infiltrated with an oily anaesthetic solution—such as benzyl salicylate—under pressure, with the same object in view. The fibrous compartments separating the fat lobules should afterwards be disrupted with the cutting edge of the wide-bored needle employed, to prevent recurrence.

Surgery.—Surgery has no place in fibrositis in which herniation is not a cause of the pain. Removal of irreducible fat herniae which cannot be disrupted by injection, however, remains the logical procedure, and in well-chosen cases is highly successful. These structures, however, are sometimes surprisingly difficult to locate in a well-covered person and local analgesia is essential, as the patient's subjective impressions are often necessary to ensure that it has been rightly localized. It must not be forgotten that herniations are sometimes multiple. In cases of panniculitis with obesity, persistent pain which cannot be remedied by other means can sometimes be cured surgically by undercutting the painful area. Presumably the sensory nerves responsible are cut in the course of this procedure. In two cases of persistent severe pain situated in an enlarged fat-pad on the medial aspect of the knee-joint the whole pad was excised, with permanent relief of pain, to the satisfaction of the sufferers, who had found no relief from less drastic procedures.

Several papers have been published in America during the last few years confirming the part played by fat herniae in the causation of pain in the lumbar and gluteal regions. The largest series is that of Hertz, of Cleveland. In a personal communication in 1948 he stated that since 1944 his series of such cases had reached a total of 229, 68 of which he had operated upon. At his last follow-up 62 of these had maintained complete relief from pain. Hutcherson, in a paper read before the Association for Surgery of Trauma in July, 1948, reported 42 such cases operated upon, with permanent relief of pain in 40. In England I know of no published series, although I have received accounts of isolated operations of this type which have been successful. Among surgeons who have communicated with me in this way I wish to thank Messrs. L. E. C. Norbury, S. L. Higgs, Rodney Maingot, Harold Edwards, J. C. R. Hindenach, and David Trevor.

Summary

It is thought that an aetiological classification, although desirable in the case of a "disease entity," must remain impracticable in the case of a syndrome. The classical conception of fibrositis is briefly reviewed, and a classification based upon the structure of the nodules found at the source of the pain is tentatively proposed.

Evidence is produced that the fat tissues of the body are subject to pathological variations which cause pain. This pain is generally labelled as "rheumatic" or "fibrositic." When this occurs in the neighbourhood of joints the condition is often wrongly termed arthritic.

It has been shown that several recognizable "rheumatic" syndromes can be the result of an abnormal retention of fluid by fat lobules in certain situations. When this occurs they endeavour to swell, but, being confined by indistensible fibrous tissue, they are unable to do so, and tension pain results. This oedema shows no sign of being inflammatory in nature.

In some such cases a flaw occurs in the fibrous covering, resulting in herniation of the enclosed fat lobule into an adjoining layer. Such herniations may be of three types, which are described.

The origin of this selective swelling is probably endocrine. The direct effect of cold may unmask it.

The syndromes described as being due to this abnormality of fat are: (*a*) "fibrositis," which occurs in any of the several normally occurring fat-pads which are described, and (*b*) panniculitis, which occurs in abnormally deposited fat, mostly in predictable sites. Panniculitis may occur with or without general obesity. In either case the chief sites of pain tend to remain similar. Adiposis dolorosa (Dercum's disease) would appear to be only a quantitative variation of this combination.

Treatment must be directed concurrently along several lines if success is to be achieved. Those discussed include diet, endocrines, diuresis and dehydration, local injections, physiotherapy, and surgery.

The indications for surgery are limited but tend to afford dramatic relief. These are the removal of irreducible fat herniae, and to a smaller extent removal of localized areas of painful tissue in severe cases of panniculitis.

The illustrations are from a film-strip kindly made for me by Mr. E. Wilson in the Photographic Department of the Royal Society of Medicine.

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RHEUMATOID ARTHRITIS IN THE YOUNG*

BY

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[WITH PHOTOGRAVURE PLATE]

The aetiology of rheumatoid arthritis still remains unsolved, but fresh light has been cast on the problem by a wider view of the clinical picture. The term "rheumatoid disease" has been wisely suggested (Ellman and Ball, 1948), and realization of the extensive nature of the condition, with less emphasis on the locomotor system, is a great step in the right direction. Arthritis has obviously attracted the most attention, but concentration on this single manifestation has certainly hampered search for the cause. That is why a study of the disorder can most profitably be undertaken in the young, for it is then that disease in general appears in its most acute and unadulterated form.

My remarks will be chiefly confined to certain aspects of Still's disease, which all now agree is no more than an acute form of rheumatoid arthritis in children. Further, I will try to correlate several striking features which have appeared fairly constantly in the group I have been studying with some of the rarer syndromes described by the able physicians of the past.

The study of rheumatoid arthritis, even in children, has to my mind centred too largely on the disease after it has become well established. Investigators have often not the opportunity of witnessing the onset and must rely largely on second-hand accounts from the child's relatives. In this way important developments may be missed.

Acute examples of a disease are also more likely to give information than insidious cases. I have therefore chosen for study from my case notes 20 children all of whom suffered from considerable fever and the usual general acute manifestations first described by Still. In nearly all of them careful clinical observations were made early in the disease, if not from the onset; subsequently the progress of the children was followed for many years, sometimes into adult life.

The account deals particularly with the early stages and general aspects of the disease; following the principles already expressed, arthritis will receive only short consideration.

*Read in opening a discussion in the Section of Rheumatology at the Annual Meeting of the British Medical Association, Harrogate, 1949.

Mode of Onset†

This is more or less abrupt and could often be dated to a day. In most instances there was considerable fever and the disease began with *arthralgia*. At first there was little obviously wrong with the joints until attempts were made to move them, which were painful. Sometimes they were puffy and, though somewhat stiff, movement was not greatly restricted. Later definite *arthritis* set in, but at this early stage it was usually migratory, flitting from joint to joint and lasting only for a few hours (see Chart I). Redness of the surrounding skin has been observed in some instances, and the glistening appearance, swelling, and pain so resembled gout that blood uric acid levels were estimated but were always found to be normal. Sometimes the onset was like that of rheumatic fever, but failure of adequate salicylate therapy soon proved this diagnosis to be wrong.

In a few cases joint manifestations were minimal or entirely absent, and the main clinical features were listlessness, anorexia, adenitis, an unexplained fever, and a rash. The diagnosis then generally inclined towards glandular fever or brucellosis, but this was not supported by laboratory tests.

Adenitis.—This constantly occurred at some period, but with few exceptions could not be counted as one of the earliest features. Once present the enlarged glands persisted throughout the acute phase and disappeared only if the child made a good recovery. When a more chronic form of rheumatoid arthritis developed glandular enlargement continued until the disease became "burnt out."

CASE	SEX	AGE AT ONSET	RASH	SPLEEN	LIVER	JAUNDICE	INITIAL LEUCOCYTOSIS W.B.C. per cmm.	LATER LEUCOPENIA	PERICARDITIS	FEVER: IRREG. CONT.	FEVER: PERIODIC	RESULT
1 C.B.	M	10/12	+	±	++	SLIGHT	18,000 P. 67	1500 P.15 L.85	P.M.	+	±	* LEUKÆMIA DEATH
2 T.D.	M	2 4/12	+	±	–	–	24,000 P 84	–	–	+	–	ARTHRITIS AMBULANT
3 J.C.	F	6 6/12	+	+	–	–	31,000 P 92	–	–		+	COMPLETE RECOVERY
4 A.B.	M	10 6/12	–	–	–	–	17,000 P. 72	–	–	+ →	±	ARTHRITIS
5 W.C.	M	5 2/12	+	–	–	–	22,000 P 78	–	–		+.	RECOVERY
6 E.L.	M	3	–	–	–	–	14,400 P 66	2,900 P.53 L.44	P.M.	TERMINAL		* DEATH
7 R.H.	M	6	+	–	–		22,800 P 77	–	+		+	ARTHRITIS
8 I.M.	M	7 6/12	+	–	–	–	20,000 P. 80	–	–	+ →	+	MIGRATORY ARTHRITIS
9 M.N.	M	2	+	–	–	–	29,000 P.82	—	–		∓	DEATH (GOLD) *
10 K.S.	M	4 3/12	+	+	+	–	12,700 P.84	–	–	+ →	+	MIGRATORY ARTHRITIS
11 W.H.	M	2 7/12	+	++	+++	SLIGHT	19,200 P.57	–	–	+ →	+	NO ARTHRITIS HEPATIC CIRRHOSIS
12 G.H.	M	6	+	+	+ later	+	21,200 P. 82	–	–		+	SLIGHT ARTHRITIS DEMENTIA
13 M.H.	F	3 9/12	+ later	+	+	–	NORMAL	–	?(PLEURISY)	+ ←	+	ARTHRITIS
14 A.B.	M	9/12	–	+ later	+ later	–	19,000 P 75	–	?(PLEURISY)	+ ←	+	SEVERE ARTHRITIS
15 A.B.	F	2 2/12	+	+ later	+ later	+	28,200	3,400 P.71 L.24	+		+	SLIGHT ARTHRITIS RECOVERY FROM LEUCOPENIA
16 M.C.	F	9	–	±	+	–	17,500 P. 72	–	–	+ ⇄	+	SEVERE ARTHRITIS
17 J.C.	M	4	+	+ later	–	–	22,600 P. 87	–	+	+ →	+	ARTHRITIS AMBULANT
18 E.M.	M	5	+	+	+ later	–	16,500		+	+ ←	+	RECOVERY
19 S.D.	F	3	+	–	–	–	13,900 P. 76	–	–	±		ARTHRITIS
20 D.P.	M	3 9/12	–	++	+	–		3,000 P.3 L.97	–		+	* LEUKÆMIA DEATH

Table incorporating the chief clinical points referred to in the text. Adenitis was an early feature in all cases. Subcutaneous nodules appeared only in Cases 8 and 12 at a later stage. Gold therapy in Case 9 provoked a severe and fatal reaction almost at once.

Splenomegaly.—This was not prominent at the beginning and was found to any degree in only just over one-third of the cases. Later enlargement of the spleen became more obvious.

The Rash.—This appeared in 15 children, usually at the onset, and varied in intensity more or less with the temperature. At a later stage it vanished altogether, and that is no doubt why it has not been greatly stressed in the literature. The rash had a fairly widespread distribution on the limbs, the trunk, and occasionally the face. It was sometimes urticarial at first but without the usual pronounced irritation; soon it became maculo-papular and was often migratory. With the associated adenopathy it is not surprising that cases have been wrongly suspected of being rubella. Rarely the macules fused into a widespread erythema; occasionally petechiae appeared and the capillary fragility was found to be raised.

Fever.—In his original description Still stated that the fever might be continuous or might occur in bouts. Both types were seen in 16 of the 20 patients: usually a prolonged intermittent or a high irregular continuous temperature in the opening stages, giving way to periodic febrile attacks later (see Charts I and II). The periods of pyrexia were not so clearly defined as in Hodgkin's disease or melitensis, or undulant, fever, but careful analysis clearly showed their phasic character, which varied considerably from case to case and from bout to bout (see Charts I–III). In fact, the temperature in some children never remained normal for long; nevertheless the "switchback" appearance of the curves was typical (see Chart IV).

Both joint involvement and the rash tended to lessen or to vanish completely when the temperature dropped. Adenitis and a raised B.S.R., on the other hand, persisted throughout the acute and active stages of the disease. During the bouts of fever the child was very miserable, obviously ill, and in pain. Improvement followed the fall in temperature, but the clinical condition slowly deteriorated with each successive rise. Eventually the febrile attacks ceased and a typical clinical picture of varying degrees of rheumatoid arthritis remained (11 cases). Complete recovery with no obvious residual arthritis was the final result in three (cf. Hench, 1949), death occurred in four from causes which will be discussed later, and in two the acute stage has not yet terminated.

Blood Picture.—With any prolonged fever anaemia is to be expected, and it was not surprising to find before long a reduction of the red cells to between three and four million per c.mm., with a corresponding drop in the haemoglobin. More significant was the pronounced leucocytosis, almost invariably present at the beginning of the disease (18 out of 20 cases). Counts between 17,000 and 30,000 white cells per c.mm. were commonly registered, with a pronounced polymorphonuclear cytosis in the region of 80 to 90%. Very soon, however, the number of white cells returned to normal and leucocytosis was seldom repeated (see Table and Chart IV).

On rare occasions and at any stage the disease appeared to become too much for the bone marrow. Severe leucopenia then set in. Both neutrophils and lymphocytes might be equally affected, but agranulocytosis has also been observed (Plate, Figs. 1 and 2), and in two cases the final clinical picture and bone-marrow biopsy were indistinguishable from that of *lymphatic leukaemia* in an aleukaemic phase (Cases 1 and 20). Leucopenia developed in four cases—three patients died (Cases 1, 6, and 20), and one recovered rapidly and soon had a normal blood count (Case 15).

Other Clinical Features and Complications

Pericarditis is a well-known manifestation, and occurred in six of the series. It may in fact precede all other manifestations and be mistaken at first for acute rheumatic pericarditis (Chart III). Doubt arises when the illness does not follow the usual course and the myocardium is not found to be greatly involved. Sometimes the physical signs are not very obvious; pericarditis is then masked by the acute febrile disturbance and is observed only at necropsy (Cases 1 and 6).

†The following section should be read in conjunction with the accompanying Table, in which the main features of the 20 cases are summarized.

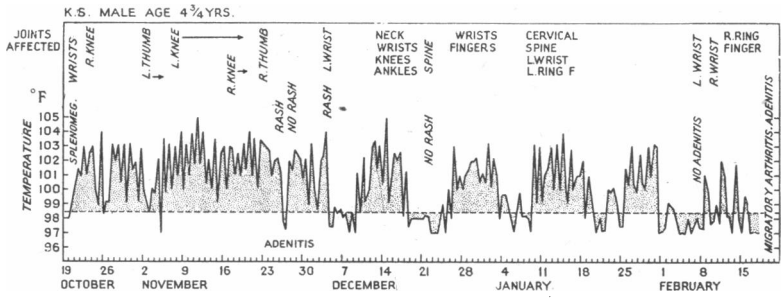

CHART I.—Temperature chart of a boy (Case 10) in the early stages of his disease, showing at first an irregular continuous form of pyrexia which subsequently became phasic in character. The migratory nature of the arthritis is also well demonstrated.

Pleurisy and pneumonitis are commonly associated with the pericarditis, but occasionally they may occur on their own (Cases 13 and 14) (cf. Ellman and Ball, 1948).

Jaundice figured as an occasional complication in Still's original account. In the present group it appeared in four cases and the liver became enlarged in nine. In one remarkable instance (Case 11) the liver and spleen reached well below the umbilicus, and laboratory tests at the time demonstrated impaired liver function. After a year's illness all signs of rheumatoid arthritis vanished; the boy appeared well and active, but was left with an enlarged spleen and *cirrhosis* of the liver (see Chart II and Plate, Fig. 3). Amyloid disease has been described (Imrie and Aitkenhead, 1939), but it was never demonstrated during life or after death in the present series.

Only one account of *encephalitis* as a complication could be found in the literature (Spühler, 1941). This was in three adults with Still's type of acute rheumatoid arthritis. In one of my cases (No. 12) convulsions and prolonged coma suddenly developed with pneumonia, pyelonephritis, cardiac failure, and transient hyperpiesia (B.P. 200/120) as additional features (see Chart IV). The boy recovered from all these, but has been permanently aphasic and demented ever since. The aetiology of all this is obscure. Polyarteritis nodosa has lately been much debated in relation to rheumatoid arthritis, and the question naturally arose in this case, but the condition was not confirmed by muscle and vessel biopsy.

Among other early features in the series were excessive sweating, rapid muscular wasting, and generalized bone rarefaction. Later hirsuties, particularly round the labia, was often noticed in quite young girls and is difficult to explain. One other factor should be mentioned: in a few isolated instances the onset of the disease followed within a few hours of some severe trauma or shock, such as a fall down a deep chalk pit, with concussion, or sudden accidental immersion in cold water. Such occurrences may be purely fortuitous and have no bearing on the problem, but they may have some aetiological significance. Similar happenings are well known to anyone who has had wide experience of juvenile rheumatism, and serious relapses have been observed in children with acute rheumatic carditis following fractures, after surgical operation, or subsequent to a long drive in an ambulance (Bland and Duckett Jones, 1935).

Relationship to Other Syndromes

Henry Cohen (1948) in a chapter on "the rarer arthritic syndromes" has picked out one or two groups which seem to be interrelated. Some of them bear a striking resemblance in part of their clinical course to the acute form of rheumatoid disease just described. In his description of *intermittent hydrarthrosis* Garrod (1910) refers particularly to the associated skin rashes, which might be urticarial, erythematous, or haemorrhagic. Although the knee-joints were usually alone affected, others have observed a migratory arthritis. The disorder differs, however, from the disease we are considering in its more benign course and in the greater regularity of its periodic outbursts. In Garrod's cases the condition was mostly superimposed on damaged joints; in mine it was often the precursor of permanent arthritis. It is interesting to find that Garrod also mentions injury as a precipitating cause.

Pyrexia is not a great feature of intermittent hydrarthrosis; when present it coincides with the joint swellings and exhibits a fairly regular periodicity. More severe fever occurred in an allied disorder which Solis Cohen (1913) called "angioneural arthrosis." About 40% of his cases were febrile, and in a number of them the pyrexial attacks had the same phasic character.

Comparable also to a few of the cases in my series was the erythematous or gouty appearance of the joints during the attacks. More or less the same clinical picture was described by Hench and Rosenberg (1944) in a condition they term "palindromic rheumatism," which seems to be a milder form with no effect on the blood sedimentation rate, no fever, a normal blood count, and little tendency to chronic arthritis, but with widespread nodule formation. Palindromic rheumatism has also attracted the attention of Parkes Weber (1946), who finds a common thread running through all these separately described syndromes, including also those of Schlesinger (1899) and Kahlmeter (1939). In many respects their course differs considerably from that of Still's disease in its usual form, but several striking points of resemblance can be found.

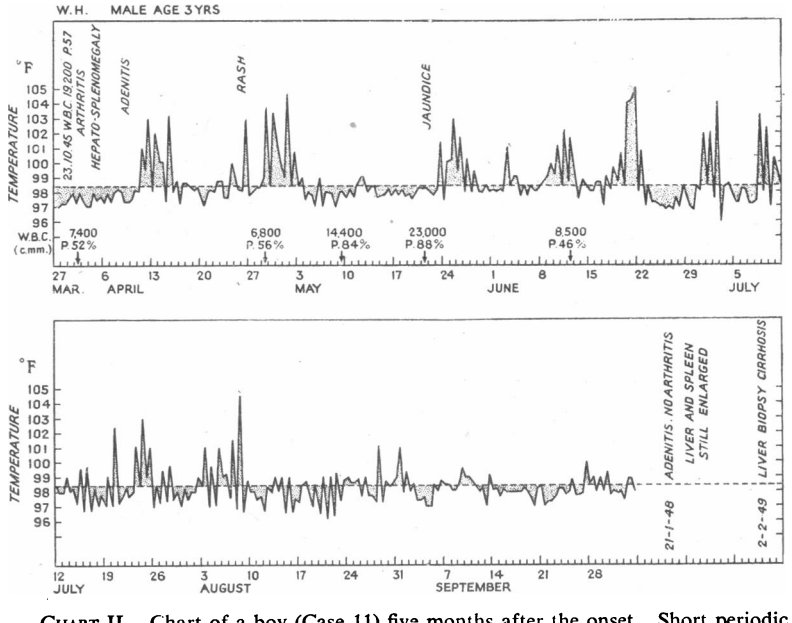

CHART II.—Chart of a boy (Case 11) five months after the onset. Short periodic bursts of pyrexia are shown. Leucocytosis, present at the onset, is no longer evident; the temporary recurrence later was associated with jaundice. There was complete recovery from arthritis, but cirrhosis of the liver developed (see also Plate, Fig. 3).

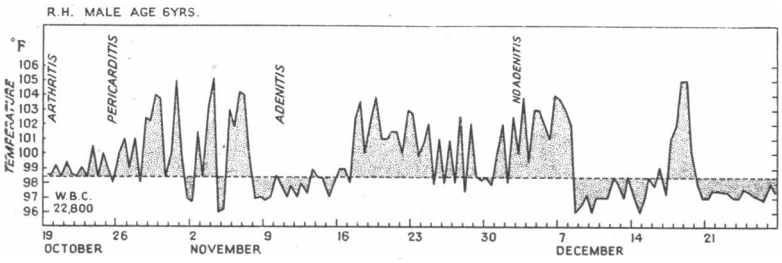

CHART III.—Chart of Case 7 with pericarditis as an early feature. Irregular bouts of fever are again obvious.

Precursor to Leukaemia

The rare termination of Still's disease in lymphatic leukaemia, which I have discussed, raises all kinds of possibilities. Preceding this there may possibly be a stage of undifferentiated leucopenia (panleucopenia), a complication of acute rheumatoid arthritis first described by Felty (1924). In his syndrome the neutrophils were not specially picked out and the low white cell count persisted for many months without the patient being in great distress or the joint affection severe. Felty remarked upon the associated pigmentation and the resemblance of his cases to those described by Banti. The glands and spleen were enlarged but not the liver, although in one case some liver impairment was suspected.

There are also one or two accounts of agranulocytic leucopenia in rheumatoid arthritis, not attributable to any specific form of therapy (Collins, 1937; Blechmann and Carcanagues, 1940). None of these cases proceeded to leukaemia, but this followed in one patient described by Türk (1938).

It seems as if the severer cases of Still's disease are in danger of developing leucopenia. From this the patient can occasionally recover, but the condition is more likely to proceed to agranulocytosis and in rare instances degenerate into leukaemia. Further research by repeated bone-marrow biopsy during the course of the disease is required in order to discover the exact sequence of events.

Discussion

Recently some form of allergy has again been suggested as the basis of the pathological process in rheumatoid arthritis, and in support of this two rare conditions—lupus erythematosus and polyarteritis nodosa—have been repeatedly quoted. Both are thought to be allergic disorders and to bear a certain resemblance to acute rheumatoid disease. This is suggested by the pathology of *lupus erythematosus*, in which the collagen tissue is chiefly affected by fibrinoid changes (Klemperer, 1948; Humphreys, 1948). The clinical features—fever, glandular swelling, arthritis, anaemia, leucopenia, and occasionally pericarditis and diffuse or focal cerebral disease—are also significantly similar to the symptomatology we have been considering. Focal myocarditis, another frequent lesion in lupus erythematosus, might be taken as further evidence of a common pathology, particularly by those who consider rheumatic fever and rheumatoid arthritis to be associated.

The morbid anatomy of *polyarteritis nodosa*, on the other hand, does not seem to bear such a close relationship, but its clinical course is somewhat similar, particularly the arthritis, fever, sweating, prostration, tachycardia, skin eruptions, and occasional involvement of the pericardium (Schlesinger and MacCarthy, 1949).

Glomerular nephritis, which is common in these two rare disorders, is not a feature of Still's disease. The link here is perhaps more with serum disease produced experimentally in animals and resulting in a number of lesions which are also found in human serum disease, nephritis, polyarteritis nodosa, rheumatic fever, and rheumatoid arthritis.

Comparison of Still's disease with these two rare disorders, whose character has only recently become clearer, and with syndromes of the past in which an infective factor has always been in doubt does seem to favour some allergic basis as the underlying cause. Its exact form is still most obscure. No one has yet discovered the nature of the antigen, explained how it produces a prolonged disease with progessive inflammatory lesions, or discovered what predisposes certain individuals and allows others to escape. Possibly some organism, normally innocuous, may be living in harmonious symbiosis with its human host and suddenly become pathological when the balance is somehow deranged by stress or trauma. This might explain the occasional effects of shock, to which attention has already been drawn.

The dual role, partly infective and partly sensitizing, that bacteria can play in the development of disease is well known. The tubercle bacillus in tuberculosis and the streptococcus in nephritis and in purpura are good examples. So far no specific infection has been constantly found to precede the onset of rheumatoid arthritis, but an infective factor is strongly suggested by the pronounced polymorphonuclear leucocytosis which was revealed in the early stages of nearly all the cases under review. Leucopenia and agranulocytosis might possibly be the detrimental effect on the bone marrow of repeated allergic reactions.

The changing character of the fever from a more or less continuously high level to an irregular periodicity might also conceivably be regarded as the successive result of an infective and an allergic process.

Mesenchymal lesions in the skin, lymph nodes, joints, heart, lungs, and spleen result from the experimental

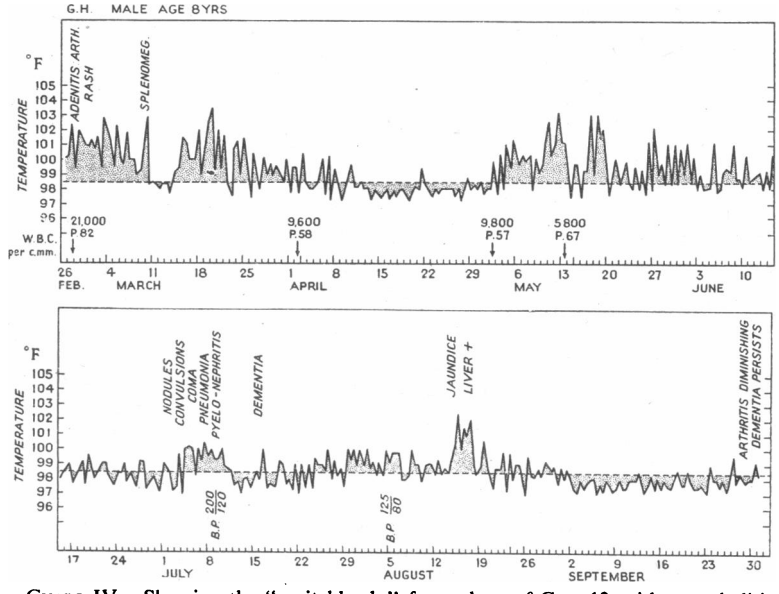

CHART IV.—Showing the "switchback" fever chart of Case 12, with encephalitis and dementia as complications. Initial leucocytosis occurred, but did not persist for long

intravenous injection of soluble antigens (horse serum) into animals, while somatic antigens (whole bacteria) produce their effect mainly in the lungs, liver, and spleen (Ehrich *et al.*, 1949; Gregory and Rich, 1946). Most of these are regions commonly involved in Still's disease, and, as has been shown, the lungs, the heart, and the liver may not escape.

Conclusions

Study of the most acute cases of Still's disease emphasizes the widespread nature of the lesions. Although the majority of cases proceed to progressive arthritis, some patients may recover completely. Characteristic types of pyrexia can be recognized at different stages, and significant effects on the blood picture have also been observed. In rare instances the clinical picture may change from Still's disease to leukaemia. The close resemblance of Still's disease, particularly at the onset, with certain rare syndromes of an allergic nature suggests that rheumatoid arthritis may have a similar aetiological basis. Should this be so, some form of primary bacterial invasion seems to be likely in the course of events.

I am indebted to several of my medical colleagues for allowing me to study their cases, and to Dr. Bodian and Mr. Martin for the histological sections and microphotographs reproduced in the photogravure plate.

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The current issue of *Planning* is the three-hundredth in the series which PEP began some 17 years ago. It looks back on the early history of PEP between its foundation in 1931 and the outbreak of war. A brief account of its origin mentions the men who were most concerned with the development of the group. In the early days PEP was given some special grants by the Rockefeller Foundation, but over the period 1933–9, 12 major and two minor reports and more than 150 broadsheets were published on a budget of no more than £56,000 for these seven years. There is a brief description of the *ad hoc* working groups set up by PEP, and an attempt is made to assess its contribution to contemporary knowledge and ideas. The first major report to be prepared by the Civic Division of PEP was that on *The British Social Services*, and this was followed in December, 1937, by the *Report on the British Health Services*. Three groups which did important work during the war were the Social Security Group, the reconstituted Health Group, and the Population Policies Group. The report on *Population Policy in Great Britain*, however, was not published until April, 1948.

PSYCHOGENIC RHEUMATISM

BY

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In a large hospital to which patients are referred suffering from "rheumatism" considerable diagnostic experience is needed in order to classify the various conditions included in this omnibus term. There is no agreement at present on the scope of the term; it is customarily taken to indicate a group of painful disorders of the locomotor system. It is obvious that a large number of syndromes come under this heading. While the arthritides, neuralgias, and other somatic conditions as a rule present straightforward signs and symptoms, patients are continually encountered whose experiences do not seem to fit in with any recognized syndrome and in whom physical signs are absent or minimal. In such cases one may be tempted to regard the patient as suffering from pain which is primarily of psychogenic rather than organic origin. If this hypothesis is correct, it is plain that rheumatic disorders of this type call for particular care in diagnosis and for treatment more far-reaching than analgesics and physiotherapy.

In recent years there has been a growing disinclination to accept all rheumatic complaints as somatic in origin. Even before the war one of us had coined the term "polyalgia syndrome" to describe the symptoms of which some patients complained. In England, Ellman *et al.* (1942) studied 50 patients with "fibrositis": 23 had no physical signs, although they had been diagnosed as suffering from somatic disease; 25 showed hysterical tendencies. An interpretation of the rheumatic complaint in psychological terms was given by these workers. An important contribution was made by Flind and Barber (1945); many of the subjects investigated by them were found to be suffering from psychiatric rather than organic states. Hench and Boland (1946) gave a full description of psychogenic rheumatism in a paper on the incidence of rheumatic disease in the United States Army; this condition was the third most common diagnosis made in their series of 800 patients.

If psychogenic rheumatism is to be accepted as an entity both by those who diagnose and treat rheumatic disease and by psychiatrists, our concept of it needs clarification and acceptable diagnostic criteria must be established. This demands close collaboration between clinician and psychiatrist: it is the purpose of this paper to show the results of such an endeavour.

Fifteen patients regarded by the clinician as suffering from psychogenic rheumatism were referred for detailed psychiatric examination. In each case the patient was first examined by routine clinical methods. The haemoglobin and sedimentation rate were estimated. The group contained 14 women and one man; the distribution by age was: 15–29, four; 30–39, three; 40–49, four; 50 and over, four. The preponderance of women in this group was in accord with the assumption that psychogenic rheumatism in clinical practice is considerably commoner in women. A control group of patients suffering from a painful disorder of which the cause was known was investigated by the same means. This group consisted of subjects of the same age and sex distribution, and broadly of the same social class. The diagnoses were: osteoarthritis (3),

G. H. JENNINGS, J. L. HAMILTON-PATERSON, and F. O. MACCALLUM: FATAL CASE OF POLIOENCEPHALITIS DUE TO POLIOMYELITIS VIRUS

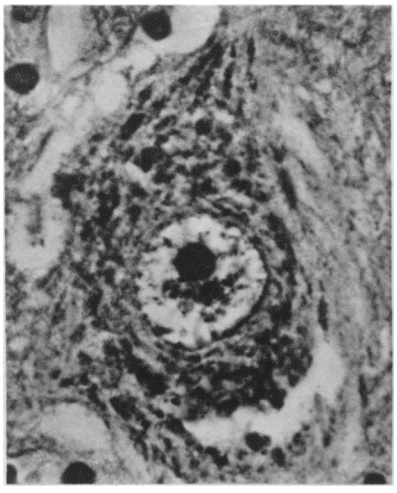

FIG. 1.—Normal anterior horn cell. H. & E. (× 1200.)

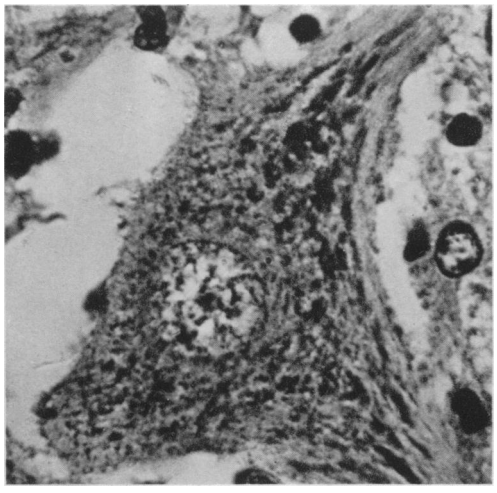

FIG. 2.—Anterior horn cell showing loss of nuclear membrane and nucleolus; partial granulation of tigroid substance. H. and E. (× 1200.)

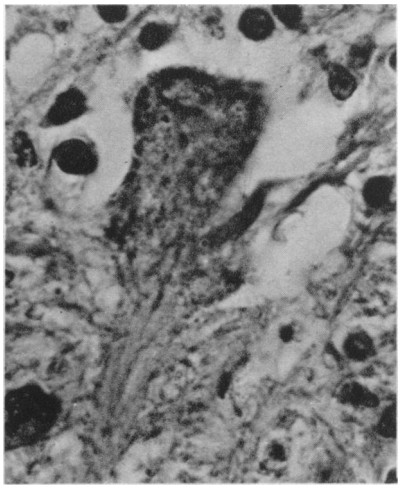

FIG. 3.—Complete loss of nucleus and complete granulation of tigroid substance. H. and E. (× 1200.)

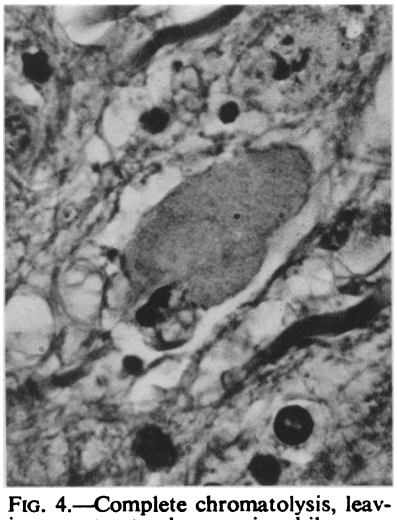

FIG. 4.—Complete chromatolysis, leaving a structureless eosinophil mass. H. and E. (× 1200.)

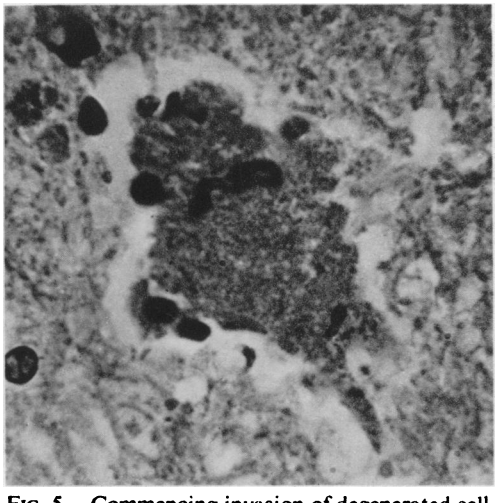

FIG. 5.—Commencing invasion of degenerated cell by histiocytes (monkey). H. and E. (× 1200.)

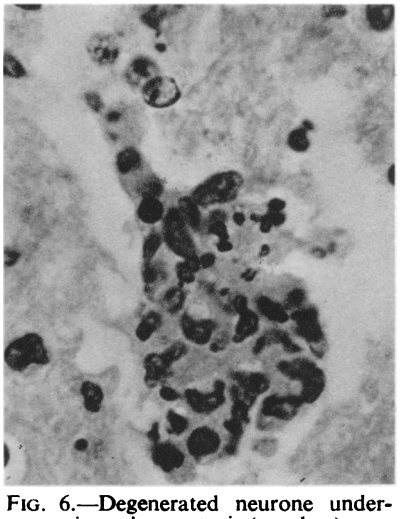

FIG. 6.—Degenerated neurone undergoing phagocytosis (monkey). H. and E. (× 1200.)

BERNARD SCHLESINGER: RHEUMATOID ARTHRITIS IN THE YOUNG

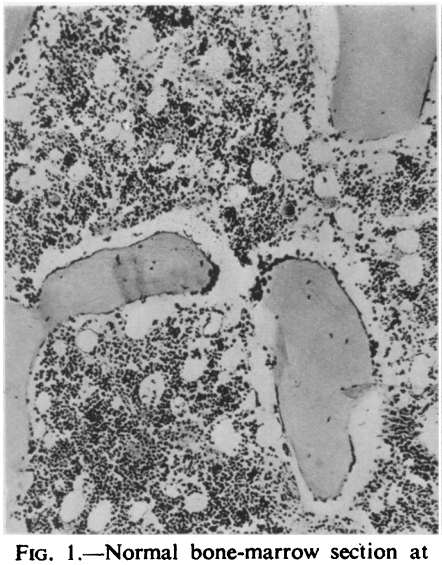

FIG. 1.—Normal bone-marrow section at necropsy.

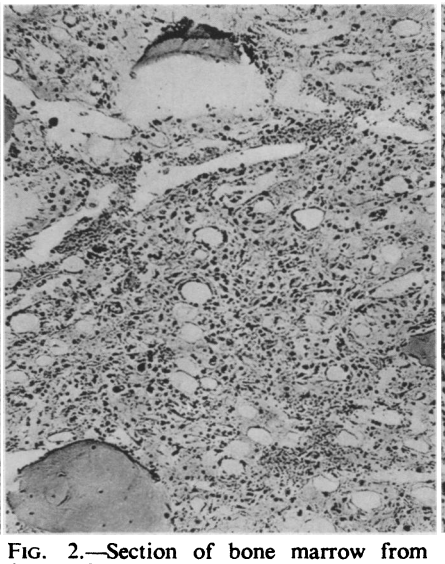

FIG. 2.—Section of bone marrow from femur of Case 1. Hypocellular, with areas of necrobiosis.

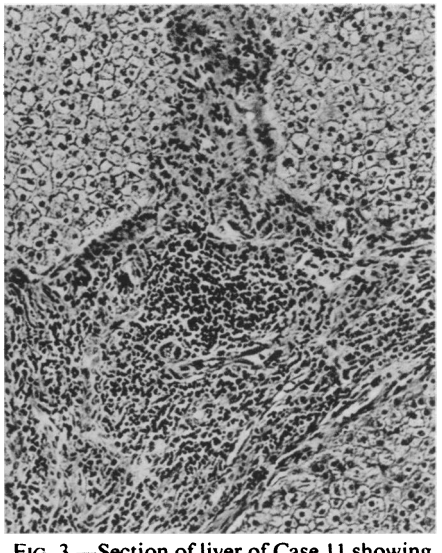

FIG. 3.—Section of liver of Case 11 showing perilobular fibrosis and infiltration of inflammatory cells.