Scoliosis and Trendelenburg sign in a painting by P P Rubens

In an article on Rubens' painting "The Three Graces," Dequeker suggests that hypermobility is a medical explanation of the seeming scoliosis and Trendelenburg sign in the middle figure. 3 But the posture of this middle figure should probably be interpreted as an artistic phenomenon without medical reference.

Sculptors in classical Greek and Roman periods often used the contrapposto posture. In this, by putting most weight on one leg, the other leg can be shown in a relaxed and semi-flexed position. This undulating between tension and relaxation will animate the figure. A person with normal muscular function and a normal back can perfectly well pose in this way with relaxed hip abductors on the weightbearing side, a descending hip on the opposite side, and a compensating scoliotic posture. This posture is facilitated by support from the arm as in Rubens' painting. If the person tries to take a forward step, relaxation of the muscles of the weightbearing hip can no longer be maintained, and the positive Trendelenburg sign will disappear.

In the Renaissance period the use of this contrapposto posture was revived. During his stay in Rome Rubens eagerly studied the then recently excavated Laokoon sculpture with its three distorted figures. 3 He often used such distorted postures in his paintings to give the impression of vigorous muscular characters capable of performing great tasks. The best example is probably "The Debarkation at Marseilles" in the Maria de Medici cycle from 1622 to 1625 for the Luxembourg Palace in Paris. 1 Here, three young women, nereides, with curved muscular backs at the bottom of the picture nearly seem to carry the ship of Maria de Medici.

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References

Author's response
Dr Hansen's remarks about our recent article in the Annals are pertinent and have to be taken as an alternative explanation for the observed functional scoliosis and positive Trendelenburg sign. I am grateful for this artistic-historical information. However, this does not exclude the possible diagnosis of benign familial hypermobility syndrome. In several other paintings by Rubens, where the three sisters (sisters) of the graces are represented, clinical signs of hypermobility can be seen. In the painting "The Judgement of Paris" (London National Gallery) a positive Trendelenburg sign and scoliosis can be seen in the two blond sisters who are now in a walking position without support. In one of them the right wrist is in 90° hyperflexion. In the painting "Sine Cerere et Baccho friget Venus" (Brussels Koninklijke Musea voor Schone Kunsten), subluxation of the left wrist is seen in the dark blonde sister and hyperextension of the distal interphalangeal (DIP) joint of the fourth finger in another sister with brown hair. Hyperextension of a DIP and metacarpophalangeal finger joint and hyperflexion of a wrist joint is also seen in the brown haired sister of the painting "The Monna and Saints." (Antwerp, Sint-Jacobskerk).

1, as well as Sven Hansen, am fully aware that errors of diagnosis are commonly made either by seeing where none exists or by interpreting at face value a pathological appearance that is only the expression of an artistic convention. The observations made in P P Rubens' painting, representing the sitters for "the graces" painting who are Rubens' second wife Helen Froment and her younger sisters, are very suggestive of the diagnosis of benign familial hypermobility syndrome and not a purely artistic phenomenon.

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Comparison of WOMAC with SF-36 for OA of the knee or hip

Angst et al compared WOMAC with the SF-36 as tools to assess the outcome of a three to four week inpatient rehabilitation programme for people with osteoarthritis of the knee or hip. They concluded that both instruments capture improvement in pain levels, but functional improvement can be better detected by WOMAC. We have reservations about the use of SF-36 in this context.

We too provide residual musculoskeletal rehabilitation of usually three weeks duration and have been searching for a suitable instrument to assess quality of life at the time of discharge from our programme. We have rejected the SF-36 for the following reasons. A large majority of the questions in the SF-36 relate to the subject's experience over the past four weeks. The condition of most of our patients improves considerably over the three weeks of treatment. It is therefore not appropriate to ask how they have been over the previous four weeks. We note that the period of treatment in the report by Angst et al varies from three to four weeks.

It is not only the length of time which makes the use of the SF-36 inappropriate in this setting, many of the questions assume the subject is living an everyday life. For example, inquiry is made about "both work outside the home and housework," “other activities at home”, and “normal social activities with family, friends, neighbours, or groups.” Obviously if a person is devoting time and energy to an inpatient musculoskeletal rehabilitation programme they are in no position to be truly engaged in any of these work or social activities.

The while the outcomes of our similar residential rehabilitation programme for people with osteoarthritis are in accordance with those of Angst et al, we do not feel it is appropriate to use the SF-36 to measure improvement at discharge. It is of course quite reasonable to use it before admission and at three or six months’ follow up.

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Authors' response
In their letter commenting on our article, 1 Jones and Leighton deal with two major problems which might arise in the application of the SF-36 to inpatients. We would like to stimulate discussion about this issue by our following response.

The first problem concerns the fact that many of the SF-36 items ask about subjective health status over the past four weeks at the time of administration of the questionnaire. Jones and Leighton suggest, therefore, that the results at the end of an inpatient rehabilitation (three or four weeks) reflect some kind of an average of the health status during that rehabilitation period in which most of the patients have improved considerably. We agree that this assessment is unlikely to show the maximum of improvement that may be expected at the day of discharge from the clinic or shortly thereafter. However, one can assume that the result overestimates the health status for the time periods close to the day of administration of the questionnaire (for example, at the day of discharge) owing to the fact that the response is based on the patient's memory. The same problem, but in the opposite direction, would arise if we administered the SF-36 two or four weeks after the day of discharge. Thus we possibly miss the maximal effect, which may last only a few days, but we do obtain an assessment of a certain time period, which is likely to be more valid and more clinically important than that of a single day. To take account of this point, we also reported results.
of the three month follow up (that is, two months after discharge) in our study in order to reflect the course of the effects and whether the different responsiveness of the SF-36 compared with the WOMAC remained consistent. In addition, we will publish further results of three-month evaluations up to the two-year follow up of our patients during the next year.

The second issue deals with the fact that some items ask about activities of daily living and social participation which are not demanded or hardly possible to perform during a stay in the clinic. These are mainly the items contained in questions 4 (a–d) and five (a–c) comprising the role physical and role emotional scales. For this reason, we reported these two scales as part of the SF-36 for the sake of completeness, but we did not include them in the analysis of the comparison of WOMAC and the SF-36. Nevertheless, item 8, which is the bodily pain scale, is also affected by this problem. Müller et al dealt with this issue recently.1 The authors created a modified SF-36m, which was adapted in items 4, 5, and 8 to the situation of a clinic stay. They concluded that bodily pain and role emotional did not show significantly different effects from those obtained by the original SF-36, but that the role physical scale was slightly more restricted by the SF-36m.

We used the SF-36 for three reasons. Firstly, the SF-36 assesses health status comprehensively—that is, not only pain and disease-specific scales as physical function, etc but also psychometric dimensions and dimensions of social participation. As a result, it gives an overall assessment of the patient’s health status which is compatible with the WHO’s new ICF or the future ICF concept defining health.2 Secondly, the SF-36 can also be administered to “healthy” people and to patients with different diseases, which allows a comparison of the results with those for other patient groups and the general population. Thirdly, the SF-36 is one of the best tested, best known, and most widely used health measure all over the world.

Is pamidronate effective for acute rheumatic pain?

Parenteral pamidronate is licenced in the United Kingdom for the management of Paget’s disease, tumour related hypercalcaemia, and metastatic bone pain, where it can rapidly relieve symptoms.1 It is also widely used for the prevention and treatment of osteoporosis, although this represents unlicensed use of the drug, and there is some evidence that it can be rapidly effective for pain relief in patients with osteoporotic vertebral fractures.4,5 It has been used with some effect for the management of anklyosing spondylitis,6 but the full extent of any ankylosis properties of the drug has not been fully explored. These properties became apparent to us quite by chance in the three cases described here.

Case reports

Patient A

A 25 year old female nurse with known anklyosing spondylitis was admitted to hospital with worsening back pain and right buttock pain uncontrolled by regular opiate analgesia and a variety of potent non-steroidal anti-inflammatory drugs. Parenteral methylprednisolone was prescribed, followed by pamidronate 30 mg for “bone protection”. In the event, pamidronate was given but not methylprednisolone, deferred owing to unexplained pyrexia. Shortly after receiving her pamidronate, her intractable pain was so greatly improved that methylprednisolone was declined and she was discharged three days later. The improvement seen has been sustained for over six months. The unexpected analgesic effect of pamidronate in this case led to its use in two subsequent cases.

Patient B

A 38 year old housewife with chronic low back pain was admitted with a short history of acute back pain and a modestly raised C reactive protein (14 mg/l). Isotope bone scan showed increased uptake in the fifth lumbar intervertebral disc. Magnetic resonance imaging identified abnormal signal from this disc suggestive of discitis. An infective cause was suggested by fibrosis, microvascular occlusion, and proliferation of the vascular intima. The reported prevalence of antiphospholipid antibodies (aCL) in SSc varies from 0 to 25%,9 and reports of clinical associations have been variable.9,10 To our knowledge, only one study has examined antibodies to β2 glycoprotein I (aβ2 GPI) in SSc and shown a correlation with pulmonary hypertension and raised mean pulmonary artery pressure.11 In our study we examined the frequency of aβ2 GPI and aCL in SSc and Raynaud’s phenomenon (RP).

Twenty six patients with SSc (16 diffuse, 10 limited) were included in this retrospective study. Informed consent was obtained. All 16 patients with diffuse SSc and one patient with limited SSc patients met American Rheumatism Association criteria for SSc and Raynaud’s phenomenon (RP).

Systemic sclerosis (SSc) is a multisystem disease in which organ dysfunction is influenced by fibrosis, microvascular occlusion, and proliferation of the vascular intima. The reported prevalence of antiphospholipid antibodies (aCL) in SSc varies from 0 to 25%,9 and reports of clinical associations have been variable.9,10 To our knowledge, only one study has examined antibodies to β2 glycoprotein I (aβ2 GPI) in SSc and shown a correlation with pulmonary hypertension and raised mean pulmonary artery pressure.11 In our study we examined the frequency of aβ2 GPI and aCL in SSc and Raynaud’s phenomenon (RP).

References


Antibodies to β2 glycoprotein I and cardiolipin in SSc

Although the disease is multisystem in nature, in which organ dysfunction is influenced by fibrosis, microvascular occlusion, and proliferation of the vascular intima. The reported prevalence of antiphospholipid antibodies (aCL) in SSc varies from 0 to 25%,9 and reports of clinical associations have been variable.9,10 To our knowledge, only one study has examined antibodies to β2 glycoprotein I (aβ2 GPI) in SSc and shown a correlation with pulmonary hypertension and raised mean pulmonary artery pressure.11 In our study we examined the frequency of aβ2 GPI and aCL in SSc and Raynaud’s phenomenon (RP).

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αβ2-GPI and aCL were measured by enzyme-linked immunosorbent assay (ELISA; INOVA Diagnostics, Inc, San Diego, CA and Hemagen Diagnostics, Inc Waltham, MA, respectively). Commercially obtained HEP-2 slides (Immuno Concepts, Sacramento, CA) were used for indirect immunofluorescence (IIF). Samples were tested for antibodies to topoisomerase 1 (Scl-70), U1 ribonucleoprotein (U1-RNP), and Sjögren’s syndrome antigens A and B (SS-A/SS-B) by double immunodiffusion.

Student’s t test (two tailed) was used for comparison of means, and Fisher’s exact test (two tailed) for analysis of frequencies. Age distributions were compared with the Mann–Whitney test because healthy controls described their age in decades, not years.

Table 1 summarises the demographics and laboratory data for the study group. The patients with SSc were significantly older than both the healthy controls (p<0.005) and the patients with RP (p=0.02). All mean laboratory values were within the normal range. Figure 1 compares the values for tests among the study groups except αβ2-GPI IgG, where all tests were negative. IgM αβ2-GPI were found in two patients with SSc (8%), one patient with RP (4%), and none of the healthy controls (p>0.05). Three (12%) patients with SSc, five (22%) with RP, and one (5%) of the healthy controls had positive tests for IgG or IgM antinuclear antibodies (p>0.05). The sera positive for aCL were not the same as those positive for αβ2-GPI.

The two patients with SSc positive for αβ2-GPI had mean disease duration of 19 months; both had cutaneous manifestations and one had hypoxia with decreased carbon monoxide transfer factor (TLC0). The three patients with SSc and aCL had mean disease duration of 112 months. One had hypoxia (with normal TLC0 and non-restrictive pulmonary function tests), one had restrictive lung disease and digital ulcers, and one had oesophageal hypomotility. None of the study participants had thrombo-cytopenia or a history of deep venous thrombosis. Twenty two per cent of the group with Raynaud’s disease had aCL, which is higher than the 8.7% reported by Vayssairat et al. Patients with positive tests did not differ from those who had negative clinical manifestations or laboratory values.

All of the patients with SSc and RP and 13% of the healthy controls had positive IIF tests on HEP-2 substrates. None of the patients with SSc had antibodies to topoisomerase I (Scl-70) or SS-A/SS-B. No IIF pattern correlated with αβ2-GPI or aCL.

In our study we found that the frequency of antibodies to β2-GPI and aCL was low in scleroderma, 8% and 12% respectively. There were no clear clinical or laboratory correlations with a positive test.

Acknowledgments

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References


Table 1  Demographics and laboratory results in patients with SSc, RP, and normal controls

<table>
<thead>
<tr>
<th></th>
<th>Scleroderma (n=26)</th>
<th>Raynaud’s phenomenon (n=23)</th>
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<td>4</td>
<td>5</td>
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<tr>
<td>50+</td>
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<td>9.2 (SD 1.0)</td>
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BUN, blood urea nitrogen; CK, creatine kinase.
*p:Comparison of age distribution versus Ssc.

Figure 1  Comparison of αβ2-GPI and aCL antibody levels in patients with SSc, RP, and normal controls. The numbers on the ordinate represent optical density values converted to SMU (standard IgM αβ2-GPI units), MPL (1 MPL unit = the binding of 1 µg/mL IgM aCL), or GPL (1 GPL unit=the binding of 1 µg/mL IgG aCL). The arrows indicate the cut off values for each dataset.

- Healthy
- Raynaud’s
- Scleroderma

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Recombinant hepatitis B vaccination can also polyarthritis and positive rheumatoid factor. Tetanus toxoid injection has been associated exists, allowing an inference about the influ-arthritides. Often, a close temporal relation mination showed swelling of the right ankle, mid-foot, metatarsophalan-geal of the left big toe, and tenosynovi-tis of the long flexor of the right middle finger. This was associated with conjunctivitis and chlamydial urethritis, with raised levels of anti-chlamydial IgG. This plasma visceral-ity was raised at 1.69 mPa.s; haemoglobin was 139 g/l, rheumatoid factor negative, and HLA-B27 positive. Plain radiographs of the left ankle, foot, and right hand showed no abnormalities. He was treated with indomet-acin and an intra-articular injection of triamcinolone to the left ankle as well as minocy-cline for himself and his partner. The conjunctivitis settled within a few days, the ulcer healed overnight, but the joint took six months before becoming quiescent, by which time he was able to stop the indometacin. Chlamydia IgG was negative by the time.

Physical examination at the second presen-tation showed swelling of the right joint ankle, with tenderness and synovial thickening. The subtaloid, mid-tarsal, and metatarso-pannicular joints were fully mobile with no swelling. The erythrocyte sedimentation rate was raised to 36 mm/1st h.

Initially, triamcinolone was injected into the right ankle, indometacin 50 mg three times a day was prescribed, and he was given elbow crutches to stop him weight bearing on the right leg. Two weeks later there was partial improvement. Prednisolone (20 mg a day decreasing by 5 mg weekly) and enteric coated sulphasalazine (500 mg twice daily) were added. One month later the ankle syn-vorits and pain had settled.

Discussion
Several strands of evidence link different vac-cine to the development of a spectrum of arthritis. Often, a close temporal relation between vaccination and the onset of arthritis exists, allowing an inference about the influence of a particular vaccine. On this basis, hepatitis B vaccination has been associated with the development of rheumatoid arthri-tis, comprising a symmetrical inflammatory polyarthritis and positive rheumatoid factor. Recombinant hepatitis B vaccination has also produced such pictures. Symmetrical small joint polyarthritis but with negative rheuma-toid factor has been described in association with intravascular Baxillus Calmette-Guerin (BCG) vaccine used as immunotherapy for bladder carcinoma.

The spectrum of arthritis associated with vaccination is illustrated by the induction of large joint monartarthritides by combined diphthe-ria, poliomyelitis, and hepatitis B vaccine. In one of these cases, synovectomy was cura-tive until a booster vaccination five years later caused recurrence and, indeed, it has been suggested that rechallenge with vaccine may be associated with more severe symptoms. Further evidence for the role of vaccines in arthritis comes from monitoring of adverse drug reactions, with one survey indi-cating a causal link between rubella vaccina-tion and acute and chronic arthritis, especially in women.

The mechanisms underlying arthritides asso-ciated with vaccination are not yet fully understood. A cross reaction between bacte-rial lipopolysaccharide epitopes and synovial antigen, leading to an idiotype-anti-idiotype immunological response enhanced by HLA-B27 expression, may provide one model. However, HLA-B27 expression is not a pre-requisite for arthritis linked to vaccines although its presence may predict a more pro-longed and severe course. Vaccines may also trigger autoimmune responses by binding to critical antigen binding clefts on the major histocompatibility complex class II molecule, thereby triggering T cell proliferation. The impact of vaccination associated with vac-cination can be severe, with prolonged and significant morbidity lasting many months. Hassan and Oldham reported Reiter’s syn-drome with joint swelling and conjunctivitis lasting many months, whereas Bracci and Zoppini additionally reported fevers and lymphadenopathy with the hepatitis B surface antigen vaccine (Engerix B). As well, in our case, appropriate treatment, includ-ing non-steroidal anti-inflammatory drugs and intra-articular or oral steroids, can be useful in limiting the duration and degree of symptoms.

Vaccination has also been shown to cause necrotising vasculitis. Leucocytoclastic vasculi-tis has been induced most often, but polyar-thritis nodosa-like and systemic vasculitides have also been reported in a few instances. In children, two other syndromes may occur after rubella vaccination (and natural infection): (a) the “arm syndrome”, in which brachial radiculoneuropathy causes arm and hand pain, and peripheral symptoms that are worse at night; (b) “catcher’s crouch”, a lumbar radiculoneuropathy causing pain in the popliteal fossa on arising in the morning, which is exacerbated by knee extension and improves in a “catcher’s crouch” position. Both syn-dromes occur one to two months after vaccination. Although the initial episode may last up to two months, relapses may occur for up to a year, eventually resolving completely without permanent sequelae.

Our case highlights a relationship between vaccination and arthritis and the ability of vaccine to retrigger a reactive arthritis in a susceptible person. Although the mechanisms of vaccination and arthritis are not clear, there is sufficient evidence to suggest that some vaccines may cause joint disease or adversely affect pre-existing joint problems. It would therefore be important to warn patients awaiting vaccination about the possible ad-verse effect on joint symptoms.

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References

Asymptomatic splenic infarction in Wegener’s granulomatosis
Wegener’s granulomatosis (WG) is a necrotis- ing, granulomatous vasculitis that classically involves the clinicopathological triad of upper and lower respiratory tracts and the kidney. Less frequently, the disease may affect other organs as well. Serious and occasionally fatal complications within the spleen occur in many autoimmune rheumatic diseases, and prompt recognition of these complications is important. In nongeriatric patients with WG, the spleen was commonly affected: 78–100% of patients had splenic lesions with a combination of necrosis, vasculitis, and inflammatory formation. Clinical presentation of apparent splenic disease is rare, however. We wish to report briefly the case of a 47 year old woman who presented with manifestations of classical WG and radiological evidence of splenic infarcts.
Splenic involvement in WG has included such abnormalities as splenomegaly, capsular adhesion, impaired splenic function, and infarcts. Infarction may occur as a result of a distal occlusion of the splenic artery or its branches, because splenic parenchymal arteries are end vessels that do not communicate with one another. There are few reports on splenic infarction on post mortem in patients with WG. Histological examination frequently shows massive or multiple areas of splenic necrosis, usually associated with extensive central arteritis, splenic trabeculitis, follicular arteriolitis and necrosis, disseminated viscer al granulomata, and capsulitis. On CT, splenic infarcts classically and more commonly appear as peripheral, well defined, wedge shaped areas of low attenuation. However, other patterns of infarction have been recognised. These include multiple heterogeneous low attenuation lesions; regions of normal enhancement centrally with peripheral low attenuation; and large, low attenuation hypodense lesions that may have a rim of enhancing tissue peripherally. Examination with ultrasound in combination with duplex sonography of splenic blood supply permits non-invasive diagnosis of splenic infarction. The diagnosis can be confirmed by magnetic resonance imaging or CT scan, which permits assessment of the extent of splenic infarction.

Splen ic involvement in WG may be more prevalent than previously believed. Pain in the left upper quadrant and left shoulder and fever may be present after splenic infarction, but many patients remain asymptomatic. Consequently, cross sectional imaging is not often carried out and the lesion may frequently go unrecognised. Unless there are signs of imminent rupture of the spleen or haemorrhage, splenic infarction on post mortem in patients with WG is usually unrecognised. These include multiple heterogeneous low attenuation lesions; regions of normal enhancement centrally with peripheral low attenuation; and large, low attenuation hypodense lesions that may have a rim of enhancing tissue peripherally. Examination with ultrasound in combination with duplex sonography of splenic blood supply permits non-invasive diagnosis of splenic infarction. The diagnosis can be confirmed by magnetic resonance imaging or CT scan, which permits assessment of the extent of splenic infarction.

Two cases of *Mycobacterium avium* septic arthritis

The unusual and memorable case reported by Ter Borg and Terrmee serves as a useful reminder that atypical mycobacterial infections, although uncommon, need to be considered in immunocompromised patients. We present here two case reports of patients with pre-existing rheumatic disease receiving immunosuppressive treatment, who developed septic arthritis due to *Mycobacterium avium intracellulare.*

Case one

A 51 year old woman presented in 1999 with Raynaud’s phenomenon, facial telangiectasia, sclerodactyly, and a positive antinuclear antibody. She complained of exertional dyspnoea, and subsequent high resolution computed tomography of the chest disclosed a ground glass appearance, indicative of active alveolitis. A diagnosis of scleroderma with interstitial lung disease was made, and treatment with prednisolone 30 mg and azathioprine 100 mg daily was started. In August 2000, she complained of pain and stiffness in the left shoulder, and this was treated with an intra-articular steroid. Six months later, she developed a left shoulder effusion, from which 50 ml of serosanguinous fluid was aspirated. Culture, Gram stain and initial bacterial cultures of the fluid were negative, and Ziehl-Neelsen stain made a good clinical response.

**References**

Case two
A 36 year old man presented in 1993 with arthralgia, proximal muscle weakness, and a creatine kinase of 12 000 U/l. Muscle biopsy confirmed the diagnosis of polymyositis. Treatment with prednisolone (initially 60 mg daily) and azathioprine 150 mg was started. In 1997 he developed axillary lymphadenopathy, and subsequent biopsy showed M tuberculosis. A good clinical response was achieved with rifampicin, isoniazid, and ethambutol. In 1999 synovitis developed in the left knee and right wrist. Both joints were aspirated and injected with corticosteroids after initial Gram stain, Ziehl-Neelsen stain, and bacterial culture were negative. Eight weeks later, M avium was cultured from fluid in the left knee, and treatment commenced with clarithromycin, ethambutol, and rifampicin was started. Histology from a right wrist tenosynovectomy six months later demonstrated granulomas, and culture confirmed the presence of M avium. At present the patient continues to receive treatment with prednisolone 7.5 mg daily and azathioprine 150 mg daily, together with the antimycobacterial therapy, but clinical evidence of septic arthritis remains.

Discussion
Infective arthritis due to M avium is rare, most commonly occurring in immunocompromised subjects, such as those receiving immunosuppressive drugs, or HIV positive patients. The most commonly affected joint is the knee. Up to 40% of patients with atypical mycobacterial septic arthritis have received prior intra-articular corticosteroid injection in the affected joint. Diagnosis of these infections rests on culture of the synovial fluid (approximately 15%), or culture of surgically obtained specimens, though the often insidious nature of the infection may lead to a delay in diagnosis of many years. Antimycobacterial treatment is given (dependent on sensitivities), with or without surgical synovectomy. Prognosis is variable, but most patients can expect to make reasonable or good functional recovery.

In summary, we report two cases of septic arthritis due to M avium, in patients with previously diagnosed inflammatory arthritis, who had received azathioprine and systemic and intra-articular corticosteroids. Arthritis with significant synovitis is not a common feature of dermatomyositis or scleroderma and therefore M avium should be considered as a diagnosis in patients receiving these drugs.

Successful radiosynoviorthesis of an olecranon bursitis in psoriatic arthritis
We describe the case of a 45 year old male patient who for more than 10 years had psoriasis with typical manifestations at knees and elbows. The family recalled psoriasis of the grandfather. Without any trauma or specific injury, an olecranon bursitis and an arthritis of the left elbow developed in 1999 as the initial manifestation of psoriatic arthritis. Three months after developing the bursitis, the patient came to the rheumatological outpatient clinic for his first visit. The clinical findings showed a patient with good general condition (weight 186 cm, weight 93 kg), blood pressure 120/80 mm Hg, rheumatic pulse rate 68 beats/min; psoriatic skin lesions at knees and elbows; no reduction of spine mobility. The left elbow showed an olecranon bursitis with a diameter of 50 mm. The remaining musculoskeletal system was not affected.

The laboratory results were within the normal range, HLA-B27 was negative, anti-nuclear antibodies negative, functional tests of liver and kidney were normal.

Radiographic findings showed that sacroiliac joints and the left elbow joint were normal. Sonography showed an olecranon bursitis with a large effusion (fig 1A). Diclofenac 100 mg twice daily was given for the first two weeks but did not produce any effect. After that, the olecranon bursa was punctured aseptically, and a crystal suspension of 10 mg trimcinolone hexacetonide was injected. Two days later, the bursitis relapsed completely. Further therapeutic options were surgical bursectomy or, alternatively, radiation synovectomy. After having received complete information, the patient gave his consent to treatment by radiosynoviorthesis. After aspiration of 9 ml of a serous effusion, 55 MBq rhenium-186 was instilled into the olecranon bursa, and then, to avoid radiosynovitis, 5 mg trimcinolone was injected. Radiosynovography scanning immediately after the injection and three days later showed that the radiouclide was distributed uniformly in the bursa. There were no local signs of an infection.

A physical examination three months after radiation synovectomy of the olecranon bursitis showed regular clinical findings. Arthrosonographic results had also normalised (fig 1B). Even six months later the bursitis was not reactivated. Radiation synovectomy is often used as an alternative, or in addition to, surgical synovectomy. Definite indications are chronic persisting synovitis, intermittent hydroxys, relapsing synovitis after surgical synovectomy, proliferative arthropathy, and activated osteoarthritis resistant to other treatments. Some studies have reported successful concomitant treatment of Baker’s cysts in the treatment of psoriatic arthritis, but radiosynovectomy. A few cases of synovitis of Baker’s cysts have been reported. In our case, neither the treatment with a non-steroidal anti-inflammatory drug (200 mg diclofenac daily) nor the local treatment with trimcinolone hexacetonide after a decompression aspiration led to improvement. An alternative to surgical bursectomy, radiosynoviorthesis with rhenium-186 was performed. The patient improved quickly and started working again the following day. The follow-up examinations, after intervals of three and nine months, confirmed the continuing success.

As far as we know this is one of the first reports on radiosynoviorthesis in an isolated bursitis. This case gives cause for hope that radiosynoviorthesis represents a successful alternative treatment to operational intervention for chronic inflammation of the bursa.

References
Multiple sclerosis in the course of systemic sclerosis

We describe the case of a young woman with longstanding systemic sclerosis (SSc), who later developed multiple sclerosis (MS), and discuss the possible explanations for this rare co-occurrence.

A 30 year old white woman was admitted to the department of neurology of our institution with 10 days’ history of vertigo and diplopia. A year earlier the patient had had an episode of paraesthesiae of her right leg, which resolved spontaneously within two to three weeks. Since the age of 22, she had been suffering from xerostomia. A five day trial of intravenous methylprednisolone 500 mg/day resulted in moderate relief of her symptoms and treatment was started with interferon β to prevent progression of the neurological process. At present, the patient has been receiving interferon β for two years and there is no evidence of any further neurological compromise.

One can suggest three possibilities for the coexistence of the neurological syndrome and the SSc in this patient. Firstly, MS occurring independently from SSc might account for the neurological deficits, given the laboratory findings and the patient’s sex and age, and the prevalence of MS in the general population. However, it is also possible that there is an association between the two conditions, because MS, like SSc, is also believed to be autoimmune in nature, and the pathogenetic role of T cells is crucial in both processes. Furthermore, MS has been increasingly reported in association with other autoimmune diseases not primarily affecting the nervous system. If any of the above possibilities is present, the prognosis and therapeutic approach of our patient should match those of typical MS. The coexistence of SSc and MS is rare and, as far as we know, has been described in only four patients. Rapidly progressive and finally gripping MS developed in their early twenties, whereas SSc appeared later in the course of the MS in all four patients. Interestingly, unlike these cases, our patient presented in her thirties with a mild form of MS, several years after the onset of SSc.

A third possibility exists that, neurological manifestations of this patient might have been part of her primary disease—that is, SSc. Involvement of the central nervous system (CNS) in this disease is considered uncommon, and secondary to vasculopathic damage. The fact that our patient had prolonged visual evoked potentials, suggestive of optic neuropathy, is rather in favour of MS, although this abnormality has been reported in SSc. On the other hand, a significant percentage of patients with systemic lupus erythematosus may present with CNS disease, and, some of them with oligoclonal banding in the CSF. Brain or spinal cord disease, or both, with clinical features and laboratory findings indistinguishable from MS has been reported in Sjögren’s syndrome too, although CNS involvement in this syndrome has been a matter of serious debate.

In the absence of guidelines for the management of such patients, we considered our patient as a case of classical MS and, therefore, she was not deprived of the possible benefit of a disease modifying treatment, such as interferon β.

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References

www.annrheumdis.com
Heavy cigarette smoking and RA
(Masi AT, Aldag JC, Malamet RL. Ann Rheum Dis 2001;60:1154.)

The authors of this letter, in a further analysis of their data, found that four heavy smokers in the control group were incorrectly included in the 168 subjects matched to the 42 pre-RA cases who had baseline negative rheumatoid factor (RF$^-$) status. They should be correctly reassigned to the 48 matched controls for the 12 pre-RA cases who had baseline positive rheumatoid factor (RF$^+$) status.

The correct assignments place 11 (23%) heavy smokers in the 48 controls for the 12 pre-RA RF$^+$ cases. Those 12 cases include two (17%) heavy smokers. The 168 controls for the 42 pre-RA cases who had baseline negative rheumatoid factor (RF$^-$) status should correctly include eight (5%) heavy smokers. Those 42 cases include 11 (26%) heavy smokers. The new correct figures are shown in bold in the table.

The correct assignments strengthen the findings in this prospective, community based study that baseline heavy cigarette smoking was an independent risk factor from baseline positive rheumatoid factor status.

### Table 1

Numbers of pre-RA cases and matched controls reporting heavy cigarette smoking (CS 30+/day) at baseline by relevant categories and odds ratios (ORs) with 95% confidence intervals (95% CIs) for developing ACR+ rheumatoid arthritis

<table>
<thead>
<tr>
<th>Categories</th>
<th>Pre-RA cases</th>
<th>Respective matched controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number</td>
<td>CS 30+/day (%)</td>
</tr>
<tr>
<td>Pre-RA RF+</td>
<td>12</td>
<td>2 (17)*</td>
</tr>
<tr>
<td>Pre-RA RF$-$</td>
<td>42</td>
<td>11 (26)</td>
</tr>
<tr>
<td>Entry and post-RA RF$-$</td>
<td>15</td>
<td>4 (27)</td>
</tr>
<tr>
<td>Conversion of pre-RA RF$-$ to RF$^+$</td>
<td>27</td>
<td>7 (26)</td>
</tr>
</tbody>
</table>

*No association of CS 30+/day with pre-RA RF$^+$ (p=0.99).
†Conversion of RF$^-$ at baseline to RF$^+$ after clinical onset of RA.
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20–24 Feb 2002; Geneva, Switzerland
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Successful radiosynoviorthesis of an olecranon bursitis in psoriatic arthritis

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