An Address

ON

ACUTE RHEUMATISM, ITS ALLIES AND ITS COUNTERFEITS.*

BY F. DE HAVILLAND HALL, M.D., F.R.C.P.,

SENIOR PHYSICIAN TO THE WESTMINSTER HOSPITAL.

It is to be hoped that the time is coming when we shall be as successful in stamping out acute rheumatism as we have been in the case of malaria and typhus fever in this country, and as it would seem we are about to be in tuberculosis. It is difficult to estimate the amount of misery that would be prevented could we but limit the spread of acute rheumatism. Under 30 years of age I should estimate that four-fifths of all the cases of valvular disease of the heart are due to acute rheumatism, and I suppose that there is no more pitiable sight in a hospital ward than a patient gradually dying from heart failure resulting from the endocarditis of acute rheumatism.

I would lay especial stress on the importance of recognizing that in children slight pains, commonly described as growing pains, an attack of tonsillitis or erythema may be the only manifestation of the rheumatic diathesis, and yet quite sufficient to set up endocarditis with all its lamentable sequelae.

The whole subject of rheumatic fever has been much clarified since its probable microbic origin has been acknowledged, and there is much evidence in favour of the *Diplococcus rheumaticus* described by Drs. Payne and Poynton as being the specific organism. Just as the discovery of the tubercle bacillus cleared up many doubts and difficulties which formerly existed in tuberculous cases, so the recognition of the rheumatic organism will enable us to form a truer conception of its nature, and we shall be able to exclude many conditions which were in the past embraced under the head of rheumatism, but which we now have every reason to believe are of a widely different pathology. Time was when the joint lesions of acute rheumatism were thought to be characteristic, but we now know that they may be simulated by pyaemic conditions—as in a case to which I shall direct your attention later on—and by gonorrhoeal rheumatism, to say nothing of gouty arthritis. In children, moreover, as I have just mentioned, the disease may run its course with hardly any, if any, joint mischief, the heart bearing almost the whole brunt of the disease. We must, therefore, regard rheumatic fever as a general disease with localization in the heart and joints, and not a local disease of the joints with symptomatic fever.

It is quite unnecessary for me to enter into a description of the symptoms of an attack of acute rheumatism. To all present I am sure that these symptoms are only too familiar, as rheumatic fever is unfortunately very common in our damp climate. I would, however, like to make a few remarks on some of the chief complications of this disease. Among these, affections of the heart take the first place. It is difficult to overestimate the frequency with which the heart is affected in cases of rheumatic fever and the evil effect this has upon the life-history of the individual. Even as far back as the time of Sir Thomas Watson it was recognized that the chance of the heart being involved during an attack of acute rheumatism, which is very great before puberty, diminishes as life advances. He writes:¹

I have known only two persons pass through acute rheumatism with an untouched heart prior to the age of puberty, and in these two I am by no means certain that the articular disease was genuine rheumatism.

Life Assurance.

The damage done to the individual is becoming more and more appreciated by assurance companies. For my part, I believe that the companies would save money if they declined to accept any applicants who had suffered from an attack of rheumatic fever. In my small book on

*The Medical Examination for Life Assurance*² I have laid down the following rules:

An attack of acute rheumatism, sufficiently severe to keep the patient in bed two or three weeks and to incapacitate him from his occupation for six or seven weeks, would require the addition of an equivalent to seven years at the age of 30, and three years must have elapsed since the attack. If there has been more than one attack, the life would not be assurable until at least ten years after the last attack. An applicant with a rheumatic history had better not be accepted under the age of 25 years.

Increasing experience in life assurance has in no way tended to diminish my fear of acute rheumatism. Indeed, I am inclined to add a rider to the above rules and to suggest that cases with a rheumatic history should only be taken for an early endowment policy.

According to Sir William Church's³ statistics, 80.55 per cent. of the cases under 10 years of age had signs of old or recent endocarditis. The percentage gradually dropped to 34.64 in those over 40, with an average percentage of 57.50—that is, his figures show conclusively that more than half the cases of acute rheumatism have endocarditis—a truly formidable indictment against the disease, and a warning not to neglect the earliest indications of acute rheumatism.

Endocarditis of rheumatic origin may come on during fetal life, and be the cause of some of the varieties of congenital heart disease. Indeed, a fetal endocardial murmur has been recognized before labour. At the Lumsian Lecture on March 25th, 1909, Dr. Norman Moore exhibited the hearts of a woman and her fetus, both showing signs of endocarditis. The mother had acute rheumatism.

It is more difficult to detect pericarditis than endocarditis, so that one need not be astonished to find that Sir William Church's statistics show that only 14.87 per cent. of men and 9.95 per cent. of women with rheumatic fever suffered from pericarditis. My late colleague, Dr. Sturges, insisted that in many cases we have to deal with a pancarditis—that is, that a myocarditis exists in connexion with the pericarditis and endocarditis, or even independently of either. It is probably to this myocarditis that we owe the sudden deaths from acute dilatation of the heart which occasionally take place during the course of acute rheumatism. The possibility of the myocardium being affected should be borne in mind during convalescence from acute rheumatism, and the patient should be kept in the recumbent position for at least two or three weeks after the temperature has become normal and the pulse quiet. Indeed, I think that to be on the safe side four to six weeks would not be too long. Unfortunately, with the demand on hospital beds, this counsel of perfection can rarely be carried out in hospitals, but more might be done to impress upon private patients the necessity of prolonged rest in the recumbent position after an attack of acute rheumatism. If this could be done much less would be seen of valvular disease of the heart.

While on the subject of pericarditis I would like to draw attention to a physical sign present at a very early stage of the disease, which I do not think has received sufficient attention—I mean cantering action of the heart; and by cantering action I understand the presence of three sounds in the cardiac rhythm. Two of the sounds being, of course, the first and second sound of the heart, and the third being due to the commencing pericardial friction, before it has sufficiently developed to give rise to the to-and-fro friction rub.

Pleurisy.

Pleurisy is another complication which I have seen from time to time during the course of acute rheumatism. The fact that it chiefly affects the left pleural cavity would seem to indicate that it arises in connexion with some cardiac infection, and, indeed, that has been my experience. I have usually seen a pleural effusion in patients who have suffered also from pericarditis. It has been stated that 10 per cent. of all cases of acute rheumatism get pleurisy, but this seems to me too high a percentage. Possibly more cases would be detected if all patients were carefully examined, but the pain attending these examinations in rheumatic subjects is often a bar to the necessary investigation.

Tonsillitis.

I think that it is now generally agreed that pharyngitis is a very common complication of acute rheumatism;

* A paper read before the Croydon Division of the British Medical Association.

indeed, it has been stated that sore throat is present in 80 per cent. of all cases of rheumatic fever. Owing, however, to the attention of the patient being concentrated on his arthritic symptoms, he will often omit to mention spontaneously the fact that his throat is sore, and this possibly accounts for the discrepancy in the percentage of cases reported by different authorities. The throat is usually affected a day or two before the onset of the articular pain, but occasionally there is no complaint of throat trouble until after the rheumatic affection has fully declared itself.

It has been suggested that the tonsils are the chief portals for the entrance of the poison of rheumatic fever, and there is much to be said in favour of this view. When sore throat is the chief feature of a rheumatic attack cardiac lesions are seldom seen, though cases do occur in which endocarditis and pericarditis are met with even when articular pains are entirely absent. Such a case I reported at the meeting of the British Medical Association in 1889, in the discussion on "Tonsillitis and its Relations to Rheumatism." The patient was a girl of 8, with a sharp attack of lacunar tonsillitis. As her medical attendant informed me before I saw the child, she seemed much worse than the throat condition would account for. On examining the heart I found pericardial friction and an apical murmur. It may be argued that the cardiac symptoms were not due to the poison of rheumatism, but arose from the absorption of a septic material from the tonsil, and that the symptoms were really of septic origin and not rheumatic.

Until we have some certain method of recognizing the *Diplococcus rheumaticus*, or whatever germ be the cause of rheumatism—and I most emphatically believe that it is a disease of microbic origin—we shall always be confronted with the difficulty of distinguishing between anomalous cases of acute rheumatism and septicaemia. In rheumatic sore throat the margin of the soft palate, the tonsils, and the posterior wall of the pharynx will be found of a deeper red colour than normal and somewhat swollen. In the more severe cases there may be some oedema of the mucous membrane and especially of the uvula.

The symptoms due to this pharyngitis are sometimes so slight that, as I have already stated, the patient will omit to mention them unless questioned on the subject, but in the more severe forms dysphagia, pain extending up to the ears, and difficulty in speaking occur.

As regards the different varieties of tonsillitis, the lacunar is the form which is most commonly associated with rheumatism.

Chorea.

Chorea has long been regarded as a manifestation of the rheumatic state. Indeed, Dr. Bright, of Guy's Hospital, as early as the beginning of the last century, clearly recognized the relationship, but it has required constant iteration to keep this fact before the profession. It has always been a matter of wonder to me how some able physicians could disregard the very strong evidence in favour of the connexion. My late colleague, Dr. Sturges, a man of a singularly well-balanced mind, did not believe in any intimate connexion between the two diseases, though he was candid enough to write as follows: "Cases in which chorea arises in the course of rheumatism are so striking and so similar that notwithstanding the comparative rarity of the conjunction it is difficult to resist the conclusion that the one affection actually gives rise to the other."⁴ When this conjunction takes place, in the great majority of instances in my experience the joint trouble has preceded the chorea, which came on either while the rheumatism was abating or during convalescence. If the chorea should make its appearance in the acute stage of the rheumatic affection, the patient is in a lamentable plight, the constant choreic movement aggravating the pain of the original disease.

As regards the way in which chorea is produced by the rheumatic poison, an analogy may be drawn between this disease and diphtheritic paralysis. We have very good ground for believing that the toxins produced by the Klebs-Loeffler bacillus give rise to the paralysis by acting on the peripheral nerves; we may therefore assume that chorea is probably caused by the toxins of the rheumatic bacillus affecting the motor areas in the brain. This is a much more reasonable suggestion than that chorea is brought about by a shower of minute emboli being carried

to the motor centres, which was the opinion at one time held.

The Skin Affections of Rheumatism.

Many cases illustrating the connexion between erythema multiforme and rheumatism have been placed on record. The varieties most frequently met with in rheumatic subjects are those known as erythema papulatum and erythema marginatum or circinatum.

The eruption of erythema papulatum consists of spots varying in size from a pin's head to that of a sixpenny piece, of irregularly rounded outline and slightly elevated. The colour is of bluish-pink; a slight haemorrhage may take place into the spots, so that when they fade they leave behind them a faint mark resembling a bruise. The eruption appears in crops.

Erythema marginatum occurs in circles of various size up to the diameter of several inches. The circle is formed by a slightly raised pink ring with a central area of a livid hue.

Erythema nodosum is far less commonly met with in the course of acute rheumatism than the varieties just mentioned. With regard to the relation of erythema nodosum to rheumatism, Stephen Mackenzie came to the following conclusions: "(1) That erythema nodosum is frequently associated with definitely rheumatic symptoms—for example, arthritis, sour sweats, sore throats, etc. (2) That heart disease (endocarditis) may arise during an attack of erythema nodosum, both in cases in which arthritis is present and in cases in which there is no affection of the joints. (3) That these conclusions justify the inference that erythema nodosum is frequently, if not generally, an expression of rheumatism even when no other definitely rheumatic symptoms are present."⁵

Urticaria may also be one of the earliest symptoms of an attack of rheumatic fever. Hence the appearance of any of the eruptions which I have just mentioned, after exposure to cold and damp, with some concomitant feverishness, should suggest the possibility that it is the precursor of an attack of acute rheumatism.

Angio-neurotic oedema is sometimes met with in rheumatic cases. This occurs as puffiness of the face, hand, or other part of the body. Pain and itching are almost absent; at the outset there may be a little pricking when the patches begin to swell. Whether it is more than a mere coincidence in the rheumatic attack has yet to be settled. A scarlatiniform rash has been described; this is probably scarlet fever with joint symptoms.

Desquamation has occasionally been noticed in rheumatic cases. Sudamina and miliaria rubra occur when there is profuse sweating. Herpes labialis and herpes zoster have been described as complications of acute rheumatism.

"Peliosis rheumatica" is the term applied to an eruption of small bright-red spots, the size of a lentil, situated chiefly upon the legs below the knees, which gradually change in colour to brown or yellow. It is accompanied by pain in some of the joints, and valvular disease of the heart has resulted from it. It is a rare disease, and its exact relation to rheumatism is uncertain. Both purpura simplex and purpura haemorrhagica have been met with in rheumatic cases.

Subcutaneous Nodules.

In Hillier's book on *Diseases of Children*, published in 1868, an account is given of a case in which subcutaneous nodules occurred, and various Continental writers have described cases in which nodules have been observed. It is, however, to Dr. Barlow (now Sir Thomas) and Dr. Warner that is due the credit of pointing out their frequency and importance as clinical evidence of the rheumatic diathesis. The paper presented by these authors to the International Medical Congress in 1881 gives an admirable account of the whole subject, and a *précis* of the 27 cases upon which the paper is based. Indeed, I may safely assert that nothing of importance on the subject has been added to their original observations. It was one of the best pieces of clinical work that was presented to the Congress.

These nodules vary in size from that of a mustard seed to that of an almond. They are, as their name implies, strictly subcutaneous, and are not connected with the skin. They are free from redness and pain, and only very slightly tender.

They most commonly occur about the joints, especially the malleoli, the back of the elbows, and the margins of the patellae. They are also met with on the pinna of the ear, the temporal ridge, the superior curved line of the occiput, the spinous processes of the vertebrae, the spine of the scapula, the crista ilii and the extensor tendons of the foot and hand. These nodules are fairly common in children, but rare in adults. As they do not give rise to pain they require to be looked for—or should I not rather say to be felt for?—as they may sometimes be more easily recognized by the touch than by the sight.

They occur discrete or in clusters and in number from one up to fifty or more. Sometimes only a single crop appears; in other cases two or three crops occur. They last from a few days to four or five months. Individual nodules greatly increased in size while under observation, and on the other hand, some subsided and then grew again. They disappear after the rheumatic attack is over. None of the nodules became bony, or infiltrated with urate of soda, or suppurated. Dr. Barlow and Dr. Warner⁶ describe the growths as "round or oval semitransparent fibrous bodies, in appearance like boiled sago grains." Microscopically they are found to be composed of "wavy strands of fibrous tissue, with caudate, spindle-shaped, nucleated cells and abundant vessels."

As regards their anatomical site, they have been found attached to tendons, to the deep fasciae, and to the pericranium. They have been found in the pericardium after death. These nodules are without doubt evidences of rheumatism; they are usually associated with the severer forms of this disease, and the more numerous and the larger they are the worse is the prognosis. In all the cases recorded by Dr. Barlow and Dr. Warner some morbid condition of the heart was present, and eight out of the twenty-seven patients died from the result of the heart disease, a clear indication of the grave prognosis afforded by the presence of these nodules. Dr. Cheadle's experience is even more unfavourable than this. He regards the eruption of large nodules as almost equivalent to a sentence of death. "They mean persistent cardiac disease, generally uncontrollable, and marching almost infallibly to a fatal ending."⁷ I think that I have now said enough to prove the great diagnostic and prognostic value of subcutaneous nodules.

Dr. A. E. Garrod⁸ has pointed out that nodules closely resembling those met with in young rheumatic patients are occasionally seen in sufferers from rheumatoid arthritis with no obvious rheumatic antecedents, and who have reached a period of life at which true rheumatic nodules are extremely rare.

A condition which seems to be intermediate between the erythematous and subcutaneous nodules has been described by Dr. Cayley⁹ under the title of Multiple Cutaneous Fibrous Nodules Associated with Rheumatism. Dr. Cayley pointed out that in his case "the nodules had all the characters of rheumatic fibrous nodules, except that they were situated in the skin as well as in the fasciae." It has been suggested that circumscribed scleroderma has a possible etiological relation to rheumatism.¹⁰

Diagnosis from Pyaemia.

One of the difficulties which occurs in the diagnosis of rheumatic fever is to distinguish it from that variety of pyaemia which comes on without any very obvious source of suppuration. A case of this kind was admitted under my care into the Westminster Hospital some years ago.

The patient was a man aged 21, who complained of joint pains and sweating. Eventually the pain became confined to the right shoulder. I had some doubt as to the correctness of the diagnosis of acute rheumatism, as the temperature chart was unlike that met with in this disease, but resembled the temperature chart of suppuration or malaria. It was not, however, until the day before the patient's death that I recognized the error in the diagnosis. At the necropsy one or two pints of a sanio-purulent fluid were found in the right pleural cavity, septic abscesses in the base of the left lung, pus in the right shoulder-joint, and diffuse intermuscular abscess in the left hand and lower end of arm.

Now that we recognize that acute rheumatism should be classed among the diseases due to microbic infection, it can easily be understood that there may be considerable difficulty at times in distinguishing it from other forms of infection. In reference to this point, Fagge and Pye-Smith¹¹ write: "Sometimes, in spite of all care, diagnosis

is impossible till after death, and difficult even then." They mention a case which occurred to Dr. Moxon¹² in Guy's Hospital.

A patient was admitted who had nine days before been attacked with headache, sickness, and rigors, and in whom these symptoms were followed by profuse sweating and by pains in the joints. Salicylic acid was prescribed, but the temperature rose and delirium set in, so that cold baths were employed on several occasions. The question whether the disease could be pyaemia was formally discussed and negatived in favour of the diagnosis of acute rheumatism. He made no complaint of the thigh, as being more painful than other parts, when he was being moved into or out of the bath. Yet at the autopsy not only was there osteitis of the lower part of the shaft of the right femur and of the adjacent epiphysis, but the bone was denuded of its periosteum, and there was a large collection of pus beneath the muscles. There was also suppuration about the shoulder and pyaemic abscesses in the lungs and heart.

Dr. Moxon's case very closely resembles mine, and I felt that I was in very good company when I made the erroneous diagnosis. The moral I would venture to draw from these cases is that in cases of acute rheumatism presenting any unusual features, the possibility of pyaemia should be borne in mind, and careful search should be made for the source of suppuration. Especial attention should be paid to the state of the ears, as one of the most frequent primary lesions is caries of the petrous bone.

An important distinction between acute rheumatism and pyaemia is stated to be that in rheumatism the pain and swelling fly about, leaving one joint, and after a few hours attacking another. Now this is just what happened in my case, and it was this that determined me to adhere to my diagnosis of acute rheumatism. Failure to respond to salicylates should awaken suspicion that the case is not one of rheumatism.

Pyaemia most commonly affects boys and young men, and, as a rule, the skin is dry instead of sweating as in rheumatism; the joints are more frequently hot and of a deeper red colour.

Rigors are uncommon at the onset of acute rheumatism, their occurrence, therefore, should suggest pyaemia rather than rheumatism.

Diagnosis from Influenza.

The onset of influenza is sometimes very difficult to differentiate from that of acute rheumatism, more especially if the former be accompanied by arthritis. There is, however, little or no effusion into the joints in the influenzal cases, but the ends of the joints are tender and enlarged and the seat of most of the pain. In some of the cases the joint affections proved to be rheumatoid arthritis, and others developed into various forms of neuritis. It used to be held that influenza did not lead to valvular disease, but extended experience has shown that this is not true, as cases of endocarditis and pericarditis of influenzal origin have been reported. On the other hand, simple dilatation and various cardiac neuroses, such as bradycardia, tachycardia, and angina, are only too commonly the result of this disease.

It will be found that the salicylates usually relieve the pain and pyrexia of influenzal cases, but the after-history of these cases is different from that of rheumatic arthritis.

Infective Endocarditis.

Infective endocarditis may present joint affections, sweats, and purpuric rashes, and was formerly regarded as rheumatic in origin. In fact, the transition in the symptoms from septic cases to ordinary rheumatic endocarditis is marked by insensible gradations. Still, it is recognized that most malignant cases are produced by streptococci, diplococci, or staphylococci, though the presence of the lesions of former rheumatic endocarditis favours the outbreak of the disease.

Antitoxin Arthritis.

The arthritis which occasionally follows the use of antitoxin is certainly non-rheumatic. As regards the joint affections of scarlatina, at one time they were regarded as nothing more or less than a rheumatic manifestation. Recent investigation, however, has shown that these cases may be grouped under four heads:

1. A simple synovitis, which occurs about the sixth day, and is six times more common than all the rest put together.

2. Septic.
3. Tuberculous.
4. True rheumatism.

The last only occurs during a late state of convalescence, and is always subacute. The early type with which it is usually confounded never recurs, is not accompanied by sweating, does not fly from joint to joint, rarely if ever affects the heart or shows any other rheumatic sequelae, and is specially apt to develop in the tendons on the back of the hand rather than in the wrist-joint. The true rheumatic type is rarer even than the purulent forms, and appears in quite an insignificant proportion of cases.

Gonorrhoea.

The affection of the joints occurring as a result of gonorrhoeal infection is sometimes confounded with rheumatic arthritis. Gonorrhoeal synovitis is usually the result of a gonorrhoeal urethritis, but Mr. Clement Lucas¹³ has recorded 23 cases of gonococcus joint disease in infants secondary to purulent ophthalmia, and a few years ago I admitted into the Westminster Hospital a nurse with arthritis due to purulent ophthalmia contracted by infection from an infant she was nursing. Gonorrhoeal arthritis of great intensity may occur in a newly married woman from an old gleet in the husband. I saw such a case in consultation a few years ago, and I had great difficulty in quieting the suspicions of the patient's mother, who was very insistent in knowing what was the cause of her daughter's shoulder and elbow being affected.

Gonorrhoeal arthritis usually comes on in early adult life—that is, more than half the cases occur between 20 and 30. When a patient has once suffered from gonorrhoeal arthritis a recurrence may occur as the result of passing a bougie, exposure to wet and cold, and excessive sexual intercourse.

Now that it is generally recognized that gonorrhoea is something over and above a mere affection of the urethra, we need a term to cover all the complications which may occur as a result of gonorrhoeal infection. I would suggest "gonorrhoeemia"; and, just as we speak of pyaemia and pyaemic conditions, so we might talk of gonorrhoeemia and gonorrhoeic states.

The symptoms of gonorrhoeal arthritis may come on at any period during the course of a gonorrhoea, most commonly between one week and three months. Generally the discharge has degenerated into a slight gleet, or even apparently disappeared, or there may be only the evidence of a stricture to excite suspicion. At the onset the patient is feverish, and suffers from malaise and anorexia. The tongue is furred. The patient is restless and depressed; sleep is interfered with by the pain. The pyrexia is almost always moderate, and in some cases the temperature remains about normal. Hyperpyrexia does not occur. Unlike ordinary acute rheumatism, there is little tendency, even in young subjects, for the heart to be affected, though some cases of malignant endocarditis due to the gonococcus have been recorded.

The inflammation is usually the lower extremities, the joints of the upper extremities being comparatively rarely attacked. The knee is the joint pre-eminently liable to attack. The plantar fascia is very frequently affected. The pains are worse at night, and are of an aching, grinding character. The lesion seems to affect the synovial membrane and ligaments of the joints. Any of the joints may become affected, and Mr. Brodhurst records a case in which finally ankylosis of every joint occurred. The temporo-maxillary joint is not infrequently attacked.

The joints of the fingers are very seldom attacked, a point of distinction from subacute cases of osteo-arthritis in young subjects. Each joint which becomes affected suffers for many days or even weeks, and there is not the rapid metastasis from joint to joint as occurs in rheumatic fever. Physical signs are well marked—that is, the joints are much swollen, both from intra-articular effusion and from exudation around. A very characteristic symptom of gonorrhoeal arthritis is the implication of the fibrous structures, especially of the loins, the plantar and palmar fascia, the tendo Achillis, and the sheaths of the nerves—for example, the sciatic. The muscles or their aponeuroses appear to suffer, so that the patient may complain of universal pain.

A peculiar form of inflammation of the eye frequently accompanies gonorrhoeal arthritis. Usually first one eye, then the other is attacked. There is injection of the small radiating vessels of the sclerotic which surround the cornea, that is, a scleritis, sometimes accompanied by iritis and catarrhal ophthalmia. As regards the diagnosis of the disease the presence of a discharge is obviously not sufficient to establish the point, for a man suffering from gonorrhoea may be attacked with rheumatic fever or gout. The chronicity of the disease, the implication of the fascia and other fibrous structures, the failure of salicylates to give relief, the moderate amount of pyrexia, the absence of cardiac complications and the presence of ophthalmia will usually suffice to determine the diagnosis. In doubtful cases a bougie should be passed in order to detect any stricture that may be present.

A gentleman aged 33 was sent to me on account of a painful and swollen knee, which was considered to be either gouty or rheumatic, but treatment directed to these conditions failed to give relief. It then occurred to me to inquire into the state of his urethra. The patient was found to have a stricture, the outcome of gonorrhoea contracted fifteen years previously. As soon as this had been thoroughly treated the patient lost his joint trouble, and has remained free from it.

Pneumococcic Arthritis.

Just as at one time gonorrhoea was regarded simply as a purulent inflammation of the urethral mucous membrane, so pneumonia was considered to be solely an inflammation of the lung substance. But even before the discovery of the pneumococcus it began to be realized that pneumonia was not infrequently associated with conditions that could only be explained on the ground of infection. Since Weichselbaum and Fraenkel in 1886 demonstrated the presence of the diplococcus in most cases of lobar pneumonia, we now know that the infection of pneumonia may be very widespread. As, for example, pneumococcic empyema, pneumococcic inflammation of the anterior mediastinum, of the meninges of the brain, of joints, and most important of all, the pneumococcus appears to be the most frequent of the micro-organisms giving rise to malignant endocarditis. On the present occasion, however, I must limit my remarks to pneumococcic arthritis. The cases of this disease hitherto reported have been few in number. I have only seen one myself, and that was in a child. On going over the *Transactions of the Clinical Society*—that storehouse of cases of unusual nature—I can find mention of only one case of pneumococcal arthritis.¹⁴

It is to be noted that in almost all the cases the arthritis followed the onset of the pneumonia at intervals varying from a few days to a fortnight. Therefore, if a patient suffering from, or recently convalescent from, pneumonia has any arthritic symptoms, the probability of this being due to pneumococcal infections should be borne in mind. In cases which occur independently of pneumonia the diagnosis can only be made by a bacteriological examination, though in a case reported by Mr. Barnard,¹⁵ where the knee-joint was opened, "the cavity of the joint was full of clots, which so closely resembled those seen in pneumococcal empyemata that a diagnosis of pneumococcal arthritis was at once made." This was confirmed by bacteriological examination.

The disease is one attended with a very high mortality. Of 31 cases collected and analysed by Dr. Cave,¹⁶ 23 died and 8 recovered. As a rule it affects adults or persons of advanced years, and males more frequently than females. The arthritis is commoner in the upper extremity than the lower, and in only about one-third of the cases was there a multiple joint affection. There is a very great difference in the clinical picture presented by these cases. In the mildest form there may only be slight pain and swelling limited to a single joint, whereas in the gravest cases there may be intense inflammation and distension of the joint and oedema of adjacent parts, going on to destruction of the ligaments and cartilages.

I do not pretend that I have by any means exhausted all the conditions which may simulate acute rheumatism. For instance, I have said nothing about gouty arthritis or rheumatoid arthritis; but I think that I have brought forward quite sufficient material to give rise to a good discussion, and it is very often the case that there is much more to be learned from the discussion than from the paper which opened the debate. I trust that this may be

the case on the present occasion, as I am only too conscious how sketchy my paper has been.

REFERENCES.

- ¹ *Lectures on the Practice of Physic*, 2nd edition, vol. ii, p. 284.
² Third edition, p. 26. ³ *Allbutt's System of Medicine*. ⁴ *On Chorea*, 2nd edition, p. 47. ⁵ *Clin. Soc. Trans.*, vol. xix, p. 225. ⁶ *Trans. Internat. Med. Congress*, 1881, vol. iv, p. 117. ⁷ *Lancet*, 1889, vol. i, p. 876. ⁸ *Allbutt's System of Medicine*, vol. iii, p. 91. ⁹ *Clin. Soc. Trans.*, vol. xxvii, p. 272. ¹⁰ *Ibid.*, vol. xxii, p. 86; *ibid.*, vol. xii, p. 70. ¹¹ *Principles and Practice of Medicine*, 3rd edition, vol. ii, p. 699. ¹² *Ibid.* ¹³ *Medico-Chirurgical Trans.*, vol. lxxxi, p. 161. ¹⁴ Vol. xxxvi, p. 161. ¹⁵ *Trans. Clin. Soc.*, vol. xxxvi, p. 161. ¹⁶ *Lancet*, January 12th, 1901.

An Address

ON THE

DIAGNOSIS AND TREATMENT OF MORBID CONDITIONS OF THE PLEURA.

DELIVERED AT A JOINT MEETING OF THE BURNLEY DIVISION OF THE BRITISH MEDICAL ASSOCIATION AND THE BURNLEY MEDICO-ETHICAL SOCIETY.

By T. R. BRADSHAW, B.A., M.D., F.R.C.P.,
 PHYSICIAN TO THE LIVERPOOL ROYAL INFIRMARY.

It is impossible in the time at my disposal to discuss adequately the morbid conditions of the pleura, and rather than touch lightly on them all I will pass over some in silence, so as to be able to deal more fully with the others.

INDICATIONS FOR PARACENTESIS IN PLEURAL EFFUSION.

If I were asked to state in a sentence the indications for paracentesis in pleural effusion, I should say that every effusion that was big enough to diagnose was big enough to tap. This would be with the obvious reservation that effusions apparently small and unattended by any special local or constitutional disturbance should be left for a reasonable time in the hope that they may undergo spontaneous absorption. When the physical signs point to a large effusion, or when the symptoms, such as dyspnoea, indicate serious embarrassment of the heart or lungs, the operation should be performed without delay. But the persistence of signs of a small effusion is no less an indication for paracentesis, as may be seen from various considerations. In the first place, the extent of the physical signs is not always proportionate to the size of the effusion, which may be much greater than it appears to be. In the second place, until we see some of the fluid, we can never be certain as to its nature, whether serous, purulent, or hydatid; and thirdly, the prolonged presence of a small collection of serous effusion in the pleura is not to be looked on with indifference. Apart from the mechanical compression which it exerts on the lung, if we accept the teaching of Sir A. E. Wright and his school, we must believe that a tuberculous effusion having a lower tuberculo-opsonic index than the other fluids of the body favours the existence of the bacilli in the parts which it bathes, and so ought to be got rid of as speedily as possible.

THE PERFORMANCE OF PARACENTESIS.

Having decided that paracentesis is to be performed, we should select a trocar and cannula of sufficient calibre to allow pus to flow through it. I employ the medium sized cannula supplied with Potain's pneumatic aspirator. I find Potain's pneumatic aspirator the most convenient for general use, but good results may be had with a simple tube acting as a siphon. The practice of making a preliminary exploration with a syringe and a fine needle is superfluous and undesirable. If fluid is found, the practitioner proceeds to employ the aspirator, and so two punctures are inflicted where one would have sufficed. If the result is negative the operator is in doubt whether the small needle was not blocked. The accidental puncture of the lung with a clean needle is not followed by any serious consequences. Certain points need to be especially attended to. The apparatus must be tested with water each time it is employed. The trocar and cannula must be introduced far enough to

make it certain that the pleura has been reached, the flow must be slow, and not more than 50 oz. should be withdrawn at the first operation. The remainder will probably undergo absorption; if it does not, it is not a serious matter to repeat the operation. It is well to tell the patient or his friends that you have not taken it all away.

MALIGNANT DISEASE.

The presence of blood in such amount as to give a red colour to the fluid is not uncommon, and is of no special significance, but a brownish or chocolate colour due to altered blood suggests the possibility of the existence of malignant disease. Sometimes the character of the cellular elements may also suggest the same condition. Malignant disease of the pleura or of the lung in such a position as to cause signs associated with the pleura is, however, such a rare event in the experience of any one individual practitioner that it is difficult to construct a clinical type for the disease. In general I should say that we ought to suspect the possibility of malignant disease in any case of pleurisy which, as regards its signs, symptoms, and progress, does not conform to the types with which we are familiar. Signs of pressure on structures not usually involved in pleural effusion, or of pressure not relieved by the removal of the fluid, strongly suggest a new growth. In one case there was oedema in the mammary region, enlarged axillary glands, distension of one jugular vein, and a slightly brassy cough. The trocar was felt to enter a hard mass. In another case there was paralysis of one vocal cord. In a woman lately under my care one of the earliest circumstances to arouse suspicion was finding that the effusion was of the colour of strong tea, and it was found to contain epithelial cells. Hard glands were present in the neck and axilla, and there was distension of the jugular veins on both sides. In another case there was nothing to suggest malignant disease until the pleura was opened for what seemed to be an ordinary empyema. It was then found that the pus lay in a cavity in a conical mass of new growth which extended for a considerable distance into the lung. In some cases one remains in doubt up to the very last whether one is dealing with malignant disease or tubercle, and even at the *post-mortem* examination a skilled pathologist may be unable to distinguish between them until a careful microscopic examination has been made. This actually happened in the following case:

A medical practitioner, aged 57, had pneumonia of the right lung in 1902, and made a good recovery. On June 8th, 1904, he consulted me with a large pleural effusion on the left side, the dullness reaching up to the clavicle, and the heart being pushed over to the right side. During the following three and a half months he was aspirated eight or nine times. Latterly the effusion was blood-stained, and its removal made little or no difference in the physical signs, and there was evidently a solid mass in the lung or pleura. During this time he was almost constantly in bed, complained of pain in the stomach, often severe, and got thin and weak. Tubercle bacilli were looked for in the sputum, but none were found. The temperature was moderately raised. The liver was enlarged. Towards the close there was dysphagia and severe dyspnoea, and a pericardial rub was heard. I could not make up my mind between a diagnosis of tubercle and of malignant disease, though I inclined to the former. He sank gradually, and died of asthenia on September 24th.

At the necropsy the naked-eye appearances led to the belief that the case was one of malignant disease. The right lung was normal, but the left lung was collapsed, the pleura being from $\frac{1}{2}$ to $\frac{3}{4}$ in. thick. The mediastinal glands contained yellowish masses, and there were nodules in the pericardium. Microscopically bacilli were found in the left pleura and in one of the glands, and miliary tubercles in the collapsed lung.

HAEMORRHAGIC EFFUSION.

A remarkable condition, of which I have seen two instances during the last eighteen months, is the presence in the pleura, without any obvious cause, of an effusion consisting of almost pure blood. I am inclined to think this may be dependent on some constitutional condition, allied to the haemorrhagic diathesis or the purpuric state, as seems to have been the case in the following instance:

A railway inspector, aged 57, fell against a fender and fractured one or two ribs on March 8th, 1908, and was treated as an out-patient at the Royal Infirmary. I did not see him at that time, but I was informed that no effusion was suspected until about March 18th, when extensive dullness was observed and a needle inserted. No fluid, however, was found. On March 27th—three weeks after the accident—exploration was again