the patient with an underlying bronchial carcinoma, and, we would suggest, was probably a reflection of the underlying condition.

The impressive increase of the γ-globulins in these patients sets several problems. Is this increase the cause or the direct or indirect consequence of the polymyositis? Is it that the increased γ-globulin reflects a reaction of the organism against a pathogenic agent, or are there autoantibodies?

It would be of interest to know if any immunological reaction could be elicited between this abnormally increased γ-globulin and products prepared from fresh surgical material such as an amputation. This, together with more extended studies on this subject, we propose to pursue.

Summary

The essential and striking electrophoretic changes in six patients with proved polymyositis were a markedly elevated γ-globulin fraction followed by a decreased albumin; such a pattern, while not of course specific for this condition, would, we feel, be highly suggestive of polymyositis in a patient with muscle disease.

The α_2-globulin fraction was raised only in the one patient who had both dermatomyositis and an underlying bronchial neoplasm. In the other two patients with skin involvement it remained at the upper limit of normal.

The E.S.R. was strikingly raised only in the patient with the underlying neoplasm—in the remaining two cases with skin involvement it was normal in the one and slightly raised in the other. In the remaining three patients with polymyositis alone it was consistently normal with a single isolated exception, when the fall was only 18 mm. in one hour (Westergren).

The glycoproteins were estimated in two patients, revealing an increased γ-glycoprotein fraction in the one with polymyositis alone and showing a tissue-destruction pattern in the patient with an underlying bronchial neoplasm.

We would like to thank Dr. W. H. McMenemey for his advice and encouragement and the various members of the consultant staff of Maida Vale Hospital for allowing us access to their patients. We are also grateful to Dr. Rowland Hill for permission to include a patient under his care at the West End Hospital. We are further indebted to Dr. McMenemey and Dr. H. C. Grant for the pathological reports and to Dr. H. R. Townsend for the E.M.G. studies.

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"Presiding at a prize-giving ceremony [on October 24] at the Middlesex Hospital School of Nursing, Lord Astor of Hever expressed regret that he had to give up the chairmanship of the hospital because he is to live in the south of France. The matron, Miss Marjorie J. Marriott, expressed appreciation of his work for the hospital. He had been a member of the board for 40 years and chairman since 1938. His weekly visits to the hospital had been greatly appreciated by patients and staff. When he was appointed chairman there were 380 nurses, compared with 890 at present, and the hospital had 450 beds, compared with nearly 1,000 now." (*The Times*, October 25.)

RHEUMATOID PERICARDITIS

A REPORT OF FOUR CASES

BY

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As in other collagen diseases, an increasing incidence and variety of systemic lesions are being recognized in rheumatoid arthritis. These include cardiac lesions, which have been shown to be frequent in most necropsy studies (Baggenstoss and Rosenberg, 1941; Fingerman and Andrus, 1943; Young and Schwedel, 1944; Bauer and Clark, 1948). By contrast, clinical studies have demonstrated heart lesions rather infrequently during life (see review by Rogen, 1947).

After excluding conditions common in elderly subjects, such as ischaemic and hypertensive heart disease, the following cardiac lesions have been reported from post-mortem studies on rheumatoid arthritic subjects.

1. Macroscopic sclerosis of valve cusps and chordae tendineae with microscopical inflammatory and degenerative changes in valve cusps and myocardium. These are similar to the lesions of rheumatic heart disease and have been found in from 6% (Bennett, 1943) to 66% (Young and Schwedel, 1944) of necropsies on rheumatoid subjects. The clinical recognition of rheumatic heart disease is very much less frequent even in patients subsequently found to have cardiac lesions at necropsy (Baggenstoss and Rosenberg, 1941, Young and Schwedel, 1944). Whether these lesions represent an undue disposition to rheumatic heart disease among rheumatoid subjects or whether they are part of the rheumatoid disease process remains uncertain.

2. Rheumatoid granulomata, histologically similar to the subcutaneous nodules of rheumatoid arthritis, have been reported in the pericardium, myocardium, valve cusps, and aorta. Such lesions are infrequent and probably affect only 1 to 3% of patients (Sokoloff, 1953).

3. Rheumatoid aortitis with aortic-valve incompetence is more commonly seen with ankylosing spondylitis, but 2 of the 22 cases reviewed by Clark *et al.* (1957) and one described by Hope-Ross *et al.* (1960) suffered from peripheral arthritis only.

4. Pericarditis or adhesions have been reported in from 11% (Fingerman and Andrus, 1943) to 50% (Young and Schwedel, 1944) of patients dying with rheumatoid arthritis. Here again clinical recognition lags far behind the pathological incidence.

The following four cases of rheumatoid pericarditis represent the only cases diagnosed in a series of 197 rheumatoid patients admitted to the Charing Cross and Royal Northern Hospitals between January, 1956, and March, 1962. Two of the four patients were seen personally and the other two are described from their case records.

Case 1

The patient, a man aged 48, was admitted to hospital in February, 1962. In 1956, after a period of kneeling to lay a concrete path, he developed painful swollen knees, followed by pain and stiffness of other joints. His arthritis was controlled with prednisolone, chloroquine, and salicylates, but for six months prior to admission he had received no corticosteroids. Three days before admission he wakened with moderately severe anterior chest pain and dyspnoea, but continued his work in spite of discomfort until an exacerbation on the morning of admission. There was no exacerbation of his arthritis. For several years he had suffered from mild bronchitis.

Examination revealed a mild fever (100° F.; 37.8° C.), a regular tachycardia (100/min.), but no other abnormal signs in the cardiovascular system. Blood-pressure 115/85. Rhonchi were heard over both lungs. There were subcutaneous nodules in the olecranon regions with ulnar deviation of the fingers and swollen right fourth proximal interphalangeal and left wrist-joints.

Investigations.—Hb 13.2 g./100 ml.; leucocytes 11.000/c.mm.; sedimentation rate, 97 mm. in the first hour (Westergren); total serum proteins, 7.5 g./100 ml.; electrophoresis showed excess of gammaglobulin. Antistreptolysin O titre, 100 Todd units. Rose–Waaler test positive (1/128). L.E. cell tests negative. Coombs test negative. Serum transaminase levels normal. Serial cardiograms showed the changing pattern of pericarditis with initial ST elevation progressing to inversion of the T waves in all leads (Fig. 1). Radiography showed some cardiac enlargement and erosions of proximal interphalangeal and metacarpophalangeal joints.

Progress.—Three days after admission a loud pericardial friction rub was heard, and this persisted for 10 days, at which time a recurrence of pain and fever was terminated with prednisolone.

Case 2

A man of 49 was first admitted to hospital in November, 1961. One year previously he suffered a transient attack of painful swollen metacarpophalangeal joints. One month before admission this trouble recurred and he also developed a pleuritic pain over the left anterior chest. Examination was negative except for swelling of the small hand-joints, with impaired percussion note and poor air entry over the lower lobe of the left lung. Blood-pressure 120/80. At this time a chest *x*-ray examination showed collapse of the left lower lobe, but the heart appeared normal. After a course of penicillin and breathing exercises he was discharged, though the chest pain persisted. In January, 1962, a further chest *x*-ray film, taken because of his persistent pain, showed re-expansion of the left lung, but a high right diaphragm with shadowing in the right lower zone suggested partial collapse of the right lower lobe. The cardiac shadow was now unequivocally larger (Fig. 2).

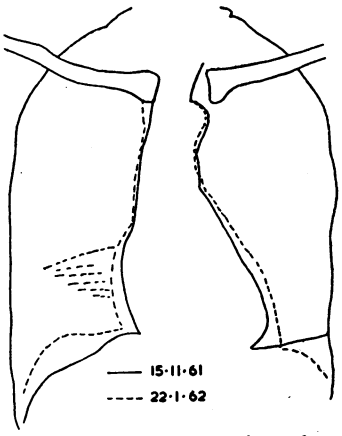

FIG. 2.—Case 2. Tracings from radiographs to show change in heart size.

The patient was readmitted, but physical examination was unchanged and a pericardial rub was not heard at any time.

Investigations. — Hb 12.6 g./100 ml.; leucocytes 3,600/c.mm.; sedimentation rate 97 mm. in first hour (Westergren); blood urea 27 mg./100 ml., total serum proteins 7.2 g./100 ml.; electrophoretic pattern showed excess of gammaglobulin. Rose–Waaler test positive (1/64), L.E. cell tests negative; Coombs test negative; Wassermann reaction negative. Serial cardiograms showed initially low-voltage T waves progressing to inversion in all leads (Fig. 3). Serial chest *x*-ray films showed enlargement of the heart shadow reverting to normal size.

Progress.—Because of persistent chest pain the patient was given prednisolone and rapidly became symptom-free.

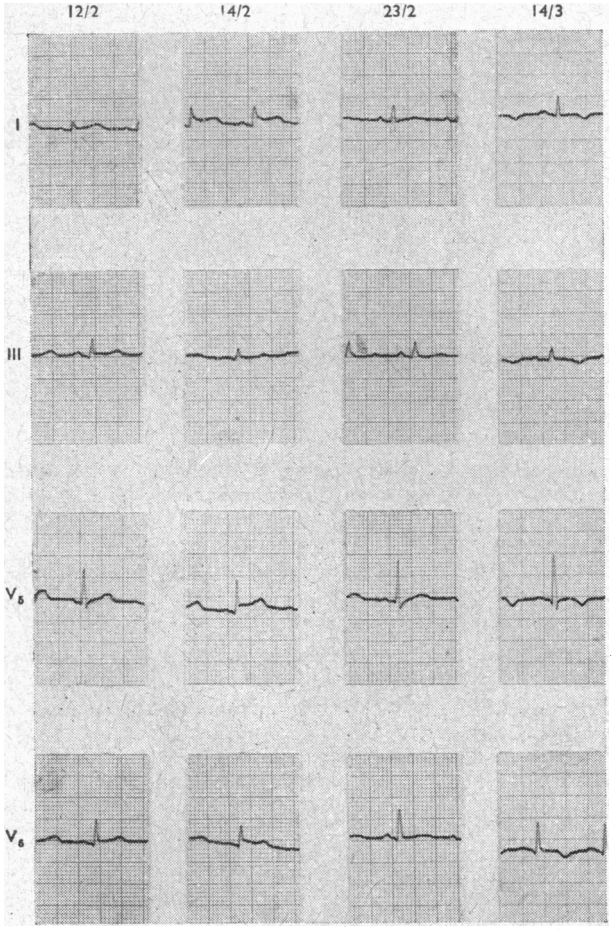

FIG. 1.—Case 1. Serial cardiograms showing initial ST elevation progressing to T-wave inversion four weeks later.

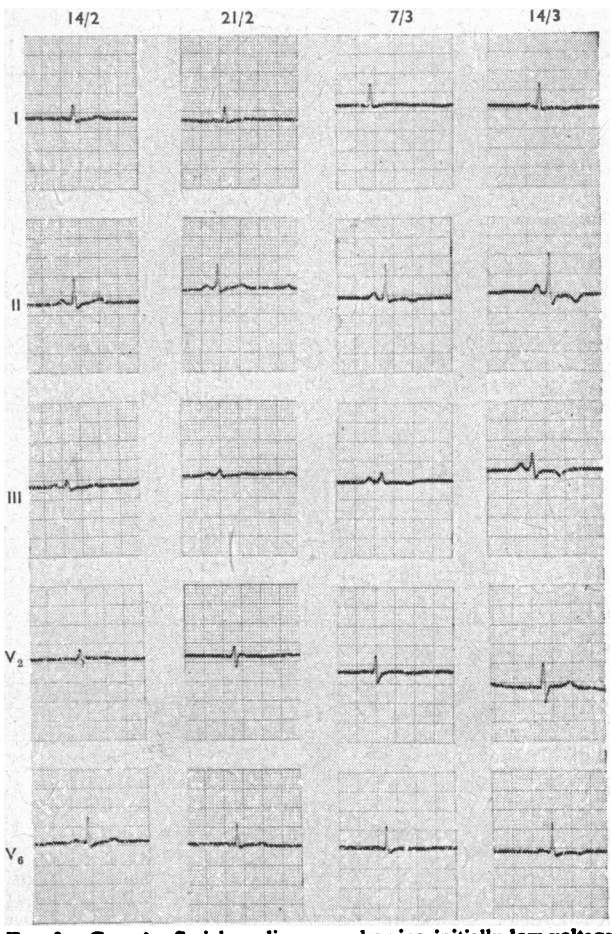

FIG. 3.—Case 2 Serial cardiograms showing initially low-voltage T waves becoming inverted four weeks later.

Case 3

A man of 45 was admitted to hospital in July, 1958. In 1946 he developed arthritis in several joints, and over the subsequent years was treated with gold, salicylates, and one short course of A.C.T.H. In 1955 a transient episode of chest pain led to cardiographic examination, which was normal.

During the year before admission he experienced three unprovoked attacks of dyspnoea lasting up to 12 hours and for three months his joints had been more painful.

Physical examination revealed no fever. There was a regular tachycardia of 100/minute, and a loud pericardial friction rub was audible for the first four days after admission. B.P. 125/75. There was ulnar deviation of the fingers, subcutaneous nodules at the elbows, small effusions into the knee-joints, and tender metacarpophalangeal joints.

Investigations.—Hb 10.8 g./100 ml.; leucocytes 11,000/c.mm.; sedimentation rate 28 mm. in first hour (Westergren). Rose–Waaler test strongly positive, L.E. cell test negative. Chest *x*-ray examination showed slight cardiac enlargement. Cardiogram showed inverted T waves in leads II, III, and V7, with low-voltage T waves in all other leads.

Progress.—The patient was treated with dexamethasone, and in 1962 had no cardiac symptoms though his arthritis remained troublesome. At this time a cardiogram was normal and radiography showed a normal heart size.

Case 4

A woman of 37 was admitted to hospital in February, 1960, with a history of Raynaud's phenomenon for two months and aching pain in the right posterior chest for six weeks. For two weeks she had noticed painless swelling of the hands and knees with some ankle stiffness.

Physical examination revealed an ill-looking woman with occasional fever up to 99° F. (37.2° C.). No abnormal signs were found in the cardiovascular system. B.P. 110/70. Both wrists were swollen, movement at the right elbow was painful, and there were effusions into both knee-joints.

Investigations.—Hb 12.4 g./100 ml.; leucocytes 12.000/c.mm.; sedimentation rate 61 mm. in the first hour (Westergren). Antistreptolysin O titre, 180 Todd units; Wassermann reaction negative; Rose–Waaler test negative; L.E. cell test negative on five occasions. Chest and joint *x*-ray films showed nothing abnormal. Cardiogram on admission normal, but two weeks later the T wave was biphasic in lead I and inverted in all other leads.

Progress.—With rest and analgesics the arthritis subsided sufficiently for her to be discharged after two months. Over the next two years she slowly improved, and when last seen in 1962 she was symptom-free and had a normal sedimentation rate.

Discussion

The association of pericarditis with arthritis is usually believed to suggest a diagnosis of systemic lupus erythematosus, and in each of the above cases this was considered and, so far as was possible, excluded. The sex of three of the patients and the presence of subcutaneous nodules in two weigh heavily against this diagnosis, quite apart from the negative L.E. cell tests. The fourth patient had arthritis with repeatedly negative L.E. cell tests, and while this makes the diagnosis of systemic lupus erythematosus unlikely it does not exclude it. Only further observation may clarify whether the diagnosis of probable rheumatoid arthritis is correct in this case.

However, there is no strong reason why the presence of pericarditis should suggest systemic lupus erythematosus rather than rheumatoid arthritis, for post-mortem studies have shown pericarditis in from 47 to 80% of cases of systemic lupus erythematosus (Griffin and Vural, 1951; Dubois, 1953; Jessar *et al.*, 1953), compared with the still considerable proportion of 40 to 50% of rheumatoid arthritic patients reported by most authors (for example, Young and Schwedel, 1944; Bauer and Clark, 1948). Certainly the pericarditis of systemic lupus erythematosus is more frequently recognized during life, for Jessar *et al.* (1953) diagnosed it in 23%, Dubois (1953) in 43.5%, and Harvey *et al.* (1954) in 48% of patients. By contrast, the reports of pericarditis complicating rheumatoid arthritis are mostly single case reports, though Goslings (1958) and Tarpley (1961) have presented several cases. This seems curious, for in most other respects the pericarditis of systemic lupus erythematosus and that of rheumatoid arthritis are very similar. Both may vary from a frankly painful illness to a clinically silent condition (Harvey *et al.*, 1954; Goslings, 1958), and the few pathological studies which fortunately included the active stages do not indicate that the pericarditis of rheumatoid arthritis is any less acute than that of systemic lupus erythematosus (Young and Schwedel, 1944; Bywaters, 1950). Likewise the clinical and cardiographic changes in both diseases suggest relatively short episodes of acute pericarditis though the reversion to a normal cardiographic pattern takes several months.

To some extent the discrepancy between the number of cases of rheumatoid pericarditis diagnosed in life and at necropsy may be due to a failure of recognition on the part of both patients and physicians. With the background of chronic pain seen in rheumatoid arthritis any further pain due to pericarditis might not provoke further complaint from a patient conditioned to accept pain, or serious consideration by a physician engrossed with joint manifestations. Certainly it is difficult to believe that the pericarditis of rheumatoid arthritis is so very much less painful than that of systemic lupus erythematosus.

Other factors making the diagnosis more difficult are no doubt the frequency with which rheumatoid arthritic patients have their pain sense dulled by analgesics and their inflammatory mechanisms suppressed by steroid therapy. Even when the diagnosis is suspected, confirmation may be difficult, for in the absence of a pericardial rub it is easy to dismiss as insignificant minor cardiographic signs such as low T waves seen on a single tracing. In such a case serial cardiograms with changing T waves or serial radiographs showing fluctuations in the heart size will usually confirm the diagnosis. Fortunately for the patient, rheumatoid pericarditis does not seem to be harmful, so failure to diagnose it is probably not important.

It is of interest that two of the above patients and three of the six cases reported by Tarpley (1961) showed coincidental pleurisy. Also, the sex incidence (three of the present series and four of Tarpley's were males) shows the same male preponderance as has been reported by Horler and Thompson (1959) and Ward (1961) in cases of pleurisy complicating rheumatoid arthritis.

Summary

Four cases of pericarditis complicating rheumatoid arthritis are described. It is suggested that this combination is not uncommon but usually remains undiagnosed. This is largely because the pericardial pain, which may be trivial, is overshadowed by the joint pains and tends to be disregarded by patients and physicians.

I am grateful to Drs. J. F. Buchan, W. E. Clarke, R. Greene, and N. S. Plummer, under whom these patients were admitted, for permission to publish the case summaries.

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WHIPWORMS IN A CAMBRIDGESHIRE VILLAGE

BY

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The human whipworm, *Trichuris trichiura*, is the third most common helminthic infestation, only the roundworm and hookworm surpassing it in world frequency (Watson, 1960). In Western Europe it is generally regarded as an interesting but unimportant parasitic inhabitant of the bowel in a small percentage of the population, and most doctors are not aware of its existence as the cause of a disease entity. Only one previous case of the whipworm having caused symptoms has been reported in this country (Ross, 1942). The present paper concerns two cases of severe infestation with *T. trichiura*.

Case 1

In September, 1961, a 2½-year-old girl was brought to the surgery with an eight-months history of passing four or five loose stools daily. In the past few weeks she had become listless, anorexic, and somnolent. She often complained of abdominal pains, and, despite the looseness of her motions, had to strain hard to pass them. Physical examination revealed a very pale, chronically ill little girl who was both apathetic and irritable. Her skin was dry, but she was not emaciated, weighing 26 lb. (11.8 kg.). The liver and spleen were not palpable, there was no abdominal tenderness, and digital examination of the rectum was negative. The stools were watery and very offensive, and contained some mucus and blood. Microscopy revealed ova of *T. trichiura* ++++. No *Salmonella* or *Shigella* organisms were found. A blood count showed: haemoglobin 5.7 g./100 ml.; packed cell volume 25%; M.C.H.C. 22.8%; M.C.D. 7.3 μ; white cells 16,000/c.mm., with 17% eosinophils.

Treatment was started with "imferon" 2 ml. intramuscularly twice weekly for two weeks and dithiazanine iodide 25 mg. four times a day for five days. The child began to improve very rapidly both in her general condition and in her diarrhoea. Her motions became less frequent but were still loose and offensive. Examination of them after treatment revealed that *T. trichiura* ova were still present in very large numbers. She was given a second course of dithiazanine iodide, the dose being raised to 100 mg. four times a day. After this treatment no ova were seen in the stools. A repeat blood examination showed a haemoglobin of 11.8 g./100 ml. and a leucocytosis of 14,000 with an eosinophilia of 25%.

On November 9 the child was seen again, having passed 100 ml. of dark red blood per rectum. Digital examination of the rectum was negative, but examination of the stools showed that she was still passing *T. trichiura* ova in large numbers. She was given a third course of dithiazanine iodide, 200 mg. three times a day, which failed to eradicate the infestation.

On December 6 sigmoidoscopy was performed on her as an out-patient under general anaesthesia. Mr. R. E. B. Tagart, who performed the sigmoidoscopy, reported as follows: "The bowel contained large amounts of liquid faeces. The instrument was passed to 20 cm. from the anus. The bowel wall was reddened, oedematous with complete loss of vascular pattern, and bled on very light contact. A number of whipworms were seen moving sluggishly and were removed with forceps. They were found to be attached to the bowel wall by an anterior filiform process. Through the sigmoidoscope, they can be distinguished from threadworms, which are commonly seen in the sigmoid colon and rectum of children, by the more uniform calibre of the body of the whipworm. The threadworm tapers gradually to a point."

It was then decided to try a course of hexylresorcinol enemata by the method described by Basnuevo (1956). In this a stock powder is made up to the following formula:

Hexylresorcinol	1 g.
Colloidal aluminium hydroxide ..	12 g.
Colloidal kaolin	12 g.
Gum acacia	to 50 g.

A fresh colloidal solution is made up in a mortar by adding 100 ml. of water to each 15 g. of powder, giving a dilution of hexylresorcinol of 1:300. The recommended enema volume for infants and young children is 12 ml./lb. (26.4 ml./kg.) body weight, and for older children is 100 ml. per year of age. It is important to clear the bowel with a saline enema both the night preceding and again two hours before treatment. The emulsion of hexylresorcinol is allowed to flow slowly through a catheter introduced 15 cm. into the rectum. After half the volume has been run in the patient is turned on to the right side to encourage the flow of emulsion to the caecum. Basnuevo recommends a course of three enemata 5 and 10 to 15 days apart respectively.

On December 21 the first enema was administered in the patient's home after the district nurse had given the two saline bowel wash-outs. No difficulty was experienced and the buttocks were well smeared with petroleum jelly to prevent skin burning when the enema was expelled. Stools were collected for three days following treatment and carefully filtered through black nylon mesh. Many hundreds of whole and partly disintegrated *T. trichiura* were collected together with threadworms (*Enterobius vermicularis*). After the second hexylresorcinol enema the child passed fewer worms, although the mother reported seeing "clumps of whipworms in the stools" after the third day of collection. These, unfortunately, were not kept. No worms were found in the stools after the third enema. Stools examined two months later showed that there were very few ova still being passed. Clinically there was an impressive and gratifying improvement in the child. She was bright, cheerful, and full of energy. The stools were well formed and were passed once a day.