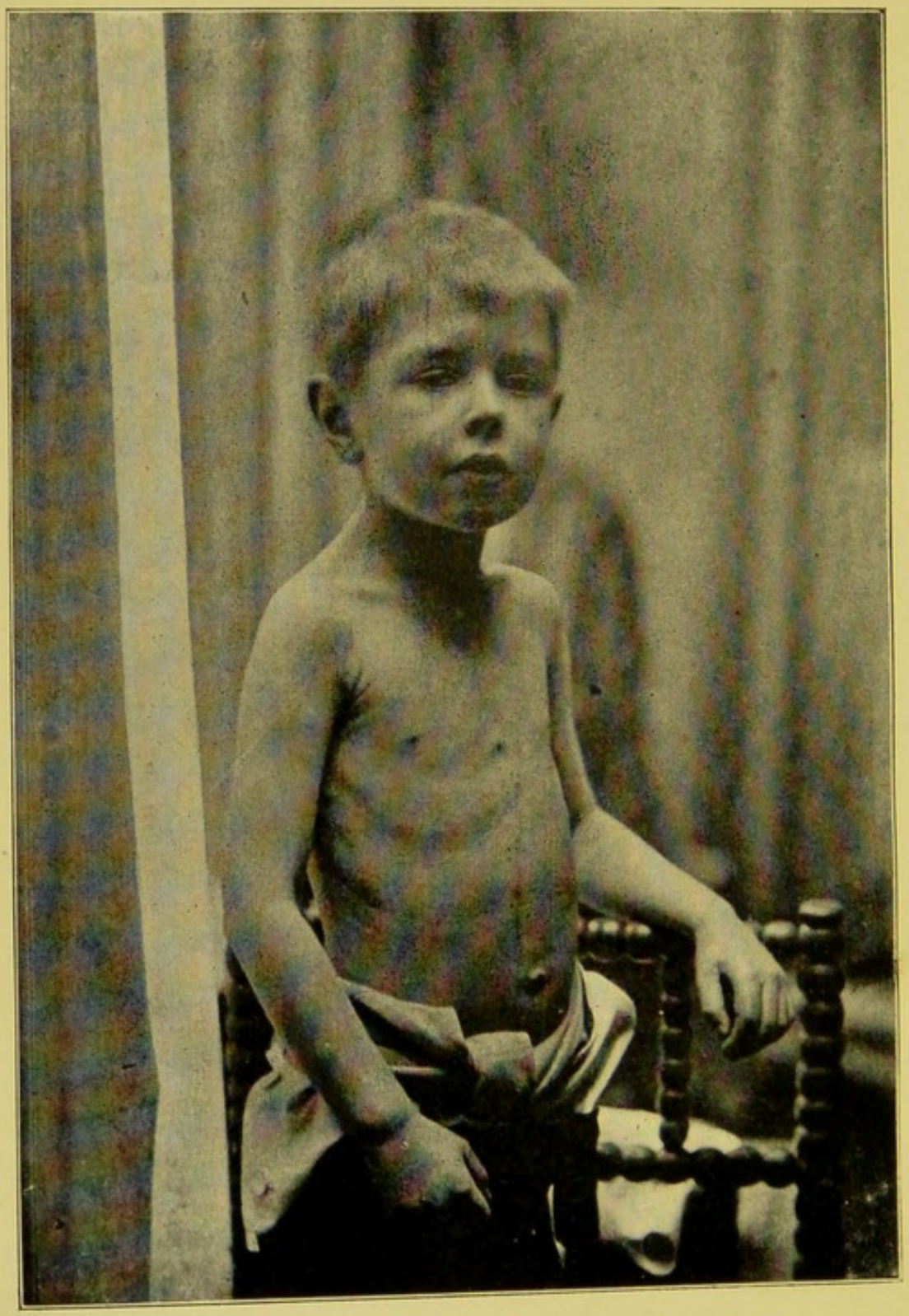
Occasional lectures on the practice of medicine : addressed chiefly to the students of St. Mary's Medical School; : to which are appended the Harveian Lectures on the Rheumatism of Childhood; / revised and corrected up to date by W.B. Cheadle.

THE

PRACTICE OF MEDICINE



E. B., æt. 4, Jan. 25, 1896. Chronic Constipation from infancy.

The colon much distended. (*From a photograph.*)

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OCCASIONAL LECTURES

ON

THE PRACTICE OF MEDICINE

ADDRESSED CHIEFLY TO THE STUDENTS OF ST MARY'S MEDICAL SCHOOL

TO WHICH ARE APPENDED THE

HARVEIAN LECTURES ON THE RHEUMATISM OF CHILDHOOD

REVISED AND CORRECTED UP TO DATE

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TO

MY OLD PUPILS AND FELLOW-STUDENTS

AT ST MARY'S

IN COMPLIANCE WITH WHOSE REQUEST

THESE LECTURES ARE PUBLISHED

Oct. 1, 1899

PREFACE

These Lectures were designed for the assistance of students in the every-day work of their profession, by directing attention to some common errors in medical practice, and by setting forth the importance of certain points in relation to the diagnosis and treatment of disease which are not dealt with in detail in the text-books, are rarely treated adequately in clinical teaching, and which are therefore apt to be neglected or overlooked in actual practice.

19 Portman Street,
October, 1899.

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ON

THE PRACTICE OF MEDICINE

LECTURE I

THE USE AND ABUSE OF TONICS

The object of the lecture—The importance of practical acquaintance with the correct use of tonics—Their routine use a common error in practice—Indications for their employment in the gastro-hepatic disorders of adults and of children—Tonics in dyspepsia—In anæmia and chlorosis—In gout and the gouty state—In diarrhœa and catarrhal colitis—The use of arsenic in chorea—In diseases of the skin—Cardiac tonics: digitalis, strychnia, strophanthus, convallaria—The use of tonics in respiratory affections—Value of the hypodermic injection of strychnia and digitalis in urgent cardiac failure.

Gentlemen,—It is my object in this lecture to give some practical hints as to the right or wrong use of tonics in the treatment of disease, which I trust will be useful to you in the actual every-day work of your profession.

The subject is one which I think requires clearing up. Numerous fallacies are prevalent with regard to it. It is dealt with very imperfectly and scantily in the text-books of materia medica, and hardly mentioned

except in the most general terms, in the books on medicine.

Tonics are, as you know, of various kinds, and differ in their mode of action. They are classed accordingly as blood tonics, heart or vascular tonics, gastric tonics, and so on. It is not my purpose to enter into any description of the special properties of each. I shall content myself with discussing their use in certain well-known disorders, and endeavour to explain when and how they may be administered with advantage, and in what particular form, and when withheld. We are apt to prescribe our tonics in somewhat routine and haphazard fashion. Iron or quinine, or strychnia, or bark, or arsenic, are given without always discriminating adequately which is the most appropriate to the special condition we have to treat at the moment; or, indeed, whether a tonic is really needed at all; for tonics are constantly prescribed in conditions when they are not only useless, but even harmful. Indeed, I am not sure that, taking all cases together, the harm done by the mistaken use of tonics is not greater than the good effected by their proper employment. So constantly do I come across this perverse use of tonics, that I have become impressed with the need of drawing attention to it in this special lecture.

Now the results of experience are a valuable assistance to theoretical knowledge. A good scientific acquaintance with the physiological action of drugs, and the theoretical grounds for their use in disease, is an essential basis and groundwork on which to develop

a reliable treatment. Practical experience is necessary, however, to temper the hard lines of science, and of theory deduced from it. The actual result of treatment by a certain remedy is by no means always in exact accordance with what might be expected, and it is here that practical experience comes in. Deductions which naturally follow from premisses based upon their physiological action alone, have to be modified by knowledge of the innumerable small influences which come into play, such as the effect of concurrent ailments, of the constitutional state, the idiosyncrasy of the individual. Medical treatment, in spite of the wonderful progress which has been made in our knowledge of the physiological action of drugs, is still, in large degree, empirical; our prescriptions in the first instance often a series of small experiments. From these we learn gradually how drugs affect the disease, the combination of diseases, the individual; and it is here, as I have said, that experience comes in and enables us to adapt the canons of physiological therapeutics to the particular disease, or condition, or individual peculiarity; a knowledge which can be gained in no other way and which cannot be formulated. Every practitioner must do this in large degree for himself by careful observation; but something—much of real practical working value—can be imparted from the stores of experience accumulated by others, and I offer you a portion of mine.

With regard to the particular subject with which I am concerned to-day—the use of tonics—you will find

this matter constantly facing you when you enter upon actual practice. Nearly every patient with whom you have to deal, at any rate every one who has reached years of discretion, will ask you for a tonic. People who are ill in any way nearly always think that they are, as they express it, 'below par,' or want 'stringing up' and 'feeding up,' and suggest that you should give them strengthening medicine, something to brace them up and invigorate them at once, a direct elixir of life; remedies which they regard as depressing are not popular with the public, they prefer stimulants and tonics, and feeding.

Yet the sensation of feeling relaxed, limp, unfit for exertion bodily or mental, depends on different causes in different cases. The condition of debility due to muscular weakness, or weakness of the nervous system, may result from an insufficient supply of nutriment in the first instance. There may be want of appetite, so that too little food is taken, or the food may be bad in quality, or it may be imperfectly prepared for absorption by defective digestive power, or the blood may not nourish the tissues because it is deficient in oxygen, or because it is distributed to the tissues too slowly and sparsely, from defect of the circulatory apparatus.

Frequently, however,—and this is the point I especially wish to impress upon you,—the sense of debility—nay, actual weakness itself—is caused by the accumulation of waste products in the body. This accumulation arises either from wear and tear of tissue, from overwork, or from excess of material; more being

taken in as food than the digestive organs can deal with, or the tissues make use of. Sometimes it has its origin in imperfect preparation by enfeebled digestion, or imperfect excretion by the organs concerned in its removal—the kidneys, the liver, the bowels, and the lungs.

In whichever way this accumulation arises, these waste products, and especially nitrogenous waste products, act as poisons to the nervous system, and produce, or increase, the sensation of prostration and debility. They clog the organs of elimination, especially the liver, the kidneys, and the bowels, and the machinery of digestion. Consequently to stir up appetite by bitters, or impede elimination by giving iron or arsenic at the outset, as is so frequently done, makes matters worse instead of better.

It is in this relation I find that in actual practice tonics are most commonly misapplied. They are constantly prescribed when, although toning up is desirable, the condition of things existing at the moment is fatal to their beneficial action. The first step is to get rid of poisonous waste; the second to keep this down during tonic treatment afterwards, by alkaline and aperient remedies in combination, and by a light and appropriate diet.

Gastro-hepatic disorder.—Take in the first instance an extremely common case, one which will frequently confront you, if your practice falls amongst the high living and luxurious people of the leisured well-to-do middle and higher classes, who are overfed and live richly, or even those who eat largely of plain food,

take a good deal of stimulant and little exercise. Most healthy persons who lead sedentary lives eat too much animal food, especially meats. Just reckon up what an excess of nitrogenous food a man takes in the soup and fish and entrées, and meat and game, of a full dinner, to say nothing of what he has at breakfast and luncheon. And then the fats and sugars of rich dishes add to the digestive disorder. A patient consults you perhaps on account of languor, weakness, inability to work, a feeling of depression, disturbed sleep, loss of appetite, dyspepsia, flatulence, and sense of weight and discomfort in the stomach and hepatic region; he will almost infallibly do so with the full conviction that he is simply 'run down,' requires 'stringing up,' 'strengthening,' feeding up, and ask you to give him a tonic. On inquiry, you will find, very likely, that he has been taking tonics, iron possibly, on his own account, or on medical advice, and not improbably that he has been taking abundance of what is called 'extra nourishment'—soups, and beef tea, and milk, port wine, or whisky to restore his strength.

On examining the man, you will discover further that not only is his appetite poor, as he complains, but that his tongue is coated, his bowels sluggish and confined, or loose and disordered, and his urine loaded with lithates or phosphates. Now these persons, judging from the feeling of depression and disinclination to work which oppresses them, are almost invariably possessed by this idea, that they are *overworked*, when they are really only choked up by the waste products of too much food, especially

nitrogenous meat foods, rich dishes, and drink. And you will find this a delusion most difficult to dispel. In some instances, indeed, these patients have, in addition, the pressure of over-brainwork. It is not, however, the *work* which does the harm so much as the worry, and anxiety, and late hours, and bad air of close rooms, and the drink to stimulate flagging effort so constantly associated with hard brain work, which are the main source of evil. When these factors are also in action they greatly aggravate the condition I have described.

Not infrequently, however, the overwork is a delusion; founded on the belief that since the sufferer feels tired and overdone he must necessarily be overworked. He may be, in proportion to his powers at the moment, but overwork is not the cause of his illness. A case in point will serve to illustrate this.

Some time ago I was consulted by a patient—a clergyman—on account of languor, depression and exhaustion; as he expressed it, he was overworked and overdone—worn out. He was a thick-set, burly man, who looked robust enough; and on inquiry I learnt that he was the vicar of a small country parish with a population of only a few hundred people; and that almost the whole of his work was comprised in taking two services on Sunday. During the rest of the week he did little except prepare one fresh sermon, an old one serving for the second discourse required. I discovered that he had been given a strong tonic of iron and quinine, and that two sisters, who lived with him, had been

assiduously feeding him up with beef tea and milk in addition to his ordinary meals, and with a full amount of port wine; and, moreover, the sisters insisted that he should avoid exertion and take ample rest. Accordingly he had his breakfast in bed, and lounged through the day on the sofa or in an easy chair, taking no exercise, going from bad to worse. He had all the signs of overloaded organs and defective excretion which I have described; and I explained to him that what he was suffering from was not overwork but over-eating and drinking and too little work; a too sedentary life. He was loth to believe me, but consented to follow my advice to take a lighter diet with abundance of fresh vegetables, fresh fruit and more exercise. He carried out my instructions loyally and was quickly free from all his depressing ailments; for which benefits he gave me all the credit and warm thanks.

Observe then that, although in this condition a man feels depressed and 'down,' what he wants at the moment is not a tonic, but relief to his overworked organs; the tonic comes later. Get the machinery into perfect working order before driving it at higher pressure. It is no use, indeed worse than useless, to stimulate appetite, to try to put more food into the body when there is already a glut of waste, and when the overtaxed digestive and excretive organs are quite unfit to deal with much new material. A few doses of alterative, with alkali to neutralise uric acid, and light, easily digested food, until the tongue is clean and excretion freed, are necessary to prepare the

ground for tonics. Calomel is the most efficient eliminator; a very good plan of giving this alterative in these cases is to administer the calomel in small doses of one-sixth grain, or quarter grain to half grain in cachet or tablet, in conjunction with a dose of effervescing saline mixture every four hours up to six doses. If the calomel has produced an aperient effect before that number has been completed, it must be stopped and repeated in a few days if required. Then, when the tongue is clean, and other signs of digestive disturbance have disappeared, tonics will come in. The form of tonic is important; strychnia, nux vomica, quinine, and bark are most useful. If you give iron or arsenic in these cases, you will probably bring back the trouble. Iron and other metals, by forming an insoluble salt with uric acid, interfere with its excretion, and, indeed, iron is seldom wanted; the patient is not usually anæmic, unless he has advanced to secondary kidney or heart disease. If you *have* to give iron, however, give a mild salt such as the citrate, in combination with an alkali; the bicarbonate, or citrate of soda or potash in full doses of twenty to thirty grains; or the sulphate together with the sulphate of magnesia and strychnia. Quinine may be given in similar fashion, or with an effervescent saline. This combination of tonic with saline laxative is especially useful if there is constipation.

One of the most useful combinations in these cases is a nerve tonic such as tinct. of nux vomica, combined with full doses, *i.e.* twenty to thirty grains, of citrate

of potash and an equal amount of sulphate of soda. These additions of alkali, or saline aperient, or both, are essential to secure free excretion and enable the tonic to be tolerated. If more aperient is required it can be given in the form of a non-irritant drug, such as an accompanying pill of rhubarb, or cascara, or euonymin.

The Gastro-hepatic disorder of Children.—A condition very closely analogous to that of over-fed and under-exercised adults is met with in great frequency in children, and the misuse of tonics is perhaps more common with them, and equally futile and injurious.

One of your most frequent patients, when you enter upon family practice, will be the child who has no appetite; the mother and nurse will tell you that it eats next to nothing; and yet it may be plump and well-fed and healthy-looking enough. Sometimes, it is lean and ill-nourished, of sallow complexion. You will generally find that the bowels are constipated, the tongue greatly coated, and the breath offensive. Frequently, but not always, these children have what are styled 'liver' attacks—as, indeed, they are—every month or six weeks, or every few months, as the case may be—distinguished by vomiting, headache, pyrexia—a temperature of 101° to 103° perhaps for a day or two; and that these attacks are relieved at once by a dose of calomel or other alterative purgative. Not unfrequently they suffer also from the itching papular eruption of lichen, or prurigo, sometimes accompanied by urticaria, all significant of gastro-intestinal irritation.

You will learn further, on cross-examination of the

mother and nurse, that the child has to be coaxed to take its food, and often is indeed actually forced to take it against will and appetite. Frequently also that, in order to make up for the want of appetite, it is given milk at odd times, and biscuits or beef tea. You will discover also, almost to a certainty, that the child has been persistently treated by tonics; tonics of all kinds, one after another, for years past without any benefit, iron being the favourite; or dosed with Parrish's food and cod liver oil, to the further disturbance of digestion, destruction of appetite, and clogging of the organs of excretion. Such cases I see every week. What these patients want is not a tonic, but some relief to over-worked organs of digestion and excretion, such as is afforded by a calomel purge to begin with, followed by a daily laxative such as sulphate of soda with the carbonate of magnesia until the tongue cleans, and a light diet, with avoidance of all forcing of food and of all persuading to it. Then, as the digestive organs work freely, appetite comes back without fail in natural course. The over-stuffing and tonic treatment of children whose appetites fail from some simple reason of the kind, is one of the most common faults in their medical treatment and management.

Dyspepsia.—Another condition in which tonics are often given with harmful rather than beneficial effect is dyspepsia. Not because in such condition all tonics are inadmissible, but because they are given wrongly combined, or prescribed at too early a stage. It is, I find, the common practice in such cases to order vegetable bitters combined with an acid.

In the vast majority of cases of simple dyspepsia there is too much acid in the stomach already—either from excessive secretion or as the result of fermentation.

In a very few instances only, viz. in the atonic dyspepsia of extremely delicate feeble persons, is there a deficiency of acid. In nearly every case in fairly healthy persons, who mostly suffer as the result of over-eating or imperfect mastication or bolting of food or the like—the dyspepsia is irritant; *acid* dyspepsia—and you must give an alkali such as bicarbonate of soda with your calumba, or nux vomica, or gentian, and give it in full doses of half a drachm to a drachm *after* meals. The small doses of five or ten grains usually given are absurdly inadequate and are usually given before meals. This stimulates acid secretion instead of neutralising it.

In the atonic cases, if you give an acid, give pepsin with it, and if anæmia is present and you feel bound to give iron, avoid strong preparations, such as the perchloride; give a mild form, such as the citrate, in combination with an alkali, at any rate at first. Similarly, if you give quinine, give it in combination with an alkali in an effervescing mixture. The quinine may be kept in solution by three or four drops of hydrobromic acid, with citric acid, in the acid mixture, to neutralise the potash and soda of the separate alkaline mixture which is added at the time of taking.

Anæmia, Chlorosis, and Gastric Ulcer.—Iron and arsenic are appropriate remedies in anæmia and chlorosis, but often great harm is done by their

administration in such cases where there is concomitant dyspepsia; still more if erosion or ulceration of the gastric mucous membrane has resulted, or is imminent. Strong acid preparations such as the perchloride of iron and acid chloride of arsenic are not well borne. It is better to give the citrate of iron with liquor arsenicalis and an alkali such as bicarbonate of soda; or iron may be given in combination with aloes. Usually in chlorosis there is gastric disturbance from feeble secretion of gastric juice and impaired muscular power; and in these it is well to commence treatment by bismuth, soda, and proper dieting, before attempting the use of iron and arsenic. If there is even a suspicion of ulceration, this precaution is still more essential.

Should there be no gastric trouble, one of the most effective methods of administering iron alone or in combination with arsenic is in a weak solution taken in quantity.

Iron, as a restorer of hæmoglobin, is clearly taken up slowly, with difficulty, and in very small quantity. Large doses, or strong preparations, are no more effective in hæmic power than smaller ones, even if they do not upset the stomach; and the good effect of chalybeate waters, where large quantities of a weak solution are taken, suggested to me a similar plan in ordinary medicinal treatment. An excellent form is that of an iron lemonade made by adding thirty drops of liq. ferri perchloridi to a pint or half a pint of home-made lemonade—to this ten drops of the liq. arsenici

hydrochlorici may be added for a time, and this pint or half-pint of iron and arsenic lemonade may be taken in small quantities of half an ounce or an ounce at a time at intervals, so that it is consumed in twenty-four hours. The lemonade has no disagreeable flavour, and this method is pleasant as well as effective. This dilution of iron and admixture with salines is one secret of the virtues of iron waters, and where the means of the patient permit it the waters of Schwalbach, or Spa, or Franzenbad, taken with an appropriate regimen, are better than artificial mixtures.

As a mere blood restorer, however, raw meat juice, with its native hæmoglobinate of iron, is more efficient than any artificial chemical preparation of the drug. With this, sunlight, fresh air, and especially the pure air diathermancy and lessened pressure of high mountain altitudes, 3,000 to 6,000 feet above the sea, aided perhaps by chalybeate waters, are blood-restoring and tonic agencies of greater potency than any other.

Gout and the Gouty State.—Gout, again, is a condition in which tonics are not well borne, unless carefully guarded by alkalies—and, indeed, iron and arsenic, like all other metals, such as lead and zinc, are usually not tolerated in any form. The reason apparently lies in the fact which I mentioned previously, viz. that these drugs interfere with the excretion of uric acid—the irritant agent in gout—by combining with it to form an insoluble urate difficult of excretion; and indeed, owing to this property, lead is one of the chief factors in producing the gout of lead poisoning. It is remarkable how

sensitive gouty persons are to iron. If you mention tonics, they say immediately, 'Oh! you must not give me iron—I cannot take iron, it never agrees with me, it upsets my stomach, or gives me a twinge in my fingers and toes.' I had for many years an old gentleman as a patient who had severe attacks of gout from time to time, and when he was under my treatment for some other ailment, I used to try the experiment of secretly adding iron to his medicine. He always found me out. After a day or two he began to feel uncomfortable and irritable, and have twinges of pain in the foot, and would say, 'I am sure there is some iron in the medicine you are giving me, it always disagrees like this.' The tonics best suited to the gouty are vegetable bitters, which can be given with full doses of an alkali or a saline laxative, nux vomica, calumba, gentian, or even quinine, if given with an alkaline effervescent saline, after the fashion I have described.

What I have said refers to persons who have had a clear attack of genuine gout. It is true, however, also of all who have the gouty habit or tendency. Even if they have not suffered from any active form of the disease, people of gouty stock are liable, although in less degree, to gastric or liver disorder, or to pass uric acid —even women and children with this gouty taint in them do not take iron well, and it must be guarded by alkali and laxative.

In the case of dyspeptic and constipated children with this tendency tonics of any kind usually make matters worse. Many of them, especially young

children of three to five years old, suffer from what are called night terrors; they wake up in the night in a state of terrible alarm, screaming and crying, and cannot be pacified. It is indeed difficult to get them to rouse up to attend to what is said, so dazed are they. The mothers say it is 'nerves,' and the doctors too frequently endorse this view without qualification, and prescribe tonics with perhaps bromides in addition. Now what these children suffer from is simple nightmare; truly enough a nervous disorder, but due to a very simple reflex cause, liver disturbance and constipation; they have coated tongues, foul breath, no appetite. A few doses of grey powder or calomel, followed by a daily laxative for a week or two will relieve the nightmare, which tonics alone serve merely to aggravate. I propose to treat of this more freely in another lecture.

Dysenteric Diarrhœa: Catarrhal Colitis.—Another condition in which tonics are frequently misused is the chronic form of dysenteric diarrhœa—chronic catarrhal colitis—most commonly met with in children. The patient is always anæmic, has little appetite, a coated tongue, and is languid, soft-fleshed, and feeble. Therefore tonics are prescribed. The condition of the mucous lining of the large intestine is the prime source of trouble and failing health. This can be relieved by bismuth, opium, grey powder, ipecacuanha, with the assistance of careful dieting; not by tonics alone. When the colitis is subdued appetite comes back, and general health revives. And similarly with regard to other forms of chronic diarrhœa, tonics alone are insufficient without

bismuth, chalk, and opium, in the first instance to remedy the fundamental fault from which all others spring. I have recently had under my care a boy of eight who had suffered for years from chronic relapsing colitis, with discharge of mucus, offensive stools, occasional blood, coated tongue, failure of appetite. He was very anæmic as such patients are, but the cause of the anæmia was the intestinal ailment. He had, of course, been treated by iron in all forms, as well as Parrish's food, cod liver oil, tonics of all kinds, but he remained as anæmic and languid as ever. The fundamental cause had not been touched. Under bismuth, ipecacuanha, opium, grey powder, and appropriate diet, he grew steadily well.

Chorea.—Take again cases of chorea. Arsenic has, I believe, a favourable influence in chorea, and shortens its course. But when chorea is rheumatic in origin, which is the rule, if there is any sign of joint trouble, or rise of temperature, *i.e.* if the rheumatic virus is still active, arsenic does harm, puts up the temperature, increases rheumatic arthritis or carditis. Salicylates with alkali, or quinine with alkali, should be given until the rheumatic trouble subsides, and then when the temperature has remained normal or subnormal for some days, arsenic may be prescribed with advantage.

Diseases of the Skin.—Take again the case of skin diseases; I constantly meet with cases of acute eczema, of acne, of prurigo, of lichen, and urticaria, treated by arsenic and iron, and made worse. There are few errors in treatment more common than this: arsenic

is an admirable remedy in certain skin affections, but not under these conditions. Alkalies, purgatives, or laxatives are the efficient remedies during the acute stage of eczema, and throughout the whole course of acne and prurigo, and lichen urticatus. The three latter are caused by constipation or indigestible food, or some source of irritation in the alimentary tract; and arsenic, a powerful stimulant to the intestines as well as the skin, capable in itself of producing erythematous eruptions, is injurious in all these cases, and iron likewise, although in less degree.

Heart affections.—Lastly, take the heart tonics of which digitalis, strychnia, strophanthus are the chief. Their most frequent and best known use is in toning up the heart muscle in cases of organic disease, or functional debility of that organ.

It is impossible for me to go into details of their action and use in all varieties of heart disease, but I may lay down a few broad rules for your guidance.

When I first began practice, the treatment of cardiac disease, as far as direct action upon the heart was concerned, was pretty well summed up in a sentence —'in disease of the heart, give digitalis and iron.' We have learnt much from physiology since then, yet cardiac tonics are still prescribed in a loose and indiscriminate fashion. Digitalis, which has the power of increasing the force of the muscular contractions of the heart and arterial tension, has also the property of lengthening the diastole; and this must be borne in mind when employing it. Thus, digitalis is

especially useful in mitral regurgitation, enabling the ventricles to drive more blood into the arteries from increased propulsive power, contracting the mitral and tricuspid orifices, and so lessening leakage, and by lengthening the diastole, and reducing the number of beats, affords the ventricles more time for refilling, and for recovery of energy. This *latter* property of heart-slowing belongs far more to digitalis than to other cardiac tonics except, perhaps, to convallaria. Digitalis is especially serviceable, then, in all cases when the heart beats fast and feebly, with short, ill-sustained contractions following each other in rapid succession, with short diastolic intervals of rest.

There is some difference of opinion as to the use of digitalis in aortic disease, but I think it is of doubtful safety in cases of pure aortic regurgitation, where the reflux is great, and the systemic arteries empty; for to lengthen diastole causes further depletion of arteries, and risk of fatal syncope from cerebral anæmia, and from deficient supply to heart muscle by the coronaries.

In these cases, strychnia is more suitable, since its action is almost purely stimulant, and sustaining to the cardiac muscle—and it is most potent when given by hypodermic injection. Caffein has a similar but less sustained action. Strophanthus and convallaria appear to hold a place intermediate between digitalis and strychnia. Digitalis is, however, admissible, in cases where aortic regurgitation is complicated by mitral reflux, and may be combined with strychnia. Digitalis again is unsuited to those cases where the heart acts

with abnormal slowness, a condition sometimes associated with fatty degeneration. On the other hand, digitalis is most valuable in cases of simple atonic condition of the heart muscle, with or without dilatation, where no valvular lesion exists. Thus in the feeble heart of anæmia, in the feeble dilated condition which follows acute disease, notably influenza, these cardiac tonics, and especially digitalis alone, or combined with strychnia and iron, are of immense service.

Diseases of Organs of Respiration.—The use of cardiac tonics is, however, by no means limited to conditions in which the heart itself is the organ primarily affected, either organically or functionally; they constantly prove most powerful aids in the treatment of diseases of respiration, both acute and chronic; in pneumonia, in bronchitis, in emphysema, in chronic fibroid, phthisis, and the like.

In acute pneumonia, the right heart is embarrassed by the obstruction to the pulmonary circulation; its action is disturbed, and its muscular contractions rendered short, rapid, and ineffective, by the exciting stimulus of the febrile state. After a period of this extra strain and forced effort, the muscular power of the ventricle becomes seriously weakened, the patient becomes more or less cyanosed, from the defective pulmonary circulation, and his dyspnœa more marked. It is *then* that, aided by pre-application of leeches, or even venesection, digitalis comes in, and is of such signal service; it calms, invigorates, restores; increases the power of the cardiac contractions, enables the wearied, embarrassed organ to recruit, by lengthen-

ing the period of diastolic rest. Strychnia and caffein help in the same direction, and strophanthus and convallaria act as substitutes for digitalis, if, as occasionally happens, the latter causes sickness or too great disturbance of cardiac rhythm.

I remember numerous cases in which the judicious use of cardiac tonics, and especially digitalis, as it seemed to me, turned the scale in favour of life.

I will mention very briefly one which will interest you as being especially creditable to one of my house physicians. The patient, a woman of upwards of fifty, came into hospital for cirrhosis of the liver with ascites. She was tapped several times and was apparently doing well, when she developed acute pneumonia. Such patients bear acute disease very badly—the circulation is feeble, and the tissues and organs ill-nourished—death seemed a certain result. The woman rapidly became deeply cyanosed, and hardly sensible. I gave an absolutely unfavourable prognosis—but my house physician, after a time, increased the dose of *ten* drops of tincture of digitalis which I had prescribed to *fifteen* drops every four hours; and, to my surprise, and his, the patient completely recovered; in my experience a unique event in such grave conditions.

In acute bronchitis and broncho-pneumonia, likewise, and in the pneumonia and lung collapse of whooping cough, especially in the broncho-pneumonia of children, I have seen remarkable results follow the judicious administration of digitalis. These drugs, strychnia and digitalis, are I find very generally given nowadays in these respiratory affections. The mis-

take usually made, is in resorting to them too early—from the very outset—or too late, when the heart has hopelessly broken down. They should be held in hand until the heart shows some slight indication of requiring aid of the kind, evidenced by commencing blueness, or the least flagging, or loss of tone in the pulse—not stimulating the heart unnecessarily, but giving it help on the first sign of its being required.

These two drugs, strychnia and digitalis, are both most effective when administered by hypodermic injection. They act more quickly and more powerfully than when given by the mouth. This method is, however, only necessary in extreme or urgent cases, and in emergency. The use of strychnia in this way—first advocated by the present Dr. Habershon, I think—is now an established practice. I have, however, never seen digitalis so administered, except at my instance; yet a hypodermic injection of three to five drops of tincture of digitalis is as safe, and often more potent than strychnia in reinvigorating a failing heart, and in urgent cases may be given in this way.

I remember well the first case in which I used it, that of a lady dying of puerperal fever; she was seemingly moribund, absolutely pulseless, cold. A hypodermic injection of twenty minims brought back the pulse to full beat, for a time; the patient recovered as one from the dead, and lived twenty-four hours longer.

Gentlemen, I have by no means exhausted this most attractive subject of tonic treatment, which seems to lead on indefinitely; but time fails, and I cannot venture to pursue it further.

LECTURE II

THE CLINICAL USES OF OPIUM

The importance of a proper use of opium—Disadvantages attending hypodermic injection—Some important physiological effects of the drug—Conditions modifying the action of opium—Its value in insomnia; delirium; exophthalmic goître; morbus cordis; respiratory affections; irritative dyspepsia; gastric ulcer; colic; diarrhœa of various forms; typhlitis; typhoid fever; peritonitis; intestinal obstruction; intussusception; biliary and renal colic; diabetes—Opium invaluable also in aiding euthanasia—Caution required in its use in Bright's disease; when the heart is fatty and much dilated; in cirrhosis of the liver—Care required in stopping the drug when it has been used freely.

GENTLEMEN,—The subject of my lecture to-day, viz. 'The Clinical Uses of Opium,' is, I think, one well deserving of your attention. Practice in this respect has become more and more narrow and routine. The use of opium in these days is chiefly limited to its administration in the form of hypodermic injections of morphia for the purpose of easing pain or of procuring sleep; or in suppositories; or in small doses by the mouth for the relief of cough or diarrhœa. It is given, moreover, as a rule, irregularly, in occasional doses only. Continuous and systematic treatment by opium is rarely seen, except in cases of peritonitis.

Its therapeutic value has, I think, a wider range.

Hypodermic injection of morphia has, no doubt, certain advantages. Of these the most striking are its quickness of action, the fulness of its effect, and the greater anodyne influence obtained when so administered. On the other hand, there are certain disadvantages connected with this method of administering opium. For one thing, it is by no means so safe as the older methods; nor is the effect produced so steadily maintained. There are also certain difficulties inseparable from the hypodermic administration of the drug. In ordinary practice the physician cannot always be at hand when its employment is desirable, so that the duty has to be handed over to the nurse, or even, in some cases, to the patient, which, considering the abuse to which it may lead, is by no means satisfactory. Again, the frequent punctures produced by repeated injection and sometimes the careless use of an unclean needle lead to the formation of abscesses.

A former patient of mine, a confirmed morphinomaniac, suffered from a constant succession of such abscesses, and was for a long period in a state of chronic pyæmia from this cause.

Moreover, in certain cases, where it is desirable to keep the patient under the constant influence of opium, there is a distinct advantage in giving it by the mouth; or where a local effect on the alimentary canal is desired, as in gastric ulcer, or in diarrhœa. But this method, again, has its drawbacks; for the appetite and the gastric secretion are thereby diminished, and from

this reduction in the amount of gastric and intestinal secretion and lessened peristalsis come loss of appetite, dyspepsia, and constipation. The common tincture and crude opium cause the most disturbance in this way, the more refined preparations comparatively little.

As regards opium, then, the manner of its administration, as well as the quantity and form of preparation required, varies in different cases. In each of these the question whether opium will be administered with the maximum advantage and the minimum disadvantage, by hypodermic injection, in the form of suppository, endermically by means of blister, or through the mouth, has to be carefully considered.

It is not necessary that I should describe to you in full detail the physiological effects of opium and the alkaloids derived from it; but some knowledge of them is essential to a correct therapeutic use of the drug. It will be sufficient for me to remind you of the leading influences it exerts upon the different organs and their functions.

1. With regard to the nervous system, as you will remember, its influence is chiefly exercised on the brain. Given in small doses of a quarter to half grain its first effect is to produce a certain degree of excitement; this is speedily followed by a sense of increased energy and mental power, which, in its turn, gives way to an agreeable feeling of languor, passing into sleep.

In moderate doses (one to two grains) the stage of excitement is short, and deep sleep is soon induced;

while as after-effects headache, nausea, coated tongue, and loss of appetite prove troublesome. By such doses the brain is rendered anæmic, its arteries and veins becoming comparatively empty.

In large doses opium quickly produces sleep, which passes into coma, and the patient is in danger of death by asphyxia. Opium induces sleep by its direct depressant effect on the functional activity of the brain; this is followed by cerebral anæmia, which also favours sleep.

With regard to the motor system, opium does not seem to exert any influence on the muscles, while its action on the motor nerves is doubtful. It has, however, a very definite effect on the sensory system, the sensory nerves being first excited and then paralysed, so that any pain which may be present is either relieved or completely abolished. The effect produced by opium on the brain centres may be summarised in the order of importance thus: there is

1. Loss of voluntary motion.
2. Loss of co-ordination.
3. Paralysis of the centres in the medulla.

Respiration is affected in two ways: the respiratory centre in the medulla being depressed, and the bronchial secretion, like most other secretions, lessened.

The action of opium on the circulation is indicated by a distinct dilatation of the cutaneous vessels, which may even amount to the production of a red eruption. The vasomotor centre does not seem to be affected by small, but is paralysed by large doses. The blood

pressure is only slightly affected. On the heart little or no direct influence is produced. All the secretions are lessened by opium except the urine, which may sometimes be increased, and the sweat, the secretion of which is always stimulated.

On the alimentary canal the influence of opium in very large quantity has been ascertained by experiment to be the production of a tetanic contraction of the stomach and intestines, while the peristalsis ceases. Thus experimentally administered to animals, opium produces a single copious evacuation of the intestinal contents. In moderate doses peristalsis is lessened, so that in this way, as well as from the diminution of the intestinal secretions, constipation is induced. In small doses, on the other hand, opium sometimes increases the peristalsis and so acts as a purgative. Especially is this the case when the existent constipation is due to reflex spasm, *e.g.* from ovarian irritation, from peritonitis, or from intestinal obstruction. The action of opium in peritonitis, according to Lauder Brunton, is probably twofold. (1) It lessens the peristaltic movements of the intestines, and so diminishes the local irritation. (2) It lessens the reflex activity of the centres through which the irritation acts in causing dilatation of vessels, and thus it diminishes the peritoneal congestion. Dr. Brunton quotes a saying of Sir Robert Christison to the effect that not only coryza but probably all inflammations could be nipped in the bud by opium if it could only be given sufficiently early and sufficiently freely.

Certain conditions exert a modifying influence on the action of opium :—

1. *Age.* In infancy, the brain being larger in proportion to the rest of the organism than in adult life, opium has a greater effect; following the rule that in mammals the influence on the brain is in proportion to its degree of development in relation to the other nerve centres; so that even one minim of the tincture has been known to prove fatal to infants between six and twelve months old. In old age, also, opium exerts a more powerful influence than in middle life.

2. *Idiosyncrasy.* While some persons are very readily excited by opium, even in small doses, and are almost intolerant of it, others are but little influenced even by large doses of the drug. There is a marked difference between individuals in this respect.

3. *Habit* has a most important effect on the degree of susceptibility shown by the patient to the action of opium and morphia. The habitual use of the drug may cause merely delay in its action when administered medicinally, or produce only a weakening of its effect, while in other cases both results may follow. These facts may possibly be explained by the lessened absorptive power of the intestine. Thus, it is said, opium eaters can take large doses of perchloride of mercury without being affected. The slow absorption of the drug may allow of its conversion into oxydimorphine, which counteracts the soporific action of morphine.

A case has been recorded in which two pints of the tincture were taken in a day. I have myself admin-

istered ten grains of hydrochlorate of morphia subcutaneously in divided doses in twenty-four hours to a regular morphia taker without producing any marked effect, and as many as forty-seven grains are said to have been taken in a day under similar conditions.

Such being the physiological powers of opium, it has naturally found large application in disorders of the nervous system. Perhaps the condition it is most commonly employed to combat is that of *insomnia*. Want of sleep results from many different conditions. It is produced by physical discomfort of various kinds, such as dyspepsia, pain, cardiac distress, and the like; and in such cases the sleeplessness is cured by the removal of the particular cause of discomfort. Alcoholism, chronic or acute, is also a common factor in the production of insomnia; while the condition may also appear as an early symptom in incipient mania. Want of sleep occurs in the specific fevers also, and in cases of morbus cordis (perhaps from the consequent fulness of the cerebral vessels), in old age (probably for the same reason), and in Bright's disease as a result, perhaps, of the disturbance of the cerebral circulation. Apart from these it arises also from over-stimulation of the brain, *e.g.* through worry or overwork, which keep up a condition of active hyperæmia in the organ. In the simple insomnia of old age and in that of excessive brain stimulation, opium and the bromides are often the most useful hypnotics. This ailment is an exceedingly common one, and its treatment by narcotics has led to a great abuse of them, especially of alcohol,

the bromides, opium, chloral, and sulphonal. So much has been said of the ill effects of the constant use of opium that people have become afraid of it, and have turned to other soporifics, so that now the most commonly used and abused narcotics are not opium and morphia, but chloral and sulphonal. The effect of the constant and prolonged use of these drugs is, however, equally or more disastrous than that of the purer and milder preparations of opium. The habitual chloral and sulphonal taker becomes exceedingly nervous; his limbs tremulous; he grows more and more sleepless, and often hysterical; while in the case of chloral, a sudden end is not infrequent as the result of an overdose. With chloral, habitual use does not appear to lead to tolerance of the drug in larger and larger doses, as is the case with opium; so that as the quantity is increased the limit of safety is suddenly reached without warning, and a fatal dose taken.

Where sulphonal and chloral have been taken habitually, and nervous breakdown has followed, the substitution of opium is often most useful. In such cases it is best administered by the mouth in repeated small doses. I have just had under my care a medical man whose nervous system and general health had completely broken down under the continual use of sulphonal for insomnia. He was nervous, apprehensive, his hands trembled, his appetite declined. He had grown thin and feeble, and more and more sleepless. Under doses of ten drops of nepenthe with five drops of liq. strychniæ every eight hours, and a

cup of beef tea and port wine at bedtime, sleep came. He was able to abandon the use of sulphonal, gained fresh vigour and appetite, tremor ceased, the opiate was gradually reduced, and he is now well, enjoying natural sleep without the aid of any narcotic.

In the *insomnia* of delirium tremens the old plan of using full doses of opium is, I am convinced, a dangerous one. By this method patients have been sent to sleep, but sometimes to their last sleep. In such cases opium is best given in full but safe doses, combined with digitalis.

In the *insomnia* of fever or pneumonia, if there be no lividity present, opium in moderate doses is the safest narcotic, since, when given by the mouth, without the sudden shock of hypodermic injection, it does not depress the heart.

Opium is sometimes of signal service also in the *delirium of acute disease*, such as that of fever or pneumonia; while at other times it may prove absolutely futile. A distinction must be drawn here between cases where the delirium is due to active hyperæmia of the brain and those in which it is the result of failing circulation. In the first case opiates should be used, and ice applied to the head; in the second, stimulants, digitalis and strychnia are the proper remedies. In these cases of excitement and delirium in the course of fever, chloral, bromide, and henbane or hyoscine may be used in combination with or in the place of the opium.

Great benefit is sometimes obtained from opium in cases of neuralgia. In these the opium acts most

effectually when applied locally and hypodermically or endermically.

Another nervous disease in which opium is of the greatest service is *exophthalmic goître*. Although it seems probable, from the recent researches of Prof. Greenfield and others,[1] that the physiological disturbance which occurs in this condition,—palpitation, pyrexia, emaciation, vomiting and diarrhœa,—is the result of the excessive secretion of thyroid juice; yet the starting point of the disturbance would appear to be the vasomotor centre of the medulla. In any case—whether primarily or secondarily—the nervous system is profoundly affected, and the drug which exerts the most favourable influence is opium. Tachycardia is, as you know, one of the most troublesome and prominent symptoms in exophthalmic goître, the heart-rate running up in some cases to 150 or 200 per minute, and accordingly belladonna in full doses, or digitalis, or convallaria, on account of their slowing action on the heart, are the remedies most usually prescribed. These drugs are useful, and have their place in the treatment of the disease; but opium in full doses and at regular intervals not only soothes the nervous distress and palpitation, but arrests the diarrhœa and vomiting, which all other drugs seem powerless to control. In these complications the chief danger lies. When cases of exophthalmic goitre end fatally it is almost always from persistent and uncontrollable vomiting and diarrhœa. Opium alone will arrest it.

[1] Bradshaw Lecture, 1893.

There is no condition in which opium may be employed more beneficially than in certain *diseases of the heart and circulation.* Thus in mitral disease, and other forms of affection which are constantly accompanied in their later stages by distress and cough, restlessness and sleeplessness, it is of great service. Many authorities deprecate its use in these conditions on account of its action on the respiratory centre; but an extensive experience has taught me that it may be administered without fear if given in moderate doses of the finer preparations, and by the mouth, as by this method the shock and the sudden depressant action on the heart, which render the use of the hypodermic method dangerous in such conditions, are avoided.

I remember well the case in which my attention was first drawn to the value of opium in these cases of cardiac distress. The patient, a woman of sixty, was brought into the Alexandra Ward in a state of urgent dyspnœa. She had a constant cough and somewhat profuse hæmoptysis. Physical examination disclosed mitral stenosis and regurgitation, advanced hypertrophy and dilatation of the right heart, a large extent of dulness over the posterior portion of the right lung, with fine crepitations—due, no doubt, to an infarct—with basic congestion of the left lung. There was some cyanosis and extensive dropsy. The pulse was small and irregular. The patient was unable to sleep and in a condition of restlessness and distress from the sense of difficulty of breathing painful to witness. Stimulants such as ether and ammonia were admini-

tered without material effect. The case seemed to me hopeless—the patient at the end of the last stage of cardiac disease. I expressed this opinion, and that there was nothing to be done but to relieve suffering and promote the euthanasia. With this view I prescribed ten drops of liq. opii sedativi every four hours. I had no expectation of seeing the woman again alive, but, to my surprise, at my next visit she was wonderfully better. She had slept, was able to take food, and the dyspnœa was no longer extreme. The improvement was maintained under the continued administration of opium—aided later by digitalis—and in a few weeks she went out of hospital convalescent.

Let me give you another instance. A boy of twelve, in the children's ward, with mitral disease, adherent pericardium, and resultant engorgement of both lungs, had frequent pulse and palpitation, with harassing incessant cough which became so much aggravated on lying down that he could not sleep. He began to lose ground rapidly, chiefly from want of rest. He was then given ten drops of nepenthe every four hours. Rapid improvement followed. The cough gradually ceased, the pulse rate fell from 120 to 90, good sleep was obtained, and appetite returned. It is worthy of note how the opium in this case acted as a tonic; instead of impairing appetite it increased it by giving ease and sleep. This patient went out convalescent, to return in a few months in almost exactly the same condition —sleepless from incessant cough due to congestion of the bases of the lungs. The same treatment proved

efficacious a second time, and he again went out convalescent. You may indeed see an example of the singular benefit conferred by opium in such conditions in the case of a young fellow in the Albert Ward, with advanced aortic regurgitation, and great hypertrophy and dilatation. A few days ago he was in constant and increasing distress from tumultuous action of the enlarged heart, cardiac pain and dyspnœa preventing rest and sleep. Ten drops of nepenthe every four hours have, in his case, also produced a marked improvement. His heart has become quieter, the distress is relieved, and he is able to sleep and eat once more.

The terrible agony which occurs in *angina pectoris*, when the patient is oppressed with a sense of impending death, and there is intense dyspnœa, is relieved by opium. Formerly the custom was to give a drachm of laudanum by the mouth; the action is, however, slow, while hypodermic injection will give instant relief. This, however, is not, as has been maintained, altogether unattended with risk. I have known death follow its use in a case of greatly dilated heart.

Except in a desperate case, therefore, or unless nitrite of amyl be not available, I should not give a hypodermic injection, or only a small one, and administer it very slowly. In angina, nitrite of amyl or brandy, followed by the continuous use of nitroglycerine regularly, or opium in frequent and moderate doses, is the best treatment, together with the usual precautions against mental excitement, physical exertion, and overloading of the stomach.

Passing on now to diseases of respiration we find that in one affection where great results might have been anticipated, viz. *whooping cough*, the influence of opium has proved disappointing as a means of controlling the paroxysms. Chloral and bromides, and, in the later stages, Indian hemp, are more efficient sedatives.

In *asthma* the effect of opium in the shape of hypodermic injection is most striking, and is produced in a few seconds. It is, perhaps, the most effective of all means of relieving asthmatic spasm. Here, again, however, its use is not altogether devoid of danger. As I have stated previously, when the heart is greatly dilated and feeble, the shock caused by the sudden intrusion of the drug into the circulation may cause fatal arrest of its action.

Idiosyncrasy also supplies an element of danger in this condition. The injection of one-sixth of a grain of morphia has produced fatal coma in a girl of nine suffering from severe asthma. In old-standing cases, therefore, it is safer to trust to other remedies, such as the tincture or compound powder of stramonium.

In *bronchitis* opium is, in my experience, the only drug which has much power in assuaging the severity of the cough. One of the best forms is the old-fashioned paregoric, which may be given in half-drachm doses, together with iodide of potassium and ipecacuanha, for the purpose of increasing and liquefying the bronchial secretion, which opium tends to diminish, although in a less degree than belladonna and atropine. Opium relieves cough by lessening the excitability of the

respiratory centre, and caution must consequently be observed if any degree of cyanosis be present.

In *phthisis*, as in bronchitis, opium is the only remedy which will effectually relieve the distressing cough. Other sedatives, with the exception, perhaps, of inhalations of conium, have small power in this respect, and conium inhalations are apt to nauseate the patient. The objection to the free use of morphia in phthisis lies in the fact that it increases the sweating, already too profuse, and interferes, to some extent, with appetite and digestion. The first may be neutralised by the addition of belladonna or atropine; the second to a great degree by avoiding the coarser preparations of opium, and using morphia or nepenthe with hydrochloric acid and gentian; the treatment in both cases being continuous, by moderate doses at short and regular intervals. This regular administration is essential. The common plan of giving a dose of sedative only occasionally, when the cough is troublesome, is to apply the remedy too fitfully and too late.

The only conditions of the *stomach* in which opium comes effectively into play are those in which vomiting and pain are conspicuous symptoms. In *irritative dyspepsia*, for example, where there is pain or discomfort, opium is extremely useful combined with hydrocyanic acid and full doses of sodium bicarbonate. In *gastric ulcer* opium is sometimes of signal service; it relieves pain, and by its power of arresting peristalsis, it lessens the churning movements, and gives the injured organ rest. Again, opium relieves pain, and by

its direct influence on the vomiting centre when absorbed, as well, possibly, as by reflex action in soothing the gastric nerve filaments, it relieves vomiting. For this purpose it should be given in small doses frequently repeated, as three to five minims of liq. opii with five grains of bicarbonate of soda in one or two teaspoonfuls of water. When the stomach is irritable it will often retain medicines in small bulk, when a larger quantity is at once rejected. If the vomiting is severe, hypodermic injection must be resorted to. Opium may also be given in this condition in the form of suppository; or by the dusting of one-sixth of a grain of morphia on the raw surface of an epigastric blister. These measures are, of course, subsidiary to the privation or limitation of food by the mouth, and rectal feeding, which are essential in all severe cases of gastric ulceration.

Passing on to *diseases of the intestine* we find that in many of these opium is of immense service. It does good chiefly through its power of lessening peristaltic movement, either by paralysing the motor fibres of the vagus, or by stimulating the inhibitory fibres of the splanchnic. Take, for example, that painful condition known as *colic*. Intestinal colic is due to a muscular spasm, or cramp of the bowel, set up by some irritant matter, such as indigestible food, part of the gut being, at the same time, unduly distended by gas. In these cases opium, by virtue of its power of arresting peristaltic contractions, gives relief. A special form of colic which occurs in children, deserves mention in this regard. It is usually a sequel of diarrhœa, which

leaves behind it increased irritability of the intestinal muscle, producing cramp, and is liable to recur very persistently. The child is seized from time to time—sometimes every day or two, sometimes once or twice a day—with more or less severe abdominal pain, generally in the region of the navel. It is usually found that there is tenderness in this one particular spot. In these cases opium does wonders. Belladonna is more often given, but inasmuch as it increases peristalsis it tends to aggravate the condition. Bismuth is constantly given without result. Tonics are tried, and usually make matters worse. Opium, properly administered, and its constipating effect neutralised by some mild laxative, will effect a cure. I think the best form is Dover's powder, with or without grey powder, according to the state of the bowels; one to one and a half grain of Dover's powder being given regularly every eight hours to a child of three years old, and so on in proportion.

Take again *diarrhœa* of various forms; how essential opium is to the efficient control of it! It acts by lessening at the same time both secretion and peristalsis. It is an indispensable adjunct of all astringent or antiseptic remedies for this condition. The first are usually ineffective alone; the second, acting only by arresting the decomposition of the intestinal contents, and take effect slowly; 'while the grass grows the steed starves,' and hence the necessity for opium to restrain the activity of the gut until the abnormal conditions which produced it subside.

In the severe form of 'infantile diarrhœa,' termed

'*cholera infantum*,' where there is profuse purging, death largely resulting from the draining of the vessels of fluid through the constant watery discharges, opium is of the first importance. Nitrate of bismuth soothes the catarrhal mucous membrane, and is of great service if given in full doses of five to ten grains every four hours for a child three months old, with two to three grains of chalk to neutralise irritant acids, and enemata or hypodermic injections of water to replace lost fluid; and a bland digestible liquid diet is essential. These remedies, however, require time to act; meanwhile it is necessary to arrest the drain quickly. Opium alone will do this effectually.

Many practitioners are afraid of giving opium in any form to little children; and in the case of very young infants a few weeks old it has its risks, unless minute doses are given at first and their effect carefully watched. The text-books, indeed, almost invariably deprecate the use of opium in such cases in young children, yet I am sure that I have constantly saved life by its employment, where its use has not been ventured upon on account of the infancy of the patient. It may be given in doses of one-eighth to half a grain of Dover's powder, according to age, with a quarter of a grain to half a grain of grey powder, according to age, every four hours; or one-eighth to one-half a drop of nepenthe added to nitrate of bismuth in full doses. It will arrest profuse diarrhœa when added to these other remedies, which they seem quite powerless to control without it.

In the *dysenteric diarrhœa or catarrhal colitis* of children opium relieves the pain, the vomiting (which is often a conspicuous symptom at the outset), and the tenesmus. Here, again, the best form is Dover's powder with grey powder; that is, giving opium with ipecacuanha and grey powder at first, when sickness is prominent, and with bismuth and ipecacuanha when the intestinal symptoms prevail, in other words, when the diarrhœa is more urgent than the vomiting.

There is another form of diarrhœa common in children, but met with also in adults, that called *lientery*, in which opium is invaluable. This kind of diarrhœa is usually very imperfectly described in the text-books, and certainly not given the importance it merits. It is characterised especially by the feature that the bowels are excited to act by taking food. This is the result of an undue irritability of the intestine, and chiefly of the small intestine, although the whole bowel is, in fact, in a condition of hyper-excitability. That this form of diarrhœa does start in too active peristalsis of the upper bowel is proved by the character of the stools. They are large and yeasty —chyme-like; the contents of the stomach much in the state in which they pass from the pylorus into the duodenum, without due digestion in the intestine. Food taken into the stomach quickly stimulates peristalsis, which, starting in the duodenum, runs down the small and large intestines, causing premature expulsion of their contents. Absorption necessarily takes place imperfectly, because the food has been imperfectly prepared for

that process and passes out too quickly. In fact, the condition of things is in some degree such as would obtain if the anus were placed at the duodenum. Now opium, by its power of restraining excessive peristaltic action, has a powerful influence on this condition, and in conjunction with bismuth and ipecacuanha if necessary, and proper diet, rarely fails according to my experience to effect a cure.

This power of opium to restrain peristaltic movements is most useful again in *typhlitis* and in *typhoid fever*. An ulcer on the point of perforating may heal if the part is kept absolutely at rest. So, too, when hæmorrhage has occurred, the administration of opium facilitates the natural arrest of bleeding by keeping the part quiet and motionless. In the same way, the peritonitis caused by a slow perforation may be limited to the locality at which the accident has occurred.

This leads me to speak of the use of opium in *peritonitis*. I believe that in general suppurative peritonitis, whether set up by perforation of the bowel, pelvic hæmorrhage, or pyæmia, the case can only be dealt with surgically—by laparotomy and flushing. But in local adhesive peritonitis—tubercular, influenzal, or perityphlitic—opium does much. By keeping the parts at rest and by restraining the activity of the local vasomotor centres, it lessens the vasomotor dilatation, and aids the subsidence of inflammation. In the various forms of *intestinal obstruction* opium plays an important part in treatment, possibly by its power of relieving spasm of the bowel. In fæcal

obstruction, for example, it sometimes acts favourably by virtue of this property, and aids effectually the use of repeated enemata.

In the case of *intussusception* opium is of great benefit. By arresting peristaltic action it prevents the further invagination of the gut, which results usually from the too active peristalsis of the small intestine forcing the ileo-cœcal valve before it. By stopping peristalsis it stops further intussusception. Opium should always be given, therefore, where this affection is suspected, to keep things *in statu quo* until the condition can be reduced by inflation or water distension, or, failing these, surgical operation.

In cases of *impacted gall-stone* and in *renal calculus* opium gives a great relief, not only by easing the violent pain and vomiting, but also by relaxing spasm and thus facilitating the passage of the stone.

In *diabetes*, again, opium and its alkaloids have great power in reducing glycosuria. They act probably by reducing the irritability of the afferent fibres in the nerves of the abdominal viscera, diminishing the irritation of the vagus by dulling it and its reflex agents, and thus reducing its activity in causing dilatation of the hepatic vessels.

The beneficent effect of opium in aiding *euthanasia* is a matter which, I think, hardly receives the consideration it deserves. Where we cannot cure, we can by this 'best gift to man' ease distress, both bodily and mental, and pass the patient peacefully and almost painlessly across the dreaded river.

In conclusion, let me add a word of caution. Remember that in cases of *Bright's disease* opium, especially if administered hypodermically, is liable to produce profound and fatal coma.

In cases of *fatty heart*, and *largely dilated heart*, the hypodermic injection of morphia is attended with risk, and in cirrhosis of the liver also opium has an increased toxic effect.

Remember also that children are susceptible to its influence in inverse proportion to their age. I have known fatal coma set up in a child of six months by half a drachm of tincture of opium, given as a rectal injection; and a child of fourteen months completely narcotised by 2½ grains of Dover's powder.

And lastly, remember, what is usually ignored, that if opium has been given freely, *the sudden stoppage* of it produces great nervous depression, often severe vomiting and diarrhœa. This is the effect of sudden privation in the case of opium eaters and morphine takers, and I have seen similar results follow in the case of patients who have been given it systematically, when it has been abruptly discontinued. In exophthalmic goître, for example, its sudden stoppage may be most disastrous. In these cases it is wise to let your patient down slowly and safely by a gradual reduction of the dose.

LECTURE III

PREVALENT FALLACIES IN THE DIAGNOSIS AND TREATMENT OF CERTAIN MINOR DISORDERS OF CHILDHOOD

The importance of correct treatment in dealing with minor ailments—Night terrors—A description of their characteristics—Erroneous views as to their pathology—The usual cause—Reflected irritation or discomfort—Most often disturbance in the alimentary canal—Occasionally caused by adenoids or by mental excitement—Faulty treatment of night terrors—The essential points in treatment—Examples—The periodic pyrexia of childhood—The cause of this condition—Its treatment—Lichen urticatus, its cause and treatment.

MANY advantages pertain to the position of a hospital physician or surgeon, and amongst them this—viz. that in the course of his work he is brought continually into contact with a large number of other medical men, both students and practitioners, of every grade.

Thus, he is enabled to observe the pathological and clinical views, and the therapeutical fashions of the time, which prevail in the general medical world.

I have in this way gathered a certain amount of useful information; on the other hand I have become acquainted with certain gaps in clinical knowledge, fallacies in diagnosis, and practical errors in treatment,

which are rife. I shall do service, I think, in bringing some of these before you—things you will not find in books—results of personal observation and experience.

Some of these, perhaps, you will think of no great importance. A correct knowledge and aptitude in dealing with minor ailments, some of which are exceedingly prevalent and troublesome, will, however, help you greatly when you enter upon private practice; and, indeed, it is always worth while to do even small things well.

In the first place, I propose to speak of some disorders of childhood, many of them common, but which nevertheless are frequently misinterpreted, and treated in consequence upon mistaken lines, and therefore treated unsuccessfully.

Night Terrors in Children.—One of the most troublesome disorders, and one certainly much misunderstood, is what is known as *Night Terrors*, of which I made brief mention in the lecture on Tonics—a source of anxiety to parents, and of worry and vexation to the medical attendant.

I dare say you have not heard much about these night terrors of children. They are hardly mentioned in the text-books, and are rarely seen in hospital. Yet you will constantly be consulted with regard to them when you get into practice. They occur in children from two or three years old, until seven or eight—or even later—although after seven or eight the disturbance more often takes the form of mere fits o restlessness in sleep, talking and groaning, and occa sionally somnambulism.

The attacks usually occur in this fashion. The child, always a restless sleeper, tosses about or grates its teeth, or talks in sleep. Then, some time during the night it wakes up screaming, evidently in extreme terror. I say wakes up—but it does not wake up completely. It does not appear to be fully conscious, does not recognise its mother or nurse, or where it is, and takes no notice of their soothing talk, but goes on crying, screaming, and talking incoherently—delirious as it is termed—for perhaps half an hour or more, without anyone being able to pacify it. At last the excitement subsides, it takes notice once more, and is eventually soothed off to sleep again.

Now these night alarms after a time affect the child with a vague fear during the day, and especially as night approaches, so that it is often afraid to go to bed, or to be left in the dark, and the nurse or mother has to sit with the child until it falls off to sleep.

At first they are almost invariably put down to the much-abused thread worm, and a worm powder is given with only temporary benefit. After a time the frequent recurrence of these 'head symptoms' or 'delirium' as they are often called gives rise to great anxiety on the part of the parents. Their fears are moreover strengthened by the fact that the child is obviously out of health—languid yet excitable—has poor appetite, its bowels are out of order from constipation, and it does not thrive. The brain is thought to be affected, or in danger of becoming so, and pictures of meningitis, water on the brain, and convulsions rise up.

Children who suffer from Night Terrors are, indeed usually, but not invariably, of emotional neurotic type and stock, and the medical man who is called in, observing this, agrees that the child's brain is too active, that there may be danger of meningitis or other brain disease if the organ is unduly stimulated, and advises, judiciously enough, that all causes of excitement should be avoided. If the patient is old enough to do lessons, he usually advises also that all school work should be given up, that no lessons of any kind are to be given on any account, the child's mind to be left absolutely fallow. With this a dose of calomel is prescribed to set the liver in order, and some bromide to soothe the excited nervous system. These measures give relief, but only for a time. It is found that the symptoms return, to be relieved again by another dose of aperient, and a fresh course of bromide, and so the case goes on.

These alarming fits of incoherent, half-conscious terror are, however, in the vast majority of cases not indicative of brain disease, nor, indeed, of serious disease of any kind. They are nothing more or less than children's nightmares; bad and terrifying dreams. Anyone who has suffered from a nightmare will remember the horror and distress it causes, how one wakes up in abject dread, with beating heart and sweating at every pore. Even the bravest men are cowards when attacked by this night demon. And how real for the time the situation seems! It is with difficulty that one can at the moment bring oneself to believe it is a

mere dream. You will understand, then, how intensely a small child suffers; and, imaginative as it is, how difficult for it to realise that its terrors are unsubstantial dreams.

The night terrors of childhood are produced by causes similar to those which set up the nightmare of adults; not indeed late and heavy dinners or suppers, but some internal disturbance and irritation, having its seat almost invariably in some region of the alimentary canal. Sometimes obstruction to respiration caused by adenoid growths or enlarged tonsils sets up disturbed sleep and occasionally night terrors; but as a rule the seat of irritation is in the alimentary canal.

The brain phenomena are indeed, as a rule, purely reflex; sometimes it may be that the brain irritation is set up by toxic material absorbed into the circulation from the digestive tract. The disturbance is more rarely central in origin, yet primarily cerebral in some instances, as when the patient has been unduly excited by a pantomime, or a children's party, or a visit to the Zoological Gardens, and wakes up horribly frightened, dreaming of bears and lions.

These cases are, however, the exceptions; they do not recur unless the particular excitement is renewed. The rule is that the brain disturbance has its source in the alimentary canal, and is either reflected thence or, less probably, induced by irritant matter generated there.

If you carefully examine these children who suffer habitually from night terrors, you will almost invariably find well-marked symptoms of disordered

function in the digestive tract; the tongue coated, the breath, perhaps, offensive, the appetite poor, the patient sallow, ill-nourished, dark under the eyes, and the bowels habitually confined; never open, perhaps, without an aperient; medicine being given twice or three times a week; and the patient subject to periodical feverish attacks with headache. In some instances there is simply constipation, without disorder of health in other ways, a good appetite and clean tongue, with plumpness of body. This, however, is exceptional.

The rule is, coated tongue, poor appetite, sallow complexion, dark rings under the eyes, want of flesh, with constipation, prominent upper abdomen, and often skin eruptions of lichen papules and urticaria. Such obvious indications of gastro-intestinal disorder are most commonly attributed to the presence of thread worms. Thread worms are indeed often there, but they are a *consequence* not a *cause*. The retention of fæcal matters in the intestine with mucus and disordered secretions afford a nidus for the parasites. These in themselves are of small importance, and their influence greatly overrated. They play but a small part. Round worms and tape worms are another matter.

Causes of the Digestive Disorder.—The disordered state of the digestive tract is often due to over-feeding. Nurses and mothers are apt to press food, particularly milk, upon children with poor appetite, until the digestive organs are overtaxed; and this is especially true where the appetite fails owing to some fault of function in liver,

or stomach, or bowels; the disordered organs, instead of having their work lightened to give them a chance of recovery, have their burden increased, with the result of greatly aggravating the condition. You will see this especially in the well-to-do and luxurious classes of society.

Be the cause of the liver inaction, coated tongue, constipation, and loss of appetite, what it may, this condition is, in the vast majority of cases, the source of evil, the fault which lies at the bottom of the brain disturbance during sleep; and the proof lies in the fact that the night terrors, the talking, and tossing, and restless sleep disappear when the bowels are kept in order and the tongue gets clean. Appetite returns, the child begins to gain flesh and colour, and it thrives.

Treatment of Night Terrors.—A dose of calomel should be given to begin with and repeated once a week for several weeks in succession. In addition some saline aperient daily, such as carbonate of magnesia with rhubarb or cascara added; or the bicarbonate and sulphate of soda, with cascara to quicken it if necessary; giving the dose always twice a day, and after meals. An excellent prescription for the purpose is—

Magnesiæ Carb.	ʒvi.
Spir. Ammon. Aromat.	ʒii.
Pulv. Rhe	gr. xxiv.
Spir. Chloroformi	ʒiss.
Aquæ Menth. pip.	℥vi.

The dose being one, two, or three teaspoonfuls, according to age, and to the effect produced.

Another useful formula is—

Sodæ Bicarb.	ʒiij.
Sodæ Sulphatis . . .	ʒvi.
Spir. Chloroformi . . .	ʒiss.
Aquæ Menth. pip. . . .	ʒvi.

The dose as before, twice a day. If necessary, aromatic extract of cascara may be added to quicken the aperient effect.

Occasionally, if the nervous symptoms are severe, it is advisable at the outset to give a full dose of bromide at bed-time. This may be discontinued when the bowels begin to act satisfactorily, and the excitement subsides.

The Essential Point in Treatment.— The essence of successful treatment in these cases is to secure a free and full action of the bowels, with soft semi-liquid stools every day, without break or intermission; and this can only be effected by the daily administration of a laxative. Occasional doses of aperient once or twice a week are ineffectual; the evil is only temporarily removed, and accrues again in the intervals.

No strong purgatives should be used, or bowel tone will be eventually impaired, but alkaline saline laxatives, with the addition of the milder vegetable aperients, such as cascara, rhubarb, or senna if required.

I insist upon these main points since I find that they are very commonly neglected in practice.

As a rule, the child is given an occasional purge, and steadily dosed with bromide, which only temporarily relieves or masks the disorder; or iron and

tonics are prescribed, which in the engorged condition of the digestive tract and impaired excretion of waste, serve to make matters worse; they aggravate the liver trouble, increase headache and coating of the tongue, and lessen appetite instead of improving it.

A few weeks ago I was consulted with regard to a boy of four, who suffered from the affection in its most extreme form. The night distress was so severe, and the after excitement and dread so great, that serious mental disease was apprehended, and it had been thought advisable to take him from his family and place him apart, with a trained nurse, under conditions of absolute quiet and rest, and close watching. This produced some amelioration of symptoms so far as the general excitability and day fears were concerned; the night terrors, however, continued to recur as severely as before.

I found that the boy had a heavily coated tongue and offensive breath; that his appetite was bad, that he was pressed to eat, and that he suffered from habitual obstinate constipation—that the bowels rarely acted without medicine, and that he was troubled by so-called 'heat spots,' lichen urticatus, a symptom specially associated with bowel inaction.

The patient had been given an occasional alterative purge—always with advantage—and bromide and tonics, including iron, steadily. The general result followed; the digestive organs remained completely disordered as before, and no real progress was achieved.

Under systematic treatment on the lines I have

laid down, the bowels soon began to act daily and efficiently, the tongue cleaned, the appetite returned, the eruption disappeared, the night terrors became less intense and before long disappeared, the childish fears were gradually forgotten, and in the course of a few weeks the boy was well.

Let me remind you again, however, that although gastrohepatic disorder with constipation is the most common source of night terrors, they are occasionally due, although rarely, to post-nasal obstruction and to mental excitement. It has been stated that these nervous explosions in some instances herald the advent of meningitis or epileptiform seizures; no example of this, however, has come within my knowledge.

The Recurrent or Periodic Pyrexia of Childhood.— Another disorder having its origin in similar defective function of the abdominal organs, due to over-feeding or imperfect elimination, is what may be called the recurrent or periodic pyrexia of children. It consists in attacks of feverishness, headache, and vomiting, which occur from time to time. They are, I find, generally misinterpreted, and erroneously treated.

You will infallibly be called upon to attend these cases, which are common enough; and you will probably be greatly puzzled by them unless you learn their nature here, for little is said about them in books, and they are not often met with in hospital practice.

The symptoms and course of the affection are as follows:—

The child becomes feverish, with temperature of

101° or 102°, 103°, or even 104°; complains of headache, is sick, refuses food, its tongue coated and bowels confined, urine lithatic. At first this concurrence of symptoms suggests the onset of some serious disorder such as scarlet fever, or influenza, or meningitis. When, however, the patient is well again after a few days, and the attacks recur from time to time, it is of course obvious that this is not the case, and they are styled 'bilious attacks,' or it is supposed that the child is troubled by that *bête noire* of mothers and nurses, the comparatively innocent threadworm.

The patient is given a dose of some active aperient, and is presently relieved. The fever subsides, the tongue becomes cleaner, and the appetite in some degree returns.

In the course of a few weeks, often—as I have said, with great regularity every four or six weeks—sometimes after a longer interval of two or three months—the attack recurs. The same process of treatment is followed successfully at the moment, and again the attack passes away as before.

It is noted, however, that the child does not enjoy full health, is fretful, is subject to headaches; is sallow, often ill-nourished, with dark rims under the eyes and poor appetite. The tongue is coated, the bowels constantly confined. The child does not thrive; it is given tonics, therefore, as a matter of course, and cod liver oil, and 'fed up'—usually overdosed with milk.

Now here again the whole secret of the condition lies in the fact that the functions of digestion and excretion are imperfectly performed.

Although, however, this faulty state of the chylopoietic function is seen to be connected with the 'bilious attack,' or 'bilious fever' as it is termed, its true significance as the 'fons et origo mali' is imperfectly appreciated. The good effected by a calomel purge does not escape notice, and this is repeated from time to time when an attack threatens.

This intermittent administration of an active aperient, however excellent in its place, is not alone sufficient. It allows the evil to recur and then remedies it. For every step forward there is a slide backward. Occasional purges do not prevent, they only delay the return of the disturbance.

Moreover, the plan commonly pursued in the intervals between attacks, tends to make matters worse.

As I said, the patient is usually sallow, ill-nourished, anæmic, and of poor appetite, and to amend this the child is dosed with tonics by mother and doctor. The former gives cod liver oil, Parrish's food, the latter almost invariably prescribes iron and quinine, and both are agreed to try to feed up the patient. The result is further embarrassment of the liver, increased coating of the tongue, lithatic urine, perhaps of high specific gravity, 1020 to 1035, and aggravation of headache, precipitating thus the attack of 'bilious fever,' which it is the object to avoid. The mistake arises from a false conception of the pathological state present. The cart is put before the horse —the consequence deemed the cause.

The headaches and want of appetite are referred to

anæmia and malnutrition, whereas, all these conditions are the direct result of the gastrohepatic and intestinal inefficiency, imperfect elimination of waste material from the bowels, and imperfect preparation of nutritive material.

It would appear that owing to this deficient discharge of waste from the bowels, there is a gradual accumulation of peccant substances, which remain in the blood or are reabsorbed into it; a fæcal poisoning. Hence in the course of a week or two this accumulation of toxic matter, when it reaches a certain point, produces the symptoms described; viz. the headache, and fever, and vomiting, of the periodic attacks. The result is closely analogous to that which occurs in granular kidney.

Treatment of Recurrent Periodic Pyrexia.—Now the rational plan of treatment to remedy this state of things is not that almost invariably adopted of allowing the evil to culminate, then to remove it for a time, foster it again by full feeding and tonics, and again remove it.

This is to establish a vicious circle which may run on indefinitely.

The way to deal with it is to prevent its recurrence by never allowing such accumulations of toxic materials to arise. This can only be done by providing for the due removal of fæcal matter from the bowels day by day. This is effected by the regular daily use of some simple alkaline saline aperient such as rhubarb and magnesia, or soda bicarbonate and sulphate, aided by cascara if required, or some other laxative of the like kind.

The daily mild aperient is what is wanted, not the occasional strong aperient once or twice a week, although a dose of calomel at the outset and once a week afterwards, for a time, is a most useful adjunct, and, indeed, necessary in obstinate and extreme cases. Iron, quinine, strychnia, or other tonics may be added to the saline laxative if anæmia or debility persist.

In this way, gentlemen, with the aid of proper dieting, you will, I think, never fail to cure these recurrent attacks of fever, nausea, headache, and perhaps, vomiting, so prevalent in growing children.

Another affection associated with, and dependent upon this same deficient elimination and intestinal irritation, marked by chronic obstinate constipation in children, are the skin eruptions Lichen Urticatus, Prurigo Mitis, Lichen Prurigo, the 'heat spots' of mothers and nurses.

The eruption consists of hard papules, scattered over the body, most numerous on the back, arms, legs, feet and hands, and often associated with it are the wheals of urticaria. The irritation is great, the itching sometimes being so severe as to seriously interfere with sleep and affect nutrition. This eruption is usually attributed to purely local causes, such as the irritation of flannel, the bites of fleas or other parasites, or a previous eruption of Varicella.

This is an error; local irritation may play a part in some instances, but the affection occurs not only amongst the poor, but quite as often, or more often, amongst the well-nurtured children of the rich, clothed in fine linen, and innocent of any parasitic irritations

beyond a most occasional flea bite, and who have never suffered from Varicella. The irritation of skin, as in the previous disorder I have described, is reflex or toxic, produced by irritation of the alimentary tract, or by waste matter absorbed therefrom into the blood.

It is set up by simple constipation—or by food containing indigestible materials, such as oatmeal, figs, raisins, jams containing figs and seeds, and particularly and almost invariably when these articles of food have been given for the relief of constipation.

I find that these ailments, after the fashion of treating all skin eruptions, are usually given arsenic or iron, which, interfering with elimination, increase the eruption and the irritation.

Local applications are used, which give little or no relief; then cod liver oil and steel wine and maltine are tried without better success; and the almost invariable result is that the medical man is driven to the unsatisfactory statement to the parents that nothing can be done further, but that they must wait patiently until the child grows out of it in the course of a year or two.

As a matter of fact the eruption is easily curable by systematic regulation of the bowels by daily alkaline aperient, after the manner I have previously laid down, so as to secure full daily slightly relaxed motions, together with the avoidance of irritant stimulating articles of diet, such as the oatmeal, prunes, figs, raisins, and the like, to which I have referred, and which are so constantly given with the view of aiding the action of the bowels.

LECTURE IV

CHRONIC CONSTIPATION AND DILATATION OF THE COLON

The importance of the subject—Usual methods of treatment—The essential principle to be observed—The three main factors in the production of constipation—Important causes—The symptoms arising from constipation—The symptoms classified according to their severity into four groups—The lines of treatment—The four modes of action of purgatives—The importance of the first three of these in rational treatment—Salines, their action and value—In severe cases drugs also needed to increase muscular tone—Strychnia—Belladonna—Dinner pills—Massage—Electricity—Enemata—Diet—Fluids—Exercise—Fresh air—Examples of treatment—Constipation with dilatation of the colon in childhood—Examples illustrating the treatment of the condition—Sometimes due to partial obstruction—Explanation in some cases obscure—Condition though pronounced may be curable—Severe constipation—The symptoms may simulate those due to organic brain lesions or tuberculosis—The value of simple mild aperients in the treatment of disease.

You will perhaps think that the subject which I have chosen to-day is commonplace and of very secondary interest and importance. Moreover, I treated of it in some of its aspects in a previous lecture. There is, however, more to be said on this matter.

Constipation is no doubt a common condition, and its treatment at first sight seems very simple and straightforward. Every doctor thinks, I imagine, that he knows how to treat constipation, but my experience on the point hardly warrants this assumption. Truly, if there is no mechanical obstruction to the bowel, nothing is usually easier than to force a free passage

through it by calomel, or colocynth, or jalap, or castor oil, or croton oil; if one purgative will not do it another will, or an enema will wash out accumulations from the colon. But this is not the whole question—the matter is by no means so simple. The mere exhibition of a purge is a very limited portion of the management of a case of constipation when chronic or recurrent.

The temporary relief of the bowels is merely a first step. If the constipation is only a chance and exceptional difficulty, a free purge may end the trouble and cure the complaint. But if the constipation is *recurrent, habitual, obstinate,* it is necessary to go deeper in order to effect a cure, viz. to alter the conditions which give rise to the torpidity of the bowels. The very ease with which the immediate difficulty can be temporarily overcome, is in itself a constant source of erroneous treatment. The knowledge that a brisk purgative will set the matter right for the time frequently tempts both the patient and his medical adviser to be content with this remedy, and to rely upon its repetition from time to time, as a sufficient means of meeting an ever-recurrent trouble.

For if the bowels become confined, what is there to be done except to give a purge and open them?

Yet the relief given by a brisk purge, if frequently repeated, tends to defeat its own end; and to retard, not hasten, ultimate cure. The various purgatives lose their power after a time. Stronger and stronger drugs have to be resorted to; the constipation grows more and more difficult to overcome; and at length the

bowels refuse to act altogether without the stimulus of powerful medicines or enemata.

The colon, its muscular wall enfeebled, and rendered atonic by over-stimulation, and habitually distended with fæcal accumulations and gases, and perhaps repeated copious enemata, becomes greatly dilated, lengthened, and tortuous; and the last state of the patient is apt to be worse than the first. The attempts to cure have aggravated the condition.

I have been surprised to find what a purely routine and rule of thumb practice prevails in the treatment of this disorder.

Three devices only seem to be usually adopted, viz. first, administration of purgatives, and usually stimulating purgatives from time to time, sometimes two or three times a week; secondly, the use of repeated enemata; and thirdly, the use of foods which owe their virtue to the fact that they contain a certain amount of indigestible material such as coarse oatmeal, cabbage, prunes, figs, and other fruits.

For slight and casual constipation these remedies are occasionally sufficient; in real chronic obstinate constipation, however, they invariably fail. Adults are as a rule much more sensibly treated than children, they are given perhaps a daily dinner pill, or, better still, sent to drink laxative saline waters at some suitable spa. Children, however, rarely or never get this chance. The treatment is almost invariably limited to the three routine devices, viz. the casual use of purgatives, enemata, and stimulating diet.

I hope to show you presently a more efficient and reasonable plan.

Now the permanent cure of constipation is of great importance to health and absolutely essential to the comfort of life.

In the case of adults habitual constipation and the necessity for constant resort to drugs are sources of trouble and worry, and of disordered function in other organs; of pain and discomfort, and often of impaired health from chronic fæcal poisoning, while not unfrequently serious or even fatal complications arise, such as obstruction, or ulceration, and even perforation of the gut from the pressure and friction of hardened fæces.

In children, the question is in certain aspects more serious still; for it affects not only present health and comfort, but robust development, and the working conditions of the alimentary canal for the whole of after life.

As a general proposition, it may be affirmed perhaps that adults suffer most from the consequences of neglect of constipation, children from the too vigorous and mischievous treatment of it; for here mothers and nurses come in as well as the doctor.

The treatment of chronic constipation is then a subject of considerable importance, and deserves more attention than it usually receives. It lies for the most part outside hospital practice. It is a frequent source of trouble to the practitioner, and sometimes of discredit to him; and if I can help you to deal with it

satisfactorily, it is a service for which I believe you will be grateful hereafter.

In the first place then, if treatment is to be successful it must comprise something more scientific and comprehensive than the mere administration of occasional aperients.

As I have said, it is not enough to open the canal when blocked, or sweep it clear of accumulations which have lodged there. The essential point is to establish, if possible, such a habit of daily evacuation that accumulation of noxious waste and its evil consequences may not recur; to modify the condition *permanently*, if the age and condition of the patient render such a consummation possible. In order to do this the causes which give rise to it must be thoroughly understood, removed, neutralised, or compensated.

There are many different factors concerned in the production of constipation, and these act in varying degrees in different cases. Three main conditions are necessary to the proper action of the bowels, viz.:

1. Sufficient fluidity of the intestinal contents, so as to permit their easy propulsion through the tube.

2. Sufficient peristalsis and muscular action to effect such propulsion.

3. An unobstructed condition of the intestinal tube so as to allow free passage.

Now these essential conditions are interfered with and rendered imperfect and ineffectual by several causes.

Take the *first* essential condition, for instance, the

deficiency of fluidity of contents of intestine; this may be due to increased excretion of water through the lungs, the kidneys, and the skin, unless the loss is compensated by abundant supply in drink. Liquids are absorbed from the alimentary canal with remarkable rapidity to make up for the loss by excretion. This diminishes the fluid poured into the intestine by osmosis and secretion; more liquid being taken out and less poured in. Thus the contents of the intestine become drier and therefore more difficult to pass on. Constipation arises from this cause largely in—

1. Training for athletics, when there is violent sweating and little drink allowed, although it partly arises also from increased oxygenation, which tends to lessen peristalsis.

2. Sweating diseases, *e.g.* acute rheumatism, phthisis.

3. Diabetes, when there is great drain of water through the kidneys.

4. Nursing women owing to the drain of fluid by lactation.

5. Loss of fluid by vomiting, as in stricture of pylorus, and the like.

6. *Deficient supply of liquid in the food.*—This tells chiefly in the case of children, who require a large quantity of water, but who, after infancy, are frequently greatly restricted in amount by mothers and nurses, through prejudice and mistaken views of physiology.

7. *Imperfect secretion of the glands of the mucous lining of the intestines.*—This is aided by the use of

astringents, as, for example, the free use of astringent wines, or, in the case of children, of lime water or hard drinking water. It is also a result of the reaction after the flux of intestinal catarrh.

The want of fluid is, however, a comparatively minor cause of constipation, except perhaps in the case of imperfect secretion of the glands of the mucous lining.

The want of the second essential condition, viz. *efficient peristaltic action*, is more frequently the source of trouble; when the contents of the stomach, liquefied by digestion, enter the duodenum they are gradually dried in their passage through the canal by absorption of the nutrient liquids.

When *unduly delayed* by inefficient peristalsis the result is like that caused by insufficient secretion: they are desiccated; become hard and more difficult to move, and are consequently detained in the sluggish large intestine.

Now the causes of this defective peristaltic movement are numerous, and often complex. One is a lack of sufficiently stimulating property in food; *e.g.* food which contains little residue (as milk and other extremely digestible foods), or when the stimulating acids of food are neutralised by hard water, or lime water. The constipation so common amongst milk-fed children is an example of this. Too great uniformity of food acts in the same direction.

A more potent cause of impaired peristalsis, and I think the most common cause of all, is dulling of the

reflex excitability of the intestine, so that it fails to respond to the normal stimulation of its contents.

There are numerous agents which effect this by diminishing motor nervous influence, or exciting inhibition through irritation of the splanchnic, the great inhibitory nerve of muscular movement of the intestine —as, for instance, lead, opium, meningitis, myelitis, or other organic affections of the brain and cord or nerves, or depressing functional nervous disorders such as hypochondriasis and hysteria. Another cause lies in the enfeebled condition of the intestinal muscle in anæmia and debility.

By far the most frequent source of impaired reflex excitability of the intestine, however, are prolonged inaction on the one hand, and over-stimulation by strong irritating purgatives, or the too frequent use of enemata on the other, so that it becomes deadened, and ceases to respond to the ordinary stimulus of food.

The effect is constantly aggravated by dilatation of the gut, chiefly of the colon, due to distension by accumulations of fæcal matter and the gases of fermentation, and in some cases by the constant use of repeated large enemata. The over-distended bowel loses its contractile power, just as the other hollow viscera, the stomach and the bladder, do in a similar condition, this being aided by the atony and degeneration of muscular fibre, which eventually results from pressure and want of use. These conditions are further complicated in the case of old persons, and women who have borne children, by laxity and enfeeblement of the

abdominal muscles, and in the case of little children by wilful resistance to evacuation, from dread of the pain of passing large hard masses through the anal ring.

The third condition essential to free action of the bowels, the absence of mechanical obstruction in the gut, is interfered with in the case of congenital atresia ani, the partial plugging of the rectum by the fibrous overgrowth of piles, or the stricture of the bowel by cicatricial contraction after ulceration in dysentery, or by adhesive peritonitis, or by malignant growths.

These special cases I shall pass by as largely surgical, and limit my remarks to constipation due to functional causes and its results.

The *symptoms* produced by chronic constipation are many, although they are by no means present in every case. They consist of flatulence and distension, possibly dyspnœa, palpitation, faintness, and vertigo in consequence and headache.

In addition, coated tongue, offensive breath, want of appetite, sometimes vomiting, dry hard stools, and in children often thread worms. Sensations of heat and cold in the limbs, numbness of legs, pains in the back, sacrum, and thighs, abnormal sensations in the genitals due to pressure on the posterior nerve roots and plexuses. Similarly varicose veins may appear in the legs owing to pressure on the iliac vessels.

Intermittent pyrexia is another symptom due to fæcal poisoning; and lastly, depression of spirits or hypochondriasis supervenes, or, in the case of children, night terrors—sometimes of such extreme form as to simulate cerebral disease or mental aberration.

As I have said, all these symptoms are not by any means present in every case.

Nay, there may be absolutely no symptoms at all, except the simple inaction of the bowels. This, however, is the exception. The cases may be roughly divided into four classes :

1. Those in which the health of the patient is apparently unaffected. They are comfortable, of fair appetite, well nourished, occasionally uncomfortable but otherwise bright, but are usually of sallow look, and have dark rings under the eyes.

2. Those cases where there is slight disturbance of general health—the stools are light coloured, dry, and mortar like ; there is some loss of appetite, coating of tongue, languor, headache. The patients, if young, are of yellowish sallow complexion, with dark rings under the eyes. There may be no vomiting, no emaciation. In adults, there is usually more or less hypochondriasis. In children, night terrors, of which constipation is the most common source, and the transverse colon under the false ribs may be seen obviously dilated and prominent, the false ribs splayed out, the stomach displaced upwards, and the heart's apex pushed up to the fourth space or nipple.

3. The third class comprises the cases with similar symptoms, but where the attacks of headache and vomiting, with coated tongue, offensive breath, and a rise of temperature to 100° or 102° or more, are chiefly *periodic*, occurring at intervals usually of a few weeks. They are due, no doubt, to poisoning by waste products

and styled sick-headaches or bilious attacks. Although these attacks are periodic, the coating of the tongue and constipation are usually persistent and continuous, but worse at the period of culmination. The colon is commonly greatly dilated, the stomach and heart displaced upwards. This class of case is met with both in children and adults.

4. The fourth class of cases includes those with graver symptoms still, when the vomiting and headaches are more severe, when there is often great emaciation and impairment of nutrition, with nervous disturbance, such as depression of spirits, hysteria, hypochondriasis—in children night terrors of extreme form. Such cases are frequently diagnosed erroneously as primary hysteria, cerebral tumour, meningitis, or even mania.

Treatment.—In the first place, then, all merely occasional or intermittent treatment is futile as far as cure is concerned.

To give purgatives two or three times a week even, simply removes the accumulations which have arisen in the intervals. It gives temporary relief, but does not cure the habit; on the contrary, if the purgatives used are of an irritant or stimulating character it increases it, as I have said, by dulling the reflex excitability of the bowel, so that after a time the gut will not act without strong stimuli.

The *first rule*, then, in the treatment of habitual constipation is that it should be *continuous*. The bowels must be *kept* free. The usual practice of

waiting until they fail to act before giving an aperient is useless for purpose of cure. There is always one step backwards for the step forwards. The important point is never to let arrears accumulate and distend the bowels. This essential of continuous treatment and daily action of the bowels is most imperfectly recognised. The remedies given must be administered daily for a considerable period, and you must warn your patients that progress will be slow, and that if they wish for a successful issue they must be prepared to persevere for weeks or months, or, if the case be one of great obstinacy, for even a longer period still.

The *second rule* is that these remedies should be as little irritating and stimulating as possible. Purgatives, remember, produce their effect through four different properties or influences :—

1. By increasing the secretion of the intestinal glands and producing osmosis *into* the intestinal canal.

2. By retarding absorption of fluid *from* the intestinal canal.

3. By giving tone to the intestinal muscle.

4. By exciting peristaltic movements.

We should endeavour in these cases to obtain the regular daily evacuation of the bowel by the first three means, viz. by increasing the flow of fluid into the canal, by retarding absorption from it, and by giving tone to the muscular structure of the bowel, rather than by exciting peristalsis by the direct stimulation of irritant drugs. For this purpose the saline laxatives, the carbonate and sulphate of magnesia, the phosphate

and sulphate of soda are of great service; they excite discharge of fluid from the glands and vessels of the small intestine.

The flow proceeds until there is water enough to bring down the solution of salts taken to 5·6 per cent. When a 20 per cent. solution is used (= 96 grains, or about ʒj$\frac{1}{2}$ to the $\bar{3}$j) this is calculated to take from one to one and a half hour.

Salines act by arresting absorption as well as by inducing flux—prevent fluid from being taken out of, as well as promote the flow into, the canal. The low diffusibility of the salt impedes the absorption of secreted fluid.

Now for the practical application of these rules. In the first place, before beginning systematic treatment, it is well to clear the bowels thoroughly by calomel, or a good dose of castor oil.

After this, in the case of adults, the best method is, no doubt, to send them to some spa, such as Kissingen, when possible, to go through a course of aperient waters there. This plan is, however, for obvious reasons, only available in a few cases; the majority of patients cannot afford it; and it is necessary to imitate the action of the waters by medical prescription. The secret of the curative effect of these waters appears to be chiefly the daily administration of dilute saline solutions in quantity. We cannot do this under the conditions of home treatment, but we can give a daily saline mixture, *e.g.* half a drachm to a drachm each of carbonate and sulphate of magnesia, or the same

salts of soda with some carminative such as spirit of chloroform, ginger, and peppermint water twice a day after meals. A similar prescription answers well for children, the dose being proportioned according to age and effect. For children of one to two years, however, five to ten grains or more of the heavy carbonate of magnesia, given with two or three minims of spirit of chloroform and some syrup in one to two drachms of water, is usually an acceptable and efficient aperient. This, or similar doses of phosphate of soda, should be given every morning, or twice a day; whilst for very small babies the daily use of the ordinary fluid magnesia for a time is usually all that is required.

Salines, however, will not always act alone. They possess little power of exciting peristalsis; the fluid stools which they induce pass down the intestinal tube chiefly by the force of gravity, with the aid of ordinary peristalsis; so that it happens sometimes, especially with grown persons, and in cases when the colon is dilated or atonic, that the liquid contents of the bowel, passing readily down the more actively peristaltic small intestine, are retained in the inert and sluggish large intestine, and there re-absorbed. The purgative which had given promise of acting fails, and in such conditions salts alone are liable not only to fail, but to cause considerable griping pain, distension, and discomfort.

In mild cases of chronic constipation then, although simple saline laxatives alone are usually sufficient—if they are pushed as they should be, until they produce

one or two loose actions daily—it is often necessary to give something in aid, at any rate at the outset, and in this relation strychnia and belladonna are of the greatest service, especially the former. These drugs act through the nerve centres of the spinal cord, increase the contractile power of the muscular coat, and aid peristalsis by improving tone, without impairing reflex excitability like irritant purgatives.

An excellent prescription for an adult is half a drachm to a drachm of sulphate of magnesia or soda (or even more), with three to five drops of the liquor strychniæ and three drops of dilute aromatic sulphuric acid, with ten drops of spirit of chloroform in half an ounce to an ounce of peppermint water, given twice a day. The mixture should be given after meals, since it acts more gently when taken on a full stomach. To this may be added three to five drops of the tincture of belladonna, or more on occasion. In appropriate cases sulphate of iron or quinine may be substituted for the belladonna.

When dyspepsia is present and it is desirable to give the *carbonates* with the sulphates in alkaline mixture, tincture of nux vomica may be substituted for strychnine.

In very obstinate cases, however, salines, even with the aid of strychnia and belladonna in full doses, will be insufficient; and it may be necessary to give, in addition, at first, some purgative of stimulant action. For this purpose cascara, or euonymin, is the mildest and best; but if they should prove insufficient it may be

necessary to give rhubarb, senna, or aloes. One of these with the tonic saline should be given *for a time only*, and the object of endeavour to gradually reduce them and get the bowels to act by salines and nerve tonics alone. In cases of the temporary constipation of anæmia, aloes and iron may be given with salines and strychnia.

To ensure success, to change the bowel habit, remember it is essential, not only to keep the bowels open every day, but somewhat loosely once a day, for some time. A daily evacuation, if scanty and confined, will lead to no permanent recovery.

In the case of grown persons a mild dinner pill may be substituted for the saline mixture, as more convenient and agreeable, as for example one of a grain each of euonymin and cascara, with one quarter grain of extract of nux vomica, to be taken after each meal.

Or, if this is not sufficient, a grain to two grains of rhubarb, or half a grain to a grain of glacial extract of aloes, may be substituted for the cascara. The treatment by pills of more or less stimulating drugs is far less satisfactory, as a means of *cure*, than the saline method; but it is not easy to get busy adults to take mixtures steadily; and if a course of mineral waters at some spa is not available, daily dinner pills, or a regular pill every night, may be the only plan practicable.

The treatment I have laid down is applicable to those cases in which cure may be hoped for eventually.

Children are always curable no matter how

obstinate or extreme the condition may be; young adults nearly always; persons of mature age not unfrequently; old people rarely or never. In the case of the latter then there need never be any scruple about resorting to appropriate aperient pills without attempting cure by the saline method, which in such patients almost infallibly proves a failure.

Massage.—The action of aperients is in all cases greatly assisted, and cure, where it is possible, hastened, by massage of the abdomen. It should be applied along the track of the colon from the right iliac fossa to the left, and the abdominal muscles kneaded. Massage is especially useful when the colon is dilated and atonic, and when the abdominal walls are flabby and relaxed. In the latter case, in elderly patients, a firm elastic abdominal belt should be worn to support the pendulous belly.

Electricity properly applied is useful in atonic cases, but it is unsuited to the case of children who dislike and dread it, and it is, according to my experience, inferior in its results to skilled massage.

Enemata, again, are useful in clearing the large bowel on occasion. But copious injections are to be regarded as *temporary* measures merely, to be resorted to in order to get out of a pressing difficulty, not as a means of cure. Their frequent use indeed does harm. It eventually causes dilatation and want of tone, so that after a time the bowel refuses to act without some assistance of the kind. The same is true, although in less degree, of small glycerine enemata, which appear

to act partly by causing flow of fluid, partly by stimulation, and the piece of soap so popular with nurses in the case of infants comes into the same category. They should not form a part of regular treatment. Small enemata of cold water, however, have a useful tonic action.

Diet is a most important agent in the cure of chronic constipation, yet one great mistake is almost invariably made with regard to it. Foods which contain large amounts of indigestible material, such as oatmeal, brown bread, figs, prunes, raisins, currants, jams, are almost universally ordered, with the object of stimulating the bowels to act. Now these means are not often effective alone. If they *do* act, well and good ; but if they do *not* act—which is the rule—they increase the difficulty by adding so much insoluble material to the accumulation of fæcal matter. These articles, therefore, should never be persisted in if the bowels are not acting freely. The diet should, however, embrace a good variety of food in which fruits and soft green vegetables form a considerable part. A monotonous highly digestible dietary lacks stimulating properties. Astringent drinks such as coarse clarets or burgundies and tea should be avoided, white wines and coffee being preferred to them.

Moreover especial care should be taken with regard to drinking water. Hard water should be avoided ; most drinking water contains lime in some form ; this is astringent, and in some chalk districts it affords a perpetual draught of chalk mixture, which adds greatly

to the constipation, and indeed it is often the chief cause of it.

Nevertheless a full quantity of liquid is essential. Salutaris water or Apollinaris water, pure distilled water, or seltzer water may be taken freely: a glass before breakfast makes a good beginning for the day.

Exercise, especially active exercise, such as riding, cycling, or outdoor games, is of service; quick movements aid the flow of bile into the intestine, and tend to shake down the contents of the bowel: exercise gives vigour and tone to all muscles and to those of intestine and abdomen amongst the rest.

Fresh air, again, is another useful agent, although physiologically *hyperoxygenation* tends to *lessen* peristalsis, as *carbonisation* of blood tends to *increase* it, hence perhaps the constipating effect of change of air, especially to the sea, where ozone is plentiful. By helping the vigour and full nutrition of the body it must, however, eventually have a favourable effect upon the muscles of the intestine and abdominal wall, and thus further efficient peristaltic action in the end.

Having thus placed before you, gentlemen, the principles and general methods to be adopted in the treatment of chronic constipation, let me illustrate the subject further, by relating some special cases, which exhibit the affection in its different phases.

The first is a simple one, of common type; one which you will come across constantly in ordinary practice.

M. H., a girl of eighteen, came to me in February

1894 complaining of obstinate constipation, with which she had been troubled several years. I learnt that for this she had, as usual, consulted many doctors and taken almost every known aperient. In spite of this matters had gone from bad to worse, so that on one occasion the bowels had not been open for three weeks. At the time of her visit they had not been open for upwards of a week, and this was a not unfrequent occurrence. Her tongue was much coated, she had a bad taste in her mouth on waking in the morning, tainted breath, and a poor appetite, the latter condition being the one which most distressed her parents and friends. She was conscious of no other ailment she said, but I found that she was anæmic, languid, indisposed to exertion.

On inquiry I learnt that the two common errors which beset aperient treatment had been made in her case. The *first* was that stimulating drugs such as aloes, podophyllin, colocynth and the like had been used in frequent repetition, and the *second* that the dosing had been intermittent, the aperient resorted to at intervals when the bowels had not been open for some days.

After a preliminary clearing of the bowels by a brisk purgative, the patient was given an aperient mixture containing a drachm and a half of sulphate of magnesia and thirty drops of liquid extract of cascara in each dose, twice a day after meals. This, however, proved quite insufficient, and the quantities were increased until the effective dose was reached, viz. 2½

drachms of sulphate of magnesia with forty-five drops of liquid extract of cascara and five drops of liquor strychniæ twice a day. This produced a full liquid action daily; and a remarkable improvement in general health quickly followed, without the aid of massage, which she could not afford, or any other adjunct. Appetite returned, languor disappeared, and, for the first time for several years, the girl found herself in such physical weil-being as to enjoy life keenly.

The doses were reduced very gradually. It is instructive to note that it took nearly four months, after many futile attempts, before the quantities could be reduced to one and a half drachm of salts and thirty drops of cascara, and eighteen months before the medicine could be left off altogether. Now she is quite well, rejoicing in freedom from all need of the pills and draughts from which she suffered so long.

The preceding case, gentlemen, is an excellent sample of numberless instances of like affection which have come under my observation, and which you will meet with frequently in your experience. I might adduce others in which the more severe symptoms such as headache, vomiting, pain in the back, were prominent, but the essential condition and principles of treatment are the same in all. I might also relate to you many instances of chronic constipation in old persons, incurable, often fatal through complications of fæcal poisoning and obstruction. These, however, are of less active interest because, although much can be done to *modify* and *relieve* the condition, the dis-

order has passed beyond the stage at which cure is possible. I cannot describe these in detail here. I will give one note of warning, however. When obstinate constipation occurs in an old person, remember that the colon is probably greatly dilated, empties itself with extreme difficulty, and requires daily assistance by aperients or enema to accomplish it. The situation is always one of lurking danger from blockage. If obstruction arises in such cases, the repeated use of large enemata of olive oil with castor oil; massage, castor oil internally if it can be retained, and the stoppage of all food by the mouth except water and strained beef tea, are the most important items of treatment.

I pass on to cases of obstinate constipation which begins in early life. In these complete cure during childhood is of the first importance to the health, and vigorous development and future well being, of the growing organism. They present also special features of their own, notably that great distension of the upper abdomen due to dilatation of the colon. It is recognised by the prominence and extensive tympanitis of the transverse colon lying between the umbilicus and the false ribs and the stomach above; the swelling outline of the transverse colon is often very noticeable and in marked contrast to the normal condition of the region of the small intestine below. This prominence of the region of the transverse colon is a distinctive feature of the condition. This dilatation of the gut moreover splays out the lower ribs, forces the stomach and the

heart upwards, and in extreme cases so far displaces the latter that the apex is found beating in the third interspace above the nipple, to its great embarrassment. The functions of the lungs and stomach are likewise seriously interfered with by the upward pressure. Instances of such displacement are not uncommon. I have notes of many. In one instance (Frank F.) the distension was so considerable that a broad elastic waistband had been applied by the medical attendant to combat it.

In another instance, in a child of three years (C. M.), constant distension had set up dilatation of the colon, which caused transverse swelling of the abdomen above the umbilicus, in the epigastrium and under the false ribs, so that the tympanitic note of the bowel and stomach extended to within half an inch of the nipple, and the heart's apex was thrust upwards; beating exactly under the nipple itself. This state of things had been brought about by obstinate constipation which had lasted since the child was three months old, and the means made use of to relieve it—which did not lack vigour. Every known purgative had I think been given at one time or other. Here is a list the mother gave me; by no means an exhaustive one; just what she could remember at the moment: Castor oil, grey powder, calomel, senna in various forms, scammony, jalap, podophyllin, Steedman's powders innumerable, Gregory's powder, belladonna, rhubarb, cascara sagrada.

This unfortunate child had gone through all this before it was three years old.

Yet this was not the worst.

Every drug in turn failed after a time, and latterly, for more than a year indeed, copious enemata had been given almost daily. If the first was retained a second was given, occasionally a third, the first two being retained; large enemata of half a pint to a pint; not unfrequently all three were retained, so that the gut, already filled by accumulated fæces and gases therefrom, was further forcibly distended by repeated copious enemata. No wonder the large intestine was found dilated and atonic as I have described!

By continuous treatment on the lines I have indicated—strychnia, belladonna, salines and massage—the bowels began to act steadily, and in two months' time the distension had subsided, and the boy eventually made a complete and permanent recovery.

I have, however, seen cases more extreme still, for example, that of a boy five and a half years old (Arthur H.), who had suffered from obstinate and extreme constipation for nine months. The same vicious plan of intermittent treatment by repeated strong purgatives had been followed, until every drug failed to act. *Then*, as usually happens, copious enemata were resorted to, until at last they had little effect, only acting after being repeated for days together, large volumes of fluid being retained. At this juncture the boy was brought to the Children's Hospital. The abdomen was enormously distended above the umbilicus, in marked contrast to the lower portion; excessive drum-like resonance extending across as high as the nipple line. The

abdomen measured 21¾ inches in circumference above the umbilicus. The heart's pulsations could be seen in the first, second, and third left spaces; the apex was just above and outside the nipple; the patient was suffering from constant colic, with loud rumbling noises, apparently due to the action of ineffectual purgatives and enemata.

A bandage was firmly applied to the upper abdomen, with daily massage along the track of the colon; opium and belladonna were given with cardamoms and ginger to relieve the colic, and the bowels solicited by small castor oil enemata.

But the plan did not answer, the bowels refused to act. The boy became excessively anæmic and feeble, the pulse smaller and smaller, the face cyanosed, the fingers blue; râles appeared in the lungs, dyspnœa became urgent, vomiting supervened. The upward pressure of the dilated colon, which increased rather than lessened, was producing serious results upon heart, lungs, and stomach; extreme distress supervened, and signs of cardiac failure, with rapid flattening pulse; fatal collapse seemed imminent.

Under these circumstances it was decided to give relief by puncture of the transverse colon; and this was done in two places by a thoroughly carbolised hypodermic needle. Gas issued freely—a large quantity escaped—the distension went down, and immense relief followed. The extreme distension and grave symptoms were removed, and under tonic and laxative treatment, with prolonged massage and galvanism for

some months, and after a succession of relapses, the boy eventually perfectly recovered; and what is more, he *remained* well when seen five years afterwards. The question has been raised whether these cases of great dilatation of the colon in young subjects are not always due to some congenital narrowing of the gut near the anal extremity. No doubt in a number of cases this is so. In a case of partial atresia ani under my care, post-mortem examination disclosed a colon so enormously dilated as to fill up almost the whole abdomen. On the other hand, a number of cases have been recorded in which no stricture of any kind could be discovered, as in one published in the 'British Medical Journal,' July 29, 1893, by Dr. Walker, of Peterborough. In this instance distension was enormous, the transverse colon being 16 inches in circumference, but no obstruction was found on post-mortem examination. Similarly in a case recorded by Dr. Rolleston and Mr. Haward ('Lancet,' May 30, 1896) no organic obstruction could be detected—and so in numerous other instances—in both old and young subjects. In all instances obstinate constipation was a leading feature, dating in many cases from early infancy. The only question is as to the part played by the constipation. That it is one factor by causing distension there can be little doubt. Whether in the case of children some congenital deficiency of the muscular structures of the intestine—or intestines—or degeneration from continued mechanical pressure and disuse may not be also concerned is uncertain. In the case of old persons the intrusion of

the latter factor is most probable. At all events, dilatation of the colon in varying degree is common in childhood as a result of chronic constipation, and is curable by appropriate treatment. Moreover, the remarkable instance of it which I have related, in which, although the dilatation was so enormous as to endanger life, the patient made a perfect and permanent recovery, proves that even in these pronounced conditions constipation may be the sole cause without any organic congenital deficiency behind it.

Cases of nervous disturbance stimulating actual cerebral disease, when the vomiting and headache are severe, and there is marked emaciation, occur more rarely ; an example of the kind is afforded by the case of a boy who was sent into this hospital a short time ago as suffering from cerebral tumour. He had vomiting, headache, constipation, and extreme marasmus. A few doses of calomel and a daily laxative restored him to good health in the course of a few weeks. I have met with several other cases of the kind when the headache and vomiting and wasting have led to a like mistake in diagnosis. Sometimes the nervous phenomena are wanting. In another instance which came under my care, in a girl of twelve, the emaciation and malnutrition developed were so extreme that the diagnosis of tubercular consumption had been confidently made. There was no physical evidence of this but the contrary, and the cure of an obstinate constipation restored the patient to perfect health. Similarly only a short time ago, a case of supposed tubercular peritonitis

yielded to similar treatment. There must of course in these cases, where emaciation and malnutrition are so marked, be something more than mere retention of fæces in the colon. There is probably imperfect intestinal digestion and absorption also, possibly as the result of retained waste and mechanical pressure. Be this as it may, cure of the constipation by the measures I have laid down, cures the malnutrition.

No drugs have, I think, brought me more credit or earned me the cordial thanks of patients than these simple aperients.

You may perhaps remember the well-known lines inscribed on the tombstone in Cheltenham Parish Churchyard :

> Here lies I and my two daughters,
> Killed by drinking Cheltenham Waters.
> If we had stuck to Epsom Salts,
> We shouldn't have been lying in these here vaults.

I cannot indeed endorse this severe indictment of the Cheltenham Waters ; but the last two lines might, perhaps, with slight alteration, serve as a suitable epitaph in certain cases :

> If I had had more Epsom Salts,
> I shouldn't be lying in these here vaults.

LECTURE V

RICKETS IN ITS MEDICAL ASPECTS

Rickets a general disease, not a disease of the bones alone—It affects also muscles, bones, ligaments, mucous membranes, blood, viscera, nervous system—Evidence of implication of these structures—Importance of the nervous disorders of rickets—Their characteristics—Reflex hyper-excitability due to defective nutrition of structures not yet fully developed—Laryngeal spasm—Tetany—General convulsions—Treatment of Laryngismus stridulus—Immediate - Prophylactic and radical—Tetany, its clinical manifestations—Treatment—General convulsions—Treatment—Value of chloral and bromides in combination—'Croup,' diagnosis and treatment—Catarrh of bronchi - Of the intestine—Importance in all these conditions of treating the underlying rachitic state.

Rickets is a general disease, affecting the nutrition and development of the whole of the growing organism during the first few years of life. It is not to be looked upon as a mere disease of the bones, and of interest only in its surgical aspect; as it is usually represented and regarded.

The most marked physical changes, what may be styled the coarse and obvious features of the affection, are indeed met with in the bones. Ossification, which is in the height of active process, is both retarded and perverted; the bones grow irregularly, and remaining largely cartilaginous and soft, yield to pressure to

which they are subjected by the force of gravity, and the weight of the atmosphere, and to the traction of the muscles acting upon them. Thus arise the various distortions and deformities which have given to the disease its name.

The striking and prominent affection of the osseous skeleton has attracted most attention, concentrated it upon this one special feature, and has led to a too narrow view of its clinical aspects and of its pathology.

So that until recently, Rickets has been considered chiefly as a disease of the bones; it has indeed been classed as such in text-books, and its pathology and ætiology investigated almost entirely in this direction without much consideration of its medical aspect.

Yet rickets in its most critical and dangerous phases belongs rather to the province of the physician than to that of the surgeon. A large number of ailments, some of them of the most serious character, which depend upon and are the outcome of the rachitic state, are entirely medical in their nature. For, let me impress upon you, the defects of growth and nutrition in this disease are not limited to the bony framework of the body. *All* the chief structures are involved. For example—the muscles are wasted and enfeebled, showing defective structure under the microscope; the muscular debility of the rickety infant is indeed one of the most striking and characteristic features of the disease in the early period. It is often far more conspicuous than the bone fault, and it is astonishing to find how little stress has been laid

upon it by writers on the subject. A rickety child of two years old or more is often unable to walk or stand, or even in extreme cases to support its trunk in the erect posture, so as to sit up; and this not because its bones are soft and distorted, but because its muscles are feeble. This loss of muscular power is in some instances so extreme that this inability to stand, or walk, or sit up is not infrequently mistaken for paralysis. I have seen a number of cases of supposed paraplegia in which the enfeeblement of rickets alone existed. Sir William Jenner observed a remarkable example of this muscular debility; the patient, a child of six, could not change her position in bed, or even raise her arm an inch from the bed without assistance.

Look again at the condition of the ligaments. The rickety child gets knock-knee or yielding ankles and flat foot, not because of the affection of the bones, but from the relaxed and yielding condition of the ligaments and muscles which hold them together.

Consider next the state of the mucous membranes. The rickety child is liable beyond all other children to catarrh of stomach and bowels, and bronchi; not because it has a disease of the bones, although this aggravates the condition in the case of bronchitis, owing to the added difficulty of respiration due to the yielding of the thoracic walls; but because the mucous lining of the lungs and alimentary tract is especially irritable and sensitive, and exhibits an abnormal tendency to catarrh.

Consider, again, the imperfection of the blood condition—the anæmia which is a part of rickets—not due to any bone fault, but the result of the profound defect which involves the nutrient fluid as well as the tissues which it supplies.

The profuse sweating is another evidence of general affection of the organism due possibly to the stimulating effect of waste matters developed from unused materials. These probably act as do septic matters, by irritating the sweat glands, and affecting similarly the bones and cartilages; causing in these latter hyperplasia. The fibrosis of other organs such as the liver and spleen which occurs in rickets is probably a result of the same excitation.

Lastly, there is the general affection of the nervous system; the controlling brain being functually backward, while the reflex nervous system displays a morbidly increased excitability; so that various forms of spasms and convulsions are readily set up. These nervous disorders are often of extreme and even fatal intensity.

You see, therefore, gentlemen, that, as I stated at the outset, rickets is not a mere affection of the bones, but a general affection involving the nutrition and development of the whole of the growing organism during the period of its early development.

Having thus cleared the ground and placed before you the wide range which rickets embraces, I turn to the results of the disease as they are displayed in the nervous system.

The morbid changes met with as far as they have

yet been made out are not striking. The increased size of the head in rickets is suggestive of an enlarged brain, but the increase in size is due chiefly to thickening of the cranial bones; according to some indeed the brain is even smaller than normal, the extra space in the cranium being filled by effusion of fluid into the ventricles. According to other observers, the enlargement is due to increase of the neuroglia, and a fibrosis such as that found in other organs, the liver and spleen, for example; and this view has therefore analogy in its favour. There can be no doubt whatever that the nervous structures share in the general malnutrition, although the material signs of this yet discovered are comparatively slight.

If we turn to the symptoms which indicate a morbid condition of the brain and cord they are distinct and striking enough. The nervous disorders of rickets form one of the most marked features of the disease, although the relation is very generally overlooked or underestimated in actual practice.

Sir William Jenner, in his classical Lectures on Rickets, was I think the first to notice the exceptional tendency of rickety children to convulsive seizures, and this relation was fully established later by Dr. Gee. Convulsions do, indeed, occur altogether apart from the rickety state in very young infants, and in special circumstances, when the reflex or central irritation is excessive. As within my own experience, in sunstroke, in meningitis, in the entrance of a round worm into the bile duct; in the gastric disturbance due to the

ingestion of profoundly irritant food; in uræmia, in extreme anæmia; and at the outset of acute disease and specific fevers. In the vast majority of cases of convulsions in children, however, there is the underlying constitutional state of rickets. The nervous system is in a state of hyper-excitability, and in this condition slight causes of irritation, which would be harmless in the case of a healthy child whose nervous system is in a state of stability, set up clonic or tonic motor spasm.

One cause of this reflex hyper-excitability would appear to be defective nutrition of the motor centres; their protoplasm is probably as ill-nourished as we know the protoplasm of muscle to be in such cases. Look at the myoidema of wasting diseases such as starvation and phthisis. The ill-nourished muscle is hyper-excitable, and contracts under the slight stimulus of a tap which would fail altogether to produce such a contraction in healthy muscle. There is again the analogous case of tetany, a kind of motor spasm met with, in the case of children, in the rickety alone, but in adults in those whose nutrition has been exceptionally lowered by lactation, by typhoid, by diarrhœa. And, lastly, patients who die of starvation frequently have severe convulsions; as, for example, in a case of severe and fatal cirrhosis of the liver in a boy under my care some years ago, practically a condition of slow starvation. The convulsions appear to be due to anæmia of the brain—just as in death from bleeding.

You know the example of the strychnised frog,

which remains quiescent until the stimulus of a shake or jar sets up convulsive spasm. Rickety children are in much the same condition of nervous tension and extreme sensitiveness to reflex stimuli. With them, at any rate when the disease is advanced or extreme, a mere breath of cold air upon the face is sufficient to induce laryngeal spasm. The presence of indigestible food in the intestine or irritation of the dental nerves n teething, may cause general convulsion, set up tetany, and intestinal catarrh.

There is probably an imperfect cerebral control by the higher centres in addition to the exaggerated excitability of the motor centres to reflex stimuli. The nervous system is not fully developed in infants, and, as Sir W. Gowers has pointed out, only portions of it are structurally complete. Extensive tracts of fibres have not yet acquired their white medullary substance, and until the axis cylinders are thus clothed, the fibres have little conducting power, which is essential to the due transmission of central control, although it is probable that such conducting power is not absent altogether. Moreover, the lower centres are further advanced in development than the higher, which adds to the imperfection and difficulty of control. Hence the readiness with which reflex disturbance is set up in early childhood. Now, in rickets these conditions are exaggerated; it is a disease of defective nutrition, chiefly from imperfect supply of materials essential for growth and development; functional power depends upon structural perfection; the parts last developed must

suffer from the general delay more than those parts that have been perfected earlier, and have been longer in fuller use. In rickets, therefore, the lower motor centres in the spinal cord and medulla already hyper-excitable are under less than the normal amount of control by the higher central centres. I lay great stress upon these points of ætiology and pathology because they furnish the key to effective treatment.

The forms of motor disorder associated with rickets vary from simple spasm of the glottis, slight twitching of muscles, carpopedal contractions, or tetany, up to the most extreme forms, viz., severe general convulsions.

Laryngismus stridulus, *tetany*, and *general convulsions* are the positive, comparative, and superlative of the convulsive state in children. They are all distinguished by the occurrence of muscular spasm.

In the first a transient spasm of laryngeal muscles, which close the glottis, lasting only a few seconds at a time.

In the second, tetany, a persistent tonic contraction of the flexors and adductors of the hands and feet varying in degree, liable to exacerbations, but never ceasing absolutely.

In the third—general convulsions—the whole of the voluntary muscles are usually involved, and there is loss of consciousness. There may be complete relaxation between the attacks, or more or less persistent rigidity of certain groups of muscles, as the flexors of the hands and feet.

The first of these, *Laryngismus stridulus*, is rarely

met with apart from rickets of which it is a common symptom.

It is quite possible that you have never seen a case, for such affections are not often met with in the hospital wards, since they occur chiefly in infants under age. Further, although infants thus affected are common enough in the children's out-patient rooms, and in private, the condition is transient and occasional only. The reflex apparatus of the glottis, necessarily highly sensitive in order to protect the air passages from the intrusion of foreign bodies, is in rickets excited to action by slight catarrh, by crying or laughter causing tickling of the fauces, by a breath of cold air, or even the emotional disturbance produced by fright or anger, or the vexation of crossed purpose, such as the refusal of a toy or its removal. Attacks are especially liable to occur on first waking from sleep, when the central control has been in abeyance; particularly in the early morning, when sleep has been prolonged and profound.

The laryngeal spasm is marked by arrest of respiration, lasting usually only a few seconds, and ending as suddenly with a prolonged crowing inspiration as the air is drawn in through the narrowed glottis—much as in whooping-cough—but in the case of laryngismus there is no cough unless laryngeal catarrh is the exciting cause. The spasm sets in suddenly and quickly, as a rule without warning, and it varies in intensity. Sometimes there is merely a slight crowing sound with each inspiratory effort for a time—like the crowing which a child makes when it is tossed

in the air, exactly the same in fact—from the same cause.

In other cases the muscles of respiration may remain fixed, and breathing be arrested until the child becomes greatly cyanosed. Just at this point—when actual asphyxia appears imminent, the spasm relaxes, and the child breathes once more. In a few cases, but comparatively rarely, the closure of the glottis continues too long for life, and death occurs suddenly from suffocation.

It does not do to speak lightly of this contingency, remote though it be; be careful to give a guarded although a favourable prognosis. I remember a friend of mine, an authority on this disorder, who was called in to a case of the kind, and, being asked by the anxious parents whether the attacks involved any danger to life, ventured to assure them that the risk was so small, they might be quite easy on that score. He had only just left the house, however, when someone ran after him and fetched him hurriedly back as the child had a second attack: on reaching the room again he found the patient dead.

The only cases I have seen which resemble laryngismus have been in children who suffer from faucial and nasal obstruction; they are liable to become greatly cyanosed during a fit of passionate crying and screaming, but I have never known any serious result to follow.

An attack of spasm of the glottis in a rickety child is not unfrequently the precursor of a general convulsion, and its occurrence must always be regarded as a most

significant indication that the condition of the nervous system has become unstable, and one in which dangerous general convulsion would be readily set up, by slight cause, at any moment.

1. Treatment of Laryngismus stridulus.—In these cases treatment must be based upon three distinct principles, viz. :—

(*a*) To relieve the dangerous spasm instantly when it occurs.

(*b*) To ward off attacks for the moment by removing the source of irritation, and by soothing the irritability of the motor centres.

(*c*) To remove the constitutional condition which underlies the attacks, viz. the rickety state.

For the first purpose a dash of cold water in the face, to induce sobbing respiration, is the readiest remedy; a finger passed into the fauces to induce the act of vomiting is also an approved method of unlocking the dangerous spasm. In first attacks, unexpected and unprepared for, these are the only means instantly available; but for subsequent seizures, if they are at all severe, it is well to have hot water kept ready to hand; a sponge wrung out in this should be applied to the throat over the larynx. For the second purpose, *i.e.* that of warding off immediate attacks, the chief means is to soothe the abnormal excitability of the motor system, and keep it dulled until such time as the source of irritation is removed, and the stability of the nervous system re-established. For this purpose chloral and the bromides are the only reliable drugs, each useful alone, but most effective when given together,

the combination possessing greater power than any other remedy in restraining reflex motor spasm.

For a child three months old one-quarter to one-half a grain of chloral and three grains of bromide every four hours for three or four doses; continued as frequently or less frequently afterwards according to the effect produced, the approach of undue drowsiness being carefully watched. For a child six months old three-quarters of a grain to one grain of chloral with four grains of bromide in the same way. If the child becomes so sleepy that it cannot be awakened sufficiently to take notice or take his food, the limits of safety have been over-passed. The use of sedatives of this kind has been decried—but in them alone lies present safety—they enable the patient to tide over the period of most active danger. Moreover, do not intermit their use too hastily. I remember well one case of this kind, in which premature abandonment of the controlling influence exercised by chloral and bromides proved fatal. The child, a year and ten months old—with marked rickets, had frequent and dangerous attacks of laryngismus on the least excitement or stimulation. At the end of a fortnight the attacks had entirely ceased under the steady administration of chloral and bromide, and I thought that the drugs might be discontinued with safety. Three days later the laryngismus returned, and in an extreme spasm the child died.

In the next place, any source of irritation should be removed. In the case of laryngismus there is usually some slight catarrh of the larynx, which may

be treated by ipecacuanha and citrate of potash. If pushing teeth and swollen gums are sources of irritation, a timely use of the gum lancet, now too little resorted to, will often suffice to arrest a threatened attack. Remember that the spasm of laryngismus is liable to be set up by a blast of cold air, or a fit of crying or of laughter, or any emotional excitement such as anger or great pleasure, and the child must therefore be carefully guarded and protected from all stimulation of the kind. It must not be played with, or tickled, or tossed, or excited by any one of the many ways affected by enthusiastic nurses or mothers or admiring visitors—lest a spasm be excited which might prove fatal.

The third object of treatment, viz. the removal of the underlying constitutional state, rickets, must be carried out systematically. Do not be content with the routine administration of cod liver oil and so called Parrish's food. These are often useful but are insufficient alone. Supply carefully those elements of food, the lack of which, or the removal of which by vomiting and diarrhœa, is the chief cause of the disease ; a full supply of animal fat in the form of cream, and animal fat and proteid in the form of milk, and animal proteid in the form of raw meat juice being the most essential. For full details of the proper dieting of rickety infants I must refer you to my articles in 'Allbutt's System of Medicine.' I will merely draw your attention to the fact that this essential point in treatment, viz. the steady cure of the underlying constitutional defect which is the root of the evil, is constantly neglected,

the symptoms to which it gives rise being alone dealt with.

Tetany, or *Tetanilla*, as it is sometimes termed, the second of the three nervous disorders associated with rickets, is a most curious and interesting affection, in which the prevailing feature is tonic spasm of the muscles, chiefly those of the hands and feet. Laryngismus is almost invariably an accompaniment of tetany in children, and general convulsions a frequent culmination of the seizure. As I mentioned to you, I think, tetany occasionally arises in adults as a sequel of exhausting disease, or severe constitutional drain ; in women during lactation, and as a sequel to diarrhœa or typhoid fever. Such cases are, however, rare. Tetany is especially a disease of early childhood and of the rickety state, and is usually an accompaniment of diarrhœa in connection with this condition.

In the spasm of tetany the thumbs are first affected, being forcibly adducted and drawn into the hollow of the palm, the tips pressed forcibly against the middle phalanx of the third finger, and sometimes even driven into the skin. The fingers are drawn together and overlap, and the palm is hollowed, so that the hand becomes cone-like; the 'accoucheur's' hand of Trousseau. The wrist is slightly flexed, and in severe cases, when the muscular spasm is great, the back of the hand and wrist may become blue, swollen, and even slightly œdematous from the pressure of the contracting muscles on the venous circulation.

The feet are affected in like manner, but usually in

less degree. The toes are adducted, flexed, and overlap, so that the forepart of the sole of the foot is hollowed and concave by the drawing inwards of its borders; the dorsum is arched, and in severe cases swollen, congested, and shiny from pressure, like the backs of the hands.

The morbid irritability of the motor nerve system in tetany is shown by the fact that contractions of the orbicularis and levator anguli oris can be induced on stimulating the facial nerve by scratching the skin with the finger over the point of its emergence from the skull, the region of the pes anserinus.

In some cases the spasm is said to extend to the muscles of the trunk, causing slight opisthotonus, and occasionally to those of the jaws, causing trismus; but if this is so it is very rare and beyond my personal experience. The muscular cramps are painful, and when extreme, acutely so. We learn this from the statement of adults, and it is noted accordingly that a child who suffers from tetany, cries incessantly if the contractions are severe, and screams when handled. The tetanoid state persists during sleep and even under chloroform. It continues a considerable time, often for weeks, and is apt to recur. Remissions occur from time to time; these are not absolutely complete, some degree of rigidity still remaining. The spasm can be excited afresh by pressure on the main artery or nerve, probably the latter, for the motor nerves are in a state of hyper-excitability, not only in the limbs but in other parts.

The electrical excitability of the nerves of the most affected parts is also increased to both faradism and galvanism ; and as Erb has shown, the mode of reaction to the voltaic current is reversed, contraction being first excited by positive instead of negative closure, and a prolonged ' tetanus ' contraction following anodal and cathodal closure, the only condition I believe in which anodal ' tetanus ' has been observed in man. It is said that this increased neuro-muscular excitability may sometimes be found in rickety children who are free from attacks of tonic spasm.

As the predisposing cause of tetany is the rickety state, so also almost invariably a protracted diarrhœa so liable to occur in rickety subjects is the exciting cause. This must be controlled by careful feeding, by the use of sedative astringents, such as bismuth and chalk, with the addition of small doses of opium in some form. In the case of very young children the diet must be free from all materials likely to set up intestinal irritation. Thus, if they are fed with cows' milk this should be diluted and peptonised for a time, and raw meat juice and cream given in addition if required. In a severe case a wet nurse should be obtained, or asses' milk substituted for the cows' milk. But the food must not be suddenly cut down to barley water alone, as is often done. Nutritive material is essential to restoration of nerve tone, and animal broths, chicken tea, or Valentine's meat juice should be given together with cream if milk is withheld for a time. With older children all coarse or irritant foods, such as oatmeal, brown bread, jams

and fruits with skins or seeds, should be avoided, and the diet limited to bread and milk, with cool beef tea, or chicken tea, or meat juice. The same principles of treatment must be observed as those I have laid down with regard to laryngismus, viz. chloral and bromide of ammonium in doses of one-quarter to two grains of the former, and two to five grains of the latter to relieve tonic spasm and protect against convulsion. I have never given larger doses than the above in these cases, but I should not hesitate to increase these amounts if need arose, carefully watching the effect. Children bear chloral well. Sir S. Wilks gave to a boy of three years old with tetany, ten grains of chloral three times a day with the best effect. Bouchut gave thirty grains to children of two to five years, sixty to children of seven years old, in order to produce anæsthesia for surgical operations.

When the crisis is over, a diet should be carried out, calculated to cure the rachitic state, care being taken to avoid disturbance of digestion by over-feeding.

General Convulsions.—The two preceding forms, more especially laryngismus, are the frequent forerunners and accompaniments of general convulsions in rickety children. Convulsions are not confined to rickety children of course, but they are most common and most readily excited in the rickety state. I need hardly describe to you in detail the phenomena of a convulsive fit. But it may be useful to indicate the symptoms which, in addition to laryngismus, indicate its probable approach. Amongst these are twitching

of the muscles of the face, and twitching and contractions of the hands and feet, flexed toes and fingers, as in tetany, or with the fingers and toes abducted, so that they are widely apart. In addition to these a most significant warning is a sudden access of coldness of face, hands, and feet, like that met with sometimes in hysteria. If any of these symptoms occur in a child, especially if it is rickety, be on your guard—watch for the advent of convulsions, and take precautions accordingly.

The treatment of general convulsions is to be carried out on the same principles as before laid down for laryngismus and tetany.

For the immediate relief of an actual convulsion the warm bath is a time-honoured remedy—useful, no doubt, in soothing nervous excitability—probably applied before you arrive on the scene. A more effectual remedy, however, is chloroform, if the convulsion is not complete, and the child respires sufficiently to inhale it. In severe cases, the administration of medicines by the mouth is difficult, often impossible. The only device then available, one adopted in the first instance as a last resource, has proved the most effectual of any, viz. the administration of chloral and bromide by rectal injection. The doses given in this way must be larger than those administered by the mouth—two to three grains of the former to five or ten of the latter according to age—in half an ounce of water, care being taken to ensure the injection being retained by holding the soft parts round the tube together so as to prevent reflux under the first straining of the rectum.

I have now under observation an infant of three months, recovering from repeated severe convulsions induced by over-feeding with milk. It has had two to three injections daily of two grains of chloral, and three of bromide of ammonium with complete success.

As the convulsions subside—indicated by their becoming less severe, less frequent, and the consciousness in the intervals between more perfect—doses of from half a grain to one grain, with two or three of bromide of ammonium, may be substituted for the enemata.

A dose of castor oil to clear out the intestine is a necessary preliminary when the exciting cause is the presence of indigestible matter in the bowel. Do not, however, commit the common error of purging the child constantly and severely. Remember that diarrhœa in itself is a cause of convulsion, and the repeated irritation and constant drain of nutriment involved by free purging impairs nervous stability and favours the recurrence of convulsive disturbance. If the gums are swollen and tender, and the teeth are obviously pushing, lance them freely. If the child is taking cows' milk, this should be stopped for the time, and asses' milk, or peptonised milk, or other bland nutriment given instead, with Valentine's meat juice in doses of ten to twenty drops in a dessert-spoonful of water, or half an ounce of chicken tea every four or six hours.

Lastly, when recovery from the acute stage has taken place, measures such as I have previously indicated by diet and otherwise should be taken to remove the rachitic state—the prime evil which underlies all.

Let me again repeat that this most important part of treatment is constantly neglected, or imperfectly carried out.

Even when convulsions arise in children not obviously rickety, the subjects are, in the vast majority of cases, flabby, pallid, anæmic, ill-nourished; and it is important in these cases also, as well as in rickets, to lessen the hyper-excitability of the nervous system; to restore it to full vitality and stability by establishing perfect nutrition of structure.

The other disorders associated with rickets which came within the province of the physician are catarrhs of mucous membranes; of the larynx, trachea, bronchi, or intestine.

They are not limited to rickety children only, but rickety children are more especially prone to them, and the disorder is more dangerous in these subjects than in robust infants.

You give ipecacuanha, or antimonial wine, warm moist air to breathe to relieve these symptoms—that is the first step.

Then in the case of laryngitis (croup), ascertain whether this is catarrhal or diphtheritic. Examine the fauces to see whether there is any sign of membrane there. If there is any appearance of film secretion, remove a little of it with a clean brush, for a cultivation test of the presence or absence of the specific Loeffler bacillus.

If you are in any doubt as to the nature of the laryngitis, give an hypodermic injection of antitoxin as a preliminary precaution. The degree of acuteness of

the attack, the fact whether it has come on suddenly in the night after the first sleep, the occurrence or absence of previous attacks—the absence or presence of albumen in the urine—the co-existence of bronchitis, will all help you to form a conclusion.

If the laryngitis or croup is simply catarrhal, always proceed to ask the further question : Is this the case of a delicate, over-cared for child merely, kept in close, warm rooms, hardly allowed to breathe the fresh air of heaven, until its respiratory mucous membrane has become hypersensitive, or has the child a constitutional tendency to catarrh? If so, is this merely an inherited oversensitiveness of the mucous lining of the air passages ; or is it acquired and due to rickets? And similarly with regard to the bronchial catarrh of children : ask yourself the same question, and examine the patient carefully for signs of rickets in the head, shape, open fontanelle, the beaded ribs, large epiphyses, soft, bent, or bending bones, head sweats and the like. If you find these marks of underlying defect, treat the rickets as soon as the special incidental catarrh subsides.

Curing the rickety state means curing the special tendencies to catarrh. But this is not all. Remember that in these affections of the larynx and bronchi, which involve more or less obstruction to the indrawing of air into the lungs, the correction of the undue softness of the rickety bone is of great importance. The intercostal muscles and diaphragm cannot act effectively on the soft walls of the thorax, the lower

chest falls in largely during inspiration instead of expanding, air enters very imperfectly, collapse of lung takes place, and the danger of the attack of catarrh is enormously increased. Permanent contraction and pigeon breast may eventually follow. And in the same way with catarrh of the intestine, evidenced by recurrent attacks of diarrhœa, correct this disorder by appropriate remedies such as grey powder and Dover's powder, or bismuth and chalk, and opium, and proper dieting, and then ascertain if the underlying cause of rickets exists, and remember that in the case of an infant, if the diarrhœa is not the result of rickets, a continued drain of this kind will set up the rickety state.

Cure the rickets if already there. Prevent it if it is merely prospective. Remember that it is chiefly a diet disease; to be cured by correct feeding and good hygiene rather than by drugs.

LECTURE VI

SOME PRACTICAL POINTS IN THE TREATMENT OF DISEASES OF THE LUNGS AND AIR PASSAGES

I. General principles. Bronchitis.

Routine treatment deprecated—Importance of the secondary effects that result from primary disease of a particular organ—Back-working in lung disease—Bronchitis—Remedies to be chosen suitable to the stage of the disease—Doses given usually too small—Drugs in the early stage—Precautions in their use—Drugs in the later stages—External applications—Steam—Failure of the right heart—Two lines of treatment: i. Stimulation of the heart; ii. Abstraction of blood to relieve it—Cardiac stimulants—Digitalis—Strychnia—Caffeine—Strophanthus—Convallaria—Alcohol—Venesection—Leeches—Cupping—Cautions as to the use of depletive measures—Alimentary disturbances in bronchitis—Their treatment—Dangers of gastric distension and dilatation—Chronic bronchitis—Recurrent bronchitis with persistent cough—'Stomach' cough; usually due to a limited patch of bronchial catarrh—Change of climate necessary for these cases.

One evil result of learning medical treatment almost entirely from books, and partly also of the dogmatic way in which therapeutics are necessarily taught to the student, is that it creates a tendency to a mechanical and routine practice. In a certain disease give a certain drug or drugs.

With many of us I am afraid, as far as my observations go, the practice of medicine is little more than a repetition of the same routine treatment in all

cases of the same disease; given the disease, the appropriate remedy is ready labelled for use in its pigeon-hole, and is brought out and applied without doubt or hesitation.

The student on his first entrance into practice, having little personal experience, is naturally tempted, or indeed obliged almost, to rely upon what is put down as the proper treatment, or to give what he has seen given in like cases. And thus he is led to treat all cases much alike, according to regulation formulæ.

Ipecacuanha and squill and ammonia for bronchitis; aconite, or an ice-bag perhaps, for pneumonia; salicylate of soda for acute rheumatism; stramonium for asthma; digitalis for heart disease; and so on.

Now this rule of thumb procedure has the advantage of simplicity, and it saves much anxious thought and mental labour.

It cannot, however, be regarded as satisfactory to the patient, or as a high form of medical art.

I am anxious to impress upon you the necessity for following a more perfect way.

You cannot treat all cases of the same disease alike, upon the same cut and dried plan; for all cases are not alike. Indeed, almost every case presents some point of difference to every other.

There is the difference of age, and condition, and habits of the individual subjects to be considered.

Also, whether the disease is pure and simple, set up by some chance cause in a body otherwise healthy, or, whether it is grafted upon some general disease; or

again, one expression only amongst many others of some general disease, affecting many organs in varying degree, such as gout, or Bright's disease, or tuberculosis.

There is also another practical point very generally overlooked, viz. the secondary or resultant disturbance produced in related organs by the special primary affection of the organ first concerned.

The bodily machinery—as your work in anatomy and physiology has no doubt painfully made plain to you—is extremely complex and intricate.

And the various organs, with their different functions, have a close relation to and dependence upon one another; especially is this the case with regard to the organs of respiration and circulation.

A break down in one organ involves the disturbance of other organs which work in connection with it, and whose functions depend upon it. You are familiar enough no doubt with the consequence of mitral disease—the back-working as it is called.

How the leakage or obstruction at the mitral valve, through which the blood stream from the lungs flows into the left heart, leads in the first place to delay and accumulation in the lung, itself insufficiently drained and emptied; how, as a consequence of this, the right ventricle finds difficulty in pumping its contents through the overloaded pulmonary vessels, and becomes over-distended and over-taxed.

How, accordingly, the systemic veins which discharge into the right heart, unrelieved of their blood

charge, become distended also, and thus the portal system lying behind again, becomes choked and clogged, while the viscera which drain into it, the liver, the stomach, the spleen and alimentary canal suffer likewise ; and even the kidneys, at the end of the long vascular chain, do not escape the effect of the primary blockage at the mitral valve, but become congested, and leak albumen and sometimes blood.

With this you are no doubt familiar; it is one of the illustrations most commonly used in clinical teaching.

There is, however, an equally striking and important example of a similar series of results which follow obstruction, only just one stage further back, which is hardly noticed, viz. the back working in lung diseases.

And this is a clinical point, bearing on treatment, to which I wish especially to ask your attention.

Just look what occurs in common diseases such as bronchitis, and pneumonia, and all chest affections indeed, in which the circulation through the lungs is impeded.

The organs behind the lungs in the direction opposite to that of the blood flow, suffer from the damming back of the stream, become overloaded with blood, and their important functions gravely interfered with and disordered. The right heart, liver, stomach, intestine, kidneys, all become involved, evidenced by falling pulse, cardiac dilatation perhaps, coated tongue, loss of appetite, impaired digestion, possibly albuminuria.

Further still, the blood imperfectly oxygenated, travelling with undue slowness, fails to carry with sufficient fulness and freedom the materials for the restoration and cleansing of the tissues.

In this way, too, the functions of the central nervous system become disordered and impaired, and semi-coma, delirium, and irregular respiration arise.

How potent this influence of back pressure on nutrition really is in pulmonary obstruction, is well illustrated by a case now under my care. The patient had extreme pneumonia eight weeks ago. The whole of one lung became hepatised, and the process of resolution has been extremely slow, and is only partially complete. Previous to the attack the patient enjoyed perfect and robust health. The obstruction to the pulmonary circulation was very great. At the end of five weeks I observed that the fingers were becoming bulbous, or clubbed, as it is termed, a result met with in various forms of obstruction to the circulation through the lungs; and this has now become marked. Possibly the change may have commenced even earlier still. This may seem a small thing, but it is of considerable significance.

The pathological condition in these cases of lung disease then is a complex one, involving many organs; and although the secondary disturbances are important, and their correction essential to the cure of the patient, attention is usually directed almost exclusively to the primary central affection of the lungs, while the secondary disturbances are little regarded.

No doubt the cure of the lung trouble eventually allows the dependent organs to recover also. That, however, cannot be done in a moment; it is a matter of time.

Meanwhile, various functions languish, and this retards the cure of the lung affection.

Much may be done to relieve the organs suffering disorder from the primary lung affection, pending the restoration of this leading offending organ; and this relief of dependent organs, by the improvement it effects in the general working of the machinery, reacts upon the initial disease, largely assists, and materially hastens, the cure of it.

We can influence the vicious circle at many points, and of the means of doing this I shall speak later.

It is of the utmost importance, therefore, when you begin the campaign against a disease, before you decide upon the treatment of any given case, to review thoroughly the whole pathological field; to examine the whole area of possible disturbance, and to do this continually from time to time throughout its course.

Picture to yourself the exact condition, not only of the chief organ affected, but of every other organ lying in the chain of connection which can be affected by the central disturbance, or which can have suffered from the same cause as that which set up disorder there.

I propose to examine how this works out practically in certain diseases of the lungs. I shall take bronchitis pneumonia, and pleurisy as examples, since they are

the most common and the most suitable for the purpose of illustration. And first as to treatment of the primary affection.

When bronchitis occurs in uncomplicated form in healthy persons, it is one of the diseases most simple in its pathology; a catarrhal inflammation of the mucous lining of the bronchial tubes; redness and swelling only at first, then secretion; at the outset scanty, viscid, white, expelled with difficulty; later, more copious, less viscid, greenish or yellowish in colour, more easily dislodged and discharged. With these conditions also there is in many instances bronchial spasm, an asthmatic element, invariably present in asthmatic subjects, and playing a part in others far more frequently than is usually acknowledged.

These cases of simple bronchitis are more serious (1) in proportion to the extent to which the smaller bronchioles are involved; (2) in very young subjects with yielding chest walls; and (3) in old persons whose respiratory and heart power is impaired.

It is not my purpose, however, in the present instance to enter into the pathology of the disease or its symptoms further than is necessary to elucidate treatment.

The position, as far as the lungs are concerned, is oughly, inflammatory swelling of the lining of the air tubes, causing obstruction to the passage of air to the air cells. Such obstruction being increased by the viscid mucus clogging the pipes, and often also by spasmodic contraction of the bronchioles.

This is the condition we have to seek to remedy in the first instance.

In this relation I would remark, in the first place, that the approved remedies, such as ipecacuanha, antimony, iodide of potassium, squills, ammonium carbonate, senega, spirit of nitrous ether, paregoric, belladonna, are generally given in an indiscriminate fashion. All these remedies are not adapted to all stages of the disease. Some of them are appropriate to one, some to another. Yet they are constantly jumbled together haphazard, without regard to this, or the consideration whether their special physiological action is suited to the condition existing at the moment.

In the early stage of dry swollen mucous membrane, when secretion is scanty and viscid, the relaxing expectorants, which ease the vascular pressure and increase secretion, are to be given. Such are antimony, ipecacuanha, iodides, alkalies, apomorphia, jaborandi, external warmth, and warm moist atmosphere.

Then, when secretion has become plentiful, loose, and easy, the drugs which aid its expulsion, and diminish it, come in, such as ammonium carbonate, ammonium chloride, senega, squill, and the balsams and volatile oils, such as benzoin, tolu, turpentine, together with remedies which increase the activity of the respiratory centre, *i.e.* which render respirations both quicker and deeper, so that more respiratory work is done—such as strychnia, ammonia, digitalis, atropine.

Lastly, oxygen may be helpful at any stage, when there is cyanosis from deficient aeration, and is often indeed of vital service.

Yet how often are drugs, such as ammonia, senega, squill, belladonna, which tend to dry up the mucous membrane, given in the early stage, frequently in combination with drugs like iodides and alkalies and ipecacuanha, which liquefy and increase secretion.

So that one set of drugs probably largely neutralises the effect of the other; belladonna and senega are especially evil in this early stage in my experience, and with respect to belladonna we know physiologically that its active alkaloid, atropine in full doses, completely arrests secretion. If a sedative has to be given, it should be in the form of opium or Indian hemp, which, while more sedative, arrest secretion less.

Remember cough is essential to the clearing of the bronchi. It is nature's remedy; to be controlled if too violent, but not arrested.

Another defect in common practice in cases of bronchitis is that the doses of the drugs given at the outset of the attack are usually far too small, and are not repeated with sufficient frequency to be really effective.

Take such a prescription as this, for instance, which is typical, I think, of the usual medication:

Potassæ Citratis	gr. v. to gr. x.
Vini Ipecac.	♏ v.
Liq. Ammon. Acet.	℥j.
Tinct. Camph. Co.	♏x. to gr. xv.
Aqua ad	℥j.

Every four or six hours.

Do you believe that it is possible that five or ten grains of citrate of potash, or five minims of ipecacuanha

wine, or a drachm of liquor ammoniæ acetatis, or ten or fifteen drops of paregoric, can have any material or immediate influence upon the pulmonary condition?

We know that much larger doses are necessary to produce a distinct physiological effect.

The only exceptions to the rule that the doses usually given are too small are iodide of potash, antimonial wine, and aconite.

The first, curiously enough, as far as the power of increasing secretion is concerned, is most effectual in small doses. Antimony cannot be given in full doses for long without causing serious depression, and perhaps severe vomiting.

In young vigorous adults, where there is extreme wheezing and distress from dry swelling of the bronchial membrane, a few doses of antimony at the outset, say ten minims of antimonial wine every four hours, from four to six doses, are often of great service in hastening secretion; but it should not be given continuously for long, on account of its powerful action in depressing the circulation, and causing fatty degeneration of tissue; antimony is stated, indeed, to have caused actual consolidation of the lung when given in large doses.

Aconite is given occasionally in bronchitis, and was at one time in great vogue in the treatment of pneumonia. In my judgment, from its property of depressing the action of the heart and paralysing the action of the respiratory centre, it is a most mischievous and even dangerous drug in all respiratory and cardiac disease, and should be avoided altogether.

Use the appropriate remedies then, give them in sufficient doses at the outset, and repeat them at short intervals, so as to produce the effect you want quickly.

Give, for example, iodide of potassium or sodium, or ammonium in two to three grains, or the citrate of potash or of soda in twenty or thirty grains, and ten minims of ipecacuanha wine, with twenty minims of compound tincture of camphor, if the cough is troublesome, every two hours for the first six or eight doses, and then every four hours.

Bear in mind, however, that some individuals are very intolerant of iodides, and others of ipecacuanha, and that you must be on your guard against early iodism or vomiting; and regulate the dose accordingly or substitute other drugs of similar action, such as alkalies in full doses. This holds also with regard to apomorphia, which is apt to set up vomiting, and if used too freely may actually cause blocking of the air tubes by excessive secretion; and it is moreover a cardiac depressant. I have small experience of its use.

With regard to jaborandi and its active principle, pilocarpine, which produces copious secretion from the bronchial mucous membrane, the same caution must be given; and, indeed, the free flux which it sets up in the salivary and sweat glands, and its depressing action on the heart, render it generally an undesirable remedy.

The stimulating expectorants useful in the later stages do not require to be given with the same frequency, or in such full doses—except in urgent cases of great accumulation of mucus. In such

conditions an emetic will sometimes clear the lungs when expectorants have failed, but their unnecessary and frequent use is to be deprecated. I have seen life lost from the exhaustion resulting from excessive and uncontrollable vomiting set up in this way, by the free use of emetic doses of ipecacuanha.

And what about the time-honoured domestic remedy of poultices? In bronchitis they are often greatly overdone and abused.

It has been shown by experiment that the application of warmth to the surface lessens the congestion of the bronchial lining, and that cold increases it. In using poultices we must be guided by the feelings of the patient in some degree. A few find them grateful and soothing, but many oppressive; and the disturbance and wear and tear caused by the frequent fresh application of them is liable to cause distress and exhaustion. This is especially the case with children and feeble subjects. In them poultices do harm rather than good, and some stimulating liniment, and a light covering of cotton wool, should be substituted.

If poultices *are* used they should be applied to the back and sides of the chest only; on the front of the chest their weight adds to the existing difficulty of respiration by impeding the easy movement of the chest wall.

Similarly with regard to steam. A warm moist atmosphere is soothing to irritable bronchi, but in practice the use of the steam kettle is liable to be greatly overdone. I have seen patients almost steamed to

death, sweating at every pore, and oppressed to the last degree from exposure to a fierce blast of vapour under a canopied bed. When the atmosphere is very dry, as during the east winds of spring, a moderate amount of watery vapour is no doubt beneficial, and the addition of carbolic acid by the use of a solution of pure acid (1 in 80) is a soothing measure.

Later terebene, eucalyptus, or pine oil inhalations, aid the recovery of the bronchial tone. As I pointed out previously, however, this direct attack upon the bronchial mucous membrane by drugs and poultices and inhalants does not comprise the whole art of treatment in bronchitis.

There are the effects of the pulmonary obstruction upon other organs to be dealt with. Of these, the additional work thrown upon the right heart is the most important. If that organ shows signs of embarrassment or failure, as evidenced by any degree of cyanosis, or quickened enfeebled pulse beat, something must be done to relieve it.

This can be effected in two ways, viz. (1) through strengthening the heart's action by the administration of drugs and foods which increase heart power: and (2) by abstraction of blood.

Thus the cardiac tonics, digitalis, strychnia, caffein, strophanthus, convallaria, which also have the property of stimulating the respiratory centre, are invaluable in the treatment of bronchitis and pneumonia.

Digitalis, especially in full doses of ten minims every four hours for twenty-four or forty-eight hours, or even

longer if well borne, has sometimes a marvellous effect in enabling the struggling heart to meet the obstructive difficulty. Cases recover under this treatment which have appeared hopeless.

I remember well one remarkable instance of the power of a cardiac tonic in obstructive bronchitis, that of a boy of four or five years, whom I saw in consultation, suffering from the extreme clogging of the bronchi and extensive collapse of lung which is so liable to occur as a consequence of whooping cough. Hardly any air entered the lung, moist râles were universal, cyanosis was marked, distress great, the pulse rapid, irregular, and feeble. I gave a most gloomy prognosis, but advised that in the extremity six minims of tincture of digitalis should be given at once, followed by three minims every four hours ; a full dose for a child of that age. I was surprised to learn some time afterwards from the medical man with whom I saw the case, that the digitalis had apparently turned the scale and the boy recovered. In urgent cases tincture of digitalis may be given by hypodermic injection in somewhat smaller doses.

Strychnia is a valuable adjunct to digitalis or substitute for it, when the latter causes vomiting, as it is sometimes apt to do. Caffein is another useful remedy of the kind, and strophanthus, and convallaria, although, I think, of inferior power to the two preceding.

Alcohol must be administered with caution if there is cyanosis, on account of its narcotic influence on the respiratory centre.

With regard to the use of these cardiac tonics again a word of warning. Do not be too hasty in the resort to them. Better too soon than too late no doubt, but remember that too early stimulation is liable to exhaust the heart before the great need comes; await the proper moment indicated by faltering pulse or commencing cyanosis, or the undue prolongation of the disease.

With respect to the other means of relieving the embarrassed right heart, by drawing off blood from the overloaded systemic veins which empty into it, this can be done most quickly and efficiently by bleeding from the arm. It is of signal value in some cases where the cyanosis is great, the heart dangerously overtaxed, and showing signs of failure; and it is especially useful in those cases where bronchitis supervenes upon mitral disease. When the cyanosis is less extreme, sufficient relief can be given by leeches. Four, six, eight, or even more, according to circumstances, should be applied between the shoulders about the middle of the scapula—the region where the bronchial veins discharge into the intercostals. By drawing from the systemic tributaries there you lessen the flow into the intercostals, and thus facilitate the flow out of the bronchials into them, relieving both right heart and bronchial lining. A similar but less effective plan is that of cupping.

With regard to conducting these operations, I would give a word of caution.

In order to get at the back for the purpose of cupping or placing leeches between the shoulders, the

patient, who has probably orthopnea, is made to lean forward. This posture causes embarrassment to the respiration, and action of the heart also, by interfering with the free movement of the diaphragm—especially in corpulent persons. I have once seen instant death caused in this way in a very stout deeply cyanosed patient; and more than once I have been apprehensive of it. In such cases it is better to take blood from the arm.

The relief of vascular and heart distension by abstraction of blood is not adapted to the case of young children—or even very feeble, delicate, anæmic adults.

Children bear the loss of blood badly; venesection is out of the question; and they are frightened by the application of leeches. With them leeching should be adopted in great emergency only.

Again, another point. When the circulation is embarrassed by the obstruction in the pulmonary arc, as I pointed out to you, the organs behind the lungs in the blood-current, the liver, stomach, and intestines suffer also. The thickly-coated tongue and foul mouth, the disgust for food, so marked in acute bronchitis, show this.

And as the digestive apparatus is the one by which nutriment is prepared for the restoration of the enfeebled and damaged tissues, it is of extreme importance to recovery that it should be in good working order.

This matter rarely receives proper attention. It is

true that the medical adviser almost invariably inquires about the state of the bowels and gives an aperient, a very proper and useful preliminary to other treatment, but the treatment is not continuous and systematic.

I am sure that the engorged state of the liver and congested condition of the stomach are not adequately realised—how this interferes with their functions; how the condition tends to increase; and how essential a part of the treatment of lung disease it is to rectify it. A dose of calomel at the outset, repeated at intervals and followed by the steady administration of an alkali, such as carbonate of soda, in full doses, with a saline aperient such as sulphate of soda or magnesia, aided if necessary by cascara or rhubarb, are the best means of relieving the congested state.

It is worse than useless to 'pour in the nourishment,' as the phrase runs, when the digestive organs cannot deal with it.

This leads me to another point in treatment of hardly less importance; and that is the necessity of guarding against flatulent distension of the stomach and bowels.

Flatulent distension of the stomach and bowels not only indicates disorder of the digestive process, and causes discomfort to the patient, but it seriously hampers the respiratory function by obstructing the action of the chief air pump of the lungs, the diaphragm.

Small attention is usually paid to this. Nurses are only too ready to overload their patients' stomachs. Although the simplest food, such as milk and beef tea only, may be given, the quantity may be too great, so that it is imperfectly digested and undergoes gaseous fermentation. And indeed liquid foods seem especially liable to cause flatulent distension. Moreover, in enfeebled states the stomach walls are liable to give way and the organ dilate, so that even these fluids are not passed on, accumulate in the dilated viscus, and ferment there.

A few weeks ago I was called in to see a gentleman suffering from rectal abscess, who had attacks of syncope which threatened to be fatal. They turned out to be due to an enormously dilated stomach filled with fermenting, gas-generating food; an enema of rue, a rigid diet, and a soda and aromatic mixture at once relieved the critical situation.

In a case of acute pneumonia to which I was called in recently, a fatal issue was imminent from the same cause. The crisis was over, the patient apparently out of danger, when he gradually began to have increased dyspnœa and became cyanosed. I found the abdomen enormously distended and tympanitic. The diaphragm could hardly move. This, added to the fact that one lung was useless from consolidation, and the heart weakened by pyrexia, and mechanically embarrassed also, produced a state of imminent asphyxia. A turpentine enema saved the situation. It turned out that the nurse, with mistaken zeal, had given between three and four pints of milk in the course of a few hours.

In these cases of pulmonary obstruction light compendious animal nourishment should alone be given. A limited quantity of milk, concentrated cold beef essence, with, perhaps, white fish, and raw eggs beaten up, in small quantities at a time. In severe cases small quantities of peptonised milk and meat essence only. Farinaceous foods such as rice, corn-flour, arrowroot, and sugar, which are most prone to ferment and set up gaseous distension, should be altogether prohibited.

When the acute stage of bronchitis has subsided, it frequently leaves behind it, as you know, a chronic catarrh—or if not that, a sensitiveness and instability of the mucous membrane, so that attacks are liable to recur on small provocation from chill, or draught, or fog.

If the attacks are frequently renewed, there arises that condition of chronic bronchitis, fibrosis, and emphysema, which is permanent ; aggravated from time to time by more acute attacks, and ending in permanent disorganisation of the lung and cardiac dilatation.

There is one special form of chronic or recurrent catarrh following a bronchial attack, to which I must not omit to draw your attention, since it is exceptionally puzzling, generally a source of trouble to the practitioner, and usually misunderstood.

The great symptom is a persistent cough, occasionally most violent, sometimes so much so as to set up retching, and even simulate the paroxysm of whooping

cough. It occurs most commonly, I think, as a sequel to the bronchitis of influenza; but it is not limited to this, it follows other catarrhs also. It is troublesome and distressing; a cough that will not go away; and appears to be little influenced by medicine. It is constantly diagnosed as a 'stomach cough,' but it is really due to irritation of some portion of the air passages. I am very sceptical as to the existence of such a malady as a 'stomach' cough. Hysterical coughs as they are called, apparently of purely neurotic origin, I *have* seen—but not a cough set up by gastric irritation alone. Beware of the diagnosis of 'stomach' cough; it is a frequent source of discredit; almost as much so as that of hysteria. Coughs occur in connection with gastric disturbance no doubt, the pulmonary and gastric trouble being due to a common cause, but the stomach disorder and the cough do not stand in the relation of cause and effect.

If you examine these cases carefully, you will find the condition which gives rise to the cough somewhere in the respiratory tract itself. Sometimes in the throat —a relaxed uvula irritating the fauces. Sometimes a congested irritable pharynx or larynx. More often perhaps the seat of reflex irritation lies lower still, viz. in the trachea.

In the case of the most violent and persistent coughs following bronchial catarrh, the seat of trouble is, however, almost always in the bronchioles; in the furthest twigs of the bronchial tree. The mucus is expelled from these dependent points at the base of

the lung with greater difficulty than elsewhere; and the catarrhal process lingers longest there. You will constantly find in these cases a little patch at the lowest portion of the lung, usually behind, sometimes in the lateral region, sometimes in front, but always low down, where fine moist râles are audible on full inspiration or on coughing—a limited area of persistent bronchial catarrh.

This small lesion, the existence of which is not suspected, perhaps, or if discovered, regarded as unimportant, is yet one productive of much reflex disturbance. I have seen a number of instances of the kind. Only a week or two ago I saw in consultation a patient troubled with a cough of this kind which had lasted most persistently for ten months after an attack of influenza; there was no sign of organic disease, but a small patch of bronchial catarrh at the left base.

In these conditions of residual catarrh, remember, drugs are of comparatively little use; cod liver oil, terebene, pumiline, and other turpentine preparations and volatile oils are helpful in some degree; and inhalations of benzoin or pine oil and the like do good in some cases; but they usually fail. If you lock your patient up in warm rooms in addition you merely relieve him for the time. When you let him out again, the first exposure brings the catarrh back. The only really effective remedy is climate—change of air—to some genial spot where the patient can live largely out of doors in the sunshine, drawing into his bronchial tubes soft, sun-warmed air.

LECTURE VII

SOME PRACTICAL POINTS IN THE TREATMENT OF DISEASES OF THE LUNG—CONTINUED

II. Pneumonia and Pleurisy

Pneumonia, formerly fatally overtreated—Drastic measures employed—Repeated bleedings—Tartar emetic—Starvation diet—Now apt to be dealt with as if catarrhal—The three dangers to be combated—Carbonic acid poisoning—Cardiac failure—Pyrexia—Methods of relief—Digitalis—Venesection, leeches—Danger of pyrexia overrated—Misuse of antipyretic drugs—Value of cold applications—The ice-bag—Delirium in pneumonia—Its treatment—Broncho-pneumonia—Principles of treatment—Pleurisy—Measures for relief of pain—Rheumatic pleurisy—Cold applications—Pleural effusions—Risks of delay in removal of copious pleural effusions.

In my previous lecture, gentlemen, I laid before you some important practical rules to be generally observed in the treatment of these affections, and I discussed more particularly the treatment of bronchitis in relation to them. I propose to-day to speak of pneumonia and pleurisy.

There is probably no disease which 'yields to treatment,' as it is styled, less than pneumonia, and there is no disease in which more harm can be done by wrong treatment; and yet no disease perhaps has been, and still is, so overtreated—as pneumonia; every drug

almost, and every possible therapeutic device, have been tried in this disease and vaunted as successful.

Formerly, when pneumonia was regarded as a simple inflammation of the lung, most strenuous measures were adopted to combat it—bleeding and repeated bleeding, purging, sweating, calomel, opium, and antimony.

From Galen downwards there was no difference of opinion as to the necessity for vigorous measures.

The only question was, as to which vigorous measure was most effective. Bleeding freely was the accepted and popular treatment. Celsus appears to have been the only one who even suggested moderation. He would bleed a strong man, but only dry-cup a weak one. Sydenham said that the only cure for pneumonia was in repeated bleedings.

But the Italians were the most blood-thirsty. At the close of last century, the elder Frank, in his public practice in Pavia, bled his patients twelve or fifteen times a piece. The mortality, Grisolle tells us, was enormous.

A few years later, bleeding appears to have almost died out. At Vienna especially it became rare for a single bleeding to be practised. Yet the immunity did not last long, and the few physicians who spared blood-letting, were styled heretics.

The sanguinary treatment reached its height in the present century. It is difficult to realise how short a time ago this truculent practice was prevalent.

Just when it seemed that the routine practice of venesection was being broken into, and the belief in its

universal necessity shaken, it was revived at Parma and Bologna by Rasori and Tommasini, at the beginning of this nineteenth century.

They bled remorselessly. Incredible as it may appear, they often took away from their patients some 10 lbs. of vital fluid.

The plan adopted was to bleed morning, noon, and evening the first day, taking 2 lbs. of blood; bleed twice the second day, taking also 2 lbs. The remaining 6 lbs. by bleeding once a day, larger or smaller quantities being withdrawn, according to circumstances.

The whole amount of blood in the body is estimated at 12 to 15 lbs., so the unfortunate patient deprived of 10 lbs. by bleeding, and of the virtue of some 3 lbs. from the exudation into the lung, would have been left in some cases with something less than nothing, if he had not replenished his stock during the three or four days during which the process was going on.

To this severe bleeding, Rasori added tartar emetic in increasing doses; larger and larger until tolerance was established. Just picture to yourselves the enfeebled condition of a patient with only a few ounces of blood left in his body, with his small remaining vitality sapped and depressed by full doses of antimony! And, as a result unfortunately, some patients with not very severe pneumonia, died just as convalescence was supposed to be established.

This practice continued with more or less severity everywhere on the Continent and in England.

In France, Bouillaud, as late as 1835, practised what

he called his jugulant treatment, blow upon blow. He thought he could strangle a pneumonia by three days blood draining. Yet he was content with some 5 lbs. of blood. In Edinburgh, about the same time, Gregory taught that with bleeding and water gruel, all other means might be dispensed with. The confessed mortality never appears to have been less than 10 per cent., and was usually much higher.

Gradually the method was modified by Louis, Laennec (at first a great bleeder) in his later days, Watson, and Trousseau.

The first real light leading to a more rational procedure was afforded by homœopathy, when Fleischman published statistics showing a lighter mortality of 6 per cent.

Barthez, finding that children left untreated rarely died, recognised the essentially benignant character of the disease, which showed a mortality of only 2 per cent. if left alone ; and Hughes Bennett confirmed this by the success of his restorative treatment of wine and beef tea, showing a mortality of only 3 per cent.[1]

Modern practice, although toned down from the mischievous and deadly activity of Rasori and of Bouillaud, and a marked improvement even upon the modified antiphlogistic treatment in vogue during the past half of the present century, is still as a rule faulty ; a survival of the old treatment based upon erroneous

[1] For most of the previous account of the treatment of pneumonia in former days, I am indebted to the admirable monograph on the disease by the late Dr. Sturges.

conceptions of pathology. We still realise imperfectly that all pulmonary inflammations are not alike, and continue to treat croupous pneumonia much as we do bronchial catarrh. The acute infective inflammation of the lung in true croupous pneumonia cannot be amenable to the same remedies as the simple catarrhal inflammation of bronchitis. The ipecacuan and iodide, and alkalies, and ammonia, and squills, and poultices, and steam, which, from their known physiological action, may reasonably be expected favourably to influence catarrhal inflammation of the mucous membrane, cannot stay the microbic invasion of the specific infection of pneumonia; and yet this is the system largely followed still in spite of recent discoveries in pathology.

We constantly find a patient, with all the signs of acute croupous pneumonia, taking diligently a mixture of ammonia and squill and ipecacuan, swathed in hot heavy poultices, in a heated steamy atmosphere, and this although he is oppressed with dyspnœa, and has a temperature of 104° to 105°. Pounds of linseed poultice are put on to the top of his chest, and although his body temperature is already raised to the level of safety, he is heated up still more, and the natural cooling transpiration interfered with by the hot, moist covering.

Jacket poultices especially, those which envelope the whole chest, so much thought of in years gone by, are instruments of torture, and they not only add to the sufferings of the patient, but materially interfere with

his chance of recovery. The patient does not require heating but cooling.

Only a few weeks ago I was called to see a patient in the country, a child with acute pneumonia of the most severe form. The lung was solid from base to apex. The respirations 50, the pulse 140, the temperature 105°. The child was restless, distressed from fever and embarrassed breathing, and yet, to aggravate fever and respiratory difficulty, its chest was enveloped in a heavy jacket poultice. The removal of this and substitution of an ice-bag, lowered the temperature, gave great relief, and proved, I believe, the turning point to recovery.

While some practitioners lag behind in this old routine procedure, others, impressed by the marvellous progress in our knowledge of the physiological action of drugs, advance too rapidly ; are prone to a mischievous activity in the use of powerful agents, far more deadly when misapplied than ipecacuan, and ammonia, and squills, and poultices, such as veratrum, veratria, aconite, antipyrin, antifebrin, calomel, quinine in enormous doses, and so on.

There is, however, a more reasonable treatment, consistent with modern physiological and pathological knowledge.

The lines on which it should proceed are these. First, observe the golden rule in medicine, viz.—*To do nothing unless you see your way clearly to doing good.*

This rule is more vital in pneumonia than in almost any other affection.

There are, however, obvious dangers to be encountered which we are often compelled to fight against by active measures. They are chiefly three, viz. :—

1. Carbonic acid poisoning from deficient aeration.
2. Heart fai ure ; the greatest danger of all.
3. Pyrexia, the most feared and fought against, but in reality in this disease, I believe, the least dangerous of the three.

With regard to the first, nothing can be done directly, at the moment, to increase the aerating surface of the lung, encroached upon by the exudation into the air cells. Yet much can be done in the way of supplying the damaged aerating apparatus with pure oxygenated air.

It is of great importance that your pneumonia patient should be in a large well-ventilated room, freely supplied with fresh warm air, but not overheated, or saturated with steam. Remember croupous pneumonia is not a catarrh. When there is great cyanosis and tendency to asphyxia, inhalations of oxygen are of service. They give immediate relief, the blueness declines to almost a natural colour ; but the improvement is transient, and the inhalation has to be frequently repeated. I confess that I have been disappointed in the power of oxygen to unmistakably and materially influence the final result. It is, however, a good and reasonable device in all cases to keep an oxygen cylinder slowly discharging into the room or under the bed canopy, especially if the room be a small and close one, or if the weather is foggy, still,

and oppressive; and if cyanosis occurs to give regular hourly inhalations.

Cyanosis is due, not only to the choking of air vesicles, but also in part to the obstruction to the pulmonary circulation, which results from capillary occlusion and deficient aeration; and measures, such as bleeding and leeches, which lessen pressure in the right heart, and heart tonics, which increase its driving power, help secondarily to relieve cyanosis.

This brings me to the second great source of danger, viz. Failure of the overtaxed heart. I need not repeat in detail what I said upon this point in speaking of the treatment of bronchitis. The remedies are first strengthening, stimulating foods, especially the meat teas and essences, or raw meat juice, and milk.

Being careful always not to overfeed, and thus over-distend the stomach, and interfere with respiratory movement. One and a half to two pints of beef tea, and two pints of milk (peptonised if necessary) in the twenty-four hours, given in small quantities at a time, are amply sufficient: in addition cardiac tonics, digitalis, strychnia, and the like; ammonia, bark, with alcohol in full quantity, unless cyanosis be great. In this case, or if there be extreme drowsiness and tendency to coma, it is well to trust to strychnia and digitalis rather than to alcohol.

I have, I am convinced, seen life saved more than once by the wise use of cardiac tonics. Pneumonia is a disease of short duration, and we can venture to stimulate freely when heart failure threatens without

exhausting the heart's reserve force. In one case under my care, of which I spoke in the lecture on Tonics, recovery took place in a condition apparently hopeless from the very outset, under the free use of digitalis.

Let me briefly recall it to you.

The patient, a woman of fifty-five, who had been tapped several times for ascites due to atrophic alcoholic cirrhosis, and who was feeble and emaciated as well as dropsical, was taken while in hospital with acute pneumonia; she became delirious, and lapsed into a semi-conscious condition, which is called a typhoid state, with dry tongue, fluttering pulse, and marked cyanosis. In a disease of enfeebled nutrition like cirrhosis, recovery from such extremely grave symptoms seemed impossible, and I expressed this opinion. Digitalis was given in the full dose of fifteen minims of the tincture every four hours, and six ounces of wine. After a time the pulse revived, and, to my unqualified surprise, the patient had a favourable crisis and recovered. There can be no reasonable doubt that this was directly due to the powerful sustaining influence of the drug and wine. In pneumonia, digitalis is usually well borne, but you must be watchful of its effect upon the pulse; and if the rate falls to 60 or less, or if its rhythm becomes irregular, owing to the diastole now and again being unduly prolonged, moderate the dose, and add strychnia, or substitute it in full dose. Do not, however, stop it abruptly, as is so often done, unless the disorder of pulse is marked. The heart feels quickly the loss of the stimulant and is apt to run down again

dangerously. This is a mistake which is frequently made not only with digitalis but with many other drugs of powerful action as well, such as opium and alcohol, for example; and I have seen disaster follow.

Do not again, on the other hand, bring your cardiac tonics into play too early, although since, as I said, true pneumonia is, as a rule, a short disease, this is not so important as in disorders which run a more protracted course, like typhoid or broncho-pneumonia.

Of the other means for relief of the right heart, viz. abstraction of blood, venesection should be resorted to only in extreme cases, when cyanosis is great, and it is obvious that if life is to be saved some decisive measure must be resorted to. Bleeding from the arm is not suited to the case of children. Moreover, although I have seen venesection give great temporary relief, I have never seen it finally turn the scale in favour of life. Leeches may be advantageously substituted for venesection in less urgent conditions of over-loaded heart.

With respect to the third source of danger, viz. Pyrexia, its importance is, I think, overrated. Although the range of temperature runs high in this disease, I have never known a patient with simple pneumonia actually to die of hyperpyrexia. The extraordinary revival of the patient, immediately after the crisis, when active disease is over and the temperature falls, seems to show that the pyrexia has done no great harm to the tissues and heart muscle. Moreover, as Dr. Sturges pointed out, in all fatal cases with high temperature

post-mortem examination has disclosed a lung damaged beyond recovery; and the high temperature must be regarded as rather a consequence of this, than in itself a cause of death. Further, temperature affords by no means a correct measure of the degree of danger in pneumonia. In some of the most fatal forms of pneumonia (as for instance in alcoholic and septic pneumonia), the range is often not very high, although the danger may be extreme. Do not, therefore, make it the great object of your treatment, as so many do, to keep down the temperature by hook or by crook. Above all, abstain absolutely from tampering with the so-called antipyretic drugs, such as aconite, antifebrin, veratrin, antimony, or salicylate of soda. They lower temperature, but at the fatal cost of increased heart feebleness. I have I feel confident seen death hastened by them, and statistics give unfavourable results with regard to many, such as antipyrin and calomel, for instance,[1] and even as to large doses of quinine of twenty or forty grains which produced marked cardiac weakness, twitching of muscles, and tremors.[2]

Although, however, I think the danger of high temperature in pneumonia has been exaggerated, and that great harm is often done by attempting its reduction, especially by antiseptic drugs, and making it the chief object of treatment, yet pyrexia is a source of distress, disturbance, and degeneration, which may be beneficially controlled by therapeutic art. The only

[1] Botkin and Posadski, St. Petersburg, *Clin. Gaz.*: 1885–6.
[2] Ripley: *Ann. Univ. Med. Soc.*, vol. iii. p. 348.

really safe and satisfactory means of doing this is by the application of cold to the surface. The credit of first using the application of external cold to the reduction of febrile temperature belongs to Dr. Currie, of Liverpool, one hundred years ago. He, with Dr. Brandreth and Dr. Gerard, adopted it in typhus, scarlet fever, smallpox, and ague, and it was actually in general use in Liverpool in 1797.

Niemeyer, however, as far as I know, was the first to advise the use of cold in pneumonia, by means of compresses of cold water wrung out and changed every few minutes. This principle was extended by Jurguensen and Liebermeister, who used cold baths.

Compresses are, however, troublesome and inefficient, and cold baths, I think, objectionable, on account of the wear and tear involved in lifting the patient about, and the exhaustion this causes.

Far better than either of these are cold sponging, the wet sheet, or the ice-bag advocated by my colleague Dr. Lees. The first and last are most easily applied and the most easily regulated.

In ordinary cases cold and tepid sponging answers all purposes. I have chiefly used the ice bag with the object of reducing body temperature, and for this it answers admirably, and is usually grateful to the patients, even to children. Possibly it helps also to arrest the spread of the disease and to hasten resolution. I have so far seen nothing which leads me to the certain conclusion that pneumonia can be cured by the use of ice-bags, although pyrexial symptoms are relieved

and the course of the disease is certainly not unfavourably influenced by their application. I must, however, confess that I have not used this method energetically and systematically except in extreme and urgent cases.

Dr. Lees is disposed to think that he can arrest the advance of pneumonia by the application of ice, the change being evidenced by distinct improvement in the physical signs; and that the disease may abort altogether if the ice treatment be applied vigorously within twenty-four hours of the initial rigor. I hope that he is right, but I must suspend judgment. He regards the action as local and direct, by cooling of the affected area; and points out that if this area is large, several ice-bags are required. He wisely insists upon the importance of keeping the rest of the body warm by hot bottles, and of watching the effect of the treatment upon the general body temperature and circulation, suspending the applications if these become too depressed.

A troublesome symptom, and one usually of grave augury, present especially in cases of severest type, and in alcoholic subjects, is active delirium, which is sometimes almost maniacal.

An ice-cap or ice-bags to the axillæ, with alcohol in full quantity of from ten to twenty ounces daily, and tincture of digitalis in doses of ten to twenty minims every four hours, have proved the most efficient remedies in my hands, especially in alcoholic subjects. Bromides with hyoscyamus are sometimes useful, but chloral should be avoided from its depressant property. Hypodermic injections of morphia and hydrobromate of

hyoscine must be used in urgent cases when other means have failed.

Another symptom which is difficult to deal with in pneumonia is sleeplessness. Narcotics, such as opium and morphia, are not advisable if there is extreme cyanosis, but at the commencement of the attack are safe and effective. Later, bromides, henbane, and alcohol, and tepid sponging, must be chiefly relied upon.

So far I have been speaking of pure croupous pneumonia with regard to which treatment is straightforward and well-defined.

When I turn to broncho-pneumonia, and that curious creeping mixed form which accompanies influenza, or the hypostatic pneumonia of typhoid and the like, the matter is not so simple. In catarrhal pneumonia complicating bronchitis the treatment is mainly such as I have laid down for bronchial catarrh. Ipecacuanha, iodides in moderate doses, alkalies in full doses; while cold sponging and a local ice-bag appear grateful and harmless under due precautions when the temperature runs high, or the area of pneumonic consolidation is extensive.

When pneumonia, whether croupous or catarrhal, is secondary to influenza, typhoid, or to mitral disease of the heart, full stimulation and the use of the cardiac tonics are especially necessary to ward off the exceptional tendency to fatal heart failure.

The *last acute pulmonary affection* of which I intend to speak, viz. *Pleurisy*, is in many respects far more amenable to medical treatment than pneumonia.

Time will only permit me to discuss one or two leading points in connection with it.

The vessels of the costal pleura are in intimate communication with those of the skin and subcutaneous tissues. We can relieve the circulation in the pleura by relieving that of the superficial vessels within our reach.

One of the great troubles in pleurisy at the outset is pain. It distresses the patient, whose every breath is painful, every cough agony; it prevents sleep, and raises the temperature. It is caused, no doubt, by the rubbing of the two swollen tender surfaces of the inflamed membranes together.

A few leeches applied over the painful spot and followed by an ice-bag usually give immediate relief. These are far more effective than the mustard poultice, and turpentine stupes, and fomentations, and glycerine of belladonna, usually applied. More efficient too, and more satisfactory in ultimate results, than strapping the side, which, although it eases pain by arresting movement, tends to favour extensive close adhesion of the two surfaces of the pleura. You may observe the success of this treatment by leeches and cold in several cases now in hospital, notably those in Alexandra Ward.

In many cases pleurisy is of rheumatic origin, as in the cases of Minnie P., a girl in Alexandra Ward, and Florence S., in Albert Victor Ward, where the pleurisy was accompanied by articular rheumatism, and possibly in those of Caroline J., in the Alexandra Ward, and Edward W., in Albert Ward, where pericarditis has

accompanied the pleurisy, although the joints are unaffected. This view is supported by the fact that acute rheumatism and pleurisy are both extremely prevalent, apart from each other, in London at the moment.

Only a few days ago I was consulted in a case of acute pleuropneumonia, in which there were profuse sour perspirations of typical rheumatic odour, but no joint affection. The patient has improved rapidly upon large doses of alkali and quinine. I believe it is rheumatic in origin. Sir W. Broadbent, who had previously seen the case, came to the same conclusion.

Seeing that many of these pleurisies are rheumatic, is it safe, you will ask, to use the ice-bag? I believe so, with proper precautions against general chill.

Look at the case of Florence S., in Albert Victor Ward, with double pleurisy, pericarditis and effusion, and joint affection. The ice-bag has been applied freely, the pleurisy and pericarditis have subsided most satisfactorily, no rheumatic joint trouble or bronchial catarrh has followed. Similarly in two other rheumatic cases. In the case of Florence S., in Alexandra Ward, only has there been any return of joint affection. But such relapses are common when cold is not applied, and it has in this instance been slight.

Lastly, I must say one or two words with regard to pleural effusion. I cannot deal with the matter in detail here, but there are some points which I should like to impress upon you.

If the effusion is copious, even if it is serous only, remove it without delay, especially if it is on the left side, where it causes greater displacement and embarrassment of the heart than on the right. The patient is in a position of some danger, although he may be apparently breathing quietly, with little or no distress ; yet the oppressed, displaced heart, is liable to stop suddenly on slight exertion. Twice I have seen fatal consequences follow delay in removing large effusion into the left pleura.

In one instance the patient was in the country, where an aspirator could not be procured until next day. During the night the patient woke from sleep, sat up in bed, and died on the spot from syncope.

In the other case, the doctor in attendance lived at a distance. It was late at night, the symptoms were not urgent, and we agreed that aspiration might safely wait until the morning. In the night the patient was allowed to get out of bed to micturate, and died instantly of cardiac syncope.

In both these instances the effusion was on the left side, but I imagine the danger is only slightly less when it is into the right pleura. In all cases of copious effusion therefore aspirate without delay.

LECTURE VIII

THE FORMS OF CHOREA AND THEIR TREATMENT

Chorea—The origin of the name—Two forms—Grimacing or habit chorea—Genuine or rheumatic chorea—Habit chorea—Symptoms—Relation to second dentition—Allied conditions in adults—Treatment—Prognosis—Acute rheumatic chorea—Symptoms—Complicated relation to rheumatic fever—Motor disturbance of central origin—Treatment—Remedies harmful or unreliable—Remedies that have proved serviceable—Rest—Arsenic—Iron—Strychnia—Chloroform—Chloral—Salicin and alkalies—Indications for the use of these drugs—Importance of frequent examination of the heart.

You have probably formed your conception of chorea chiefly from the cases you most commonly see in the hospital wards, of which there are several under my care at this moment. These cases are indeed examples of what may be called genuine or true chorea—ordinary chorea characterised by disorderly muscular movements, such as jactitations of the limbs, grimacing, and the like; and this is the important chorea, and the one of which I shall chiefly speak, since, from its close association with acute rheumatism, it is liable to be accompanied or followed by serious disease of the heart—the result of endocarditis, pericarditis, and I believe frequently also myocarditis, and simple dilatation.

The term chorea was originally applied to an entirely different disorder, to which it was much more appropriate, one which seems to have been more allied to hysterical mania than to anything which we can recognise as chorea now, and it is unfortunate that Sydenham should have caused confusion by adopting the title for a totally distinct affection.

The name was given in the first instance to the wild dances of the religious enthusiasts of the middle ages. They were possessed by an irresistible impulse to muscular action, increased to a veritable mania by mutual incitement and imitation—or the sound of music.

A most graphic description of this original mediæval chorea is given by Sir Thomas Watson :—

'Some, impelled by a strange and unavoidable necessity, executed measured and regular movements with surprising energy, rapidity, and perseverance. When music was performed in their hearing the movements became an actual dance, and when crowds were collected together, the mania was apt to spread from person to person by a sort of imitative infection, realising the fable of Orpheus.'

It has been suggested that the term 'Chorea Sancti Viti' is in reality but a vulgar corruption of 'Chorea Sancti *In*viti' (the dance of the unwilling saint), and took its rise in the misfortune of some holy person who chanced to be seized by one of these invincible but unwilling impulses to caper.

There are many forms of involuntary spasmodic

muscular movements, some ordered, some disordered, which have been classed with chorea, but I wish to direct your attention more particularly to the two forms with which you will have chiefly to deal in actual practice, and which it is of great importance that you should learn to distinguish one from the other —not always an easy matter—viz. :

(1) 'Grimacing.'—What is called—or rather miscalled—'Habit Chorea,' and (2) 'Acute Genuine or Rheumatic Chorea.'

The first of these you will probably see little of in your student's course. These cases do not come into hospital, and are not common, I think, in the out-patients' rooms. They are met with chiefly amongst the more neurotic children of the well-to-do, the reverse of true chorea, which is more common amongst the poor.

Habit Chorea has, I think, been badly named. It is not at its outset, and in its origin, an acquired habit. It is involuntary, a local chorea, reflex in origin, and analogous to the twitching of muscles seen sometimes in the first dentition.

Like rheumatic chorea, it is met with chiefly in childhood, but it has a shorter range period, being limited pretty closely to the age of from eight to twelve. It rarely appears earlier, and rarely later.

This grimacing chorea is characterised by spasmodic contraction of certain muscles of the face, neck, and occasionally of one or other arm or shoulder. As far as my observation goes, it never affects the lower limbs.

The patient may be restless or fidgety, but there is no general jactitation, as in true chorea. In this lies one important mark of distinction. Grimacing resembles true chorea in that the movements are involuntary, but differs in that they are limited.

The same muscles are always affected in the same way, and there is no derangement of general health; no association with rheumatism or other general disease.

The involuntary movements vary in different cases, the most common being winking of the eyes, corrugations of the nose, sniffing and snorting of the nostrils, rapid and repeated elevations of the eyebrows, little shakes of the head, shrugs of one or both shoulders.

In some instances, facial spasms have been accompanied by stammering. In two cases within my experience this has been of the most severe kind; in one, it became so extreme that at last no intelligible speech could be uttered. The attempt to speak brought on such acute stammering that no effective articulation was possible. Both these patients recovered perfectly after many months.

Now in true chorea also speech is sometimes entirely lost—in severe cases—lost even for weeks or months. The muscles of articulation cannot be co-ordinated to produce the proper succession of sounds; but there is no stammering as in these cases of the grimacing form, and herein lies one point of distinction.

I have said that these cases are met with in

children chiefly between the ages of eight and twelve. They are usually neurotic and emotional children—frequently of neurotic stock. There is a neurotic basis, but the immediate exciting cause is not so obvious. The disturbance has nothing to do with rheumatism. The spasms are not set up by fright, or by overwork, or by great excitement, the immediate *excitants* often—not the prime *cause* of true chorea—although they are aggravated by them. There is no breakdown of health. Except for the grimacing the children are usually well and hearty. Not even the much-maligned thread worm can be charged with participation in this case.

The only clue I can find to their causation is the fact that the period of life at which they arise corresponds closely with that of the second dentition, and the affection usually reaches its height with the eruption of the eye teeth and late molars, and ceases as dentition is complete. I am inclined, therefore, to think that the spasm is due to the disturbance of second dentition, and analogous to the twitchings and convulsions of the first dentition. The special implication of the facial muscles to which the disorder is often entirely limited, supports this view. I saw yesterday a boy of nine and a half who came to me with twitching movements of one shoulder: no face grimacing. He was under my care one and a half year ago with grimacing, in the form of incessant winking of the eyes. At that time two upper incisor teeth of great size, and the first molars, were pushing

through the gums. When they were through the grimacing ceased, and he has remained free until now. At this moment, the bicuspids and canines are swelling out the gums.

These concurrences may be simple coincidences, but I have seen many of the kind, and I shrewdly suspect that second dentition and grimacing stand in the relation of cause and effect.

Although this choreic spasm usually arises in the period of childhood named, some of the movements may become permanently established and remain during life.

We are, most of us, I dare say, acquainted with people who are troubled by uncontrollable tricks of this sort. I can call to mind several. One man snorts and sniffs in the most unpleasant way when he is addressing you, another jerks his head away every few minutes. Sir Thomas Watson mentions the case of a lad in his service who had the awkward trick of moving his head so that he seemed to be giving his master and his friends a familiar nod, and he was obliged to part with him on that account. I am not sure whether these strange movements are survivals of the grimacing chorea of childhood, or have arisen independently in later life.

The most painful example of facial spasm which I have seen, due, no doubt, to some local irritation of the motor branch of the fifth nerve apparently seated on the left side, was in a patient of Sir Edward Sieveking's, in this hospital. He had violent spasmodic contractions

of the masseter and temporal muscles, so that his mouth shut with a loud snap of the teeth every few seconds. The result was that not only was it difficult for the man to eat or sleep, but the constant attrition wore the teeth away to the level of the gums, and he was obliged to apply an india-rubber pad to ease the shock and erosion of tissue. The spasms almost disappeared under large doses (℥ss to ℥i.) of succus conii three times a day. When the toxic effect of loss of power in the lower limbs was produced, the snapping of the jaws became very feeble and nearly ceased. When the drug was withdrawn, however, the spasms returned as fiercely as before. It was proposed to cut the motor branch of the fifth nerve on the left side, but the man declined and went out no better than when he came in.

Treatment of Grimace Chorea.—As I said, the patients are usually in excellent general health, and little is needed beyond care to keep them so; simple aperients, if required, simple diet, avoidance of undue excitement, and, above all, of harsh punishment or constant rebuke or teasing. Such things infallibly do harm; the peculiarity should be little noticed or altogether ignored. Nerve tonics, such as arsenic or strychnia, are useful in certain cases accompanied by anæmia or debility; but as a rule treatment by drugs is not required. Be sure, however, in all these cases to satisfy yourself thoroughly that the affection is really grimacing, and not a slight case of true chorea. The limitation to the face and non-implication of limbs, the

ability to hold out the tongue steadily, and the history will help you to discriminate. Never omit, however, to examine the heart carefully from time to time. It is in the rheumatic connection and liability to carditis of true chorea that all danger lies.

In this grimacing chorea the parents are commonly distressed by the grotesque movements. You may reassure them, and tell them that, although the movements may persist or recur for months, they will eventually finally disappear when the second teething is fully completed. All the cases I have seen, including the stammerers, have got absolutely well before the time of puberty.

Acute Rheumatic Chorea.—I turn now to acute, genuine, or rheumatic chorea. I will not describe in detail the features of an affection with which you must be more or less familiar. It has been described as an exaggerated fidgetiness, an exaltation of that condition of unrest which is characteristic of childhood. The movements are irregular, jerking, sudden—affecting facial muscles and causing various grimaces. The tongue pushed in and out incessantly, fingers, arms, trunk, lower limbs, in constant jactitation, but it is to be noted that the lower limbs are never affected in the same degree as the upper, and never alone.

Moreover, there is marked loss of muscular power, sometimes developed to distinct paresis. The patient, in extreme cases, is unable to stand, or even sit up in bed. Most frequently, but not always, the paresis is unilateral.

The violence of the movements may be so great as to necessitate keeping the patient in a padded bed, or bound down by a sheet. In some severe cases the power of speech is entirely lost, it may be for months.

In one case under my observation, speech was absolutely lost for eighty-one days—nearly three months.

In the case of a girl of sixteen with rheumatic purulent pericarditis and violent chorea, in Alexandra Ward, some years ago, speech was entirely lost, and the patient was utterly unable to describe her sufferings or state her wants.

In some rare instances these cases of violent chorea end fatally, as in both these mentioned ; the patient dies exhausted by the incessant exertion or want of sleep, aided often, as in the preceding instance, by pericarditis. It is most dangerous in the rheumatic cases, in females between thirteen and twenty-three, and in pregnancy.

On the other hand, the jactitations may be limited to slight fidgeting of arms and legs, with grimacing and tongue movements.

The movements cease entirely during sleep. Occasionally mental aberration follows, and the emotional factor is always well marked.

Now this true acute, or rheumatic chorea is like grimacing chorea, a disease of childhood especially, but its age range is greater.

It is most common between six and fifteen. It is

most often met with in girls, two or three times as frequently as in boys. It is of uncertain duration—the average being about thirteen weeks, and it is especially liable to recur.

One of the points of great interest in relation to chorea is the singular connection it has with heart disease. Endocarditis, pericarditis, myocarditis, with the consequences of these in valvular incompetence, dilatation, and hypertrophy, are closely associated with chorea. Dr. Lees and Dr. Poynton have shown that simple dilatation without valvular lesion is also found in chorea as in rheumatism.

It is in this connection that the gravity of an attack of true chorea centres. It is again closely associated with acute rheumatism, and the question of the relation between chorea and rheumatism is one which is of great interest, and has been largely discussed.

It is impossible to state the question in detail here; for a full presentation of the subject, I must refer you to my address at the meeting of the British Medical Association in 1895; my lectures on 'Rheumatism in Children'; and the article upon the same subject in Professor Allbutt's 'System of Medicine.'

I will merely point out that chorea arises not only in direct connection with acute rheumatism of the joints, but with endocarditis, pericarditis, erythema, scarlatina, apart from any joint affection, and, above all, with that special rheumatic sign, the eruption of subcutaneous nodules. Chorea, moreover, is especially

liable to occur in members of families in whom rheumatism is rife, even when it is unattended with any other rheumatic sign.

Of the three cases under my care now, two have a rheumatic history, the third has had scarlatina.

The chain of evidence is a remarkable one, and the conviction is gaining ground that in the majority of cases, at all events, chorea is one of the expressions of the rheumatic state; and that the cardiac affections of chorea are invariably of rheumatic nature.

That the motor disturbance in chorea is of central origin appears certain from the fact that the movements cease during sleep; the hemilateral paresis, and the emotional excitement, which is a constant symptom, the occasional temporary dementia, the influence of excitement upon the movements, all point to the cortical area as its seat, and to irritation of this, or hyperexcitability of this, as the disturbing agent. Morbid anatomy has so far failed to identify the changes.

The emboli, perivascular hæmorrhages, the patches of degeneration found in some fatal cases, have been absent in others, and no constant morbid change has yet been established.

Treatment of True Chorea.—Perhaps there is no disease for which more specifics have been vaunted than for chorea. Iron, sulphate of zinc, strychnia, arsenic, by way of tonics. Antimony, antipyrin, conium, bromides, chloroform, chloral, opium, henbane, belladonna, as sedatives.

Shower baths, galvanism, ether spray to the spine,

massage, gymnastic exercises, have all been credited with wonderful powers.

Yet many experienced physicians doubt whether active treatment—by drugs at least—has any beneficial action.

The fact is chorea has a tendency to get well, as a rule slowly, but occasionally very rapidly, under proper care and rest in hospital, and when the administration of any particular remedy chances to coincide with the amendment, the medicine is credited with the success really due to nature.

No doubt many drugs are useless, some directly hurtful; but there are one or two which give valuable assistance in chorea, and the same may be said of the other adjuncts of treatment, such as gymnastics, massage, and the like.

It will be well, perhaps, in the first place to put aside the remedies which have in my experience proved unreliable or hurtful, and thus narrow down the list to the few which serve a distinctly useful purpose, and then to explain how and when these are to be used in varying conditions of the disease.

You may, I think, put aside, to begin with, sulphate of zinc, antimony, antipyrin, conium, morphia, bromides, henbane, belladonna, amongst the drugs.

Sulphate of zinc had a great reputation at one time, and was given in increasing doses. I used it largely in former days with no decided result.

Antimony was used by Trousseau and others in large doses of tartar emetic, with the view of calming

the motor excitement. The practice was to give it in three series of three days each, with intervals of four or five days between—five grains in the first series, eighteen grains in the last. Trousseau took objection to the plan on the ground that the time required to carry it out was too long. My objection is that the plan is injurious —even dangerous. Two of the only three fatal cases of chorea I have seen were treated by full and repeated doses of antimony. This slightly calmed the symptoms but produced extreme depression without bringing sleep, and the drug was, I fully believe, the chief cause of the exhaustion and collapse which finally proved the cause of death. In chorea there is apt to be not only endo- and pericarditis, but also primary dilatation of the heart, with a feeble irregular pulse; and powerful cardiac depressants, like antimony, are completely contra-indicated. Therefore eschew antimony altogether.

Antipyrin, which has recently come into vogue, is objectionable on similar grounds.

Conium appears to have no effect, unless pushed to the final extent of causing paresis of lower limbs and staggering gait, and the patient is no better when the drug is suspended. It only temporarily paralyses, it does not cure.

In the same way morphia is a temporary sedative only, and complicates matters by disordering digestion and bowel action.

Bromides are chiefly useful in cases of great excitement and in combination with chloral. Belladonna and henbane have, in my hands, been quite ineffectual.

The shower-bath, although it may be useful in the case of adults, does harm to children; it frightens little children terribly, and makes the chorea ten times worse than before.

Galvanism in like manner terrifies children and aggravates the disorder. It can do no good, since the seat of disease is in the cerebrum—out of reach.

Ether spray to the spine, at one time in vogue, is useless. Cooling down the cord could do no good; it is based on a mistaken idea as to the centre of disordered action, which is in the brain—not the cord.

Massage and gymnastic exercises are perhaps of some value in giving tone and lessening hyper-excitability, but they are in no sense curative, although useful aids to cure.

This leaves us with iron, strychnia, arsenic, chloral, to which must be added two powerful agents for good—rest in bed, and avoidance of all exertion and excitement.

Let us now see how these are to be used. First of all, rest in bed. It is necessary to calm down motor excitement and also to guard the heart from strain in its enfeebled, possibly injured state. With this avoidance of mental excitement also, *i.e.* mental rest as well as bodily rest. As you know, the immediate exciting cause of chorea is frequently shock or fright, or mental excitement, and any recurrence of this is apt to produce relapse.

A most striking instance of the evil effect of excitement and emotion came under my notice a few years ago. I was summoned to see a girl of eight years old at the Grand Hotel. I found her tumbling about on

the sofa in a most extraordinary manner. When I entered the room the movements became so violent and uncontrollable that she rolled off on to the floor, and it was only by the nurse and mother forcibly holding her that I was enabled to examine her.

I found that she had had chorea following upon slight articular rheumatism, with endo- and pericarditis a few weeks before, in Dublin. When free from choreic movements, the parents brought her over to London for further advice, two days before my visit. The day after her arrival, that is the day before I saw her, she was taken sight-seeing, and in the evening to the pantomime. The following morning the violent chorea I witnessed set in. Under rest and arsenic, however, she quickly recovered.

Rest and quiet, then, is the first prescription.

Arsenic, Iron, Strychnia.—With rest, arsenic in increasing doses, if there is no fever, and digestion is undisturbed. I am not sure that tonics have much influence, but if any one does good it is arsenic. Arsenic appears to have a two-fold effect—it relieves the anæmia, increases red corpuscles, and it is also said to diminish the irritability of the motor nerves. It appears to be especially beneficial in paretic or paralytic chorea. I collated a number of cases some years ago, and found that the duration in hospital of cases treated by arsenic was twenty-four days, as compared with a forty days' stay of those not thus treated.

You will watch, of course, the effect of increasing doses—the first toxic sign, the silvery coating of the tongue, vomiting, the suffusion of eyes and skin, ery-

thema, urticaria or albuminuria. Watch, moreover, for any sign of neuritis apt to follow the free use of arsenic. I have now a patient suffering from general peripheral neuritis, the result of treatment by arsenic for severe chorea. If any of these effects occur, suspend the drug ; give a mild purge and recommence with smaller doses. Iron is used only as an adjunct to relieve anæmia, strychnia to tone up the flabby, and possibly fluttering, irregular heart.

Let me give you a further warning as to the use of arsenic. If there is any pyrexia, and, above all, if there is any sign whatever of rheumatic or cardiac affection, do not give arsenic. It interferes with elimination and increases pyrexia. Give salicine (rather than salicylate of soda) with citrate of soda in full and frequent doses. If there are any drugs which have a favourable influence in the arrest of rheumatic cardiac inflammation, they are salicine and alkalies.

Chloroform and Chloral.—In cases of severe jactitation, preventing rest and sleep, not only may a padded bed, and binding down by a sheet be necessary, but even chloroform may be required to induce quietude and sleep. I have, however, as a matter of fact, never met with an instance in which chloral, given by the mouth, was not sufficient for the purpose. It must be administered in moderate doses, frequently repeated, *i.e.* in doses of three to five grains, for a child from seven to twelve years old, every four hours. For this purpose of giving peace and rest it is invaluable. I am convinced that I have seen life saved by it more than once. It

does not cure, but it relieves wear and tear, ensures sleep, and gives time for restoration of strength, and thus prevents fatal exhaustion. In spite of the frequently enfeebled state of the heart, I have never observed any ill-effects from the continued use of chloral in this way.

In one instance which I remember, inability to sleep was caused in a very curious way. I was called in to see the patient, a girl of nine years old, chiefly on this account. The child was becoming exhausted for want of sleep. The jactitations were severe, but not so extreme as to prevent sleep altogether in most cases. On examining the patient I found one wrist red, swollen, and intensely tender. The incessant movement and tossing about of the inflamed limb caused excruciating pain, and this was the prime cause of insomnia. I suggested the arm should be bandaged to the side, and the child slept comfortably afterwards without hypnotics.

Finally, let me repeat the warning as to constant watchfulness. Examine the heart frequently. Look for nodules, for any sign of joint affection, for pyrexia. Remember the heart inflammations of rheumatism and chorea are apt to come on insidiously and unexpectedly, and are constantly overlooked.

If any of these signs arise, treat the case as one of rheumatism rather than chorea; with salicin and alkalies, or with quinine and alkalies, rather than with salicylates, unless the joint symptoms or pyrexia are severe. For the danger in chorea lies not in the chorea itself, except in rare cases of extreme severity, but in the cardiac inflammations which accompany it.

LECTURE IX

INFANTILE SCURVY

The prevalence of scurvy in childhood—Symptoms identical with those of adult scurvy—Morbid anatomy also identical—Rickets and scurvy essentially different diseases although often associated—Causation of infantile and adult scurvy identical—Due to lack of fresh food—Diet on which scurvy has developed—Feeble anti-scorbutic power of cow's milk—Effect upon milk of peptonisation and pancreatisation—Scurvy more common in the children of the wealthier classes—The condition often mistaken for stomatitis or rheumatism—Sometimes for congenital syphilis or anterior poliomyelitis—If hæmaturia or albuminuria, for nephritis—The diagnostic features of the disease—Illustrative examples of scurvy—Prognosis—Prevention—Treatment.

Appendix—List of sixty-one cases, showing the exact diet, progress, and issue in each instance.

TWENTY years have elapsed since I first drew attention in this country to the occurrence of scurvy in infants in sporadic form. The views I ventured to put forward were subsequently supported by the investigations of Dr. Barlow into the morbid anatomy of these cases, which he showed to be identical with the changes found in the scurvy of adults. Since then these results have been abundantly confirmed by other observers in this country, on the Continent, and in America, and the conclusions adopted by Dr. Barlow

and myself have, I think, been very generally accepted. As a proof of the interest which the question has excited, and of the unexpected prevalence of the disease, I may mention that no less than 106 cases were reported to the New York Academy of Medicine in 1894, in addition to a large number recorded in Germany and in this country. In the tenth volume of the American Pædiatric Society, published in 1898, a report on the subject is published, founded upon no less than 379 cases.

My purpose in this lecture is to give the results of my own clinical experience. It is not my intention therefore to describe the symptoms and morbid anatomy of the disease in detail. For that I would refer you to my article on the subject in the fifth volume of Professor Allbutt's 'System of Medicine.'

General Summary of Symptoms.—The symptoms and morbid changes met with in well-marked examples of sporadic infantile scurvy correspond not only individually but as a composite whole with the series of phenomena met with in the scurvy of adults occurring in epidemic form. The pallid, earthy complexion, the progressive anæmia, the spongy gums and carrion odour of the breath, the extreme muscular feebleness, the tendency to syncope, the various hæmorrhages and their seat, the hæmaturia or albuminuria, the œdema, the periosteal and muscular swellings of the limbs and their exquisite tenderness, the special implication of the lower extremities, are the same in both. The fact that the bones suffer somewhat more severely in pro-

portion to other tissues in the case of children is explained by the extreme formative activity which prevails there during the period of infancy.

Morbid Anatomy.—As the symptoms correspond with those of adult scurvy, so do the morbid changes met with after death—extravasations of blood under the periosteum, especially of the leg bones, fractures of the long bones usually by separation of the epiphysis from the shaft; sometimes also similar changes in the arm bones, scapulæ, and skull. Hæmorrhages and serous infiltration of the deep muscles—sometimes into the tissue at the back of the orbit, causing proptosis, hæmorrhages into the medullary cavity of the long bones, under the skin, from the bowels, from the kidney—ecchymosis in the internal organs, the lungs, pleura, spleen, kidney, intestinal glands. Put concisely, fragility of the capillaries, anæmia, extreme muscular debility, are the morbid changes which determine the symptoms of the scorbutic state, and afford the key to its pathology.

There are, however, in many instances of the disease in children other signs and symptoms in addition to those of scurvy proper, as seen in adult life, viz. those of an underlying rickets, such as bending of the ribs, enlargement of the epiphyses of the long bones, head sweats, laryngismus, and the like. In most cases, however, these signs are slight, and in some no indication of rickets whatever can be discovered.

The fact of the frequent association of infantile

scurvy with rickets led to the assumption of some essential pathological connection between the two conditions. The earlier observers in Germany regarded and described infantile scurvy as an acute form of rickets, and the disease has also been styled scurvy rickets, from the supposed pathological relation between the two affections. The essential independence of scurvy has, however, as far as I can ascertain, been generally accepted by the leading authorities in Europe and America, and is, I think, established by the following considerations.

Although, as has been stated, a certain degree of rickets is usually present, this is not a constant or invariable accompaniment, and there is moreover no relation, correspondence, or proportion between the degree of rickets and the degree of scurvy, nor indeed between the degree of rickets and the co-existence of scurvy at all. In severe and advanced cases of rickets where the bone changes are extreme, where there is marked cachexia, head sweats, laryngismus, and all the signs of severe and progressive disease, just the cases in fact where, if any pathological relation existed between the two, the scurvy changes might be expected to be pronounced, there is no affection of the gums, no sub-periosteal hæmorrhages, no muscular or subcutaneous extravasations, no hæmaturia, no hæmorrhages elsewhere. Rickets is not in itself hæmorrhagic in any degree. Moreover, as proved by clinical observation and post-mortem examination, all signs of rickets may be entirely absent according to positive

observations by Dr. Barlow and by myself, confirmed by a case recently recorded by Dr. Westhrup, of New York.

A final proof of the distinction between rickets and scurvy is afforded by the action of antiscorbutics. The rachitic state is no doubt powerfully influenced by dietetic treatment, but the effect is slow and gradual, in marked contrast to the immediate and rapid amelioration which follows the substitution of antiscorbutic diet in the case of scurvy. There is nothing in the whole range of medicine—not even excepting the effect of thyroid extract in myxœdema—more striking and remarkable than the marvellously rapid recovery, which follows the administration of fresh elements of food in these cases of infantile scurvy.

So that the scorbutic symptoms are not a mere excessive development of severe or acute rickets, but a morbid condition distinct from it although oftentimes superadded to it.

The explanation of the frequent although not invariable association of rickets with scurvy is afforded by the fact that the infant's diet which produces scurvy, by a distinct defect of another kind produces rickets also. A scurvy diet of dried food is usually also a *rickety* diet, deficient in fat and proteid, and perhaps phosphates as well as in anti-scorbutic property.

Causes.—As the symptoms and morbid anatomy of infantile scurvy are identical with those of the same disease in adult life, so the causes which produce it are identical also.

In the case of adults the affection has been traced to the lack of fresh food, and especially to the lack of fresh vegetable food, and the scorbutic state is intensified and fostered by conditions of malhygiene, by hardship, exposure, foul air, want of light and probably also by the prolonged use of salt provisions.

The nature of the anti-scorbutic element which exists in fresh food has never been satisfactorily determined, although there is evidence which suggests that it may consist in the presence of organic, vegetable acids in combination with potash. It has been suggested [1] that it is rather the simple incompatibility of food or the presence of some toxic agent or ptomaine developed by fermentation. This, however, appears to be negatived by the fact that scurvy gets well without change of the food which has produced it, except only the addition of an anti-scorbutic; if exactly the same diet be continued and anti-scorbutic alone added, the disease ceases. Of course this might be due to the action of the anti-scorbutic as an antidote to the ferment poison, but it is far more probable that it is due simply to the supply of a missing element. Neither has the exact nature of the blood change been ascertained with certainty, although the researches of Busk, Garrod, Ralfe and others point to defective alkalinity, owing to neutral salts, such as the chlorides being increased at the expense of the alkaline salts. Hence dissolution of blood corpuscles, degenerations of muscles, cells and

[1] Trans. American Pæd. Soc. 1898: Report of Committee of American Pæd. Soc. 1898.

other tissues, fragility or permeability of capillaries, hæmorrhages and ecchymosis. Whatever the nature of the element in which the protective virtue lies, it is certain that it exists in fresh foods, in greatest abundance in fresh vegetables, while it is defective or absent in dry farinaceous foods and preparations, and in salted provisions.

In the case of children, the cause has been traced with equal certainty to the deficiency of the fresh element in food. The natural, ordinary food of infants is milk alone. No instance of a child becoming scorbutic, when at the breast, has, as far as I know, been recorded—with the exception of one or two doubtful cases where imperfect details of diet are given—save only in those instances where the suckling mother has been herself suffering from scurvy at the time.

Period at which it arises.—The occurrence of infantile scurvy is therefore, as might be inferred, almost limited to the first two years of life, *i.e.* the period of bottle feeding, and most often between six and eighteen months. Later in childhood the wider range of food given, including almost invariably such anti-scorbutics as potato and other fresh vegetables and fruits, prevents its occurrence. In forty cases under my observation, twenty-three were under one year, ten between one and two years, three between two and three years, and two three years old, in one the age unrecorded. The only instances I have met with in older children are those in which there has been deficiency of fresh element in diet from some unusual

circumstance, as in the cases noted by Dr. Barlow, where a curious morbid antipathy existed to vegetables and fresh meat.

Further, no instance has come to my knowledge where scurvy has supervened on an ordinary diet of fresh cow's milk, unaltered, except by boiling. The aliment fresh milk must then contain the antiscorbutic element, since children fed on it alone do not contract scurvy; it is the normal anti-scorbutic food of infants.

Deficiency of fresh milk in the diet.—A careful examination of the conditions of diet in a large number of cases establishes the prime fact that children who get scurvy are brought up on a diet deficient in fresh milk. In sixty-one cases under my immediate observation, in which the details of feeding could be ascertained with precision, in the great majority no fresh milk had been given for a considerable period before the attack; in the majority of these, none at any period of feeding, and in the rest only at the commencement of the hand feeding. In the remaining cases very small quantities only had been given, and in some of these for a few weeks only. Lastly, in none of these cases had the deficiency of fresh food caused by privation of milk been compensated by the addition of any other fresh elements of anti-scorbutic virtue. So far with regard to defective supply of the normal anti-scorbutic food of children.

Foods on which scurvy developed.—The next point is with regard to the food they received in place of milk.

In a few cases the food was limited absolutely to dried farinaceous preparations, such as oatmeal or malted food made with water only.

In a number of instances (five) the scorbutic condition developed upon a prolonged diet of one of the proprietary preserved infants' foods, consisting of malted flour combined with dried animal matter, such as desiccated milk, and prepared by the simple addition of water, without any fresh element whatever. The number of other cases (five) arose upon simple condensed milk. More still upon the lengthened use of so-called humanised milk, with, in some cases, sterilised milk in addition.

The largest number of cases, however, have resulted from prolonged diet of peptonised and pancreatised food. Some on peptonised milk, both fresh and condensed, but the greater proportion on pancreatised farinaceous food, in which the added milk is peptonised in the process of preparation.

Feeble anti-scorbutic power of milk.—The anti-scorbutic virtue of cow's milk, as Dr. Parkes showed long ago, is comparatively slight and imperfect, so that large quantities are required to render it effective in either preventing or curing scurvy. It is possible also that it varies to some extent according as the cow is fed on dry food or on grass and roots. The relatively low, anti-scorbutic power of even fresh milk partially explains the occasional occurrence of scurvy in children who have fresh milk in very small amount.

From what has been stated above, it is clear that

prolonged heating, especially at high temperature, as in the process of desiccating, condensing, sterilising, impairs or destroys the already feeble, anti-scorbutic power of milk. Two cases of scurvy have come under my notice upon a diet of simple, sterilised milk, and other cases have been reported to me. The leading authorities in America and Germany, where it is more largely used than in this country, deprecate on this ground the permanent feeding of children on milk sterilised by protracted heating at high temperatures. The mere raising of milk to the boiling-point appears to have no serious deteriorating influence.

Effect of the peptonising or pancreatising process on anti-scorbutic power.—More curious and interesting still, however, is the effect of peptonisation and pancreatisation. Even fresh milk treated in this way, as in ordinary peptonised milk, and that added to pancreatised food, appears to be profoundly affected, and has the least scorbutic power of all. In this fact probably lies the key to the secret of the exact nature of the anti-scorbutic property in food.

Special incidence of the disease on the children of the well-to-do.—Another interesting fact comes out in connection with the development of scurvy in these artificial foods, and preparations of milk, viz. that the majority of cases occur amongst the children of the better class. Although the children of the poor are by no means exempt, scurvy is much less common amongst them than in the children of the well-to-do. These cases are rare in our hospital wards. Of the sixty-one

under my own observation, fifty-five were private cases and only six hospital patients.

This incidence on the children of the higher class is explained partly by the consideration that the artificial foods so productive of the disease are chiefly used by them. They are too expensive for the poor. The poor, however, on the other hand, use largely condensed milk and farinaceous preparations such as corn flour, which are eminently scorbutic foods. The reason why scurvy does not follow more frequently is to be sought in the fact that the children of the poor begin to share the food of their parents more early than in the class above, and thus have additions to diet of which the powerfully anti-scorbutic potato always forms a part.

It is worthy of note in connection with this point, that this incidence of scurvy on the rich, as compared with the poor, is the converse of what obtains in rickets, which is far more common and severe amongst the poor. This affords additional evidence as to the difference in the individual pathology of the two affections so often concurrent.

Lastly, I may remark, that infantile scurvy appears to be increasing in prevalence, especially in America, owing no doubt to the more extended use of artificial foods.

Diagnosis.—The recognition of a case of infantile scurvy is not difficult when the typical signs of periosteal tenderness and swelling are accompanied by spongy gums and subcutaneous hæmorrhages. And yet

it is remarkable how seldom the affection is recognised, even when the characteristic features of the disease are present. Indeed, the condition of the gums has even led people astray, two of the most extreme cases of spongy gums I have seen having been diagnosed as stomatitis. When the swollen, purple, spongy condition of gum is absent, as is apt to be the case in very young subjects, when the teeth are not actively pushing, and sometimes, in slight cases, when the teeth are present, the real nature of the affection is still more liable to be misunderstood.

The most common error is to mistake these cases for rheumatism. The swelling, immobility and tenderness of the limbs, often accompanied by slight pyrexia, at first sight, indeed, simulate rheumatism very closely. Scurvy may, however, usually be distinguished by the swelling being periosteal and muscular, almost invariably limited to the shafts of the long bones and deep muscles, often near the joint, but not affecting the joint itself as in rheumatism. In one instance in my experience, the periosteal swelling was over the malar bone, and in another the periosteal swelling of the lower part of the shaft appeared to extend to the adjacent joint structures. Such cases are, however, extremely rare.

Other diagnostic points are the intense character of the tenderness, far beyond anything seen in rheumatism, and the fact that infantile scurvy is practically limited to the period of bottle feeding, when rheumatism is almost unknown. I have seen only one doubtful case

of acute rheumatism in early infancy, although a few instances have been recorded by Senator, Henoch, and others. Possibly these may have been cases of scurvy, which was at that time hardly recognised. The existence of other signs of scurvy, such as spongy gums, subcutaneous or other hæmorrhages, hæmaturia or albuminuria, and a history of scurvy diet would determine the diagnosis. These are, however, not always present. The decision must then be deferred until the crucial test of anti-scorbutic diet can be applied. This quickly clears up all doubt.

Occasionally these cases of scurvy, in which periosteal affection is marked, are mistaken for syphilitic disease of the ends of the long bones. In this there is periostitis, with pain on movement. But the dread of movement is less marked, the joints are usually more implicated, while the history of scurvy diet, the presence of special signs of scurvy, the absence of syphilitic history and signs, serve, together with the diet test, to distinguish between the two conditions.

Sometimes the extreme immobility of limbs, due not to paresis but to extreme dread of the pain of movement, may be mistaken for the early stage of infantile palsy (anterior poliomyelitis) in which there is often considerable hyperæsthesia. Scurvy may be distinguished by the more intense tenderness, the greater intolerance of movement, the absence of the extreme flaccidity of muscle seen in infantile palsy, in which the affected limbs are limp and fall loosely down and the toes droop. The existence of periosteal swell-

ing and other signs of scurvy, and later the persistence of faradic contractility and tendon reflex which disappear in anterior poliomyelitis.

In another group of cases when hæmaturia or albuminuria is the first symptom which attracts notice, the condition may be referred to Bright's disease, or some other affection of the kidney. From this it is to be distinguished by the microscopic evidence afforded by the urine, and by the absence of other signs of kidney implication, by the presence of other symptoms of scurvy, by the diet history and the diet test.

When the periosteal swelling is great, accompanied by a rise of temperature, usually present at first when hæmorrhage is extensive, the condition is not infrequently mistaken for deep-seated abscess, and in many instances incisions have been made in order to let out suspected pus; with the result of finding blood clot only.

Other conditions for which infantile scurvy has been in my experience mistaken, are an extreme form of stomatitis, when the affection of the gums has been great; for pneumonia, when there has been hæmorrhage into the lung; for meningitis, when there has been proptosis and squint from post-orbital hæmorrhage, and for tubercular disease of the knee or hip-joint when the long bones in the immediate neighbourhood of these joints have been involved. Mild cases are usually mistaken for rickets or for simple anæmia and debility. With careful examination and search for the

characteristic signs of scurvy, with the aid of the diagnostic points described, and, above all, by the diet history and diet test, the true nature of these cases can generally be made out.

In all cases of rickets with limb tenderness, in which there is probably a scorbutic complication, the questions of diet should be carefully investigated.

I may now relate one or two cases in illustration of some of the points I have laid before you.

Case I.—The most severe and typical case of infantile scurvy I have met with was the one which first disclosed to me the true nature of the affection.

The patient was a child sixteen months old, whom I was asked to see in consultation with the late Mr. Sumner, of St. John's Wood, in 1877, on account of what was presumed to be a singular form of stomatitis supervening on rickets. It presented a very striking appearance. Large purple masses, having all the appearance of raw flesh, protruded between the lips; these on examination proved to be the gums swollen to this degree, livid, spongy, and bleeding. The spongy protrusions had, I learnt, been greater still, having been partially excised because they were thought to interfere with feeding. The mucous lining of the mouth, especially over the palate, was similarly purple and spongy, and oozing blood, and so swollen as to be almost in contact with the tongue. The breath was horribly offensive with the odour of carrion.

The complexion was pallid and earthy, the skin was harsh and dry, and there were purpuric blotches

on the lower limbs, which were extremely tender; the child screamed out on touch or movement of them. The ankles and feet were œdematous, but no muscular or periosteal swellings were observed. The child was so feeble that it could not sit up, and fell over on being placed upright.

The skull, wrists, and chest presented all the characters of well-marked rickets.

I had never seen a like case before; the condition of the gums and the horrible fœtor of breath were suggestive of scurvy, but I knew of no such affection having ever been observed as occurring in infants, except in conjunction with the same affection in adults as part of an epidemic of so-called sea scurvy.

An inquiry into the infant's diet, however, strongly supported the view that the condition was scorbutic.

The child had been suckled until six months old, being given in addition oatmeal and water, and at one time condensed milk. At the end of the six months the infant was weaned, and, incredible as it may seem, was fed solely on oatmeal, rusks, and water; milk was stopped altogether because it was found to disagree. At ten months some mutton broth was given in addition to the farinaceous food and water. This diet had been continued without any change or addition up to the time of my visit, so that for ten months the infant had no milk or potato, or fresh food of any kind, except a small quantity of broth! With this exception it had been fed upon farinaceous food alone for this long period. The diet, therefore, contained no anti-

scorbutic element, and being destitute of animal fat and deficient in proteid, was a rickety diet as well as a scurvy diet.

I proceeded to test the matter further by treatment. So far this had been addressed solely to the state of the mouth and the rickety condition—chlorate of potash, bark, iron, cod liver oil, with astringents to the gums locally. Anti-scorbutics, such as fresh milk, potato gruel, and raw meat were substituted for the rusks and oatmeal.

As medicines, steel wine and bromide of potassium were given for a short time to relieve the dangerous attacks of laryngismus from which the child suffered as a result of the rickety state. Immediate improvement followed, all the fungous swelling of the gums disappeared in the course of a week; recovery was uninterrupted, so that the child was practically well in the course of a few weeks, and running about at the end of three months. The test by treatment was decisive as to the nature of the ailment.

Case II.—The next case possesses unusual interest, and illustrates another feature of the affection.

The patient was a child twelve months old, sent to me from the country on account of an obstinate and unexplained hæmaturia. The child was extremely pallid and cachectic in appearance, and lay limp and motionless in its mother's arms. It cried on being moved or touched, the limbs were obviously extremely tender, and bruises appeared when it was handled. It was feeble, drowsy, and listless, and became panting

and breathless upon the least exertion or excitement; sometimes so faint that it was feared that it would die at the moment. About a month previous to my seeing it the urine became very dark, and the medical man in attendance found that it contained blood; on several occasions blood was passed from the bowels. On examining the urine I found it loaded with blood and albumen. The child had a slight degree of rickets only, evidenced by an unduly open fontanelle, some beading of the ribs, and enlargement of the wrists. On examining the legs they were found to be extremely tender; there were distinct periosteal swellings on the front of each tibia, the feet were œdematous, and there was a large purpuric blotch of ecchymosis under the skin in the right iliac region. Lastly, the gums round the incisor teeth were swollen and purple.

The history was this: after three months' feeding on milk and water the child had an attack of vomiting and diarrhœa: fresh milk was stopped, and a malted farinaceous food, with a small quantity of condensed milk, substituted for it.

The inference drawn from the physical state of the child and its diet record, that the mysterious and unexplained hæmaturia was due to the scorbutic state, was confirmed by the crucial test of anti-scorbutic treatment. No drugs of any kind were given, but potato gruel, raw meat juice, and cream. Some difficulties arose with regard to the feeding in this case. The stomach proved sensitive, and the new food was rejected. However, by reducing the quantity tolera-

tion was obtained, and then the amount was gradually increased without further drawback. In a fortnight all trace of blood and albumen had entirely ceased, and the tender swelling of the limbs and sponginess of gums had disappeared. In less than a month the child was well.

Case III.—The next case serves as an illustration of the errors in diagnosis which result from the pain, and tenderness, and immobility of the limbs, especially the legs.

In this instance the patient, a child of eight months, had been sent up from the country to Mr. Howard Marsh, as a surgical case of obscure hip-joint disease. Mr. Marsh, however, who had previous experience of similar conditions at the Children's Hospital, Great Ormond Street, on examining the child, at once came to the conclusion that it was suffering from infantile scurvy, and asked me to see it with him.

The child was lying in that state of absolute motionlessness of the lower limbs which is so characteristic.

Both knees, or rather the bones above and below the joint, were exquisitely tender, and the left swollen; the tibia above the right ankle was tender and periosteum swollen; no muscular extravasations could be detected.

The least attempt to handle the limbs, or move the child, or even to approach it, caused it to scream with fright and dread of pain. The little patient was indeed constantly crying from this pain of movement, for it was carried about and dandled with the idea of soothing

it to sleep. There were no purpuric extravasations or bruises, and the urine was free from blood and albumen.

The complexion was pallid and earthy, and the child's muscles flabby and feeble. The gums were purple and swollen round the base of the teeth, a symptom quite pathognomonic when present; and another significant fact was, that on one occasion the child had brought up blood on coughing, although nothing abnormal could be detected in the lungs. There was no sign of rickets beyond some beading of the ribs. A patent infant food of malted flour formed the staple of the child's diet. Milk had disagreed, and only half a pint was allowed—nothing else was given. A diet of dry farinaceous food with such a small allowance of milk, which is very weak in anti-scorbutic power, and no other fresh element is a scurvy diet. The test of full anti-scorbutic diet at once clenched the diagnosis. On potato cream, raw meat juice, and a slowly increased amount of fresh milk, the child made a rapid recovery in the course of three weeks.

Case IV.—I give this case as an illustration of the disease in one of its milder forms. The difficulty of diagnosis is in the first instance in such conditions. The patient was a child nine months old, which had come under the care of Dr. Liveing for eczema. Dr. Liveing referred it to me on account of pain and tenderness of the lower limbs concurrent with rickets. The signs of rickets were distinct but not extreme—a large fontanelle, absence of teeth, beading of the ribs.

The child was plump, of good fresh colour, being

only slightly anæmic. It lay quite motionless, its legs drawn up, the least attempt to straighten them, or offer to touch them even, causing it to cry out. The periosteum of each tibia was obviously swollen, and extremely tender. There was a faint bruise on the right cheek near the ear. The gums appeared to be slightly swollen, and somewhat darker in colour than normal, but could not be positively identified as spongy. There was no albumen in the urine. The child had been fed entirely upon a preparation of farinaceous food with desiccated milk without the addition of any fresh element. It seemed *primâ facie* probable that the case was one of scurvy, yet the child was so unusually well-nourished and healthy in look, that in the absence of any decided affection of the gums I had some doubt whether the condition was really scorbutic. To test the matter, anti-scorbutics were added to the previous dried food, viz., potato, fresh milk, raw meat juice. Before, however, this had time to take effect, a symptom developed which was really decisive. A violent fit of crying produced a copious extravasation beneath each lower eyelid, so that the child appeared with two black eyes, and this evidence of the nature of the ailment was finally confirmed by a rapid and complete recovery on the anti-scorbutic diet. In three weeks the child was kicking its legs about quite well.

Case V.—I give this next case as one which exhibits, in addition to the ordinary signs, some of the less common symptoms of the disease, and discloses a new source of error in diagnosis.

In July 1895 I saw, in consultation with Dr. Hannan, of Camden Town, a child nine months old, suffering from what appeared to be pneumonia, accompanied by some strange and unusual features. I learnt that the chief symptoms were pyrexia, a small patch of pneumonic consolidation in one lung, great tenderness of the legs, and within the last few days the supervention of cerebral symptoms in the form of proptosis and strabismus of the right eye.

At my visit I found a large fat anæmic child lying on its mother's lap with motionless limbs, the right upper eyelid swollen and œdematous, and discoloured by ecchymosis drooping over the eyeball, which was projected forwards, and turned outward in external squint. The left eye was normal. Both legs were acutely tender and swollen. The swelling involved the periosteum just above the ankles, which, together with the dorsum of the foot, were œdematous, but the arms were unaffected.

My suspicions were aroused by the condition of the limbs and the state of the eyes, and on looking into the mouth, the gums round the two upper incisors, the only teeth through, were seen to be purple, swollen, and spongy. The appearances presented are shown in a coloured drawing taken at the time.[1]

On examining the chest, a small patch of faint bronchial breathing could be detected at the base of the right lung. This Dr. Hannan told me had been at one time much more pronounced. The temperature was

[1] This is reproduced in the article on Infantile Scurvy in vol. v. of Professor Allbutt's *System of Medicine*.

101 degrees. The child showed but slight evidence of rickets. The forehead was somewhat prominent and the wrists large, the rib beads distinct; teething was delayed. But the fontanelle was only the size of a sixpence, and its margins not thickened, the chest normal in shape, and there were no head sweats. The child had been brought up entirely on condensed milk with malted food, without change or addition—a highly scorbutic diet.

The case was clearly one of scurvy, with hæmorrhage under the periosteum of the tibia, into the back of the orbit, into the upper eyelid, and no doubt a small hæmorrhage into the lung giving rise to the physical signs found there.

Potato gruel and raw meat juice were ordered, and when I saw the patient again a fortnight later, the gums were only slightly red, the proptosis and swelling of the eyelid and œdema of the limbs had disappeared; the periosteal swellings and tenderness had gone, with the exception of a small one on the left tibia.

It appeared that progress had been somewhat delayed by the inability to take potato, which caused sickness. During the first week a fresh periosteal swelling had appeared, accompanied by temporary pyrexia. Vegetable soup and grape juice were substituted for the potato, and the child was quickly well.

Case VI.—This case is instructive as an example of scurvy in its mild form when many typical symptoms are wanting. The patient was a child of eight months, whom I saw in consultation, on October 10, 1899.

The symptoms were extreme tenderness of the lower limbs, with some swelling about the ankles, accompanied by a rise of temperature 99° to 99·5°. There was great dread of movement, the child lay with its legs perfectly still, and screamed if they were moved or touched.

No distinct periosteal swelling could be made out, but the part affected appeared to be above the joints at the lower part of the tibia, rather than in the ankle itself. The child showed no obvious marks of rickets; it had four teeth, and the gums appeared to be perfectly healthy. There had been no hæmorrhages of any kind. The urine was free from albumen.

As in the majority of these cases, the condition had in the first instance been considered to be rheumatic; salicylate of soda had been given for five days with the effect of lowering the temperature somewhat, but without affecting in any way the swelling and tenderness of limb. The question of scurvy had been duly considered, but in the absence of any affection of the gums, or other hæmorrhagic sign, it was regarded as improbable, and discarded.

Against the diagnosis of rheumatism there were the facts that this affection is almost unknown at such an early age, that salicylate treatment was ineffectual, and that the limb swelling seemed to be of the end of the bones rather than in the joints.

In favour of the diagnosis of scurvy was the latter fact, the extreme degree of tenderness and dread of movement, and, above all, that the child had been fed throughout upon a diet which is scorbutic if too

long continued without additions of fresh material, viz., a predigested food for the first four months, 'humanised' milk for a later period since. So although there was no affection of the gums, we inclined to regard the condition as scurvy.

To test the diagnosis we gave the child no medicine but potato gruel, and raw meat juice. I learnt from the medical attendant nine days later that our conclusion had proved to be correct, the child was getting rapidly well.

Case VII.—The last case which I have to bring before you is one of the disease in its early stage, incipient scurvy, in which the symptoms are present only imperfectly and in slight degree.

The patient, a boy eleven months old, was brought to me from the country on September 17, 1894, on account of three symptoms, viz. extreme muscular debility, great pallor, and slight diarrhœa. The history was that he was suckled for the first two months and did well; at that period he was weaned, and placed upon milk and barley water and a malted farinaceous food. In spite of variations in the strength of the milk and water, and the kind of malted food given with it, the child suffered from colic, flatulence, and diarrhœa, and did not thrive. Two months before its visit to me, the mother and nurse came to the conclusion that the child could not digest milk in any form, and, without consulting the doctor, stopped the milk entirely, and gave only malted food made with water, and a small quantity of Valentine's meat juice; so that for

the last two months he had no fresh food of any kind—a diet absolutely scorbutic.

The child was obviously extremely anæmic, with a sallow earthy pallor, and great muscular flabbiness and debility. There was, however, no tenderness of limb, no periosteal or muscular swelling to be found, no hæmorrhage of any kind, no albumen in the urine.

On examining the gums, however, they were seen to be somewhat darker in colour round the roots of the incisors, and distinctly swollen. There could be little doubt that the condition was one of commencing scurvy. A diet of asses' milk and raw meat juice produced immediate and steady, although not rapid, improvement. In view of the disturbed digestion, and the fact that scorbutic symptoms were not urgent, no potato or other powerful vegetable anti-scorbutic was given, hence the comparative slowness of recovery.

Prognosis.—The prognosis in infantile scurvy is almost uniformly favourable. It is an eminently curable disease.

If it is recognised before extreme symptoms have set in, recovery is rapid and certain.

Before the nature of the affection was recognised, however, the rate of mortality ran high—upwards of 20 per cent. Since then the death rate has fallen remarkably. Out of the sixty-one cases previously mentioned, two only have proved fatal. Both were in hospital patients. In one instance the child had fallen into such a condition of extreme debility, that it took food with difficulty, so that curative treatment was

necessarily slow, and it died from hæmorrhage into the lung three days after admission.

In the second case there was extreme swelling of the femur, which was treated surgically for abscess, and the scorbutic condition was not recognised, or treated.

Danger to life, however, does exist until the scurvy is relieved, and from two sources, viz. from hæmorrhage into some vital organ, such as brain or lungs, and from syncope and exhaustion. In no instance, however, it will be seen, within my experience, has a fatal case occurred after the patient has been for a few days under anti-scorbutic treatment.

Prevention.—The obvious means of preventing infantile scurvy lies in the avoidance of the patent prepared dry foods made without the addition of fresh milk; and also the prolonged use of those in which, although consisting wholly or partially of fresh milk, it has been altered by the pancreatising, or peptonising process, or by sterilising at high temperature, or the system adopted in the preparation of so-called humanised milk. These adaptations are serviceable for a time, but are not suited for permanent diet without the addition of some fresh element.

In some cases, in which the child exhibits an intolerance of cows' milk, even if boiled and diluted, it may, in the absence of wet nurse or asses' milk, be necessary to give a food rendered more bland and digestible by one of the processes named. If so, it is essential that such altered milk should not be given

continuously for more than a few weeks, without the addition of some fresh living material. These prepared milks may usually be gradually replaced by fresh cows' milk through the device of admixture in slowly increasing proportion. If this cannot be borne without digestive disturbance, the want of anti-scorbutic property must be supplied by the addition of a little cream with raw meat juice; or meat tea, in which fresh vegetables, such as carrots and potatoes, have been boiled and strained out.

Treatment.—The treatment of infantile scurvy when it has actually developed is almost entirely dietetic. No drugs of any kind are required, merely the administration of fresh foods which possess the anti-scorbutic property in high degree. The child should, in the first place, be given fresh milk if it can digest it. This may be raised to the boiling point, but not sterilised by long heating, not humanised, nor peptonised. Fresh milk, however, as pointed out previously, possesses very moderate anti-scorbutic power, and is insufficient alone to effect the rapid cure of scurvy, just as it is oftentimes unequal to prevent it if given in small quantity.

For really effective anti-scorbutics we must resort to vegetable juices. Fresh green vegetables themselves, such as the cruciferæ, so efficacious in the scurvy of adults, are not available in the case of young bottle-fed infants. A most efficient substitute is, however, to be found in the potato, which Dr. Baly proved to possess such remarkable virtue in the epidemic of scurvy at the Milbank Penitentiary more than fifty years ago. Even

young children can usually take potato without digestive disturbance when it is properly prepared. The most satisfactory method is to reduce a well-steamed floury potato to finest powder by rubbing it through a fine sieve. The powder should then be well beaten up with a little boiled milk until it is of the consistence of thin cream. This should be added to the food the child is taking—in very small quantities at first—half a teaspoonful to each bottle. If this agrees, the amount may be gradually increased to a dessert-spoonful, or even a tablespoonful if the child is a good age. Another effective plan, suited to more delicate digestive power, although less rapid in its action, is to administer the vegetable juices through the medium of beef tea or chicken tea, in which potatoes and carrots have been boiled and strained out. A small cup of this may be given once or twice a day.

The fresh element in food should be further strengthened by the addition of raw meat juice. This possesses anti-scorbutic power, but, like milk, in minor degree only; for experience shows that in any feasible quantity it is unequal alone to effect the rapid cure of scurvy, or to prevent its development when a small amount of it forms the sole addition to an otherwise scurvy diet. The comparative feebleness of raw meat juice and of milk in anti-scorbutic power is not generally known, and a too high estimate of their value in this respect has, in some cases, led to erroneous conclusions as to the nature of the disease, when it has arisen where milk or raw meat juice has formed a

small portion of the patient's diet. As with fresh milk, as before stated, so with raw meat juice, a considerable quantity is required, if it is the sole anti-scorbutic given, to either ward off or cure scurvy.

The special value of raw meat juice in these cases does not lie in its anti-scorbutic property, but in its great hæmic virtue. It contains iron in the most assimilable form in its hæmoglobin, and it is the most powerful of all remedies for the restoration of the blood in anæmia, which is so prominent a symptom in scurvy. Grape juice, orange juice, lemon juice, baked apples, are all useful anti-scorbutics, especially in the case of older children.

When potato and raw meat juice are well borne and freely taken, the result is immediate and almost magical. If the gums are spongy and swollen all sign of it frequently disappears in a few days; the swelling of the limbs goes down, the extreme tenderness subsides, so that in the course of a week or ten days the child no longer screams on the approach of any one near it, and indeed no longer dreads movement or handling. In the course of from a fortnight to three weeks, if the feeding progresses without interruption, the patient is well.

In addition to anti-scorbutic diet, which is the chief agent of cure, and so rapidly effective alone, fresh air and sunlight, as in the case of adult scurvy, are useful aids in relieving the anæmia and restoring vitality.

With regard to local treatment little is required.

The child should be kept absolutely at rest, lying on a soft pillow, its tender limbs swathed in cotton wool, and moved as little as possible. In extreme cases, when the gums ooze with blood, and there is the fœtid carrion-like odour of breath, a mouth wash of weak carbolic solution (1 in 80) or of boracic acid or permanganate of potash, is useful as a disinfectant and deodoriser. Local astringents are hardly required, diet is really all-sufficient for the cure of the spongy gum.

Let me again affirm that the only internal remedies required are anti-scorbutic foods. Drugs give little or no help, and many constantly given are positively mischievous: all depressant remedies, for example, are harmful and to be sedulously avoided. Iodide of potassium, for instance, is frequently prescribed with the fallacious idea of causing absorption of material effused under the periosteum. I doubt whether it has any such power; if it has, its aid is unnecessary; for the effused blood disappears with marvellous rapidity without it, and iodide is depressant and therefore objectionable. Iodide of iron, again, still more frequently given, is not much better. Both these iodides, indeed, if pushed far enough, produce, in the case of children, a condition of blood and vessels in some respects akin to that found in scurvy, viz. iodic purpura.

Cod liver oil and steel wine are useful in the later stages for the relief of any underlying rickets. But in the active stage of scurvy they are better omitted, since they have no anti-scorbutic virtue, and are liable

to cloy appetite and interfere with the ingestion of essential food.

In these cases raw meat juice is better than any pharmacopœial preparation of iron, and fresh cream than cod liver oil.

Appended is a list of sixty-one cases under my own observation in which the diet was ascertained with precision.

APPENDIX

Table of Sixty-one Cases of Infantile Scurvy showing the Age at which the Disease appeared, the Exact Diet of Artificial Food on which the Scorbutic State developed in each instance, and the Results of Treatment by Anti-scorbutic Diet.

Name	Age	Date and Reference	Food	Result
A. B.	16 months	1877	Oatmeal and water only	Recovery.
W. Hospital out patient	3 years	1877	Suckled 1 year, bread and butter, beef tea. Chief diet, bread and tea : brandy and water occasionally : German sausage, but no other meat or vegetable	Recovered after 5 weeks.
S. Hospital out patient	16 months	1877	Cow's milk and patent infant's food of slightly malted flour, equal parts. Last 4 months bread and milk. (This latter statement not reliable)	Rapidly well.
S. Hospital out patient	1 year 11 months	1879	Bread and milk once daily, beef tea, broth, gravy, bread. On Sunday only a little meat and potatoes	Convalescent after 5 weeks.
G.	16 months	1881 Nov. vol. 7, p. 210	Patent infant's food of malted flour and desiccated milk-powder, arrowroot, isinglass	Almost well in 27 days.
A. Hospital out patient	2 years 9 months	1881 Nov.	Bread and butter, tea, very little milk	Convalescent in 3 weeks.
L. Hospital out patient	1 year 2 months	1888 Jan.	Bread sop chiefly. (Bread and milk made her sick)	Fatal from hæmorrhage into lung.
H. Hospital out patient	1 year 1 month	1888 April	Suckled 6 weeks. Patent infant's food of slightly malted flour, and condensed milk to 11 months. Pint and a half cow's milk, and oatmeal for last 2 months. (This latter statement not reliable)	Quite well at the end of 3 weeks.
S.	1 year	1888 vol. 13, p. 17	Patent infant's food of malted flour, condensed milk, 1 teaspoonful to 3 bottles	Well in 3 weeks.
G.	8 months	1888 vol. 13, p. 65	Peptonised condensed milk and patent meat essence for 6½ months	Recovery, duration not known.

Name	Age	Date and Reference	Food	Result
P.	2 years	April 1888 vol. 14	Patent farinaceous infant's food and desiccated milk entirely	Recovery.
H.	1 year 10 months	June 1889 vol. 14, p. 305	Condensed milk and patent slightly malted food 11 months	12 days later recovering rapidly.
W.	8 months	Nov. 1889 vol. 14, p. 524	Patent food of malted flour for a fortnight, then milk added by teaspoonful reaching ⅓rd milk in 7 months	Recovered.
H.	1 year 2 months	Nov. 1890 vol. 15, p. 370	Nursed 3 months, then a patent infant's and invalid's food made from flour and milk predigested by pancreatisation for 5 months, then 'all kinds of food,' milk, cream, peptonoids, Revalenta, Swiss milk, since September only Revalenta and beef tea, no milk	Nov. 21 rapidly recovering.
S.	11 months	July 1891 vol. 16, p. 296	Swiss milk, cow's milk for a short time, then a patent food of malted flour and half a pint of milk	Recovery.
P.	7 months	Nov. 1891 vol. 16, p. 422	A patent food of flour and milk predigested by pancreatisation	Recovery.
H.	1 year 2 months	Feb. 1892 vol. 17, p. 88	Humanised milk, patent food of highly malted flour alone. Beef tea, egg, frame food for a short time lately	Quite recovered in 3 weeks.
S.	8 months	June 1892 vol. 17, p. 277	Condensed milk, patent food of flour and milk predigested by pancreatisation the last 3 months	In 8 days' time recovered rapidly.
L.	11 months	Feb. 1893 vol. 18, p. 55	Cow's milk tried at first, disagreed, patent food of highly malted flour and whey for 14 days, then condensed peptonised milk 1 month, then patent food of malted flour and desiccated milk-powder, oatmeal, corn flour, nursery biscuits, with humanised milk 1½ pint	Recovered.
S.	9 months	May 1893 vol. 16, p. 215	Humanised milk for 8 months	A fortnight later was getting rapidly well.
H.	2 years	July 1893 vol. 18, p. 263	Patent food of flour and milk predigested by pancreatisation, oatmeal with butter and sugar, humanised milk 6 months	Quite recovered by the end of a month.
W.	1 year	Aug. 1893 vol. 19, p. 39	Patent food of slightly malted flour, less than a pint of milk in 24 hours	Recovered.
B.	7 months	Oct. 1893 vol. 19, p. 67	Patent food of flour and milk predigested by pancreatisation. Patent meat juice and raw meat juice the last 2 months	Almost well in 10 days.

Name	Age	Date and Reference	Food	Result
S.	6 months	Jan. 1894 vol. 19, p. 208	Patent food of malted flour and desiccated milk-powder, milk four parts to three of water	Recovered.
F.	3 years	Jan. 1894 vol. 19, 232	A patent food of malted flour, nursery biscuits, with half milk and half water, humanised milk	Recovered, duration not known.
P.	9 months	Jan. 1895 vol. 21, p. 1	Peptonised milk the first 3 months, last 6 months patent food of highly malted flour, cow's milk one part to three of water, less than a pint in 24 hours	Recovery perfect at the end of 3 weeks.
Dr. S.'s case	10 months	Jan. 1895 vol. 21, p. 14	First 6 weeks cow's milk and water, then for 2 months humanised milk, then for 1 month 'kindermilch,' then for 4 months a patent food of malted flour and milk, next peptonised milk, then 'kindermilch,' small quantity of raw meat juice and bread jelly	Recovered.
T.	7 months	Feb. 1895 vol. 21, p. 54	Humanised milk and sterilised milk	Recovered.
Case from Bournemouth	—	—	Particulars missing	Recovered.
B.	10 months	Feb. 1892 Consultation book, p. 77	'Kindermilch' the first 8 months, last 2 months patent food of malted flour, 2 months milk and water equal parts	Heard child quickly recovered.
P. (twins)	7 months	Consultation book, p. 80 March 9, 1892	Patent food of flour and milk predigested by pancreatisation 2 months	Recovered at once.
M.	7 months	Consultation book, p. 142 Nov. 1892	Patent food of flour and milk predigested by pancreatisation	Recovered.
G.	8 months	Consultation book, p. 212 Feb. 1893	Patent food of malted flour, half a pint of milk only	Rapidly recovered.
T.	10 months	Consultation book, p. 346 Dec. 1893	Cream of malt 3 months, patent food of flour and milk predigested by pancreatisation 7 months	Rapidly recovered.
B.	10 months	Consultation book, p. 388 March 1894	First 7 months humanised milk, since then sterilised milk	Recovered.
Br.	10 months	Consultation book, p. 452 July 1894	Condensed milk and a patent food of highly malted flour	Recovered.
D.	9 months	Consultation book, p. 664 July 1895	Patent food of malted flour and desiccated milk-powder, and another of flour and milk predigested by pancreatisation	Recovered.
G.	8 months	Consultation book, p. 621 July 1895	Patent food of flour and milk predigested by pancreatisation, humanised milk	Recovery.
P.	11 months	Sept. 1895 vol. 22, p. 9	Milk and water in equal parts, patent food of highly malted	Rapidly recovered.

Name	Age	Date and Reference	Food	Result
			flour and another of malted flour. Last two months the former food with water and a little patent meat juice only	
S.	9 months	Dec. 1895 vol. 22, p. 193	Patent food of highly malted flour and fresh milk, said to have a pint and a half daily, milk observed to deposit sediment	Recovery.
G.	10 months	Dec. 24, 1895 vol. 22, p. 207	The first 2 months by hand, cow's milk and water entirely, then for 3 months peptonised condensed milk only, then milk and water in proportions of 2–3 with patent food of highly malted flour and another of malted flour, with less than one pint of milk in the 24 hours for 2 months. For the last 3 months peptonised condensed milk with an occasional crust buttered over with cod liver oil and maltine	Recovery complete in 1 month.
H.	7½ months	Nov. 17, 1896 vol. 22, p. 246	By hand. Condensed milk 1 month, then a patent food of malted flour and cow's milk, less than a pint of the latter in 24 hours	Recovery. Dec. 21 quite well.
B.	8 months	Feb. 8, 1897 vol. 24, p. 77	At first by hand upon a patent food of highly malted flour with cow's milk, this caused diarrhœa. Then upon a patent food of malted flour and milk, 2 pints of scalded nursery milk	Recovery in 1 week.
S.	8 months	June 16, 1897 vol. 24, p. 245	Humanised milk until the last fortnight, then a patent food of milk and flour predigested by pancreatisation with condensed milk	Recovery in 14 days.
T.	8 months	Jan. 8, 1898 vol. 25, p. 254	Brought up on humanised milk for 3 months, then a patent food of malted flour and desiccated milk-powder, never fresh milk or food	Quite well on potato and raw meat juice.
L.	9 months	Oct. 14, 1898 vol. 26, p. 216	Cow's milk and water and a patent food of malted flour, at first ⅓rd milk and ⅔rd water, then the patent food alone and then with milk (nearly 2 pints). The milk had been sterilised for the last 3 weeks	Recovery in 14 days.
W.	17 months	Jan. 17, 1899 vol. 27, p. 51	Beef tea 6 weeks, a patent food of flour and desiccated milk-powder with beef tea for another 6 weeks (no milk)	Rapid recovery.
H.	8 months	May 5, 1899 vol. 26, p. 244	A patent food of malted milk for 6 months, then unsweetened condensed milk	Recovery.

Name	Age	Date and Reference	Food	Result
M.	16 months	May 25, 1899 vol. 27, p. 274	Nursed 5 weeks, then sterilised milk with equal part of water. Frame food and a patent food of slightly malted flour ever since	Recovered in 3 weeks.
C.	9 months	Feb. 8, 1897 Consultation book, p. 200	For the first 3 weeks a patent condensed milk, for the last 6 months humanised milk entirely, the last fortnight humanised milk and a cup of chicken tea	Recovery in 3 weeks.
S.	14 months	March 1, 1897 Consultation book, p. 216	For the first 6 months humanised milk, for the last 8 months sterilised milk with a patent food of flour and milk predigested by pancreatisation. The last month beef tea was added	Complete recovery in a month.
W.	5 months	May 1, 1897 Consultation book, 96-98, p. 228	Fed since birth on sterilised milk	Rapid recovery.
M.	9 months	June 8, 1897 Consultation book, p. 251	Bread jelly and peptonised condensed milk alone for 4 months	Rapid recovery.
Dr. W.'s case.	12 months	July 16, 1897	Patent pancreatised food only. No fresh food until last few days	Rapid recovery.
C.	10 months	Nov. 23, 1897 Consultation book, p. 311	Breast fed 1 week, humanised milk 7 weeks, sterilised milk 32 weeks	Recovery in 10 days.
W.	12 months	Dec. 2, 1897 Consultation book, p. 318	After 1 month fed upon humanised milk, latterly a patent food of highly malted flour and a patent meat juice	Rapid recovery.
B.	9 months	May 11, 1898 Consultation book, p. 393	For 6 weeks breast fed, milk and barley water until 2 months, then a patent food of flour and milk predigested by pancreatisation until 8 months, for the last fortnight a patent food of malted flour	Recovery.
W.	13 months	Sept. 21, 1898 Consultation book, p. 443	Wet nurse for 4 months, then humanised milk only	Recovery.
W.	6¾ months	Dec. 20, 1898 Consultation book. p. 477	A patent food of malted flour and desiccated milk-powder for 6 months	Recovery in a fortnight.
T.	14 months	April 13, 1899 Consultation book, p. 51	Breast fed for 6 months, then humanised milk to 10 months with a little cream, then sterilised milk and 2 tablespoonfuls of cream	Rapidly well.

[The references in this list are to the private Case-books.]

A note on Scurvy

Professor Wright. Lancet aug. 25th 1900.

He considers that the condition is one that supervenes upon the ingestion of a considerable excess of mineral acids over bases, and that it is an acid intoxication.

He prescribes for it the salts of oxidizable organic acids E.S. for an adult. 3ss doses of bicarb of Soda. bicarb or Sodium acetate. or. 3i doses of Sodium lactate.

Jackson & Vaughan Harley. Lancet. Aug. 28. 1900.

hold that it is due to tainted food. and is essentially ptomaine forming.

THE RHEUMATISM OF CHILDHOOD

These Lectures in their original form were delivered before the Harveian Society in 1888, and published subsequently in the 'Lancet.' They have been thoroughly revised for publication in the present volume with the kind assistance of Dr. Poynton, who has himself done good work in connection with the subject. The alterations consist chiefly in the addition of new matter which has accrued during the ten years which have elapsed since their first publication, so as to bring the subject fully up to the level of present knowledge.

19 Portman Street, W.
1899.

THE RHEUMATISM OF CHILDHOOD

LECTURE I

Rheumatism not to be regarded as a mere affection of the joints—Arthritis only one of many rheumatic phenomena - Various phases of the rheumatic state – General characteristics of the rheumatism of childhood as compared with that of adults – Comparative insignificance of the joint affection—Greater prominence and frequency of other phases – Tendency of the various phenomena of the rheumatic series to appear independently—They may occur singly, or in various combinations, in varying order of sequence, at different intervals of time—Examples—Influence of sex—Influence of inherited tendency—The rheumatic phenomena considered in detail—Arthritis—Special characters in childhood—Variation in the chief symptoms from the adult type.

THERE is perhaps no serious disease more familiar to us than acute articular rheumatism; it is one of the disorders most commonly seen in the wards of a general hospital; it is constantly encountered in private practice; and I must confess that when I chose Rheumatism as the subject of the Lectures which you have done me the honour to ask me to deliver, I was almost afraid that it might be deemed too trite and commonplace, destitute of sufficient novelty and interest. But there is a certain advantage in treating of a matter with which the audience are familiar, and I trust I shall be able to present it to you

in some new aspects, and to attract attention to certain points of great interest, hitherto, I think, insufficiently considered; and thus I hope that my choice may be justified, and any unfavourable criticism in this respect eventually disarmed.

I must premise, at the outset, that in these Lectures I shall use the terms 'rheumatism' and 'rheumatic' in the strictest sense, as applying only to that form which is distinguished as acute or genuine rheumatism, of which what is known as rheumatic fever is the most extreme expression in adults, but which appears with every degree of acuteness and severity, and also, as I hope to show, in many different phases.

Acute articular rheumatism is not only an extremely common disease, but it has very striking and obvious symptoms by which it is readily recognised; the swollen, tender, painful joints, the fever, the profuse sour-smelling perspirations, render a typical case unmistakable and distinct; yet this is a picture of the disease very rarely seen in childhood. Even in its milder forms, genuine articular rheumatism preserves its special features; some may be absent, others modified; yet the stiffness, tenderness, and swelling of joints usually indicate the nature of the affection. But this, again, does not represent rheumatism completely as seen in childhood. We are so accustomed to associate the term 'rheumatism' with this condition of the joints, to regard arthritis as the chief and essential feature, and any associated affections of other parts as mere complications, that it is difficult at first to realise that articular inflammation is only

one of many direct and sometimes independent manifestations of the rheumatic state. This is, however, the one central idea which I wish to enforce. The ordinary conception of acute rheumatism, as essentially characterised by articular inflammation, is based upon observation of the disease as we see it in adults; in them the arthritis is one of the most constant, prominent, and characteristic of the morbid phenomena which arise. The names rheumarthritis, polyarthritis, polyarthritis synovialis, and polyarthritis rheumatica acuta, reflect this narrow view.

A study of the disease in children, however, leads to a far wider conception. In the rheumatism of early life other morbid conditions appear prominently and constantly, which may claim, equally with the arthritis, to be regarded as direct results of rheumatic activity; in childhood arthritis cannot be regarded as alone typical, essential, and representative.

The most complete and comprehensive manifestation of the various phases of rheumatism belongs, indeed, to the period of childhood; it appears then, under the simplest conditions; this presentation of the disease should be regarded as representative, and the changes which take place in the phenomena with advancing age regarded as modifications of the earlier and more perfect form. I will ask you, gentlemen, to put aside, at any rate for the moment, this limited and narrow view of rheumatism as inseparably associated with arthritis, a view based upon observation of the disease in grown persons, and to look at it from a new standpoint.

There are certain affections which have been observed to be so frequently associated with acute rheumatism that the existence of some pathological connection between them has come to be very generally accepted, although the extent and intimacy of the association may be a matter of controversy. Endocarditis and pericarditis, for example, are so constantly seen as immediate accompaniments of articular rheumatism, even in adults, that there is no question of their relation to it; and in the case of children the connection is still more frequent and more intimate. Pleurisy, again, and tonsillitis are allowed to be not unfrequently accompaniments of articular rheumatism in both periods of life. But with these I think the list ends as far as adults are concerned. If we turn to children, however, we learn that the scope of acute rheumatism is wider still. Other manifestations appear in undoubted association with it in early life, and must be included in the series of rheumatic phases, although some decline or disappear in later life. Subcutaneous tendinous nodules, chorea, and exudative erythema are the most common developments of rheumatism. There are other affections which have been deemed by some to be minor expressions of the rheumatic diathesis. I have not satisfied myself upon this point, and I wish to limit the list to those generally accepted as having some connection with it. The claims of endocarditis, of pericarditis, of pleurisy, of tonsillitis, of exudative erythema, of chorea, of subcutaneous nodules, will hardly, I think, be seriously

disputed. I shall have something to say on this point when I come to speak of them in detail; but for my present purpose I shall assume the connection and speak of these seven phases, together with arthritis, as the rheumatic series.

Let me not be misunderstood in this matter however. I do not say that the rheumatic poison, or whatever the morbid influence may be, is the sole cause of these affections associated with rheumatic arthritis, any more than it is the sole cause of inflammation of joint structures. They are set up by other causes, just as arthritis, for example, may be set up by mechanical injury, by the poison of gout, or that of septicæmia or pyæmia. Yet the rheumatic poison is the most common exciting cause of arthritis, and so with other members of the rheumatic series. Take, for instance, pericarditis, endocarditis, and pleurisy. The most common cause of the first two, at any rate, is rheumatism. Yet these same inflammations of the pericardium, endocardium, and pleura are now and again set up by the septic or pyæmic poison, or by that of uræmia. Tonsillitis, erythema, and chorea in like manner are undoubtedly set up by other exciting causes as well as by rheumatism, although the latter is a frequent and potent one. The one manifestation of the series most closely identified with rheumatism, and almost diagnostic of the rheumatic state, is the evolution of subcutaneous nodules. These are, however, occasionally met with in rheumatoid arthritis, although such instances are rare.

It appears, then, that the rheumatic virus (whatever its exact nature and by whatever physiological machinery it acts) which produces the articular inflammation produces in like manner inflammation of the fibrous tissue of the pericardium, endocardium, and pleura, and that of fasciæ and tendons. It has clearly an irritant inflammatory effect upon fibrous tissue, not of the joints alone but of these other structures. It affects, moreover, mucous membrane and skin, and disturbs nervous centres. The presence of some subtle micro-organism would explain this widely operative influence of the rheumatic poison. The presence of such agent has not, however, been satisfactorily established. Thus, for example, an anærobic bacillus, isolated by Achalme and others from the blood of rheumatic patients, suffering from an acute attack of rheumatism, is considered by these observers to be the actual cause of the disease. On the other hand, diplococci, which have been frequently demonstrated in acute rheumatism, are by others supposed to be the specific cause. And again, Gustav Singer, in a monograph upon this question,[1] doubts the existence of a specific micro-organism, and inclines to the view that rheumatism is but a special variety of pyæmia, which owes its origin to an infection by staphylococci or streptococci. The conception of rheumatism, then, which I shall endeavour to put forward and establish is this broad one; that the terms 'rheumatism' and 'rheumatic' must be held to include many various morbid expres-

[1] *Aetiologie und Klinik des acuten Gelenkrheumatismus.*

sions—the series of phenomena I have laid down—and must not be regarded as a special inflammation of tendons and ligaments or synovial membranes, or as a condition of which this is always the chief feature, accompanied by complications and sequelæ. The term 'diathesis' is quite inadequate; there is something more than mere tendency—a common factor concerned in the production of those different phases—and forming the link between them.

The rheumatism of childhood exhibits a marked contrast to the disease as it is seen in mature life. The articular affection, which is regarded as the very type and essence of the latter, has not yet become the chief feature, but is usually slight and subordinate, and, indeed, may be absent altogether from a seizure undoubtedly rheumatic in its nature; while other phases, again, less marked in the disease of adult life, or even absent from it altogether, rule as prominent and characteristic features. The joint tissues are less susceptible; the other fibrous tissues more so. Subcutaneous nodules, which are so frequent and significant in early life, practically disappear with the advent of puberty; and chorea, so common in connection with the rheumatism of childhood, disappears as full maturity is reached. Endocarditis and pericarditis, again, frequent as they are in adult life, are more frequent still in children—*i.e.* they tend to decline as age advances.[1] In the rheumatism of early life arthritis is at

[1] The statistics of the Collective Investigation Committee of the British Medical Association (*British Medical Journal*, Feb. 1888) give 72 per cent. of heart disease in rheumatism in the case of children,

its minimum; endocarditis, pericarditis, chorea, and subcutaneous nodules at their maximum. As life advances this is gradually reversed; the joint affection becomes more prominent, constant, and typical of the disease, and reaches its maximum; while the other phenomena decline and tend to die out. So that, if the picture of the disease had been drawn originally (as it should be) from the rheumatism of childhood, the articular affection would not have been taken as representative, or endocarditis and pericarditis spoken of as complications. Endocarditis or chorea would have been taken as the primary essential phenomena, and the articular affection as a complication. Yet none are really to be regarded as complications. When, for instance, any of these phases—pericarditis, nodules or chorea—occur in immediate association with arthritis, they are in no way set up by the arthritis; they may come before it or after it, or occur quite independently. And so with the articular affection: the various phases are not complications or sequelæ, but direct manifestations of rheumatic activity.

Another point of distinction between the rheumatism of childhood and that of later life is the tendency of the various phases to arise independently and apart from each other. They do, indeed, occur grouped together commonly enough, as in adult life; but often also the series of rheumatic events is spread out, scattered over a period of months or years, so

as compared with 46 per cent. in male adults. In the case of female adults the difference is not so marked—*i.e.* they retain in some degree the special proclivity of childhood.

that the history of a rheumatism may be the history of a childhood. There may be, for instance, an endocarditis at one time, a chorea at another, a tonsillitis at another, without any other manifestation at the moment, and yet each may be as essentially an expression of rheumatism as the articular affection which may perhaps have happened long before or does not appear until long after. Look, for instance, at Series VIII., in the abstracts of cases in the table, showing the succession of rheumatic events—the first event was chorea, the articular affection not occurring until eleven months later; or at Series II., where the first event was an endocarditis which had occurred years at least before any arthritis was observed; or at Series X., where there were two attacks of chorea before the arthritis appeared. There was nothing, under the present system of taking articular affection as the key to the condition, to show that the chorea or endocarditis were rheumatic at the time they occurred. More usually, however, two or three phases occur more or less closely together; as, for instance, the common coincidence of arthritis with endocarditis (as in Series I.), or chorea, nodules, and endocarditis.

The rheumatic series as seen in children may, indeed, be complete or incomplete in any degree. The whole series may follow in succession or it may be limited to a single event even—an arthritis only, or a chorea only, or an endocarditis only, without subsequent development of any other rheumatic seizure. Again, the combinations of the different

phases of the rheumatic series may follow any order of sequence. Sometimes—most commonly—an arthritis first; sometimes a chorea first, or an endocarditis. We are so accustomed to regard arthritis as the starting-point, and other manifestations as complications or sequelæ, that the possibly rheumatic nature of the affections which come before it is liable to be overlooked. Yet these manifestations, developed previously to arthritis, cannot be looked upon as complications or sequelæ, for there is no arthritis to complicate or follow. They are direct and independent results of the rheumatic disturbance. Further, these phases of the rheumatic series may be either closely associated in point of time, singly or in groups, or scattered, with varying intervals between. Here, again, we are so accustomed to look at the short series of adult rheumatism, minus chorea and nodules, occurring in conjunction with arthritis, that even a group of significant affections occurring apart from arthritis would hardly be recognised as rheumatic. And so on with the other members of the morbid series, some of which, again, may be repeated more than once. Clinical examples, from cases actually observed, will serve to illustrate most clearly some of the various combinations and the wide range and protean character of rheumatism in children. The most frequent of all is the one with which we are so familiar in adults.

Series I.—(1) Arthritis; (2) endocarditis; these may recur more than once without other phases. H. B. K——, a boy of nine, was brought to the Hospital

for Sick Children in January, 1888, with rheumatism of the joints and advanced mitral disease. Here the arthritis recurred almost yearly after the first attack in 1882, and probably the endocarditis also, for when examined in 1888 there were marked thrill, a double murmur at the apex, and enormous hypertrophy and dilatation; while the chorea did not appear until six years after the first arthritis. Often, in childhood, to these two manifestations are added the development of nodules, and perhaps pericarditis or pleurisy.

Series II.—C. H. B—— was brought to the Hospital for Sick Children in May, 1888, for pains in the knees and in various joints. Small subcutaneous nodules were found on the knees, elbows, and malleoli. He had had pains in the back of the knees and in the ankles for the previous five weeks, never before. There was a double mitral murmur with great hypertrophy. Pleurisy developed on June 7th; then a fresh crop of nodules on June 9th; pericarditis on June 11th, and again on July 14th; a fresh crop of nodules on July 14th. With this came excited action of the heart; the pulse rose to 140, marking the advent of fresh endocarditis and probably of pericarditis. Pleurisy (slight) followed on August 16th; after this, gradual failure, and death on September 6th. In this case the primary endocarditis must have occurred some years before the pains in the joints were first noted, which was only five weeks before admission, whereas the heart disease was of long standing, as evidenced by the great hypertrophy. It

CLINICAL EXAMPLES OF DIFFERENT COMBINATIONS

SERIES I.	SERIES II.	SERIES III.	SERIES IV.	SERIES V.
H. B. K., boy aged nine. (Hosp. Sick Children, 1888.)	C. H. B., boy aged five. (Hosp. Sick Children, 1888.)	G. S., aged seven. (Hospital Sick Children.)	H. C., aged ten. (Hospital Sick Children, 1878.)	A. W., aged sixteen. (St. Mary's Hosp.)
1. ARTHRITIS, ENDOCARDITIS. (1882.)	1. ENDOCARDITIS (?)	1. ARTHRITIS, ENDOCARDITIS.	1. CHOREA, ENDOCARDITIS. (1875.)	1. CHOREA, ENDOCARDITIS. (1874.)
				MITRAL DISEASE found with —
2. ARTHRITIS, ENDOCARDITIS. (1884.)	2. ARTHRITIS, evolution of NODULES. (March, 1888.)	2 CHOREA five months later.	2. MITRAL DISEASE found. (1878.)	2. ARTHRITIS four years later. (1878.)
3. ARTHRITIS, ENDOCARDITIS. (1885. Recurring almost yearly for 6 years.)	3. Second crop of NODULES, PLEURISY, PERICARDITIS. (June, 1888.)	3. CHOREA after two months' interval. (Father had rheumatic fever; mother had chorea as a child.)	3. Arthritis absent. (Mother rheumatic fever and heart disease.)	—
4. ARTHRITIS, ENDOCARDITIS. (1886.)	4. Third crop of NODULES, fresh ENDOCARDITIS, fresh PERICARDITIS. (July, 1888.)	—	—	—
5. ARTHRITIS, ENDOCARDITIS. (1887.)	5. PLEURISY, PERICARDITIS (recurrent). (Aug., 1888.)	—	—	—
6. ARTHRITIS, CHOREA. (1888.)	—	—	—	—

OF VARIOUS PHASES OF THE RHEUMATIC SERIES.

SERIES VI.	SERIES VII.	SERIES VIII.	SERIES IX.	SERIES X.
A.B., aged twelve. (St. Mary's Hosp., 1884.)	F. M. C., aged seven.	W. S., aged four years and a-half. (Hosp. Sick Ch., 1887.)	F. M., aged five. (Hosp. Sick Ch., 1888.)	John T., aged seven. (Hosp. Sick Children, Dec. 1887.)
1. ARTHRITIS, ENDOCARDITIS. (1881.)	1. ARTHRITIS (severe). TONSILLITIS. (1878.)	1. CHOREA, probably accompanied by ENDOCARDITIS. (Nov. 1886.)	1. ARTHRITIS. (Sept. 1887.)	1. CHOREA, attributed to fright. (Nov. 1886.)
		Interval of eleven months.		
2. CHOREA. (Dec. 1884.) Old-standing MITRAL DISEASE discovered.	2. PURPURA RHEUMATICA. (1883.)	2. ARTHRITIS (first attack). (Oct. 1887.)	2. ARTHRITIS (second attack). (Jan. 1888.)	2. CHOREA (second attack), likewise attributed to fright. (Aug. 1887.)
3. PERICARDITIS. (Jan. 1885.)	3. CHOREA. (1883.)	3. CHOREA (second attack), SUBCUTANEOUS NODULES, ENDOCARDITIS, ERYTHEMA MARGINATUM. (Nov. 1887.)	3. CHOREA, eruption of NODULES, ENDOCARDITIS (first attack). (March, 1888.) ARTHRITIS (third attack). (April.) ERYTHEMA MARGINATUM.	3. ARTHRITIS, evolution of NODULES. (Nov. 1887.) ENDOCARDITIS. (Dec.)
—	4. TONSILLITIS, constantly recurring for several years. (1883-4-5.)	4. EMOTIONAL ATTACKS, CHOREA (continued), FRESH NODULES. (Dec. 1887.)	4. CHOREA (second attack), eruption of NODULES (second attack). (Dec. 1888.)	4. CHOREA (third attack), EMOTIONAL ATTACKS, LARGE NODULES (second eruption), ARTHRITIS (second attack), ENDOCARDITIS, PERICARDITIS, PLEURISY. (June, 1888.)
—	—	5. ERYTHEMA MARGINATUM, fresh eruption of NODULES, CHOREA (relapse), ARTHRITIS (second attack). (Jan. 1888.)	—	5. ENDOCARDITIS, PERICARDITIS. (July, 1888.) DEATH, Aug. 1888.
—	—	6. Fresh eruption of ERYTHEMA, fresh crop of NODULES, TONSILLITIS. (Feb. 1888.) DEATH, March, 1888.	—	—

is to be noted, too, in this case how endocarditis, pericarditis, and pleurisy coincided with the appearance of fresh crops of nodules.

Another common combination is that represented in

Series III.—(1) Arthritis and endocarditis; (2) chorea. George S——, aged seven years and a half, was admitted for right paretic hemichorea. He had had rheumatic arthritis seven months before, accompanied by endocarditis evidenced by a loud mitral murmur. Two months later he had chorea; then a second attack, for which he was admitted. There was a family history of rheumatism; the father had had rheumatic fever; the mother chorea as a child.

Sometimes the arthritis appears to be missing. Thus:—

Series IV.—Henry C——, aged ten years and a half, was admitted to Great Ormond Street, June 1878, with mitral stenosis and regurgitation and great hypertrophy. He had never had any joint affection, but he had had chorea three years before. It was found, moreover, that his mother had had rheumatic fever, and was suffering from valvular disease of the heart resulting from it. I think that in this case the morbus cordis must be reasonably attributed to rheumatic endocarditis; the chorea to rheumatic influence likewise, although no arthritis had appeared up to the time of observation.

The combination in which the articular affection appears last in the series throws much light on such cases as the preceding. Here is an example:—

Series V.—Alice W——, aged sixteen, admitted into St. Mary's Hospital complaining of pain, stiffness, and tenderness of knees and ankles. On examining the chest, well-marked mitral disease of old standing was found—viz. a loud regurgitant murmur, with great hypertrophy and dilatation. The original endocarditis must have taken place years before. Yet, observe, she was not known to have had any joint affection before the present attack ; but she had had chorea four years before, and, moreover, her mother had had rheumatic fever ; her mother's sister had had rheumatic fever. Can there be any reasonable doubt that both chorea and endocarditis (which, no doubt, occurred with the chorea four years before the articular rheumatism) were both of them rheumatic? Yet, until the articular affection appeared, which was delayed to the age of sixteen, this case was exactly similar to the preceding. If the chorea and accompanying endocarditis had been recorded at the time of their occurrence, there would have been nothing to stamp them as rheumatic, and the endocarditis would have been styled 'choreic.' The appearance of acute articular rheumatism afterwards, and the family history of the disease in mother and aunt, revealed the link between the different phases.

Another combination, where arthritis and endocarditis commence the series, and pericarditis and chorea follow later on, is thus shown :—

Series VI.—A. B——, a girl of twelve, was admitted into St. Mary's Hospital in 1884 for severe

chorea. She had had rheumatic fever three years before; she had also had endocarditis, as evidenced by a loud mitral murmur. The chorea rapidly got well under arsenic; but just at this point acute pericarditis came on. There was not the slightest affection of the joints, yet I take it both chorea and pericarditis were rheumatic.

You will observe that either endocarditis or pericarditis is present in each of the preceding series. Yet these most serious of all the rheumatic phases, present, as I have said, in nearly three-fourths of articular affections in children, are sometimes wanting. Happily this was so in the case of one of my own boys, and his history also illustrates the connection of two of the less prominent phenomena of the rheumatic series—viz. purpura rheumatica and tonsillitis.

Series VII.—F. M. C——, a boy of seven, had acute articular rheumatism, with tonsillitis, in 1878. There was no endocarditis or pericarditis, or other manifestation—arthritis and tonsillitis only; and for several years there was complete quiescence of rheumatic activity. Then, in 1883, or five years after the arthritis, purpura rheumatica appeared; this was unaccompanied by any other phase. Then, about one year later, chorea and emotional attacks occurred. Finally, he had tonsillitis, recurring frequently and severely for four or five years.

In all the examples yet given the rheumatic series has been incomplete, as indeed it is most commonly, many of the minor phases especially being wanting.

This is, however, partly due no doubt to the imperfection of the record, many of the cases being taken before the significance of subcutaneous nodules, tonsillitis, and erythema was fully recognised.

I will add, therefore, in conclusion of this part of the subject, three more combinations, in which the series is fuller and almost complete.

Series VIII.—Look at the remarkable succession of rheumatic events, occurring chiefly in groups, given in this series. W. S——, a boy of five, was recently under my care in the Children's Hospital. The first event was an attack of chorea in November, 1886, when the boy was between three and four years old. This was almost certainly accompanied by endocarditis, for a double mitral disease of old standing was discovered on his admission to hospital a year later. The second event was an attack of articular rheumatism (the first), in October, 1887. The third group of events was a second attack of chorea, accompanied by an extraordinary development of subcutaneous nodules, and also by fresh endocarditis and erythema marginatum, in November, 1887. The fourth group of events was a series of emotional attacks, accompanied by further evolution of subcutaneous nodules and continued chorea, in December, 1887. The fifth group of events comprised a second eruption of erythema, a second eruption of subcutaneous nodules, a relapse of chorea, and (last in order) a second attack of arthritis, in January, 1888. The sixth group of events consisted of a third eruption of nodules, a third eruption of erythema, and tonsillitis,

in February, 1888, followed by death in March. Here I would ask you to observe that the very first event of the series was chorea (probably accompanied by endocarditis), the first appearance of rheumatism of the joints being delayed until one year later; so that, if we take the joint affection as the test, there was nothing, when the chorea first occurred, to indicate its rheumatic nature; and it would no doubt have been classed as non-rheumatic, and the accompanying endocarditis simply termed 'choreic.' The rheumatic relation was only established by subsequent events, and the last phase was tonsillitis, in an unusual place, late in the series.

Series IX.—The next example (F. M——, aged five, Hospital for Sick Children, 1886) is not quite so complete, but represents a common sequence of events. The first was articular rheumatism in September, 1887. There was no other manifestation at the time. The next event was a second attack of articular rheumatism four months later (January, 1888); and this, again, was unaccompanied by any other manifestation. The third event came three months later still, when chorea developed, and this was accompanied by an eruption of large subcutaneous nodules, and with these the first sign of endocarditis, and also another phase—erythema marginatum. The fourth event, a month later still, consisted in a fresh eruption of nodules only, followed by convalescence and quiescence until December, 1888, when the boy was readmitted to hospital with a fifth rheumatic development, consisting of chorea and

another eruption of nodules. This case presents the unusual feature of two attacks of arthritis without appreciable endocarditis. A soft murmur was first found, without hypertrophy, in the third period, together with the chorea, an evolution of nodules, and a slight arthritis of one wrist only.

Series X.—The last example which I have to give exhibits the rheumatic events in different order. J. T——, a boy of seven, was admitted to the Children's Hospital in December, 1887. The first event in his case was chorea, accompanied at the time by no other phase of rheumatism, and attributed to fright (November, 1886). The second event was a second chorea, likewise unaccompanied by any other manifestation of rheumatism, and attributed to fright (August, 1887). Up to this period there had been no arthritis, but two months later it came in a group of phases comprising the third manifestation—viz., arthritis, evolution of large nodules, endocarditis (November and December, 1887); then a period of rest for nearly six months; after which came the fourth group of events, comprising a third attack of chorea with emotional seizures, a second eruption of nodules, pleurisy, pericarditis (June, 1888); and lastly, the fifth and final manifestation—recurrent endocarditis, pericarditis (in July, 1888), and death (August, 1888), when the pericardium was found enormously thickened, the aortic and tricuspid valves thickened, and the mitral thickened to a remarkable degree. In this case, intensely rheumatic, and one which ended fatally, it is deserving of notice that two attacks

of chorea at intervals of a year, both attributed to fright, preceded the first known attack of articular rheumatism; and also that the persistent endocarditis and pericarditis marched *pari passu* with the evolution of nodules.

Such, gentlemen, are some of the combinations of the rheumatic series. They are all taken from actual cases; but the list is not exhausted; the varieties are numerous, as numerous perhaps as the combinations and permutations possible with the eight phases I have named as commonly rheumatic. But the brief summaries I have given will serve perhaps to exhibit the disease in its different outbreaks, extending over months or years, as one connected whole, instead of the fragmentary view afforded by a single phase, or the less numerous phenomena of the rheumatism of adult life.

Before I pass on to the consideration of each phase of rheumatism in detail there are two other characteristics of rheumatism in childhood to which I must make some allusion. One is the incidence of the disease upon the two sexes; the other the influence of inherited constitutional predisposition.

Taking first the influence of sex. The incidence of articular rheumatism upon males and females exhibits marked contrasts and variations at different periods of life. I ask your attention to this, because it has an important bearing upon the rheumatic connection of chorea and heart disease, which I shall have to point out later. Taking males and females of all ages together, articular rheumatism is somewhat more

common in the former. The statistics of the Collective Investigation Committee of the British Medical Association[1] yield in 655 cases 375 males to 279 females.[2] Up to the age of twenty, however, the balance is the other way. For this period there is a preponderance of females. The Collective Investigation Statistics yield 108 males to 120 females.[3] After twenty the males are enormously in excess—viz. 263 to 158.[4] Yet further, this preponderance of females over males in early life is not uniform at all periods of this first twenty years, but shows a remarkable variation, which is, I think, of considerable significance. Taking periods of five years from one to twenty years, it appears that in the first, one to five years of age, boys preponderate—viz. 5 to 1. At the next quinquennial period, between five and ten years, they become nearly equal—viz. 15 boys to 14 girls. At the next period, eleven to fifteen years of age inclusive, comes a remarkable change. The proportion is suddenly and decisively reversed. During this quinquennial period girls suffer

[1] *Collective Investigation Record*, vol. iv. 1888, p. 67.

[2] The statistics collected by Senator from various sources (Ziemssen's *Cyclopædia*, vol. xvi. p. 18) give somewhat conflicting results; but collectively yield a similar preponderance of males—viz. 1,499 to 1,425 females.

[3] *Collective Investigation Record*, vol. iv. p. 67.

[4] The general preponderance of girls over boys is borne out by other statistics. The records of cases of articular rheumatism at the Children's Hospital in Great Ormond Street for twenty-six years give 336 girls to 327 boys. In this case the limit of age is for one period ten years, and for another twelve, so that it excludes a large proportion of cases between eleven and fifteen, when girls most predominate. Dr. Goodhart's statistics give 42 girls to 27 boys (*Diseases of Children*, p 510).

from articular rheumatism in great preponderance—viz. 47 girls to 25 boys, or nearly two to one. After fifteen there is another change; the greater liability of girls gradually declines up to twenty, so that at the close of this period males again preponderate—viz. 76 to 67.[1] The greater proclivity of females, which has been noted up to twenty years of age, is then, in reality, chiefly due to their extraordinary liability to the disease during this particular period of from eleven to fifteen. A strong confirmation of this special susceptibility of young girls to rheumatic arthritis is afforded by the fact that the rule holds with regard to scarlatinal rheumatism. According to the statistics of 652 cases of scarlatina at the South-Western Fever Hospital, noted by Dr. Gresswell, to whom I am indebted for calling my attention to the point and for this information, it appears that the incidence of scarlatinal arthritis in girls and boys between three and ten years of age is about equal, just as in ordinary rheumatic arthritis—viz. 4 boys in 176 cases, 5 girls in 179 cases; between ten and fifteen, boys 0 in 42, girls 4 in 71, again in harmony with the results in ordinary rheumatism. The numbers are small, but as far as they go support the special susceptibility of girls to rheumatic arthritis.[2] So that, if these statistics hold

[1] *Collective Investigation Record*, vol. iv. 1888. (Analysis of cases there given.)

[2] The susceptibility appears, however, to be more prolonged than in simple rheumatism. Taking the period ten to twenty-five years, the numbers are: boys, 2 cases of arthritis out of 80 cases of scarlatina; girls, 12 cases out of 118. Yet this is, of course, on the whole, a further confirmation of the special liability of young females.

good, girls from the age of ten to fifteen are about twice as liable to articular rheumatism as boys, in striking contrast to all other periods, when males preponderate. It will appear later that this greater liability of girls to rheumatic arthritis during this period corresponds with a similar liability to endocarditis and chorea.

The second personal factor, viz. hereditary predisposition, is one of the most potent. Instead of being overrated, it is, I think, underrated. The tendency to rheumatism is transmitted as strongly as the tendency to gout. This influence appears more striking in children than in the case of adults: partly, perhaps, for the reason that the constitutional tendency existing is usually excited into activity before maturity—if it is there, it comes out in childhood; and partly, perhaps, because of the greater ease with which the history of relatives is obtained in the case of children. The parents supply information about their own generation and that of their children; while grown persons, amongst the poor at any rate, know little, and forget much of the generation which preceded them. The estimates of the extent of this transmission vary according to the minuteness of inquiry made, and the evidence allowed as sufficient to establish the existence of the rheumatic state. Sir A. Garrod traced it in about 25 per cent. Dr. A. E. Garrod,[1] taking adults and children together, estimates it at 35 per cent. in rheumatic patients, as compared with 20 per cent. in non-rheumatic patients. These estimates are necessarily under the mark, since they

[1] The *Lancet*, July 21, 1888.

are founded solely upon the occurrence of well-marked attacks of acute articular rheumatism, and chiefly in adults, all evidence afforded by minor attacks or combination of other phases, such as heart disease, chorea, nodules, &c., being rarely taken into account. Dr. Goodhart,[1] taking children alone, found satisfactory evidence of rheumatic affection in 41 out of 69, or 57 per cent. The results furnished by private cases yield the most reliable data, and this is especially true with regard to children.

In thirty-two consecutive cases out of my private note books (in all of which careful and minute inquiries have been made), kindly collated for me by Dr. Poynton, in twenty-three, that is 70 per cent., there was a definite history of rheumatic fever in near blood relations. If chorea and erythema be taken as sufficient evidence of rheumatism, the proportion is raised to thirty-one out of thirty-three, or 93 per cent. If cases of arthritis, chorea, and morbus cordis are taken together, 180 cases give 103 with a definite family history of rheumatic fever, or 58 per cent. If chorea (exclusive of grimacing) and morbus cordis are accepted as evidence of acute rheumatism, the proportion rises to 137 or 80 per cent. with a definite family history of acute rheumatism. I may give a few examples to illustrate this influence of family predisposition.

George L——, aged eight, was admitted to Great Ormond Street for advanced mitral disease of the heart. He had had rheumatic fever four years be-

[1] *Diseases of Children*, p. 512.

fore, followed by two attacks of chorea. His father had rheumatic fever as a child, and died of valvular disease of the heart. His father's sister had rheumatic fever three times. The father's brother had subacute rheumatism.

Take another instance. George H. S——, aged nine years and five months, was admitted to Great Ormond Street with slight articular rheumatism, ushered in by tonsillitis. On examination a loud mitral regurgitant murmur and an aortic regurgitant murmur were found, with great hypertrophy. He must have had endocarditis long before, yet there was no history of any previous joint affection. His mother had had rheumatic fever; his mother's sister rheumatic fever, followed by chorea; another sister of the mother rheumatic fever; the father had rheumatism, but of doubtful nature.

A case is recorded by Steiner in his 'Diseases of Children,' which shows remarkable family proclivity to the disease. A rheumatic mother had twelve children, and eleven of them had rheumatism before the age of twenty.

In most instances the inheritance is from one parent only; but as might be inferred, when the proclivity is inherited from both parents, the tendency is greatly intensified; not only is the liability to the disease increased by the double inheritance, but its severity and persistence are increased also. Dr. Goodhart, in vol. xxv. of 'Guy's Hospital Reports,' relates a case where, 'with a rheumatic strain in both father

and mother, five out of six children under fifteen—*i.e.* all but a baby of fourteen months—had either articular rheumatism or heart disease. A boy of fifteen had had rheumatic fever twice, and had mitral regurgitation; the second, a boy of ten, the same; the third, a girl of eight, died of mitral disease; the fourth, a girl, had rheumatic fever after scarlatina, followed by mitral disease; the fifth, a boy of four, was laid by all winter with rheumatism.' The most extreme case of the kind which has come under my personal observation is the following: A girl nine years old, the child of a medical man, had chorea in its most severe form: the jactitation was violent in the extreme, speech was entirely lost for eighty-one days, and feeding was difficult. Repeated attacks of endocarditis, pericarditis, erythema, paresis of limbs, acid sweats, and pains in the joints followed, and successive crops of subcutaneous nodules in such profusion that no less than 200 were stated to be present at one time. Finally, in spite of all treatment, at the end of nine months of almost continuous illness she died of cardiac dilatation and failure, a case of extreme inveterate uncontrollable rheumatic affection in all its phases. The family history was charged with rheumatism on both sides. The father had subacute arthritis and muscular rheumatism; his sister died at eight years old of heart disease after acute rheumatism and chorea; his wife, the patient's mother, had suffered from acute rheumatism, heart disease, and chorea; her nephew, a cousin of the patient, had rheumatic fever and heart disease, and a niece subacute rheumatism.

Now it occurred to me that this extraordinary tendency of rheumatism to develop in certain families might be due to some special faults of locality or circumstances, but careful inquiry into a number of cases showed me that they arose in very various localities, in members of the family when in different places and under different conditions.

Having thus reviewed broadly the general characteristics of the rheumatism of childhood—viz. the comparative insignificance of the articular affection; the prominence and importance of other phenomena, some of which, such as nodules and chorea, are limited to early life; the tendency of these phenomena to appear independently, scattered singly or in groups through a considerable period; sometimes one, sometimes another, appearing as the primary phase, and the others in varying order of succession; the influence of sex, and of hereditary predisposition – I pass on to the consideration of the various phases in detail. The first—the arthritis, or articular rheumatism—differs in many notable features from the acute articular rheumatism of later life. I have alluded already to the fact, sufficiently recognised, that the articular affection is usually comparatively slight and may even be absent altogether. It is, however, I believe, nearly always present at some period of the rheumatic efflorescence. I have now under my care at St. Mary's Hospital, Kate B——, a girl of fifteen, who was admitted with severe chorea. There was no sign of articular affection, or of any other condition suggestive of rheumatism.

The heart's sounds were clear and natural. There was no history of any previous rheumatic affection of any kind to be obtained by most careful inquiry, there was no history of rheumatism of any form in the family, and the case was put down as an instance of non-rheumatic chorea. Twelve days after admission, however, she suddenly complained of pain and stiffness of the right wrist and back of the hand, which were found to be slightly but unmistakably swollen and tender. The arthritis subsided in forty-eight hours. Three days later a similar pain and tenderness of the right wrist and hand developed, subsiding as quickly as the first. It would certainly have escaped serious notice had not my house physician been on the watch for the possible appearance of some rheumatic sign. Such cases give the key to many instances of unexplained heart disease and chorea.[1] The arthritis is, indeed, less extreme in every way; there is less swelling and tenderness and pain. It is a comparatively rare thing to see a little child lying still and motionless, bound hand and foot, not daring to move or turn for fear of pain—the condition so characteristic of the disease in an adult. There is often merely a little pain and a little stiffness and tenderness, limited perhaps to a single joint or set of joints, hardly attracting notice, soon forgotten, often overlooked altogether, and frequently not severe enough for the doctor's aid to be called in. It is subsequent

[1] This has subsequently received further illustration and confirmation. Since the above was written, and three weeks after the occurrence of the transient arthritis above recorded, pericarditis has supervened. The chorea has disappeared, and there is no articular affection.

heart disease which sends these cases to hospital. Often when medical advice is sought the ailment passes for nothing but a slight feverish attack. Such cases are constantly described as 'low fever.' And, as the inflammatory condition is slight, so many signs met with in severe rheumatic arthritis are wanting. The profuse sweating, the intensely acid, sour-smelling perspiration, which is one of the typical symptoms in the acute rheumatism of adults, is rarely seen in that of children, the rarity being in inverse proportion to age. It only begins to appear with more severe joint affection as age advances, and is rarely extreme before puberty. Other conditions, again, associated with the profuse sweating are also wanting. The sudamina and miliary eruption which so often accompany it in adults are rarely if ever seen in children. And as the inflammation of joint structures is slight, so also is the pyrexia. The temperature seldom runs high; and this is the more remarkable because, as a rule, it rises readily in children—goes up suddenly for a slight cause to 104° or 105°, perhaps from such trivial disturbance as a dose of irritant food or a passing chill. Yet here, in this eminently pyrexial affection, it seldom rises in children above 101° or 102°; 103° and 104° are comparatively uncommon, and such rise is usually of short duration. Look at the case of S——, for instance, given in Series VIII. As you will see, during his stay in hospital from December, 1887, to March, 1888, a period of four months, he had articular rheumatism, eruption of nodules, endocarditis, and tonsillitis, in

addition to chorea and erythema, and died of the disease at last. Yet his temperature was chiefly subnormal, rising only once to 101° for a single night, and once to 100° for a like period. Take, again, the case of John T——, given in Series X. He was in hospital for nine months almost continuously. He had, amongst other rheumatic phases during his stay, arthritis, endocarditis, pericarditis, pleurisy, a pulse of 120 to 130, and the disease steadily progressed to a fatal ending. He died of recurrent pericarditis. Yet his highest temperature was 101·5° ; for two short periods only did it reach 101° ; for the rest it rarely reached 100°. Hyperpyrexia, which occurs now and again in the acute rheumatism of adults, and is in them so grave and dangerous, often absolutely uncontrollable, is rare in childhood. I have never seen a case of fatal hyperpyrexia in a child, and I can find no instance in which the temperature has shown a persistent tendency to run up rapidly beyond control to a fatal height. Dr. Fagge observed [1] that in fourteen cases of fatal rheumatic hyperpyrexia at Guy's Hospital all but two were over twenty years of age, and these were a boy of nineteen and a girl of eighteen. In Dr. Wilson Fox's [2] twenty-two cases of rheumatic hyperpyrexia the youngest was a girl of seventeen. Dr. Barlow [3] mentions a case of fatal rheumatic hyperpyrexia in a girl of thirteen, but he does not give the range of tempera-

[1] *Principles and Practice of Medicine*, vol. ii. p. 546.
[2] *Treatment of Hyperpyrexia*, pp. 71–77.
[3] *Brit. Med. Jour.*, September 15, 1883, p. 513.

ture, or state that hyperpyrexia was the direct cause of death by running up uncontrollably to a fatal height.

The slight arthritis of childhood often assumes a misleading aspect, and it is sometimes difficult to distinguish it from other ailments involving pain and tenderness of parts. Their recognition is of immense importance, since a deadly endocarditis or pericarditis may be insidiously developing concurrently. I give one or two clinical examples of errors of the kind.

M. T——, a girl of three, was observed to be slightly feverish and ailing on February 10. On the 12th the great toe of one foot was found to be swollen and tender. On the 13th she was brought to me. The child had a white and dry tongue; pulse 120; temperature 103°. The swelling and tenderness of the toe were supposed to be due to chilblain. No other joints were affected, but the pulse and temperature suggested articular rheumatism. The heart-sounds were clear. Two days later (February 15) both feet and ankles were slightly stiff, tender, and swollen, and movement of them was painful; temperature 102°. On examining the heart, a soft murmur was audible at the apex. The following day the pain and stiffness of joints were almost gone; temperature 99°. The cardiac murmur had become full and blowing. This murmur persisted for many weeks, and then gradually disappeared. The father's sister and her child had both had acute rheumatism, and the former pericarditis and mitral valvulitis also.

Take another case I saw in consultation a few years ago—a little girl four years old. The child had had, for a week or ten days, difficulty in putting the right heel down, and was supposed to have incipient talipes, for which she had been galvanised steadily without result. The difficulty in putting down the heel had subsided, but the child still showed the greatest disinclination to walk. I could find no deformity or distortion, but I found both knees unmistakably swollen and tender, especially the left. The temperature was 100°. It turned out, too, upon inquiry, that the girl had suffered from pains and stiffness in the knees and ankles from time to time for the previous six months; and further, the mother had had rheumatic fever. I had no doubt that the joint trouble was rheumatic. Under salicin and citrate of potash all tenderness and stiffness disappeared, and in a day or two the child was running about as usual.

Here is another example. R. C——, a boy five years old, was brought to me because he had suddenly become unable to walk when he got out of bed in the morning. He was most reluctant to attempt it, but when made to do so he went on tiptoe with bent knees; any attempt to straighten the knees gave pain. The ham-tendons were rigid and very tender. There was no swelling. The temperature was 103°. The heart's action was much excited, but there was no bruit. It appeared that the mother had had rheumatic fever and acute pericarditis, and examination showed

a loud mitral murmur. Salicin was given freely, and in twenty-four hours the stiffness and tenderness had so far declined that the child was able to walk, and the temperature had gone down to 100°.

Another example which I think throws great light upon cases of valvular disease of the heart of unexplained origin is that of Ernest C——, a boy who was admitted into the Children's Hospital with a loud mitral regurgitant murmur, heaving, diffused impulse, bulging præcordia, and other signs of hypertrophy and dilatation. He had never been observed to have any articular affection or chorea, or other sign of rheumatism. There was no family history of rheumatism. Three weeks after admission he suddenly complained of pain in one knee, and could not bear to straighten it or use it in walking. The tendons on each side of the popliteal space and its lower border were found tense, swollen, rigid, and extremely tender. There was a slight rise of temperature. In view of the mitral disease, there could be little doubt that the affection of the knee was rheumatic, and that the valvular disease was likewise the result of rheumatic endocarditis, possibly accompanied by some slight arthritis which had escaped with little notice and long been forgotten. This affection of the tendons of the hamstring muscles —the biceps, semi-tendinosus, semi-membranosus, gracilis, and sartorius—and walking with bent knees on the tips of the toes to avoid tension of them, is, I think, very characteristic. I have seen it now many times in rheumatic children, and it is analogous to

the stiff neck and torticollis which is sometimes in like manner the first or sole feature of a rheumatic attack. Conversely, other conditions are often mistaken for articular rheumatism. Essential paralysis in its early stage, when there is great hyperæsthesia, is one of them. The chief points of distinction are that in essential paralysis there is extreme flaccidity of muscle, the limb falling limp and loose, drooping of the toes, and the fact that the tenderness in general is not confined to joints and tendons. Later the disappearance of faradic contractility and tendon reflex would be decisive. I have, however, now under my care at Great Ormond Street a boy who had considerable rigidity about the knees, the extension of which gave great pain. The tenderness was, however, general, the toes drooped, and faradic contractility had almost disappeared in the affected muscles.

Another condition which is liable to be mistaken for slight rheumatic arthritis is syphilitic disease of the ends of the long bones. In this there are tenderness and swelling from accompanying periostitis, and there may be even some arthritis, the limbs being kept motionless from pain on movement. It may be distinguished by the presence of other signs of congenital syphilis, and by the history possibly; but the age at which it occurs—viz. in the first few months after birth—is almost diagnostic. Rheumatic arthritis is almost, if not quite, unknown in early infancy. I have never seen it under two years, although Dr. Goodhart has noted some doubtful cases in children of two

or three months, and Henoch records one at ten months. Senator has collected several cases in infants, one at four weeks old.

Another condition which has been mistakenly regarded as rheumatic is the tenderness, swelling, and immobility of the limbs which arise in infantile scurvy —the so-called scurvy-rickets. This is to be distinguished by the swellings being limited usually to the shafts of the long bones, although I have once seen a periosteal swelling on the malar bone and swelling and tenderness of the joints. Such, however, are rare exceptions. Other diagnostic points are the existence of spongy gums, subcutaneous hæmorrhages, and albuminuria. And then infantile scurvy is limited to the bottle-feeding period—*i.e.* the first eighteen months or two years, when articular rheumatism is almost unknown. Rheumatism of the right hip-joint has been mistaken for an early appendicitis. The joint tenderness and neuritis of influenza, the swelling of the wrists in tetany, the arthritic hæmorrhages of hæmophilia, the joint trouble of strumous disease, and pyæmic arthritis, are all conditions liable to be mistaken for articular rheumatism. I have only time to call attention to them as possible sources of error.

LECTURE II

Anæmia and leucocytosis in rheumatism—Tonsillitis, its relation to rheumatism—Erythema exudativum—Erythema nodosum—Purpura rheumatica—Hæmorrhage from mucous membranes—From bladder and kidneys—Venous thrombosis—Chorea, its place in the rheumatic series—Subcutaneous nodules, their character and structure—Mode of evolution—Rare in adult life—Pathological and clinical significance—Pleurisy—Occurs in two distinct ways: (1) late, in relation to cardiac disease, as a result of mechanical congestion; and (2) as a primary manifestation of rheumatism.

Anæmia.—The effect of rheumatism in producing anæmia in adults is sufficiently well known, but in children this is still more remarkable; the extreme pallor and the hæmic murmurs are often most notable. Dr. Goodhart[1] thinks, further, that even children of rheumatic parentage are often habitually anæmic. Others, again, have regarded anæmia as a predisposing cause of rheumatism. It may be that the inherited rheumatic taint gives the tendency to anæmia, and thus the rheumatism is the antecedent of the anæmia, not the anæmia of the rheumatism. Be this as it may, however, where the rheumatic state is actively developed, anæmia proceeds apace in children. The presence of the rheumatic poison appears to be inimical to the red corpuscles or their hæmoglobin; it either promotes

[1] *Diseases of Children*, p. 515.

their disintegration or interferes with their production. Trousseau[1] affirms that there is, perhaps, no acute disease which produces anæmia so rapidly as rheumatism; but this is not so. The rheumatic poison is not so rapidly destructive of red blood as that of diphtheria, which causes marked blanching in the course of a few days; but still its effect is decided and unmistakable. This influence of rheumatism in producing anæmia is greatly aggravated when it is accompanied by valvular disease of the heart and pericarditis. The deficient circulation through the lungs—the deficient oxygenation—may, in part, account for this; and it is accompanied, also, by progressive emaciation. Of this, however, I shall speak in the concluding lecture. I have now under my care a child with chorea and pericarditis. There is no arthritis, but the patient has had articular rheumatism before. The anæmia is extreme; the pallor that of a marked chlorosis.[2]

Tonsillitis.—The claim of tonsillitis to be ranked as one of the rheumatic series is, I think, well established. Tonsillitis is a common disorder, and

[1] *Clin. Med.*, vol. iv. p. 454.

[2] Dr. Garrod (*Med. Chir. Trans.*, vol. lxxv. pp. 199–263) describes the anæmia as being of two kinds, viz. (1) an acute oligocythæmia developing in the acute stage and rapidly recovered from, and (2) a more chronic chlorotic condition developed as a sequel to the attack, and often of long duration. There is also a leucocytosis, according to M. Hayem and Dr. Garrod, which is usually quite moderate, but was so extreme in a case of fatal rheumatic endo- and pericarditis under my care that the condition had on this ground been erroneously regarded as a splenic leukæmia.

often arises independently; but it occurs so frequently in direct and immediate association with articular rheumatism that some pathological connection cannot be doubted. Trousseau[1] recognised a rheumatic sore throat, and showed how this was apt to alternate at one time with stiff neck and torticollis, at another with joint pain and lumbago. Attention was again drawn to the almost forgotten observation of Trousseau by Dr. Kingston Fowler,[2] in 1880, who published an account of twenty cases of acute rheumatism ushered in by tonsillitis. The statistics of the Collective Investigation Committee[3] show that tonsillitis was associated with rheumatism in 158 cases out of 655, or 24·12 per cent., in addition to twenty cases of sore throat, the exact nature of which was not described. If these were added the proportion would be 27·17 per cent.[4] It is assumed, I think, generally, that tonsillitis always precedes the articular affection, and usually comes immediately before it—ushers it in, as the phrase goes; and this is no doubt the rule, but it is by no means invariable. Tonsillitis may occur at any period of the rheumatic series, although most often it comes first—immediately preceding arthritis.[5] In all but three of the 158 cases just quoted the tonsillitis preceded the arthritis at various intervals; in two of

[1] *Clin. Med.*, vol. ii. p. 466. [2] *Lancet*, December 11, 1880.

[3] *Brit. Med. Jour.*: Dr. Whipham's Report, Feb. 25, 1888, p. 391.

[4] Dr. Fowler estimated it as occurring in 80 per cent. of cases.

[5] Dr. Archibald Garrod and Mr. Cooke, in a paper published in the *Lancet*, July 21, 1888, found the percentage of cases of tonsillitis with rheumatic family history exactly the same as the percentage of articular rheumatism with rheumatic history—viz. 35 per cent.

the remaining three cases the tonsillitis accompanied the joint affection, and in the third followed it. In the fatal case of W. S——, shown in Series VIII., it came last of all. In the case of William K——, a boy of eight, recently under my care, it likewise occurred last of a series beginning with arthritis and endocarditis; four months later, chorea; at the close of the chorea, acute tonsillitis. My own boy, of whose case I have spoken as having exhibited almost all the phases of rheumatism except carditis, had tonsillitis during the period of arthritis, and constant, repeated, and severe attacks during the period of chorea, and for a considerable term after all other signs of rheumatism had ceased. Of four other children, not one has suffered from tonsillitis, although all have been brought up together under like conditions. Whether the tonsillitis set up by rheumatism presents any distinctive characters, I cannot say with certainty. Trousseau affirmed that it could be distinguished by its ephemeral character, lasting only from thirty to forty-eight hours, and he describes the rheumatic sore throat as something more than tonsillitis, involving the pharynx, soft palate, and uvula. In some cases which I have observed, the diffused character of the inflammatory redness and swelling was very marked; but although I think this is a common feature I do not think that it is an invariable one. Follicular tonsillitis and suppurative tonsillitis also occur. It is probable that the same chill which creates the condition favourable to the rheumatic

invasion, may also set up tonsillar inflammation apart from it. It is probable that tonsillitis may occur as a solitary expression of the rheumatic state. In many cases it arises in rheumatic subjects quite apart from the articular manifestation. Of its concurrence with another rheumatic affection—viz. chorea—I have seen three examples quite recently, and in all these the chorea occurred in a child who had had articular rheumatism at another epoch. But of its connection with endocarditis or pericarditis apart from arthritis I have no certain knowledge. Since tonsillitis is set up by many other causes as well as by rheumatism, the decision whether a given case of tonsillitis not immediately associated with articular rheumatism is of rheumatic nature must be based upon a comprehensive survey of the patient's life history and family predisposition, as well as of the accompanying symptoms. If it occurs in a child with a strong family history of acute rheumatism, or in one who has had articular affection, this is presumptive evidence in favour of the tonsillitis being rheumatic. Even if there has been no arthritis at any time, the concurrence of other members of the rheumatic series—such as subcutaneous nodules, chorea, endocarditis, pericarditis, or erythema—would afford evidence weighty in proportion to the particular phases developed, the completeness of the combination, and the extent of the series.

Erythema exudativum.—The connection of erythema exudativum with the rheumatic state appears much more clearly in the case of children than of

adults. With the latter it occurs occasionally; with children, according to my experience, it is common. The statistics of the Collective Investigation Committee give only thirty-two cases out of 655, or not quite 5 per cent.; but of the whole number very few are children—only fifty-one under twelve years of age. Dr. Barlow and Dr. Warner found that, of twenty-seven patients with rheumatic nodules, eight had erythema papulatum or marginatum, one urticaria, and one purpura—*i.e.* ten out of twenty-seven, and these in close association with the evolution of the nodules. Out of twenty consecutive cases of erythema papulatum or marginatum noted by Dr. Archibald Garrod,[1] ten had acute articular rheumatism, seven of these cardiac disease also, two chorea, three subcutaneous nodules, and five more had articular pains or a family history of acute rheumatism. It appears in various forms—erythema marginatum, erythema papulatum, erythema nodosum, and urticaria, the first named being the most common. The erythema may occur at any point in the rheumatic series; but it is usually, at any rate, associated with the development of active rheumatic disturbance of some other kind, and occurs chiefly in the marginate or urticarious form. These varieties are not infrequently associated with endocarditis and pericarditis in the more serious cases. In eight cases of extreme gravity which have come under my care within the last year, four of which were fatal from persistent recurrent pericarditis and endocarditis,

[1] *Treatise on Rheumatism*, 1890.

erythema of this kind occurred in one-half—two of the fatal cases and two others. Dr. Barlow, in his excellent introduction to the discussion of rheumatism at the meeting of the British Medical Association at Liverpool in 1883, refers to a case under his observation in which erythema occurred as one of a group in the rheumatic series, associated with endocarditis, pleurisy, and pneumonia, but without arthritis; the arthritis had occurred years before. He mentions another case, also under his own observation, where erythema appeared as one of a group of rheumatic phenomena, which included nodules, chorea, and fatal pericarditis, but no arthritis; that had occurred in a previous explosion of rheumatic activity. Dr. Barlow also gives two other cases in which exudative erythema ushered in rheumatic seizures—including endocarditis and pericarditis, with arthritis following.

With regard to the special form erythema nodosum, it would appear that, although it has, so to speak, an arthritis of its own, it is associated sometimes with true articular rheumatism. The eruption is attended in itself with pain of joints, and sometimes swelling. But it also arises in connection with genuine acute articular rheumatism. Sir Thomas Watson observed it to occur both before and after this affection, and quotes Rayer as having seen it in the same connection. Dr. Stephen Mackenzie records 108 cases. Acute and subacute arthritis or cardiac lesions occurred in sixty-seven, and what is most suggestive in two instances endocarditis developed

with the eruption without joint affection. In twenty consecutive cases recorded by Dr. Garrod, eleven had arthritis, six a family history of rheumatism. A case under my care at Great Ormond Street is interesting as illustrating the rheumatic connection, the concurrence of endocarditis, and also the occurrence of this eruption at an early age. Walter G——, aged two years and eight months, was admitted on October 1, 1888, with well-marked erythema nodosum on both shins. There was no arthritis, or any history of its previous occurrence at any period, yet the child had a well-marked mitral regurgitant murmur, undoubtedly organic. The father had had 'rheumatism,' and the father's sister rheumatic fever twice. In another case under my care at St. Mary's, in February, 1889, a boy, F. V——, aged eight years and three months, erythema nodosum appeared together with subcutaneous tendinous nodules, apart from any other rheumatic manifestation except tonsillitis. The tendency of erythema nodosum to occur especially in young girls—who are also most liable to rheumatic arthritis, to endocarditis, and to chorea—is in agreement with the view of its rheumatic nature. Exudative erythema, however, although often rheumatic, is, like arthritis and tonsillitis, set up by other causes than rheumatism. The facts that erythema occurs in cholera and in septicæmia as the result of poisoning by certain drugs, and the urticarious form by food-poisoning, suggest a similar influence of the rheumatic poison, and we should look at the appearance

of erythema in any given case as probably indicative of the existence of some irritant matter which is in circulation acting either directly or by reflection upon the skin. Such irritant may be the rheumatic virus; whether it is so or not must be determined by careful examination of the evidence afforded by the presence or absence of other members of the rheumatic series, or by their previous occurrence, together with the existence or otherwise of family predisposition.

Purpura Rheumatica.—This appears especially in rheumatic subjects, and, like erythema nodosum, has a local *quasi*-arthritis of its own; when it appears on the legs and ankles, the ankle-joints and feet are swollen, tender, and painful. Sometimes it occurs independently of any other rheumatic manifestation at the time. In one case, the patient whom I have mentioned before, a boy of eight (Series VII.), had had acute rheumatism severely twelve months before. The eruption appeared after standing about in the wet grass while very hot from playing cricket—a profuse crop of large purpuric spots on both legs, mostly about the ankles. There was much swelling of the ankle-joints, which were so tender that he could not bear to walk or move them; but no other joints were affected, and there were never any constitutional symptoms or the smallest rise of temperature, although it was taken frequently, from apprehension of a second attack of rheumatic fever. From these two circumstances—viz. that the ankles only were attacked, and that there was never any fever or general illness—I

conclude the swelling was not directly rheumatic, but a local œdema, from purpuric extravasation or thrombosis of venules. Purpura does, however, arise in the course of acute general articular rheumatism, as I have observed more than once. A remarkable example of this occurred in a girl of eighteen, a patient under the care of my colleague, Dr. Lees.[1] The purpura was extreme, and the attack of acute rheumatism ended fatally. Purpura rheumatica, therefore, while sometimes concurrent with active, acute, articular rheumatism, occurs also apart from it in rheumatic subjects, and is then probably a minor expression of the rheumatic state, although far less common than exudative erythema.

Hæmorrhage. — Hæmorrhage occasionally takes place from mucous membranes. I have twice seen hæmorrhage from the bladder in the course of rheumatic fever, accompanied by the painful passage of clots per urethram – and once a simple hæmaturia. In neither of these cases was there concurrent purpura.

Thrombosis.—Now and again thrombosis of veins takes place in the course of acute rheumatism, as in typhoid and other infective fevers. I have seen it in the axillary and subclavian, the jugular, and in both iliac veins ; in all instances under my observation the rheumatism was accompanied by valvular disease of the heart, and the impaired efficiency of the heart mechanism was probably a factor in the production of coagulation.

[1] *Lancet*, Oct. 1899.

Chorea.—This is one of the most interesting members of the rheumatic series. When associated with articular rheumatism it usually follows the arthritis; it is sometimes concurrent with it; and sometimes, again, as I have shown, and as M. Roger noted long ago, it precedes the joint affection. Instances of this are shown in the clinical examples given in the preceding lecture. Chorea has furnished several curious pathological problems, which have given rise to frequent discussions and controversies, some of which continue to rage unsettled to this day. All are agreed, I think, that there is a certain connection between chorea and rheumatism. As Dr. Barlow has well observed, there is no other general disease or fever of childhood with which chorea has any such association, with the single exception of scarlet fever, and that in far less degree; and scarlet fever, significantly enough, is the one fever especially associated with acute rheumatism. We never see a measles chorea, or a whooping cough chorea, or a mumps chorea. The only points in dispute, then, are the nature of such relation between chorea and rheumatism, and its closeness and constancy. Some hold with M. Roger, that all chorea is rheumatic—that it owns no other source; others that the relation is comparatively slight and rare. Statistics have been supplied on each side, some supporting one view, some another, varying perhaps in degree according to the unconscious bias of the compiler. Let me state clearly my own position on this question. I do not

think the evidence warrants the assumption that chorea is invariably of rheumatic origin, although recent observations tend to connect it more and more closely with the rheumatic state. I must say of chorea, as of tonsillitis, erythema, endocarditis, pericarditis, and arthritis—in fact, of the whole of the rheumatic series except subcutaneous nodules—that it is produced by other causes as well as by rheumatism. But I am convinced that rheumatism is the by far most common and potent factor. Other factors, however, must be taken into account in forming any satisfactory explanations of the genesis of chorea. There is a physiological basis in that greater mobility of nervous system and motor readiness of expression so well described by Dr. Sturges, and in the more excitable temperament of quick, intelligent children, and of girls as compared with boys. This all tells and plays a part in the development of chorea. Yet it is singular how small the association with other neuroses appears to be, at any rate as far as the individuals are concerned; for these are found, according to the Statistics of the Collective Investigation Committee,[1] in ninety-seven cases only out of 439, or a little more than 22 per cent., and these ninety-seven include seventy-nine cases of headache and migraine, so that if there is any pathological affinity it is with those special forms only. Taking the question of inheritance, there is what is termed a neurotic family history in no less than 46 per cent. At first sight this seems significant, but on examination the neurotic

[1] *Coll. Invest. Record*, vol. iii. p. 54.

affections included are such a motley crew that their presence in the record does not carry much weight ; for the list embraces paralysis, drunkenness, tubercular meningitis, diabetes, sunstroke, spinal injury, and sciatica. There is not, as might fairly have been expected, a large proportion of hysteria (7·5 per cent.). The only connection which comes out with any significance is chorea itself (14 per cent.), and this probably represents a rheumatic as much as a neurotic relation. And while I am upon this subject, let me pause for a moment to explode a time-honoured fallacy in the etiology of chorea—that is, that it spreads by imitation. This was true of the dancing mania of the middle ages, which spread amongst enthusiasts by a sort of hysterical contagion. And children learn certain tricks of grimacing. But they never acquire true chorea by imitation any more than they acquire nodules, or paresis, or endocarditis by imitation. Dr. West,[1] indeed, speaks of it as occurring occasionally, and of the necessity arising for changing the position of patients, to prevent the involuntary mimicry by one child of the movements of another. In the Collective Investigation Report[2] two cases are recorded as mimetic, but no details are given. I have been connected with the hospital in Great Ormond Street for more than twenty years. Cases of chorea are extremely common there ; in the girls' wards, especially, there are often five or six or more at a time.

[1] *Diseases of Infancy and Childhood*, seventh edition, p. 236.

[2] Vol. iii. p. 48.

It is very rarely, indeed, that there are not some. The chorea patients are placed indiscriminately with other children, and yet I have not seen a single instance of its spread by imitation. I believe the separation of children to prevent mimetic development of chorea is entirely unnecessary. The belief in its production in this way is a survival of some old tradition, or has arisen from mistaking a trick of grimacing for true chorea. The statement has got into text-books, and, like many other time-honoured fallacies, holds its ground there, simply because it is taken for granted without inquiry. In speaking of grimacing, I may here allude, in passing, to the minor chorea, which consists in mere winking of the eye and twitching of the lips or nose, without jactitation of the limbs. Grimacing is, I believe, a local chorea, connected with the eruption of the second teeth, and especially of the eye-teeth, and not, I think, essentially connected with rheumatism.

Now turning to true chorea, there is, even in the rheumatic cases, often something more than the constitutional basis of mobility of the nervous system, influenced and played upon by the rheumatic poison. There is frequently another factor—nervous shock. Sometimes the rheumatic invasion seems to be the direct exciting cause, as well as the predisposing influence; when, for instance, as often happens, chorea immediately follows, or is concurrent with articular rheumatism, and endocarditis or pericarditis. But shock or mental excitement is frequently found as the immediate exciting cause of the choreic disturbance in

rheumatic subjects. Witness the case of J. T——, Series X., eminently rheumatic, yet having two attacks of chorea ascribed to fright long before the first arthritis; and this shock or excitement is a common exciting cause enough in rheumatic cases. A remarkable instance of the effect of mental excitement, which I have mentioned elsewhere, came under my observation a year or two ago in a case of rheumatic chorea. A little girl of eight years old had acute rheumatism, with endocarditis and pericarditis, followed immediately by severe chorea. The chorea got completely well in the course of a few weeks, and the child was brought up to London for change. The day after her arrival she was taken to the pantomime for the first time, and became wildly excited. The next morning I was sent for to see her; chorea had returned so violently that she had to be held by her mother and nurse to keep her falling from the bed. I have never seen more extreme jactitation. This was developed suddenly in a single night. Again, a public schoolboy under my care for chorea following articular rheumatism, on his recovery returned to school and to work. His chorea did not recur until he go into a quarrel and scuffle with another boy. The next day he was again choreic as before. I would insist on the fact that there is nothing antagonistic between the agency of nervous shock or excitement and rheumatism. They are concerned together as factors in many cases of chorea, as in the two examples just quoted. Yet fright chorea and rheumatic chorea are spoken of as if distinct. If fright is the immediate exciting cause in a

given case it is labelled 'fright chorea,' as distinguished from rheumatic chorea. Such distinction is fallacious and misleading, and has confused the etiology of chorea. The rheumatic state is the most common predisposing cause. Sometimes the choreic disturbance follows on this without obvious immediate exciting cause. In other cases a nervous shock stirs it suddenly into action; fright acts equally on rheumatic and non-rheumatic.

Turning now to the vexed question of the degree of closeness and constancy of the connection between chorea and rheumatism, I would point out that the only evidence dealt with by most writers on the subject is the occurrence of acute articular rheumatism either preceding or accompanying the chorea. But there is a great deal of evidence in addition, some of it of quite recent development, which bears upon the question; this cannot be ignored; it must at least be taken into consideration and duly weighed in order to come to a correct judgment in the matter. Let me indicate some of these neglected data. In the first place, it appears from the Statistics of the Collective Investigation Committee, as previously stated, that between the ages of ten and fifteen, girls have a remarkable proclivity to acute articular rheumatism as compared with boys during the same period, which is that of the maximum incidence of chorea. This corresponds with a similar greater proclivity during that same period of girls as compared with boys to organic heart disease associated with acute rheumatism, and to organic heart disease associated with chorea. The special proclivity

of girls to chorea is more protracted, extending throughout childhood and adolescence. This singular harmony of relative incidence of these affections upon the sexes is possibly capable of more than one interpretation, but it is *primâ facie* very suggestive of a close pathological connection.[1]

Further, there is the evidence of arthritis occurring after the chorea. This is the least common sequence. The proportion is not large, yet, according to my observation, much more than is indicated by the thirteen instances out of 655 cases of rheumatism furnished by the Collective Investigation Record. Of these, nine are under the age of nineteen; three, twenty-four; one, twenty-seven. The explanation lies probably partly in the fact that these cases comprise a singularly small proportion of children. A considerable number of children with rheumatic chorea must be carried off by heart disease before reaching maturity, and those who survive are forgetful of slight choreas which are usually classed as fidgetiness or nervousness.[2]

[1] Collating the cases given in the *Collective Investigation Record*, it appears ('Tables of Cases,' vol. iv.) that, for this period of eleven to fifteen, acute articular rheumatism is met with in girls as compared with boys as 47 to 25, or nearly 2 to 1; rheumatic heart disease in about the same proportion - viz. 28 to 16, or nearly 2 to 1. Chorea associated with antecedent or concurrent articular rheumatism occurs in girls as compared with boys in still higher proportion—viz. 116 to 32 (vol. xiv. p. 47), or more than 3 to 1; organic heart disease in connection with chorea (collated from cases in vol. iii.) also in greater proportion—43 to 13, or more than 3 to 1; chorea generally, 131 to 47, or not quite 3 to 1 (vol. iii. p. 47).

[2] That this is so I have many times had proof from private cases—children brought for nervousness and restlessness, who, on examination,

Be this as it may, however, there are a certain number of cases of chorea connected with rheumatism usually excluded from the estimate.

Then, again, there is the evidence afforded by the association of chorea with other conditions which are themselves found in close relation to articular rheumatism—*i.e.* the other members of the rheumatic series. This evidence is of weight cumulative in proportion to the number and importance of the phases combined. Thus the occurrence of endocarditis or pericarditis in association with chorea would have a certain significance. When a case of endocarditis or pericarditis arises in connection with chorea, even if there be no arthritis, since rheumatism is the chief cause of endocarditis and pericarditis on the one hand, and so closely associated with chorea on the other, the concurrence of these—each *per se* allowed to be frequently rheumatic—affords a considerable presumption that they are together rheumatic. The recent valuable observations of Dr. Lees and Dr. Poynton, showing the frequency of acute dilatation of the heart in acute rheumatism, coupled with their further observation that acute dilatation is also common in chorea, affords another link of connection between them.[1] There is no other general morbid state so closely associated with chorea as rheumatism. There are only two diseases largely and closely associated with endocarditis—viz. chorea

turn out to have the jactitations of slight chorea. These cases do not come into hospital, and amongst the poor they probably never come under the observation of a doctor.

[1] *Med. Chir. Trans.*, 1898.

and rheumatism. Explanations, more or less plausible, have been suggested as to the cause of the endocarditis of chorea apart from rheumatism, as, for example, that it is *dynamic*, due to attrition of the edges of the valve segments when the heart is acting with undue force. The heart does not, however, act with increased force in chorea, on the contrary, it is enfeebled greatly. Moreover, no dynamic theory will explain the *pericarditis* of chorea when it occurs without articular affection. The significance of this fact has, I think, been overlooked.

The association of erythema nodosum or tonsillitis with the endocarditis or pericarditis would greatly strengthen the presumption of the rheumatic nature of an accompanying chorea; while the presence of nodules, so intimately associated with rheumatic activity, would be, I think, conclusive. The recent recognition of the rheumatic relations of the exudative erythemata and nodules has, indeed, brought much new light, and the evidence which these affections afford has yet to be fully collected and appraised. The history of a strong family predisposition to rheumatism, again, would possess a certain value. Look at the case of W. S—— in Series VIII., and that of J. T—— in Series X. In both these, as I pointed out previously, the chorea occurred long before the joint affection; and there was nothing to identify it as rheumatic at the time it occurred except the accompanying endocarditis, and in the latter case the fact that the child's mother had had rheumatic fever; yet later came the fatal

series of events stamping it as rheumatic—the endocarditis, pericardits, nodules, and arthritis.

Again, a boy of eight (Charles B——),[1] the child of well-to-do people, had general chorea of moderate intensity. He had never had arthritis, he had no nodules, and there was no sign of cardiac affection. I learnt, however, from the boy's mother, a young woman, that she suffered frequently from painful swelling of the finger-joints ; that her brother, a young man of thirty, suffered from valvular disease of the heart, on account of which he had been refused for life assurance ; that her mother and two of her mother's sisters suffered from subacute rheumatism, and another of her mother's sisters had had rheumatic fever. No neurotic taint of any kind could be made out. Does not such a history afford at least a presumption that the chorea known to be so often rheumatic owed something to the rheumatic connection in this instance? It is, moreover, to be noted that this history was only elicited by careful cross-examination. The answer to my first question, as to whether there was rheumatism in the family, was in the negative. Had I been content with it I should have failed to trace the rheumatic taint.

Another case (D. D——)[2] which came under my care is more suggestive still—that of a little girl of four, brought to me for a slight general chorea. She had never had articular rheumatism. There were no nodules to be found, and the heart's sounds were

[1] Note-book, XIII. p. 467. [2] Note-book, XIV. p. 7.

normal. On inquiry I learnt that her mother had had rheumatic fever, and died of heart disease, the mother's sister had had rheumatic fever and severe chorea. Does not such a history raise a strong presumption of the rheumatic connection in the child's chorea?

Again, I received into the Children's Hospital a boy of five, with chorea and small nodules. He had no arthritis, and apparently no fresh endocarditis. The pulse was quiet; the temperature normal. The only sign of rheumatic activity was the development of the nodules, and with it the chorea. Yet he had rheumatic arthritis more than ten months ago, and has a double mitral murmur.

Take, again, the case of H. C——, Series IV. He had chorea in 1875, accompanied no doubt by endocarditis, for he was admitted with advanced mitral disease in 1878. He had never had arthritis as far as was known. But his mother had had rheumatic fever and mitral disease. Is it not probable, to say the least of it, that the endocarditis and chorea were both rheumatic, the arthritis possibly having occurred without being noticed, or being yet to follow (as we have seen it sometimes do) later in the series? Evidence of this kind must be taken into account, although it is difficult to estimate it statistically.

When all these considerations which I have pointed out have been allowed due weight, there remains still a certain proportion of cases in which no rheumatic connection of any kind can be traced. The unsettled question is as to its measure and degree. Various estimates have been made. The value of such estimation is, to

my mind, very doubtful. In most instances the data are very imperfect. We may, I think, for example, at once put aside as inadequate all statistics based—as nearly the whole of them are—solely upon antecedent attacks of well-marked articular rheumatism, since they take into account only one phase, in severe form, occurring previously or concurrent with the chorea. Such statistics are necessarily defective, because—1. They omit many slight cases of joint or tendon affection, such as those of which I gave examples in the first lecture, conditions constantly unnoticed or forgotten, or, if observed, not regarded as rheumatic.[1] 2. They omit all cases in which the joint affection comes *after* the chorea. 3. They fail to take into account all the evidence afforded by the occurrence and concurrence of subcutaneous nodules, of endocarditis, of pericarditis, of erythema, and of tonsillitis; so that, unless well-marked articular rheumatism has likewise occurred before or with the chorea, these are classed as non-rheumatic. 4. They omit to take into account the

[1] It is significant, in relation to the genuine rheumatic character of so-called growing pains, that a family history of rheumatism is found in 49 per cent. of all 'vague pains,' almost identical with the 52 per cent. in definite acute articular rheumatism, and more than double the percentage found in diseases of all kinds, as stated by Dr. Sturges and Dr. Archibald Garrod—viz. 20 per cent. (*Collective Investigation Record*, vol iii. p. 48). The case of Kate B——, given in the first lecture, illustrates this point also. She was admitted for chorea. No history of articular rheumatism either in the patient or her family could be obtained, and the case was recorded as one of non-rheumatic chorea. Twelve days later she developed slight arthritis, lasting forty-eight hours. Three weeks later still pericarditis appeared, the articular affection and chorea having entirely ceased.

evidence afforded by family predisposition. Statistics, such as those of the Collective Investigation Committee, likewise necessarily under-estimate the rheumatic element, although in less degree. They do, indeed, take into account not only previous acute articular rheumatism, but also those cases in which it accompanied the chorea or followed it immediately. But by the very nature of the case, being a report on chorea at the time, they do not take into account cases where the joint affection occurs some time afterwards, and apart from the chorea. Many cases, again (slight joint affections, not being identified as rheumatic by the observers), are probably classed as 'vague pains.' Many, again, are no doubt altogether overlooked. Moreover, in these statistics, as in others, the significance of mitral disease, of erythema, of tonsillitis, and of nodules, alone or in combination, is not estimated or admitted into the rheumatic proof; and how little this most important and common piece of evidence, the evolution of nodules, is looked for and recognised, and how wanting these statistics must be in this respect, appears from the fact that, of 439 cases of chorea recorded, in only seven instances were nodules observed. Why, we have sometimes had as many in the wards at Great Ormond Street at the same time! There are six at the present moment. The statistics of the Collective Investigation Committee, based upon this single rheumatic event of articular affection before, during, or immediately after the event, give a 32 per-centage of chorea positively rheumatic. If to these be

added the cases of vague pains the percentage is 46·2. The estimates of two of the most sagacious clinical observers of our time come near to this. Dr. Stephen Mackenzie found 44·76 per cent. almost certainly rheumatic, and for reasons similar to those I have put forward, he regards this as representing very imperfectly the rheumatic connection of chorea. Dr. Barlow finds in forty-four out of seventy-three, or 60 per cent., sufficient evidence of rheumatism, and points out that the existence of progressive heart disease and the imperfection of the record render it probable that many other cases should be included. In eighty-four cases minutely recorded and specially investigated by myself, I find satisfactory evidence of acute rheumatism in the patient or immediate relatives in sixty-two—*i.e.* in 75 per cent. Leaving out the family history, the estimate would coincide very closely with that of Dr. Barlow. From private patients of the better class more reliable information can be obtained than from the poor. Out of fifty-five consecutive cases of chorea in private patients compiled for me by Dr. Poynton, in which the family history was ascertained with precision, there was a definite history of rheumatic fever in the patient or near blood relations in twenty-seven, or 52 per cent. : in two there was a family history of chorea, in one of morbus cordis, in two of erythema nodosum and chorea. If these are accepted as evidence of the rheumatic taint, the number would be thirty-three out of fifty-five, or 60 per cent. Now of these fifty-five cases of chorea ten are instances of face chorea : these are no

doubt usually included in such estimates, and I have so included them. If they were excluded, as I believe they should be, the number of rheumatic cases out of forty-five would be twenty-eight or thirty-three, that is according to the evidence accepted=64-75 per cent., which agrees closely with Sir Dyce Duckworth's recent estimate of 78 per cent. In addition to this more certain evidence there are the suggestive observations of Dr. Lewis of Philadelphia, Dr. Newsholme, and Dr. Garrod. The former notes a remarkable connection between the annual curves of rheumatism and of chorea, that of rheumatism following the elevations of chorea just a month later. This agrees with the statistics at St. George's Hospital. Dr. Newsholme, in his Milroy Lecture in 1895, shows that rheumatism is especially a disease of large towns; chorea is a disease of large towns likewise—an urban, not a country disease. Dr. Garrod informs me that in chorea as in rheumatism an increased amount of hæmatoporphyrin may be confidently looked for in the urine. It may then, I think, be concluded that in the majority of cases at least chorea is a phase of rheumatism.

It is not my purpose to enter into the question of the minute morbid anatomy of chorea, or the exact mechanism by which the motor disturbance is effected —whether by embolism in the motor tract, or by thrombosis there, or the irritant effect of the rheumatic virus, or the mysterious influence of nervous shock. All these in the present state of precise pathological knowledge can only be matters of speculation. But I

may point to the possibility of some proliferative change in the neuroglia akin to that of the fibrous tissues elsewhere as a point which needs examination.

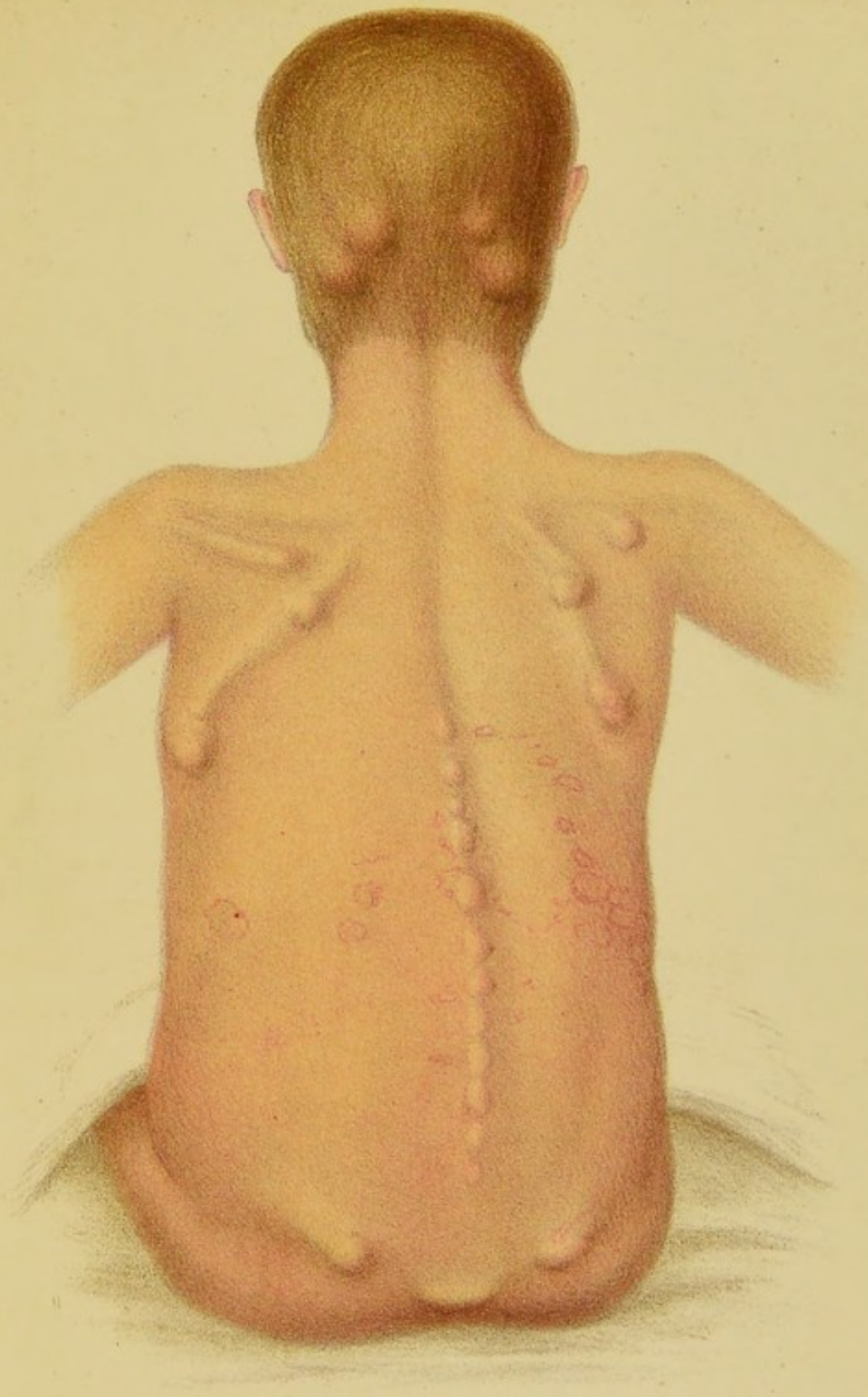
Before leaving the subject of chorea, however, I should like to call attention to the relation of nervous excitability to rheumatism. The rheumatic children who develop chorea are intellectual, highly strung, excitable, and nervous, often emotional, just as other choreic children. The question arises, is the nervous instability due to the influence of the recent active rheumatic state induced by it? The extraordinary emotional attacks observed in some cases of rheumatic chorea (as in the examples of W. S——, Series VIII., and J. T——, Series X.) would favour this. Or is the nervous instability part of the original constitution of the child, and chorea produced when this is acted upon by a second factor—viz. rheumatism? Or do the two tendencies run together in the original diathesis?—the child with a tendency to rheumatism inheriting therewith a mobile nervous system, as suggested by Sir Dyce Duckworth.[1] I am inclined to think the latter: that nervous excitability goes with the rheumatic diathesis, and that it is stirred up to active eruption of motor disturbance in chorea by some direct exciting cause—fright, or acute rheumatism, or pregnancy. I have seen this excitable tendency in the single child of a family in many instances. The rheumatic child is the excitable, emotional child. The rheumatic tendency

[1] Address to the Thames Valley Branch of the British Medical Association at Richmond (*Brit. Med. Jour.* Jan. 3, 1885).

and the nervous excitability run together in the same inherited constitution or diathesis.

Subcutaneous Tendinous Nodules.—I now turn to the next phase or manifestation—viz. the evolution of subcutaneous nodules. The actual discovery of these nodules is no new thing. They were observed by Hillier, who gives an excellent account of a typical case in his book on the Diseases of Children, published in 1868. The case was one of chorea with pyrexia and organic mitral murmur, but no articular rheumatism. Cases have since been reported by Meynet and others. I had long been familiar with them as occurring occasionally in the course of articular rheumatism ; but the credit of pointing out their frequency and great importance as clinical signs in the various manifestations of rheumatism belongs to Dr. Barlow and Dr. Warner.[1] These subcutaneous nodules are not uncommon in children—much more rare in adults. I have seen only three cases in grown persons, scores in children. This is perhaps partly the reason why they have not been more frequently observed, for we take our ideas of rheumatism from the disease as seen in adults. A second reason why they are not observed is that they are not known or looked for ; and a third reason why they often escape notice is their small size in many cases. They are sometimes to be felt rather than seen. In one case now under my observation, two of these growths, the size of a hempseed, on the tendons of the outer malleolus, only become visible when the skin is

[1] *Trans. Int. Med. Cong.* 1881, vol. iv.



Mintern Bros. Chromo lith.

NUMEROUS LARGE SUBCUTANEOUS NODULES.

W.S. Æt. 4½. (Series VIII).

Chorea-Endocarditis-Arthritis-Subcutaneous nodules,
Erythema marginatum=Tonsillitis (Pericarditis ?).

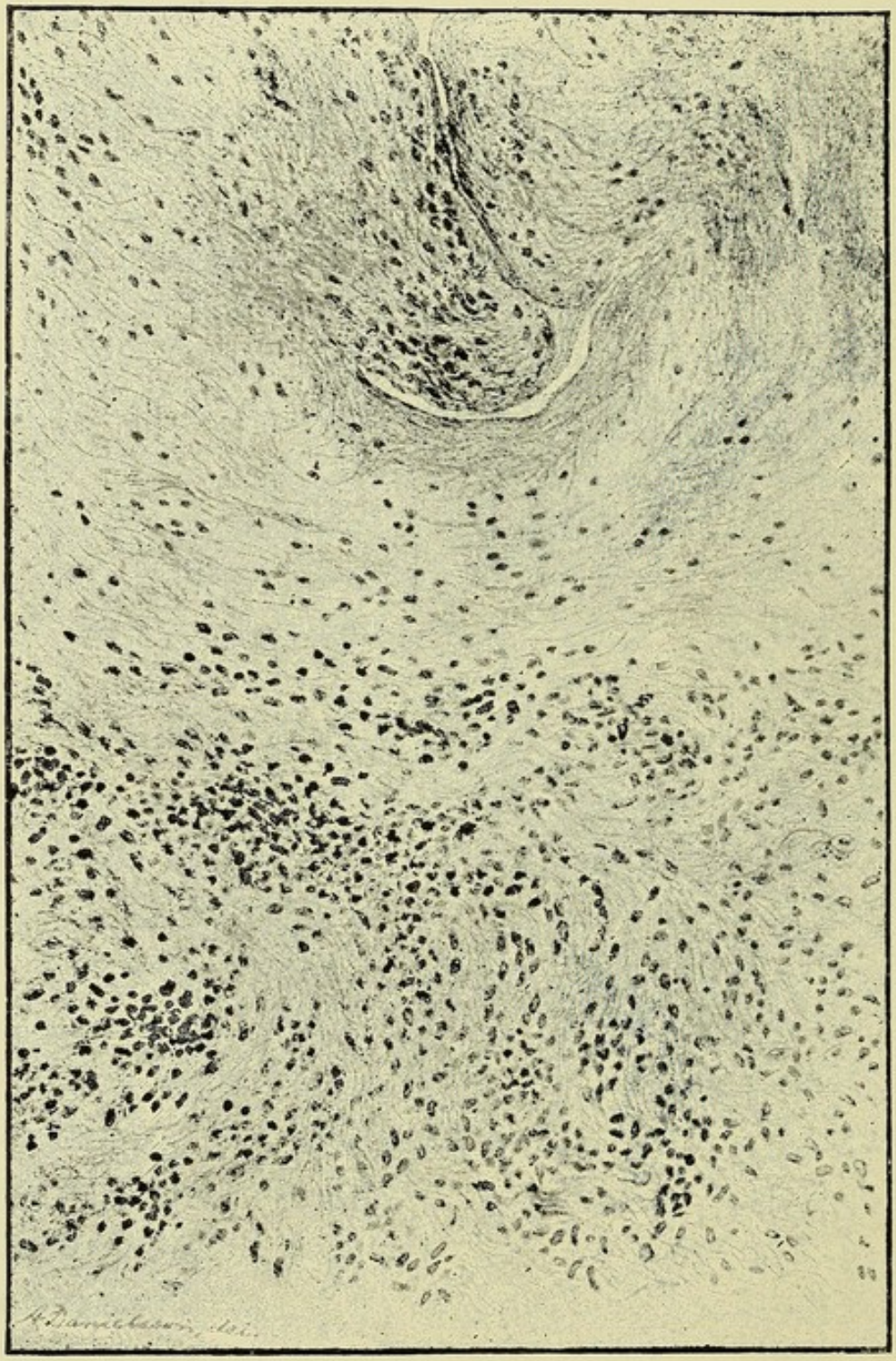
Fatal.

(Sketched from Life).

drawn tightly over them, although they can be detected readily enough by touch. In the 655 cases of articular rheumatism given in the 'Collective Investigation Record' (vol. iv.), there are only thirty-six in which nodules are recorded. Of these only five are in children under sixteen; but then the returns embrace a very small proportion of children. The chorea returns[1] yield only twelve cases out of 439. Yet I have at the time of writing this five under my care in hospital, and there are two more under my colleagues; all these are, except one of articular rheumatism, cases of chorea. The nodules vary in size from that of a hempseed to that of an almond or larger; in extreme instances they may attain that of half a walnut, as in the case of W. S——, Series VIII. (represented in the coloured illustration), and also in that of J. T——, Series X., and some others. The nodules lie under the skin, and are connected with fascia or tendons—in relation with fibrous tissues. They are not tender, except slightly in rare instances. There is no redness of the skin over them, except occasionally from friction or pressure. They are found most commonly upon the back of the elbow, over the malleoli, and at the margin of the patella. They are also found not unfrequently upon the head, especially along the superior curved line of the occiput, the temporal ridge, and now and again upon the extensor and flexor surfaces of the hands, on the extensors of the feet, the vertebral spines, the spine of the scapula, and the crista ilii. In extreme cases—

[1] *Coll. Invest. Record*, vol. iii.

FIG. I.



Microscopic appearances in a section of subcutaneous tendinous nodule in acute rheumatism, showing active proliferation and cell-infiltration of fibrous tissue. (John T——, aged seven years and a half; Hospital for Sick Children, Great Ormond Street; died August 6, 1888. Chorea, arthritis, endocarditis, pleurisy. nodules, pericarditis.)

arthritis, or cardiac inflammation, or pleurisy. For example, in the case of two children under my care, in whom nodules have developed in conjunction with chorea, the temperature has never been above normal. When the nodular growths are exposed by dissection, they appear as 'oval semi-transparent fibrous bodies, like boiled sago-grains.'[1] Examined microscopically in thin section, they exhibit wavy bands of tissue, with caudate and spindle-shaped cells and abundant nuclear growth, and they are highly vascular. They consist, therefore, of nuclear growth or fibrinous exudation in process of development into fibrous tissue in all stages of transformation. These appearances are shown in the section under the microscope, for which I am indebted to Mr. Priestley, formerly registrar to the Children's Hospital, and are represented in the drawings given in the next lecture. More recent investigations by Dr. Poynton and Dr. Still, in a paper read before the Path. Soc. April 28, 1898, show that the centre of a recent nodule is formed by a structureless material, which, when stained by special methods, gives a colour reaction similar to that given by fibrin. Thus it would appear that these nodules are localised manifestations of the rheumatic poison, and that there is at these points a fibrinous exudation and vascular dilatation precisely analogous to that which occurs in the pericardium in acute pericarditis. This exudation, which forms as it were the basis of the nodule, is probably rapidly absorbed in those cases in which the local irritation is slight, and in

[1] *Trans. Int. Med. Cong.* 1881, vol. iv.

this way the occasional rapid disappearance of the nodule seems best explained. On the other hand, more usually there is cellular exudation and later some fibrosis and shrinking, the nodule disappearing gradually rather than rapidly. A subcutaneous nodule is a proliferation of fibrous tissue, or of fibrinous exudation developed into fibrous tissue, according to the later researches of Dr. Poynton and Dr. Still ; [1] just such as that developed in the interstitial framework of the liver or in the nerve-sheaths by undue stimulation of alcoholic or other irritation. As Dr. Dickinson has well said,[2] the tissues of a child, and especially the fibrous tissues, are more readily excited to sprout in this way than those of adults, as shown by the rapid growth of fibrous tissue in hypertrophic cirrhosis and interstitial nephritis in children ; the quick hypertrophy of muscle in cardiac disease and the marvellous growth of sarcomatous tumours are analogous examples. On this point I shall have more to say in the next lecture, when I come to speak of endocarditis and pericarditis.

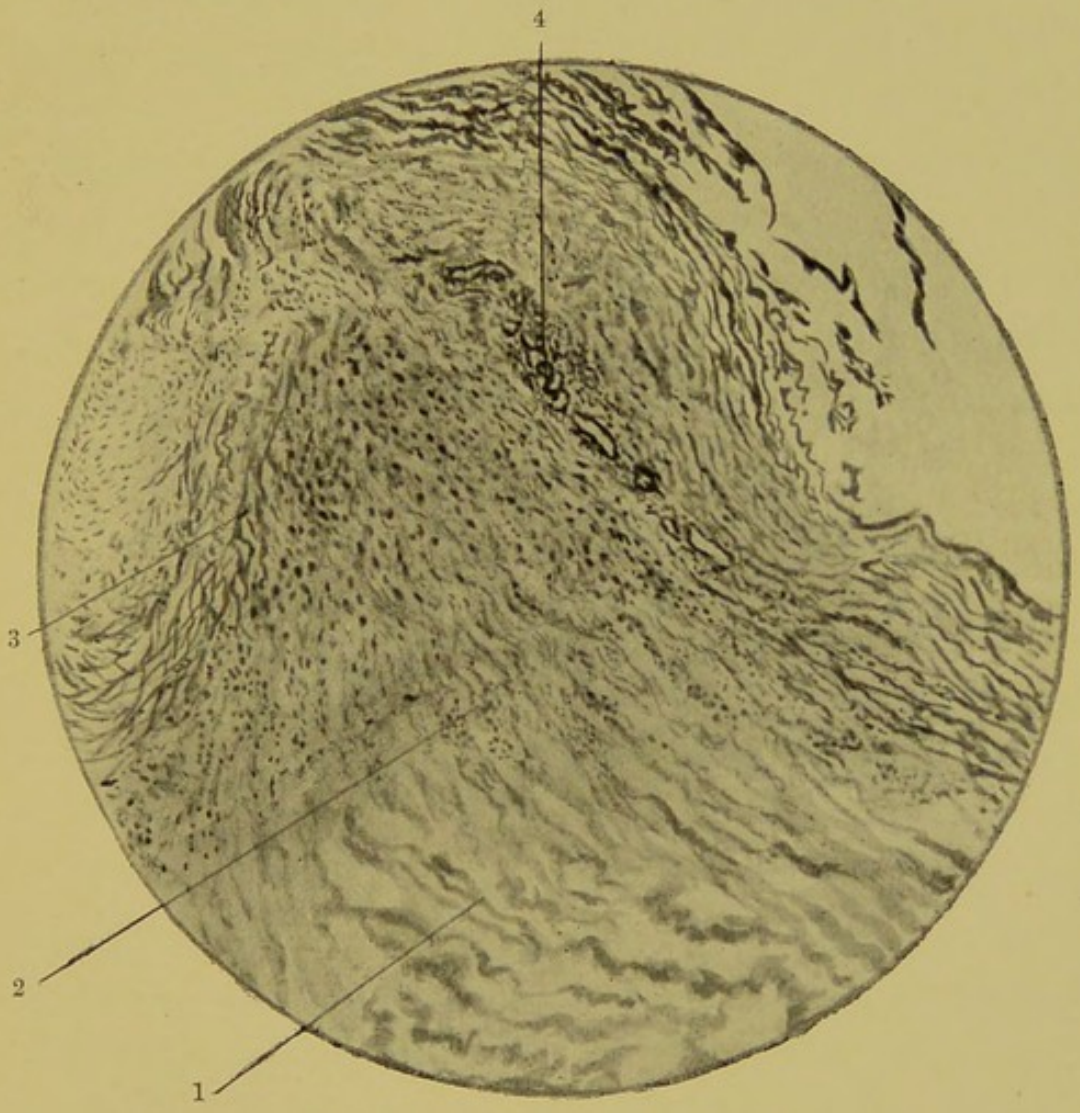
The connection of these nodules with rheumatism is extremely close. As far as I can judge, they rarely own any other origin or connection. In nearly all cases in which I have seen them, there has been either rheumatic joint affection at the time or at some period of the patient's history, or such a concurrence of rheumatic events, one or more—such as endocarditis,

[1] *Pathol. Soc. Trans.*, 1899.

[2] Introductory Address at the Hospital for Sick Children (*Lancet*, November 3, 1888).

FIG. II.

The Sketches II., III., and V. were made from the sections shown at the Path. Soc. in April, 1899, to illustrate the paper on the pathology of the rheumatic nodule by Dr. Poynton and Dr. Still. They are reproduced by the kind permission of the Pathological Society.



From a Sketch by Dr. A. W. Sanders.

Microscopic section of a subcutaneous nodule of three weeks' duration.

1. The fibrinous exudation in the centre of the nodule.
2. The zone of cellular infiltration.
3. Swollen fibrous tissue.
4. Tortuous capillaries.

pericarditis, chorea, and erythema—that there could be no doubt as to the nature of the condition. In nineteen out of twenty-seven cases recorded by Dr. Barlow there was arthritis; in six more there was distinct joint pain, and other evidence of the rheumatic state. In nearly every case recorded by others, the rheumatic connection has been clearly traced. The exception is in the case of rheumatoid arthritis, in the course of which I have, in adults, twice seen a copious eruption of large fibrous nodules, and a similar development has been observed by others. It is stated that they have occurred in influenza,[1] but of this I know nothing. As evidence of rheumatism they are, I think, indisputable, but there is something more than this. Not only are these nodules especially connected with rheumatism, but they are specially connected with the graver forms of it; and they are signs which are serious apparently in proportion to their size and number. Look at the case of W. S—— (Series VIII.) and that of J. T—— (Series X.). What a list of rheumatic events! In the first, repeated endocarditis, chorea, arthritis, and almost certainly pericarditis, accompanied the evolution of nodules. In the second, endocarditis, chorea, pleurisy, and pericarditis. Both cases ended fatally in spite of all treatment. Nothing availed to stop steadily progressive endocarditis and pericarditis. Salicylate of soda, salicin, free doses of alkalies and quinine, and mercurial inunction were equally futile. Of six cases of this kind, where the nodules were numerous and large,

[1] Washbourne: *Path. Soc.*, April 8, 1899.

which came under my care at the Children's Hospital during the last year, four were fatal. In all cases but one there was chorea. In every one there was endocarditis. In three, and probably in four, there was pericarditis. In two there was pleurisy.

So far as my present experience goes, I regard the eruption of large nodules, such as are shown in the Plate, as almost equivalent to a sentence of death. They mean persistent cardiac disease, generally uncontrollable, and marching almost infallibly to a fatal ending. General experience agrees as to the grave significance of these fibrous growths, which were at first looked upon as unimportant curiosities. Dr. Barlow and Dr. Warner [1] first recognised their serious import. In every one of the twenty-seven cases observed by them there was organic heart disease; in eight there was pericarditis; in twelve there was progressive valvular disease; and eight were fatal in spite of all treatment. Dr. Money, who has since investigated the subject, found nodules in half the cases of rheumatism in which well-marked heart disease occurred, and my experience is quite in accord with these observations. The grave importance of the occurrence of nodules in rheumatism is further borne out by the statistics of 150 fatal cases of rheumatism in children under twelve compiled and analysed by Dr. Poynton.[2] In eighty-seven fatal cases since 1879

[1] *Trans. Int. Med. Cong.*, 1881, vol. iv.

[2] Dr. Lees and Dr. Poynton, *Med. Chir. Trans.*, 1898. Appendix C, p. 443.

—*i.e.* since the existence of nodules has been noted—they were found in forty-seven, and in eight more their presence was doubtful, making 54 per cent. This, however, under-estimates their frequency, for it has been shown by Dr. Poynton and Dr. Still, in a paper read before the Pathological Society in April, 1899, that nodules can be demonstrated after death which could not be seen or felt during life. I could give examples of the association of these nodules with every other phase of the rheumatic series, as well as with endocarditis and pericarditis and chorea and pleurisy already mentioned; and in all these cases the evidence they afford as to the rheumatic nature of the affection is of the highest value, and I believe decisive. But their greatest interest lies in the fact of their frequent association with chronic recurrent endocarditis and pericarditis of the most deadly form, and in their connection with chorea.

Pleurisy.—Pleurisy occurs in association with rheumatism in two distinct ways. It arises frequently towards the end of rheumatic heart disease—partly, perhaps, as a result of the mechanical congestion of the pleura, caused by the valvular defect, or by pericarditis, or by extension from the latter. Of this late pleurisy, instances are common enough, as in the cases of Series II. and X. Of it I shall say nothing further, since it is probably due as much to cardiac as to primary rheumatic causes. But pleurisy likewise occurs in rheumatism as an initial phenomenon, coming immediately before, together with, or immediately after

arthritis, as a direct result of rheumatic influence. It is likely enough that it occurs quite independently, apart from all other phases, yet still a rheumatic manifestation. Probably many of the simple pleurisies and pleuro-pneumonias we see and regard as idiopathic would be found, on careful inquiry, to be of rheumatic origin. This is a point which deserves further investigation. Cases such as the two following are, however, very suggestive. For the particulars of the first I am indebted to Dr. Lewis, of Hamilton Terrace, with whom I saw the case.

On Dec. 22nd, 1887, a boy of ten travelled up to town from the seaside in extremely bitter weather. On the 24th he complained of pain in the ear, and subsequently had purulent discharge from it. On the 25th he was sick twice. At this point Dr. Lewis saw the boy, and found him with a temperature of 104°, and sweating profusely. No physical signs of any kind could be detected. On the 26th the fever and sweating continued ; the temperature was 104·2°. On examination of the chest, pleuritic friction was heard at one spot, and this had greatly extended next day. Three days later he complained of pain and stiffness in the wrists, knees, and ankles, which were discovered to be swollen and tender. Signs of pneumonia had also developed. Salicin was given freely, and in forty-eight hours the articular symptoms subsided. The next day the temperature came down rapidly, and there was considerable collapse, which passed off, and the boy got quickly well. The heart remained unaffected.

The second case is that of a boy who was under my care at St. Mary's.[1] He was admitted with signs of some consolidation and pleuritic effusion on the left side, and cough. Three days later both wrists and knees became stiff, tender, and swollen; the temperature was 103°. Under salicin the arthritis quickly subsided, and the boy made a quick and rapid recovery.

It may be that in such cases the pleurisy is simply the direct result of chill, just as the articular rheumatism is the result of chill, both being due to a common cause. Yet, pleurisy comes sufficiently often later still in the course of acute articular rheumatism, when it can hardly be attributed to the initial chill, and when there is no serious cardiac change to account for its origin in mechanical congestion, to render its immediate dependence upon the rheumatic virus highly probable.

The pleural exudation too in fatal cases is usually fibrinous, and resembles the pericardial exudation, both in this characteristic and in its microscopic appearances.

[1] F. G. H——, Albert Ward, December, 1888.

LECTURE III

Pericarditis—Its connection with other phases of rheumatism—Special characteristics in childhood—Subacute, recurrent, dry—Tendency to fibrosis and chronic thickening rather than to effusion—Myocarditis—Endocarditis; subacute and recurrent likewise—Special liability of children and of young girls—Connection of endocarditis with other phases of rheumatism—The endocarditis of chorea—Relation of pericarditis and endocarditis to the evolution of nodules—Morbid changes in nodules and cardiac valves analogous—Significance of this—Different forms of valvular disease—Mitral stenosis—Early signs—Double second sound at the apex—Hypertrophy and dilatation—Simple acute dilatation—Comparative rarity of dropsy—The mode of death differs from that met with in adults—Scarlatinal rheumatism—Rheumatoid arthritis—Special points in treatment.

PERICARDITIS is admitted into the rheumatic series without question. It is, I think, undisputed that inflammation of the pericardium owns rheumatism as its most common cause. Pericarditis may appear at any point in the rheumatic procession of events—first or last, alone or in combination with other phases. Most often it comes late, after endocarditis, especially when the heart is already hypertrophied and dilated. Sometimes it is associated with the valvular inflammation, is often accompanied by the evolution of nodules, and not unfrequently arises in connection with chorea. The development of pericarditis in

association with chorea apart from articular rheumatism is a link of association between them which is of considerable significance. Examples of different combinations and the place of pericarditis in them are shown in the illustrative cases, especially in Series II., VI., and X., and in the case of Kate B——, given in the first lecture, as well as in some examples which I am about to put before you. But the pericarditis with which we are most familiar—the acute general pericarditis of rheumatic fever—is not representative of the disease in children. The extreme distress; the panting, shallow respiration; the fluttering, irregular pulse; the strange delirium; the physical signs of friction all over the cardiac area as lymph is poured out; the extensive dulness, often following quickly as serous effusion takes place, spreading high up to the second or third rib; the muffled heart's sounds; the raised apex—all these classical signs are either wanting, or largely modified, in the rheumatic pericarditis of childhood in its most usual form.

Examples of acute general pericarditis of this character do indeed occur in the acute rheumatism of childhood, as well as in adults, but they form the exception. I had under my care at St. Mary's a girl of seven, Elizabeth S——,[1] who developed most acute pericarditis with chorea, and died of it in ten days. There was not the slightest accompanying articular affection, not the smallest joint pain or tenderness; the temperature ranged between 98° and 100° only except

[1] Children's Ward, December, 1888.

for one night, when it reached 101·5° ; but—most evil sign—the pulse ran up to 130 and 140. The child had had slight articular rheumatism seven months previously, and again a fortnight before admission and prior to the chorea. Her father had had rheumatic fever ; her sister, aged twelve, subacute rheumatism. There was a rumbling mitral murmur. The child was thin, pallid, and feeble. The progress of anæmia since the advent of pericarditis was most remarkable ; in the last few days of life it became extreme. Moreover—what I will ask you to mark especially—there were no nodules, so common an accompaniment of cardiac inflammation in a child, whether it be endocardial or pericardial, as I pointed out before. This very acute inflammation appears to be less identified with the development of nodules than the more chronic form.

Let me give another example of intensely acute general pericarditis, associated with equally extreme chorea, probably rheumatic. M. B——, a girl of fifteen, was admitted under my care at St. Mary's Hospital in August, 1885, with chorea of the most violent kind, rapid respiration and panting dyspnœa. She had been ill about three weeks, but it was impossible to obtain any clear history, for the people who brought the patient could give us little information about her, and the girl herself was unable to utter a word. She made frantic attempts to speak, but could not articulate, uttering only unintelligible sounds. The jactitations were so violent that she had to be fastened down in bed, and it was not possible to

examine the chest satisfactorily, so constant and uncontrollable were the movements. By the aid of two nurses I was just able to make out that the area of cardiac dulness was increased, the heart's sounds—as, indeed, extreme feebleness of the pulse showed—were almost inaudible, and a faint friction-sound could be detected at one point. The diagnosis was rheumatic chorea and pericarditis with some effusion. Before evening, a few hours after admission, the patient died. On post-mortem examination, extensive pericarditis was found, the pericardial sac being greatly distended with turbid, flaky, sero-purulent fluid, and the surfaces thinly coated with recent lymph. There was also inflammatory thickening of the mitral valve. In this instance, the chorea, endocarditis, and pericarditis occurring in the same individual stamped the case as almost certainly rheumatic.

Instances of extremely acute pericardial inflammation like these occur as they do in adults. As I explained to you, however, this is not the most frequent and typical form of pericarditis in children. It usually occurs insidiously; a slight pericardial rub is noted perhaps, which may cease or continue without much change; the child is seen to be restless and uncomfortable, and complains of pain in the præcordial region; the pulse quickens to 120 or 130; the anæmia increases to a marked degree; the chorea, if present, becomes a little aggravated perhaps, or curious emotional attacks come on, the child being moved to tears or laughter by a word; the temperature is slightly raised, perhaps to

100° or 101°, but often remains normal if there is no accompanying arthritis, or pleurisy, or pneumonia; and with this a mitral murmur develops, or an existing one grows rougher, and subcutaneous nodules begin to appear on the elbows and knees, or ankles or occiput. The pericardial rub continues, or subsides after a few days, to reappear again after an interval perhaps, or, although it never reappears, the rapid action of the heart continues, in spite of digitalis, or belladonna, or strophanthus, or ergot; fresh nodules come out; the area of cardiac dulness increases, showing advancing dilatation, and there is muffling of the sounds over the mid-cardiac region, but no sign of effusion; the heart is clearly growing larger, and the pericardium thicker, and emaciation and anæmia proceed apace. The child daily grows more pallid, weak, and wasted; the pulse grows more feeble; and so, without extreme dyspnœa or dropsy, the patient sinks slowly from exhaustion and heart failure.

Such, gentlemen, is a picture of the pericarditis of childhood in its most usual form, so frequently associated with the evolution of subcutaneous nodules, often with endocarditis, and sometimes with chorea. It represents very closely the case of J. T——, Series X. And mark what is found after death: usually the heart is dilated; and the two surfaces of the pericardium glued together by a thick layer of adhesive lymph; the pericardium itself greatly thickened; the walls of the sac tough, dense, fibrous tissue, an eighth of an inch thick, perhaps; the chronic inflammatory process

spreading sometimes from the external sac to the anterior mediastinum, so that these are matted together in a thick fibrous mass—'indurative mediastino-pericarditis,' as seen in the specimen from one of these cases now before you.

Take, again, the case of C. H. B—— (Series II.), a boy of six, who was admitted to the Hospital for Sick Children on May 8, 1888. The boy had never complained of anything until five weeks before, when he had stiffness in that typical place which I mentioned in the first lecture—viz. the hamstring tendons—and in the ankles; but they were not noticed to be swollen or tender. Then he had pains in the shoulders, and then in the chest, for which he was poulticed; but he was never kept in bed, nor was it thought necessary for a doctor to see him, although he was observed to be very breathless on exertion. The pains were supposed to be growing pains merely; rheumatism was not thought of. Latterly he had grown very excitable and nervous, and had twitching of the limbs. On examination, he was found to have a double mitral murmur with a highly accentuated second sound, and considerable hypertrophy; the dulness reached to the left edge of the sternum, and upwards as high as the third rib; the apex-beat was in the fifth space, three-quarters of an inch outside the left nipple—so that the valve disease must have been of considerable standing, due to endocarditis of much earlier date than the late articular pains. There was also discovered a crop of subcutaneous nodules on the elbows,

knees, and malleoli, so small as not to attract attention unless carefully looked for. The boy was extremely anæmic; the pulse suspiciously rapid (124). By the end of the month these nodules had attained considerable size, and on June 9th a fresh development of still larger nodules took place on the back of the head, the malleoli, and the knees; with this, pericardial friction occurred two days later, and great pain in the cardiac region. In a week's time all pericardial friction had disappeared, but the boy remained pale and anxious, the pulse was quick and irregular, pleuritic friction was heard at the base of both lungs, and the systolic mitral bruit had become musical. A month later (July 14th) a third crop of nodules of large size appeared on the knees and elbows. The cardiac excitement continued, the pulse ranging from 130 to 140; pain in the chest came on; emaciation and anæmia were progressive. No friction could be heard, but the increasing area of cardiac dulness, the rapidity of pulse, which neither digitalis nor any other drug could control, showed no diminution; and thus, in spite of all treatment, the boy went from bad to worse, and died from heart failure on September 6, just four months after admission. It is to be noted how in this case the grave symptoms proceeded *pari passu* with the eruption of large nodules, rapid pulse, progressive anæmia and emaciation, and how steadily and resistlessly the disease marched on in this way to a fatal end, uncontrolled by any remedial agent.

Take, again, another case, that of Arthur C——, a boy of eleven, who was under my care at Great Ormond Street Hospital. He was first admitted in September, 1888, with subacute arthritis, great anæmia, a loud double mitral murmur, and great hypertrophy. He quickly went out convalescent, but was brought back on November 12th, again failing in health. On the 22nd the temperature, which had been normal, rose to 101°, and well-marked double tonsillitis followed. This was over in three days, and the temperature fell to normal; but a week later (November 28) he became restless, his respirations went up to 32 and his pulse to 112, he had headache, and vomited. The following day (the 29th) the first sign of nodules was detected on the knees and elbows. The heart's action became more excited; by December 6 it was violent, and the anæmia had greatly increased; the nodules had grown larger, some to the size of small peas, and a pericardial friction-sound was audible. The temperature fell to normal, but the pulse kept between 100 and 112, and there was dyspnœa at times. The friction continued, the anæmia increased, and the prognosis became most grave. Note here again, the rapid pulse, the increasing anæmia, and the development of nodules concurrently with the development of progressive intractable pericarditis.[1]

Rheumatic pericarditis, then, in early life is apt

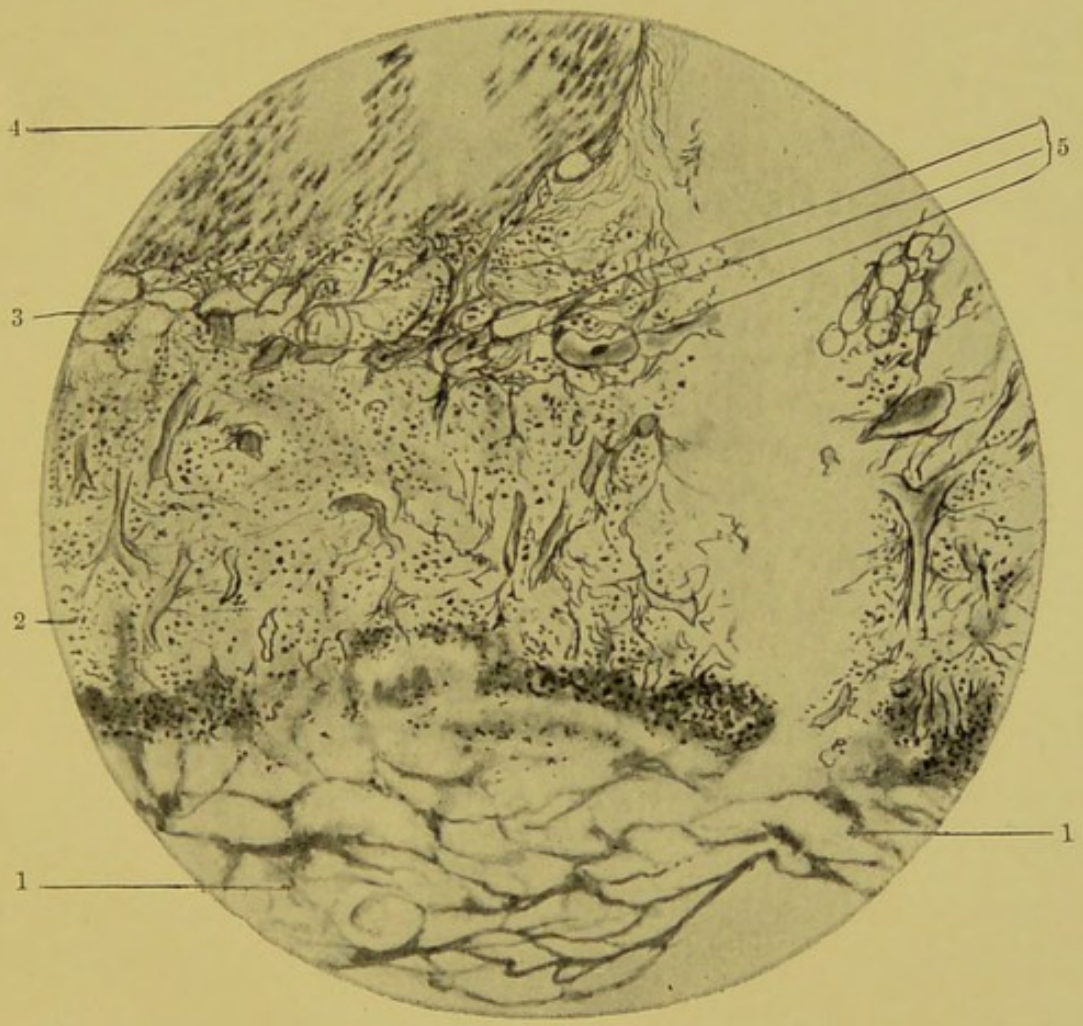
[1] This unfavourable prognosis has since been verified. The pericarditis continued in spite of treatment, anæmia and dyspnœa increased, and the boy died about six weeks later. Post-mortem examination revealed general dry pericarditis, with great thickening, and nearly complete adhesion.

to be subacute, persistent, recurrent, and progressive; going on, not for days only, but for weeks or months; the inflamed membrane slowly or intermittently exuding, not serum, but adhesive lymph, causing adhesions more or less complete, and development of fibrous tissue, so that the pericardium becomes thickened, sometimes enormously, as in the specimen I show you. This form of subacute progressive pericarditis might almost be called 'nodular,' so frequent is the evolution of these significant bodies associated with it. Their eruption appears to have a deep meaning; when they are numerous and large, they indicate a condition so grave, and apparently often so uncontrollable, that, as I said before, I have come to regard them as almost a sentence of death.

What is the pathological relation of the fibrous nodule to the fibrosis of the pericardium? It would seem as if the same virus or influence which stirs up the inflammatory change in the fibrous tissues of sheaths and tendons, and which results in the formation of nodules, stirs up in the same way fibrous organisation of lymph or proliferation of the fibrous tissue of the pericardium. Dr. Barlow found in one case that the pericardial adhesions had a distinctly nodular character;[1] and Dr. Angel Money,[2] in a similar case, with extreme pericardial adhesion, found a distinct nodule extending from the pericardium inwards, and invading the heart's substance. I believe that the fatal issue is largely dependent upon

[1] *Brit. Med. Jour.*, Sept. 15, 1883, p. 511. [2] *Ibid.*

FIG. III.



From a Sketch by Dr. A. W. Sanders.

Microscopic section of the pericardium in acute pericarditis.

1. The fibrinous exudate.
2. The layer of cellular exudation.
3. The swollen fibrous tissue of the visceral pericardium.
4. The myocardium.
5. Numerous capillaries.

the tightening grip of the adherent contracting thickened pericardium, strangling, as it were, the muscle and muscular movements of an often already crippled heart; possibly also the heart-muscle is weakened by fibrous interstitial invasion and by concurrent myocarditis acute dilatation results; hence the rapid pulse and the progressive feebleness of cardiac action. The effect of the continued constriction of the heart by the tough, thickened pericardial casing in early life upon the due growth of the heart itself was first brought home to my mind some years ago by the following remarkable case.

A girl, aged nineteen, was admitted into St. Mary's Hospital with general dropsy, for which no satisfactory cause could be made out. She had no signs of either cardiac or renal disease, but she had considerable ascites and hydrothorax, for which she was repeatedly tapped. At last she died. On post-mortem examination, the heart was found tightly enclosed in a bag of dense unyielding fibrous tissue, the adherent pericardium enormously thickened; the heart itself was as small as that of a child of ten years old; there was no other lesion of any kind. There could be no doubt that the girl had had plastic pericarditis as a child with subsequent fibrosis, and the embarrassed heart, tightly embraced by the contracting tissue, could not grow; at length, becoming unequal to carry on the circulation of the enlarging vascular system, dropsy followed. Sir T. Watson[1] observed a similar result. He says:

[1] *Principles and Practice of Physic*, 4th ed. vol. ii. p. 301.

'When the adhesion takes place during youth, as in connection with acute rheumatism it is very apt to do, it seems to prevent the further growth of the heart, and virtually leads to atrophy of that organ or a disproportionate smallness of its cavities, vessels, and general size'; and Bouillaud mentions the case of a woman, aged sixty-one, whose heart was the size of that of a child of ten or twelve, and marked by furrows and opaque spots from old pericarditis.[1] Usually, however, in the case of children, the pericarditis persists or recurs, or it comes late, when the heart is already dilated and hypertrophied from valvular disease, and the new clog, the tightening grip of the pericardium, is more deadly than it would be to the normal organ. The heart is not small, but enlarged, and although one may believe that its further development might be interfered with, yet death usually takes place before the disproportion between heart-growth and the development of the rest of the body has become conspicuous or extreme.

Myocarditis.—In some cases of acute rheumatism that have ended fatally, extensive changes have been found in the myocardium. The late Dr. Sturges attached much importance to the widespread inflammation of the heart that occurs in childhood, and called the condition a rheumatic carditis.[2] It is uncertain at present to what extent this myocardial inflammation occurs, and

[1] Payne, Jones, and Sieveking's *Pathological Anatomy*, 2nd ed. p. 344.

[2] Heart inflammation in children.—Dr. O. Sturges, Lumleian Lect., 1894.

whether it is coincident with or secondary to the pericarditis. Cellular exudation can be most frequently demonstrated immediately beneath the pericardium, but there is some evidence to show that the changes in the heart wall may be widespread and occur independently of pericarditis. In those rare cases of acute rheumatism in which death occurs with cardiac dilatation, but without pericarditis, fatty changes have been found in the myocardium. Similar changes have been found in cases of acute pericarditis, and they may be widespread. In cases of recurrent rheumatism recent foci of inflammation have been found in the heart wall, when the pericardium itself has been in a condition of sclerosis from the previous attacks, and the vessels in the myocardium sometimes show a fibrosis around their walls which is probably the result of the previous rheumatic attacks. It is possible that the clinical importance of these myocardial changes is very great, though this must greatly depend upon the extent to which the rheumatic poison acts specifically upon the cardiac muscle.[1]

Endocarditis.—Endocarditis occurs acutely in the course of articular rheumatism in children, just as it does in adults. The position of endocarditis as one of the rheumatic inflammations is like that of pericarditis, established and allowed.[2] Yet in childhood it

[1] A contribution to the microscopy of rheumatic heart disease.—Dr. Poynton, 1899; *Royal Medico-Chir. Trans.*

[2] How close the association between endocarditis and the rheumatic state appears from the following data furnished by my note-book of private cases. In ninety-four consecutive cases, in which the history was accurately ascertained, which have been analysed for me by my

often occurs quite apart from any concurrent affection of the joints; and it may develop at any period in the rheumatic procession of events, early or late, in combination with arthritis or pericarditis, or chorea, or nodules, and with any or all of these combined. Usually it comes early in the series, and recurs later, in contrast to pericarditis, which is more apt to appear towards the end. An example of this is given in Series II., where it came first. The rule is: endocarditis early; pericarditis late. But as with pericarditis, so with endocarditis; the acute form in connection with severe articular rheumatism is far less common than one slight and trivial apparently at the time, accompanying an equally slight articular affection or chorea; all sign of it perhaps disappearing for a season, yet recurring and persisting until the injury to the valve becomes serious and finally fatal. It is not, as it is so commonly with adults, a sharp attack of endocarditis accompanying the articular affection, ceasing with it, and followed slowly by chronic afterchanges in the valve or muscle; but subacute, insidious, progressive.

In the first lecture I endeavoured to show how an extremely slight and transient arthritis, hardly noticeable, not recognised as rheumatic, probably not coming under a doctor's observation at all, might be accom-

friend Dr. Poynton, in sixty-two (*i.e.* in 66 per cent.) there was a definite history of acute rheumatism in either the patient or in immediate blood relations. If chorea and morbus cordis are accepted as evidence of rheumatic taint, the numbers are seventy-three out of ninety-two, or 77 per cent.

panied by valvulitis, running on unsuspected at the time, and only discovered afterwards when great cardiac changes have developed. Dr. Sibson, whose name will always be honoured in the Harveian Society, affirmed that the more severe the rheumatic attack, the greater the tendency to cardiac inflammation. Probably this is so. Severe attacks of articular rheumatism are, however, rare in young children; and, light as the arthritis usually is, children are nearly twice as liable to cardiac inflammation as adults.[1]

Valvular disease of the heart in childhood is not invariably the result of rheumatic endocarditis. There are certain exceptions. We may exclude the cases of congenital disease and those where the endocarditis is set up by one of the specific fevers, or the septic or uræmic poison. There will remain for consideration three classes—viz. (1) those cases in which the valvular affection arises in association with distinct attacks of articular rheumatism; (2) those associated with chorea in which no articular affection is observed; and (3) those in which no connection with any other morbid condition of any kind can be traced—what may be called the 'unexplained cases.'

Now, with regard to the first class, cases of heart affection connected with articular rheumatism, the organic change would, I think, be generally allowed to be due to rheumatic endocarditis. And these form the largest proportion. Dr. West[2] gives statistics of 135

[1] *Collective Investigation Record*, vol. iv. p. 71.

[2] *Diseases of Infancy and Childhood*, 7th ed. p. 553.

cases of organic valvular disease, of which in 60 per cent. rheumatism was 'either known or asserted on good grounds to have been the starting-point of the mischief'; and he quotes the estimate of M. Roger as 78 per cent., and that of M. Cadet de Gassicourt as 81 per cent. Dr. Goodhart's statistics yield 62 per cent.[1] My own statistics of 195 cases, specially examined and investigated for the purpose, including the evidence of rheumatic fever in parents or immediate blood-relatives, give 79 per cent.[2] All these are based upon the occurrence of articular rheumatism only, and the absence of evidence of this in any given case is not to be taken as positive proof of its non-rheumatic nature.

The only question which arises, then, is with regard to the two remaining groups, those associated with chorea alone, and the group of unexplained cases. Are these the result of rheumatic endocarditis? Taking the latter first, their number is not large. Dr. Goodhart, out of 248 cases of heart disease, found all but 55 associated with either rheumatism or chorea.[3] If from this remainder we take the congenital, septic, uræmic, and fever cases, there are few left unexplained, and of these few many are probably rheumatic, where slight arthritis has been overlooked or been absent. There remain, then, practically only the choreic cases —*i.e.* the cases where organic heart disease arises in connection with chorea, but is not known to be associated

[1] *Diseases of Children*, pp. 528–9. [2] *Lancet*, October 31, 1885, p. 795.
[3] *Diseases of Children*, p. 529.

with any manifestation of articular rheumatism. Are these to be included as examples of rheumatic endocarditis? In my judgment most of them—possibly all. The antecedent probability is largely in favour of their rheumatic origin. My reasons are these. The cardiac affection of chorea is, as a rule, organic, not merely functional. Functional murmurs do no doubt arise occasionally in chorea—*e.g.* the ordinary hæmic murmur of the pulmonary orifice. But that the mitral murmur of chorea is not usually of functional origin seems to be established by the following considerations:—

1. If the mitral murmur were what is called hæmic, it is strange that in this particular disease the mitral valve should be the chief seat of it, such functional disturbance being in other conditions usually associated with the pulmonary and aortic orifices. There are, no doubt, functional mitral murmurs due to dilatation, but if a functional mitral murmur did exist, *à fortiori* a pulmonary one should exist also, but it does not. Moreover, the mitral murmur of chorea comes early as a rule before anæmia and debility arise.

2. The hypothesis that the mitral murmur of chorea is commonly spasmodic or paretic, as suggested by Dr. Sturges, although plausible and ingenious, is a pure hypothesis, unsupported by positive evidence. It is rendered improbable by the fact that, as a matter of clinical observation, the murmur does not arise especially in connection with irregular action of the heart, as I have repeatedly satisfied myself; and, more-

over, as Dr. Osler[1] points out, the general immunity of involuntary muscle in all choreic disturbance is against the theory of spasm; while, as to paresis, the murmurs have no special association with paretic chorea.

3. In fatal cases of chorea endocarditis is almost invariably found *post mortem*. There is no other disease in which it is so constant.[2]

4. In fatal cases of valvular disease arising in association with chorea, the changes found in the valves *post mortem* are identical with those from endocarditis from other causes, while the valve chiefly affected is the mitral valve, the one most liable to endocarditis.

These considerations are borne out by careful clinical observations, such as those of Dr. Stephen Mackenzie and Dr. Osler. The former found the murmur of chorea persistent in 60 to 80 per cent. from one to five years afterwards.[3] Dr. Osler, independently pursuing a similar inquiry, comes to a like result. In 110 cases of chorea examined two years afterwards, organic heart disease was found in fifty-four.[4] A large proportion of choreic murmurs, therefore, persist or reappear, and in some instances do not develop until after the chorea, when all motor and paretic disturbance has long ceased.

In a small proportion of cases the mitral murmur of chorea is probably atonic or functional without

[1] *American Journal of Medical Sciences*, vol. xciv. new series, p. 374.

[2] *Ibid.* p. 375.

[3] *Trans. Int. Med. Cong.* 1881, vol. iv. pp. 100, 104.

[4] *American Journal of Medical Sciences*, vol. xciv. new series.

valvular lesion, due to dilatation and imperfect closure such as does undoubtedly occur in acute rheumatism, and also in chorea, according to the recent researches of Dr. Lees and Dr. Poynton.[1]

The valvular affection of the heart, then, in chorea is, in the vast majority of cases, organic, due to endocarditis or myocarditis. Is the carditis rheumatic? The evidence in favour of this is extremely strong. First, there is the frequent association of articular rheumatism with chorea and endocarditis or dilatation; secondly, the frequent association of articular rheumatism with the two independently, with chorea on the one hand, and with endocarditis and dilatation on the other; thirdly, the especial association of endocarditis and dilatation with those cases of chorea which are also associated with articular rheumatism. The great proportion of cases of endocarditis which arise in connection with chorea arise in chorea connected with articular rheumatism. Endocarditis picks out, so to speak, the rheumatic cases. In eighty-four cases of chorea of which I took precise notes there was a history of articular rheumatism in either the patient or near blood-relatives in sixty-two. In the remaining twenty-two there was no such history. Of the sixty-two cases of chorea with rheumatic history, forty-three, or 69 per cent., had organic heart disease. In the twenty-two choreas without rheumatic history there were only six cases of organic heart disease, or 27 per cent.[2] The statistics of the Collective Investigation Committee tell

[1] *Med. Chir. Trans.* 1898. [2] *Lancet*, October 31, 1885, p. 794.

the same story. They yield 50 per cent. of heart affection in choreas with rheumatic history, as against 35 per cent. in those without rheumatic history; and these statistics, as stated, necessarily give too low an estimate of the rheumatic influence, since the only test of rheumatism admitted is the antecedent or concurrent association of articular affection. Yet the evidence afforded by nodules, by pericarditis, and by erythema exudativum is of considerable weight. How suggestive, for example, are cases of chorea such as that of the child I exhibited at the last lecture, who has a plentiful crop of nodules accompanying endocarditis, yet in whom there is no history of articular affection at any time, either in the patient or her family; [1] and that of the girl under my care at St. Mary's Hospital,[2] who came in with chorea, and afterwards developed pericarditis, with only the slightest and most transient articular affection, lasting no longer than twenty-four hours. Lastly, there is the indirect evidence afforded by the significant fact to which I drew attention in the last lecture—namely, the singular harmony which exists between the incidence of articular rheumatism, of chorea, of rheumatic chorea, and of choreic heart disease, upon the sexes

[1] Sarah M——, admitted to the Hospital for Sick Children, November 15, 1888, under Dr. Barlow. Since the above was written a very significant series of events has occurred—namely, acute tonsillitis, followed after an interval of a fortnight by pain, swelling and tenderness of one small joint only, that of the right middle finger; this lasted two days, and then disappeared. On the same day pericardial friction was discovered, and still continued when this note was taken a week later.

[2] Kate B——, Albert Victor Ward, January 1889.

between the ages of eleven and fifteen. If the statistics are to be trusted, girls are from two to three times as liable as boys to articular rheumatism, to chorea, to rheumatic chorea, to heart disease in rheumatic chorea, and to heart disease in chorea where there is no history of rheumatism. And apart from all direct evidence of rheumatism, when endocarditis appears in association with chorea, in view of the fact that rheumatism is the chief cause of endocarditis on the one hand, and that it is the only disease closely associated with chorea on the other, is it not more reasonable to suppose, when endocarditis and chorea occur together, that rheumatism is at the root of both, rather than that the valvular inflammation is set up by disordered muscular action, or that it depends upon some mysterious condition connected with chorea of which we know nothing?

It has been suggested that strain, or shock, or violent action of the valves is the source of choreic endocarditis. This is, I think, untenable, for many good reasons. In the first place, the heart does *not* act violently in chorea, but feebly; tension is lessened, not increased. Secondly, in diseases such as exophthalmic goître, where the valves flap to with great force, valvulitis is not thereby set up, although a functional murmur is sometimes developed. Thirdly, heart disease does not arise especially in connection with chorea associated with fright, when a sudden strain might be postulated, but in connection with chorea associated with rheumatism equally whether

fright is or is not present as a co-factor. The most cogent argument of all, however, is that drawn from pericarditis. Pericarditis arises in chorea quite apart from any articular affection as often in proportion to its general frequency as endocarditis. How is this to be accounted for? Neither the strain hypothesis, the paretic hypothesis, nor the spasm hypothesis will explain choreic pericarditis any more than it will explain choreic nodules or choreic erythema. So far the only reasonable explanation of the endocarditis of chorea, as of the pericarditis, is that it is of rheumatic origin. As Dr. Barlow has surmised, and as I shall endeavour to demonstrate presently, the active morbid change in the cardiac valves in subacute endocarditis appears to be analogous to that in the subcutaneous fibrous tissues giving rise to nodules. Such nodules are frequently developed without concurrent articular rheumatism, but with endocarditis, in the course of chorea. Is it not probable that in choreic endocarditis a subacute change of this kind goes on, and that many of the murmurs at least which come and go, or develop afterwards, are really due to this cause, rather than to functional disorder? The endocardial change may be sometimes just as passing or recurrent as we have seen the nodular development to be in many cases.

Turning now from the evidence that the endocarditis of childhood is chiefly rheumatic to the consideration of the condition in itself, I have said that it is less acute, severe, and passing than with adults; that it

is especially liable to relapse, and recur, or smoulder on for weeks or months. This is well shown in the case of W. S—— (Series VIII.), where endocarditis, beginning in 1886, occurred again in 1887, and then continued with arthritis, chorea, erythema, and nodules, pretty constantly, until the rheumatic series closed in death in March, 1888. The cases of H. W. B—— (Series I.), C. H. B—— (Series II.), and J. T—— (Series X.), afford additional illustrations.

In the case of B. H——, a boy aged six years and a half, under my care in Great Ormond Street Hospital, a similar slow recurrent endocarditis was observed to be going on. The boy never had any sign of what is ordinarily termed rheumatism, except some pains in the feet attributed to an accident, with pain in the cardiac region and sweating; but there is a history of rheumatism on the mother's side. He has a well-marked mitral regurgitant murmur and reduplicated second sound at the apex. Nodules were developed; he grew more pale and ill; had two attacks of sudden dyspnœa, with signs of circumscribed pneumonia, evidently embolic. Fresh nodules continued to appear; the murmur grew harsher and more musical. There can be little doubt that chronic endocarditis was going on, and that the injury to the heart-structures was progressive.

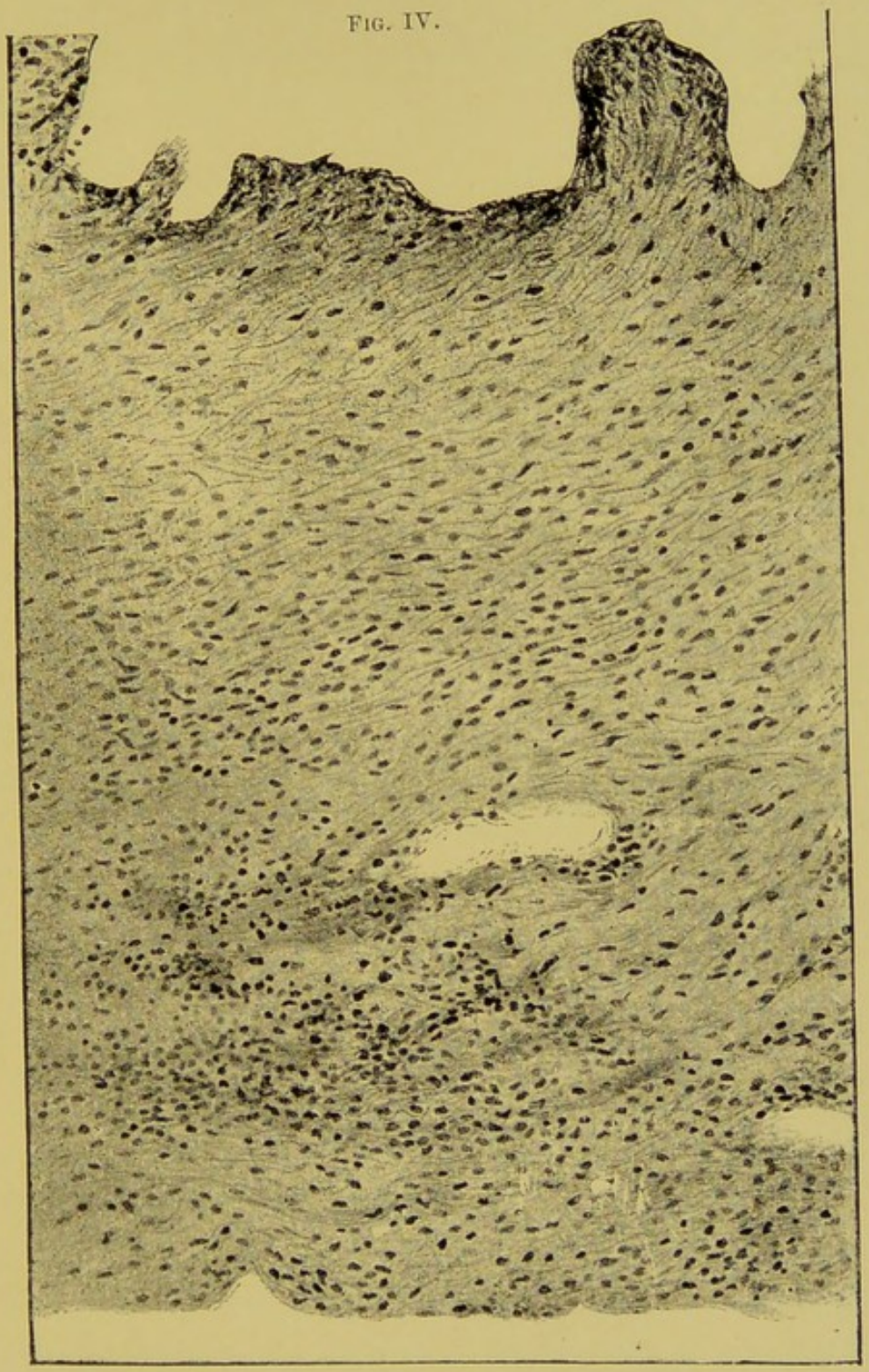
A more striking instance still is that of H. G——, a boy aged fourteen, whose case I have published before,[1]

[1] Lectures on Heart Disease in Children (*Lancet*, October 17, 1885, p. 703).

and which I shall therefore allude to only very briefly here. The patient had large nodules on the flexor tendons of the palms and fingers, accompanied by subacute recurrent endocarditis, which had probably commenced with an attack of slight arthritis about a year previously. The joint rheumatism had received no serious attention; the heart was not examined. The boy continued to follow athletic sports, with the result of setting up extreme regurgitant aortic disease and dilatation of the left ventricle. It is interesting to note, in passing, that the boy's sister had had chorea without any articular rheumatism; his grandmother, rheumatic fever.

The eruption of subcutaneous nodules is associated with grave progressive endocarditis as closely as with progressive pericarditis; and I think an examination of the pathological process going on in the valves in endocarditis of this kind explains the connection. The inflammatory process in the valves, as Dr. Barlow ventured to anticipate seven years ago, appears to be identical with that met with in the nodules—viz., fibrinous exudation, cell infiltration, spindle cells in process of transformation into fibrous tissue, wavy bands of fibres, and vessels. The microscopic appearances of a thin section of the mitral valve of the patient, Ellen M——, are shown in the specimens exhibited, for which I am indebted to Mr. Priestley, and reproduced in the drawing (fig. IV.), and those in a subcutaneous nodule in fig. I., p. 269, and also the microscopic features as observed more recently by Dr. Poynton

FIG. IV.



Microscopic appearances in a section of the mitral valve in a case of rheumatic endocarditis in a child, showing proliferation and cell-infiltration of sub-endothelial fibrous tissue. (Ellen M——, aged eight years and a half; Hospital for Sick Children, Great Ormond Street; Dr. Barlow; March 28, 1888. Arthritis, nodules, morbus cordis, purpura.)

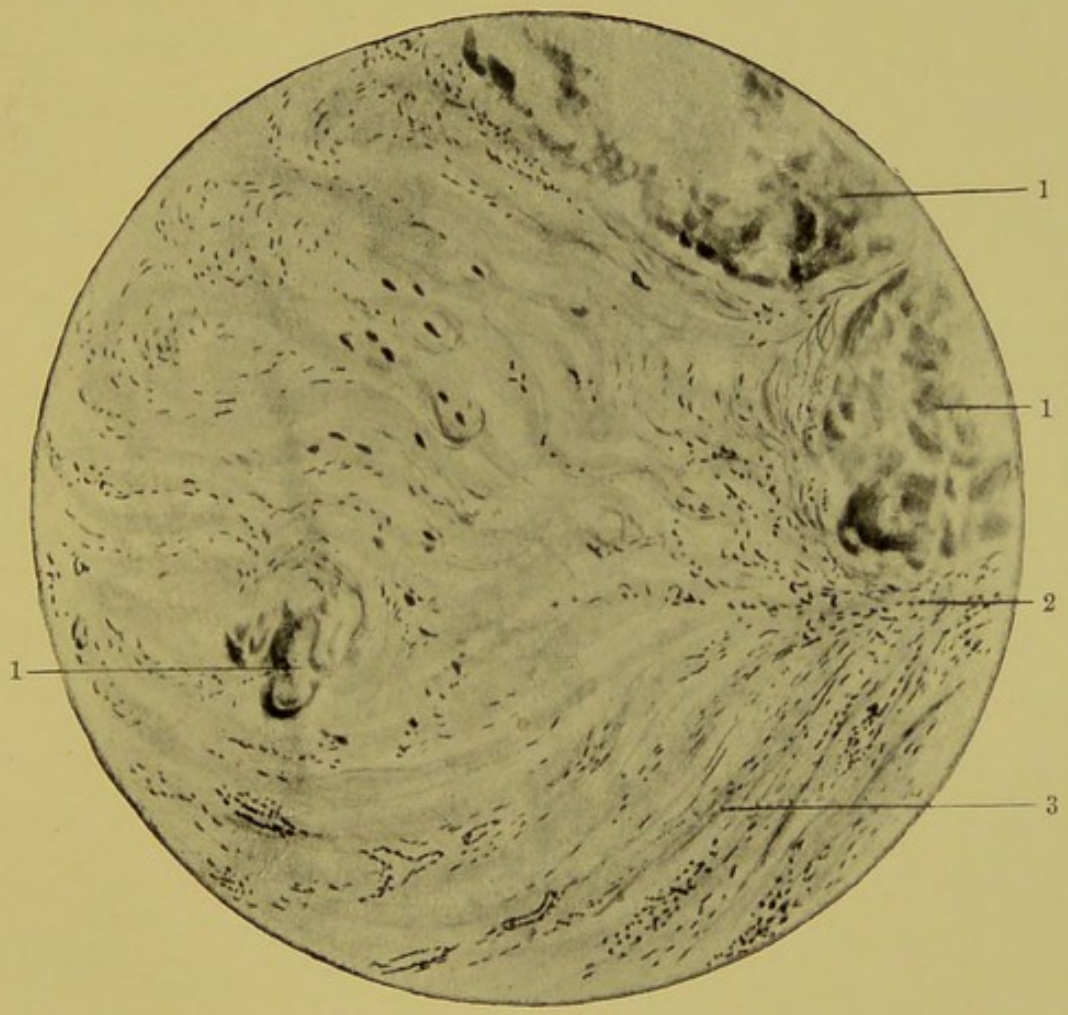
and Dr. Still, as shown in figs. IV. and II. p. 270. The proliferation and cell infiltration of the fibrous tissue or fibrinous exudation into it, which forms the framework of the valve is the most conspicuous feature, and it is to the infiltration of this layer that most of the swelling is due. There is, moreover, infiltration of the whole thickness of the valve with round cells, or leucocytes. In some specimens there can be seen distinct proliferation of the endothelium, and in some a deposit of fibrin on the surface in which leucocytes are visible. But the chief change—the greatest and most important change, that which causes the thickening and rigidity—is the proliferation of fibrous tissue, which is the leading feature of the morbid process seen in the subcutaneous nodule. Now, looking at the affection of the fibrous tissues of the joints, of the subcutaneous fascia, of tendons, of the pericardium, and of the endocardium, it appears that the *materies morbi* of rheumatism, whatever its nature, sets up similar irritant or inflammatory exudation changes in fibrous tissues in all these parts. But since the nodules are rarely found in adults although so common in children, and that endocarditis and pericarditis are likewise more common in children, while arthritis, on the other hand, is less severe, it would appear further, that in early life the fibrous tissues of subcutaneous fasciæ, of tendons, of pericardium, and of endocardium are far more sensitive to the morbid stimulus, more easily excited to proliferation, than the same tissues in later life; while, on the contrary, the joint fibrous tissues are less susceptible to

the poison in children than in adults. It seems clear, moreover, that the susceptibility of subcutaneous fibrous tissues and the susceptibility of the fibrous tissues of endocardium and pericardium go together; and this circumstance, as suggested before, together with simple dilatation, afford a possible clue to the nature of the evanescent mitral murmurs of rheumatic arthritis and chorea. The rapid development of the nodules, in some instances in the course of a few days even, and their equally quick disappearance on occasion, suggest that the murmur which arises and disappears, and which is styled functional, is sometimes caused by the development of similar inflammatory cell-growth in the valves and chordæ, which may subside in them as quickly as we see it does in a subcutaneous nodule, or by acute dilatation. The correlation of nodules and valvulitis gives the appearance of the former great clinical significance, as probably indicative of a similar change going on in endocardium or pericardium, or both. When the nodules are few or small, they must be regarded as serious signs; when large and numerous, they are grave warnings of the existence of a condition of ultimate danger.

Ulcerative endocarditis is most rare in the rheumatism of children, since it is met with chiefly in patients broken down by drink and disease. The earliest age at which I find its occurrence recorded is in a patient of my own at the Children's Hospital, Annie I——, a girl of eight.[1] Senator observed it in a boy

[1] *Post mortem Book*, vol. iv. 1887.

FIG. V.



From a Sketch by Dr. A. W. Sanders.

Microscopic section of a portion of the mitral valve from a case of acute endocarditis. The valve had been deformed by a previous rheumatic attack, and the section has cut it obliquely. The vegetations (1, 1, 1, in the fig.) appear from this account to be imbedded in the substance of the valve.

1. Fibrinous exudate.
2. Layer of cellular exudation.
3. Swollen fibrous tissue.

of fourteen, and Dr. Ord in a girl of sixteen. It possesses no special features which require notice here.

Rheumatic endocarditis in children, as in adults, attacks mitral, aortic, and tricuspid valves, the former by far the most commonly ;[1] mitral regurgitation is the most usual resulting lesion, although mitral stenosis is far more frequent than is generally supposed. It is in the latter that the chief interest centres.

It is well known that mitral stenosis is met with most frequently in young women. Of 263 cases collected by Sir Dyce Duckworth,[2] there were 177 females and 86 males. And this relation between the sexes probably holds good in the case of children, in accordance with the great preponderance of heart disease in girls between eleven and fifteen. Mitral stenosis is in its origin especially a lesion of early life. I find thirty-three cases with presystolic murmur and twenty-four with reduplicated second sound at the apex, indicating commencing stenosis, out of 273 cases of organic heart disease in children of which I have accurate record. The youngest patients were two boys of four. Yet the age of the youngest patient in Sir Dyce Duckworth's list was fourteen. Dr. Fagge had no instance under ten ;[3] Dr. Hayden two only of seven years old.[4] In the analysis of 150 fatal cases by

[1] In Dr. Poynton's analysis of 150 cases of fatal rheumatism the mitral valve was affected in 149 ; the aortic valve in 51 ; the tricuspid valve in 36 ; the pulmonary valve in 3 (*Med. Chir. Trans.* 1898, Appendix C, p. 443).

[2] *Etiology of Mitral Stenosis* (pamphlet).

[3] *Guy's Hospital Reports*, Series 3, vol. xii.

[4] *Diseases of the Heart* (Dublin, 1875) p. 964 *et seq.*

Dr. Poynton referred to before, marked mitral stenosis is only stated in nine cases. This is partly explained probably by the fact that permanent stenosis is a gradual change, and any *marked* degree of it would not in most cases appear until early adolescence. The presystolic bruit and reduplicated second sound in the early stage indicate a stenosis from rigidity and imperfect opening only, which may be but a temporary change.

It was formerly taught that mitral stenosis is less often rheumatic than any other valvular affection. As a matter of actual fact, this form of heart disease—in children at any rate—is especially rheumatic, and because it is so associated with the rheumatic endocarditis of childhood I lay stress upon it here. Dr. Sansom found 50 per cent. with a history of articular rheumatism or of pains.[1] Sir Dyce Duckworth's cases yield 60 per cent. My own statistics are still more remarkable. Taking children alone, I find this striking evidence of its rheumatic relation. Of fifty-seven cases of presystolic murmur, in forty-four there was acute rheumatism in the patient ; in twenty of these forty-four in the family also; in four more in the family alone, leaving nine only in which there was no rheumatic history. Putting out of the question family history then, there was personal history of rheumatism in forty-four out of fifty-seven, or 79 per cent.

It is in the period of childhood that mitral stenosis most commonly commences. The reason why it has not been more frequently observed is perhaps due to the fact that it is at this period usually in the initial

[1] *Lettsomian Lectures*, 2nd ed. p. 130.

stage. It is, as Dr. Sansom has shown,[1] the special product of the slight, subacute, slow, recurrent rheumatic endocarditis which is characteristic of childhood. The stenosis does not generally reach the degree when it is proclaimed by the loud, vibrating, prolonged presystolic murmur, until a few years have passed. The slight rumble, or the reduplicated second sound, escapes notice, or attention is drawn away from it by an accompanying regurgitant bruit. Hence it is discovered later, with startling frequency, just after childhood. Cases are met with in which this condition is found to exist without any rheumatic attack to explain it. In these instances I believe it has had its first origin in childhood, arising unnoticed with a slight arthritis, or a crop of nodules, or a chorea, or alone, as the sole manifestation of a rheumatic outburst.

The earliest sign of mitral stenosis is reduplication of the second sound *at the apex*. Dr. Sansom has observed generally,[2] that 'reduplication of the first or of the second sound is an early sign of stenosis.' But I would limit the statement to reduplication at the apex audible over the mitral area only, or at least having its maximum intensity there. Dr. Sansom gives two cases[3] in which this local limitation was observed, but he does not differentiate them from the rest. Now, although reduplication of both first and second sounds, audible over the aortic and pulmonary valves at the base of the heart, occurs in certain cases of mitral

[1] *Lettsomian Lectures*, 1886, p. 80. [2] *Ibid.* p. 22.
[3] *Trans. Med. Soc.* vol. v. p. 204.

stenosis, it is not this with which I am concerned; that is not an early sign according to my experience. It is reduplication *at the apex* which is the significant early sign; basic reduplication usually comes later, with a pronounced presystolic murmur or with mitral regurgitation, and it is explained readily enough by the difference in tension in the pulmonary artery and aorta, causing asynchronous closure of their respective valves. It appears, however, to be generally assumed that this asynchronism of the pulmonary and aortic valves will account for *all* reduplication. But it certainly entirely fails to explain reduplications audible only at the apex. If the doubling of the sound were caused by the asynchronous closure of the aortic and pulmonary valves, it would be audible over their position—*i.e.* at the base of the heart—but it is not. Dr. Sansom saw this difficulty and to him the credit of a more correct explanation is due. He suggests that the *first* of the two second sounds is the normal one caused by the simultaneous closure of the pulmonary and aortic valves, and the *second* by the sudden tension of the mitral flaps as the ventricle relaxes. The blood in the auricle, in a state of increased tension, drives open the mitral door, and, rushing round the sides of the ventricle, gets underneath the curtains—the anterior one of which, as Dr. Macalister has shown, is stretched tight from the basal ring to the top of the papillary muscle—and thus gives rise to a sound of tension.[1] This explanation is, I think, correct in the main—so

[1] *Lettsomian Lectures*, 2nd ed. p. 123.

far as this, at least: that one of the two second sounds —viz., the second—is mitral in origin. It is not audible at the base; only *one* sound is audible over the aortic and pulmonary area, while *two* are audible over the mitral area. The one which is heard over the mitral only must be produced there. It seems to me that the most probable explanation is that the *first* of the two second sounds is the normal one caused by the click of the basic valves, as Dr. Sansom suggests, the pulmonary element being accentuated and rendered louder by increased resistance in front. The *second* of the two second sounds is due to the smack or click of the forcible opening of the swollen, rigid mitral with shortened chords, kept closed with difficulty by the contracting ventricle, and springing back suddenly as the ventricle relaxes and exerts its suction power, aided by the increased tension in the left auricle, rather than by the auricular contraction, which occurs later. The suction power of the relaxing ventricle has been shown by Marcy and Fick, and Goltz and Gubler, to be considerable at the beginning of diastole, possibly as much as 23 millimeters of mercury, independent of respiration.[1] This view is supported by the fact that, after the *second* of the two second sounds—*i.e.*, the one which Dr. Sansom and myself take to be the *mitral* sound—there is in some instances a soft bruit, a distinct diastolic murmur, probably due to the rush of blood through the narrowed aperture caused by the auricular contraction which comes, not at the very beginning of

[1] Macalister: *Brit. Med. Jour.*, October 28, 1882, p. 123.

diastole, but a moment after its commencement. Reduplication of the second sound at the apex, then, is, I think, the earliest sign of swelling and rigidity of the mitral flaps, and consequent imperfect opening of the valve. Whether the explanation I have given be accepted or not, there can be no question as to the connection of this morbid sound with early mitral stenosis, and of its clinical significance.

Hypertrophy and Dilatation.—These changes proceed more rapidly in children than in adults. The first develops apace, in accordance with the general law before mentioned—that the tissues respond to stimulation and grow more rapidly in childhood; and the second—dilatation—perhaps because the tissues are more soft and yielding. Look at the case of Harold G——, with acute regurgitation previously narrated. The first attack of acute rheumatism was only sixteen months before I saw him, and he had already enormous enlargement—chiefly dilatation—of the left ventricle. Observation of these cases in hospital show that they advance at a rate unknown in later life.

The following is an example of rapid dilatation. George B——, aged six, was admitted into Great Ormond Street Hospital on April 29, 1879, with general cardiac dropsy. He had had scarlatina with articular rheumatism six months before. There was a soft systolic murmur audible at the apex. After death the heart was found to be somewhat hypertrophied and still more dilated, weighing seven ounces—as large as that of a child of eleven or twelve. It is worthy

of note that, although there was a distinct systolic mitral bruit heard during life, there *was no valvular lesion of any kind, except dilatation* of the orifice ; and no history of any rheumatic affection previous to the scarlatina.

Simple Acute Dilatation.—In discussing the nature of the heart murmurs in chorea I stated that in a small proportion of cases their mitral murmur might be due to imperfect closure of the orifice in consequence of atonic dilatation. The case of George B——, just given, where the fatal heart failure and dropsy which accompanied it was found to be due to simple dilatation without actual valvulitis, is an example of this. The recent important observations of Dr. Lees and Dr. Poynton,[1] previously alluded to, seem to establish the fact that simple acute dilatation is common in rheumatism and chorea, and is probably the earliest indication of rheumatic heart affection—the signs of it being increased quickness and feebleness of pulse, increased area of cardiac dulness, especially towards the left, and sometimes a faint, soft, systolic bruit at the apex, with accentuation of the pulmonary second sound.

As hypertrophy develops rapidly in children, so compensation is usually for a time exceptionally complete, and, as a further result of this effective compensation, great enlargement of the liver and spleen, pulmonary apoplexy, and extreme dropsy are rare in the younger children, becoming more common as age advances.[2] It is an unusual thing to see a little

[1] *Med. Chir. Trans.* 1898.

[2] In the 150 fatal cases collated by Dr. Poynton dropsy was described as

child blue, turgid, and waterlogged—a sight so sadly frequent in the last stage of mitral disease in adults.[1] Dr. Goodhart has observed these facts, and attributes them to reduction of the whole blood supply as part of the general wasting, which is so conspicuous a feature. This probably plays some part in addition to efficient compensation. But there is still another reason. Children with severe heart disease, as a rule, die from other causes before the stage of grave tricuspid leakage is reached. For in children the mode of death from mitral disease differs from that we see in later life. Instead of engorged liver and lungs, with blueness, extreme dyspnœa, and general dropsy, there is rapid wasting, progressive anæmia, feebleness, and death from asthenia, due to the enfeebled blood supply from the dilated heart and weakened muscle, or to the strangling grip of pericardial exudation and adhesion.

Scarlatinal Rheumatism.—The account of the various phases of rheumatism would be incomplete without some reference to the scarlatinal affection. An articular inflammation appears now and again in the course of scarlet fever, which can in no way be distinguished from that of acute rheumatism. It is often accompanied by endocarditis or pericarditis, and

'much' or 'considerable' in twenty-five, but in only eight had this dropsy been of long standing (*Med. Chir. Trans.* 1898, Appendix C, p. 443).

[1] In cases where there was considerable dropsy, there was almost invariably more valvular disease than is the rule in the fatal rheumatism of childhood (Dr. Poynton; *Med. Chir. Trans.* 1898, Appendix C, p. 443).

sometimes by chorea. Henoch[1] records a case in which acute arthritis appeared in the first week, followed by mitral murmur and chorea. This scarlatinal rheumatism, although it may come late, in most cases arises early; Dr. Barlow noted it as early as the third day.[2] In a series of cases observed by Dr. Ashby, of Manchester, the symptoms supervened with great regularity at the end of the first week. The endocarditis or pericarditis which comes late in the course of scarlatina are possibly uræmic; but uræmia does not set up inflammation of joints. Dr. Ashby inclines to the view that the arthritis is of septic origin, due to foul throat, or otitis, or empyema, and not a true rheumatism. The theory of septic poisoning, indeed, carries us a step further than the uræmic, for septicæmia produces arthritis as well as pericarditis and endocarditis; and septicæmia does undoubtedly arise in scarlatina in the way mentioned, as I have seen more than once. But then the articular affection of scarlatina is not especially associated with bad throats, or otitis, or empyema, or with other signs of septicæmia; moreover septicæmia will hardly explain early scarlatinal rheumatism, or the occasional concurrence of chorea. It seems clear that either genuine acute rheumatism does occur in the course of scarlatina, or else that the scarlatinal virus itself occasionally produces an inflammation of joints and serous membranes, and a nervous choreic disturbance analogous

[1] *Diseases of Children*, p. 30 (*Eng. Trans.* Sampson Low, 1883).
[2] *Brit. Med. Jour.*, September 15, 1881, p. 510.

to and indistinguishable from that set up by the rheumatic poison. It is curious to observe, too, as previously mentioned, that the especial liability to acute articular rheumatism, which is so marked in girls, extends to this scarlatinal form, as shown by Dr. Gresswell; the numbers being twenty girls to nine boys, the inequality between the sexes being most marked after ten years of age.

Rheumatoid Arthritis.—I had it in mind when I planned these lectures to say something of rheumatoid arthritis. But the shortness of time and the length of the subject prevent my finding place for it. I will merely say that, although it is uncommon in childhood, it does occur, and, as Sir A. Garrod long ago pointed out, sometimes in its most severe and intractable form. The disease in children appears to be due to the same causes as in later life, and to differ in no material respect from the type with which we are familiar in adults.

Treatment.—Almost the whole interest and importance of rheumatism in children centres in the cardiac inflammation and its results. The one great aim and object of treatment should be to minimise this danger as much as possible; and the first point which I would urge is the necessity of being constantly on guard against an insidious attack of endocarditis or pericarditis. I have shown how it may accompany the slightest articular affection—may even arise without accompanying arthritis of any kind; associated with chorea, or an exudative erythema, or an eruption of subcutaneous nodules; or it may arise apart from any

known rheumatic phase with an indeterminate febrile attack of apparent insignificance. It is essential, then, in children especially, to examine the heart carefully in every case of the slightest articular affection—even a stiff neck or a stiff knee; and in chorea, in tonsillitis, in erythema, in an evolution of nodules, and, indeed, in every pyrexial condition of every form. Whenever there is suspicion of rheumatic inflammation—even if no cardiac affection be perceptible—enforce absolute rest in bed. Complete physical repose and external warmth are of the first, possibly of vital, importance. Cases of slight rheumatism are, as a rule, treated far too lightly by both parents and doctors. What a vast difference it would have made in the future condition of the schoolboy, H. G——, for instance, if, instead of being allowed to follow athletic sports, and thus strain his heart's muscle and court fresh chill, he had been kept secure in rest and warmth. Dr. Sibson found that a much larger proportion of cases—more than two to one—treated by absolute rest escaped permanent heart mischief than those allowed free action.

With regard to drugs I may point out that the heroic treatment by large and repeated doses of salicylate of soda is rarely called for in the rheumatism of children, since the articular affection is usually slight, and the pyrexia as a rule not severe. Salicin may be given in place of salicylate of soda in most articular cases with advantage, as being less depressant—and with the salicin, alkalies. The general evidence of the statistics of the Collective Investigation Committee

supports the conclusions of Dr. Fuller and Dr. Dickinson, that cardiac inflammation is less frequent and pronounced under their influence than under any other form of treatment. It would be interesting to note now full treatment by alkalies affects the development and duration of subcutaneous nodules, a point not yet ascertained. In the case of pericarditis, however, there is one remedy which has seemed to me to have a powerful influence for good, viz., the local application of cold. We are indebted to my colleague, Dr. Lees, for a revival of this method. The application of an ice-bag to the præcordia is most beneficial and also most grateful to the sufferer. It is reasonable to suppose that as the vessels of the skin in the præcordial region communicate with those of the pericardium, the effect on the two will be the same, and the hyperæmia of the pericardium thus be reduced by the application of cold to the external surface.

My object in this course of lectures, however, has not been so much to indicate treatment as to explain and illustrate the many different phases of rheumatism, and thus lay down a solid foundation upon which the therapeutics of the affection in its varied forms may be securely established; and this I trust I have in some measure accomplished.

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