PALINDROMIC RHEUMATISM

BY

STEPHEN MATTINGLY

Department of Rheumatology and Physical Medicine, The Middlesex Hospital

Palindromic rheumatism was first described by Hench and Rosenberg in 1941, and three years later a detailed account of their 34 cases appeared in the *Archives of Internal Medicine* (Hench and Rosenberg, 1944). A number of individual case reports subsequently appeared in the literature (Ameen, 1954; Cain, 1944; Ferry, 1943; Ginsburg, 1948; Grego and Harkins, 1944; Gryboski, 1948; Hopkins and Richmond, 1947; Lewitus, 1954; Mazer, 1942; Neligan, 1946; Paul and Logan, 1944; Paul and Carr, 1945; Perl, 1947; Rotés Querol, 1956; Salomon, 1946; Scheinberg, 1947; Thompson, 1942; Parkes Weber, 1946; Wingfield, 1945; Wirtschafter, Williams, and Gaulden, 1955; Wolfson and Alter, 1948; Wassmann, 1950; Zentner, 1953). However, there have been few reports of series of patients with this syndrome (Ansell and Bywaters, 1959; Dames and Zuckner, 1961; Rotés Querol and Lience, 1959) and although reference is made to 140 cases seen at the Mayo Clinic (Ward and Okihiro, 1959) these have not yet been reported in the literature (Ward, 1965).

Hench and Rosenberg (1941) coined the term "palindromic rheumatism" to describe recurring attacks of joint pain and swelling. Usually only one joint was affected at a time, although practically every joint in the body had been affected, including the temporo-mandibular joint. The shoulder, knee, wrist, and small joints of the hand were most frequently involved and pain in the neck was common. The onset was often sudden and although an attack could start at any time of the day or night it frequently began in the late afternoon. Each attack lasted a few hours or a few days, but rarely as long as a week. Pain was most severe a few hours after onset and often prevented sleep; it could be very disabling and even lead to admission to hospital. Joint swelling was usually evident and the overlying skin was often red. The interval between attacks was very variable and irregular, unlike that in intermittent hydrarthrosis, and between attacks the joints appeared normal.

One third of their patients experienced so-called "para-articular attacks" affecting the soft tissues near joints or even overlying muscles away from joints.

Painful non-pitting tender swellings an inch or more in diameter, and occasionally much larger, appeared over the forearms, back of wrist, or heel. Sometimes the finger tips became swollen and transient intra-cutaneous or subcutaneous nodules were observed on the hands, but usually disappeared within a few days.

Patients suffering from palindromic rheumatism remained well and did not lose weight. Attacks were usually afebrile. Radiographs were normal in most cases or showed coincidental degenerative changes. Laboratory investigations usually gave normal results, although there was sometimes a transient rise in the erythrocyte sedimentation rate during an attack, the white cell count might show a relative lymphocytosis, and the serum fatty acids were increased in some patients. In a few cases, biopsy of a joint during an attack revealed a non-specific acute inflammation which subsided rapidly; the fibrinopurulent joint fluid contained many polymorphs but was sterile on culture and contained no urates.

Most patients experienced a succession of attacks affecting different joints at irregular intervals; they might occur a few times a year or many times a week, and might continue indefinitely over 20 years or subside spontaneously. No treatment was of value. Hench and Rosenberg (1944) concluded that "despite thousands of attacks suffered during a grand total of 307 years of illness, not a single joint had been crippled permanently". The characteristics of palindromic rheumatism as described by Hench and Rosenberg (1944) may be summarized as follows:

Recurrent attacks of joint pain and swelling at variable and irregular intervals lasting a few hours or a few days.

Any joint affected but especially fingers, wrists, shoulders and knees.

Para-articular attacks and transient nodules.

Good health; normal blood tests and x rays.

Good prognosis: no effective treatment.

Their claim that the affected joints remained undamaged even after many years appears to be sub-

stantiated by Fig. 1, which shows the duration of the disease in 48 patients reported in the literature, including their own 34 cases. However it should be noted that a third of the patients had had their disease for less than 5 years.

Many rheumatologists believe palindromic rheumatism to be an atypical form of rheumatoid arthritis and have described cases which subsequently developed arthritis (Boland and Headley, 1948; Bywaters, 1949; Kuhns, 1945; McEwan, 1960; Ropes and Bauer, 1945; Ward and Okihiro, 1959; Ansell and Bywaters, 1959). For example, McEwan (1960), found that seven out of fourteen patients with palindromic rheumatism gave a positive test for the serum rheumatoid factor, and six developed rheumatoid arthritis within a few years.

Boland and Headley (1948) first reported the favourable response to treatment with gold thioglucose in three patients with typical palindromic rheumatism, and further cases were described by Ginsburg (1948), Dames and Zuckner (1961), and Lewitus (1950). However, remissions were often incomplete and the period of follow-up was less than 2 years. In view of the large number of treatments previously tried and found wanting (including purine-free diets, vaccines, colchicine, eradication of septic foci, antihistamines, and sulphonamides, to mention only a few), it is rather surprising that gold

injections had not previously been tried and that so few reports on chrysotherapy have appeared in the literature.

This paper is based on a study of twenty patients (twelve men and eight women) with palindromic rheumatism seen at the Middlesex Hospital between 1955 and 1965, and an attempt is made to answer the following questions:

- (1) Do patients with palindromic rheumatism develop signs of permanent joint damage if followed up for long enough?
- (2) If so, is this rheumatoid arthritis?
- (3) What is the effect of treatment with gold injections?

Middlesex Hospital Series (1955-1965)

During the 10-year period 1955-1965, thirty patients were recorded as cases of palindromic rheumatism. Some of them did not have the syndrome described by Hench and Rosenberg (1944), since joint pain and swelling persisted for weeks at a time, and these have been excluded from this study. Thus only twenty patients (twelve men and eight women) with typical palindromic rheumatism have been reviewed so far, although a further eight cases have been brought into the study group during the past year.

The age at onset of the disease is shown in Fig. 2 (opposite).

The first attack usually occurred without warning in a

Fig. 1.—Duration of disease in 48 cases reported in the literature without serious joint damage, by sex (25 men and 23 women).

PALINDROMIC RHEUMATISM

Fig. 2.—Age at onset of palindromic rheumatism in twelve men and eight women.

patient who was otherwise fit, although in five cases it was preceded by illness or operation (Table I). Four women experienced their first attack during the menopause.

TABLE I
PRODROMAL ILLNESS IN FIVE PATIENTS WITH PALINDROMIC RHEUMATISM

Case No.	Prodromal Illness or Operation
11	Exacerbation of chronic osteomyelitis
12	Sterile abscess of rib incised
14	Acute chest infection
19	Severe attack of measles
20	Laparotomy for sterile tubovarian abscess
	Thyrotoxicosis recently treated with radioactive iodine

Any joint could be affected, apparently at random, and attacks were usually monarticular. However, sometimes one joint was involved immediately after another and, although each joint only remained painful for a day or two, the patient became disabled by a migratory polyarthritis. The joints most affected included the shoulders, knees, wrists, ankles, and small joints of the hand (Table II), the hips, elbows, and feet being less often involved.

TABLE II
JOINTS MOST AFFECTED BY ACUTE ATTACKS IN TWENTY PATIENTS WITH PALINDROMIC RHEUMATISM

Affected Joint	No. of Patients
Knee	17
Wrist	16
Ankle	15
Shoulder	14
Metacarpo-phalangeal	14
Proximal interphalangeal	11
Elbow	9
Foot	9
Hip	8
Temporo-mandibular	7
Cervical spine	7
Big toe	3

Seven of our patients had experienced acute attacks

in the neck or jaw, and three had acute attacks affecting the big toe simulating acute gout. Each attack lasted 1 to 3 days, although they sometimes subsided after a few hours or persisted as long as a week. The interval between attacks was very variable, even in the same patient; they might recur several times a week or only once or twice a year. They usually started in the late afternoon and pain was usually severe enough to prevent sleep. Five patients were admitted to hospital during an acute attack.

Most of these twenty patients have remained in good general health throughout their illness without weight loss; ten have developed the clinical and radiological signs of a low-grade polyarthritis, but only two men now aged 64 and 75 are much disabled by their joint disease after 25 years. Moreover, seventeen out of the twenty patients under review have some clinical evidence of joint damage or tendon lesion, if one includes slight stiffness of the wrists or spindling of an interphalangeal joint; eight patients have tendon lesions in hands or feet, including two with rupture of the thumb extensor; three patients have recurrent olecranon bursitis, and three have developed subcutaneous nodules on elbows or fingers, biopsy of one showing typical histological changes of rheumatoid disease. Detailed clinical information about these twenty patients is given in Table III (overleaf).

Only four patients have had so-called para-articular attacks; in two, tender swellings appeared on the finger tips in response to pressure and persisted for about 24 hours.

An unexpected and previously unreported finding was a family history of palindromic rheumatism and a high incidence of joint disease in the mothers of my patients (Table IV, overleaf).

At least two of these mothers developed palindromic rheumatism at the same time as their sons. One young woman had a mother with "rheumatoid arthritis" and a twin sister and younger brother with "palindromic rheumatism". It has not yet been possible to examine these relatives.

TABLE III
CLINICAL PARTICULARS OF TWENTY PATIENTS

Radiology	Case No.	Sex	Age (yrs)		Duration of Disease (yrs)	Serum RA Factor	Tendon Lesions	Olecranon Bursitis	Sub-cutaneous Nodules	Miscellaneous Findings	Present Condition	
			At Onset	Now								
Normal	2	M	32	58	26	—	—		Transient	Familial web toes Recurrent conjunctivitis	Stiff shoulder	
	5	M	22	36	14	+	+					
	6	M	30	40	10	—	—					
	9	M	42	51	9	—	—					
	10	M	45	50	5	+	+		Osteomyelitis Recurrent conjunctivitis	Swollen ankle		
	11	M	38	42	4	+	—					
	13	F	30	43	13	+	—				Calcified opacities +++ on chest x ray	Slight swelling PIP Slight swelling several PIPs Stiff wrists
	15	F	34	42	8	—	—					
16	F	50	57	7	—	—						
17	F	46	52	6	—	—	Transient					
Arthritic	1	M	27	64	37	+	+	+	+	Pulmonary T.B. Bronchitis Bronchitis (see Fig. 4)	Polyarthritis	
	3	M	51	75	24	+	+	+	+		Polyarthritis	
	4	M	21	37	16	+	+	—	+		Mild polyarthritis	
	7	M	43	51	8	+	+	+	—		Mild polyarthritis	
	8	M	54	60	6	+	+	—	—		Mild polyarthritis	
	12	M	36	41	5	+	+	—	—		Mild polyarthritis	
	14	F	36	47	11	+	—	—	—	Severe measles (see Fig. 3)	Mild polyarthritis	
	18	F	48	53	5	—	+	—	—		Polyarthritis Mild polyarthritis Mild polyarthritis	
	19	F	46	50	4	—	—	—	—			
	20	F	42	44	2	—	—	—	—			

TABLE IV
FAMILY HISTORY OF JOINT DISEASE IN TWENTY PATIENTS WITH PALINDROMIC RHEUMATISM

Disease	Relationship				
	Mother	Father	Brother	Sister	Aunt
Palindromic rheumatism	3		1	1	
Rheumatoid arthritis	3			1	1
Gout	2	1			

Investigations

Although most of these twenty patients had clinical evidence of permanent damage to joint or tendon, only ten had radiological evidence of arthritis; this was almost entirely confined to the small joints of the hands and feet and only three patients had involvement of a knee, elbow, or mid-tarsal joint. Erosions were usually present at the metatarso-phalangeal and metacarpo-phalangeal joints, although they were often minimal, and three patients with an erosive arthritis of the metatarso-phalangeal joints (Fig. 3)

Fig. 3.—X ray of feet of Case 3, a woman aged 36 with 11-year history of palindromic rheumatism. The hand x ray was normal.

PALINDROMIC RHEUMATISM

had normal hand x rays. In a few patients, x rays showed marked destructive changes affecting the interphalangeal joint of the big toe (e.g. Fig. 5,

overleaf) similar to that seen sometimes in psoriatic arthropathy, although none of these patients had psoriasis (Figs 3 to 5).

Fig. 4.—X rays of (a) hands and (b) feet of Case 4, a janitor aged 37 with a 16-year history of palindromic rheumatism. The joint x rays were normal for the first 8 years. Note destructive changes at interphalangeal joint of big toe.

(a)

Fig. 5.—X rays of (a) hands and (b) feet of a typical case of a man aged 60 with a 30-year history of palindromic rheumatism. The joint x rays the first 20 years.

(b)

Fig. 6 (opposite) shows the duration of the disease in December, 1964. In some cases radiological evidence of arthritis was noted within 6 months of

onset, but in others it was delayed for over 10 years (Fig. 7, opposite).

The results of laboratory investigations were frequently normal. Only four patients developed a transient anaemia, haemoglobin values generally remaining within normal limits even after 20 years. White cell counts were normal and no relative lymphocytosis or eosinophilia was seen. The erythrocyte sedimentation rate (ESR) measured by the Westergren method was often normal, even during acute attacks, although the ESR (Wintrobe) at the same time was frequently as high as 40 mm./1 hour. However, a few patients did have an ESR (Westergren) of between 20 and 40 mm./1 hour, and in one case the rate rose to 90 mm./1 hour during an exacerbation of the arthritis. The Wasserman reaction and gonococcal complement-fixation test were negative in all cases.

A sporadic search for L.E.-cells in the blood in twelve patients was unsuccessful. However, eleven out of the twenty patients had positive tests for the serum rheumatoid factor, including four with normal x rays (Fig. 8, overleaf). Three patients with radiological evidence of arthritis had negative tests, but none had had their joint disease for more than 5 years. Tests for the serum anti-nuclear factor were positive in three out of fifteen patients tested.

PALINDROMIC RHEUMATISM

313

Fig. 6.—Duration of disease in twelve men and eight women when assessed in December, 1964, showing severity of rheumatism.

Fig. 7.—Duration of disease in twelve men and eight women before x-ray changes were first apparent.

It may be significant that tests for serum proteins usually gave normal results and only in three patients was there some slight increase in gamma globulins on electrophoresis of the serum proteins.

Three patients had a random serum uric acid between 6.0 and 7.5 mg./100 ml., but subsequent tests failed to confirm hyperuricaemia and these results may have been due to ingestion of aspirin. The serum cholesterol was within normal limits in all cases, and estimations of serum calcium, phosphate, alkaline phosphatase, and blood urea gave normal results although they were not measured routinely. Random estimations of serum non-esterified fatty

acids revealed some abnormally high values (1,500–2,500 micro-equiv./litre), but this was not a constant finding even in the same patient at different times and its significance has still to be evaluated.

Effects of Chrysotherapy

Fifteen patients were given one or more courses of sodium aurothiomalate (myocrisin) by intramuscular injection. The weekly dose was usually 20 mg., but in one case it was only 10 mg. and in four others it was 50 mg. Most patients were given a course totalling 1.0 g. and injections were then stopped.

Fig. 8.—Results of tests for rheumatoid factor in twenty patients, related to presence of x-ray changes and duration of disease.

although recently we have tended to maintain the patient on chrysotherapy indefinitely, giving smaller doses and increasing the interval between injections as the joints have improved.

In three patients it was impossible to assess the value of chrysotherapy, but in twelve the gold injections appeared to stop the acute attacks, often within a few weeks and usually by the time 200 mg. myocri-

sin had been given. However, two patients relapsed when I attempted to increase the interval between injections from 2 to 3 weeks. Moreover five patients relapsed within 2 to 4 years after gold injections were stopped. One patient has so far had a remission lasting 7 years (Fig. 9) although he has recently had some further joint pain, and two others have had remissions lasting 4 years.

Fig. 9.—Effects of chrysotherapy in twelve patients over a period of 7 years.

It should be noted, however, that six patients developed gold dermatitis; one rash was trivial, four lasted several months, and in one patient an exfoliative dermatitis developed requiring several months' in-patient treatment with British anti-Lewisite and prednisone. A further patient, not included in this present series, has recently developed an acute thrombocytopenia after a total dose of only 250 mg. myocrisin.

Discussion

Ten of the twenty patients with palindromic rheumatism studied so far have developed radiological signs of a low-grade erosive polyarthritis and their serological tests suggest a diagnosis of rheumatoid arthritis. Other patients with normal *x* rays have soft tissue lesions compatible with that diagnosis or positive tests for the serum rheumatoid factor.

Although gout has been suspected on many occasions, and has frequently been suggested by the radiologist, there is little evidence to support this diagnosis; no patient has had a persistent hyperuricaemia or developed gouty tophi. On two occasions biopsy of a subcutaneous nodule or tendon sheath to establish the diagnosis has revealed rheumatoid changes, and examination of joint fluid in a few patients for uric acid or calcium crystals has proved negative. No calcification of cartilage or soft tissues has been seen radiologically, even after 25 years, and attacks of joint pain in chondrocalcinosis usually last longer than a week. Fat metabolism may be abnormal in these patients, but the results of investigations so far are inconclusive and inconsistent. It may be of interest that Reed and Sosman (1942) have described the case of a young woman with palindromic rheumatism who was known to have Gaucher's disease. Cold and emotional stress may precipitate acute attacks (Ginsburg, 1948; Zentner, 1953), and some of my patients noted that their attacks were brought on by anxiety.

No patient has yet developed the systemic manifestations of lupus erythematosus, and the one patient with a light-sensitive rash has a family history of sensitivity to sunlight and may have intermittent porphyria, although investigations so far are inconclusive. None of the twenty patients has psoriasis although the destructive changes in the interphalangeal joint of the big toe are suggestive of psoriatic arthropathy.

Palindromic rheumatism may be a syndrome rather than a specific disease, and conditions such as

gout, systemic lupus erythematosus (Piguet, 1957), and chondrocalcinosis must be considered in the differential diagnosis. It is, however, difficult to escape the conclusion that it is frequently an atypical or prodromal form of rheumatoid disease. Ward and Okihira (1959) considered that over a third of their 140 patients seen at the Mayo Clinic had developed rheumatoid arthritis (Table V), although Ward (1965) states that "the diagnosis of palindromic rheumatism is often made without the observance of very strict criteria and this leads to the

TABLE V
FOLLOW UP OF 140 PATIENTS WITH PALINDROMIC RHEUMATISM AT THE MAYO CLINIC (WARD AND OKIHIRO, 1959)

Clinical Status after 5 years						No. of Cases
Complete remission	..	..	..	..	..	11
Continued attacks	..	..	..	..	..	73
Rheumatoid arthritis	..	..	..	..	..	50
Gout	..	..	..	..	..	3
Systemic lupus erythematosus	..	..	..	..	..	3
Total	..	..	..	..	..	140

inclusion of many cases of episodic rheumatoid arthritis; however, even if strict criteria are adhered to, a significant number of patients will develop an illness indistinguishable from so-called rheumatoid arthritis although the prognosis is likely to be better than in a group of patients with classical rheumatoid arthritis".

Certain facts should be remembered concerning palindromic rheumatism. The sex incidence appears to be equal. Patients usually remain well without weight loss or anaemia. Permanent joint damage can occur within a few months of onset, particularly in women, but may be delayed for many years; in these cases, however, careful clinical examination may reveal signs of minor joint damage or soft tissue lesions even though the *x* rays are normal. Some patients never develop significant joint damage in spite of thousands of attacks, and at any time these can cease spontaneously.

Half my patients have a slowly progressive polyarthritis and I believe that the majority of patients with palindromic rheumatism have an episodic form of rheumatoid disease, but this tends to be low grade and to have a relatively good prognosis. This must be taken into account when considering treatment. Nevertheless, patients may be severely disabled by frequent attacks of joint pain and swelling, and the only effective treatment so far appears to be chrysotherapy.

Gold injections appear to suppress the acute

attacks which may cease temporarily after as small a dose as 50 mg. sodium aurothiomalate (myocrisin). However, the patient often relapses within a few years after stopping the injections and may even relapse during the course when the interval between injections is increased. Moreover, there was a high incidence of toxic side-effects in my small group of patients, even on a weekly dose of 20 mg. myocrisin; it may be wise to limit the weekly dose to 10 mg., increasing the interval between injections once there has been a favourable response. Boland and Headley (1948) suggested that chrysotherapy might act as psychotherapy in susceptible subjects and a trial of placebo injections would certainly prove interesting and should be safer! The only other treatment reported to have much effect is steroid therapy, but it is doubtful whether long-term steroid therapy is ever justified in these patients. If attacks are frequent and severe, and gold is contraindicated, a course of ACTH injections might be considered. If attacks are infrequent it may be wisest to treat the patient symptomatically during the attack.

Palindromic rheumatism is not as rare as the literature would suggest. Hench and Rosenberg (1944) estimated the incidence at 1:1000 of patients attending rheumatic clinics, but Rotés Querol and Lience (1959) found the incidence to be 1:500. Certainly the diagnosis is made more frequently once the syndrome is recognized. In the acute attack the patient is often thought to have gout, and once there is clinical or radiological evidence of joint damage the diagnosis is changed to that of rheumatoid arthritis. It is important to consider the diagnosis of palindromic rheumatism when patients complain of episodic joint pain and swelling in view of the prognosis and possible response to chrysotherapy.

Summary

Twenty patients with typical palindromic rheumatism were studied during a 10-year period, 1955-1965. Over half the patients developed signs and symptoms of a low-grade polyarthritis. Chrysotherapy appeared to suppress the acute attacks, but toxic side-effects were common and relapses frequent. Palindromic rheumatism is probably an episodic form of rheumatoid disease, although some patients never develop permanent joint damage, and the prognosis is generally better for these patients than for those with classical rheumatoid arthritis.

I am grateful to Mr. Drury, the medical artist, and to Mr. Turney and his staff in the photographic department,

the Middlesex Hospital, for their help in illustrating this paper. I am indebted to Dr. A. C. Boyle who has allowed me to study patients under his care.

- Ameen, L. (1954). *J. nerv. ment. Dis.*, **120**, 253.
 Ansell, B. M., and Bywaters, E. G. L. (1959). *Ann. rheum. Dis.*, **18**, 331.
 Boland, E. W., and Headley, N. E. (1948). *Ibid.*, **7**, 246.
 Bywaters, E. G. L. (1949). *Ibid.*, **8**, 1.
 Cain, J. C. (1944). *J. Amer. med. Ass.*, **125**, 1037.
 Dames, R., and Zuckner, J. (1961). *Arch. interamer. rheum.*, **4**, 18.
 Ferry, J. L. (1943). *J. Indiana med. Ass.*, **36**, 348.
 Ginsburg, M. (1948). *Ohio St. med. J.*, **44**, 707.
 Grego, J. G., and Harkins, H. N. (1944). *J. Mich. med. Soc.*, **43**, 401.
 Gryboski, J. S. (1948). *Bull. U.S. Army med. Dept.*, **8**, 550.
 Hench, P. S., and Rosenberg, E. F. (1941). *Proc. Mayo Clin.*, **16**, 808.
 ——— (1944). *Arch. intern. Med.*, **73**, 293.
 Hopkins, J. J., and Richmond, J. B. (1947). *Ann. intern. Med.*, **26**, 454.
 Kuhns, J. G. (1945). *Arch. Surg.*, **51**, 289.
 Lewitus, Z. (1954). *Rheumatism*, **10**, 70.
 Mazer, M. (1942). *J. Amer. med. Ass.*, **120**, 364.
 McEwen, C. (1960). In "Arthritis and Allied Conditions", 6th ed., ed. J. L. Hollander, p. 246. Lea and Febiger, Philadelphia.
 Neligan, A. R. (1946). *Brit. med. J.*, **1**, 205.
 Paul, W. D., and Carr, T. (1945). *Arch. phys. Med.*, **26**, 687.
 ——— and Logan, W. P. (1944). *J. Iowa St. med. Ass.*, **34**, 101.
 Perl, A. F. (1947). *Canad. med. Ass. J.*, **57**, 382.
 Piguet, B. (1957). *Rev. Rhum.*, **24**, 303.
 Reed, J., and Sosman, M. C. (1942). *Radiology*, **38**, 579.
 Rotés Querol, J. (1956). *Rev. esp. Reum.*, **6**, 345.
 ——— and Lience, E. (1959). *Arch. interamer. Rheum.*, **2**, 491.
 Ropes, M., and Bauer, W. (1945). *New Engl. J. Med.*, **233**, 592.
 Salomon, M. I. (1946). *N. Y. St. med. J.*, **46**, 622.
 Scheinberg, D. (1947). *J. Tennessee med. Ass.*, **40**, 260.
 Thompson, J. L. (1942). *Med. Ann. D.C.*, **11**, 189.
 Ward, L. E. (1965). Personal communication.
 ——— and Okihira, M. M. (1959). Quoted by R. W. Lamont-Havers (1960). In "Arthritis and Allied Conditions", ed. J. L. Hollander, p. 708. Lea and Febiger, Philadelphia.
 Wassmann, K. (1950). *Acta med. scand.*, **139**, 55.
 Weber, F. Parkes (1946). *Lancet*, **2**, 931.
 Wingfield, A. (1945). *Brit. med. J.*, **2**, 157.
 Wirtschafter, Z. T., Williams, D. W., and Gaulden, E. C. (1955). *Acta med. scand.*, **153**, 119.
 Wolfson, S. A., and Alter, M. S. (1948). *Ann. rheum. Dis.*, **7**, 159.
 Zentner, A. S. (1953). *Bull. Menninger Clin.*, **17**, 64.

Rhumatisme palindromique**RÉSUMÉ**

On étudia vingt malades atteints de rhumatisme palindromique typique pendant une période de 10 ans, 1955-1965. Plus de la moitié de ces malades développèrent des signes et des symptômes de polyarthrite bénigne. La chrysothérapie semblait supprimer les attaques aiguës, mais les effets secondaires toxiques étaient communs et les rechutes fréquentes. Le rhumatisme palindromique est probablement une forme épisodique de la maladie rhumatismale, bien que certains malades ne développent jamais de lésions articulaires permanentes, et le pronostic est généralement meilleur pour ces malades que pour ceux atteints d'arthrite rhumatismale classique.

Reumatismo palindrómico**SUMARIO**

Se estudiaron veinte enfermos con reumatismo palindrómico típico durante un período de 10 años, 1955-65. Más de la mitad de estos enfermos desarrollaron signos y síntomas de poliartritis benigna. La crisoterapia parecía suprimir los ataques agudos, pero efectos secundarios tóxicos fueron comunes y recaídas frecuentes. El reumatismo palindrómico es probablemente una forma episódica de la enfermedad reumatoide, aunque algunos enfermos nunca desarrollan lesiones articulares permanentes y el pronóstico es generalmente mejor para estos enfermos que para los con artritis reumatoide clásica.