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Lumleian Lectures

ON

RHEUMATIC FEVER AND VALVULAR DISEASE

Delivered before the Royal College of Physicians of London on March 23rd, 25th, and 30th, 1909

BY

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Lumleian Lectures

ON

RHEUMATIC FEVER AND VALVULAR DISEASE.

LECTURE I.

Delivered on March 23rd.

The Clinical Aspect of Rheumatic Fever.

Mr. President, Censors, and Fellows of the College,—The learned person after whom the lectureship is named, to which you have been pleased to appoint me, inherited his estate from a grandfather who fought at Flodden Field. He was himself educated at Queen's College, Cambridge, where he entered in May, 1549, and he retained a taste for learning throughout a long life, in which, as Lord Lumley, he took an active part in the dangerous politics of the time. His wife, a daughter of the twelfth Earl of Arundel, was as well read as her husband, and among their learned friends was Dr. Richard Caldwell, an Oxonian, sometime President of our College, who with Lord Lumley founded this lectureship in 1581. The fine books which belonged to Lord and Lady Lumley are evidence of their learned tastes, and were purchased at our benefactor's death for Henry, Prince of Wales. They are preserved in the Royal Library in the British Museum.

It seemed proper in expressing my own gratitude to the College to mention our debt to the founders of this lectureship, but I will not say more of their merits or achievements lest you should feel as did King James I. on April 13th, 1603, when the Dean of Durham at Lumley Castle addressed him so elaborately on the history of the family that he grew tired and called out, "Oh! man, gang na further; let me digest the knowledge I have gained, for I did na ken Adam's name was Lumley."[1]

Rheumatic fever, on which I propose to lecture, is so common a disease that every physician has seen very many examples of it, yet its natural history is still obscure in several directions. Its name takes us back to a pathology long anterior to the revival of learning, and acute rheu-

[1] Stow's Chronicle.

matism, the commonest synonym for that name, indicates a pathological confusion not wholly dispelled from the medical thought of our own day. How little consideration had been given to the subject for many centuries is shown by the fact that the Aphorisms of Boerhaave, the elaborate summary of the whole medical teaching of the greatest medical teacher of his time, which were printed at Leyden in 1709, do not mention any of the numerous conditions to which the term "rheumatism" has been applied. Boerhaave was professor at Leyden, a city where the frequent chimes seem perpetually to remind one of the heroic conduct of its citizens in the defence of their liberties and of their noble choice of a University as the reward of their valour. His lucid teaching, his unremitting study, his vast learning, his delightful conversation, and his universal kindness made him the object of the respect and the admiration of many generations of students from every part of Europe. One of these, Gerard van Swieten, whom religious intolerance drove from Leyden to Vienna, where he became the most distinguished physician in the empire of Maria Teresa, published a famous commentary on the Aphorisms of Boerhaave which began to appear in 1741, and is to this day the best book for any man to read who desires to ascertain the state of the knowledge of medicine in the middle of the eighteenth century. Van Swieten, in the commentary on the 1490th aphorism of his teacher—"There is a disease allied to the arthritis, gout and scurvy, which is very common, and is called the Rheumatism"—relates that this subject was first treated in the third edition of the Aphorisms, published in 1722. It "was now mentioned because Boerhaave had himself that year suffered excruciating tortures from this disorder. ..... As long as he had the care of the botanical garden he was constantly there before sunrise; not only in the summer but in the spring and autumn seasons, that he might prepare everything ready for his academic lecture, which he read at 7 o'clock in the morning. I well remember that in the year 1721 he suffered rheumatic pains in his scapula and neck, which were exceedingly troublesome, as in one of the hottest summers he gave advice to a vast concourse of people, in a very cold part of the house in which he lived, not so warmly clad as was requisite: but in a short time after the disorder gave way; but the following year it returned with the greatest violence. ..... When he was well and spoke before his scholars concerning the Rheumatism, about the end of June in the following year, he said for near three months he had endured the most excruciating tortures and at length was so relaxed, that no motion, and scarce any sensation, remained in his lower limbs. He added that, as soon as the pain remitted, he began to run over in his mind all the authors, both ancient and modern, that he had read; for the attention to his own pains excited him: but at the same time he remarked, he had

not found anything of consequence in any, excepting Sydenham. ..... Under this name (Rheumatism) he every year described to his pupils the malady he suffered." Thus the malady of Boerhaave, whatever may have been its true pathological species, was the cause of the introduction of a chapter on rheumatism into every subsequent general treatise on medicine.

Sydenham's remarks on rheumatism prove that he had recognised rheumatic fever, and he is, I believe, the first physician who did so and deserves to be regarded as the discoverer of the disease, though parts of his account show that, like all subsequent writers on the subject, he did not precisely define it nor always distinguish its pains from others of different origin. His description of the symptoms is clear and nothing of the kind is to be found in any earlier writer. He says: "It begins with shivering and shaking and presently heat, restlessness, and thirst, and other symptoms follow, which accompany a fever. After a day or two, and sometimes sooner, the patient is troubled with a violent pain, sometimes in this, sometimes in that joint, in the wrists and shoulders, but most commonly in the knees: it now and then changes place and seizes elsewhere, leaving some redness and swelling in the part it last possessed. At first for some days the fever and the symptoms above mentioned happen sometimes together; but the fever goes off by degrees, the pains remaining, and sometimes rage violently. ..... It may be asked why medical authors have not touched upon it, unless it be supposed this is a new disease. However it be, it is frequent enough now, and though it seldom kills anyone when the fever is off, yet upon the account of the violence of the pain, and the continuance of it, it is not contemptible." Another sentence of Sydenham—"When this disease is not accompanied with a fever, it is often taken for the gout, though it differs essentially from that, as plainly appears to anyone that well considers both these diseases"—probably suggested the writing of a classical passage in the "Commentaries on Diseases," published in 1802, of the Dr. William Heberden whose portrait in extreme age is in our Censors' room. Heberden, in this passage, gives the distinguishing characteristics of gout and then says: "These appear to me to be the marks of the genuine gout, in almost every one of which it differs from what I would call the rheumatism. For this does not begin in the foot preferably to any other part, and it seldom continues long in the same place, but will be perpetually wandering over the whole body, even during the first fit, which has been known to last for several months. Rheumatic pains will come on suddenly and without any preparatory symptoms. In rheumatisms the chief pain arises from moving the part affected, which while at rest gives for the most part rather the sensation of lassitude, than of anguish and torture. The discolouring of the skin if there

be any is not a deep red but rather a faint blush. A severe fit of the rheumatism often happens without ever returning throughout a very long life, and hardly ever makes periodical returns like the gout. Rheumatic pains very rarely desert the muscles and joints to seize upon the vital parts. Very young children will labour under violent rheumatisms; and particularly those who have in them by inheritance the seeds of a gout, with which they are to be afflicted when they arrive at manhood. These are according to my judgement the principal characters of the two distempers; but it must be owned that there are cases, in which the criteria of both are so blended together, that it is not easy to determine whether the pains be gout or rheumatism."

That children are attacked by acute rheumatism is almost the only addition made to the description of Sydenham by the observant Heberden, while his general definition of the disease is less precise than that of Sydenham. It was certainly this admirably drawn contrast of Heberden's which suggested a similar passage in the writings of Sir Thomas Watson, whose medical knowledge and pleasant style caused his book to be widely read and often republished: "In gout the small joints are first and chiefly affected, especially the joint of the great toe; in rheumatism the large. The redness of the gouty inflammation is more bright and vivid than that of the rheumatic; and the fluctuations between agony and ease are more complete and more frequent. Gout usually affects one joint only at a time: Rheumatism usually several at once. The inflammation in Gout is attended with turgid veins and with more œdema than in Rheumatism; and is followed, in the majority of instances, by desquamation and itching, phenomena which we do not notice at the close of rheumatic inflammation. Gout is not attended with those drenching acid sweats which are so characteristic of acute fibrous rheumatism. The gout is decidedly hereditary: rheumatism though probably hereditary too is much less distinctly so. The gout occurs rarely or never, whereas rheumatism is not very uncommon before the age of puberty. In gout though many functions suffer, and especially the digestive functions, there is no tendency to carditis: in rheumatism with far less general disturbance, but more fever, that tendency is very well marked. Gout is often, rheumatism is never, associated with chalk stones."

These connected passages from the writings of the three great English masters of medical description—Sydenham, who wrote in the time of Dryden; Heberden, who was a contemporary of Johnson; and Watson, whose last entrance to our Comitia, when the whole College stood up to show him respect, I can remember—all contain confusions from which the expressions of more modern physicians are not wholly free. Sydenham's account, at first clear and admirably describing rheumatic fever, goes on to associate it with quite different conditions, as he also does in his

account of the case of the apothecary Malthus in his Epistle to Dr. Brady on the Epidemical Diseases from the year 1675 to the year 1680. Heberden's clinical distinctions show that what Sydenham thought might perhaps be a new disease had not at the end of another century become perfectly familiar to physicians. Watson alone mentions the "tendency to carditis" which had first been observed by Dr. David Pitcairn, who taught at St. Bartholomew's Hospital, to which he was physician from 1780 to 1793, that valvular disease was a frequent result of rheumatic fever.

The history of the attainment of exact knowledge about each particular disease is generally that some prominent symptom has first been noticed and has long been assumed to be the whole disease, and that progressive observation has gradually demonstrated that this prominent symptom may be a result of several distinct morbid conditions. Thus the single disease of past generations of medical authorities comes to be divided into a number of diseases. These for a time continue to be spoken of as if they were related conditions, and finally the partial relation of some and the absolute distinctness of others come to be recognised. The wide meaning of the original term is forgotten and it is applied in some different way or less often falls into disuse. It is easy, for example, to recognise in old writers under the heading "Asthma" cases of some forms of tuberculosis of the lungs, of emphysema, of fibrinous bronchitis, and of laryngismus stridulus, and under that of "Diabetes" of chronic interstitial nephritis as well as of the group of pathological conditions now included under that general designation. The term "rheumatism" is at present undergoing this process. "And rheumatism I send to rack the joints" is a line of Dryden quoted by Johnson in his dictionary, and shows the general sense attached to the term since the poet's time. Physicians were very little more particular and were content to say with Heberden: "The rheumatism is a common name for many aches and pains, which have yet got no peculiar appellation though owing to very different causes." From this general statement the terms "chronic rheumatism" and "acute rheumatism" became fixed in use, and this terminology, added to certain misunderstood phenomena, led to the view still prevalent among the public, and not always rejected by practitioners of medicine, that the pains common in the muscles and joints of the aged and continuing with intervals for years are a long lasting form of the fever with swelling and pains in the joints which occurs in attacks of a few weeks' or days' duration in the young. The morbid anatomy of these two groups of cases is sufficient to demonstrate their absolute difference. In what is called chronic rheumatism well-marked arthritic changes are found, such as bony induration, ankylosis, degeneration or destruction of cartilage, while permanent morbid changes

in joints form no part of the post-mortem appearances of acute rheumatism, as these may be studied in those patients whose death has been due to the consequences of valvular disease in the first half of life. It is clear from this evidence that the several forms of chronic arthritis have no relation to rheumatic fever. Since it is desirable to avoid the suggestion of any pathological relation to chronic rheumatism the term "acute rheumatism" should fall out of use and the words "rheumatic fever" be invariably employed.

Various forms of septic arthritis, of which gonorrhœa-rheumatism is one, are imperfectly distinguished from this condition on the side of arthritis, while on the side of endocarditis several conditions due to ascertainable bacilli are no doubt sometimes placed under the heading of "Rheumatic Fever." I shall venture to consider some of these conditions without relation to bacteriology which at present fails to give that precise guidance which it is likely in future times to offer and without the confirmation of which, clinical determinations can only be regarded as approximate.

In the attempt to define clearly rheumatic fever it is necessary first to consider two diseases which are sometimes confused with it: gonorrhœal rheumatism and progressive destructive endocarditis, often called "malignant endocarditis" or "ulcerative endocarditis."

The close relation in time to scarlet fever which exists as regards the arthritis and endocarditis which sometimes appear in that disease makes it unnecessary from the clinical point of view to discuss its differentiation from rheumatic fever. It would promote clear understanding if the arthritis which sometimes follows a proved urethral growth of gonococcus were included under the general heading of "Gonorrhœa," a term sufficiently expressing that infection, and if the name "gonorrhœal rheumatism" were to be altogether disused. Before the modern improvements in surgery which have made cases of pyæmia very rare it was possible to see in the post-mortem room the body of some patient with a suppurating operation wound, several of whose joints and perhaps his pericardium contained pus. This condition of pyæmia is the type of what occurs when an arthritis succeeds the onset and accompanies the progress of a gonorrhœa. The centre of infection in the body in such a case is the urethra, and if the heart should be affected, as it occasionally is in the endocardium or pericardium or both, the source of the organisms which infect it is the urethra just as the suppurating wound is the centre of infection and the source of the arthritis in pyæmia. The observation which in my experience may be made in every such case of complicated gonorrhœa, that the arthritis is never cured while any urethritis remains, shows that the gonococci in the urethra are the stock whence those of the joints are derived. The comparative rarity of endocarditis

in such cases shows that the endocardium, like a joint, must be regarded as a point on the periphery of the infected circle, of which the centre is the urethra. This arthritis in gonorrhœa might generally be distinguished from rheumatic fever, quite apart from discovery of the urethritis, and its true nature may safely be conjectured from clinical observations, though the patient have no urethral pain and only an occasional urethral discharge, or one in which the presence of the gonococcus has not been demonstrated. The swelling and tenderness are not confined to joints or even to their immediate neighbourhood, but are spread in the line of tendons or over the area of aponeuroses and fascia. The plantar fascia is very often affected. There is a sense of stiffness in the affected joints and both this stiffness and the pains last longer than in rheumatic fever. Endocarditis is not a necessary part of the disease and, as a rule, no physical signs indicating it are present. Permanent damage to a joint often seems about to occur and sometimes does occur.

Out of 11 cases under my care at St. Bartholomew's Hospital during the years 1903 to 1907 the plantar fascia was painful in four, and large areas of the calf and forearm in two others, in addition to the arthritis of particular joints. Marked stiffness was present in seven and led to rigidity of the wrist-joint in one. A raised temperature persisted for three weeks or more in five of the cases without signs of any pleurisy or pericarditis. In one case there was on admission disease of the mitral valve causing regurgitation and in this case pleurisy also occurred during the illness. In the other 10 there were no signs of endocarditis throughout the illness. Permanent damage to joints took place in six cases, slight in five, and accompanied by extensive ankylosis in one. The whole aspect of the disease is seen to be entirely different from that of rheumatic fever in the duration and character of the pains as well as to some extent in their locality, and in the absence of infection of the endocardium and the frequency of permanent damage to joints. The temperature chart is not the same as that of rheumatic fever which is under treatment by salicylates. This difference may be stated briefly by saying that the approach to the line of 100° is generally more frequent during the first three weeks and is more often continued into several weeks than in rheumatic fever.

The clinical distinction of ulcerative endocarditis from rheumatic fever is sometimes difficult, and most observers have, I should think, long remained in doubt whether they have before them two distinct diseases or merely varying results of the same infection. The bacteriological evidence does not at present solve the problem. The two diseases, however, may generally be distinguished by clinical observations. In a case of ulcerative endocarditis the patient is often remarkably contented or even cheerful and has not the pained position and expression common in

cases of rheumatic fever till the pain is relieved. This frame of mind, a sort of settled good temper, quite different from the optimistic mental state seen in patients with tuberculosis, is well marked in a little girl now in one of my wards who has been there since July, 1908, and who, in spite of high temperature and prolonged illness, is invariably placid and uncomplaining. The joints are in most cases of ulcerative endocarditis free from pain and from swelling. Out of nine cases under my care no joint pain existed in seven. The temperature chart, however long the disease may last, always shows high temperatures, usually every day, always for most days of each week. In some cases the temperature reaches 104° F. often; but in others 103° or 102° is the highest point, and in either case this high degree is reached again and again. Sometimes no cardiac murmurs are heard, more often they are plain and vary a little from week to week. Evidence of emboli, such as enlarged spleen, hæmaturia, or hemiplegia, is often observable, while the arteries of a limb are sometimes blocked or acute aneurysms appear on them or on other vessels. The illness may go on for a very long time, but the patient never recovers. Sometimes it is clear that the patient has had rheumatic fever and had a heart damaged by that disease before the ulcerative endocarditis began. Out of the nine patients above mentioned four had had rheumatic fever years before, and one had a heart which had probably been damaged in relation to scarlet fever. These symptoms, therefore, make it possible to distinguish a case of ulcerative endocarditis from one of rheumatic fever. It may be added that treatment by salicylates is without any effect on the symptoms or temperature chart of ulcerative endocarditis. My conclusion, from a clinical point of view, is that rheumatic fever is a distinguishable disease of different origin from the other morbid conditions which include arthritis amongst their symptoms.

The cardinal features of rheumatic fever are endocarditis and arthritis. Endocarditis is always present. In most cases, but not in all, it produces some permanent alteration in one or more valves. This endocarditis of rheumatic fever is the chief cause of the varieties of valvular disease met with in patients under 30 years of age. If the heart in a slight case is only auscultated at the beginning and end of the disease the sounds may sometimes seem to be unaltered and it may be assumed that they have been normal throughout; but if the heart is examined daily changes in its sounds will, in my experience, always be discovered, which, taken with the evidence of the frequency of definite valvular disease as a result of rheumatic fever and with the frequency of a history of rheumatic fever in cases of valvular disease belonging to the first half of life, justify the belief that these variations in sound may be taken as evidence of the presence of endocarditis, even though no permanent valvular lesion be produced.

Out of one hundred cases of rheumatic fever under my care in St. Bartholomew's Hospital murmurs indicating valvular disease were distinct in all but one. In the solitary exception, that of a man, aged 24 years, who had first had rheumatic fever the year before that in which I saw him, pericarditis was present with a very loud friction sound which may have obscured an endocardial murmur. The friction sound remained after the fever had disappeared and I was never able to hear any murmur. Many of the patients gave a history of previous attacks, some of them certainly were in their first attack. The slight degree of arthritis which is sometimes present in a first attack of rheumatic fever in a child often leads to its non-recognition by a parent and patients who are suffering from the disease later in life and who have suffered from it more than once before—a very common case—are often found to be very inaccurate in fixing the date. When a child with rheumatic fever is said never to have had an attack before, but is said to have had chorea a year before, it will be far more often true that another attack of rheumatic fever preceded the chorea than that the existing attack is the first one. A patient of the age of 28 years may easily remember an illness of childhood in which he was confined to bed with severe pains in the joints, but slighter attacks of pain are easily forgotten. Every man has his own memory by which to test the powers of memory possessed by others. The recollection of physical pain and of slight illness is especially transitory. Few pains are more severe than that of toothache, but who can mention the dates in his life or even the years in which he had toothache? Even when an event is impressed upon the mind by some special act in relation to ourselves its precise date is rarely remembered even by highly trained minds. The Fellows of this College each receive half-a-crown in the College on the Monday after Palm Sunday, but how many of them could write down on what day of the month those Mondays were or who on each particular occasion sat to their right hand and their left in the Comitia? We generally expect too much from the memory of our patients. I have therefore not attempted to say how many of these one hundred patients with rheumatic fever were suffering from their first attack. 63 were males and 37 were females. As regards age they were thus distributed :—

63 Males.

To 5 years	1	To 25 years	5
,, 10 ,, ...	14	,, 30 ,, ...	4
,, 15 ,, ...	20	,, 35 ,, ...	3
,, 20 ,, ...	13	,, 40 ,, ...	3

37 Females.

To 5 years	3	To 25 years	5
,, 10 ,, ...	4	,, 30 ,, ...	4
,, 15 ,, ...	10	,, 35 ,, ...	3
,, 20 ,, ...	7	,, 40 ,, ...	1

In 44 males and 29 females the mitral valve alone was affected ; in nine cases, all of males, the aortic valves alone were affected ; in eight males and eight females both the mitral and the aortic valves were affected ; in one male the pulmonary and aortic valves were affected ; and in one case, as already mentioned, a very loud friction sound made it impossible to determine the presence or absence of valvular disease. In seven other cases, in all of which the mitral valve only was affected, pericarditis was present, so that pericarditis occurred in only 8 per cent. of the whole. The question of whether endocarditis or pericarditis was the more common in rheumatic fever was frequently discussed by the generation which flourished between 1830 and 1860, but at the present day the reply must be that while pericarditis is a somewhat rare incident of the disease there is evidence of endocarditis in every case.

Taking the history of 100 cases of valvular disease lately under my care in which the patients were of the age of 30 years or less, 60 gave a distinct history of rheumatic fever, and in 100 cases in which the patients were over 30 years of age, 46 gave a history of rheumatic fever These 200 patients were none of them included in the 100 cases of rheumatic fever mentioned above. The 200 cases show the frequent occurrence of rheumatic fever as a cause of endocarditis. It is probably correct to say that three-fourths of the cases of valvular disease which are seen in patients under 30 began in the endocarditis of rheumatic fever, and that half of the cases of valvular disease in patients over 30 began in the same disease. The 100 cases may be taken as proving that endocarditis is invariably present in rheumatic fever. It must be regarded as the central condition in the disease. If no evidence of endocarditis is to be found throughout an illness of which a symptom is arthritic pain then that illness is not rheumatic fever, and the arthritis has some other cause.

Sir William Selby Church, one of the latest writers on rheumatic fever, does not believe that endocarditis is invariably present. "It is difficult," he says, "perhaps impossible, to determine during an attack when endocarditis begins, for it gives rise to no special symptoms, but in a large majority of cases if no endocardial murmur be present during the first ten days of an attack the endocardium escapes." This is certainly the prevalent view, but my own observations lead me to a different conclusion. I have found that in every case of first attack of rheumatic fever the presence and sometimes onset of endocarditis may be inferred from the alterations in the heart sounds. In later attacks where valvular disease with a loud murmur or murmurs existed before the onset of the arthritis, minute differences of sound are less easily detected, but progressive alterations in the valves as well as, in many cases, diurnal or weekly alterations of sound, with rises of temperature after

arthritic changes have ceased, satisfy the observer that endocarditis is present.

The deepest symptoms of rheumatic fever are alterations of the heart sounds and can only be discovered by a trained observer. The most obvious are pains in the joints accompanied by swelling and often by redness of the skin over them. The patient himself is distressed by the pain and anyone at his bedside may see the swellings or the redness. This part of the disease is matter of common knowledge, while detection of the endocarditis belongs to the arcana of medicine, discoverable only by a skilled method of observation and a trained sense of hearing. This prominence of the external over the internal symptoms was still more marked when, owing to the want of any remedial drug, the pains lasted for more days than they do at present, and at the same time the practice of auscultation was less assiduously pursued than it now is. The well-known fact that the size or number of the joints affected has no direct relation to either the severity of the endocarditis or to the seriousness of the consequent valvular disease supports the view that the endocarditis is the primary lesion in rheumatic fever.

The point of entrance of the organism which seems likely to exist, though its presence has not been proved, has sometimes been suggested to be the tonsils. A slight degree of sore-throat and sometimes a severe angina faucium may be present at the beginning of an attack of rheumatic fever, but is not found sufficiently often to justify the belief that swollen tonsils are the first anatomical change of the disease and that it is through them that the organism enters the system.

The common expression that the pain flies from one joint to another—say, from the left ankle to the right knee—is, of course, merely a relic of the Hippocratic doctrine of metastasis. When an empyema[2] succeeded a pneumonia, or an obvious pleurisy was followed by a pneumonia, the Hippocratic school held that one disease had been converted into another, and thus it was easy for them to regard an inflamed ankle on one side as converted into an inflamed knee on the other. This form of words belonging to an obsolete doctrine ought at the present day to be avoided in teaching medicine at the bedside, since expressions, as distinct from mere terms, of archaic pathology easily continue to have influence on the mind of our own day in a way detrimental to a clear understanding and view of disease.

Whatever the point of entrance and line thence of travel, the endocardium is to be regarded as the invariable centre and primary region of growth of the organism of rheumatic fever and the swollen joints as so many colonial settlements proceeding thence.

The affected joints of rheumatic fever always recover com-

[2] Cf. ΠΕΡΙ ΠΑΘΩΝ, cap. ix.

pletely ; not a single case of stiff joint was to be found in the cases of the last six years in my wards. The knee-joint is one which is very often attacked in rheumatic fever, perhaps more often than any other. The presence post mortem of a mitral valve showing thickened edge, thickened tendinous cords, or small growths upon its cords or edge may be taken as indicating that the person in whose body it is found has had rheumatic fever. When I have opened the knee-joints in young patients who have died from the results of disease of the mitral valve I have never found the remains of changes in the structures of the joint, neither thickening nor degeneration. Some of these cases may have acquired their affection of the mitral valve otherwise than by rheumatic fever, but the percentage of cases in which mitral disease is the product of rheumatic fever is so large that the exceptions as regards other ways of origination of mitral disease do not affect the argument. In old hospital notes any accounts of cases of rheumatic fever terminating in stiff joints, or of stiff joints attributed to past rheumatic fever, may be suspected to have been examples of some form of septic arthritis or of progressive or degenerative arthritis and not of true rheumatic fever.

After the endocardium and the joints the temperature of rheumatic fever next deserves consideration. The well-known papers of Dr. Henry G. Sutton in the Guy's Hospital Reports for 1865 and 1866, entitled "Cases of Rheumatic Fever Treated for the Most Part by Mint Water, Collected from the Clinical Books of Dr. Gull," unfortunately give no evidence as to the temperature attained or continued when the disease was left to follow an uninterrupted course, for the thermometer had not then come into regular use in the hospitals of London.

In the 100 cases in my own wards which I have already mentioned the effect of a treatment by salicylate of soda must be allowed for. Doses of not less than 10 grains nor more than 20 were given during the febrile period of the disease, for one, two, or three days every four hours, then every six hours for a period regulated by any remains of pain or unusual rise of temperature, and succeeded by a longer continuance of administration three times a day. This was the general plan of treatment, the patient being kept in bed with a few exceptions till the temperature had been normal for three weeks. The commonest temperature chart was one in which the temperature on admission being 100° or higher it rose to a less height the next day, and perhaps still less on the third day, and then continued normal for some days, after which it rose to 99° or a little above 99° on some one day or two days of a fortnight or three weeks and then steadily continued normal or subnormal for three weeks, after which the patient left the hospital. The slight rise on one day, or continued for two or three days, of the week succeeding a pause of several days which itself succeeds the first definite fall of

temperature to the normal point is characteristic. When the chart presents a different aspect circumstances which explain the greater or longer rise can generally be discovered. A few examples may be given :—

A girl, aged 10 years, was admitted to the hospital for what seemed to be her first attack of rheumatic fever. On admission her temperature was 101·5° F. ; on the next day it was 99·8°, and it was then normal till the sixteenth following day, when it rose to 99·2°. It was then normal for two days, then it rose to 99°, then it was normal for four days, and then it rose to 99°, after which it remained normal.

A girl, aged 16 years, who had had a previous attack, had a temperature of 99° F. twice in the first week and one of 98·6° in the second week, and no further rise.

A woman, aged 20 years, who had had three previous attacks, was admitted with a temperature of 99° F. and on the next day her temperature was 99·8°, on the day after it was 98·6°, on the day after that 98·8°, and then it was normal or subnormal for 13 days. It then rose on two successive days to 99·2° and then it became normal for eight days, then it rose to 98·8°, was next normal for five days, and after that continued for some weeks more, with rises of a degree or a few tenths of a degree separated by intervals of several days, finally reaching a continuous normal temperature.

A woman, aged 19 years, in her first attack had on admission a temperature of 102·8° F. On the next three days it was successively 101·8°, 99·8°, and 99·4°, and was then normal for two days, then it reached 99°, and was then normal for a fortnight, then it was once more 99° and then it continued normal for three weeks.

A boy, aged nine years, was admitted with a temperature of 102·4° F. On the next two days his temperature rose to 99° and then it was normal for 15 days. After one day at 98·8° it was normal for three days, then on one day 98·8°, and then normal for 18 days, after which he left the hospital.

Variations or interruptions of this course of temperature are generally due to ascertainable causes and in rheumatic fever, as in all other diseases, a constant consideration not merely of the daily temperatures but of the whole chart is important. Pericarditis always produces a rise in temperature of some days and may thus be inferred before a friction sound is audible. A boy, aged four and a half years, who had had pain in his knees for three weeks was admitted with a temperature of 100·8° F. and his temperature approached, or slightly exceeded, 100° for eight succeeding days. On the second day of his admission a friction sound was heard over his heart and was audible for two days. The abnormal rise of temperature was certainly due to pericarditis. Pleurisy in the absence of bacteriological evidence as to its nature must be believed to be an actual part of the cycle of rheumatic fever, and, of course, affects the chart. Bronchitis, common in patients with rheumatic fever, whose hearts are embarrassed by valvular disease of long standing, similarly affects the course of the temperature, though it remains uncertain whether it or the pneumonia which sometimes occurs are a true part of the rheumatic fever. Attacks of sore throat and common colds are other influences to be considered in contemplating the temperature chart. Whoever studies cases of rheumatic

fever will become convinced that slight rises of temperature are never unimportant, and that considerable rises of temperature at exceptional times can generally be explained if sufficient search be made.

The grounds for the diagnosis of rheumatic fever are: (1) evidence of the results of endocarditis; (2) a multiple transient arthritis; and (3) a temperature chart showing an initial rise followed after a fall by occasional rises with intervals of normal temperature and with evidence of the existence of some disease for a considerable period, seldom less than three weeks. The diagnosis ought not to be made till these three factors are ascertained. If endocarditis be clearly present without arthritis ulcerative endocarditis, and not rheumatic fever is likely to be the true diagnosis. The same is true if there be a temperature chart of high oscillations with uncertain evidence of endocarditis but signs of disturbed heart and no arthritis. If there are no signs of endocarditis and a multiple arthritis at present, then a gonococcal infection and not rheumatic fever is the probable diagnosis. The same is true if, though there be signs of endocarditis and of multiple arthritis, any of the affected joints come to show signs of permanent damage. It should be added that a culture may be the only way of ascertaining whether the gonococcus or some septic organism is the origin of the condition which after some resemblance in symptoms to rheumatic fever leaves the patient with a stiff joint.

The term "subacute rheumatism" should never be used, for it suggests an absence of importance which attaches to no case, however slight, of rheumatic fever. Just as the prevalence of the idea that *petit mal* in some way means that the patient said to have it is not an epileptic, while in fact his next fit may be a most serious one, does harm, so the term subacute rheumatism, by suggesting that the case is of less importance than one of rheumatic fever, does harm. The pains are very slight in such cases, but the endocardium is none the less affected and they are often merely periods of slight endocarditis leading to or threatening more severe manifestations in the future.

Every case of chorea in which there is a cardiac murmur, accompanied by rise of temperature, must be regarded as connected with an attack of rheumatic fever, perhaps best as the continuation of such an attack. It is probable that some cases of chorea in which a valvular murmur can be heard but in which the temperature, even when taken regularly for weeks, shows no rise, are also actual continuations of rheumatic fever, and there are perhaps some cases in which the endocarditis is so nearly quiescent that there are neither murmurs nor rises of temperature and which yet have grown out of an attack of rheumatic fever and may pass on into another such attack. Spasmodic and incoördinate movements in children may be

due to several causes, but it is important to recognise that what is sometimes called rheumatic chorea is a definite species of which a cardiac murmur due to the same endocarditis as that of rheumatic fever and a temperature showing occasional rises are symptoms. The first question when a case of chorea is before us is to determine whether it belongs to this species, and if it does the patient for purposes of treatment may be assumed to be in the course of an endocarditis less severe than it is in a definite attack of rheumatic fever but capable of aggravating or producing injury to the cardiac valves and especially to the mitral valve.

The administration of sodium salicylate so commonly relieves the pains in the joints as well as reduces the temperature in rheumatic fever that whenever it is found in any case that sodium salicylate has no effect, even in a large dose, the suspicion will arise that ulcerative endocarditis, a gonococcal infection, or some other condition and not true rheumatic fever is present.

The disease belongs to the first half of life, though, like measles, mumps, and some other morbid conditions of childhood and youth, it may appear for the first time in the second half of life, but here again the difficulty of certainty as to a patient's true history often raises doubt as to whether his earlier life has been free from any attack. In the absence of bacteriological evidence it is impossible to be certain, but as a result of the observation of a few supposed first attacks between the ages of 35 and 45 years I incline to the belief that rheumatic fever does now and then attack a patient at that age. Before the diagnosis is made great care should be taken to ascertain that no septic source, such as a uterine one in women, exists, or has recently existed, in the patient.

The duration of rheumatic fever is indefinite. The exact day of its commencement can rarely be fixed. In many cases the state of the heart proves that the disease has been in progress for some time before the patient is aware that he has a definite illness. The date of its absolute termination is always obscure. The difficulty of determining it is increased by the knowledge that endocarditis may continue to affect the heart—in other words, that the organism which produces it may continue to live within the heart, capable of fresh development, long after the temperature has become normal. What is commonly called an attack of rheumatic fever probably continues not less than three weeks after a normal temperature is reached. Certainly as this period is approached the temperature shows less tendency to those occasional rises which are common in the first month of the illness. The fact that a patient has not yet recovered from an attack may be indicated by slight rises of temperature, fresh pains or swelling of joints, or even by chorea with or without rise of tem-

perature. Since there is generally no further opportunity of infection it is probable that the organism remains in the heart capable of reproducing under favourable circumstances the former phenomena. Thus the duration of the disease really depends on the presence of circumstances which are favourable to the development of an organism capable of long existence in the human body. "When a man is infected by bilharzia," said the very learned Professor Looss to me in course of a conversation on parasitology in Cairo this winter, "it may be some comfort to him to reflect that every entozoon dies after a sufficient lapse of time. What we have to do is to ascertain what its lifetime is." To learn its lifetime is equally part of the study of every micro-organism. The bacillus of enteric fever commonly ceases to be maleficent within three months of its introduction into the human body. Sir George Paget related to me the history of a patient of his who developed signs of tuberculosis of the lung at 30 years of age, was never quite free from signs of it, and died from well-marked pulmonary tuberculosis when over 90 years of age. The tubercle bacillus had lived in his body for full 60 years. The duration of life of the organism of rheumatic fever is certainly less than that of the tubercle bacillus. Its active presence, as shown by its effects, lasts longer than that of the pneumococcus in lobar pneumonia. I shall venture to offer a hypothesis as to its approximate duration when I come to consider the prognosis.

The conclusion to which I wish to attain in this lecture is that every case of rheumatic fever, whether the pains in the joints are slight or severe, is a case of endocarditis, and that the condition of the endocardium is not to be regarded as a frequent complication but as the essential and invariable feature of the disease. I cannot help wishing that a name absolutely extricating this serious disease from the confusion involved in the term "rheumatism" could be found. Medical terms, inconstant as their meaning is from century to century, are difficult to destroy. It is no less difficult to bring them into use. The "dothienenterite" of Bretonneau was appropriate enough but is almost as little used as the "pædosteosplanchnocace" of Daniel Whistler. Dr. Charles Badham's term "bronchitis," issued just a century ago, owes its firm place in the English language as well as in medical practice partly to the circumstance that no patient likes to think that his lungs are affected, since he knows that people often die from lung disease, but is willing to account for his cough by a name which does not convey a definite anatomical idea to his mind. Remembering this unwillingness of men and women to hear that they have diseases associated with the lungs or heart, I feel that "heart fever," which would be an appropriate term for a disease of which the most serious feature is endocarditis, would have little chance of coming into use instead of

rheumatic fever. Were it not for this difficulty on the side of public opinion, which is what chiefly regulates the use or neglect of words, the term "heart fever" might with advantage be adopted for that morbid condition in which endocarditis, transient arthritis, and raised temperature are always present.

LECTURE II.

Delivered on March 25th.

Valvular Disease.

Mr. President, Censors, and Fellows of the College, —It is remarkable that neither in the notes of the Lumleian lectures of 1616, in which Harvey shows that he had attained the conception of the circulation of the blood, nor in his "Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus" of 1628, in which his discovery is fully set forth, is there any allusion to valvular disease. The retardation of the blood in the veins produced by narrowing of the mitral orifice with the consequent dropsy seems to us an illustration of the course of the blood from the right heart through the lungs to the left auricle, and the changes in the pulse produced by disease of the aortic valves demonstrate the continuity of the blood stream from the left ventricle to the remotest arteries, but Harvey does not seem to have noticed the effects on the circulation of any alterations in the valves, or, indeed, any other than the normal condition of the valves. There is only one allusion to disease of the heart, and that an obscure one, in the "De Motu Cordis." Andreas Laurentius is mentioned as quoting the case of a man the left ventricle of whose heart was found post mortem to contain turbid, fœtid, acrid fluid. Laurentius thought that some turbid urine during the patient's life had therefore come from the heart. It seems possible that the disease of which fainting was a symptom may have been ulcerative endocarditis. Harvey evidently was rather incredulous about the observation.

Moses from Pisgah saw the good land which he longed to enter stretched before him, but was never to know its plains and hills, its deserts, its forests, and its towns. So Harvey, having seen clearly the course of the circulation and the precise normal action of the heart, was not permitted to see further into the land of truth he had obtained for mankind, nor was it till nearly a hundred years after he lectured to this College that a knowledge of valvular disease began to grow up.

The belief in the existence of polypus of the heart, about which so much was written in the eighteenth century, shows that the consequences of the doctrine of the circulation of

the blood were not grasped for a long time. These polypi, which are very clearly described by Tulpius in 1641, with a beautiful drawing, and some twenty years later in Malpighi's "Dissertatio de Polypo Cordis," were clots sometimes ante-mortem, as in that which was removed from an aneurysm by Borelli, and sometimes post-mortem clots, as in those described as occurring in the right ventricle. Huxham in 1742 described to the Royal Society polypi in the hearts of two sailors in language which shows that he failed to grasp the impossibility of circulation with such obstructions:—

> The polypi were very nearly of the colour of the buff formed on the surface of highly pleuritic or rheumatic blood, when quite cold or rather whiter. They were vastly tough and seemed to be formed of various laminæ very closely connected, though here and there a bloody vein, as it were, was interspersed. They were not only firmly attached to the fleshly columnæ of the heart but were also sunk and inserted strongly into the intercolumniæ or sulci and that even to the very bottom of the ventricules. ..... The polypus taken out of the left ventricle of the same heart was also very considerable and rather more firm and compact than that of the right but of the very same colour, and firmly implanted into the sides of the ventricle quite down to the mucro cordis. Its branches were shot a great way into the subclavian and carotid arteries but very little down the aorta.[1]

Matthew Baillie's demolition of the doctrine of polypi[2] may perhaps be regarded as the final step in the understanding of Harvey's discovery of the circulation:—

> Polypus consists of a mass of coagulable lymph, which fills up some of the large cavities of the heart, particularly the ventricles, and extends into the neighbouring large vessels. ..... In order that the circulation may be carried on it is necessary that the cavities of the heart be free for the transmission of blood, and if any one of its cavities should be plugged up, the circulation would necessarily be stopped altogether. A polypus, however, plugs up the cavity of the heart in which it is formed so entirely as to prevent the circulation. ..... These circumstances seem to contradict strongly the opinion that polypi in general are formed during life.

Thus it took from 1628 to 1795 for Harvey's discovery to be thoroughly understood in the world of medicine. The above passage in Matthew Baillie's "Morbid Anatomy" may be regarded as the final preparation for the accumulation of knowledge on valvular disease. A few observations on disease of the valves may be found in earlier medical books, of which the best is perhaps that of Dr. James Douglas.[3]

> I lately opened a young man in St. Bartholomew's Hospital, that died of the palpitation of the heart, whose violent beating and prodigious subsultory motion for some months before his death, was not only easily felt by laying the hand on the region of the heart, but seen to rise and fall by raising the bed cloaths that covered it, and, which is almost incredible at some times, the trembling and throbbing made such a noise in his breast as plainly could be heard at some distance from his bedside.

[1] Philosophical Transactions, vol. xlii., 1744.
[2] Morbid Anatomy.
[3] Philosophical Transactions, 1715.

After describing an adherent pericardium and the normal right side of the heart, Douglas mentions the increase in size of the left ventricle and says :—

> The valvulæ called mitrales placed at the orifice of this ventricle are much thicker in substance than ordinary. ..... The semilunary valves at the mouth of the aorta ..... were very much preternaturally affected ..... very thick contracted, as it were, and furled together, and of a whitish colour, and in all appearance, if the person had lived longer, they had turned bony or undergone a petrifaction.

Everyone who reads this account will perceive how very near Douglas went to the discovery of the uses of auscultation. The loud noise which was to be heard proceeding from the cardiac region was probably a systolic murmur due to the rigidity of the aortic valves which was discovered post mortem. Huxham, in the paper which I have already quoted, says that in one of the sailors with polypi which he discovered post mortem, "I observed one of the semilunar valves of the aorta beginning to grow bony." There was, however, no real understanding of valvular disease till the time of Laennec and of his school.

I have maintained in my first lecture that endocarditis often producing permanent valvular disease is the central feature of rheumatic fever. What evidence is there of the extent in the heart of this endocarditis? Excluding cases of ulcerative endocarditis, the presence post mortem of disease of the mitral valve is the best obtainable anatomical evidence that the subject of the necropsy has had rheumatic fever. The mitral valve is rarely affected by chronic degenerative change or by syphilis. It may be affected by endocarditis and so deformed in the course of scarlet fever, of gonorrhœa, of influenza, of sepsis, or of pneumonia, but the number of cases due to these conditions taken together is small compared to the number due to the endocarditis of rheumatic fever. This endocarditis sometimes affects a large area of the interior of the heart. A woman, aged 19 years, whom I examined post mortem, was a good example of this. Her heart weighed 19 ounces and the pericardium was universally adherent. The endocardium of the right auricle was normal. The tricuspid orifice was wider than natural; the edge of the valve was greatly thickened and studded on its upper surface with numerous minute growths. One part only was free—a quarter of an inch at the anterior end of that part of the valve which is attached to the ventricular septum. At the root of the cords which proceed from the anterior part of the ventricular septum to the valve were numerous minute growths. The point of origin of this group of cords —they have no projecting musculi papillares—was thickened and these minute growths were on this thickened base and on the lower part of the cords. The other vertical cords showed no thickening, but there were some transverse

thickened cords and a little roughness on the under surface of the valve. There was a small linear thickened patch in the right ventricle on the septum. Two of the pulmonary semilunar valves were adherent to one another and on each of these were numerous minute growths and one more prominent one. There were several much thickened patches of endocardium in the left auricle and there was a small rough patch on the auricular septum. The mitral orifice was somewhat dilated. The valve was fringed by a thick border of minute growths and all the tendinous cords were thickened. There was a general thickening of the aortic flap of the mitral valve. On the upper and anterior part of the ventricular septum in the left ventricle there was a thickened patch as large as a silver penny of Queen Victoria. The aortic valves were incompetent, each had numerous thickly-set small growths on the outer surface below the edges of the valves and with a connecting fringe of growths from valve to valve. In the hollows between the valves there were also minute growths. The valves were adherent to one another and were slightly everted. There were no signs of ulceration in the endocardium and no emboli in the viscera. This was an example of very widespread endocarditis. Taking 20 other cases (ten males and ten females) examined by me post mortem in which there was a probable history of rheumatic fever, as they came in my notebook, the following was the evidence of the area of endocardium affected :—

Males.

10. Right auricle thickened patches, tricuspid, mitral, wall of left ventricle, aortic valves showing thickening.
19. Tricuspid, mitral, aortic valves showing signs of old endocarditis.
19. ,, ,, ,, ,, ,, ,, ,, ,,
21. ,, ,, ,, ,, ,, ,, ,, ,,
24. ,, ,, ,, ,, ,, ,, ,, ,,
27. Tricuspid, wall of right ventricle, mitral, showing signs of old endocarditis.
28. Mitral, wall of left auricle, showing signs of old endocarditis.
31. Mitral, aortic valves showing signs of old endocarditis.
35. ,, ,, ,, ,, ,, ,, ,,
37. Tricuspid, mitral, wall of ventricle, aortic valves showing signs of old endocarditis.

Females.

5. Tricuspid, mitral thickened.
8. Left auricle, mitral, left ventricle, aortic thickened.
15. Mitral valve deformed.
16. ,, ,, ,,
20. Wall of right auricle, tricuspid, wall of right auricle, mitral thickened.
20. Mitral, wall of left ventricle, aortic valve deformed.
21. Mitral, aortic valve deformed.
21. Left auricle, mitral, aortic valve deformed.
29. Mitral, inside of outer wall of ventricle, valve deformed.
44. Tricuspid, right ventricle, left auricle, mitral valve deformed.

In none of the males were the signs of endocarditis confined to a single region, and in only two of the females was the

mitral valve the only affected area. The first case shows how very widespread an endocarditis may be in rheumatic fever, and the others support the view that endocarditis tends to be widespread on the left side of the heart and sometimes extends to the right side.

Specimens of hearts are now gradually accumulating in museums in which naked-eye appearances resembling those of the endocarditis of rheumatic fever are proved to be due to various bacilli. Thus, in a male patient, aged 39 years (under the care of my colleague, Dr. J. A. Ormerod), who was ill for three months and whose necropsy I happened to witness, extensive recent endocarditis involving the mitral valve was found and gonococci were cultivated from the growths. The patient had died from endocarditis, which was a part of gonorrhœa. Here is the heart. There are large, soft, recent growths on the aortic valves with considerable destruction. In another heart showing endocarditis belonging to our collection at St. Bartholomew's Hospital, which I show you, a pneumococcus was cultivated from the growths, and in another the bacillus coli was found in abundance in the endocardium. The history of a few cases in my wards in which valvular disease due to an acute endocarditis was found and in which the only acute disease of the patient's past history was small-pox, added to the fact that pericarditis occurs in small-pox, makes it seem probable that an endocarditis may sometimes be due to the organism of variola. A passage in the Loimologia[4] of Dr. Nathaniel Hodges may, perhaps, point to what is in itself likely, the occurrence of endocarditis in plague:—

> I was sent for to a youth of about 14 years of age who had continued free of the infection, after his mother and the rest of the family had been visited by it, when all on a sudden he was seized with such a palpitation at heart that I and several others could hear it at some considerable distance, and it continued so to do till he died, which was soon after.

It was in the eager search for knowledge on this and other parts of the morbid anatomy of plague that my former clinical clerk, Dr. C. T. Raikes, lost his life in Singapore, and I cannot allude to the post-mortem appearances of plague without commemorating the keen observation and intelligent devotion to the increase of knowledge of this young Oxonian.

The course of the following obscure case showed an unexpected occurrence of endocarditis and subsequent valvular disease.

> A boy, aged 10 years, was admitted to St. Bartholomew's Hospital with a rash on his face, legs, and arms, and ulcerated lips and some sore throat. The spots were pimples with bullæ in the centre and gradually faded away and the mouth healed. The throat gave a pure culture of streptococcus. A cultivation of his blood gave a growth in broth of yellow staphylococcus and some streptococci. The tem-

[4] Second edition, London, 1720.

perature was 100° for two days, and then became normal. On his admission, on March 8th, 1907, his heart sounds were normal and so continued till April 8th. Then a short presystolic murmur was audible just within, and a little below, the left nipple, and a well-marked thrill was felt at the apex. On April 22nd the thrill was still distinct though the murmur was fainter. The rash was most like varicella, but as the nature of the disease was obscure I asked four of my colleagues to express their opinion on it. Dr. Ormerod, Dr. H. H. Tooth, and Dr. J. H. Drysdale were of opinion that the condition was of septic origin and Dr. S. West suggested that the rash belonged to the class of bullous erythema. The murmur and thrill were present when the patient left the hospital, otherwise well, on April 26th.

There were no joint symptoms of any kind and no chorea followed the illness. It was probably an example of endocarditis and consequent valvular disease originating in some sepsis. I suspect that cases of what is called heart failure in influenza may sometimes be due not only to affection of the myocardium but to endocarditis which may sometimes interfere seriously with the action of the valves without causing a murmur. Dr. T. J. Horder has demonstrated the presence in the endocardium of the influenza bacillus. There are two specimens in the St. Bartholomew's museum. In lobar pneumonia it is not common to find clinical evidence of endocarditis, but there is no doubt that the pneumococcus is a cause of endocarditis. Here is a heart showing growths on the tendinous cords and edge of the mitral valve from which pneumococci were cultivated.

It is interesting to observe that in these cases as in those of ulcerative endocarditis valvular disease, often the result of long past endocarditis of rheumatic fever, is often present, as if an already damaged endocardium formed a favourable ground for the growth of streptococci and other organisms.

The view of 30 years ago was that endocarditis was an occasional complication of rheumatic fever. I have tried to show that endocarditis is its central feature. It is clear that endocarditis has many true species, some determined bacteriologically, some resting their specificity on clinical observation alone. Fresh species will certainly be discovered. The nature of the endocarditis of rheumatic fever seems at present to rest on clinical observations alone. It is none the less a good working species. Let us proceed to examine its effects upon the heart.

I may begin the subject by showing you these two specimens. This heart is that of a woman, aged 20 years, whose post-mortem examination I lately attended. It shows some old thickening of the cords and edges of the flaps of the mitral valve and on one flap a small raised red patch of recent endocarditis. Seeing that the uterus contained a fœtus I asked the demonstrator to open its heart and we saw a similar patch of recent endocarditis on the mitral valve of the fœtal heart. I have myself never heard a murmur which I could attribute to an alteration of the fœtal heart sounds and having once asked the late Dr. Matthews Duncan if he had he-

said that he had never heard any sound from which he could diagnosticate fœtal endocarditis. The heart I have shown you is the only one in which I have ever seen a true fœtal endocarditis in progress. It was clearly part of the maternal infection and it is interesting to observe that while congenital malformations described as due to fœtal endocarditis are generally on the right side of the heart in this case it was the mitral valve that was affected exactly as it was in the mother.

The tricuspid valve is found to show signs of endocarditis in a good many cases post mortem. Thus it was affected in nine out of the 20 cases mentioned above. I do not agree with those who maintain that tricuspid disease can be frequently discovered by auscultation. I incline to doubt any determination of it during life in which pulsation of the veins of the neck has not been clearly made out. And here I wish to point out the importance of teaching men as a matter of habit never to arrive at the diagnosis of any form of valvular disease from physical signs alone. Too great care cannot be given to the minute study of the physical signs in every heart, and to compare the observations of different days greatly assists to the complete knowledge of the valvular state. If the observer will write down precisely what he hears and on the next occasion do the same without looking at his former note he will learn whether what he hears is constant and if inconstant how it varies. But when the precise physical signs have been made out they must be considered in relation to the movements of the heart, to the character of the pulse, and to the general state of the patient before an opinion is expressed on the nature of the valvular disease. The most striking illustration of the importance of the pulse in relation to the interpretation of a murmur occurs when obstruction at the orifice of the pulmonary artery is the sole lesion. The loud systolic murmur audible is not always distinctly plainer at the second left intercostal space than at the right or on the sternum, and it is to be heard over a considerable area of the chest. It might easily be mistaken for an indication of obstruction at the aortic orifice but that the pulse is absolutely normal. When this is remembered the determination of obstruction at the pulmonary valves becomes one of the most certain among interpretations of cardiac murmurs. A minute search will show faint traces of affection of the right side of the heart due to rheumatic fever in a proportion of cases, but such are very rarely indeed to be found on the pulmonary valves, and still more rarely in sufficient size to cause a murmur. I have never met with an example of regurgitation at the pulmonary orifice. The clinical evidence of affection of the right heart in rheumatic fever is very small.

The left side of the heart and particularly the mitral valve, or the mitral and aortic valves, and rarely the aortic valves

alone, are the parts of the endocardium most often deeply affected in rheumatic fever, while the degenerative changes of later life and the effects of syphilis are more often seen in the aortic valves. We speak of mitral obstruction or stenosis and of mitral regurgitation. Since mitral regurgitation is often due to thickening and shortening of the tendinous cords, and since that condition is often associated with thickening of the edge of the valve and the projection of minute growths from it, it is clear that in many cases in which the chief effect is regurgitation some of the physical signs of obstruction may be present, while, on the other hand, extreme narrowing of the orifice by adhesion of the flaps of the valve to one another, though its physical signs are nearly the same as in the other case, has a different effect on the heart and on the circulation as a whole. The commonest physical signs of mitral obstruction are a thrill confined to the region of the apex beat and a presystolic murmur heard in the same region, and these are accompanied by an unequal pulse of diminished volume. The thrill is often systolic in time; sometimes presystolic or including both periods. It indicates some rigidity of the edge of the mitral valve and is never absent in that form of mitral obstruction which is due to the firm adhesion of the two flaps of the valve. A thrill may, however, be present without this adhesion, when there is a thickening of a part only of the edge of the valve or a rigid projecting growth upon its edge. If there is no localised thrill at the apex there is no mitral obstruction. If at the same time a murmur thought to be presystolic has been heard at the apex, further investigation must be made, and it will generally be discovered that it is in fact diastolic and is due to aortic regurgitation, even though it may for a time have been heard at the apex and not at the right base. Changes of position, raising of the arms, and the administration of nitrite of amyl will assist the observer.

Is a definite thrill at the apex ever due to any other cause than mitral obstruction in some form? The answer to this question is that such a thrill sometimes occurs when no other change is present but rigidity of one or more aortic valves. In some such cases the thrill may be felt more plainly still at the second right intercostal space, though definite at the apex. In others, though a thrill is only to be felt at the apex, it is certainly not due to the mitral valve and is due to the aortic valves. A constant thrill felt at the apex only is always present when the flaps of the mitral valve are adherent and thickened and also when a part of the edge of the valve is rigid or a rigid growth projects from it. When a patient has had a systolic murmur loudest at the apex and while the temperature is raised a thrill comes to be felt there which was not there before, it is often the case that adhesion of the mitral flaps has taken place and a permanent mitral stenosis been established.

Such permanent stenosis, when it is the immediate issue of an attack of rheumatic fever, embarrasses the heart, but is unaccompanied by any hypertrophy of the left ventricle. Therefore, in a case of mitral stenosis, if there be well-marked hypertrophy of the left ventricle and no chronic interstitial nephritis, other valvular disease, or adherent pericardium, it may be assumed that the primary effect of the endocarditis was mitral regurgitation, that this was the cause of the hypertrophy, and that adhesion to one another of the flaps of the valve was a subsequent event. When mitral stenosis has been proved by repeated examination to be present, and provided there is no chronic interstitial nephritis and it is known that no different valvular lesion existed in the past, and the size of the heart, including the left ventricle, is increased, then, and perhaps then only, may the diagnosis of adherent pericardium be safely made.

Here the importance of examining the heart by means of electric light may be urged. It sometimes gives an exact notion of the nature of the enlargement present. The murmur of mitral stenosis is always the most confined in area of all cardiac murmurs. The murmur of mitral regurgitation is the most widely heard of cardiac murmurs. It is loudest at the apex and generally loud over the whole front of the chest and is audible in the left axilla and at the angle of the left scapula and often over the whole back, but when it is so it is always louder at the angle of the left scapula than at the right supraspinous fossa. Is this widespread area of sound present in every case of mitral regurgitation? To this question a negative answer must be given, for if the heart is acting feebly after a patient has been lying in bed for some weeks the murmur may be very faint and not audible in the axilla or at the angle of the scapula, while a week later, when he has been up and the muscular action of the heart is stronger, while the temperature chart shows that no further endocarditis has taken place, the murmur may easily be heard at the angle of the left scapula.

Here must be considered the question of systolic murmurs which are not associated with any history of endocarditis or with any rise of temperature and which disappear altogether: the murmurs of chlorosis and of anæmic states. These may be heard at the angle of the scapula when they are loud in front. They resemble the systolic murmur of mitral regurgitation, and I believe them to be due to a temporary mitral regurgitation due to the feebleness of parts of the cardiac wall, including the musculi papillares. This hypothesis accounts for their relation to chlorosis and seems to me to rest on a basis of observed fact which is not present to support the hæmic theories. Such murmurs should be regarded as indicating a temporary mitral regurgitation, and the œdema of the feet which occurs in those who have such murmurs as due to the valvular defect.

Is a systolic murmur loudest at the apex always a proof

that a patient has mitral regurgitation? This cannot be asserted, for the systolic murmur of aortic obstruction is sometimes, though rarely, louder at the apex than at the base, and I have met with a few cases in which it was audible at the apex only. I cannot prove that this was due to a thickening of the cusp nearest the ascending part of the mitral valve, but if this were so it might explain the conduction to the apex. Such a murmur becomes faint beyond the apex and though loud there is not to be heard at the angle of the left scapula. Here, again, the character of the pulse must be taken with the auscultatory results.

It may be proper here to object to such phrases as "a mitral murmur was heard at the apex"; "an aortic murmur was heard on the sternum." By thus mixing the premiss and the conclusion in the description of a murmur the value of a record is much diminished. What is heard should be stated separately from the interpretation of that observation. The record of what was heard is an observation of scientific value only to be impugned by evidence of a defect of the sense of hearing in the observer. The clear statement of exactly what was heard, of the time of the murmur in the cardiac cycle, of the place where it was loudest, and of the area of the chest over which it was audible, enables future readers to accept or reject the conclusion drawn by the observer. The mixed statement leaves a conclusion which the future reader has no opportunity of considering, and his acceptance or rejection of it can depend on the authority only of the person making it.

The definite character of the pulse makes the determination of aortic valvular disease, whether obstructive or regurgitant or both, comparatively free from difficulty. The murmurs are the loudest of all valvular murmurs when loud, though they are occasionally barely to be heard. Aortic murmurs may sometimes be heard without touching the chest wall. I have once at St. Bartholomew's Hospital heard a murmur not indeed so loud as that described by Douglas which I mentioned in my last lecture but loud enough to be heard as one stood near the patient without stooping. It was systolic and was due to aortic obstruction. I have since heard murmurs in three cases, all of aortic valvular disease, which were audible without touching the chest.

A woman, aged 50 years, complained of pain in her left shoulder and down her left arm occurring in paroxysms. These had increased for six years in frequency and when they came on she felt stifled and could not move. I myself saw her in one of these paroxysms, and there could be no doubt that it was an attack of angina pectoris. The impulse of her heart was always greatly increased and the apex beat was in the sixth left intercostal space an inch outside the nipple line. A diastolic thrill was to be felt over the sternum at the level of the second intercostal space. A very loud diastolic murmur was audible in the second right intercostal space, down the sternum, at the apex, at the angle of the left scapula, and in the right supra-spinous fossa. The murmur was loudest at the second right intercostal space

and was preceded by a very faint systolic murmur. The pulse at the wrist was of the kind often compared to a water hammer. The diastolic murmur was distinctly audible three measured inches from the patient's sternum without stethoscope and without touching the chest or any part of the body. She was admitted to St. Bartholomew's Hospital a month later and the murmur continued to be audible without contact for a week. Then it became less loud and the anginal attacks less frequent, and it was no longer audible without a stethoscope though it always remained audible with one.

A labourer, aged 41 years, who had shortness of breath and palpitation had also attacks of angina pectoris. The impulse of his heart was exaggerated and the apex beat was three-quarters of an inch outside the nipple line in the sixth left intercostal space. A double thrill, systolic and diastolic, was to be felt all over the cardiac area. At the second right intercostal space a loud systolic and equally loud diastolic murmur was to be heard, loudest there but audible down the sternum, at the apex, at the angle of the left scapula, and at the right supraspinous fossa. The pulse at the wrist was very sudden. Both these murmurs were audible without touching the chest at a distance of three measured inches from it. After rest in bed for 14 days the murmurs could no longer be heard without touching though plain with a stethoscope.

A man, aged 22 years, had a much increased cardiac impulse. His apex beat was in the sixth left intercostal space just outside the nipple line. A very loud diastolic murmur was to be heard at the base, audible all down the sternum, and this was easily heard without touching at one inch and a quarter from the chest wall.

The murmur of aortic valvular obstruction and that of regurgitation are sometimes very faint or only audible now and then. If the patient be made to raise his arms above his head a faint murmur often immediately becomes distinct and the same effect may sometimes be produced by the inhalation of nitrite of amyl, but the former method is the better. The murmur of aortic obstruction may often be heard at the right supraspinous fossa, and this is a useful physical sign, for if a systolic murmur be heard in the right supraspinous fossa and nowhere else on the back it is a very certain sign of aortic obstruction, a fact which has not been generally observed and is not mentioned in books on auscultation, not even in the book which we have all read with enjoyment, in which Dr. S. J. Gee has in such measured and well-chosen language set forth one of the best arranged statements of knowledge on auscultation and percussion in English. The diastolic murmur of aortic regurgitation may also sometimes, but much less often, be heard in the right supraspinous fossa.

In a complicated case of valvular disease, when it is wished to arrive at an exact conclusion as to the valves affected, it is often a good plan to listen first at the back and hear what can be heard there without prejudicing the mind by listening first to the louder sounds in front.

The murmur of mitral regurgitation is often very loud at the angle of the left scapula ; the murmur of aortic obstruction is sometimes very distinct in the right supraspinous fossa. The murmur of mitral regurgitation, it is true, is often very loud over the whole back, sometimes even including both supraspinous fossæ, but this is not always so,

and in a fair proportion of cases it will be possible to distinguish the areas of the two murmurs. The murmur of aortic obstruction will rapidly become fainter as the stethoscope is moved obliquely downwards towards the dorsal vertebræ, and the murmur of mitral regurgitation will be made out to be louder near the angle of the left scapula than near the dorsal vertebræ or beyond them to the right. It may be audible up to the right supraspinous fossa, but when that is reached an immediate increase in the volume of sound is perceptible, for two murmurs each produced in the systole are heard together. A good many systolic murmurs of aortic obstruction are not audible in the right supraspinous fossa while nearly all murmurs of mitral regurgitation are audible at the angle of the scapula. In cases where both murmurs are audible behind I have proved this method of distinction by repeated observations and recommend it to the College.

A character of murmurs produced by aortic disease, when heard in the front of the chest, is their apparent nearness to the end of the stethoscope. This is often of great assistance in distinguishing each murmur when those of mitral regurgitation and of aortic obstruction are both present.

A single observation will never enable one to dogmatise about a complication of murmurs and however erroneous the opinion of some other man may appear to a listener to a heart it is wise not to say this or that cannot be heard till repeated examination has been made. Murmurs, indistinct or inaudible, often become plain as the action of the heart alters.

It is often very difficult to explain the alterations of murmurs. An adherent pericardium, not, I think, by its thickness but owing to its interference with the muscular rhythm of the heart, may produce confusion as to the valves affected. I hope this discussion of a few parts of the subject of valvular murmurs is not too trite for the College. There seems to me to be so much that is difficult in the subject of valvular disease that I have ventured to discuss not the whole of it but a series of parts in which I chance to have had good opportunities of observation.

The first attempt in England to explain the pathology of rheumatism was that of Dr. Clopton Havers of St. Catharine's College, Cambridge, in his "Osteologia Nova," published in 1691. Few have read the book, yet its account of the structure of bone has secured for its author the mention of his name with the Haversian canals by every student of medicine since his time. He advanced the view that rheumatism was due to a sort of jelly "concreted upon the superficies of the muscles." He mentions in support of this hypothesis that rheumatism was due to this mucilage or jelly a case of a curious tumour of the leg, perhaps a hydatid, since it contained "many smaller pieces of white

curd mixed with a serum," which he thought showed that mucilaginous glands existed in the muscles and became diseased, and that they were the source of the outpouring which constituted rheumatism. Since the time of Havers many pathological hypotheses have arisen on the causation of rheumatism. None of those relating to rheumatic fever need be recapitulated. Its resemblance to diseases produced by definite organisms is too close for any other hypothesis of origin to be at present probable. It might obviously be included among what Sydenham would have called the epidemic diseases of the year. The pain which it produces we know how to relieve, but when it is considered that it is the origin of most of the valvular disease of the first half of life and of some proportion of the valvular disease of the second half of life, and through them of all the distresses of slow death by dropsy and instant death by affection of the muscular tissue of the heart, as well as of all the diminution of working power which is a necessary result of valvular disease long before it has begun to threaten life, we anxiously look to the bacteriologists for a conclusive demonstration of the nature of the organism which produces it and of its natural history as an important aid to the prevention of rheumatic fever and the diminution of its incidence on the population and of its long train of disastrous consequences.

LECTURE III.

Delivered on March 30th.

Prognosis and Treatment.

Mr. President, Censors, and Fellows of the College, —The book called Προγνωστικόν in the Hippocratic writings seems at its beginning to promise to treat its subject in the widest sense in which the word can be used. τὸν ἰητρὸν δοκέει μοι ἄριστον εἶναι πρόνοιαν ἐπιτηδεύσιν. "He seems to me to be the best physician who knows how to know beforehand what will happen.' "He will treat diseases best when he shall be able from knowledge of the patient's actual state to foresee the condition of the future." These are sentences of the preamble, but as the book goes on the subject is restricted to acute diseases and to a discussion of the interpretation of symptoms, not as bearing on particular maladies, but of the meaning of symptoms in relation to all forms of acute disease. The famous passage on the Facies Hippocratica occurs in this book, and, well known as it is, I will venture to quote it, since it is a perfect illustration of how the Prognosticon deals with its subject:—

> In acute diseases the physician should make the following observations: he should examine first of all the face of the patient and should notice if his countenance is like that of men in health, but chiefly if it is like its own natural condition. This would be the most favourable condition, and the more it seems to differ from that the greater the danger. The features have attained the most extreme degree of alteration when the nose is pointed, the eyes sunken, the temples flattened, the ears cold and shrunken, the lobes of the ears prominent, the skin of the forehead dry, stretched, and rough, the skin of the whole face yellow, or black, or livid, or leaden. If from the onset of the disease the countenance shows these appearances, and if other symptoms do not furnish sufficient suggestions, the patient should be asked if he hath long watched, if he has had a severe diarrhœa, if he has suffered from hunger; the answer yes on one of these matters makes it right to think of the danger as less imminent. Such a morbid state when one of these causes has thus altered the countenance will be decided within a day and a night. But if the patient remembers no such cause and if the disease does not come to an end within that time it must be known that death is at hand.

Gnashing of teeth, movement of the hands, rapid breathing, sweats, the state of the hypochondrium, pulsations in the hypochondrium, dropsy, coldness of the hands and feet, sleep, the intestinal discharges, the urine, vomiting, expectoration, pains with fever, crises, pains in the head, sore throat, returns of fever—all these are discussed in their most general

application to acute disease. The only special application to any single morbid condition is in the remarks on pneumonia and empyema. I may mention incidentally, because I do not remember to have noticed that it is pointed out in any of the commentaries, that Hippocrates here shows that he knew that empyema sometimes follows pneumonia.

Such is the Hippocratic treatise on prognosis, an attempt to arrive at general laws upon the interpretation for good or evil of particular symptoms. At the present day the word prognosis, I observe, generally suggests to the practiser or student of medicine a single inquiry and no more. Will the patient live or die? A sounder use of the word is to include under it the whole probable future of the disease after the diagnosis has been made in each particular case. The constant practice of such a way of considering each case is certainly to the advantage of the patient, for it cannot but have an important effect upon his treatment. It is in this sense that I propose to apply myself to the consideration of the prognosis of rheumatic fever and of valvular disease.

The first question to be discussed is the duration of rheumatic fever. How long does the organism continue to live on in the endocardium? Does it after an attack die out of its condition of multiplication, so that we can say the disease is absolutely at an end, as does the pneumococcus of pneumonia? Or does it, like the tubercle bacillus, live on indefinitely in the body, maintaining its lodgment, for a time not spreading, then again multiplying and doing more injury, sometimes finally dying out? Take the first ten cases you meet of rheumatic fever, what light do they throw on this question?

A girl, aged six years, had rheumatic fever in September, 1905, and was in St. Bartholomew's Hospital for six weeks. She came in again in December, 1905, for six weeks. She was an out-patient for six months for her consequent valvular disease, mitral regurgitation, and during these months occasionally complained of joint pain. Then came an interval in which she seemed well; from August, 1907, onwards, till on Dec. 4th, 1907, she had definite rheumatic fever and was re-admitted to the hospital.

Is not the appearance of such a case as regards its lasting but intermitting effects very like one of tuberculosis? Would anyone doubt with such a history of manifestations in a case of tuberculosis that the whole was due to the effects of the original settlement of bacilli in the body? Would the intermission of raised temperature and of other symptoms from August to December in the second year give rise to any doubt on the subject of the whole being one attack of tuberculosis?

A woman, aged 24 years, had well-marked rheumatic fever in July, 1908; in March, 1908, she had had an attack and had had one in 1904.

Supposing this patient had had hæmoptysis in 1904 and well-marked pulmonary symptoms in March and July, 1908, should we not have expected to find tubercle bacilli in her

sputa in July, 1908, and should we not be certain that her illness began in 1904?

A woman, aged 25 years, who came into St. Bartholomew's on account of palpitation and shortness of breath in January, 1908, had mitral obstruction and regurgitation but no present pains in her joints or fever. She had had rheumatic fever in 1899, and in 1904 had been admitted to St. Bartholomew's Hospital for rheumatic fever and had stayed in hospital for six weeks. She had had rheumatic fever again in October, 1906, and was in bed for ten weeks. Since then she had had occasional pains in the joints.

If this patient had had dysentery in India in 1899, had got over it and had returned to England, and had been admitted to the hospital in 1904 with a chronic diarrhœa, with blood and mucus discharged from the bowel, and after treatment had got well and left and had come back again in October, 1906, with a similar diarrhœa, and if after seeming to recover she had now and then passed a little blood and mucus, would not much the most probable explanation be that her Indian infection was the cause of all the prolonged symptoms and that if she died we should find imperfectly healed old ulceration with scar tissue near it in the large intestine? Should we not venture to state as we went round the ward that that anatomical change was then present in her intestine and that the whole dated from her attack of dysentery in India in 1899?

A woman, aged 24 years, was admitted to one of my wards on Oct. 3rd, 1908, with rheumatic fever, of which the pains had begun on Sept. 21st. The pain and swelling of joints were well marked and she had mitral regurgitation. In 1900 she had chorea which lasted for seven months.

Might not this case be compared to that of a patient who had an attack of pleurisy on the left side, the nature of which was undetermined at the age of 16 years, and who had developed well-marked tuberculosis of the lung on the left side at the age of 24 years? Would it not be possible that the original infection had remained, that the pleurisy was due to a settlement of the tubercle bacillus which had since been so inactive as to produce no symptoms noticed by the patient, and which in 1908 under some favouring circumstances had been able to grow and produce the tuberculosis? If the original pleurisy had been shown to be tuberculous should we not at once admit that the tubercle of the lung eight years later was a probable development of the same infection? I have chosen this comparison because while we cannot be certain that an attack of chorea which we have not witnessed was of the variety which undoubtedly belongs to the series of phenomena of rheumatic fever, the presumption is strong that any chorea which occurred in the early life of a young patient with rheumatic fever was of that variety.

A woman, aged 24 years, was admitted to hospital on July 8th, 1908, with valvular disease, mitral regurgitation and aortic obstruction and

regurgitation, but without symptoms of rheumatic fever. She had had rheumatic fever when aged eight years and had had 12 attacks since the first. The last attack was some time before May, 1908, so that she had 13 definite attacks in 15 years.

In the absence of an exact history of each attack and of the intervals we can only apply our knowledge of the frequency with which some pains appear between serious attacks, and the probability seems to be that this was one long period of rheumatic fever—that is, not of continuous fever, but of continuous disease due to the original infection, and to the constant presence of the organism to which rheumatic fever may be believed to be due.

A woman, aged 18 years, was admitted to hospital with rheumatic fever on August 24th, 1908. She had mitral obstruction and regurgitation. She had been admitted on three previous occasions with rheumatic fever and had thus had it under observation in 1898, 1900, 1902, and 1908.

Here again, if the analogy of tuberculosis has any force it may be applied to enable us to accept the probability that a particular organism was present in her system, probably in her endocardium, for ten years.

A boy, aged 16 years, was admitted to hospital on April 24th, 1907 with rheumatic fever. He had at the time mitral regurgitation and aortic obstruction and regurgitation. In 1896, when aged five years, he was in bed for six months with rheumatic fever; in 1897 he had chorea for seven weeks; in 1898 rheumatic fever; in 1899 rheumatic fever for eight weeks, and in 1904 another attack.

Supposing this boy had acquired the hæmatozoon of tertian ague in a malarious region in 1896 and had soon after moved to a healthy part of England we should have had no difficulty in believing that a recurring tertian fever was due to the parasite. I have myself observed a case in which a tertian ague continued at intervals for nine years, and have read of longer liability to the fever. The possibility of obtaining the hæmatozoon from the blood of course supplies a test of the continuity which must be absent while the micro-organism of rheumatic fever is unknown.

A boy, aged 12 years, was admitted to hospital with rheumatic fever in January, 1907. He had mitral regurgitation. In the summer of 1903 he had had an attack of rheumatic fever and since had had pains in his joints with fever at intervals.

A boy, aged 11 years, was admitted to hospital with rheumatic fever and had mitral regurgitation. His present attack seemed to have begun on Nov. 1st, 1906, and he had a similar attack three years before.

A boy, aged nine years, was admitted to hospital with rheumatic fever on Sept. 24th, 1904. He had disease of both mitral and aortic valves. In 1903 he was in the hospital with rheumatic fever for nine weeks and was readmitted in May, 1904, and was under observation till the beginning of August.

The history in this last case for more than a year was nearly continuous, and in this, as in the former two, the

analogy of a development of the tubercle bacillus may be used to explain the course of a disease dependent on the organism of rheumatic fever.

It would, of course, be easy to mention many more such cases, but I have perhaps said enough to satisfy you that there is nothing unlikely in the view that the several attacks of rheumatic fever from which so many patients suffer are really successive developments of an organism which remains in the endocardium throughout the series of attacks. Thus, using the word rheumatic fever for the condition present when this organism is in the system, whether developed or in a potential state of development, I arrive at the conclusion that rheumatic fever has a variable duration. It sometimes, though rarely, lasts two months and no more. It frequently lasts from three to ten years and may last longer still. A short attack is more probable after 30 years of age than before. A long attack is most likely to occur when the disease begins in early childhood.

The answer to the question whether the patient will live or die in the first attack must, of course, be that he will not die in it. The cases of death said to be due to a first attack of rheumatic fever are generally diminished in number when minutely examined. Thus, a woman, aged 26 years, in St. Bartholomew's Hospital, under the care of the late Dr. Reginald Southey, had a loud systolic murmur, plainest at the apex, and some swelling of the right knee and fever. She had been ill for seven days, and she died three days later. It was supposed that she had rheumatic fever, but the post-mortem appearances seemed inconsistent with this view, though the endocardium was affected, and there were no signs of ulcerative endocarditis. The pericardium was adherent and in one part was calcified. The whole heart was dilated. The endocardium of the right auricle showed 18 small white specks, circular and encircled by an area of engorgement. These seemed on microscopic examination to be localised hæmorrhages of old standing, but what their nature was not discovered. There were 250 in the right ventricle, some in the left auricle, and many in the left ventricle. The valves had no such specks on them, nor had they any recent growths on them, though both the tricuspid and the mitral were incompetent. There was a small patch of recent lymph on the wall of the left auricle. The peritoneal surface of the intestines, the kidneys, and the liver contained similar specks, and it seemed possible that the disease was, in fact, a pyæmia, and that these were minute abscesses of various dates. As the post-mortem examination was made in 1879 the test of cultivation was not used.

That the illness beginning with a first attack of rheumatic fever will ultimately by the way of valvular disease be the cause of death is true of a large percentage of cases. In a smaller percentage the valvular disease, while not itself the immediate cause of death, is a chief contributing cause in

some such acute disease as pneumonia. In another group of cases of valvular disease descended from an attack of rheumatic fever, a pericarditis perhaps of the same infection, perhaps of a pneumococcus or other organism, is the cause of death. In a small percentage again the rheumatic fever is a remote cause of death, since it has prepared the endocardium to receive the infection of ulcerative endocarditis or of influenza. In ten cases of ulcerative endocarditis an endocardium damaged in long past attacks of rheumatic fever was found in five. Thus rheumatic fever must be regarded as leading to a large sacrifice of life in at least four different ways of death. Will the endocarditis certainly leave permanent damage to some valve or valves? To this the answer is that a small percentage of patients receive no permanent damage, that a further small percentage receive damage, the signs of which after a lapse of time disappear, but that a large proportion of patients do acquire permanent valvular disease. It must be added that the proportion of cases which do not end in permanent valvular disease may certainly be increased by judicious treatment, just as judicious treatment diminishes the mortality in enteric fever, and that the commonest cause of a further continuance of the disease is letting the patient get up too soon.

The true result of an attack of rheumatic fever on the valves of the heart cannot be determined till after the patient has been up and about for some time and the tone of the myocardium has been completely restored. The injury to the valves may then prove to be much less than was before expected, and now and then it may prove to be unexpectedly greater. The valvular damage can as a rule be accurately determined three months after the patient's convalescence. The commonest valvular lesion after rheumatic fever is mitral regurgitation. The more often the febrile attacks are repeated the greater will be the degree of injury to the valves. In every case of rheumatic fever it is safe to predict that there will be no permanent damage to the joints. These are the chief points in the prognosis of rheumatic fever itself.

The prognosis of the several forms of valvular disease may properly begin with the general statement that death in mitral disease tends to occur gradually with long-preceding dropsy, and that in aortic disease, while the same method of termination may occur, there is also the permanent risk of a sudden termination of life.

The post-mortem appearances of the heart in cases of gradual death from valvular disease show that it is the enfeebled or degenerate condition of the myocardium which is the cause of the general dropsy and of the patient's death. The post-mortem appearances in cases of sudden death with disease of the aortic valves sometimes include a degenerate myocardium but in other cases show muscular tissue in which degeneration has not begun.

The temporary recovery which patients make from a con-

dition of cardiac embarrassment, even if associated with extensive general dropsy, shows that an enfeebled myocardium may attain strength again and be able for some time longer to do its work. If at the supposed end of an attack of rheumatic fever a patient is left with distinct mitral regurgitation what will be the effect of that lesion upon his physical future and in what ways is that future affected? The first danger before him is that endocarditis is merely dormant, a danger to be met only by taking the fullest pains to ascertain that the attack is at an end. Further, rheumatic fever always means increased injury to the first affected valve with the possibility of injury to other valves. Let us suppose that no further endocarditis occurs. The imperfection in the circulation caused by the damaged valve is likely to be remedied by hypertrophy of the parts of the myocardium affected, of which the left ventricle is part. The apex beat, therefore, is found lower down and further to the left than in a normal heart. The degree of hypertrophy of course depends on the difficulties to be overcome. To what extent will it go?

The heaviest heart due to mitral regurgitation alone which I have myself met with post mortem weighed 22 ounces. It was that of a man, aged 20 years, who had no chronic interstitial nephritis and no adherent pericardium or other valvular lesion than mitral regurgitation. The degree of hypertrophy in a man does not often make the heart exceed 16 ounces in cases of mitral regurgitation. A patient, aged 12 years, with mitral regurgitation may in the course of from six to eight years attain such a good working heart that he only feels the presence of the valvular lesion after great exertion. Alcohol will sometimes relieve the cardiac uneasiness which he feels after fatigue, and I have seen a few cases of chronic alcoholism which seemed traceable to the endeavour thus to overcome the sensations produced by an overworked heart with mitral regurgitation. The heart that has accommodated itself in course of years to a considerable mitral regurgitation will go on working well for years, and even into old age if it be not affected by fresh endocarditis of any kind and is not exposed to unfavourable conditions, of which too great physical exertion and prolonged mental distress or anxiety are the most important, and the next in importance such diseases as pneumonia and influenza. These are particularly dangerous to such hearts in two ways —the pneumococcus or the bacillus of influenza may attack the old damaged endocardium or they may originate pericarditis; and in either condition, besides the embarrassment due to it, considerable enfeeblement or further change in the myocardium must be expected and generally occurs. In this way such an attack is often fatal. Over-physical exertion seems to strain the muscular tissue and thus leads to great irregularity in the action of the heart, but rest and treatment may restore the heart to its former condition.

Prolonged mental distress seems to have even a more severe effect on the condition of such a heart than too great physical exertion, and the restoration in such cases is often a matter of extreme difficulty. The condition of the heart seems directly traceable to the disturbance of the mind or of the feelings, and as the illness goes on with these unrelieved the condition of the heart itself seems to react on the mental state and to aggravate it. Such patients sometimes recover very slowly; sometimes the natural ultimate failure of a damaged heart seems hastened and they die from dropsy, but I have seen several in whom an attack of pericarditis—not extensive or with great effusion—terminated life.

In mitral regurgitation aches are occasionally felt in the heart wall, not confined to one spot but extending from base to apex. Very severe spasmodic pain is also sometimes felt which might be mistaken for angina pectoris, but when watched is seen to be different, in that it lasts a much longer time, often several hours, and does not give any sense of actually impending death. The occurrence of simple emboli and of right hemiplegia due to such an embolus are occasional incidents of a case of mitral regurgitation as also of aortic valvular disease and add further to the long list of the consequences of rheumatic fever.

If instead of merely allowing regurgitation the mitral orifice is greatly narrowed by adhesion of the flaps of the valve to one another, so that there is well-marked mitral stenosis, the patient will have a still less easy life, more palpitations, a more frequent sense of cardiac irregularity, and less power of work, and will altogether be in a more distressing condition, and one in which there are fewer possibilities of improvement by way of hypertrophy or in any way. The patient with mitral regurgitation, as we all know, may often live to old age; the patient with mitral stenosis very rarely indeed. The narrower the orifice the fewer will be the patient's years. The heart may be capable of very nearly regular action in a few cases, but even in these very little extra exertion is sufficient to make it embarrassed and very irregular. The lungs are permanently engorged and attacks of bronchitis are frequent. A distress which recurs after relief, and with each recurrence grows worse, is pain in the liver. The tenderness to the touch may be extreme and often there is a sense of stretching there which amounts to acute pain as the patient lies still. Sometimes there is a similar pain in the spleen. The urine becomes diminished in quantity and albuminous. The general discomfort may be increased by piles; ascites and anasarca of the legs and arms appear, and a continued reduction in the quantity of the urine which diuretics affect but little points to the constant engorgement and gradual hardening of the kidneys. Treatment and rest give relief, but it is rarely long-continued, and the patient's life, even if carefully regulated, consists of little more than longer or shorter

intervals between painful attacks. At last the cardiac irregularity and tumultuous action are incessant, the dropsy becomes inveterate, the engorgement of the liver cannot be diminished any more, constant œdema of the lower lobes of the lungs is present, the imperfect aeration of the blood affects the brain, and the patient has temporary delusions when awake and dreadful dreams during restless and often interrupted sleep. Mitral stenosis is the most distressing of all the common forms of valvular disease. It gives the patient least ease and soonest terminates his life. If it has begun in the girlhood of a woman she seldom lives beyond 40 years of age if she is married and has children, or much later than 50 years if she lives unmarried under the most comfortable circumstances. I have seen very few men with mitral stenosis who reached the age of 50 years. I think it is the cares of a household rather than childbearing which makes the lives of married women with mitral stenosis shorter than those of spinsters, for during pregnancy the patient is often much less distressed by her heart than at any time, though after childbirth she sometimes perishes owing to the onset of a fresh endocarditis. The addition of tricuspid stenosis increases the obvious irregularity of the heart and adds to the distress and diminishes very much the duration of life. From each side of the heart simple emboli may be shot into the circulation and may produce hæmoptysis or hæmaturia, while other hæmorrhages may occur as results of venous engorgement.

The hypertrophy produced by mitral disease seldom increases the heart to more than twice its natural weight. That produced by disease of the aortic valves often does so and may be the origin of a hypertrophy reaching 30 or even 38 ounces. It is important to bear this general fact in mind in cases in which the cause of a systolic murmur is difficult to determine. If there is very great hypertrophy (and no other present cause, such as adherent pericardium or chronic interstitial nephritis), if with a heaving impulse the apex beat is in the sixth space and beyond the nipple line, then that hypertrophy alone makes it probable that aortic disease is present. Such cases generally terminate in a sudden death, and of all causes of transition from what seems fair health to immediate death aortic valvular disease is the most frequent. This sudden death may be very long deferred. Thus a patient who probably acquired aortic valvular disease in 1837, after a well-regulated life in which it rarely troubled him, died suddenly from his aortic disease while dressing one morning in 1900.

Angina pectoris is to be regarded as a likely incident in this form of valvular disease. Its occurrence at once, and generally correctly, suggests a near termination, but sometimes the patient goes on living a very long time. A man whose aortic valves became diseased between 1877 and 1880 found that hard mental work affected his heart soon after 1890

when he was 43 years of age, and in 1898 he had definite angina pectoris which never left him but continued for ten years. I have met with several other instances where a man who seemed likely to die very soon yet lived ten years under similar circumstances.

Such are a few of the points of prognosis in rheumatic fever and in valvular disease. They all involve a repeated consideration of the patient's state and of his account of it. In receiving such accounts of past attacks and pains a literal interpretation of words must not be used nor too much information expected. Johnson in his noble poem on the death of Levett, as he repeated it to Boswell—

> " In misery's darkest caverns known
> His ready help was ever nigh,
> Where hopeless anguish pour'd his groan
> And labour steals an hour to die "

—has a last line afterwards altered by him, which expresses with the force of true poetry an observation on human life made among those who can but just earn their living, the justice of which we, who have all of us known and respected in the wards of our hospitals many such people, can confirm. How many a man and woman have we seen who seemed barely able to spare the time to die and who had amid laborious occupations neglected to record in the mind or to remember illnesses not great enough to prevent going to work.

There is another cause which obscures a man's life-history and which makes him subdue the sensation of cardiac irregularity and go on till the time for resting his heart is almost past and degeneration of the myocardium has begun. Alcohol at this serious and fatal cost obscures his condition from himself and keeps him going when he ought to be in bed. It does much to obscure the gradual, and often to be relieved, course of the valvular disease due to rheumatic fever and leads men to think symptoms sudden which are in reality but the last steps of a very gradual descent. Cases of this kind, of which there are very many, tend to spread the belief that death from valvular disease is a more rapid process than it really is.

Such are the points of prognosis which have struck me most.

The treatment of rheumatic fever proposed by Sydenham was based on the theory that it was an inflammation supported by the observation that the blood drawn from the rheumatic patient presented the same appearances as that drawn from one with pleurisy, a condition then universally described under the pathological heading inflammation. His first proceeding was to order ten ounces of blood to be taken from the arm on the side affected. The next day the bleeding was repeated ; after one or two

days there was a third bleeding, and after three or four days more a fourth bleeding, which was generally the last. A cooling julep, which was little more than a draught of sweetened water, was to be taken at the patient's pleasure. The painful joints were to be relieved by a poultice of white bread tinctured with saffron or by the repeated application of a cabbage-leaf. The diet was one of barley and oatmeal broth, all meat or meat broths being absolutely forbidden. The patient was allowed to drink small beer, everybody's daily drink at that time, or ptisans of barley, liquorice, or sorrel boiled in water. He was to keep some hours every day out of bed. On the alternate days to the bleedings enemata of milk with sugar were given and for eight days after the last bleeding. After that a purge was to be taken in the morning and the same evening a large dose of diacodium in cowslip water. Then the patient was allowed gradually to return to his wonted way of living with one caution—that he should drink no wine and no spirits and should avoid salted and spiced meats and anything difficult of digestion for a long time. "Pains," says Sydenham, "will thus be lessened, but will remain about for a long time."

Dr. Peter Mere Latham, who wrote in 1845 and whose knowledge of the practice of his day went back to a few years before the battle of Waterloo, had seen rheumatic fever treated by bleeding, by opium (2 to 5 grains every 24 hours), by calomel, by colchicum, and by drastic purgatives. When he was a student at St. Bartholomew's Hospital from about 1810 to 1814 the treatment of rheumatic fever usual there was to give a dose of liquor ammoniæ acetatis three times a day and an opiate at night—a humane method perhaps traceable to the practice of the enlightened David Pitcairn. He himself thought bleeding expedient in many cases. His view about it in the reign of Queen Victoria was much the same as that of Sydenham in Charles II.'s. He had used opium with success, though as, unlike Sir William Gull, he had never thought it right to leave cases quite untreated, he had, he admits, no sufficient standard of comparison to tell him whether he had done more than relieve pain. On the whole, he thought that the best plan of treatment was that by large (10 to 20 grains) and repeated doses of calomel followed by purgatives. I was a clinical clerk at St. Bartholomew's Hospital about a quarter of a century after the end of Dr. Peter Mere Latham's active life in his profession, beyond which his honoured age lasted for many years. His method was entirely obsolete and the general method of treatment of rheumatic fever was by alkaline salts. This method and several others have since become obsolete, and for the present, so far as the Pharmacopœia is concerned, it seems clear that the salicylates are much more efficient than any remedy of past times. They lead to a rapid cessation of the pain

and swelling of the joints, and when continuously administered in the dose proper to each patient over several weeks they seem, so far as the temperature chart and the absence of further symptoms enable one to judge to prevent the further development of the organism in the endocardium or to destroy it altogether. The difficulties of administration which occur in particular individuals can generally be overcome by a little ingenuity in prescribing. A more rapid extermination of the organism is desirable, and for this we have yet to seek a drug. For the present that which we have does a good deal for the relief of the patient's pains while it is not inoperative in the process of terminating his malady.

We rightly regard the keeping the patient sufficiently long in a condition of absolute rest in bed as one of the greatest modern improvements—as compared with the practice of past times—in the treatment of enteric fever, and I think we may believe that there is no practiser of medicine in England who fails to carry it out thoroughly. I wish to urge the importance of a similar unanimity with regard to rheumatic fever. My experience leads me to the belief that there is always fear of re-development of the disease if the patient is allowed to leave his bed till his temperature has been absolutely normal, without any rise whatever above the normal line, for three weeks at least from the last rise. After this he ought long to be watched day by day with the aid of a temperature chart; and if in the week of his leaving bed or later any definite rise is observed, he ought to go back to bed for a further three weeks. If no rise occur he ought still to continue under observation for three months, if possible, taking salicylates or similar drugs in adequate but diminishing quantities, and all this time his life should be so ordered that no strain is put upon his heart. If he is a boy at school he is not to take part in games all this time. If he is at home he is to take carefully regulated exercise and to have one rest or more in the day as well as a long night.

Perhaps in hospitals it may prove that three weeks of normal temperature is not enough before allowing the patient to get up. This may be discovered when three weeks have come to be generally adopted as a minimum period. I am certain that an earlier day of getting up only leads to prolongation of the disease.

One point which I wish to urge about the valvular disease due to rheumatic fever is the importance, when we see such cases, of taking the temperature or asking for a temperature chart. Thus only can we be certain that endocarditis is not present and that our treatment ought not to require that the patient stay in bed. Most physicians have seen children who walked to the hospital and who had the day before played about who, when examined, had raised temperature and such marked alteration of cardiac sounds that it was certain they

had had endocarditis for several days. Arthritis was, of course, present, but of some small joint and causing only a little pain. Thus, when seeing a case of valvular disease in a young person, the first question to which the observer must ascertain the answer is not, when had he rheumatic fever, but has he got rheumatic fever at present. Thus the observer will come to know whether he has to treat a condition of acute endocarditis or one in which he is to try to relieve the heart in difficulties which it suffers from this or that simple or complex valvular defect.

As to this last part of the subject, it would be obviously impossible within the limits of these Lumleian lectures to deal with it. If, like the accomplished Sir Charles Scarburgh, versed alike in medicine, in anatomy, in Greek, and in mathematics, I were to hold the lectureship for 38 years I might perhaps, if I had his abilities as well as his time, exhaust all that there is to be said on valvular disease in all its forms. Or if like the learned and judicious Dr. Richard Powell, the first describer of facial palsy, I were to hold the lectureship for ten years, I might make considerable progress in the statement of sound principles of treatment and the refutation of much that is foolish which has been said about it; but my measure is smaller and my course of lectures is finished. Your time and mine will not have been wasted if I have persuaded you, and through you our profession at large, (I.) that rheumatic fever is a single definite disease; (II.) that endocarditis is always an essential part of it; (III.) that its duration may extend over many years, and that these circumstances, but half demonstrated as they necessarily are at present, are still the safest indications of method in the treatment of the disease.

Lord Lyndhurst, the Chancellor, once said in conversation, "I consider that the worst exaggerator is the person who understates." The remark was original and wise. It is illustrated in the books of many writers on rheumatic fever. They understate the conclusions which their own experience would allow. I have tried to avoid this fault and to state the conclusions to which my own experience has led me without abatement and without reservation. I have ventured to risk the censure which might attend such a course because I believe that the adoption of these opinions would not merely lead to a saving of lives but also to the prevention of many of the fatigues, disappointments in work, checks in usefulness, and other inconveniences which follow the valvular disease consequent on the endocarditis of rheumatic fever.