CHRONIC INFECTIOUS ENDOCARDITIS

By WILLIAM OSLER[1]

An endocarditis with fever as its only symptom may be prolonged for weeks or months under many different circumstances. Following rheumatic fever in a child an endocardial complication may keep up a temperature of from 100° to 101° for several months, during which time there may be no other symptoms and the general condition may remain fairly good. In chronic valvular disease in the stage of broken compensation slight irregular fever may persist for months, associated with the presence of fresh endocarditis. As a rule, the form of endocarditis to which we give the term infective, septic, or ulcerative runs its course under three months. That occasional instances were characterized by a very protracted course was noted by Wilks, Bristowe, Coupland, and Lancereaux. In my Goulstonian Lectures 1885, I stated that this type had the following characteristics: the fever was irregular and intermittent, resembling ague; the cold, hot, and sweating stages might succeed each other with great regularity; in the intervals fever might be absent; two or three paroxysms could occur in the course of a day. In many of the instances the disease was prolonged to three or four months, and I give the notes of a case of Bristowe's, in which the condition persisted for five months. The recurring chills usually led to the diagnosis of malaria and also gave rise to the opinion widely held, particularly by French writers, that ulcerative endocarditis could be caused by this disease. The cases to which I wish to call attention in this communication are of this chronic character, not marked specially by chills, but by a protracted fever, often not very high but from four to twelve months' duration. At the time of the delivery of the Goulstonian Lectures I had not seen a case of this type. In the past twenty years I have seen ten cases of this form, two of which I have already reported (*Practitioner*, 1893). I have put them together in tabular form to indicate their main features.

[1] Read at the Association of Physicians of Great Britain and Ireland, Edinburgh, June 12, 1908.

[Q. J. M., Jan. 1909.]

It has long been recognized that malignant endocarditis is really an acute septicaemia with localization on the endocardium, but the symptoms are not necessarily due to the local lesion. The clinical picture is a septicaemia sometimes of a typhoid type, sometimes like a pyaemia—then again with predominant meningeal symptoms, occasionally with pronounced cardiac features. The pneumococcic, the gonorrheal, and the streptococcic forms present, as a rule, a picture in which the heart-symptoms are in the background. Cases of infection with these organisms may run an identical course without any endocarditis. On the other hand, there is a large group of cases in which the endocarditis plays a more important rôle and the vegetations and ulcerations appear to be directly responsible for the fever and the associated symptoms. As a rule, the valves involved are already the seat of a sclerotic change. The source of the infection is rarely to be determined. Thus, in only one of the series here reported was there an external lesion. The patients in this series were all adults, five women and five men. In six there was a past history of rheumatic fever; eight had old mitral lesions, two aortic, well compensated, and not giving any trouble at the time of the onset of the symptoms. It was not always possible to get a definite history of how the attacks began. In five of the cases there were chills and fever, mistaken for malaria. Cough and loss of weight in some cases suggested tuberculosis. The slight fever without any localizing symptoms may raise the suspicion of typhoid fever. In my series these have been the three diseases the diagnosis of which has been suggested. Once established the fever becomes the dominant, and for months may be the only, symptom. This is the most striking peculiarity of the cases. Week after week, month after month, the daily rise of one and a half or two degrees may be the only indication there is of an existing mischief. In Case I, in which the fever lasted for thirteen months, the patient's sister, a trained nurse, had decorated the room with yards of the temperature charts; fever with an occasional sweat were the only symptoms. The appetite remained good and she lost very little in weight. There were no embolic features and from month to month there were few, if any, changes in the cardiac condition. In this very protracted form chills are not nearly so common as in the more acute cases, nor is the fever so high, not often reaching above 102·5° or 103°. It is of a remittent type, not falling to normal at any period of the day. With the occurrence of a chill the temperature may rise to 104° or 105°, but in none of the cases was there the type of fever in which the paroxysms recur with great regularity—quotidian or tertian, as we see so often in the acute forms of ulcerative endocarditis. Another peculiarity is the occurrence of periods of apyrexia, usually towards the end, but in one or two of the cases there were afebrile interludes which gave deceptive promise of recovery. It is well recognized now that fever is not an invariable accompaniment of endocarditis. Following pneumonia there may be for months a slight toxaemia with little or no fever in connexion with a patch of endocarditis.

The cardiac features in this group are usually well marked, but as a rule

indeed for several months, there may be only fever, and unless there have been special features pointing to the heart, such as the development of a diastolic murmur or the great intensification of a mitral bruit, it may be impossible to settle the diagnosis. There are, indeed, cases in which from beginning to close no heart murmur has been present. By far the most suggestive features are: (1) a knowledge of the existence of an old valve lesion. This was present in every one of my series. (2) The occurrence of embolic features, sudden swelling of the spleen, with friction in the left flank, sudden attack of haematuria, embolism of the retinal arteries, hemiplegia or the blocking of a vessel in one of the limbs. (3) The onset of special skin symptoms, purpura, and more particularly the painful erythematous nodules to which I have referred. Present in seven of the ten cases, these are of definite diagnostic import. They are in all probability caused by minute emboli. (4) The progressive cardiac changes, the gradual increase in the dilatation of the heart, the marked change in the character of a mitral murmur, the onset of a loud rasping tricuspid murmur, or the development under observation of an aortic diastolic bruit.

With carefully made blood-cultures one should now be able to determine the presence of the septicaemia. This was easily done in three of my more recent cases. An onset with chills and fever and slight swelling of the spleen almost always leads to the diagnosis of malaria, more particularly in regions in which this disease prevails, but in not one of my cases was there any difficulty in excluding this by careful microscopical examination of the blood. It was not always possible to convince the physician. With slight cough tuberculosis may be suspected, as happened in two or three cases of my series. For many weeks the patient may present nothing but a pyrexia, of doubtful origin, or a cryptogenetic septicaemia, and as he may look very well and may feel very well, and there are no special symptoms, and with a heart-condition that may have remained unchanged for years, it is not easy to reach a positive diagnosis. The blood-cultures and the presence of the painful erythematous nodules and the occurrence of embolism furnish the most important aids.

The anatomical condition in these cases is quite unlike that of the ordinary ulcerative endocarditis. In the three specimens I have had an opportunity of studying there was no actual ulceration, but large proliferative vegetations, firm and hard, greyish yellow in colour, projected from the endocardium of the valves like large condylomata, encrusting the chordae tendinae and extending to the endocardium of the auricle. The condition is quite unlike the globose vegetations of the pneumococcal and gonorrhoeal endocarditis or the superficial ulcerative erosions of the acute septic cases.

The organisms responsible for this condition have been carefully studied. In my series cultures were made in six cases. In three they were negative. In two streptococci were present, in one a staphylococcus. While, as a rule, this condition is much more commonly caused by the streptococcus other organisms may be present. Thus Fraenkel has reported one instance of a pneumococcus endocarditis persisting for nearly six months (*Deutsche med. Woch.*, 1900). Of

sixteen cases of this chronic form, the clinical course of which extended from four to eight months, Harbitz (*Deutsche med. Woch.*, 1899) found pneumococci in four, streptococci in nine, and in eight other micro-organisms. Lenhartz (*Deutsche med. Woch.*, 1901), who has reported sixteen cases with a duration of from three to seven months, found staphylococci and streptococci the common organisms, the pneumococcus once and the gonococcus once. In the majority of cases it seems to be a mild streptococcus infection, possibly by a special form. Possibly in some instances there may be a special resistance on the part of the host, but these are points which must be settled by future investigations. These are cases in which the possibility of successful vaccine treatment should be considered. It was tried in two cases of my series, but in both rather late, and in neither did it seem to have special influence. Horder has treated a case of this chronic type with a vaccine prepared from the patient's organism, but without success. The results in the acute forms are discussed by him in the *Practitioner*, May, 1908. Abstracts of the cases are here given.

Case I. J. M., aged 28. I saw this patient with Dr. Mullen of Hamilton, Ontario, in 1888, during my occasional visits to that town, on my way to Toronto. A point of special interest is that the sister of the patient, a trained nurse, had kept a very accurate temperature chart from July 17, 1888, to July 7, 1889, nearly twelve months. Sheets of the four-hourly temperature charts, pinned up on the wall of the bedroom, provided a very remarkable picture.

The patient had had good general health, but at twelve years of age had had rheumatic fever. In February, 1888, she got cold and had pain in the chest. Early in the summer she began to feel badly and had attacks of faintness and the fever came on in the afternoon. When she returned to her home in the first week of July the temperature was as high as 104° in the evening, and she was thought to have typhoid fever. The fever persisted and she had profuse sweats. I saw her in the end of September, and though a systolic murmur was present I did not appreciate that the condition was one of endocarditis. I saw her again at Christmas time, when she seemed very much the same, except that she had been having severe rigors followed by very high fever and profuse sweating. This was the first case in which I noticed the remarkable skin lesions. She had a great many crops which were at first thought to be urticaria. Dr. Mullen's description is most characteristic: 'The spots continue to appear at intervals. They are erythematous, some as small as a pea, others a centimetre and a half in diameter with a white point in the centre. They often pass away in a few hours and rarely last longer than the evening of the day on which they appear. They are not numerous. The commonest situation is near the tips of the fingers, which for a short time become swollen.' These spots were seen more or less throughout the illness, less towards the close than at the early part. At this visit at Christmas we made up our minds that the condition was one of endocarditis. The heart murmur had intensified and there were signs of dilatation of the organ. I saw her again in April, 1889, when there was little or no change, except that she was weaker. She died July 7, 1889, more than thirteen months from the onset of the illness. Dr. Mullen very kindly sent me the heart for dissection. The mitral valves were a little thickened; the orifice admitted two fingers. The margins on the auricular side were covered with large vegetations, many of them extending on to the wall of the left auricle. The chordae tendinae were shortened and thickened and encrusted with vegetations. There were signs of old infarcts in the spleen and kidneys.

Case II. T. B., aged 43, admitted to the private ward, Johns Hopkins Hospital, March 13, 1892, complaining of weakness and fever. He had had very good health, with the exception of an attack of typhoid fever twenty years previously and chronic malaria when a lad. Early in December, 1901, he began to have loss of appetite, malaise, and fever with enlargement of the spleen. The fever was of an intermittent type, ranging from 102° to 103°. He had occasional sweats. The spleen was enlarged, and very naturally the condition was thought to be malaria. Throughout the winter the temperature persisted and he had cough, and there was a loud systolic murmur detected at the apex. When admitted to hospital the examination was everywhere negative, except in the heart, the impulse of which was in the sixth interspace, three centimetres outside the nipple line. There was a loud systolic murmur of a musical quality heard as far as the angle of the scapula. The sounds at the aortic cartilage were clear. There was no anaemia. The patient was under observation from March 15 to May 10. The temperature rose daily to between 102° and 103°; about four or five o'clock in the afternoon he sweated. He gained slightly in weight. He complained a little of pain on the left side in the splenic region. Throughout May and June the temperature range was from 97° to 103°. In July the fever was less marked. There were several days when the temperature was almost normal. Early in July for the first time the petechiae appeared. At intervals there were very profuse sweats. Throughout August and September there were groups of days in which the temperature was normal or subnormal, sometimes as low as 95°. He died September 16, about ten months from the onset of his illness. The autopsy by Dr. Block showed an extensive mitral disease. The ventricular surfaces of the valves were studded with enormous masses of vegetation. The chordae tendinae were thickened and encrusted with firm yellow outgrowths. The aortic valves and those of the right side were normal. The spleen and kidney showed infarcts.

Case III. Florence M. D., aged 21, seen March 16, 1899. The patient was well and strong as a girl; at seventeen she had severe anaemia. Through the summer she was very well, but tired easily on exertion. In October she began to have feelings of chilliness and irregular fever, and sometimes the joints were a little stiff and sore, but never red. The doctor thought she had slight rheumatic trouble and gave her salicylates, but she never got perfectly well and grew pale and nervous. She had a little cough and it was suggested that she might have tuberculosis. Early in February she had a severe chill. Subsequently she had slight ones at intervals, following which the temperature would rise to 103°. It was then discovered for the first time that she had heart trouble. As she had a cough as well, it was decided to send her South and she was brought to see me on the way through Baltimore.

She was a tall, well-nourished girl, looking a little pale. I was surprised to find the temperature above 103°. The pulse was rapid. The heart's action was violent, the apex beat in the fifth space outside the nipple line. There was a very intense apical systolic murmur, transmitted loudly to the back and also heard in the left sternal margin. There was a soft bruit at the aortic cartilage. There was no swelling of the joints, but on the radial side of the first phalanx of the right index finger were three raised red spots, each about a centimetre in diameter and very tender. They appeared that day, and she stated that they came at various places on her hands and feet and lasted two or three days. The patient was so ill that they were not able to proceed on their journey, and I saw her at intervals for the next six weeks. Symptoms of severe endocarditis increased. Early in April she had left hemiplegia and the spleen enlarged. Numerous crops of the painful spots of the skin came out, four or five at a time, usually about the hands and feet, occasionally in the forearms and legs. She could tell at once when a fresh one started because of a peculiar hot and tingling

sensation. Then it grew red, became swollen and very tender. After lasting from twenty-four to thirty-six hours they gradually faded. They were not specially connected with the tendons. They were in the skin and perhaps a larger number occurred in the palmar surfaces of the hands, particularly about the pads of the fingers. She died April 25, between seven and eight months from the onset of the illness. There was no post mortem.

Case IV. Mary B., aged 19, seen with Dr. J. K. Mitchell of Philadelphia, June 16, 1890. The patient had had rheumatic fever as a child but had got fairly well and strong. Her illness began with chills and fever which were thought to be malarial, but no parasites could be found in her blood. When I saw her there were signs of an old mitral lesion—apex beat outside the nipple, impulse forceable, and a loud systolic murmur propagated to the back. The spleen was enlarged. The temperature ranged from 102° to 103° and she had profuse sweats. Crops of painful spots appeared from time to time upon the hands and feet, and a few on the skin of the flanks. This was the second case in which I had seen them. They were red, raised, from 3 to 5 mm. in diameter, and often very painful. The fever in this case lasted about seven months. Towards the end embolic symptoms occurred, with hemiplegia.

Case V. July 16, 1902, I saw with Dr. Samuel Ward of Albany, Mr. B., of Cincinnati. I had seen Mr. B. about for several days and noticed that he was not looking very well, but was surprised to find on examining him that he had an old heart lesion, well-marked aortic insufficiency, a loud aortic systolic murmur, and a rough murmur of mitral regurgitation. The patient stated that he had been having malarial fever since the end of May. He had been subject to the disease since 1879. In 1884 he had typhoid fever. He had had attacks of arthritis which were called gout, in one of which in 1898 he had some affection of the heart. The attack at the end of May did not yield to the usual remedies of quinine and Warburg's tincture, and early in June he had a severe chill followed by fever and sweats. He had been up and about, but he had had fever ever since, the temperature occasionally rising to 103°.

From the outset I had no doubt of the nature of the trouble, and had no belief in the malarial theory of the fever, though he came from a malarial district and he had had attacks. Under these circumstances it is always possible to have malarial complications, but there were no crescents in the blood and no pigmented leucocytes. Dr. Ward ascertained that in the second week of April he had bruised his foot on one side, which became red and inflamed, and hot poultices had to be applied. He was in bed for four days. It is quite possible that this may have been a local focus of infection. I saw the patient at intervals with Dr. Ward through August. He had three severe chills. The temperature became more irregular and reached a higher point. He was removed to his home at Cincinnati under the care of Dr. R. W. Stewart. A pure culture of staphylococcus aureus was obtained from the blood. There were no embolic features. He died September 16, 1902, about four months from the onset of the fever.

Case VI. Dr. B. T., aged 33, seen September 25, 1902. Early in May while hard at work he began to have fever. As he had been to the eastern shore of Maryland, it was thought to be malaria. Once or twice a week his temperature would rise to 101° or 102°, sometimes with a chill. He lost in weight, but was able to continue work, and in July while away for a holiday he seemed better, though he still had occasional attacks of fever and sweats. For the previous six weeks he had had daily temperature from 100° to 101° and had sweated at night. Occasionally he would feel very cold and at night when getting into bed the teeth would chatter. He had consulted one or two professional

friends who thought he possibly had chronic malaria, and pulmonary tuberculosis was suggested. He had become a little thinner and paler.

He had been a remarkably healthy man with a very good family history. He never had had rheumatic fever or chorea. He had not had gonorrhoea. On close questioning he stated that in April or May, he forgot which, he had a little swelling and tenderness in some of the joints, but it was quite trifling. In 1890 in an examination for Life Insurance Dr. Chew found aortic insufficiency, but he had never had the slightest cardiac inconvenience.

The patient was a very well-built, well-nourished man, looking a little pale. The right wrist-joint was a little tender on pressure, there were no subcutaneous fibroid nodules. There was a well-marked collapsing pulse. The apex beat was outside the nipple line, not forcible. There was a little diffuse pulsation to right of sternum and second interspace. At the apex the heart-sounds were flapping and clear. At the second right intercostal space there was a short, rough systolic, and a well-marked diastolic murmur of slightly wiry quality was heard down the sternum. The spleen was not enlarged. The patellar tendons on either side were tender on pressure. He assured me that the heart features were very much like those which Dr. Chew had noted in 1900, and I felt convinced that the case was one of endocarditis. Throughout October he became worse and was confined to bed. On November 26, when I saw him, he had changed remarkably. He was very pale. Visible pulsation was seen everywhere in the smaller vessels. The spleen was enlarged. The heart had become more dilated, but there was very little change in the murmurs, except that there was now a loud apical systolic. He had had several very painful spots about his fingers and toes, lasting for a day or two. The blood-cultures were negative. I saw him again on December 8, and he was much worse. His feet were oedematous, with petechial spots here and there. He died in January, about eight months from the onset of the fever.

Case VII. Dr. R. H. T. In 1889 and again in 1890 I was consulted by Dr. T. for an old mitral lesion which was associated with slight enlargement of the left ventricle. As a boy he had had a mild attack of rheumatic fever. For the next ten or twelve years I saw Dr. T. at intervals and never found any special change in his heart. He was a man who lived a very active life and was able to do a great deal of work, though with limitations. During the year 1903 he was not very well and throughout February he had an irregular fever, never very high, not often reaching 102°. He felt very well and he had no chills. From early in March until his death, October 3 (eight months), he was confined to bed and was under the care of Dr. H. B. Thomas, to whom I am indebted for the copy of the temperature chart. I saw him at intervals. Briefly summarized, the main features were, first, fever, which rarely rose above 102°. After June it became a little higher and a little more irregular and sometimes reached 103°. In August and September it was lower, and after September 17 until his death it was normal. There were no chills. He had occasional sweats.

The condition of the heart was very interesting. In June and July when I saw him the pulse was good, heart's action regular, and there was very little change in the mitral murmur, which presented practically the same characteristics with which I had been familiar since 1889. He had no cardiac distress, as a rule, but just before he was moved in the summer there were two attacks of what were supposed to be angina.

The only embolic features were two attacks in the vessels of the retina in July. He had no painful spots on the skin, but he had painful fingers. On one of my visits he had a well-marked, localized red spot about three millimetres in extent on the pad of one finger. He died suddenly October 3, after an illness of more than eight months. The post mortem, by Dr. MacCallum, gave the

following: vegetative and ulcerative endocarditis affecting tricuspid, mitral, and aortic valves and wall of left auricle; rupture of chordae tendinae and encrustation with vegetations; embolic occlusion of anterior coronary artery at orifice; embolic necrosis of myocardium, cardiac hypertrophy and dilatation; infarctions of various ages in the spleen and kidney; focal haemorrhages in the intestines; acute splenic tumour; the vegetations everywhere were firm, yellowish white, and from the mitral orifice a great mass projected into the auricle and there were large irregular masses on the aortic valves. The cultures showed a streptococcus.

Case VIII. In November, 1906, I saw with Dr. Fuller England in Winchester Mr. W., aged 36. He had been under the doctor's care many years previously for acute rheumatism which had left his heart damaged. There was a loud mitral systolic, but there was perfect compensation. Through the summer of 1906 he was not very well and complained of shortness of breath, and in July had frequent attacks of shivering. He began to have inability to rest comfortably at night in the recumbent posture. He lost in weight and became anaemic. He had also slight fever. When I saw him he had been for some weeks in a nursing home. His temperature had ranged from 100° to 101·5°. It was very frequently subnormal in the morning. He had profuse sweats. There was some little doubt at first in the diagnosis, as he had tenderness in the region of the spleen and a dilated stomach. There was a history of tuberculosis in his family.

The patient was very pale and looked thin and ill. There were the signs of old mitral disease with moderate hypertrophy of the heart, a loud thrill and a very intense apical systolic murmur. There was slight infiltration of the bases of both lungs. The spleen was enlarged, but at the time of my visit there were no embolic features. Cultures were made from the blood and a streptococcus was obtained. Numerous injections of a polyvalent serum were made which seem to have reduced the fever slightly, and it caused a good deal of drowsiness. For a month before his death there were numerous embolic patches on the skin with purpura. The patient lingered until December 8. The temperature chart is very interesting. The fever was never high, not once passing above 102°. Towards the end, for the month before his death, it was rarely above 100°. Anti-streptococcic serum seemed to have reduced the fever very much.

The entire duration was about six months. A point of interest in the diagnosis is that the case began with symptoms of shivering, sometimes a definite chill, and as he had an enlarged spleen it was suggested at first he had malaria. Then the distension of his stomach and indefinite swelling in the left side of the abdomen aroused the suspicion of cancer. Later, a slight cough, the fever, the infiltration of both bases, and the man's general appearance suggested tuberculosis.

Case IX. May 8, 1907. I saw in Washington, with Dr. Hardin, Dr. J. C., aged 52, well known in connexion with his work on yellow fever. He had had the ordinary diseases of childhood, typhoid fever in 1886, yellow fever in 1900. He passed the physical examination for the Army in 1902. For several years he had known that there was a lesion of the mitral valve which was detected in a Life Insurance examination. On the evening of February 18 he felt chilly and did not rest well. The next forty-eight hours he was depressed, had cough, and his temperature rose to 102·8°. From that time until the day I saw him he had had regular fever, rarely reaching above 102·5°. He had sweats, more particularly in the early morning hours. As he had a little cough and had lost in weight, it was very natural that tuberculosis was suspected. Dr. Ruffin, Dr. Thayer, Dr. Barker, and others saw him and it did

not seem possible to arrive at a satisfactory diagnosis, as the physical signs were so slight and there was nothing but the fever.

He looked very well, not specially changed in appearance since I had last seen him. There was no alteration in the skin. I made a careful examination, which was negative everywhere except the heart. There was slight enlargement of the left ventricle and there was an apical systolic murmur propagated beyond the mid axilla, and there was a loud pulmonic second sound. His physicians could not determine that there had been any special change in the condition of the heart or in the murmur. He complained of very peculiar spots on his skin, chiefly about the arms and fingers, sometimes on the toes and feet. They came in crops, lasting from one to five days. Each spot was raised, a little red, and felt like a localized infiltration of the skin. They were chiefly on the fingers and on the palms of the hands, sometimes along the forearm. When I saw him, two or three were just disappearing. I did not think that there was any question as to the nature of the case. The mitral lesion, the irregular, persistent fever, and the spots suggested strongly the chronic septic endocarditis. Throughout the summer the condition remained practically the same. The fever persisted, the oscillations of temperature a little greater; he continued to have occasional eruptions of the spots on his fingers, the crops lasting for two or three days. There were no other signs, no audible change in the heart lesion. On September 15, 1907, he suddenly lost power of speech and got right hemiplegia, and he died in fourteen hours. About fifteen blood cultures were taken, all negative. The duration of this case was exactly seven months.

Case X. January 13, 1908. I saw, with Dr. Ward and Dr. Powel of Southampton, Alice A., aged 20. Five years previously she had rheumatic fever, a severe attack with cardiac complications and very slow recovery. Twelve years previously she had a very deep-seated gland removed from the right side of the neck. It was probably tuberculous. The hypoglossal nerve was involved and it had left her with atrophy of one side of the tongue. The previous winter she 'came out' and had a very busy season. She danced and skated and seemed very well. In February she had tonsilitis, not a very severe attack, but she had not been quite well since. She was pale and was often weak and nervous. This was attributed by her mother and the doctor to a love affair which had worried her. Some weeks later she began to have a slight fever and the doctor at first suspected that she might have tuberculosis, but the lungs were negative. Then through the summer she was not well, and on and off had febrile attacks, which increased in September. In October it was thought best that she and her mother should go abroad and spend the winter. On the steamer she got very much worse and it was found she had a temperature of 103°. She landed about the end of October and had been in a nursing home ever since. The symptoms had been—(1) Fever, which had ranged from 100° to 102°, only within the past week had it crossed the 103° limit; (2) she had had at times drenching sweats so that the bed-clothes had had to be changed; (3) she had lately had great irritability of the stomach, constant nausea; (4) on several occasions on the tips of the fingers there had appeared red spots, exceedingly tender swellings, looking very angry and almost, as Dr. Ward said, as though they would suppurate and then they gradually subsided.

There was no pain and no distress about the heart; the urine was clear; the sputum had been examined, as, of course, tuberculosis was at first suspected. She had wasted a good deal. I found a girl looking a little pale, but not so thin in the face as in the body. There was marked general anaemia of the skin, much more so than the face would indicate. The pulse was small, about 110. There were no petechiae. The heart was moderately enlarged, the impulse forcible, wavy, and extended from the second interspace to the fifth, an inch

outside nipple line. There was a very intense mitral systolic heard everywhere over the heart, loudly up the left sternal margin and transmitted to the spine. Though rough and harsh, Dr. Ward did not think it had specially changed in character. The second sound was everywhere clear.

Within three or four days there had been a slight infiltration at the lower lobe of the left lung. The percussion note was impaired and the breath sounds tubular. The apices and other parts were clear. The abdomen was a little swollen, nowhere tender, slightly tumid in the epigastric region; the liver was not enlarged, the edge of the spleen only just palpable. She died about seven months from the onset of the fever. It is quite possible that the onset of the attack may have been in February, when she had tonsilitis, in which case the duration was over a year.