ACUTE RHEUMATISM

BY

J. ALISON GLOVER

Damp, chill, and exposure have always been considered prime factors in rheumatic fever. The conditions of modern war—the trench or fox-hole filled with water, icy exposure, sleeping in wet clothing, and the crowded dug-out or shelter—seem to make an environment favourable to rheumatic fever, and one might expect it to be outstanding among those diseases which crowd in the train of war. Yet it finds no place in Hurst’s Diseases of War, and the various bulletins of war medicine hardly notice it. Even in times when the disease in civil practice was far more common and severe than to-day, its incidence in war was much below expectation. “Certain it is,” says Lehnbach in 1863, “that we often see regiments exposed to damp, wet, cold, sudden and violent changes of temperature, and obliged to sleep on wet ground with but scant protection and no cases of acute rheumatism.” Then it was—as it still is—a disease of recruits, occurring in training establishments (in war always overcrowded), rather than a scourge of armies in the field or fleets at sea. In the American Civil War we find that “acute rheumatism shows in its monthly rates a greater prevalence in that period of the war during which new levies were sent to the field than later when the levies have become inured to the hardships of active service.” This preferential incidence on recruits and training establishments often takes the form of “barrack epidemics.” But rheumatic fever, if not outstanding as a war disease, is important for the following reasons: it causes no small proportion of medical casualties (who require long treatment in bed); in “barrack epidemics” it may ruin for active service previously healthy recruits; it causes invaliding, and pension claims for consequent heart disabilities; as the main cause of organic heart disease in children it diminishes man- and woman-power for recruiting; and, lastly, it seems often to be the precursor of those more chronic forms of rheumatic disease to which armies in the field are particularly prone—fibrositis, lumbago, and sciatica.

Previous Wars

Table 1 summarizes estimates from the various official histories of recent wars—estimates which are never strictly comparable. In the American Civil War of 1861–4 the authors frankly point out that the contrast between the large incidence and the low death-rate, together with the low case-mortality—when the latter in civil hospitals was about 3%—alike suggest that the majority of cases assigned to acute rheumatism were, in fact, cases of fibrositis, myalgia, or conditions other than acute rheumatism. Bearing in mind Copeman’s ratio of 15% referred to later, it probably would be safe to divide the incidence of 65 per 1,000 by six.

<table>
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<tr>
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<th>Average annual incidence per 1,000 strength</th>
<th>Acute rheumatism admissions as a percentage of all non-battle admissions</th>
<th>Death-rate per 1,000 strength</th>
<th>Case-mortality %</th>
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<td>Crimea—</td>
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<td>(British, 1854–6)</td>
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* Much inflamed with cases of muscular rheumatism, etc.
† Based on 1,984 cases in Richmond (Var.) (Chimborazo Hospital).

In the Crimean figures the average annual incidence is calculated on a strength of 98,000, whereas at the beginning of the peak period, death, wounds, and disease had reduced the effective strength to 24,000. In considering the high case-mortality we must remember that in those days the average case-mortality in civil hospitals was reckoned as high as 3% (Loumis) (Glover, 1946).

In the South African War (1899–1902), which was characterized more by heavy marching on half rations than by severe battle casualties, the outstanding medical phenomena were the vast epidemics of typhoid and dysentery. But the admissions for rheumatic fever formed 6-05 of the total admissions for all diseases and non-battle injuries, and, during the whole war, amounted to 24,460—thus exceeding those for wounds, 21,292. The total killed and wounded was 27,273. The average annual admission rate was 44 per 1,000, the death-rate 0-04 per 1,000, and invalided 7-75 per 1,000. The case-mortality was 0-1%, half that of the American Civil
War. The heavy and rapid marching may have been an important factor in this war, and ox-drawn rations were often left far behind. On the march to Pretoria, for example, heavily laden infantry "averaged nearly seventeen miles a day over apparently endless prairies in blazing sun and bitter cold, swept now by hot and choking dust storms, now by rushes of icy hail, fording rivers and floundering through sand, with scanty food and shelterless bivouacs." (Official History of the War in South Africa.)

Rheumatic fever was much less prevalent in France in the Great War 1914-19 for, after four years of trench warfare, Colonel T. Hay wrote "acute rheumatic fever was not a common disease among soldiers in France, and it is improbable, therefore, that much valvular disease of the heart originated during war service." The vast majority of joint and limb pains in this war were due to trench fever and other non-rheumatic conditions.

This astounding fact—that in the wet, the mud, and the stench of the trenches in Flanders in 1915 acute rheumatism was much less prevalent than in the high and magnificent climate of South Africa—seems to show that the disease had already begun its long decline in incidence and severity.

The Decline in Rheumatic Fever

For nearly a century now, rheumatic fever has gradually shown the features of obsolescence, its incidence, and, still more, its severity steadily declining. In military history since the Crimea this trend can be seen. So in civil life it is no longer "not only one of the most prevalent, but one of the most fatal maladies incident to our precarious climate" (Macleod, 1837). No longer are more than a tenth (11.5%, Ormerod, 1852) of all admissions in general hospitals those of patients with rheumatic fever, so acute as to be readily diagnosable by high pyrexia and characteristic odour. Since 1870, their number and the proportion to total admissions in civil hospitals have fallen to a tenth of what they were, and, in peace time, there have been similar reductions in the service hospitals. The severest of its clinical features—such as hyperpyrexia, pericarditis, and nodules—are nowadays rarities, the age incidence has shifted from the younger adult age-groups to that between 5-15 years, and its social incidence falls now almost exclusively on the children of the poor, especially those of great cities. The standardized all-ages death-rate for males, 89 per million in the decade 1891-1900, had fallen to less than a quarter—22 per million, in 1937. In the military age-groups the reductions in death rates were even greater. Occasional exacerbations occurred, such as one (1915) during, and one (1920) just after the first world war, the latter perhaps an aftermath of the high level of haemolytic streptococcal infection from the great influenza waves of 1918-19. Minor peaks occurred in 1925 and 1934, but in spite of them the great decline in rheumatic fever mortality still went on.

The Inter-war Years

The inter-war years, 1919-39, were noteworthy in rheumatic fever history for three things: first, the acceleration of the decline; secondly, the invention of systematic preventive effort, especially in London, Birmingham, and Bristol; and thirdly, the strengthening of the theory (not yet conclusively proved) that infection by the Streptococcus pyogenes is the cause of the disease, a theory greatly strengthened by the differentiation of its serological types by F. Griffith (himself a war casualty), and the work of Lancefield, Todd, Coburn, and many others, which shed new light on the role played by this organism in the diseases wholly or partially due to its infection. The general trend and oscillations of rheumatic fever mortality closely correspond to those of scarlet fever mortality and thus support the view that both derive from the same infection, which in the latter case is undoubtedly Strep. pyogenes.

Barrack Epidemics of Rheumatic Fever

The investigation of “barrack epidemics” and similar outbreaks in residential schools in these inter-war years also supported the streptococcal theory. Such epidemics have certain characteristics. They occur in overcrowded communities with a high proportion of newcomers—freshly recruited adolescents. They have a regular cycle: overcrowding, a precursor epidemic of acute tonsilitis, an interval (representing the latent period in the individual) and then the occurrence of cases of rheumatic fever, usually numbering less than one-tenth of the cases of tonsilitis. A high carrier-rate (say 30 to 50%) of a single type of Strep. pyogenes is present, and the same type will be found in swabs from the tonsillitis patients. Such “barrack epidemics” of rheumatic fever obviously resemble those of cerebrospinal fever, save that the meningococcus itself produces no throat or catarrhal symptoms and so only a “silent” or “carrier” precursor epidemic. Recruits, in both cases, are particularly vulnerable in their first weeks of training, when the strain of unaccustomed exercises and environment lowers resistance to the infection by a fresh strain of haemolytic streptococcus, of which the virulence has probably been enhanced by rapid transference.

Surgeon-Commander C. A. Green (1942) has reported one such epidemic where the attack rate of rheumatic fever rose to 63 per 1,000. The recognition of this phenomenon is recent, but old records suggest that it has often occurred before. Thus in 1859, when acute rheumatism admission rates in the Army were still high, that for depot battalions was 65-4 per 1,000—more than twice that for the Foot Guards, 31-7. It is recorded that "many of the cases were the sequel of venereal disease," so perhaps the incidence stated is too high. The great differences in incidence for white troops between the 32 training camps in the United States in 1917-19 suggest the probability of "barrack epidemics" in some of them. The average incidence for all these
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The World War 1939–45: the Decline Continues

This war has involved civilians to an extent unparalleled in this country since the Anglo-Saxon invasion. Civil experience is, therefore, of great significance, especially as the war in this country began with evacuation, the unprecedented mass movement from great industrial cities of three-quarters of a million school children, the most susceptible of all the community to acute rheumatic infection. Evacuation was fortunately carried out in ideal weather, and though an epidemic of tonsillitis was noticeable (though, of course, not notifiable), in the autumn of 1939, the great decline, described in a previous section, continued through the years 1940 and 1941, and brought acute rheumatism to its nadir in 1942, in which year the crude all-age death-rate sank to 12:1 per million, scarcely half of what it was in 1939 (23), and only 56% of the previous low record of 1937 (21:5). Of actual deaths under 15, only 157 from rheumatic fever occurred in 1942, compared with 300 in 1937, and from heart disease (at least nine-tenths rheumatic in origin) only 330 (contrasted with 590 in 1937). Scarlet fever mortality showed corresponding and even greater reductions up to 1942 (Glover, 1943).

The death-rate per million under 15 years in 1938 (the last whole year of peace) for rheumatic fever was 44 and for heart disease 68; in 1942 they were respectively 18 and 39; in 1943, 27 and 40, and in 1944, 28 and 42.

Evacuation of children and hospitals and the closure of many rheumatic supervisory clinics renders impossible any comparison between pre-war and war incidence on London children; but Bristol, though later suffering severely from air raids, was not originally an evacuation area, and Prof. C. Bruce Perry has kindly provided the following information. At the Bristol cardio-rheumatic clinic the total number of patients with evidence of active infection, including chorea, dropped from 201 in 1937–8 (when there were 50,800 elementary school children in average attendance in the Bristol schools) to 63 in 1941–2 (average attendance 42,545). The decline in severity was shown by only 28 of the 63 patients having arthritis, and by the fact that patients with rheumatic nodules in 1941–2 numbered only 5, whereas in 1937–8 there had been 20. [Bristol has long been known for a high incidence of rheumatic fever (J. A. Symonds, 1834).]

In Cardiff, hospital admissions dropped from 121 in 1937 to 89 in 1941–2. In Leicester the numbers of patients attending the rheumatic clinic declined from 179 (nine months only) to 102. At Great Ormond Street Children’s Hospital, London, the percentage of rheumatic to total admissions in 1933–4 was 2:24; in 1937–8, 1:20; and in 1941–2, 0:83.

In the Glasgow Royal Hospital for Sick Children admissions for acute rheumatism numbered 70 and formed 3-3% of the total admissions; in 1931 there had been 150 and they had formed 10-1% of the total admitted.

Since its nadir in 1942, rheumatic fever has increased a little, the crude death-rate rising to 17 in 1943, and 18 in the first half of 1944; deaths under 15 rose to 233 in 1943, all figures still much below the best pre-war records of 21:5 and 300 respectively in 1937. Scarlet fever showed a corresponding trend in incidence.

Military Experience in the 1939–45 War

The original British Expeditionary Force of 1939–40, which was evacuated from Dunkirk, had but little rheumatic fever. Copeman (1940) records that, during the first four months of this campaign admissions to base hospitals No. 2 and No. 3 for “rheumatic” conditions were about 15% of total admissions and 26-0% of all admissions in the medical division. But of the patients with “rheumatic conditions” (judging by the first 100 cases admitted to a special “rheumatism” ward) only 15% suffered either from acute or subacute rheumatic fever; the majority—70%—suffered from fibrositis; 6% from rheumatoid arthritis, and 9% from osteo-arthritis (mostly traumatic in origin). Rheumatic fever, therefore, seems to have accounted for some 2-25% of total admissions to hospital. Copeman’s proportions, i.e. the percentages which admissions for rheumatic fever form of admissions for all “rheumatic diseases,” after allowing for differences in terminology, are not unlike those of earlier military records, which, however, generally put the proportion of true rheumatic fever even lower (e.g. 7-9 in 1916–17–18 and 5-2 in Italy 1917–18). They may also be compared with those for insured males of corresponding age (16–35) in civil life in England and Wales in 1922, which are: acute rheumatic fever 9-2 (subacute 20-4); rheumatoid,
2-9; osteo-arthritis, 3-8; muscular rheumatism (including lumbago), 63-6. After further experience in the Middle East, Copeman (1944) has described three types of rheumatic fever: first, the "classical" type; secondly, a "benign" type, for which he suggests the title "acute febrile myalgia," and which, though not uncommon, is non-epidemic, of short duration, and without cardiac or other sequelae; and, thirdly, a type which, while "classical" in attack and as to carditis, instead of resolving, merges into chronic fibrositis.

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These figures cannot give the incidence as it is not possible to state the populations at risk, but it is apparent that the incidence was low, that it was heavier in the first half years than in the second half years in each case, and that the proportion which rheumatic fever bears to the rheumatic group as a whole is not unlike that found in France by Copeman. The figures for female patients from the Services are much smaller and much more confined to the 18–24 group, and the percentage of rheumatic fever in the whole rheumatic group is, as might be expected, considerably higher.

**Invaliding**

All statistics relating to acute rheumatism are difficult of comparison. It is clear, however, that the rates of invaliding for acute rheumatism have greatly diminished in the last 90 years. In the American Civil War the ration was 25-1 per 1,000; in South Africa 7-75; in the first World War I estimate the figure at 8-2 per 1,000, but I do not think that this estimate relates so accurately to acute rheumatism as does the South African war figure; it probably includes other forms than acute. It is yet too early for estimates for the Army in the war just past. Surgeon-Commander Green tells me that in the one year, 1944, in the Navy, 0-26 per 1,000 were invalided for rheumatic fever with or without cardiac lesions, and an additional 0-08 invalided for allied cardiac conditions, making 0-36 per 1,000 (males) strength.

**Prophylaxis**

Prophylaxis against rheumatic fever is at present identical with that against acute streptococcal tonsillitis or scarlet fever. Good ventilation and avoidance of overcrowding are still the paramount considerations, though the importance of dust suppression grows ever clearer, and dampness in houses or between decks is a factor. Chemoprophylaxis has, probably, some promise of success, especially in specially susceptible groups, such as children known to have had previous attacks of acute or subacute rheumatism.

Outbreaks of scarlet fever, sore throat, and consequent rheumatic fever, were frequent in American camps on her entry into the war. At Farragut, for example, there was a 30% carrier rate of haemolytic streptococcus, and in one year there were about 5,000 cases of scarlet fever and 1,500 severe rheumatic attacks (Coburn, 1944). Chemoprophylaxis was considered effective here.

R. Cruickshank (1946) writes:

Mass chemoprophylaxis with sulphonamides was tried in a number of these outbreaks. Unfortunately this prophylactic measure was sometimes not instituted until the epidemic had reached its peak so that a sharp fall in incidence following its introduction was difficult to interpret. None the less, the combined evidence suggested that sulphanilamide in doses of 0-5 g. twice daily for periods of one to four weeks helped to control the epidemic spread of streptococcal throat infections. There was, however, an unhappy sequel. In the winter.
of 1944-45 (the large-scale prophylactic use of sulphonamide was introduced in 1943-44) a number of outbreaks of scarlet fever and streptococcal tonsillitis occurred due to sulphonamide-resistant strains of the same types (principally types 17 and 19) as were prevalent the previous winter, and there seems little doubt that these resistant strains derived their origin from the small doses of sulphonamides used to control the earlier outbreaks of streptococcal infection.

Experience with the chemoprophylaxis of streptococcal infection in this country has been largely negative.

Recent work by Hamburger and his colleagues (1945) has suggested that the nasal carrier may be more important than the throat carrier as a reservoir and source of streptococcal infections.

In civil life Cruickshank advises that—

Since most of the toxic and allergic reactions among rheumatic fever patients on prophylactic doses of the drug have appeared between the first and fourth weeks after commencement of the treatment, clinical and laboratory supervision is most essential during that period. While any patient who has had one attack of rheumatic fever might be considered suitable for chemoprophylaxis, a fair proportion of children do not develop cardiac complications. Prophylactic use of the drug might, therefore, be limited to children who have already had two attacks and to those who have had one attack accompanied by rheumatic carditis. Sulphadiazine, sulphamethazine, or sulphanilamide are the drugs of choice.

Since I completed the above article two further sources of information have become available. H. S. Barber (Brit. med. J., 1946, 2, 83), dealing with the results of treatment at an R.A.F. Rheumatic Convalescent Centre, gives the incidence of rheumatic fever in 1940 in the R.A.F. as a whole as 2-1 per thousand, whereas among the boy entrants and apprentices it was 10-4 per thousand for the same period. Secondly, D. D. Rutstein (Amer. J. publ. Hlth., 1946, 36, 463) states that, from January, 1941, to August, 1945, approximately 17,000 cases of rheumatic fever occurred in the U.S. Army, and from the onset of war to Jan. 1, 1945, 14,344 cases of rheumatic fever occurred in the U.S. Navy.

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