the tuberculosis in these glands, as in any other part, points to an unduly weak resistance to tubercle, and on this ground necessitates caution in prognosis.

As regards the glands themselves, there can be no doubt that the process may undergo arrest here, as elsewhere, and the only trace of its occurrence may be some fibrous induration of the gland, or perhaps a calcareous focus where a caseous spot has occurred; but my own experience has been that the larger the gland the less likely is such a result; more often, after persistent enlargement for several months, with slight variations in size from time to time, the glands eventually soften, and surgical measures become necessary.

Treatment. *Prophylactic.* Before discussing the treatment, I should like to lay some stress upon the prevention of tuberculous glands in the neck. As already mentioned, there is very strong evidence that in a large majority of the cases the disease is due to the use of tubercle-infected milk. In some places nearly 20 per cent. of samples of milk have shown the tubercle bacillus. There is no doubt that by boiling the milk this source of infection can be entirely avoided.

Clinical evidence seems to show that any inflammatory enlargement of a gland renders it more vulnerable to tubercle, and therefore any source of irritation which may cause enlargement

of glands is specially to be avoided in the child of tuberculous family, in whom on this account unhealthy tonsils, adenoids, or carious teeth call the more urgently for treatment.

Curative. The remedy which has most effect upon tuberculous glands in the neck is sea-air; a bracing climate usually does most good, such as the Kent coast, Herne Bay, Margate, Broadstairs, or Folkestone; or, on the east coast, such places as Whitby, Scarborough, Sheringham, or Felixstowe; or the north coast of Cornwall or Devon. But many such places are less suitable in the winter, and on this account, as well as for the reason that change of air in itself seems to do these cases good, permanent residence at any particular part may be less advisable than a change of place with the seasons: Hastings or Worthing, or even the milder seaside places, such as Westcliff, Torquay, or Falmouth, may be more suitable for the winter. Mountain resorts, for instance, in Switzerland, have seemed to me generally less satisfactory for these children, but if inland places are the only ones available, high ground with a bracing atmosphere is to be recommended.

Here I would add a caution: in sending a child away to the seaside or country for tuberculous glands, it is extremely important to emphasize the necessity for frequent medical inspection whilst away, for the glands, sometimes, very suddenly and rapidly soften and form an abscess, and in this way the opportunity for surgical intervention at the right moment may be lost.

Where there is extensive glandular involvement, complete recumbency for a few weeks is a very valuable adjunct to the climatic treatment. The child can be taken out in an invalid perambulator, so that there need be no interference with the open-air regimen which is essential for these cases.

Whatever treatment is adopted, these cases require generous feeding, but all the milk should be given boiled or pasteurized; it is illogical to be pouring tubercle bacilli into the child whilst we are fighting tuberculosis in the glands. Cream, it must be remembered, is nearly, if not quite, as dangerous as unboiled milk in conveying tuberculous infection, and as it is difficult to sterilize cream it is best avoided altogether. A useful substitute for it is ordinary sweetened condensed milk, which most children enjoy when it is given undiluted, with stewed fruit or otherwise.

Dripping instead of butter is taken well by many children, and is very suitable.

As to external applications, I am not satisfied that any good is obtained thereby. Any rubbing, however light, is to be strictly

forbidden. Some observers have considered that the application of counter-irritants such as iodine was positively harmful.

Recently the application of X-rays has been found of value in some cases, but this method of treatment should only be carried out by some one with special knowledge of its use.

Of drugs for internal use, perhaps the most useful is Iron Iodide, which is best given with malt. There are several preparations of it in the market, some containing cod-liver oil in addition to the malt. For children who dislike the ordinary malt preparations, a dried malt with Iron Iodide (Wander) can be used sprinkled on bread and butter. An occasional course of arsenic for two or three weeks may also be of value.

The late Dr. Eustace Smith thought highly of a mixture of Liq. Ferri Perchlor. ℥v, Liq. Hyd. Perchlor. ℥x, Glycerin ℥xx aq. ad ʒii, to be given in half a wineglassful of soda-water three times a day, after meals.

The use of tuberculin has to be considered. I have used it in many cases, but combined almost always with residence at the seaside, so that it was difficult to apportion the good effects, if any. As the sum of my experience, I can say that I have never known any direct harm to result from the use of tuberculin in these gland cases, and, on the other hand, I have not seen any sufficiently definite benefit to enable me to urge its use. I said direct harm because I have several times seen very unfortunate mischief arise indirectly from this method of treatment; parents and doctor alike, having been led to place confidence therein, have allowed surgical interference to be delayed until the glands had already broken down into an abscess, which made any complete removal impossible and inflicted upon the child many weeks or months of tedious treatment for the healing of the abscess.

What I have already said about sources of adenitis as a predisposing cause of tubercle is to be remembered also when the glands have already become tuberculous; an important part of treatment may be the removal of tonsils and adenoids, or of carious teeth, which by keeping up a chronic adenitis maintain a favourable soil for the tuberculous process.

This brings me to the surgical aspect of treatment, and here I must speak with the caution which becomes a physician, but none the less there are some points upon which I shall venture to be outspoken. With regard to the time and manner of surgical intervention, the following seems to me to be the correct attitude. If after several months' trial of sea-air, there is little

or no diminution in the size of the glands, or if after short or long stay at the seaside the glands are becoming larger, or more are becoming affected, operation should not be delayed.

Operative measures should never be postponed until the glands have already softened. The only operation which is to be advised is the complete and thorough removal of all the affected glands where practicable. This is a much more extensive and difficult operation than might be thought, for although only two or three enlarged glands may be felt, there are almost always many deeper ones also affected, which must be dissected out if the cure is to be permanent. If operation is postponed until the glands have already softened into pus and there is much periadenitis, complete removal may be impossible. When this has happened, the proper treatment is simple incision and evacuation of the pus by drainage, *not* by scraping. Any scraping of tuberculous glands is a dangerous proceeding, for it entails a risk of rubbing the tuberculous material into adjoining lymphatics or blood-vessels, and so disseminating the infection: a fatal result from tuberculous meningitis sometimes follows within a few weeks of this scraping operation.

Lastly, there is a point upon which advice is often asked. Is the child with tuberculous glands in the neck dangerous to other children ? In former years I took a light view of the risk of infection from such cases, but I have since seen several instances in which there was strong reason to believe that tuberculosis was conveyed by a patient with tuberculous glands in the neck. The risk, no doubt, is greater where there is an open wound or a discharging abscess, but even where there is no external opening it is to be remembered that tuberculous glands are frequently associated with unhealthy tonsils and adenoids, which in a certain proportion of the cases have been shown to contain tubercle bacilli. It is therefore intelligible that, by sneezing or coughing or otherwise, the infection may be conveyed by these children to others. Close association, therefore—for instance, in the schoolroom or bedroom—is to be avoided. There will, of course, be much less risk from association out of doors.

CHAPTER XXXII

TUBERCULOUS MENINGITIS

Some idea of the frequency of meningitis in childhood may be gleaned from the following figures: in the three years 1905–8 there were admitted to the Children's Hospital, Great Ormond Street, 8,948 children; of these 174 were suffering from tuberculous, 49 from cerebro-spinal or posterior basic, and 17 from suppurative meningitis.

These figures give also a very fair idea of the relative frequency of these three forms of meningitis, apart from the special variations which may be induced by an epidemic of the cerebro-spinal variety.

I have spoken of these three forms of meningitis as if they were the only varieties met with in childhood; and indeed, for practical purposes they are: even amongst the many thousands of children who pass through a children's hospital, it is extremely rarely that any other variety is seen. I have met with thickening and matting of the meninges in the posterior fossa two or three times in infants with congenital hydrocephalus, where one rather assumed than knew that the condition might have been due to an intrauterine meningitis perhaps of syphilitic origin. I have also seen one case in a child of about four years, where there was strong clinical evidence of a gummatous meningitis. It has been thought that rheumatism may possibly cause a meningitis, but I know of no satisfactory evidence that it can.

When seeing children with meningitis, I have sometimes been asked whether it might not be influenzal. It has been proved by bacteriological evidence that meningitis does occur but with extreme rarity as a result of infection with the bacillus of influenza, but my own experience has been that the supposed influenzal meningitis usually turns out to be either one of the common forms of meningitis or not meningitis at all.

Tuberculous meningitis is by far the commonest variety, so I shall take this as my starting-point in some remarks chiefly upon diagnosis and treatment. There seems to be no special sex liability: out of 150 cases under my own observation 73 were girls, 77 were boys.

In its age-incidence tuberculous meningitis follows closely the curve of tuberculosis in general; it is so rare under the age of six months that one should always hesitate to make this diagnosis in infants at this age; it is far more frequent in the second year of life than at any other period, and though gradually diminishing in degree there persists, until the end of the fifth year, a special tendency to this affection.

The adjoining chart of age-incidence shows the contrast between tuberculous and posterior basic meningitis in this respect, a point of some value in diagnosis, for it is evident that under

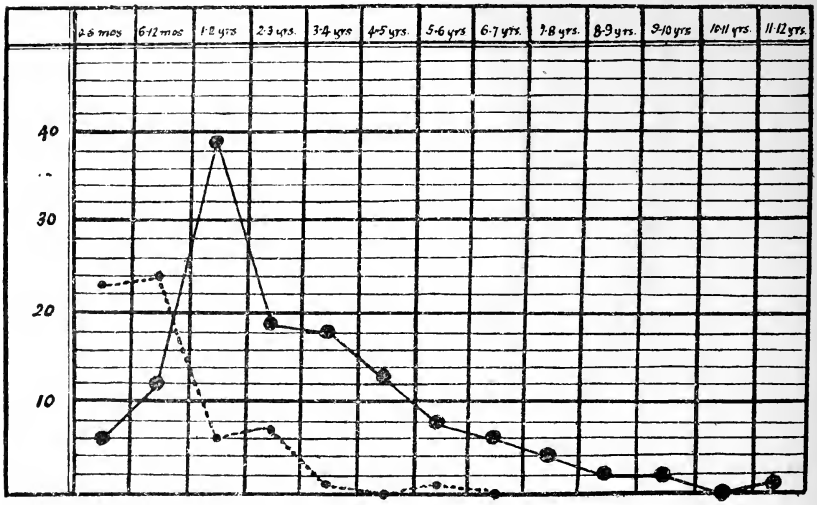

FIG. 32. Age-incidence of tuberculous meningitis (continuous line) contrasted with that of posterior basic meningitis (dotted line).

the age of six months meningitis is more likely to be of the posterior basic than of the tuberculous variety.

One other point I wish to emphasize in connexion with the age-incidence of tuberculous meningitis; namely, the very special liability to it during the first five years of life. If we can do anything in the way of prophylaxis against this terrible disease, it is chiefly during these first five years that our precautions should be taken. There are two sources of tuberculosis which are responsible for a certain number, perhaps for a large proportion, of the deaths from this disease in early childhood: to wit, living in close contact with other persons, especially nurses or relatives, who are suffering with some form of tubercu-

losis, and the use of unboiled or un-pasteurized cow's milk. In the nature of things it is impossible to produce scientific proof of this causation in any particular case, but none the less I think that clinical sequence when observed repeatedly may furnish at least a strong presumption of cause and effect, and for practical purposes may be hardly less important than scientific proof.

No one doubts that there is a risk of tuberculous infection from being with those who are tuberculous, but in the everyday care of children this is too apt to be forgotten. I have notes of cases where a father or mother has developed pulmonary tubercle; the signs were but slight no doubt, a few crepitations at one apex, hardly more, but no one thought of the danger to the little child who slept in the parent's bedroom, and two or three months later the child was dying of tuberculous meningitis. Let me quote a more striking instance: a grandfather had shown signs of pulmonary tuberculosis and was ordered abroad, what more natural than that he should visit his married son, and see his little grandchild before going? He did so, and stayed in his son's house a few weeks; about two months later I saw the grandchild with tuberculous meningitis, of which he died. In the meantime the grandfather had gone abroad, and there taken up his residence with another married son, who also had a young child; within a few weeks this child also sickened with tuberculous meningitis and died.

Probably the risk is greatest where the child is brought in contact with persons having the pulmonary form of tuberculosis, but I am not by any means sure that it is safe to allow a young child to associate with persons who have other forms of tuberculosis; for who shall say with certainty that the lungs are entirely clear in the child who has extensive tuberculosis elsewhere, say in the abdomen or in the cervical glands? Twice at least I have known tuberculous meningitis to occur in children who had been apparently in good heath until this illness, but had been living recently in close association with children having tuberculous glands in the neck.

With regard to the drinking of unboiled milk, I think there is no reasonable doubt that this is one source of tuberculous infection in general, and in particular leads to tuberculous meningitis *viâ* infection of the mesenteric glands. I have been very much impressed by cases in which a child remained apparently in perfect health upon scalded or boiled milk, and then a change was made, sometimes because the family had moved to a district in which the milk was reported to be specially reliable, the milk

was given unboiled, and within a month or two the child has died of tuberculous meningitis. I have noted this sequence sufficiently often to make me suspect very strongly that unboiled milk is a real danger in respect of tuberculous meningitis.

Parents often inquire whether a blow or a fall on the head can have caused the meningitis: certainly the sequence sometimes suggests that there may be some connexion; and it is reasonable to suppose that a severe blow on the head may lower the resistance of the meninges and so allow the tuberculous infection already present in other parts of the body to invade this part, but we have no proof of this connexion, and considering the many tumbles and knocks to which little children are liable, one must be careful in accepting a causal relation where there may be nothing more than coincidence.

The causal relation of mental strain, for instance, of school work, seems to me still more doubtful, but even this I would not pooh-pooh altogether, for we know very little about the psychical influences which may determine resistance to infection, and if a parallel were required I would point out that the strain of school work may almost certainly determine the incidence of chorea, notwithstanding the evidence which accumulates that chorea depends upon a specific infection.

Symptoms. In its most familiar form tuberculous meningitis is usually the first indication of tuberculous infection, and in this sense the term 'primary' has been applied to it, to distinguish it from the meningitis which arises either in the course of some longstanding and extensive tuberculous affection, for instance, tuberculous peritonitis, or as part of a general acute miliary tuberculosis; in either of these cases its course is apt to be somewhat different from that seen in the so-called 'primary' form, the cerebral symptoms are of shorter duration, and, indeed, may not become evident until a few days before death.

But from the pathological standpoint it may be doubted whether tuberculous meningitis is ever really primary; even though no symptoms or signs have been detected during life to raise suspicion of tubercle elsewhere, there is almost, if not quite invariably, a caseous focus to be found by sufficiently careful examination in some other part of the body, usually in the mediastinal or mesenteric glands.

This point has its clinical bearing: in the early stage when the diagnosis of tuberculous meningitis is in question, the detection of enlarged glands in the abdomen may confirm our suspicions, and at a later stage when, although it is evident that there is

some form of meningitis present, it is still doubtful whether the infant is suffering with tuberculous or with posterior basic meningitis, I have sometimes obtained valuable guidance by detecting hard and enlarged glands on careful abdominal palpation.

It is in these cases of so-called primary tuberculous meningitis that the insidiousness of onset so often makes diagnosis difficult at first, nay, I think, impossible often for a week or more. Let it be remembered that tuberculous meningitis does not pick out for its victims always, or even usually, the sickly and ill-nourished: too often it is the rosy-cheeked, healthy-looking child that falls a prey to tuberculous meningitis. I mention this because often at the early stage too much stress is laid on the previous good health and present good nutrition of the child as weighing against this diagnosis.

The first indication of the coming trouble is often for a few days nothing more than simple malaise,—the child who has been bright and active becomes unnaturally quiet, does not care to talk, wants to lie about; and perhaps, but not necessarily, complains occasionally of headache. I have sometimes thought that a particularly characteristic feature at this earliest stage is the way the child brightens up for a short time, perhaps for a few minutes, perhaps longer, and then wants to be quiet again.

Sometimes a mental alteration is the most noticeable symptom, especially in the older children; the previously good-tempered happy child becomes cross and peevish. I remember one girl about ten years old, in whom the first symptom to attract attention was alteration of behaviour at school; she had been an obedient well-behaved child, but now became so disobedient and troublesome that the teacher complained, and the parents, suspecting that there must be something more than naughtiness, brought her to hospital, where other evidence soon appeared of tuberculous meningitis.

In older patients I have known this early mental change to be mistaken for hysteria: it has also simulated acute mania. In some children an early symptom is slight mental confusion, the child uses a wrong word occasionally although otherwise quite clear and rational; or there may be more definite delirium. If the child is already walking the gait may become unsteady, perhaps definitely tremulous.

Within a few days at the most, vomiting occurs: it is, I think, the most constant of the early symptoms. In its most characteristic form it is independent of meals, and it occurs with little

or no retching, the contents of the stomach gush up without effort; but I should be sorry to lay much stress upon these features, the vomiting is often very definitely related to food, and so long as the child takes no food there may be no vomiting. Of far more importance is the fact that it occurs with obstinate constipation; to my mind this combination of apparently causeless vomiting with constipation in any child over six months of age, especially if therewith there is complaint of headache, is always suggestive of tuberculous meningitis.

The vomiting is seldom severe, it occurs perhaps only once or twice in the twenty-four hours, and seldom persists for more than a few days, though it may reappear towards the end of the illness. But to this rule there are exceptions. I have seen cases in which there was no vomiting throughout, but this is extremely rare; much more often vomiting is a marked feature of the early stage when the whole aspect of the case may be gastro-intestinal rather than cerebral. I suppose there is no commoner mistake in the diagnosis of this disease than to conclude that the vomiting is due to some error of diet or to a 'bilious attack'.

In rare cases the vomiting is very severe, even water cannot be retained, and then I have known the condition mistaken for 'cyclic vomiting'; or, again, the combination of severe vomiting with obstinate constipation has raised the question of intestinal obstruction. I remember a case of tuberculous meningitis which was sent into a surgical ward for operation for supposed acute intestinal obstruction.

The subsidence of the vomiting after a few days raises hopes which are only too soon to be destroyed, for the headache persists, and the child does not recover his natural vivacity. Headache is not always severe; sometimes, indeed, its apparent slightness has thrown doubt upon the possibility of tuberculous meningitis. Both in infancy and in early childhood, the only evidence of headache may be the putting of the hand up to the head; infants with headache sometimes beat the hand against the head; often in young children the presence of headache has to be judged from the pained knitting of the brows. It is curious how a young child will say 'no' persistently if asked whether his head hurts, although the child may be knitting his brows and putting his hands up to his head in a way that makes it almost certain that there is headache. If the fontanelle is still open its unnatural fullness and tension is noticeable usually very early in the disease.

At the same time there is an unnatural drowsiness which may indeed be the only recognizable symptom if the child is too young to talk: the child likes to be quiet with his eyes closed, and although for some days he will reply to questions and occasionally bestirs himself to take a languid interest in his toys or picture-books, it is soon evident that the drowsiness is increasing from day to day. At this stage the decubitus becomes characteristic: the child lies on his side with his legs drawn up, knees and hips flexed, the arms adducted and forearms flexed and the hands constantly picking at the lips, which are becoming dry and frayed.

He objects to the light, more by turning away from it than by any verbal complaint, and he dislikes being disturbed; if one pulls aside the bedclothes which are half covering his face, he pulls them pettishly over him again, and says peevishly, 'Go away', or 'Leave me alone'. Some children at this stage will cry out occasionally either with a simple short sharp cry, or with a more definite complaint, 'Oh! my head! my head!'

The abdomen by this time is beginning to have the empty hollowed-out appearance which at a later stage becomes still more striking, and I have often noticed a visible peristalsis of coils of intestine as if the nervous irritation affected the bowels with irregular and ineffective contractions. Another indication of nervous irritation is the so-called 'tache cérébrale': on scratching the skin gently with one's finger-nail there appears within a few seconds a bright red flush along the line of the scratch; or else a white line of anæmia quickly bordered and then replaced by a red flush.

Usually it is not until a week or ten days after the onset of symptoms that motor disturbance becomes evident in a slight droop of one eyelid, or some squint, or, more rarely, some paralysis of face or limbs; the pupils at this stage tend to be contracted. The neck is often slightly stiff, so that the child objects to having it flexed; but anything like well-marked head-retraction or opisthotonos of the rest of the spine is very rare, and would be prima facie evidence against tuberculous meningitis, and in favour rather of cerebro-spinal or posterior basic meningitis.

A symptom which is often noticeable during this middle period of the disease is a fine tremor of the hands on movement; this may be made more evident by raising the child into the semi-recumbent position. Another symptom which is of value in diagnosis is the tendency to a deep, sighing expiration at

intervals, perhaps once or twice in five minutes; this is noticeable sometimes before other symptoms have become sufficiently pronounced to make the diagnosis clear.

The pulse, which in the earliest stage of tuberculous meningitis is slightly quickened and irregular, gradually becomes slower, and at the stage now reached, the middle period or, as some writers have called it, the 'irritative stage' of the disease, it becomes slow, sometimes only sixty or even forty per minute, of high tension, and more markedly irregular.

The urine shows as a rule nothing characteristic in this disease, but in a certain proportion of cases, which my colleague, Dr. A. E. Garrod, has found to be about 30 per cent., sugar (glucose) is detected in the urine during the few days preceding the fatal termination. The amount of the sugar is small; Dr. Garrod tells me seldom more than one per cent. I have found it in several cases, and it certainly may be present as long as a week before death. Its value for diagnosis is not great on account of its late appearance, when the symptoms are already pronounced.

The temperature is often remarkably little affected; at the beginning of the disease it may be raised to 100° or thereabouts at some time of the day, but after a few days it falls to normal and remains so until the last day or two, when it may become subnormal or run up just before death to 106°–107° or even higher; but in some cases it is raised throughout, being always a little above normal and running up daily to 101° or 102°.

I have noticed in several cases attacks of collapse, in which the child turned grey and cold, the pulse became very feeble, and the temperature fell suddenly to normal or subnormal; these attacks have occurred two or three times at intervals of a few days; they have lasted perhaps half an hour or less. I have always supposed that they indicated some interference with the cardiac centres in the medulla, either by increase of pressure in the ventricles or by tubercular deposit about the medulla.

By degrees the drowsiness passes into more or less complete coma, and the final or 'paralytic' stage, as it has been called, is reached. The pulse becomes more and more rapid, 155–200 or more per minute, and its irregularity disappears; the child no longer lies curled up on his side but lies on his back, often picking with tremulous hands at the navel, or with the hands folded one over the other just over the pubes, with one or more limbs rigidly extended and undergoing slight clonic spasms from time to time. The pupils hitherto contracted become dilated, perhaps unequally, and muco-pus tends to accumulate on the cornea;

the face becomes deeply flushed, often an intense scarlet, and beads of perspiration break out, especially on the forehead ; the respiration becomes irregular or definitely of Cheyne-Stokes type ; in a day or two the scarlet flush upon the face becomes more dusky and blue as the respiration becomes more hurried and irregular, and soon the pulse, which has become small and uncountable, fails altogether and the child dies.

Such is the characteristic course of a tuberculous meningitis : in its middle and last stage so obvious that a fool cannot err therein, in its beginnings so deceptive that the most sagacious physician may fail to recognize that anything serious is amiss with the child.

Diagnosis. As I have already mentioned, the combination of constipation with vomiting and headache is the characteristic grouping of symptoms at the onset of tuberculous meningitis. If one of these symptoms is more constant than another it is constipation : all three are usually present. When a child is ailing with these symptoms the fundus oculi should always be examined, for although in the large majority of cases no change can be detected during this early period, there are cases in which our suspicions are confirmed most unexpectedly by the finding of well-marked optic neuritis. Usually pronounced change is to be found only during the last few days of the disease, when no help is required in diagnosis, but often an experienced observer can detect an unnatural fullness and darkness of the veins, especially near the disc, soon after the onset of symptoms, which may help to confirm the diagnosis.

Tubercle of the choroid is almost never seen apart from general acute miliary tuberculosis, where tuberculosis of the meninges though almost invariably present is seldom the prime cause of the clinical symptoms, which are mainly connected with the pulmonary condition ; but it is well to be on the watch for tubercle of the choroid even in cases where so-called ' primary ' tuberculous meningitis is in question ; twice at least amongst the many scores of cases of tuberculous meningitis which I must have examined, I have found a solitary tubercle in the choroid at an early stage when the diagnosis was still doubtful.

I have already mentioned some of the conditions for which tuberculous meningitis is often mistaken : teething, gastric disturbance, ' bilious attacks,' an ordinary sick-headache, these are the innocent conditions it simulates at first ; then the persistence of headache may suggest typhoid, but the points of distinction are many ; the patient with typhoid usually lies on

his back, not curled up on his side like the child with tuberculous meningitis, the fever in typhoid is usually higher and more continuous, the fullness of the abdomen, the enlargement of the spleen, the presence of the typical rose spots, the Widal reaction, are all foreign to tuberculous meningitis, in which also careful observation may detect some slight indication of cranial nerve paralysis which may give the necessary clue.

Where ear discharge has been present before the cerebral symptoms began, it is sometimes extremely difficult to distinguish between cerebral abscess and tuberculous meningitis: the brain symptoms usually turn out to be due to tuberculous meningitis in these cases because it is much the commoner disease, but none the less it is necessary to be on the watch for symptoms pointing to cerebral or cerebellar abscess. Pain or tenderness in the head localized to the side on which there is ear disease, an optic neuritis much more marked on this side than on the other, and paralysis of cranial nerves limited to this side: these are some of the points which may serve for the distinction, but the symptoms both of abscess and of tuberculous meningitis are extremely variable and it is a very difficult, sometimes, I believe, an impossible diagnosis. I have seen cases operated upon, and, indeed, advised the operation myself, for supposed abscess, where post mortem examination has shown it to be tuberculous meningitis.

I have known infantile paralysis to simulate tuberculous meningitis very closely even in the occurrence of squint, facial paralysis, and semi-coma: but the involvement of cranial nerves or nuclei is very rare in infantile paralysis. The converse error has been made occasionally of mistaking tuberculous meningitis for infantile paralysis, where, as rarely happens, the earliest pronounced symptom of the meningitis is paralysis of one or more limbs.

In infants, as I have mentioned elsewhere (Chapter XL), the nervous symptoms which accompany the high fever of acute pyelitis are sometimes mistaken for those of tuberculous meningitis, which is hardly surprising, as drowsiness, squint, twitching, and even stiffness of the neck may be present with acute pyelitis (vide p. 280). The sudden onset, however, and extremely high fever and absence of fullness of the fontanelle are all unlike tuberculous meningitis; moreover, the pyelitis is obvious on microscopic examination of the urine.

Tuberculous meningitis has to be distinguished from other forms of meningitis. Suppurative meningitis differs chiefly in

its rapid course, its whole duration is less than a week, perhaps usually three or four days; moreover, it is almost always secondary to some obvious forms of infection, generally pneumococcal such as lobar pneumonia, or empyema, or to some pyæmic condition.

Cerebro-spinal and posterior basic meningitis differ in the suddenness of their onset: there is no prodromal period of indefinite malaise, the child becomes at once acutely ill, often the mother can date the onset from a particular hour on a particular day; head-retraction, which is the exception and even when present is generally very slight in tuberculous meningitis, very quickly—sometimes even on the first day—becomes a marked and characteristic feature in the cerebro-spinal and posterior basic varieties; the temperature also tends to be higher than in tuberculous meningitis, from which also in some cases the presence of a rash, herpetic, purpuric, or a blotchy purplish erythema, may make diagnosis easy.

Whether any distinction should be drawn between cerebro-spinal and posterior basic meningitis is perhaps still doubtful. As regards clinical symptoms, I think that the cases to which the term Posterior Basic has been applied are, as a rule, of milder type than those usually described as 'Cerebro-spinal', this group also comprises chiefly infants under one year, whereas the more severe cerebro-spinal disease, though it attacks infants, frequently attacks also older children; whether sporadic or epidemic, the cerebro-spinal variety is much more frequently associated with rashes, purpuric or herpetic, than is posterior basic meningitis. But these may be merely differences of degree, and I doubt whether there are sufficient clinical grounds to justify any distinction unless, as some observers think, the diplococcus of the posterior basic disease can be distinguished by some of the recent methods of differentiation from that of cerebro-spinal meningitis.

I have laid stress upon head-retraction in the diagnosis of tuberculous meningitis from these other varieties; but this symptom indicates nothing more than the fact that there is much irritation in the medullary region, and in the exceptional cases in which tuberculous meningitis produces much inflammation and matting in this region I have seen head-retraction just as marked as in a cerebro-spinal or posterior basic meningitis: this, however, is very rare.

Nowadays other methods of diagnosis are open to us. Lumbar puncture may give information on at least three points, the tension of the cerebro-spinal fluid, the number and character of

the cells contained in it, and the nature of any bacteria which may be present.

Increase of tension tells us little more than that there is probably some organic disease of the brain: increase in the number of cells present points to inflammatory mischief; if the predominating cell is polymorphonuclear, the inflammation is likely to be of more acute variety than if most of the cells are lymphocytes: the particular micro-organism causing the inflammation is determined by the bacteriological examination of the fluid. At the Hospital for Sick Children, Great Ormond Street, Dr. J. G. Forbes [1] found tubercle bacillus in the cerebro-spinal fluid in 27 cases out of 47 cases of tuberculous meningitis in which lumbar puncture was done during life, and in a more recent series he found it in 25 out of 31 specimens. The fine lymph clot which usually forms if the fluid withdrawn is allowed to stand for a short time, offers the best chance for the discovery of the bacillus by staining.

Lumbar puncture is clearly a most valuable adjunct to diagnosis, but it is only one element in diagnosis; there are cases in which clinical symptoms give far more reliable information than does lumbar puncture; for instance, I have notes of cases under my own care in which a diagnosis of the variety of meningitis, based upon the cell contents of the cerebro-spinal fluid, would have been entirely wrong, and of other cases in which sterility of the cerebro-spinal fluid was reported when post mortem showed either tuberculous or cerebro-spinal meningitis.

These facts only emphasize the necessity for considering lumbar puncture in conjunction with clinical symptoms: either may mislead us if considered alone; the chance of error is diminished by checking the information from one source by that from another.

But valuable as lumbar puncture is where certainty of diagnosis cannot be reached by consideration of symptoms alone, and where at the same time the patient's welfare is at stake for want of certainty, there is a large proportion of cases in which any experienced physician can make a reliable diagnosis both of the presence and of the variety of meningitis without having recourse to lumbar puncture, and where this is so lumbar puncture is only to be advised if it has any therapeutic value, as it may have in cerebro-spinal meningitis.

Prognosis. To say that tuberculous meningitis is invariably fatal would perhaps be too sweeping a statement, but certainly

[1] *Quarterly Journal of Medicine*, January, 1908, i, 2, p. 109.

it does not far exceed the truth. Amongst at least 150 cases of tuberculous meningitis under my own observation, I have not seen a single recovery, nor do I know of any instance of recovery amongst the large number of cases admitted during recent years with tuberculous meningitis at the Children's Hospital. I say 'during recent years', for the record of supposed recoveries in former days, when cerebro-spinal or posterior basic meningitis was confused with the tuberculous variety, are necessarily unreliable. But face to face with the individual case may we not rather cling to the possibility of recovery until death disproves it? for there are instances on record which strongly suggest, if they do not prove, that, as with tubercle elsewhere, so in the meninges, though much more rarely, the process may be stayed.

I once saw a very remarkable improvement in a child who had been admitted with evident tuberculous meningitis; optic neuritis was present, and squint, and the child gradually fell into a semi-comatose condition; then most unexpectedly he began to improve, consciousness returned, and he became so well that he sat up and played with his toys and talked and seemed to be on the road to recovery, but the squint remained, and after a few days he again became worse and died some days later of tuberculous meningitis.

A case still more suggestive of the possibility of recovery was recorded by the late Dr. H. Ashby. A girl, aged $6\frac{3}{4}$ years, who had abscesses in her legs, developed headache, with staggering gait and some delirium; then she began to squint, optic neuritis was present, she vomited, screamed occasionally, and passed her fæces under her. With treatment by ice-bags to the head and bromides by mouth she gradually recovered and was discharged three months later; after six months she returned to hospital with symptoms of meningitis and died. Post mortem showed recent tuberculous meningitis and much matting.

That recovery has actually occurred in some cases may now be taken for an ascertained fact, since the introduction of lumbar puncture has made it possible to detect the tubercle bacillus in the cerebro-spinal fluid during life. Dr. A. E. Martin[1] collected 20 cases, which had been recorded since 1894, in which recovery had occurred from symptoms believed to be those of tuberculous meningitis, and in several of these lumbar puncture had shown tubercle bacilli in the cerebro-spinal fluid. In some inoculation of guinea pigs with the fluid withdrawn produced tuberculosis.

[1] *Brain*, Part II, 1909, p. 209.

Lastly, I must add that Dr. Nathan Raw has recently reported two recoveries of children 'with all the classical symptoms of tuberculous meningitis'.

But one has told the parents that the hope of recovery, if any, is small, and the next question is, how long will the child live? Of course there is considerable variation in the duration of the disease, but as a useful generalization I think one may say that the fatal termination is usually about twenty-one days after the date upon which the first definite symptom of illness, such as vomiting or headache, or distinct malaise was noticed. In the individual case our estimate will be determined also partly by the stage already reached, the bright flushing of the face seldom fails to appear as a warning before the end approaches, and from the first beginning of this symptom to the end of the disease is usually about four days at most. The state of the pulse also furnishes some indication; the slowness of the middle stage is, almost, if not quite, invariably followed by a gradual increase up to great rapidity during the last few days of life.

The differentiation between the three forms of meningitis, tuberculous, suppurative, and posterior basic, is of importance in the matter of prognosis. As regards duration, it may be said roughly that they last respectively three weeks, three days, and three months. Suppurative meningitis is probably as hopeless as tuberculous, and its duration, so far as can be judged from clinical symptoms, is almost always less than a week. Posterior basic and cerebro-spinal meningitis, though often fatal, are by no means always so; about 10 per cent. of the cases of so-called posterior basic meningitis in infancy recover, and recovery is not uncommon in cases of cerebro-spinal meningitis in older children, even when the symptoms are very severe.

Treatment. It is very doubtful whether treatment can do anything to stay the course of tuberculous meningitis. Formerly mercurial inunction to the back of the neck was a common mode of treatment, but I cannot say that I have seen any good from it. The Unguentum iodoformi has been used; I think I have seen definite advantage from iodoform in tuberculous peritonitis, and one can believe, therefore, that this drug may possibly be helpful in tuberculous meningitis.

Recently I have used urotropin, for the experiments of Flexner have shown that this drug, when administered by mouth, quickly appears in the cerebro-spinal fluid and acts as an antiseptic, inhibiting infection to some degree. It is conceivable that, given

in large doses at the early stage of tuberculous meningitis, it may at least assist any tendency towards recovery, and it is clear from such cases as I have mentioned above (p. 465), that even without assistance Nature makes a strong effort towards spontaneous recovery in some cases. In one case in which I used urotropin the disease certainly ran a slower course than usual, and in another where the symptoms pointed strongly to tuberculous meningitis, the child, a boy of 4½ years, had so far improved when I saw him five and a half weeks after the onset of the illness as to be running about at play. I had seen him first on the fourteenth day of the illness, and urotropin was given in doses of 7 grains every four hours, until after about six days the occurrence of hæmaturia, which large doses of this drug sometimes produce, necessitated its cessation.

To an infant 6–12 months old 3–4 grains of urotropin may be given every four hours, and to a child 3 years old 5–6 grains every four hours; but I have given as much as 10 grains every 2 hours to an infant 21 months old without ill effect, and probably it is wise to use large doses like this, at any rate for two or three days, after which, if necessary, the smaller doses mentioned may be given. Experiments seem to show that large amounts of this drug are necessary to obtain its antiseptic effect in the cerebro-spinal fluid. The effect which may necessitate its discontinuance or reduction is, as already mentioned, hæmaturia.

I have used tuberculin injections with and without the guidance of the opsonic index, but in the few cases in which I have tried it, there has not been the least improvement so far as I could observe; in this the experience of others seems to agree, but recently Dr. Nathan Raw has stated that out of four cases treated with tuberculin, two recovered after four injections; the evidence of tuberculous meningitis in these cases rested only on clinical symptoms, which, however, were considered to be quite typical.

The fact that in some cases of tuberculous meningitis there is transient return of consciousness a few hours before death when blood pressure is falling, and, presumably, intracranial tension is diminished, led me to hope that lumbar puncture might possibly restore consciousness for a time in the same way, but even for this purpose it proved useless. In one way I have known lumbar puncture to be of value in the treatment of this disease; it sometimes has a markedly quieting effect upon the clonic spasm or general convulsive restlessness of the late stage of tuberculous meningitis, which may be an important matter where the parents are greatly distressed thereby.

But if we can do nothing to ward off a fatal ending we can at least do something to prevent suffering. An icebag to the head, a quiet room and darkened windows will conduce to the child's comfort, and if the icebag does not relieve the headache, phenazone gr. i–iv, the smaller dose for an infant of one year, the larger for a child of eight or nine years, or phenacetin gr. iij–v, with caffein citrate, one grain, sometimes relieves it definitely, and if these fail and the child is restless and crying out frequently or moaning with pain, I have seen marked relief from morphia; in the later stage clonic spasm, which, if not distressing to the patient, may be distressing to the parents, is sometimes diminished by bromides to which chloral may be added.

Sooner or later nasal or rectal feeding becomes necessary as coma becomes more profound, and at this stage watch must be kept upon the bladder, for the urine may be retained. I have sometimes found the bladder enormously distended where this has been forgotten. Throughout the illness it will be necessary to keep the bowels working; as long as the child will swallow, calomel is perhaps best, as likely to produce a fluid stool and relieve blood pressure to some slight degree, but in the later stages, and sometimes in the earlier, it may be necessary to enforce action of the bowels by glycerine suppositories or enemata.

CHAPTER XXXIII

RHEUMATISM

Rheumatism is one of the many diseases which illustrate the considerable differences which may exist between the manifestations of one and the same disease when it occurs in an adult and when it occurs in a child. The conception of rheumatism as essentially a joint disease is based on its occurrence in adolescent and adult life; the wider and almost certainly more accurate conception of rheumatism as a general disease, probably of infective origin, is based chiefly on its manifestations in childhood.

In a child the articular phenomena of rheumatism become a matter of merely secondary importance; indeed, judging from clinical evidence, one would say that a child may suffer severely from rheumatism who has never had a pain in its joints in its life. Some of the most severe cases of endocarditis, cases in which the rheumatic nature of the lesion has been confirmed by the presence of rheumatic nodules, have not at any time had any joint pains whatever so far as can be ascertained.

As is well known, the joint symptoms in a child are often so slight that the parents take no notice of them, beyond dismissing them from their memory with the comforting assurance that they are 'only growing pains', so that even with careful inquiry it is often difficult to be quite certain that some such transient aches or pains may not have occurred at some previous date and have since been forgotten. Allowing, however, for such a fallacy, one sees cases of undoubted rheumatism in childhood in which joint phenomena would seem to be entirely absent.

I venture to lay some stress upon this point *in primis*, for it seems to me that we must widen our conception of rheumatism if we would understand aright the relationship of those apparently disconnected phenomena which make up the rheumatic complex in childhood.

To measure the frequency of rheumatism in childhood by the number of cases which come under treatment for the articular manifestations is entirely to misunderstand the prevalence of this disease in early life. Even if the joint symptoms alone were

to be considered, such a method of estimation would involve a large fallacy; for the joint symptoms are so slight in children that comparatively few of the cases in which they occur come under medical observation until the presence of severe cardiac affection or the more obtrusive phenomena of chorea induce the parents to seek medical advice.

Moreover, the heart affections may apparently occur alone as the earliest symptom of rheumatism in children; and whatever view may be held as to the exact relation of chorea to rheumatism, the association of chorea with other rheumatic symptoms—particularly the cardiac affections and rheumatic nodules—where articular symptoms are absent, makes it necessary to include in our statistics not only the cases of chorea in which there has been concurrent or previous articular rheumatism, but also many others, if we wish to obtain any accurate idea of the frequency of rheumatism in childhood.

The following figures may serve to illustrate this point. Out of 383 as far as possible consecutive cases of chorea which came under my observation, 183 had concurrent or previous articular rheumatism (pains in limbs or joints) with or without heart affections and nodules; 8 had no joint symptoms, but had heart disease and rheumatic nodules; 2 had rheumatic nodules without cardiac bruits or joint symptoms; 12 had systolic and diastolic apical bruits; 3 had aortic diastolic as well as apical bruits; and 48 had only systolic apical bruits, without joint symptoms or nodules.

At the very lowest estimate, therefore (excluding cases with only systolic apical bruits), 54·3 per cent. of cases which came under treatment for chorea showed positive evidence of rheumatism; and it is obvious that unless such cases are included in the statistics of rheumatism the frequency of that disease in childhood will be considerably underrated.

As a matter of fact such cases too often figure in statistics as 'chorea' only, and perhaps this accounts in part for the statement sometimes made, that rheumatism is a disease of adolescent and adult life rather than of childhood, whereas in London at any rate it is probably more frequent in the later half of childhood (six to twelve years of age) than it is in older persons.

In the Children's Out-patient Department at King's College Hospital, where only children under ten years of age are treated, in 1,000 consecutive cases there were 229 children between six and ten years of age; of these 229, 13·1 per cent. showed evidence of rheumatism (3·5 per cent. attended for joint symptoms, the

rest were cases of heart affection or of chorea, in which there was satisfactory evidence from associated symptoms or from a history of previous joint pains, that the condition was rheumatic), and if children up to the age of twelve years were included, no doubt the percentage would be considerably higher.

The frequency of rheumatism amongst the in-patients of a children's hospital may be seen from the following figures. During the years 1897 and 1898, 681 children between the ages of six and twelve years were admitted to the medical wards (including diphtheria wards) of the Hospital for Sick Children, Great Ormond Street: 149 were cases of chorea, 63 of endocarditis, and 38 of articular rheumatism. Records were lacking in 29 of the cases of chorea, but of the remaining 120, 67 showed evidence of rheumatism (either previous articular rheumatism or definite endocarditis, not including systolic apical bruits of possibly doubtful nature); so that out of the 681 children, 168 showed evidence of rheumatism—that is, 24·7 per cent.

Such figures, however, only take into account those cases of chorea in which there is evidence of past or present rheumatism in heart or limbs or in the form of rheumatic nodules; but it may well be that in the light of bacteriology chorea may prove, in some cases at least, to be directly or indirectly the result of rheumatic infection, and therefore just as truly a manifestation of rheumatism as the articular symptoms. If this were so, chorea, like the heart affections and the rheumatic nodules, might be expected to precede the joint symptoms in some cases, and might be the first, or possibly the only, clinical manifestation of rheumatism.

The recent investigations by Dr. Poynton and Dr. Paine into the etiology of rheumatism would seem to point in this direction, and such a view would well accord with clinical experience; but until further evidence is forthcoming, one can only suspect that a considerable number of the cases of chorea in children without other manifestations of rheumatism, ought to be included in our statistics if we would realize fully the widespread influence of rheumatism in childhood.

Age-incidence. From the figures given above, it is, I think, sufficiently evident that rheumatism is an exceedingly common disease in the later period of childhood. During early childhood, however, rheumatic phenomena are much less frequent, and in infancy rheumatism is almost unknown. The age-incidence is shown in the accompanying chart (Fig. 33).

Several cases of supposed rheumatism in infancy, from twelve

hours old and upwards, have been recorded, a few well authenticated, others of very doubtful nature. Amongst 1,027 children with articular or cardiac rheumatism or with chorea, I have not seen a single case under two years of age, and I have seen only eight cases in which any of these rheumatic affections were present before the age of three years.

The youngest was a boy aged two years and five months with acute articular rheumatism; his sister was said to have had her first attack of articular rheumatism at eighteen months; another case, first seen with severe endocarditis at $2\frac{1}{2}$ years, had had an attack of acute articular rheumatism a month earlier. In one case the first attack of chorea was said to have begun

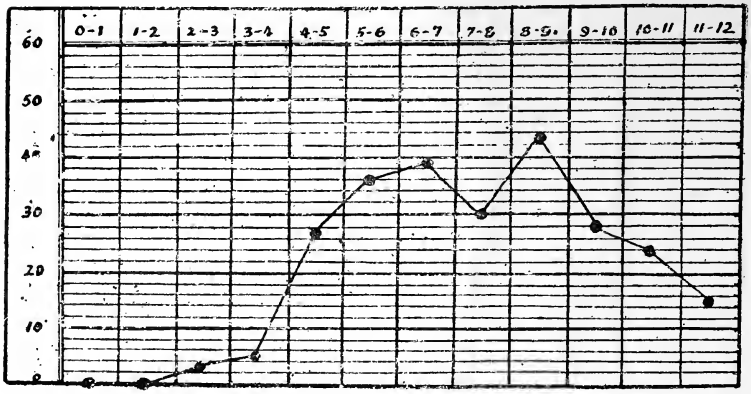

FIG. 33. Age-incidence of rheumatism in childhood.

when the child was two years old; in another articular rheumatism was said to have begun at this age; in another there was good reason to suppose that articular rheumatism occurred just before the age of two years, but I did not see the child until he was brought for rheumatism later. I had under my care a boy with chorea, endocarditis and articular rheumatism at the age of two years and ten months, and another boy in whom there was severe endocarditis, the rheumatic nature of which was confirmed by the subsequent appearance of a large rheumatic nodule on one elbow at $2\frac{1}{2}$ years.

Symptoms. The symptoms of rheumatism in childhood, as already pointed out, differ from those in adults chiefly in the much slighter character of the joint manifestations, and in the much greater prominence and frequency of the heart affections.

Reference has been made above to 'pains in the limbs' as evidence of rheumatism in children, and clinical experience shows that pains referred to the limbs, as distinguished from the joints, may be just as significant of rheumatism in children as swollen, red and tender joints. This is proved by the fact that it is quite common for children with only such vague 'pains in the limbs', for example, in the calf or thigh, to show other symptoms of 'acute rheumatism', such as endocarditis and pericarditis or subcutaneous nodules; moreover, definite swelling and tenderness of the joints may immediately precede or follow these pains in the limbs.

It is perhaps worthy of note that, even where there are only such vague and slight pains, examination of the joints may show that these are really affected. In a child under my care as an out-patient at King's College Hospital, routine examination showed considerable effusion into one knee, where the complaint of vague pains in the limbs in a child walking about with apparently no discomfort hardly suggested the possibility of such a definite joint affection.

The practical importance of recognizing the significance of these slight pains in the limbs in children—'growing pains' as they are too often called—can hardly be overrated. They may be the earliest indication of rheumatic taint, and as such should always serve as a danger signal. To disregard them is to favour the onset of more severe symptoms, and it seems likely that active treatment in this early stage may not only cause the disappearance of the pains but may also prevent those affections of the heart which are so distressing a feature in the rheumatism of childhood; certainly these vague pains call for the utmost care in prophylaxis, and a child who has once shown such symptoms requires continual care as to clothing, climate, and the prevention of exposure to cold and damp. The presence of these indefinite pains in a child makes it necessary also to keep a careful watch on the condition of the heart, for these apparently trivial 'growing pains' may be associated with or followed by cardiac disease as severe as any that occurs with the most acute articular rheumatism.

In connexion with the joint symptoms I may mention a point to which my attention was first drawn by Sir Thomas Barlow: the frequency of affection of the hip-joint in the rheumatism of childhood. This point is worth remembering in its bearing on diagnosis, for it happens occasionally that rheumatism in children remains limited to one joint for several days. Such

monarticular rheumatism is often extremely puzzling, and when it affects the hip-joint may easily suggest commencing tuberculous disease, or may lead to stranger errors owing to the very indefinite localization of pain by children. One child was sent to the Children's Hospital for perityphlitis, another for intussusception, and a third had been circumcised on account of vague pain in the right groin, which a few days later was followed by definite symptoms of rheumatism elsewhere. Another common complaint is 'pain at the back of the knee', often with no objective evidence of rheumatism in the knee-joint.

Another symptom which is of importance because it may be the earliest manifestation of rheumatism in a child, is stiff-neck. This may seem a trivial affection, but, like the vague pains and stiffness in the limbs, it may be followed or accompanied by severe cardiac rheumatism. I have seen severe endocarditis associated with rheumatic nodules in a child who had shown no other evidence of rheumatism but stiff-neck.

The frequency of cardiac affections is one of the most characteristic features of rheumatism in childhood. In 170 as far as possible consecutive cases with rheumatic manifestations, 128 had cardiac bruits, which in 93 cases were certainly due to endocarditis.

But endocarditis and pericarditis are by no means the only results of cardiac rheumatism, and in children, probably to a greater extent than in adults, cardiac dilatation is a frequent result of rheumatism, an important point to which Dr. Lees has specially drawn attention. In some cases this dilatation, associated with irregularity and rapidity of the heart, may be the only clinical evidence of cardiac affection; certainly in many cases it is the earliest indication of such affection.

It may not be out of place to mention here the wasting which so often accompanies cardiac rheumatism in childhood, and which would seem to be a more noticeable feature in children than in adults. It is no uncommon thing for a child to be brought for medical advice simply on account of wasting, by a mother who has no suspicion that the child has ever had rheumatism or has any affection of the heart, although examination reveals advanced cardiac disease, and on inquiry there is a history of vague pains in the limbs for many months past.

An important manifestation of rheumatism in children is the rheumatic nodule. This is to be found most often over the olecranon process and the condyles of the humerus and femur (Fig. 34), but also over the spinous processes of the vertebræ, the malleoli, the ends of the radius and ulna, the knuckles, and

sometimes over the occipital bone, scapula and crest of ilium, less often over tendon sheaths where these are superficial, for instance at the wrist and ankle. On dissection the nodule is seen as a tawny-yellow deposit, about the size of a millet-seed or hemp-seed in the meshes of loose connective tissue; a network of fine vessels can be seen passing on to the surface of the deposit, but it has no capsule. The histological character of these nodules shows that they are exactly analogous to the fibrinous deposit on the inflamed endocardium or pericardium, and their association almost always with endocarditis suggests that they are due

FIG. 34. Rheumatic nodules. Photograph shows nodules on patella, condyles of femur and head of tibia; girl aged about ten years with severe endocarditis.

to the same cause, the rheumatic irritant, whatever it may be. This view would agree with clinical experience, which shows that, in children at least, these nodules are almost, if not quite, conclusive evidence of rheumatism.

While in adults rheumatic nodules are rare, in children they occur with sufficient frequency to make them of considerable practical importance. In 50 consecutive cases of articular rheumatism in children under twelve years of age, no less than 23 showed nodules, that is, nearly half the cases; and even including cases of chorea (with or without evidence of rheumatism) and rheumatic heart disease without concurrent articular rheumatism,

I found nodules in 55 out of 200 as far as possible consecutive cases, i.e. in 27·5 per cent. But these statistics were drawn from cases admitted to the wards of the Hospital for Sick Children, and therefore apply only to the more severe cases, and give altogether too high a percentage for the average frequency of rheumatic nodules. In the out-patient department, where the slighter cases are included, the frequency of nodules in consecutive cases of chorea and cardiac or articular rheumatism is probably not above 10 per cent. (Some figures taken from my out-patients gave 9 in 84.)

The practical importance of these nodules in children is often great. Their presence in itself is a manifestation of rheumatism, and may therefore determine the character of other symptoms, such as pains in the limbs or cardiac bruits where these are of doubtful nature. Whether these nodules are ever the only manifestation of rheumatism may be doubtful, certainly it is extremely rare to find them without other rheumatic phenomena; but, as already stated, they may occur with chorea without other clinical evidence of rheumatism, a point which is worthy of note in considering the relation of chorea to rheumatism.

The close association of endocarditis with these nodules has also its clinical importance, for even if no signs of endocarditis are detected, the presence of nodules should always arouse a watchful suspicion as to the condition of the heart. In some of the few cases in which I have seen rheumatic nodules without evidence of cardiac affection, definite signs of endocarditis have appeared within a few days or weeks after the appearance of the nodules.

Moreover, the presence of nodules in a child has a very important bearing on prognosis; their frequent association with severe endocarditis and pericarditis makes their significance extremely grave, even where the prognosis otherwise might seem by no means unfavourable. In many such cases the cardiac disease proves fatal sooner or later; but its advance may be slow. One of the most marked cases I have seen, with numerous large nodules and endocarditis, seemed little worse when seen three years later, although many nodules were still present. I had under observation a boy who had several nodules and a systolic apical bruit during an attack of articular rheumatism; the bruit disappeared before he left the hospital, three years later no bruit was detected and the boy seemed in good health: in another case the heart was normal and the child seemed perfectly well 3½ years after an attack of chorea, during which many rheumatic

nodules had been present, with a systolic bruit over the base of the heart.

Amongst the symptoms of rheumatism in children, as in adults, must be reckoned sore throat. Tonsillitis so frequently occurs just before the onset of rheumatic pains in children that it is difficult to escape the conclusion that it bears some direct relation, possibly as medium of infection, to rheumatism ; its occurrence also is sometimes followed by an exacerbation of rheumatism.

The skin eruptions of rheumatism have not seemed in my experience to be particularly frequent in children, but their recognition is important.

The rheumatic child sweats easily, and therefore is liable to sweat-rashes, either in the form of tiny, pin's-head, clear vesicles, *sudamina*, or as papules capped with an opaque milky-white vesicle, *miliaria*. These are, however, not essentially rheumatic : they occur in any condition with profuse perspiration. Much more characteristic is the pink, very slightly raised eruption known as *Erythema marginatum*. The name is derived from the sharply defined edge of the patches, which tend to be circular, forming rings which, becoming confluent, show an irregular or gyrate margin. In a little girl under my care this curious contour had gained for her at home the nickname of ' the map girl '. This rash occurs chiefly on the trunk, especially on the chest, but also on the limbs, does not itch, and shows a great tendency to recur. Occasionally the erythema takes the form of papules rather than patches, and it may be associated with purpura. Apart from erythema, however, *purpura* is sometimes a striking feature, appearing in patches especially over one of the swollen joints during an attack of rheumatism. *Erythema nodosum* is still regarded by some as rheumatic, but there is very strong evidence from its age-incidence, its occasional epidemic occurrence, and the extreme rarity of a second attack, that it has no connexion with rheumatism.

There is one result of rheumatism in adults which is almost unknown in childhood, the so-called ' cerebral rheumatism ' or ' rheumatic hyperpyrexia '. One would have expected that the nervous instability which is so noticeable a feature in rheumatic children would have particularly favoured such a condition, but Dr. Cheadle states that he has never seen a case, and says that the earliest age at which he could find it recorded was thirteen years. One case occurred at the Hospital for Sick Children, Great Ormond Street, under the care of Dr. Gee (to whom I am indebted for permission to place it on record), of delirium with

fatal hyperpyrexia (108·4) in the course of acute articular rheumatism without cardiac affection or chorea in a boy of 6½ years.

In seeing large numbers of rheumatic children one is struck with the frequency of certain minor symptoms which, although not usually described as symptoms of rheumatism, seem to have a very close connexion therewith; and I think that the recognition of their association is often of considerable practical value.

A very common complaint in such children is that of 'pain in the stomach': in my experience this has seemed to occur most frequently in children who were suffering at the time with articular rheumatism or with occasional pains in the limbs. The pain is usually referred to the epigastrium, and is sometimes sufficiently severe to make the child cry; more often it is slight and transient, in some cases it was definitely after meals, but in others it seemed to be independent of them.

The association with the limb- or joint-pains suggests that the 'pains in the stomach' may sometimes be in the abdominal muscles, but I suspect that it is usually gastric in origin, and is due to a catarrhal condition in some way dependent on the rheumatism.

Another symptom which I have noticed less often is 'pain in the side', usually in the lower part of one axilla, with nothing otherwise to suggest pleurisy, and no sufficiently severe cardiac affection to account for it. This may possibly be due to rheumatism affecting the intercostal muscles; at any rate it is associated often with other symptoms of rheumatism in the heart or joints.

A common trouble in rheumatic children, and in the children of rheumatic parents, is headache: in some cases it may be dependent on some gastric catarrh as suggested above, in others, as Sir James Goodhart points out, it is associated with anæmia, whilst in others it may be part of the neurotic element which is so prominent in these children; but I cannot help thinking that in some cases it may have a more direct relation to the rheumatic poison whether bacterial in origin or not; the very frequent association of headache with chorea perhaps points in the same direction.

In our estimate of the wider influence of rheumatism in childhood we must take into account not only those symptoms which seem to be direct manifestations of rheumatism, but also those more distantly related phenomena which in children are so often associated with rheumatism or rheumatic heredity.

To my mind one of the most striking features in such children is the 'nervous' temperament to which Goodhart has specially drawn attention. The rheumatic child is *par excellence* the

nervous child. In many cases this is seen in an undue excitability—such children will become almost uncontrollable in their excitement over games and amusements; or, on the other hand, there may be excessive timidity; in some of the older children this is replaced by a sensitiveness or shyness which is almost morbid.

The frequency of rheumatism in association with night terrors is another illustration of this point. Sir James Goodhart found rheumatic parentage in 17 out of 37 cases, and Dr. Graham Little from a study of 30 cases concludes that 'a preponderating number of cases are found in rheumatic subjects with early heart disease'; in my own experience also the association of night terrors with rheumatism has been very frequent, and I have elsewhere pointed out a similar association in cases of day terrors.

Somnambulism is another of the neuroses to which such children are subject; they are also prone to talk in their sleep and to be restless at night after the least excitement.

Habit-spasm has also seemed to me to be specially frequent in rheumatic children; in some cases it occurred concurrently with pains in the joints or limbs, in others there was a strong family history of rheumatism. As a rule, the character of the movement, its limitation to a sudden blinking or a twitch of the nose, or a momentary lateral rotation of the head, has distinguished it from chorea; but in some of the cases with more complicated movements their nature is less certain, for the association with rheumatism naturally suggests the possibility of an unusually limited chorea, and I have more than once seen typical habit-spasm occur in rheumatic children who were said to have had chorea at some previous date.

Another ailment which I have several times seen associated with rheumatism in children is lienteric diarrhœa. This curious irritability of the intestine, which results in a desire to defæcate almost directly after—sometimes indeed before—a meal is finished, is almost certainly a nervous phenomenon. In cases under my care this has occurred sometimes in children under treatment for articular rheumatism, sometimes in the children of rheumatic families. The frequent association of such neuroses with rheumatism in children has to be remembered in considering the relation of chorea to rheumatism, but clinical experience suggests, I think, that chorea stands in a different and more direct relation to rheumatism.

There is one further phenomenon which is noteworthy in rheumatic children, and which is perhaps worth mentioning

here, although it is necessarily noticeable also in adults—the association of red hair with rheumatism and rheumatic heredity. The following observation may serve to illustrate it. In four days there were amongst my out-patients eleven children with red hair. Of these, two were attending with articular rheumatism; one had occasional pain and swelling in the knees, his mother had red hair and frequent pains in the limbs, and her brother and sister had 'rheumatic fever'; one had 'pains in the knees' and his mother had 'rheumatism'; three others showed a history of 'rheumatic fever' in the mother or father; one was attending with chorea; one had a brother attending with articular rheumatism; only two out of the eleven showed no rheumatism or chorea in themselves or their families.

Amongst 80 children with red hair (including the 11 already mentioned) 24 were attending with definite manifestations of acute rheumatism, articular or cardiac or chorea; 6 had pains in the limbs which were almost certainly rheumatic, and of the remaining 50 cases 17 showed a family history of acute rheumatism (including chorea) in parents or brothers or sisters; so that there was rheumatism either in the child or the family in 47 out of 80; i.e. in 58 per cent.

I would suggest that this colour of the hair is only the index of some fine peculiarity, perhaps in the chemistry of metabolism, which produces a soil favourable to rheumatic infection, and hence the association is only noticeable where such infection is rife. The colour of the hair may seem but a small point, but it is one of the many little indications which are sometimes of value in leading to the early detection of rheumatism in a child.

The importance of early recognition of rheumatism in children will be realized by those who are familiar with the frequency of heart disease in the rheumatic child. I suppose there are few more pitiful conditions than that of the child dying with cardiac rheumatism, and it is by paying attention to 'the day of small things', the apparently trivial aches and pains, and the various associations which make up the picture of rheumatism in childhood, that we may hope, in some cases at least, to prevent those terrible results which are too often seen from neglected rheumatism in children.

Diagnosis. But while it is most important to remember the possibility of rheumatism whenever a child complains of 'growing pains' or stiffness of the limbs, or shows some definite swelling of the joints, it must not be forgotten that there are many other causes of joint affection and pains in the limbs in children.

It may be laid down as a sound principle that a diagnosis of rheumatism in the case of a child under two years of age is almost always a mistake. One condition which is erroneously thought to be rheumatism in an infant is scurvy; such a case as the following is not very rare.

An infant, aged ten months, was sent to me for 'rheumatism'; he had been ailing six weeks with slight swelling and tenderness about one ankle; salicylates had been given with little advantage. Examination showed that the swelling was not strictly over the ankle-joint, but extended over the lower end of the tibia, which was tender, there was no effusion in the joint itself; the gums showed the purple spongy appearance of scurvy; under antiscorbutic treatment the child was well in a few days.

Another affection which is sometimes confused with rheumatism in infancy is acute epiphysitis, with which there may be suppuration in several joints; the general illness is more severe and the local symptoms more intense than is usual in rheumatism, and the occurrence of œdema, and suppuration in one or more of the affected joints, may settle the diagnosis.

I have seen a few cases under one year of age in which one joint has become swollen with evidence of slight effusion in it, with some limitation of movement but little or no tenderness; after continuing for several weeks the affection has passed off, leaving the joint apparently normal. The course of these cases has been unlike articular rheumatism, which does not remain limited to one joint for several weeks and which, moreover, is usually accompanied sooner or later by other manifestations of the disease, whereas in these there was nothing except the one swollen joint, and salicylates appeared to have no effect.

In one infant, aged six months, the right knee was affected thus for several weeks after summer diarrhœa; no evidence of syphilis could be obtained nor of tuberculous disease, and comparing this case with others that I have seen I supposed it to be an infective condition, but its limitation for so long to one joint was quite unlike articular rheumatism.

Again, I have seen arthritis in early childhood associated with a vulvo-vaginitis, which in children is most often due to the gonococcus.

A child, aged two years and eleven months, who was in the Children's Hospital under Dr. Lees, had a profuse purulent discharge from the vagina four weeks before admission. About a fortnight after the discharge began one hand became swollen and painful, and three days after this the other was affected. The hand showed swelling not limited strictly to the wrist, but extending over the dorsum of the hand and upwards slightly on the forearm. The vaginal discharge was treated, and the tenderness of the hands disappeared quickly, but the swelling remained for some weeks.

Without a bacteriological examination one cannot be certain, but it seemed most probable that this was a case of gonorrhœal arthritis.

There are cases also in which a gonococcal arthritis occurs in infancy without any apparent primary focus of infection such as an ophthalmia or a vaginitis; it is seen sometimes in male infants.

Charles H., aged seven weeks, was in King's College Hospital for diarrhœa and vomiting: a month after admission he seemed in pain, and two days later the left ankle became swollen; no other joint was affected. After four days it was opened by Mr. Carless, and about a drachm of pus evacuated which, on bacteriological examination by Dr. D'Este Emery, seemed to contain a pure growth of gonococci.

Dr. Emmett Holt[1] has reported twenty-six cases of gonorrhœal arthritis in children under three years of age. Dr. Kimball[2] has reported eight cases in infants not more than three months old, and several of these cases were males with no trace of gonorrhœal infection elsewhere. Probably such cases have hitherto been included under the head of acute arthritis of infants.

Lastly, there are cases of syphilitic synovitis in infancy associated with syphilitic epiphysitis. Dr. Melville Dunlop[3] has recorded two such cases in infants aged three months and six months.

I mention these various causes of swelling about or in the joints in infancy, not because they are common, for they are all very rare, but in order to show that there are several conditions to be thought of before we commit ourselves to the extremely unlikely diagnosis of 'rheumatism' in an infant.

I have considered the diagnosis of rheumatism in children past the age of infancy in my remarks on the various symptoms; but I will add these further cautions.

The rarity of acute pain and tenderness in the rheumatic arthritis of childhood should make us cautious where such manifestations are present. If a child cries out with tenderness when the limbs are touched the possibility of acute infective osteomyelitis is to be borne in mind, and the history of a sudden onset, perhaps with a rigor and a very high temperature and delirium and the extension of the swelling and tenderness beyond the limits of the joint, would point to this affection.

In connexion with most of the specific fevers an acute arthritis is occasionally seen in children; it may be of suppurative character and the constitutional and the local symptoms are

[1] *Med. Record*, March 25, 1905.
[2] Ibid., November 14, 1903.
[3] *Edin. Med. Journ.*, December, 1904.

likely to be more severe in such conditions than in rheumatism.

But I must point out also that in rare cases the constitutional disturbance with acute rheumatism is severe, and the joints are swollen and tender, and I have even seen œdema over a joint with acute articular rheumatism in childhood; moreover, there may be acute illness, high fever and sordes on the lips with nothing more than some stiffness of one or more joints, and until further symptoms appear the only guidance in diagnosis may be the rapid improvement under salicylate treatment.

In the child, as in the adult, rheumatism tends to flit from joint to joint; as I have already mentioned, it is occasionally limited for a few days to one joint, but a monarticular affection lasting several days should always raise suspicion that it may not be rheumatism but may be tuberculous disease or some other form of infective arthritis.

It seems excusable to mention here an instance of a form of arthritis which, though almost unknown in childhood, has been recorded in a few cases, namely gout, which Sir Alfred Garrod saw in a child aged 7 years, and Scudamore in a child aged 8 years.

Florrie P., aged 12 years, at the age of 3½ years had her first attack of gout in the right ankle, and since then, every few months, has had attacks of pain and swelling in the metatarso-phalangeal joint of the great toe, which becomes dusky red, and extremely tender; the child's temperature is not raised above 100°. The attacks subside within forty-eight hours on free administration of Vinum Colchici. The child's father has had typical attacks of gout since he was a young man; his father and grandfather also suffered with gout.

Prognosis. Rheumatism in the child must always be regarded as a serious condition on account of the special tendency in early life to heart affection. If there were only the joints to be considered rheumatism would be a disease of little gravity; it is extremely rarely that any trace of the articular affection remains permanently. I have seen one or two children in whom there occurred a chronic thickening about the joints like that described by Jaccoud as 'rheumatisme chronique fibreux', which he attributed to repeated attacks of rheumatic fever in young adults. I have elsewhere[1] recorded such a case in a boy aged five years. But this is so rare that it hardly calls for consideration in giving prognosis in the rheumatism of children. The point of greatest practical importance is to warn the parents of the liability to heart disease, and to impress upon them the extreme importance of prolonged rest if there is any suspicion of impurity or irregularity or undue rapidity of the heart sounds suggesting that there may be already some affection of valve or muscle.

[1] *Trans. Roy. Med. Chir. Soc.*, vol. lxxx.

Treatment. So far as the treatment of the joint affection is concerned there is little to be said. Under salicylate or aspirin the articular symptoms subside very quickly; to a child of ten years, if the symptoms are severe, 15 grains of the salicylate can be given every two or three hours for five or six doses and then less frequently; as a rule, a smaller dose, 10–12 grains is sufficient; if as much as 10 grains is used it is wise to give with it, as Dr. Lees has suggested, double as much sodium bicarbonate, to prevent any toxic symptoms from the salicylate.

It is the fashion now to give aspirin, which may be used in doses of gr. 5–7 for a child of six to twelve years. I have not been able to satisfy myself that it offers any special advantage over sodium salicylate; given in large doses it has the same toxic effects. Dr. Lees has recorded a case in which a child with chorea developed air-hunger and became comatose after aspirin had been given in doses of 15 grains three times daily only for seven doses, and, as he has pointed out to me, it has the disadvantage that it cannot be given with alkalies, as sodium salicylate can. Dr. Lees's valuable observations have shown that bicarbonate of soda in large doses sufficient to keep the urine alkaline effectually prevents the toxic symptoms of the salicylate group of drugs.

But how long should the salicylate or aspirin be continued? The answer to this question depends upon the view taken as to the action of this drug. There are those who deny to salicylate and the allied drugs any beneficial effect beyond the reduction of temperature and the relief of the joint symptoms; on the other hand, there has been attributed to this drug a specific effect as antagonistic to rheumatism in all its manifestations as mercury is to syphilis.

It has seemed to me probable that salicylates do more than merely relieve the arthritis of rheumatism. I have several times observed that after salicylates have been given and the joint symptoms for which the drug was administered have passed away and the temperature has become normal, the discontinuance of the salicylate has been followed after a few days by a further rise of temperature with no recurrence of joint symptoms, and this fever has subsided upon further administration of salicylates.

If the temperature in such cases may be taken to indicate activity of the rheumatic infection in other parts of the body after the joint symptoms have disappeared, it would seem that salicylate has some effect in stopping this activity. It is, I think, no evidence against the specific value of salicylate that rheumatic

endocarditis or pericarditis does not disappear under its influence, it may have a preventive value where it has no power to remove the products of inflammation or the mechanical results which a very little inflammatory exudation must produce in the heart valves; and when inflammation has occurred the drug may still have a definite value in preventing further inflammation of the part.

I throw out these suggestions as some justification for the view that the administration of salicylates should not be discontinued when joint symptoms disappear, nor even when the temperature falls to normal, but should be continued for two or three weeks after fever has completely subsided, and should be given during this time in moderately large doses, e.g. 10 grains of sodium salicylate three or four times daily, with double that dose of bicarbonate of soda to a child of eight years.

In the cases in which the joint pains tend to recur frequently in subacute form, I have used iodide, given in doses of 3–5 grains. A suitable mixture is Potassium Iodid. gr. v, Sod. Bicarbonat. gr. xv, Spirit Ammon. Aromat. ℥v, Syrup ℥xxx, Aq. Menth. Pip. ad ℥ij ter die. Local applications are hardly required in the articular rheumatism of childhood; it is sufficient to wrap the affected joint in some warm cotton-wool, and to prevent the pressure of bedclothes, if necessary, by a cradle.

It is a difficult question in regard to articular rheumatism how long the child should be kept in bed. I have long thought that not only is articular rheumatism much less frequent amongst the children of the well-to-do than amongst the poor, but that when the child of well-to-do parents does suffer with rheumatism it is less often accompanied by heart disease than in the children of the poor. If this is so, the explanation probably lies in the greater care taken by the well-to-do to prevent their children being exposed to damp and chill, and to secure rest in bed when the child is affected with rheumatic pains. It seems probable enough that when the rheumatic infection is present in the blood—of course it must travel by the blood—any extra strain thrown upon the heart by exercise may render the heart valves more liable to infection, just as we know that other infective conditions tend to occur in parts subject to stress of work or injury.

On these grounds—speculative, I admit, but supported, I think, by clinical experience—I regard it as a wise precaution to keep the child who has shown definite evidence of articular rheumatism in bed for three or four weeks after all the pains have disappeared, even though there be no suspicion of heart affection.

CHAPTER XXXIV

HEART DISEASE IN CHILDREN: ENDOCARDITIS

The term heart disease covers many conditions: it includes congenital heart disease, malignant or infective endocarditis, suppurative as well as serous pericarditis, and, in children as well as in adults, functional affections of the heart.

In this chapter only simple endocarditis, or acquired valvular disease, will be considered. I shall not attempt to give any general description of endocarditis, such as may be found in any general textbook of medicine, I wish rather to draw attention to some of the special features of this condition as it is seen in childhood.

As regards its frequency, close upon 10 per cent. of the children between two and twelve years of age admitted to the Children's Hospital, Great Ormond Street, are suffering from endocarditis; and any one who has visited the out-patient department of a children's hospital in London can hardly fail to have been struck by the large number of children attending with this form of heart disease. It would be difficult to draw any reliable comparison between hospital statistics and those of private practice, but I will venture to say from my own experience that heart disease is less common, probably much less common, amongst the children of the well-to-do than amongst the poorer classes. If this is so, it suggests that this terrible disease—to my mind one of the most distressing diseases of childhood—is to some extent preventable.

It seems to me probable that one factor in the causation of the rheumatism from which heart disease usually results in childhood, is exposure to cold and damp, and that it is partly on this account that the children of the poorer classes suffer much more with rheumatism in all its manifestations than do the children of the well-to-do. In making this statement I do not, of course, mean to imply that any exposure whatever is capable of producing rheumatism in any form unless the specific infection, diplococcus rheumaticus or whatever it may be, is present; but I do think that in some way exposure to cold and damp favours this infection, just as I see no reason to doubt

that a common 'cold' or 'relaxed throat', albeit almost certainly infective, is often induced by exposure to a cold draught. I suspect also that house infection and child to child infection may play a much larger part in the spread of rheumatism than we have yet realized, and that housing conditions may in this way play a part in determining the frequency of rheumatic affections, including heart disease and chorea, amongst the hospital class.

Most writers have pointed out the heavier incidence of endocarditis on girls than on boys. Out of 255 cases of endocarditis with or without chorea, 140 were girls, 115 were boys; but if only cases of endocarditis without previous or concurrent chorea were included, the reverse would seem to be true; out of 67 consecutive cases of endocarditis in which chorea had never occurred 40 were boys, 27 were girls. It would seem, indeed, as if, while girls more often suffer with rheumatism than boys, the limitation of the rheumatic affection to heart and joints is more common in boys than in girls.

A point of some practical importance is the age at which the liability to acquired endocarditis begins. I say acquired endocarditis to distinguish it from the intra-uterine endocarditis which I think is more often talked about as a cause of congenital heart disease than its frequency would justify, for it is, I think, extremely rare. How soon after birth may a child develop endocarditis? The answer to this question lies in the etiology of simple endocarditis in children; the age-incidence of endocarditis is the age-incidence of rheumatism: let it be laid down as a fundamental rule for guidance in diagnosis that rheumatism under the age of three years is extremely rare, and that under the age of two years it is almost unknown (see p. 472).

The practical bearing of these facts is upon the diagnosis of heart disease in children under two years of age. It is evident that if acquired endocarditis in children is due to rheumatism, the possibility of acquired endocarditis in a child under two years of age can be almost entirely excluded. There is, indeed, one form of endocarditis which does occasionally begin in infancy, namely, malignant endocarditis, but it is excessively rare and when it does occur is associated with an obvious source of infection. I have seen this in one infant at seven months and in another at two years, in both cases associated with empyema; in the absence of any such cause for malignant endocarditis, a bruit in an infant under two years of age is almost certainly either functional or due to congenital heart disease.

I have already implied that the chief cause of endocarditis in children is rheumatism; perhaps it would be rash to say that rheumatism is the only cause of simple endocarditis in childhood, but my own experience very strongly suggests this. Out of 150 cases in which I investigated this point, 142 showed evidence of rheumatism either in articular rheumatism, subcutaneous nodules or chorea. Of the remaining eight cases one was after scarlet fever, two were after supposed influenza, the rest could not be assigned to any particular cause.

Scarlet fever and influenza are generally reputed causes of endocarditis; but when one remembers how often the vague febrile symptoms at the onset of rheumatism in children are mistaken for 'influenza', and how extremely probable it is that the so-called scarlatinal rheumatism is in many cases only ordinary acute rheumatism to which this particular fever predisposes, it seems at least possible that these cases should be included under the head of rheumatic endocarditis. Even the cases in which the history threw no light on the cause of endocarditis cannot be excluded from the category of rheumatism, for the cardiac manifestation may be the first evidence of rheumatism in a child; this view is confirmed in the majority of such cases by the subsequent development of other rheumatic manifestations.

Undoubtedly a pyæmic condition in connexion with scarlet fever or any other disease may give rise to a malignant endocarditis, but so far as simple endocarditis is concerned, I cannot affirm from my own observation that it is ever due in childhood to any other cause than acute rheumatism.

Symptoms

One of the features which is particularly noteworthy in the heart disease of children is the insidiousness of its onset. There may be nothing to draw attention to endocarditis in a child until it is already so advanced that breathlessness or some other symptom results, which leads to examination of the chest and the unexpected discovery of signs of endocarditis. For instance, Walter H., aged 11½ years, was brought for wasting with languor and depression and some 'pain in the chest after food'. Routine examination showed most unexpectedly an enlarged and hypertrophied heart with a loud presystolic bruit at the apex. On further inquiry it was found that some time previously he had had an illness which was called 'influenza' with stiffness of

joints, and that for six months past he had been complaining of 'growing pains'.

A common antecedent of heart disease is this complaint of 'growing pains', which so often represent acute articular rheumatism in children. It is very important to remember that these vague aches and pains may be associated with just as severe endocarditis as the most acute attack of so-called 'rheumatic fever'. Even when the heart disease is already advanced, the symptoms for which the children are brought to the medical man are often very misleading. A not uncommon complaint is wasting. I have several times had children brought to me sclely for this when routine examination has shown that the cause of the wasting was heart disease, and generally inquiry has elicited some history of 'growing pains' or other evidence of rheumatism. Such a case was the following.

Henry S., aged seven years, was brought for wasting; no mention was made of any other symptom, but I noticed that his cheeks were purplish, and on examining the heart found it was considerably enlarged; there were systolic and diastolic bruits at the apex; there were rheumatic nodules on the elbow and on one knee. On further inquiry it was found that the boy had occasionally had some slight pains in his limbs.

Wasting is a much commoner and more marked feature of heart disease in the child than in the adult, as might be expected, for the growth of the body in childhood makes heavy demands upon nutrition, and if the circulation is impeded by heart disease nutrition will suffer more in the growing child than in the adult, who requires only nourishment enough to maintain his completed structure.

Epistaxis is sometimes the symptom for which the child with heart disease is brought; it is not uncommon in cases of severe endocarditis; it was present in 10 out of 150 consecutive cases of endocarditis.

Elizabeth B., aged seven years, was brought for nose-bleeding, which had occurred at intervals of a few weeks for two years; no other symptom was mentioned, but on inquiry it was found that she had had pains in the ankles and in the neck occasionally; and the mother had had rheumatic fever. The child's heart was found to be enlarged, there was a loud systolic murmur at the apex, with reduplication of the second sound.

Tonsillitis is the only complaint in some cases; for instance:

Lilian B., aged 12¼ years, had had repeated attacks of tonsillitis; the mother had no idea that there was anything more amiss, but on being specially asked, said that the child had occasionally complained of pains in her limbs, to which no importance had been attached. The area of cardiac dullness ex-

tended about ¾-inch beyond the right margin of the sternum, and 1 inch beyond the left nipple line, and there was a blowing systolic bruit at the apex conducted into the axilla.

It is the exception for a child with heart disease to be brought for any symptom directly traceable to the heart condition; occasionally, it is true, the child complains of shortness of breath, especially on exertion, or comes for 'palpitation', or for præcordial pain, but much more often the doctor is consulted not for heart symptoms, but for growing pains, or more definite joint rheumatism, or for stiff-neck or chorea.

The only way to avoid overlooking heart disease in children is to make a practice of examining the chest thoroughly in every child, whatever may be the reason for seeking medical advice. I have many times seen serious heart disease overlooked by neglect of this routine. Where there have been 'growing pains' or chorea, or other manifestations of rheumatism, the heart should be examined repeatedly; disastrous heart mischief might be avoided if endocarditis were detected in its earliest stage and the child then kept at rest in bed for two or three months.

There are certain points in the examination of the heart to which I should like to draw attention. Too often the only point to which importance is attached is the presence or absence of bruits, and their character or extent. Let it be recognized that a child may have endocarditis with no bruit at all: this happens sometimes at the earliest stage of the affection; a child with chorea or with swollen rheumatic joints will sometimes show slight but definite increase of cardiac dullness with rapid and perhaps irregular action of the heart, and possibly some thickness of the first sound at the apex; after these changes have been noticed for several days a definite systolic bruit becomes apparent at the apex, and gradually assumes the character of well-marked endocarditis. Occasionally I have noticed in such cases the presence of rheumatic nodules on the elbows or elsewhere several days before the bruit appeared; a fact which confirms the presence of endocarditis, for it is well known that there is an extremely close association between the presence of these nodules and the presence of inflammation of the valves.

Let it be recognized also that a careful estimation of the size of the heart is often just as important as—nay sometimes more important than—the detection of the presence and character of a bruit. In the child's heart dilatation takes place very easily and often very rapidly, it may be the earliest evidence

of heart affection. No doubt this special tendency to dilatation in childhood is partly due to the comparative thinness of the muscular wall, but it is probably due also in part to the fact that especially in children rheumatic inflammation of the heart tends to affect the whole structure of the heart, muscle as well as valve and pericardium.

In examining a child's heart, therefore, it is very necessary to determine and note carefully the 'deep dullness'. I mean by this, not merely the limits of the heart where uncovered by lung, an area which in the healthy child extends laterally usually from the left margin of the sternum barely to the nipple line—this tells us little or nothing about the size of the heart, it only shows how much is not overlapped by lungs—but the actual extent of the heart which in the healthy child extends at the widest part from about $\frac{1}{4}$-inch beyond the right margin of the sternum to the left nipple line, and sometimes nearly $\frac{1}{4}$-inch further out.

The appreciation of the actual size of the heart by percussion requires considerable training of hand and ear. I have thought that mistakes are often made by percussing from heart dullness towards lung resonance instead of vice versa. It is easier to appreciate a very slight change of note when passing from a completely resonant on to a slightly impaired note than when passing from the complete dullness of heart gradually on to the resonant lung; so that if percussion is begun in the axilla and carried gradually towards the heart the slight change of note which indicates the edge of the heart is more readily appreciated.

The limit on the right side is more easily determined than on the left. Some years ago I made experiments on the cadaver, inserting long needles through the limits ascertained by percussion, and I found that I could gauge the right margin so accurately that the needle would sometimes graze the outermost edge of the heart on the right side, whereas on the left there were frequent errors of $\frac{3}{4}$-inch or even more. In advanced heart disease, and where endocarditis is complicated by pericarditis, it is the limit of the right side which requires especially to be watched, for any considerable dilatation of the right side is quickly accompanied by symptoms of failing compensation, puffiness of face and lividity, œdema of legs, shortness of breath, ascites and enlargement of liver; and the timely application of leeches, which may be suggested by percussion before these symptoms are pronounced, is of the greatest value.

In the earlier stages of endocarditis there is often dilatation

of the left side, apparently the result of toxic or inflammatory changes in the heart muscle rather than of the direct mechanical stress to which right side dilatation is often due ; its occurrence is an important indication of the need for prolonged rest in bed.

Hypertrophy of heart is usually of much less importance as far as treatment is concerned than dilatation. It is an indication of the struggle against pressure : hypertrophy means more or less successful effort to resist, dilatation means failure. In the child hypertrophy often becomes very obvious by the bulging of the præcordium which is made possible by the yielding character of the chest wall in early life.

On auscultation there are certain differences between the signs in children and in older patients. In the child aortic bruits are much less common than in adults. In 250 cases of endocarditis I found aortic bruits only in nine, and they are even more rare in the earlier years of childhood than in the four or five years preceding puberty. When aortic bruits develop, it is almost always in cases where mitral disease has already been present for some weeks or months. If the heart is already labouring under the stress of the mitral affection, I am always glad to discover signs of aortic regurgitation, for it is my experience that when regurgitation is sufficient to produce a bruit it in some way relieves the heart, and the child generally lives longer than when mitral bruits alone are present.

I have implied that aortic disease may be present without producing any bruit. Post mortem evidence shows that although aortic disease is so rarely recognizable in the child during life, it is quite commonly present in fatal cases : in twenty-eight out of thirty-six cases of endocarditis the aortic valves were found to be affected, whereas only five of these had shown any aortic bruit during life.

Aortic disease without mitral disease is an extreme rarity, if it occurs at all in childhood. I think I have twice seen children in whom there was a diastolic bruit without any bruit at the apex, but it would be quite unsafe to conclude that in such cases there was no mitral disease ; it is quite possible, I think probable, that if these cases had been examined at the onset of the endocarditis, definite evidence of mitral disease would have been found.

As a rule aortic disease in a child shows itself by systolic and diastolic bruits, which, be it noted, are not only occasionally but usually heard better over the middle part of the sternum, or in the left second and third spaces close to the sternum, than on

the right side; occasionally there is only a diastolic bruit: but so far as my own observation goes, there is never, or almost never, a systolic aortic bruit without a diastolic. This is a point of some diagnostic importance because congenital heart disease occasionally takes the form of aortic stenosis, and in this condition a systolic bruit alone may be heard in the second right space.

The vast majority of cases, however, clinically show only mitral disease; this was so in 241 out of 250 cases. In 117 of these there was only a systolic bruit at the apex, indicating mitral regurgitation; in 124 there was also a diastolic apical bruit, pointing to mitral stenosis.

I am often told that a child has a 'double murmur' at the apex, as if this were sufficient description where systolic and diastolic bruits are present. Now I wish to insist here upon the importance of differentiation between the different kinds of diastolic mitral bruit. It is quite true they all point to mitral obstruction and to this extent have the same significance, but there is valuable guidance, both for prognosis and treatment, to be gained from more accurate timing of the diastolic bruit. Perhaps I shall make my point more clear by describing what is a common history of the heart sounds at the apex in the child who suffers with mitral obstruction. The child comes under observation first with evidence only of mitral regurgitation, a systolic bruit at the apex, the second sound is clear and well defined; soon a change is noticed, the second sound ends off less sharply, there is a 'tailing off' of the second sound; next the tailing becomes an evident reduplication and the second part of the reduplication after a few days has changed into a short puff, much shorter and less marked than the systolic bruit but still evidently a bruit, and this is the middiastolic bruit, or as it is sometimes well described, a 'foo-ti-foo' murmur, in which the first 'foo' representing the systolic bruit is more prolonged than the second 'foo'.

Now if the child be kept in bed at this stage, the order of events may be reversed, and after a few weeks the heart may show only the original systolic bruit, but if the child is allowed to get up, or further endocarditis occurs, the middiastolic changes into a presystolic murmur which appears as a slight explosive sound leading up to the systolic bruit, and is a very different sound from the loud rumbling presystolic bruit which is met with in the adult.

Even now, if proper precaution is taken to ensure thorough

rest in bed, the order of events may be reversed, the presystolic may give place to a middiastolic and this in turn gradually disappear; but if treatment fails, or indiscreet exertion is allowed, the time of the bruit undergoes a further change, and the presystolic bruit is replaced by an early diastolic, which with the systolic produces 'to-and-fro' bruits at the apex. Even when this most unsatisfactory stage is reached, under favourable conditions the bruit will sometimes retrace its steps, but I think very rarely beyond the presystolic stage.

Such then are the differences of time which occur in the bruit of mitral obstruction. I have described them thus at length because I am sure from clinical experience that the recognition of these differences affords valuable guidance in prognosis and treatment; one would feel more hopeful of the child with systolic and middiastolic, than of the child with 'to-and-fro' bruits at the apex, and the child with a presystolic bruit which is still capable of changing into a middiastolic is in a better position than the child whose presystolic bruit remains unalterable.

When a bruit has changed within a few weeks from mid-diastolic to presystolic and thence to early diastolic, it is evident that the mischief is progressing and that prolonged rest is necessary; on the other hand, where the course is in the reverse direction, we may judge from it the progress of repair, and therefrom the time during which rest must be continued.

I have alluded to the difference in character between the child's ill-marked presystolic and the loud 'rumble-and-snap' which is so often heard in the adult. This difference corresponds with the difference in post mortem appearance of the affected mitral valve in the child and in the adult: in the child there is usually little or no appreciable diminution of the mitral orifice, the obstruction is evidently due rather to the slight stiffening of the valve by inflammatory infiltration which prevents its moving freely, whereas in the adult there is often an obvious contraction of the lumen, the so-called 'button-hole mitral'.

The difference probably is one of duration; if the endocarditis is sufficient in degree and the child survives long enough, he may show a presystolic bruit of the adult type during life and similar 'button-hole' change post mortem, but this change requires, I believe, some years for its accomplishment. I found amongst 124 children with some diastolic bruit at the apex only three with the loud rumbling presystolic of adult type. Only in one of them could the duration of the mitral disease be deter-

mined with some degree of probability; in this case it had lasted seven years before the child first came under observation with the adult type of presystolic murmur, but it could not be ascertained when the murmur first assumed this character. In another case, not included in this series, which was under my care with a bruit of this adult character at the age of ten years, the boy had had only the ordinary systolic and middiastolic bruits when I saw him four and a quarter years previously, two years after his first attack of acute rheumatism. This type of bruit has, I think, a favourable significance inasmuch as it points to what one might call a cicatricial condition of the valve, and therefore usually to a quiescent stage of endocarditis; certainly the cases in which it is present are commonly stationary, so far as can be judged from clinical evidence, and remain so for years.

In children, as in adults, the supervention of a musical character in a bruit due to endocarditis is always of sinister omen, it generally points to very severe and generally progressive endocarditis, and the cases which show it are apt to do badly; but this is not always so; I have known one's fears to be falsified, the musical character has disappeared and the child has done well.

And here I should like to draw attention to a particular bruit which has somewhat of a musical character, but is neither of sinister omen nor does it indicate endocarditis of any sort. In my own notebooks I am in the habit of labelling it 'Physiological bruit', but only for want of some better name. It is heard usually just below the level of the nipple, and about halfway between the left margin of the sternum and the vertical nipple line; it is not heard in the axilla nor behind; it is systolic, and is often so small that only a careful observer would detect it; moreover, it is sometimes very variable in audibility, being scarcely noticeable with some beats and easily heard with others; its characteristic feature is a twanging sound, very like that made by twanging a piece of tense string. This bruit is found mostly in children between the ages of two and six years; as a rule they are brought for some slight ailment such as a cough, or some indigestion, and the bruit is discovered only in the course of routine examination; it does not seem to be due to any anæmia, at any rate the majority of children with it show no definite anæmia. It persists sometimes for many months; I have noted it as present in one case for two years. Whatever may be its origin, I think it is clearly functional, that is to say, not due to any organic disease of the heart either congenital or acquired; and I mention it in con-

nexion with endocarditis because I have seen several cases in which it has given rise not only to groundless alarm, but to unnecessary restrictions, so that the child has been treated as an invalid and not allowed to walk about.

Prognosis. I have already indicated some of the grounds upon which prognosis is based. So long as any evidences of active rheumatism persist the child with endocarditis is in danger of further cardiac mischief: the tendency to fresh attacks of rheumatism, chorea, joint affection, and especially heart inflammation, is at its maximum during the middle period of childhood from about five to ten years of age, and therefore the earlier the first attack of rheumatic heart affection occurs, the greater is the probability that it will be aggravated by subsequent attacks of rheumatism. As a rough statement, I think it is correct to say that the chance of survival of a child with endocarditis steadily improves after the age of eleven or twelve years has been reached.

A very important element in prognosis is the occurrence of pericarditis. There is no complication more to be dreaded than this; out of forty-eight cases in which pericarditis supervened, forty proved fatal within a few weeks after the onset of the pericarditis. Even when the child recovers from the acute stage of pericarditis, it is almost always left in a much worse position than before, the myocardium has been inflamed together with the pericardium, and this with the adhesions of the pericardium has aggravated the dilatation so that the heart already crippled by valvular disease is left barely capable of compensation, which sooner or later fails altogether.

The presence of rheumatic nodules is of great practical importance as an index of active rheumatism, and therefore must always make us cautious in prognosis. These nodules are associated specially with severe endocarditis and in this way make the outlook bad; they do not, however, make the prognosis desperate. I have seen many cases with nodules make an excellent recovery, sometimes even to the extent of losing their bruits altogether.

Serious as endocarditis must always be, both in its risk to life and in its tendency, when life is spared for some years, to render the child more or less a permanent invalid, I think it is extremely important that it should not be regarded as a condition which must necessarily go from bad to worse. There is no reasonable doubt that in rheumatic children bruits which eventually disappear altogether are sometimes, perhaps usually,

due to endocarditis. I have known loud bruits, of whose endocarditic origin no one had entertained the least doubt, to disappear after several months. I have even known a bruit which was associated with very severe acute pericarditis in a child of nine years to vanish completely so that there was no vestige of any heart disease to be detected a few months later. I have followed up cases which had been in the Children's Hospital with endocarditis and many rheumatic nodules, and have found them years afterwards doing ordinary work and showing no evidence of the former heart affection. No doubt such happy results are unusual, but I suspect that they are less rare than is often supposed; at any rate they are sufficient to encourage us in taking a hopeful view of the child with endocarditis, especially when this is of recent origin. And even where endocarditis is already advanced and causing severe symptoms, it is astonishing how much improvement will sometimes occur. I remember one child who at the age of seven years had severe mitral regurgitation and great dilatation of the heart, with general œdema, and a dropsical effusion in the pleura, great enlargement of the liver, and jaundice. Her condition looked absolutely hopeless; nevertheless she slowly improved and became so far recovered that she was able to go about and to learn dressmaking and lived nine years after her condition had seemed so desperate.

Treatment

In the treatment of heart disease, there is one remedy which above all others is of supreme importance and supreme value—rest. No change of air, no dieting, no drug, can compare with this; rest is the *sine qua non* in heart disease. And yet how difficult it is to secure! Here is a little child whose mitral disease is still at a stage when two or three months of quiet rest in bed may cause all signs of heart disease to disappear, or, failing this, may secure to the child several years of happy and useful life, and yet the parents will submit to almost anything rather than keep their child in bed. No doubt the child dislikes it, no doubt it is difficult to keep a child amused when he is in bed for weeks, especially if, as is only natural, the child being an invalid has been rather spoiled; but the child's life is at stake, and it is well to impress this fact upon the parents that even though the child may feel and look well, the presence of endocarditis, however early and slight it may appear, means serious danger which may be averted by prolonged rest.

Whilst I am emphasizing the importance of rest in heart disease, I should like to point out also the value of sleep. I suppose the surest way of securing rest, and thereby diminishing to some extent the heart's work, is by promoting sleep; occasionally it may even be necessary in a case of heart disease to obtain sleep by the use of drugs, but this is rare and is generally undesirable, for the drugs themselves may exercise a harmful influence upon the heart. In the slighter cases and in the earliest stage of endocarditis this requirement is usually easily fulfilled; the child can be accustomed to a midday sleep if the room be darkened and kept quiet, and the night sleep should be made as long as possible; but in the more advanced stages, when the child is short of breath and obliged to sit up continually, the lack of sleep becomes a real difficulty. I know of few more pathetic sights in a children's hospital than the child with severe heart disease sitting up at night leaning forward with his head resting upon his arms on the bedboard, ever and again waking with a slight start and seeking to find some comfort in his uncomfortable position, then leaning back wearily on his high-propped pillows, and so with frequent changes but always in the sitting position, getting a broken sleep. The only satisfactory treatment of the sleeplessness in such a case is the improvement of the cardiac condition by such means as I shall mention, but it is a vicious circle which is hard to break; the lack of proper sleep makes the heart worse, the cardiac distress prevents sleep; there are cases in which we are driven to use some hypnotic, it may be 10 grains of trional, or 3 grains of veronal.

And here I venture to note what has sometimes struck me as a flaw in the treatment of a child with severe heart disease: a medicine is ordered to be taken every three or four hours and the child's sleep, difficult enough to obtain perhaps, is disturbed for the administration of drugs which are of less value to the child than unbroken sleep would be.

It is evident that a child cannot always be kept in bed until the signs of heart disease disappear, for they may never disappear, and sometimes the best we can hope for is to secure good compensation so that the child may live on in comfort and in reasonable activity in spite of damaged valves. What then are the special indications for rest in bed? For the sake of clearness I will state categorically what I conceive these indications to be.

1. First and foremost, recent or active endocarditis. It is not always easy to determine when endocarditis is recent; the more

carefully the heart is watched when chorea, or any other rheumatic symptom, is present in a child, the more often will the beginnings of endocarditis be detected. As I have already pointed out, there are often indications of heart affection before any bruit is heard; rapidity and irregularity of heart with signs of dilatation may point to commencing endocarditis. Where a bruit has been present for some time the appearance of fresh bruits,—for instance of aortic bruits where there have hitherto been only mitral bruits, or a change in character of the bruit, so that instead of a blowing sound it becomes musical,—may indicate fresh endocarditis. Wherever there is reason to believe that this inflammatory process in the valves is recent or active we should insist upon rest in bed for several weeks.

2. When rheumatic nodules have recently appeared or are still appearing. The association of rheumatic nodules with endocarditis is so close that it is generally safe to conclude that when fibrin is being deposited in the subcutaneous tissue in the form of nodules, it is also being deposited in the cardiac valves to form the minute vegetations of endocarditis; and, therefore, the recent development of rheumatic nodules should be taken as an indication for rest in bed. I think that as a general rule rest in bed should not be discontinued until all rheumatic nodules have disappeared, however slight the endocarditis appears, but this is not practicable in all cases, for in rare instances these nodules will last a year or even longer; certainly a child should not be allowed to get up until at least a month after the development of a fresh nodule.

3. When the heart with endocarditis shows much dilatation with little or no hypertrophy. This condition almost always means that there is active inflammation going on; sometimes it points to pericarditis as well as endocarditis and in any case it calls for rest in bed.

4. Whenever there are symptoms of failing compensation. Any puffiness or blueness of face, or œdema of legs or enlargement of liver, or other sign pointing to failure of the right side of the heart, necessitates rest; indeed not merely until these urgent symptoms have passed off but for many weeks afterwards; and when once such failure has occurred, and the child has been allowed to get up even after many weeks resting in bed, he must only be allowed to be on his feet for quite a short time, say half an hour daily at first.

In connexion with this subject of rest there is a little point in treatment which is sometimes overlooked: a child is ordered a

medicine or some brandy, to the taste of which he happens to have a great dislike, and each time it is given there is an outburst of crying or perhaps, as I once saw where brandy was being administered, of struggling resistance, which must do harm far beyond any good likely to come from the medicine or brandy. It is generally easy to find some substitute which will avoid these scenes; but if not, the sooner this source of disturbance is stopped the better.

Drugs have little or no effect in checking endocarditis, but they are of the greatest value in combating symptoms. One might have thought that salicylate would have a specific effect upon rheumatic inflammation in the heart as it has in articular rheumatism, but so far as I have been able to judge from my own observation, salicylate exercises no curative effect unless it be of value in preventing fresh endocarditis. I am inclined to give salicylate freely if there is any articular rheumatism with the endocarditis, believing that the disappearance of joint symptoms corresponds with a diminished danger of fresh endocarditis.

If there is very little dilatation of the heart, there is nothing to be gained by cardiac tonics, or stimulants; indeed, it is conceivable that harm may be done by increasing the vigour of the heart-beat, where our object should be to rest the heart as much as possible; but as a rule dilatation very quickly becomes considerable and it is necessary to prevent its increasing. For this purpose I think that if the dilatation is only of moderate degree, and there are no symptoms of failing compensation, nux vomica is preferable to digitalis, for although it has a less powerful influence in strengthening the heart-beat, it does not increase the peripheral resistance as digitalis does. I very often use a combination of Tincture of Nux Vomica ℥iii–v with Bicarbonate of Soda, which, as Dr. Lees has pointed out, may have a value in preventing dilatation.

In more severe degrees of dilatation, where there are any signs of failing compensation, or where, with compensation just and only just maintained, the heart is irregular, the choice of drugs lies, I think, between strophanthus and digitalis; and of these two strophanthus has often proved the more useful in my hands: no doubt this may correspond with the fact that strophanthus does not cause the peripheral vessels to contract so much as digitalis does. A dose of 2 or 3 minims of tincture of strophanthus with 5 or 6 minims of tincture of nux vomica, may be given to a child of ten years every six hours.

Digitalis, in doses of 5–10 minims of the tincture, will succeed

in some of these cases with failing compensation, but its use calls for discrimination. It must be remembered that by increasing the peripheral resistance and augmenting the force of the heart-beat it not only increases the heart's power for work, but it gives it more work to do; and if the muscle be already so inflamed or degenerate that it is incapable of much increase in power, to give digitalis may be to spur a tired horse, and even though there may be slight temporary improvement, the result of the digitalis may be irretrievable cardiac failure.

Dr. D. B. Lees has insisted upon a very important practical point in the use of digitalis for those cases in which there is much dilatation of the right side of the heart—and it is worth remembering that in endocarditis as in pericarditis, it is usually dilatation of the right side of the heart that kills—it is often worse than useless to give digitalis when the right auricle and ventricle are greatly over-distended; the heart stimulated by digitalis makes a great effort to empty its over-distended cavities and exhausts itself in the effort, with the result that the thin-walled right side dilates more than ever; whereas if the tension be relieved even to a very slight degree by the application of three or four leeches over the sternum or liver before the digitalis is given, the drug stimulates successful contraction, and the dilatation is diminished.

There is another condition in which I have several times seen digitalis do harm in children, namely, where with endocarditis there was great hypertrophy of the heart. In these cases the child has complained of sudden spasms of severe pain over the præcordial region and sometimes radiating up to the left shoulder; the attacks have generally occurred only once every few days and have lasted some seconds. I have found the heart beating with an extremely forcible but not rapid beat, and it seemed obvious that the digitalis had induced spasmodic contraction of anginal character; the attacks have stopped forthwith when digitalis was omitted and morphia substituted.

I have also tried theocin sodium acetate, a cardiac stimulant which has been recommended for cases in which there is œdema or ascites; my experience of it has been too small to afford a reliable estimate of its value, but I cannot report any striking success from its use: in a boy of nine years 6 grains of theocin sodium acetate produced a severe headache each time it was given, so that it had to be discontinued. At a later period when this boy was suffering with œdema of the legs, I gave doses of 1 grain, and these produced vomiting, but

he had also vomited other drugs. I have not, however, had these ill results in any other of the few cases in which I have used this drug; and I have known the closely allied preparation theocin, in doses of 1 grain, to increase the flow of urine appreciably.

Diuretin I have also given to children with cardiac dropsy in doses of 5–8 grains, and think it is sometimes of value.

Vomiting is sometimes a troublesome symptom of dilatation of the right side of the heart. In making autopsies on children who have died of endocarditis I have often found the stomach greatly dilated and lying vertically instead of transversely; the dilatation results no doubt from the catarrh produced by chronic venous congestion, and the position from the dragging downwards of the pyloric end by enlargement of the liver. I have wondered sometimes whether this unnatural position of the stomach may not combine with the catarrh to cause the vomiting in such cases. Vomiting may of course be due to the irritant effects of drugs, especially digitalis, and it must be remembered that some children are very apt to vomit anything they dislike, so that it may be the taste of the medicine which is at fault.

The treatment is to relieve as far as possible the venous congestion of the stomach by relieving the over-distension of the right heart, but this is often just what we cannot do; our cardiac drugs, purgation by calomel, even the application of leeches, may fail, and we are driven to treat the symptom rather than its cause. A mixture of bismuthi carb. gr. x with sodium bicarbonate and nux vomica may be given, or counter-irritation may be tried in the form of a hot fomentation or a mustard plaster over the epigastrium.

Opium is very valuable in some cases of heart disease in children, but it is no easy thing to select the particular case for which it is suitable: the features which to my mind suggest that it may be serviceable are signs of much hypertrophy combined with tumultuous action; but it is very important to distinguish the cases in which enlargement of heart is due mainly to dilatation from those in which hypertrophy predominates, for opium may do nothing but harm where there is much dilatation with little hypertrophy. The forms of opium which I have thought most useful are Dover's Powder in doses of gr. ij–iij ter die for a child of eight to ten years, and the liq. morphinæ hydrochloratis 2–3 minims three times a day or every six hours.

A symptom which occurs sometimes in cases of severe mitral disease and which can often be relieved at once by drugs, is sudden attacks of severe dyspnœa lasting a few minutes, sometimes with a feeling of oppression over the præcordium. I have

found these to be relieved very speedily by inhalation of amyl nitrite, 2 minim capsules can be used. Atropin also relieves these attacks: 1 minim may be given by mouth or, if necessary, by hypodermic injection. I say, 'if necessary,' for in my opinion hypodermic medication is always to be avoided if possible in children, whatever the disease may be; for these little people are frightened by little things, and the disturbance and crying consequent on the momentary prick of a hypodermic needle can do nothing but harm when the heart is with difficulty struggling to do its work.

Amongst the measures which are only to be used when other methods have been tried in vain, is puncture of the œdematous legs in several places with a triangular surgical needle. If cardiac tonics and diuretics and aperients have failed to relieve cardiac dropsy, three or four punctures into the subcutaneous tissue of each leg will sometimes produce very striking improvement: not only does the fluid drain away into the absorptive antiseptic wool which should be placed round the leg and changed frequently, but the relief thus given to the pressure upon the veins improves the circulation so much that the heart itself will sometimes recover at least for a time from its extreme dilatation. If the mattresses are arranged so that the child's body is on an inclined plane with the legs sloping downwards, the drainage is assisted; and not only does the œdema of the legs disappear but also that of the scrotum and the fluid in the abdomen, no doubt owing to the general improvement of circulation.

The value of leeching in connexion with digitalis has been mentioned already; I will only add that the advisability of leeching must be determined by the evidence of right heart failure and especially by the signs of dilatation of the right side of the heart, not by the colour of the face or lips. I have several times hesitated to apply leeches because the child's face was profoundly white and the lips anæmic, but none the less leeching has proved of greatest benefit; on the contrary, blueness of lips and face, though often present where leeches are needed, is not necessarily an indication for leeching, it is sometimes present where the heart condition has become chronic and stationary.

Last, but not least, in every case of endocarditis the importance of keeping the bowels acting freely is to be remembered; purgation with 2 or 3 grains of calomel may do much to relieve a labouring heart, and is certainly a valuable adjunct to the treatment of the œdema and dropsy in advanced heart disease.

CHAPTER XXXV

RHEUMATIC PERICARDITIS IN CHILDREN

Rheumatic pericarditis is associated so inseparably with endocarditis in children that it seems a perversion of clinical grouping to consider them separately: my only excuse for doing so is convenience. I have never seen any case of rheumatic pericarditis in childhood in which there was not also endocarditis, and I am informed that in the whole of the post mortem records, extending over forty years at the Children's Hospital, Great Ormond Street, there is no instance of such an occurrence. Pericarditis, however, adds certain symptoms and signs to those already present in endocarditis and moreover requires special treatment, so that there is some reason for giving it special consideration.

Rheumatism is, of course, not the only cause of pericarditis in children, there are at least three other varieties: suppurative pericarditis, usually of pneumococcal origin but sometimes due to other pyogenic micro-organisms, tubercular pericarditis, and the insidious form which occurs in the 'chronic polyarthritis with enlarged glands and spleen'(a form of rheumatoid arthritis) of childhood; but all these varieties differ completely from rheumatic pericarditis in their associations, symptoms, and even in the treatment they require; they have, in fact, just as little in common with the rheumatic form as pulmonary tuberculosis has with lobar pneumonia.

I shall, therefore, confine my remarks here to the pericarditis of rheumatism, which is by far the commonest variety. I suspect that the liability to pericarditis is greatest when the heart is affected by rheumatism at an early age, say under six years, but I have no figures to show what proportion of rheumatic children in each year of life develop this symptom, and if the following table is compared with the age-incidence of rheumatism in general it will be seen that there is a fairly close correspondence which seems to contradict my impression.

Age-incidence of Rheumatic Pericarditis in 53 Cases

Age . . .	0–1	1–2	2–3	3–4	4–5	5–6	6–7	7–8	8–9	9–10	10–11	11–12
No. of cases	0	0	1	2	4	7	12	4	10	7	4	2

The sex-incidence of rheumatic pericarditis is in curious contrast to that of chorea: out of the fifty-three cases tabulated here, thirty-three were boys, twenty were girls; one might suggest as a possible explanation of this disproportion the greater physical activity in boys which may determine the affection of the heart, as the emotional instability of girls seems to determine the incidence of chorea; but this must needs be mere speculation.

The clinical picture of rheumatic pericarditis can be summed up in few words: a child with some rheumatic affection, chorea, endocarditis, or joint inflammation, begins to vomit, is short of breath, and complains of pain over the præcordium. Examination of the heart shows three characteristic symptoms: dilatation, rapid and tumultuous beat, and friction.

The importance of vomiting as an early symptom of pericarditis is worth remembering; whenever a child with any manifestation of rheumatism begins to vomit without apparent cause one should think of the possibility of pericarditis, especially if at the same time there is some rise of temperature.

The respiration is often grunting and sometimes associated with a frequent dry cough, which may be very distressing to the child and may do considerable harm by preventing sleep. The presence of pain is curiously variable, as a rule there is some, but it is not very rare for a child to state that he has no pain whatever when auscultation reveals loud friction over the præcordium. I suppose it is the absence of pain in some cases which leads to pericarditis being overlooked. The pain like that of pleurisy is sometimes referred to the epigastrium and may thus be misleading, especially when, as occasionally happens, there is also diminution or absence of the abdominal respiratory movements so that the condition may simulate, as Dr. Wynter[1] has pointed out, some acute abdominal affection.

There are two other symptoms which to my mind often suggest pericarditis; namely, a white pallor of the face with grey, livid lips, and a marked restlessness; these often appear within a few days after the onset of the pericardial inflammation. Occasionally the constitutional symptoms are severe, there is delirium with sordes on the lips, the breath has a curious sweetish odour, and the temperature reaches 103° or 104°.

The signs of pericarditis are clear enough in some cases, the friction may be so gross as to be palpable, and on auscultation

[1] *Proc. Roy. Soc. Med.*, vol. iv, Med. Sect. p. 40.

it may be obvious all over the præcordium ; but there are many others in which, especially at the onset, the diagnosis has to be made on less conclusive evidence : there may be no friction

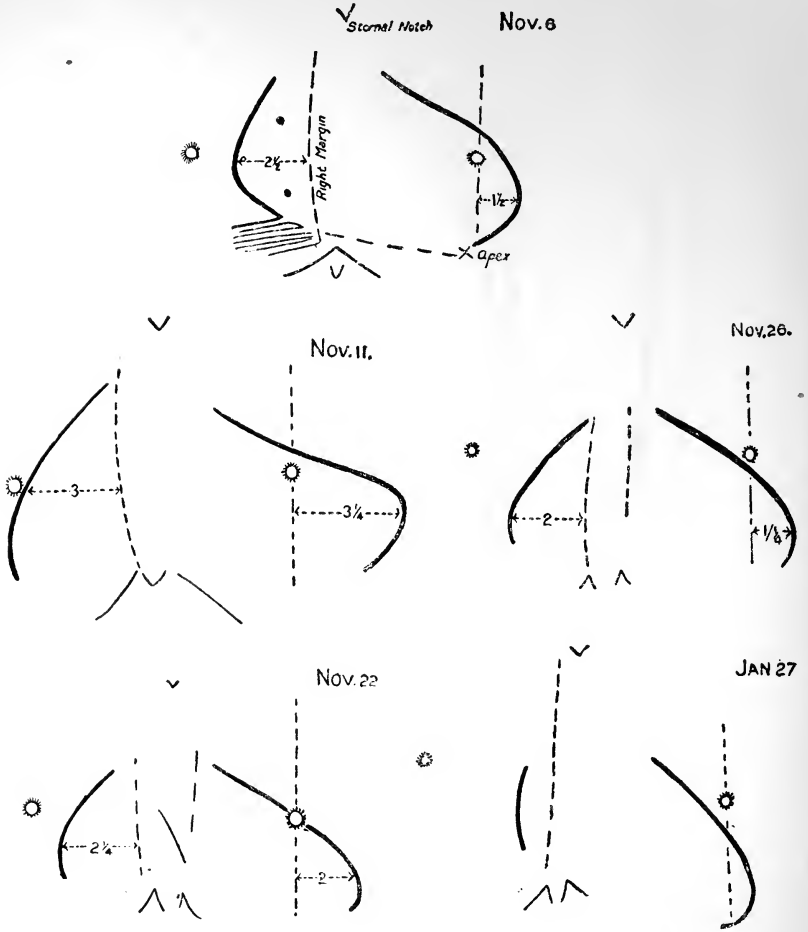

FIG. 35. Tracings of cardiac area, taken during an attack of rheumatic pericarditis in a boy aged nine years. The tracings show the gradual increase during the acute stage and subsequent diminution during convalescence.

whatever, and yet the diagnosis may be almost certain from the character of the heart-beat : it is not only unduly rapid but tumultuous, it seems to plunge and rear like a horse frightened beyond control. Even if friction is present, it is sometimes easy

to overlook, for not only may it be localized to a very small area, perhaps the size of a sixpence, and this at any part of the præcordium; but it may also amount to nothing more than a little scratchiness, which seems rather a modification of the first and second sounds than any additional sound; and it is sometimes so far synchronous with the first and second sounds that it is almost impossible for a time to say whether the abnormality is valvular bruit or pericardial friction. I remember a child with 'to-and-fro' sounds at the base of the heart, who was examined by several physicians. Some thought the sounds due to aortic disease, some to pericarditis; I believe it was only after several days that definite evidence of pericardial inflammation appeared.

Too little attention is often paid to the area of cardiac dullness in these cases. Percussion may give valuable confirmatory evidence, for dilatation occurs very rapidly, especially on the right side of the heart, with pericarditis, and if careful note of the cardiac dullness is kept day by day where pericarditis is suspected, the changes are often very striking, and the rapid extension of the dullness is a point strongly in favour of pericarditis. The tracings reproduced on p. 506 were taken from a child in my ward at King's College Hospital with the help of my then house physician, Dr. A. E. Hay; they show the rapid increase of cardiac area, and its diminution as the pericarditis subsided. Such a record is of practical value in dealing with this affection, for both prognosis and treatment depend to no small extent upon the degree and rapidity of these changes.

It is important to recognize the special tendency to dilatation in pericarditis, for the enormous size of the cardiac dullness is often mistaken for the result of pericardial effusion. The larger my experience becomes of rheumatic heart disease in children, the less ready I am to diagnose pericardial effusion. It is quite certain from post mortem evidence that where the cardiac area is largest, extending perhaps from the right nipple line almost to the middle of the left axilla, there is usually either no fluid at all—there is only a greatly dilated heart with adherent pericardium—or the fluid amounts at most to $1\frac{1}{2}$–2 ounces and evidently, therefore, played only a very minor part in the enlargement which was due chiefly to dilatation.

The following case illustrates some of these points.

Victor C., aged six years, had complained of pains in his limbs on September 22 for the first time, on the 25th his temperature was 103°, and there was some swelling of joints; on September 30 he complained of some pain in the chest, and had a short hacking cough; auscultation showed nothing definitely

abnormal in the heart. On October 2 there was a bruit at the apex and well-marked pericardial friction. He was admitted to hospital on October 4 with a temperature of 101·6°, pulse 140, his face very white, and respiration rapid with frequent short cough. The area of cardiac dullness extended three fingers' breadth to the right of the sternum, three fingers' breadth outside the left nipple line, and upwards to the second rib; there was palpable friction, which was so loud on auscultation as to mask any bruit; there was some epigastric pulsation.

The question of pericardial effusion was raised on account of the large size of the heart area, but, on the ground of its extreme rarity in childhood, it was concluded that the increase of dullness was due to dilatation.

On October 5 the area of cardiac dullness was still large, three and a half fingers' breadth outside the right margin of the sternum and four fingers' breadth outside the left nipple line, respiration was 65, and pulse 160; the boy was delirious, restless and vomiting, his colour was dusky, and the constant cough very distressing. Next day he died; only four days after the first definite symptoms of pericarditis.

Post mortem, the heart was extremely dilated and covered with yellowish fibrin; not more than 2 ounces of serum were present in the pericardium; there was recent endocarditis of the mitral and aortic valves, and some recent pleurisy at the base of the left lung, which was partly collapsed.

This case shows how deadly a disease rheumatism sometimes proves in children: this boy was well and had never had a symptom of rheumatism until September 22 and he was dead within a fortnight. It shows also how quickly endocarditis supervenes; there was no bruit on September 30, and yet by October 6 there was endocarditis of the aortic as well as the mitral valves.

The presence of pleurisy at the left base is a common occurrence with pericarditis, nor is it always the left base only which is affected, both pleuræ are sometimes inflamed or adherent. No doubt in some cases this is secondary to some pneumonia or infarction of the lung, but in others, as in the case mentioned here, there is no pneumonia. Is this pleurisy due to some secondary infection? or is it, as seems much more probable, due to the rheumatic infection? The point is of practical interest because now and then one meets with an unexplained, apparently primary, pleurisy as a clinical phenomenon in a rheumatic child and one is tempted to brush aside one's experience that an unexplained simple pleurisy in a child usually means tubercle, and to label the condition rheumatic pleurisy.

However, this is a digression; the fact remains that rheumatic pericarditis is very commonly associated with pleurisy, and I draw attention to the point because I have sometimes wondered whether the severity of the symptoms which we attribute entirely to the pericardial condition may not be aggravated by pleurisy,

which, especially in young children, is sometimes itself a cause of severe symptoms. I think it is always wise to watch the lungs carefully in a child with pericarditis, for it may be that our fomentations or ice-bags or counter-irritants should be applied to the bases of the lungs no less than to the præcordium.

Prognosis. There is, I think, no graver symptom in a rheumatic child than pericarditis. In the large majority of cases it proves fatal within a few weeks, and even if the child recover from the acute attack, too often he is left with a damaged myocardium and an adherent pericardium, so that he drags out an invalid existence for a few months or perhaps a year or two with a heart which is hypertrophied enough, but only just enough, to prevent still further dilatation; and sooner or later some slight excess of exertion or some fresh endocarditis upsets the balance of compensation and further dilatation ends in death.

Out of fifty-three cases under my own observation only nine recovered to the extent of having lost all acute symptoms when they were seen several weeks or months later. One of these is known to be still living, with a much enlarged heart and mitral bruits, three years after the acute attack of pericarditis. Of the remaining forty-three cases nine died within a fortnight after the onset of pericarditis, four others within a month, five within a few weeks—but the exact time could not be ascertained—seven within about four months; the rest could not be timed exactly but died within a few months, except two cases which died, I believe, one and two years respectively after the attacks.

Amongst all these fifty-three cases there was only one that made a recovery so complete that the most careful examination of the child three months after she left the hospital failed to detect the slightest indication of any heart affection whatever. I have quoted this case in full below.

In the individual case, I think that an important element in prognosis is the degree of dilatation which occurs; perhaps because this is to a large extent an index of the degree of myocarditis present. The condition of the lungs must also be taken into account: if there is evidence of consolidation, as there often is at one or both bases, the strain upon the heart must be increased and the chance of right heart failure greater. No doubt the pleurisy which complicates pericarditis so often must also diminish the child's chance of recovery. I found recent pleurisy or adhesions which appeared to be of the same date as the pericarditis in twenty-two out of thirty-eight cases which were examined post mortem.

Treatment. If rest is important in simple endocarditis it is still more urgently needed where pericarditis is also present, for here it is certain that the myocardium also is acutely inflamed, and it is only astonishing that muscles in such continual activity can recover at all from so acute a process. And herein lies the importance of early diagnosis; the sooner the child is put to rest in bed and kept as free as possible from all disturbance and excitement the better the chance for the inflamed heart muscle; we cannot give it absolute rest, but we can prevent the extra activity of the heart which even the slightest exertion on the part of the child necessitates.

Here I venture to draw attention to a little point—a very little point it may seem, but not to be disregarded on that account if it affects the child's welfare—namely, the clothing of the child who is ill with acute pericarditis. I have little doubt that harm is done by the considerable disturbance of the child which is sometimes involved in undressing the child when for any reason it is necessary to take off the child's bed-dress. If, every time the child's clothes are changed or taken off, the child must sit up and have his arms dragged up over his head to extricate him from a close-fitting jersey vest, or must raise his weight off the bed in the effort to free the lower parts from that plague of the sick child, a 'combination pyjama', the fluttering, rapid heart is goaded to still further excitement and the risk of further dilatation is increased.

The ideal dress is that which can be taken off and put on with the least disturbance of the child. I like for heart cases a flannel vest which opens at the side or buttons above the shoulder, and over this a flannel nightshirt which reaches to the knees and is open from top to bottom and is secured behind by not more than three buttons at most: with a bed-dress like this a child can be uncovered for any purpose with a minimum of disturbance.

It is well also to explain to those who have the nursing of the child that the very least possible amount of washing is to be done: a gentle sponge-over for the face and hands twice a day will be quite sufficient; anything more thorough necessarily entails disturbance of the child, which is much better avoided.

It may seem a contradiction of all this to insist upon the paramount importance of examining the heart carefully every day, or even twice daily; but if the child is properly dressed, so that the chest can be with ease partially uncovered, the examination can be made with extremely little disturbance, and the information

gained as to the degree of dilatation and the need for leeching or for administering digitalis may be of vital importance to the child.

The decision as to the right moment for leeching is often of vital importance. Dilatation occurs with great rapidity; within a couple of days the already considerable enlargement of cardiac dullness may have increased still further by half an inch or more in either direction, the child has become more breathless and livid, and the one thing needful is leeching—I say leeching rather than venesection, which is more disturbing to the child and has no advantage so far as I know. Four or even five leeches may be applied over the sternum and liver. I mention these places particularly, for it is well to avoid putting leeches over the præcordium to the right or left of the sternum, where the wounds would interfere with examination of the heart for the next day or two; the cardiac dullness will still require careful watching, for it may be necessary to leech again.

Directly the leeching is completed digitalis should be given, but usually not before. I think digitalis should be regarded only as a last resource in acute pericarditis; if the dilatation is so much that leeches are required, digitalis also will usually be needful, but where dilatation is less extreme, and the symptoms less urgent, I think digitalis may do actual harm by encouraging forcible contractions of the inflamed cardiac muscle, and also increasing the peripheral resistance.

If it is thought that any such cardiac stimulant should be used I prefer either nux vomica or strophanthus, but I have often thought that at the beginning of pericarditis, when the heart is most tumultuous in its action, a sedative in the form of opium—I generally use Dover's Powder, gr. ii–iv ter die or sextis horis, according to the age and condition of the child—is more useful than any cardiac tonic in quieting the action of the heart, and saving the child from exhaustion; in some cases also the dry, hacking cough is so disturbing to the child that some opiate is necessary, and for this also the Dover's Powder is useful, or, if a fluid medicine is preferred, a mixture of Tinct. Camph. Co. ℥x, Syrup. Scillae ℥x, Syrup. Tolut. ℥x, Aq. ad ℥ij, may be given every three hours.

Dr. D. B. Lees thinks highly of salicylate in the treatment of rheumatic pericarditis. In some of the few cases which have recovered I have used salicylate or aspirin, and the only case which I have ever known to make apparently a complete recovery from pericarditis was treated with ice-bag and aspirin.

Lucy O., aged $10\frac{3}{12}$, had sore throat, followed by swelling and pain in several joints. She was admitted to King's College Hospital with a temperature of 101·8°, swollen joints, and a faint systolic bruit at the apex of the heart, which was only very slightly dilated. Under salicylate, the joint symptoms quickly disappeared, but the fever continued, and two days after admission she looked very ill, her face was grey, and there was some dyspnœa. The heart became very irregular and rapid during the next few days, and the child lay very low in the bed, looking extremely white. but it was not until eleven days later that pericardial friction was heard, and by this time the heart had dilated about $\frac{3}{4}$-inch further to the left, and a few days later it was dilating also to the right. The note became impaired at the base of the left lung, and the breath sounds diminished.

Sodium salicylate gr. x every four hours had been given at first; an ice-bag was kept constantly over the heart, and after a few days aspirin gr. v ter die was substituted for the salicylate, and continued for about five weeks. Pericardial friction continued very marked for ten days, then disappeared, and within three days the heart rate fell from 120 to 88. Rheumatic nodules were found on the right olecranon a few days later.

The systolic murmur at the apex remained, but the area of cardiac dullness diminished steadily; ten weeks after the onset of the pericarditis, the systolic bruit was noted as 'barely audible', and the area of cardiac dullness had become normal. The child left the hospital, but was kept under observation, and a month later the systolic bruit had completely disappeared. When the child was last seen, nearly three months after discharge from hospital, there was not a trace of heart disease to be detected, the area of cardiac dullness was perfectly normal, the sounds were perfectly pure, and the child looked fat and well.

I quote this case at length, because, although it is unique in my experience, it makes one more hopeful of these cases and encourages one to believe that pericarditis may be amenable to therapeutics.

In this case I made use of the ice-bag: how any external application can affect an inflammation of the pericardium I cannot explain, but none the less I am inclined to think that it does. I have used in different cases poultices, hot fomentations, blisters, and ice-bags, and I have thought that the subsidence of the pericarditis was hastened by them, but I have no scientific proof to offer of their value, and in this case, as in so many questions of treatment, one must needs trust to impression—and impressions, as we all know, are too often fallacious. On the whole, if skilled nursing is available, I am in favour of the ice-bag: most children after they have had it a short time like it; the cold seems to ease pain, and I have fancied that it hastened the disappearance of signs of pericarditis.

It requires skilled careful adjustment to keep it in place, and it is advisable to place only a single layer of butter-muslin between it and the chest wall; if thicker material is used the

cold does not reach the chest wall sufficiently. If the child greatly objects to the cold, I use hot fomentations. Blisters to do any good must be repeated in several places, and they are then apt to interfere with the proper examination of the heart, which is, I think, an objection to them.

Lastly, I would caution against the indiscreet use of alcohol in acute pericarditis. Brandy or any other alcoholic stimulant quickens the action of the heart, and it may in this way do harm rather than good: a much more effective cardiac tonic which does not quicken the heart to the same extent and which is more lasting in its effect is strychnine, and although in special urgency, when strychnine is not available, it may be advisable to give brandy, I feel sure that as a general rule alcohol is best avoided in these cases. With any of the drugs which I have mentioned above, the tincture of nux vomica may be an advisable addition, if the heart shows indication of feebleness.

CHAPTER XXXVI

CHOREA

To those who visit the wards of a children's hospital chorea must appear one of the most frequent of diseases in the later half of childhood. At the Hospital for Sick Children, Great Ormond Street, where children up to the age of twelve years are admitted, out of 5,116 admissions to the medical wards 383 were for chorea, that is, 13·3 per cent. of the whole number of children admitted.

But there is an obvious fallacy in these statistics, for children with chorea are specially selected for admission owing to the urgent need for rest in bed, which can with difficulty be secured otherwise. In the children's out-patient department at King's College Hospital, where medical cases up to the age of ten years are seen without selection, I found that amongst 600 consecutive cases between the ages of five and ten years there were eighteen cases of chorea; so that only 3 per cent. of the children brought at this age suffered with this disorder. This is a much lower proportion than my impressions gained at the Children's Hospital, Great Ormond Street, would have led me to suppose; but in the out-patient department at that hospital the physicians see only selected cases, so that the perspective of the actual frequency of diseases is apt to become distorted.

The sex incidence of chorea is interesting. As is well known, girls are much more liable to this affection than boys: my own statistics showed in 150 consecutive cases 108 girls and 42 boys, a proportion of about 2·5 to 1. This difference of incidence does not correspond so closely to the sex incidence of other manifestations of rheumatism as might be expected; in 150 as far as possible consecutive cases of rheumatic arthritis or endocarditis, with no history of previous or concurrent chorea, there were 82 girls and 68 boys. If chorea were dependent upon no other factor beside the invasion of the brain by a special toxin or a specific micro-organism there does not seem to be any particular reason why girls should be so much more often affected by chorea than boys. It seems reasonable to suppose that there may be a psychical factor also in determining its occurrence, and

that the excitability of the cortex induced by the micro-organism or toxin of rheumatism is more likely to produce chorea when the cortex acted upon is that of an unstable excitable child, hence the special liability of the girl.

This view is borne out by the fact noted by some observers that after the age of puberty, when the mental characteristics of the two sexes become more distinctly differentiated, chorea, an infrequent disease at this period, becomes more and more exclusively a disease of females. The occurrence of chorea in adult women chiefly during the specially emotional and unstable time of pregnancy seems to point in the same direction.

This is a matter of some practical importance, for it may have a bearing upon the prevention of chorea in a rheumatic child. One would expect that excitement, mental strain, profound emotion, or shock of any kind, might, by increasing the instability of the child, determine chorea ; and I think there is clinical evidence that this actually happens. How common it is to obtain a history such as this : Miriam G., aged $6\frac{1}{2}$ years, was frightened one day by a soldier who pretended to shoot her ; about two days later she became choreic for the first time. She had two subsequent attacks without any history of special cause, and then a fourth attack at the age of $9\frac{1}{2}$ years, which began a few days after she had been frightened by fear of punishment for accidentally breaking some china.

Sometimes it is the strain or the excitement of school life which determines it. I have notes of cases in which chorea began in children who were working for a school examination ; in one case a girl aged ten years had been greatly excited by acting in a school play just before the chorea began. It is difficult to know how much importance is to be attached to such histories. I have inquired into a considerable number of cases of alleged causation of chorea by fright, and have often obtained what seemed to me clear evidence that slight chorea was present before the fright ; on the other hand, I have met with a sufficient number of cases in which the sequence seemed to be genuine to make me hesitate to disregard these histories altogether. In any case emotion and excitement can only be regarded as contributing factors if we accept the view that chorea is a manifestation of rheumatism.

Relation of chorea to rheumatism. For my own part I may say at once that I regard chorea as just as good evidence of rheumatism as a gumma is of syphilis, but I am not blind to the arguments which may be advanced against

this view. The strongest, indeed to my mind the only one of any weight, is the undoubted occurrence of chorea occasionally in children who show no other evidence of rheumatism either in their personal or in their family history. In a large proportion of these cases, as Dr. F. E. Batten [1] has shown, observation of the child during the next few years after the supposed non-rheumatic chorea gives clear evidence of rheumatism in articular or cardiac manifestations. If rheumatism be, as clinical and bacteriological evidence suggests, an infective disease which is not limited to joints but may affect the heart, the brain, and possibly the lung and the pleura, it would be as reasonable to deny that a tuberculous meningitis was tuberculous because there was no pulmonary tubercle as to deny that the brain affection, chorea, is rheumatic because there is no arthritis ; any one of the various manifestations of rheumatism may be the first to appear or may be the only one to appear, and in this way a child may have chorea months or years before any other symptom of rheumatism, and possibly may never develop any other symptom of the disease.

The occurrence of chorea after scarlet fever, a rare sequence, has been adduced as evidence of its non-rheumatic nature; but it might equally well be made an argument in favour of it, for it is, I think, evident that the joint affection which occurs during convalescence from scarlet fever is in a large proportion of cases ordinary acute rheumatism. I have watched several of these cases subsequently for months or years and observed in them the ordinary series of rheumatic manifestations, heart disease, nodules, chorea, &c. In the same way, where chorea has occurred shortly after scarlet fever, I have seen it associated with the other manifestations of ordinary rheumatism ; for instance, James B., aged seven years, developed scarlet fever, he had never suffered with rheumatism before, but whilst in the fever hospital convalescing from the fever he developed ' post-scarlatinal rheumatism ' during which endocarditis supervened, and about a fortnight after his discharge from the fever hospital chorea began.

There is undoubtedly a post-scarlatinal synovitis which is pyæmic and has no connexion with rheumatism, but I think that apart from this it is evident that scarlet fever in some way predisposes to ordinary acute rheumatism just as measles predisposes to whooping-cough, and hence the connexion, so far as there is any, between scarlet fever and chorea. Pregnancy

[1] *Lancet*, November 5, 1898.

chorea has also suggested a difficulty in accepting the rheumatic origin of chorea; but the clinical observations recorded by Dr. Poynton, as well as his bacteriological investigations with Dr. Gordon, suggest strongly that the chorea of pregnancy is also rheumatic.

My own experience has convinced me that a good deal of the confusion in this matter of the etiology of chorea has arisen either from loose usage of the term chorea to cover any irregularity or jerkiness of movement, or else from imperfect differentiation between chorea and habit-spasm and other functional disturbances. There is no doubt, for instance, that curious irregularities of movement may be imitated by neurotic children, and in this way have arisen epidemics of strange movements such as dancing, beating the arms against the thighs, and so on, but in these cases the movements are more like habit-spasm than chorea, they are more purposive and often more limited to one part of the body than chorea usually is, and they frequently consist only in the repetition of one particular movement.

I suspect that out of such epidemics has arisen the tradition of an 'imitation chorea'. Amongst many hundreds of cases of chorea which have been under my own observation in various institutions I have not seen a single instance of the spread of chorea from one child to another by imitation; and this in spite of the absence of any effort to screen off the choreic child from the observation of others. It seems possible that the shock of seeing a severe chorea might, like any other profound emotion, determine the occurrence of chorea in a rheumatic child, but I have never known this to occur.

The difficulty of discriminating in some cases between chorea and the various functional abnormalities of movement which are met with in children makes it, I think, very necessary to be cautious in drawing inferences from abnormalities of movement in animals. I was for a long time desirous of seeing a typical canine 'chorea', as the close relation of this disorder to distemper has been advanced as an argument against the rheumatic nature of children's chorea. A veterinary expert, who is a well-known authority on diseases of dogs, kindly brought me what he considered to be a typical example of canine chorea: the movements were entirely different in character from human chorea, they were the exact counterpart of a well-marked habit-spasm. I am assured that some cases of the canine disease resemble the children's chorea more closely, but considering how irregularities of movement dependent

upon various functional and organic diseases are mistaken for chorea even in human beings, it seems to me dangerous to lay much stress upon the irregularities of movement in animals as indicating chorea. In the case of dogs, however, I understand that they suffer both with arthritis and endocarditis, so that if chorea does occur in them it may be rheumatic.

Lastly, the extremely close association of chorea with symptoms which are admittedly due to rheumatism bears testimony to its rheumatic nature. I have already shown this by statistics in the previous chapter, which I may summarize here in tabular form.

CHOREA, 380 Cases.

Previous or concurrent articular rheumatism with or without heart disease and nodules	183
No articular rheumatism, but	
Cardiac bruits with rheumatic nodules	8
Rheumatic nodules without cardiac bruits	2
Systolic and diastolic apical bruits	12
Aortic diastolic as well as apical bruits	3
Systolic apical bruits only	48
No evidence of previous rheumatism	124

It may be admitted that a systolic apical bruit alone may not be evidence of endocarditis, and therefore objection might be taken to including this group of cases as evidence of rheumatism; but even if we exclude them, no less than 208 out of the 380 cases, 54·3 per cent., are seen to be rheumatic; there was, however, no reasonable doubt that in a large proportion of cases where there was only a systolic bruit this was due to endocarditis, and probably the error would be smaller if we included them in our estimate of rheumatism than if we omitted them; their inclusion would raise the proportion of cases showing rheumatism to 66·8 per cent.

But even so we shall under-estimate the evidence of rheumatism, for in some of the cases where there was no rheumatic manifestation in the choreic child, there was a history of definite acute rheumatism in the parents or in the brothers or sisters of the child; which must carry some weight in this connexion. Moreover, any calculation based only upon concurrent or previous manifestations of rheumatism in a choreic child must necessarily be incomplete, for, as I have already said, and as Dr. Batten's observations have shown, in a considerable proportion of the cases articular or cardiac rheumatism occurs subsequently to the chorea, which may be the first indication of rheumatism.

Symptoms. The symptoms of chorea in a well-marked case are so familiar to every medical man that I need not give any lengthy

description here. Often the first thing noticed wrong by the mother is that the child is always dropping things ; or the unfortunate child is punished by an ignorant teacher at school because she ' can't do her sums ', I have notes of several cases in which this has happened ; the inability to do lessons is, I think, not always due to the mechanical difficulty from inco-ordination, it seems probable that the power of attention may be actually diminished in chorea. In many cases the child is noticed to be ' always falling about ', as the bruises on the shins too obviously testify, and occasionally the mother has been more struck by the apparent mental change than by anything else and complains that the child has become ' so silly ' lately.

A frequent complaint in the early stage of chorea is headache ; as I have pointed out elsewhere, this is not uncommon in rheumatic children, but its occurrence specially with chorea is interesting in connexion with the observations of Dr. Poynton and Dr. Holmes[1], who found vascular engorgement of the pia mater and underlying cortex, with some small round cell infiltration of the pia-arachnoid in the immediate neighbourhood of the vessels, and here and there thromboses of small vessels both in the pia-arachnoid and in the cortex, together with degenerative changes in the cells of the cortex and the presence of diplococci, resembling those found in other rheumatic lesions, in the exudation about the vessels.

Soon the chorea is obvious enough in the child's fatuous appearance and the irregular jerky gait with a few hesitating steps and then a hurried step or two, or a sudden jerking forwards of one leg. Now and again one or other shoulder is shrugged and a hand is jerkily thrown behind the child's back ; the facial muscles, too, show irregular working, now perhaps a slight pout, now a twitch of one corner of the mouth. The monotonous speech with a tendency to blurt the words out suddenly, the occasional ' clucking ' noise made by the suction of the tongue against the palate as the child opens the mouth, the hesitancy in protruding the tongue at request, and the sudden drawing of it in again, and last but not least the extraordinary emotionalism of the child, so that he bursts out crying or laughing on little or no provocation, complete the characteristic picture of chorea.

In the limbs the inco-ordination is often much more marked on one side than on the other ; in rare instances careful observation fails to detect any chorea on one side, so that the condition is a true hemichorea. If one examines these cases more thoroughly

[1] *Lancet*, October 13, 1906.

it will often be found that the jerky irregularity of movements involves also the muscles of the trunk, so that not only is the back flexed or arched in an irregular purposeless way, but there is also a jerky inco-ordination of the thoracic and abdominal muscles so that respiration is spasmodic and irregular. The knee-jerks also are influenced by the general uncertainty of muscular action; in some cases they are exceedingly difficult to obtain, if indeed they are not actually absent, although the child does not prevent their occurrence by any voluntary or involuntary rigidity. More often, instead of a single jerk, a double or triple jerk occurs ending in complete extension of the knee, which persists for a few seconds.

The heart action is similarly affected in some cases so that its rhythm has a remarkable irregularity. I think there can be little doubt that sometimes this is due to choreic irregularity in the contraction of the heart-muscle, at any rate the sounds convey to one's mind something exactly similar to the choreic movements of the limbs. It must, however, be remembered that one of the earliest indications of endocarditis in cases where no chorea is present is sometimes irregularity of the heart-beat; moreover, rheumatism, like some other infective conditions such as influenza and diphtheria, may very quickly produce changes in the myocardium, the most obvious indication of which may be irregularity of action; we must be cautious, therefore, in attributing cardiac arrhythmia to chorea.

If the most frequent affection of the heart in chorea were merely a disturbance of co-ordination like that in the limbs, chorea would be a disease of little or no gravity; unfortunately the cardiac affection is much more often inflammatory, usually an endocarditis, occasionally a pericarditis, and herein lies the great danger of chorea.

Amongst 250 cases of chorea I noted the presence of cardiac bruits in 155; in 114 of these there was a systolic apical bruit alone which was thought to be due to mitral regurgitation; in 30 the bruit disappeared whilst the patient was still under observation; 38 cases had a diastolic bruit as well as a systolic at the apex; 3 had an aortic diastolic bruit as well as apical bruits. Five out of the 155 cases had also pericarditis, and two others in which the presence of bruits is not mentioned had pericarditis.

I have very little doubt that in other cases beside the thirty mentioned the bruits would have been found to disappear ultimately had the child been under observation for several

months, but it would be a great mistake to suppose that this proved the absence of endocarditis; on the contrary, it is, I think, practically certain that the complete disappearance of bruits due to endocarditis is not very rare (see p. 497). It has often been assumed that where there was only a systolic apical bruit in chorea this was likely to be due to some slight dilatation of the ventricle or to be merely 'hæmic'; without denying that either of these causes may occasionally account for a systolic apical bruit in chorea, I will say that my own clinical and pathological observations lead me to think that a systolic apical bruit in chorea means endocarditis in the very large majority of cases. The subsequent appearance of other bruits, the evidence of cardiac hypertrophy, the association with rheumatic nodules in some cases, the post mortem testimony to the extremely early occurrence of endocarditis in chorea (I found minute recent vegetations on the mitral valve in one case where death occurred, I believe from exhaustion, on the tenth day of a first attack of chorea), all these points taken together with the certainty that bruits due to endocarditis may disappear, make it, I think, probable that a very large proportion of the cases with only an apical systolic bruit have endocarditis.

Prognosis. Chorea involves but very small danger in itself; there are exceedingly rare cases in which it proves fatal by its violence and the consequent inability to sleep and the difficulty in feeding, but these are mostly in adolescents, not in children under the age of twelve years. Almost invariably if death occurs during chorea it is due to endocarditis or pericarditis, and the danger in chorea does not end with the attack, for the history of many fatal cases of heart disease in children shows that the affection dates from a chorea which may have been months or years before. The serious significance of chorea lies almost entirely in its connexion with rheumatism; its dangers are the dangers of rheumatism, and as a manifestation of rheumatism chorea is always a serious disease.

When once a child has had chorea, even though there may have been no other rheumatism with it, it is quite certain, if the views which I have expressed above be correct, that he has the rheumatic taint and may at any time develop acute articular rheumatism or rheumatic heart disease. It is very common for a child who has had chorea to come under treatment subsequently for an attack of acute articular rheumatism, then perhaps another attack of chorea with some endocarditis, and finally perhaps an attack of pericarditis which ends the tale.

These risks are the greater if the chorea occurs early, say at five or six years old, because the tendency to acute rheumatic affections including endocarditis seems to be greater during the middle period of childhood from about five to ten years of age than it is subsequently. In the prognosis of this affection it has also to be remembered that, like the joint pains of rheumatism, chorea shows a marked tendency to recurrence.

As regards the duration of an attack of chorea, in 50 cases in which it was possible to date the onset and cessation fairly accurately, the duration was as follows: 4–6 weeks, 17; 7–9 weeks, 18; 10–12 weeks, 8; 13–16 weeks, 4; 5–6 months, 3. But these figures apply only to those cases—the larger number—in which the disease has definable limits; there are cases of chorea in which after the acute phase of the disease has passed off there remains a slight choreic tendency which pervades the child's movements for many months. In these cases exacerbations occur from time to time, and so in varying degree the chorea drags on sometimes even for a year or two.

The severity of chorea is no criterion of the risk of heart affection nor of the duration of the attack. With regard to the latter point I have often noticed that a violent attack of chorea has ended in much quicker recovery than one in which the movements were altogether slighter.

It is important to realize that the slightest attack of chorea is just as liable to be accompanied by serious heart disease as the most extreme chorea; a fact which corroborates the view that the heart disease is not the result of the chorea but that both are independent manifestations of a more general disease, namely rheumatism.

Diagnosis. The recognition of chorea appears at first sight so simple as to offer little or no possibility of error; but my own experience has satisfied me that even with the most careful observation the diagnosis is sometimes difficult. I have seen mistakes made and have made them myself. There is, however, no excuse for the ignorance or slovenliness which labels any and every jerky irregularity of movement as 'chorea'. It should be our endeavour always to distinguish clearly between the various abnormalities of movement which result from functional nervous disturbance or from some gross disease of the nervous system and the specific rheumatic disease chorea.

I suppose that the commonest mistake is to confuse habit-spasm with chorea, although in most cases the distinction is easy. Habit-spasm, which occurs mostly in the later half of

childhood, differs from chorea chiefly in consisting of the repetition of one movement or one particular series of movements, and in being limited as a rule to one part of the body; for instance, in one case the movement is a frequent blinking, in another it is a shrug of one shoulder, in another a toss of the head and so on; the child may have more than one such spasm at once, and after a few days or weeks he may desist from one form of spasm and acquire another, but always for the time being he adheres to this one particular movement or set of movements, so that the mother can describe to the doctor exactly what movement the child shows, whereas in chorea the movements are hardly the same for two minutes together,—one moment there is a 'clucking' noise, the next moment a twitching of the fingers, then grimaces, and then perhaps a shuffling of the foot; and even when the movements are limited to one side of the body there is complete uncertainty as to the particular irregularity of movement which will occur at any moment.

Habit-spasm unlike chorea has no essential connexion with rheumatism, and therefore where chorea is in question a personal or family history of rheumatism in the child would carry some weight in diagnosis: but it must be remembered that the child who has rheumatism or is of rheumatic parentage is especially apt to be a 'nervous' child, and therefore just the type of child who is liable to contract habit-spasm; moreover, apart from any rheumatism in the child, a family history of acute rheumatism is extremely common in some parts of England: my own statistics showed this in 25 per cent. of children who were brought to hospital for non-rheumatic affections.

A variety of habit-spasm which I have known to be mistaken for chorea is 'Tic Convulsif', in which sudden violent jerks of one or other limb occur, accompanied sometimes with an explosive utterance of some sound which may be merely a grunt or may be an articulate word. One boy about ten years of age who came under my observation with this disorder would be lying quite quietly in bed when suddenly a violent contraction of limb or trunk muscles would almost jerk him out of bed. The explosive character of the movements, and the intervals of many minutes, perhaps quarter of an hour or more, between the explosions, is very unlike chorea. Nevertheless in the case I have just mentioned the disease had been called chorea.

Next to habit-spasm as a cause of confusion in diagnosis I should put that capricious fidgetiness which is natural to

some nervous children: Dr. Warner has described it as microkinesis. When one speaks to the child with this condition he shows in a marked degree what is often to be noticed in the shy child when asked to recite before a stranger: he raises one shoulder, bends his head toward that side, fidgets with his fingers, stands first on one foot then on the other, screws his face up into a troubled smile, in fact manifests a general fidgetiness which is easily mistaken for a slight degree of chorea, as might be imagined when it is remembered that chorea itself has been described as an exaggerated fidgetiness.

This 'microkinesis' is more noticeable at one time than another, especially when the child is out of health from any cause, and of course when the child is made timid or shy by being watched by a stranger; but it never becomes more than might simulate an extremely slight degree of chorea, so that the history of its being habitual to the child, and the fact that it remains always extremely slight, will generally distinguish it from chorea.

Jerky inco-ordination which may more or less closely resemble chorea occurs sometimes as a functional neurosis. I have twice at least seen the presence of worms associated with choreiform movements which subsided rapidly on the expulsion of these parasites.

But in these cases, as in almost every other instance where chorea is simulated, any one who has seen large numbers of children with the true rheumatic chorea can detect a certain almost undefinable difference in the character of the movements; they are, if I may so say, choreoid but not choreic. So too with hysteria, which occasionally shows itself in children as some perversion of movement, there is often a purposiveness about the movements which is lacking in chorea, and the child's general behaviour, his self-consciousness and the evident cessation of the abnormal movements when the child's attention is distracted, may all point to hysteria, the diagnosis of which may be confirmed by the complete cessation of the disorder within a day or two, when the child's environment is changed, for instance, on admission to a hospital, or under the usual treatment for hysteria. The following case was of this kind.

Eleanor F., aged nine years, was brought to hospital on April 28 for sudden attacks of dyspnœa with some discomfort in the epigastrium; these had occurred only during the past few days. The child was stated to have had 'St. Vitus's dance' in May and again in November of the previous year, but had had no other symptoms of rheumatism, nor was there any rheumatism in the family.

She was a spare child and rather pale, with a self-conscious appearance; the tongue was thickly furred, the heart was normal.

A fortnight later she was suddenly seized one evening with what the mother called 'St. Vitus's dance', and on inquiry the mother said that these movements were exactly the same as in the previous attacks of supposed chorea.

The movements seen were at first sight somewhat like chorea, but they differed in their more purposive character; for instance, the child, whilst standing perfectly quiet, would suddenly clasp her hands together as if washing them, then she would screw up her lips as if pouting, and wrinkle her nostrils with a sort of *risus sardonicus*; occasionally the shoulders were shrugged, but quite symmetrically; in walking, her gait was natural when she was not noticeably under observation, but when attention was specially directed to her, there was a sudden *quasi*-purposive shuffle of her feet. All these movements were diminished when the child's attention was diverted from herself by talking to her. She was admitted as an in-patient, and with the administration of valerian the movements had entirely ceased within four days.

A few months later the child was brought to hospital again with the history that she was seized with attacks of immoderate laughter without any reasonable cause; these came on particularly at meal times; they speedily ceased under treatment. In December of the same year the movements of the face and limbs began again and were similar to those seen in the previous attack. The child was admitted again, and the movements had almost entirely ceased in three days, and the child was sent out quite well seven days after admission.

A much more serious disease which may cause choreiform movements is cerebral tumour. Some years ago there was admitted to the Children's Hospital a child about ten years of age with a general lack of co-ordination which was at first unhesitatingly diagnosed as chorea; it was only after the child had been under observation in the ward that the irregularity of movement was noticed to differ vaguely from chorea, and other symptoms occurred suggesting the possibility of cerebral tumour. Examination with the ophthalmoscope showed well-marked optic neuritis, and after a short time the child died, and post mortem examination showed a new growth in the central part of the base of the brain invading the optic thalamus.

There are two other diseases which I must mention, not as bearing any close resemblance to chorea, for indeed as a rule there is no risk of confusing them, but because I have known both of them to be mistaken for chorea: namely, infantile hemiplegia and diphtheritic paralysis. In some cases of infantile hemiplegia, seen a few years after the onset, the prominent symptom is inco-ordination, especially in the movements of the hand, in which also there may be some action of involuntary nature; in a pronounced case these symptoms could hardly be mistaken for chorea, but where they have been very slight

I have known even an observer of large experience to be deceived. The history of its duration and usually sudden onset, and the evidence of some degree of spasticity in arm or leg and probably some difference in size of the limbs on the two sides will serve for diagnosis.

Diphtheritic paralysis only rarely shows as its most striking manifestation an inco-ordination which has simulated chorea: to remember the possibility of this error is to avoid it, for the history and the other symptoms of diphtheritic paralysis are sufficiently obvious.

Treatment. Before discussing the therapeutics of chorea I should like to call attention to certain points in the general treatment of the disease. First I wish to lay great stress upon the importance of complete rest in bed during the first few weeks of the disease. I see children with chorea who are being allowed to get up for two or three hours in the day, as if, provided the child spent the greater part of the twenty-four hours in bed, it would do no harm to allow him to be up for a short time. This, I think, is a mistake. The movements become worse, the duration of the attack is prolonged, and I suspect that the danger of cardiac affection is increased by allowing the child to get up. If there is one part of treatment which is more important than any other in dealing with chorea, it is complete rest in bed, and by this I mean remaining in bed entirely and for two or three weeks at least.

I must qualify these remarks on the value of rest in bed by adding that I believe there comes a time in chorea when not only is complete rest in bed unnecessary, but it may actually retard recovery. It is difficult to fix any precise date at which this period begins, but it is during the late stage of the attack, when the choreic movements after subsiding gradually seem to have reached a stage in which no further improvement occurs and there remains a certain amount of what one might call residual chorea. At this stage if the child is allowed to get up for a few hours daily and to amuse himself with toys and games an opportunity is given for the re-education of co-ordination in the limbs, whereas if the child is kept in bed his co-ordination is much more slowly regained.

This may seem simple and obvious enough when pointed out, but I venture to point it out because I only learnt it myself by comparing the results of the two lines of treatment in a large number of cases.

The question next arises how far it is necessary to isolate the

child. In hospital practice some physicians like to have the choreic child screened off from the other patients, and in private practice I have seen chorea carefully secluded in solitary quietude. In a severe case this is no doubt advisable as much perhaps for the sake of the other children, who are only likely to be shocked by the sensational contortions of a violent chorea, as for the patient himself, who can hardly be otherwise than benefited by absence of all mental stimulus. In the moderate degrees of chorea, any such strict isolation is unnecessary; but even in the slightest attack, freedom from everything which may excite or disturb the child is certainly to be advised.

I turn now to the medicinal treatment. Chorea is one of those diseases in which recovery follows the use of so great a variety of drugs that one is tempted to wonder whether the child might not recover equally well with no drug at all. One thing is certain, that the duration of chorea where no drugs are given varies greatly, it may last three weeks, it may last three months; it is easy therefore to deceive ourselves as to the shortening of the disease where this or that method of treatment is adopted.

Amongst the many remedies which I have used and seen used for chorea, I think there is none more generally valuable than arsenic. At one time at the Children's Hospital, Great Ormond Street, in the three medical wards three different methods of treatment were in vogue: in one the choreic children had rest in bed with no drugs except sometimes cod-liver oil, in another drugs many and various were tried, in the third arsenic was given. I had the opportunity of watching all these methods, and I must confess that there was remarkably little to choose between the results, but so far as there was any difference it seemed to me that arsenic given in large doses had more definitely beneficial effect than any other mode of treatment. By large doses I mean an initial dose of 10 or 12 minims of the liquor arsenicalis for a child of ten years old; this is continued for four days unless the child shows any symptom of intolerance earlier, it is then reduced and the reduction is repeated every third day by 2 minims until the dose has been reduced to 2 minims; the whole course occupies about fourteen days.

But whenever arsenic is used in such large doses a sharp watch must be kept for symptoms of intolerance; usually vomiting is the first indication, but I have seen congestion of the conjunctiva come first, and I have known a very severe gastritis with local tenderness and severe vomiting to follow so quickly upon the first retching as to be almost the first warning of trouble.

The absence of such symptoms should not, I think, tempt us to prolong the large doses beyond the duration mentioned above; for I have known peripheral neuritis to result from too prolonged use of large doses of this drug in chorea.

A much less serious result of the prolonged use of arsenic even in moderate doses (3 or 4 minims) is a brownish discoloration of the skin, especially of the trunk; this I have seen many times in children who had been under treatment for chorea for many weeks; but when the drug is discontinued it slowly disappears. The use of initial large doses, followed by gradual diminution of the dose, has seemed to me to be more effectual than the usual procedure of starting with a small dose of 2 or 3 minims which is gradually increased.

On the ground that if chorea is rheumatic it should be benefited by sodium salicylate, Dr. D. B. Lees has advocated the use of this drug in large doses; certainly if it has any good effect in chorea it can only be from large doses, for experience has long shown that it is useless for this purpose in small ones. Dr. Lees[1] recommends an initial dose of 10 grains of sodium salicylate with 20 grains of bicarbonate of soda every two hours during the day and every four hours during the night for a child of six to ten years. After two or three days the quantities are to be increased to 15 grains of the salicylate and 30 grains of the bicarbonate of soda.

It is very important that the sodium bicarbonate should be added in the proportion of two to one of the salicylate, for given in this excess it prevents the toxic effects of sodium salicylate, which may be very serious when such large doses are given. The symptoms of salicylate poisoning are very like those of diabetic coma, a curious gasping for air, associated with a drowsy condition passing into coma; in some cases there is also much vomiting. Dr. Lees has recorded a fatal case of such poisoning where sodium bicarbonate had not been given.

This method of treatment must be regarded as upon trial at present; like the use of large doses of arsenic, it may be more applicable to hospital practice where the patient is under continuous skilled supervision than to private practice. I once prescribed it to a private patient with very unfortunate results, the child was made very sick and pale and it had to be discontinued within two days. I have several times noticed pallor and a generally 'ill' look produced in children by large doses

[1] *Acute Visceral Inflammations*, p. 291.

of salicylate before the value of sodium bicarbonate as a corrective was known, and one has sometimes wondered how far diminution of choreic movements might be due merely to depression produced by drugs, just as the depression caused by any severe illness, be it acute pericarditis, or pneumonia, or any other acute disease supervening, will sometimes cause the choreic movements to diminish or disappear within a few days.

Aspirin has been recommended by some, but so far as I have observed it has no advantage over sodium salicylate, and has the disadvantage of being only slightly soluble in water.

The newer sedative preparations have been tried for chorea, such as trional, chloretone, and veronal. Trional can be given to a child of about eight years in doses of 5 grains every six hours; this can be continued if necessary for ten or fourteen days. Chloretone, to a child of the same age, can be given in doses of 3 grains every six hours. Any of these drugs can be given as powders in a tablespoonful of milk.

I have seen cases in which recovery seemed to be hastened by the use of trional or chloretone; the latter particularly has undoubtedly a quieting effect upon some cases of chorea, but a dose of 3 grains given every four hours to a child of seven or eight years sometimes produces a drowsiness which makes it necessary to stop the drug; and a further ill effect of chloretone, given too freely, has been irregularity of the heart, with feeble pulse, and grey, almost livid colour. Veronal I have perhaps not used in large enough doses to secure any good from it, and I think it is too dangerous a drug to be of any general use for children. Phenazone I have sometimes thought useful; it may be given in doses of 3 or 4 grains three times a day to a child of ten years; some have used much larger doses, but knowing how powerfully it depresses the heart in some persons, I have always avoided large doses, and even with the smaller doses I always give some form of stimulant, usually sal volatile.

If these drugs are useful there seems to be no reason why bromides should not do good, but I think it must be the experience of most that bromide is very much less valuable in chorea than might be expected. I sometimes use it in combination with small doses of arsenic, and have thought that used in this way it was serviceable.

Chloral is undoubtedly valuable in severe cases, especially where there is sleeplessness, but I confess to a dislike to the large doses which have sometimes been used with the intention and result of keeping the child in a semi-comatose condition for several days; a single dose at night of 10 or 12 grains is as

much as I care to give to a child of eight or ten years, but some physicians give as much as 10 grains every six hours; if the dose is to be repeated two or three times a day, 3–5 grains is a suitable dose.

A drug which I think is undoubtedly useful in some cases is ergot; I have seen rapid improvement follow the use of this drug in violent chorea, but like other drugs its value is by no means constant. I have used it in some cases without the least advantage. I have usually given 30–40 minims of the liquid extract three times a day, but Dr. Eustace Smith [1], who has advocated this drug for chorea, recommends as much as 1 or even 1½ drachms every three or four hours.

Zinc sulphate is probably much less used now than formerly. I have used it in doses of 3 grains increased gradually up to 10 or 12 grains three times a day, unless the child vomits, as often happens before the large dose is reached. But there is extraordinary tolerance of this drug as of arsenic by some children. About forty years ago at the Children's Hospital, Great Ormond Street, zinc sulphate was in common use for chorea, and was increased sometimes up to 25 or even 30 grains three times a day; one girl aged nine years took 34 grains three times a day; these heroic doses do not seem to have been any more successful than more recent methods.

Another drug which has fallen out of use for chorea is antimony. Dr. Charles West [2] regarded it as one of the most valuable remedies for chorea; it was given 'every four hours in what would usually be nauseating doses', presumably about a sixth of a grain; he says that it accomplishes all the good of which it is capable within four or five days, and he considers it useful especially for controlling violent chorea in its early stage.

As an addition to drugs or in place of them, certain other methods of treatment have been found useful. The wet-pack is very valuable in severe cases of chorea. The child is stripped, and then wrapped in a sheet wrung out of warm water at a temperature of about 105° F., surrounded with a blanket, and outside this a mackintosh; in this warm pack the child can remain for half an hour, and if, as often happens, he falls asleep in it, there is no need to disturb him; let the child lie thus for about an hour, then quickly dry him and put on a warmed flannel night-garment, when he will usually fall asleep quickly

[1] *Brit. Med. Journ.*, July 18, 1903.

[2] *Diseases of Infancy and Childhood*, 5th ed., p. 229.

again. This procedure may be repeated on several successive evenings with distinct advantage.

Massage has not seemed to me of much use in chorea; certainly if it disturbs and excites the child as it sometimes does, it is much better omitted. Douching or spraying over the spine with cold water has been used, and some have thought that it quieted the movements; like the massage, it may disturb rather than calm the child, and then is only likely to do harm.

I have dealt with the treatment of chorea as if it were merely a matter of reducing the irregularity of movement; but in the majority of cases there is more to be considered than this. The treatment of the heart disease which accompanies the chorea may be a much more important matter, and the practical questions, How long must the child remain in bed? When may the child return to school? Shall he be sent away for change of climate? may have to be answered rather in view of the cardiac condition than with reference to the chorea. I have already considered rest in bed where there is only chorea, but where there has also been a bruit I believe that strict rest in bed should be enforced for at least two months, and if there is any evidence of active endocarditis, for instance, recent appearance of rheumatic nodules or appearance of fresh bruits, I would continue the rest in bed much longer. I have referred to these points in the chapter on heart disease.

As to the date of returning to school after chorea, when there has been no bruit and the movements have completely disappeared, I think that an interval of three months is very advisable; nothing seems to determine a fresh attack so often as the return to school. I think that where it is practicable home-teaching, at first only for a hour or two daily and then for longer, is much to be preferred. With regard to change of climate, whether country or seaside, I think that it is well to impress upon the parents that the one thing needful for the choreic child is rest, and that no change of air is of any importance in comparison with this. I am often asked by parents whose child has slight chorea, 'Don't you think it would do him good to go to the seaside?' It is usually the greatest mistake to 'send away for a change' the child who has chorea, the excitement of going away, and the unrestricted liberty of play and running about which is almost sure to accompany the change of air, will do nothing but harm. Even when the chorea is almost over and there is left only a very slight degree of residual chorea, I think there is seldom anything to be gained from

a visit to the seaside or country, and if the chorea has left behind it a damaged heart more harm than good is likely to be done by the extra walking and running about to which such a visit usually leads.

It must, however, be remembered that chorea is a manifestation of rheumatism, and whatever the explanation may be I do not think there can be the least doubt that cold and damp conduce to fresh attacks of rheumatism, and on this score it may be advisable under certain circumstances to send the child who has had chorea or any other manifestation of rheumatism away to some climate and soil where warmth and dryness of atmosphere are likely to be found.

CHAPTER XXXVII

CONGENITAL HEART DISEASE

CONGENITAL heart disease is not a common condition; but it comes under medical notice much more commonly in childhood than at any other period, partly because its symptoms attract notice usually in infancy or early childhood and partly because comparatively few survive to adult life. Amongst 2,792 children under ten years of age in the Children's Outpatient Department of King's College Hospital, there were only sixteen cases of congenital heart disease. My figures at the Children's Hospital, Great Ormond Street, give no accurate idea of the frequency of this disease, for the cases seen by the physicians are only selected cases. I find that amongst approximately 18,000 children I saw 64 cases of this affection.

There seems to be no special sex-incidence; out of 100 consecutive cases, 48 were boys, 52 were girls.

I am not concerned here with the pathology of congenital heart disease, I shall deal rather with its clinical aspect, but I may point out that the term congenital heart disease includes various conditions which are not all of similar origin. They fall into three distinct groups, which I shall tabulate here in order of frequency according to post mortem findings in forty-four cases recorded in the registers of the Children's Hospital.

A. Abnormal persistence of openings:

(1) Patent septum ventriculorum	20 cases (only lesion in 4)
(2) Patent ductus arteriosus	5 cases (never alone)
(3) Patent foramen ovale (widely open)	1 case

B. Deformities of valves:

(1) Pulmonary stenosis	15 (only lesion in 6)
(2) Aortic stenosis or fusion of valves	12
(3) Pulmonary atresia	3
(4) Tricuspid stenosis	2

C. Abnormalities of vessels and heart cavities.

(These occur in large variety, but are of little practical importance; some, e.g. single ventricle with double auricle, or single ventricle and single auricle, are not consistent with life for more than a few hours or days, and none can be differentiated during life. In the present series of cases the following were noted):

(1) Abnormally large pulmonary artery (probably pulmonary regurgitation)	1
(2) Abnormally small pulmonary artery	1
(3) Transposition of vessels and single ventricle	1

It will be noticed that patent foramen ovale is one of the rarest of malformations according to these statistics; perhaps it is a little more frequent than they indicate, for in some of the cases which die in early infancy it is difficult to say whether patency of the foramen ovale should be considered abnormal or not, the time of complete closure of the foramen ovale is normally very variable. However this may be, it is quite certain that the normally patent foramen ovale in early infancy does not produce any bruit whatever, and it is probable that only a very wide patency can do so: this is rare in comparison with other malformations, so that there is no ground in post mortem statistics for the assumption often made at the bedside that the bruit of congenital heart disease is likely to be due to patent foramen ovale. I mention this point because I so often hear the remark à propos of a child with congenital heart disease, 'I suppose it is a patent foramen ovale,' as if patency of the foramen ovale usually produced a bruit.

Most of these conditions are abnormalities of development; they can hardly be called 'disease' in the ordinary sense of the word. What the influence may be which perverts development we cannot tell; but some light is thrown upon the problem by the associations of congenital heart disease. It is often only one of several abnormalities in a child; for instance, I have notes of one case in which there was also cleft palate, of another in which the penis was undeveloped and the testicle undescended, another with imperforate anus, another in which there was a double thumb on one hand, another in which there was congenital syndactyly, another with severe congenital talipes, and another with microcephaly. Some such congenital abnormality was associated with the congenital heart disease in 13 per cent. of my cases.

It is evident from such cases that congenital heart disease may be only one expression of a general tendency to faulty development.

Again, congenital heart disease is a recognized associate of Mongolian imbecility, it is found in about 5 per cent. of such cases; and this happens to be one of the forms of developmental imbecility in which there often seems to be an explanation in the comparatively advanced age of the mother at the time of pregnancy, a fact which suggests that congenital heart disease, like Mongolian imbecility, may sometimes be an exhaustion product. Some information might be gained from statistics of the age of the mother at the time of the child's birth. I have

no record of this, but from their place in the family some of my cases suggest the influence of exhaustion, one was a nineteenth pregnancy, another a tenth pregnancy, another a fourteenth, one eighth, and two sixth.

In discussing the etiology of mental deficiency, I shall point out that there is a liability for the reproductive function to be imperfectly established in a first pregnancy, so that the first child suffers with congenital abnormalities, while the later or last child shows them because the reproductive function is failing. I suspect that the same applies to congenital deformities of the heart. I have noted the place in family of fifty cases, no less than sixteen out of the fifty were first-born.

Closely allied to these factors in its *modus operandi* is prolonged illness or any depressing influence which impairs the mother's power of perfect reproduction ; in this way also may be explained the influence of parental syphilis which, without producing any specific manifestation of syphilis in the child, sometimes figures in the history of the child with congenital heart disease. From my own observations I should say that syphilis is quite an exceptional factor ; it appeared to be present only in two out of the series of 100 cases mentioned above, but I think these figures may slightly underestimate its frequency. Recent observations of the Wassermann reaction in cases of congenital heart disease give discordant results : Holt [1] in six cases found no positive reaction ; Findlay and Robertson [2] found a positive reaction in nine out of eleven cases, either in parent, or child, or both. It is quite conceivable that the influence of disease in the mother may be more direct, for, as Dr. Ballantyne has pointed out, imperfect development may be the effect of disease or poison acting upon the embryo, although the same disease or poison acting at a later period of intra-uterine life would produce entirely different effects, resembling more or less closely those which occur after birth. A poison which may in this way act upon the embryo is alcohol ; and I have fancied, but I am unable to produce evidence, that alcoholism in the parents is one cause of congenital heart disease.

As with other congenital malformations, it occasionally happens that more than one child is affected. I had under my care for some time two sisters, who both had congenital heart disease, which in one of them was associated with cleft palate. Another instance was a boy who was said to be one of three who had

[1] *Amer. Journ. Dis. Child.*, Sept. 1913, p. 167.
[2] *Glasgow Med. Journ.*, Dec. 1914.

congenital heart disease out of a family of thirteen. I did not see the other two children, who died in infancy.

I have spoken so far as if congenital heart disease were always due to faulty development, and I think that post mortem evidence shows that this is not far short of the truth, but it must be admitted that there is a very small residue in which there is disease exactly similar to that which occurs after birth, an endocarditis which produces thickening of valves. Dr. Poynton has recorded one case in which the mother had rheumatism during pregnancy and the child had a cardiac bruit at birth, which was found when the child died shortly afterwards to be due to recent endocarditis. Such a case must, I think, be excessively rare.

I had under my care a boy, Jack E., aged ten years, with a rough systolic bruit very localized in the fourth intercostal space close to the left edge of the sternum ; it could only just be heard in the third space and was not heard elsewhere ; he was not cyanosed but occasionally had sudden attacks of panting for breath. The mother had had rheumatism with swollen joints whilst pregnant with this child. At first sight the family history suggests a fœtal endocarditis of rheumatic origin, as in Dr. Poynton's case, but the position of the bruit was, I think, in favour of patent septum ventriculorum, and if this were so, the lesion was presumably developmental and the history of maternal rheumatism a coincidence.

I have notes of an almost exactly similar case in a girl, where in spite of the maternal rheumatism during pregnancy it seemed very improbable that the child's heart condition was due to fœtal endocarditis. I have many times seen crumpled and deformed valves in examining post mortem children with congenital disease ; but the appearances are so unlike what we are familiar with in post-natal endocarditis, that even granting that the effects of valve-inflammation in intra-uterine life may be expected to differ from those of post-natal disease, I cannot but regard it as the merest assumption to say that these have been deformed by fœtal endocarditis. Moreover, if the endocarditis were rheumatic, I think, judging from the usual course of rheumatic endocarditis in young children, it is unlikely that it should all subside before birth, leaving the crumpled, and sometimes, but not always, abnormally thick valves which I have mentioned. It would be, moreover, very remarkable if rheumatic endocarditis occurred in intra-uterine life, and yet were almost unknown, as it is, to attack a child in the first year

of life. In short, it appears to me that, whilst it cannot be denied that an acute endocarditis—possibly of rheumatic origin where infection occurs from a mother with acute rheumatism—does occasionally, but extremely rarely, occur in the fœtus, the majority of abnormalities met with in congenital heart disease are not due to endocarditis.

Symptoms. Amongst the symptoms of congenital heart disease I suppose there is none that is more striking than the cyanosis which characterizes it in some cases, but I want to emphasize the fact that there is *no* cyanosis whatever in the majority of cases of congenital heart disease. The name 'morbus cæruleus' has been responsible for many oversights and errors in diagnosis; far too much stress has been laid upon this cyanosis. I have many times seen cases of congenital heart disease overlooked all together or mistaken for acquired heart disease, because there was no blueness of lips or cheeks, and when I have pointed out that the bruit could only be due to the congenital affection, I have been met with the objection, 'But there is no cyanosis,' as if cyanosis were a necessary symptom. I have kept a record especially of this point in 100 cases; only thirty-four showed cyanosis and in many of these it was only very slight; sixty-six showed no cyanosis.

In this series the diagnosis rested upon clinical evidence, except in a few cases where a post mortem was made; but a series of cases in which congenital malformations were discovered post mortem showed even more clearly that cyanosis is the exception rather than the rule; it was present in thirteen out of forty-four cases.

In some cases cyanosis becomes more obvious as the child grows older. I have even known it to become very marked where there had been none in infancy: e.g. Reginald B. was seen by me at the age of ten months, when he had a loud systolic bruit with a marked systolic thrill over the inner end of the left second costal cartilage. I particularly noted that he had no cyanosis and no clubbing of fingers. I saw him again at the age of $7\frac{1}{2}$ years, when the bruit had become so small that it was only heard on careful auscultation, but the cyanosis had become so marked that his face was a livid leaden colour and his lips a deep blue and his fingers had become clubbed to an extreme degree.

In some cases where there is no cyanosis while the child is quiet, a little exertion such as running about or crying makes the lips distinctly blue; in others without any definite cyanosis

the fingers and toes are always slightly bluish, more rarely a bright scarlet, as I saw in one child with an abnormally large pulmonary artery and patency of the foramen ovale at six years.

In the most severe cyanosis the conjunctivæ are congested, and the tongue, as well as the lips, is a livid leaden colour; in such cases the venous congestion is very conspicuous in the fundus oculi on ophthalmoscopic examination, where the veins are not only extremely full and dark but also show a remarkable tortuosity.

Clubbing of fingers is, generally speaking, proportionate to cyanosis; it is gradually produced by venous stagnation, and therefore is not present in the earliest weeks of life; but it soon becomes noticeable when the cyanosis is pronounced. In an infant aged sixteen days, with intense cyanosis there was no clubbing; in another at the age of ten weeks it was barely noticeable; in another at eight months it was well marked.

Shortness of breath is almost always noticeable, especially on exertion, where there is any cyanosis, but it is surprising how little the shortness of breath sometimes is where the child is extremely blue; many of these 'blue children' seem perfectly comfortable and easy in their respiration so long as they are sitting quietly. Evidently there is some compensatory arrangement; I suspect that this is to be found in the state of the blood. The ingress of blood to the lungs is hindered usually by the cardiac malformation, especially by pulmonary stenosis, and under ordinary conditions of the blood the number of red corpuscles able to reach the lung to take up oxygen at each inspiration would be comparatively small; but if in a given bulk of blood the number of corpuscles were increased, the amount of oxygen which could be taken up would be greater. Some such compensatory purpose I imagine underlies the remarkable polycythæmia which is to be found in these cases with extreme cyanosis.

In a boy, George R., aged four years, with very marked cyanosis of lips and cheeks and clubbing of the fingers and toes, whose heart showed a loud rough systolic bruit all over the præcordium and over the left back, with its maximum intensity in the left third intercostal space, the blood showed 9,280,000 red corpuscles and white corpuscles 9,200 per cubic millimetre, hæmoglobin 160 per cent., and Colour Index 0·9.

The illustration (Fig. 36) shows two capillary tubes which were almost filled with blood (A) from this boy, and (B) from a normal person to serve for comparison. The lower dark part is blood-

clot, the upper part is clear serum. I am indebted to Dr. D'Este Emery for this preparation, which shows in a graphic way the much larger amount of blood-clot due to the excess of red blood corpuscles in the cyanotic case of congenital heart disease.

Interference with the circulation means interference with nutrition, and dating as it does from birth it is hardly surprising that the circulatory difficulty of congenital heart disease is associated with various manifestations of poor nutrition. Most infants with congenital heart disease are puny and small and many remain undersized delicate children as they grow older; no doubt this malnutrition is most marked in the cases in which there is cyanosis, but I wish specially to emphasize the fact that where there is not the leaden blueness nor shortness of breath, nor any other symptom specially indicating affection of the heart, failure to thrive and actual marasmus may nevertheless be the result of congenital heart disease.

I have frequently had infants brought to me solely for wasting or for not thriving, where routine examination of the chest discovered the presence of a loud bruit over the heart. In some of these cases it had been assumed that the cause of the wasting was simply digestive difficulty, and food after food had been tried in a vain endeavour to overcome the difficulty. There are times when the recognition of congenital heart disease as a cause of marasmus may be important, for it may prevent unnecessary weaning and unwise changes from food to food; moreover, it is a satisfaction to parents as well as to doctor to have a definite explanation of the failure to thrive.

FIG. 36. Blood (A) from congenital heart disease with cyanosis, showing clot greater than in (B) normal blood.

In cases with cyanosis there is often a further indication of the interference with nutrition in the curious condition of the skin and hair: the skin looks and feels abnormally thin, transparent, and glossy, the network of veins shows too conspicuously through it, the hair is remarkably soft and fine and silky.

Signs. In determining the presence of congenital heart disease it is all-important to locate carefully the maximum intensity of the bruit. Mistakes are frequently made through lack of care in this respect : a systolic bruit is heard on listening at the apex in a child of perhaps seven or eight years, and we are so accustomed to associate a bruit in this position with mitral disease due to rheumatism, that, without more than a perfunctory auscultation of the rest of the heart, the case is labelled mitral regurgitation. Had we listened more critically we should have found that the bruit was better heard over the inner end of the fourth intercostal space close to the sternum, or perhaps was louder at the inner end of the second or third left space ; a systolic bruit alone, heard best in either of these positions, is almost always either functional or due to congenital heart disease, it is not due to any acquired endocarditis.

This is the most distinguishing feature of the bruit of congenital heart disease, that even though it may be heard well all over the præcordium, it is heard loudest in a position which would be quite unusual for the bruit of rheumatic endocarditis.

The quality of the bruit is not much to be depended upon ; a very loud rasping or roaring bruit at the base, or over the mid-præcordium, usually suggests congenital heart affection, but bruits quite as harsh and loud at the apex may be heard with acquired heart disease. Often the bruit of congenital heart disease is so small that it is only to be heard on careful auscultation.

The presence of a thrill, like that of a bruit, is only characteristic in so far as it occurs in a position which would be unusual in acquired endocarditis : with ordinary rheumatic mitral disease the thrill is usually very limited at the apex; with aortic disease there is rarely any thrill, and when present it is usually felt only in the second or third right space ; therefore when a systolic thrill is felt in the epigastric angle, or over the pulmonary area, it would accord best with congenital affection.

I have spoken of the bruit of congenital heart disease as if it were a necessary sign : some of the most striking cases, so far as facial diagnosis is concerned, show no bruit at all. The child comes with intense cyanosis, the lips almost black, the cheeks deep purple, the tongue livid, and the conjunctival vessels engorged, his fingers are almost spoon-like in their clubbing, and one listens to the heart expecting to hear a loud roaring bruit ; there is no bruit whatever, the heart sounds are absolutely normal. These cases are rare. I met with four in a series of

114 consecutive cases of congenital heart disease: post mortem examination has shown pulmonary atresia, that is, complete obliteration of the pulmonary orifice.

Another congenital abnormality which is sometimes found post mortem where there has been no bruit is fusion of the aortic valves so that there are only two, one of which is much larger than the other; if the fusion is more general, making a cone-shaped opening, there may be a systolic bruit in the aortic area; with this malformation there is no cyanosis, so that if no bruit is produced it is not recognizable during life.

It might be thought that the bruit of congenital heart disease being due to a malformation would undergo no change. I have mentioned incidentally in the case of Reginald B. (p. 537) a change from a loud systolic bruit at ten months old to a very small one at $7\frac{1}{2}$ years: in that same case, a year later, when acute tonsillitis occurred and the boy was acutely ill, I could hear no bruit whatever after careful listening. I did not see the boy again, but it is probable that when the acute illness subsided the bruit might have been heard again.

I have noticed a similarly marked diminution of the bruit just before death, no doubt a result of the enfeeblement of the heart's action. In addition to a bruit the heart sometimes shows definite dilatation, but this is much less marked than in rheumatic endocarditis, and I am inclined to lay some stress upon this disproportion between the loudness and extent of the bruit and the slightness or absence of cardiac enlargement as a point of distinction between congenital and rheumatic heart disease.

Diagnosis. The diagnosis of congenital heart disease from rheumatic endocarditis depends not only on the character of the bruit but sometimes also on the age of the child. It is to be remembered that rheumatic heart disease is practically unknown under the age of two years, and therefore can be excluded in an infant. The only cause for acquired endocarditis in infancy is pyæmia or pneumococcal infection producing an infective endocarditis; this, however, is extremely rare, and when it occurs is associated almost, if not quite, invariably with an obvious primary focus, such as empyema or suppurative epiphysitis. One may say, therefore, that a bruit in infancy usually means congenital heart disease. I say 'usually' because there are cases which suggest that some bruits, even loud ones, heard in infancy, may be of functional origin, and therefore we should be cautious how we conclude that a bruit is due to congenital

heart disease simply because it is heard in an infant. The following cases may illustrate the difficulty of diagnosis.

Henry H. was brought to hospital at the age of eighteen months for wasting and convulsions; there was a loud blowing systolic murmur below the nipple and some added unusual sound of uncertain nature; no doubt was entertained that there was an organic heart affection, and the age clearly pointed to its being congenital. Eighteen months later the boy came again for some cough; he was thin and pale, there was no cyanosis, and I think never had been; there was no trace of any bruit, there was not even any thickness of the heart sounds.

Charles N., the son of a medical man, was found at the age of three months to have a loud systolic bruit over the mid-præcordium; there was no cyanosis, but the boy was not thriving, and he was seen by two physicians of large experience, who concluded he had congenital heart disease. Four years later there was no bruit whatever, the first sound in the pulmonary area was sometimes rather thick, but sometimes even this was hardly noticeable.

Was there a mistake in the diagnosis in these cases? or is it conceivable that the contractions of the several cavities of the heart became so nicely adjusted as to diminish the flow of blood over a patent septum ventriculorum, or through a narrowed pulmonary orifice, so that although the deformity persisted the bruit disappeared? I know not; but certainly the following case suggests that error in diagnosis is at least possible where a loud bruit is present in infancy.

Louisa R., aged ten months, had always been delicate; she had had diarrhœa for three weeks, of which she died. I saw her during life and found a 'whistling systolic bruit heard all over præcordium, loudest just to left of lower end of sternum and in epigastric notch: no thrill'. The lips were pale, and perhaps slightly livid, but otherwise there was no cyanosis. I had diagnosed congenital heart disease without hesitation, and was much surprised when post mortem examination showed no malformation of any sort in the heart, the foramen ovale and the ductus arteriosus were closed, the valves were normal, the only suggestion of an abnormality was a rather deep depression in 'the undefended space' at the upper part of the ventricular septum viewed from the left ventricle, but it did not amount to a sac, and one could hardly be sure that this would account for a bruit.

In later childhood certainly it is necessary to recognize the occurrence of functional bruits, particularly the twanging systolic bruit, which is usually best heard about halfway between the left margin of the sternum and the left nipple line in the fifth space or thereabouts. I have described this elsewhere as the 'physiological bruit'. I know not what its significance may be, but I think it is clearly functional, and it should not be confused with congenital heart disease.

A point which must carry some weight where a bruit is present of doubtful origin is the presence of other congenital malforma-

tions, for as I have already mentioned there is a tendency for congenital heart disease to be associated with other congenital abnormalities.

Is it possible to diagnose the particular malformation or lesion which is present ? I doubt if we can do more than guess in most cases, sometimes not even that, for the varieties of malformation are so many and their combinations so various that physical signs will not suffice for their certain diagnosis ; but none the less there are indications by which in some cases we may form an opinion as to the particular deformity, and, as I have proved by post mortem examination, correctly.

In the first place it is to be remembered that the commonest of all malformations according to post mortem evidence is patent septum ventriculorum, and I think it is highly probable, if we may judge from the rather shaky evidence of clinical experience, that this is even more frequent in comparison with other malformations than post mortem figures show ; in the second place, that pulmonary stenosis is the next in order of frequency, and that it is frequently associated with patent septum ventriculorum. One or other of these deformities, or both, will be present in the large majority of cases in which there are signs of congenital heart disease ; the aortic abnormalities which come next in order of post mortem frequency seldom produce any signs or symptoms during life.

When either pulmonary stenosis or patent septum ventriculorum occurs alone it gives rise to a bruit which may be heard over a large part or the whole of the præcordium, but has its maximum intensity in a well-defined area ; the former in the second left space or over the third left costal cartilage, close to the left edge of the sternum, the latter lower down over the fourth left space or adjoining costal cartilages close to the left edge of the sternum.

There are further points of distinction between these two deformities : pulmonary stenosis is often, but not always, associated with a thrill, its murmur is usually harsh and rough, and it gives rise to more or less cyanosis ; patent septum ventriculorum usually produces a small blowing bruit, no thrill, and does not cause cyanosis.

Occasionally there is good ground for suspecting the combination of both these deformities, where with more or less cyanosis and generalized systolic bruit there are two separate points of maximum intensity in the areas I have mentioned.

There is at least one other malformation which produces

a characteristic bruit, namely, patent ductus arteriosus. The bruit is peculiar in not being strictly systolic or diastolic, it seems to run partly or entirely through both cycles, only being increased during systole ; it is increased also by inspiration. It has usually a well localized point of maximum intensity about 1 inch to the left of the left margin of the sternum in the third left intercostal space.

The only other congenital malformation which can, I think, be surmised from clinical signs is aortic stenosis due to more or less fusion of valves, which produces a rough systolic bruit in the second right space. It is remarkable how extremely rare is congenital affection of the mitral valve, and corresponding therewith is the rarity of an apical bruit in congenital heart disease ; a bruit is often, it is true, to be heard at the apex when it is heard over the rest of the præcordium, but careful auscultation will almost always show that the maximum intensity of the bruit is not at the apex, but at a higher level or much nearer to the left edge of the sternum.

And here I venture again to insist upon the importance of localizing carefully the point of maximum intensity of cardiac murmurs. It may be admitted that there is little practical value to be attached at present to our clinical diagnosis of the different varieties of cardiac malformation. I have given my own observations for what they are worth, they are based on a comparison of clinical with post mortem findings ; but whether we can distinguish the different malformations or not, it is valuable to know that when a bruit has its maximum in such and such a position it is due to congenital heart disease and not to any rheumatic endocarditis, and this much at least we may learn from careful localizing of the bruit.

There are rare instances in which the most careful auscultation will fail to differentiate between congenital malformation and rheumatic endocarditis. In such cases the only criterion may be the age at which the bruit was first noticed, e.g. John M., aged nine years, was brought to me at the Children's Hospital, Great Ormond Street, for enuresis. He had dark auburn hair of the colour which to my mind is always suggestive of rheumatism. On auscultation I found a soft blowing systolic bruit, best heard at the apex just below and under the left nipple ; it could be traced inwards as far as the left margin of the sternum ; it was not heard above the level of the nipple. It might well have passed for the ordinary bruit of mitral regurgitation due to rheumatic endocarditis, but I found that I had seen the boy at King's College Hospital at the age of six weeks, and that the

bruit was present at that time, so that there could be no doubt it was congenital.

Such cases are, in my experience, very exceptional.

Prognosis. Congenital heart disease considerably diminishes a child's chance of long life; it is difficult to ascertain what proportion reach adult years, but it is quite certain that it is a very small one. Of the series of 44 fatal cases mentioned above, 41 were under five years of age, and as these figures were taken from the Children's Hospital, Great Ormond Street, at which children are admitted up to the age of twelve, it seems probable that the majority die under the age of five years. Out of 100 consecutive cases under clinical observation for a longer or shorter time, 77 were under five years of age, 8 were surviving at the age of five to six, 7 at seven years, 1 at eight years, 5 at nine years, 2 at eleven years. One of those who had reached six years and one who had reached seven years, both with deep cyanosis, already showed symptoms of cardiac failure, so that they probably did not live much longer, but the rest passed out of observation without any indication of speedy death.

I think that as a general rule the prognosis is bad in direct proportion to the cyanosis. I am well aware that some cases with most severe cyanosis survive even to adult life, but I think that the tendency to respiratory complications, especially bronchitis and broncho-pneumonia, and to gradual failure of the right side of the heart, is decidedly greater in these cases than in those in which there is little or no cyanosis. The cyanotic cases seem also to be specially prone to certain attacks in infancy, which I am inclined to regard as anginoid. I have seen them in several instances. The usual history is that two or three times a day, usually just after a meal, the infant suddenly becomes intensely distressed, as if in acute pain, fighting for its breath, and turning more and more deeply cyanotic; after a minute or two the attack passes off, leaving the child pale and collapsed.

I do not think that I have ever known an infant to die in one of these attacks, but death has usually occurred within a few weeks or months. I imagine that the accumulation of gas or food in the stomach presses on the diaphragm and so hampers the already labouring right heart, and that a sudden spasmodic effort of the over-distended right side occurs and produces these anginoid symptoms.

Occasionally without any evidence of pain attacks of urgent dyspnœa occur, which may even lead to convulsions.

There is a great tendency to bronchitis and broncho-pneumonia,

and the risk from them is always grave, for they throw additional strain upon the right side of the heart, which is usually already scarcely able to cope with the strain thrown upon it by the abnormal cardiac condition. I have specially noticed how badly cases of congenital heart disease with any degree of cyanosis stand whooping-cough; the cardiac dullness gradually extends further to the right, and I have seen œdema supervene with symptoms very like those of failing compensation in rheumatic heart disease.

With or without cyanosis the infant with congenital heart disease tends to be puny and feeble, and like all such ill-nourished infants falls an easy prey to any acute illness, for instance, to gastro-enteritis. Occasionally without any apparent cause death comes quite suddenly. I have known this to happen where there was no trace of cyanosis; one infant who died thus I had seen on the previous day, when I had specially remarked upon the child's healthy appearance and good colour.

Lastly, I must mention the possibility of endocarditis attacking a congenitally malformed heart. I have known this to happen; it is, of course, a mere coincidence, and only happens when the child is old enough to be liable to rheumatism, or has some infective condition which might cause malignant endocarditis; but the possibility is worth remembering, for when it does occur it may lead to most perplexing physical signs.

Treatment. One might suppose that congenital heart disease was beyond the range of treatment; but the function of the medical man is not merely to prescribe drugs, it is to advise on all that concerns health, and in the case of congenital heart disease there are many points upon which he may be called to advise. In the first place, whilst the parents are to be warned of the precarious tenure of life in these cases, they are also to be encouraged in the hope that with care the child's life may be prolonged for years, perhaps to adult life. The more painstaking and intelligent the care devoted to these children, the longer they are likely to live. I have seen children in well-to-do families live on for years with congenital heart disease which would probably have ended fatally at a much earlier date had the child been born in circumstances where time and money could not be spent so freely on its care.

From infancy upwards these children require most careful protection from cold: an indiscreet outing in the perambulator on a cold, damp, foggy day may be the beginning of the end; exposure to a sharp east wind may start a bronchial catarrh. Even apart from the risk of bronchitis the child with congenital

heart disease stands exposure and chill badly. I have more than once been told that these children turned very blue or went pale when bathed, and I think that when this happens it is wise to forbid bathing and allow only a small part of the body to be sponged each day. The clothing is to be warm: the underclothing should be flannel, and the arms and legs are to be warmly covered.

It is important to secure the most easily digestible diet in infancy, not only on account of the special tendency to malnutrition and marasmus with congenital heart disease, but also because there is a risk of the sudden anginoid attacks, to which I have alluded, if any flatulence accumulates in the stomach. The value of breast-feeding is to be insisted upon, and if the mother's milk fail, it may be advisable to use a wet-nurse; otherwise I have found peptonized milk most useful in these cases.

As the child grows older the question of education will arise, and it is, I think, clear that school is wholly unadvisable; the likelihood of careless exposure to damp and cold and of physical over-exertion is too great to be risked, no child can be trusted to take care of himself in these respects; the child with congenital heart disease should be educated at home.

It is particularly important that the child with congenital heart disease should be trained from earliest years to lie down, and, if possible, sleep, for an hour or so at midday. Such a habit formed in infancy can be continued throughout the greater part of childhood, and is an asset of no small importance when the balance of compensation is being maintained with difficulty by a heart in which congenital deformity throws an unnatural strain upon the weaker portions of the heart muscle.

The slightest 'cold' or 'cough' is never to be disregarded in these children: a little timely care, confinement to one room for a day or two, the use of a counter-irritant to the chest, the early administration of some saline expectorant and of an aperient, may save from the bronchitis which is so dangerous in the congenital heart affection.

But there are times when drugs are useful and necessary. In the anginoid attacks which I have described in infants, inhalation of amyl nitrite should be given; 1 minim capsules can be obtained and the mother may keep these handy for use when required. Carminatives are, I think, of value to prevent these attacks; a mixture of Sodium Bicarb. gr. ij, Tinct. Carminativa (B.P.C.) ℥j, Spirit. Chloroformi ℥j, Glycer. ℥v, Aq. Anethi ad ℥j, immediately after food, may be given three or four times a day.

For the symptoms of failing compensation, which sooner or

later appear in some of the cases with over-distended right heart, the treatment must be upon the lines laid down in the chapter on rheumatic heart disease : leeching, followed by digitalis and nux vomica, will improve the child for a time ; but usually the relief is only temporary, as the cause must necessarily remain unalterable. The time at which such symptoms may supervene is very variable ; in one case it was at twenty months, in another at six years, in another at seven and a half.

CHAPTER XXXVIII

NEPHRITIS IN CHILDREN

NEPHRITIS is not specially a disease of children, but it is not uncommon in childhood, and when it occurs at this age often raises questions of prognosis and sometimes of diagnosis which refer specially to this particular period of life.

There is no age at which nephritis may not occur; amongst 100 cases in children under the age of fourteen years who were under my own observation one was an infant of eight weeks who had first shown symptoms of nephritis at six weeks; another had shown dropsy at the age of five days and died at about nine weeks; post mortem showed acute nephritis. The late Dr. Ashby recorded a case of nephritis in which dropsy appeared on the second day after birth and the infant died with uræmia a few weeks later.

Even chronic interstitial nephritis, rare as it is at any period of childhood, has occurred as early as the fifth year (Guthrie). Four of my 100 cases were probably of this variety.

There seems to be no special sex-incidence, 52 were girls, 48 were boys. Probably the commonest cause of nephritis in children is scarlet fever, but nowadays, owing to the transference of most cases of this disease at an early stage to the fever hospitals, the physician at a general hospital is not likely to see many cases of nephritis due to this cause unless the renal affection persists after the patient is discharged from the fever hospital. My own figures show that such persistence is probably exceptional; amongst the 100 cases of nephritis only 22 were probably due to scarlet fever; most of these scarlatinal cases were seen in the chronic stage. This infrequency of chronic or subacute cases of scarlatinal nephritis amongst the children seen at general hospitals is confirmed by the statistics of the fever hospitals, which show that the majority of cases with nephritis recover entirely from their renal symptoms before leaving the hospital.

Apart from scarlet fever there are many causes for nephritis in children, but my own experience has been that in the majority of these non-scarlatinal cases the disease begins without any assignable cause. Possibly cold and exposure may play some part in determining the onset of this unexplained nephritis, but

it seems more probable that the essential cause is bacterial. This, however, has not been demonstrated, and at present we can only recognize that there is a large group of cases in which the nephritis of childhood is apparently primary and 'idiopathic' in the sense that we do not know its cause.

Almost all the specific fevers are occasionally complicated by nephritis. Henoch states that he saw three cases of nephritis beginning during measles; amongst my cases there were only two in which the close time relation made it probable that the nephritis was due to measles, but in one of these there was also whooping-cough, which is an occasional cause of nephritis. Amongst a very large number of children with whooping-cough I have only once seen nephritis as a complication, unless the case just mentioned was to be regarded as due to this cause.

Varicella seemed to be the cause of nephritis in a girl aged $5\frac{3}{4}$ years who, about a week after the rash appeared, became œdematous with much albuminuria and blood in the urine. Henoch recorded three cases in which the nephritis began eight to fourteen days after varicella.

Mumps I have once known to be followed by nephritis. I have not seen any case after rötheln.

Influenza was followed immediately by acute nephritis in a girl of thirteen years: three other persons in the house had had influenza within a few days before this child contracted it; none of these, however, had nephritis.

I saw with Dr. Gundlach, of Clapton, a girl aged $13\frac{1}{2}$ years, who within four days after the onset of a severe follicular tonsillitis developed a very acute nephritis which proved fatal in a few days; there was nothing to suggest either scarlet fever or diphtheria.

Diphtheria, though it very commonly causes albuminuria, has rarely been a cause of nephritis in my experience, only one out of the 100 cases was due to it.

Twice I have seen lobar pneumonia complicated by acute nephritis (p. 378).

Congenital syphilis is a recognized cause of acute nephritis, especially in early infancy; four cases have been under my care, the youngest was aged six weeks, the oldest a girl of nine years.

There was one case in my series that raised the question whether acute rheumatism may not be an occasional cause of nephritis. It was a girl aged six years, who was under treatment for chorea and was found unexpectedly to have albumen and blood with granular and epithelial casts in the urine; she had

a systolic bruit at the apex, but the cardiac condition was not such as to cause even slight albuminuria. The association may have been a coincidence, but in view of the strong evidence that rheumatism is an infective disease it would be quite in accordance with what we know of other infective conditions that the kidney should sometimes be affected.

Amongst the few cases of malaria in childhood which have come under my notice none have had nephritis, but Moncorvo, of Rio, recorded eleven cases of acute nephritis from this cause in children, and Henoch mentions one in a child of six years.

There are two other causes which though rare are worthy of mention because the likelihood of nephritis constitutes the chief danger of the disease, the one is glandular fever, the other is Henoch's purpura. The former has been in my experience an extremely rare condition, but out of three cases which I took to be glandular fever, with pyrexia, swollen glands in the neck and nothing abnormal in the throat, two developed nephritis about ten days after the onset of the illness. The nephritis which complicates Henoch's purpura has been a very severe hæmorrhagic form in the few cases which I have seen.

Symptoms and diagnosis. In the primary cases of nephritis the onset is usually quite sudden; the child who has been in perfect health up to that time, has a headache one day, is drowsy and perhaps vomits and has some looseness of the bowels, within a few hours the face is noticed to be swollen and the urine is found to contain blood with a large quantity of albumen and some casts. In the nephritis which is secondary to one of the specific fevers the development of symptoms is often more gradual.

Usually in the acute stage there is little risk of overlooking a nephritis: the puffy face and general œdema suggest examination of the urine, which confirms the diagnosis; but this is not always so: there are cases in which the facies of the child does not give the least suggestion of nephritis, although the urine contains a large amount of albumen and blood. There seems to be no very close correspondence between the amount of dropsy and the amount of albumen in the urine. I had under my care a boy of seven years who, for several weeks after an attack of nephritis, became very œdematous when allowed to get up, although the amount of albumen at that time was only a slight trace.

When the acute stage is over and the albumen has diminished to a very small amount there is often nothing in the appearance of the child to suggest the presence of nephritis, and if the child is first seen at this stage the necessity for examination of the

urine is not so apparent and the renal condition may easily be overlooked.

I had under my care for some years a child who at the age of 2½ years had general dropsy with albuminuria, hæmaturia, and casts; at the age of 6 years she was the picture of health, fat and rosy-cheeked. But every now and then she was brought to me for severe headaches, which recurred for a few days, and called for the free use of some saline aperient. I do not think it would have occurred to any one from the child's appearance that these attacks were related to any nephritis, but examination of the urine showed at all times albumen, though sometimes only in very small quantity; and I had no doubt that the headaches were of renal origin.

Even examination of the urine may mislead in this chronic stage, for there are sometimes intervals of hours or days in which the urine is entirely free from albumen.

A boy of 9½ years was under my observation for nearly three years after an attack of acute nephritis. At times his face was slightly puffy, but usually there was nothing in his appearance to suggest nephritis. His urine on some days contained no albumen and, indeed, appeared to be normal in every way, so that a single examination might have led to the conclusion that there was no nephritis; but repeated examination showed that albumen was seldom absent for more than a few days, and at intervals of a few months or weeks it increased to a large amount, blood and casts reappeared in the urine, and the boy quickly became acutely ill with more or less dropsy.

This temporary absence of albuminuria is even more liable to mislead in the rare cases of primary chronic interstitial nephritis in children, for here there may be nothing in the previous history to suggest any kidney affection, and the very rarity of such a condition in children may put us off our guard. In a boy aged eleven years who was brought for severe headache, vomiting, and giddiness, a diagnosis of cerebral tumour seemed reasonable after the urine had been examined and showed no albumen; but repeated examination showed that there was often a very small amount of albumen in the urine, and examination of the eyes showed albuminuric retinitis. In another case, a girl aged 7½ years, the absence of albumen or its extremely small amount, led to a diagnosis of 'anæmia and debility', until with repeated examinations it was found that a small amount of albumen was frequently present, and that in this child also there was albuminuric retinitis. It is but seldom, however, that any assistance in the diagnosis of nephritis can be obtained from examination of the fundus oculi in children. Albuminuric retinitis indeed is generally considered to be an extreme rarity in childhood, although so far as my own experience goes it would seem to be rather the rule than the exception in the primary chronic interstitial variety of nephritis; four such

cases have come under my observation and all four showed albuminuric retinitis. In one other case, a boy of 9½ years, who died five months after the onset of what appeared to be an acute (non-scarlatinal) nephritis, there was retinal hæmorrhage and some swelling of the retina; autopsy showed that the nephritis was of the 'large white kidney' variety.

Whilst puffiness of the face in a child should always suggest the advisability of testing the urine, it is a common symptom of other conditions beside nephritis. In chronic intestinal indigestion, the disorder which Dr. Eustace Smith described as 'mucous disease', there is often noticeable puffiness of the lower eyelids; a similar appearance is seen with cyclic albuminuria and a more general puffiness of the face with heart disease and whooping-cough.

The puffy face of whooping-cough is generally explained at once by the history of paroxysmal cough; it is only where it is associated with hæmaturia or albuminuria that doubt may arise.

A girl of 4½ years, after coughing much for ten days, began to whoop violently; four days later when I saw her the eyelids were puffy and I was told that the urine had been bright red for two days; but the colour varied much at each micturition—sometimes the urine was almost clear. I examined the urine and found it dark reddish-brown, sp. gr. 1010, with some oxalates and other crystals, some small round granular cells, and a few doubtful casts, some granular and some hyaline with blood-corpuscles; two days later there was no blood in the urine and no albumen; it was, in fact, perfectly normal. There was no œdema, except in the face, at any time.

Here the sudden onset and rapidly transient character of the urinary disturbance together with its variation from hour to hour, showed that it was due merely to a passive venous congestion, differing in no way from the venous engorgement which gives rise to the œdema in the eyelids or to epistaxis when the paroxysms of whooping are severe. And one may suppose that just as casts may be present in the urine of patients in whom heart disease and failing compensation are causing venous engorgement of the kidney, so also in whooping-cough casts may be present in the urine when the congestion is sufficiently frequent or severe.

I have twice seen in infants a few months old a general dropsy which has appeared without apparent cause and exactly resembled the dropsy of acute nephritis, but the urine therewith showed no albumen, and its only peculiarity was an extremely low specific gravity (1001–1004), an almost complete absence of colour and a scarcely appreciable percentage of urea; the kidneys in a case which I examined post mortem showed no

evidence of nephritis, but were rather unusually small for the age. This condition is of course distinct from the œdema which is sometimes seen in puny infants just after birth, the so-called 'œdema neonatorum', and is probably also entirely different in its etiology from the much less uncommon cases of more or less general œdema in late infancy or early childhood, where there is advanced marasmus dependent upon some chronic gastro-intestinal disorder. The urine in these cases also is free from albumen.

Occasionally older children develop a general œdema as if they had acute nephritis when the urine proves to be entirely free from albumen.

A boy aged eleven years was brought to me with a history that for one week his face had been puffy, and for four days his legs had been swollen. He showed no physical signs except the general œdema and evidence of some fluid in the abdomen. He looked exactly like a case of acute nephritis, and I expected to find the urine loaded with albumen: it contained not a trace of albumen, and except that the boy was passing an abnormally small amount for his age, there were no urinary symptoms; his stools were very offensive, but otherwise there was nothing beyond the dropsy to be found.

Of some value, I think, as confirmatory evidence of nephritis in children, is the character of the aortic second sound which by its accentuation may indicate increased arterial resistance quite early in the disease, although the pulse tension is not appreciably raised—and, indeed, except in the rare cases of chronic interstitial nephritis, it is but seldom that an increase of pulse tension is sufficiently definite in children to be of any practical value in diagnosis.

In the chronic cases, also, signs of cardiac hypertrophy are usually present, and in the absence of any evidence of valvular disease may confirm the diagnosis of nephritis. Amongst my notes I find records of enlarged cardiac dullness in many of the subacute cases in which œdema was still present, and in some of these the enlargement was apparently the result of hypertrophy; but the frequency of cardiac dilatation in the nephritis of children must also be remembered and no doubt accounts not only for the increased cardiac area in some cases, but also for the presence of a systolic apical bruit which was found in five out of my 100 cases.

Prognosis. Passing now to the consideration of prognosis, I would emphasize first the need for caution in regarding any child who has shown symptoms of nephritis—and I am referring now specially to non-scarlatinal nephritis—as cured. When the œdema has all gone, and the albumen has diminished to a bare

trace, and then been absent altogether for a few days, one is tempted to pronounce the child 'cured'; so he may be of his acute symptoms but not necessarily, perhaps not even usually, of his disease. If these children are carefully watched it will be found in many cases that from time to time a cloud of albumen reappears in the urine and that this cloud, although on most days it may be only just perceptible with most careful testing, increases occasionally to a considerable quantity, perhaps as the result of some slight exposure to cold. To say that the nephritis is cured in such a case is surely a mistake, however well the child may look, and however infinitesimal the trace of albumen may be; certainly not only may the albumen increase from time to time, but at any time the child may show a return of all the symptoms of an acute nephritis. I have already referred to children who have been under my care as out-patients three years or more with this latent nephritis, if I may so call it; and looking back over my series of cases, I am inclined to think that this is no uncommon result of an acute nephritis in children, if indeed it be not the minority of cases which ever make a complete recovery.

But here I would draw a sharp distinction between scarlatinal and non-scarlatinal nephritis in children. I mentioned above that only twenty-two out of my hundred consecutive cases of nephritis in children were scarlatinal in origin, and when this small proportion is compared with the well-known fact that the commonest cause of nephritis in children is scarlet fever, it is obvious that of the large number of children with scarlatinal nephritis in the fever hospitals the majority must either die or be cured, for only a very small number reappear with albuminuria subsequently at other hospitals. Happily statistics show that a very large majority of the cases of scarlatinal nephritis are entirely cured before leaving the fever hospital, and it would seem that the prognosis of scarlatinal nephritis in children, apart from the risks during the acute stage, is altogether more favourable than that of the non-scarlatinal affection; it is indeed quite exceptional to meet with a prolonged albuminuria lasting for years as a result of the scarlatinal form, whereas such a course is quite common in the non-scarlatinal cases.

This will be seen most clearly from actual figures, and for the purpose of comparison I shall avail myself of statistics taken from a fever hospital by Dr. Goodall; these, it should be stated, include adults as well as children, whereas my own much smaller figures include children only.

Out of 281 cases of scarlatinal nephritis 54 per cent. had completely lost their albuminuria within four weeks after the onset of the renal affection ; not more than 24 per cent., probably only 19 per cent., showed albumen persisting more than six weeks. Contrast with these statistics my own figures of non-scarlatinal nephritis as shown in the following table (which includes fatal cases and the four cases of chronic interstitial nephritis mentioned above).

Duration of Nephritis.	*Number of Cases.*
17 days	1 (fatal)
3–4 weeks	4 (two fatal)
1–3 months	11 (one fatal)
3–6 months	14 (four fatal)
6–12 months	7 (two fatal)
1–2 years	6 (two fatal)
2–3 years	4
3–4 years	1
4–5 years	2
Probably several years	1 (fatal)
Total number of cases	51

The duration of the albuminuria stated in this table refers to the period already reached when the child died or passed out of observation; in many cases, no doubt, it lasted very much longer. It was only in the fatal cases and in two or three which recovered that the total duration of albuminuria could be ascertained.

It is evident even from these small figures that anything comparable to the rapid recovery in three or four weeks which occurs in about half the cases of scarlatinal nephritis is quite exceptional in the non-scarlatinal nephritis of children; indeed, amongst my own series of non-scarlatinal cases, 37 out of 51, that is, fully 73 per cent., lasted more than three months. Whilst, however, the non-scarlatinal cases of nephritis in childhood tend to run a much more prolonged course, and in this respect have a worse prognosis than those of scarlatinal origin, they have at any rate the advantage that a fatal ending rarely occurs as speedily as in scarlatinal nephritis. To quote the authority already mentioned, in the 281 cases of scarlatinal nephritis there were 13 deaths, and no less than 7 of these occurred within fourteen days after the onset. In my 51 cases of non-scarlatinal nephritis there were 13 deaths ; none of these occurred earlier than the seventeenth day, and only 3 within four weeks after the onset, as will be seen from the following table :

Age of Child at death.	*Duration of Nephritis.*
1¼ years	17 days
2¼ months	3 weeks
1¾ years	4 weeks
6¼ years	3 months
3 years	4 months
3¼ years	4 months
9½ years	5 months
4¼ years	6 months
2¾ years	7 months
9¾ years	10 months
7 years	13 months
12 years	20 months
9 years	at least 2 years

Whilst, however, the fatal ending would seem to be much longer delayed in the non-scarlatinal nephritis of children than in the scarlatinal affection, the heavy proportion of deaths in this series, 13 out of 51, is noteworthy as contrasting with the much lower death rate in the fever hospital cases, 13 out of 281.

Moreover, most of the children who passed out of my observation still had albuminuria, and as experience shows were likely therefore to have exacerbations of nephritis sooner or later; it would seem, in fact, that the ultimate prognosis in cases of non-scarlatinal nephritis in children is very much less hopeful than in the scarlatinal cases. Undoubtedly a small proportion recover completely, and a much larger number lose all symptoms of nephritis except the albuminuria; but these latter cases, although, as I have said, they may live in fair health for months or years, live continually on the edge of a precipice: they have damaged kidneys and at any time a fresh outbreak of renal inflammation may occur and prove fatal. The approach of danger in such cases is almost always indicated by the reappearance of œdema, and at the same time the urine shows a great increase of albumen and perhaps blood and casts. But a child may have several relapses of this description lasting days or weeks and yet survive for several months or years, but in each relapse there is danger to life and the sources of danger are several.

In at least seven out of the thirteen fatal cases mentioned above, there were symptoms of uræmia shortly before death (intractable vomiting, convulsions, delirium or coma), but in some of these children there were other complications which no doubt contributed to the fatal result. Of extremely sinister significance is the erysipelatoid rash which is sometimes seen in cases with much general œdema. The rash is generally associated with much local tenderness and some pyrexia; it was

seen in four out of thirteen cases, and in each case was followed by death within two or three weeks at most. Any considerable rise of temperature is also of unfavourable omen, even if no obvious cause for it can be found; in the majority of the fatal cases pyrexia (102° or 103°) occurs for several days before death. In some of the children under my observation good reason for this temperature was found post mortem in a very acute peritonitis, five out of the thirteen cases showed seropus or serum with flakes of lymph floating in it in the peritoneal cavity.

In none of these cases had the peritonitis been suspected before death, but the pyrexia which accompanied it and the presence in one or two cases of considerable tenderness over the abdomen might have suggested this complication, and such a combination of symptoms in a child with much œdema and a large quantity of albumen in the urine—the conditions under which this complication seems most likely to occur—would make the prognosis extremely bad.

A very marked diminution in the average quantity of urine passed daily is sometimes the precursor of a fatal ending, but it is only when the quantity passed is really very small for the age that any prognostic significance can be attached to it. It may not be out of place here to mention that the average quantity of urine passed daily by a healthy child is much lower than is often supposed, and curious errors have arisen through ignorance of the normal average (see p. 562).

In the very rare cases of chronic interstitial nephritis to which I have referred the prognosis is probably invariably bad. The question 'How long?' however, is not easy to answer, for the disease may be so insidious that although the child is ailing no medical advice is sought until the onset of the chronic uræmic symptoms which are usual in these cases; and even then, although as in one of the four children mentioned here death may occur in seven weeks, a fatal result may be postponed for several months (in one of these cases for eighteen months). Probably one would be right in saying that very few of these children with chronic interstitial nephritis live more than two years after the first symptoms appear. In giving prognosis in any case of chronic interstitial nephritis in a child it is well to remember the possibility of cerebral hæmorrhage, which has been the cause of death in some recorded cases. One of the four cases included in my series died suddenly with no premonitory increase of the chronic uræmic symptoms which she had shown for several months; the immediate cause of death was not determined.

The termination of these cases, however, is most often by uræmia with convulsions and coma.

Lastly, in any case of prolonged nephritis in a child the prognosis will depend largely on the possibilities of environment, the care which is likely to be taken with the clothing to see that the child is warmly clad, the avoidance of exposure to damp and chill, and in some cases on the possibility of residence in a suitable climate.

Treatment. The treatment of nephritis in children is upon the same lines as in adults. In the acute stage the most important part of treatment is to promote sweating and watery evacuation of the bowels. No measure has seemed to me more generally useful than hot-packs, and if directly the child is put into the pack 2 or 3 ounces of milk diluted with about 6 ounces of hot water be given with 20–60 drops of brandy, or perhaps still better with the same quantity of rum or gin, according to the age, a diaphoretic effect is usually obtained, which can be prolonged when the child is taken out of the pack after about an hour by wrapping him in two or three layers of dry hot blankets with hot-water bottles beside him.

In severe cases where uræmic symptoms are present in the acute stage of the disease it may be necessary to repeat the packs several times a day to ensure a sufficient action of the skin. I have used hot-packs continuously for eight or ten hours, changing them every hour where there was difficulty in inducing perspiration.

If suitable apparatus is available a hot-air bath makes some children sweat more than the hot-pack. Where electric light is available, a radiant heat bath can be improvised by fixing electric lamps inside a bed-cradle which is placed over the child and covered with blankets. Care must be taken that neither the child nor the bed-clothes are burnt by the lamps. Whatever method is used it is necessary to keep a sharp watch upon the child while he is in the pack, for some children show feebleness of pulse from these applications of heat.

Diaphoretic drugs often fail altogether in severe cases unless repeated very frequently. Pot. Nitrat. gr. v, Spirit. Ætheris Nitrosi ♏v, Spirit. Juniperi ♏v, Syrup. ℥j, Infus. Scoparii ad ℥ss, may be given every two hours until six or eight doses have been given to a child of eight years; and by combining such drug treatment with the use of hot-packs or the hot-air bath diaphoresis may be increased.

In the less severe cases the hot-pack once daily or on alternate days will be sufficient, and in the interval hot poultices or hot fomentations may be applied over the lumbar region. I have several times tried dry-cupping, which can be done with very

little disturbance even to the youngest child; I have thought that it was of some value.

A dose of pulv. jalapæ co. gr. xxx or gr. xl for a child of eight years can be given every alternate night for the first week of the disease and then an early morning dose of Mag. Sulph. gr. xx, Sod. Sulph. gr. x, Syrup ℧xxx, Aq. Menth. Pip. ad ℥ij, may be substituted; the dose of the sulphates to be regulated by the effect produced. It is extremely important to keep the bowels active, and when there is much œdema, good may be done by keeping the stools loose and diarrhœal for a few days. The saline aperients sometimes produce sickness, after they have been used for some weeks, and it is necessary to find some more agreeable aperient. 'Phenolphthalein' in doses of gr. j–iij, given in the early morning on an empty stomach, may be successful, but I have sometimes found the most satisfactory result from 'Tamar Indien', which children take very readily. ½–1 Tamar is given at night.

Throughout this early stage of the disease one of the dangers to be guarded against in the child is cardiac dilatation and failure: weakness and rapidity of the heart, or increase of the cardiac dullness, may make it necessary to use strophanthus or digitalis; probably the former is the better in these cases.

When the initial severity of symptoms has passed off and there remains still considerable œdema, diuretics may be of value; sometimes diuretin, given in doses of 5 or 6 grains in some syrup and water, three times a day for children of six to twelve years, increases the flow of urine; or potassium acetate, 5 to 10 grains, may be added to the diaphoretic mixture mentioned above.

I have also used the tinctura apocyni in doses of 5 to 7½ minims for children of about eight years. Solution of Adrenalin (1 in 1000) given by mouth in doses of 1–2 minims in distilled water, three or four times daily, sometimes has a marked diuretic effect, which, however, passes off in a few days but may, nevertheless, give valuable temporary relief.

The so-called 'Imperial drink' makes an excellent diuretic which children will often take readily. The formula in use at King's College Hospital is Acid Potassium Tartrate, 1 ounce; Tartaric Acid, 1 ounce; Oil of Lemon, 12 minims; Refined Sugar, 16 ounces; Boiling Water, 1 gallon. Of this solution the child may drink half a tumblerful or more two or three times daily.

Later, when there is only slight œdema and the child is in a stationary condition with much pallor, I think there is nothing better than the old-fashioned mixture of ferrous sulphate with magnesium sulphate, which may be given with advantage for several months. I have frequently noticed that the children whose urine continues to show a slight trace of albumen for

many months or years after an acute attack of nephritis are decidedly the better for this mixture, and if they leave it off for a few weeks they are apt to come complaining of headaches apparently due to the renal condition.

The time at which the child may first be allowed to get up is not easy to fix; but there is, I think, evidence that being 'up and about' throws a strain upon renal excretion which is to be remembered where there is a damaged kidney. The so-called cyclic albuminuria occurs usually only during the hours of being up; it has been shown that in some healthy people strained position of the body will produce albuminuria; in some cases of nephritis during convalescence albumen is found to be present only when the patient gets up; and, lastly, there are cases such as one I have mentioned above in which œdema recurs directly the patient gets up. It is a good error, therefore, to keep the child in bed for a longer time than might seem necessary if we were to judge only from the child's general condition after an attack of nephritis; probably by so doing we may give the renal tissues a better chance of complete recovery.

Diet, I think, is often needlessly severe in cases of nephritis which are past the initial acute stage; no doubt in the most acute period it is advisable to avoid a highly nitrogenous diet, and the child will do best with milk diluted with barley-water, thin Benger's Food and farinaceous diet.

But after a few weeks, when the child has reached a chronic condition, even though there be some œdema remaining, such things as fish, bacon, chicken, or eggs, may be given, though it will be wise to keep measurements of the amount of urine excreted and of the albumen and urea, and to be guided in diet by the results. My experience has been that, as a rule, the child is no whit the worse for having such food, even in the sub-acute stage, whilst in cases where a trace of albumen persists for months after the acute attack I think that a little red meat also may be allowed once a day.

When the child has reached this chronic stage of slight albuminuria, with no other symptoms, the question of climate will arise, for there is no doubt that residence in a warm climate considerably increases the chance of escape from exacerbations of nephritis, if not the chance of complete recovery. On our south coast, Falmouth, Torquay, or Sidmouth, or any of the places on the south coast of Cornwall, are suitable places for these children, while for those who like to take their children further afield, the French Riviera or the Canary Islands offer choice of localities.

CHAPTER XXXIX

SOME URINARY DISORDERS IN CHILDHOOD

It is sometimes of importance to know how much urine a child should pass daily. As an easy method of remembering the average quantities, I think that the formula which I worked out some years ago is sufficiently accurate, but it applies only to children from four years old and upwards, namely, age in years multiplied by 2·5; the resulting figure represents the quantity of urine in ounces.

In younger children and infants the amount of urine is larger on account of the fluid character of their diet; during the first year it is about 12 ounces a day, and during the second year about 10 ounces. But at all ages it is subject to considerable fluctuation, e.g. a boy, aged $5\frac{1}{4}$ years, whose average for nine days was 13·5 ounces, passed one day 21 ounces and another day 6 ounces, and a child of three years, whose average for nine days was 9·2 ounces, passed one day $15\frac{1}{2}$ and another day $4\frac{3}{4}$ ounces.

This fluctuation in quantity is, I think, much more marked in infancy and early childhood than in older children, say at eight years or more; it is no doubt one manifestation of that instability of function which is one of the characteristics of early childhood. I mention this point because it has a practical bearing upon a not very uncommon occurrence in children during the first half of childhood, namely, suppression of urine for many hours without any apparent cause. When a mother says that her child has passed no urine for more than twelve hours this does not necessarily mean that the child's bladder is full.

A catheter should never be passed where there is this complaint without first ascertaining by percussion whether the bladder is much distended: usually the failure to pass urine is not due to any retention, the urine has not been secreted and the bladder contains so little that the child has no desire to micturate. Most of the cases I have seen with this condition have been children about two or three years old; one girl at $3\frac{1}{4}$ years passed no urine for $18\frac{1}{2}$ hours, and then passed no more than her usual amount. A boy of three years went 22 hours without

passing urine; an older child, a girl of twelve years in good health, was brought to hospital because she had passed no urine for 21¼ hours, the bladder was not distended, but after drinking some warm milk she emptied her bladder, which contained only 2½ ounces of urine.

There is usually nothing to account for the suppression of urine in these cases; the urine which is passed after the period of suppression is not even highly coloured in some of the cases, and there are no symptoms. The phenomenon is purely a physiological variation, and as such calls for no special treatment; indeed it would hardly call for mention were it not that there is a chance that those who are unfamiliar with it might think that a catheter was necessary. Catheterization, always to be avoided in a child if possible, is not only unnecessary but useless in these cases, the small amount of urine which is secreted will be passed in due time without any interference.

Albuminuria. The presence of a small and transient trace of albumen in the urine is not a rare occurrence in children who are out of health from any cause, particularly when they are suffering from any gastro-intestinal disturbance.

Observations of the urine from a series of forty sick children, amongst whom there were none suffering from nephritis, diphtheria, heart disease, or any of the diseases ordinarily recognized as producing albuminuria, showed that within fourteen days no less than twenty-five had shown a transient trace of albumen in the urine on one or more occasions; several of this series were under the age of two years, and I have the impression that an evanescent albuminuria is much commoner in the urine of infants and very young children than in older ones.

Where there is nothing more to be detected in the urine than a slight trace of albumen, a single examination is clearly not sufficient to show whether there is any importance to be attached to it.

Cyclic albuminuria. It is a good rule always to examine two specimens, an early morning and a mid-day one, for in some of the children whose urine shows a trace of albumen at midday, cyclic albuminuria is present. This condition is probably commoner in childhood than has been supposed. It is generally said to be commoner in boys than girls, but of ten consecutive cases from my books eight were girls. They were all between seven and thirteen years of age. They are mostly children over six years of age in whom the condition is found. The urine just after rising in the morning is free from albumen, but two or

three hours later a definite but usually very slight cloud of albumen is present on boiling.

In testing for albumen I think that boiling the upper part of the contents of a test tube three parts full, and then adding acetic acid, is much more reliable than the nitric acid test, with which I have frequently known small amounts of albumen overlooked which were easily recognized with the simple boiling.

The symptoms for which the children with cyclic albuminuria are brought to the medical man are usually of the vaguest. The child has usually been noticed to be slightly puffy under the eyes, like a child with worms or chronic indigestion; he is pale and without being ill is seldom quite well; he is nervous, complains of headaches, occasionally perhaps of nausea, and in many cases has pains in the abdomen; in two of the cases mentioned the child was said to turn faint occasionally. I have seen cyclic albuminuria in association with enuresis and nervous (lienteric) diarrhœa.

In at least three of this small series the urine showed calcium oxalate crystals; an occasional hyaline or granular cast has been found by some observers in the urine of patients with this disorder; in two of these ten cases a stray cast was found by centrifugalizing on one or two occasions.

The occasional presence of one or two casts in the urine in these cases does not apparently indicate any nephritis; but it should make us careful in diagnosis, for after a definite acute nephritis, when the albumen has diminished almost to the vanishing point, there is sometimes a period during which the albumen is present only in the middle of the day, not in the early morning. I have mentioned the time relation, but no doubt the albumen in these cases, as in most cases of cyclic albuminuria, does not depend really upon the time of day, but on the erect posture or on muscular activity.

Another possible fallacy is the contamination of urine by vaginal discharge, which is probably more likely when the child has been walking about than when she has only just risen from bed.

Beyond the slight symptoms I have mentioned, cyclic albuminuria seems to have no ill effect; the child suffers little inconvenience from his slight symptoms, and sooner or later drifts away from medical observation, so that it is difficult to follow out the course of the disorder, but I have kept several cases under frequent observation for two and three years, and have seen no further harm come of this disorder. I have known it to pass off entirely soon after puberty.

Treatment seems to have little or no effect. I have tried calcium lactate, of which 5 or 6 grains can be given three times a day to a child of eight or nine years ; in one case the child was said to be much better in general health while taking it, but I could not satisfy myself that the amount of albumen was less. There is little to be done beyond the treatment of particular symptoms as they arise. There is, I think, no need to treat the child as an invalid or as specially liable to nephritis.

Where there are calcium oxalate crystals in the urine it is wise to prohibit foods known to favour oxaluria, such as tomatoes and asparagus, but especially rhubarb, which in some healthy children will produce hæmaturia or albuminuria. A small plateful of stewed rhubarb given to a healthy girl, aged four years, at 12.30 noon produced red urine full of blood corpuscles and calcium oxalate crystals at 3 a.m. the next morning. There is, however, marked idiosyncrasy in this, for another child who took treble this quantity of stewed rhubarb in twelve hours showed oxaluria but no hæmaturia.

Uric Acid. An occasional cause of albuminuria in children is the presence of uric acid crystals in the urine. Infants often pass a considerable quantity of this 'cayenne pepper' deposit. Many children in the earlier half of childhood pass urine which is very acid and after standing a short time deposits uric acid; a much smaller number pass the uric acid crystals already formed.

It is noticeable in these cases that the child is pale and fretful, complains of feeling tired, has perhaps some nausea, and generally is ' below par '. I find that in most cases the doctor has directed that little or no red meat shall be given, and the parents having an idea that anything in the nature of meat is likely to produce this trouble have carried his directions even further, and the child is living largely upon a starchy carbo-hydrate diet. I very much doubt whether there is wisdom in this ; it has seemed to me that some of these children who pass very acid urine with uric acid in it are suffering not from excess of proteid but from excess of carbo-hydrate in the diet, and that what is needed is reduction of this element. The child who begins the day with porridge and toast and then has a meal consisting chiefly of potato and farinaceous pudding, and, later, bread-and-butter and cake with an occasional interlude of the indigestible apple or banana, may be better for the substitution of bacon, egg, or fish for breakfast, and of broth, boiled brains, fish, sweetbread, or chicken for dinner ; and of custard or junket or jelly in place of the farinaceous pudding.

Free drinking of water and the administration of potassium citrate is advisable ; malt and pancreatin may also do good.

Hæmaturia. There are some causes of hæmaturia which may be mentioned here as concerning chiefly infancy and childhood.

When an infant under the age of one year shows blood in the urine the probable cause is infantile scurvy. Hæmaturia is sometimes the only symptom of this disease, and the diagnosis has then to be made from the history of scorbutic feeding and the rapid disappearance of the blood when fresh fruit juice or potato is given ; usually, however, there are other characteristic symptoms of scurvy.

New growth in the kidney is commonest in infancy and early childhood, but it is seldom seen in the first year ; hæmaturia is not a constant symptom.

Tubercle of the kidney must be reckoned amongst the causes of hæmaturia in children, and the appearance of blood in the urine, perhaps even in sufficient quantity to redden it, may be the first indication of this affection of the kidney. Sooner or later, however, the blood in these cases is likely to be associated with more or less pus, and this is perhaps of more serious significance than the hæmaturia, which is more likely to occur in the early stage of the disease, and is certainly not always the precursor of any progressive tuberculosis ; I have seen several such cases do well, and am by no means inclined to resort to surgery even if one could be sure that the affection was unilateral.

Whooping-cough sometimes produces profuse hæmaturia when the paroxysms are violent ; the bleeding is no doubt due to rupture of small vessels, and after a day or two the blood ceases to appear in the urine. I have occasionally seen nephritis in children with hæmaturia as the only prominent symptom : the child has shown no trace of dropsy, but was brought because the urine was red. There are other causes of hæmaturia which are common to adults as well as children ; children occasionally suffer with renal calculus. In countries where bilharziosis is prevalent children are subject to hæmaturia from this cause. A boy, aged about nine years, was brought to me at King's College Hospital with hæmaturia, for which no explanation could be found until it was discovered that he had lived in South Africa, and microscopic examination showed the ova of bilharzia in the urine.

Children seem to suffer occasionally with an unexplained bleeding from the kidney like the bleeding from the nose to which many children are liable ; this ' renal epistaxis ' passes off after

a few days, leaving no trace behind it, and nothing further occurs to explain it.

Lastly, it must be remembered that a red urine, or a brown one, does not always mean hæmaturia, it may be the result of hæmoglobinuria, as can be determined by the absence of blood corpuscles, or their extreme scarcity in proportion to the colour of the urine. This disorder, in the few cases which I have seen, as in several which have been recorded by others, has been associated with congenital syphilis.

Abnormal Colour. Apart from abnormalities of colour due to peculiarities of metabolism, such as the rare congenital condition alkaptonuria, in which the urine becomes a dark blackish colour after it is passed, so that it stains the diapers, there are curious pigmentations of the urine which are specially liable to occur in childhood owing to the eating of coloured sweets.

A yellowish pink fluorescent appearance of the urine, very like that of a solution of eosin is produced by the eating of pink lozenges and various aniline coloured substances may thus be responsible for curious tints in the urine. Recently I had under observation a little girl passing bright green urine, which on examination appeared to be due to aniline dye, and was, no doubt, explained by the colour of her dress, which was exactly the same, and which she probably sucked. In one instance[1] a boy, in order to evade school, soaked a piece of turkey-red cloth in his urine, and pretended that he was passing blood.

[1] *Clin. Soc. Trans.*, vol. xxix, p. 174.

CHAPTER XL

PYELITIS IN INFANCY AND CHILDHOOD

Acute pyelitis is hardly one of the common diseases of childhood, but it is more frequent, especially in infancy, than is generally known. Admirable descriptions of this disease have been published by Dr. Holt, of New York,[1] and Dr. John Thomson, of Edinburgh.[2] Its recognition is usually a very simple matter, and yet it is overlooked again and again simply because the possibility of its occurrence is forgotten and the urine of an infant is so seldom examined. Unrecognized, acute pyelitis in infancy gives rise to prolonged fever of most severe degree, with profound constitutional disturbance, which may end in death; recognized and treated with appropriate drugs it often subsides in a few days, and even if symptoms persist for a while they quickly become less severe, and generally yield to treatment before long. The recognition of pyelitis is therefore a matter of urgent practical importance, and as a preliminary to a study of its diagnosis and treatment I shall sketch in outline the clinical picture of the disease as it commonly presents itself.

A female infant under the age of twelve months, and apparently in perfect health, is suddenly taken ill—so suddenly indeed that the mother can often tell the exact hour at which the illness began—the child seems hot, so the temperature is taken and is found to be 103°. From this time, day after day, the temperature ranges from 101° to 105°, the infant is fretful and miserable, crying out suddenly at times as if in pain, or perhaps drowsy and inclined to twitch slightly in the fingers and eyelids. Either at the moment of onset or perhaps later the infant has been noticed to turn blue and cold, or has actually shivered. Vomiting is either absent or occurs only occasionally. The stools, though perhaps not loose or costive, are nevertheless not quite as they should be, and on inquiry there has been some unhealthiness of the stools or perhaps constipation several days before the illness began. There has been nothing to call the mother's

[1] Holt, *Archives of Pediatrics*, Nov. 1894.

[2] Thomson, *Scot. Med. and Surg. Journ.*, July 1902, and *Quart. Journ. Med.*, April 1910, p. 251.

attention to any abnormality of the urine, nor is there any evidence of pain anywhere.

Careful examination fails to reveal signs of any sort; possibly the abdomen is a little full, and there seems to be some vague discomfort on palpation, but there is nothing more.

The infant is obviously acutely ill, but no explanation of the high fever and profound constitutional disturbance is forthcoming until the urine is examined, when pus and bacteria are found.

Such, in brief, is the clinical picture of acute pyelitis, which I shall now describe in more detail.

Sex. Pyelitis is almost but not quite exclusively an affection of females. Of 28 infants under one year who have come under my own observation with this disease only 3 were boys, and amongst 14 older children there were no boys. Why this predominance of girls? It is at least consistent with the view that the infection of the urinary tract travels up from below, the vulva is soiled with fæces and the bacteria pass through the bladder and ureter to the kidney.

Age. The age-incidence also would fit in with this theory. Out of 42 cases 28 were under the age of twelve months when the pyelitis began (the youngest, a girl, was ten weeks old), and 2 others were under two years. Of the remaining 12, 4 were between two and four years old, and 8 were under ten years.

The disease is evidently much commoner in infants than in older children, and the soiling of the vulva with fæces would occur most easily during this age of diapers.

One might further point out that the bowels have often been loose before the onset of pyelitis, or, if there has been no diarrhœa, the stools have been unhealthy in some way, green or curdy or offensive, sometimes only constipated.

In harmony with all these facts the *Bacillus coli* is found to be the exciting cause in the large majority of cases of pyelitis (only rarely is the *Proteus bacillus* or some other micro-organism found), and it seems reasonable to suppose that the infection comes from the bowel.

It is possible, however, that the route from the bowel to the kidney may vary, and that in some cases it is via the blood, and in some even directly by contact; at any rate it must be remembered that pyelitis does sometimes occur in boys, and that it sometimes shows symptoms very suggestive of a blood infection.

Symptoms. A striking feature in most cases is the abruptness of the onset. In one case the infant was being dressed one

morning, apparently quite well, when she suddenly 'went blue, and seemed cold and faint', from that time the child was acutely ill with high fever; in another the infant had gone out seemingly in perfect health in her perambulator, when she suddenly became acutely ill, and was thought to have a convulsion; she was brought home and found to have a temperature of 104°, which was explained only when the urine was examined four days later. I say 'thought to have a convulsion', because I think it is quite clear, on careful inquiry, that in some cases the supposed convulsion is really an attack of quite a different character, sometimes a genuine rigor, sometimes an attack in which the infant goes cold and blue, but does not actually shiver.

Dr. Thomson has laid stress upon the occurrence of shivering or rigor in the infant as an early symptom. Rigors are so extremely rare in infancy and early childhood from any cause that their occurrence with this pyelitis may be of considerable diagnostic value. I cannot, however, affirm from my own experience that they occur in a majority of the cases. I have made special inquiries upon this point in most of the cases under my own observation, and in some could obtain no history of shivering. This, of course, does not prove that rigors had not occurred, for they would easily be overlooked in an infant, but it does prove that the failure to obtain a history of shivering does not weigh much against the possibility of pyelitis as the cause of fever. It is often said that a convulsion takes the place in infancy of the rigor of the adult, but I have not observed in this pyelitis of infancy any special tendency to convulsions at the onset in those cases in which a rigor did not occur, nor do I think that a convulsion is the only counterpart of a rigor; I suspect that vomiting in early childhood sometimes takes the place of the rigor of the adult, for instance, at the onset of lobar pneumonia.

However this may be, I would point out that without any actual shivering it frequently happens that several times during the course of acute pyelitis, especially at its onset, an infant suddenly turns blue and cold, and seems collapsed. These attacks occur in such a large proportion of the cases that to my mind the combination of an otherwise unexplained fever in an infant with attacks of blueness and collapse is always highly suggestive of acute pyelitis.

Of course, not every supposed convulsion in acute pyelitis is of the same nature as these attacks which I have described; genuine clonic convulsions do occasionally occur at the onset

or during the course of the disease, but they are quite exceptional—only 3 out of 28 infants had convulsions.

To any one who has seen the condition many times, the appearance and manner of the infant is often enough to suggest the diagnosis; the extreme misery and restlessness is very striking, unless indeed, owing to the severity of the infection, the infant has already fallen into a drowsy, almost comatose state; the complexion is pale and earthy, the skin is hot and dry; when with such a condition I am informed by the doctor that nothing has been found to account for the high fever, I always suspect that pus will be found in the urine.

But perhaps it is not quite correct to say that nothing is found, for in some cases the abdomen is a little fuller than normal and the infant seems to be discomforted more than is natural by gentle palpation. It is difficult to be sure of this, for the infant is so fretful that any examination is resented; but I am the more inclined to think that this observation, made in several cases, is correct, because in older children I have sometimes found quite definite tenderness over the front of the abdomen with pyelitis, so that the condition has even simulated appendicitis.

There are cases in which pyelitis assumes the guise of acute cerebral disease: the child is drowsy or even semi-comatose, there is muco-pus on the cornea, the neck is stiff, the head perhaps definitely though slightly retracted, there may even be a squint and some slight convulsive twitching of the limbs; what wonder if the case is mistaken, as it so often is, for one of meningitis?

The temperature in acute pyelitis is profoundly puzzling so long as the condition remains unrecognized; day after day it is 103° and 104°, and often reaches 105° several times, nor does the fever show any sign of abating usually for several weeks unless the cause is recognized and the proper treatment given. With the requisite drugs the temperature falls generally within two or three days to a much lower level, if not to normal, but it is very apt to rise again after a few days, for reasons which I shall consider later.

The chart shown here (Fig. 37), from a girl aged 4½ months, is a very characteristic example of the prolonged and severe fever caused by acute pyelitis, when this is unrecognized and consequently is not treated with suitable drugs.

In striking contrast with this is the chart (Fig. 38) from an infant of the same age in whom treatment of the pyelitis was begun on the fourth day of the disease.

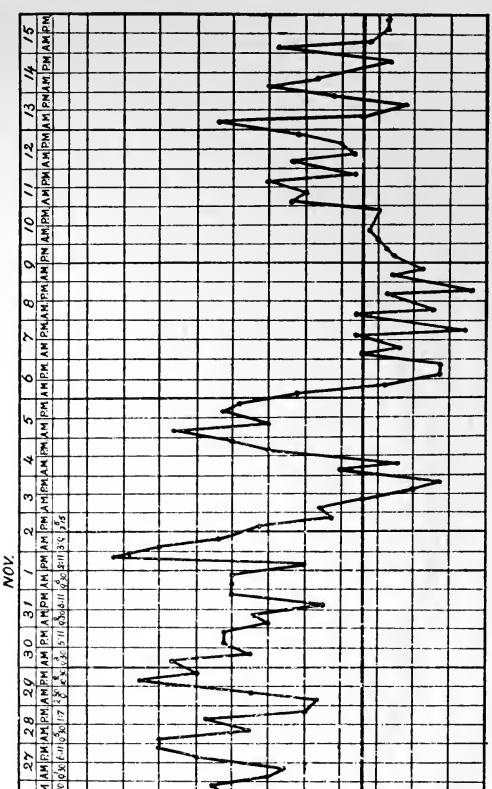

FIG. 37. Acute Pyelitis. Chart of girl aged 4½ months, showing prolonged fever due to acute pyelitis. The disease was mistaken for tuberculous meningitis, and the pyuria was not discovered until it had already persisted many months and the infant had fallen into a marasmic condition.

The one essential, of course, in the diagnosis of this disease is the examination of the urine, and this is the one point which has not been examined in most cases. 'It is a baby,' says the doctor, 'so one can't get the urine.' I have been told this again and again, and I will venture to say that in no single one of these cases have I failed to obtain the urine within a few hours—often indeed it was forthcoming within a few minutes—after explaining to the nurse the extreme importance of having it.

It is necessary to point out that the object of examination is to ascertain whether pus corpuscles are present, and therefore the urine must be obtained by some other method than by catching in absorbent wool or any other absorbent material which would

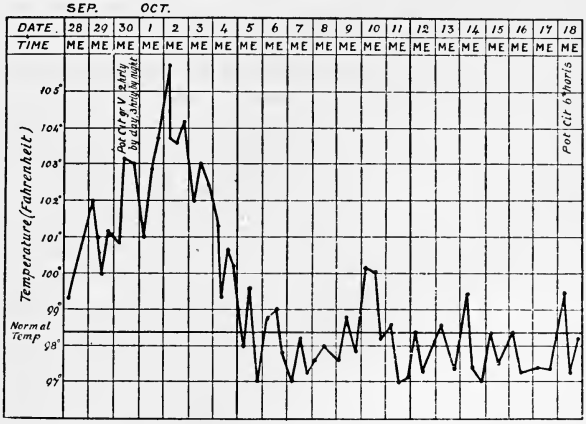

FIG. 38. Chart from a case of acute pyelitis in a girl aged 4½ months; treatment with potassium citrate begun on fourth day of disease (chart begins on second day of illness).

strain off the pus cells, so that the urine squeezed out would be useless for the purpose. A simple plan is to let the infant lie for a few hours on waterproof mackintosh until some urine is passed; a little arrangement of the mackintosh will usually secure that the urine shall collect in sufficient quantity for the purpose. A few drops will suffice, as it is microscopic examination only which is necessary to the diagnosis.

Several times I have been told that the urine had been examined and that it was 'clear and acid, with no albumen', although the aspect of the case was that of pyelitis, and further examination showed pus and albumen in the urine. Mistake is easy enough: more than once after I have suggested that the case was one of pyelitis the doctor has produced the

infant's urine in a chamber-pot of white china, and remarked, 'See, it is perfectly clear!' whereas directly it was poured into a clear glass bottle it was obvious that the urine was slightly turbid.

Again, the doctor says there cannot be pus, for he found no albumen; on inquiry I find that he used the nitric acid test, and on using the boiling test, with subsequent addition of a few drops of acetic (being careful to fill the test-tube three parts full and to boil only the upper half-inch so that the slightest turbidity may be seen by contrast), one is able to show that there is albumen, albeit extremely little, corresponding to the very small amount of pus found in many of these cases. The use of the nitric acid test is a very common cause for overlooking small traces of albumen in urine.

Again, even when the urine has been found to contain a trace of albumen, there has been so little cloudiness of the unboiled specimen, and such an apparent absence of deposit, that the doctor has not thought it necessary to examine it microscopically. It must be remembered that the amount of pus is generally microscopic in pyelitis, often only six or even fewer pus-cells are to be seen in a field (under a one-sixth objective) when the shaken-up urine is examined, and this even when the symptoms are most severe. Here I would emphasize the advantage of examining the shaken-up urine rather than urine which has been allowed to stand, or has been centrifugalized. The examination of a deposit gives no idea whatever of the relative amount of pus which is present; the deposit obtained by standing or centrifugalization from a urine which would show 8 cells to the field in the fresh or shaken-up specimen, does not differ from one which would show 30 cells, so that a valuable standard of comparison is lost. This principle applies not only to pyelitis but also to other urinary conditions, such as nephritis and hæmaturia. The centrifuge is often rather a hindrance than a help; we want to know not only that there are casts or blood-cells, but how many.

The urine of pyelitis before treatment is acid, usually very acid, and commonly shows (under one-sixth objective) an average of about 6-30 pus-cells per field, and here and there a group of perhaps 10 or 12 stuck together; usually, but not always, bacilli can be seen either singly or in clumps, and in the large majority of cases prove to be *Bacillus coli*.

Hitherto I have referred only to pyelitis in its acute stage. I want to draw attention also to the extremely misleading cases

in which the diagnosis has to be made after the disease has become chronic. These come to the doctor under the guise of marasmus; a miserable fretful infant is brought solely for wasting; at first sight there seems to be enough to account for the condition, the infant takes food badly, and the stools are unhealthy, and no one has doubted but that the trouble was simply digestive. The extraordinary fretfulness and misery, however, of the child may suggest something more, and inquiry elicits the fact that there was some febrile illness, it may be months ago, or perhaps there has been a good deal of irregular fever, which has been attributed to teething, influenza, or what not—in one such case I was assured that the infant had had tubercular meningitis, a mistake evidently due to the nervous manifestations of acute pyelitis, as examination of the urine showed. In older children also a Coli pyelitis, when it has become chronic, may show itself as a wasting disease. A girl, 9 years old, was brought to me for supposed phthisis; she was much wasted, and, with phthisis in mind, it was easy to imagine little differences between parts of the lungs on percussion and auscultation; but the temperature chart was unlike it; there were bouts of fever alternating with longer periods of normal temperature, and the child was more cachectic than her very doubtful pulmonary signs would warrant; examination of the urine showed large numbers of pus-cells and Coli-form bacilli.

Acute pyelitis, as I have already pointed out, is much less common in older children than in infants, but it is important to remember that it does occur, for it is easily overlooked; not only are there the same fallacies in the examination of the urine, but the symptoms are apt to be much less severe, the temperature is lower, and there is less constitutional disturbance. In the older as in the younger children there is rarely any local symptom or disturbance of micturition to call attention to the urine; the child is feverish, complains of headache, perhaps has a rigor or is sick, and sometimes there is definite pain in the abdomen, which may suggest appendicitis, as in the following case:

Lily S., aged $8\frac{1}{2}$ years, was admitted to hospital for pain in the right side of the abdomen and high temperature. These symptoms had begun suddenly on March 14. When admitted, on March 17, she was very drowsy, with some headache and marked rigidity of the right side of the abdomen. Appendicitis was suspected, but examination of the urine showed this to be acid with many pus-cells and Coli-form bacilli. Under potassium citrate treatment the fever rapidly subsided, and all rigidity and pain had almost disappeared by March 20, when the child suddenly became bad again, with much abdominal pain and rigidity, which, however, were now on the *left* side of the abdomen. Potassium

citrate, 22½ grains 'every two hours when awake' quickly relieved these symptoms; and the child made an apparently complete recovery. But on April 23 there was still 'an occasional stray pus-corpuscle' in the urine, and after remaining apparently quite well till August 3 the child again complained of pain in the abdomen, and was found to have fresh pyelitis, which again subsided under treatment.

This case seems to show that pyelitis may remain limited to one kidney, at any rate for a time, and that recurrence of fever and other symptoms when the child seems on the road to recovery, may indicate infection of the previously sound kidney. It illustrates also the liability to a subsequent attack of pyelitis if treatment has not rendered the urine entirely free from pus and bacteria.

Diagnosis. It is an interesting speculation how these cases were labelled in days gone by: one cannot doubt that Infantile Remittent Fever, Dentition, remarkable recoveries from supposed Tuberculous Meningitis, and many another equally wrong diagnosis served to cover the acute pyelitis of infants. I have kept a record of some of the diagnoses which had been made in cases which I have seen. 'Tuberculous meningitis' was the most frequent, and indeed some of the nervous symptoms of pyelitis are scarcely distinguishable from those of meningitis, but the very high fever and acute onset are quite unlike tuberculous meningitis, and would better fit in with cerebro-spinal, which had been suspected in some of the cases. The prolonged fever without physical signs had given rise to a diagnosis of 'Typhoid' in several, but again the acute onset should have raised a doubt as to the possibility of this diagnosis. The sudden onset and sharp fever had been attributed to a latent 'Pneumonia' in some, whilst 'Influenza', 'Gastro-enteritis' or 'Dentition' was thought to explain the symptoms in others.

These mistakes can only arise if the urine is not properly examined; there are other questions of diagnosis which are less simple. A girl with some fever and headache is found to have pus in the urine, but she has some vulvo-vaginitis—is there pyelitis or is the pus merely an accidental contamination from the vulva? The presence of bacilli in the freshly passed urine may help us, for vulval discharges are almost always due to cocci of some sort, not to bacilli; but it may sometimes be necessary to obtain a catheter specimen (a thing always to be avoided in the case of children if possible). Usually the effect of alkalinization of the urine will be the most satisfactory solution of the problem.

There are cases in which the distinction between Tuberculous

pyelitis and Coli pyelitis is by no means easy by ordinary clinical methods, especially when the pyelitis has become chronic; the tuberculous affection is not likely to show the sudden acute onset and severe constitutional symptoms of the Coli disease, and blood is often present in tuberculous urine, whereas it is very rarely found in the urine of Coli pyelitis. A bacteriological investigation of the urine may be necessary.

A secondary pyelitis may occur with congenital dilatation of the pelvis of the kidney, and may easily be mistaken for the acute primary pyelitis of infancy.

Jessie P., aged 5 months, had been vomiting for 7 weeks and wasting. The child, on admission, was very collapsed, and temperature fell to 97·4°. The urine was acid, showed about 15 pus-cells per field, and gave a pure growth of *Bacillus coli*. The child rallied from its collapse, and 3 days later the temperature was 101·2°, and subsequently 103·4°. Potassium citrate caused no improvement, although the urine became alkaline. The child died 13 days after admission. Post-mortem showed the right ureter greatly enlarged and tortuous, measuring 8 inches long, whereas the left was only 4 inches. The pelvis of the right kidney was somewhat dilated, and there were a few small abscesses in the cortex. The left kidney looked dark and congested, but otherwise normal. There was also some broncho-pneumonia.

No doubt in such a case the absence of any acute onset, and perhaps the low range of temperature, might suggest the possibility of dilated pelvis, but in the absence of any palpable enlargement the nature of the pyelitis could hardly be diagnosed.

It is difficult to account for the apparent absence of cystitis in most cases of Coli pyelitis; one would have thought the bladder must inevitably have been affected, nevertheless it is but rarely that straining or discomfort in micturition or frequent desire to pass water is present, indicating some irritation of the bladder. I mention this here because it is well to remember that such symptoms do sometimes occur where the course of the case otherwise seems to point to Coli pyelitis, and where, moreover, the effective treatment is that of pyelitis.

Prognosis. To any one who is not familiar with the course of the disease, the condition of the infant stricken with acute pyelitis may seem wellnigh hopeless, but experience shows that the outlook is generally good. Amongst 42 cases, including 28 under twelve months old, only 4 were fatal (three under six months old, and one in the second year).

Examination of the urine months after the acute attack shows that a trace of pus sometimes persists in spite of energetic treatment with alkalies during the acute stage. I suspect that this indicates insufficient dosage or too short duration

of treatment; in such cases there is occasionally, though rarely, a recurrence of acute symptoms after months of apparent good health. As a rule, with efficient treatment, recovery is not only rapid but complete: sometimes, as in the case from which the chart shown below (Fig. 39) is taken, the subsidence of the fever resembles in its rapidity the crisis of a pneumonia.

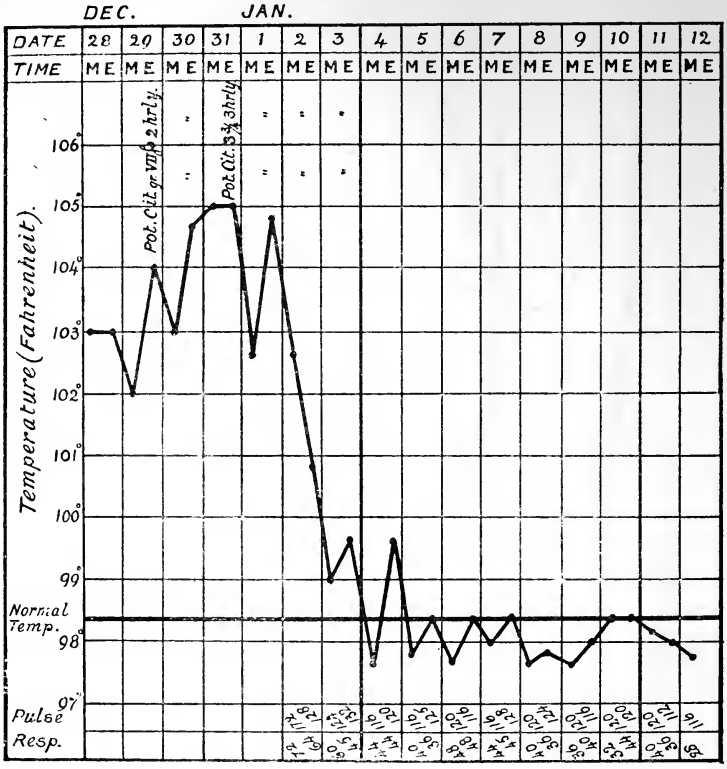

Fig. 39. Acute Pyelitis. Chart from a girl, aged 7 months, showing sudden subsidence of fever after treatment with potassium citrate.

Morbid Anatomy. In three cases which I have examined post mortem, the same characteristic changes in the kidney were found in all. No gross change could be detected in the pelvis, but spreading up from the pelvis into the pyramids were yellow streaks seen on section running upwards towards the cortex parallel to the straight tubules. This purulent infiltration was seen also in the cortex as small irregular yellow patches, some of which could be seen bulging slightly on the surface of the

kidney beneath the capsule. In some parts this infiltration could be seen to be surrounded by a narrow zone of inflammatory hyperæmia.

This invasion of the pyramids and cortex may of course occur only in fatal cases, while in those that recover the inflammation may be limited to the pelvis.

Treatment. The treatment of acute pyelitis is in most cases as satisfactory as it is simple since Dr. J. Thomson first emphasized the importance of alkalinization of the urine. In most cases this has almost the virtue of a specific: the improvement in the infant's condition is almost as rapid as the relief of scurvy by potato-cream.

The drug which is most generally convenient for this purpose is potassium citrate, but its virtue depends entirely upon proper frequency and dosage: it is useless unless sufficient is given to render the urine alkaline, and its effect is only transitory unless the alkalinity is maintained night and day continuously for at least a week or ten days. As a preliminary dose 5 grains of the potassium citrate is to be given every two hours by day and every three hours by night; this dose will do for any age in the first year of life. Strict injunctions must be given to the nurse that the urine shall be obtained as often as possible, and tested immediately after it is passed with litmus paper. The only gauge of the efficiency of the dosage is the reaction of the urine; so long as this remains acid the fever and general symptoms are likely to persist. The charts shown in Figs. 40 and 41 show the speedy effect of this treatment with potassium citrate.

It is surprising what large doses are necessary in many cases to keep the urine alkaline, 8 or 10 grains every two hours may be only just sufficient in the case of an infant, and a child of three years may require 20 grains or even more every two hours.

It is necessary, however, to point out that the use of potassium citrate has its limitations; I have noticed repeatedly that large doses, e.g. 10 grains or more every two hours for an infant of six or eight months, are apt to disturb digestion and to set up diarrhœa, which is a troublesome complication; where this has happened, I have sometimes found that bicarbonate of soda or potassium, in doses of 5–10 grains every two or three hours, has been successful in maintaining the alkalinity already induced by the potassium citrate, and the bicarbonate has not had the ill effects of this drug.

How long the potassium citrate should be continued is a very important question. Again and again I have seen symptoms

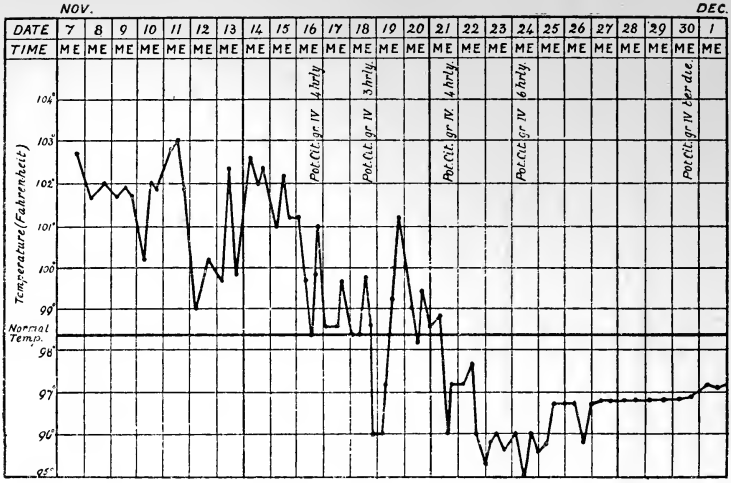

FIG. 40. Acute Pyelitis. Chart from a boy aged $4\frac{1}{2}$ months.

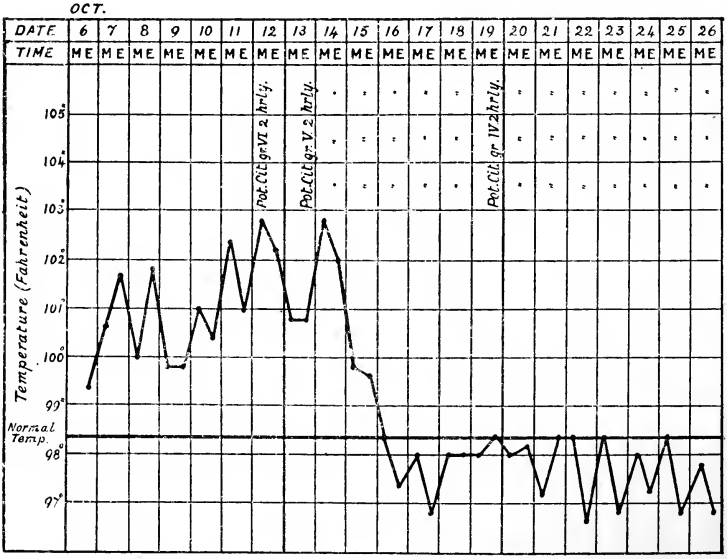

FIG. 41. Acute Pyelitis. Chart from a girl, aged $5\frac{1}{2}$ months, showing effect of treatment with potassium citrate.

recur where the drug was not continued long enough with regularity.

When the temperature falls, as it usually does after two or three days of this treatment, there is a great temptation to reduce the dose or frequency of the potassium citrate, and the mother is only too ready to give up disturbing the infant at night for medicine, which now seems unnecessary. But the result is grievous disappointment, the temperature quickly rises again, and the child seems as bad as ever until the urine has again been rendered alkaline. I would lay it down as a general rule that the potassium should be continued at the efficient dose, i.e. that which renders the urine alkaline, every two hours by day and every three hours by night, for at least ten days after the temperature has become normal.

I have referred hitherto to the temperature as if it were the only symptom which is to serve as guide in determining the duration of treatment. But there is another direction in which careful watch should be kept; every day or two the number of pus cells in the urine should be counted; only in this way can we obtain any clear idea of the degree to which the pyelitis is subsiding. This is easily done with quite sufficient accuracy for practical purposes by examining a drop of the shaken-up urine under a sixth objective, and taking the average of the number of cells counted in half a dozen fields.

Usually the diminution in the number of cells is quite appreciable after two or three days, and often it is possible to observe also a decrease in the number of bacteria present (the *Bacillus coli* is readily seen with a sixth objective). No doubt the most scientific method of determining the proper time to discontinue the alkali treatment would be repeated bacteriological culture from the urine, but this is seldom practicable; in the disappearance of the pus-cells we have a more easily ascertainable criterion, which is at least as important as the temperature, and by disregarding which we may, I think, lay the seeds of future trouble. As already mentioned, a few pus-cells can sometimes be found in the urine months after the temperature has become normal, and in such cases a recurrence of all the acute symptoms may occur; it seems possible that by having regard not only to the temperature but also to the disappearance of the pus-cells before discontinuing the alkali we might prevent this misfortune.

After laying so much stress upon the alkali treatment and its success, I must needs add that there are cases in which the alkalies fail. I have not been able to formulate any rule whereby

one could tell when this was likely to happen; it undoubtedly occurs sometimes when the pyelitis is due to some organism other than the *Bacillus coli*, e.g. the *Proteus bacillus*, but it also occurs with the *Bacillus coli*, though very rarely. When this is so, urotropine occasionally does good; 1½ grains may be given every four hours to an infant six months old, and 2½ grains at twelve months. Another drug which is certainly useful is salol, 1 grain every four hours at six months of age; this sometimes makes a useful adjunct also to the alkali treatment, where the unhealthy condition of the stool threatens further Coli infection.

In the most severe cases with profound toxæmia, where the temperature is very high, and nervous symptoms are prominent, anti-colon bacillus serum is worthy of trial. I have known it cause remarkable improvement. It is given subcutaneously in doses of 5–7 c.c. for an infant under six months of age.

In obstinate cases—and there are some in which in spite of all drugs the pyuria and irregular temperature persist—I have several times resorted to vaccine treatment; this should be carried out if possible with an autogenous vaccine, i.e. one made from the child's own urine. As initial dose 5 millions may be given subcutaneously to an infant six months old, and a week later 8 or 10 millions, which may be increased after another week to 15 or 20 millions: to a child of 3½ years I have given as many as 100 millions.

Whatever treatment is used stimulants are often necessary, especially in the acute stage, when large doses of potassium citrate are being given, as whether from this drug as some have thought, or from the disease, the child is often much depressed, and 10 or 15 minims of brandy every four hours may be an essential part of the treatment.

If any tendency to convulsions shows itself, phenazone ½–1 grain at 6–12 months should be given with 2 or 3 grains of sodium bromide, in a mixture separate from the potassium citrate, so as not to interfere with the frequent administration of the latter.

CHAPTER XLI

MENTALLY DEFICIENT CHILDREN

Most medical men are confronted from time to time with cases of mental deficiency in children, and most of us know only too well how difficult to answer are some of the questions put to us by the parents: Will the child talk? Will he learn to walk? Has his mental condition resulted from a fall? If other children are born, is there any likelihood that they too may be imbecile? Has the fact that the parents are first cousins any bearing on the condition? These are only some of the many questions which the doctor is expected to answer.

I have kept notes for several years past of all the mentally defective children who have come under my own observation in hospital and in private practice, and I shall base some rather scattered remarks on this subject upon this series of 350 cases. As will be seen, I have not been able to investigate every case with reference to all the questions in which I was interested.

Imbecility seems to be as common in one sex as in the other: my own figures show 174 boys and 176 girls. Many statistics have been published on this point, some show a slight preponderance of boys, others a slight preponderance of girls; the conclusion would seem to be that there is no special sex-incidence.

The recognition of mental deficiency in infancy is not always easy. I find from my notes that in most cases the parents have not suspected that there was anything serious amiss until the tenth to the fourteenth month, when the failure of the infant to sit up drew attention to the abnormal condition; but the medical man can often recognize the deficiency earlier than this. In one group of cases, those known as Mongols, the facies within a few days after birth is so striking and so characteristic that the mental deficiency which is associated with this particular abnormality can be foreseen even at this age; this, however, is the only variety of imbecility in which a diagnosis is possible during the first week or two of life.

It might be supposed that cretinism, which has such characteristic features at a later age, would be recognizable at birth or very soon afterwards; this, however, is not so. It seems probable

that a newborn infant has received from the maternal circulation a supply of thyroid secretion sufficient to prevent any signs of thyroid insufficiency from appearing for many weeks after birth ; it is only after this has been used up that the absence of thyroid secretion becomes evident in the appearance of cretinism. However this may be, it is certain that cretinism is very rarely recognized, and probably is seldom recognizable, until the sixth or seventh month ; the earliest age at which I have seen it myself was at seven months ; Dr. Koplik, of New York, has observed it as early as four and five weeks after birth. Formerly the name 'fœtal cretinism' was given to the condition now known as achondroplasia, which is of entirely different pathology and is not associated with mental defect.

The abnormality of the thyroid from which cretinism results is no doubt usually congenital, but it may be doubted whether the symptoms of cretinism are ever congenital. The date of recognition of this form of imbecility has a special practical importance, for the degree of physical and mental development which is attainable, albeit always somewhat short of the normal, is certainly greater when treatment with thyroid is begun very early.

Amongst other forms of imbecility one of the earliest recognized is microcephaly, partly perhaps because the contrast between the small cranium and the relatively large face attracts notice, and partly because the more severe degrees of microcephaly are usually associated with extreme fatuity of mind ; as might be expected, the more complete the fatuity the earlier the mental deficiency becomes recognizable.

Symptoms. There are certain points which should suggest imbecility in an infant, apart from the peculiarities of facies which characterize the Mongol and the cretin. The most important of these is failure to acquire certain powers at the proper age. Most infants, when held in the sitting posture, hold the head up quite steadily at the age of four months ; if an infant in good physical health fails to do this at the age of six months there is ground for suspecting some mental deficiency. Most infants distinguish their mother or nurse from a stranger quite definitely at the age of six months, often considerably earlier (at $3\frac{1}{2}$ or four months) ; failure to do so at the age of nine months would indicate mental deficiency unless there were some gross defect of sight.

A healthy infant should be able to sit up without support at the age of nine months ; if an infant without rickets or other

physical disease is unable to sit up alone at twelve months, mental deficiency is probable. The age of learning to walk varies very considerably in health, but I think it may be said that if a child is free from rickets and other physical disease, and makes no attempt to walk at the age of eighteen months, mental deficiency is at least probable.

The date of speech development is even more variable in health ; it may be very imperfect or even absent altogether for as much as two or even three years, without any deficiency of intellect, but as a general rule a child should be attempting to say single words at twelve months, and any considerable delay beyond this age may, when associated with such other evidences as I have mentioned, point to mental defect.

Far more characteristic than mere delay in speech development is the habit which some imbeciles show as early as the end of the first year of making meaningless uncouth sounds, sometimes shrill sudden cries, sometimes a drawling, senseless sound, accompanied with a fatuous smile, without apparent aim or object : these sounds are very different from the pleasant cooing and lalling of the normal infant, which, though inarticulate, are not meaningless ; they convey the idea of a purposive expression of pleasure and contentment.

Unnatural movements are often a symptom of mental disorder. I have noticed a tendency in some imbecile infants to deflect the eyes without reason to one side ; in others the normal co-ordination of the limbs is not acquired, the arms and legs are flung about in a clumsy, purposeless way at the age of nine or ten months, when an infant should be beginning to use its limbs in a definitely purposive manner, particularly in grasping at objects. At four months old some normal infants will grasp at an object with definite purpose to reach it, though the aim is usually very inaccurate at this age ; at six months the grasp is sometimes perfectly accurate, though the infant is apt to grasp with fingers only, not using the thumb. If an infant fails to grasp at objects, particularly at its feeding-bottle, when it is nine months old, mental deficiency is highly probable, and similar failure at twelve months old makes mental deficiency almost certain, provided of course that there is no defect of sight.

A curious symptom of mental deficiency, and one which is apt to be puzzling in early infancy, when there may be nothing otherwise to suggest imbecility, is frequent causeless crying and screaming. I must confess that, as a rule, one has only recognized the significance of this in retrospect. The mother has brought

her infant to me at the age of two or three years or thereabouts, with obvious signs of idiocy, and has volunteered the statement that the child as an infant was always screaming, without any apparent cause; but sometimes I have heard this complaint, and heard the crying too, when an infant has come to me with signs suggestive of mental deficiency in early infancy, and in such cases this symptom has contributed to the diagnosis.

Of course crying and screaming are common enough in infancy, with little or no ascertainable cause, and he would be a rash person who would attach any weight to this symptom by itself, but in some of those cases where the most careful investigation of all the ordinary causes fails to show any reason for the constant crying, the possibility of mental deficiency is to be kept in mind. I use this expression advisedly: it would be worse than folly even to hint at such a possibility to the parents until other much stronger evidence has appeared.

Exactly the reverse is the story in some cases. 'My baby is so good, it never cries'; the imbecile infant is often abnormally placid. I am not referring now to the stolid placidity which characterizes the cretin, the '*chrétien*' or '*christian*' disposition to which some have traced the term 'cretin'. Perception would seem to be so dull in many varieties of idiocy that the ordinary stimuli fail to produce the usual result, and the mother mistakes for 'goodness' what is merely dullness.

In later infancy and in early childhood the tendency to rhythmic movements which is so often to be seen in older imbecile children becomes noticeable. The child will sit rocking the trunk to and fro with only slight pauses for hours together, or turning the head slowly from side to side as in negation; and sometimes such movements are accompanied with a crooning sound. These rhythmic movements in young children are not necessarily indicative of imbecility, they are seen in children who are in nowise mentally deficient, but, as I have pointed out elsewhere, they are even then apt to be associated with some eccentricity or oddness of temperament.

Akin to these movements is another curious habit of some imbeciles, the passing slowly to and fro of the outspread fingers in front of the eyes; this is done in such a position usually that the fingers are between the face and the window or other source of light. The children who do this have, I think, always some considerable defect of vision, and the explanation which Mr. Donald Gunn, formerly ophthalmic surgeon at the Children's Hospital, Great Ormond Street, has suggested to me seems a reasonable

one : the child being unable, owing to its defective sight, to see and take pleasure in the complex details of its surroundings, derives pleasure from the simple alternation of light and darkness produced by the fingers ; perhaps also in part from the feeling that he controls this phenomenon himself.

Classification. Various attempts have been made to classify imbeciles, and I know of no scheme that is wholly satisfactory. Even such a rudimentary distinction as congenital or acquired is impossible in many cases. A common history is such as this : Amy W., aged ten months, the child of first cousins, had many convulsions from the age of three days until the age of three months ; she has had none since, but now at the age of ten months it is clear that she is idiotic. Who shall say whether the idiocy has resulted from the convulsions or whether the convulsions were only a symptom of a congenitally ill-developed brain ? I suppose there can be no doubt that in the large majority of cases imbecility is due to developmental disorders during intra-uterine life, but there is a small residue of cases in which it is undoubtedly due to causes acting after birth, and it is at least possible that it may be due in some cases to causes acting during birth.

For practical purposes I think a classification based as far as possible upon etiology is the most useful, and for this reason it seems well to adopt some such grouping as that proposed by Dr. W. W. Ireland, without attempting any strictly logical classification. One may recognize the following groups as developmental imbecility.

Varieties.	*Number out of the total* 350.
Genetous	177
Mongolian	77
Microcephalic	22
Cretin	10
Hydrocephalic (sometimes acquired)	1
Spastic (sometimes acquired)	26

The following may be classified as acquired imbecility :

Varieties.	*Number out of the total* 350.
Eclamptic	10
Epileptic	20
Inflammatory	1
Syphilitic mental degeneration (general paralysis)	4
Amaurotic family idiocy	0
Traumatic	2
Hypertrophic	0

It will be seen that if we omit the hydrocephalic and spastic

cases, most of which should probably be regarded as developmental, no less than 288 out of the 350 cases were developmental, and I suspect that these figures understate the proportion, for cases classified as eclamptic or epileptic are open to much uncertainty as to the acquired or developmental character of the mental defect.

The above grouping calls for some explanation.

The term *Genetous* does not mean merely congenital—microcephalic idiocy is congenital, but it is not genetous in the sense here intended. Dr. Ireland devised it for the grouping together of the residue of cases which cannot be classified in a precise way on pathological grounds. I have followed him in this except that I have regarded the Mongols as a separate group on account of their well-defined clinical characteristics.

It will be seen that about half the cases of imbecility are of the genetous variety: simple developmental imbecility it might be called, for all we can say of the child is that he is born idiotic owing to some unexplained failure in the development of the brain. There is nothing distinctive in the appearance or conformation of imbeciles of this group; great stress has been laid on the frequent occurrence in them of the so-called 'stigmata of degeneration', the high, narrow or V-shaped palate, the ill-formed external ears, abnormalities in shape or size of the skull, and congenital malformations of all kinds.

Undoubtedly these are of some value as confirmatory evidence where there are indications of mental deficiency; but it would be very unwise to lay any great stress upon them, for they are frequently met with in association with perfectly normal intellect.

Microcephalic idiocy is merely a clinical grouping, and even as such is ill-defined: as the name implies, extreme smallness of the cranium is its distinguishing feature, and in a well-marked case the type is obvious enough, the small cranium contrasts strangely with the relatively large face below it, reminding one of a pear with the narrower end above; but what degree of smallness of the head constitutes microcephaly? More than half the cases of imbecility, including all varieties, have the head below the normal size, but very few of these would be classified as microcephalic. In a consecutive series of 100 imbeciles I found that in 69 the maximum circumference of the head was below the normal average for the age; in 43 of these the head was not less than an inch below the normal; only in 11 was the circumference above the normal.

I find in my own notes that I have usually classified a case

as microcephalic when the maximum circumference was 2 inches or more below the normal average; but the measurement alone is not, I think, sufficient. I have seen children whose head must have been fully 2 inches below the normal average in circumference, but it was not out of proportion to the face or to the rest of their physical development, and the child's mental condition was perfectly normal; it is the disproportion between the size of the cranium and the size of the face which should distinguish the microcephalic imbecile. The vault of the cranium is arrested in its growth, whilst the other parts of the skull continue to enlarge; consequently, as the child grows older, the forehead comes to slope backwards in a characteristic manner.

Even when such disproportion is present the degree of intelligence does not always correspond to the size of the cranium. My colleague, Dr. Thursfield, showed me a girl aged $4\frac{10}{12}$ who was attending school and whose intelligence seemed to be almost normal (I thought she was a little too babyish for her age); the cranium measured only $15\frac{1}{2}$ inches (4 inches below the normal !) in circumference and was strikingly small in proportion to the face, so that the child had the typical appearance of microcephaly.

Some degree of spasticity of the limbs is not infrequent with microcephalic imbecility. I noted this association in five out of twenty-one cases.

To classify an imbecile as microcephalic is merely to describe the appearance of the patient, it tells us nothing of etiology or pathology, for there are many different causes which may lead to this arrest in growth of the brain, and even the gross changes are by no means always the same in the brain of the microcephalic idiot. In some cases the arrest of development affects the whole brain uniformly, in other cases the arrest seems to affect only parts of the brain; for instance, in one case I found the occipital lobes normal on macroscopic examination, whereas the parietal lobes were so rudimentary as to be only just definable and the convolutions of the frontal region were extremely minute. In some cases the only abnormality found has been an unduly small brain with the convolutions much less complex than normal, in others there is obvious sclerosis of all or part of the convolutions. Sometimes in spite of the small size of the head hydrocephalus has been found in the microcephalic brain.

Nor if we turn to etiology has microcephaly any more distinctive a significance. I have seen well-marked microcephaly associated with congenital syphilis, and the late Dr. Ashby

recorded a case in which post mortem examination of a syphilitic infant with microcephaly showed some endarteritis narrowing the lumen of the vessels; but probably only a very small minority of cases of microcephalic imbecility are due to syphilis. I had under my observation twin sisters, both microcephalic and both spastic; there was no evidence of syphilis. Dr. Ireland quotes a record of four microcephalic idiots in one family and of five in another.

In most cases of microcephaly the fontanelles close very early. I have found the anterior fontanelle in such a case completely closed at the age of six months, but it is clear that this is not the cause of the arrested development of the brain, for I have found the fontanelle still open at the age of two years and five months in a case of microcephalic idiocy. It is but a few years since operations were done for the relief of this form of idiocy by removing a coronal strip of bone from the vault of the cranium with the idea of allowing the brain freedom to expand: the operation was based on erroneous pathology. The hindrance to the development of the brain lies not in any mechanical restriction by too early closure of sutures, but in some fault of intra-uterine origin, in some cases perhaps an interference with the blood-supply to the developing brain, in other cases perhaps the influence of some poison carried in the maternal blood to the growing structures of the embryo or fœtus.

Hydrocephalic idiocy requires no explanation. In some cases it is found where the hydrocephalus is of congenital origin, and presumably the impairment of mental function also dates from birth; in other cases the hydrocephalus has been acquired, usually as the result of cerebro-spinal or posterior basic meningitis, and the child previously intelligent has become mentally defective from the pressure of the ventricular distension upon the cortex. It is, however, surprising how seldom mental deficiency results from this cause; even with enormous hydrocephalus where post mortem shows extreme thinning of the cortex, the mental function is sometimes injured very little if at all.

Spastic or paralytic idiocy is the condition which is so often associated with the cerebral palsies of childhood; and with no form more often than with spastic diplegia. Both spastic diplegia and spastic paraplegia are usually of congenital origin, so that the mental deficiency in such cases must be considered as belonging to the developmental group, but in the more common form of spastic cerebral palsy, infantile hemiplegia, the

condition is usually of post-natal origin, and the accompanying mental deficiency will then fall into the group of acquired mental defects. In this latter variety of spastic palsy, especially when it is of post-natal origin, the mental defect is seldom very marked, the child is apt to be too facile, too easily pleased and too ready to make friends with strangers, but any degree of mental defect which would be described as idiocy is quite the exception, whereas in spastic diplegia the mental affection is usually considerable, amounting sometimes even to complete fatuity.

With spastic paraplegia the degree of mental defect is less as a rule than with spastic diplegia, but more than with infantile hemiplegia.

I have already mentioned that microcephaly is often associated with some degree of spasticity. It would seem indeed as if the pathological process which arrests the development of the brain in the microcephalic idiot is in some cases very similar to, if not identical with, that which occurs in the spastic paralysis of childhood in which the cranium is usually below the normal size, and the naked-eye appearances of the brain are similar, sclerosis and shrinking of convolutions being prominent changes in both conditions.

Eclamptic and epileptic idiocy. Turning now to the varieties of acquired imbecility, I have adopted Ireland's distinction between eclamptic and epileptic imbecility: meaning by eclamptic that imbecility which may be produced by a severe bout of convulsions, and by epileptic the idiocy which results from chronic recurring epilepsy: in the former case the mind is wrecked by a single storm, in the latter it degenerates owing to a series of shocks.

I have already referred to the extreme difficulty which there is in determining whether idiocy is the *result* of either of these two causes, or is merely an associated symptom of some congenital abnormality of brain. Probably in many cases where imbecility in a child is attributed to either of these causes, the abnormal condition of the brain was really developmental and therefore congenital.

Amongst my own notes I can find very few instances with satisfactory proof that idiocy resulted from a bout of convulsions; to prove such a connexion it would be necessary to have clear evidence that the child was mentally normal before the convulsions, and this is just what it is most difficult to ascertain. The cases I have grouped as eclamptic were only possibly of this

nature; several had had many convulsions during the first few weeks of infancy, e.g. Fred M., aged two years, was much asphyxiated at birth and had convulsions from birth till four months old; since then there have been no more convulsions, the child is now a fatuous idiot. In these cases with convulsions during the first few months after birth it cannot be denied that the mental deficiency may have resulted from the convulsions, but it is impossible to prove.

In the following case there was some evidence that the child had had some intelligence before the convulsive attack.

Ernest T., aged $2\frac{3}{4}$ years, had begun to stand at nine months and to talk at twelve months old; he was, however, only using single words at the age of $2\frac{1}{2}$ years, and had never acquired clean habits, when a severe convulsion occurred which lasted four hours, and was followed by much screaming for a fortnight. Since this time he has been completely idiotic, making meaningless noises and taking very little notice.

A more convincing case is one which I have described elsewhere (p. 660) in which the child had learnt to walk and talk at the usual age, and had seemed perfectly bright and intelligent up to two years and eleven months, when a bout of convulsions followed by others occurred, and the child became idiotic.

In estimating the part played by convulsions or by epilepsy in the causation of idiocy, it must be remembered that these are common in cases where it is quite certain that the child was mentally defective before they began. Out of 239 imbeciles I found that 101 had had convulsions or epileptic attacks at some time or other, mostly in infancy; but only in 30 of these 101 cases did it seem likely that the mental defect might be the result either of the convulsions or of the epilepsy.

Epilepsy is much more often productive of mental defect when it occurs in infancy than when it occurs in later childhood; and I fancy from my own observation that petit mal in an infant is of more gloomy prognosis in this respect than major attacks. The infant who has sudden attacks of momentary loss of consciousness, with the head falling forward on the chest, or with a momentary spasmodic flexion of the trunk, almost always becomes more or less mentally defective. Such attacks are, I think, certainly precipitated by the worry of dentition in infants predisposed to epilepsy, and they sometimes cease when dentition ceases; but the mental ruin is irreparable; the child may become decidedly brighter when the attacks cease, but I have never seen restoration to normal intelligence.

Inflammatory idiocy, that is, idiocy depending upon en-

cephalitis or meningo-encephalitis, is a condition of post-natal onset. In some cases it is probably identical in its pathology with those cases of infantile hemiplegia in which as the result of some infection an acute polioencephalitis occurs affecting the motor area. If this process be more extensive, there will be not only spastic hemiplegia but also mental deficiency, as sometimes happens; but if the encephalitis be confined to other parts of the cortex, the mental functions may be affected alone. It is noteworthy that just as infantile hemiplegia sometimes occurs directly after one of the specific fevers, so imbecility occasionally comes on after measles or some other specific fever, and is perhaps in such cases due to encephalitis. I have very little doubt that such an encephalitis occurred in the following case.

A boy aged $1\frac{3}{4}$ years, a bright healthy child, was seized with convulsions whilst out in his perambulator; the temperature was raised, and a few hours later reached 105°; in spite of vigorous treatment with ice-bags, bromide, chloral, &c., the child remained for more than a week in a semi-comatose, convulsive condition, with some general rigidity and occasional clonic spasms; lumbar puncture gave no information; after several weeks the rigidity passed off, the knee-jerks were normal, and the plantar response was flexion; the child began to take some notice of surroundings, but there was very little return of intelligence; the child remained completely idiotic, and when seen again four years later could not have been distinguished from a genetous idiot.

Traumatism is probably invoked as an explanation of mental deficiency far more often than the facts would justify, but rare though they may be, cases do occur in which a blow upon the head results in mental defect.

Cecil A., aged $2\frac{1}{2}$ years, fell off his chair twelve months before admission to the hospital, and struck the back of his head against the fender. From that time he gradually lost the power of speech and walking, which he had previously acquired, and ten months later he began to have 'twitchings' with tenderness over the back of the head and much excitability. During the six weeks preceding admission he had fits with increasing frequency until about thirty occurred per diem. He now seemed dull, with evident mental deterioration, he was quite conscious, but took no interest in his surroundings; his temperature for three weeks after admission was raised and irregular, then it became normal for nearly three weeks, rising again to 103° and 104° for a week before death. During the last few weeks of life the child became rigid and apparently blind, and there was frequent vomiting. The fundus oculi was normal. Post mortem examination showed all over the occipital and parietal regions of both sides extensive thrombosis of veins, which were hard like whipcord and pale; the thrombosis was evidently not of recent occurrence, but it was uncertain whether it could have occurred so long as fourteen months before death, the date of the fall. There was no gross meningitis, the pia mater on the vertex and under surface of the frontal lobe was slightly

more opaque than normal, the convolutions were markedly wasted, so that the sulci gaped between them, the superficial part seemed unusually soft, while the deeper part felt much harder than normal, and was evidently sclerosed; the only part of the brain which seemed normal was the cerebellum, which looked unusually large by contrast with the small, wasted cerebrum.

Syphilitic mental degeneration. Acquired imbecility, or more properly speaking mental degeneration, as a result of congenital syphilis is now considered identical in some cases, if not in all, with general paralysis of the insane, and is described as juvenile general paralysis. I confess I do not altogether like the term 'general paralysis' applied to these cases, for it has come to suggest a chronic affection, whereas, as I shall show, a similar pathological change may occur as an acute disease in children. I have notes of seven such cases which ran the ordinary chronic course: four girls aged respectively four, four and a half, nine, and about eight years, and three boys, two aged seven years, and one aged ten years at the time of the onset, all showing well-marked evidence of congenital syphilis. The history in these cases is this: usually after the age of five years, at some time in the later half of childhood, the child becomes dull and slow, then perhaps complains of headache, and begins to lose his memory, the speech also becomes slow and thick, and epileptiform attacks, if not the earliest symptom, are likely to occur at some period of the disease, the legs gradually become weak and the gait uncertain, the pupils may be unequal; in two of my cases the sight was impaired from optic atrophy associated with choroiditis. The knee-jerks in some cases are much increased. Sooner or later some rigidity of limbs appears, the child becomes more and more demented, is unable even to sit up and lies in bed becoming gradually emaciated, and usually within three or four years after the onset dies of exhaustion.

The brain in these cases shows opacity and slight thickening of the pia mater, sometimes adhesions of the dura mater, some sclerosis and atrophy of the convolutions and often some dilatation of the ventricles; there is apparently a meningo-encephalitis.

In the seven cases to which I have referred, the affection ran a chronic course, only one was under observation until death at the end of two years, in the others the affection had already lasted periods varying from six months to two years. But I doubt if the disease is always of such long duration. I have seen one case in which exactly similar pathological changes were found after seven weeks' illness, and another in which

the symptoms were of only thirteen days' duration. I shall quote the description I have given elsewhere [1].

A boy aged twelve months, who had had snuffles at the age of five weeks, with severe thrush and subsequently some rash about the buttocks, and whose eyes showed choroido-retinitis and opacities of the vitreous, was admitted to hospital for convulsions with rigidity and wasting; he died after seven weeks' illness. I found the dura mater adherent to the pia mater chiefly at the vertex, with small hæmorrhages on the inner side of the dura, which was much thickened; the pia was slightly thickened, especially in the neighbourhood of the blood-vessels on the surface of the brain; the convolutions showed a dirty yellowish coloration and were hard and in places shrunken; the cerebellum was normal.

Wilfred S., aged ten years and eleven months, was born with pemphigus, the details of his infancy otherwise were unknown. Later he had interstitial keratitis. Ten days before admission to hospital under the care of Dr. Lees he had severe frontal headache and vomited, some days later he had a fit and another on the day of admission. He died three days after coming into the hospital.

The pia mater was thickened, grey, fibrous, and opaque over the left side of the vertex of the brain and also about the quadrate space and the sylvian fissures at the base; there was some sclerosis and wasting of one convolution in the right frontal region. The pia mater down the posterior surface of the cord was somewhat thickened. There was no exudation of lymph; in fact, none of the ordinary appearances of acute meningitis.

I quote these two cases to show that the clinical course of the disorder known as 'general paralysis' may be very acute. No doubt the pathological process may be, as in the former of the two cases just mentioned, of much longer duration than the clinical symptoms; it is only in those that last longer that the dementia becomes a prominent symptom. Dr. Ashby reported a case in which complete idiocy resulted from meningo-encephalitis in a syphilitic infant; the disease began with convulsions at eight months old and ended fatally after lasting six months.

With regard to the two remaining groups of acquired imbecility I shall say very little, for they are extremely rare.

Hypertrophic idiocy. Under the name of hypertrophic imbecility have been recorded cases in which the child has been normal until a year or two old, when he has begun to suffer with headache and has become progressively duller, and has died usually before puberty. In other cases the course has been similar except that the child has always had a large head and been backward; on post mortem examination the brain has been found exceptionally large and heavy with no hydrocephalus but with increase of neuroglia. I have seen several imbeciles with heads considerably above the normal size, cases which I

[1] Power and Murphy, *System of Syphilis*, i, p. 324.

thought might be of the hypertrophic variety; but I have satisfied myself from clinical and pathological observation that it is not possible to exclude the presence of hydrocephalus merely from the shape of the head, so that without post mortem examination the diagnosis of this variety cannot be made with any certainty.

Amaurotic family idiocy I have only twice seen. One case was that of a Jewish infant who was one of a family of twelve children, of whom four others had died with this disease. The infant, whom I saw with Dr. J. M. Twentyman, had been healthy for several months after birth and then had gradually become more and more dull and fatuous, and then rigid in its limbs; the eyes showed the characteristic cherry-red spot in the macular region of the fundus. The infant died when it was two years old.

The second was also in a Jewish child, a girl, who at the age of four months was noticed to be weak in the back. This weakness increased slowly, and sight seemed to be lost. Some spasticity had appeared by the time the child was eighteen months old. The typical cherry-red spot was easily seen at the macula lutea in both eyes. The child's mental condition seemed to be already completely fatuous by the time she was eleven months old. She was the first child of the family.

The course of this disease is invariably progressive towards a fatal end at about two years of age. Its morbid anatomy has been investigated carefully by several observers, but beyond the fact of degenerative changes in the brain, nothing has been found to explain its origin; its limitation almost exclusively to the Jewish race, and its occurrence in several children of a family, but only in one generation, are very remarkable.

Causes of Mental Deficiency

Apart from the possible causes of mental defect which I have already mentioned in connexion with particular types of imbecility, there are some more general causes which are worthy of consideration.

Alcoholism in either parent is, I believe, a potent cause of mental deficiency in their offspring. It is difficult, however, to prove the connexion by any statistics. I inquired carefully from the mothers in fifty cases; in fourteen of these the father was said to be drunk more or less frequently; the complete absence of intemperance in the mother is no doubt unreliable as she was the person questioned. Tredgold [1] obtained a history of alcoholism 'in the antecedents' in 46·5 per cent. of his 150 cases, but noted a point which is probably of importance, that

[1] *Archives of Neurol.*, vol. ii, p. 328.

there was also in most of these cases a family history of insanity or other neuropathic taint. Some of my cases showed parental alcoholism with no other evidence of neuropathic inheritance.

There is experimental evidence[1] that alcohol in the mother's blood not only reaches the fœtal circulation, but may cause malformations in the fœtus; one may conjecture that if alcohol can modify the gross physical structure it must still more readily cause the fine changes in the brain which are sufficient to cause mental defect.

The influence of paternal alcoholism is not capable of explanation, but as the finest peculiarities of feature can be transmitted from the father to the child, it seems likely enough that any toxic condition in him may affect the development of the child.

Syphilis plays, I think, a larger part in the production of idiocy than has been supposed. Amongst my 350 cases of mental deficiency, there were 13 cases of undoubted congenital syphilis and 4 others were children of probably syphilitic parents; so that syphilis was a possible factor in the causation of mental deficiency in nearly 6 per cent. of the cases. The results of the Wassermann test have been so widely different in the hands of different observers, that it is difficult to know what value to attach to them: Dean found 15 per cent. positive; Findlay and Robertson found 59 per cent. positive.[2] To state the proportion of cases of mental defect showing congenital syphilis, however, is only to compare the frequency of syphilis with that of other causes of idiocy; it is at least as important to ascertain the frequency of mental defect in congenital syphilis. Out of 142 consecutive cases of congenital syphilis I found 10 showing either congenital idiocy or subsequent mental degeneration, a proportion of 7 per cent.

The congenital mental defect produced by syphilis is not always of one type: amongst the syphilitic cases mentioned, most were 'genetous', but two were Mongols, one was paralytic, one was microcephalic.

The occasional association of parental syphilis with Mongolian imbecility in the offspring, and the still more rare occurrence of actual evidence of syphilis in the Mongol imbecile himself, are interesting as throwing some light upon the relation of syphilis to imbecility. It is quite certain that in the large majority of cases Mongolian imbecility occurs where there is no reason whatever to suspect syphilis either in parent or child, and the small, too simply convoluted brain suggests simply a hypoplasia of brain as if Nature had lacked the energy to make a brain of the usual size and complexity. And, indeed, this suggestion may

[1] *Vide* Ballantyne's *Ante-natal Pathology: The Fœtus*, pp. 273, 274.

[2] *Glasgow Med. Journ.*, Dec. 1914.

be nearer the truth than its untechnical form would imply, for it is well known that this particular type of imbecility occurs usually in children who are born when the mother is already approaching or past the age of forty years, and is therefore nearing the end of her reproductive period; the Mongolian imbecile is an exhaustion product.

It seems probable, therefore, that syphilis, when it occurs in such cases, acts not by producing any specific syphilitic disease in the child or in its brain, but by impairing the mother's power of reproduction, just as advancing age or even the strain of domestic anxiety may do. The influence may thus be indirect. In some cases, however, it is certainly direct. As Dr. Ballantyne [1] has pointed out, a poison acting upon the growing structures in a very early stage of intra-uterine life tends to cause malformations or abnormalities of development: the same poison acting upon the fully or almost fully developed structure will produce diseases like those of post-natal life. I have already referred to a case in which microcephaly in a syphilitic infant was associated with narrowing of the blood-vessels by syphilitic endarteritis.

In some cases of imbecility with syphilis the occurrence of actual syphilitic disease of the nervous system at some period of intra-uterine life is, I think, suggested by examination of the fundus, which often shows syphilitic choroiditis; and this in some infants is almost certainly of intra-uterine origin.

And here let me emphasize the importance of ophthalmoscopic examination in determining the cause of mental defect. I have notes of cases in which one might have puzzled in vain over the problem had not examination of the eyes revealed a retino-choroiditis, which in a young child may probably be taken as proof positive of syphilis. Opacities in the vitreous, and remains of former iritis, may point in the same direction. I have already mentioned the red cherry-tinted discoloration of the macula lutea which is to be seen in amaurotic family idiocy. I have once or twice found coloboma of the choroid in imbeciles, which was at any rate interesting as indicating that the mental condition was also due to developmental defect, and could not be referred to any asphyxia at birth or to any of the causes of acquired mental defect.

Asphyxia. That asphyxia at birth can produce mental deficiency I have little doubt, but it is impossible to prove. Out of 133 cases in which I investigated this point 25 had suffered with asphyxia at birth.

It seems that in some of these cases the mental defect is due to hæmorrhage damaging the brain substance, just as paralysis

[1] *Ante-natal Pathology: The Embryo*, chap. i.

may be due to hæmorrhage from venous congestion, for instance, in whooping-cough. If this were so one would expect that in some cases paralysis might be associated with the mental defect due to this cause; and this does happen. There was spastic paralysis of limbs in three of the twenty-five cases mentioned, and in two out of these three cases it was specially noticed that the asphyxia had been very severe.

But without hæmorrhage it seems quite likely that prolonged non-aëration of the blood may have a toxic effect upon the cells of the brain, and at this early age may so affect their function that idiocy results.

Instrumental labour. The relation of asphyxia in the newborn to idiocy is closely connected with another possible factor at the time of birth, namely, instrumental labour. It is natural enough that a mother seeing her infant's head deeply indented and perhaps severely lacerated by forceps should attribute any subsequent defect to injury from this source; but is there any evidence to support this view? It is clear that certain forms of imbecility cannot possibly be due to this cause, for instance, Mongolism, cretinism, hydrocephalic idiocy, traumatic idiocy due to post-natal traumatism, amaurotic imbecility, and syphilitic mental degeneration; but in the genetous, paralytic, and microcephalic varieties, and in that very doubtful group of the cases in which convulsions or epilepsy may be the cause or may be the manifestation of the abnormal brain condition, instrumental labour might conceivably be the cause.

If it could be shown that instrumental labours were much more frequent in these varieties of idiocy than in cases of normal intelligence, this would at least be strongly suggestive; and my own figures show—I must say contrary to my own preconceived idea—that instrumental labour figures with such remarkable frequency in the history of imbeciles of these classes, that the possibility of mere coincidence is, I think, quite excluded. I investigated 136 cases (including 98 genetous, 13 spastic or paralytic, 10 microcephalic, 8 epileptic, and 7 eclamptic): no less than 28 out of the 136, that is 20·5 per cent., were instrumental labours (21 out of the 98 genetous, 4 of the 13 spastic, 1 microcephalic, 1 eclamptic and 1 epileptic).

The significance of these figures is seen on comparison with statistics of children of normal intelligence. At the Children's Hospital, Great Ormond Street, excluding all cases of mental deficiency and cerebral palsy, I found that in 100 consecutive cases there were only 3 instrumental labours, whilst amongst cases in the Maternity Department of King's College Hospital, amongst 1,214 children born, only 2·2 per cent. were delivered by forceps.

Amongst the well-to-do the proportion of cases of instrumental delivery of children of normal intelligence would probably be much higher than this; so that in comparing idiots with normal children with reference to the effect of instrumental labour, it is very necessary to compare children of the same social class; in the statistics on this point, therefore, I have included only cases seen in hospital practice. Although these figures go a long way to prove that instrumental labour is a much more important factor in the production of imbecility than has been supposed by some observers, it must not be assumed that traumatism is necessarily the *modus operandi* of the instrumental delivery in producing mental defect. It must be remembered that the labour which necessitates the use of forceps is also one which is likely to result in more or less severe asphyxia, and this rather than traumatism may be the cause of the mental condition; in nine out of the twenty-eight cases of instrumental labour there was also more or less severe asphyxia.

Consanguinity of parents is popularly supposed to be a potent cause of idiocy in the offspring. I recently read in a lay journal that all children of first cousins were idiots! Dr. Tredgold[1] says in regard to the marriage of first cousins, 'as a cause of idiocy I do not think this need be seriously thought of.' Probably the truth is somewhere between these extremes. It is likely that marriage of first cousins has some influence in producing abnormalities of one kind or another in their offspring. My colleague, Dr. A. E. Garrod[2], has pointed out that in cases of alkaptonuria the proportion of families showing first-cousinship of parents is nearly 50 per cent. (8 out of 17 families). He quotes also Ascoleo as finding that in 5 out of 24 families in which albinos occurred the parents were first cousins.

It may be that consanguinity of marriage only increases the chance of reproducing abnormalities which have already been present in previous generations of the family, and that when the offspring of first cousins are congenitally imbecile this is merely due to neuropathic heredity in the sense that consanguineous marriage increases the chance that a neuropathic taint, like any other abnormality, may reappear in the offspring: a view which would be in accordance with Mendel's law of heredity.

This view, however, would not in any way diminish the risk of consanguineous marriage; indeed, considering the large proportion of families in which there is neuropathic taint of one sort or another, it would rather tend to emphasize the risk of first-cousin marriages.

[1] *Archives of Neurol.*, vol. ii. [2] Croonian Lectures, *Lancet*, July 4, 1908.

To estimate the influence of consanguinity we must either take a large number of first-cousin marriages and ascertain the proportion of these which resulted in idiocy, and then compare with these figures similar statistics in the case of non-related parents, or we may adopt the less satisfactory but more feasible method of comparing the proportion of consanguineous marriages amongst the parents of idiots with the proportion amongst the parents of families in which there is no idiocy. It is useless merely to quote the proportion of cases of idiocy in which there was consanguinity of parents, with no standard of comparison, for even if it were assumed that the consanguinity was causal, this proportion would only tell us how frequent consanguinity is in comparison with other causes of idiocy; it would tell us nothing whatever about the degree of risk of idiocy from consanguineous marriage.

Dr. Karl Pearson's statistics[1] taken from observations collected by Dr. Garrod, at the Children's Hospital, Great Ormond Street, showed that amongst families of the hospital class, that is, the poorer class, there were in 700 families only 0·86 per cent. marriages of first cousins. Taking figures exclusively from the same class, indeed most of them from the same hospital, I found that amongst 166 idiots the family history showed seven first-cousin marriages, that is, 4·2 per cent. If this disproportion is confirmed by larger statistics, it can hardly be doubted that first-cousin marriages do play a part in the production of idiocy.

I must point out that it is very necessary in comparing statistics on this point to compare only those which are taken from families in the same social class, for it is well ascertained that first-cousin marriages are considerably more frequent amongst the well-to-do than amongst the poorer classes. It may be suggested that on this account idiocy should be relatively commoner amongst the well-to-do than amongst the poor. No statistics are available on this point, but it must be remembered that other factors such as alcoholism and syphilis are probably much commoner amongst the poor than amongst the well-to-do, and this would tend to counterbalance the affect of consanguinity on the relative frequency of idiocy in the two classes.

Place in family. There is a factor in the production of imbecility which has attracted but little notice except in the cases of Mongolism, namely, place in family. It is well known that Mongolian imbecility occurs with much greater frequency in children born when the mother is approaching the age of the

[1] *Brit. Med. Journ.*, 1908, i, p. 1395.

menopause than at an earlier age, and therefore this type of imbecility is usually seen in a child who comes late, if not last, in a family : but it is at least as remarkable that in other types of imbecility it is the first child of a family who is specially liable to be affected : no less than 80 out of 177 consecutive imbeciles (excluding all Mongols) were first-born children. (In these figures I have been careful to describe as first-born only children who were the result of first pregnancies ; I have not called a child first-born if it was preceded by a miscarriage.)

An explanation which suggests itself at once for this special incidence on the first-born is the greater frequency of difficult labour and also of asphyxia in the first-born than in later children ; but my own series of cases shows that although in some of the first-born labour had been instrumental and in some there had been asphyxia, in most of them there was no history of either difficult labour or asphyxia. This explanation is also made the less probable by the fact that some abnormalities of development which are more liable to occur in the first pregnancy than in the later ones could hardly be related to any influence acting at the time of birth only ; a striking illustration of this is so-called congenital hypertrophy of the pylorus which, whether it be due to abnormality of nervous system or to primary abnormality of muscular development in the pylorus, affects first-born children with remarkable frequency (nearly half the cases).

The fact that first children and late or last children are specially apt to be affected by abnormalities of development, mental and otherwise, seems most naturally explained by supposing that in the one case the reproductive function in the mother has not yet become fully established, while in the other it is already failing. The fault in these cases seems to be one of maternal reproductive function, not necessarily of general health or strength ; the mother whose first child is imbecile is not usually a very young mother nor one whose health is delicate. I inquired into the age of the mother in sixty-nine of the first-born imbeciles ; only five of these were under the age of twenty years, including two just under the age of nineteen, when the child was born ; most of them were between twenty and thirty.

Nor is the mother whose late-born child is a Mongol necessarily in any way unhealthy. The mothers of Mongols are commonly healthy women, their failure is, so to speak, merely physiological ; the reproductive power is flagging before it ceases altogether. It is probable in both cases that any cause

which tends to exhaust a mother's general vigour will increase the likelihood of imperfect reproduction : it would be difficult to offer any scientific proof of this, but I am strongly inclined to think from my own clinical observation that great stress of domestic anxiety, no less than exhaustive illness, in the mother before or during early months of pregnancy may result in imbecility in the offspring.

Relative age of parents. Great disparity in the age of the parents is a point to which the laity attribute considerable importance in the production of imbecility as well as of other defects in the offspring. Does the fact that the father is much older than the mother in any way prejudice the child's health ? I can give little more than impressions by way of answer to this question. Certainly in comparison with other causes of imbecility it must be a very infrequent cause if it is one at all. I investigated the relative age of the parents in 217 cases. In 130 the difference of age was not more than five years (in 85 the father was the older, in 45 the mother), in 36 the age of the parents was equal ; in 32 cases there was a difference of five to nine years ; in one case the mother was eleven years older than the father ; in 13 the father was older than the mother by ten to fourteen years, in one case by twenty-six years. But these figures do not show what proportion of marriages between persons of very unequal age results in imbecile offspring ; this proportion I have been unable to ascertain, but it must be known before we can judge whether such marriages have any real influence in producing imbecility of offspring.

My impression is that where the father is much, e.g. twenty years, older than the mother the children are very apt to be weaklings in physical constitution, and I should expect therefore that disparity of age in parents might cause also defect of cerebral development ; but I know of no proof that this is so.

I have discussed the various possible factors in the causation of imbecility at some length because parents whose children are mentally defective are generally anxious to know the cause. The scientific mind is less ready to assign causes than is the mind untrained in science, but none the less it is well that we should know how much, or rather how little, it is possible to determine with reference to this point.

Family incidence. There is another very practical question which is closely related to etiology, and which I have many times been asked by the parents of an imbecile child: if after the birth of the imbecile child they have other children, is it

likely that any of these will also be imbecile ? I think it may be said without hesitation that with certain exceptions to be mentioned below, the risk is so extremely small as to be a negligible one. I have notes of seventy families in which an imbecile child had younger brothers or sisters : only in a single instance was one of these younger children also an imbecile. In one other family there were two imbeciles, both genetous, but these were twin children (the father was alcoholic).

It might be supposed that in the case of Mongolism particularly the exhaustion of the mother would be still more likely to produce Mongolism in a subsequent child, but although I have notes of several cases in which a Mongol was last but one of a large family, I have not seen any instance in which there were two Mongols in a family.

The exceptions to this generalism are amaurotic family idiocy and epidemic cretinism ; the former of these is almost certain to occur in several children of a family, the latter is commonly seen in several members of a family when cretinism is endemic, but not in this country.

Prognosis. I know of no more difficult question in prognosis to answer with kindness and tact and withal with honesty, than the anxious, almost imploring question of a mother who is trying to persuade herself, against the evidence of her own senses, that her child is not an imbecile, when it is patent to the doctor and perhaps to every one except the parents that the child is an imbecile : 'Will he grow out of his backwardness ?'

Or, perhaps worse still, the parents have not even suspected the possibility of imbecility, and the child of twelve or fifteen months is brought only because he cannot sit up, and this has been attributed to his back being weak !

What is the medical man to say ? The knowledge that their child is an imbecile comes as a terrible shock to the parents : it means the hopeless and irreparable ruin of some of their most cherished hopes, and therefore a medical man may well consider carefully how he may cause as little distress as possible. In the first place the choice of words is not a matter of indifference ; the terms idiot and imbecile are usually to be avoided altogether, parents are much less distressed to be told that their child 'will always be backward' or 'mentally defective' than to hear that he will 'always be an imbecile'.

When the parents are aware of the child's mental deficiency, the point upon which they usually expect an opinion from the medical man is the degree of mental development which the

child may be expected to attain. Such questions as the following arise directly mental deficiency is detected and therefore almost always before the child is three years of age: Will he walk? will he talk? will he be able to take an ordinary place in society? will he be able to earn his own living?

Obviously each individual case must be judged on its own merits; for instance, the outlook is far worse in every respect in the drivelling fatuous idiot who takes no notice at all than in the child who recognizes its parents and toys and pictures and takes an interest in its surroundings, albeit in a somewhat silly manner; but I shall venture to state certain generalisms which may, I think, be of value in this connexion.

Almost all feeble-minded children of whatever degree of imbecility make some progress in mental development during the first few years of life; very few mentally deficient children fail to walk, though walking may not be acquired until the child is four years old or more. In the very large majority of mentally deficient children, speech is acquired sooner or later, though it may always be very imperfect; most imbeciles, even when speech is not acquired till three or four years old or later, can be taught to be clean in their habits, and learn to make some sign when they want to pass urine or fæces. There is no likelihood that the child whose mental deficiency is recognizable in the first two or three years of life will be able to take an ordinary place in society, or to earn his own living.

While the child is yet in its earliest years, it is only right to comfort the parents with the assurance that there will almost certainly be some progress in mental development within the next few years; the extent of this progress cannot be foreseen, it will certainly fall short of the normal, but it is sometimes considerably more than the child's appearance might lead one to expect.

As to the acquirement of the power of walking, I think it may safely be said that unless the child is extremely fatuous it will almost certainly learn to walk, though this may not be accomplished until the child is five or even six years old.

Dr. Caldecott kindly made some investigations for me at Earlswood Asylum upon this point, and ascertained that out of 443 cases only four failed altogether to walk, and fourteen could walk only with assistance; so that only 4·06 per cent. failed to attain full power of walking.

The acquirement of speech is more doubtful. Dr. Caldecott has investigated this point also, and furnished me with statistics

showing that out of 442 imbeciles, 355 could talk intelligibly; 87, that is, 19·6 per cent., failed to acquire speech. Fully four-fifths therefore of imbeciles may be expected to talk.

One is sometimes asked another question in prognosis: will the child live? Generally speaking the chance of survival of a mentally defective child is, I think, smaller than that of normal children; the feeble-minded child offers but feeble resistance when attacked by disease; he is, I think, specially prone to succumb to pulmonary disease, such as bronchitis or broncho-pneumonia or tubercle. In one variety of imbecility, Mongolism, it is probable that a large proportion of the cases die during the first five years of life from pulmonary disease, and although I am unable to put forward any statistics illustrating this point, I should say that amongst other forms of imbecility also the mortality under the age of five years is probably considerably higher than that amongst normal children.

In mentally defective children at a later age the mortality in institutions (Earlswood and Royal Albert Asylum) has been shown by Dr. Shuttleworth to be about nine times as great as the average mortality amongst normal children; his figures, quoted by Dr. Ireland, show the deaths per thousand to be as follows.

Age.	*Imbeciles.*	*Normal Children.*
5–10 years	50·1	6·10 per thousand
10–15 years	33·9	3·35 per thousand

Dr. Caldecott found that at Earlswood Asylum, amongst 1,000 consecutive deaths, 39·2 per cent. were due to tuberculosis (30·5 per cent. to pulmonary tuberculosis, only 1·1 per cent. to tuberculous meningitis); next to tubercle epilepsy was the commonest cause of death, 12·4 per cent. being due to exhaustion from this cause, whilst 4·9 per cent. died in the status epilepticus; pneumonia, lobar or lobular, accounted for 10·4 per cent. of the deaths. These three diseases, tubercle, epilepsy and pneumonia were by far the most frequent causes of death. These figures, however, no doubt underrate the mortality from tubercle and pulmonary disease among the mentally deficient; for at Earlswood Asylum patients under the age of six years are not eligible for admission, except by payment, and consequently the number of patients under this age is comparatively small; it is well known that the mortality from both these causes amongst children of normal intelligence is much higher under the age of five years than over; and therefore, if, as Dr. Caldecott's figures show, there is a special

tendency to tubercle in the mentally deficient, this would probably be even more marked amongst younger children.

Certain it is that mental deficiency does not preclude the possibility of long life; in Dr. Caldecott's statistics there were several imbeciles who had attained the age of sixty and some who had reached their three-score years and ten. Amongst 1,000 deaths, 243 occurred at or after the age of twenty-five years, and 11 of these were over the age of sixty-five.

The figures for imbeciles, it must be remembered, apply to the inmates of institutions where great numbers are living together, and where on this account there may be certain disadvantages which may tend to increase the mortality, but the general conclusion from them that the probability of reaching adult life is much less in these than in normal children is, I think, applicable to mentally defective children under any condition of environment.

Treatment. Drugs play but a small part in the treatment of mental deficiency, save in cretinism, where thyroid, administered either as the fresh gland or as a dried preparation or extract of it, restores the cretinous child almost to a normal condition of mind and body. I say 'almost' because even with the best results the intellect is, I think, always more or less below the normal, though I have seen instances in which the child would easily have passed for normal in ordinary conversation; one female cretin, for instance, at the age of $9\frac{1}{2}$ years was able to attend an ordinary school and at the end of the term was sixteenth in a class of thirty-one girls, but she was in a class two below that in which she should have been at her age. She would have been quite capable of earning her own living in such an occupation as housework.

The earlier the administration of thyroid is begun the greater the intellectual development is likely to be. Some care, however, is needed in the administration of thyroid; too large doses have resulted in fatal syncope in an infant; and apart from this rare disaster other symptoms will sometimes show that the dose is excessive: one of the first indications that the drug is taking effect should be a gradual decrease in the weight, owing to the disappearance of the excessive subcutaneous fat, but if the dose is too large this decrease takes place unduly rapidly and therewith the infant looks pale and ill, the pulse becomes rapid and perhaps irregular, and the temperature shows irregular elevation.

A further caution which applies only to cases in which the child is already beginning to walk when the thyroid treatment

is begun, the child should be allowed to be on its feet only very little, for owing to the rapid growth in length of the bones, under the thyroid treatment, bending is apt to occur under the weight of the child's body, and one difference between the pictures ' before ' and ' after ' treatment, may be the presence of bandy-legs in the latter.

For an infant of about six months, a dose of $\frac{1}{4}$-grain of Thyroid Extract (Burroughs, Wellcome & Co.) twice, and then three times a day will be sufficient at first, increased gradually to $\frac{1}{2}$-grain ter die at a year old ; or the Thyroideum Siccum (British Pharmacop.) gr. $\frac{1}{20}$ may be given and increased to gr. $\frac{1}{10}$ or gr. $\frac{1}{8}$ ter die at one year, the dose can then be increased gradually up to gr. $1\frac{1}{2}$ or 2 grains of the extract, or gr. $\frac{1}{4}$ of the thyroideum siccum and given twice a day instead of three times. It is noteworthy that any increase of dose beyond a certain limit, which seems to vary in different cases, produces no corresponding improvement : this limit must be ascertained by experience.

For the maintenance of the improved condition I have found $1\frac{1}{2}$ to 2 grains twice a day of Burroughs, Wellcome & Co. Thyroid Extract (Tabloids) to be sufficient for children from about four years old onwards.

There is another class of drugs which are of value in the treatment of a certain type of mental deficiency, namely, the sedatives such as bromide, chloral, and phenazone. In some cases of mental deficiency associated with epilepsy, especially in the cases of petit mal in infancy where the child is becoming duller and duller in intellect with the frequent recurrence of the epileptic attacks, I have seen very obvious improvement in the mental condition when the frequency of the attacks was reduced by the use of one or other of these drugs. Moreover, the administration of sedatives is specially called for at this period when, as I have satisfied myself by my own observations, the irritation of dentition, however it may do it, does undoubtedly determine the occurrence of epileptic attacks.

On the other hand it is a mistake to give large doses of bromide to these children, for the drug may not only interfere with the child's general health, making it look pale and miserable, but may produce the very condition we are trying to avoid, namely, dullness of intellect. A combination of phenazone with the bromide often makes it possible to get good results from a much smaller dose of the bromide than would otherwise be required ; the phenazone does not affect the child's general health or

brightness in the same injurious way as the bromide ; to a child a year old, a mixture of Sodium Bromide gr. ij, Phenazon gr. j, Spirit. Chloroformi ℥j, Glycerin ℥x, Aq. Anethi ad ʒj, may be given twice or thrice a day.

It is advisable in some cases to continue such a mixture as this throughout the teething period if the fits tend to recur frequently, say at intervals of a fortnight or less. But the treatment of mental deficiency in children is usually a matter of management and training rather than of drugging. When a mentally defective child is brought to a medical man his part is to encourage patience and perseverance with the hope that even if the child can ' never be quite like other children ', nevertheless he may, and if a very young child, almost certainly will, make some progress in mental development, provided proper efforts are made to cultivate his mental faculties.

In this task a patient and tactful nurse is essential during the first few years of the child's life—one who is quick to see what arouses the child's interest, and to use this object, picture, toy-animal, or whatever it may be, to stimulate and train the child's power of attention, for therein lies one of the most important factors in mental progress. If a mentally defective child can be taught to give his attention to an object even for a few moments, there is hope of further attainment ; attention like other mental processes can be cultivated, and the nurse should make persistent efforts to engage the child's interest, and so to increase his capacity for attention.

Such training may be made profitable in other ways. The simple turning over of the leaves of a picture book, or the holding of a doll is valuable in teaching the finer movements of the hands, especially of the thumbs which are usually so difficult for imbecile children ; thus little by little more complex processes of mental and physical activity can be taught. By frequent naming of the picture or toy in the simplest manner, as ' pussy ' or ' dolly ', or sometimes better by imitating the sound made by an animal which is shown in picture or toy, the child will come to associate what he sees or hears with certain sounds or names, and will sooner or later begin to repeat them himself and so gradually acquire speech.

The speech of the mentally defective is seldom distinct, often scarcely intelligible ; but in no respect is the value of careful and patient training more evident than in the improvement of speech which can be attained in these children, especially in those whose mental deficiency is of minor degree.

Another point to which I must refer in connexion with the infantile period of an imbecile's life, is the teaching of cleanly habits. It is remarkable how even with severe degrees of mental deficiency infants of a year or eighteen months will learn to make some sign such as a grunt or a particular cry to indicate their desire to defæcate ; cleanliness in regard to micturition is often not acquired until later. Training in these cleanly habits depends upon the carefulness of the nurse in attending at once to the child's indications of his need; if the child discovers that a grunt brings quickly the attention he needs, he will soon acquire the habit of grunting for this purpose. Many imbeciles who do not acquire any speech until they are four years old or more, have become accustomed to make their needs known in some such way, or by pointing to the chamber-utensil when they were less than two years old.

As the child grows older other methods of training can be used. The love of music, which is so common in the mentally defective, can be used to improve their speech and gait, class singing and musical drill are used with excellent results for this purpose in some of the schools for such children. Manual occupations of more or less simple kind may be employed, progressing from the simple fitting of pegs or bricks of different shapes into corresponding holes in a board, or threading beads, up to complex processes such as paper mat-work, or basket-making ; it is astonishing how skilful some of these children become.

When the child is six or seven years old the advisability of sending him to some institution or asylum has to be considered ; this question will arise chiefly where the severer degrees of imbecility make education in a day school for 'backward' children impracticable. I think any one who has seen the working of many of these institutions will agree that although it may be hard for the parents to part with their child, in no other way as a rule can the feeble-minded child obtain such expert care and teaching : the value of the institution environment, with its special adaptations to the capacity of the children, lies not in any intrinsic value of this or that simple work or occupation which the child may learn, but in the fact that participation in routine with other children of like capacities is in itself a source of happiness ; and that even the simplest accomplishment learnt gives a pride and joy to these children which makes one feel that it is only right to put them where their capacity for such enjoyment will be increased to the utmost.

CHAPTER XLII

MONGOLIAN IMBECILITY

MONGOLIAN imbecility has such well-defined characteristics, both physical and mental, which distinguish it from other forms of imbecility that it is worthy of separate consideration, especially as it is by no means an uncommon condition.

My own statistics taken as far as possible from consecutive cases showed 77 Mongols amongst 350 imbeciles. This proportion (22 per cent.) may be higher than larger figures would warrant. Dr. Shuttleworth considers that Mongols constitute 'perhaps nearly 5 per cent.' of all mentally deficient children. My observations were taken for the most part at the Great Ormond Street and King's College Hospitals, and it is probable that amongst imbeciles seen at a hospital the proportion of Mongols is much larger than in asylums, for the Mongol has less chance than most imbeciles of other types of surviving to the age when admission to an asylum is practicable.

The most striking point about the Mongolian imbecile is the facies, and the characteristics of this will be best illustrated by the photographs here given (Figs. 42 and 43). The downward slant of the palpebral fissures towards the nose at once attracts attention, and it is this peculiarity present normally in the Mongolian races of mankind which has given to this particular type of imbecility its name. The mother of a Mongol imbecile under my care stated that the neighbours called her child 'the Chinese baby'. The face as a whole is rounded and somewhat 'squat'; the bridge of the nose is much flattened and appears unduly broad, an appearance which is increased by the very marked epicanthic fold which is usually present. Owing to the flattening of the nasal bridge the nose looks small and *retroussé*. The cheeks, especially in children beyond the age of infancy, are usually very high-coloured. The tongue is broad, and in the worst cases tends to loll out of the mouth. In infants it often presents no abnormal appearance otherwise, but in later childhood the tongue has a very remarkable and characteristic appearance, probably due to hypertrophy of the papillæ. In some cases the surface looks like velvet with very coarse pile;

in others, where the condition is more marked, it has an almost mammillated appearance, and the whole dorsum of the tongue is divided up by branching fissures into irregular areas. In the child mentioned above the mother herself had noticed this, and said the tongue looked 'as if scored all over'.

FIG. 42. Mongol imbecile. Photograph of case under Dr. R. Hutchison showing typical slanting palpebral fissures, depressed bridge of nose, and protruding tongue.

Dr. John Thomson has made the interesting suggestion that these curious appearances of the tongue may be induced by the habit which many Mongols show of sucking the tongue.

The palate is often high and narrow, but by no means always; a perfectly normal palate is sometimes found.

One of the most noticeable characteristics of these imbeciles

is the brachycephalic condition of the skull. The head in almost all the cases I have measured has been below the average in its maximum circumference, and in many it has been obvious that the contraction was mainly in the antero-posterior diameter.

Taking the maximum circumference of the head in eight Mongols, and comparing these with the average maximum

Fig. 43. Mongol imbecile.

circumference at the same age, in a series of children of normal intelligence, the following results were obtained.

Age.	*Circumference in Mongols.*	*Circumference in normal children.*
4 months	14¾ inches	15·5 inches
13 ,,	16¼ ,,	18 ,,
14 ,,	16¾ ,,	18·2 ,,
17 ,,	17¾ ,,	18·4 ,,
22 ,,	17⅛ ,,	18·5 ,,
3¼ years	17⅞ ,,	19·2 ,,
4¾ ,,	18¾ ,,	19·5 ,,
9¾ ,,	19⅝ ,,	20·25 ,,

In addition to the small size of the head the shape is also peculiar ;

the occipital region tends to be flattened so that the back of the head loses its rounded contour.

The eyes frequently show some strabismus, but more commonly, in my experience, in older children than in infants. The squint is probably due in most cases to errors of refraction, which are exceedingly common in all forms of imbecility and idiocy. I have also met with nystagmus occasionally in Mongolian imbeciles.

Cataract is more frequent in these children than has been generally recognized. Mr. A. W. Ormond [1] found some form of lens opacity in 59·5 per cent. (25 out of 42 cases). The opacities were usually of the dot variety, and sometimes so faint that they required careful search to detect them. In some, however, they were more extensive, and I have seen cases in which there was a large cataract easily recognizable, even without dilatation of the pupil. These more fully developed opacities are, according to Mr. Ormond, of the lamellar variety. Blepharitis is extremely common; indeed, the majority of Mongols show more or less soreness of the eyelids from time to time.

In several of the cases under my care the ears have been abnormally small, although not otherwise misshapen. The hair is normal in amount, and there seems to be no special tendency to any particular colour, points in which this condition differs from cretinism.

The trunk and limbs are well formed and well proportioned. These children are usually well nourished, and often tend to obesity. The stature is below the average, but not to any such degree as is seen in the dwarfing of cretins. The following measurements of children at various ages (the first four from the same case) show the shortness of the Mongol imbecile in comparison with the normal.

Age.	*Height of Mongol.*	*Height of normal child.*
$2\frac{1}{12}$ years	$29\frac{1}{2}$ inches	33 inches
$2\frac{10}{12}$,,	31 ,,	35 ,,
$3\frac{9}{12}$,,	$32\frac{1}{2}$,,	36 ,,
$4\frac{3}{12}$,,	33 ,,	38 ,,
$1\frac{2}{12}$,,	$27\frac{1}{4}$,,	$28\frac{1}{2}$,,
$1\frac{6}{12}$,,	28 ,,	31 ,,
$4\frac{9}{12}$,,	$33\frac{2}{3}$,,	39 ,,
$9\frac{9}{12}$,,	$47\frac{1}{2}$,,	$51\frac{1}{2}$,,

The hands are short, and the relative shortness both of the little finger and the thumb is very noticeable. Special attention has been drawn to this shortness of the little finger in association with a curving towards the ring finger as a diagnostic point between Mongolian and cretinoid imbecility, and, also, to the

[1] *Ophthalm. Soc. Trans.*, vol. xxxii, 1912, p. 69.

fact that although the hand is short in both these conditions, the fingers in the Mongol are tapering, whereas in the cretin they tend to be square at the tip.

The hands and feet are often blue and cold; the circulation is sluggish, as is common in other forms of imbecility.

The great toe sometimes shows a curiously wide separation from the adjoining toe.

In many of these Mongols, especially in infancy, respiration is accompanied by a snorting, snuffling sound, partly, no doubt, on account of the smallness of the naso-pharyngeal space, the result of the antero-posterior shortening of the base of the skull, and partly on account of the tendency to catarrh of the nasal and respiratory passages, which is so common in these cases, but it seems likely that this respiratory obstruction is increased in some cases by a further anatomical peculiarity—the projection backwards of the vomer into the naso-pharynx—which is described below.

During infancy many of these imbeciles when they are pleased make a long respiratory crowing sound, an exaggeration of the crow of pleasure which a healthy infant makes. It resembles the crow of a severe laryngismus stridulus, except that the Mongol makes it voluntarily and as a sign of pleasure.

Some other curious habits are particularly common in this form of imbecility. The eyes are often rolled upwards momentarily so that, except for the lower edge of the cornea, only 'the whites of the eyes' are visible; or the eyes are turned suddenly in an extreme lateral direction as if the child were 'looking out of the corners of its eyes' in a sly way. At two or three years old the child will still hold up its hands with outspread fingers in front of its face and talk to them with unintelligible sounds.

In all respects these children are backward in development. Dentition begins late and is apt to be irregular; in one case there were no teeth at fourteen months; in another only one tooth, and that a molar, at seventeen months; in a third no teeth until two years, when one of the molars appeared. The fontanelle closes late; thus in one case it was still open at four and three-quarter years, in another it closed at three and a half years. In the latter case walking was first acquired at two years and five months, and at that age the only attempts at talking were vague sounds interpreted by the mother as 'gee-gee' and 'dad-da', and even at the age of four years he could say only a very small number of simple words and a few short sentences, such as 'drop of tea'. Another child made no

attempt to talk at three years old, but at five and a half years she could say a few words such as 'mamma', 'up', 'go', but no sentences, and at the age of five and a half years she still made no attempt to walk. The 'Chinese baby' mentioned above only began to talk when she was over three and a half years old, at which age she also began to feed herself; she could not stand until she was two and a half years old.

The voice in these cases is characteristically deep and gruff. The mental condition is usually rather backward than imbecile, if I may draw such a distinction. These children take considerable interest in their surroundings and soon learn to distinguish their parents or nurses from strangers; they are also affectionate and sometimes jealous of attention paid to others; one boy at the age of two and a quarter years would attempt to hit his baby brother on the head if the mother nursed the baby instead of himself, and another child at the age of four years would attempt to push away any such interloper. In general, however, they are not spiteful and are sometimes quite lovable children. They are usually stolid and obstinate, quick at mimicry, but slow to learn where any process of reasoning is required. At three or four years old they will take interest in pictures, and enjoy scribbling with a pencil, and later some of the highest grade Mongols can be taught to read simple words and even to write after a fashion.

The Mongol at ten years old is like a child of five, but often rather like a precocious than a dull one. He may, indeed, be the *enfant terrible* in the smart things he says, and in his keen observation of people's foibles and peculiarities. I have known a Mongol boy at nine years to be quicker in repartee than many normal children; but though the sense of humour is prominent, it is such as one expects in a younger child.

As in other forms of imbecility there is often a marked fondness for music, and some of these children can hum a tune fairly correctly at about four years old. Many Mongols can be taught to be clean in their habits quite early, if proper attention is paid to the grunt or other sign which they make when they require to micturate or defæcate; the 'Chinese baby' made a grunting sound before defæcation at one year, so that it was possible to leave off napkins at that age, but this, I fancy, is rather exceptional. Another case had made no sign whatever of his needs in this respect up to the age of four years, but a few months later he always went of his own accord to the chamber when he required attention.

The morbid anatomy shows little to account for the condition. The brain has usually been below the normal weight ; for instance, in a Mongol aged twenty-two months I found that the brain weighed 31¼ ounces instead of 33½ ounces, the normal average for that age. But otherwise there is nothing remarkable in the naked-eye appearance of the brain, the convolutions are well formed and of normal consistence. I noted in one case a curious projection backwards of the vomer into the naso-pharynx. Instead of projecting backwards only just beyond the posterior nares, as the vomer does in the normal infant, it extended backwards almost to the posterior wall of the pharynx, so that it could be felt by digital examination from the mouth, as a partial septum in the naso-pharynx, with its edge sloping upwards and backwards from the posterior nares to the roof of the pharynx. In another case under my care at the Hospital for Sick Children, Mr. Stansfield Collier examined the naso-pharynx at my request during life, and found the same condition. He tells me that he has found it in other Mongolian imbeciles ; it is, however, certainly only an occasional occurrence.

This projection backwards of the vomer when present no doubt accounts partly for the obstructed respiration, for it is evident that the very small naso-pharyngeal space is thus made still smaller, and when the mucous membrane over the vomer is in a swollen and catarrhal condition, as part of the frequent 'colds' to which these children are subject, the available space must be diminished still further. It is evident that a very small quantity of adenoid overgrowth would be sufficient to obstruct breathing in these cases.

The thyroid gland is found to be normal.

Etiology. A large proportion of Mongol imbeciles are the offspring of women who have already borne many children. Out of 73 cases in which I noted this point :

9 were	. . .	1st children.
7 ,,	. . .	2nd children.
25 ,,	. . .	3rd, 4th, or 5th children.
18 ,,	. . .	6th, 7th, or 8th children.
14 ,,	. . .	9th–13th children.

(The place in family in these figures was calculated by counting pregnancies, not only live-born children.)

When it is remembered that there are comparatively few families in which there are six to thirteen children, the large proportion of Mongols who were late children of large families becomes the more striking, as evidence that this form of imbecility is an exhaustion product. But is it the number of preced-

ing children or is it the age of the mother which determines this incidence upon the late-born child of a family ? This is not an easy question to determine, but there is some evidence that the age of the mother is of more importance than the place in family.

In 53 cases I noted the age of the mother at the birth of the Mongol child ; it was as follows :

Years of the mother's age.	*No. of Cases.*
18	1
20–29 inclusive	6
30–32 ,, ...	2
33–35 ,, ...	8
36–39 ,, ...	20
40–45 ,, ...	14
46–50 ,, ...	2

It will be seen that 36 out of 53, that is 67·8 per cent., were the offspring of women between thirty-six and fifty years of age.

If the advanced age of the mother be the chief factor we should expect to find that this applied equally when a Mongol imbecile was the first or second child in a family. I can only offer a small series of observations on this point : out of six Mongols who were second children five were born of mothers between 36 and 40 years old. But in the case of first-born Mongols this factor is by no means evident : the ages of the mothers in seven cases were respectively 18, 23, 25, 28, 32, 33, and 34 years.

This contrast between the ages of the mothers of first-born and second-born Mongols suggests that there is some other special influence determining the occurrence of this condition in the first-born : as I have shown elsewhere, there are facts which seem to indicate that the power of perfect reproduction is often not fully established in the first pregnancy, so that various congenital abnormalities tend to occur specially in first children ; in this way Mongolism may be due to immaturity of reproductive power, although it is more often due to exhaustion of this power.

Whether the Mongol is born early or late in a family it is noteworthy that any child born subsequently is almost always mentally sound, a fact which is difficult of explanation.

There are no doubt other causes besides mere age which may render a woman's reproductive power imperfect ; her vigour may be diminished by the strain of repeated child-bearing, but it may also be affected by disease, for instance, by syphilis or tubercle. If syphilis ever has any share in the causation of Mongolian imbecility it is probably only in this indirect way, it impairs the mother's vigour and therewith her power of perfect reproduction. Tuberculosis similarly may have an indirect

effect; a family history of phthisis is very common, and tuberculosis is the cause of death in many Mongol imbeciles, but there is no reason for supposing that tubercle has any specific influence in producing this condition. I have seen cases in which it seemed likely that the worry of a great domestic anxiety had contributed to this exhaustion imbecility.

Diagnosis. The diagnosis of Mongolian imbecility is easy after a typical case has once been seen. The two conditions which are most often confused with it have been, in my experience, cretinism and congenital syphilis.

The points of distinction from cretinism are: (1) the facies, the obliquity of the palpebral fissures, the high-coloured cheeks, the round squat face; all these distinguish the Mongol from the cretin, whose puffy eyelids, sallow earthy complexion, thick lips, large mouth, and splayed out nostrils are so characteristic; (2) the skull in the Mongol is brachycephalic and usually small, in the cretin dolichocephalic and often large; (3) the hair and skin; in the cretin the dry harsh skin, and the scanty, coarse, and often sandy-coloured hair contrast with the skin of the Mongol, which in the earlier years of childhood is normally soft, while the hair is abundant; (4) the hands; the short stumpy hands are common to both, but in the Mongol there is also a disproportionate shortness of little finger and thumb, with curving of the former towards the ring finger, and the tapering tips of the fingers contrast with the square ends of the cretin's fingers; (5) first appearance of symptoms; while the Mongol shows the characteristic appearances from birth, there is usually nothing to attract notice in cretinism until some months after birth.

The confusion with syphilis arises almost always from the presence of snuffling with marked depression of the bridge of the nose, two characteristic features of Mongolian imbecility which are not in themselves evidence of syphilis. The presence of the other characteristics of Mongolian imbecility, the history, and the absence of other syphilitic symptoms will generally suffice for the diagnosis from syphilis, and in some cases careful observation shows that the noisy respiration is not altogether like the snuffling of congenital syphilis; it has with it a snorting character which is common in other forms of idiocy, especially with contracted skull, as in severe microcephaly.

In the diagnosis of Mongolian imbecility in infancy and early childhood it is well to remember that a facies somewhat resembling that of Mongols is occasionally met with in people of

perfectly normal intelligence, but in some of these cases certainly, perhaps in most of them, there are obvious points of difference, and in addition the characteristic shape of the skull and of the hands may be lacking. But this resemblance at least suggests caution in prognosis in any case of supposed Mongolian imbecility in infancy; and in conjunction with the special symptoms of this particular form of imbecility it is necessary to take into consideration the presence of the general symptoms of mental deficiency.

Prognosis. Prognosis in these cases can be given at an early age rather more definitely than in some forms of imbecility and idiocy, for on the whole Mongol imbeciles resemble one another almost as closely in their course as they do in their facies. As to life, the prognosis is bad. A large proportion of these children die within the first few years of life; it is the exception for them to reach adult years. There seems to be a very marked tendency to respiratory disease. Bronchitis and broncho-pneumonia are the causes of death in many of those who die in infancy or in early childhood, while those who live on to later childhood or to adult life succumb, probably in the majority of cases, to pulmonary tuberculosis. Dr. A. E. Garrod [1] has drawn attention to the occurrence of congenital heart disease in this form of imbecility. It was present in three out of my series of seventy-seven cases, and I have known this to be the cause of early death. Short, however, of fatal disease these children are very prone to recurring attacks of bronchitis; nasal catarrh is a frequent trouble, and the eyelids become red and sore with repeated attacks of blepharitis.

As to the mental condition, if the case is seen in infancy, a rough estimate of the possibilities of development can be given from the general experience of such cases. So far as concerns the immediate future, the child will be backward in every respect, but he will learn to walk and talk, and will take an interest in toys and simple games and picture-books.

The possibility of teaching him any useful occupation can hardly be foreseen, but for the comfort of the parents it may be said that some of these children can be taught simple reading and writing and may learn some simple work such as gardening or farm work. Dr. Shuttleworth mentions the case of a girl who was not only taught to read and write but could also do laundry work and cookery, and I have known a Mongol girl, at thirteen years, to be capable of ordinary simple housework. But there is no chance

[1] *Trans. Clin. Soc. Lond.*, 1898.

of the Mongol imbecile ever being able to earn a livelihood or to look after himself altogether; he can only do simple work, and that only under more or less supervision. Nevertheless, it is no small gain if he can learn to do anything that is useful, for it gives him a purpose in life and is a source of pride and pleasure.

No drug treatment has been of any avail. The administration of thyroid gland in particular has been in my experience, as in that of others, absolutely useless, except to reduce excessive fat. Various other organic preparations have been tried without success.

Nevertheless, much can be done to make the lives of these children happier by such simple instruction and amusement as is provided in our large institutions for the feeble-minded, and for the poorer classes it is probably wisest to send the Mongol at the age of five to seven years to some such institution, in order that he may have such teaching and occupation as is specially adapted to his individual case, and this can hardly be obtained elsewhere.

Where it is possible by home teaching or by the special advantages of private 'homes', to avoid sending the child into crowded institution life, it is probably better to do so; for apparently the crowding together of such children, with their special tendency to tuberculous infection, increases the risk of tuberculosis.

Wherever these Mongol imbeciles are kept, the special tendency to catarrhal complications makes constant medical supervision necessary.

CHAPTER XLIII

NERVOUS CHILDREN

If any apology were needed for the use of a somewhat unscientific title I should say that there is no technical term which so well expresses the condition which I wish to emphasize here as the homely word 'nervousness'. Vague as the term may sound it represents a very definite type of temperament which is by no means uncommon in children, and the recognition of which is a matter of no small importance; indeed, it were much to be desired, in the interests of the children, that parents and school teachers, as well as medical men, were more fully aware of its practical bearings.

The manifestations of this nervous temperament in children are extremely varied, not only in kind but also in degree; at one end of the scale there is the simply timid or excitable child, at the other end is the child who shows hysterical symptoms as severe as any that occur in adults.

It is chiefly with the minor and commoner manifestations that I propose to deal in this chapter, for it is these which appear to me to be most often neglected or perhaps misunderstood.

And, first, I would point out that the nervous temperament may be evident at a very early age; at three and a half or four years a child will often show well-marked evidence of its nervous tendencies. I have seen even severe hysterical phenomena at so early an age as four years.

But I suspect that even in the first few months of life an undue excitability of the nervous system may show itself in a tendency to start at any slight noise, and certainly the infant who, apart from any disturbing cause in digestion or dentition or general health, has always been a 'bad sleeper', sleeping little and waking easily, is apt to turn out a nervous child at a later age. The occurrence also of convulsions in infancy upon a very slight occasion and apart from the influence of rickets, foreshadows, I think, in some cases a nervous temperament in later years.

Perhaps one of the commonest features of the nervous child is an abnormal degree of excitability. Nervous instability is to some extent normal in childhood, and the excitability which is

one of its expressions is therefore to some extent normal also, and the stolid child may be as abnormal in one direction as the over-excitable child is in the other. It would be difficult to define in words the limit between normal and abnormal excitability in childhood, but in practice it is often very obvious when the limit is passed. To take a well-marked instance, a bright intelligent boy, aged eight years, was brought to me with the history that amongst other symptoms of the nervous temperament he was so excited by music that the singing of Stainer's 'Amen' at church would rouse him to a state of excitement in which he was, so the mother said, 'almost past control'.

A frequent complaint with reference to these children is that they become unduly excited in their games; and I have been told sometimes by parents that they were afraid to send their child to a children's party for fear of the after-results; such children will become wildly excited at the time, with the result that subsequently they are pale and exhausted, and in many cases sleep is disturbed, the child talks in his sleep and keeps turning restlessly from side to side.

Closely related to the excitability of these children is what I may call their impressionability. An extreme instance of this came under my observation in a boy about seven years old, who, after overhearing a conversation in which a vivid description was given of a girl with melancholia, for several days refused to answer questions, took no interest in games, and avoided all companionship; with judicious neglect the condition passed off after several days. This was a quick, intelligent boy, who suffered much with headaches, walked in his sleep, and was of a very excitable temperament. The same boy when listening to a pathetic story would be so deeply affected that he would burst into tears. A striking instance of this feature in the nervous child is that recorded by Charcot, in which a girl of about thirteen years after being taken to a spiritualistic séance was seized with hysterical convulsions.

In some cases sensitiveness is extreme; a girl, aged nine and a half years, was brought to me because she was low-spirited and extraordinarily timid; the child's sensitiveness was distressing, she seemed unable to look one in the face, and started nervously when she found one was looking at her.

Some children similarly will blush whenever one looks at them or speaks to them; others will begin to cry and display an emotionalism only less in degree than the child with chorea.

Timidity in some of these cases is very striking; one little girl,

aged three and a half years, whilst out driving with her mother in a brougham would scream in terror if the carriage jolted over a rough piece of road, and whilst travelling in a train would be terrified if the train went under a bridge; she was brought to me again at a later period with habit-spasm, and showed other evidences of the nervous temperament. Another little girl, aged six years, was said to be 'terribly nervous'; she was so timid that she shook and trembled if there was a knock at the door. She suffered much from headache and talked in her sleep about her school lessons.

Disorders of sleep are specially common in these children; one child will suffer from insomnia, another is simply restless and sleeps fitfully, and others will have night terrors or walk in their sleep.

Insomnia is by no means an uncommon complaint in children, and is often very difficult to remedy. Its occurrence does not, of course, necessarily mean that a child is unduly nervous; in some cases it means digestive disturbance, particularly flatulent dyspepsia; in others it means obstructed respiration, not necessarily of very marked degree, from adenoids or enlarged tonsils; in others, again, it is the result of some fault in the child's surroundings, too little or too much bedclothing, or a noisy room or too much light in the room; but apart from all such causes there are many cases in which the child lies awake quietly for hours, and nothing abnormal can be found except that the child is one of markedly nervous temperament, a fact which is worth recognizing, for not only does it give us the comfortable feeling that we know something, however vague, about the cause of the trouble—a thing which parents always crave to know from the doctor—but it gives also some practical guidance. The child's daily life must be so arranged that all excitement and mental exertion, particularly the strain of lessons, is to be reduced to a minimum; and if the insomnia still persists it may yield only, as do so many other nervous irregularities, to change of scenery and air.

A boy of seven years was brought to me with the following history: he was fond of school, and the examinations were just past. For the last five or six weeks his sleep had been disturbed; he lay awake for about three hours before falling asleep, and then cried out in his sleep about his school. The mother said he was 'a very excitable child', and worried much over school, sometimes crying over his home lessons. I advised keeping him away from school altogether for a time, and about three weeks later

I heard that he was sleeping well and had entirely ceased to talk in his sleep.

The pressure of school examinations is certainly in some cases the exciting cause of these disorders. I remember one girl who at such times repeatedly walked in her sleep, to the great uneasiness of her parents; and in a boy, aged six years, the somnambulism was specially noticed to have begun since he first went to school, since which date he had also suffered frequently from headaches.

Night terrors are common in such children. For instance, a little girl, aged four years, was said to be 'very nervous', and was afraid to be left alone in the bedroom; even the ticking of a clock frightened her, so that it had to be stopped. She talked in her sleep, and lately, soon after falling asleep, she had started up in bed, seemingly in fright, talking of 'guys and clowns'; she failed at such times to recognize her mother, and when awakened had some difficulty in going to sleep again. Sometimes several of these attacks occurred in one night.

The much rarer phenomenon, to which the name of 'day terrors' has been given, usually occurs in children of nervous temperament. I have recorded some of these cases elsewhere,[1] but I may refer to one of them here as illustrating this point. A boy, aged three years, for the last six weeks has had sudden attacks of screaming with fright, often many times a day; whilst sitting quietly with the rest of the family, or busy at his play, he will suddenly begin to scream, turn pale, and look strange about his eyes; he rushes to his mother and hides his face in her lap, saying that he is frightened. Sometimes he says that 'somebody is coming after him'; but he cannot describe his imaginary pursuer. The attack lasts from two to three minutes. He has had night terrors with visual hallucinations for some weeks past, and is 'a very excitable child'. He is easily frightened by trivial things; for example, on crossing a railway bridge a few days ago he saw the smoke of a train and screamed with fright. He is quick and intelligent, perhaps precocious; he is shy, but when this is overcome he talks in the rapid eager fashion of an excitable child.

The speech of these children often shows evidence of their nervous instability, not only in its eager hurry, but also in some degree of stuttering. The stuttering in some of these cases seems to be simply a failure of speech co-ordination to keep pace with the child's haste to express his ideas, an occurrence which is quite common and natural in very young children whilst they are

[1] *Lancet*, February, 1900.

learning to talk. We are all familiar with the momentary difficulty of the little one whose words seem to tumble over one another in the eagerness of his talk, but in an older child the momentary stutter is often significant of a lack of nervous control which should have been acquired by this time. In other cases—and these are the more troublesome—the stuttering seems to be due to a disturbance, perhaps an actual inhibition of co-ordination in the mechanism of speech, owing to the child's sensitiveness or timidity; a result exactly analogous to the disturbances of movement,—for instance, tremor or loss of power in the limbs, which may occur as the result of emotion.

The nervous temperament of a child is often very apparent on auscultation of the heart. In examining large numbers of children one is struck by the fact that while in the majority, unless the child cries or shows obvious signs of being frightened, there is no great quickening of the heart beat or other alteration in its character, in certain children the effect of examination is not only great rapidity of the heart beat but also a characteristic rhythm, the beat becomes sudden and short. This quick staccato or slapping beat is, I think, a sure sign of the nervous temperament, but it is present only in a minority of nervous children. A girl, aged ten years, who was brought for some nasal catarrh, showed no sign of being frightened or excited, but I noticed that her heart rate was 144 per minute, and the beat was of the sudden slapping character which I have described. I noted it as evidence of a nervous temperament, and this was confirmed a few days later by the appearance of a definite habit-spasm, a frequent upward toss of the head.

Less characteristic than this nervous staccato rhythm, is irregularity of the heart beat. In some nervous children this phenomenon is very noticeable when the child is first examined by a stranger. Both alterations may occur at the same time, the beat becoming not only irregular but quick and slapping. It is important to bear this possible cause for cardiac arrhythmia in mind, otherwise quite unnecessary anxiety may be raised.

The nervous child shows his instability also in his temperature; a trivial ailment, a little constipation or a slight bronchial catarrh, which in another child would scarcely cause a deviation from the normal, will send up the temperature several degrees in these children; indeed, it may not require even an ailment to do this, an evening at the pantomime or the excitement of a tea-party may be quite sufficient to raise the tempera-

ture to 101° or 102°. It is in these children also that there occurs a more alarming and puzzling pyrexia. In some cases this takes the form of an evening rise to about 100° for many weeks or even months without any apparent cause—and, what is more important, without any injurious influence on the child's health ; in other cases there are recurrent bouts of more severe fever every few weeks or months. I have considered fevers of these kinds more fully in a previous chapter (Chapter XIX).

Habit-spasm is a very common occurrence in nervous children. In one child there is a sudden twitch of some of the facial muscles, in another a shrug of one shoulder, in a third an oft-repeated apparently involuntary sniff, and with such spasms there are often other manifestations of the nervous temperament. A boy, aged eleven and three-quarter years, was brought to me for a sudden twitch of both eyelids and of the angle of the mouth ; this had been present six months. He was a studious boy, forward in his school work ; spent most of his leisure time in reading, and was more fond of books than of play. He had a shy manner, and seemed unable to look one straight in the face ; he talked much in his sleep at night. Another boy, aged nine and a half years, with twitching of the right ala nasi, and an occasional contraction of the frontalis and of the orbicularis palpebrarum muscles, was said to be ' a very excitable child ' ; he dreamed much, frequently cried out in his sleep, and had had definite night terrors a few months previously ; he had also had occasional nocturnal enuresis. He was a spare child, very bright and intelligent, talked in a rapid excited way and gave one the general impression of being, as the mother stated, an excitable child.

The occurrence of nocturnal enuresis in such nervous children is by no means uncommon, and perhaps this relationship has not been sufficiently recognized.

The restless condition of the higher centres in these children during sleep is evident in the dreams and night terrors to which they are so liable, and it may be that a similar restlessness in the lower centres manifests itself in nocturnal enuresis.

Probably closely allied to this enuresis is the so-called ' lienteric diarrhœa ', the ' diarrhée nerveuse ' of Trousseau, which is specially associated with this particular temperament. A boy, aged ten years, for two years had been obliged to leave the room directly after each meal, and sometimes before the meal was finished, to relieve the bowels. He was an emotional boy and easily cried if spoken to roughly ; he talked much in his

sleep; his brother suffered with night terrors. A girl, aged seven years, was brought to me with the history that for three months the taking of a meal was often followed almost immediately by an action of the bowels; the slightest excitement had the same result. For two months there had also been enuresis at night. She was 'a highly nervous child', and suffered with headaches from time to time.

Fæcal incontinence, a disorder chiefly of boys between the ages of six and nine years, is another manifestation of this nervous temperament. I have seen it associated with habit-spasm, insomnia, enuresis, stuttering, night terrors, and occasionally with the nervous diarrhœa which I have just mentioned. Sometimes the nervous character of this disorder is seen in its relation to school pressure, and complete holiday from lessons may be a necessary adjunct to drug treatment.

The frequency of headaches in nervous children is very noticeable, and perhaps more so in those who have reached the school age than in the younger ones. A little over-exertion, the excitement of an entertainment, the slightest pressure of school work, may be sufficient cause for a headache. Some of these headaches are undoubtedly of the nature of migraine, but others—and I fancy the majority—are of different character, and almost defy pathological classification; some of them are probably related more or less closely to the rheumatism which is so common a feature in the personal or family history of these nervous children.

The connexion between rheumatism and the nervous temperament in childhood is very close, and rheumatism in the parents would seem to be almost as important a factor in the production of this temperament as rheumatism in the child. But there are other factors which enter into its causation, and foremost among them is neurotic heredity. In many cases the mother or father shows evidence of the nervous temperament, or it may be hysteria, epilepsy, or insanity. Some of the nervous children under my observation have been the offspring of drunken parents; in others, again, 'great wits,' 'to madness near allied,' have figured in the family history.

But nervous instability in children is not always congenital, often the history is that the child has become nervous only during the past few weeks or months. In such cases the presence of worms in the intestine may be the cause of the trouble. So marked is the effect of worms in producing nervous symptoms such as I have described, that it has been suggested that they

may produce some toxin which acts specially upon the nervous system; experiments, however, have given contradictory results, and I am the less inclined to attribute the nervousness to any specific effect of the worms, because I have often observed that the passage of much mucus in the stools, as in the condition which Dr. Eustace Smith has called 'mucous disease', is associated with an exactly similar nervous condition. It seems probable, therefore, that any chronic irritation of the intestine, whether by digestive disorder or by parasites, may cause general nervous instability; certainly in many of these cases where a child is said to have become nervous recently strict regulation of the diet, most often, perhaps, curtailment of starch and sugar, is an important part of treatment.

Treatment. The management of the nervous child calls for no small amount of tact and common sense on the part alike of parents, nurses, and teachers, and a volume might be written on the bringing-up of such children; but here one can only touch on some of the more important points on which the medical man is sometimes consulted.

A weighty matter in the care of these children is the choice of the nurses and teachers to whom they are to be entrusted. The combination of sympathy and discretion which is needed in dealing with the nervous child is not easily to be found, but it is very important for the welfare of the child. By sympathy I mean not only a love of children but also that readiness to divine the workings of a child's mind, and to see things from a child's standpoint, which is perhaps a natural gift rather than an acquired ability. The need for a wise discretion is urgent and continual in dealing with these children, and if this discretion be important in nurses and teachers it is even more important in the parents themselves.

As I have already pointed out, the nervous temperament manifests itself at a very early age, and the special care which is needed in the management of these children must begin in the nursery. The lines of this special care are indicated to some extent by the traits which I have described above as characterizing the nervous child; some of these traits are to be regarded rather as danger-signals than as dangers in themselves, and most of them indicate sufficiently clearly the sources of harm which are to be avoided. The sensitiveness, the impressionability of these children must be specially borne in mind from the first. Listen to some of the hallucinations and delusions in their night terrors, and it is evident that these are

but the echo of the unwholesome fiction with which they have been entertained in their waking hours.

Look at some of the picture-books which are in vogue for nursery use. See the impossible but fearful ogre whose special delight is to devour small children, or the mysterious and awful witch who hovers round the bed at night, apparently for the express purpose of carrying off juvenile offenders against nursery law. Is it any wonder that the child fears the dark when it is peopled with such appalling imagery ? It is difficult for us to conceive of the unquestioning realism of early childhood, but of the harm done to a nervous child by filling its imagination—and these nervous children are *par excellence* the imaginative children—with the terrible and the unlovely there can, I think, be no doubt. I would protest against the thrilling ghost story and other harrowing tales for the nervous child ; perhaps I may be forgiven for illustrating my point by a quotation from that ' Uncommercial Traveller ' whose powers of observation were so often used in the interests of the little people that he loved. After narrating the gruesome tale of Captain Murderer, one of ' Nurse's Stories ', wherewith that typical domestic was wont to entertain her youthful charge, he continues, ' The young woman who brought me acquainted with Captain Murderer had a fiendish enjoyment of my terrors, and used to begin, I remember—as a sort of introductory overture—by clawing the air with both hands and uttering a long hollow groan. So acutely did I suffer from this ceremony, in combination with this infernal Captain, that I sometimes used to plead I thought I was hardly strong enough and old enough to hear the story again just yet. But she never spared me one word of it, and, indeed, commended the awful chalice to my lips as the only preservative known to science against " The Black Cat ",—a weird and glaring-eyed supernatural Tom, who was reputed to prowl about the world at night sucking the breath of infancy, and who was endowed with a special thirst (as I was given to understand) for mine.'

But it is not only in this direction that books may be harmful ; some of these nervous children are children of unusually studious habit ; they will devote to reading all the time which should be spent in bodily recreation, and show a disinclination for games which is unhealthy and to be discouraged. When I am told that a little girl aged eight years does not care to join in games, but enjoys reading Longfellow and Milton, I am not surprised to find that the child has a troublesome

habit-spasm, and, as I find in my notes of the case, is 'very excitable'.

If the evil effects of sensational fiction, whether in books, pictures, or entertainments, is great, the effects of fright on the timid child are still more serious. Not only functional neuroses of longer or shorter duration but also syncopal attacks, convulsions, and even idiocy have been traced to fright in such children, and I would plead for these timid children that their fears be respected. The mixture of cowardice and ignorance which can even threaten to shut one of these nervous children or, indeed, any child in a dark room as a punishment, is nothing short of contemptible; and when one hears nurses or parents playing upon the fears of a child to enforce obedience by threats or feints of highly improbable but none the less terrifying punishments, one feels that such people are wholly unfit to be entrusted with the care of any child, much less of a nervous child.

The nervous child is to be treated gently; his nervous texture, if I may use so vague a term, is more delicate and requires more careful handling than that of a coarser fibred child. It is a great mistake to attempt to 'harden' such a child by exposing him purposely to that which he fears; the shrinking, timid child is not to be made 'manly' by a course of exposure to the ridicule or bullying which he will almost certainly meet with at any large school; nor will the nervous and sensitive child be any way the better for that foolish proceeding which some parents call 'knocking it out of him'; on the contrary, the nervous condition may easily be aggravated, and what was first an unimportant and almost natural characteristic of the child may take the form of some functional neurosis which may be very difficult to cure.

Perhaps one of the most important points in the management of these children—and even for the medical man the treatment of the nervous child is largely a question of management rather than a dose of medicine three times a day—is the matter of schooling. I have touched incidentally upon some of the common results of school pressure on these children, and my own experience has more and more convinced me that to the nervous child schooling must be given in small doses well diluted.

Long school hours are bad in every way; for many a nervous child of seven or eight years one to two hours' schooling in the morning, or even less, may be as much as can be advantageously given, and there are those for whom school must be forbidden altogether for a time. Public schools have seemed in my

experience to be wholly unsuitable for such children. I have seen nervous symptoms seriously increased by the methods of education adopted in these schools. It is useless, and worse than useless, to attempt wholesale methods in the education of nervous children; they must be dealt with individually. Even the very subjects of study require selection and adaptation for the individual child. For one child 'sums' are not merely the bane of his existence, but may and do cause a mental strain, the result of which may be sufficiently obvious when some neurosis develops; to another 'spelling' is a bugbear; and to a third 'music' may be prejudicial. For the younger children with this nervous temperament I believe that the Froebel system is the proper and best method of education, while for the older ones home teaching by a governess or tutor is often to be preferred to any school instruction. In one respect Froebel did not follow the practice of his great forerunner, Pestalozzi. 'Although Pestalozzi,' says one of his pupils, 'objected on principle to corporal punishment, he gave us every now and then boxes on the ear right and left.' And this is a point of importance. In the training of the nervous child corporal punishment of any kind must be absolutely forbidden; the effect of such punishment may indeed be disastrous. In one case, for instance, it was followed almost immediately by hysterical convulsions, and there can be little doubt that such affections as habit-spasm, which are just those for which the child is punished by an ignorant parent or school teacher, may be seriously aggravated in this way.

The value of physical training for these children is great. I believe that disciplined exercise, say in a well-regulated gymnasium or in the form of Swedish drill or even in dancing, accustoms not only the motor centres but also the higher centres to that ready and well-ordered control which is so often lacking in the nervous child. To obtain the full value of such training, however, the exercises or drill must not be an event of once or twice a week only, as in some schools, but should be part of the daily routine and, moreover, must be carried out with accuracy and care. In visiting schools, both private and public, I have been disappointed to see how much of the value of such physical training is lost by the slovenly and perfunctory way in which the children are too often allowed to perform the exercises.

As I have already said, there are children for whom schooling must be prohibited altogether for a time; and I would add that there is many a nervous child for whom a few months of

freedom to run wild in the country, or at the seaside, will be of far greater and more lasting value than the most intimate knowledge of multiplication tables, or even the more subtly instilled wisdom of Gift I in the Kindergarten.

I have made no mention hitherto of drugs, for these play quite a subordinate part in the treatment of this condition; but drugs have their place, and there are times when the administration of bromide or a course of arsenic may be the best possible thing for the child

Lastly, it must be pointed out that there is no rule of thumb by which we may be guided in the treatment of the nervous child; each case requires to be weighed carefully, not only in regard to its particular symptoms, their kind and degree, but also as to practical possibilities, in the way of modifying environment, or directing education in its widest sense.

CHAPTER XLIV

HABIT-SPASM

Amongst the many functional nervous disorders to which children are subject, one of the commonest is habit-spasm. This name has been given to certain oft-repeated and seemingly purposeless movements which most commonly affect the face or head and which, although they may change in character from time to time, are for varying periods persistent in kind, whether the movement be a simple twitch of one muscle or a more complicated action of several muscles. The term 'tic' is sometimes applied to this disorder, but lacks the descriptive merit of 'habit-spasm', which I shall therefore use. Trivial as the movements may be, their frequent repetition is most worrying and distressing to parents, and so the child is brought sooner or later to the medical man, and I will venture to say that there are few disorders of childhood out of which the medical attendant is likely to gain less credit than these cases; they look so easy and they are so difficult to cure. But if there is no drug which acts as a specific for habit-spasm there are many details in the management of these children upon which we may give valuable advice, and the value of our directions is likely to be in direct proportion to our appreciation of the relations and significance of habit-spasm.

The most frequent form of habit-spasm is a rapid blinking of the eyes or a more forcible closure of the eyelids, described by the parents as 'screwing up the eyes'; this movement, in the slighter or more forcible degree, was present in 47 out of 100 consecutive cases under my care; sometimes it was the only spasm present throughout the affection, but more often it was the first to appear and was subsequently replaced or accompanied by some other form of spasm. Much less common is a movement of the eye itself: in 5 per cent. of the cases the child had a habit of suddenly turning the eyes upwards, and still more rarely there was a sudden lateral deviation or a rolling movement of the eyes.

Next in frequency to the sudden blinking or closure of the

eyes which I have mentioned are various movements of the face, perhaps most often a twitch of the nose, which may be twisted to one side, or a sudden drawing up of one angle of the mouth, or the twitching may affect the forehead, causing a sudden frown or an elevation of the eyebrows. Grouping together these various facial contortions, I noted one or other of them in 48 per cent. of the cases. A common spasm is a sudden antero-posterior jerk of the head, a rapid affirmative nod; sometimes it is a sudden lateral rotation as in negation, or the head is rotated always towards one shoulder only, or it may be momentarily thrown upwards. Some such head-jerk was present in 30 per cent. of my series. The limbs are less often affected, but the upper limb more frequently than the lower. The upper limb was affected in 22 per cent., the lower in 9 per cent. In the upper extremity the commonest movement is a sudden elevation of one or both shoulders; this was present in several of my series, but all sorts of curious movements are seen. For instance, a girl aged eleven years and four months developed a habit of frequently flipping the end of the mid-finger against the end of the thumb first in one hand and then in the other, as if snapping her fingers; this continued at frequent intervals all day for three weeks, then it ceased and was replaced by a habit of suddenly jerking her head upwards. Another child, a girl, began at the age of eight years and ten months to strike herself frequently on the chest with her hand at home and at school, but soon this stopped and she began to blink frequently, and later developed a sudden antero-posterior jerk of her head; another girl who, at the age of 6½ years, had begun to blink and then had developed an antero-posterior jerk of the head, began at eight years to abduct her arms suddenly and beat them against her thighs like a bird flapping its wings. Another girl, nearly twelve years old, made frequent sudden clawing movements with her hands.

In the lower limbs equally curious movements occur. A boy of eight years would at frequent intervals 'paw the ground like a horse' whilst out walking; the affection had begun with blinking of the eyes two months earlier; then this ceased and he acquired a habit of frequent grunting and at another time a sudden head-jerk. A girl about ten years old who eighteen months earlier had begun to interpolate a curious sound between every few words in speaking, and a little later had changed this habit for a frequent clearing of the throat, was now said to jerk one leg forward quite suddenly at short intervals when

walking. A girl aged 6½ years, who was brought first for blinking, and when this ceased for continual 'hawking', had now begun to pause suddenly in walking and turn one foot over on its outer side and then the other foot similarly. Another girl, aged 8¼ years, had a curious habit of raising herself suddenly on her toes whenever she began to speak.

Trunk movements are more uncommon; a child to whom I have already referred as flipping her fingers was brought a year later for a sudden bending of her trunk to one side, a sort of pleurosthotonos, but quite momentary; it occurred usually whilst out walking. A boy, aged seven years, showed a sudden twitch of the facial muscles, drawing his mouth over to the right side, and at the same time his whole trunk was jerked slightly forward.

A common form of habit-spasm is the frequent repetition of some particular sound, which may be like that made in 'clearing the throat' or a more obvious 'hem', or may be of more articulate character, resembling some syllable or word which the child utters in season and out of season in the most inopportune manner; rarely the sound is much more like one of the involuntary sounds produced by healthy persons, such as hiccough or a gurgling sound in the throat. One or other of these varieties was present in 14 out of my 100 cases. As examples of these I may mention a girl, aged 9½ years, who first showed a rapid affirmative jerk of the head which recurred sometimes twice in a minute; she made also at short intervals a slight grunting sound and for a time had occasional momentary adductor spasm of the arms; these habits disappeared almost entirely after a month's stay in the country, but about two years later she was brought again for a return of the antero-posterior jerks of the head and a very frequent inspiratory sniff. A boy, aged eleven years and four months, who began with a frequently repeated 'ahem', and then passed from one habit-spasm to another, has now begun to roll his eyes up frequently and twitch his face, and at the same time makes a sudden hissing noise between his teeth at short intervals. A girl, aged eight years, who attended first for blinking and, when this stopped, for a sudden shrug of the shoulders, uttered a sound like 'pistol' at irregular intervals in the most irrelevant manner.

Such are the irregularities which are grouped together as 'habit-spasm', but, as will have been noticed, the term 'spasm' hardly seems equally applicable to all; the jerk of the head, almost like the result of an electric shock in its suddenness,

seems much more appropriately termed spasm than does such a movement as flapping the arms against the thighs or the even more quasi-purposive movements which sometimes alternate with a simple spasm; twice I have seen a simple twitch of face or nose replaced by a frequent sudden licking of upper or lower lip, so that the lip became reddened and sore, and one child who attended for years with first one habit-spasm and then another had at one time a troublesome habit of licking with her tongue objects which happened to be near her, but this, like the rest of her curious habits, passed away soon, only to be replaced by some other.

To this account of the individual movements I would add that, whilst in most cases the movement occurs singly, whether the child shows only one or more of the different forms—I mean, for instance, that a sudden jerk of the head and perhaps a grunting noise recur at irregular intervals of a few seconds or minutes—yet in some cases I have seen the particular spasm to occur in a series of perhaps six or eight repetitions in rapid succession, after which the child would show no spasm for several minutes, when another series would occur. There is in these cases, as it were, a grouped habit-spasm, a sort of habit clonus. Another feature of the disorder, which I have illustrated incidentally in this description of the movements, is the limitation of the spasm to different parts at different times, sometimes to one part only; in one child there is only blinking, in another blinking and a twitch of the nose, in a third, perhaps, an added shrug of the shoulders; the particular spasm is defined and settled; there is none of that complete uncertainty as to the whereabouts of the next jerk which is so characteristic of chorea, where the movements vary continually from moment to moment. Yet a further peculiarity, which I have also mentioned incidentally, is the striking tendency to change after a varying period of days, weeks, or months, from one form of habit-spasm to another; as one mother said to me of her child, who attended at hospital with habit-spasm for eight years, 'as fast as she gets rid of one movement she seems to get another'.

A remarkable feature of habit-spasm, and one upon which I would lay some stress, for it may be of some weight in diagnosis, is its diminution or cessation in many cases so long as the child is conscious of being specially under observation; sometimes, indeed, the medical attendant has to rely entirely upon history in his diagnosis; for the movements may cease directly the child is conscious that he is being watched by the medical

man, but directly this inhibitory effect of observation is removed —as it may be by letting the child sit quietly aside whilst the mother is being questioned—the habit-spasm becomes evident.

So far I have spoken chiefly of the movements which are the characteristic feature of habit-spasm, movements which vary from a simple twitch of one muscle up to a complex co-ordinated movement such as pausing in walking to turn each foot over on its side ; I have mentioned also the occurrence of apparently involuntary utterance of sounds which similarly vary from a simple grunt up to a complex articulate utterance, as in the case where ' pistol ' was the repeated sound. But there is yet another manifestation of the disorder, which has been described by some writers under a separate heading as ' psychical tic ' ; the child in some cases, and these generally with very marked, sometimes almost violent affection of movement, shows curious psychical disorder ; a girl, aged eight years, whose earliest symptom had been simple blinking of the eyes, which was replaced a little later by shrugging of the shoulders, subsequently was brought to me with a complaint that she had begun to utter foul language, apparently without any reason or even seeming to be aware what she was saying. Another child, a boy aged 10½ years, who had begun with a simple noise like clearing the throat at short intervals and then had developed the habit of frequent frowning and an upward jerk of the head, was brought to me two years later with delusions of ' microbes on his hands ', so that it was difficult to get him to take his food. Such cases undoubtedly represent the more severe and intractable degrees of the disorder, but I have specially mentioned the early symptoms in these two instances to show that they differ in no way from many another case of habit-spasm in which there is less evidence of psychical disturbance, and I say ' less evidence ' advisedly, for although but few cases show such pronounced disturbance as I have described, there are many more in which extreme excitability, great passionateness, or a morbid waywardness shows evidence of psychical disturbance. To this point I shall refer again ; here I would only deprecate the specializing of any such group of cases, as if ' psychical tic ' were some affection quite distinct from the mild case of habit-spasm which shows only blinking of the eyes. Similarly every gradation may be observed in one and the same child, from a simple twitch of the eyelids or nose up to the violent and sudden jerk which has been described—with no sufficient ground, as it seems to me—under a separate heading, as if

quite distinct from the slighter degree of spasm, as 'tic convulsif'.

Etiology. Turning now to the etiology of habit-spasm, one might have expected that, being a functional neurosis, it would be much commoner in girls than in boys. My own figures, however, showed 53 girls to 47 boys; the excess of girls affected, therefore, is only slight. I would point out that the same holds good of hysteria in childhood, where the proportion according to my own figures is 1·5 to 1, a proportion which contrasts with the 10 to 1 which is stated to be the relative frequency in adult life. The sexes are less sharply differentiated,

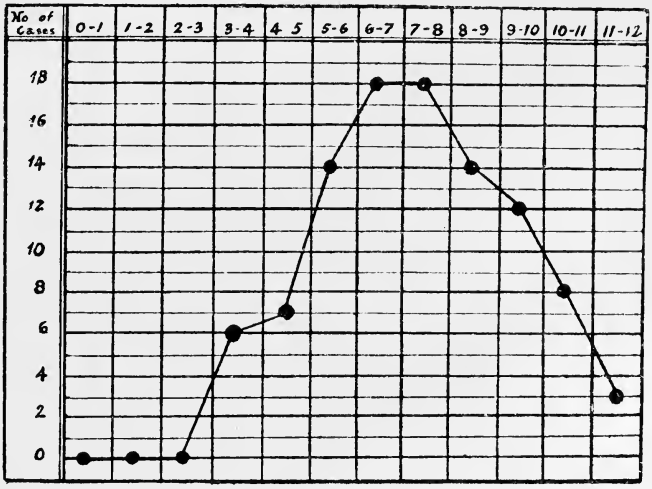

Fig. 44. Age-incidence of habit-spasm. Chart showing age at onset in 100 cases.

so far as concerns the tendency to functional nervous disorders, in childhood than in later life. With these proportions I would specially contrast the sex-incidence of chorea, where the proportion of females to males has been shown to be approximately 2·5 to 1. The age-incidence of habit-spasm is of some interest, as it has been suggested that it bears some relation, which I shall mention later, to the cause of the disorder. It is shown in the accompanying chart, in which the age at onset is recorded in 100 consecutive cases under my care.

The one fact which stands out pre-eminent in the history of children with habit-spasm is nervous instability, and this is well seen in the various disorders with which habit-spasm keeps

company. A boy aged 11¾ years, who was brought for a frequently-occurring shrug of both shoulders, was said to be a very timid child, often suffered with headaches, was very restless, and talked much in his sleep; the shrugging of the shoulders was noticed to be worse when he came home from school. Another boy, aged seven years, who was brought for twitching of the face and jerking of the body forward, and later for some blinking, was said to be 'very nervous', talked much in his sleep, had had enuresis, and the habit-spasm was more whenever he became excited. Another child, a girl aged 6½ years, who attended for many months with various habit-spasms, had recently walked in her sleep; at the onset of the habit-spasm she had begun to suffer with enuresis, and a little later was said to be 'so dreadfully nervous, she cries if you speak to her'.

There is no need to multiply examples of this sort, but to sum up the associated manifestations in my series of cases they were as follows: in a large proportion of the cases the child was said to be extremely nervous and excitable; in some cases the child was extremely irritable or passionate and difficult to control. Disturbances of sleep were very common, the sleep was often noticed to be restless, and in 20 per cent. there were more definite disorders of sleep; the child talked or walked in its sleep, had night terrors, or suffered with troublesome insomnia. Headaches were common and enuresis was noted in 10 per cent. of my cases. That peculiar form of looseness of the bowels which occurs immediately after each meal, 'nervous diarrhœa' as it has been called, was noted in two cases. Stuttering occurred in two cases, and I have noted in three cases a vasomotor instability showing itself in extraordinarily ready flushing or extreme liability to urticaria. Convulsions had occurred in infancy or early childhood in 7 per cent. of the cases—an occurrence which, as Dr. J. A. Coutts pointed out, often foreshadows nervous instability in later years. To this description of the accompaniments of habit-spasm in children I would add one more which, though not a disorder, is like the 'genius which is near allied to madness', a common association of nervous tendencies—I mean quick intelligence. Most of these children are sharp, responsive, 'quick in the up-take.' Often I have been told that they are 'always reading' or 'mad on school' (I quote the mother's words). The child with habit-spasm is to be found at the top of the class and not uncommonly in a class above his age. As a further point in connexion with their nervous temperament I would note that these children are

almost never the 'fat, sleek-headed, such as sleep o' nights', they are nearer akin to the lean Cassius—they are spare of build.

With all these indications of the nervous tendency I would class the perhaps more than average frequency of rheumatism in the family history of these children; in 14 out of the 100, the mother or father had had rheumatic fever; in three others a sister or brother; in seven others one of the grandparents; in seven others an uncle or aunt had had rheumatic fever; in six others the child himself had had pains in the joints, possibly or probably rheumatic. These figures perhaps understate the frequency of such a history, for I have counted only histories of definite rheumatism, omitting cases in which there was 'rheumatism' of uncertain nature. The nervous temperament of the rheumatic child and the child of rheumatic parentage is a very real clinical fact, whatever the pathological explanation may be, and it is in this respect, no doubt, that rheumatism may play some part in predisposing to habit-spasm. As might be expected, a family history of neurosis is very common in these cases, but I have only occasionally seen habit-spasm in either of the parents.

Given the nervous tendency, there are still other factors in many cases determining the onset, or it may be the return of the habit-spasm; and first I should place local irritation, not because of its frequency—for in my own experience it has only been in the minority of cases that any such could be detected—but because its detection may be of much practical importance in treatment. The local irritation may be so slight that the child is barely, if at all, conscious of it, and yet the removal of the irritant, if it be removable, certainly diminishes or stops the habit-spasm in many cases. The age-incidence suggests one possible source of irritation, the onset of habit-spasm corresponds in so many cases with the onset of the second dentition, a process which in some children produces appreciable local worry and might be expected, like the teething of the infant, to produce reflex disturbance. I have seen several cases also in which caries of the teeth seemed to be an exciting cause of habit-spasm, and dental treatment has caused a rapid improvement. I am satisfied also that in some cases the blinking which is so commonly an early manifestation of habit-spasm is started by a local cause, sometimes by a slight follicular conjunctivitis, so that when the lid, especially the lower, is everted its inner surface is seen to be unduly red and granular, sometimes and perhaps more often by some

error of refraction which, so my ophthalmic colleague, Mr. L. V. Cargill, tells me, is most commonly hypermetropic astigmatism. Similarly in the nose I have seen inflammation of the mucous membrane on the septum associated with a constantly recurring screwing up of the nose, and I doubt not that sometimes the 'clearing of the throat' which becomes a habit and is replaced subsequently by other entirely different habits, may be started by some temporary naso-pharyngeal catarrh or by the more chronic catarrh which is so often associated with adenoids.

This leads me to raise another point which may emphasize the necessity for early detection of these local sources of irritation—I mention it as a suggestion rather than as an assertion. It has seemed to me that habit-spasm once induced by a local irritation begets habit-spasm elsewhere, so that a child who owing to some error of refraction has acquired the blinking habit may soon develop a twitching of the nose, and then, perhaps, lose both of these spasms and develop a shrug of one shoulder or a jerk of the head.

It seems likely that more remote irritation may occasionally set up habit-spasm. I have noted particularly the presence of worms in several of my series of cases, but one must remember that worms are exceedingly common in children without habit-spasm and, moreover, even if worms do play any part in the production of habit-spasm, it may be rather indirectly by increasing the general nervous instability of the child than by any more immediate effect of the irritation.

Other factors which play no small part in the causation of habit-spasm in children are mental strain and mental shock. There can be no doubt that the present system of compulsory education, enforced as it too often is by the bullying ignorance of attendance officers and attendance committees, involves a degree of mental strain which is harmful to many a nervous child. And let it be understood that in the influence of school there is to be considered not merely the strain of acquiring knowledge—this, indeed, may be but trivial, particularly for some of these children who suffer from habit-spasm, for they are often particularly quick at learning—but also the strain of excitement in competition, the dread of failure and punishment—a very real strain with some of these nervous children—and in some cases also the continual dread of that most thoughtless of torturers, the schoolboy who finds his delight in teasing or terrifying a timid boy. Amongst the well-to-do all these difficulties can be avoided by home education, where the nervous

child has, perhaps, his hour or two hours of lessons, and not only the hours but the subjects and the methods of education can be adapted, as they should be, to the requirements of the individual child: to attempt, as the modern tendency is, to adapt the child to its education may be an inevitable procedure in wholesale education, but it is none the less unsatisfactory, and particularly unsatisfactory in dealing with such children as these with habit-spasm. The effect of school on habit-spasm is sometimes very striking, the spasm diminishes when the child is kept away from school, and increases markedly or returns directly schooling is commenced again; there is sometimes other evidence of the nervous strain of school at the same time in the appearance or increase of sleep-walking, night terrors, or headaches when the child returns to school. It may be also that in this relation to school life, especially to the earliest years of schooling, lies the explanation of the very striking age-incidence of this disorder, as shown in the chart on p. 639.

Excitement of any kind contributes to the production of habit-spasm. I have repeatedly known the good effects of treatment undone by some exciting experience: the sensational doings of Bluebeard at the theatre made one child who was under my care much worse, and in another the newly-conferred dignity of trousers increased the habit-spasm considerably. More potent still is fright or mental shock of any sort: a boy, aged seven years, who had been improving, became much worse after being knocked down by a bicycle; another boy, aged eight years, whom I have already mentioned as 'pawing the ground like a horse', became worse after pinching his finger in the door of a railway carriage. Similarly—and this is a point worth remembering—the mental shock of a surgical operation sometimes originates or increases habit-spasm. In the case of a boy, aged nine years and four months, the habit-spasm began directly after circumcision, and after various forms of habit-spasm had appeared for many months he gradually improved; the teeth, however, were very carious, and five were extracted, with the result that his habit-spasm again became bad; in another the habit-spasm seemed to have been started by the operation for removal of tonsils and adenoids. Lastly, I would mention exhaustion as a factor which at any rate aggravates if it does not start habit-spasm. I have purposely avoided particularizing as to the kind of exhaustion, for I doubt if it be possible to separate physical exhaustion altogether from nervous exhaustion. In several cases I have been told that the habit-spasm was always worse

when the child was tired; in others it was definitely worse in the evening, or when the child was allowed to stay up later than usual.

If I might throw out yet another suggestion for consideration it would be in the form of a question: May not habit-spasm arise in the involuntary perpetuation of what began as a voluntary movement? I suspect that in some of the cases of local irritation to which I have referred the child began by screwing up its eyes or twitching the nose voluntarily, and as a result of frequent repetition the movement changed from the voluntary into the involuntary. It has even seemed to me possible that such a natural movement as a toss of the head to one side, which began in the effort to throw back the irritating curl which will fall over the face, may sooner or later, in a nervous child, pass into the involuntary jerk of a habit-spasm; in the same way I believe that I have seen wilful imitation of a parent's habit-spasm pass into an involuntary facial twitch in the child.

Diagnosis. The diagnosis of habit-spasm is usually simple enough, but it is sometimes mistaken for chorea. The points of distinction are these: habit-spasm is a repetition of one movement or of one set of movements, chorea shows movements varying quite irregularly from minute to minute; habit-spasm is generally diminished or temporarily abolished altogether by observation, whereas chorea is usually exaggerated by observation (in either case, however, the reverse sometimes occurs); habit-spasm is usually much more limited in distribution than chorea; a twitch persistently limited to the face or head would be almost unknown in chorea, whereas it is a common form of habit-spasm. But I am quite sure that there are cases in which the diagnosis is extremely difficult; indeed, when I collected from my notes a series of cases of habit-spasm for this chapter, I found myself obliged to omit several cases which I had headed '? Chorea ? Habit-spasm'; and the difficulty arises in two ways, the movements of habit-spasm—e.g. in the shoulders and arms—may approximate in character and extent to those of a limited and slight chorea; and whilst habit-spasm may be in some cases more extensive in distribution than usual, chorea, as we all know, is sometimes so slight that it is only detected by a shrug of one shoulder or a twitch of one hand.

And there is a further difficulty, as Dr. F. Warner pointed out: the nervous child often shows fidgety movements, very like chorea, especially under observation, and this 'microkinesis' as it has been called, is not uncommon in children with habit-

spasm. Imagine, now, a child with this fidgetiness of his limbs brought with a complaint that he twitches his shoulders ; there is perhaps a shrug of the shoulder and in addition there is a general fidgetiness which might pass for a slight chorea : it may be no easy matter to decide offhand whether the shrugging of the shoulder is choreic or is due to habit-spasm. In such cases repeated, and perhaps prolonged, observation may be necessary before a diagnosis can be made. Even the presence of rheumatism is not conclusive evidence in favour of chorea. I have already pointed out that a family history of acute rheumatism is not uncommon in cases of habit-spasm ; more importance is, I think, to be attached to acute rheumatism in the patient, though even this is not conclusive, for some of my cases had apparently had definite articular rheumatism, though none had heart disease.

Treatment. I have discussed etiology at some length because therein lies the key to the management of these cases of habit-spasm, but I must refer to some of these points again. First, I would emphasize the importance of the diagnosis between chorea and habit-spasm in regard to treatment. By far the most important part of treatment in chorea is rest in bed ; the child with habit-spasm, on the contrary, does not require to be kept in bed at all : a good scamper at the seaside, or the varied interests of farm-life or country, will do more to get rid of the habit-spasm than anything else. I doubt if there is any treatment so effectual as a change to seaside or country for these children, generally marked improvement at least is obtained in this way, but unfortunately it is not always permanent, and even if the habit-spasm has ceased altogether it is apt to return when the child goes back to town or to school. Of the influence of school I have already spoken ; it may certainly be taken as a general rule that school should be forbidden and for a time no lessons should be allowed at all. But as the habit-spasm diminishes, an hour or two daily of home teaching by a governess or tutor may be begun tentatively. Not only the duration of lessons but also the choice of subjects may have to be restricted ; one subject or another may be particularly difficult to a particular child ; to one ' sums ' are a bewildering misery, to another ' spelling ' is no better ; and in all such cases those subjects should be left in abeyance for a time ; the worry of them is undoubtedly prejudicial to these nervous children and will perpetuate such a condition as habit-spasm. Obviously education is no small difficulty in a disorder like this, which may continue with intermissions for several years, and

whilst the well-to-do can get over the difficulty to some extent by such restriction of lessons as I have described, the poor have no such opportunity; half-time attendance is resented or absolutely refused by school authorities, and the only alternative is to keep the child away from school altogether for a much longer period than would be necessary if more ideal methods of education were practicable.

Having said so much about the necessity of avoiding school, I should like to qualify this by saying that in exceptional cases school, so far from being injurious, seems to have a quieting influence upon the habit-spasm. One may suppose this to occur when the home influences are such as to encourage neurotic tendencies; in any case, it is worth bearing in mind as a justification for trying the effect of school for a time.

Another practical question asked by parents is this: Should the child be scolded or punished for his 'tricks'? I have made notes on the effect of scolding in some of my cases; in none was it said to have done any good, in some it was said definitely to have made the child worse. The necessity for avoidance of all forms of excitement and fatigue follows from what I have said of etiology, and in this connexion I must mention in particular the bad influence of late hours on such cases. The need for removing sources of local irritation has already been suggested. The eyes and the teeth are the parts most often requiring attention; worms also should be treated. As I have mentioned the possibility of a naso-pharyngeal catarrh occasionally starting the 'hem' which constitutes one form of habit-spasm, let me say a word against the indiscriminate removal of adenoids in such cases. The nervous disturbance caused by the fright of the operation or the anæsthetic has made some cases of habit-spasm so much worse that I am satisfied that only in cases of necessity and after other means have been exhausted should operation be done.

The drug treatment of habit-spasm does not offer any large field for selection. I have found most useful a mixture of arsenic with bromide: liquor arsenicalis, 2 or 3 minims with potassium bromide, 5 to 10 grains, three times a day. This may be continued three or four weeks—the dose of arsenic to be reduced if any toxic symptoms arise—and then after an intermission of about a fortnight repeated again for three or four weeks. Another drug which has seemed to me of value in some cases is ergot; from ½ to 1 drachm of the fluid extract can be given with 4 or 5 minims of tincture of nux vomica three times

daily. I have also tried valerian with distinct advantage. In a bad case which some would have described as 'tic convulsif' I tried electricity, first faradism and then galvanism, with some temporary improvement, but not enough to justify strong recommendation. General massage I have known to cause decided diminution of the habit-spasm in a severe case, but massage is always to be used with caution in the functional nervous disorders of children, for whilst in some it acts as a sedative, in others it has only a disturbing and exciting effect and is therefore harmful. Lastly, for an intractable case simple change of environment, especially living amongst strangers for a time, will sometimes do more than any drug treatment.

CHAPTER XLV

CONVULSIVE DISORDERS IN INFANCY

The subject which I propose to consider in this chapter is convulsive disorders in infancy; and I use the term 'convulsive disorders' advisedly, to comprehend not only the different grades, if I may so say, of general convulsion, but also certain local affections which, although not usually considered under this head, are nevertheless convulsive in character. A proper appreciation of the relation between these various convulsive manifestations has a very practical value both in prognosis and in treatment, the two points which I wish specially to consider.

As our starting-point we may take what is ordinarily recognized as an infantile convulsion. I need hardly describe such an attack, every medical man is familiar with it: an infant suddenly becomes livid about the lips and about the alæ nasi; its eyes are fixed in a vacant stare, consciousness is lost, the limbs stiffen for a moment, and then face, arms, and legs twitch irregularly, and the eyes perhaps show a nystagmoid jerking to one side or the other, or perhaps a transient squint; at the same time the fontanelle is bulged, and the child becomes more and more livid. Gradually twitching and rigidity subside, consciousness is regained, and the child lies back pale and exhausted. Such an attack may occur as an isolated manifestation, to recur perhaps after weeks or months, perhaps never; but, on the other hand, it may form part of a series of convulsive attacks, between which the infant may not even regain consciousness, and is, in fact, in a condition exactly resembling the status epilepticus of the adult.

The seriousness of such attacks as these no one is likely to overlook, but there are other minor manifestations which I venture to think are often overlooked, or, if not overlooked, regarded too lightly by the medical man. Those who have attended the out-patient department of a children's hospital must have heard mothers talk of 'the inward convulsions',—a vague complaint, I admit, but one to which the mother attaches no little importance. Now I shall put in a plea for these 'inward convulsions'; I would strongly advise medical men never to disregard such a complaint from a mother; the symptoms to which she refers

may be the first sign of coming danger, and may give a valuable indication for the prevention which is better than cure.

As I have said, it is a vague complaint, and the symptoms which the mother includes under this term are not the same in all cases. In one case the child screams, goes a little livid around the mouth, and then perhaps turns its eyes upwards, and at the same time turns its thumbs in strongly towards its palms. In another there is a momentary twitch, extremely fine it may be, of the forearm or of one leg, or perhaps only of the fingers, with no apparent loss of consciousness, and, indeed, with so little disturbance that only a careful observer would notice that anything unusual had occurred. In a third there will be a sudden start of the whole body as if from an electric shock, so momentary that if it occurs during sleep, as it sometimes does, it scarcely disturbs the infant, who perhaps cries slightly after it, but is soon sleeping quietly again. Trivial as these symptoms may appear in themselves, there can be no doubt that they are just as truly convulsive as the major attacks which we call infantile convulsions; they stand, indeed, in much the same relation to these major manifestations as petit mal does to major epilepsy, and at any moment they may run on into the more general convulsion.

Sometimes, however, the mother's phrase is less accurate, and she applies it to the paroxysmal screaming of an infant with flatulence and colic; unless, indeed, she dignifies that condition by another term from the maternal vocabulary, 'the screaming convulsions,' which may have no justification in pathology, but may serve to remind us that the same colic which gives rise to the paroxysms of screaming may also give rise to actual convulsions.

Here I shall mention an occurrence which I think is not sufficiently generally recognized, namely, that a child may literally 'cry himself into a fit'.

I have several times had children brought to me because when they cried they 'went black in the face', and the parents were terrified lest any harm should come of it. Now any one who is not familiar with such cases might easily pooh-pooh the attacks and say, as I have known said, that they were merely the tantrums of a spoilt child and required only firm discipline. It may be perfectly true that the child is a spoilt child, and that the crying is due to mere passion because the child cannot have its own way, but the 'going black in the face' is none the less an indication of real and very serious danger; what starts as a mere

bout of crying ends in a spasmodic closure of the glottis during the violent inspiration of crying, the child becomes more and more cyanosed, and, as I have seen happen in some cases, passes into a state of complete unconsciousness or into a general convulsion.

Such a case was the following :

Percy P., aged three years, had been subject to convulsions at intervals since early infancy ; but now the attacks were said to come on when he cried from any cause, for instance, when he could not get his own way or happened to fall or hurt himself ; sometimes he passed urine in the attacks. Such was the account given to me, and, rather sceptical of the mother's tale, I proceeded to examine the child, who was apparently healthy in every respect. The child objected to being examined and began crying passionately, he went bluer and bluer and then a livid leaden colour, and lost consciousness. Gradually he revived ; and I then attempted unwisely to look in his mouth, which again started an outburst of crying, and the child again became unconscious and was undoubtedly in imminent danger of his life.

In another boy, aged about two years, who was under my care and who had also been subject to convulsions in early infancy, definite convulsions occurred whenever he cried violently ; the attacks began in an exactly similar way to that just mentioned ; he was a self-willed child and in a paroxysm of passionate crying would hold his breath, become livid, and pass into complete unconsciousness with some twitching or rigidity ; after the attacks he was pale and listless, and there was no doubt that they interfered considerably with his general health.

Such cases are quite distinct from laryngismus stridulus, which I shall mention subsequently ; they have no connexion with rickets nor with any of the special associations of laryngismus stridulus. The children who thus ' cry themselves into a fit ' have usually shown other evidence of a convulsive tendency ; and in the two cases I have just mentioned it was clear that this tendency was not due to rickets, for it had been present since a few weeks after birth.

Whatever the exact pathology of these attacks may be, it is evident that a state of partial asphyxia is induced, and any one who has seen the very similar manifestations of a severe attack of laryngismus stridulus, or the attacks of laryngeal spasm which I have described elsewhere (p. 335), in the newborn, will realize that this ' going black in the face with crying ' may indicate a condition fraught with real danger.

In all these conditions, whether the convulsive manifestation be of the major or of the minor variety, there is a common basis in an abnormal nervous instability, and on this I would lay some stress, for it forms a link between these and the other convulsive conditions to which I shall refer. It would seem that this nervous

instability may be congenital or acquired ; in one case an infant shows a tendency to convulsions almost from the day of its birth, in another it is only in later infancy, when rickets has had time to develop, that a convulsive tendency appears. This difference may be of some prognostic significance ; at any rate, I strongly suspect that the infant who suffers repeatedly with convulsions under the age of six months is more likely to show evidence of nervous instability, in convulsions or otherwise, in later years than is the infant who only develops its convulsive attacks under stress of rickets ; though, of course, it must be understood that the child who has already shown the congenital tendency will be specially prone to show its nervous instability even more markedly when the influence of rickets is superadded. Certain it is that there are children who from earliest infancy show a nervous instability which remains for years, and may manifest itself in convulsions at the onset of every acute illness which befalls the child. I would not recommend any medical man to prophesy in such a case, but I remember one case in which the family doctor gained no little credit from having foretold such an occurrence ; he had seen the child at the age of three months, when it had had repeated convulsions, and recognizing the unstable nervous equilibrium of the child, he told the mother that if ever the child had an acute illness it would probably begin with a convulsion. Two years later, when I saw the child, it had just begun an acute bronchitis with a temperature of 103·8° and a convulsion.

There can, I think, be little doubt that in certain cases also infantile convulsions foreshadow other manifestations of nervous instability which appear in later childhood ; for instance, a boy of ten years had convulsions from early infancy up to the age of three years; since then he has suffered with night terrors, and occasionally walks in his sleep ; he is extremely ' nervous ', and after any little excitement suffers with insomnia. In another child convulsions occurred occasionally up to the age of $3\frac{1}{2}$ years, and at the age of six years he came to the out-patient clinic, with a marked habit-spasm, twitching of the facial muscles, and blinking of the eyes. Another child who had suffered with convulsions in infancy came under my care for hysterical dyspnœa at the age of twelve years.

Of course we must be careful in tracing any relation between conditions so common as infantile convulsions on the one hand, and habit-spasm, night terrors, or the various neuroses of childhood on the other, but the sequence is at any rate sufficiently

frequent to justify the suspicion that both are dependent on one underlying cause, a congenital nervous instability, and to this extent the earlier manifestations may foreshadow the later. Dr. Coutts,[1] in an interesting paper on this subject, has pointed out that infantile convulsions are often followed by neuroses, not only in childhood but also in later life.

But when we come to consider the convulsive conditions which are associated with rickets it is evident that here we have to do with an acquired morbid state ; it is only after the development of the rickets that the convulsive tendency appears, and it passes off as the rickety condition improves. And in this connexion I should like to draw attention to an interesting point, namely, that the nervous symptoms of rickets are not necessarily proportionate to the bony changes present ; one child with extreme rickety deformity of skull, thorax, and limbs will show no tendency to convulsive manifestations, whilst another with comparatively slight rickety change in the bones will suffer from almost all the possible nervous symptoms of rickets. It would seem indeed that, as has been noticed in many other diseases, the stress of rickets may fall upon different tissues in different children, and I have seen cases which strikingly suggest that in certain families, as well as in certain individuals, there is, if I may so say, a tissue proclivity, which determines the incidence of rickets on certain tissues. However this may be, I would emphasize the fact that with slight evidence of rickets in the bones there may be marked nervous symptoms, and vice versa, with severe bony change there may be little or no nervous disorder.

The acquired nervous instability of rickets may manifest itself in local as well as in general symptoms, and I wish to lay stress particularly on these local manifestations, for a proper appreciation of their significance has a very practical value.

Laryngismus stridulus. Laryngismus stridulus is one of these ; it is a common condition in the out-patient room of a children's hospital ; a rickety infant, on being awakened from sleep, or perhaps more often still when he begins to cry, suddenly seems to hold his breath, his chest is fixed, his face becomes more and more cyanosed, his features are contorted as if in terror, and just as asphyxia seems imminent inspiration occurs with a long-drawn crowing sound very like the whoop of pertussis. Now what has happened ? Some slight stimulus, perhaps a deeper inspiration than usual bringing cold air in contact with the glottis, has started a spasmodic or convulsive closure of the

[1] *Trans. Internat. Med. Congress*, 1887.

glottis,—a tonic convulsion, in fact, of that particular part. The close relation of such attacks to general convulsions is seen in the history of many of these cases: in a large proportion—35 out of 73 cases, i.e. 47·9 per cent.—general convulsions have occurred within a few weeks before or after the first appearance of laryngismus stridulus. The attack itself may end in a general convulsion, but this may be a result rather of the extreme cyanosis than of an extension of the convulsive phenomena.

Laryngismus stridulus is closely connected with rickets: the bone manifestations of rickets were found in 63 (94 per cent.) out of 67 cases of laryngismus stridulus. Its onset is almost exclusively between the ages of four months and eighteen months. In 73 cases under my own observation the age at onset was as follows:

Age at onset.	*No. of Cases.*
Birth to 4 months	0
4–6 months	19
6–12 months	41
12–18 months	10
20 months	1
3–4 years	2

The age-incidence would harmonize well with rickets as the chief factor in the causation of this disorder: six to eighteen months is the age at which the morbid changes of rickets are most active, and a tendency to convulsive disorders is one of the dominant features of rickets. But although it is, I think, clear that rickets plays a very large part in the production of laryngismus stridulus, there are probably other factors, for unlike most of the manifestations of rickets which occur at no special season of the year, laryngismus stridulus has a very striking seasonal variation, as the accompanying chart (Fig. 45) shows.

One might suggest that during the winter months an infant is likely to be kept indoors much more than in summer weather and the unwholesome atmosphere of stuffy rooms may aggravate the nervous instability of rickets, but beyond this mere speculation I have no explanation to offer of this seasonal incidence.

I would call attention, however, to the similarity between this chart and the seasonal chart of spasmus nutans (p. 771), another functional nervous disorder of infancy.

A very remarkable association with laryngismus stridulus is craniotabes: twenty-one out of fifty-five cases showed this affection of the skull. Statistics of a series of cases of craniotabes show even more strikingly how close is the connexion between

craniotabes and laryngismus stridulus : seventeen out of twenty-two cases of craniotabes had laryngismus stridulus. I have considered the significance of craniotabes elsewhere (p. 96), I only mention it here because its association with laryngismus stridulus is so close that it sometimes affords valuable confirmation of diagnosis when we have to judge of the presence of laryngismus stridulus from a mother's account without seeing one of the attacks.

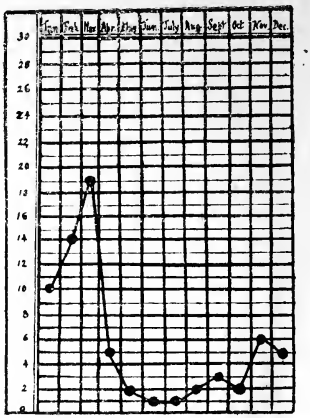

FIG. 45. Chart showing seasonal incidence of laryngismus stridulus.

There are two other nervous manifestations which are commonly associated with this laryngeal spasm, namely, facial irritability and tetany.

Facial irritability. As an indication of a convulsive tendency in rickety children 'facial irritability' is, I think, of considerable practical value, and has not received the attention which it deserves. It is seen most often in association with laryngismus stridulus, but it occurs also apart from this and occasionally even apart from rickets. Out of seventy-one cases of laryngismus stridulus, in which I noted this point, fifty-seven showed this facial irritability and three showed a similar excitability of the nerves only in the limbs. The name 'facial irritability' is unsatisfactory, for the sign to which it refers is usually present in the limbs as well as in the face,—it was so in the majority of the fifty-seven cases just mentioned—and, as I have stated, I have found it occasionally in the limbs when it could not be obtained in the face ; it would be better to speak of it as 'nerve irrita-

bility'. A gentle tap with the tip of the finger over the motor branches of a nerve produces a twitch in the corresponding muscle; perhaps the easiest place to demonstrate this is on the face, a tap on the facial nerve, where it emerges on the face just in front of the ear, produces a twitch of the orbicularis palpebrarum and sometimes of several of the neighbouring muscles; similarly a tap over the lower branches of the facial nerve, just below and external to the angle of the mouth, produces a twitch at the inner canthus of the eye and sometimes of the muscles of the nose on that side. Similarly an extensor jerk of the wrist is produced by tapping over the musculo-spiral nerve where it turns round the outer side of the humerus, and eversion of the foot, so that the outer edge is jerked upwards, by tapping over the external popliteal nerve where it turns round the outer side of the fibula. This nerve irritability is often very valuable as a danger signal: so long as a child shows this sign it is in danger of general convulsions. I am inclined to go further, and say that the degree of risk can be judged to a certain extent from the degree of the nerve irritability; where many of the facial muscles are thrown into twitchings by each tap or by mere stroking over the facial nerve, there would seem to be a greater liability to general convulsions than in those cases where the response is only slight or limited to a single muscle, usually the orbicularis palpebrarum.

Not only is this sign thus a useful indication of danger, and of the need for prophylaxis, but it may also guide us to some extent in the course of treatment, for it is often very noticeable that the diminution or cessation, under the influence of bromides, of convulsions which are dependent on rickets, occurs *pari passu* with diminution or disappearance of nerve or facial irritability. The effect of the drug on this symptom may indeed be taken as the outward and visible sign of the effectiveness of our treatment on the tendency to general convulsions, and may in this way serve as a gauge of the necessity for continuing or increasing our anti-convulsive measures. This nerve irritability is rarely found apart from rickets, and is usually associated also with laryngismus stridulus, and with another convulsive disorder which is also localized in its distribution, namely, tetany; it is, however, occasionally seen in infants who show a tendency to general convulsions during the first two or three months of life, when rickets is rare; I have seen it also in later childhood with epilepsy.

Tetany. The relation of tetany to general convulsions is very close; in a considerable proportion of the cases it is preceded or

followed by convulsions, which may also supervene at any time whilst the tetany spasm is still present in the limbs.

The tetany position of the hands and feet is so familiar that it hardly needs description here. The thumb is drawn in across the palm so that its tip is between the ring- and middle-finger; the metacarpo-phalangeal joints are semiflexed and the phalangeal joints fully extended, so that the hand assumes the position shown in the illustration. The wrist and elbows are usually kept

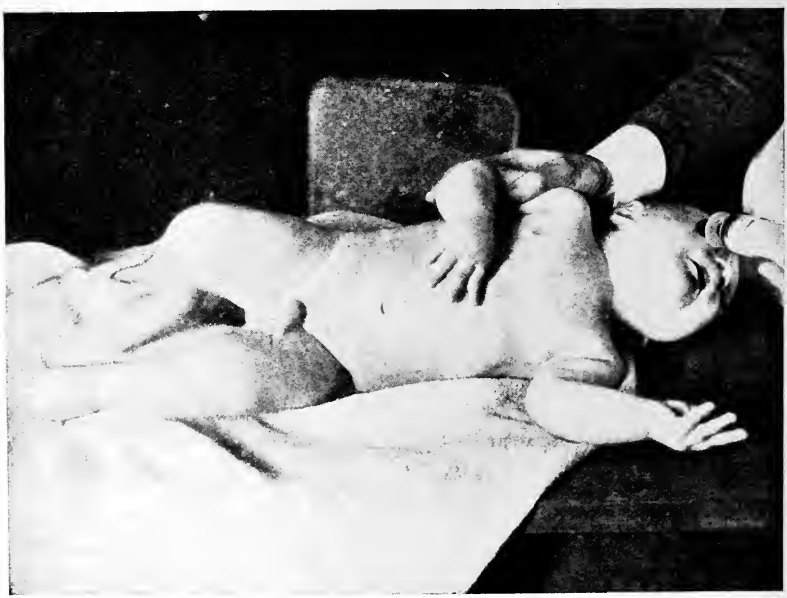

FIG. 46. Tetany. Showing characteristic position of hands. The forefinger of the right hand shows over-extension of the phalangeal joints, which is common in severe cases. In the left foot the tetany position, the crowding together and pointing of the toes, is just visible.

semiflexed. The feet are often similarly affected, the toes are crowded together, and often the ankle is extended. Very rarely spasm of some of the muscles of the trunk has been recorded; I have not seen such an affection myself.

In some children tetany evidently produces no pain, for the child will play unconcernedly with his toys, scooping them about the table when he is unable to grasp them owing to the spasm of his hands: pain seems to occur just as the spasm comes on, and in cases where the tetany position is intermittent and lasts

only for intervals of a few minutes, as it sometimes does, the child may cry out as it comes on, but where it is continuous, as it often is, for hours or days, it seems to be painless after its onset.

With the tetany spasm there is sometimes well-marked œdema of the hands and feet, and this may extend a little way up the

FIG. 47. Tetany. Showing spasmodic flexion of the wrist with the tetany position of the fingers.

limbs. I have known tetany to be mistaken for acute nephritis on account of this œdema, and the mistake might seem to be confirmed if the urine were examined, for some albuminuria—usually, however, quite a slight cloud—is also a common feature of tetany. It seems likely that the œdema is purely mechanical, being produced by slight obstruction of the venous flow in the limbs owing to the spasmodic contraction of the muscles. The

albuminuria is probably a manifestation of the toxæmia which seems to be the exciting cause of tetany.

Tetany in children is almost always associated with rickets and is evidently comparable to the other convulsive manifestations with which it often keeps company, laryngismus stridulus, facial irritability and convulsions; but there is also associated very closely with tetany in almost all instances some definite gastro-intestinal disturbance; usually there has been diarrhœa either shortly before or at the time of the onset of the spasm, sometimes the stools without being loose have been green and extraordinarily offensive.

This association is so constant that one can hardly doubt that the bowel condition plays an important part in the production of tetany, and probably also of the albuminuria which goes with tetany, for, as I have pointed out elsewhere, apart from tetany diarrhœa in infants is often associated with slight albuminuria.

The causal relation of gastro-intestinal disturbance to tetany receives further confirmation from the occurrence of tetany with so-called cœliac disease, and also with chronic dilatation of the colon, and sometimes after irrigation of the bowel (see p. 242).

Tetany is not always the obvious condition I have described, in which the characteristic position of the hands and feet is present spontaneously; there is a latent tetany which is only to be detected by a special method which I shall illustrate by the record of an actual case.

A rickety infant, aged nineteen months, had laryngismus stridulus nine weeks ago; three weeks ago the infant had general convulsions; it has now very marked facial irritability, and although there is no apparent trace of tetany in the hands or feet, the effect of compressing the arm by encircling it just below the insertion of the deltoid muscle with one's forefinger and thumb, so as to compress the brachial vessels and nerves, is to produce in about thirty seconds the characteristic tonic spasm of tetany in the hand.

This artificial method of exciting the tetany spasm is known as 'Trousseau's sign'; and, as in this case, the presence of this sign is almost always associated with those other convulsive manifestations to which I have already referred, and is therefore an indication of danger.

This latent tetany clearly differs from the ordinary variety only in the degree of tendency to spasm; for when spontaneous tetany has recently passed off it is often possible to reproduce it by Trousseau's method: the child with latent tetany is, so to speak, on the verge of spontaneous tetany.

I find amongst my notes 55 cases of tetany, of which 25 were of the spontaneous variety, and 30 of the latent; 13 of the former were associated with laryngismus stridulus, and 29 of the latter.

The close connexion with convulsions is seen in the fact that 28 out of my 55 cases had convulsions either shortly before or during the tetany, and this convulsive tendency is confirmed in most cases of tetany by the presence of facial—or, as I would prefer to say, nerve—irritability. It is in this tendency to convulsion that the chief danger of this disorder lies, whether the tetany be spontaneous or only produced by Trousseau's method; and this brings me to the question of prognosis.

Prognosis. What is the immediate risk entailed by an infantile convulsion, and what are the more remote risks?

In estimating the danger to life the first point to be taken into account is the age of the infant; there can be no doubt that the danger is much greater during the first year of life than subsequently. It has been calculated that 89·5 per cent. of the deaths from convulsions occur in infants under a year; and this high proportion is not, I think, altogether due to the fact that convulsions are commoner at this period than in older children, for it seems probable that there is no such disproportion between the frequency of convulsions in the first year and in the second as to account for this percentage of mortality in the earlier period. The following statistics are quoted by Lewis (Keating's *Encyclopæd.*) from Jamieson, who found that out of 365 fatal cases of convulsions in children under five years of age, 236 occurred within the first twelve months, and out of this number 165 occurred under the age of six months.

Age.	*Number of deaths.*
0–1 year	236
1–2 years	73
2–3 ,,	32
3–4 ,,	13
4–5 ,,	11

On the whole, it would seem also that the earlier the date of convulsions in the first year the greater the danger to life.

In considering the immediate prognosis, naturally the severity of the convulsion, and its duration, must be taken into account; such a condition as I have already mentioned as comparable to the status epilepticus of adults is fraught with much more serious risk to life than the slight twitching with little or no loss of consciousness which occurs in many infants. At the same time one must always remember that even when they occur within a few days after birth, convulsions of extreme severity may end in perfect recovery, and that a fatal result is very exceptional. As long ago as 1789 it was pointed out

by Underwood[1] that 'children are much oftener supposed to die of convulsions than they really do, for though a convulsion frequently closes the scene, it has generally arisen from the great irritability of their nerves, and violence of the disease under which they have laboured'.

But apart from the risk to life there are other very serious risks from infantile convulsions which must be considered. There can, I think, be no doubt that in a certain number of cases, probably an extremely small proportion of the whole number of infants who have convulsions, a severe attack of convulsion results in permanent impairment of intellect. Take, for instance, such a case as the following. A boy, aged 5½ years, had been in apparently perfect health, up to the age of six months, then he had thirty convulsions in two days; since then he has only had convulsions on two occasions, once at the age of twelve months, and again at 3½ years; but he is a boisterous, noisy idiot, he only learnt to walk at four years, and is dirty in his habits. His head is of normal size, his palate is high. Labour was natural and easy, and there was no difficulty in establishing respiration; there is, in fact, no apparent cause for idiocy apart from the severe convulsions.

It is, of course, extremely difficult to prove a causal relation in any such case, but it seemed even more apparent in the following. A girl, aged 8½ years, had learnt to walk and talk at the usual age, and seemed perfectly healthy up to the age of two years and eleven months; then she had ten convulsions in one day, and subsequently often eight or nine a day. Since the onset of these attacks she has ceased to talk, and has become imbecile; her tongue lolls out, and she takes little notice of her surroundings.

Happily such a result would seem to be extremely rare, but when it occurs it is probably more often from convulsions in very early infancy than from those occurring later. It is to be remembered, however, that while infantile convulsions are probably only a rare *cause* of idiocy, they are particularly common in association therewith. Statistics from 100 consecutive cases of idiocy or imbecility under my own care showed that infantile convulsions occurred in at least 22 per cent. of idiots; and if one included convulsions occurring after the age of two years (the end of infancy), the proportion would be 28 per cent., but out of these 100 cases there were probably not more than three in which there was any likelihood that the convulsions

[1] *Treatise on Diseases of Children*, p. 82.

were the cause of the idiocy. Whilst, therefore, the risk of idiocy resulting from convulsions would appear to be small, we must remember that convulsions, if not the cause, may at any rate be the first indication of anything abnormal in an infant who is subsequently found to be an idiot.

But apart from idiocy there are other abnormal mental conditions which are probably in some cases the result of infantile convulsions. I have already mentioned that neuroses in older children may be a later manifestation of that same nervous instability which allowed the occurrence of convulsions in infancy; but it seems almost certain that occasionally infantile convulsions are the cause of mental abnormalities with which there is little or no impairment of intellectual power. Some of these infants who have had a severe attack of convulsions grow up eccentric even in their childhood, odd children with curious habits, hardly imbecile in the slightest degree, and yet 'not like other children'. Then, again, it seems almost certain, as I have pointed out elsewhere,[1] that a severe bout of convulsions in infancy may cause an arrest of moral development, with the result that the child from its earliest years is passionate, spiteful, or perhaps an incorrigible liar or thief.

Fortunately all these mental disturbances are rare in comparison with the frequency of convulsions, and so far as I have seen, they would seem to result almost exclusively from very severe or prolonged attacks of convulsion; but they are worth remembering in all cases, and must 'give us pause' when we are asked, will the child's mind be affected?

It is not only the higher functions of the brain, however, which may suffer permanent damage, but also the motor functions. Some of the cerebral palsies of children, particularly the so-called 'infantile hemiplegia', may be the direct result of a severe convulsion. Whether the intense venous congestion during the attack causes some hæmorrhage on the surface or into the substance of the brain, or whether this venous congestion so profoundly modifies the cells of the cortex that their function is henceforth impaired, may be uncertain, but of the clinical sequence there can be no doubt. Here again, however, prognosis must be wary; there are cases in which a complete hemiplegia, differing in no way from the initial stage of an ordinary permanent infantile hemiplegia, results from a severe infantile convulsion, and, after lasting for several hours, gradually disappears completely. In one infant, aged nineteen months,

[1] Goulstonian Lectures, *Lancet*, 1902.

who was under my care at King's College Hospital, the left side was paralysed for four hours after a convulsion. In another, paralysis, also of the left side, remained for three hours only. In both these cases the paralytic affection was merely transitory, but had they been seen during the paralysis there would have been nothing to distinguish them from cases of infantile hemiplegia beginning with a convulsion ; and it is to be remembered that fully 50 per cent. of cases of permanent infantile hemiplegia begin with a convulsion, although perhaps only a very small proportion of these have been caused by the convulsion.

Blindness lasting for several days or weeks has been observed after convulsions. Mr. Sidney Stephenson and the late Dr. Henry Ashby have recorded such cases : out of eleven cases under three years of age, including their own and those reported by others, ten had completely recovered sight, only one seemed likely to be permanently blind : in some of these cases, however, the convulsions were only a symptom of gross disease of the brain, for the child was left with permanent spastic paralysis.

There are other results which, although less serious, have some practical importance. In the later months of infancy, when speech is in process of development, a convulsion may result in stuttering, and if we regard stuttering as a disturbance of co-ordination we might almost have expected that a convulsion might produce it. Squinting also is undoubtedly in some cases a direct sequel and result of such an attack, whether the squint be temporary, as it is in some cases, or permanent, as it is in others ; and this fact of the occurrence of squint, apart from more serious results, is worthy of note, for the question of commencing meningitis is so often raised by convulsions, and may be extremely difficult to determine. Take, for instance, the following case.

An infant three months old had been restless and sleeping badly for five days, and then had eight convulsions within a few hours. Two days later the doctor noticed slight retraction of the head, and the infant became much flushed at times. The bowels were very costive ; there was no vomiting. On the ninth day of the illness more convulsions occurred, the infant was apathetic, there was a squint, and the fontanelle was bulged. At this stage I saw the infant, and found it as described, drowsy, and with very marked bulging fontanelle, but with no definite squint, although this was said to have been present earlier in the day. There was no paralysis anywhere, but there was an occasional momentary twitch of the hand. There was no stiffness of the neck at that time ; the abdomen was natural. On the whole, the balance of evidence seemed to be against meningitis, and this opinion was confirmed by a steady convalescence under chloral and bromide.

Another point which must be considered in prognosis is the

relation of infantile convulsions to later epilepsy. When a child of two and a half or three years is brought with a history of convulsions from early infancy it is no easy matter to say whether the present attacks are to be regarded as epileptic or not; and, indeed, who shall decide where the line is to be drawn?

There are cases in which convulsive attacks occur at intervals from early infancy onwards to the end of childhood and into adult life; but, so far as my own experience goes, I should say that out of the enormous number of infants who have convulsions an extremely small proportion become epileptic, at any rate before the age of puberty. This, be it observed, does not necessarily contravene the fact that of cases of epilepsy in later life a considerable proportion, about 12 per cent. according to Gowers, or 40 per cent. according to Osler, have begun during the first three years of life as infantile convulsions.

Even when infantile convulsions have persisted beyond the end of the second year they usually cease before the end of the fourth year, and often before the end of the third year.

But without any direct continuity it seems likely enough that, like the neuroses to which I have already referred, epilepsy also should be an occasional manifestation in later life of the nervous instability which conduced to convulsions in infancy.

With regard to the prognosis of the local convulsive conditions to which I have referred, it will be gathered from what has been already said that the chief risk both of laryngismus stridulus and of tetany lies in the tendency to general convulsions which these local affections indicate; but the former at any rate has also a danger of its own, namely, the possibility of asphyxia during the closure of the glottis by the convulsive spasm; and although no doubt this is a more likely occurrence where the attacks are severe, I would strongly advise the medical man never to say that a case with apparently mild laryngismus stridulus is free from risk, for at any moment a more severe attack may occur and end fatally. I have seen such an unexpected ending more than once, and have had occasion to regret the favourable opinion which I had formed. I remember in particular an infant of seven months who had what appeared to be quite a slight degree of laryngismus stridulus; it was well nourished, with only a moderate degree of rickets, and some facial irritability. It was attending as an out-patient, and one day on its way home in the tram-car it was suddenly seized with an attack of laryngismus stridulus; the parents rushed with it

to a doctor's house which they were passing, but the child was dead by the time they reached there.

Tetany *per se* involves practically no risk to life, although in very rare instances affection of the respiratory muscles by the tetany spasm has ended fatally. But such affection of the respiratory muscles must, I think, be an extreme rarity ; amongst a very considerable number of cases of tetany in infancy and early childhood which have come under my notice I have never seen such an occurrence.

In all these conditions, however, the risk of general convulsions is a very real one, and I come now to the consideration of their treatment.

Treatment. There are few of the exigencies of childhood which are more alarming and distressing to parents than convulsions in a child, and it is necessary in most cases to adopt some active treatment immediately, almost as much for the parents' sake as for the child's.

The traditional warm bath has, at any rate, the merit that it is almost always available ; and if it does no good, it is at any rate not likely to do any harm. The water should be at a temperature of 100°–105° F., and the child should be kept in it, immersed to the neck, for about five minutes. In some cases this seems to have a quieting effect, and perhaps the benefit is increased by placing a cold or iced water compress over the child's head.

Far more effectual than the bath is the administration by rectum of bromide or of chloral ; and here I would insist upon the greater effectiveness of chloral, and its value in any case of prolonged or frequently repeated convulsions. I have sometimes seen a small dose of chloral put a stop to the convulsions which had continued in spite of large doses of bromide.

But whether bromide or chloral is used, the drug will often require to be given with a free hand ; and especially in dealing with the serious condition which I have compared with the status epilepticus of the adult, heroic doses may be necessary.

Even very young infants take chloral well in small doses. I saw with Dr. Key, of Southsea, an infant fifteen days old who had been in convulsions at short intervals for twelve hours. Potassium bromide had been given by the mouth in doses of $3\frac{1}{3}$ grains, which for the last three hours had been repeated hourly; some diminution of the convulsions had occurred, but there were still frequent twitchings of the limbs. Chloral gr. $\frac{1}{2}$ was then ordered immediately, and to be repeated alternating with

the bromide every three hours; and no further convulsions occurred. By the rectum a grain dose of chloral might have been given to an infant at this early age, and if necessary might have been repeated in two hours' time; whilst for an infant three months old 2 grains might be given by rectum at intervals of three hours if necessary. At the age of twelve months $2\frac{1}{2}$ grains may be given by the mouth, or double that dose by rectum if the case is urgent, but probably in most cases a dose of 1 grain by mouth repeated every three hours will be sufficient.

It should be borne in mind in using chloral for infants and young children, that when given by mouth it sometimes causes gastro-intestinal irritation, with consequent vomiting or looseness of the bowels; and for this reason it is more suitable as an occasional resource than for continued daily administration.

A drug which is sometimes of value, both for recurring infantile convulsions and for frequent epilepsy in children, is urethane; I have known this to be successful where bromide, phenazone and chloral had all failed: to an infant of nine months I have given $1\frac{3}{4}$ grains three times a day, but I have known a smaller dose than this to succeed, for instance, to a child of nineteen months I gave 1 grain three times a day, and there was complete cessation of the convulsions which had been occurring for seven weeks, sometimes as many as twenty or thirty times a day: in this case 5 grains of sodium bromide three times a day had had little or no effect: within thirty-six hours after the urethane was begun the convulsions stopped, and when my last note was made thirteen days later there had been no recurrence. To older children, say at six or ten years, I have given 3–8 grains three times a day.

Morphia has been recommended, and I have seen it do good in these cases; as much as a forty-eighth of a grain may be given hypodermically to an infant six months old. I have, indeed, given more than this, but I would advise rather to begin with less, and always to feel one's way carefully in the use of morphia hypodermically for infants; a sixtieth or even a hundredth of a grain may be quite sufficient. It is better to err on the side of safety.

Amyl nitrite has sometimes done good; it may be given by inhalation from capsules containing 1 minim.

If these measures fail the inhalation of chloroform is often of value, although prolonged administration may be necessary. Other remedies which have had their advocates are lancing of the gums where these are swollen and tense, the application

of one or more leeches behind the ears, and the administration of oxygen. Lumbar puncture has also been recommended.

More satisfactory than any remedial measures is the prophylaxis of convulsions. As I have already pointed out, there are several little indications, such as the presence of 'facial irritation', or of those slight manifestations which the mother calls 'the inward convulsions', which may apprise us of the tendency to the more serious general attack, and in such circumstances much may be done in the way of prevention.

Perhaps the commonest exciting cause of a convulsion is constipation. I select this out of many exciting causes which may call for treatment, because I feel sure that, especially in the convulsions which occur during the first few months of infancy, constipation is so often the determining cause that regulation of the bowels should be our first thought in prophylactic measures. In rickety infants the administration of cod-liver oil has a very striking effect in reducing the nervous instability, and this effect is noticeable before there is any amelioration of the osseous changes. When there is marked facial irritability, or where convulsions have already occurred, I usually order 1 or 2 grains of bromide with an emulsion of cod-liver oil, and the rapid disappearance of the convulsive tendency therewith is often very noticeable.

I am fond of phenazone for infants or young children who show a tendency to convulsive attacks of any sort: this drug seems to me to be much less depressing to the general health than bromide, its prolonged use does not produce the pale, unwholesome look and general depression and dullness which bromide does: there are also cases in which bromides, even in small doses, very quickly produce a bromide rash, sometimes an acneiform eruption looking almost like small pustules and occurring mostly on the limbs, sometimes a warty patch like fungating granulation tissue, which, if it occur on the face and be followed by keloid, as has happened, may be a lifelong disfigurement. If the nature of the eruption is quickly recognized it slowly passes off after the bromide is discontinued, but I must point out that the first appearance of a bromide rash may be several days after the administration of the drug has ceased. It is obviously desirable to avoid such eruptions if possible, and by using phenazone we may enable the child to do without bromide altogether, or if these two drugs are used in combination we may use a much smaller dose of the bromide than would otherwise be required.

With regard to the treatment of laryngismus stridulus, it has been held by some that the rational treatment of this disorder is to give 'tone' to the nervous system by cold douches to the back, by fresh air, and by good hygiene in general, and that the administration of bromides is unnecessary. But whilst I would not for a moment dispute the value of such hygienic measures, I would insist upon the very real risk of sudden death during the convulsive spasm of the larynx, and on this ground it is certainly advisable to give bromide in laryngismus stridulus, combining this drug with Ol. Morrhuæ, and, of course, not neglecting the hygienic treatment. There can, I think, be no doubt that the attacks are rapidly diminished, both in frequency and in severity, by the administration of bromides, or of such sedatives as chloral or phenazone, and in so risky a disease we cannot afford to adopt any line of treatment which means a slower, if not less sure, cessation of the attacks.

During the spasm of laryngismus stridulus a hot sponge applied over the larynx, or cold water splashed over the chest or face, may shorten the attack. In one fatal case, in which I happened to be at the bedside when the attack occurred, the spasm was so prolonged and the closure of glottis so complete, that I believe nothing short of tracheotomy could have saved life; but who would not hesitate—perchance, as in this case, hesitate a few seconds too long—to adopt such treatment for a condition which his experience tells him is usually quite transient? The treatment of tetany involves not only the prophylaxis of general convulsions on the lines which I have already indicated, but particularly careful attention to the state of the bowels; the very striking relation of tetany to a preceding diarrhœa or some abnormal character of the stools strongly suggests that the toxic influence, whatever it may be, which results in tetany, arises usually from the gastro-intestinal tract, and that our therapeutic measures must deal with this. As in laryngismus stridulus, the use of bromides is not only of value in relieving the local spasm, but has the advantage also of reducing the risk of supervention of general convulsions.

CHAPTER XLVI

EPILEPSY IN INFANCY AND CHILDHOOD

Epilepsy is a disease of all ages, but its beginnings often date from childhood, sometimes from infancy. According to Sir William Gowers, one-eighth of all cases commence during the first three years of life, but the most frequent age of onset is from fourteen to sixteen years.

Every medical man is only too familiar with these distressing cases, but when the disease occurs in childhood he is confronted with an anxious parent, and must needs answer questions as to régime and prognosis which have special aspects in the case of the child. Even as to diagnosis problems will arise which are peculiar to infancy and childhood. Is the attack a simple infantile convulsion ? Or, if the child be at the school age, is the attack merely the reflex result of some dietetic vagary or of the indiscretions of a children's party ? Is it an isolated occurrence which may never be repeated, or is it the precursor of an epilepsy which will blight the child's prospects for life ?

There are many such points, with reference to which experience of epilepsy as seen in the adult will help but little in the case of the child. It is only by studying the disease as it affects infants and children that we can hope to appreciate the peculiar features and to solve the special problems which it presents at this age.

Sex. There seems to be no special liability of either sex. Boys and girls are equally affected. Of 100 consecutive cases from my notebooks, 50 were boys and 50 girls.

Age. It is not always easy to be sure from the history at what age epilepsy first occurred, for often there have been convulsive attacks in infancy, which may or may not have been of this nature ; sometimes the persistent recurrence of the attacks, as the child grows older, seems to justify the assumption that they were from the beginning epileptic ; sometimes there have been only two or three attacks in infancy, which were thought to be ordinary infantile convulsions, then, after an interval of months or years, attacks have begun which run the course of epilepsy ; who shall say whether the earlier attacks in such a case were really the first manifestations of epilepsy ? There are indeed

no valid distinctions between the symptoms of an ordinary infantile convulsion and an attack of major epilepsy, the distinction between the two affections, so far as there is any, lies in the course, not in the symptoms; the one being dependent upon a nervous instability which in greater or less degree is a physiological feature of early life, tends to subside as the child grows older; the other being due to some morbid erethism of the brain which is independent of age, is likely to persist for years, if not for life.

There are, however, cases in which even in infancy it is possible to be quite sure of the epileptic character of the attacks. I refer to those in which there are symptoms of petit mal. The infant who suddenly jerks the head forward on to the sternum and then for a moment looks dazed, or who shows a sudden flexion of the trunk with rigid extension of the arms and momentary loss of consciousness, is undoubtedly epileptic.

In a certain number of cases the doubt must remain, and with this reservation the following statistics give some idea of the frequency with which the epilepsy of childhood dates from infancy. In 42 per cent. of children with epilepsy—and my figures in this chapter include only children under twelve years of age—the attacks began during the first two years of life. In at least 28 per cent. the infantile attacks merged directly into the epilepsy of later childhood, and in the majority of cases in which this happened the attacks during infancy could not have been distinguished from ordinary infantile convulsions.

It is hardly necessary to point out that the statement that epilepsy is frequently a sequel of convulsive attacks in infancy does not by any means involve the entirely distinct proposition that infantile convulsions commonly lead to epilepsy; the contrary indeed is the fact. An extremely small proportion of the children who have infantile convulsions ever become epileptic.

The age at onset is shown in the following statistics of 100 cases:

Age at onset.	*No. of cases.*
Birth to 2 years	42
2–3 years	18
3–4 years	5
4–5 years	5
5–6 years	5
6–7 years	6
7–8 years	8
8–9 years	3
9–10 years	4
10–11 years	2
11–12 years	2

It is evident that the majority of cases begin during the first three years of life, and that there is no period of special liability during the rest of childhood unless the very slightly increased frequency of onset during the seventh and eighth years can be taken as an indication that the second dentition is an exciting cause of epilepsy, as has been affirmed by some observers.

Etiology. Any full description of the etiology of epilepsy would be out of place here. There are some causes, however, which are peculiar to childhood and there are others upon which the study of epilepsy at this age has some special bearing.

In children one seems to see more clearly than in the adult how largely epilepsy is an inborn vice. The exciting cause is but the spark that fires the train. The necessary antecedent is the congenital tendency, for which in a large proportion of the cases heredity is responsible; not necessarily the inheritance of an epileptic strain, but often only of a neuropathic taint, which in the child's parents or near relatives may have shown itself as insanity, or alcoholism, or in less pronounced degree as asthma, migraine, or neurosis of one kind or another. The inborn tendency of the child seems to be shown by the early occurrence of the attacks in many cases. A considerable proportion occur within the first three months of life, some even within the first few days. The epileptic in fact is, in the majority of cases, an epileptic from birth, albeit the first manifestations of his morbid tendency may be delayed for months or years.

Life has its periods of special danger, when the flashpoint of nervous explosion is at its lowest, periods which may be passed in safety by the person of average stability, but which, to the victim of neuropathic inheritance are likely to be a time of catastrophe. That these correspond with periods of special activity of physiological development is surely more than a coincidence. In the adult no one will deny that the periods of active functional change, the time of pregnancy, and the age of the climacteric, to which perhaps there corresponds a certain age in the male also, are periods of special stress when such affections as epilepsy and insanity are only too likely to make their appearance. Surely there is nothing unreasonable in supposing that the time of the first dentition, perhaps also of the second dentition, and the age of puberty, may also be critical periods for the child of neuropathic tendency. As a matter of observation, I am satisfied that undoubted epileptic attacks sometimes recur with great frequency throughout the period of dentition, and then either become very infrequent or entirely

cease when the last tooth has made its appearance. In such cases, sooner or later, the attacks are likely to return, but their temporary cessation corresponds so exactly with the end of dentition that it seems to me impossible to doubt the connexion.

With regard to the second dentition the association is less striking, but the slight increase in the frequency of onset during the time when the second dentition first becomes active, namely, between the ages of six and eight years, is probably more than a mere accident of statistics. As I have elsewhere (p. 641) mentioned, habit-spasm shows an increased frequency of onset between the ages of six and eight years; acquired enuresis also might be instanced as another nervous disorder, the onset of which is specially incident to this period.

Puberty is by common consent acknowledged as one of the periods at which the first manifestations of epilepsy are most likely to occur.

One is apt to think of epilepsy as distinguishable from other convulsive conditions, particularly the convulsions of infancy and the much rarer reflex convulsions of later childhood, by the apparent causelessness of its outbreaks, but this is by no means an infallible guide. At all periods of infancy and childhood genuine epileptic attacks may be excited by some ascertainable irritation, be it the presence of worms in the bowel, the retention of hard scybala in the colon, an indigestible meal, or some unusual excitement. Nevertheless, in a general way it is true that the epileptic attack is traceable but seldom to any definite exciting cause.

Whether the presence of adenoid hypertrophy or enlarged tonsils is ever sufficient to cause epilepsy is in my opinion very doubtful. I have known the attacks to cease for a time after removal of the naso-pharyngeal obstruction, but as a rule the operation has not the slightest beneficial effect upon epilepsy. Nor again am I convinced that the worry of an adherent prepuce is any but an extremely rare exciting cause of the attacks.

Among the less recognized causes of epilepsy in childhood congenital syphilis must be included. Its *modus operandi* is not obvious, for there is no evidence in such cases usually of any organic lesion affecting the brain, and, apart from any other symptoms of syphilis which may be present, the epilepsy runs the same course as in non-syphilitic children. In a case which showed for many months symptoms of ordinary petit mal, the subsequent localized character of the attacks led to trephining and the discovery of syphilitic meningitis. In the category of

syphilis have also to be reckoned a small proportion of the cases in which epilepsy is associated with cerebral palsy dependent upon organic lesions, to wit, infantile hemiplegia, spastic diplegia, and paraplegia, with all of which epilepsy is a frequent concomitant. These cases, however, I have not included in the statistics given above, which refer only to so-called idiopathic epilepsy.

Some have attributed to masturbation a part in the production of epilepsy. I must have been consulted about a large number of children for masturbation, but only in one single case have I known this to be an exciting cause of epilepsy, and I am strongly of opinion that, apart from a pre-existing tendency to epilepsy, this habit has no such risk attaching to it.

Symptoms. In infancy, as in later childhood, epilepsy may be of the minor or the major variety. The latter is more likely to cause doubt in diagnosis in the infant than in the older child. In children past the age of infancy the major attack presents features like those in the adult. The child, with or without a sudden cry, falls to the ground unconscious ; there is increasing cyanosis, with rigidity which is soon replaced by clonic spasms of the limbs, face, and trunk, perhaps with frothing of the mouth and jerky grunting respiration as the spasm of the respiratory muscles allows, until the paroxysm seems to exhaust itself and the child lies pale and flaccid, dazed, or drowsy, as consciousness slowly returns. In the infant an attack exactly similar, except of course for the falling which the recumbent position at that age prevents, commonly passes for an ordinary infantile convulsion, until its persistent recurrence and the absence of any of the usual causes of infantile convulsions raise the suspicion that it may be epileptic. There is no difference of symptoms whereby we may distinguish between the two.

Very different is the case when symptoms of petit mal occur in an infant. Perhaps the commonest manifestation of this variety of epilepsy in infancy is a sudden jerk of the head forward. With this flexion of the neck there is often associated a sudden rigid extension of the upper limbs, with the hands clenched and the arms slightly adducted. The attack is almost instantaneous, and at first the jerk of the head may be so slight as hardly to attract the parent's attention. Sometimes the petit mal takes the form of a momentary slight ' start ' which seems to affect the whole body, and is perhaps associated with a barely perceptible quivering of the eyelids. In comparison with the distressing spectacle of the major attack, or an ordinary infantile convulsion, these momentary spasms may appear but a trivial matter ; nevertheless

it is of the utmost importance that their real significance should be recognized. Such symptoms should be regarded without hesitation as epileptic, for experience shows that they continue into later childhood, and in many cases are replaced by or alternate with attacks of major epilepsy.

In older children petit mal manifests itself in various ways. Often the child stops suddenly in his play or in the act of eating, looks vacant for a moment, and then proceeds as if nothing had happened; or perhaps in the midst of talking he suddenly turns pale, shows a momentary upturning or quivering of the eyes and becomes confused in his speech, but quickly finds his words again, and the attack is over. I have seen several cases in which the only symptom was a momentary trance-like condition with fixed gaze, so that, if walking across a road, the child suddenly stopped, stood still for a few seconds, and then went on its way as though nothing had happened. A dangerous form of the disease in these days of motors! Sometimes the attacks were described as a sudden 'dreaminess'; the child took no notice of a question, and then seemed startled when one spoke to him. It is not uncommon for attacks of petit mal to terminate in a deep audible sigh.

Both after minor and major epilepsy there sometimes occur in children, as in adults, automatic actions. A child of three under my care for severe attacks of major epilepsy invariably pulled himself up by the sides of his cot when the attack was over, and groped his way round it in an uncertain, fumbling way, quite unconscious of his surroundings, until, after three or four minutes, he regained full consciousness and seemed surprised at the position in which he found himself.

The danger of this epileptic automatism was well illustrated by the following case:—

Henri D., aged 12½ years. Had suffered with convulsions at 2 years old, and on several occasions up to the age of 5 years. These then ceased, and he seemed healthy until at the age of 9 years he became liable to curious attacks, consisting of sudden lapses into a condition resembling somnambulism. During the attack the boy always fumbled with the sleeves of his coat, as if trying to pull them down, and he would often walk forward, and if he came to a wall or other object against which he could support himself would lean against it until full consciousness returned. On one occasion an attack occurred when the boy was approaching the bank of a river: he walked straight on into the river and stood in the water fumbling as usual with his coat-sleeves; his father, who was near, called to him, and the boy came out, still unconscious of his surroundings; after walking a few steps he recovered consciousness, seemed confused, and asked why his clothes were so wet.

In addition to these categories of major and minor epilepsy, there is another which often passes unrecognized for a time, the so-called ' épilepsie larvée ', or ' masked epilepsy '. I have seen several cases in which the chief symptom was a sudden pain in the navel or epigastrium, e. g. :—

William B., aged $10\frac{1}{2}$, since the age of 9 months has had sudden attacks of pain in the epigastrium ; this lasts a few seconds, during which there is sudden pallor, succeeded by flushing, after which the boy becomes pale and asks to lie down and goes to sleep for a long time. This occurs several times a day. With large doses of bromide these attacks were greatly reduced in number and sometimes checked altogether for several weeks.

A sudden loss of speech is sometimes the only evidence of masked epilepsy, as in the following case :—

Mary A., aged $10\frac{1}{4}$ years, is subject to curious attacks of difficulty of breathing which come on just as she is going to sleep. She gets out of bed making violent respiratory efforts, but there is no cyanosis, and she is able to come downstairs to her mother. This respiratory difficulty passes off after two or three minutes, leaving a numbness of the lips with difficulty in producing consonants or with complete loss of speech for a few minutes. The child is conscious, and has even tried to write what she was unable to say. The whole attack lasts only a few minutes. It has only once been associated with convulsive movements of the face. Under bromide there was great diminution of the attacks.

In another case the child, a girl of $9\frac{1}{2}$ years, just after falling asleep at night, woke unable to speak. Her tongue ' wobbled about ' and saliva ' dribbled ' from her mouth, but she understood what was said to her, and was sufficiently conscious to feel for her pocket-handkerchief on account of the ' drivelling '. The attack lasted about two minutes and recurred about once a fortnight.

Sleepiness is sometimes the chief manifestation of a masked epilepsy. Both major and minor attacks are often followed by more or less drowsiness or prolonged sleep, but the attacks to which I refer consist of little else, for instance :—

George G., aged $5\frac{3}{4}$, had what was supposed to be an ordinary infantile convulsion at 14 months. More recently he had shown a curious tendency to fall asleep at odd times, without warning. At any hour of the day he would sit down and go to sleep for several minutes. The nature of these attacks was not apparent until eventually the sleepiness began to be associated with epileptic twitching.

Still more pronounced was the somnolence in the following case :—

David A., aged $9\frac{3}{4}$ years, at the age of $9\frac{1}{2}$ was found one morning in bed so deeply asleep that even with strong smelling-salts he could not be roused for nearly four hours. On one occasion, in order to wake him, he was placed in

a bath, but although he was able to stand in it he remained in a state of stupor, and when he woke two hours later knew nothing about what had taken place and complained of some headache. There was nothing to indicate that these lapses into profound stupor were preceded by any epileptic convulsion. There was no history of involuntary defaecation or micturition during the attack. On the whole, it was thought that the attacks probably represented a masked epilepsy, and this was confirmed by the subsequent history, for ordinary attacks of major epilepsy followed, which persisted for two years and then ceased under treatment.

The relation of epilepsy to sleep is very remarkable: there is in many cases a special liability to attacks just as the patient is falling asleep and also just before awaking. I have notes of cases in which the fits were limited to these times. I would compare with this the behaviour of some cases of enuresis, and also of night terrors, both of which tend to occur during this intermediate state between sleeping and waking. One may suppose that the lower activities of the brain are still sensitive to stimuli after the higher controlling functions have become dormant, and that the abnormal erethism of the epileptic brain is therefore still less in check when sleep is just beginning or just ending.

The manifestations of epilepsy are often curiously localized, although the subsequent course of the disease shows that it is of the idiopathic variety rather than a symptom of some gross lesion of the brain; thus the epileptic movements may be limited at first to one side, or even to one limb, although subsequent manifestations are of the ordinary general distribution. Sometimes these localized outbreaks leave behind them a temporary weakness of the affected limb or side which may last some hours. I have elsewhere (p.661) mentioned a similar occurrence in connexion with ordinary infantile convulsions, and, as in that case, it is important to realize that such weakness does occur as a temporary phenomenon, and does not necessarily point to any permanent cerebral palsy or organic lesion.

Various subjective symptoms may be the only manifestation of epilepsy in childhood. For instance, in one child, aged $7\frac{1}{2}$ years, there were frequent sudden sensations of falling replaced at times by definite attacks of major epilepsy. In another, the child, aged $4\frac{1}{2}$ years, complained of the sensation of falling backwards, and seemed much frightened by it, although she never actually fell. This child had suffered with an ordinary epileptic fit. In another the sensation was one of giddiness. Such subjective symptoms may form an aura before an ordinary attack of major epilepsy, but the cases to which I am now referring show, in some at least of their attacks, no manifestation beyond these subjective symptoms.

An aura, in my experience, is not very common in children. I have seen several cases in which the child complained of some pain in the abdomen, usually at the navel, just before the attack begins. In some instances nausea immediately preceded the onset, and the child would run to his mother saying that an attack was coming. I have seen cases in which the attack was preceded for some hours, or even for a day or two, by extraordinary perverseness and fractiousness, which were quite unnatural to the child at other times.

This brings me to the mental condition of the epileptic child. There is no more serious aspect of epilepsy in childhood than its effect upon the mind. The result depends almost entirely upon the age at which the epilepsy begins, but the variety of epilepsy is also a consideration. To the lay mind the major attack very naturally suggests more danger to the intellect than the slight hardly-noticed quiver of the eyelids or jerk of the head which may be the only manifestation of petit mal. But these latter attacks, beginning under the age of one year, are almost invariably followed by idiocy, and the earlier they begin the more profound is the degree of the idiocy; whereas major attacks, indistinguishable at the time from ordinary infantile convulsions, though their subsequent persistence may show them to have been epileptic, are much less likely to interfere with the development of the intellect in any great degree. Beginning a little later, during the second or third year of life, epilepsy rather modifies the development of the intellect than arrests it, and it is in children who have been first affected at this age that one sees the mental peculiarities so characteristic of many epileptic children. The child seems to lose that subtle restraint which goes with the higher degrees of intellectual refinement. There is a lack of normal shyness and modesty. The child talks to strangers too readily and with too easy a familiarity; at the same time, he is abnormally impatient of control, flies into a passion on the slightest thwarting, and strikes out petulantly at his mother if she expostulates with him. The power of concentration is diminished and the child's attention wanders too easily; nevertheless there is no gross impairment of intelligence, and the child passes for a normal child albeit wayward and capricious.

Diagnosis. To the distinction between epilepsy and infantile convulsions I have already referred: there are several disorders which must be remembered as simulating epilepsy in childhood. 'Fainting attacks' are a common cloak for epilepsy. In children,

before the beginning of the second dentition, fainting is extremely uncommon, but in later childhood, especially as puberty approaches, it is by no means rare. It is often no easy matter to distinguish between fainting and epilepsy from a mother's description; especially when she is naturally anxious to obtain a verdict in favour of the less serious affection. Inquiry may elicit the fact that there was some rigidity during the attack, or that there was twitching. Either of these, however slight in degree, would be conclusive against fainting.

Children are not exempt from hysteria, and I have seen hysterical attacks resembling severe major epilepsy in a girl about 9 years, who frequently fell to the ground kicking and struggling violently; she showed no loss of consciousness, and the purposive character of her movements and the deliberate manner of her falling left no doubt as to the hysterical nature of the fit.

It must be remembered also that malingering is not unknown in childhood, and I have seen deliberate mimicry of an epileptic attack by a child aged 3 years, who had suffered with genuine and severe epilepsy. The child, finding himself under observation, would suddenly hold up the right arm and screw up the left side of his face, and then laugh at the onlooker.

A habit which may be mistaken for epilepsy is masturbation. I have on several occasions been consulted for supposed epilepsy in such cases. The stiffening of the legs, the clenching of the hands, and the reddening of the face, followed by pallor and exhaustion, make up a picture not unlike epilepsy, but there is no loss of consciousness, and careful observation shows that the movement is purposive and is directed to the excitement of a sexual orgasm. This habit of masturbation, as I have elsewhere pointed out (p. 743), occurs frequently in infancy, especially in girls, and the suspicion of masturbation may be confirmed by the finding of some soreness of the vulva, which frequently accompanies this habit. I know of no evidence that this orgasm ever passes into an epileptic attack, unless the child is already the subject of epilepsy. When this is so, the act of masturbation may occasionally be the exciting cause of an attack of epilepsy, as I have known happen in one case, where actual loss of consciousness with epileptic symptoms followed directly upon some of the bouts of masturbation, though not upon all.

The possibility of organic cerebral disease as the cause of epileptiform attacks must always be borne in mind. Both at the onset and in the later stages of the cerebral palsies of childhood such attacks are common (see pp. 702, 709), and tumours, and even

meningitis, may first manifest themselves by an epileptiform attack.

Prognosis. There are few diseases in which it is more difficult to give a forecast with any confidence than in epilepsy : but this point I would specially urge,—do not be too gloomy in prognosis. Parents and doctors alike are too apt to think that ' once epileptic, always epileptic ' is the inevitable fate of the child who has shown any evidence of this disorder. Undoubtedly there are many cases in which such a view is correct, but happily there are also many in which it would be cruelly wrong, and it is in childhood particularly that a hopeful prognosis is most likely to be justified. The favourable prognosis of epilepsy at this age was noticed even in ancient times. ' They who are troubled with the falling sickness before they attain the age of fourteen may be freed from it.' So wrote Hippocrates (I quote from Sprengell's translation), and his observation was, I think, sound. I could quote many cases in which, even after epileptic attacks had recurred for several years during the middle period of childhood—I mean from about five to ten years of age—they then ceased for years, and apparently, as is shown by the histories of adults who had suffered with the disease in childhood, in some have never returned.

The frequency of attacks is no guide in prognosis ; often indeed the child who is having a dozen or more attacks of major epilepsy every day proves much more amenable to treatment than one who is only having one attack every few weeks or months. To the uncertainty of the effects of treatment, which must make us wary in any case of giving prognosis before a thorough administration of drugs has been tried, there must be added another element of uncertainty which may baulk our most careful calculations. Epilepsy, like enuresis, is a disorder which sometimes apparently without rhyme or reason, after we have wellnigh exhausted the pharmacopœia with little or no effect, suddenly ceases spontaneously. If I had to single out one variety of epilepsy as of less hopeful prognosis than another, it would be petit mal. At all periods of childhood I think this is generally less amenable to treatment than the major variety ; and in infancy certainly it is of far more sinister significance than the major attacks, from another aspect, namely the effect upon the mind.

I have already described the mental changes which are likely to result from epilepsy occurring in infancy or early childhood; here I will only repeat that petit mal in the infant leads almost, if not quite, invariably to idiocy. The older the child at the

time of onset of the epilepsy the less the danger of mental deterioration.

It is remarkable how little effect is produced upon the mental powers by epilepsy in children, even when it recurs many times daily, provided that it has begun later than the end of the third year. With a very frequent recurrence of attacks, even in late childhood, there may be temporary dullness and slowness of mental response, but this disappears as the attacks become less frequent: with a condition of 'status epilepticus' in late childhood I have occasionally known delusions and mental confusion to occur, but even these clear away within a few days after the bout of epilepsy has subsided. It would seem indeed, as one might expect, that it is during the earlier stages of mental development, when the processes of reason are only just being acquired, that epilepsy is most disastrous to the intellect.

Treatment. There are certain general points which seem to me worth bearing in mind in connexion with the treatment of this disease. First, that as in many other nervous disorders, morbid habit probably plays no small part in epilepsy. 'Principiis obsta' is a good precept in the treatment of any disease; it is specially applicable to this one, for it seems likely that by preventing the attacks in childhood we may prevent the acquirement by the brain of the epileptic habit, which unchecked would become in later years an ingrained vice and render the patient a lifelong epileptic.

If this be so, it is surely important that treatment should be continuous over a long period. I am convinced that when bromides or any other drugs arrest the fits they should not be discontinued until many months have elapsed since the last attack; the longer we can arrest the outbreaks the more the chance of accustoming the brain to a better habit.

Secondly, that even if we cannot arrest the attacks altogether we may be able to lengthen the intervals between them: and this is especially of importance in infancy, when a diminution of the number of attacks may mean a less degree of permanent mental impairment.

Thirdly, that whilst in the majority of cases of epilepsy no exciting cause is found, it is never right to assume that this is so without investigation, for there are those in which a mere off-hand prescription of bromide or any other sedatives would be thoroughly bad treatment, where faults of digestion or local irritation, or the daily regimen, particularly as to diet, may be responsible, in part at least, for the attacks.

To deal with this last point first: the epileptic child should live a life as free as possible from excitement and emotional disturbances; a children's party, a severe scolding, a sudden fright—any such cause may determine an attack.

If there is much nasopharyngeal obstruction by adenoid hypertrophy or enlarged tonsils, it may be wise to have them removed, but it should be explained to the parents that only very rarely has such an operation any beneficial effect upon the epilepsy. I have known severe and frequent fits to cease on removal of the obstruction, but as a rule the epilepsy has not been checked even temporarily.

In every case care should be taken that the bowels are acting regularly and well, and that any digestive disorder is set right; and in this connexion it must be remembered that defective teeth are a very common cause of indigestion in infancy, and cases are on record in which the irritation of carious teeth has seemed itself to have caused epilepsy in children; both West and Tomes mention such.

Apart from dietary to ensure good digestion, certain special diets have seemed to be of value in some cases, namely the purin-free diet and the salt-free diet.[1] The purin-free diet consists of milk, eggs, butter, cheese, rice, macaroni, tapioca, white bread, cabbage, lettuce, cauliflower, sugar, and fruit. The salt-free diet consists of milk, fresh butter, eggs, fruit, white bread made without salt—or made with sodium bromide instead of sodium chloride—weak tea, coffee or cocoa, and sugar.

I have seen cases in which a purin-free diet certainly seemed to do good; but I have seen others in which a diet the very reverse in character seemed more successful, and I have seen many more in which diet did not seem to have the slightest effect upon the epilepsy. As this is so, I am accustomed to regard the special dietetic treatment of epilepsy as so uncertain and so entirely empirical, that I always reserve it for cases in which the much more reliable drug treatment has failed, which, moreover, is far less irksome to the child and his parents than the restricted choice of these special foods.

Turning now to the drug treatment of epilepsy in children, I know of nothing more generally useful than the bromides, and I must say a word upon the reluctance which both the profession and the laity often show to the prolonged and thorough administration of bromide for this disease. 'Surely,' says the mother, 'it is so dulling.' It is perfectly true that some children are made

[1] Aldren Turner, *Practitioner*, 1906, p. 545.

temporarily not only dull and sleepy, but miserable and peevish, if very large doses of bromide are given, but the quantity which can be tolerated without this effect varies in different cases, and in the vast majority of them the dose required to control the epilepsy is not sufficient to cause any mental dullness; in any case the dose should be pushed up, if necessary, until dullness is apparent, and if any occurs, the bromide can be replaced in part and reinforced by other drugs which I shall mention.

Nor is the objection that bromide makes the child look 'seedy' one which can be urged as any general reason for not using it. It is astonishing what large doses children will often take for many months not only without looking ill, but with marked improvement of their general health, owing to the cessation of the epilepsy. Many children of 9 or 10 years will take 25 or 30 grains three times daily for months and months with nothing but advantage. On the other hand, there are cases in which the child becomes very pale and heavy-eyed, and looks, as the saying is, 'seedy,' whilst taking much smaller doses, e. g. 10 or 15 grains at 9 years thrice daily. For such the drug must generally be given in much smaller doses, combined with some other drug of value for this disease.

As I mentioned in the preceding chapter, there are cases in which even in small doses bromide causes an eruption on the skin; in these some other drug must be substituted altogether.

The doses of bromide must be determined by experiment in the individual case. It often happens that with a moderate dose of bromide the attacks become less frequent, but when they do occur they are more severe; where this is so the bromide should not be stopped, but should be pushed until the attacks are more completely controlled, as is generally possible.

Infants, I think, stand prolonged administration of large doses of bromide less well than older children, and for this reason it is advisable to supplement this drug with phenazone. An infant of six months with petit mal may take a mixture of Sodium bromide gr. ijss, Phenazone gr. ss, Spirit chloroform ℥ jss, Glycerine ℥ v, Aq. anethi ad ℥j ter die, and at one year the dose of phenazone may be increased to 1 grain and the bromide, if necessary, to 4 or 5 grains; but one would not hesitate to try a watchful administration of larger doses of bromide if these failed to check the attacks, for the stakes are high; it is the child's intelligence we are trying to save.

In these infantile cases especially, chloral is often more effectual than bromide, but owing to its disturbing effect upon digestion,

its prolonged use is generally to be avoided. It is particularly suitable as a temporary measure when the attacks happen to be specially severe or frequent, and at any time it may be useful to add a small dose of chloral to a bromide mixture, so that it may not be necessary to give a large dose of either. To an infant of six months ½ grain of chloral with 1–1½ grains of sodium bromide may be given; whilst a child of 18 months or 2 years may take 1–1½ grains of chloral three times a day, for weeks if necessary; but this drug should be discontinued or diminished as soon as possible.

For older children the addition of digitalis to a bromide mixture is sometimes of undoubted value. Such a mixture as the following has been very successful for children of 7 or 8 years. ℞ Sod. bromide gr. v, Pot. bromide gr. v, Ammon. bromide gr. v, Tinct. digitalis ♏ iii, Syrup, ℥ j, Aq. menth. pip. ad ℥ ss ter die. It has been supposed that by mixing the three bromides a more sedative effect is obtained than from a similar dose of any one bromide. I am not clear that this is so, but I have seen excellent results from such a mixture, as I have also from the use of strontium bromide. Borax is sometimes distinctly beneficial as an addition to a bromide mixture: 2½ grains may be given thrice daily at one year and 5 or 6 grains at six years.

Another bromide preparation which is worthy of trial for children past the age of infancy is bromocarpin, which to a child of three years may be given in doses of 20–30 minims thrice daily, and to a child of six years in doses of half to one drachm thrice daily.

When there seems to be a special liability to the acneiform or warty eruption which sometimes comes from bromides, bromipin may be given by rectum: I have used 1–2 drachms of the weaker 10 per cent. solution for rectal injection twice daily at the age of eighteen months. Usually, however, in such cases it is best to discard bromides altogether, and a valuable substitute is urethane, of which 1–1½ grains may be given thrice daily to an infant a year old, and 3–5 grains to a child of six years. This drug dissolves readily in water and has but little taste, so that it is easy to administer. In some cases urethane is more effectual than bromide, and epilepsy which has seemed intractable has yielded to it; but like most other drugs it will often fail, and sometimes in cases where bromide proves successful.

In one child, who was not benefited by bromide, thyroid seemed to check the epilepsy to a marked degree; but this must, I think, be exceptional, for in other cases it had no good effect whatever.

In older children, as in infants, chloral is sometimes more effectual than bromide, and may be given alone or in combination with bromide; 3 grains may be given thrice daily to a child of six years, whilst a child of ten years may take 4 grains, increased if necessary to 5 grains. It is well, however, to discontinue chloral as soon as possible and always to give the smallest dose which is effectual.

In the rare cases in which a child passes rapidly from one fit to another, remaining in the status epilepticus for hours—a very rare occurrence, I think, in childhood, but one which I have seen in two or three instances—it may be necessary to give this drug by rectum, and for this purpose it must be given with a free hand. 15 grains may be given by rectum to a child of five years, whilst as much as 20 grains may be necessary for a child of ten years. The prolonged administration of chloroform is the most effective treatment if chloral fails to control the fits.

CHAPTER XLVII

INFANTILE PARALYSIS

Of all forms of paralysis in childhood infantile paralysis or acute anterior poliomyelitis is the most frequent. And yet, familiar though this disease may be, it is constantly raising difficult practical questions for the clinician, whilst for the pathologist it has remained until quite recently an entirely unsolved riddle so far as its cause is concerned.

It may now be taken as proved by the researches of Flexner and others that infantile paralysis, at any rate in its epidemic form, is an infective disease, but there are still many points which require elucidation. The nature of the virus itself is still uncertain, and though it may be assumed that the ordinary sporadic disease, with which we are all familiar, is infective and probably due to the same infection as the epidemic variety, yet we must keep an open mind until more evidence is available; for, as I shall point out, the clinical course and the apparent exciting causes of some of the sporadic cases at least suggest the possibility that similar pathological changes may be due to dissimilar causes.

The consideration of its clinical features has important bearings upon the pathological obscurities of this disease, and as a clear mental picture of the morbid anatomy of infantile paralysis is essential to the proper understanding of the questions at issue I shall describe briefly at the outset the main facts of its morbid anatomy as far as they are known. It may be taken for granted now that the clinical phenomena—the paralysis, the muscular wasting, the arrested growth, and the coldness and blueness in the affected limbs—are all due to a primary lesion in the spinal cord, with the reservation that in certain cases peripheral nerve lesions may be associated with the spinal lesion, and that possibly in rare cases a clinical picture hardly, if at all, distinguishable from that of poliomyelitis may result from a primary peripheral nerve affection. To the spinal cord we must look for the characteristic changes, and it is little use seeking to determine the character of the initial lesion or its cause from the changes found in the spinal cord months after the onset of the disease: the shrinkage of the anterior horns,

the disappearance or degenerated state of nerve-cells in the anterior cornua, the diminished size of the anterior roots, the ascending degeneration which may occur, these tell us little or nothing of the beginning of the disease. It is from the examination of cases which have died during the first few days of the disease that we may hope, sooner or later, to determine its cause; at present but few such early examinations have been recorded. My colleague, Dr. Batten, has recorded a carefully studied case which died thirteen days after the onset, and I quote his description of the changes found.

'The vessels in the grey matter are thrombosed and surrounded by perivascular exudation; there are scattered hæmorrhages throughout the grey matter and exudation of small cells. The congestion is almost limited to the ventral portion of the cord.'

In addition to these changes in the substance of the cord, there occur more or less increase of vascularity and small cell infiltration of the spinal meninges.

Now, upon the interpretation of these early changes there exists considerable doubt. Is the thrombosis ever the primary lesion, or is there always first an inflammation of which the thrombosis is merely a part? In either case is the primary lesion necessarily the result of invasion by some micro-organism? and if so, is the infection one peculiar to this disease? or, on the other hand, may the lesion result from many different causes, infective and otherwise? These are the questions raised by the morbid anatomy of infantile paralysis, and if it be not possible to give a definite answer to any of them, yet we may at least obtain some hints towards their solution from a consideration of the clinical facts of the disease.

Boys would seem to be rather more liable than girls. My own figures show in 100 cases 57 boys to 43 girls.

The age-incidence is of some interest; it is shown in the accompanying chart (Fig. 48) of 100 cases amongst children under twelve years of age. The disease may occur even in adults, but it usually begins before the end of the third year, and is far more frequent during the second year of life than at any other period. Out of 100 cases in which I have noted this point, 84 began within the first three years of life, and there is a very striking maximum incidence in the second year; 38 began between the ages of one and two years, whereas only 20 began in the first year of life. The earliest age of onset among my cases was the seventh week. I draw attention to the special incidence upon the second year of life because it contrasts with the age-

incidence of infantile hemiplegia which has its onset most often in the first year.

With reference to the age-incidence I would point out also, but without laying any great stress upon the fact, that such undoubtedly infective diseases as suppurative, posterior basic or cerebro-spinal, and tubercular meningitis have their maximum incidence during the first three years of life, so that it might be

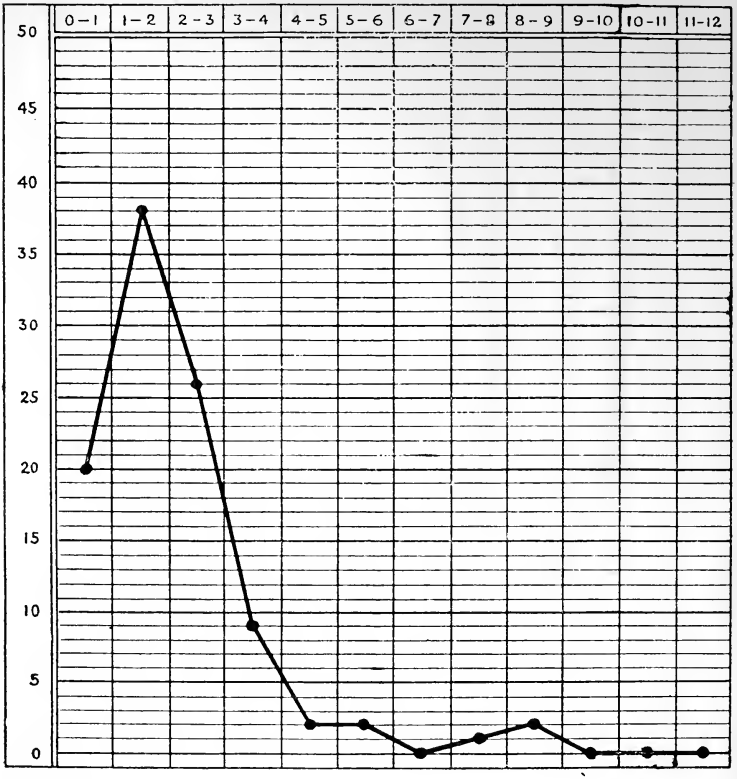

FIG. 48. Age-incidence of infantile paralysis. Chart shows age at onset in 100 cases.

suggested that the central nervous system is specially liable to infective disease at this period (see p. 707).

More important is the seasonal incidence which is shown in the chart below (Fig. 49). In 71 out of 100 sporadic cases in which the month of onset could be determined, the disease began between the beginning of June and the end of September; and in 50 out of the 100 cases the onset was either in August or September. This incidence during a particular season strongly suggests

an infective character; but undue stress must not be laid upon it, for it must be remembered that diseases which I suppose no one imagines to be infective have sometimes a very striking seasonal incidence—for example, spasmus nutans; and it is quite possible that light, temperature, and barometric pressure may influence the seasonal incidence of a disease apart from any infective agent.

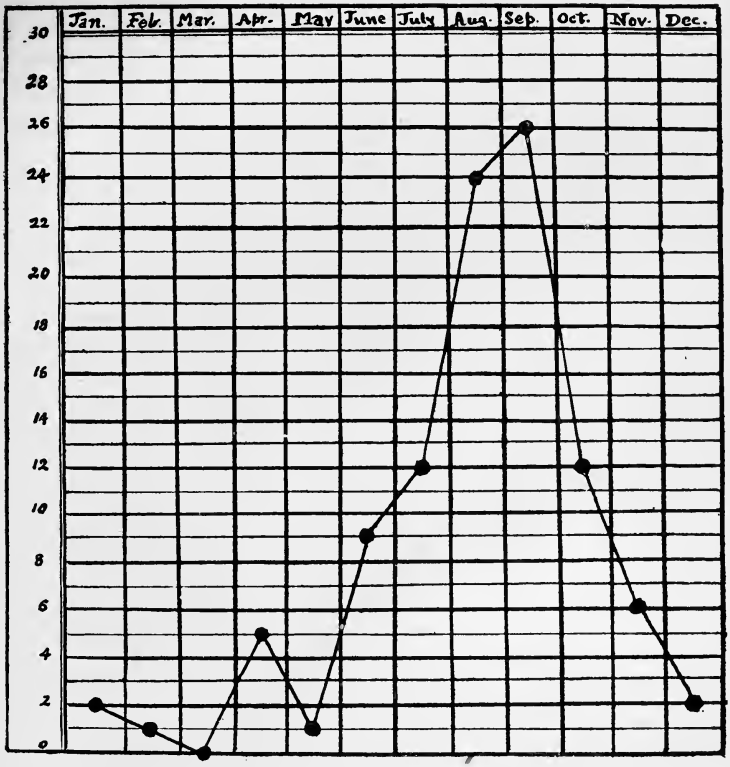

FIG. 49. Seasonal incidence of infantile paralysis. Chart shows month of onset in 100 cases.

A further point which harmonizes with the infective origin of infantile paralysis is the relation of this disease to specific fevers; amongst my notes of 100 cases there are 7 in which the paralysis supervened during the acute stage of a specific fever or during convalescence therefrom: (1) fourteen days after the appearance of measles rash; (2) four weeks after measles; (3) eight days after onset of scarlet fever; (4) while in hospital convalescing from scarlet fever; (5) during typhoid;

(6) during whooping-cough; (7) eight days after vaccination, paralysis of vaccinated arm. The tendency for one specific fever to follow another is often apparent in children, who within a few months will pass through two or three specific fevers, and is, I think, a reasonable ground for assuming that if any particular disease tends to follow specially upon specific fevers the disease in question is likely itself to be of specific infective nature. But there is another possible explanation; thrombosis may certainly result from typhoid fever, and it seems likely enough that after other acute illnesses degenerative changes in vessel walls and feeble circulation, perhaps with dilatation of the heart, may favour the occurrence of thrombosis apart from any specific infection peculiar to infantile paralysis; on this view the relation to specific fevers might be held to support the idea of a primary thrombosis as the cause of the spinal cord condition. It is noteworthy that infantile paralysis follows directly, not only the specific fevers, but also acute diarrhœa; in three of my cases acute diarrhœa was followed after twenty-four hours duration by infantile paralysis, and in another case paralysis was found after the child had been ill three weeks with diarrhœa. A primary vascular lesion would accord also well with the cases in which traumatism has seemed to be the determining factor. Parents are apt enough to attribute illnesses to blows and falls, the occurrence of which just before the illness may be a mere coincidence; but it is as easy to attach too little importance to them as too much, and even if a disease be undoubtedly of infective character it may still be true that a blow or fall may produce either at the part struck a *locus minoris resistentiæ*, or by its generally depressing effect a general vulnerability of tissues. For instance, a boy, aged five and a half years, went to school apparently well; whilst there he was hit a severe blow in the back by another boy; in the afternoon shortly after the blow he fell whenever he tried to walk: both legs were paralysed; the right recovered gradually, the left remained paralysed. In two other cases the onset of infantile paralysis was three days after a fall downstairs, but in both the onset was accompanied by fever and in one by vomiting, as is common in cases where there is no history of traumatism.

The strongest argument in favour of an infective origin, apart from experimental evidence, is the occurrence of epidemics of infantile paralysis—a *résumé* of thirty-five epidemics has recently been collected by Drs. Holt and Bartlett,[1] and Dr. F. E. Batten[2]

[1] *Amer. Journ. Med. Sci.*, May, 1908.

[2] *Proc. Roy. Soc. Med.* (Epidem. Sect.), June, 1911.

has collected information as to similar outbreaks in Great Britain —a fact which seems also strongly to support the view that the disease is due to some specific infection. Whilst, however, I draw attention to this almost conclusive proof of an infective origin, I would point out that in most of these epidemics there have been certain features very unlike those we are familiar with in the ordinary sporadic infantile paralysis. In the first place, the mortality has been widely different from that of the sporadic disease, which is very rarely fatal. In the epidemic recorded by Caverley at Rutland, Vermont, U.S.A., there were eighteen deaths out of 126 cases—i.e., nearly 15 per cent. Again, in an epidemic in France there were three deaths among 13 cases. Moreover, it is remarkable that in some of these epidemics some cases have shown symptoms so like cerebro-spinal meningitis that great doubt has arisen as to their nature, and in a recent epidemic in Australia one view regarded the epidemic as including two distinct diseases—acute anterior poliomyelitis and cerebro-spinal meningitis. In the Rutland epidemic horses, dogs, and fowls were affected, and in a horse the necropsy showed that there was definite meningitis. In some of the epidemics some of the cases have resembled rather a polyneuritis, and this also has been demonstrated at autopsy. Lastly, I would point out that complete recovery, which is so rare in ordinary sporadic infantile paralysis, has been quite a common occurrence in some of the epidemics. Caverley reported fifty complete recoveries in 126 cases; Altmann, in an Australian epidemic, reported four complete recoveries in 18 cases.

I mention these facts, not to throw any doubt upon the occurrence of epidemic anterior poliomyelitis, but only to suggest that until, by the more exact methods of experimental medicine the specificity of infantile paralysis in all varieties of its occurrence has been proved, the exact relationship of the recorded epidemics to the sporadic disease which we call infantile paralysis remains uncertain.

The brilliant work of Flexner and his colleagues at the Rockefeller Institute has proved that at least in the epidemic form of the disease there is a specific infection; and his results are so important that I cannot do better than summarize them here. An emulsion was made of the spinal cord from children who had died during the acute stage of epidemic anterior poliomyelitis; this was injected into the brains of monkeys through a trephine hole, and it was found that after a period of about 6–10 days paralysis of limbs appeared and autopsy showed changes in the spinal cord of the monkeys exactly similar to those found in children. (As the incubation period may prove to be of practical importance in

preventing the spread of this disease it should be mentioned here that Dr. Batten, from his study of epidemics, concludes that in human beings this period is shorter, not exceeding four days.)

The spinal cord from these monkeys was in turn emulsified and injected into other monkeys, who also developed paralysis after a few days and showed the characteristic changes of acute anterior poliomyelitis in the spinal cord. In passing the disease from monkey to monkey it was found that transmission was possible not only by intra-cerebral inoculation, but also by injecting the emulsion into the peritoneal cavity or into veins or into nerves. Further experiments showed that transmission was possible also by subcutaneous inoculation, and by bringing the virus into contact with the scarified mucosa of the nasopharynx. The exact nature of the virus remains, however, unknown. Flexner, after examination of the nervous tissues and of the cerebro-spinal fluid from children and from monkeys, was entirely unable to find any micro-organisms having the ordinary characters of bacteria or protozoal parasites, and even found that after admixture of the comminuted spinal cord with glycerine for seven days, a procedure which in the case of the virus of vaccinia destroys any admixture of bacteria without destroying the specific virus, the spinal cord still retained its power of producing acute anterior poliomyelitis when injected into monkeys. It seems, therefore, clear that the infecting agent is not an ordinary bacterium.

A filtrate of the cord, which was made into a fine emulsion and passed through a Berkefeld filter, so that a clear, bacteriologically sterile filtrate resulted, was still capable of transmitting the disease. It was thought, therefore, that the infecting agent of epidemic poliomyelitis belonged to the class of the minute viruses which, like those of rabies and vaccinia, are capable of passing through fine filters and which have not hitherto been discoverable with certainty by any microscopic examination.

More recently, however, by prolonged culture of pieces of cerebral cortex in human ascitic fluid, to which has been added a fragment of sterile fresh kidney from a rabbit, Noguchi has been able to cultivate, after about five days, certain very minute globoid bodies, in chains, pairs, and small masses, which produce in inoculated monkeys the characteristic lesions of poliomyelitis. Flexner and Noguchi [1] consider that this organism fulfils the requirements of a specific virus.

As to the method of conveyance of acute anterior poliomyelitis to human beings, no conclusive evidence is yet available, but

[1] *Journ. Experiment. Med.*, vol. xviii, 4, 1913, p. 461.

certain facts discovered by Flexner are at least suggestive and may prove to be of extreme practical importance. The virus, as already mentioned, can be inoculated in monkeys by simple application to the scarified mucous membrane; it has also been obtained from the nasal mucous membrane of monkeys after they had developed the disease from inoculation in other parts. Levaditi and Pastia have also shown its presence in the tonsils and pharyngeal mucosa. It seems, therefore, at least conceivable that one portal of entry in children is the nasopharynx, including the tonsils. Lastly, Flexner has shown that if the common house fly is allowed to feed upon the fresh spinal cord from monkeys which have been recently paralysed by infection with acute anterior poliomyelitis, and is then killed and emulsified, the bacterium-free filtrate from these flies is capable of transmitting the disease to monkeys.

An extremely important observation is that made by Osgood and Lucas, quoted by Flexner, that the virus can survive in the nasal mucosa for nearly six months, so that long after the acute stage of the disease has passed a monkey can still act as 'carrier' of infection. Flexner and Clark themselves have observed persistence of the virus in the nasal and pharyngeal mucosa four weeks after the onset of the paralysis. Clearly, if these facts hold good for human beings as well as for monkeys, they may have an extremely important bearing upon the prevention of this disease.

I have quoted these experiments at some length because, whatever future observations may bring forth with regard to the relation between epidemic anterior poliomyelitis and the ordinary sporadic instances of the disease, it is clear that the epidemics are due to a specific infection, and until the contrary be proved, it is, in my opinion, only right that we should assume that the sporadic disease also is infective, and, moreover, that it is possibly contagious, and we should take precautions accordingly.

Onset and symptoms. The clinical course of infantile paralysis gives but conflicting evidence on the problems of its pathology. Most striking, to my mind, are the remarkable differences in the mode of onset, which may be classified in three groups:

(1) An acute illness, more or less severe, lasting a few days, with pyrexia and drowsiness, and perhaps with vomiting, headache, constipation, and some pain in the back or limbs, is found to have left the child paralysed in one or more limbs: e.g. Alice S., aged eighteen months, on October 9 was restless and seemed vaguely ill; on October 10 she vomited three times and was feverish; October 11 she seemed less ill, but on October 12 she

was noticed to be unable to move her limbs or head, and as she improved she remained paralysed in the left arm and leg.

This is the commonest mode of onset and would fit in well with the idea of an infective disease.

(2) An insidious onset which perhaps cannot be dated at all; the first thing noticed is weakness, and this perhaps only when there is already considerable wasting: e. g. Charles H., aged two and a half years, came with flaccid paralysis of the left leg. The onset was entirely unnoticed, and it was only when he should have walked at sixteen months that the weakness of his left leg attracted attention. In some of these cases one is assured the weakness has come on gradually, but it seems likely that it is the wasting rather than the weakness which has gradually increased and has given rise to this statement.

(3) Sudden, perhaps instantaneous onset: e.g. Violet S., aged two years and four months, was running about at play; she fell down, and on being picked up was found to be paralysed in the right leg. I have seen another case with exactly similar history, where a little girl running across a room suddenly dropped down and was found to be paralysed in the legs. In both these cases the subsequent condition seen by me was that of typical infantile paralysis. Such cases suggest very strongly the occurrence of some vascular lesion, obstruction or hæmorrhage. In this group, perhaps, should be included the not uncommon cases of which the following is an instance: Willie S., aged three years, went to bed apparently perfectly well; in the morning he was found to be paralysed in the right leg, though otherwise showing no sign of illness; the paralysis remained permanent.

In connexion with the onset of the disease I would draw attention specially to the evidence of widespread disturbance which is noticeable in some cases at this stage. Not only is there fever with malaise, but in 9 out of 100 cases I have noted convulsions at the onset; a boy, aged four and a half years, whose illness had begun with a convulsion, followed by vomiting, headache, and drowsiness, fell into a semi-comatose condition, his face became paralysed on the left side, he passed his urine and fæces under him, and after a few days lost power in the left shoulder muscles and the right leg. All these symptoms disappeared within three weeks from the onset, except the paralysis of the right leg, which persisted with wasting of muscles and ' reaction of degeneration '.

In such a case it seems evident that there is something more than a mere local affection of the spinal cord, thrombotic or otherwise; there is a profound constitutional disturbance suggestive

of some acute infective disorder. The facial palsy in this case was of interest as showing that parts at least as high as the pons may be affected. My colleague, Dr. F. E. Batten, has published a case in which microscopic examination proved that the facial paralysis was the result of congestion which 'had destroyed the nucleus of the seventh nerve'; and if the disease may produce inflammation of the grey matter at this level there seems to be no reason why it should not produce it also in the grey matter of the cortex. On this view the polio-encephalitis which is supposed by some to be the usual primary lesion in 'infantile hemiplegia' may be of a nature identical with the poliomyelitis which is the usual lesion in 'infantile paralysis'.

Similarly, 'infantile paralysis affecting the brain' may be the explanation of the widely spread polio-encephalitis which probably underlies some of those cases in which, after a sudden onset of convulsions with loss of consciousness and pyrexia, a child is left idiotic with general spasticity. But until we have bacteriological evidence by which we may test the identity of such conditions it would be premature to state that these cortical lesions are etiologically identical with infantile paralysis.

It seems clear that for some reason the part of the spinal cord remote from the brain is affected most often and most severely. My own figures showed in 100 cases:

Left lower limb paralysed in	32
Right lower limb	23
Both lower limbs	16
Both lower and both upper limbs	6
Both lower and right upper limb	2
Both lower and left upper limb	1
Right lower and right upper limb	1
Right lower and left upper limb	2
Left lower and right upper limb	1
Left lower and left upper limb	1
Both upper limbs	2
Left upper limb	5
Right upper limb	8

It will be seen that the lower limbs, one or both, were the only part affected in 71 out of 100 cases; that the lower limbs were affected together with the upper in 14 cases, and that the upper limbs alone were affected only in 15 cases.

These figures refer to the permanent paralyses, but it must be remembered that during the few days or weeks following the onset of the disease there is often much more extensive paralysis, which clears up, leaving, it may be, only one limb or one group of muscles affected.

I have not referred in these figures to paralysis of trunk muscles,

which, though much less common than that of limbs, is sometimes a marked feature; twice, at least, I have seen the diaphragm paralysed, and cases have been recorded in which the muscles of the abdominal wall have been paralysed on one side or both, so that the abdomen was puffed out in an extraordinary manner when the child cried or coughed. The spinal muscles were affected in several of my cases, giving rise to more or less lateral curvature; the following case illustrated both the extensive recovery from initial paralysis and the persistence of trunk affection. Winifred P., at the age of eighteen months, became feverish and ailing one day in August; after three days it was found that she was weak in all her limbs, but especially in the right leg and left arm. After a few weeks the limbs had gradually recovered completely, but months afterwards there was severe lateral curvature left as a result of affection of spinal muscles.

The results of infantile paralysis are not limited to the loss of muscular power; in many cases there arise gradually secondary deformities which add in no small degree to the disability already present; such deformities, which include several varieties of talipes, claw-hand, &c., are dependent chiefly on unopposed action of the unparalysed muscles, but partly, for instance in talipes valgus, on stretching of ligaments owing to absence of muscular support.

It is sometimes stated that there is no affection of sensation in infantile paralysis, and this no doubt is true for the disease in its chronic stage, but at the onset I have often found marked tenderness in the affected limbs, so that the child cries whenever the limbs are touched; in some cases also there is pain in the affected limbs at this stage. Diminution of sensation is much rarer than tenderness; it has been observed during the first few days of the disease.

As in a case I have quoted, incontinence of urine and fæces, or retention of urine, sometimes occurs at the onset, but although these irregularities may indicate affection of the sphincters in some cases, they are probably more often due to the negligence in these matters which is common in children with any acute illness or disablement; I have never known any sphincter affection to persist more than a fortnight after the onset.

The tendon-jerks are rapidly lost in the affected limbs, but I would point out that this loss depends entirely upon the distribution of the paralysis; there may be extensive paralysis in the legs with no loss of knee-jerk, so long as part of the quadriceps extensor, particularly the vastus internus, is intact. Out of sixty-four cases in which the lower limb was affected I

have noted that the knee-jerk was present in the paralysed leg in no less than twenty-four cases. The plantar reflex has shown extensor response in some of my cases, signifying probably, not interruption of pyramidal tracts, but simply weakness of flexor muscles so that the unparalysed extensors predominate.

Diagnosis. When the limb is already wasted, cold and blue, with loss of tendon-jerks and with flaccid paralysis, the diagnosis is usually easy enough, but at the onset a diagnosis may be impossible for several days, and the febrile illness, with tenderness or pains in limbs and back, has been mistaken, naturally enough, for all sorts of febrile ailments ; acute rheumatism it was supposed to be in a little girl, who, with some fever, complained of tenderness about one shoulder and inability to move it; and this, perhaps, is one of the commonest errors. Influenza the illness was called in another case, while in the case mentioned above (p. 692) where facial paralysis was associated with stupor and fever before the paralysis of limbs appeared, a confident diagnosis of meningitis was made. Even when extensive paralysis of limbs is evident, it must be remembered that there are other conditions beside anterior poliomyelitis which occasionally cause such symptoms in children. I have seen acute myelitis paralyse all four limbs in a child of six years, but the occurrence of anæsthesia, with retention of urine, cystitis, and a tendency to bed-sores, all pointed to this diffuse inflammation of the cord. Neuritis also may hardly be distinguishable from poliomyelitis, unless the occurrence of prolonged tenderness and much pain in the limbs may be taken as indicating peripheral nerve-lesion rather than affection of spinal cord, but in some of the epidemics of supposed infantile paralysis neuritis was found to be associated with the cord-lesion, and it may be that the specific *materies morbi*, if such there be, of infantile paralysis can act upon nerves or spinal cord indifferently ; certainly there are cases in which pain and tenderness, associated with very extensive paralysis and a very slow recovery in almost all the affected parts after many weeks or months, strongly suggest a polyneuritis rather than a spinal cord lesion, but the distinction may be impossible.

Prognosis. There are few diseases which within so few hours or days may so completely mar a life by permanent and hopeless crippling as does infantile paralysis. And yet, disastrous though its results may be, it is a disease which, at any rate in its ordinary sporadic form, very rarely endangers life. When infantile paralysis kills it is almost always during the first week or two of the disease, and death is due to paralysis of respiratory muscles from affection of the upper part of the cord, particularly of

that part of the cervical cord which supplies the diaphragm. But it is possible that more deaths may be due to this disease than are commonly attributed to it ; for if it affects the cervical cord there seems to be no reason why it should not affect the medulla, and it may be that some of the sudden and unexplained deaths in children may be due to infantile paralysis affecting the medulla, and so damaging the respiratory and cardiac centres that death occurs before any paralysis of limbs has appeared.

Does complete recovery ever occur in infantile paralysis ? It is usual for some of the limbs or muscles originally paralysed to recover completely within a few days or weeks after the onset, and if the lesion in the cord may thus subside in one part without permanently damaging its functions there seems to be no *a priori* improbability that all the parts affected may sometimes recover. There is evidence, as already mentioned, that this has happened in some of the epidemic cases, but naturally it is difficult to prove any sporadic instance. A child was brought to the Children's Hospital with infantile paralysis of one limb ; it was stated that another child in the family had become paralysed similarly within ten days after the onset in this child, but had completely recovered.

The possibility of complete recovery is strongly suggested also by the not uncommon cases in which the weakness left some months after the disease is so slight that it might be overlooked were it not for the wasting and shortening of the limb, which is more evident than the weakness.

If complete recovery occurs it is certainly to be looked for only in the first few weeks after the onset ; later, the only question is as to the degree of improvement possible. And here I would give two pieces of advice : (1) Not to take too gloomy a view because the paralysis, say three months after the onset, is extensive in distribution and the muscles flabby and wasted. No doubt a complete loss of reaction to faradism and galvanism makes it improbable that the muscle will recover, and a ' reaction of degeneration ' is only less discouraging, but I would strongly advise the medical man not to base his prognosis merely on electrical reactions. The origin of nerve-supply of a muscle extends probably through two or three segments of the spinal cord, and it is always possible that, though the main sources may be irreparably damaged, some may be so little affected that some small portion of the muscle may retain its function and, by compensatory overgrowth, restore to some extent the lost power. Moreover, where some muscles have escaped the child gradually

learns to adapt its remaining powers to the altered mechanical conditions. The power of walking may thus improve steadily for years, not because there is any real recovery in affected muscles, but because the child learns to use to the best advantage those which remain, and the extra work thrown upon these muscles, or surviving portions of muscles, increases them in size and power. (2) Not to take too bright a view because the paralysis is very slight and limited in extent. It is a striking feature in some cases of infantile paralysis that the arrest of growth in the bones of the limb may be altogether out of proportion to the paralysis. A boy who, at the age of $3\frac{3}{4}$ years, had infantile paralysis in the left leg, had so little weakness at the age of ten years that all movements seemed to be good, but, nevertheless, he limped badly in walking, for the affected leg was 2 inches shorter than the other.

Treatment. It is clear that in some cases, at least, if not in all, infantile paralysis is an infective disease, and there is some evidence that, at any rate in its epidemic form, it may not only spread by direct contagion from person to person, but may also be conveyed indirectly by a healthy person who has recently been in contact with a child who has just developed the disease.

Experience, however, shows that in the sporadic cases such infection must be extremely rare, if it occurs at all, and, therefore, it hardly seems necessary to enforce any rigorous isolation. Inasmuch, however, as the possibility of contagion exists, it would seem at least wise to exclude children from the sick room of the child who has recently become affected with anterior poliomyelitis, so long as there is any fever, or, if practicable, for at least a fortnight from the onset of the disease.

In view of the ascertained infectiousness of the nasal secretion in monkeys after inoculation with this disease, it would also be a wise precaution to spray the nostrils with some antiseptic such as eucalyptus during the first two or three weeks after the onset.

Admittedly such precautions rest at present upon no very solid basis of clinical evidence, but they have the merit of simplicity and seem worth the little trouble they may give if perchance thereby the spread of so serious an infection may be prevented.

When acute anterior poliomyelitis has been recognized within a few days of its onset, urotropin is the drug which seems likely to have most value. It has been found by Flexner and Clark that in monkeys the administration of urotropin in large doses by mouth is speedily followed by its appearance in the cerebrospinal fluid, where it exercises an appreciable effect in lengthening the incubation period of the inoculated disease, and can even

entirely prevent paralysis. To obtain these effects, however, the urotropin was given before the animal was infected; it remains to be seen whether this drug can diminish or prevent the occurrence of paralysis when administered to children who have already become infected with the disease.

I know of no observations upon the amount of urotropin which it is safe to administer to a child. I have given as much as 10 grains every 2 hours until 24 doses had been given to a child 21 months old, but in a case where a dose of 7 grains was given every 4 hours to a boy of about 7 years for several days, Dr. P. Vosper tells me that hæmaturia occurred which ceased soon after the drug was stopped. I have seen other cases in which hæmaturia resulted from large doses of this drug. Certainly in so serious a disease as infantile paralysis it is advisable to give this drug as freely as is consistent with safety during the first few days after the earliest appearance of paralysis, or indeed before this, if there is any ground for suspecting that the illness may be the preliminary stage of acute poliomyelitis (for instance, in the case of a child who is suffering with acute fever of obscure origin in a neighbourhood where cases of infantile paralysis have recently occurred).

It is very doubtful whether any other treatment has any beneficial effect during the acute stage of the disease. It is conceivable that in the first few days after its onset good may be done by measures directed to reducing the hyperæmia of the spinal cord, particularly by application of ice-bags over the affected portion or by vigorous counter-irritation, e.g. with mustard plasters. I have used and seen used various measures of this kind, but as some of the initial paralysis almost always recovers whether any local treatment has been used or not, it is difficult to determine whether the amount of residual and permanent paralysis is influenced by such applications. I have also used ergot internally. A child of eighteen months will take 10 minims of the liquid extract, and a child of five will take 30 minims three or four times a day; aconite and eserine have also been used; but here also the treatment rests on theory, not on demonstrable results. Examination has shown that in the affected portion of the cord some inflammatory exudation persists for several weeks. It seems reasonable, therefore, to give iodides for a considerable period, perhaps two or three months, in the hope of promoting absorption and so relieving the pressure which no doubt hinders repair.

Apart from these measures of theoretical utility can we influence

the condition in the spinal cord ? One would fancy, to hear the extravagant claims which have been made sometimes for electrical treatment or massage of the paralysed limbs, that such treatment had some influence upon the seat of the disease ; but surely one might as well water the branches of a dead tree in the hope of reviving its root as stimulate the muscles with the idea of restoring the destroyed cells in the spinal cord. What we can do—and the only thing we can do by such treatment—is to promote the nutrition of those muscles or parts of muscles whose nerve-supply has escaped destruction, and so to raise their working capacity to its utmost. There is no doubt that just as the athlete by continual use of one muscle or set of muscles increases it both in size and power, so by frequently repeated stimulation, whether by electricity or by specially adapted exercises, and by flushing the muscle with blood, as is done by massage, we can increase both in size and power those muscles or parts of muscles whose representation in the cord has not been destroyed. With this purpose electricity, massage, and carefully planned exercises are all of value after the first two months of the disease, but I doubt whether electricity has so great a superiority over massage as to commend its use for those children who never seem to lose their dread of it, and to whom its daily application means daily misery. But no treatment is of much avail which is not capable of home application, for it requires to be used, not once or twice a week, but twice or thrice daily, and this is seldom practicable unless the parents or nurse can carry it out themselves. While I am convinced that in expert hands the application of galvanism is of considerable value, I am not at all sure that its random application by mother or nurse does much good, and as they can be taught to carry out efficient massage with excellent results, I am inclined to recommend massage as the more generally useful treatment. Not less valuable than either electricity or massage are carefully adapted exercises for cases in which a limb is only partially disabled ; but they must be planned with care and discretion, for it is obviously possible to do harm rather than good by encouraging the unbalanced action of muscles which are already tending to cause deformity by their unopposed power of contraction. Where any voluntary contraction of a weakened muscle is possible, or where by extra development of unparalysed muscles some compensatory advantage may be gained, the surest method of increasing power in these muscles is by exercising them frequently.

I have said nothing of the surgical measures which are occa-

sionally advisable in infantile paralysis,—tenotomy, fixation of joints by 'arthrodesis', and the recent methods of tendon-grafting; each has proved its value in particular cases, but their consideration does not fall within my province. On one matter, however, which is perhaps more surgical than medical, I venture to speak. Is it wise to encase a child's leg in a rigid apparatus which effectually prevents any exercise of the very muscles which being weak require to be used? I am well aware that there is a Charybdis as well as a Scylla to be avoided, and that some deformities—e.g. talipes valgus or genu valgum—may be aggravated in some cases by walking without artificial support; but none the less I think that sometimes a very simple support, perhaps even a flat-foot pad or some stiffening in the side of the boot, assisting but little the action of the weakened muscles, might give the limb a better chance of improvement than does the elaborate and cumbersome instrument which has been worn. Certainly if such apparatus must be used, it should be left off for some hours daily when the child is not walking, and in these intervals the muscles should be improved by massage and exercise. Lastly, I would point out that while in all cases there comes a time when no further improvement is possible, this may not be until two or three years or even more have elapsed since the onset; recovery from paralysis there is probably little or none after the first few months of the disease; but improvement, in the sense of increased ability to perform certain actions, there certainly is for much longer, and this, as I have said, depends upon extra development of the muscles or parts of muscles which have escaped.

CHAPTER XLVIII

THE CEREBRAL PALSIES OF CHILDHOOD

The relative frequency of the various forms of cerebral palsy in childhood is probably fairly shown by the following statistics of 100 consecutive cases under my own observation: 51 were infant hemiplegia, 26 were spastic paraplegia, 22 were spastic diplegia, 1 was spastic monoplegia.

As to sex my own figures show in all forms a predominance of boys: of the 100 cases, 61 were boys, 39 were girls; the incidence in the different forms was as follows:

	Boys.	*Girls.*
Infantile hemiplegia	32	19
Spastic paraplegia	14	12
Spastic diplegia	14	8
Spastic monoplegia	1	0

Infantile hemiplegia differs from the paraplegic and diplegic forms of cerebral palsy in being usually of post-natal origin; but there are many cases in which no date of onset can be assigned, the weakness and spasticity were only noticed when the infant became old enough for its defective movements to attract attention; in some of this latter group of cases there seems to be no more reason for assuming the condition to be of post-natal origin than there is for assuming under similar circumstances that a case of spastic diplegia or paraplegia is of congenital origin. This point was investigated in 49 of my cases of infantile hemiplegia, in 33 of these a more or less definite time of onset was assigned, in 16 the date of onset was entirely unknown, the weakness was discovered, so to speak, accidentally when the infant was some months old.

The age at onset in the thirty-three cases is shown in the following table:

INFANTILE HEMIPLEGIA.

Age at onset.	*Number of Cases.*
Congenital	2
Under 6 months	4
6 months to 1 year	4
1–1½ years	6
1½–2 years	6
2–3 years	6
3–4 years	4
6 years	1

Of the sixteen cases with no definite date of onset, one was known to have been paralysed before it was three months old, the remaining fifteen were first discovered to be abnormal about the end of the first year or a little later, when the child's backwardness, especially in walking, drew attention to the hemiplegia. In eight of these cases convulsions had occurred at some period of infancy, and may have marked the onset of the palsy, although it was not noticed at the time, but in the remaining eight cases there had never been a convulsion, and except in one case, in which some acute febrile illness had occurred at the age of five months, there seemed to be no ground whatever for assuming that the paralysis was of post-natal origin. Out of forty-nine cases, therefore, two were certainly congenital, and at least seven others were possibly, if not probably, congenital.

I lay stress on this point, for some writers have drawn a sharp distinction between infantile hemiplegia, on the one hand, as an acquired palsy, and spastic diplegia and paraplegia, on the other hand, as congenital or birth palsies. It is at least possible that a much larger proportion than is usually supposed of the cases of infantile hemiplegia may be congenital.

Relation to convulsions. The number of cases lacking evidence of post-natal onset would be even larger if the convulsions in the cases where the paralytic condition is of doubtful date were to be considered as the result of antecedent damage or disease in the brain, rather than as marking, if not causing the onset of the paralysis. Whilst, however, it seems unjustifiable to assume that the paralysis dates from the convulsions in an infant who is discovered several months after their occurrence to be hemiplegic, I think that the congenital origin of the palsy in such cases is certainly made more doubtful by the history of convulsions; for it is an undoubted fact that of the cases which become paralysed at some definite date a large proportion (22 of 32 cases in my series) begin with convulsions.

The exact relation of these initial convulsions to the hemiplegia is uncertain; it is generally held that both the convulsions and the paralysis are the result of some vascular cerebral lesion, inflammatory or otherwise, but I am by no means inclined to accept this view for all cases. Often there is a history that months before the onset of the palsy the child had suffered once or several times with convulsions, which differed in no way from ordinary infantile convulsions, and, like the majority of these, left no trace whatever of their occurrence; then the child had

an unusually severe attack of convulsions and was left paralysed on one side; for example:

Henry E. had had occasional infantile convulsions since the age of two months; at the age of fourteen months he had a severe bout of convulsions, lasting twenty-four hours, which left him with permanent hemiplegia.

With such a case may be compared the occasional occurrence of temporary hemiplegia after an infantile convulsion; sometimes the hemiplegia lasts a few hours, sometimes a few days; for instance:

Florence P., aged two years, had had three convulsions two months previously which appeared to be ordinary infantile convulsions and left no ill effects, but now, after another convulsion, the left arm and leg were found to be paralysed; the paralysis remained very marked for three hours after the convulsion and then passed off so completely that four days later no effect whatever of the convulsion was to be found.

Had the paralysis remained permanent in this case we should have called it infantile hemiplegia.

If it be asked how an infantile convulsion could cause hemiplegia, one might answer that in severe convulsions there is venous congestion, which may well originate some vascular lesion in the brain, just as a severe paroxysm of whooping-cough undoubtedly sometimes does, and it may well be that with rupture or thrombosis affecting only very minute vessels a transient paralysis occurs, whereas with more extensive hæmorrhage or thrombosis a permanent paralysis results.

It is allowed that a severe attack of convulsions may leave a child idiotic; and if the occurrence of such an 'eclamptic idiocy' be admitted—and my own observations lead me to think that although quite exceptional, imbecility as a result of infantile convulsions does occur—then it seems only reasonable to suppose that the storm which wrecks the mental function in one case may wreck the motor function in another.

Asphyxia. That intense venous congestion may damage the brain permanently is shown, I think, by the occurrence of idiocy after severe asphyxia at birth. I have seen so many cases in which this sequence occurred that I have no doubt in my own mind of the causal relation of asphyxia to idiocy: if this be so, one would expect that motor disturbance might result from the same cause. I have noted this point specially in nineteen cases of infantile hemiplegia; four of these gave a history of instrumental birth, but only one was said to have had severe asphyxia at birth; this child had convulsions for twenty-four hours after birth, but had no more up to the time when I first saw him at the age of

three years. It was noticed only when he began to crawl that there was weakness of the left side ; no date of onset was assigned for the hemiplegia, which seems most naturally referred to asphyxia or to the convulsions immediately following the asphyxia. The possibility of traumatism from the use of forceps cannot be excluded in such a case, but damage to the brain from forceps is probably much less frequent than damage from asphyxia.

It might be expected that with so general a condition as asphyxia any damage to the brain would be more likely to be diffuse than localized ; and that, therefore, such a cause would figure more largely in cases of generalized motor and mental impairment such as occurs in spastic diplegia, than in the cases of infantile hemiplegia. My own figures are too small to carry much weight, but so far as they go they show that this is so. In 17 cases of spastic diplegia and 13 cases of spastic paraplegia in which I have noted this point, 11 were known to have suffered with more or less asphyxia ; and in 6 of these the asphyxia was known to have been severe : the asphyxia occurred in 7 cases of spastic diplegia, in 4 cases of spastic paraplegia. On similar grounds it might be expected that convulsions would also be followed by generalized affection rather than hemiplegia ; sometimes this is so, but a localized lesion seems to be commoner as a result of convulsions.

Premature birth. The frequency of premature birth was particularly noticeable in my series of cases of spastic paraplegia, of whom at least 11 out of 25 were premature. A large proportion also were firstborn children—9 out of 21 cases of spastic paraplegia, and 5 out of 22 cases of spastic diplegia ; both these affections would seem to occur much more often in the earlier children than in those born later in a large family, therein contrasting with such a condition as Mongolian imbecility, in which the later or last children of large families are specially apt to suffer. The incidence on firstborn children seems to support the view that birth injuries, whether from traumatism or asphyxia, are a factor in cerebral palsies ; it is, however, noteworthy that several of the firstborn children were also premature, and no doubt on this account less likely to have met with injury from difficult labour ; but premature labour does not necessarily mean easy birth, two at least of the premature infants were delivered instrumentally, and two others were known to have suffered with asphyxia at birth.

Relation to syphilis. Amongst the various causes to which the cerebral palsies of childhood have been attributed, con-

siderable stress has been laid upon syphilis. The practical difficulties in determining whether the parents have had syphilis are so great that I am unable to offer any reliable statistics on this point, but so far as inherited syphilis in the child is concerned my own figures show that this plays but a small part. Of 51 cases of infantile hemiplegia none showed evidence of syphilis, but in 2 the relation of miscarriages to the birth of the infant raised a suspicion of syphilis in the mother. Of 26 cases of spastic paraplegia only 1 showed undoubted congenital syphilis, but another showed Parrot's nodes on the skull, which some would consider evidence of syphilis, and in 2 others a history of miscarriages suggested maternal syphilis. Of 22 cases of spastic diplegia 1 showed evidence of congenital syphilis. Ophthalmoscopic examination was made in many of these cases but not in all; in 2 of the cases mentioned patches of choroiditis were the only evidence of syphilis; and as fundus changes may be the only evidence it is possible that the proportion of cases showing syphilis may be slightly greater than appears from these figures, but certainly it is not high. Wassermann tests by various observers have confirmed these conclusions, but one series [1] showed a positive reaction in 13 out of 33 cases of spastic diplegia.

Polio-encephalitis. The mode of onset in some cases of cerebral palsy beginning after birth, with a short period of malaise and pyrexia preceding the appearance of convulsions and paralysis, has suggested the possibility of some infective cause, and the nature of the lesion—in many cases apparently an encephalitis either of recent or remote date—has suggested that the disease is identical in its pathology with infantile paralysis (acute anterior poliomyelitis). A further point which might be urged in favour of this identity is the age-incidence, which is very similar in these two conditions.

About 90 per cent. of cases of acute anterior poliomyelitis begin under three years of age, and 50 per cent. during the second year of life, while of cases of infantile hemiplegia beginning after birth, about 80 per cent. begin within the first three years of life, and about 40 per cent. in the second year. Moreover, in both conditions the paralysis is apt to occur during or shortly after one or other of the specific fevers. With regard to infantile hemiplegia it has been stated that this association is noticeable in about one-third of the cases (J. Taylor); but my own series of fifty-one cases showed it only in five—a proportion which corresponds remarkably closely with that observed in acute anterior

[1] *Glasgow Med. Journ.*, Dec. 1914.

poliomyelitis, in which, according to my own figures, about 10 per cent. of cases occur during or shortly after one of the specific fevers. The five cases were :

1. Fourteen days after measles convulsions occurred, with loss of consciousness for three days ; the child was found to be paralysed on the left side.

2. During diphtheritic paralysis the child became paralysed suddenly on the right side without convulsions.

3. Convulsions occurred seven weeks after measles ; left hemiplegia remained after the convulsions.

4. During a paroxysm of whooping-cough a convulsion occurred and the child remained paralysed on the right side.

5. During convalescence from scarlet fever, a boy, aged $3\frac{10}{12}$, who had suffered with convulsions occasionally since two years old, had a convulsion which left permanent hemiplegia.

It is well known that one specific fever is often followed after a short interval by another; it seems, in fact, as if one infection predisposed to another, so that sometimes a child will pass through two, three, or even four specific fevers within a few months ; there may even be a special affinity between particular infections, as is seen in the frequency with which whooping-cough follows after measles, or acute rheumatism after scarlet fever. The tendency of infantile hemiplegia and also of acute anterior poliomyelitis to occur during or just after specific fevers gives therefore some support to the view that both are due to some specific infection ; and as already pointed out, there are reasons for suspecting that both may be due to the same specific infection.

Plausible as such a theory may appear, there is much that might be urged against it. The facts with regard to the association of infantile hemiplegia with specific fevers are susceptible of other interpretations. Cardiac dilatation, enfeeblement of cardiac action, degenerative changes in vessel walls, are all recognized results of specific fevers, and might well favour the occurrence of embolism or thrombosis. In the case mentioned, where hemiplegia occurred during diphtheritic paralysis, I found subsequently localized softening in the internal capsule, a condition almost certainly the result of embolism or thrombosis ; and again in cases where hemiplegia occurs during whooping-cough the lesion is explained most naturally as the result of venous congestion, perhaps with hæmorrhage, as has been found in some cases at autopsy.

Further, for two of the points which form perhaps the strongest arguments in favour of the specific nature of acute anterior poliomyelitis, there is no parallel in infantile hemiplegia : these

two points are seasonal incidence and epidemic occurrence. About 60 per cent. of the cases of acute anterior poliomyelitis in this country begin during the months July, August, and September (see chart, p. 687); infantile hemiplegia, when its onset can be dated, appears to begin with almost equal frequency at any time of the year, as is shown by the following table of 20 consecutive cases in which the month of onset could be ascertained:

Jan.	*Feb.*	*Mar.*	*Apr.*	*May*	*June*	*July*	*Aug.*	*Sept.*	*Oct.*	*Nov.*	*Dec.*
0	4	2	2	2	1	4	0	1	2	1	1

Acute anterior poliomyelitis has several times occurred in epidemics; if infantile hemiplegia be due to the same specific infection it might be expected that it would at least occur with special frequency when such an epidemic was rife. I have never myself observed any special prevalence of infantile hemiplegia when acute anterior poliomyelitis has seemed to be specially prevalent in London. Dr. James Taylor has mentioned an instance in which one of two children in a family developed infantile paralysis at the same time that the other developed infantile hemiplegia, but such an occurrence is very rare.

It is particularly noteworthy that in the experimental production of acute anterior poliomyelitis in monkeys the intra-cerebral inoculation of the virus does not result in a spastic paralysis of cerebral type, but in a flaccid spinal paralysis with the lesions of acute anterior poliomyelitis.

It is evident, therefore, that even under conditions which would seem specially to favour the occurrence of a cerebral paralysis the virus of acute anterior poliomyelitis maintains its affinity for the spinal cord, and does not produce a spastic paralysis such as is seen clinically in infantile hemiplegia.

This limitation of the effects of the virus of acute anterior poliomyelitis to the cord under artificial conditions which would seem specially to favour the occurrence of cerebral paralysis, hardly gives support to the view that infantile hemiplegia is commonly caused by this particular virus.

Whilst, therefore, the cerebral lesion in some cases of infantile hemiplegia appears to be similar to the cord lesion of acute anterior poliomyelitis, the evidence of any specific infection in the cerebral cases seems to be much less conclusive than in the spinal cord affection, and until bacteriological investigation shall have furnished us with more exact knowledge of the pathogeny of these two conditions, there seems to be no sufficient ground for

regarding infantile hemiplegia as having any nearer kinship to infantile paralysis (acute anterior poliomyelitis) than an occasional resemblance in its morbid anatomy.

Symptoms. The characteristic features which are common to all the cerebral palsies are weakness and spasticity of the affected limbs, with retention and exaggeration of tendon-jerks. I need not describe these symptoms in detail, the names of the several varieties of palsy sufficiently indicate the distribution of the spastic paralysis ; but I should like to point out some features which are of importance in diagnosis. The weakness is usually very much less than the spasticity, and both are so slight in some cases that the cause of the child's slight talipes equinovarus, or of his easily stumbling in walking, may be overlooked altogether until careful examination shows that there is a little clumsiness also in the grasp of the hand, or that the knee-jerk is unduly brisk, and that the plantar reflex is of extensor type.

In all forms of cerebral palsy where the legs are affected there is a tendency specially to spastic adduction of the thighs and pointing of the toes, with inturning of the foot, so that the position assumed is like that of talipes equinovarus, but the spasm is intermittent, coming on chiefly when the child attempts to stand or walk, so that the position of the feet has not the fixity of a congenital talipes equinovarus, or of that due to contraction of muscles after infantile paralysis.

There are also cases in which the loss of co-ordination is the most marked feature, and the child is brought with what at first sight may be mistaken for a chorea. This applies especially to cases of infantile hemiplegia, in which within a few years after the onset there are sometimes involuntary movements of the affected limbs, but much more often in the upper limb than in the lower. In some there is an involuntary slow 'squirming' movement of the hand which is known as athetosis ; in others there is only an occasional fidgety movement very like that of chorea, and in others a coarse tremor or jactitation only upon attempting voluntary movement.

The face is curiously little affected by the cerebral palsies ; in most cases of infantile hemiplegia the difference between the two sides of the face is barely noticeable a few years after the onset, although it has been quite definite at first.

In these hemiplegic cases also the leg recovers to a much greater extent than the arm, which indeed tends rather to become more incapacitated through spastic clenching of the hand and flexion of the wrist as the child grows older : there is often only slight limping on the affected side when the hand of

that side is almost completely useless through spasticity or athetoid movements.

This tendency to recovery in the face and legs in infantile hemiplegia makes it easy in some cases to mistake the condition for a monoplegia affecting the arm, but as the figures given above show, a true monoplegia is so rare, that we should always examine the other limbs and the face very carefully before concluding that any case was primarily a monoplegia.

Almost more important than the paralytic symptoms are the mental disturbances which are so often associated with the cerebral palsies of childhood: the child with spastic diplegia is almost always more or less imbecile; there is also usually a considerable degree of mental weakness with spastic paraplegia.

With infantile hemiplegia the intellect may escape damage altogether, but many cases show a slight degree of impairment. If the onset was under eighteen months, they are backward in acquiring speech and walking, and in any case they are backward at school, their attention is easily distracted, they are 'facile', laugh too easily, and make friends with strangers too readily, and in general give the impression of being 'weak' mentally, without any such deficiency as could be called 'imbecility'.

The moral control also is apt to suffer: some of these cases of infantile hemiplegia, which otherwise might pass for mentally normal, are unnaturally passionate, spiteful, and seem to lack sense of right and wrong to a degree which can only be regarded as morbid. A large proportion of cases of infantile hemiplegia develop epileptiform attacks sooner or later, whether the palsy began with a convulsion or not; in the majority of cases I think this complication appears before puberty.

Diagnosis. The condition for which the cerebral palsies are most likely to be mistaken in infancy and early childhood is infantile paralysis. The chief points of distinction are the flaccidity of the limbs in the spinal cord affection contrasting with the more or less spasticity in the cerebral palsies, the affection of a group of muscles rather than the whole limb in infantile paralysis, the loss of tendon-jerks in the former, their exaggeration in the latter, in which also Babinsky's sign (extensor plantar reflex) is present if the legs are affected.

But there are cases in which mistake is easy, for the spasticity is sometimes very slight with cerebral palsy; and the action of unopposed muscles or the contraction of paralysed muscles in infantile paralysis may simulate the deformities of spastic paralysis, but, as I have mentioned, the contraction in the latter

comes on chiefly when the muscles are in use; it may be absent at other times, whereas that of infantile palsy is permanent.

The distribution sometimes helps in diagnosis; facial weakness associated with paralysis of the arm or leg of that side points to the cerebral affection; but the face has often recovered completely so that it gives no assistance, and the affection of both the arm and leg of one side is not very rare with the spinal cord paralysis.

In any case of hemiplegia, whether of sudden or of gradual onset, in a child, the possibility of some progressive form of cerebral disease is to be remembered, such as tumour, cerebral abscess, and even meningitis, for occasionally tuberculous meningitis begins with a sudden onset of hemiplegia.

On the other hand, as I have mentioned, a hemiplegia following convulsions may be transitory, and, after a few hours or days, disappear completely.

Prognosis. There is no likelihood of complete recovery; there are very slight cases of cerebral palsy in which the affection of the limbs is so slight that after a few years the condition is only just noticeable, but in the majority of cases spasticity, especially in the arms, tends rather to increase than to diminish.

Where there is paraplegia or diplegia, if the mental condition is not very bad, there is a good prospect that the child will learn to walk, probably at a very late age; even where at two or three years of age the adduction of the legs is so much that the feet cross one another directly the child attempts to stand, walking is usually acquired eventually.

In the hemiplegic cases the chief trouble is the tendency to spacticity in the forcarm and hand; the elbow is flexed and drawn in to the side of the chest, the wrist is strongly flexed and the finger clenched over the thumb, which is drawn into the palm of the hand.

If the child is seen in infancy or shortly afterwards, the likelihood of some mental affection is to be remembered, but in the hemiplegic cases one may fairly comfort the parents with the assurance that if there is any mental alteration it will probably be slight. The probability of epileptiform attacks occurring sooner or later must also make prognosis guarded.

Treatment. The most important item in treatment is the prevention of spastic contraction. Even when contraction has become so marked that the hand is almost useless, I have seen great improvement from long-continued perseverance in passive movements, but I think there is no doubt that some of the most troublesome deformities produced by the spasticity of

cerebral palsy might be prevented, or at any rate minimized, by the use of passive movements in the early stage of the affection.

There is no need for massage in these cases; indeed, it hardly seems logical to apply massage to muscles which are already doing more than is desired, and it is not likely that any massage applied to those muscles which are being overcome by the stronger ones in these cerebral palsies can so stimulate them as to counterbalance the tendency to spastic contraction. On the other hand, passive movements regularly and frequently applied so as to overcome by force the spastic deformity, are, as experience shows, of decided value; they are most applicable to the forearm and hand, where slow pressure directed to abducting the thumb, and extending the fingers, wrist, and elbow, will overcome the usual deformities

These passive movements, each done slowly several times in succession, should be repeated three or four times daily, and if there is already considerable contracture, which from neglect has become continuous, it may be necessary for a few weeks to wear a splint so arranged as to prevent the deformities in the intervals between the passive movements.

With such measures I have seen decided improvement, but it is only by patient perseverance that the deformity can be prevented from recurring; and this entails considerable patience on the part of the parents until the child is old enough to realize the importance of the movements, and to do them for himself with the sound hand, as some children can with infantile hemiplegia.

At the same time, it is important to make the child use the affected arm and hand as much as possible, and toys and games should be selected with this object in view. The lower limbs are to be treated on similar lines, and by simple dancing and drilling in the milder cases the gait may be improved.

At a later stage it is often necessary to have some special form of apparatus to prevent the tendency to the equinovarus position, and considerable improvement is to be obtained in some cases by operative measures.

Drugs have little place in the treatment of the cerebral palsies; when I have seen them in the early stage I have generally given potassium iodide, but I admit only with vague ideas of possible absorption of inflammatory exudation in any encephalitis which may be present.

The epileptiform attacks are usually more intractable than epilepsy apart from cerebral palsy; but bromides, phenazone, borax, or urethane may at least diminish their frequency.

CHAPTER XLIX

HYDROCEPHALUS

Hydrocephalus, in the ordinary clinical sense of the term, with which alone I am concerned here, is an enlargement of the head produced by excess of fluid within the ventricles of the brain.

It is necessary to make this clear at the outset, for the pathologist sometimes talks of the moderate dilatation of the ventricles in tuberculous meningitis as 'hydrocephalus', but clinically such a case is not one of hydrocephalus, and nothing but confusion results from terming such cases, as the older clinicians of half a century ago did, 'acute hydrocephalus'; nor, again, am I acquainted with any condition which justifies the term 'external hydrocephalus'. It is perfectly true that there are cases in which, as a result either of operation or of accident, there occurs a perforation of the thinned cortex over the dilated ventricles of the ordinary 'chronic internal hydrocephalus', and in this way a secondary collection of fluid collects in the subdural space; but this is only a secondary result of hydrocephalus, and much less is the term external hydrocephalus applicable to those cases in which shrinkage of the brain, such as occurs in some idiots with cortical sclerosis and sometimes in children who are extremely wasted, results in an outpouring of compensatory fluid over the surface of the brain.

Sir W. Gowers refers to a congenital condition in which excess of fluid occurs in the subdural space and produces progressive enlargement of the head, without abnormal distension of the ventricles. Such a condition must be extremely rare.

Hydrocephalus, in the sense which I have indicated, an enlargement of the head from fluid distension of the ventricles, is a disease arising exclusively in infancy and early childhood.

When the fontanelles are closed and the sutures firmly united, such a condition cannot arise unless, as is said to have happened in extremely rare cases, the cranial bones become greatly thinned along the sutures, so that these yield. The hydrocephalic enlargement of the head, which is occasionally seen in an older child or in an adult, almost invariably dates from infancy; such cases

are rare only because the child with hydrocephalus so rarely survives.

Out of 51 consecutive cases under my own observation, only one began after the end of the first year (at fifteen months), and 8 between the age of six months and one year. Thirty-two out of the 51 showed the hydrocephalus at birth or within the first three months of life.

Hydrocephalus is usually classified as (1) congenital, (2) acquired; but this distinction has very little practical importance. It is necessary, however, to point out that the term 'congenital', as applied to this affection, may be used to refer to the origin or to the date of its appearance, for although in some cases hydrocephalus is noticeable at the time of birth, in many where post-mortem examination has proved that the cause was antenatal the enlargement of the head has not been noticed until the child was 4–10 weeks old. In 28 at least out of the 51 consecutive cases mentioned above, the hydrocephalus was apparently of congenital origin, and in 15 of these 28 the large size of the head had been noticed at birth.

It would seem, therefore, that fully half the cases of hydrocephalus are of the congenital variety.

Morbid Anatomy. To understand properly the important questions which arise with regard to the prognosis and treatment of hydrocephalus, it is essential that one should be perfectly clear as to its anatomy, particularly as to the path of the cerebro-spinal fluid, which is shown in the accompanying diagram (Fig. 50). The cerebro-spinal fluid, in passing from the two lateral ventricles above to the theca or reservoir which surrounds the spinal cord below, has to pass by the lateral foramina of Munro into the median 3rd ventricle, thence by the narrow iter into the 4th ventricle, which lies between the medulla and cerebellum; the 4th ventricle is closed in below by an extremely thin transparent reflection of pia arachnoid, which passes from the inferior surface of the cerebellum to the medulla; in this reflection there are three openings for the passage of the cerebro-spinal fluid, a median one—the foramen of Majendie—and two smaller openings, one on each side—the foramina of Luschka—which adjoin the roots of the vagus nerves. Hydrocephalus, at least in a large majority of the cases, is purely a mechanical condition, and is due to obstruction at some part of this path.

In congenital cases the iter, as a result of abnormal development, may be partly or entirely absent, or its lumen may be

extremely narrowed; or there may be some deformity of the cerebellum, for instance, its lobes may be elongated posteriorly, and hang down into the foramen magnum of the skull, packing this so tightly that there is no passage for the cerebro-spinal fluid; or there may be cystic malformation of the cerebellum, causing it to bulge into and obstruct the ventricles; or, again, there may be adhesions between the cerebellum and medulla, perhaps as the result of some intra-uterine meningitis, so that there is no exit below from the 4th ventricle. All these conditions I have myself found in congenital cases of hydrocephalus.

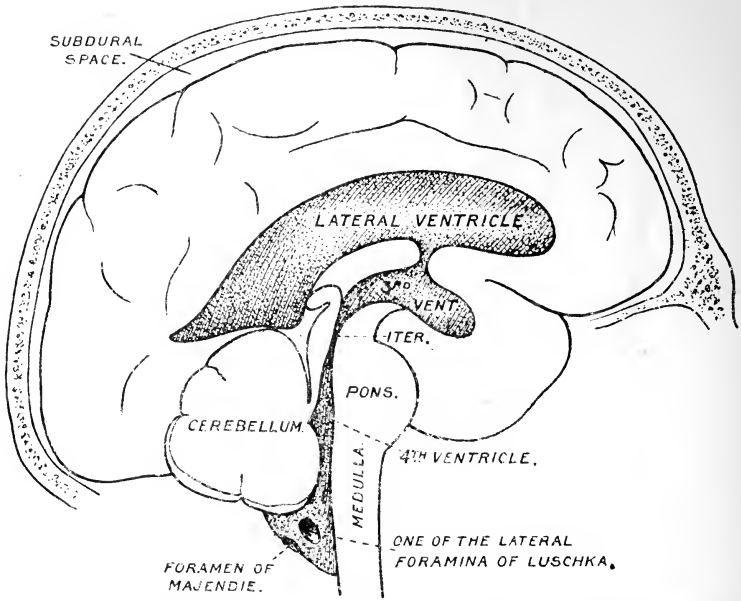

FIG. 50. Diagram to show path of the cerebro-spinal fluid from the ventricles of the brain to the theca of the spinal cord.

In acquired cases the commonest cause is posterior basic meningitis, whereby the pia arachnoid reflection from the cerebellum to the medulla becomes greatly thickened, and the foramen of Majendie and two foramina of Luschka are obliterated by inflammatory material. In no less than 11 out of 17 cases in which the condition was acquired the hydrocephalus was the sequel of posterior basic meningitis. Very much less commonly, tumour, generally a cerebellar tumour, by pressure upon or bulging into the 4th ventricle, acts as the obstructing cause.

The iter or the 3rd ventricle also occasionally becomes blocked by a tumour.

I have spoken so far as if the only cause of hydrocephalus were obstruction of the passage of the cerebro-spinal fluid. I am quite aware that there are cases in which no obstruction can be demonstrated. These are difficult to explain, but to my mind the failure to find obstruction is not convincing proof that they are not due to obstruction. The ordinary methods of removing the brain at autopsy destroy the relation of parts at the base of the skull, and it is quite conceivable that a slight abnormality in the packing, so to speak, might cause obstructive pressure. The exact arrangement of the posterior reflection of pia arachnoid is usually not seen at all when the brain is removed in the ordinary way, and any abnormality of the foramina here, or any slight adhesions between the cerebellum or medulla, would easily be overlooked.

On the ground that the choroid plexuses play an important part in the production of the cerebro-spinal fluid, it has been supposed that some abnormality of their function, whether from inflammatory change or not, may lead to hydrocephalus where there is no obstruction; we may, in fact, suppose that there is a loss of balance between secretion and absorption, the cerebro-spinal fluid is produced more rapidly than it can be carried off, and hydrocephalus results. This view rests merely on conjecture and is necessarily incapable of proof. Nor is there, I think, any good ground for supposing, as some have done, that lack of support for the brain may lead to dilatation of the ventricles. It is undoubtedly true that there are curious cases in which a congenital defect of ossification of the cranium, so that a large part of the skull remains membranous much longer than it should, is sometimes associated with hydrocephalus. But there are exactly similar cases in which the defect of ossification is not associated with hydrocephalus, so that lack of support in these, at any rate, has not led to dilatation of the ventricles.

These cases of congenital defect of ossification with hydrocephalus should rather be regarded as illustrations of the tendency to association of congenital deformities. This is very noticeable with hydrocephalus, and may be of value in deciding whether the hydrocephalus is of congenital origin. Amongst 36 congenital cases of which I have notes, 10 showed congenital abnormalities; there was spina bifida in 4, congenital heart disease in 2, supernumerary finger in 1, a Meckel's diverticulum in 1, talipes varus in 1, and Mongolian imbecility in 1.

A factor which seems to play some part in the causation both of congenital and acquired hydrocephalus is congenital syphilis. Amongst 51 consecutive cases I noted undoubted syphilis in three, probable syphilis in one, and the sister of another case had interstitial keratitis. In one of these syphilitic cases the hydrocephalus was not noticed until the age of nine months, in another at three months, in another at birth. How syphilis causes hydrocephalus is not very clear. Possibly the matting of the cerebellum on to the medulla and the thickening of the pia arachnoid reflection here which is found in some congenital cases is the result of syphilitic meningitis of intra-uterine origin; in some cases it may be that syphilis acts only indirectly, the disease in the mother predisposing to malformation in the offspring.

In one case I have known hydrocephalus follow after a fall upon the head at one year old, an occurrence which suggests some inflammatory adhesions resulting from the accident.

Symptoms. Most of the symptoms of hydrocephalus are sufficiently obvious at a glance, the enormous rounded head contrasting with the small and often delicate-featured face, the abnormally high forehead, the downward rotation of the eyes, so that the white sclerotic is seen above the cornea and the pupil is half covered by the lower eyelid, the presence of nystagmus in some cases and more often of divergent squint. Several of these characteristics are seen in the adjoining illustration (Fig. 51). Not only is the union of sutures and fontanelles delayed so that the anterior fontanelle at three or four years (in one of my cases at $7\frac{1}{4}$ years) is still open, and at six months measures perhaps six inches instead of one inch transversely, and extends forwards to the bridge of the nose and backwards to the posterior fontanelle, which I have known to be still open at two years, but there is also interference with the ossification of the bones, so that they remain thin and papery, and the intracranial pressure may even cause absorption of the bone so that gaps somewhat like patches of cranio-tabes appear. The bulging of the fontanelle, which is often very striking, forms a reliable index of the degree of intracranial tension, and in this way may be of practical importance, as I shall show when discussing treatment. The scalp is stretched by the distension of the cranium, so that the hair appears sparse and thin, and the distended veins show unduly through the thin skin. I have seen several cases in which the distension was so great, and the thinning of the cortex, skull, and scalp so considerable that the head was actually translucent—a bright light showed red through the enlarged head.

The shape of the head in hydrocephalus is generally stated to be round, in contrast to the square head of rickets ; but occasionally the hydrocephalic head, though enlarged in all directions, is dolichocephalic—that is, elongated in the antero-posterior direc-

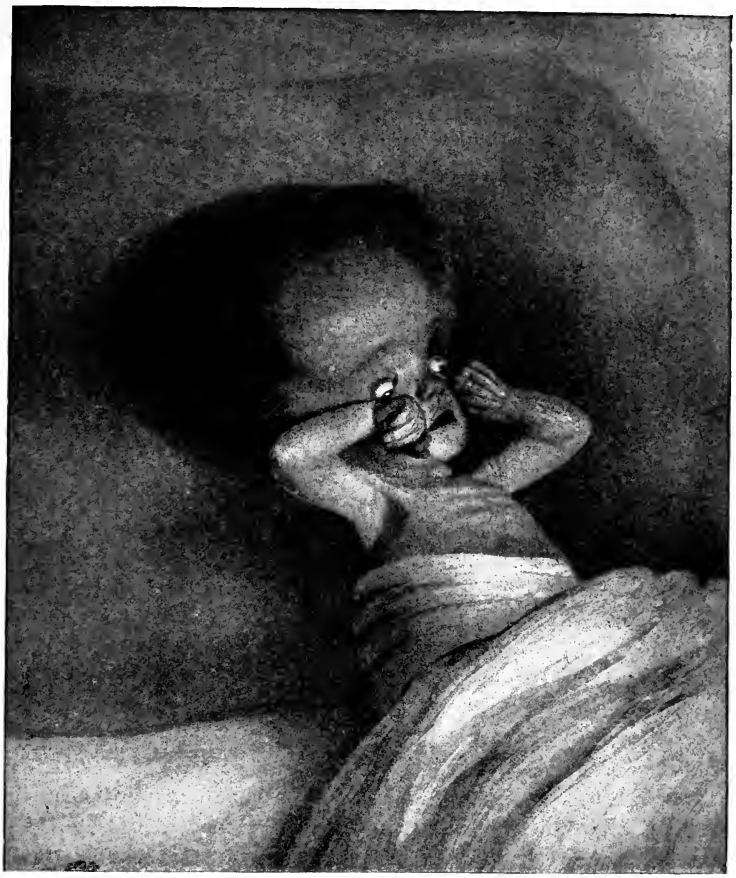

FIG. 51. Hydrocephalus, showing high forehead and downward rotation of the eyes, which in this case was so extreme that the pupils were completely covered by the lower eyelids, and the infant had learnt to pull down the eyelids in order to see. From a case under the care of Dr. F. E. Batten.

tion ; in some cases, also, the increase in the vertical dimension is the most striking change, as the photograph (Fig. 51) shows. No doubt these differences depend upon slight differences in the resistance offered to expansion in the various directions, and the effect of an artificial increase of resistance is sometimes shown

by the asymmetry of the head where the hydrocephalic infant has been habitually carried on one arm with its head pressed against the nurse's breast, or habitually lies on one side ; in such cases the side of the head which is not subjected to the pressure expands more than the other.

The enlargement of the head in hydrocephalus is often enormous, but to appreciate the degree it is obviously necessary to bear in mind the normal measurement for the age. The most generally useful is the maximum circumference, which has already been given (see p. 4), for the various ages in the healthy child: the normal average at birth is 13 inches, at one year 18 inches, and at seven years 20 inches. With hydrocephalus the circumference is often as much as 20 inches at three months, 22 inches at one year, and 24 inches at five years. The largest I have seen, a congenital hydrocephalus, measured 38½ inches at the age of nine weeks. I have seen other congenital cases in which the circumference was 26–28 inches at one year old. The greatest degrees of enlargement are seen as a rule in those cases where the hydrocephalus is of congenital origin. The rate of increase during the first six months is commonly about a quarter of an inch a week, very rarely it is as much as ¾–1 inch per week, but after this the rate of enlargement gradually diminishes, no doubt owing to the greater resistance offered by the hardening of the skull bones. The increase in the second year is often at the rate of about ½ inch in three months, and in the third year about an inch in twelve months. In favourable cases the rate slowly diminishes until perhaps at five or six years the fontanelle and sutures are closed, and the rate of increase is little more than the normal for the age.

The downward rotation of the eyes is so early a symptom of hydrocephalus that it is difficult to suppose that it can be due to any direct pressure, in some cases it is so marked that the pupil is almost covered by the lower lid ; in the case shown in the photograph the infant had learnt to draw down the lower lid in order to see. I have seen one case in which some proptosis of the eyes occurred, but this is very rare.

Some defect of vision is present in many cases ; blindness of central origin, lasting a year or more, is a not uncommon result of posterior basic meningitis, apart from hydrocephalus, so that it is found where hydrocephalus has resulted from this form of meningitis. More or less defect of vision, however, may arise from various changes in the fundus oculi.

Ophthalmoscopic examination is always worth making in

cases of hydrocephalus. Optic neuritis is almost never seen with congenital hydrocephalus (a curious fact throwing doubt upon the part played by pressure in the causation of optic neuritis); its presence almost always points to cerebral tumour as the cause of the hydrocephalus. Choroidal changes are found in some cases; sometimes symmetrical patches of choroidal atrophy suggestive of a congenital abnormality: sometimes a choroiditis or choroido-retinitis pointing to congenital syphilis.

The general symptoms produced by hydrocephalus are often remarkably slight. The child may be quite well and happy, and even while the head is steadily and rapidly enlarging there is usually no evidence of headache. The mental condition during infancy may seem unimpaired, but if the child survives it usually becomes evident that there is some mental alteration; he is too facile, too easily pleased, talks at six or seven years in a babyish way, like a child of two or three. When one considers how greatly the cortex must be compressed it is remarkable that the mental change is usually so slight. One might have thought that reduction of the cortex to a thickness of 5 millimetres or even less would involve complete loss, or at any rate extreme perversion, of function.

Sitting is delayed, as the child is unable to support the weight of the head. Walking is always acquired very late. One child could not stand at $7\frac{1}{4}$, another could just walk, with assistance, at four years, another at five years. The difficulty in walking is dependent partly, no doubt, on the enormous weight of the head; in one child I found the head weighed four-fifths of the total body weight; but there is also in many cases, both of congenital and of acquired hydrocephalus, some spasticity of the limbs, especially of the legs, with pointing of the toes, which interferes with walking and makes the gait awkward. In such cases the knee-jerks are usually increased, but ankleclonus is hardly ever present. Occasionally there is evidence of more localized damage: in one case with congenital hydrocephalus the right arm and leg were spastic, as in an infantile hemiplegia.

Where the affection is due to a tumour, paralysis or spasticity corresponding to the position of tumour may be present, and throw light upon the cause of the hydrocephalus. Convulsions occur almost as frequently with congenital hydrocephalus as with the acquired form, but they are less frequent than one might expect—they occur in about a third of the cases.

Diagnosis. There is very rarely any difficulty in the diagnosis of hydrocephalus; the only doubt which is likely to arise is in

the case of rachitic enlargement of the head. The rickety head, with its flat top, its square shape, its much slighter degree of enlargement, and the associated evidence of rickets elsewhere, sounds easy enough of recognition, and no doubt it is so as a rule, but there are cases of slight enlargement of the head in which, though the evidence of rickets is indubitable, the possibility of some degree of hydrocephalus also is by no means easy of exclusion; nevertheless I know of no connexion whatever between rickets and hydrocephalus (see p. 95), and unless the history showed evidence that the supposed hydrocephalus was either congenital or was due to one of the usual causes of acquired hydrocephalus, the presumption would certainly be against the existence of hydrocephalus. The very rare condition known as 'hypertrophy of the brain' in which enlargement of the head, with overgrowth of the neuroglial elements of the brain, is associated with mental defect, might be confused with hydrocephalus, but there is none of the rapid enlargement of the head which occurs with hydrocephalus.

It is not always easy to distinguish between a congenital and an acquired hydrocephalus; it must be remembered that posterior basic meningitis, the usual cause of acquired hydrocephalus, though it most commonly occurs between 6 months and 12 months of age, sometimes occurs at 10 or 12 weeks old: and cerebral tumour occasionally begins before the age of 3 months, as the following case may show:

Mabel A. was seen at 5 months old for hydrocephalus; the head then measured 19 inches in circumference, there was no optic neuritis, there was some vertical nystagmus, and the right hand was slightly spastic. The enlargement of the head was first noticed when the infant was 2 months old. Having regard to this onset at 8 weeks, I had no doubt whatever that the case was one of congenital hydrocephalus. It passed out of my care, and I heard subsequently that the child died at the age of 3 years. A post-mortem examination showed that the hydrocephalus was due to a new growth in the optic thalamus, bulging into and almost filling the 3rd ventricle.

Prognosis. Hydrocephalus is an extremely fatal condition: very few cases survive beyond the end of the third year. Death results from gradual exhaustion, with coma and convulsions, but complications are apt to occur, the tense skin over the enlarged head easily develops a bed-sore by pressure on the pillow, and the sores becoming septic may lead to pyæmia or to suppurative meningitis. A similar end may result from the giving way of the skin over a spina bifida associated with the hydrocephalus.

But it is very necessary, in view of the problems of treatment,

to emphasize the favourable possibilities of hydrocephalus. It is not inevitably a fatal condition; there are cases in which the abnormally rapid enlargement after continuing for two or three years gradually ceases spontaneously, and the child lives on for several years, weakened, it is true, in intellect, but perhaps only to an extremely slight degree, leading a happy and comfortable existence, and generally a bright lovable child, though perhaps somewhat quick tempered, and inclined to be passionate.

The following cases may illustrate such favourable exceptions :—

Alice R. had been hydrocephalic at least since six months of age, probably earlier; the condition was apparently of congenital origin. When she was first seen, at $2\frac{1}{4}$ years, the head measured $22\frac{3}{16}$ inches, there was divergent squint and she could only just stand with assistance; after this the head ceased to enlarge at an abnormally rapid rate: at 6 years the head was $23\frac{1}{4}$ inches and she talked brightly, and could walk, but with difficulty. At 9 years she goes to school, can write her name fairly well, and reads words of one syllable, and answers questions quite brightly and intelligently, but she is just a little babyish in her manner.

Boy, aged 19 years, looks as if he were 13 years. At 3 months old had posterior basic meningitis, followed by hydrocephalus, which steadily increased for about two years; then the abnormal enlargement ceased spontaneously. The boy's head is obviously too large, but the disproportion is not so striking as it would be with similar enlargement in a younger child. The head measures $24\frac{1}{2}$ inches. He is somewhat childish in his ways, but talks quite sensibly, and is intelligent enough to be entrusted with bringing his younger brother, aged 8 years, to hospital.

In which cases spontaneous cure is possible we cannot tell; it is quite clear, as these two cases may serve to show, that congenital as well as acquired hydrocephalus is capable of spontaneous arrest; but I know of no way in which we can determine in any particular case whether this is likely to occur; it is not even clear how the arrest is brought about when it does occur. It is conceivable that, if the obstruction is a partial one, when the intracranial pressure reaches a certain degree the secretion is so retarded that a balance is struck between secretion and absorption; it is also possible that with a sufficient pressure the partially obstructed passage may be forced, so to speak, and the fluid then leaks through in sufficient quantity to prevent further accumulation.

This latter view would be consistent with the clinical fact that a gradual diminution in the bulging and tension of the anterior fontanelle is one of the favourable signs which precede arrest of the hydrocephalic enlargement. Another phenomenon which is of good augury is diminution in the size of the fontanelle; this

almost always coincides with diminution in the rate of enlargement of the head, and if the fontanelle approaches actual closure, the hydrocephalus is almost certainly becoming stationary.

One point upon which some comfort may be given to the parents is the disproportion of the head to the body; this is so striking in infancy and early childhood that they are distressed at the thought of the child's appearance if he should survive; the disproportion, however, becomes less and less marked as the child grows older, when the body continues to develop after the hydrocephalus is arrested.

Treatment. It is clear that the scope for drugs is extremely limited in the treatment of hydrocephalus; in the congenital cases, if the trouble be not, as it may be, due to some malformation blocking the path of the cerebral fluid, the only cause which might conceivably be removable by drugs is syphilis, and even this is little likely to respond to the usual treatment, for in the rare cases where it is not merely an indirect factor, predisposing to malformation, it has probably produced fibrous adhesions which no mercury or iodide can remove.

In the acquired cases there is, it is true, the possibility, in the early stages of a hydrocephalus, that some inflammatory exudation blocking the foramina in the pia arachnoid between the cerebellum and medulla may still be capable of absorption. The process may then possibly be hastened by the administration of iodides and mercury, especially where the exudation, instead of being due, as it nearly always is, to the diplococcus intracellularis of posterior basic meningitis, is due to syphilis. In the rare event also of its being due to a tumour, drugs may have some value. But these possibilities are the exception: in most cases, as post-mortem evidence shows, there is obstruction by fibrous adhesions. The question, therefore, which has to be determined is whether operation should or should not be done. On that point certain general considerations should be borne in mind: hydrocephalus is not an inevitably fatal condition, spontaneous arrest occurs in about one in ten cases, according to my own figures; whatever method of operation is adopted, it is very seldom that any good results; often a fatal ending occurs within forty-eight hours, and even if no ill effects follow the operation, the intracranial tension is seldom relieved for more than a day or two at most.

On the other hand, it must be stated that operation has in very rare instances been followed by cessation of further hydrocephalic enlargement; and therefore where the increase in size

of the head is very rapid and persistent, and the intracranial tension is great, it may be justifiable to give the child the very small chance which operation affords.

The simplest operation is tapping, and there are two situations in which this has been done : (1) through the lateral angle of the anterior fontanelle ; (2) in the lumbar region of the spinal canal. The latter, lumbar puncture, is rarely if ever advisable, for although there are cases in which there is free communication between the spinal canal and the dilated ventricles of the brain, these are extremely rare ; almost always there is some obstruction at or above the level of the medulla, so that lumbar puncture can have no effect whatever upon the hydrocephalus.

Tapping through the lateral angle of the fontanelle is a much more reliable method of removing fluid from the lateral ventricles, and if the hydrocephalic brain were merely a closed tank with no inlet, no doubt this would be an efficient procedure ; but, as it is, experience shows that the brain fills up again within a day or two after the removal of the fluid. This might be expected, seeing that the fault in nearly all cases is not an abnormality of secretion which might conceivably be influenced by removing a little of the fluid, as seems to happen in some cases of pleural effusion, but is a purely mechanical accumulation of normally secreted fluid, owing to obstruction of outlet. Even as a temporary palliative this tapping of the ventricles very rarely does any good ; as a rule it affords no relief to any symptoms such as convulsions or rigidity, it may indeed produce them. If, however, it is decided to adopt this procedure, care must of course be taken that strict asepsis is observed, and only just enough general anæsthetic should be given to prevent the child from feeling the puncture. A fine trocar and cannula should be used, pushed in at right angles to the surface of the scalp at the lateral angle of the anterior fontanelle. The pulse must be watched carefully, for after a few ounces of the fluid have been removed (and it should be allowed only to trickle out slowly) the pulse is apt to become feeble, the child's colour may turn bad, and it would be dangerous to continue the drainage. I have had quantities varying from 4 to 7 ounces removed at a time, but with drainage to this amount have seen symptoms of collapse. Usually there is some leakage through the puncture afterwards, so that careful aseptic dressing is necessary.

Far more rational is the operation of subdural drainage devised by Sir Watson Cheyne and Dr. Sutherland. This attempts to establish permanent drainage by creating an artificial route by

which the cerebro-spinal fluid can pass out of the ventricles into the lymphatic system. The pacchionian bodies which lie along the longitudinal sinus serve as lymphatics, which pass on fluid from the subdural space into the blood-stream, and if the fluid could be brought into contact with these by allowing it to pass out of the ventricles into the subdural space, it should be absorbed. To accomplish this an opening in the skull is made and a rectangular tube is inserted so that one arm passes through the cortex and projects into the ventricle while the other arm lies on the surface of the brain in the subdural space. The tube is sutured at its angle to the adjoining dura mater to keep it in position, and the wound is then closed. Very fine metal tubes have been left thus as a permanent internal drain, but decalcified bone tubes have also been used.

In theory this operation leaves nothing to be desired, in practice it is only rarely successful. The procedure is less simple than it sounds: the tube may become blocked passing through the cortex or subsequently: the thinned cortex is very apt to tear, so that instead of trickling through the tube the fluid comes out with a big gush, and the child's life is endangered thereby. Even when the operation has been completed with apparent success, hyperpyrexia and death have followed in several cases within the next forty-eight hours. Twice at least I have seen arrest of hydrocephalus after subdural drainage, and I must have seen at least ten or a dozen in which it not only failed, but apparently hastened death; nevertheless, there can, I think, be no doubt that subdural drainage is an operation which offers some hope of permanent benefit to the child.

A much simpler procedure, based on the same physiological principles, namely, the establishing of a communication between the intraventricular cavities and the subdural space, so that the fluid may be drained off via the pacchionian bodies, has been tried with success in some cases. It consists in puncturing the corpus callosum. This is done by passing a trocar and cannula through the anterior fontanelle at a spot about half an inch from the middle line, so as to avoid the longitudinal sinus; when the cranial cavity has been reached the trocar is withdrawn, and a long blunt, probe-like rod is passed down through the cannula until it is felt to penetrate the tense corpus callosum. A few drops of cerebro-spinal fluid may then escape through the cannula, showing that an opening has been made into the ventricle: the rod and cannula are then withdrawn. A general anæsthetic is of course necessary. The pressure of the fluid in

the distended ventricles should suffice to keep the puncture in the corpus callosum patent whilst the fluid slowly passes into the subdural space, so that, in theory at any rate, a permanent drainage is established. My colleague, Dr. Theodore Thompson, adopted this procedure in a case under his care with excellent result.

Obviously operation is never to be recommended lightly for hydrocephalus: careful measurements of the maximum circumference of the head should be taken and recorded every week for several weeks, and only when the rate of increase and its persistence make all hope of spontaneous recovery remote should the question of operation be mooted. The exact time when this point is reached must vary in different cases, but it is certainly later in those of comparatively slow increase than in those where this is more rapid (vide p. 718). It must be remembered, however, that surgery has little chance of success when operation is delayed until the cortex is thinned to an extreme degree, and the brain is a mere thin-walled sac of fluid; and even if life were prolonged by the operation at that stage, the mental condition is likely to have been permanently damaged.

By whatever method the fluid is removed from the ventricles it is wise to apply a firm bandage around the head directly afterwards, to give some support to the flaccid brain.

A point to be remembered, also, is the great liability of the hydrocephalic child to pressure-sores on the scalp, where the head rests upon the pillow. This is due partly to the tight stretching of the scalp over the distended cranium and partly to the fact that the child in such cases is often unable to lift or even to turn the heavy hydrocephalic head.

To prevent this very serious complication—serious because of the risk of pyæmic infection from it—it is important to keep the scalp thoroughly dry, for which purpose a dusting powder of borated talc may be ordered; the position of the child's head should be frequently changed, and if there is the slightest tendency to a pressure-sore a ring pad of wool and silk should be arranged so as to keep the irritated part off the pillow.

CHAPTER L

ENURESIS AND FÆCAL INCONTINENCE

Amongst the functional disorders of childhood, one of the commonest is enuresis, and I suppose there is hardly any which causes more petty trouble and annoyance to parents and shame-faced misery to a sensitive child than this, at first sight, trivial complaint. Moreover, though so much has been written on enuresis and its treatment, it remains a bugbear to the medical man, who knows only too well that, though fortune may sometimes favour him, reputation is more often lost than won in the treatment of this disorder.

In many cases enuresis can hardly be said to have an onset; the child has never fully acquired that higher control of the reflex act of micturition which should come about the end of the first year of life.

Micturition in the newborn infant, as in the fœtus *in utero*, is purely a reflex function; the higher centres in the brain only gradually gain control of the function so that it becomes subject to the will. The contraction of the bladder and the relaxation of its sphincter are dependent upon centres in the lumbar cord, and for stimulation of this centre under ordinary conditions a certain moderate degree of distension of the bladder is requisite. It seems clear from the character of the incontinence in different cases that the defect in the nervous control is not the same in all cases. In some it would seem that the chief fault is an undue excitability of this lumbar centre, so that the accumulation of only a very small quantity of urine in the bladder suffices to stimulate the centre to such a degree that the cerebral control is insufficient to prevent micturition. These are the cases in which incontinence is associated with great frequency of micturition, or pollakiuria, as it has been called. In other cases the lumbar centre shows no undue excitability, but the control by the higher centres is unduly weak, so that although it may suffice as long as these are actively exerting their control, yet directly the higher centres become less watchful, so to speak, the spinal centre works uncontrolled. These are the common cases in which incontinence occurs only during sleep, or perhaps

also in waking hours if the child happens to be absorbed in his play or other occupation.

But the whole explanation of enuresis is no simple matter. I have sometimes wondered whether there may not be an afferent rather than an efferent or central failure of the nervous mechanism in some cases: it seems clear from the accounts of some of the older children that sometimes when enuresis occurs in waking hours there is no consciousness of the act until the child discovers it from the wetness of the underclothing. Then, again, what is the meaning of that curious and not very rare occurrence. that a child after voiding his urine voluntarily into a chamber, will sometimes within five minutes pass water involuntarily into his clothes; or, as I am sometimes told, a child asks for the chamber but is unable to pass any urine, and yet almost directly afterwards passes urine involuntarily? Such cases suggest a defect of co-ordination like that of the stutterer who becomes absolutely speechless in his struggle to get a word out, and finally blurts it out with uncontrollable force. Again, there are cases in which the trouble seems to be something more than a mere disorder of the control of micturition, for not only is there frequency of micturition and enuresis, but the amount of urine is above the normal, and the child complains of thirst—symptoms reminding us that the secretion of urine as well as its retention in the bladder is under nervous control, and that both may be disordered at the same time.

The controlling function of the higher centres, like other functions of the brain—for instance, speech—is acquired more slowly by some children than by others. Control during waking hours is usually acquired before control during sleep. Occasionally an infant can be trained to be clean in his habits while awake before he is a year old, but control during sleep is seldom acquired until the child is at least eighteen months old, and is usually imperfect and dependent upon very constant care on the part of the nurse until the age of two years. The persistence of bed-wetting, and even more of incontinence when the child is awake, after the age of $2\frac{1}{2}$ years, in spite of careful training, may be regarded as abnormal and described as enuresis.

Boys and girls seem to be equally subject to this disorder; of 200 cases under my own care 102 were boys, 98 were girls. Enuresis is a persistence of the infantile condition as frequently as it is an acquired disorder; in 142 cases in which this point was noted the incontinence dated from birth in 67

cases, and was said to have begun at some time after infancy in 75 cases.

Where enuresis is an acquired disorder it very rarely begins later than the end of the ninth year. In 60 per cent. of these cases the onset is between the ages of five and eight years—that is to say, about the time when the second dentition begins, a period in which children would appear to be particularly liable to certain functional nervous disorders, if we may judge from the similar age-incidence of stuttering and habit-spasm.

As control during sleep is acquired later than control during waking hours so it is the earlier lost ; and, as might be expected, it is very rare to find enuresis during waking hours if there is not also enuresis during sleep ; this occurred only in 4 per cent. of my cases, whereas the reverse condition of incontinence during sleep without enuresis when the child is awake is the commonest occurrence ; it was noted in 52 per cent. In 44 per cent. the enuresis was both when the child was asleep and when he was awake. I have referred to sleeping and waking rather than to night and day, for the diurnal enuresis in some cases is only when the child happens to fall asleep in the daytime ; and this is a point of some interest in regard to the etiology of nocturnal incontinence, for some writers have laid stress on the concentration and acidity of the urine during the fasting hours of sleep as accounting for the incontinence at night; whereas the fact that some children, when they fall asleep in the daytime, even for half an hour, suffer with enuresis, seems to show that, at any rate in these cases, it is the lessening of cerebral control by sleep rather than any special character of the urine which causes the incontinence to occur specially in the night.

When there is enuresis at night only there is sometimes exaggerated frequency of micturition by day, and this frequency of micturition, evidently indicating some undue excitability of the spinal centres, is not very uncommon in children apart from enuresis altogether. I wish to lay some stress on this pollakiuria occurring without incontinence ; for its near relationship to enuresis does not seem to be generally recognized. There are, of course, cases in which some irritating quality in the urine, such as the presence of uric acid granules, or oxalates, or bacterial contamination, are sufficient cause for frequent micturition, but the cases to which I refer are those in which the urine is perfectly normal, and it seems clear that the condition is purely a nervous one ; moreover, it is amenable to exactly the same

sedative treatment as ordinary enuresis, particularly to belladonna and its allies.

Diseases, like men, are often known by the company they keep, and the nervous character of enuresis is more than hinted at by many of its clinical associations. I suppose that quite the commonest association is the presence of threadworms in the child with enuresis. How often one hears that the child who has recently begun to wet the bed has been going thin lately, and has become ' so nervous ', and worms have been found in the stools ! But what is the connexion between threadworms and enuresis ? At first sight the traditional explanation that the local irritation in the rectum causes the involuntary emptying of the bladder seems likely enough ; but I think this is certainly not the whole explanation. One of the results of the presence of threadworms is a general increase of nervous instability, an increase which is often so striking that some have even suspected that it may be due to some special poison produced by these worms. It is evident from other associations that nervous instability, however produced, is a potent factor in the causation of enuresis, and it is probable, therefore, that the effect of worms is due, at least in part, to their general influence upon the nervous system.

Certainly, whether they have worms or not, the children who suffer with enuresis are commonly the nervous children. They are unduly excitable or excessively timid ; they suffer with ' nervous ' headaches easily ; often they have night terrors ; occasionally they walk in their sleep. Several times I have seen enuresis with habit-spasm of face or limbs. I have also seen it associated with that curious nervous disorder which is perhaps best known as ' nervous diarrhœa ', the condition in which each meal causes forthwith an urgent desire to defæcate, although the stools may not be loose at all. Incontinence of fæces also, a disorder which perhaps even more constantly than enuresis is linked with nervous instability, is occasionally associated with enuresis. Stuttering is another nervous disorder which sometimes keeps company with enuresis—a fact which lends support to the idea that a similar defect of co-ordination may occasionally underlie enuresis. I have also noted occasionally a history of convulsions at some former period in these children, an occurrence which probably foreshadows, in some cases, a tendency to nervous instability.

Another association which may be more than a coincidence is the occurrence of enuresis in rheumatic children : the rheu-

matic child is peculiarly apt to be a nervous child, and herein may be the connexion with enuresis ; certainly I have noted this association sufficiently frequently (11 times in the 200 cases mentioned) to make me always watchful for rheumatism in the child with enuresis. This point was impressed upon me some years ago by the case of a boy aged seven years, who was brought for enuresis. I found nothing else abnormal and treated the boy, not very successfully, for several months, when he made some complaint which made me re-examine his chest, and I found to my surprise that quite insidiously he had developed well-marked mitral disease, which was soon accompanied by other evidence of rheumatism.

Fright is occasionally the starting-point of enuresis, as it is apt to be of other functional nervous disorders in children : a boy aged 4½ years, bitten by another boy, seemed much frightened, became extremely timid, and complained of headaches, a week later he began wetting the bed, and a few weeks later developed a habit-spasm.

In a few cases I have been told that a boy with enuresis was given to masturbation, and it seems likely there may be some connexion between this and enuresis, if not by any local effect, certainly by the increased nervous irritability which so often accompanies masturbation, and partly by the impairment of general health which sometimes results therefrom.

The general health of a child often plays a large part in the production of enuresis. Control may be perfect so long as the child is in good health, but directly any upset occurs, perhaps a slight failure in digestion or a ' cold ', he begins to wet the bed ; it is by no means uncommon for the onset of enuresis to date from some acute illness ; I have noted its onset after most of the specific fevers.

I have emphasized the close relationship between enuresis and many of the common nervous disorders of children in order to bring into prominence the fact that incontinence of urine in children is often, perhaps usually, not merely a local disorder ; it is a manifestation of general nervous instability, either temporary or permanent.

Nevertheless local causes may play some part in its production. It is obvious that if the nervous mechanism is barely sufficient to control micturition when the stimulus is only of normal degree, it is likely to fail if the stimulus be increased, as it may be, by some irritating quality of the urine. It is customary to make much of excessive acidity of the urine as a cause of enuresis.

Now, without disputing the occasional part played by such acidity, I venture to think that it is a very exceptional cause, and that excessive acidity is often asserted where there is no evidence whatever that the acidity found is anything more than a transient occurrence, which could not account for the persistent enuresis. The passage of uric acid sand or of oxalates no doubt plays a part occasionally; but, again, I doubt whether much stress can be laid on these unless they are almost of daily occurrence. Sometimes quite unexpectedly pyuria will be found with enuresis, indicating some cystitis which may be the cause of the incontinence, but this order of sequence must not be assumed too hastily. I have little doubt that in girls the cystitis is sometimes not the cause, but the result, of the enuresis; it has seemed clear that enuresis was present long before the cystitis, and the underclothing constantly foul with decomposing urine has been a source of infection upwards through the urethra. Frequent change of underclothing has proved beneficial both in the treatment of the cystitis and in the prevention of its recurrence.

Dr. H. Thursfield [1] has pointed out that sometimes without any evidence of cystitis bacteria swarm in the urine in cases of enuresis—a point of practical importance if the treatment with urotropin, which he recommends, proves to be successful.

In the examination of the urine there are other possibilities to be borne in mind, rare though they may be. Twice I have had boys brought to me with enuresis when the urine showed abundant sugar and the disease proved to be diabetes mellitus. In another case, where the enuresis was associated with great thirst and polyuria, the condition proved to be diabetes insipidus (in a boy, aged three years, passing about 8 pints of urine per diem with a specific gravity of 1,000). The enuresis in the latter disease seems to show that failure of control of the bladder may be due rather to the rapid filling of the bladder than to any irritating quality in the urine; and in some children without any morbid excess of urine it seems evident that so long as the bladder fills but slowly the urine can be retained perfectly, whereas when the bladder fills more quickly it becomes intolerant and enuresis occurs. For this reason, many children are more or less free from enuresis in hot weather in spite of the concentration of the urine which occurs then, whereas in cold weather, when the skin is excreting less and the kidneys more, the enuresis returns or becomes worse.

Prognosis. The outlook in enuresis is usually good; it is

[1] *Clin. Journ.*, October 11, 1905.

a disorder which is very prone to relapse, but it rarely persists even as late as puberty, and extremely rarely into adult life, and even then I have known it to cease on marriage. Incontinence by day generally responds to treatment much more readily than nocturnal incontinence, and I have thought that the cases with much frequency of micturition, with or without enuresis, are usually favourable cases for treatment. But each case is a law unto itself; sometimes after resisting treatment for many weeks the enuresis will suddenly cease without apparent cause—perhaps after all treatment has been abandoned. In some cases a few doses of belladonna will effect a permanent cure, while in others, apparently no worse, no drug of any sort seems to have the least effect. We may well be cautious, therefore, in our praise of this or that mode of treatment, and must not too readily take credit for recoveries which may be nothing more than 'Fortune's testimonials'.

Treatment. It is undoubtedly true that Nature, left to herself, gradually works a cure in many cases of enuresis, and probably would do so in many more if no effort were made to cure them otherwise. But this seems to me no justification for shirking the risk of failure in treatment, and assuring the parents that there is no need to adopt any medical treatment, for the child 'will grow out of it'. Such a course means months or years of trouble and expense to parents, and of discomfort and misery to the child —often quite unnecessarily; and it seems only right, therefore, that a thorough course of treatment should be tried in every case before such a policy of despair is adopted.

Failure in treatment is too often due to half-hearted measures —a bottle or two of medicine has been given in a desultory way from time to time, but no settled course of treatment has been thoroughly tried. Often the fault lies rather with the parents than with the doctor; they will not give the opportunity for a course of treatment, which must necessarily extend over several weeks—perhaps over two or three months; and unless this is clearly understood at the outset the result is likely to be only disappointment for all concerned.

Before deciding on the mode of treatment the urine will have to be examined, and I must confess that my own experience has been that, although occasionally some abnormality may be found in the urine, in the vast majority of cases the urine is perfectly normal and gives no guidance whatever. Occasionally a concentrated acid urine may call for the use of potassium citrate; the presence of uric acid crystals may be met by cutting

down the carbo-hydrates as far as practicable—a measure which I believe to be more useful in such cases than cutting off meat and other nitrogenous food, as is usually done ; or again, the presence of pus or bacteria in the urine may indicate the need for urotropin, which can be given to a child of about five years in doses of 4 to 5 grains three times a day.

Far more often an important indication for treatment is found in the presence of threadworms, and I think it is often wise even when their presence is denied to give one dose of santonin gr. i–ij with calomel gr. j–i½ according to the age to make this point certain, for there is little chance of stopping enuresis whilst worms are present.

In the majority of cases, however, no exciting cause offers itself for treatment, and then nothing is more generally useful than the time-honoured belladonna. But to obtain the best results from belladonna it must be given on a definite plan, and for a period of several weeks. The random bottle of belladonna is responsible for much discredit in the treatment of enuresis. The parents should be given to understand at the outset that the drug is a potent one, and that the child must be seen at intervals of a few days by the doctor in order that the dosage may be carefully increased as the child is able to tolerate it. If this is clearly understood there is some hope of doing good with belladonna.

Many failures are due to the use of doses which are far too small. I have seen cases given up as incurable in which no more than 5 minims of the tincture three times a day had been tried. Children usually tolerate belladonna well, and at any period beyond infancy 5 minims of the tincture should only be considered as an initial dose. If the parents are intelligent and are forewarned of the possible toxic symptoms, it is usually safe to begin with 10 minims for a child over five years of age. The dose should be increased by 2½ minims every fifth or sixth day until either the enuresis is stopped or the limit of tolerance is reached.

If the enuresis is controlled, say by 17½ minims three times a day, it is well to ' make assurance doubly sure ' by pushing the dose slightly further by an additional 2½ minims, if tolerance allows. The dose should be maintained at this level, just beyond the minimum efficient dose, for a fortnight, and then reduced by decreases of 2½ minims once a week and so gradually discontinued.

A word of caution with regard to belladonna : children, like

adults, have their idiosyncrasies to drugs, and while some will tolerate 30 minims of the tincture of belladonna three times a day, I have occasionally met with marked intolerance even of so small a dose as 5 minims. Another caution which applies especially to such a drug as this : see that the dose is measured with a medicine glass, not with a spoon ; the domestic teaspoon will often hold 2 drachms, sometimes as much as 3 drachms. Nor is even a medicine glass necessarily reliable if it be one of the cheap measures which are often used. I found in one case that a drachm dose measured by a cheap medicine glass was actually 2 drachms, and smaller errors were found to be quite common.

There are many cases in which, after the belladonna has been pushed up to 17½ or 20 minims with decided diminution of the enuresis, the disorder is still not completely controlled, and yet any further increase of the belladonna produces toxic symptoms which are sufficient at any rate to alarm the parents. Under these circumstances a useful procedure is to keep the belladonna at the topmost dose which is well tolerated, and to combine with it tincture of lycopodium, 12½ minims at first, with increases of 2½ minims as may be required. A child will usually take 20 minims of this drug without ill effect, sometimes more, and certainly in some cases with most gratifying result.

The liquor atropinæ has been recommended by some as more effectual than the tincture of belladonna, but it must be remembered that in respect of alkaloids 1 minim of the liquor atropinæ is equivalent to 28 minims of the tincture of belladonna ; and I suspect that the supposed greater efficacy of the atropine solution is due to the fact that a stronger dose is used rather than to any intrinsic difference between the two drugs. It is, however, possible that some difference in the action of these two drugs may exist, if the tincture of belladonna contains, as is said, hyoscyamine and little or no atropine. I have used the liquor atropinæ and seen good results from it, using it in increasing doses, and commencing with ½ minim ; but toxic results are apt to occur when 1 minim is reached ; and on the whole it has seemed to me less convenient for manipulation of the dosage than the belladonna tincture, without any compensatory advantage. With either drug the occurrence of dryness of the throat, which children sometimes describe as ' sore throat ', or much flushing, or complaint of dimness of vision, and even more, any indication of delirium, make it advisable to reduce the dose just sufficiently to avoid these symptoms.

As an addition to the belladonna and lycopodium, whether these are used together or separately, nux vomica is undoubtedly helpful; some, indeed, use nux vomica alone and find it effectual. The tincture of nux vomica should be used in large doses, for instance, 6 minims for a child aged five years. Occasionally potassium bromide or phenazone will succeed where belladonna has failed; I have tried in vain to find any feature in the case which might indicate when these are likely to be useful; one might have thought that a high degree of 'nervousness' would be an indication, but this apparently forms no reliable gauge, and success may be obtained with them where least expected. Another drug, which will sometimes succeed where belladonna fails, is the fluid extract of rhus aromatica. The dosage is much the same as that of the tincture of belladonna. Ten minims three times a day may be given to a child aged five years, and the dose can be increased gradually up to 20 or 25 minims, but I have known it to produce sickness when a 15-minim dose was reached. Like belladonna this drug will sometimes stop enuresis almost at once, in other cases it fails completely. In a boy aged seven years, to whom I had given tincture of belladonna in doses gradually increased up to 20 minims three times a day without success, the fluid extract of rhus aromatica, 12 minims three times a day, stopped the enuresis at once. In a girl aged seven years, who had had enuresis three weeks, the enuresis ceased permanently after a week's treatment with fluid extract of rhus aromatica, 20 minims three times a day.

Excellent results also have followed the use of ergot in some cases which I had found most intractable with other drugs. To a child aged five years, 20 minims of the liquid extract can be given three times a day, and for an older child 30 minims is a suitable dose. I usually combine nux vomica with it.

At one time I used the liquid extract of salix nigra extensively, having found it useful in cases of masturbation, and arguing that it might, therefore, have some sedative effect on the spinal centres which would be of value in enuresis; the results, however, did not justify my hopes.

Turning now from the drug treatment to other measures, I shall utter a word of protest against certain procedures which have been advocated in recent years. We are told that good results have followed the application of silver nitrate to the urethra or to the neck of the bladder; that a course of massage of the sphincter of the bladder by a finger in the rectum has also done good; that injection of various fluids into the sacral

portion of the spinal canal, or even into the tissues outside the canal, has cured this disorder.

I do not doubt the sequence, but I do doubt very gravely the desirability of any such measures, some of which not only savour of quackery, but involve a totally unnecessary infliction of distress and pain. It is clear that in enuresis, as in other functional nervous disorders, there is a large psychical element, and as these disorders may sometimes be induced, so also they may sometimes be stopped, by any profound psychical impression ; hence enuresis will sometimes cease after any surgical operation, just as it will cease sometimes after smart corporal punishment. The latter mode of treatment is generally condemned, and rightly so, for even if it were often successful—it is as likely to do harm as good—it would still be liable to serious abuse, and, as every one knows, has led to gross cruelty. The manipulative and surgical procedures to which I have alluded are certainly quite unnecessary in the vast majority of cases, and if the disorder be so obstinate that it is advisable to try the effect of psychical impression, a much less objectionable method is the application of the galvanic current, one pole over the sacrum and one over the pubes, a mode of treatment which is not only free from danger, but is also said to be effectual in some cases.

In this connexion I must mention two operations which are sometimes recommended as if they were almost specific in the treatment of enuresis, namely, removal of adenoids and circumcision. It seems likely enough that the child whose health is being impaired by chronic starvation of air, through naso-pharyngeal obstruction, may have not only his general health, but also his nervous stability, and therewith his control of micturition, improved by removal of adenoids. But even this I cannot affirm from my own experience, and I suspect that when cessation of enuresis follows removal of adenoids, it may be due simply to psychical impression, and that any other operation would have done equally well. I have known most intractable enuresis stop the very night after removal of adenoids, but within a few weeks it was as bad as ever again—surely an instance of profound psychical impression. Again and again I have known adenoids removed without the least effect on enuresis. I have sometimes, as a matter of interest, treated enuresis by drugs with success before adenoids were removed, in order to demonstrate the curability of the disorder apart from any operation on adenoids. Circumcision similarly has usually no

beneficial effect whatever on enuresis. I have even known enuresis apparently started by circumcision, and I have known it to become worse after circumcision; but I doubt not that the boy who is cured by the pain or nervous shock of corporal punishment might be cured equally well—or rather, equally badly—by the psychical impression produced by circumcision.

Lastly, I must refer to the general management of these cases. It is, I think, seldom necessary to restrict greatly the amount of fluid which the child may take in the forenoon, but during the latter half of the day it should be limited, especially at the last meal, and after this none should be allowed. Tea and coffee are to be forbidden altogether, partly on account of their diuretic effect and partly because of their exciting effect upon the nervous system. In most cases I do not think there is much to be gained by any rigorous dieting, but where there is—as is so exceedingly common—difficulty in digesting carbohydrates, or a condition of intestinal catarrh, such as Dr. Eustace Smith has called 'mucous disease', I am satisfied that the reduction of starch and sugar, and especially the exclusion of potato and raw fruit, are decidedly helpful. I have known enuresis to stop at once when green vegetables and fruit were stopped altogether and potato reduced to an extremely small amount; possibly the presence of triple phosphates in the urine, where the reaction is neutral or alkaline, from fixed alkali not from ammoniacal decomposition, may be an indication for this line of treatment.

The effect of school on these children is worth considering. Schooling, if it entails worry and mental strain, seems to perpetuate the enuresis in some cases. Boarding-school has seemed to me most unsuitable for them on every ground: if the child's weakness is cause of ridicule at a school, whether from teachers or school-fellows, the sooner the child is removed from that school the better. In hospital practice one hears too often that a child has asked the teacher for permission to leave the room and has been refused, and consequently has wetted his clothes. In such a case it is not the child but the teacher who ought to be ashamed of himself, and parents should be advised to take steps to prevent ill-treatment of this kind.

Incontinence of Fæces

Incontinence of fæces, that is, apart from organic nervous disease and mental deficiency, is much less common than incontinence of urine, but is near akin to it, and sometimes associated with it. So far as my own experience goes, boys would seem to be much more liable to it than girls; out of 20 consecutive cases 16 were boys, only 4 were girls. Like enuresis it may be a persistence of the infantile lack of voluntary control, or may be an acquired disorder; in 13 out of 19 cases, in which I ascertained this point, the incontinence was acquired, and in 9 of these the onset was between the ages of six and nine years. It was associated with enuresis in 13 out of the 20 cases; but even when this is so the fæcal incontinence is usually by day only, whereas the enuresis, if it be not both in day and night, is in the night only.

In the etiology of fæcal incontinence nervous instability plays at least as large a part as in enuresis; almost all the children with this disorder are highly 'nervous' children, and often show other manifestations of their nervous temperament, such as night terrors, insomnia, stuttering, habit-spasm, and I have seen it, like enuresis, associated with rheumatism.

But, as in urinary incontinence, there is often a local exciting cause in some abnormal condition of the intestinal contents; the stools, without being actually diarrhœal, are abnormally offensive, or slimy, or perhaps fermenting; the bowels, in some cases, are open two or three times a day, and most often are inclined to be loose, but there may be no looseness whatever. The bowels in some of my cases were only open once a day, and sometimes the stools were of constipated character.

I have mentioned the frequency of micturition which sometimes accompanies, and sometimes occurs altogether apart from enuresis. It has seemed to me that there is a very similar condition related to fæcal continence, namely, the so-called 'nervous diarrhœa' or 'lienteric diarrhœa'—misleading names, for the stools are not necessarily loose at all; the only characteristic feature is urgency of defæcation directly a meal is taken; the presence of food in the intestine seems to excite an expulsive effort at once, just as the presence of a very small quantity of urine in the bladder excites micturition in the cases of so-called 'pollakiuria'.

This nervous disorder of the bowels, though usually found apart from incontinence of any sort, is associated in some cases

with fæcal, and in others with urinary, incontinence ; moreover, its affinity for both these disorders is suggested by its frequent association with those manifestations of nervous temperament which I have mentioned as frequent with urinary and fæcal incontinence. It is readily amenable also to the same treatment as is required for incontinence of fæces.

This incontinence is much more easily checked than is enuresis ; indeed, in the majority of cases, even when it has been present for weeks, a few days' treatment will set it right. There is, however, a tendency to relapses, which may occur more than once after several months of perfect control.

Treatment. The most effectual treatment, in my experience, is the administration of Dover's Powder, in doses of 1½ to 3 gr. three times a day, according to the age of the child, and a mixture of belladonna with potassium bromide, to which arsenic and nux vomica may usefully be added in some cases. At the same time all food likely to irritate the bowel, especially fruit, fresh or dried, must be prohibited ; and all drink should be given either cold or only just warm. The injection of drugs into the tissues about the rectum, as has been done, seems to me to be an entirely unnecessary infliction of pain.

CHAPTER LI

DISORDERS OF SPEECH

For the better understanding of the disorders of speech which are met with in childhood I shall mention first some facts in connexion with the development of speech.

The first indication of speech is the lalling which a normal infant begins at about four months old. That this is a real attempt at imitation of speech is, I think, shown by the fact that the easiest way to induce an exhibition of this baby-prattle is to talk to the infant. The sounds which are made in this earliest infant-speech are labial sounds : lul-lul-lul or bub-bub-bub or mum-mum-mum : while at a later period other sounds are formed, such as goo-goo, de-de, da-da at eight or nine months old.

After a few weeks there is added to this simple repetition of a syllable some inflection of voice, which marks a further stage in the approach to actual words. At the age of six or seven months the child begins to associate persons with names ; I mean that it understands which is Mamma and which is Dadda when these words are used : and I have been assured confidently by a mother that her infant actually said ' Mamma ' between the ages of five and six months ; but if so this is certainly quite exceptional. More often at the age of nine or ten months the child is still in the stage of lalling, but as I have said is forming guttural and dental sounds as well as the labials.

Between ten months and twelve months many infants begin to say such simple words as ' Mamma ', ' Dadda ', with evident understanding of their application, and by fourteen months an infant should be saying many simple words, such as ' up ' and ' go ', easily. About the age of eighteen months an infant should form simple sentences, such as ' Baby go tata '.

The normal development of speech depends upon several factors. There must be not only the speech centre presiding over the motor mechanism of speech, but also the auditory centre with all that appertains to the perception of sound, the perfect hearing and interpreting of which constitutes the basis of speech ; the visual-perceptive centre must play its part :

moreover, there must be adequate connexions between these centres, and above and over all, utilizing and co-ordinating them must be that function which we recognize as intellect.

Most of the disorders of speech can readily be referred to failure in one or other of these contributing functions : failure which, nevertheless, is seldom an insuperable bar to the acquisition of speech, for Nature has so carefully safeguarded the all-important means of communication which distinguishes Homo Sapiens from the lower animals, that where hearing is absent, speech can still be taught by employing visual and tactile sensation to educate the mechanism of speech, and even where sight and hearing are both absent it is still possible by tactile sensation alone to cultivate the power of speech, as was proved by the famous case of Laura Bridgman, the blind and deaf child.

In the few remarks which I shall make here upon the disorders of speech I shall refer first to certain anomalies in its development, and then pass on to mention some of the acquired disorders of speech in children.

Absence of speech. Children are often brought to the medical man because at the age of two years or more they have not begun to talk. What are the possibilities which must be considered ?

1. **Simple delay of development.** Let it be remembered that the time of first talking, like the time of eruption of teeth or of learning to walk, has normally a considerable range of variation, some infants will say words fairly distinctly at ten months, others equally intelligent and healthy will say nothing until the age of eighteen months, or even several months later. In some cases this appears to be due to what one might term physiological laziness, or perhaps to the absence of what the Germans expressively term the 'Sprechlust', the instinctive desire to talk.

The child shows a quick understanding perhaps of what he hears, but when he wants to draw attention to an object or to ask for anything, instead of attempting to name it he makes an inarticulate grunting noise and points to the object, and perhaps at the same time pulls his nurse or mother towards it, or describes what he wants by a kind of dumb show. Such children, even when they have begun to say a few words, will often continue for many months to make use of the inarticulate grunts and pointing or dumb show, whenever they can thus avoid the use of words, and sometimes if they are shown that their wish will not be obtained until the required word is

used they will promptly articulate the word distinctly and as quickly relapse into grunts and pointing if they think they can get what they want without the trouble of speaking. I have several times noticed this delay in the development of speech to occur in families, e.g.

A girl, aged 2¾ years, would never talk unless obliged to do so by failing otherwise to get her desire, she preferred to make a noise like 'um', 'um', and point to the object to which she wished to draw attention. She was a bright intelligent child, and about two years later talked as freely and well as any ordinary child. Her brother, about four years younger, and, therefore, born too long afterwards to have been influenced by her example, showed an exactly similar unwillingness to talk; at the age of two years he would never use an articulate word if he could avoid it.

Another type of simple delay is that in which there is no attempt at speech until long past the usual age, perhaps not until two or three years old and then quite suddenly, so that the day or even the hour can be fixed, the child begins to talk almost fluently. Mr. Edmund Gosse, in his recent volume *Father and Son*, writes, 'I was slow to speak. I used to be told that having met all invitations to repeat such words as "Papa" and "Mamma" with gravity and indifference, I one day drew towards me a volume and said 'book' with startling distinctness.'

A more striking example of the sudden acquisition of speech at a later age has been recorded by Dr. Bastian[1]. I quote it in full:

The patient was the son of a leading barrister, he was then twelve years old and had been subject to fits at intervals. The first fits occurred in infancy when the patient was about nine months old. Towards the end of the second year these fits seemed to have ceased, and the child appeared sufficiently intelligent, to be well, in fact, in all respects except that he did not talk. When nearly five years old, the little fellow had not spoken a single word, and about this time two eminent physicians were consulted in regard to his 'dumbness'. But before the expiration of twelve months, as his mother reports on the occasion of an accident happening to one of his favourite toys, he suddenly exclaimed 'What a pity!' though he had never previously spoken a single word. The same words could not be repeated nor were others spoken, notwithstanding all entreaties, for a period of two weeks. Thereafter the boy progressed rapidly and speedily became most loquacious.

As another instance which, although not illustrating the sudden acquisition of speech, may serve to prevent pessimism in prognosis in cases of delayed speech development, I may quote the history of a girl whom I first saw when she was under the care of my colleague, Dr. Voelcker (he has recorded the case fully in

[1] *Aphasia and other Speech Defects*, p. 5, Lond., 1898.

the *Transactions of the Clinical Society*, vol. xxxiii), she was then aged seven years and did not talk at all, beyond saying 'Eh' and imitating a few simple sounds such as 'bee', 'baa', 'bye' and 'gee'.

She had had convulsions up to the age of three years, but not since; she had learnt to walk at twelve months. I saw this child again at the age of fourteen years, her speech was then perfectly fluent and showed nothing abnormal and the girl seemed of average intelligence.

2. **Defective intellect causing delay of speech development.** Judging from my hospital experience I should say that the commonest cause of delay of speech development beyond the end of the second year is mental deficiency. Almost all imbeciles and mentally defective children are late in acquiring speech, often making no attempt to talk at four or five years old, and when they do speak the articulation is usually very imperfect. But in these cases also it is seldom necessary to give a gloomy prognosis as to the ultimate acquirement of speech: the large majority of mentally defective children, almost all indeed except the most fatuous of idiots, learn to talk eventually, and it is remarkable how much their articulation can be cultivated by patient and skilful teaching.

I have elsewhere quoted figures furnished to me by Dr. Caldecott, of Earlswood Asylum, showing that out of 442 imbeciles only 87—that is 19·6 per cent., failed ultimately to acquire speech.

Dr. Shuttleworth's figures at the Royal Albert Asylum, Lancaster, showed complete absence of speech only in 13 out of 589 imbeciles, but 95 others made only slight attempts or a few articulate sounds, so that altogether 108, that is 18·3 per cent., could hardly be said to talk.

3. **Deaf-mutism.** Next to deficiency of intellect, I should say that deafness is the commonest cause of inability to talk, and the deafness may have been acquired or it may be congenital. Rather over 50 per cent. of the cases of deaf-mutism are due to acquired deafness.

It is noteworthy that the deafness which causes failure of speech development is not necessarily complete. I have seen cases in which it was quite clear that the child heard sounds if they were particularly loud or of a particular character, but did not hear voice sounds with sufficient distinctness to imitate them, and consequently did not learn to talk. In this connexion I should like to point out also what I think is not generally recognized, that imperfect speech, for instance, clipping the

ends of words so as to say 'Că' for cat, and 'dŏ' for doll, is sometimes due to imperfect hearing: speech is normally acquired by imitating the sounds heard in the speech of others, and if the child catches these sounds only imperfectly it is natural enough that he should reproduce them imperfectly.

Otitis media is so common in infancy that it is only remarkable that deafness is not more frequent than it is: any acute illness, but especially any catarrhal condition of the naso-pharynx, may lead to otitis media; all the specific fevers, especially measles and scarlet fever, are at times complicated by it. But an acquired deafness is not always due to ear disease, there are cases in which deafness and consequent mutism are the result of posterior basic meningitis: the deafness is then probably of central origin like the blindness which is left by this disease; but unlike the blindness, which usually disappears after several months, the deafness remains permanent and complete.

Posterior basic meningitis is most frequent in the first year of life and therefore before speech has been acquired, but if it occur in the second year of life when the child has already begun to talk, the limited vocabulary which has already been acquired is usually lost and the child remains dumb unless taught by special methods. Such a case was the following:

Lily H., aged four years, was a healthy child and was talking well at the age of two and a half years, when she became acutely ill one day with vomiting and screaming, and next day had marked retraction of the head and some general opisthotonos, which lasted for four weeks with frequent vomiting; during this time sight was lost and she ceased to talk. There was apparently no ear-affection.

Eighteen months later, there was no evidence of hydrocephalus, the child seemed perfectly intelligent, sight had completely recovered, but the child was a deaf mute, hearing was entirely lost.

Congenital deafness is responsible for absence of speech development in the remaining portion of this group. The deafness is dependent sometimes on congenital abnormalities of the internal ear, more often probably upon abnormalities of the auditory centre in the brain. Deaf-mutism from this cause shows a remarkable tendency to run in families: there was in the Children's Hospital, Great Ormond Street, a boy who was a congenital deaf-mute; he was one of a family of eight children; five boys and three girls: four of the boys, and one of the girls were deaf-mutes.

Congenital word-deafness. This curious condition is presumably allied in its pathology to the cases of congenital deaf-mutism but differs in the site of the abnormality. The auditory

centre would seem to be normal but there is no adequate connexion between this and the part of the brain which should interpret what is heard; the child hears speech but it conveys no meaning, so that the child pays no special heed to it, and therefore is easily mistaken for a deaf child. Moreover, as words mean nothing to him, he naturally makes little or no effort to imitate them, and so does not learn to talk.

The child with congenital word-deafness often appears absolutely blank when spoken to, indeed might be 'stone-deaf' for all the notice he takes, but let a clock strike or a musical-box play and at once it is evident that the child hears and takes interest: moreover, it is clear in some of these cases from the child's general behaviour that he is perfectly intelligent, or at any rate nothing more than a little backward in other respects. Moreover, if patient effort has been made to associate certain sounds with certain *things* that he can *see*, the child may be able to name an object in a picture, although he may fail to understand the same word when mentioned apart from the picture: for instance, if one points to a dog in a picture the child if he has been taught will say 'dog', but if one asks him without the picture before him, 'Where is the dog?' the word may convey nothing to him.

The child can repeat words which he hears spoken but does so mechanically, without understanding them. Medical inspection of school children has made it evident that this condition is less rare than was supposed; children suffering with it were formerly mistaken for mentally deficient or for deaf children. The following cases were of this kind:

Victor M., aged $6\frac{2}{12}$ years, brought to hospital because he is unable to talk; he has had earache at some time, but it is not known that he has ever had any otorrhœa: nothing abnormal can be detected in the ears. The boy is a first child, he has never had convulsions. He looks and seems in every way perfectly intelligent; he is fond of drawing things from sight. If shown certain things, for instance, bread on the table, he can name the article; and he evidently understands simple orders, for instance, 'Shut the door'; but he watches his mother's face carefully when she speaks and evidently gathers her meaning partly from the movements of her lips.

His hearing may be defective but he certainly hears, and apparently hears ordinary speech well. He does not speak at all except to name things which he can see, or to repeat a few words said to him, after he has watched the face of the speaker.

Catherine B., aged $4\frac{3}{4}$ years, had never talked: she had some otorrhœa at two years old, but before this had made no attempt to talk, only imitating sounds made by animals. When she was brought to me at $4\frac{3}{4}$ years she was an unusually well-grown child, looking as if about seven years old: she appeared to be perfectly healthy, and looked quite intelligent.

She would have passed for a normal child, until one spoke to her, when her manner at first sight suggested complete deafness. I have noted that she 'took absolutely no notice of the sound of my voice, in fact might have been stone-deaf, dumb, and blind, for all the notice she took of anything said to her'.

She showed no sign of shyness, but simply appeared absolutely vacant when questioned or addressed: I tried to coax her to talk but her face remained completely expressionless. I was beginning to think that, in spite of the mother's statement that she could hear, she must really be deaf, when I showed her a doll which made a long-drawn but not very loud squeaking sound, this caught her attention and with scarcely a movement of the muscles of her face she imitated the sound.

Her mother then quietly hummed a tune to her and the child, still with a remarkable lack of expression on her face, began humming the tune in unison with her mother, and when her hand was moved to beat time, she continued spontaneously beating time quite correctly. Her cry was curiously monotonous, like the speech of a deaf person. It was, I think, clear that the child's hearing was little if at all defective; and that the condition was really one of congenital word-deafness.

Dr. C. J. Thomas[1] has recorded four very typical examples of congenital word-deafness, all in boys. I shall quote his description of one, at the age of $9\frac{1}{2}$ years, as it exemplifies the condition well.

'Healthy boy who would pass for intelligent on inspection. He understands no questions put to him, but if allowed to watch the lips will give his name when asked, his articulation being clear and good.

Hearing: hears all notes blown softly upon the mouth-organ, responding each time by raising the hand. Hears a bell softly struck at a distance of 30 feet. Repeats all simple sounds, "ah," "oo," &c., softly spoken to him, and will imitate many words without understanding them in the least.

Sight: V = 6/9.

Vocabulary: has learnt to name most of the objects surrounding him, "book," "keys," "chair," "table," &c., he calls his chin his mouth, and calls the mouth-organ a "blow".

General: recognizes letters and can give the sound of the majority. Drawing

[1] *Internat. Mag. School Hyg.*, 1905.

very good, imitates well. He plays naturally and intelligently with the other children and is quite able to take his own part with them.'

Voluntary absence of speech. There are certain children of abnormal mental condition who might easily pass for deaf and dumb unless their history were obtainable from those who are constantly with them. When a stranger speaks to them they will take absolutely no notice, neither coaxing nor scolding elicits a word from them, and yet they show no sign of shyness or timidity : their face betrays no consciousness that any one is speaking to them.

The child can talk, for the parents say that he will speak to them at home, but with strangers he is obstinately dumb. A boy was brought to me at the age of eleven years, with a history that he had had convulsions from time to time until he was seven years old, and did not learn to talk until he was six. He was a very unruly child and very passionate, dull, but not by any means imbecile.

He had a habit of wandering away from home by himself, with the result that he was frequently lost, and being found by the police in various parts of London, apparently homeless, was taken to the nearest police-station, where no information whatever could be obtained about him, as the boy appeared unable to talk.

The boy was brought to me for this trouble, and I endeavoured to get him to talk, but in vain, his face showed no consciousness whatever that any one was looking at him or speaking to him. After about twenty minutes of fruitless effort, I gave up the attempt : a few minutes later, as the boy went out of the room with his mother, he turned and said in the most natural way, 'Good-morning'. I have seen other cases very similar, the unwillingness to talk is not, I think, due to any contumacy or bravado : these children are, so far as my experience goes, always mentally abnormal, they are usually backward, sometimes to a considerable degree, and like the boy I have mentioned passionate or eccentric children.

Loss of speech. I have already pointed out that the child who becomes deaf within a few months after the acquisition of speech is likely to lose the words he has learnt, and to remain a deaf-mute until taught by special methods. There are conditions in which speech is temporarily lost ; a severe fright has occasionally abolished speech for a time. Buckland [1] tells of a boy aged nine, who was seized in Ratcliff Highway by a tiger

[1] *Curiosities of Natural History*, vol. iii, p. 249.

which had escaped from Jamrach's Store; the boy was rescued with no further injury than a bite, apparently slight, in one shoulder, he was so frightened, however, that he spoke not a word for four hours.

Kussmaul mentions the case of a boy, twelve years old, who was unable to speak for nine days after a fright.

In chorea speech is sometimes completely abolished for several weeks; a girl under the care of Dr. Lees in the Children's Hospital was unable to speak for eight months, although the choreic movements during the greater part of this time were by no means violent.

I have also seen absence of speech for a week or more in a child convalescent from typhoid.

It is seldom that a child becomes aphasic from gross lesions in the brain: in a boy, aged eleven, with chorea and infective endocarditis speech was suddenly lost when left hemiplegia, probably of embolic origin, occurred; not a word was spoken until the twentieth day after the onset of the hemiplegia, he then gradually recovered normal speech.

Faulty Speech

The speech of children is often faulty in production: the faults may be classified under two general headings (1) Stuttering; and (2), what I shall call for lack of a better term, Defective articulation. German writers distinguish these as Stuttering and Stammering, but in England the term stammering is so generally used as synonymous with stuttering that it seems almost too late to apply it to faults entirely distinct from stuttering, such as lisping, and failure to pronounce certain sounds, for example, th, r, and s: the terms dyslalia and paralalia, though somewhat pedantic, convey well the distinction between stuttering and defective articulation.

1. **Stuttering** (dyslalia) is a defect of co-ordination. Articulation involves a highly complex series of movements, involving the muscles of the tongue, the jaw, the palate, and, not least important, the muscles of respiration; the perfect co-ordination of these movements is only gradually acquired, and hence occasional stuttering is so common in children of two or three years, whilst they are learning to talk, as to be almost a normal feature at this age.

How often one notices that a little child will articulate quite regularly and smoothly so long as he talks slowly, but directly he becomes eager and excited his co-ordination fails, he stumbles

in his speech ! Stuttering at this age is indeed exactly analogous to the stumbling in walking which is so common, not only in the child who is learning to walk, but also during the first two or three years of life, when the child attempts to walk fast or to run. Both in walking and in speech the failure of co-ordination and the tendency to stumble is specially noticeable when the child hurries and also when he is tired.

I draw attention to these everyday facts of early childhood because, as I shall point out, they throw some light upon the occurrence and treatment of stuttering in older children. It is also necessary to point out the innocence of occasional stuttering during the first two or three years of life, because parents are apt to mistake it for the more serious disorder which is met with in older children and to be unnecessarily anxious about it.

Occasionally, it is true, co-ordination is never fully acquired, what is innocent and natural in earliest childhood persists as the much more serious inconvenience of later life : but as a rule this stuttering of the first two or three years passes off after a few months, leaving no tendency to this fault of speech.

The stuttering of older children as a rule begins about the middle period of childhood, often about the commencement of the second dentition, i.e. at six or seven years old. Boys are very much more liable to stutter than girls : out of forty consecutive cases brought to me for this disorder twenty-nine were boys.

Stuttering is associated in a large proportion of cases with other evidence of nervous instability or defective nervous control : amongst the forty cases I have mentioned five suffered with habit-spasm, six others had enuresis, four (including one of those who had enuresis) had epilepsy, one had asthma and occasional incontinence of fæces, one had had rheumatism with chorea three times, seven others talked or walked in their sleep or had night terrors ; in several other cases the child was said to show great nervousness or excitability.

The basis of stuttering is defective nervous control : or that ill-defined but very real entity, the nervous temperament ; the determining cause may be some depressing illness, or a shock of any kind, perhaps even the irritation of threadworms. I have noted the onset of stuttering after specific fevers, particularly scarlet fever, diphtheria, and whooping-cough. I have notes of two cases in which it began just after a fright : one of these was a boy whom I first saw at eleven years old for stuttering and enuresis. A few years previously he had been lost for

a whole day and was taken to a police-station by the police, from this time the stuttering began. In another case it began after a fall downstairs.

Knowing how very easily nervous children contract morbid habits, I can quite believe that stuttering may originate in some cases by imitation, perhaps even by wilful imitation. I have seen cases in which one or other of the parents stuttered as badly as the child about whom I was consulted. One little boy, aged six years, was brought to me with the history that he had amused himself a short time previously by mimicking the stuttering of a playfellow who lived next door, and had quickly fallen into the habit of stuttering in earnest. The boy was a highly nervous child who talked in his sleep and suffered also with habit-spasm.

In almost all the cases I have mentioned the child was rather above the average in intelligence: the child who stutters is usually the quick-witted, thoughtful, sensitive child. Charles Lamb was writ down 'an inveterate stutterer', but as he says of himself, 'his conceptions rose kindlier than his utterance', and it was Charles Kingsley who wrote of his difficulty in speech, 'that fearful curse of stammering which has been my misery since my childhood'.

In some children the stuttering is only noticeable when the general health is poor, in most the difficulty is aggravated whenever the child is tired, for instance, at the end of long school hours.

Stuttering takes various forms: in some cases it consists in prolongation or repetition of the first consonant; for instance, one child, when asked how old she was, said 'F-f-f-f-five', the 'f' sound was simply prolonged: in others it is repetition of the first syllable, for instance, a boy called 'Benny' told me his name was 'Ben-ben-ben-ben-benny'. Much more rarely it is the last syllable of a word which is repeated.

Another variety consists in a spasmodic indrawing of breath, which for a moment prevents the beginning of a sentence; another is spasm of the muscles of articulation, so that there is complete speechlessness for several seconds. With any of these varieties there are often associated spasmodic movements of the face, in severe cases also of the head and limbs.

All these faults occur chiefly at the beginning of a sentence, or over those words upon which the main emphasis should fall, and certain sounds are more difficult than others, perhaps particularly the explosive sounds 'p', 'b', 't', 'd', hard 'g', and

'k'; though none are entirely free from the difficulty, and even the vowel sounds which usually give no trouble may be a stumbling-block to the severe case.

Stutterers are usually free from stuttering in singing, and also in some cases in reading aloud poetry or rhyme. In whispering also there is no stutter except in severe cases.

Treatment of stuttering. The presence of stuttering as a transient phenomenon in early childhood during the normal development of speech, shows that the co-ordination necessary to perfect articulation is only gradually acquired, it has in fact to be learnt. And one may assume that what is possible in the earliest years of life is possible also for the stutterer in later years, he can with perseverance acquire the necessary co-ordination.

But in order to do this he must learn to accustom not only the muscles of articulation but also those of respiration to orderly action. For this purpose regular breathing exercises are of value, but they must be done daily and preferably twice a day to be of real help. Next the child should practise daily reading aloud and singing: the latter particularly may be useful as an introduction to the more difficult exercise of reading aloud; and for the reading or reciting, nursery rhymes or simple poems should come first, as the rhyme assists co-ordination; and so gradually the child may reach ordinary prose.

But whatever exercises are used care must be taken that faulty speech is not allowed to go unheeded: if the child stutters he should be asked to say the word, or read the sentence again. Often it will be found that by taking a deep inspiration before beginning a sentence a child can avoid stuttering altogether.

Before all else there are two rules to which the stutterer must pay attention if he is to be cured: *Speak slowly: speak quietly.*

But the treatment of stuttering consists not only in the education of speech; it involves also in many cases attention to the general health. It is very noticeable in some children that they stutter only when they are 'run down': and it may be more necessary to advise a stay at the seaside or a course of arsenic and nux vomica than to give directions for the training of speech. There can, I think, be no doubt that the presence of worms in some way enhances nervous instability, and a few doses of santonin may be a necessary part of the treatment of stuttering. The effect of fatigue is also to be remembered, both physical and mental exertion must be limited.

Defective articulation (paralalia), the fault which Germans would describe as stammering in contradistinction to

stuttering, is a normal occurrence during the development of speech. It consists essentially in the substitution of an easy sound for one which the child finds more difficult. This only adds to the charm of baby talk: it is pretty enough to hear the little one talk of 'dŏin' in de tain wif muvver', but in an older child such faulty speech is a serious drawback.

By the age of about four years these natural imperfections of baby talk should have disappeared, and their persistence after this age must usually be regarded as abnormal.

Amongst mentally defective children a very large proportion show some defect of articulation; most commonly, I think, a simple slurring of word-endings, e.g. the child says 'că' for 'cat', 'dŏ' for 'dog', and so on; but very usually in association with other faults, for instance, the child substitutes 'd' for 'th', says 'dă' for 'that', or omits consonants almost entirely, saying 'ah-ah' for 'father'.

Amongst children of normal intelligence various faults are met with; two of the most familiar are lisping, the substitution of 'th' for 's' (sigmatismus interdentalis), and the use of 'w' in place of 'r'; so that the child says, 'wound the wugged wock the wagged wascal wan': but other faults are hardly less common, omission of consonants both in the middle and at the end of words, or substitution of sounds which seem to follow no particular rule: for instance, a highly intelligent boy of six years used the sound 'pf' to replace many other sounds; for 'blackbird' he said 'pfaf-bir', for 'goat' 'pfo', for 'cock-a-doodle-doo' he said 'pfof-a-boo-boo'. A 'mouse-trap' was 'pfar-ta'.

A girl, aged $5\frac{1}{2}$ years, used a 'y' sound chiefly; 'ladder' was 'yadder', and 'house' was 'youse', and so on: her brother, aged $4\frac{3}{4}$ years, omitted all consonants except at the beginning of words, he even managed to get through 'rhododendron' without a single consonant except the initial one, it was 'Ro-o-ee-ah'!

I have seen a few cases in which this omission and substitution of sounds was carried so far that speech was entirely unintelligible, and might have passed for some barbarian language; the child spoke with fluency and with obvious intelligence, a nursery rhyme was only recognizable by the rhythm, but it was clear the child had understood and learnt from the normal speech of his parents: such cases have been described as 'Idioglossia'.

They differ in no respect except in degree from other cases of defective articulation or 'paralalia', in which only certain sounds are at fault: moreover, like these, the fault may be rectified

by training; for instance, I saw a girl, Alice C., aged $6\frac{2}{12}$, whose language was entirely unintelligible, with the exception of the word 'Daddy'; for 'Daddy, come and play with me', she said 'Daddy, tar-tar tay te tee': for 'Rosie Griffiths' she said 'Essie Disser'; for 'pencil', 'Tan-too'; she understood apparently perfectly well what was said to her. I saw the child again when she was nearly thirteen years old, she had been to school and her speech had gradually become almost completely normal.

Many of the children who show one or other of these various defects of speech are of perfectly normal intelligence, but I think it must be admitted that the presence of such faults in a child of six or seven years or more should make one cautious in prognosis, for such children are apt to be more or less backward in mental development; and in this respect are less satisfactory than the stutterer, who is usually fully up to, if not above, the average intelligence.

Treatment. The first essential in the correction as in the prevention of defective articulation is good example. It is as true of speech as it is of morals that 'evil communications corrupt good manners': a child cannot be expected to be clear and accurate in articulation if his exemplar is a nurse or a parent who lisps or has some other fault of speech. And here I would emphasize the need not only for freedom from gross faults of articulation in those with whom the child is in constant association, but also for distinct speech; let it be remembered that every word and syllable has to be acquired by a child by imitation of what he hears, and it is no wonder if his articulation is faulty when those around him are mumbling and indistinct in speech.

Attention paid to the careful education of a child's speech during the first four or five years of life would do much to prevent some of the defects of articulation which are most common.

And here I cannot refrain from mentioning a practice which in my opinion is as mischievous as it is foolish, in talking to little children, namely, the use of those very faults of articulation which are often so troublesome to correct as the child grows older; a mother says to her child, 'Dood boy,' or, 'Baby do in de tain,' correctly imitating the child's way of talking, as if the baby understood her any the better because she uses incorrect sounds: such a practice is only likely to favour the persistence of defective articulation in the child.

I need hardly point out that there is a great difference between the use of the conventional terms of babydom such as 'puff-puff',

or 'gee-gee', or 'bye-bye', and the use of slovenly articulation; there is no harm in a child learning to say 'puff-puff', but there is harm in encouraging him in his faulty way of saying it as 'fuff-fuff'; the child should be taught to articulate these baby words distinctly and correctly.

Faults of articulation can usually be cured by careful training if the child is of normal intelligence, and even in the mentally defective it is surprising how great an improvement can be obtained by patient teaching. The old familiar nursery rhymes are excellent for speech cultivation: the child finds a pleasure in repeating them, and if taught to say them clearly and distinctly may profit more from 'Jack and Jill' and 'See-saw, Marjory Daw', than from many a more recondite study.

CHAPTER LII

SLEEPLESSNESS, LOSS OF APPETITE, AND SOME OTHER SYMPTOMS

In this chapter I shall consider certain symptoms which are not uncommon in childhood, and which are often puzzling both as to significance and treatment.

Insomnia. How often one hears the complaint, 'My child will not sleep'! There are infants who from their birth are 'bad sleepers', they sleep lightly, waking on the slightest disturbance, and often they will lie awake without any apparent cause so much of the day and night that the mother almost believes her own exaggerated statement that they 'never sleep'.

In children beyond the age of infancy also there is the same trouble; some children will lie awake for several hours after being put to bed, others will fall asleep quickly but wake a few hours later and remain awake for two or three hours, with the result in either case that next day the child is languid and looks pale and dark under the eyes.

When the trouble dates from earliest infancy I think it usually indicates an abnormal nervous excitability and foreshadows a nervous temperament in later years, certainly some of the children who suffer with insomnia at a later age, say six or seven years, have always been more or less troubled in this way, and were babies who slept very little. In many of these older children careful investigation fails to detect anything to account for the insomnia, beyond the fact, which is generally sufficiently obvious in such cases, that the child is of nervous excitable temperament.

A very common cause of sleeplessness in infants is dentition. Whether the local worry prevents sleep, or whether the general increase of nervous excitability which undoubtedly accompanies dentition in some infants makes them wakeful, I know not; probably both factors play their part, certainly in some cases there is evidence that, as the mother says, the infant is 'feeling his teeth', for he frequently 'pulls at his gums' and crams his fingers into his mouth in a way that clearly means discomfort.

Both in infants and in older children sleeplessness is sometimes

dependent upon indigestion: distension of the stomach or intestines with flatulence is, I think, a common cause, perhaps by interfering with the movements of the diaphragm and making respiration less comfortable than it should be.

Obstructed respiration, as every one knows from experience with a common 'cold', makes sleep difficult: in infants even more readily than in older children, owing to the smallness of the parts, coryza or adenoids will prevent nasal respiration and interrupt sleep; the infant drops off to sleep, but after a short time is awakened by the difficulty of breathing, he cries pettishly for a few minutes, then falls asleep again, only to be disturbed soon by the recurring difficulty of nasal respiration. In older children large tonsils and adenoids are apt to prevent sound sleep, and the benefit of operation in some of these cases is, I think, due in part to the sounder and more refreshing sleep which is made possible by the freer respiration.

The value of fresh air in promoting sleep is very noticeable in the case of infants, who often suffer with sleeplessness whenever it becomes necessary for any reason to keep them indoors for a few days: older children similarly may be sleepless for lack of an open window.

Treatment. The medical man is often at a disadvantage in advising upon the treatment of insomnia through ignorance of the details of the child's environment at night. There are children—those of nervous temperament—whose sleep is disturbed by noises or lights which would not affect a less sensitive child in the least; there are others who do not sleep because their bed-clothing is too heavy or too light. A romping exciting game just before going to bed keeps some children awake for hours. The nervous child too, like the nervous adult, easily becomes the victim of auto-suggestion, 'they can't, because they think they can't.' In no disorder is the rôle of habit more evident than in sleeplessness; if this habit can but be broken for a few nights only, the insomnia may be cured.

In every case the diet must be considered: it may need revision especially in the direction of reducing such elements as make for flatulence, excess of sugar, barley-water, starch-containing patent foods in the case of the infant, fruit, potato, porridge and suchlike, in the case of the older child. In the latter case also special attention must be paid to the last meal. I see children occasionally of eight or nine years who are being allowed to take as their supper part of the late dinner of their parents, including soup or fish, milk-pudding and fruit. A child should

have his last substantial meal at 4.30 or 5 p.m., not just before going to bed, when a big basin of bread and milk or even undiluted milk is too much for some children.

If sleep is difficult the milk at bedtime should be given with one-third water, and perhaps a teacupful of Benger's Food with diluted milk may be better than plain milk and water. If the child wishes for something to eat, plain biscuits or rusk are, I think, better than bread just before going to bed. I am often asked whether if the child lies awake for hours at night it is advisable to give food : I think that it is, and nowadays, when it is possible to keep food warm for hours by Thermos flasks, there is no difficulty in giving some warm milk and water at night, which with a biscuit may help to get the child to sleep again. A warm bath at night seems to soothe some children and promote sleep.

Occasionally I think sleeplessness is due simply to going to bed too early ; a mother rightly anxious that her child of six or seven years should have plenty of sleep sends him to bed at 6.30 p.m., with the result, especially on light summer evenings, that he lies awake for two or three hours, when if he had been kept up half or three-quarters of an hour later he would have fallen asleep easily.

Children vary in the amount of sleep which they require. In general a newborn infant should sleep about twenty-one hours out of the twenty-four ; at three months an infant should sleep about nineteen hours and at six months sixteen hours a day ; from one to five years of age a child should sleep about fourteen hours a day ; from five to seven years about twelve hours, and from seven to ten years about eleven hours. During the rest of childhood ten hours is sufficient. In these averages I have included the midday sleep, which I would have continued, if possible, until a child is seven years of age. But there are children who not only are incapable of but do not require so much sleep, and sometimes it is better to allow a child to stay up a little later than to send him to bed to lie awake.

But in spite of any modification of the daily routine, some of the nervous children remain sleepless for hours every night, and the medical man is expected to supply sleep in a bottle of medicine. Under these circumstances it is useful to point out to the parents that the trouble is due in large part to the child's nervous temperament, and that though it would be easy to drug the child to sleep by large doses of hypnotics, such treatment is undesirable, the better course is to change the child's environment altogether

for a time; almost invariably a few days at some country or seaside place restores sleep to the child, and after a little while a better habit is induced.

For some of the very excitable children a short course of phenazone and arsenic may be useful, but rather as an adjuvant to such measures as I have mentioned than as sole therapeutic.

I have occasionally tried bromides, chloral, and trional, but they are usually quite unsuccessful unless such large doses are used that one would hesitate ever to recommend them.

Anorexia. Loss of appetite is common enough at all ages, but it is particularly puzzling sometimes in childhood, for there may be no obvious reason, and the refusal to take food is so obstinate that parents become alarmed.

In infancy teething is quite the commonest cause of loss of appetite; many babies for a week or two before a tooth is cut will refuse some feeds entirely and take only a small part of others, and no amount of coaxing will induce them to take more. In these cases the most favourable time to get food taken is often when the infant is half asleep.

Children a little past the age of infancy, at about three or four years old, often suffer with such complete anorexia that after the child has eaten a few mouthfuls any further attempt to eat is followed by retching and actual vomiting; they will refuse the things for which most children crave,—cakes, sweets, jam and such things have no charm for them; the child will sit at table without displaying the least interest in the meal, and if pressed to eat will burst into tears. I have seen scolding and coaxing tried in vain, compulsion ends with vomiting.

Most of the children who show this profound anorexia are pale and unwholesome looking; but sometimes there is absolutely nothing in the child's appearance to suggest anything amiss, the child though taking little food gets enough to maintain nutrition and might pass for a healthy child. But I think that almost always careful inquiry will detect some of the vague symptoms which are associated with indigestion (see Chapter XIII), and in many cases the bowels are costive.

Chronic constipation both in infants and in older children, but more often I think in the latter, is a cause of lack of appetite.

The appetite like sleep is greatly affected, especially in infants and very young children, by the amount of fresh air taken; if from any cause the child is kept indoors for a few days the appetite quickly flags.

Treatment. In the treatment of anorexia the first thing is

to assure the parents that in the absence of any signs of organic disease the lack of appetite need cause no anxiety. Both in infants during dentition and in children of three or four years this trouble will last sometimes for many weeks, without any serious harm, the child may lose a little weight, but this will soon be regained when appetite returns. In the case of the teething infant it is worth while to try some phenazone, gr. ½ twice daily at six months, gr. i thrice daily for an infant of one year; and if the bowels are not well open, ½ grain of grey powder with a grain of bicarbonate of soda, and if necessary a grain or two of compound rhubarb powder should be given three times a day.

For the older child, after a regular daily action of the bowels has been ensured, any of the various preparations which contain phosphorus may be useful: Parrish's Food, the Syrup Ferri Phosph. Co. (B.P.C.), in doses of 1 drachm for a child of four years, or Byno-hypophosphites (Allen & Hanbury), ½ drachm for a child of this age, or plain Malt Extract (which contains phosphates), a small teaspoonful three times a day, or a mixture of nux vomica, 2 minims with 5 minims of dilute phosphoric acid and glycerine and water; any of these given just after meals may help in these cases.

Children usually have so great a dislike to the vegetable bitters, such as gentian, that I have rarely prescribed them, but I have known these to be as useful in children as they are in adults when given with some bicarbonate of soda shortly before meals. A cold bath in the morning in summer with Tidman's sea-salt, or a warm bath in winter with an almost cold douche, may help to stimulate appetite; and care must be taken that the child is out in the open air several hours a day, but without fatigue: these children are often unfit for much walking, they are easily tired and look pale if made to walk far, so that their outing should be as much as possible riding in perambulator or go-cart or carriage.

Lastly, I would point out that a child easily becomes the *malade imaginaire*, and that anxious parents constantly watching the child at table and remarking on the smallness of his appetite may be fostering the trouble they deplore; there is a sense of distinction in being peculiar, and the child will sometimes eat more when no notice is taken of him; a meal in the nursery with younger children to distract attention may be better taken than a meal in the dining-room with mother.

Difficulty in swallowing. I mention next, as being in some instances connected with anorexia, certain cases in which a child

is said to have difficulty in swallowing. Leaving out of account rare cases in which the trouble was due to some real œsophageal obstruction or gross disease of some sort, I find in my notes sixteen cases in which there was this tale. The youngest was aged three weeks, the oldest ten years; nine were between one and two years old; and the second year of life is, I think, the usual age at which this trouble occurs. I shall perhaps make the condition clear by mentioning actual cases.

Wilfred T., aged one year and five months, was brought for 'something wrong in his throat'; he had always had difficulty in swallowing fluids; after taking the fluid into his mouth, he held it at the back of his mouth for some time as if unable to swallow. Given some water to drink in my presence, he took it into his mouth and tilted his head slightly backwards with his mouth widely open, so that the fluid could be seen unswallowed in his throat; in this position he remained for a few moments like a person gargling, but he made no noise and then gradually he swallowed the fluid.

Arthur S., aged one year and three months, was fed at the breast till twelve months old, and ever since birth had been awkward in swallowing. Since weaning there was more difficulty in swallowing fluids, though he took sopped bread easily. Given some milk in my presence he kept it at the back of his mouth like a person gargling with his mouth open and made six or seven masticating movements as if he were eating solids, then, after a minute or so, he swallowed the fluid. There was no abnormality of any sort to be seen in the throat, the palate moved well. The child was apparently bright and intelligent. Five months later the difficulty had almost gone; only when he took a large mouthful of fluid he occasionally went through the same performance.

Charles A., aged one year and five months, was brought because for the last five weeks he did not swallow solids properly. He was said to keep them in his mouth until they were taken out; he swallowed fluids readily. I gave him a finger of bread-and-butter which he took with pleasure: he filled his mouth with it, leaving a piece hanging out between his lips. He sat thus quite unconcerned for four minutes, making no effort even to draw into his mouth the piece of bread which was hanging out. From some movement in the sublingual region I thought he was gently sucking the mouthful, but he made no attempt to swallow it, and at the end of the four minutes he quietly spat the whole mouthful out. A month later the child was swallowing solids almost normally.

The last case shows that the difficulty may occur with solids as well as with fluids. In both cases I fancy the difficulty is due simply to lack of education: the co-ordination necessary for swallowing is less readily established in some children than in others; and when the child has been accustomed only to fluid food the change to solid food requires a new effort of co-ordination which he cannot or will not make. There are many infants who object strongly to the first introduction of solids into their diet

and will spit and retch if solid food such as sops or pudding is forced upon them.

But it is, I think, clear that the delay in swallowing is not always due merely to infantile caprice, there is in some cases a genuine defect of co-ordination ; for in two of my series, a boy of three years and a girl of twenty-one months, fluids were occasionally regurgitated through the nose when the child drank ; in neither case was there anything to suggest paralysis of the palate.

The difficulty is noticed specially when the child passes from one method of feeding to another ; a girl of sixteen months swallowed normally so long as she took food from breast or bottle, but when an attempt was made to give fluid from a cup she took it into her mouth but kept her mouth open so that some of the fluid ran out before she swallowed the remainder. She was apparently a perfectly intelligent child, but the trouble first noticed at six months still persisted when she was eighteen months old.

Amongst my cases there were some in which the supposed difficulty in swallowing was simply a manifestation of anorexia, it was ' a can't of won't '.

Violet B., aged 6¾ years, was brought to me for loss of appetite. She had recently taken a great dislike to food, and actually vomited at the sight of it. Compelled to take food into her mouth, she would keep it there unswallowed, and had kept it so for two hours ; she said she ' could not swallow it '.

Kenneth K., aged 4$\frac{10}{12}$ years, had no appetite, was occasionally sick in the night, was a very nervous boy, and had attacks of ' recurrent pyrexia '. The mother said, ' he takes food in his mouth and keeps it there, says he can't swallow it.'

It is surprising how long such children will keep the food in their mouths unswallowed ; I saw myself a large bolus of bread in the mouth of a boy aged 2½ years, three hours after he had taken it, according to the mother's statement.

As I have indicated, there are cases in which difficulty of swallowing in children is dependent upon more serious cause :

Esther W., aged 9¾ years, was brought to me with a history that, from birth until the age of three years she had had difficulty in swallowing fluids : they regurgitated through the nostrils. Her speech was like the speech of a severe diphtheritic paralysis ; the soft palate moved hardly at all. Her sister was a cretin.

In this case there was apparently a congenital paresis of the soft palate.

The condition known as congenital ataxia, in which there is

some coarse ataxy of the limbs, noticed generally about the end of the first year, though no doubt present from birth, with nystagmus and curious drawling speech, is associated with some difficulty of swallowing: a girl who was under my care with this condition at $4\frac{3}{4}$ years had always had difficulty in swallowing fluids, she had to drink slowly and in small quantities.

I have seen one case in which difficulty of swallowing in a child seemed to be due to hysterical spasm of the œsophagus, it persisted in spite of the passing of an œsophageal bougie, but disappeared after one or two applications of electricity.

Knee-elbow position in sleep. This symptom may be mentioned here, not as being in itself of any importance, but because it seems to be an indication usually if not always of digestive disorder. I have noted it most frequently in children from two to five years of age; the child lies on his face with the knees drawn up upon the abdomen, and generally the forearms flexed and drawn in under the chest, so that the child rests in the prone position chiefly upon his elbows and knees. In most cases the child who sleeps thus has a large abdomen, the result of unhealthy fermentation in the intestine from unsuitable food, especially excess of starch. Sometimes there are other indications of digestive disturbance in the character of the stool, for instance, excess of mucus. I have also seen that curious perversion of appetite which is known as 'pica' associated with this symptom, and pica, as I have pointed out (p. 781), seems to be due in some cases to faulty digestion.

I fancy that the explanation of the knee-elbow position is relief of pressure upon the diaphragm, as when the child lies thus the pressure of the distended intestines falls chiefly upon the anterior abdominal wall, which is more yielding than the posterior, and by its bulging takes more of the pressure off the diaphragm. No doubt what begins as an unconscious effort to relieve discomfort may persist subsequently as simple habit.

'Falling about.' Under this somewhat untechnical phraseology I shall refer to a complaint often made by parents with regard to children about three or four years old, that they stumble more often than is natural at that age in walking and running. There are many causes which may account for such a complaint,—a cerebral tumour, or infantile hemiplegia, which causes the toes to be pointed so that the child is apt to trip in walking, or the disorder known as congenital ataxia, or chorea, or diphtheritic paralysis,—but in these and the various other conditions which may interfere with walking there are usually

characteristic symptoms which make the cause of the stumbling obvious enough; in the cases to which I refer there is usually no obvious evidence of disease to account for the mother's complaint. The falling is not due merely to unsuccessful efforts in learning to walk, for the child has usually been able to walk for at least a year or two, and sometimes the mother dates the trouble definitely from a few weeks before medical advice is sought, and says that it is increasing rather than diminishing.

There are, I think, two causes for this 'falling about'. In some cases there is a defect of co-ordination precisely similar to the stuttering which is so common at this age. The child of three or four years may talk fluently so long as he talks slowly, but if he becomes excited and talks quickly the speech co-ordination cannot keep pace with his words and stuttering occurs; moreover, at this age the child who talks fluently when well and vigorous is very apt to stutter when health is depressed from any cause. So it is with walking, and especially with running, the co-ordination recently acquired is sufficient under ordinary circumstances to prevent stumbling, but if the child becomes unduly nervous and excitable as children are very apt to do with digestive disorders, with worms, or with any slight disturbance of health, it becomes insufficient and action outruns co-ordination: the child in fact stutters in walking.

In another group of cases there is a more tangible cause, namely, laxity of ligaments, which goes usually with flabbiness of muscle. These are the children whose tibiæ can be wabbled from side to side, knocking loosely against the condyles of the femur; the ligaments of other joints are equally lax, and the result is that fixation of the joints in walking is less sure than it should be.

Such a condition is familiar in rickets, but it is also quite common apart from any definite rickets.

Treatment. In the last-mentioned cases the legs should be massaged twice daily, and a douche of hot and cold water alternately may be used over the legs while the child is in his morning bath. The amount of walking must be limited, for the laxity of the joints and lack of tone in the muscles are apt to lead to slight degrees of flat-foot or knock-knee if the child runs about too much. Where the trouble appears to be merely an insufficiency of co-ordination as I have suggested, the diet may need revising, or santonin powder may be necessary for worms. In both groups of cases a tonic containing nux vomica will be useful.

CHAPTER LIII

HEAD-NODDING WITH NYSTAGMUS IN INFANCY

UNDER various names, such as 'spasmus nutans', 'head-nodding of infants', and 'head-shaking with nystagmus', there has been described a curious and somewhat uncommon disorder which is worthy of attention, if only on account of the unnecessary anxiety which it arouses in the minds of those unfamiliar with its occurrence. The earliest published record of which I am aware is one by Ebert in 1850, but it was not until the late Dr. W. B. Hadden of the Hospital for Sick Children, Great Ormond Street, published his series of observations in 1890 that the disorder became generally recognized. Thirty-seven cases of this affection have come under my own observation; twenty-two of the patients were boys, and fourteen were girls (the sex was not noted in one case); this disproportion, however, may be accidental, for in a series recorded by Dr. J. Thomson,[1] there were fifteen boys and twenty girls. The disorder is almost limited to infancy, i.e. the first two years of life, although, as I shall point out, in rare cases it lasts beyond this limit. In twenty-eight out of thirty-four cases in which the age at the onset could be determined, the disorder began between the ages of five months and twelve months; of four which began earlier only one was said to have begun as early as the age of three months, and two began after the first year, both before the age of fourteen months.

Sketched in outline the clinical picture is somewhat as follows: as the child sits on his mother's lap there is, perhaps, nothing abnormal to be seen for a minute or two, then, as the child fixes his gaze on some object, the head begins to nod at the rate of about eight or ten nods within five seconds, after which there is a pause of varying length; and so, with irregular intervals, short series of noddings occur more or less frequently so long as the child is in the sitting position. Hardly less striking than this rhythmic unsteadiness of the head is the curious way the child

[1] *International Contributions to Medical Literature*, Festschrift, May, 1900.

has of looking at objects out of the corner of his eyes with the head slightly averted, and the face turned slightly downwards as if he were unaccountably shy.

The other feature which attracts attention is the exceedingly fine rapid nystagmus, which is peculiar in being so much more marked in one eye than in the other that it may appear to be actually limited to one eye, a point which the mother herself has usually noticed. These three symptoms, the head-nodding, the tendency to look out of the corner of the eyes, and the nystagmus, constitute the characteristic features of spasmus nutans which I shall now consider in more detail.

Symptoms. The rhythmic movement of the head is not necessarily an antero-posterior affirmative nod as the term 'head-nodding' might seem to imply; indeed, so far as my own observations go, a lateral shake of the head as in negation would appear to be a commoner movement. Sometimes also the movements vary, being at one time antero-posterior, at another negation, whilst more rarely there is a simultaneous combination of the lateral and antero-posterior movements, producing a sort of pendulum movement. In twenty-eight cases I have noted the variety of movement: fifteen patients showed only lateral rotation (negation); eight showed only antero-posterior nodding; four either of these movements at different times; and one the combined pendulum movement.

The rate of movement varies from 60 to 120 nods per minute; the series of nods lasts sometimes only for four or five seconds, and sometimes as long as thirty seconds. I have noted in one case three bouts of nodding in six minutes, but they may be more frequent, or may occur much less often; there is no regularity in the intervals, they depend largely on the child's occupation. I have thought that the nodding occurs chiefly when the child fixes his gaze upon an object in a sort of abstraction, but that the movements cease directly the child is aroused to active attention. In some cases I have specially noted that the movements could be stopped by attracting the child's attention. Perhaps there is some variation in this respect, for one mother informed me that the movements were specially noticeable when the child was watching children playing in the street, and another said that her child showed the nodding chiefly when not interested by anything in particular. The head movement occurs only when the child is sitting with the head unsupported; it ceases, therefore, when the child is lying in a cot or leaning the head against the mother's arm. This is a point upon which I would

lay some emphasis, for it is of some importance in diagnosis; the condition might easily be overlooked if the child is seen only lying in a cot. Moreover, there is another movement, head-rolling, which must be distinguished from the head movement of spasmus nutans, and which occurs chiefly when the child is lying down. The range and vigour of the movements vary considerably in different cases and in the same case at different times; in some they are so slight as to be easily overlooked, while in others they are obvious to all. Twice I have seen the antero-posterior nodding so forcible as almost to shake the child's bonnet off, but this is, I believe, quite exceptional. But whether the movements be slight or vigorous, they are obviously involuntary, and in this way differ from the much commoner 'head-rolling', which has the appearance of a voluntary movement.

I have already alluded to the occurrence of head-nodding when the infant is sitting gazing at some object in an absent-minded sort of way. And this leads me to remark on the curious tendency of these children with spasmus nutans to fall into an absent-minded stare, a sort of 'brown study' which seems hardly natural for an infant. But there is nothing whatever in these moments of abstraction to suggest petit mal. The child takes notice directly attention is drawn to sight or sound; there is no change of colour, no tendency to any epileptiform manifestation—in fact, no ground whatever for regarding this peculiarity as due to petit mal. I wish to lay great stress upon this point, for it has been stated that children with spasmus nutans show a special liability to petit mal. So far as my own observations go, I have not seen the least tendency to epilepsy of any sort. Very remarkable is the habit these infants have of looking out of the corner of the eyes while the face is turned in the opposite direction and slightly downwards. So characteristic is this when present—and I think it is present in many cases—that it should at once suggest spasmus nutans. The parents themselves have often noticed it. In one of my cases it was the first complaint that the infant had recently begun to look out of the corners of his eyes 'in a funny, coy way', which had made the father, a medical man, fear some serious cerebral disease. I have no explanation to offer for this curious symptom. I have heard it suggested that it is due to an effort to obtain fixation of the eyes which nystagmus renders difficult, but this can hardly be so, for I have seen this sideway gaze very marked in two of the exceptional cases where there was no nystagmus. 'Gaze' I say rather than 'glance', for during this action perhaps even

more than at other times the infant is apt to stare fixedly as if in one of those 'brown studies' to which I have referred. This phenomenon may be noticeable several times within five or ten minutes.

The nystagmus which with the head movement constitutes the chief feature of spasmus nutans is characteristic and peculiar in certain respects, and chiefly in two points: (1) its unilateral predominance. It is almost invariably much more marked in one eye than in the other—indeed, in some cases it appears to be actually unilateral. This unilateral predominance, apart from any unilateral lesion of the media or fundus, is, so far as I am aware, excessively rare apart from this particular disorder; (2) its onset without apparent cause in an infant a few months old, and its complete disappearance after a few weeks or months. Such temporary nystagmus in infancy without ocular lesion or any evidence of gross cerebral disease is almost peculiar to spasmus nutans. The nystagmus differs also from that due to some other conditions in being exceedingly fine and rapid. It may be vertical, horizontal, or rotary, and Dr. Thomson has observed it to be convergent instead of conjugate in some cases. I have seen it associated with vertical nystagmus of the upper eyelid, but this is unusual. I have not observed the rhythmic contraction of the pupil (hippus) which some writers have recorded; Dr. Thomson noted this in four out of thirty-five cases.

The onset of nystagmus in spasmus nutans may precede the head-shaking by several weeks or even months. In one of my cases it was stated to have been present three months before the head-nodding began; more often the head movement is the first symptom that attracts attention, and the nystagmus may appear later. From this variation in the order of occurrence of these symptoms it might be conjectured that cases would occur in which one or other symptom was lacking altogether, and there can be little doubt that not only does the characteristic head-nodding occur sometimes without nystagmus at any period of the disorder, but the nystagmus may be the only manifestation of spasmus nutans, if the affection may be so called when head-shaking is entirely absent. The evidence of this lies in the occurrence in infants of nystagmus which has the characteristic features mentioned above, and which shows the same age-incidence, seasonal variation, and relation to rickets, and the same temporary character, a duration of weeks or months, as does the nystagmus which is associated with head-nodding. I have included in my series of thirty-seven cases three in which was

shown this nystagmus alone, and two in which was shown the characteristic nystagmus, associated not with head-nodding, but with the much commoner quasi-voluntary movement, head-rolling, an association which is perhaps to be regarded as accidental. Of the thirty-two cases with head-nodding, twenty-nine patients showed nystagmus sooner or later (in two of these it was extremely slight and perhaps open to doubt); only two showed no nystagmus at any time.

With this description of the symptoms of spasmus nutans I would mention the case of an infant, aged five months, in whom not only were the antero-posterior noddings of the head unusually marked, but there was also some shaking of one arm. These symptoms had lasted for some days when I saw the child, and I heard from Dr. C. W. Cooke of Willesden, under whose care the child was, that they passed off soon afterwards without leaving any ill effect. In one other case I have noted some shakiness of the arms, and Dr. Thomson mentions this symptom as present in two cases in his series.

Etiology. I turn now to the consideration of the etiology of spasmus nutans. Rickets is present in a large proportion of the cases. In twenty-four out of thirty-one cases in which I have noted this point the patients showed some degree of rickets, but only four showed severe osseous rickets; in most it was particularly noted that the evidence of rickets was definite but slight. The occasional absence of rickets shows that rickets is not an essential factor; moreover, in the rare cases in which the disorder begins at as early an age as two or three months, rickets is not likely to have been a causal factor even if it is found later. It is, I think, clear that spasmus nutans does not stand in so close a relation to rickets as do such conditions as laryngismus stridulus, facial irritability, and tetany—phenomena which in infancy are almost always associated with well-marked and often severe degrees of rickets. It is, indeed, noteworthy how seldom spasmus nutans keeps company with any of these phenomena, which are so closely associated together that the presence of any one of them raises an expectation of the others and which, like convulsions, are well recognized as expressions of the nervous instability of rickets. In my own series one patient showed facial irritability; in Dr. Thomson's series of thirty-five cases two showed laryngismus stridulus with or without facial irritability, and two others showed facial irritability without laryngismus. As a predisposing cause, however, rickets must be regarded as a frequent factor in the etiology of spasmus nutans.

The relation of this disorder to dentition is specially noteworthy. The onset of spasmus nutans is most commonly between five and twelve months of age, the period when the worry of dentition begins; the affection rarely persists after the end of the second year, the end of the first dentition. Moreover, it was evident in some of my cases that just before the eruption of a tooth the head movements and also the nystagmus were increased, while they at once became less when the tooth was through the gum. My own opinion is entirely in agreement with that of Henoch, who regarded the irritation of teething as a possible cause of spasmus nutans. I think, however, that it is quite likely that other forms of peripheral irritation may also act as exciting causes, and some support is given to this view by the closely comparable phenomenon to which Mr. W. T. Lister has drawn my attention, the occasional production of temporary nystagmus by ear-syringing. Preceding illness or injury would seem to have determined the onset in some of my cases; in one with nystagmus alone this began three days after an attack of diarrhœa and vomiting; in another it began during convalescence from diphtheria; in two others during convalescence from infantile scurvy; in one case head-nodding began four days after an attack of diarrhœa, in two just after bronchitis, and in one a few hours after a fall on the head. Its occurrence in rickety children and after the exhaustion of various illnesses or the shock of an injury, and its very close relation to dentition and the exacerbations just when a tooth is in process of eruption, all suggest that spasmus nutans is a functional disorder depending upon an acquired or congenital nervous instability with some peripheral irritation as an exciting cause.

The defective-light theory. Of recent years there has been put forward a theory which is worthy of careful consideration—namely, that spasmus nutans is due to living in ill-lighted dwellings; dimness of light, it is supposed, causes deficient fixation of vision, and, moreover, the infant throws his eyes into an unnatural and strained position in attempting to look towards the window. To this strain upon the ocular muscles is due the nystagmus; the head movements are secondary to the nystagmus. This is the view put forward by Raudnitz[1]. The arguments adduced in its favour are: (1) the fact that infants with spasmus nutans often live in dark dwellings; (2) the rarity or non-existence of the disorder amongst the wealthier and consequently better housed class; (3) the onset of the disease nearly always during

[1] *Jahrbuch für Kinderheilkunde*, Band xlv, p. 145.

the dark months of the year; and (4) the analogy with miners' nystagmus, with which there are known to be associated in rare instances some rhythmical swaying movements of trunk and head.

Now I venture to think that in spite of the above apparently strong arguments there are grave difficulties in accepting the defective-light theory.

1. It is quite certain that spasmus nutans may occur in infants living in well-lighted dwellings. I have inquired specially into this point in thirty-one cases: in four of these I was able to visit the home, in the rest my inquiry was only by careful interrogation, not by actual visiting, and is therefore less convincing than actual visitation would be. In seven cases only did the home appear to be ill lighted, and in some of these the child was hardly taken out of doors at all. In twenty-four out of the thirty-one cases, including the four which I visited myself, there appeared to be no reason for regarding the room in which the child chiefly lived as ill lighted, and in several cases it was specially noted that the rooms were light and sunny. But in considering the question of light, it is obviously necessary to take into account to what extent the child lives indoors. Some of my patients were taken out daily. One of those which I have mentioned as living in ill-lighted rooms was out in the open air daily for three to four hours, and in one case it was particularly stated that the child was taken out much every day and 'in all weathers'.

2. The disorder is undoubtedly seen chiefly in hospital practice, but I have seen it in three cases in private practice amongst people in comfortable circumstances and well housed, once in the infant of a medical man whose nursery was an excellently lighted room. It must be remembered also that rickets, which most observers have regarded as a predisposing cause, is commoner amongst the hospital class than amongst the well-to-do.

3. The seasonal incidence of the disease is, indeed, remarkable. In 26 of my cases the onset could be dated with some degree of accuracy: January, 7; February, 7; March, 1; April, 0; May, 0; June, 1; July, 1; August, 0; September, 2; October, 1; November, 1; and December, 5. In seven others the date could be determined with less certainty, thus: January, 1; February, 2; March, 3; and November, 1. So that out of 33 cases 22 began within the three months December to February. Only two began during the five months April to August. These figures agree with those collected by Dr. Thomson, and addition

of his statistics to mine shows that 85 per cent. of the cases of spasmus nutans have their onset during the five months November to March.

But the special incidence of the disorder in these comparatively dark winter months is not necessarily connected with deficiency of light, and I would point out that laryngismus stridulus, another nervous disorder associated with rickets, and one which can hardly be attributed to deficiency of light, has a very similar seasonal incidence (see chart, p. 654); its onset is, it is true, rather more often in March than in January, but according to my own statistics no less than 80 per cent. of the cases have

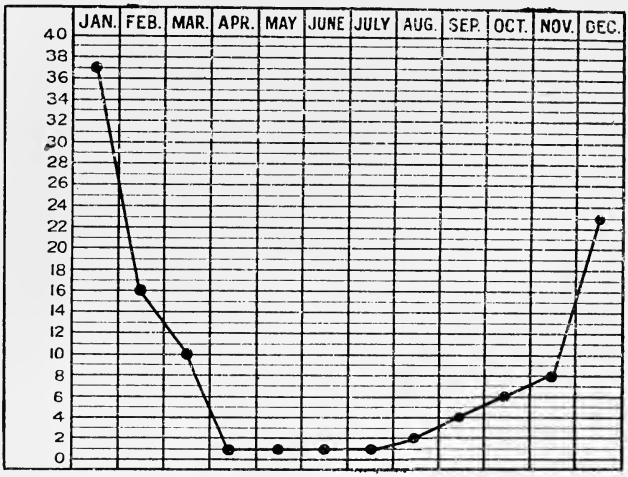

Fig. 52. Seasonal incidence of Spasmus Nutans. Chart showing the month of onset in 110 cases (statistics collected by Dr. J. Thomson combined with the author's series).

their onset during the five months November to March. It would seem, therefore, that during this season there is some influence apart from deficiency of light favouring the onset of certain nervous disorders in connexion with rickets.

4. The analogy with miners' nystagmus can hardly be said to support very strongly the defective-light theory, for it is the opinion of experts now that such nystagmus is due much less to the bad light than to the strained position of the eyes in this occupation; and nystagmus is known to occur from a similar cause in other occupations in which a strained position of the eyes is usual, although the work is done in broad daylight.

Moreover, the miners' nystagmus does not show, so far as I have been able to ascertain, the unilateral predominance which is so striking a feature in the nystagmus of spasmus nutans.

Lastly, this theory seems to assume that the head movements are secondary to the nystagmus; whereas clinical experience shows that the head movements often precede the nystagmus, and occasionally no nystagmus appears at any time.

Prognosis. Spasmus nutans usually passes off after a few months, leaving no ill effects; occasionally it lasts only from two to three weeks, more often it lasts from three to twelve months; it rarely lasts beyond the end of the second year. I have, however, seen nystagmus still present in one case at the age of four years.

As already stated, I know of no special relation between spasmus nutans and epilepsy, minor or major; indeed, I have not seen a single case in which the child has developed epilepsy either during the persistence of, or subsequently to, spasmus nutans. I suspect that the tradition of such an association has arisen from the description given by Ebert of cases of epilepsy with clonic jerking of the head under the same heading 'Spasmus seu Eclampsia Nutans', an unfortunate confusion of disorders which differ widely both in their symptoms and in their course. In one of my cases a single convulsion occurred in the ten months during which spasmus nutans lasted, but it appeared to be an ordinary infantile convulsion such as any rickety infant may have, and no others occurred during the subsequent period of observation (five months).

I have been asked several times whether the disorder will leave any injurious effect upon the intellect and I have kept notes in several cases of the child's progress in walking, in talking, and in intelligence. I have not found them more backward than other children, unless the degree of rickets happened to delay development, which it rarely did; but spasmus nutans, being an index of nervous instability, is, I think, likely to occur in children who will show nervous peculiarities at a later age. (I am not alluding to imbecility, though there would seem to be some special liability to this disorder in Mongol imbeciles; two cases in my series were 'Mongols', and Dr. Thomson includes two cases in his series of thirty-five cases, and alludes to another.) In several cases I have thought that the child, long after all trace of the disorder had disappeared, was a highly nervous or eccentric child, without showing any lack of intelligence. One patient was sent to me subsequently for 'spitefulness and screaming', another at the age of four years because it was 'so nervous and

screamed at the slightest thing'. Another at three years and four months for pica (dirt eating). Apart, however, from this possible foreshadowing of later tendencies to neurosis, it is probably safe to give an unqualified good prognosis.

Diagnosis. Spasmus nutans must be distinguished from a very rare congenital condition of nystagmus with head-nodding. This disorder, unlike spasmus nutans, is permanent, not transitory. In a case under my care, a boy aged seven years, apparently of average intelligence, showed head-nodding very like that of spasmus nutans, but the nystagmus, which dated from birth, was coarser and less rapid than that usually seen in spasmus nutans and showed no unilateral predominance. The eyes, as in spasmus nutans, showed nothing abnormal apart from nystagmus.

Another curious movement to be distinguished from spasmus nutans is the head-rolling which is seen in infancy, and sometimes in children just past infancy. It occurs chiefly when the infant is lying down—the child rolls his head monotonously from side to side on the pillow. The movement appears to be quite voluntary, whereas that in spasmus nutans appears to be involuntary; it is not associated with nystagmus.

Clonic antero-posterior jerkings of the head occasionally form the chief manifestation of epilepsy, which has then unfortunately been called 'eclampsia nutans' and by some writers 'spasmus nutans'. The attacks consist of a series of forcible noddings of the head or bowing movements of the whole trunk. Dr. West states that as many as fifty or more jerks of the head may occur in succession. The child at the time has a vacant appearance, probably with more or less complete loss of consciousness. Nystagmus, if present, is only likely to occur as part of each attack, whereas in spasmus nutans it is to be seen just as often when the head is at rest as when the nodding is present. The head-jerking may occur without any spasm of the limbs, but sooner or later more widespread spasm is likely to occur, so that the attack resembles the more ordinary attacks of epilepsy; but even when the clonic spasm of the head is the only manifestation, the sudden onset of each attack, the more jerky character of the movement, the vacant appearance or loss of consciousness, and probable change of colour, all make the distinction from spasmus nutans obvious; indeed, it is probable that whatever confusion has arisen is owing less to any resemblance between the disorders than to the unfortunate use of the term 'eclampsia nutans' for this form of epilepsy, which bears no relation whatever to spasmus nutans.

Treatment. The very variable duration of this disorder and its frequent fluctuations in degree make it difficult to estimate the value of treatment. Diminution both of the head-nodding and of the nystagmus sometimes follows the use of sedative drugs, and I have thought that phenazone was more valuable than bromide in these cases. At six months old half a grain, and at one year old one grain of phenazone may be given. In some cases I have given a combination of bromide with cod-liver oil apparently with good results.

The part played by rickets in predisposing to spasmus nutans suggests also the advisability of inquiring into the feeding and correcting any fault which may favour the rachitic tendency. I have no doubt also that cold or tepid douches as the infant sits in a warm bath tend to reduce the nervous instability, and that confinement in a close, ill-ventilated room, whether well or ill lighted, increases this instability and is therefore to be forbidden. For this reason I have urged the parents to keep these children out of doors as much as possible, and I suspect that in the greater liability to confinement in a stuffy room with closed windows during the cold months lies the explanation of the seasonal incidence of spasmus nutans rather than in any deficiency of light.

CHAPTER LIV

ON CERTAIN MORBID HABITS IN CHILDREN

THERE is no period of life at which habits are so readily established as in childhood, but fortunately—for, like rank weeds, bad habits grow faster than good—childhood is a plastic age when the deep ruts of habit may yet be smoothed away, and custom moulded to new tracks.

A troublesome habit is that of masturbation, a practice only too common, as every medical man knows, in boys at the school age, but common also in girls, and—a fact not nearly so well known—by no means rare in infancy. For some reason the large majority of children brought to the medical man for this habit in the first few years of life are girls. I doubt whether it is really commoner in girls than in boys, possibly the mother mistakes for an innocent phenomenon the manifestation of the habit in the boy; but there is one reason which may account for its frequency in girls, namely, the great liability to vulval irritation from lack of cleanliness or from actual vulvo-vaginitis. I am convinced from my own observations that in the large majority of these cases in girls the habit has been started by some such local irritation, and what began as a mere scratching or rubbing to palliate itching or discomfort, has ended in the habit of masturbation. No doubt the child is led thereto by the discovery that the act brings with it some concomitant gratification.

I find amongst my notes thirty-nine cases, of whom thirty-one were girls; five of these thirty-nine cases were brought for this habit when under the age of twelve months, and six in the second year; the youngest was eight months old when brought, but several were known to have begun the habit earlier than this; two began it at five months, two at six months, three at seven months; in one girl whom I saw at the age of two and a half years for masturbation the mother, an intelligent woman, was quite certain that the habit began during the first week of life. In twenty-two out of twenty-five cases in which I have noted this point, masturbation began before the age of two years.

The manifestations of this habit are so often unrecognized by

medical men that I shall quote the mother's description of the symptoms in a few cases, as far as possible in her own words.

Elsie D., aged one year and eleven months, was brought for 'attacks' which began at the age of eleven months; 'she throws her head back and gets into such a heat that beads of perspiration stand on her forehead, at the same time she screws her legs up and crosses her thighs and takes no notice of anything; after this her face, which has become very red, turns pale. This occurs many times a day if the child is left sitting alone.'

Irene W., aged nine months, when sitting on her mother's lap crosses her left thigh over the right and 'works her body about'; whilst doing this she throws her head back, flushes and perspires, and then goes very pale.

Annie W., aged seven years, was said to 'wriggle' when sitting on a chair, and at the same time went very red and then turned pale and seemed sleepy.

Mabel S., aged eighteen months, since the age of six months has had a habit of frequently 'clenching her hands and wriggling till she goes dull in the eyes'.

Maud J., aged two years, was brought for attacks of 'stiffness of the legs'. Since she was twelve months old she has had these 'attacks' five or six times a day, always when sitting up, never in bed; she crosses the thighs rigidly, goes very red, breaks out in a sweat, and then turns pale.

In other cases the child was said to 'rise as if trotting while sitting on a chair, making a grunting noise, and turning pale afterwards', or to 'sway herself backwards and forwards on her chair, and then perspire very much and go red and then white', or again, the child is said to 'stand and rub herself against the corner of a chair, making a straining noise and then turning pale'.

In most of these cases in infancy and in early childhood the habit is practised in the day-time, not at night—at any rate, it is not noticed at night; only in few cases does the child use her hands to rub herself.

From the symptoms which I have described it can be understood that the habit may easily be mistaken for some epileptic manifestation. I have twice at least seen such cases in consultation where a diagnosis of epilepsy had been made. The mistake is the more natural when the mother mentions, as I have noted in several of my cases, that the child 'fixes her eyes' or 'stares' during the paroxysm, and also that she 'quivers' or 'waves her hands in a sort of tremor', and that after the attack the child is sleepy or actually goes to sleep.

This last symptom is, I think, occasionally accountable for the persistence of the habit; the child learns that sleep comes with the exhaustion from the orgasm and, by a kind of auto-suggestion, finds difficulty in getting to sleep until she has induced weariness by masturbation; one little girl who was brought to me for this habit at about eight years old, when reproved, said, 'But I can't go to sleep if I don't do it,' and I have no doubt that her explanation was a sound one.

I have noted in almost every case that after the orgasm the child turned pale; in some cases the mother added that after it the child 'looked dark under the eyes', and usually the child was 'quite exhausted'. In several cases the mother said that the child had become very nervous and fretful when the habit was most frequent.

These symptoms indicate the harm that is likely to come from this habit in early childhood; the general health is depressed to some extent by the repeated exhaustion, the child becomes pale, lacking in vivacity, and often nervous and peevish.

Beyond this I have seen no harm, unless I was right in suspecting that in one child who had had previous convulsions, the orgasm of masturbation sometimes passed directly into an epileptic attack; but here, as I think probable in all such cases, the tendency to epilepsy was present independently of the masturbation, which was only a determining cause of particular attacks.

No doubt in later life the habit may do serious harm by increasing sexual excitability and so predisposing to immorality, and herein I think lies one important reason for making every effort to stop this habit before it has become firmly established. In the meantime we can assure the parents that the physical harm from this habit has been grossly exaggerated by unscrupulous quacks for their own base ends, and that there is not the least danger of the child becoming idiotic as a result of this practice—idiots are often addicted to this habit, but it is the result and not the cause of their mental condition.

I have already referred to the part played by vulval soreness and irritation in producing this habit in girls; let me here point out, especially to those who have the responsibility of deciding whether a boy should be circumcised, that the exposure of the glans penis, especially of the corona, by circumcision seems sometimes to be the exciting cause of masturbation in boys. No doubt an adherent prepuce—and an unduly long or tight prepuce is usually adherent—may be and often is a cause of local worry, and may thus induce this habit, but none the less,

I think that in many cases circumcision is done most unadvisedly. A boy is deprived of Nature's covering for the sensitive glans, which is left exposed to the constant friction of clothes, so that the poor child is willy-nilly kept in constant mindfulness of this part, when, perhaps, by merely separating the adhesions and pulling the foreskin back daily during the first few years of life circumcision might have been avoided and all this local irritation prevented. Even where circumcision is necessary, there is no need whatever to cut the foreskin away wholesale, leaving corona and glans exposed; nowadays some surgeons have realized this, and I see boys with a prepuce left sufficient to cover the corona and the greater part of the glans, a condition of things far less likely to favour the habit of masturbation than the flayed and unprotected state which is too often seen after circumcision.

The underclothing of boys is sometimes calculated to increase the irritability of these parts: an undergarment which allows the penis to be continually slipping in and out through the opening in front is a bad one; many little boys are clad in a combination garment so arranged that this is inevitable; a little care and common sense can easily prevent this source of irritation.

Treatment. One great factor in the successful treatment of this habit is early recognition. If only masturbation is detected in infancy it is seldom difficult to stop, but when it has been going on several years there is usually great difficulty in breaking the habit. It seems probable that in a very large proportion of cases this habit begins in infancy, and as the infant practises it without concealment the failure or success of treatment at this age is evident. It should be impressed upon parents that they must not be satisfied until it is quite certain that the habit is stopped. Whether any evidence of inflammation is detected in the vulva or not, it is, I think, wise to insist upon the necessity of sitting the child in a warm bath for a few minutes twice a day, with the thighs well separated, and if there is definite evidence of local irritation, a calamine lotion, such as that in use at the Children's Hospital (Zinci Oxidi gr. xxx, Calaminæ Prep. gr. xxx, Glycerin. ℥xxiv, Liquor Calcis ℥xxiv, Aqua ℥j), may be applied after the bath, or, if there is definite vulvo-vaginitis, a half per cent. solution of protargol should be used locally.

In infancy mechanical devices are more likely to be successful than at a later age, when I think they are seldom of any permanent value. In these earlier years, however, mechanical restraint is very rarely necessary, for drugs are often effectual. I have found the liquid extract of salix nigra particularly valuable in

several of these cases, almost always it reduced the frequency of the habit considerably, and in some cases it stopped it apparently altogether. In the case of a girl of two and a half years, 5 minims of the liquid extract given three times a day stopped the masturbation in a few days; in a boy aged one year the habit was much diminished in frequency by $2\frac{1}{2}$ minims given similarly.

On the other hand, an older child, a boy of seven years, was none the better for 10 minims of the extract three times a day; and I must point out that the drug produces unpleasant symptoms when given in large doses. A dose of 15 minims ter die given to a boy of nine years made him feel giddy each time it was given, and a dose of 20 minims given thrice daily to another child of nine years, gave her a transient headache after each dose.

I have also used belladonna in combination with bromide with good effect: to a child of twelve months a mixture of Tinct. Belladonnæ ℥iv, Sod. Bromid. gr. ij, Syrup ℥xx, Aq. Anethi ad ℥j, may be given three times a day, and the dose of belladonna may be cautiously increased if necessary. Occasionally I have used phenazone with the bromide, but I think it is less valuable than belladonna in these cases.

Whatever drug or other treatment is used, it must be impressed upon the parents that at all costs the habit must be broken, and that whenever the child attempts to do it, he or she must be stopped by force if necessary. This will mean some determination on the part of the parents, for infants will scream passionately when interfered with during the act.

In older children a little judicious bribery is worthy of trial, the child should be under constant supervision by a trustworthy and tactful nurse, and a promise should be made that a successful avoidance of lapses for a stated interval shall secure some coveted reward. Some such method is, in my opinion, far preferable to punishment, which is only likely to add cunning and deceit to bad habit.

Pica: or Perverted Appetite

Very curious is the habit which has been described as pica, or dirt-eating; it is seen mostly in the later half of infancy, from one to two years of age, but, as Dr. John Thomson has pointed out, it begins sometimes in later childhood, when from any cause the general health fails, and the child becomes anæmic. Dr. Thomson[1] has recorded eleven cases, in nine of which the habit began before the child was two years old.

[1] *Edinburgh Hospital Report*, vol. iii, p. 81.

I have notes of fourteen cases which have been under my care, seven boys and seven girls; not including three cases of hair-eating which ought perhaps to come in a different category.

The history of the child with pica is sufficiently shown by the following cases:

Ivy J., aged 4½ years, was sent to me by Dr. H. M. Stewart of Dulwich, since the age of fifteen months she had had a craving for what her mother described as 'rubbish', she was particularly fond of green stuff, such as raw cabbage-leaf and potato-peel, a fortnight before I saw her she had pulled down a clematis in a neighbour's garden and eaten the leaves, she would also eat the wax faces of her dolls, had eaten five between Christmas and February, and at times had eaten sand and mortar and had also eaten part of her pinafore; when taken to consult a doctor, she was found eating the papers in his waiting-room.

She was a very passionate child, but apparently of normal intelligence. Her appetite for ordinary food was very poor. She had had threadworms since the age of two years.

The only acute disturbance which had resulted from her morbid appetite was after eating privet leaves, when acute gastric symptoms had occurred.

Muriel J., aged 2½, at the age of fourteen months began to eat earth, and when placed in her 'pram' to stop this she sucked the mud off the wheels and ate it; she had a great fondness for eating mortar, which she picked out of walls; she had eaten the tops off a dozen safety matches, and candle-grease was also a favourite. She ate the legs and arms off all her dolls if they were made of 'composition', so that only china dolls could be used. She had a very bad appetite for ordinary food, and was so passionate that the day before I saw her she had thrown a scissors at her sister when thwarted of some desire: she had also suffered with night terrors.

She sometimes passed earth with her stools and had also vomited earth. Her mother stated that she herself had suffered at the age of sixteen years with a craving for blotting-paper and linen, and used to get out of bed at night to eat towels.

Grace D., aged 2½ years, since the age of eighteen months has been in the habit of eating mud and dirt: she will pick the dirt out from between the boards of the floor, or mud off the soles of boots, and eat it; she will also eat coals and earth, and if prevented by force she cries. She is a very excitable child and is quite unnaturally timid. The mother's brother is insane.

Stewart H., aged 1½ years, was brought because for the last two months he had taken to eating mud, hearth-stone, bits of brick, soap, or 'anything he can get hold of': he was particularly fond of the white plaster off toy horses.

His appetite for normal food was bad: the bowels had been constipated, and occasionally after eating such things as those mentioned he retched.

The child was very irritable, and during the persistence of the dirt-eating habit he had begun to sleep badly, talking in his sleep and starting up in terror at night: he was intelligent and showed no signs of disease except some rickets.

Three months later he was taken to Scotland, with the result that his general health improved greatly and his appetite became good, and he lost his craving for unnatural food altogether.

Mud and mortar seem to be special favourites with these

children; coal, cinders, and gravel were also mentioned in some of my cases. In nine out of my fourteen cases the habit began in the second year of life; in one only it began in the first year (at eight months); in two it began in the fourth year; in two I have not noted the time of beginning, but in one of these the habit was present at $3_{1}^{4}_{2}$, and in the other it was known to have been present at $2\frac{1}{2}$ years.

Now what is the significance of this curious perversion of appetite? As I have mentioned, there was nothing in any of the cases to which I have referred to suggest any mental deficiency; imbeciles often show a similar habit of dirt-eating, but in them it is less strange, for it is associated usually with an extreme degree of mental deficiency.

Some light is thrown upon the point by the disorders with which pica is associated. It goes, I think, in the majority of cases with definite indications of the 'nervous' temperament; one child I had seen a few months earlier for spasmus nutans, another a few months after the pica ceased attended for enuresis, another subsequently developed stuttering and somnambulism, others, like the cases I have mentioned, show an abnormal passionateness or excitability.

No doubt these nervous symptoms are aggravated by more or less digestive disturbance set up by the abnormal material eaten, but I think that the development of other nervous disorders in some cases after the pica has entirely ceased, and the family history in others, go to prove that the nervousness is partly at least cause rather than effect.

Another association which I think plays an important part in the causation of this perverted appetite is digestive disorder.

Again I admit the difficulty of proving how much of this is cause and how much effect. In almost all cases the appetite for ordinary food is extremely poor, in fact it is often this rather than the dirt-eating which excites the mother's anxiety: the abdomen is usually large, the stools sometimes contain mucus, and the bowels are costive or irregular.

It is natural enough that such symptoms should be induced by the indigestible substances eaten, but in some cases it has seemed to me clear that there was digestive disturbance before this habit began, and I suspect that this is so in the majority of cases and that the consequent discomfort, hardly felt as such perhaps by the child, plays some part in exciting the habit of dirt-eating in a nervous child. This is confirmed, I think, by

the effect of treatment: an important part of the treatment is the improvement of digestion.

Prognosis. I have not seen any serious harm come from this habit, but it is obvious that there must always be a risk of acute gastro-intestinal disturbance. The most noticeable feature in my cases has been the dull pallor of the face. As Dr. Thomson says, 'Their complexion has not the rosy tint of healthy childhood: it lacks clearness and is dull and unhealthy looking. They are hollow-eyed, often with a hungry and unhappy look'. I would add that they are almost always fretful and irritable while the habit is in force.

The duration of the habit is often months, or even some years if no special measures are taken for its cure: in one of my cases the dirt-eating persisted from eight months to two years and four months, when it ceased spontaneously: in another it stopped when the child was about two years old: in another at two years and eight months: in another, where it began at fifteen months, it was still troublesome at $4\frac{1}{2}$ years. It usually yields to treatment, but I have known relapse to occur.

Treatment. The first essential in treatment is to prevent the child obtaining the dirt, coal, mortar, or other injurious substance for which it craves; the second is to improve its general health, especially its digestion. For this purpose a combination of liquid malt with nux vomica makes a useful mixture, and it may be advisable to give with this or separately a grain or $1\frac{1}{2}$ grains of phenazone, according to the age, to allay the nervous irritability which is one part of the disorder.

But there is no part of the treatment more valuable than a few weeks at a bracing seaside place, or if this is not attainable, at some high-standing breezy inland country place. At the same time it will be necessary to aid digestion by the most careful dieting: if the child is living chiefly on milk, it may be well to add sodium citrate to it, or to peptonize it partially for a time, and care must be taken that the food is not such as to set up fermentation in the bowel, or to keep up a mucous catarrh by its irritating residue; I need not repeat here what I have already said elsewhere on the subject of feeding and indigestion, these cases of pica call for careful adaptation of the diet to the digestive capacity of the particular child.

Teeth-grinding

Teeth-grinding is exceedingly common in childhood, and is sometimes puzzling as to its significance. It is seen, or rather heard, under such diverse conditions that it will be well to mention first those in which we can be certain of its exciting cause, and then to consider those in which its relation to associated disorders is less defined.

Grinding of the teeth occurs sometimes in the waking condition, but far more often during sleep : so far as I have observed in children its occurrence during waking hours is almost, but not quite, restricted to those cases in which there is either organic disease of the brain or imbecility. I shall refer subsequently to the rare cases in which this is not so.

Teeth-grinding in a marked and persistent form is to be heard in the child with chronic meningitis, especially with posterior basic meningitis, where it sometimes alternates with the curious champing movements of the lower jaw to which Dr. Lees and Sir Thomas Barlow have drawn attention. It is also to be heard sometimes where there is chronic hydrocephalus from any cause, and it is not uncommon in imbeciles, where it is likely that there is some structural abnormality of the brain.

In all these conditions it may reasonably be assumed that there is direct cortical irritation.

Next there are cases in which local irritation seems to be the cause, for instance, I have met with it in children who were worried by teething, both towards the end of the first dentition and during the early stage of the second dentition. In two girls, aged respectively about six and eight years, the habit began with the approach of the permanent incisors and when these were cut rapidly passed off. I have seen it also with extensive caries of teeth, and in the absence of other apparent reasons it seemed at least possible that the dental irritation was responsible for the habit.

I have noted teeth-grinding in association with otorrhœa, and considering how definitely middle-ear irritation is responsible for various functional nervous symptoms such as head-retraction and head-rolling, I think it is likely enough that it may also start the habit of teeth-grinding.

But commoner than any of these sources of irritation is disturbance in the intestines. There is a popular notion that

teeth-grinding usually means the presence of worms in the bowel; I think it may safely be affirmed that while this is true for some cases, in the majority of children who grind their teeth there are no worms, but there is chronic indigestion; they are often pale children with large abdomen and rather puffy about the eyes, and they are constipated or their stools are said to contain mucus: the child is usually thin and said to be wasting as children with chronic indigestion do; moreover, there are often associated with teeth-grinding those other indications of a restless brain which are closely connected with digestive disorders, sleep-talking, somnambulism, and night terrors.

I have more than once seen children with teeth-grinding assume the knee-elbow position in sleep, a habit which I think almost always indicates digestive disorder, particularly chronic distension of the abdomen from fermentation and flatulence in the stomach or bowel.

There are other associations which may throw some light upon the significance of teeth-grinding even if they have no bearing upon its exciting cause: enuresis is common in children who grind their teeth during sleep, habit-spasm also I have seen with teeth-grinding. I have known laryngitis stridulosa (spasmodic croup), which I take to be a respiratory neurosis, to occur in the child who was given to teeth-grinding; and, lastly, the children with this habit are often notably excitable, nervous children.

Now it is always necessary to be careful in attributing any causal relation to such common conditions as worms or indigestion and, *a fortiori*, to physiological processes such as dentition; association does not prove a causal relation, we have to reckon with the possibility or probability of coincidence; but none the less the cumulative evidence of experience goes, I think, for something, and from the facts which I have mentioned I think we may reasonably surmise that teeth-grinding means cortical irritation, which may be direct as in the case of posterior basic meningitis or may be reflex from some peripheral source, be it the teeth themselves, the ear, or the intestine; moreover, the liability to this habit will be the greater where the cortex is specially excitable as it is in the child of nervous temperament.

The limitation of teeth-grinding as a rule to the time when the child is asleep corresponds no doubt with the diminished control by the higher functions of the brain at this time, and is comparable to the much greater frequency of enuresis during sleep than when awake; and one might expect that if the

nervous instability were much increased or the stimulus more powerful, the teeth-grinding might occasionally occur like enuresis when the child was awake. As I have already mentioned, this happens with gross lesions of the brain and in the mentally defective, but it happens also, though very rarely, in the child who shows neither of these causes.

A boy, aged 7½ years, was brought to me on account of a very slight elevation of the temperature, which for seven weeks had been noticed to be up each night nearly to 100°. The boy was said to be a nervous, excitable boy; but was chiefly remarkable for his intellectual attainments; he talked like an intelligent boy of about fifteen, he read French and English well, took a keen interest in the scientific nomenclature of insects and was regarded in fact as a 'prodigy'.

Whilst he was in my consulting-room and unoccupied during my talk with his parents he began grinding his teeth horribly, and I found that some of his teeth were worn down by this habit.

It was noteworthy in this case that the boy had been under treatment recently for indigestion, his bowels were so costive that he had to take a daily aperient, and he suffered with nausea frequently. The slight rise of temperature at night for many weeks with nothing more than slight digestive disturbance is, as I have pointed out elsewhere (Chap. XIX), a common occurrence in the nervous child.

Here there was clearly a combination of an exceptionally nervous temperament and active brain with indigestion and constipation: and therein lay the explanation of this unusual occurrence of teeth-grinding during waking hours without mental deficiency or gross cerebral lesion.

Treatment. The treatment of teeth-grinding is sufficiently indicated by its causes. It is seldom necessary to give sedatives such as bromide or phenazone; the chief point to be investigated where imbecility and gross brain-disease can be excluded and where the presence of worms has been disproved, is the diet; in most cases this will need careful revision, not only as to the food taken, but also as to the time of meals. Dr. Bazett, of Hendon, tells me of a girl of nearly fourteen years in whom troublesome teeth-grinding ceased when the taking of a substantial meal immediately before going to bed was discontinued; in some cases I think the source of the trouble is fruit, which so often causes the various symptoms of indigestion in children and, lastly, a regular action of the bowels may be the one thing lacking.

Head-rolling and head-banging

There occur in infancy and early childhood certain curious movements more or less rhythmic in character, and sufficiently unusual to cause some anxiety to parents and to others who are unfamiliar with them. It is important that the medical man should be aware both of their occurrence and of the significance of these movements, for, although in themselves they may be but of little importance, they are sometimes a valuable indication of underlying conditions.

It is no uncommon complaint that an infant 'keeps rolling his head from side to side' on his pillow, and there may be corroboration of the mother's statement in the thinness of the hair at the back of the infant's head. As the infant lies in his cot he rolls his head monotonously from side to side with occasional pauses if his attention is attracted; the movement may continue even when the child is asleep, as I have noted in two cases, but in both it was only during light sleep; it ceases almost invariably when the child is sitting up. This head-rolling occurs chiefly in infants under the age of two years. Nineteen out of twenty-nine cases under my own observation occurred within the first two years. The oldest child I have seen with it was a girl aged five years, who was in hospital for cleft-palate. The head-rolling in this case was chiefly during light sleep, and, as usual, only when the child was lying down, but it had the unusual feature that it was accompanied by a low-pitched crooning noise, a sort of 'auto-lullaby'.

The curious rolling of the head is apt to arouse fears in the parents' mind of 'something wrong with the brain', and even to the medical man it may suggest the possibility of meningitis. With regard to this latter association, let me say at once that head-rolling very rarely, if ever, means meningitis. What, then, is its significance?

A large proportion—fifteen out of nineteen cases in which the point was noted—showed more or less rickets; and in four of my twenty-nine cases other nervous disorders, usually associated with rickets at this age—viz. laryngismus stridulus with facial irritability in three, and facial irritability alone in one, were present. But rickets can hardly be an essential factor, for the head-rolling began in one of my cases of the age of four weeks, and in some I have specially noted that rickets was absent. Whether there be rickets or not, there is usually another factor—namely, peripheral irritation of some sort.

It is to this particular form of peripheral irritation that I wish to draw attention specially in connexion with these cases of head-rolling. In no less than fifteen out of twenty-nine cases—i.e. in more than half the cases—head-rolling was associated sooner or later with evidence of middle-ear irritation. It would seem that head-rolling may be caused by middle-ear affection where there is no other symptom suggestive of ear trouble, and long before any discharge appears from the ear: in one case head-rolling began four weeks, in another five weeks before ear discharge began; while in another two months elapsed before the persistent head-rolling was explained by a discharge from the ear. The movement in such cases has nothing in it to suggest pain; indeed, the infants have usually appeared to be entirely free from pain, except, perhaps, for a few hours before otorrhœa began. In some cases ear discharge has preceded the onset of head-rolling, and the unhealthy condition of the middle-ear remaining after discharge has ceased may still be sufficient to cause continuance of the movement. This seemed to be shown by one case in which head-rolling had begun synchronously with otorrhœa, and persisted in spite of the cessation of the ear discharge; after head-rolling had continued many weeks the aural disease again became evident by recurrence of discharge, explaining, as it seemed, the persistence of the movement. But middle-ear catarrh may exist without perforation of the membrane at any time, and, therefore, without discharge, and probably without causing pain at any time, certainly without causing any manifestation of pain which can be recognized as such in an infant; there can, I think, be little doubt that some cases of head-rolling are due to such latent catarrh in the middle-ear; indeed, the existence of such a cause was proved at autopsy in some cases. In two cases in which death was due to acute colitis, head-rolling had been specially noted during the illness, but there was nothing else to suggest ear disease during life: autopsy showed in one case pus in one middle-ear, in the other case pus in both middle-ears. In a case of tuberculous meningitis, where, during the early stage of the disease, there had been head-rolling, there was nothing during life to account for the movement, which might very naturally have been regarded as due to the meningitis, had not autopsy shown pus in both ears, which, in the light of other cases illustrating the usual relation of head-rolling to middle-ear catarrh, made it probable that the head-rolling here was due, not to the meningitis, but to the accompanying middle-ear affection.

It may well be that such a latent middle-ear catarrh accounts for many of the cases of head-rolling for which there is no obvious cause, and that the proportion of cases due to ear disease is higher even than can be demonstrated from clinical or post mortem evidence. But there is no proof that head-rolling is due in all cases to this particular form of peripheral irritation. It seems likely enough that dentition is the exciting cause in some cases. The age-incidence, of course, suggests this : twenty-one out of twenty-nine cases began between the ages of five months and two years, and it would be strange indeed if the obvious irritation of tense gums were not as efficient as the much less obvious irritation of an apparently painless middle-ear catarrh in producing this head-rolling. Such a case as that of a boy, aged seventeen months, in whom the eruption of each fresh tooth was stated to have aggravated the head-rolling, lends support to this view ; but as in other disorders where the causal relation of dentition has been asserted, conclusive evidence is difficult to obtain.

Head-banging

In connexion with head-rolling may be mentioned another curious movement, seen during the first few years of life, namely, head-banging. In some cases this takes the form of beating the head with the fists or with any available implement, in others the child beats the head against some hard object, oftentimes bending downwards deliberately to beat its head against the floor, or banging its head against the side of the cot, or against a neighbouring wall if this happens to be more convenient.

I am not referring now to cases in which some slight thwarting of a child's wishes is immediately followed by an outburst of fury, in which the child dashes himself down on the floor and bangs his head again and again against the floor ; such behaviour is not very rare in excitable, unstable children during the first three or four years of life, and sometimes indicates a morbid defect of self-control which may show itself in troublesome ways later on. The head-banging to which I wish to draw attention is something quite different from this—it occurs without any apparent provocation from without, often indeed when the child is quite unaware that he is being observed. It is sometimes sufficiently forcible to cause bruising of the head, but usually the child does himself no harm, although the possibility of injury naturally distresses the parents.

Of twenty-eight cases of which I have kept notes all were under five years of age, and twenty-two were between the ages of six months and two years. Rickets was not more frequent in these cases than in any series of children under three years of age amongst the hospital class: nine out of twenty-two cases showed rickets associated with head-banging, but my own statistics showed that 44·6 per cent. of children under three years of age brought to hospital for disease of any kind have rickets.

Head-banging sometimes occurs in a child who at other times has shown head-rolling; a fact which suggests a similar causation. My own series showed that in six out of twenty-eight cases head-banging bore some relation to ear disease, and that like head-rolling it may precede by several weeks the appearance of discharge from the ear, or may be due to a latent middle-ear catarrh. It is noteworthy, also, that the banging in these cases with ear disease is usually not a beating of the ear but of the frontal or occasionally of the occipital region.

My impression, however, is that head-banging less often indicates ear irritation than does head-rolling, and so far as statistics on this point are of any value the figures I have mentioned confirm this impression.

The fact that more than three-fourths of the cases occur between the ages of six months and two years makes it probable that head-banging, like head-rolling, is due to the worry of dentition in some cases. It seems possible also that headache, from whatever cause, may sometimes excite this movement, but I have never seen head-banging in association with meningitis or cerebral tumour.

Body-rocking

A less common movement than those I have already described is an antero-posterior rocking of the trunk. It occurs in infancy and early childhood, but almost exclusively in the sitting position, and therefore only in children old enough to sit up, not in infants under nine months of age.

Usually it is a simple swaying to and fro of the trunk as the child sits; rarely it is more complicated, as in a girl aged one year and nine months, in whom I noted a combination of a rising movement with the antero-posterior, so that the child appeared to be 'rising' as a rider does in trotting, and this she did at the rate of four times in ten seconds, for periods of ten or twelve

seconds, when there would be a pause, and the movement was then resumed after a few seconds. This movement would continue thus at short intervals for hours, and even whilst being watched by three doctors beside her bed the child continued the movement quite unconcernedly. In this particular case the movement ceased during sleep, but in two others it occurred chiefly during sleep, when the child sat up asleep, rocking itself backwards and forwards. Twice I have noted in children respectively of three years and seven months, and four years, a less common variety of body-swaying, a slow side-to-side movement like that of a metronome ; I counted the rate in one case and found it twenty-eight per minute. In one of these the rocking was chiefly during light sleep, and if the child was roused he would lie down and substitute for the body movement head-rolling such as I have already described.

The substitution of one curious movement for another is not uncommon ; the little girl who showed the ' rising ' movement exchanged it when she was lying down for a rhythmic lifting of trunk and pelvis. In a boy aged eighteen months the to-and-fro rocking was replaced at times by a banging of his head against the sides of his cot ; another boy, aged $6\frac{1}{4}$ years, would sometimes substitute for his to-and-fro rocking during sleep a rhythmic rocking in the knee-elbow position with his face buried in the pillow. The boy at the same time made a monotonous cooing noise, and I have noted a similar accompaniment in another case ; in both apparently, as in the case of head-rolling with this accompaniment, a sort of self-soothing lullaby.

In itself this body-swaying is of no importance, but as a little indication of a child's temperament and tendencies it is, I think, worthy of notice. The children who show this curious movement are generally odd children—not in the least defective in intellect, indeed often rather above the average in this respect, but children of peculiar temperament, sometimes extraordinarily reserved, sometimes unduly self-conscious, sometimes showing some more definite evidence of nervous abnormality, as in the boy aged $6\frac{1}{4}$ years already mentioned, who was brought at one time for temporary fæcal incontinence, a disorder which usually indicates a neuropathic taint.

The formation of abnormal habit, whether rhythmic or otherwise, is often an indication of wider abnormality in the higher functions of the brain, and it is interesting to compare with this body-rocking of children who show no intellectual defect the very similar rocking which is often seen in imbeciles.

I must refer again in this connexion to a movement which must be distinguished from the harmless body-rocking to which I have referred, namely, that which sometimes accompanies masturbation in the sitting position. I have notes of several such cases. For instance, a girl aged five years, was brought with the history that she would several times a day, when she thought herself not observed by her mother, rock herself backwards and forwards as she sat on her chair, at the same time turning red in the face, and perspiring, and then turning white as if exhausted. Inquiry showed that masturbation occurred at night also, and it was evident from the child's appearance that her health was being impaired.

One must distinguish also from the head movements mentioned above the 'head-nodding of infants' or spasmus nutans, which I have considered in the previous chapter, where I have also mentioned the occasional occurrence of head-jerking as a manifestation of epilepsy.

Treatment

In the treatment of the movements which I have described, the first point to be determined is the underlying cause; it may be possible to relieve a middle-ear catarrh, it may be advisable to give some drug, perhaps even in very rare cases to lance the gums to relieve the worry of dentition, but often it is impossible to find any cause, and we are driven to treat the symptoms.

It has seemed to me that phenazone in such cases is sometimes more useful than bromide, but a combination of the two drugs may be still better. Only let us be careful that our treatment is not worse than the disorder; if the movement is doing no harm, and we can assure the parents that its continuance can have no ill-result, it may be wiser to practise a wholesome abstention from drugs than to give any of these sedatives, and indeed, it may be that by careful attention to the diet and hygiene of the child, particularly in the matter of baths and fresh air, we may do more to reduce his abnormal tendencies than by many bottles of medicine.

CHAPTER LV

CONGENITAL SYPHILIS

The frequency of syphilis in children no doubt varies considerably in different parts of the world and in different cities; and this variation depends partly on the frequency of acquired syphilis in the adult population, and partly on the possibility of securing thorough and effective treatment for the syphilitic adults in the particular locality. My own statistics would seem to show that amongst the children who are brought for hospital treatment in London congenital syphilis is not a very frequent condition: amongst 4,830 consecutive out-patients at King's College Hospital in the children's department, where only children under the age of ten years are seen and where fully a third of the patients are under two years of age, there were only twenty-nine cases of congenital syphilis, i.e. less than 1 per cent., or to be more exact 0·6 per cent. These figures include only cases in which the presence of syphilis was beyond dispute; but even when doubtful cases are included the proportion is not above 1·5 per cent.

Such statistics, however, give very little idea of the extent to which the inheritance of syphilis damages the community; to estimate this it would be necessary to have some record of the number of children who fail to reach live birth, or die shortly after birth, as a result of the syphilitic taint. Some idea perhaps of this early mortality amongst the children of syphilitic parents may be gained from the following figures in which only those still-births, miscarriages and early deaths are included which immediately preceded or followed, or formed part of a series immediately preceding or following the birth of the patient in whose family history they occurred; there were 39 still-births, 36 miscarriages, and 25 deaths attributable to congenital syphilis in the family histories of 87 children who were under treatment for congenital syphilis; moreover, out of these 87 children, 13 died whilst under observation.

I shall not consider here the much rarer condition, acquired syphilis in children. The infection of a child after or during birth by contact with a syphilitic person who is still in the infective stage depends upon such a combination of favouring

coincidences that it must necessarily be very rare. I have only seen two, or perhaps three cases myself in which the possibility of such acquired syphilis could be entertained; it must be seldom, indeed, that such infection can be absolutely proved by the presence of a primary chancre on the child.

The introduction of the Wassermann test may necessitate some revision of our ideas of the frequency of syphilis, but it would be premature to accept statistics based on this test at present, for we have yet to learn the fallacies attaching to it, and fallacies there must be, for the results obtained are sometimes not only inconsistent with clinical facts and pathological findings, but actually contradictory of themselves, as, for instance, where a child examined by different skilled observers showed within the same week first a positive and then a negative reaction apart from any influence of treatment. There is no doubt that, in conjunction with other evidence of the disease, a positive reaction must carry considerable weight, but in view of the undoubted fallacies, dependent partly upon variations in technique and partly upon other less known factors, it is not sufficient to base a diagnosis upon this test alone. On these grounds one must view with suspicion statistics in which children, who admittedly showed no trace whatever of the disease, were counted as syphilitic solely on the strength of a positive Wassermann reaction. By such methods some observers have found 10–30 per cent., or even more, of children attending hospitals to be suffering from syphilis. On the other hand, a recent investigation[1] amongst the newly born in the East End of London showed a positive Wassermann reaction only in 4 out of 677 infants, i.e. in 0·59 per cent.; a proportion which agrees curiously closely with that determined solely on clinical grounds as mentioned above.

A distinction has been drawn between inherited and congenital syphilis. Some writers would apply the term 'inherited' only to those cases in which the syphilis dates from the time of conception, whilst they would call 'congenital' only those in which the syphilis was transmitted at some later period of intra-uterine life, in other words, where the mother became infected during pregnancy. No doubt such a difference in the date of transmission does exist, but it is more than doubtful whether there exists any corresponding difference in the resulting manifestations of the disease, and in the absence of any special clinical significance for these terms it seems a pity to complicate the subject by what for all practical purposes is a distinction without a difference, so I shall speak indiscriminately of 'inherited' or 'congenital' syphilis.

[1] Report to Local Government Board by Dr. P. Fildes, 1915.

Transmission of Syphilis to Offspring

Perhaps one of the most striking features in the family history of syphilitic children is the frequent absence of any history of syphilis in the mother.

In past times various suggestions have been made to account for this seeming anomaly. The fact which is embodied in Colles's law that a mother who has shown no symptoms of syphilis may suckle her infant who has congenital syphilis without fear of becoming infected, whilst a wet nurse would be infected by the same infant, seems to show that the mother has somehow acquired immunity. It has been suggested (Coutts) that this immunity is derived from the syphilitic fœtus, which *pari passu* with its disease develops an antitoxin, and imparts this to its mother in sufficient quantity to confer immunity upon her. Such a view, however, assumes that the father can beget a syphilitic child without giving the disease to the mother. So far as clinical symptoms are concerned this undoubtedly is so, but in the light of the more recent methods of investigation it would seem that the mother of a syphilitic child, although she may never have shown a single symptom of syphilis, has nevertheless been infected, for her blood gives a positive reaction with the Wassermann test.

The various modes of transmission, classified according as symptoms of syphilis are present in one parent or in both, might be arranged in order of frequency thus: (1) father syphilitic, mother apparently healthy; (2) father and mother both show symptoms of syphilis; (3) mother syphilitic, father healthy; and (4) mother acquired syphilis during pregnancy. Does the mode of transmission make any difference to the disease in the infant, either in the character or in the severity of the symptoms? So far as my own observations go there would seem to be no corresponding difference, unless it be that when the mother has acquired syphilis after the time of conception the infant is apt to be affected more severely than in other cases; but even if this be so—and I do not feel at all sure of it—the difference may depend merely on the recency of the disease in the mother.

And this brings me to another question: Does the stage of the disease in the parents influence in any way the character of the disease in the offspring? At first sight it appears obvious that the later the stage of the disease in the parents the less the severity of the disease in the child. How common is a record of successive pregnancies like this: (1) miscarriage at $4\frac{1}{2}$ months; (2) miscarriage at $5\frac{1}{2}$ months; (3) eight months child, lived two hours; (4) boy with marked symptoms of congenital syphilis who survived; and (5) apparently healthy, now aged five months. Or this, where the mother

showed secondary symptoms of syphilis during her first pregnancy : (1) still-born ; (2) still-born ; (3) lived four days ; (4) lived 19 days ; and (5) idiot who survives with choroiditis and other marked symptoms of syphilis. Moreover, it has been shown that in the still-born or short-lived infants of syphilitic parents there are often severe syphilitic lesions which certainly cannot be present in the later infants who survive. It is, of course, possible that the increasing duration of successive pregnancies and the longer life of the fœtus may depend upon diminishing placental disease—i. e. the decreasing severity of the syphilis may be in the mother and in the maternal portion of the placenta, not in the fœtus ; but in face of the frequent occurrence of syphilitic lesions in the fœtus this explanation hardly seems probable for all cases. It must be admitted, however, that there are certain facts which are difficult to reconcile with any theory of progressive diminution in the intensity of the transmitted disease. The intensity of the disease may be greater in a later than in an earlier child ; occasionally the disease may even be absent in one child and reappear in a later child. The following history illustrates these points : (1) twin, premature, both still-born ; (2) survived, shows interstitial keratitis ; (3) (4) and (5) all miscarriages ; (6) apparently entirely free from syphilis; (7) and (8) still-born. I have no record of the subsequent children until (14) a girl, who came under my observation at six years and eight months with hæmoglobinuria, a condition which in children is usually associated with congenital syphilis. Even more curious are the cases in which one of a twin is healthy while the other has congenital syphilis. Several such cases have been recorded. One came under my own observation, an infant with severe and fatal syphilis who died at the age of seven months. His fellow-twin was apparently perfectly healthy.

Symptoms

Earliest appearance of symptoms. According to my own statistics in nearly 80 per cent. of cases of congenital syphilis the disease shows itself within two months after birth. The following table[1] shows this :

Date of First Appearance of Symptoms in 104 Cases.	
In first month	54
In second month	26
In third month	16
Between end of third month and end of sixth month	3
Between end of sixth month and end of twelfth month	3
One year to eighteen months	1
Eighteen months to two years	1

[1] Power and Murphy, *System of Syphilis.* Art. Congen. Syph.

The practical importance of these facts is appreciated when the question arises whether a woman can safely be engaged as a wet-nurse? As I have already mentioned, a woman may show no symptoms of syphilis although her child is undoubtedly syphilitic. How soon may we expect manifestations of the disease in the infant? It is clear from the above table that even at the age of three months there is still a possibility that symptoms may yet appear in the infant, but the likelihood is then small. Unfortunately it is often impracticable in this country to obtain a wet-nurse whose infant is as much as three months old, more often the woman who desires employment in this capacity wants to be employed as soon after her confinement as possible: it may be laid down as a general rule in the selection of a wet-nurse that it is best to obtain one whose child is not younger than two months old. As I have pointed out elsewhere, a discrepancy of one or two months between the age of the wet-nurse's child and the foster-child is of little importance, and certainly of far less moment than the risk of syphilitic infection.

Nowadays, however, the Wassermann test affords an additional safeguard in this important matter of the selection of a wet nurse.

I have assumed that the fact of syphilis in the infant indicates danger of infection from the mother to any healthy child she may wet-nurse; but, as already pointed out, it is possible that the infant may inherit syphilis though the mother shows no symptoms of the disease: admittedly in such cases her blood will give a positive reaction with the Wassermann test, but it is by no means certain that where this is the only evidence of the disease in a woman her milk can infect a foster-child. I know of no proof that it can, but I also know of no proof that it cannot: a few cases are on record which prove that a syphilitic woman may suckle a healthy infant without giving the disease. Mr. Lucas has recently published one such[1] but it would require a large number of these cases even to prove that infection was the exception; judging from the analogy of tubercle, it is probable that, rare though it may be, infection may be transmitted through the milk. Certainly where so much is at stake it would be unwise to take the risk.

In the series of cases tabulated above there happens to be no case illustrating the delayed manifestation of congenital syphilis which is sometimes called *syphilis hereditaria tarda*. I had under my care a girl who came under treatment at the age of $7\frac{1}{2}$ years for synovitis of both knee-joints; no symptoms of syphilis had been noticed until a month previously, when interstitial keratitis had appeared. In some of these cases no doubt earlier symptoms have been so slight that they have

been overlooked or forgotten, but in others where the child has been carefully watched there seems to have been no symptom until the later years of childhood.

Rarity of manifestations at birth. It is customary to state that inherited syphilis very rarely manifests itself at birth; Diday even questioned whether symptoms of syphilis were ever present at birth. Undoubtedly it is very seldom possible to make a positive diagnosis so early, but I suspect that symptoms which are seen in the light of subsequent events to have indicated syphilis are present at birth in a larger proportion of cases than is usually supposed. Perhaps the most characteristic is the so-called syphilitic pemphigus; this was present at birth in 2 out of 100 cases under my observation. Jaundice, the result of intercellular cirrhosis, is occasionally present at birth; snuffling was noted after careful inquiry as present on the day of birth in 11 per cent. of my cases; some of the changes in the fundus oculi, particularly choroido-retinitis, are almost certainly intra-uterine in origin, and therefore present at birth; enlargement of the liver and spleen are also present at birth in some cases.

A curious association of congenital syphilis may be mentioned here, for it is noticeable at birth in some cases, namely, an unusually abundant crop of hair, usually, I think, dark in colour. This was present in 4 per cent. of my cases and may perhaps be of some value as confirmatory evidence in conjunction with other symptoms; alone it certainly has no diagnostic value. Here also may be mentioned the so-called syphilis hæmorrhagica neonatorum, which, although not observed actually at birth, has usually appeared within a few days after birth. Some observers have thought that spontaneous hæmorrhages in the newly-born—e.g. from the stomach or bowel—are commonly due to congenital syphilis. I can only say that in my own experience such an association has been exceedingly rare; most cases of melæna neonatorum and other hæmorrhages in the newly-born are not in syphilitic families, and most syphilitic infants show no special tendency to hæmorrhages. There can be no doubt that the association exists, I have seen it myself more than once, and microscopic change, particularly endarteritis presumably syphilitic in origin, has been demonstrated in the small vessels in some fatal cases of melæna neonatorum; but none the less I think that the frequency of this association has been exaggerated and that it is actually very rare.

Order and frequency of symptoms. I shall consider the symptoms of congenital syphilis as far as possible in order of occurrence; but it will be understood that while some symptoms

usually occur during the first two or three months of life, and others later in the first or second year, there is no rigid sequence, and while it is useful for practical purposes to know how late or how early any particular symptom usually occurs, considerable variation is to be expected.

Marasmus. This may be mentioned first not only because it is noticeable in greater or less degree in a large majority of syphilitic infants, but also because in its gravest form it frequently dates, if not from birth, at any rate from a few days after birth. It has seemed to me that wasting occurs in two different forms in congenital syphilis; the first and commoner is the moderate degree of wasting which accompanies almost any illness in infancy. This is associated with well-marked manifestations of syphilis and ceases when the accompanying symptoms of syphilis subside; it scarcely influences prognosis. The second is a progressive marasmus which often begins before any other manifestations of syphilis have appeared, and which continues even when associated symptoms of the disease have disappeared completely under mercurial treatment; the wasting is independent of any fault in feeding or irregularity of the bowels, it may occur when the infant is breast-fed and free from vomiting and all other ordinary causes of marasmus; sometimes, and herein lies the urgency of diagnosis, it responds to treatment with mercurials, but more often, I think, it fails to respond to any treatment, and day after day the infant loses weight and after a few weeks, generally within three or four months after birth, he dies. In families where there is known to be syphilis in the parents, either from their own admission or from the occurrence of congenital syphilis in previous children, this grave form of marasmus may be recognized as due to syphilis before other manifestations are present, and in any infant under the age of three months who wastes persistently in the absence of the ordinary causes of marasmus, the possibility of congenital syphilis should be considered, for, as I have said, although mercurial treatment often fails to arrest the wasting in such cases, occasionally it will save the infant.

Snuffles. Snuffling occurs almost always within the first three months, usually within the first six weeks; it was present in 70 per cent. of my cases. The condition varies greatly in degree from a slight stuffiness of the nose up to a profuse discharge of pus, perhaps blood-stained, continually running from the nose, so that the infant cannot suck and is half asphyxiated if it tries to close its mouth; these severer degrees are, however, quite rare, and corresponding with this rarity is the fact that depression of the bridge of the nose results only in a small minority

of the cases which have snuffles, and even more rare as a result is ozæna. The depression of the bridge of the nose may be produced very rapidly; in an infant recently under my care with very severe snuffles the bridge of the nose had markedly fallen in at the age of twelve weeks, so that had the child lived he would have been permanently disfigured. Vigorous and early treatment, with local antiseptic applications by syringing, as well as internal mercurialization, might do much to prevent this nasal deformity.

I need hardly point out that infants, like older people, are subject to 'cold in the nose' and snuffling may be due to this simple coryza with no taint whatever of syphilis. Adenoids also are not very rare even in infants a few weeks old and may be associated with some catarrh causing snuffling respiration. There are also some forms of idiocy, particularly Mongolian imbecility and microcephaly, in which a small naso-pharynx with a special tendency to catarrh is responsible for snuffling and snorting respiration quite apart from syphilis.

Skin eruptions. These generally appear soon after the snuffles and almost always within the first three months. They are certainly very uncommon after the age of six months; in one of my cases the rash appeared at eight months and in another at one year. Some characteristic eruption was present in 69 per cent. of my cases. I shall not attempt to describe all the skin affections which have been observed in congenital syphilis; the most characteristic might be grouped as follows:

1. Macular, small reddish-brown, 'raw-ham-coloured' areas, generally roughly circular, but running together into irregular areas, dry and sometimes finely desquamating on the surface; occasionally these patches are yellowish-brown or fawn-coloured and have a dry, glazed surface. This eruption may be present on any part of the body but is particularly characteristic in its tendency to affect the middle third of the face, i.e. if the face be divided into three parts vertically, the middle portion including the chin and upper lip, the nose, and inner end of the eyebrows, is specially affected and sometimes the only part affected by the syphilitic rash.

2. Erythematous, diffuse in distribution and occurring specially about the perineum and down the inner side of the thigh and leg as far as the instep of the foot; the skin may be dry and glazed and at the edge of the affected area there are often outlying small round patches of similar character.

3. Desquamative; either of the preceding is sometimes associated with a little fine branny desquamation of the affected areas, but apart from these there is a profuse desquamation of the

palms and soles which, occurring in an infant under the age of three months, is very characteristic of congenital syphilis.

4. Fissures, usually radiating outwards from mucous surfaces, e.g. the mouth and anus; these generally occur within the first few weeks of life and are often deep enough to leave scars which last for the rest of life.

5. Bullous, the 'syphilitic pemphigus', which, as already mentioned, is present at birth or within the next few days.

6. Condylomatous; these moist warty-looking, flat-topped slightly raised patches usually of about the size of a threepenny-bit or smaller and situated in moist parts, for instance, about the anus or in the mouth, must be mentioned here, for although they differ from the other skin lesions of congenital syphilis in their tendency to appear and reappear long after infancy, they are, I think, most common in the first and second years of life. Lastly, I would draw attention to the frequent occurrence in syphilitic infants of skin affections which exactly resemble such ordinary non-syphilitic lesions as eczema and erythema intertrigo; for instance, behind the ears there is often a very ordinary eczema, whilst on the face there is a characteristic syphilitic rash in an infant who shows other well-marked evidence of the disease. Similarly a seborrhœic condition of the scalp or an ordinary erythema intertrigo about the groins and perineum frequently coexists with definite manifestations cutaneous or otherwise of congenital syphilis.

Whilst considering the skin lesions of this disease I must mention two symptoms which are very characteristic of inherited syphilis. I have already referred to the abundant crop of hair which is sometimes a noticeable feature at birth in the syphilitic; a few months later the opposite condition (a more frequent and much more characteristic symptom) is sometimes present (it occurred in 6 per cent. of my cases)—a thinning of the hair, so that it becomes sparse and scanty, never leaving complete baldness as in alopecia areata, but leaving so little hair that the baldness at once attracts notice and the more so as it commonly begins over the vertex and leaves for some time a fringe of thicker hair around the side of the head somewhat after the style of a monk's tonsure. The other symptom is onychia, seen usually in early infancy and, I think, usually associated with some cutaneous lesion, especially perhaps with the desquamative lesions; the nails of the fingers and sometimes of the toes become dry and shrivelled and discoloured and soon are shed altogether, leaving a small base of newly-formed nail which if the infant is under treatment with mercurials may speedily grow to a perfectly healthy nail.

Enlargement of the spleen. Widely varying statistics have been recorded on this point; some have placed the frequency as low as 10 per cent., some as high as 78 per cent. This variation probably depends in part upon difference of ideas as to what is to be called enlargement; if easy palpability be the standard, then 45 per cent. of my cases showed splenic enlargement, but including only cases in which the enlargement was at least one and a half fingers' breadth (about 1 inch) below the costal margin the proportion was 22 per cent. It is quite clear that absence of splenic enlargement weighs very little against the diagnosis of congenital syphilis, and mere palpability, I think, is of very little value as positive evidence, for the spleen is often to be felt in infants who are free from all suspicion of syphilis; when, however, the spleen extends an inch or more below the costal margin, especially in an infant under the age of six months, it has, I think, some value as confirmatory evidence.

The virus of syphilis, the spirochæte pallida, which must now be regarded as the specific organism, evidently exerts some irritative action on the spleen, for in autopsies on infants with congenital syphilis there is frequently seen a recent capsulitis, and in later stages patches of thickening of the capsule and some increase of connective tissue in the substance of the organ remain as evidence of past reaction; to these changes apparently the enlargement is due in some cases. Very rarely has any gummatous deposit been found in the spleen in children. I have elsewhere [1] recorded two cases (one associated with lardaceous disease) and collected four other published instances. In some cases the enlargement of the spleen has been secondary to a syphilitic cirrhosis of the liver.

Seeing that the peritoneum over the spleen sometimes shows distinct inflammatory change one might expect that occasionally a more general peritonitis would result, and this becomes more probable when it is admitted, as it is by some writers, that syphilis may cause peritonitis during intra-uterine life. By the kindness of Dr. A. E. Naish of Sheffield, I had the opportunity of examining the viscera of a still-born infant, whose birth had been hindered by fœtal ascites. The intestines were found to be matted together by adhesions; the liver showed intercellular cirrhosis. There was no evidence of syphilis in the family history, but the condition of the liver made such a cause at least probable. Of syphilis, however, as a cause of acute peritonitis after birth very little is known; one case probably of this nature has been described by West [2] in a syphilitic infant

[1] *Path. Soc. Trans.*, vol. xlviii.

[2] *Diseases of Infancy and Childhood*, 1865, p. 650.

whose abdomen became enlarged and extremely tender at the age of four weeks; but the peritonitis, if such it was, gradually subsided and the infant recovered. In an infant, aged four weeks, who was brought to me with a well-marked syphilitic rash, the abdomen became distended and the child died in about forty-eight hours from the onset of symptoms; autopsy showed a very acute peritonitis with turbid serum and lymph in the pelvis; in the liver there was very advanced intercellular cirrhosis. Whether the peritonitis in this case was solely due to syphilis or, like the suppuration which sometimes occurs in a syphilitic epiphysitis, was due to a superadded infection without any apparent source, is uncertain, but it seems at least possible that the association of acute peritonitis with syphilis was more than a coincidence.

Laryngitis is one of the most frequent of the early symptoms. It was present in 14 per cent. of my cases. It occurs usually soon after the onset of the snuffles or rash. A hoarse aphonic cry in an infant under the age of three months should always suggest the possibility of congenital syphilis. I have twice had the opportunity of examining the larynx in this condition after death: in one case at the age of four months, where the hoarseness had been very marked during life, the only change detected was slight thickening and roughening of the mucous membrane over the arytænoid cartilages; there was no change in the cords. In the other at the age of eight weeks there was much thickening of the epiglottis with some ulceration of the mucosa near its base; there was extensive ulceration of the vocal cords also, chiefly at the anterior part.

Epiphysitis was present in 11 per cent. Some observers have found it even more frequently, e.g. Scherer found it eleven times in fifty cases. The earliest onset in my cases was at two weeks, and six out of eleven began during the first three months (six out of Scherer's eleven cases also began under the age of three months). It is one of the causes, if not the only cause, of the so-called 'syphilitic pseudo-paralysis' and should always be suspected when an infant during the first half-year loses the use of one limb, especially the upper limb, without apparent injury. In my own cases the arms were affected alone in five, and with the legs in three, out of eleven cases.

The following is a typical example of this affection:

Herbert G., aged two months, began to scream fourteen days ago when his arms were moved, and seemed to have lost the use of both arms. He has had snuffles from birth, but has had no rash; some thickening is felt about the upper epiphysis of the left humerus and also about the upper epiphysis

of the radius in each forearm; there is no movement of the arms or forearms, the child cries if they are handled. The mother was treated ten months for syphilis about three years ago; she then gave birth to a child who had snuffles severely and died at five weeks; the patient was the next child. Inunction of mercury over the abdomen produced rapid diminution of the epiphysial thickening and also of tenderness, and when the infant was seen about a week after this treatment was begun, there was already slight movement of fingers and wrists, and a week later there was good movement of the upper limbs.

This syphilitic epiphysitis might be mistaken for infantile scurvy, but the latter disease rarely occurs under the age of six months and practically never under four months. Infantile paralysis also hardly occurs under the age of six months, and rheumatism can be excluded for it is almost unknown in infancy. The tenderness and pain on movement will usually suffice to eliminate a paralysis of cerebral origin which, moreover, is seldom limited to one limb as this syphilitic pseudo-paralysis usually is. The history of onset some weeks after birth serves to distinguish it from the palsy which results from damage to the brachial plexus during birth. The diagnosis is important, for with mercurial treatment the pseudo-paralysis disappears completely in a week or two; local treatment is seldom necessary, beyond protection of the flaccid limb from mechanical injury. But if the syphilitic origin of the condition is overlooked, apart from the suffering entailed to the infant, there is a risk of separation of the epiphysis and also of suppuration, which I have seen occur about the affected epiphysis with undoubted syphilis, perhaps from superadded pyogenic infection of the damaged tissue.

Dactylitis is less frequent than epiphysitis, if indeed it is to be distinguished therefrom; some observers have thought it was an epiphysitis, others have rather described it as a periostitis or an osteomyelitis. It was present only in 2 per cent. of my cases. Out of nine which I collected seven were under one year, including five which occurred at four weeks; none were over two years of age.

The fingers are more often affected than the toes and usually more than one finger is affected. There is a fusiform swelling usually of the proximal phalanx. Dr. Carpenter in his monograph on congenital syphilis mentions affection of the metacarpal bones in one case, and I have seen one similar case.

The condition can hardly escape observation, the difficulty is to distinguish between syphilitic and tuberculous dactylitis. It has been said that the syphilitic tends to be symmetrical in the two hands, but this was not apparent from the nine cases I have mentioned nor do I know of any reliable point of distinction; the age-incidence will sometimes assist, for tubercle is

excessively rare under the age of three months and by no means common under the age of six months, whereas most of the syphilitic lesions of infancy including dactylitis are almost as likely to occur under three months of age as later ; the diagnosis must usually depend upon the presence of other symptoms of syphilis which are seldom lacking.

Orchitis, present in five out of sixty-four males, i. e. in nearly 8 per cent. of my cases, is another symptom which in early infancy is almost pathognomonic of congenital syphilis. The testes are enlarged to two or three times the normal size, are very hard and quite free from tenderness. The condition may easily be overlooked unless specially sought ; its detection is important, not only as an aid to diagnosis but also because early administration of mercury may prevent loss of function in these organs. It differs from tuberculous disease not only in affecting the body of the testis first and chiefly, whilst the epididymis is seldom appreciably affected, but also in being more often a bilateral affection. In three out of my five cases it began at the age of twelve weeks, a time at which tuberculosis of any sort and especially of the reproductive organs is very rare. The orchitis usually begins under the age of five months.

Brain affections and indeed nervous affections of any sort are usually stated to be very uncommon in congenital syphilis. According to my own figures 10 per cent. of children up to the age of twelve years with this disease showed cerebral affection of one kind or another, and even if only infants were included the proportion seemed to be nearly as high.

Of fifteen cases with well-marked congenital syphilis and affections of the brain, seven were cases of progressive mental degeneration (i. e. juvenile general paralysis) commencing at about five to eight years of age ; three were idiots from birth (one of these three was markedly microcephalic); one was hydrocephalic, and one hemiplegic. It seems likely that even 10 per cent. is below the actual proportion, for I have not included some cases of cerebral palsy in which the history suggested the likelihood of congenital syphilis without sufficient evidence to make it certain. It is recognized also that some children with congenital syphilis become epileptic in the later years of childhood, and I would add that it is not uncommon for syphilitic infants to die with convulsions shortly after birth. The relation of syphilis to mental deficiency I have already considered (pp. 594, 597).

It has been customary in textbooks to mention congenital syphilis as a cause of meningitis in infancy ; but if by meningitis is meant a simple inflammation of the pia-arachnoid—I mean

as distinct from the pachymeningitis with cortical sclerosis which is found in cases of juvenile general paralysis—then meningitis is certainly of extreme rarity in congenital syphilis. There can be little doubt that what was formerly described as a chronic basal meningitis due to syphilis in infants was identical with the disease which we now know as posterior basic meningitis or cerebro-spinal meningitis and which, however chronic it may be, is the result of infection with known micro-organisms and has no connexion with syphilis, although there is of course no reason why it should not occur in a syphilitic infant. I have never seen such an association myself. But apart from this posterior basic meningitis which, as is now generally admitted, has nothing to do with syphilis, is there any form of lepto-meningitis (inflammation of the pia-arachnoid) which can be attributed to inherited or congenital syphilis ? Cases have been recorded in which syphilitic infants with microcephaly have shown thickening and adhesions of the pia mater—sometimes at the vertex, sometimes at the base—evidently the result of meningitis during intra-uterine life. I have more than once found adhesions and opaque thickening of the pia-arachnoid about the medulla and under surface of the cerebellum in cases of congenital hydrocephalus where it seemed clear that there must have been meningitis before birth. In some such cases it seems certain that syphilis is the cause, in others it can only be suspected.

Sir Thomas Barlow[1] has recorded a case in which old thickening and calcification of the pia mater was found with arteritis and choroiditis in a syphilitic infant aged ten months, but here there was no history of definite meningitic symptoms at any time and the date of the inflammation was quite uncertain. In a syphilitic boy aged $10\frac{11}{12}$, who died after thirteen days of acute symptoms, I found a chronic inflammation of the pia mater which was thick and opaque over parts of the vertex and showed some thickening with adhesions in the Sylvian fissures and between the medulla and cerebellum ; there was also some opacity of the membranes down the posterior surface of the spinal cord ; this case showed also some sclerosis of the cortex and comes into the category of juvenile general paralysis (see p. 595, where I have considered this case more fully).

Eye affections. A striking feature in all the nervous affections of congenital syphilis and to a less extent with other manifestations of this disease in children is the frequency of syphilitic eye changes ; in at least 21 per cent. of cases with or without

[1] *Path. Soc. Trans.*, vol. xxviii, p. 287.

nervous manifestations some syphilitic eye affection was present; in 12 per cent. there was choroiditis, and these figures no doubt understate the frequency, for the point was not specially noted in all my series. With the nervous affections they are much more frequent, ten out of fifteen cases with cerebral manifestations showed some eye affection which in nine out of the ten was a choroiditis or choroido-retinitis; the remaining one showed only keratitis. These statistics, although they deal only with small numbers, may serve to emphasize the value of ophthalmoscopic examination in the diagnosis of congenital syphilis. In view of the probable intra-uterine onset of the eye changes in some cases, it seems likely that in cases where from the known presence of syphilis in the parents or in the other children syphilis is suspected in an infant, examination of the fundus oculi may be of value in determining whether antisyphilitic measures should be adopted before other symptoms have appeared: nine out of sixty syphilitic infants in the first year of life showed iritis or choroiditis.

A very characteristic affection is interstitial keratitis, which is often associated with the Hutchinsonian type of permanent teeth, and begins most often between six and twelve years of age. In most cases it begins in one eye several weeks before the other is affected, but I have seen both affected within a week. The haziness of the cornea, with spots of opacity in its substance, and the fine blood-vessels ramifying over it and passing deeply into the cornea, afford very positive evidence of syphilis. Sight is almost completely lost in some cases for many weeks or months owing to the dense opacity of the cornea; but the prognosis is much better than appearances would suggest, for with free administration of iodide and mercury internally and the use of an ointment of the yellow oxide of mercury locally the opacity gradually diminishes, and if the cornea does not become quite clear as it may do in a mild case, it at least becomes sufficiently clear to allow of good vision.

Anæmia of a very profound degree is sometimes a result of congenital syphilis, most often, I think, towards the end of the first or in the second year. This anæmia may occur after all other manifestations of the disease have disappeared, so that at the time it is the only indication of syphilis. This was impressed upon me some time ago by the case of an infant whom I had treated for syphilis at the age of five months; he passed out of my observation for a time and at the age of about eighteen months he was brought again with profound anæmia. I had forgotten the previous symptoms (syphilitic epiphysitis and

condylomata) and was in great doubt as to the cause of the anæmia, which was the only symptom except some diffuse thickening of the epithelium of the tongue. The anæmia remained a puzzle until after some months large condylomata appeared at the anus.

The extreme pallor of the face with a brownish-yellow tinge, especially about the nose and cheeks, is very striking, and may even in the absence of other symptoms suggest syphilis, but it is certainly not pathognomonic. This anæmia of congenital syphilis is not associated usually with any special enlargement of the spleen beyond the moderate degree which is common in this disease, and herein lies one difference from the so-called 'splenic anæmia', the relation of which to congenital syphilis is still *sub judice*, and in which enormous enlargement of the spleen is a characteristic symptom. Examination of the blood shows nothing to distinguish this profound anæmia of congenital syphilis from profound anæmia secondary to other conditions, for instance, rickets.

As in the following case there is often a marked diminution of hæmoglobin, but poikilocytosis is uncommon :

Doris G., aged 2½ years, was one of a twin, and had been preceded by two miscarriages; there were marked Parrot's nodes, and the yellowish pallor suggested syphilis. The blood showed hæmoglobin 40 per cent., red blood corpuscles 3,032,000 per cub. cm., white corpuscles 15,500 per cub. cm. Differential count of white corpuscles showed polymorphonuclears 61 per cent., large mononuclear 2·5 per cent., small lymphocytes 32 per cent., large lymphocytes 3 per cent., eosinophiles 1·5 per cent.

Whether a study of the blood will draw any hard-and-fast line between this syphilitic anæmia and the so-called 'splenic anæmia of infants' is still a moot point. Undoubtedly the blood in the latter group of cases presents features which might suggest a disease *sui generis*, but the very close association between this 'splenic anæmia' and congenital syphilis and rickets must, I think, make us very cautious in accepting any such view. The *café-au-lait* tint which is sometimes described as characteristic of congenital syphilis differs considerably from the yellowish anæmia. The predominating tint is brown rather than yellow, and it is seen not in well-nourished infants as the anæmia is, but almost always in puny, emaciated infants and at an earlier age than that at which the anæmia usually occurs.

Parrot's nodes and craniotabes. The relation of these to congenital syphilis has been much disputed. By some they have been regarded as indubitable evidence of syphilis, by others as a product of syphilis and rickets combined, and by others again

as due to rickets alone. If I may simply state my own opinion, formed as the result of some special observation on this point, I should say that neither of these symptoms can be taken as evidence of syphilis; that both of them are found far more often without syphilis than with it; that both of them may be produced by rickets alone; but that both of them are occasionally found with congenital syphilis apart from rickets. Of 100 cases of congenital syphilis under my observation only 2 showed Parrot's nodes and both these showed rickets also. I think it is quite possible that under the name 'Parrot's nodes'

FIG. 53. Parrot's nodes. Photograph of skull, showing characteristic Parrot's nodes. In this case there was roughening of the outer surface over the bosses, which had a honeycomb appearance: the child had both rickets and congenital syphilis.

two conditions structurally different though resembling one another clinically are confused together, one being the result of syphilis and the other of rickets; at any rate I can affirm from my own observation that these bosses on the skull show anatomical differences which seem to support this view.

In some cases the thickening is found to be due to a very vascular thickening of the diploe, while the outer surface of the thickened part remains normally smooth; in other cases there is deposit of new bone on the outside of the skull which has a rough honeycomb appearance. The latter condition is certainly

found in syphilitic infants. I do not know that it is ever found in pure rickets. In either case the bossing is usually, but not always, symmetrical, affecting the parietal and frontal bones near the anterior fontanelle, and so producing the natiform or hot-cross-bun skull.

Craniotabes has seemed to me to be very rarely due to syphilis (see p. 97); indeed, in examining the heads of syphilitic infants for craniotabes I have been struck with the firmness of the bones, which have often seemed to become firm and hard rather earlier than in infants who showed no evidence of syphilis; in my own experience craniotabes has been associated most often with laryngismus stridulus and tetany, which are both recognized as rachitic manifestations, and in most of these cases there has been no suspicion of syphilis. There is no doubt that congenital syphilis may cause extensive absorption of the bones of the skull as in the following case:

Edward H., aged ten months, was brought for a rash on the face, buttocks, and head. He was slightly hoarse, and had condylomata about the anus; the fundus oculi showed choroidoretinitis; but the remarkable feature of the case was the presence just in front of the frontal eminences of two symmetrical gaps, one in each frontal bone, triangular in shape—the base of the triangle being above—and measuring about one inch in the transverse line of the forehead, whilst the distance from base to apex (which reached nearly halfway down the forehead) was also about one inch. The bone appeared to be entirely absent in these areas, and I concluded—wrongly, as events proved—that it was an unusual example of congenital defect of ossification. The child was treated with mercury and iodide for the other syphilitic manifestations, and in the course of a few weeks it was found that the holes in the frontal bones had completely disappeared, being filled up with hard thick bone with a somewhat uneven surface. No swelling of any sort had been noticed over these areas before the absorption of the bone; so that it was unlikely that it had been preceded by any gummatous deposit.

Nephritis. Less common than any of the affections already mentioned is nephritis as a result of congenital syphilis. Several such cases have recently been recorded by Dr. Carpenter, Dr. Sutherland, Dr. Walker, and others. I have had four syphilitic children with nephritis under my observation. One developed general œdema at the age of six weeks and died six weeks later; the kidney showed acute nephritis. Another showed symptoms of nephritis at the age of three years and three months, became dropsical with ascites and died: intercellular cirrhosis of the liver was found and an acute nephritis chiefly of interstitial character.

A third came under treatment at the age of three months with 'pseudo-paralysis' of one arm, enlarged spleen, anæmia,

dropsy, and albuminuria. The epiphysitis rapidly cleared up under mercury, but the child died shortly afterwards of suppurative meningitis, and the kidney showed acute parenchymatous nephritis. The fourth case was a girl aged nine years who had been under the care of Dr. Thursfield with hæmaturia and interstitial keratitis, the left kidney was thought to be unduly palpable; under iodide treatment the urine became free from blood but albuminuria continued and many casts were found in the urine.

In five out of ten cases in which the character of the nephritis has been noted, the inflammation was chiefly or entirely interstitial: a fact which has suggested that the rare cases in which chronic interstitial nephritis is found in childhood may possibly in some instances be explained by congenital syphilis.

Teeth. Passing now to the symptoms which occur in later childhood I must mention the characteristic changes in the teeth which were described by Sir Jonathan Hutchinson. The upper central incisors are chiefly affected, they are unduly small and, partly in consequence of this, appear widely separated. They are also abnormal in shape, the cutting edge is usually narrower than the base and its corners are round instead of sharply angular; most characteristic is the edge, which shows a crescentic notch, as shown in the accompanying photograph (Fig. 54) of a boy aged ten, who had also disseminate choroiditis.

Similar changes are present sometimes but in less marked degree in the lower central incisors. The first molars sometimes show a characteristic but less obvious change, being small and dome-shaped owing to defective development of the cusps. I have not found these dental changes to be frequent, but when they do occur they are of great value in the recognition of syphilis in later childhood.

Is the primary dentition affected in any way by congenital syphilis? It has been stated positively that the characteristic teeth of congenital syphilis are seen only in the permanent dentition, never in the temporary set. I venture to think that like most rules this one also has its exceptions. A male infant, aged fourteen months, was brought to me at the Hospital for Sick Children, Great Ormond Street, for screaming and colic. His central upper incisors were both of typical screwdriver shape, narrower at the edge than at the base; they showed at the cutting edge a well-marked shallow crescentic notch, and in addition showed that undue separation from one another which is common in syphilitic teeth. I do not think that a more characteristic specimen of syphilitic teeth could well be found; there was no

other evidence of syphilis in the child, but the family history was suggestive—(1) still-born at sixth month ; (2) premature, eight months' child, lived nine weeks ; (3) seven months' child, deaf and dumb ; (4) patient. Smale and Colyer, in their treatise on

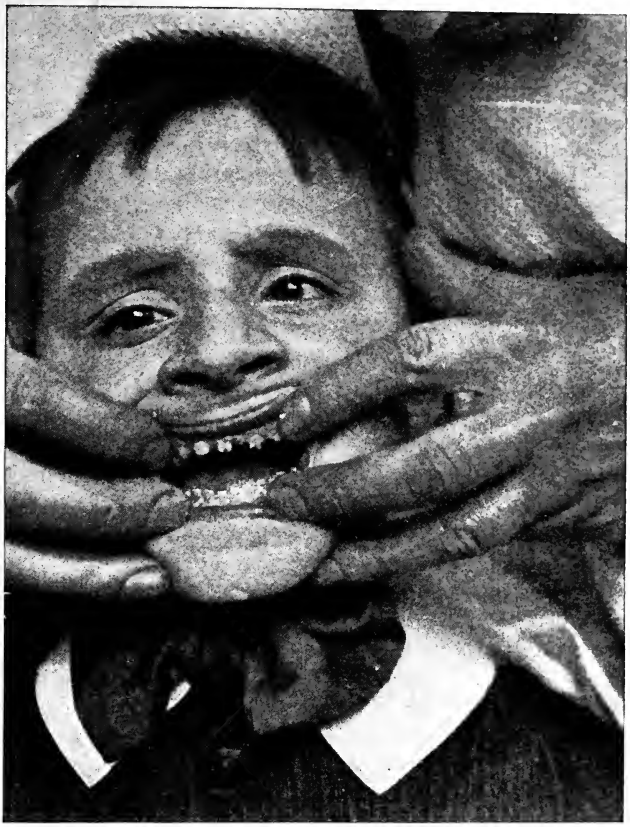

Fig. 54. Notched upper incisors in congenital syphilis (Hutchinsonian teeth). Photograph shows characteristic dwarfing and notching of the upper central incisors, which are also widely separated from the lateral incisors and especially from each other. The lower central incisors showed a much shallower notch at their cutting edge. The boy had interstitial keratitis.

Diseases and Injuries of the Teeth, refer to a case recorded by Mr. Oakley Coles,[1] of 'peg-shaped temporary teeth which were very characteristic, occurring in a child the mother of whom had long been the subject of syphilis '.

[1] *Trans. Odont. Soc.*, vol. ix, p. 258.

Apart from these extremely rare affections of the milk-teeth, it has been thought that syphilis has some influence upon the date of appearance of the temporary teeth, but opinions are divided, some have thought dentition was delayed and others that it was hastened. In some cases certainly the first teeth appear rather earlier than usual, and in view of the fact, which I think is indisputable, that the skull is often more firmly ossified than usual in the early infancy of a syphilitic child, one would rather expect that dentition would occur early. The exact influence of syphilis in this respect is difficult to determine, for syphilis is complicated in a very large proportion of cases by rickets.

Periostitis. In the limbs and joints during the later half of childhood from six years of age till puberty there are sometimes very characteristic syphilitic affections. A chronic periostitis is not very rare, especially in the tibia, affecting the diaphysis, and producing a thickening which gives the bone the appearance of convexity forward and has thus given rise to the term '*sabre tibia*'.

In some cases the bone is also affected, becoming inflamed and soft so that it bends, but I think this is quite the exception, the convex appearance is more often due to periosteal thickening only. The periostitis apparently causes but little pain; in some of my cases there have been vague aching pains and there has been some slight tenderness on pressure.

I have thought that this condition was specially frequent with the mental affections of congenital syphilis; for instance, four children with syphilitic mental degeneration (juvenile general paralysis) all showed periostitis of the tibia.

Joint affections. Joint-lesions are not very rare in congenital syphilis; a symmetrical synovitis was present in the knees in three of the 100 cases mentioned above, and one of these three showed also symmetrical synovitis of the ankles. But more obscure in its pathology is a curious affection of the joints which closely simulates osteo-arthritis. I have notes of four such cases; all were boys between the ages of five and twelve years, and in all there was limitation of movement in many joints, with some thickening about the joints, which in some certainly appeared to be due to osteophytic change; but there was also some thickening of soft tissues, probably of the capsule of the joint, and in one case at least there were probably fibrous adhesions in the joints. There was no enlargement of lymphatic glands, and in three of the cases the spleen could not be felt. In all the four cases there was some syphilitic eye affection; three showed interstitial

keratitis ; one showed patches of choroidal atrophy. The joint affection does not appear to be continuously progressive : the joints became affected successively during a period extending over several weeks or months, but apparently before anti-syphilitic treatment had been commenced they had already become quiescent, leaving the movements of the joints more or less impaired. The thickening about the joints seemed to diminish very slowly, being still noticeable two or three years after the onset of the affection.

Enlargement of lymphatic glands. A point of some practical importance is the relation of enlargement of lymphatic glands to congenital syphilis. It is not uncommon in children with this disease to find many of the superficial glands slightly enlarged so that they are easily palpable, but there is nothing in such a condition to differentiate it from similar palpability of glands which is often noticeable, especially in poorly nourished children, without any obvious cause and without any taint of syphilis ; but there seems to be no doubt that occasionally a considerable local enlargement of glands, which might easily be mistaken for tubercular enlargement, occurs as a result of congenital syphilis, and that without any local source of irritation. Holt states that in his experience this adenitis is usually a late manifestation of congenital syphilis. Hutchinson [1] refers to one such case where the cervical glands became greatly enlarged in a girl of fifteen years with interstitial keratitis, and the enlargement rapidly subsided under treatment with potassium iodide. In a boy aged eight years who was under treatment for several years at the Hospital for Sick Children, Great Ormond Street, for various syphilitic manifestations, including interstitial keratitis and gummata in the liver, there appeared at one time considerable enlargement of glands forming a visible tumour in one groin and palpable also in the iliac fossa ; this subsided very rapidly on treatment with potassium iodide and mercury internally : there was no apparent local cause for the adenitis.

Deafness. Lastly, and I put it last because it is usually the latest to appear of the symptoms of congenital syphilis, there is the affection of the ears which is specially characteristic of this disease, and which is perhaps due to an insidious inflammation of the internal ear : the patient becomes rapidly deaf so that hearing is completely lost within a few weeks. This affection belongs rather to adolescence than to childhood for it begins usually after puberty.

[1] *Syphilis*, p. 450.

Contagion in Inherited Syphilis

I turn now to a very important practical point, the contagiousness of inherited syphilis: Is there any risk that the infant may give the disease to any one else? I have already referred to Colles's law, which emphasizes the extremely important fact that a mother can suckle her child who has congenital syphilis without risk of infection. It is hardly possible to exaggerate the importance of this, for the life of a syphilitic infant often depends upon the continuance of breast-feeding; the special frequency of gastro-intestinal disorders and marasmus in these infants makes it a matter of vital importance that the mother should suckle her child if possible for the full nine months. A few cases have been recorded in which a syphilitic infant was supposed to have infected the mother, but in some of these there was a doubt whether the infant might not have had acquired syphilis, and in any case the number is so small that such infection must be excessively rare and the risk is so infinitesimal that it can hardly be taken into account when the difference which suckling makes in the chances of the infant's survival is considered. But Colles's law, be it remembered, states also that the infant with inherited syphilis, although he does not infect his mother, will infect a wet-nurse; and this raises the broad question as to the contagiousness of inherited syphilis. On this point there is a wide difference of opinion. 'The lesions of congenital syphilis,' says Diday, 'differ from those of ordinary syphilis by an infinitely greater power of contagion. And what is at the same time remarkable and deleterious in them is, that they transmit this same property to the person who receives the disease through them.' A modern writer has even stated that 'Hundreds and thousands of cases of innocent syphilis have had their origin in innocent babes'.

On the other hand, Dr. J. A. Coutts in his Hunterian lectures on infantile syphilis says, after excepting one doubtful case, 'I have personally never known an instance where syphilis was contracted from an infant with the inherited complaint, and this in spite of many thousands of healthy children and adults having been exposed to infection from cases under my care.' He mentions also that as a result of special inquiry from eighteen medical men connected with children's hospitals he could hear of only eight cases of supposed infection from children with congenital syphilis, and four of these eight cases took the form

of exceptions to Colles's law and might therefore by some be rejected as unreliable. In my own practice I have not met with a single case of infection from an infant with inherited syphilis. Dr. W. R. Grove of St. Ives, Huntingdon, showed me a family in which such infection apparently occurred. I shall quote the case from his own record,[1] as it is of importance in emphasizing the possible danger.

An infant who had had snuffles a few days after birth, and whose mother was known to have had syphilis, developed condylomata at the anus and some sores in the mouth at the age of seventeen months. About a month later the grandmother, in whose care the infant had been left, developed two primary sores of syphilis, one on the lower lip, the other on the tonsil, and shortly afterwards showed a syphilitic rash. About the same time a daughter aged twelve years, and shortly afterwards a son aged seven years, developed a primary sore on the tonsil followed very soon by a syphilitic rash. The baby had been fed with a spoon with which the grandmother and her granddaughter had tasted the food from time to time during the feeding of the infant, and it was supposed that the grandson also had used the spoon unwashed.

I have heard of two other cases in which infection (not of the mother) seemed to have occurred, and I am entirely in agreement with Dr. Coutts's conclusion, 'that inherited syphilis is occasionally contagious is certain, but it is equally certain that the virulence of such contagion has been grossly and vastly overestimated.'

The practical outcome of these observations is clear; it is only right to warn parents that such a danger does exist, especially so long as syphilitic skin lesions, condylomata, or other specific affections of mucous membranes are present. Kissing of the syphilitic infant should be avoided altogether, and those who must handle the child should wash their hands carefully after each handling; the filthy habit of moistening the 'comforter' or 'teat' in the mother's own mouth before giving it to the infant, objectionable and dangerous as it is in any circumstances, becomes even more so when children, following the example of their elders, do the same for the syphilitic infant. A mother, whose little girl aged about eight years was born before syphilis appeared in the family, told me that this child frequently took the teat from the syphilitic baby's mouth and moistened it afresh in her own. Such a practice should certainly be forbidden.

[1] *Brit. Med. Journ.*, 1906, vol. i, p. 1400.

Treatment

The treatment of congenital syphilis is simple enough so far as choice of drugs is concerned; indeed, if the early manifestations only are to be considered there is no choice—mercury is the one essential. There are, however, two points which require consideration in the use of this drug, the mode of administration and its duration.

The simplest and most convenient method in the large majority of cases is, I think, to give mercury by the mouth as grey powder. With this, as with any other form of mercury, the risk of producing diarrhœa in an infant who is perhaps already puny and wasted, as so many syphilitic infants are, has to be remembered; but it is, I think, avoided to some extent by giving small doses rather than the large doses recommended by some writers. Half a grain three times a day with a grain of Sod. Bicarb. and 2 grains of Pulv. Cretæ Aromat. can usually be given safely to an infant a few weeks old without causing diarrhœa, and this dose is quite sufficient in many cases to cause a steady subsidence of symptoms. If there is a tendency to diarrhœa the combination of grey powder with $\frac{1}{8}$ to $\frac{1}{2}$ grain of pulv. ipecac. co., according to the age of the infant, may be used. Sometimes, where $\frac{1}{2}$-grain doses of the mercurial have been sufficient to cause the symptoms of syphilis to disappear, fresh manifestations of the disease have appeared subsequently although the infant was still taking the drug regularly. In such cases the dose may be increased to 1 grain three times a day, or, as suggested below, a combination of the internal with the external administration of mercury may be more useful.

Whether such small doses of grey powder are likely to affect the teeth subsequently, is, I think, very doubtful; but the possibility of such a result is at any rate a reason for using the smallest dose which is effectual. The risk of salivation or of stomatitis from mercury is very small in infants; I have known it to occur only two or three times amongst a very large number of infants to whom mercury was given for one cause or another.

As an alternative—and one may be wanted, for there are infants who will vomit powders of any sort—the liquor hydrargyri perchloridi is sometimes useful, and may be given in doses of 3 to 5 minims three or four times a day to an infant a few weeks old, and in doses of 10 minims or even more for an infant

of six to twelve months. My own experience of this drug is that it produces looseness of the bowels more often than the grey powder does, and for this reason is less satisfactory. To prevent this it may be combined with small doses of castor oil, as in the following formula: Liq. Hyd. Perchlor. ℥v, Ol. Ricini ℥iv, Mucilag. Acaciæ ℥xv, Aq. Anethi ad. ʒi, ter die, for an infant two months old; and, if necessary, Tinct. Camphoræ Co. in minute doses may be added.

Some observers have recommended calomel in doses of $\frac{1}{12}$ to $\frac{1}{6}$ grain twice or three times in the twenty-four hours, as a more rapidly effective form of mercury than grey powder; its tendency to cause diarrhœa, however, is greater, and will probably necessitate the use of opium in some form.

Where rapidity of action is very important, as in the very severe cases where an infant appears likely to succumb speedily to the intensity of the syphilitic virus, a combination of internal administration of grey powder with external administration by inunction of unguentum hydrargyri has seemed to me better than large or very frequent doses of the drug internally. I usually order a piece of the ointment the size of 'the tip of the little finger', or of 'an average green pea', which corresponds roughly to 15 grains; this is gently rubbed over the abdomen or the inside of the arm or thigh each night, and left covered with a flannel bandage till the next morning, when it is thoroughly washed off. The risk of dermatitis from inunction—albeit a small one, especially if the skin is properly cleansed after the application, and the area chosen is varied from day to day—makes this method, I think, less suitable as a routine treatment than the use of grey powder. As a cleaner method of external application warm baths containing about 10 grains of perchloride of mercury in about 2 gallons of water have been recommended. I have never used this method, which seems open to objection on account of the large quantities of corrosive sublimate which must be entrusted to unskilled and sometimes careless persons. No method of external application seems to allow of so accurate a control of dosage as the administration by mouth.

I shall make no reference to the intramuscular injections of mercury and intravenous injections of iodine which have been used by some in congenital syphilis, except to state that in my opinion such methods are unnecessary in the syphilis of childhood, for by the combination of inunction and oral administration it is easy to get a child very speedily under the influence

of mercury to any desired extent. Methods which involve pain and risk of inflammatory complications are always to be avoided, especially for children, if the desired result can be obtained otherwise.

These objections apply equally to the treatment of congenital syphilis with '606', salvarsan. At present the number of cases in which this treatment has been adopted is too small to justify any general conclusions as to its value in the congenital form of the disease. Cases have been reported in which injection of this drug into the suckling mother was followed by rapid disappearance of symptoms of syphilis in her breast-fed infant. These indirect methods of treatment through the mother are always highly unsatisfactory, as they render it impossible to regulate with any accuracy the dose received by the infant.

If salvarsan is to be used at all in congenital syphilis it will usually be advisable to administer it directly to the infant, and for this purpose doses varying from $\frac{1}{3}$–1 grain (0·02–0·06 grm.) have been given: to older children 2–3 grains may be given.

The intramuscular injection of salvarsan into an infant is certainly not without danger. Necrosis of the gluteus muscle has occurred more than once with fatal result when the injection has been made in this part. Other fatal cases have been recorded, and in view of the harmful results which have already been recorded from its use in adults, and the now ascertained fact that relapses may occur in spite of this treatment, it hardly seems expedient to use so painful a procedure when we have already an efficient remedy in the simple oral administration or inunction of mercury.

Intravenous injection of neo-salvarsan has been recommended as preferable to the intramuscular method. A sterilized saline solution containing $\frac{3}{4}$ grain in one drachm is injected slowly into a vein in the arm or in the scalp. The injection may have to be repeated several times at intervals of about a week, and the dose may be increased up to 2 or 3 grains. In this way it is claimed that the Wassermann reaction can be rendered negative, and the symptoms of syphilis rapidly disappear. It is not denied that vomiting, rigors, cyanosis, and even coma and death may result from the intravenous injection, but, apart from such effects, the pain and terror involved can only be justified if the beneficial results are not to be obtained by any less distressing method of treatment. Certainly in the large majority of cases of congenital syphilis the simple treatment with mercury by mouth or skin effects a cure without the

slightest distress to the child. The duration of treatment is much longer, it is true, but most parents would prefer this inconvenience to the risks and suffering involved in the salvarsan or neo-salvarsan treatment.

The place of iodides in the treatment of congenital syphilis is a question of some importance. It is sometimes taught that mercury should be given to the infant and iodide to the older child in congenital syphilis, on the general ground, no doubt, that the earlier symptoms are usually of 'secondary', the later of 'tertiary' character. The two stages, however, do not necessarily correspond with any age limit, and there are certainly many cases in which potassium iodide is just as necessary for an infant as for an older child. Gummata are sometimes present in early infancy, and some of the bone-lesions, epiphysitis and periostitis, are, I think, often more successfully treated by a combination of iodide-treatment with mercury than with the mercurial alone. Potassium iodide is taken well by children, and in some of the lesions of later childhood where there is no response to small doses, the dose may be increased until 15 or 20 grains or even more are taken three or four times a day.

The duration of treatment with mercury is a point on which there is some difference of opinion.

Great stress has been laid on the injury to teeth which results from prolonged administration of mercury during infancy; it has been recommended not to continue the treatment much beyond the disappearance of symptoms. But in some cases this might mean administration of mercury for three or four weeks only, and so short a course of treatment is certainly not sufficient to prevent a recurrence of symptoms. I have seen fresh manifestations of syphilis occur in an infant after a very short interval each time the mercury was stopped in this way. A regular administration of mercury to the syphilitic infant for at least twelve months and if circumstances allow for eighteen months, even where all symptoms are in abeyance after the first few weeks of the treatment, seems to be the most satisfactory routine.

Another matter to which reference must be made is the feeding of the syphilitic infant; and here I should like to insist again upon the vital importance of breast-feeding for these infants.

There seems to be a dread in the minds of some medical men that a mother may be infected by her own child when the child has congenital syphilis and the mother shows no sign of the disease; and I have sometimes found that a mother had been advised to stop suckling at once when it was found that the

child had syphilis. Even if it be taken as proved—and the evidence is so slight that it can hardly be called proof—that exceptions have occurred to Colles's law that a mother cannot be infected by her infant when it has inherited syphilis, these exceptions must be so exceedingly rare that for practical purposes they may safely be disregarded. Moreover, there must be weighed against such an extremely small and doubtful risk the very real and great difference which breast-feeding makes in a syphilitic infant's chances of survival. Amongst the causes of death in congenital syphilis during infancy, one of the most frequent is marasmus : it would almost seem as if there were some inherent difficulty of assimilation in these infants at birth ; some of them waste almost from the day they are born, although no other evidence of syphilis may be apparent until wasting is already advanced ; and the marasmus often occurs without any obvious gastro-intestinal disturbance in an infant who is properly fed at the breast. Apart, however, from this tendency to marasmus, there seems to be a special liability also to gastro-intestinal disorders ; a large proportion of the infants who come under treatment for congenital syphilis are brought, not for any specific manifestations, but for persistent digestive disorder in spite of careful feeding. It is therefore all-important that the food should be as easy of assimilation as possible, and for this reason no method of artificial feeding can replace the mother's milk. To deprive the syphilitic infant of his mother's milk may be to deprive him of his chance of life. Suckling, however, can only be done by the mother : no wet-nurse must be allowed to suckle an infant with congenital syphilis, for however rare infection from congenital syphilis may be, it seems clear that a wet-nurse can be, and in several cases has been, infected by suckling an infant who had this disease.

Lastly, I would point out that apart from the marasmus to which I have referred, not only infants but also older children with congenital syphilis are so often poorly-nourished, pale and ' unwholesome-looking ' even when free from any definite manifestation of the disease, that good food, and perhaps malt and cod-liver oil, sea-air and good hygiene in general, play no unimportant part in the treatment of syphilis in children.

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