RHEUMATIC FEVER IN YOUNG ADULTS

BY

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During and immediately following World War II the opportunity presented itself to observe numerous cases of rheumatic fever in young adults. The present study was undertaken from patients admitted to the Brooke General Hospital over a period of one year. There were four hundred patients admitted for rheumatic fever during this time. Observation of each patient included the history, physical examination with special attention to the heart, complete blood count, examination of urine and stools, sedimentation rate, electrocardiogram, X-ray, and fluoroscopic examination. The diagnosis was based on these findings. Only if at least one major manifestation such as polyarthritis, carditis, chorea, old history of rheumatic fever, or nodules was present with several of the minor ones, such as fever, rash, epistaxis, leucocytosis, elevated sedimentation rate, and anaemia was the diagnosis made. Two hundred and two of these patients, all enlisted personnel of the Army, were diagnosed as having rheumatic fever, and were followed for several months, the average stay in the hospital being six months. Many have been observed for one year or more before discharge.

Clinical Features

Age of Patients. The patients' ages ranged from 17–39 years; the largest groups were 18 years with 65, and 19 years with 63 patients. Between the ages of 17–20 there were 166 (82%); between the ages of 21–29 there were 29 (14%) and from the ages of 30–39 there were 7 (3%).

Number of attacks before and after enlistment. There were 40 patients (20%) who gave a previous history of definite rheumatic fever. This compares with 24 per cent of the group reported by Quinn (1947). Both these figures are smaller than that of 36 per cent found by Manchester (1946) in his study of rheumatic fever in naval personnel. One hundred and sixty-two patients had their initial attack after enlistment.

Family history. Twenty-four (12%) of the 202 patients gave a positive family history of rheumatic fever

Month of onset. The greatest number of cases occurred during the months of January, February, March, and April. As shown by other investigators, rheumatic fever may occur at any time of the year but is most frequent in the spring and autumn months.

Prodromal disorders. There were 137 patients (77%) who had a preceding upper respiratory infection. The diagnoses in order of frequency were: catarrhal fever, tonsillitis, pharyngitis, laryngitis, and bronchitis. The term "catarrhal fever" implied a non-specific febrile illness of the upper respiratory tract. Many of the patients had throat cultures that showed a haemolytic streptococcus. There were 19 patients who had scarlet fever before their rheumatic fever and 2 who had fever of undetermined origin. Forty patients gave no history of any preceding infection. There were four instances of unusual infections including acute parotitis, acute mastoiditis, pneumonia, and appendicitis. The interval between the prodromal infections and onset of rheu-
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Rheumatic fever was up to 10 days in 91, between two and four weeks in 61, and four to ten weeks in only 10 patients.

Thus the number of patients who gave a history of no preceding infection is higher than the 10 per cent found by Quinn (1947), but approaches to the 19 per cent of Manchester (1946).

Type of onset. One hundred and seventy-three patients (86%) gave a history of acute polyarthritis. There were 12 patients whose onset was one of general constitutional illness with fever, malaise, muscular aching, and anorexia. Four patients had chest pain as the initial symptom, while 10 gave low back pain as their initial symptom. Two patients complained of abdominal pain and in one, an eighteen-year-old boy, the onset was with acute chorea.

Incidence of fever, leucocytosis, and sedimentation rate. Fever was present on admission in 181 patients (89%). Leucocytosis was present in 118 patients (58%). A raised sedimentation rate was the most consistent positive laboratory finding on admission. One hundred and ninety-four patients (96%) had elevation of the sedimentation rate whereas only eight (4%) did not. Of these eight, four gave a previous history of rheumatic fever; two followed scarlet fever; one followed a haemolytic streptococcal throat infection; and one had acute pericarditis. Five of these patients showed no permanent cardiac involvement.

The sedimentation rate thus proved to be the most consistent laboratory test and also was valuable in following the progress of the disease in each patient. Nevertheless it is clear that rheumatic fever and rheumatic heart disease can occur without any elevation of the sedimentation rate. The height of the sedimentation rate showed no relation to the severity of the disease, but the duration of elevation seemed to have some significance. In 123 patients who had a raised sedimentation rate for less than eight weeks, 53 developed carditis; in 55 who showed a raised sedimentation rate for longer than eight weeks 42 were found to have cardiac lesions.

Response to salicylates and weight fluctuation. The response to salicylates was recorded as good in 151 (74%) patients; in 29 (14%) it was fair, and in 9 there was no response. Thirteen did not receive salicylates. Eighty-seven (43%) patients lost weight during their illness; in some instances this was as much as fifty pounds.

Initial joints involved. In 173 (86%) patients joint pain was the initial symptom. There were only 26 instances in which non-weight bearing joints were involved. One hundred and thirty-five patients incriminated the weight bearing joint with one of the upper extremities. In 12, the initial joint involved was not clear.

Some of the unusual instances of initial joint involvement included two cases of a painful, swollen, tender temporo-mandibular joint and seven patients who complained of a swollen, painful great toe. There were ten patients in whom initial joint pain followed trauma to that particular joint. Glazebrook and Thomson (1941a) noted eleven cases in their series in which the first joint affected was the site of the recent trauma.

DEVELOPMENT AND INCIDENCE OF MURMURS

Each patient was examined frequently in various positions and in both phases of respiration by a member of the staff. Twenty-four patients had significant murmurs on admission. There were 178 patients who had no murmur on admission. Ninety-five (53%) of these suffered significant cardiac damage from this attack. Eighty-three patients (47%) showed no evidence of residual cardiac damage.

The percentage incidence of these murmurs is shown in Table I. There were seven cases of a presystolic apical murmur (Table II). This murmur was described as low-pitched, rumbling, less often blowing, heard in late diastole, often crescendo in character and associated with a systolic murmur. The average duration of the time from admission to when the murmur was first heard in these seven cases was 149 days. Glazebrook and Thomson (1941b) discussed the changing heart murmurs in acute rheumatism and noted the early appearance of a presystolic murmur in some cases. In three of their patients it developed within four, six, and nine weeks of the onset of the
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TABLE I
DEVELOPMENT OF MURMURS IN 202 PATIENTS WITH RHEUMATIC FEVER

<table>
<thead>
<tr>
<th>Murmurs on admission</th>
<th>Time after admission PS heard (days)</th>
<th>Duration of raised ESR (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical systolic</td>
<td>48</td>
<td>27</td>
</tr>
<tr>
<td>Aortic systolic</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Aortic and apical systolic</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Aortic and pulmonary systolic</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Apical and pulmonary systolic</td>
<td>13</td>
<td>7</td>
</tr>
<tr>
<td>Mitral presystolic</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>Aortic diastolic</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>Pulmonary “functional” murmurs</td>
<td>95</td>
<td>53</td>
</tr>
</tbody>
</table>

TABLE II
DEVELOPMENT AND INCIDENCE OF APICAL PRESYSTOLIC MURMURS

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Previous Attacks</th>
<th>Murmurs on admission</th>
<th>Time after admission PS heard (days)</th>
<th>Duration of raised ESR (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>19</td>
<td>none</td>
<td>none</td>
<td>150</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>20</td>
<td>none</td>
<td>none</td>
<td>75</td>
<td>36</td>
</tr>
<tr>
<td>3</td>
<td>19</td>
<td>1</td>
<td>AS</td>
<td>240</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>17</td>
<td>none</td>
<td>AS</td>
<td>180</td>
<td>16</td>
</tr>
<tr>
<td>5</td>
<td>18</td>
<td>none</td>
<td>AS</td>
<td>157</td>
<td>36</td>
</tr>
<tr>
<td>6</td>
<td>18</td>
<td>none</td>
<td>AS</td>
<td>92</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>18</td>
<td>none</td>
<td>AS</td>
<td>150</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>Average</strong></td>
<td><strong>22.1</strong></td>
</tr>
</tbody>
</table>

PS=presystolic. AS=apical systolic. ESR=sedimentation rate.

disease. The earliest onset in the present group was 75 days. In the seven cases, there were four who showed cardiac enlargement.

The relation between the clinical appearance of the presystolic murmur and anatomical mitral stenosis is not certain. It is doubtful whether anatomical stenosis could develop in such a short time. In 1935, Bland et al. studied the necropsy findings and clinical records of 100 patients who had rheumatic fever. They felt that “after the first year and often becoming more extensive during subsequent years the structural alterations of the mitral leaflets assume increasing importance. Although considerable deformity may develop during the second year, scarring of sufficient extent to produce stenosis at the mitral orifice is rarely present before the third year.” In their total series mitral stenosis was considered to be present in 68 instances. In these 68 cases with mitral diastolic murmurs only 21, less than one-third, actually had anatomical stenosis of the mitral orifice.

There were 15 instances of aortic diastolic murmurs (Table III). The average time from admission to when the murmur was first observed was 66 days. There were 7 patients in whom this time was 38 days or less. In these 15 patients there were 3 with demonstrable cardiac enlargement by X-ray and fluoroscopy. Two of these gave a previous history of rheumatic fever.

Three patients developed both aortic diastolic and mitral presystolic murmurs while under observation. In one, the aortic diastolic was first observed in 90 days and the mitral presystolic in 180 days. In the second, the appearance of the murmurs was in 37 and 157 days respectively. In the third, the time intervals were 37 and 150 days respectively.

One interesting observation that was made was the development of the so-called pulmonary "functional" murmur; this was observed in 21 patients. A murmur was not present in any of the patients of this group upon admission. It consisted of a grade II blowing systolic murmur heard at the pulmonic area and which changed with respiration or position but did not completely disappear. The exact significance of this development is not clear. It may mean that in these
cases of friction fever.

cutaneous nodules had chest pain over the arthritis but these findings were present when his clinical picture showed no abnormalities and the sedimentation rate and other laboratory findings were normal. Another, admitted in May, was about to be discharged in November when a final routine cardiogram showed changes which progressed as in the first case. The patient felt well and all laboratory data were normal. No friction rub was heard and the chest

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\textbf{Table III}

\begin{center}
\begin{tabular}{|c|c|c|c|c|}
\hline
Case & Age & Previous Attacks & Murmurs on admission & Time after admission ADM heard (days) & Duration of raised ESR (weeks) \\
\hline
1 & 20 & 1 & none & 60 & 9 \\
2 & 17 & none & ASM & 90 & 16 \\
3 & 17 & none & ASM & 150 & 6 \\
4 & 19 & none & none & 55 & 16 \\
5 & 19 & none & none & 152 & 4 \\
6 & 24 & none & none & 28 & 24 \\
7 & 18 & none & ASM & 37 & 36 \\
8 & 19 & none & none & 38 & 8 \\
9 & 19 & none & ASM & 60 & 4 \\
10 & 19 & none & none & 21 & 15 \\
11 & 19 & none & none & 28 & 24 \\
12 & 29 & none & none & 60 & 4 \\
13 & 18 & 1 & ASM & 150 & 44 \\
14 & 18 & none & ASM & 37 & 15 \\
15 & 19 & 2 & none & 26 & 24 \\
\hline
\text{Average} & & & & 66 & 17 \\
\hline
\end{tabular}
\end{center}

ADM=aortic diastolic. ASM=apical systolic. ESR=sedimentation rate.

particular cases some form of pulmonary valve or pulmonary artery involvement actually existed. Collection of lymphocytes, Aschoff bodies, edema and disruption of the elastica have been described by Paul (1927) as occurring in the pulmonary artery.

\textbf{Other Signs Associated with Rheumatic Fever}

\textit{Spontaneous epistaxis} occurred during the initial few days of the acute rheumatic attacks in 21 (10%) of the group.

\textit{Rheumatic pneumonitis.} In 3 patients there were chest X-ray findings which pointed to this condition. These findings were present during the acute initial stage when the patient had fever and polyarthritis. Physical findings over the chest were usually absent. Whether these were cases of rheumatic pneumonia as described by Epstein and Greenspan (1941) could not be proven.

\textit{Subcutaneous nodules} appeared in 7 patients and in 4 instances were quite extensive, appearing over the occipital scalp, cervical and thoracic vertebrae, elbows, wrists, knees, and ankles. Subcutaneous nodules appear in about 20 per cent of cases in both rheumatic fever and rheumatoid arthritis but their pathology has proven to be different (Fingerman and Andrus, 1943).

Four patients showed the typical erythema marginatum which is associated with rheumatic fever.

\textit{Pericarditis.} There were eight patients who had signs of fibrinous pericarditis in the series. Three of these had a friction rub heard during the first week of illness. None of these three had chest pain and only one had electrocardiographic changes. The other five had no pericardial friction but had changes in the electrocardiogram which consisted of initial elevation of the S–T segments in the limb leads followed by inversion of the T waves in the limb and chest leads. Only one of these 5 patients had chest pain. Two of these had features of special interest. One who was admitted in February did not develop these cardiographic changes until five months later when his clinical picture showed no abnormalities and the sedimentation rate and other laboratory findings were normal. Another, admitted in May, was about to be discharged in November when a final routine cardiogram showed changes which progressed as in the first case. The patient felt well and all laboratory data were normal. No friction rub was heard and the chest
roentgenogram was normal. The T wave inversions persisted until March, five months later, when they became stable and remained inverted.

There were two cases of pericarditis with effusion. Both had electrocardiographic changes but only one had chest pain.

**ELECTROCARDIOGRAPHIC ABNORMALITIES**

All of the patients had serial tracings taken. These showed significant changes in 124 cases (Table IV). Prolongation of the P-R interval with normal sinus rhythm was the most frequent abnormality, occurring in 64 patients. In a study made by Engleman (1947) of patients admitted to an Army Service Force rheumatic fever centre, 50 per cent showed abnormalities of the electrocardiogram as compared with 61 per cent in the present series. The most common were heart block and significant T wave deformities in leads I, II, and IV.

Partial auriculoventricular heart block with Wenckebach phenomenon and dropped beats occurred seven times but was never an isolated finding.

**TABLE IV**

<table>
<thead>
<tr>
<th>Electrocardiographic Changes observed among 202 Cases of Rheumatic Fever</th>
</tr>
</thead>
<tbody>
<tr>
<td>Changes observed in 124 cases</td>
</tr>
<tr>
<td>T wave abnormalities</td>
</tr>
<tr>
<td>S-T segment changes</td>
</tr>
<tr>
<td>Pericarditis</td>
</tr>
<tr>
<td>Ventricular premature beats</td>
</tr>
<tr>
<td>Arrhythmias (auricular flutter, auricular tachycardia)</td>
</tr>
<tr>
<td>First degree A-V block</td>
</tr>
<tr>
<td>Second degree A-V block</td>
</tr>
<tr>
<td>Complete A-V block</td>
</tr>
<tr>
<td>Intraventricular branch system block (right)</td>
</tr>
<tr>
<td>Complete A-V dissociation</td>
</tr>
<tr>
<td>P wave abnormalities</td>
</tr>
<tr>
<td>QRS abnormalities</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
</tr>
</tbody>
</table>

There were thirteen instances of sinus bradycardia. The rate ranged from 42–58 a minute during the acute onset of the disease eventually returning to normal. Glazebrook and Thomson (1941c) found that bradycardia was a frequent occurrence in the acute attack in adolescents and young men. This bradycardia was considered to have some prognostic significance as half the patients showing it developed permanent cardiac damage compared with only 30 per cent of those without it. In the present group seven of the thirteen exhibited residual cardiac damage.

No correlation was found to exist between the severity of the clinical picture and the electrocardiographic findings.

The relationship of rheumatic fever to rheumatoid arthritis has been discussed by other investigators (Baggenstoss and Rosenberg, 1941; Bayles, 1943; Fingerman and Andrus, 1943; Young and Schwedel, 1944). In the present group there were several patients who came from the same areas at the same time and whose clinical course was that of rheumatoid arthritis. One patient whose case is described below showed manifestations of both diseases.

A man, aged 19 years, developed a sore throat and subsequently swollen and painful interphalangeal joints of the second and third fingers of his left hand. When he was admitted on March 10, three weeks after the onset of illness walking was difficult because of pain in his legs. Physical examination showed the heart not to be enlarged. Grade II apical and pulmonary systolic murmurs were present. The left knee was red, warm, swollen, and painful. The electrocardiogram showed a prolongation of the P-R interval. An aortic diastolic murmur was heard for the first time approximately 37 days after admission. All of the proximal interphalangeal joints of the fingers became swollen and quite tender and there was slight swelling and tenderness of both elbows. There was
no leucocytosis. A mild anaemia was present with 3,800,000 red cells and 11·5 g. of haemoglobin (Sahli). Urine was normal. The sedimentation rate (Westergreen) was 38 mm. in one hour. The electrocardiogram was normal on May 13.

During his stay in hospital the patient's temperature remained normal. On June 12 he developed large subcutaneous nodules over the occipital region, the cervical and dorsal vertebrae, elbows and wrists. In September a presystolic apical murmur appeared. The weekly sedimentation rates remained elevated. A chest X-ray was normal. Films of the hands showed moderate decalcification of the bones in the fingers and wrists. The patient's proximal interphalangeal joints showed fusiform swellings, and were moderately tender.

DISCUSSION

It is not within the scope of this paper to present a complete epidemiological study of this group of patients or the epidemiology of rheumatic fever in the American Army as a whole. Quinn (1947) stated that "the highest rate in the Navy is found in the recruit training centres and service schools. The duty status of the patients revealed nothing of epidemiological importance." Manchester (1946) observed four major factors involved in the occurrence of rheumatic fever in service personnel: (1) infection of the respiratory tract due to the haemolytic streptococcus; (2) length of service; (3) seasonal influences; and (4) antecedent rheumatic fever. In the present study all factors played an important role.

It is interesting to compare some of the findings of McCue and Galvin (1948). In their study of 225 patients with rheumatic fever the average age of onset was 7·9 years. A family history was obtained in 17 per cent compared to 12 per cent in ours. Fifty per cent gave a history of a preceding upper respiratory infection compared to 68 per cent in the present group. Joint pains were the most frequent complaint in 157 of their patients. In their group 48 per cent showed residual cardiac damage compared to 53 per cent of our patients in their initial attack. There were no deaths in our group during the initial attack.

The two patients who developed abnormal electrocardiograms resembling those of pericarditis were of special interest. Both were clinically normal and were about to be discharged from the hospital. Neither was observed to have a friction rub. This phase of the disease might have been due to a pure delayed "hypersensitivity" phenomenon. It is suggested that all cases of rheumatic fever should have frequent electrocardiograms.

As a new decade in medicine starts with drugs such as adrenocorticotropic hormone (ACTH) and cortisone, many physicians may lose sight of the fact that rheumatic fever is still almost a pure clinical diagnosis. We have no specific diagnostic test. Physicians in daily practice must rely on their own clinical acuity without which—and without appreciation of the natural history of the disease—no proper evaluation of treatment can be made. It is hoped that clinical studies such as this can provide some measure of help both in diagnosis and treatment.

SUMMARY

A clinical study of 202 enlisted personnel of the American Army with rheumatic fever between the ages of 17 and 39 is presented.

There were 40 patients with a history of rheumatic fever prior to enlistment and 162 with their first attack after enlistment.

A history of preceding upper respiratory infection was given in 137 patients. Nineteen had preceding scarlet fever. Forty gave no history of a preceding infection.

The sedimentation rate was elevated in 194 patients. This was the most consistent laboratory finding and constituted a valuable guide in following the course of the illness. There appeared to be some relationship between the duration of elevation and development of cardiac consequences.

Ninety-five patients (53%) developed murmurs while under observation, in 7 of whom the murmur was mitral presystolic. The average duration from admission to the time the murmur was
first heard was 149 days. There were 15 cases of aortic diastolic murmurs. These developed during observations after an average interval of 66 days. Pulmonary "functional" murmurs appeared in 21 patients.

There were eight instances of fibrinous pericarditis, two with effusion. Of the eight, three had pericardial friction but no chest pain. Two showed no electrocardiographic changes. Two patients were observed to develop electrocardiographic abnormality typical of pericarditis five months after their initial attack. Both showed normal laboratory findings.

Electrocardiographic changes were observed in 124 cases (61%). Prolongation of the P–R interval with normal sinus rhythm was the most frequent abnormality.

One patient developed simultaneously signs and symptoms of both rheumatoid arthritis and rheumatic heart disease.

The diagnosis of rheumatic fever and rheumatic heart disease still belongs in the realm of the clinician. It is a disease which can be easily overlooked when all the criteria for diagnosis are not present and may exist in the active phase even with no elevation of temperature or sedimentation rate. About one half of any group can be expected to develop permanent cardiac damage.

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