OBSERVATIONS ON THE NATURAL HISTORY OF ACUTE RHEUMATIC FEVER

BY

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It was remarked in the *Lancet* recently that clinical medicine is the study of man in his environment. It would seem therefore that the more simple the environment can be rendered the less complicated should become the study of his disease.

An unusual opportunity to study the natural history of certain rheumatic diseases in a body of young healthy males was offered by working in a hospital which supplied the medical needs of several large bodies of troops who were stationed, as was the hospital, in a remote desert. Thus they were isolated from all contacts (other than those within their units) for a period of nearly a year, and they were totally divorced from those complicating factors which are associated with civilized environment. As a result it is believed that a clearer understanding of the genesis and development of rheumatic fever and of its relation with fibrositis was obtained. Subsequent experience in Malta soon after the siege (during which a large proportion of the population had been living on a minimal diet in underground caves), allowed an investigation which further conditioned these views. Later, certain invasion troops were seen after a journey involving hardship and exposure unusual even for such "tough" troops as this "spearhead" comprised. This also provided material for study, and some of the results are incorporated in this paper.

Aetiology of Rheumatic Fever

Rheumatic fever, it is generally agreed, shows unmistakable signs of being due to an infection. It is not primarily caused by the factors of exposure, fatigue, etc., with which it is nevertheless often associated at onset. In this series of cases it was established that the possibility of the infection having been transmitted by extraneous contacts was remote owing to their isolated situation for ten months, and the fact that their last experience of a city was merely one night in transit four months before. They had left England two years previously and had been since in Burma continuously. If therefore the cause of the cases under consideration was infection transmitted by human agency it would seem probable that it had laid dormant for at least fourteen months, and possibly for much longer, as the disease is not common in Burma. If a period of latency be possible for months, the possibility that this period might last for years must also be considered. With

this in view two categories of cases may be briefly reviewed.

(1) *Cases (10) with history of previous attacks.*—It seems reasonable to suppose that these attacks were recurrences of previous ones in England, in view of the facts that the disease is known to relapse, and the unlikelihood of any fresh infection having been met with in the circumstances. If so the infection must have lain dormant during the years intervening since the last attack (a minimum of 2 years, and in some cases 10–15 years), until conditions again became favourable for its recrudescence. Enquiry in this group revealed that in two cases the mother had suffered from acute rheumatic fever.

(2) *First attacks in desert (32 cases).*—It is reasonably certain that these cases had had no contact with any case of active rheumatic fever for a very considerable period, as apart from crossing India 14 months previously they had no urban contacts since leaving England two years before. It seems, therefore, to be a possibility that some at any rate may have acquired their infection before leaving England, but that the circumstances had not hitherto favoured the development of the disease clinically. These cases did not (except in one instance) arise in units in which the relapse cases, referred to above, developed, nor could contact with them be traced. The first case of rheumatic fever admitted to hospital was a first attack, and was the only case recorded in his unit during our stay. The possibility that certain of these infections might even date back to childhood was considered, and it may be significant to record that in this group nine cases gave a convincing history of acute rheumatic fever in one or more parents—generally the mother. It was noted that all the cases suffering with first attacks reported an exceptional previous freedom from any illness, suggesting a high degree of general resistance. The actual attack of rheumatic fever was, however, in all the desert cases preceded by severe or protracted illness of various other types, and in many cases, in addition, by exceptional exposure and strain which would be likely to have lowered such resistance. These observations seem to favour the possibility that the causative agent of rheumatic fever may, like that of tuberculosis, lie dormant in the healthy body the resistance of which is high, and produce signs of disease only when

circumstances conduce to a marked lowering of this.

The cases studied were in widely dispersed units and the question of droplet infection within these units was investigated and dismissed, with the exception of the four cases discussed later under that heading.

INFECTION IN PARENTS AND CHILDREN

The relation between infection in the parents and in their children has been referred to above. It appeared in this series of observations that the proportion of parents who had suffered with the disease was higher in the group of patients who developed first attacks in the desert than in the group who were suffering a relapse of the disease contracted originally in urban surroundings.

At my request C. T. Potter investigated this matter in a series of 405 rheumatic children attending his O.P.D. at the Queen Elizabeth Hospital for Children, and reported as follows: Of 290 children (under 7 years old) with growing pains, 10.7 per cent. of their parents had had rheumatic fever. Of 115 children (under 7 years old) with acute rheumatic fever, 7.6 per cent. of their parents had had rheumatic fever. On this he comments that if the disease is the result of transmittable infection, the proportion of children who suffer with rheumatic fever, and whose parents also give evidence of infection, is unexpectedly low. The observations recorded above may explain this, however, since the organism may in fact be directly transmissible from parent to child; but if the latter's resistance is at the time good it may remain dormant until some infection of another type, such as streptococcal tonsillitis, lowers resistance sufficiently, later in life. It is suggested that the series of first attacks in the desert discussed above may be examples of this mechanism. In cases where the resistance is never lowered to the necessary extent it would appear possible that the syndrome of rheumatic fever will never develop, although the subject is in fact harbouring the organism. This rationalizes the clinical observation that the disease is familial but not strictly hereditary.

Such further problems as whether there may, therefore, be "carriers" of rheumatism, and whether an attenuated form (e.g. fibrositis) might develop in infected persons with high immunity, must await the discovery of the causative organism.

Nature of Infecting Organism

The streptococcus is widely implicated as being the causative organism of rheumatic fever by virtue of the fact that it is so frequently a precursor of the rheumatic attack. It is also fairly generally agreed that it is unlikely to be the sole causative agent, but, beyond this, exact knowledge does not yet go. Birkhaug's theory of a circulating endotoxin formed by localized streptococci (toxic focus) has not been confirmed, and does not seem to fit the facts as observed in this series; nor does the theory of super-

sensitivity as the result of previous streptococcal infections, suggested by Zinsser and Yu.

Gibson and Thomson (1933) found that one fact emerged from their studies—i.e. an association between infection with haemolytic streptococci and rheumatic fever. This, they said, was not a simple matter of cause and effect, and some other factor must be postulated, such as the presence of another infective agent not yet defined, possibly with some allergic process as a contributing factor. Only in some such way, they think, can the close epidemiological relation between haemolytic streptococci and rheumatic fever be reconciled with widespread distribution of streptococcal infection and the comparatively limited incidence of rheumatic fever. This view of the matter would fit in with the present observations if we except the need to invoke an allergic process.

Regarding the *Micrococcus rheumaticus* of Poynton and Paine, as recent observations have shown that certain cases of chronic meningococcal septicaemia not uncommonly masquerade as rheumatic fever in both adults and children and are indistinguishable except by means of blood culture (*Lancet*, 1940) the suggestion was recently made that this organism may in fact have been a form of meningococcus, which the facilities at their disposal at that date did not permit them to recognize (*Lancet*, 1942). This likelihood is reinforced by the account they give (Poynton and Paine, 1913) of a fatal case of true rheumatic meningitis following an attack of acute rheumatic fever. Necropsy revealed a healing lesion of the mitral valve and turbid fluid over the whole base of the brain; in films from this "diplococci" were seen in numbers. Also in a fatal case of "chorea" which they describe numerous diplococci were found in the perivascular spaces of the pia mater.

Observation of the cases of rheumatic fever which occurred in the Middle East showed that, although a preceding illness was almost invariable, this was by no means usually due to the streptococcus. Streptococcal sore throats, and more commonly streptococcal furunculosis, were not uncommonly the forerunners of an acute attack of rheumatic fever, but such unassociated diseases as the dysenteries, sandfly fever, and malaria were even more common. It seemed, therefore, as though the role of this "preceding" disease must merely be to lower the general resistance sufficiently to allow some further and so far unknown organism, which was either latent or recently acquired, to produce symptoms. The alternative would be to suppose that rheumatic fever is merely a non-specific but peculiar type of host response to all these various types of organism, conditioned possibly by sensitization, as has been postulated by Davidson and others. If the former view be adopted the role of the streptococcus may be considered to be a non-specific one, as in the case of the other diseases mentioned, and the fact that in England and America it comes so largely into the picture may be explained on the grounds that it is the commonest intercurrent infection of a self-

limiting nature encountered in temperate climates; and more especially at those times of year when rheumatic fever is most commonly met with. In the same way those diseases mentioned above as being the common precursors of rheumatic fever in the Middle East are the commonest infections met with in these latitudes.

If this is so, then the streptococcus cannot strictly be said to have a specific relation with rheumatic fever, although its common association in temperate climates is explicable. In unscientific terms it might be suggested that the pathological function of the "preceding infection" is merely to awaken the dormant specific organism. In some cases the latter, lying in a milieu of high resistance, merely "turns in its sleep"—the clinical consequence being possibly the syndrome of "acute febrile myalgia" described below—whilst if circumstances are temporarily favourable it may be awakened fully, and bite viciously at its host.

Streptococcal infection in the throats of children, about ten days before, was correlated with relapse of their rheumatic fever by Coburn in the Northern States of U.S.A., where, as in England, this is the commonest type of subacute infection met with. This did not seem to occur so commonly in the Southern States, however, where it is reasonable to suppose that conditions approximate more to those in the Middle East. He suggested that rheumatic fever is due to a reaction of tissues to the products of the haemolytic streptococcus in predisposed individuals. It might be suggested that such "special predisposition" is in reality latent specific infection, and that the streptococcal infection allows it to become active by lowering body resistance. When these American children were removed to Costa Rica it was found that they did not relapse, although streptococci were still found in the throats of certain of them. This suggests that in the absence of the third element (the physical factor) generally found by us to be necessary—i.e. exposure, cold, etc.—the virulence of this streptococcal infection was no longer sufficient to depress the child's resistance to the stage at which the specific rheumatic organism could become pathogenic. It is generally found that this physical factor is an important one in the development of a case of rheumatic fever, and indeed sometimes may even replace the need for the usual preceding infection if it is sufficiently severe (see next section).

Such evidence as is presented by this series of cases seems to point to rheumatic fever being caused by some organism at present unknown. If it is accepted as unlikely that the present series of cases had met with any source of fresh infection in the desert, it must be considered probable that this organism is able to remain latent in the tissues of healthy individuals possibly from childhood until resistance is lowered by various means amongst which streptococcal infection takes a high place, environmental factors coming next.

The analogy of tuberculosis arises in the conjunction of these three factors; or (if the conception

of the specific organism as being of the nature of a virus—as originally suggested by Schlesinger and Amies be preferred), the analogy of the activating role of secondary mixed infections and climatic conditions on the virus of the common cold must be considered as being somewhat comparable.

SPREAD BY DROPLET INFECTION

It has been suggested by Glover and others that rheumatic fever may be an infectious disease, and he reported a number of instances in which outbreaks of rheumatic fever had occurred in communities of young adults. This, he thought, probably resulted from droplet infection from the upper respiratory passages.

This matter was investigated in all the cases in this present series, and only in the case of one workshop unit, from which four cases were received, did it appear to be likely. They were all received during the month of January and the first week of February, 1943, and they had been living in close contact with a fellow craftsman who had had a first attack of rheumatic fever when aged 6, and another lasting six weeks when he was 15. He was now 22 and had suffered with a bad cold for the previous month, although he did not complain of any exacerbation of the chronic backache, which was a legacy from his last acute attack. None of the four patients had caught the cold, although they slept in the same hut, in which there were twenty other men. The clinical features of their rheumatic attacks were similar. They started with a short period of malaise, then stiffness leading to pain in one or more joints, which swelled up on second or third day, when they were admitted. They were sweating and their temperatures rose to 102°–103° F., although they did not admit to feeling really ill. The blood sedimentation rate rose during the attack and slowly resumed the normal about four weeks after the onset. The attack appeared to respond readily to salicylate treatment. In none of these cases had any rheumatism occurred previously (*vide*, later, "benign" type rheumatic fever). A watch was kept upon this unit, but no further cases occurred during the next three months. This unit had no connexion with other units in which cases arose. Being Base troops their standard of fitness was not equal to that of the divisional units, from which all the others were drawn. The following record is of the first case admitted.

Sgt. Age, 25 years. Service, 2½ years. No previous rheumatic history in self or family. Tonsillectomy when 8 years old. Seven days previous to admission slight sore throat (Gram+diplcocci) and on next day complained of stiffness, acute pain, and, later, swelling of right wrist. On following day left ankle painful, and

CHART 1.

metacarpal-phalangeal joints of the right hand swollen slightly. After two more days of fleeting pain and B.S.R. 15 mm. the pain and all swelling disappeared and did not return. Two weeks after onset B.S.R. was 7 mm. and he returned to his unit. Four months later, perfectly fit and no recurrence.

Preceding Factors

Even if we admit the presence in the patient's tissues of an unknown primary infective organism, we must also recognize certain important preceding factors which are almost invariably associated with the onset of the illness. These are: (a) preceding infections of various (non-rheumatic) types, as already mentioned; (b) physical factors, which include unaccustomed or severe exposure, strain, or cold. These two additional factors appeared to be jointly operative in the majority of cases of rheumatic fever which developed in this series, more particularly in the case of the primary attacks. Where the "dosage" of either (a) or (b), however, is extreme, it may prove sufficient to provoke the attack without the necessity for the other factor being operative (*vide*, later, "Cases due to physical factors only").

NATURE OF PRECEDING INFECTION

Although both the primary and the relapse cases were preceded in nearly all instances by some debilitating illness, both the nature of this and its time relation with the onset of the rheumatic fever were so variable as to render it extremely unlikely that there could be any specific connexion between them. These illnesses were for the most part those which are most common in the Middle East. An analysis of the preceding illnesses in the first twenty-five cases of rheumatic fever yielded the following result: malaria (all types), 8; dysentery (both types), 7; sandfly fever, 6 (in four of these cases two attacks or relapses); streptococcal infections, 4. In most cases the sufferer felt subjectively that he had never entirely recovered from the effect of these at the onset of the rheumatic fever. There was generally a history of exposure in addition occurring during this period. It seemed likely that these illnesses plus the physical exposure acted non-specifically by reducing the general powers of resistance of the body, and so allowing the hypothetical specific organism already present in the tissues to take advantage of this favourable circumstance. Streptococcal infection, although it is the most common preceding infection in England was comparatively rare in this series. Streptococcal sore throats had occurred before the attack in eight of the forty-two cases and streptococcal furunculosis in five. The role of the streptococcus in this connexion has been more fully discussed above under the heading of "the nature of the infecting organism."

It was noted from their histories that all the men infected had had a high degree of immunity to disease in general, rheumatic fever being in most cases their first period of illness since childhood. But for the exceptional circumstances of their present life it

seems reasonable to suppose that many of the cases in this series (who were all category A1 young men), would never have developed rheumatic fever, even though the evidence seems to point to the fact that they were harbouring the specific organism.

A homely analogy may be used to illustrate this relation between the preceding infection and the latent specific infection. In stoves some rapidly burning spirit such as methylated often serves to heat up the heavier but less spontaneously inflammable oil, which will, however, if it reaches the right temperature, initiate even more intense and prolonged combustion. The specificity of methylated spirits for this purpose is perhaps comparable with that of the streptococcus in the rheumatic syndrome, since it is the substance most commonly met with in this connexion in our country, but other volatile spirits (*cf.* organisms) can produce the same effect.

PHYSICAL FACTORS

These were principally exposure, which includes cold and wet. Whilst the men were in the uplands of Persia they slept mostly in the open. It was extremely cold weather, in many places there was snow, and it rained not infrequently. They slept on the ground. The training during the daytime was intensive and strain was the other principal factor, operating mostly upon those men who had been debilitated by recent illness. In the case of civilian patients investigated later in Malta it is probable that malnutrition also played a part. In the cases which occurred amongst our Sicilian invasion troops exposure and strain were the sole preceding factors, no history of any antecedent illness being obtained. This was the only series of cases in which this seemed to be so.

It was noticeable that where physical factors were operative they were either unusually severe, or else the man was unaccustomed to them. This reinforces the wisdom of the Army policy of progressive "hardening." It appeared that a smaller "dose" of the physical factor was needed to provoke an attack of rheumatism when the antecedent illness had been severe and vice versa—as would perhaps be expected. The site of the initial pains tended to be localized by the nature of the exposure or strain. For example, in several cases where the man had slept on cold ground without a groundsheet the initial lesions were on the side of the body on which the patient had slept, whilst in the cases localized by strain certain joints which had been subjected to this were the first to be affected.

CASES DUE TO PHYSICAL FACTORS ONLY

The following are condensed case histories of men of commando troops who fell ill after a combined operation. Their attacks were very acute, but ran a "benign" course (*vide*, later, Cases (d) and (e)).

O. A. B. Age, 20 years. Single. Service, 2 years. Father and mother no rheumatic history. Patient had no sickness of any sort except a mild attack of "lumbago" last year as a result of a strain. Last three months

fighting in Tunisia, followed by intensive "invasion tactics" course, which included sleeping in open in wet clothes after swimming ashore. During daytime very long marches with full equipment in great heat. Journey of two days in landing barges in which there was little room to lie down. No shelter from fierce sun, and was rather sea-sick. On last night it rained and he was wet through. On disembarking could not bear weight on left knee as it was very painful. Thought this was due to stiffness and tried to "walk it off," but failed. Went sick next day and was admitted with temperature 103° F. Suffered with typical attack of rheumatic fever of "benign" type (*vide* section on clinical types). Uninterrupted recovery and B.S.R. returned to normal in a month.

C. D. Age, 20½ years. Single. No parental or personal history of rheumatism or other sickness. Was a ghillie before joining paratroops two years ago. One brother and one sister—no sickness. Exceptional exertions involving heavy-weight carrying in Algeria for one week prior to embarkation. During a forced march in great heat had felt giddy and "fainted" but had not gone sick on recovery. For two months had been sleeping in the open on the ground; it was very cold and there was a heavy dew. During the invasion he was shot down into the sea, and spent four hours in the water. The day after he had felt stiff all over, and next morning woke up after a bad night with very tender wrists, knees, and ankles, which all swelled during course of day and became acutely painful. Temperature 100° F. and pulse 100. Admitted.

O. E. Sweating profusely and joints (as above) much swollen and painful. Said he did not feel ill, however, Eyes congested and catarrhal. Constipated. No headache. Temperature was 102.6° F. and pulse 108. There were acutely tender points on the internal aspects of the joints. Next day temperature 100.2° F. and effusion much less, but tender spots, which correspond with points on the tendons of surrounding muscles, as before. B.S.R., 25 mm. This case also ran a "benign" course, and the patient rejoined his unit in five weeks, apparently normal.

It did not seem that these cases differed in any way from those provoked by the more usual mechanism of a preceding illness.

GENERALIZED STRAIN

In the only two cases in which cardiac lesions were observed to arise *de novo* the history was of severe strain undergone whilst on a "battle course," in conjunction with a preceding indisposition (sore throat in one, dysentery in the other). There was no exposure experienced.

It is possible that there may be further and less commonly encountered factors which should be mentioned under this heading, although they did not affect the series of cases under consideration. Such factors may include hyperthyroidism, menstruation, etc., and, as already mentioned, sub-nutrition.

Nature of Rheumatic Fever: Clinical Observations

The two chief elements of any case of rheumatic fever are (1) the general signs and symptoms, which include pyrexia, malaise, sweating, raised B.S.R., etc., which are common to most generalized infections, and need not be examined further at this

stage; and (2) the local signs and symptoms, which comprise (a) muscle and tendon stiffness and pain, (b) joint stiffness, pain, and swelling, and (c) cardiac lesion.

LOCAL SIGNS AND SYMPTOMS: (a) AND (b)

Close observation of these strongly suggests that they are due to a similar "fibrositic" mechanism—probably a localized oedema of the synovium of the affected tendons in the first place, the difference in symptoms and signs depending merely upon the localization and extent of the lesion. A detailed description of the onset of a relapse of rheumatic fever in the first case studied in detail will bring out all the points regarding stiffness, pain, and swelling which emerged as the result of subsequent clinical observation.

L./Cpl. G. H. Age, 32 years. The patient had had two previous attacks of rheumatic fever, the first when aged 12 and the second when aged 15. There were no cardiac lesions, and he was passed into the Army two years before.

A week prior to admission to hospital he had complained of pain in the right ankle for twenty-four hours. The joint then swelled up, this sequence being followed in the left knee on the day of admission. The day after admission (temperature 100° F.) he said that he thought the right knee was going to be affected and also the elbow-joints; these showed no abnormality. On close questioning he described the subjective sensation as a "tightness" round those joints which rendered him rather unwilling to move them, "as they did not feel quite right." By the evening this was interpreted as a definite stiffness; some very localized creakings could be felt over one biceps tendon, and could be heard through a stethoscope. On waking next morning there was "definite pain in all these joints." Close examination showed, however, that this pain was in fact situated in certain tendons around the joints, and that pressure on these areas exacerbated the "joint pain," whilst pressure on other tendons, or areas of the same tendon, as well as along the joint line had no effect. This pain was increased by movement of the joint. The area of tendon affected in each case seemed to be extremely small—the greatest length of tenderness in any particular tendon being apparently about ¼th inch. Examination at hourly intervals throughout the day showed that these tender areas were not constant, portions which had been tender at 8 a.m., for instance, not being so at midday, whilst others had at that time become first stiff and then tender, the new tender area being equally clearly demarcated. About six o'clock some increase in joint fluid was detected in the knee-joint, although the pain was less than it had been in the morning. The fluid increased progressively for a few hours and then remained constant during the next day. Throughout this period he described the knee-joint as painful, although the focus of this pain was found to be located in the tendinous junction of the quadriceps muscle with the patella (a common site). After this the fluid gradually dispersed, then the pain, and lastly the stiffness and creaking, in the inverse order of their appearance. Subsequently both wrists swelled, the sequence of events being exactly similar to that already described. The tendon sheaths principally affected were those of the superficial layer of the flexor surface where they pass beneath the anterior carpal ligament.

Subsequently all patients with pain and swelling of the joints were closely observed and as the result of these observations it is thought that the arthritis of rheumatic fever is a secondary phenomenon arising by direct extension from a primary tenosynovitis of the muscle tendons which surround the joint. This will explain the almost invariable experience that joint fluid from such cases is sterile. In the case of the knee-joint there may be a direct connexion between the tendon sheath and the joint cavity *via* the supra-patellar bursa, and this may account for the temporary lessening of the pain which is not uncommon whilst the joint cavity is filling up, since the tenderness and pain in the tendons following the period of creaking is no doubt due to the tension of the inflammatory fluid at that point, and its relief will therefore depend on it being able to disperse either along the sheaths or into the joint, where this is possible, before natural resolution starts.

The commonest sites for these initial areas of tenderness round the larger joints appeared to be: (a) In the wrists—any of the tendons might be affected; the tender areas were, however, always in the neighbourhood of the surface markings of the carpal ligaments under which they pass. (b) In the knees at the junction of the quadriceps tendon with the patella, and on the tendons on the internal aspect of the joint and in the semi-tendinosus tendon posteriorly. (c) In the ankles; over the tendons of the peroneus longus and brevis on the outer aspect, and the tibialis posterior on the internal aspect—as they hook round the malleoli. (d) In the elbows on the flexor aspect, not infrequently in the biceps tendon: this is not inserted very near the joint, nevertheless this lesion is subjectively interpreted by the sufferer as joint pain, and no doubt any subsequent inflammatory effusion into the joint cavity might take place by direct extension of inflammation from this tendon sheath to the synovium of the joint.

To recapitulate, the analysis of some forty cases of rheumatic fever showed that two stages precede the actual swelling of the joints. First, a feeling variously described as tension, stiffness, or creaking, referred to the joint. There is disinclination to move the joint, sometimes interpreted as “weakness.” This lasts from 2–24 hours, and probably represents a condition of progressive swelling of certain tendons or their sheaths around the joint. The second stage is that of pain, originating in one or more points tender to pressure, and mostly situated in or over the larger tendons. This pain is largely referred over the joint, and is spoken of by the patient as “joint pain.” It is probably due to an increase in tension due to localized effusion in the tendon sheath, and each area remains painful for 5–48 hours. Swelling can often be palpated at this stage along these areas. These points of tenderness are quite definite and discrete, and there may be a number of them round a single joint. (The pain can be abolished at each by an injection of procain.) The third stage is that of actual effusion into the

neighbouring joint cavity either from primary affection of the synovium of the joint, or quite often by secondary extension from that of the affected tendons.

(c) CARDIAC LESIONS (ASCHOFF NODULES)

No opportunity of studying this manifestation of the disease arose in the present series. Their occurrence in established cases of rheumatic heart disease and their histological structure is, however, well recognized. A subcutaneous nodule from the elbow, one of many which developed in the course of a severe first attack of rheumatic fever in a native boy, was removed and its structure was reported to be indistinguishable from that of an Aschoff nodule (Dr. F. Marsh).

It is suggested as the result of these observations that in rheumatic fever the general pyrexia and malaise and the local synovitis of tendons and joints which occur early are principally due to a non-specific reaction to infection such as may occur temporarily in the course of several other febrile diseases—e.g. scarlet fever or Malta fever. True subcutaneous nodules, however, are evidently reactions to a more specific process, and are evidence when they occur that this is beginning to predominate, and consequently that the prognosis is becoming graver. It is thought that Aschoff's nodules in the heart may be of the same nature and significance as these; their special importance lying only in their localization in a “vital organ.”

Even in “benign” attacks of rheumatic fever in which these specific manifestations do not occur it seems probable that the initial symptoms (which it is suggested above may be largely non-specific) may become intensified and “fixed” by the specific agent, the prognosis probably varying with the degree of this action.

Clinical Types of Rheumatic Fever

Three types were met with and will be discussed separately: (1) The classical type. (2) A benign type. (3) Classical type which merged into chronic fibrositis.

The classical type, with general fever and peripheral tendon lesions leading to joint swelling as described, needs only brief further reference here. Two cases of permanent cardiac damage occurred. There was seldom much difficulty in diagnosis, and if they had not recovered within six weeks they were generally sent home, as it was considered that even after recovery they would be liable to relapses. A differential diagnosis which sometimes offered difficulty was undulant fever.

“ACUTE FEBRILE MYALGIA”

A benign form, for which the name “acute febrile myalgia” is suggested, and which it is believed as the result of subsequent experience will be comparatively common if looked for. It should not be regarded merely as a “mild” attack of rheumatic fever, since this diagnosis as commonly employed

implies that the myocardial damage may be progressive although the signs and symptoms are outwardly mild. Cardiac damage does not appear to follow this form at all. It responds well to salicylate therapy. In this form it is probable that the non-specific element is maximal and the specific factor (whatever it is) minimal. The onset of the attack is the usual one in rheumatic fever—malaise, pyrexia, sweating, fleeting muscular pains. The pyrexia is sometimes less in evidence than in normal attacks, however. After the first twenty-four hours there is also very little malaise, although pain and, later, swelling remain severe. The blood sedimentation rate is considerably raised, but is generally approaching the normal again (as is the patients general condition) in about a month. In these cases there was no previous history of rheumatism, although there was always a history of preceding illness and/or exposure or strain which had “pulled him down,” as in the case of the classical attacks described above. As in these cases, the preceding illnesses were of widely varying types, and appeared to act by lowering the resistance of the patient to the attack of some more specific organism. It suggested that those cases may represent the attacks of the specific organism of rheumatic fever on a soil of unusually high resistance temporarily lowered by certain preceding factors.

The reason for separating this type as a clinical entity from rheumatic fever proper lies principally in its apparently benign (non-toxic) course. In nearly all the cases observed the patient was well and B.S.R. normal again within a month; and no sequelae, cardiac or otherwise, were observed four months later (except in one case in which some fibrositis persisted). No relapses were reported. The prognosis in subacute rheumatic fever is rightly regarded as so charged with malign possibilities that it seems important to recognize that a benign form may exist. The natural tendency of the medical officers in charge of these cases was at first to take the usual grave view and to recommend that they should be boarded out of the Army. It was chiefly the fact that the men themselves seldom thought seriously of their condition after 24 hours, but merely reported sick with an account of some muscular pain or joint swelling, that led us ultimately to modify our own view. The name (benign) “acute febrile myalgia” accurately describes the syndrome, and will emphasize the difference in prognosis from acute rheumatic fever of normal type, although these cases must not be confused with “acute epidemic myalgia” since no epidemic factor is present.

Even within the framework of this syndrome considerable variation in severity was found to occur. At one end of the scale was the patient who suffered with tonsillitis accompanied by mild fleeting arthralgic pains, which persisted for a longer period than usual after the throat infection had subsided and whose B.S.R. also remained raised. Such patients are unlikely to be admitted to hospital in civilian life, but are probably frequently seen by the

family doctor. At the other end of the scale the condition will merge into the classical type of rheumatic fever and the line of demarcation may prove difficult to determine. The features indicating these cases to be of the benign type seem to be (a) the short initial period of pyrexia and the patient's subjective feeling that he is not very ill; (b) the subsidence of the B.S.R. within a comparatively short period; and (c) freedom from tachycardia during early convalescence. It would probably be wise in civilian practice to take an electrocardiographic tracing in addition before deciding that the case was definitely of the benign type.

Some Illustrative Cases

A few typical summarized case histories of patients with “acute febrile myalgia” are quoted below. All of these men were returned to their units in about a month, and when followed up four months later were reported to be perfectly fit. It would seem therefore that the distinction made here between acute rheumatic fever and “acute febrile myalgia” is at least as useful clinically as that commonly drawn between acute typhus fever and its milder form of Brill's disease, or between smallpox and alastrim.

Case 1.—I. J. Age, 22 years. Service, 2 years. No previous personal or family history of rheumatic troubles. At time of attack was in poor health owing to a recent attack of bacillary dysentery, followed by chronic streptococcal furunculosis. No sore throats. In peacetime was law-student. Athletic. Unmarried. Plays hockey twice a week. Woke up one day after last match with stiff and somewhat swollen wrists and ankles. Difficult to walk, and was put on to light duty for two days. These symptoms cleared up, but as knees were also getting stiff and patient complained of sharp pains in neck and shoulders was admitted to hospital with temperature of 100·6° F.

Lightly-built wiry man with good appetite. Did not feel ill, but now complained of pains in quadriceps and wrists. No joint swelling. N.A.D. on clinical examination. Temperature 99° F. for first two evenings. B.S.R. 21 mm. Hb. 76 per cent. C.I. 0·9. W.B.C. 3,950 per c.mm. Sod. Sal., gr. x, tds. Uneventful recovery. B.S.R. 2 mm. on twenty-first day after onset. Returned to unit four weeks after admission. Subsequently had further attack of furunculosis, but no recurrence of rheumatism.

Case 2.—K. L. (R.A.). Age, 33 years. Service, 2 years. No previous or family history of rheumatic troubles; married, two children. In civilian life was decorator; worked outside in summer only. No previous illness since measles and whooping-cough in childhood till dysentery (Flexner) on arrival in Middle East six months previously. This was “cured” in hospital, but he had had diarrhoea since and had not felt well. No sore throats. Woke up one morning with synovitis right knee. Next morning pain, acute round both ankles but no swelling; “went sick” and was put on light duty. Next day other knee painful and also both shoulders and back. Did not feel ill at any time, but T.=101° F. Admitted to and seemed well in c.c.s. No temperature third day, so discharged

on fourth day; but relapsed four days later with pain in knees and admitted to hospital.

Healthy looking patient with good appetite. Said he felt well but rather tired, and had sweated last two nights. Nothing abnormal discovered except that movements of right shoulder and elbow were restricted and painful. Urine normal. B.S.R. 12 mm. $T.=100^{\circ}\text{F}$. Blood count normal. The following is a chart of the tempera-

CHART 2.

ture during both attacks. On Feb. 3 (3 weeks later) there was no pain or stiffness anywhere. B.S.R. 3 mm. One month later "is doing full duty and feels very well." B.S.R. 5 mm. Four months later no recurrence.

Case 3.—M. N. Age, 27 years. Service 3 years. No personal or family history of rheumatism. Married, one child. "All children's diseases," "pleurisy" when 10 (bed 1 week). No sore throats. Non-venereal urethritis 18 months ago; cured. Depressed and not up to mark for last month. Woke up on morning of Dec. 21 with severe pain in both thighs, made worse by movement, and palpitations. That day he joined in a three-day "exercise" under rough conditions and sleeping in open with snow on ground. On his return his left knee was swollen, hot, and red, and he had severe pain in both hips and buttocks. He was admitted and next day both wrists also painful, but B.S.R. only 3 mm. (Dec. 28). B.P. 120/70. Slight systolic murmur at apex of heart. By Jan. 1 he still had stiffness of elbows, but did not feel ill. B.S.R. 10 mm. On Jan. 28 B.S.R. 3 mm.: murmur gone. Feels very well. Discharged. An electrocardiogram was subsequently done and reported as normal. On enquiry at end of April his M.O. stated that he was perfectly well and doing full duties and murmur had not reappeared.

The following typical cases are more briefly summarized:

Case (a).—O. P. Age, 25 years. (A very mild case.) Severe tonsillitis, and developed slight pain in right patella region on second and third day. On third day his temperature was 100°F . and right knee slightly swollen. On fifth day throat well and temperature normal, but B.S.R. 17 mm. and pain in left shoulder. On sixteenth day felt well, but B.S.R. was 26 mm. On twenty-seventh day the B.S.R. was again normal (9 mm.) and patient had had no further pains and resumed normal work.

Case (b).—Q. R. Age, 23 years. No previous rheumatism. On Dec. 5 caught a severe cold, which was followed by pains in knees and swelling of ankles. Next day pain at bottom of back and round right hip, which kept him awake. Slight sore throat and temperature 101°F .; he was febrile for four days, this being highest temperature reached. Pulse rate, 100. Heart normal. Dec. 9 was admitted, as both knees and ankles swollen and painful. No fluid in joints. B.S.R. 18 mm. Put on to sod. sal. gr. xx tds. Next day temperature and pulse normal. W.B.C. 8,000 per c.mm. (77 per cent. polymorphonuclears, 21 per cent. lymphocytes). On Dec. 14 seemed very well. B.S.R. 1 mm. On Dec. 19

returned to his work. Four months later reported "very fit."

The following case is of interest as the resistance appeared to be lowered and the disease activated as the result of T.A.B. inoculation, and not of illness.

Case (c).—S. T. Age, 22 years. No previous rheumatic or other illness. Very occasional sore throats during last few months. Ten days previously was vaccinated and given routine T.A.B. injections and had felt seedy since. Sudden onset of pain in both shoulders going round to upper back, knees, and right ankle: $T.=101^{\circ}\text{F}$. Pains disappeared and temperature was normal after two days of sod. sal. (gr. 120), but B.S.R. 35 mm. After first twenty-four hours in bed felt quite well. Eight days later B.S.R. 5 mm., and after thirteen days was discharged. Four months later reported perfectly fit and doing full duty.

Case (d).—"Benign" rheumatic attack provoked by burns. Italian soldier aged 22. Admitted Nov. 2 with superficial burns both lower limbs. These had produced considerable shock. $T.=102^{\circ}\text{F}$.; P. 100; R. 20. No malaria. Two days later onset sudden acute pain both legs during night and much sweating. $T.=102^{\circ}\text{F}$. P. 70. Very restless and in much pain next two days—chiefly right hip (which he could not move) and both knees and wrists swollen. B.S.R. 58 in 1 hour. Sweating constant. Much better in another two days and didn't feel ill. Left knee still swollen. Burns healing. Nov. 15 no further pains or swelling.

Case (e).—U. V. Age, 25 years. This case would seem to have been provoked principally by unaccustomed exposure (as were several others). No personal or family history of rheumatism. Sore throats for two to three days each year for last five years; not this year. Sleeping in open on right side in intense cold. After two weeks of this woke one morning with pain right hip and knee, which later swelled up. Did not feel ill but was admitted. Temperature 99°F . Slight increase in joint fluid of right knee. B.S.R. 58 mm. Pyrexia lasted only for two days, and after two weeks B.S.R. was 21 mm. and only slight stiffness remained. Three weeks from admission B.S.R. 3 mm. and patient seemed absolutely fit and was discharged.

CHART 3.

Charts of the B.S.R. in 6 typical cases of "acute febrile myalgia" showing that in most cases this has resumed a normal level a month after onset of the attack.

Cases E and F show a common form of "lag" curve; the B.S.R. not starting to rise until well after the onset of the disease. This type is generally normal again about a month after it starts to rise.

To summarize, it is suggested that a benign form of rheumatic fever exists which is not uncommon and for which the name "acute febrile myalgia" is

proposed, in order to avoid the implications attaching to the diagnosis of ordinary rheumatic fever. The onset is similar to that of the classical attack, but fever rarely lasts for more than 48 hours. It responds to salicylates. The patient rarely feels ill after the first two days, and the blood sedimentation rate returns to the normal in about a month (see chart). There are no cardiac or other sequelae.

CLASSICAL TYPE MERGING INTO FIBROSITIS

It was noticed in several cases that the acute attack instead of resolving gave place to chronic muscular pain and stiffness from which the patient was seldom entirely free. This seemed to occur equally after primary attacks as after relapses. These patients also became unduly sensitive to exposure and to muscular strain, and they generally appeared unlikely to return to a high army category. Later a neurotic element was sometimes added, but this appeared to be the effect and not the cause of the chronic pain. The psychiatrist was asked to examine several cases and agreed in general with this conclusion.

The rheumatic attack which runs this course is often clinically not a severe one and none developed cardiac complications. It was noted in those cases which resulted from relapses that the primary attack had occurred after the age of 15 or 16; that is at a rather later age than is usual. Several of these cases are reported in full below.

RHEUMATIC FEVER AND CHRONIC FIBROSITIS

It is common experience that patients are not infrequently seen with chronic fibrositis which they attribute, apparently rightly, to an attack of rheumatic fever. In view of the lack of certainty regarding the specific causative agent of the latter, and our almost complete aetiological and pathological ignorance regarding fibrositis, no authority has publicly connected the two diseases. Detailed history, taking in such cases as have given a story of this type has, however, served to show that the two diseases may in certain cases stand in the relation of cause and effect. This can be shown by recording one or two such cases in full.

Case X.—Age, 29 years. Service 2½ years. Category A1. (A first attack of acute rheumatic fever leading to chronic fibrositis.) Father suffers with severe muscular rheumatism in his shoulders. Mother, no special history. No illness except whooping-cough and measles when about 10 years old. No previous rheumatism. Was a solicitor's clerk in peacetime. Unmarried. One year before admission had a carbuncle on left leg and was in hospital nine days. In the next two months (March and April) this occurred on right leg and recurred after healing. In December a similar lesion appeared on the left chest wall, and just as that was healing another turned up on right side of neck (haemolytic streptococcus). At this time he began to complain of pains in legs and lower back. Later his feet became very painful and he could hardly walk. He felt "washed out" but not really ill; could not eat much. No headache. He was admitted to hospital with T. = 101° F., and this rose to 103° soon afterwards. He still did not feel very ill,

but the pain in the legs and arms was severe enough for three nights to prevent consecutive sleep. On the first night he sweated profusely, and after this the pain moved to the shoulders for a time.

On examination he was a rather pale but well-developed man, with injected conjunctivae. Tongue heavily coated. Liver and spleen not felt. All usual tests for specific infections were negative. Urine, normal. He had a healing boil on right side of neck, but no adenitis. Throat, normal. Knees and ankles very swollen owing to effusion: sodii. sal. gr. xx, t.d.s. On the evening of the sixth day after admission the temperature was 100·6° F., and this ended his pyrexia, which had ranged between 99°–103° F. He felt much better, but fairly severe pains still flitted between his legs and shoulders. The boil had healed, and no joints were swollen, although the knees were still very tender. The heart appeared to be normal. B.S.R. was 10 mm. (Westergren). Three weeks after admission he felt well and continued afebrile. There was, however, severe pain in the left shoulder, chiefly the deltoid region, and this was worst in the mornings. The B.S.R. was 2 mm. and his Hb. 94 per cent. W.B.C. 8,000 per c.mm. (polymorphs 45 per cent.; lymphocytes 43 per cent.; large mononuclears 7·5 per cent.; eosinophils, 3 per cent.; and basophils, 1 per cent.). The pain remained in the shoulders and sometimes the neck, but he made an otherwise uninterrupted recovery.

He was seen two months later and still complained of these pains, which he said were unchanged. Examination showed the presence of typical fibrositic "myalgic spots," which were extremely tender and the source of the pains he complained of. He was otherwise well and heart was normal. Four months later the report stated that he was exactly in the same condition, with the additional fact that he was now extremely sensitive to draughts and wet weather, both of which factors would provoke severe pains in his shoulders and neck.

Case Y.—Aged 22 years. (A case of juvenile rheumatic fever with one subsequent attack and subsequently a gradually progressive condition of fibrositis becoming established.) Father (a plumber) often suffers severely, his joints swell, and he has to go to bed during attacks. Patient's paternal grandfather died of "rheumatic heart disease." His mother's side of family showed no case of rheumatism. His sister aged 20 complains frequently of severe pains in shoulders. The patient is also a plumber in a dockyard and frequently gets wet through. After this, with an interval of 1–2 days, during which he feels no harmful effect he generally has to spend several days in bed. He has two young children who are not rheumatic. His only other sickness has been measles as child and pleurisy (bed 2 weeks) aged 12. He started work at the age of 15, but after 2 months was taken ill with rheumatic fever, which kept him in bed for 6 months. The attack started gradually with "growing pains" which progressively got worse and spread from legs to arms and shoulders. Medicine did not help, and he developed fever and sweated at night heavily. (He volunteered the statement that the sweat had a pungent smell.) He states that he came out in crops of subcutaneous nodules on knees, wrists, and elbows, "which interested the doctor" but which were not painful or tender. They were of sudden onset and each crop lasted about 2 weeks. He felt very ill during this period, and said that it was agony to move arms or legs, and the joints were padded with wool.

At the end of March, after 2 weeks convalescence on sod. sal. and ultra-violet light he gradually recommenced work, and had resumed full work in April, the onset of the attack having been in August. He appeared to

have completely recovered until the following September, when he noticed twinges of pain in his shoulders after carrying heavy lead pipes. Two days rest and sod. sal. did not help, but they gradually passed off. Since then attacks of this sort have occurred every 2 to 4 weeks, and prevented him working for 2 or 3 days owing to the pain in shoulders and knees. Does not generally have to go to bed unless these are the result of a wetting. At the onset of the war these attacks were becoming more severe and lasted up to 7 days. He had joined the Territorial Army in 1938, and was called up without medical examination at outbreak of war. Since then had another attack of rheumatic fever in Army, which was not nearly so severe as his previous one and lasted only a month. He managed to carry on in a sedentary employment in England (and was in fact better than he had been in peace time), but on the voyage the pain became very bad, and since arrival in the Middle East he had spent most of his time in hospital (Dec.-March).

On examination he was a rather pale and slightly-built man. No abnormalities discovered other than fibrositic "nodules" around buttocks, sacrum, lumbar region, and shoulders. Many of these were extremely tender, and the pressure on these would refer the pain widely and reproduce his "attacks." No pyrexia. Heart, clinically normal. Blood count normal. Urine normal. B.S.R. 8 mm. Electrocardiograph: slight prolongation of P-R interval. Procaine injection of tender nodules produced improvement, but other spots subsequently appeared to replace these, and no permanent cure by this method seemed to be possible. He was re-graded and given a sedentary job again, in which he found himself able to carry on.

Case Z.—Age, 32 years. (A case of chronic fibrositis of 12 years' duration, dating from an attack of rheumatic fever and exacerbated by an attack of "acute fibrositis" lasting three weeks a year later.) Mother is practically a cripple with rheumatism. Father died young. Patient had no rheumatism as child, nor had his sister. No previous illness. He started work in a shipbuilding yard when 16—most of the time up to his knees in water. After a few months of this he began to contract sore throats and colds at very frequent intervals, often laying him up for many days at a time. Latterly he had several attacks of bronchitis. At the age of 20 he had a sudden attack of rheumatic fever in January, with pyrexia, sweatings and hot painful joints. He was in hospital for 6 weeks and felt very ill. He resumed work in the summer, but was conscious of tender spots in his back and shoulders, and he suffered with occasional slight attacks of generalized muscular pain. These have subsequently persisted. In March of the next year the mild pain in his back became intense and spread, and he was again in hospital for 3 weeks. He was pyrexial only on the day of his admission, and did not feel very ill at any time. The condition responded to treatment and he returned to work under his previous conditions. He was able to continue with this until the outbreak of war without interruption, except for a period every winter during which the pains in the back and shoulders became exacerbated and he had to retire to bed and be treated medically for a few days. A few years ago he broke his leg and clavicle in a cycling accident, and he has noticed that "rheumatism" has also settled down at these sites, although the alignment seems to be perfect.

During the last 3 years in the Army he has been in an indoor job and has managed not to go sick in spite of several sore throats. He was seen as an out-patient on account of the pains in the back which still persisted.

On examination looked healthy and the heart appeared to be normal. Many "myalgic spots" which were acutely sensitive to pressure were found along the edges of the trapezius, rhomboid, and lumbar muscles. The blood count and the B.S.R. were normal. Injection with procaine into these spots appeared to relieve the condition very considerably, and he returned to his work.

COMMENT

From a study of these and similar cases one is struck by the clear-cut nature of their history. From this it appears reasonably certain that the fibrositis, from which they now suffer, dates its origin from the first attack of rheumatic fever, and it is often rendered more chronic by a second attack. There seems little doubt that these attacks were genuinely of rheumatic fever, and the definite family history of rheumatism in each case is noteworthy.

There does not seem to be any suggestion that it is "chronic rheumatic fever" rather than fibrositis from which these patients are now suffering, since in the cases quoted the patients were well in themselves, no cardiac lesions had developed, and the temperature and B.S.R. were normal. It was also in all cases the soft tissues and not the joints which were affected, except during the original acute attacks. The "trigger points" and the distribution and reference of the pain are now typical of chronic fibrositis. There seems, therefore, no reason to differentiate this end result from fibrositis of other aetiology. It is submitted that this view is rationalized by the ideas as to the basic nature of rheumatic fever which have been put forward earlier in this paper.

It has been observed elsewhere (Copeman, 1943) that certain areas of the body in susceptible people can be rendered prone to fibrositis as the result of attack by one of several of the common fevers, and that if reactivation by these or other infections occurs before resolution has taken place the condition will become chronic. The aetiology of chronic fibrositis in these cases, although infective, is not therefore a specific one. Acute fibrositis indeed seems to be a common temporary host reaction to many separate types of infection, and will sometimes persist. In the case of rheumatic fever it seems possible that it is this non-specific element of the fever which initiates the disease and a specific rheumatic factor which later intensifies and prolongs the process in certain cases; and which will in time presumably also produce cardiac lesions. The prognosis would seem to depend upon the speed and dosage in which this specific factor follows up the attack of the initiating non-specific factor. In the cases described of "acute febrile myalgia" the appearance of the specific factor would appear to be sufficiently delayed to enable the patient (whose normal resistance is high) to establish adequate defence. It might therefore be regarded as the clinical link between acute rheumatic fever and chronic fibrositis. In debilitated patients—i.e. those in whom resistance is normally low (or in whom the "dosage" of "preceding factors" has been high)—the disease

will run its classical course, the specific element blending from the start with the initial non-specific febrile fibrositis which seems to be the basic element of the disease.

Summary and Conclusions

An exceptional opportunity was presented for studying the natural history of rheumatic disease in a body of healthy young adults who were isolated from civilized environment for a considerable time in a remote desert. Later, other unusual opportunities arose for studying certain cases of acute rheumatic fever.

It is generally agreed that acute rheumatic fever shows unmistakable signs of being a specific infection. It was established, however, that the possibility of an external source of infection being the cause of the 32 cases suffering with first attacks and 10 cases who had had previous attacks in peacetime was remote. The possibility that the specific infective organism might have been able to lie dormant in the tissues of apparently healthy hosts is therefore examined.

The nature of the infecting organism of rheumatic fever is discussed in the light of clinical experience, and a suggestion is offered to explain the common association of the streptococcus in temperate climates.

The infection gave no evidence of being spread by droplets except in one small series which is described.

It was observed that preceding factors were present in almost every case of acute rheumatic fever, were of two types, and that both were generally operative: (1) A non-rheumatic infection whose role appeared to be to lower the patient's general resistance. This possibly allowed of a successful attack by the hypothetical specific organism. These "preceding infections" were not always streptococcal in type, but comprised the diseases such as dysentery, sandfly fever, and even malaria, which are most commonly met with in the country of observation. (2) A physical factor which comprised chiefly cold, wet, or fatigue in unaccustomed "dosage." A small series of cases is reported in which physical factors in extreme degree appeared to be alone responsible. In a series of cases seen in Malta soon after the siege it is probable that malnutrition was also a factor.

An attempt was made to arrive at the essential nature of rheumatic fever from close clinical observation of this series of cases. Every case showed: (1) General signs and symptoms, such as are common to most general infections. (2) Local signs

and symptoms which comprise muscle and tendon stiffness and pain leading to joint stiffness, swelling, and pain. It was thought that even these symptoms and signs were possibly not in the first instance due specifically to rheumatic fever, but to a non-specific "fibrositic" process (possibly secondary to the fever): the arthritic process arising secondarily by direct extension from this. Cases of true rheumatic fever will, however, show later the specific "stigmata" of subcutaneous nodules and cardiac lesions in addition. It is suggested that these two specific lesions are of similar nature as regards their morbid anatomy.

This view leads to the conclusion that acute rheumatic fever is basically an acute febrile and progressive form of fibrositic reaction occurring in localized areas of tendon sheaths adjacent to joints. The "joint pain" complained of appears to be really referred from these areas, and later, when effusion occurs in the joints, it is generally a secondary extension of the inflammatory process from the affected tendon sheaths, and not a primary arthritis of blood-borne origin. It is only when the specific element comes into the picture in sufficient "dosage" that the condition becomes differentiated from the polyarthritis which may occur as the result of other types of infection such as the gonococcal or meningococcal. (A method of estimating this specific factor would determine prognosis more accurately than will clinical observation alone.)

Three clinical types of rheumatic fever were observed: (1) The classical type, which ran its accustomed course. (2) A "benign" type which has not, it is believed, been previously described. This appeared to be non-epidemic, of short duration, and probably not uncommon. For this syndrome the name "acute febrile myalgia," is suggested. Typical case-histories of patients suffering with this type of disease are recorded. (3) The classical type which, instead of resolving, appeared to merge into a condition of chronic fibrositis, which was shown to be difficult to differentiate from fibrositis arising from other causes.

My thanks are due to Brigadier Evan Bedford, and Lt.-Col. W. L. Ackerman for reading this paper and making suggestions.

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