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. 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 proceeds 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. 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 Debarakation at Marseilles” in the Maria de Medici cycle from 1622 to 1625 for the Luxembourg Palace in Paris. 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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Authors’ response
In their letter commenting on our article, 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. The question of the week of the questionnaire. The editors will decide as before whether to accept or reject the response. Providing it isn’t libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on “read eletters” on our homepage.
of the three month follow up (that is, two months after discharge) in our study 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 assessments 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 four (4a–4d) and five (5a–5c) comprising the role physical and role emotional function. For this reason, we report 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. The authors created a modified SF-36m, which was adapted in items 5, 4, 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 responsive in 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. It also includes a number of disease-specific scales as physical function, etc and is responsive in the SF-36m. This was adapted in items 4, 5, and 8.

Weber et al recently.

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Weber et al.
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 Sjogren’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 IgM, 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 anti-cardiolipin (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 hypoa (with decreased carbon monoxide transfer factor (TcO2). The three patients with SSc and aCL had mean disease duration of 112 months. One had hypoa (with normal TcO2 and non-restrictive pulmonary function tests), one had restrictive lung disease and digital ulcers, and one had osseopag hypomobility. None of the study participants had thrombo cytopenia or a history of deep venous thrombo sis. Twenty two per cent of the group with Ray naud’s disease had aCL, which is higher than the 8.7% reported by Vannier et al.19 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 1 (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

This research is supported by the Canadian Institutes for Health Research. Dr Schoenroth is supported by the Alberta Heritage Foundation for Medical Research. Dr Lonzetti is supported by scleroderme Quebec.

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References

Recurrence of reactive arthritis after a booster dose of tetanus toxoid

We report the case of a 24 year old man who developed a recurrence of reactive arthritis after receiving a booster of tetanus toxoid.

Case report

A 24 year old man presented with acute swelling of the right ankle. Two weeks before presentation, he had been given a booster tetanus toxoid vaccination. Within a few days of the injection, he felt pain and noticed swelling of the right ankle. The swelling and pain worsened, such that at presentation he was walking with a pronounced antalgic gait. There was no preceding history of trauma, infection, or any past history of psoriasis, iritis, inflammatory bowel disease, or inflammatory back pain. There was no family history of ankylosing spondylitis.

Three years previously he had presented to another rheumatologist with acute synovitis of the right ankle. He had been treated with indomethacin and an intra-articular injection of triamcinolone to the right ankle as well as minocycline for himself and his partner. The joint was assessed to be stable and no further treatment was advised. The patient only took six months before becoming quiescent, by which time he was able to stop the indomethacin. Chlamydia IgG was negative by this time.

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

Initially, triamcinolone was injected into the right ankle, indomethacin 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 sulfasalazine (500 mg twice daily) were added. One month after the ankle synovitis and pain had settled.

Discussion

Several strands of evidence link different vaccine preparations to the development of a spectrum of arthritides. Often, a close temporal relation exists, allowing an inference about the involvement of vaccines to the development of a spectrum of arthritides. Often, a close temporal relation exists, allowing an inference about the involvement of vaccines to the development of a spectrum of arthritides.

The spectrum of arthritis associated with vaccination is illustrated by the induction of large joint monarticular by combined diphtheria, poliomyelitis, and tetanus toxoid vaccine.1 In one of these cases, synovectomy was curative 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.2 Further evidence for the role of vaccines in arthritis comes from monitoring of adverse drug reactions, with one survey indicating a causal link between rubella vaccination and acute and chronic arthritis, especially in women.3

The mechanisms underlying arthritis associated with vaccination are not yet fully understood. A cross reaction between bacterial lipopolysaccharide epitopes and synovial antigen, leading to an idiotype-anti-idiotype immunological response enhanced by HLA-B27 expression, may provide one model.4 However, HLA-B27 expression is not a prerequisite for arthritis linked to vaccines' although its presence may predict a more prolonged and severe course.5 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.6 The impact of vaccination associated with vaccination can be severe, with prolonged and significant morbidity lasting many months. Hassan and Oldham reported Reiter's syndrome with joint pains and conjunctivitis lasting many months, whereas Bracci and Zoppini additionally reported fever and lymphadenopathy with the hepatitis B surface antigen vaccine (Engerix B).7,8 However, as with other vaccines, articular pain, and abnormalities. He was treated with indomethacin and an intra-articular injection of triamcinolone to the left ankle as well as minocycline for himself and his partner. The joint was assessed to be stable and no further treatment was advised. The patient only took six months before becoming quiescent, by which time he was able to stop the indomethacin. Chlamydia IgG was negative by this time.

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Our case highlights a relationship between vaccination and arthritis and the ability of vaccine to trigger 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 advisable to warn patients at the time of vaccination about the possible adverse effect on joint symptoms.

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References

Case report

A 47 year old woman during the past month developed fevers to 38.6°C associated with weight loss, diffuse arthralgias, anaemia, and erythrocyte sedimentation rate of more than 100 mm/1st h. During the past three months she complained of nasal congestion and occasional episodes of epistaxis. A chest x ray examination showed a left upper lobe density, and her family doctor prescribed oral amoxicillin in combination with clarithromycin, without improvement. Ten days before admission gross haematuria was noted and a freshly collected urinary sediment showed the presence of red blood cell casts. Renal function declined rapidly with a serum creatinine level of 600 µmol/l and the patient was referred to the hospital for further investigation. A positive cANCA titre (1/160) was found using the indirect fluorescence technique and a positive antiproteinase 3 result on enzyme linked immunosorbent assay (ELISA; 66 U/ml). WG was considered on the basis of ANCA analysis results and the multisystemic nature of the disease and a percutaneous renal biopsy was performed under computed tomography (CT) guidance. Unspecification of renal biopsy specimens with light microscopy and immunofluorescence showed an acute necrotising segmental pauci-immune glomerulonephritis with crescent formation in more than 50% of the glomeruli. An impressive and rather unexpected CT finding was the presence of well defined areas of low attenuation within the spleen, consistent with infarction, within the larger hypodense lesions (fig 1). A search for lupus anticoagulant and anticardiolipin antibodies was negative and no abnormalities of blood clotting could be detected. A trans-oesophageal echocardiogram failed to detect cardiac sources of emboli.

Treatment was started with 500 mg methylprednisolone intravenously per day for three consecutive days together with cyclophosphamide at a dose of 2 mg/kg body weight per day orally. After three days methylprednisolone was continued at 48 mg/day orally. Pulmonary and renal function progressively recovered and serum creatinine was 150 µmol/l on discharge.

Today, three years after the initial presentation, the patient is in stable remission and serum creatinine is 125 µmol/l. A recently performed CT scan showed a considerable volume reduction and scarring of the spleen (fig 2).

Discussion

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 visceral 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.

Splenic 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 signs of infection, splenic infarcts are often carried over and may be missed.

Figure 1 Abdominal CT scan showing a normal liver and a spleen with well defined areas of low attenuation, consistent with infarction.

Figure 2 Abdominal CT scan three years later shows volume reduction and scarring of the spleen.
Successful radiosynoviotherapy 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 special straining, 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 (height 186 cm; weight 93 kg), blood pressure 120/80 mm Hg, rhythmic 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 ranges, 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 radiosynoviotherapy. 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. Radiosynovectomy was performed after the injection and three days later showed that the radionuclide 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 hydrops, relapsing synovitis after surgical synovectomy, hemophilic arthropathy, and activated osteoarthritis resistant to other treatments. Some studies have reported successful concomitant treatment of Baker’s cysts in the treatment of synovitis, but radiosynovitis with rhenium-186 for the treatment of Baker’s cysts is not usual. It is possible, however, by infusion of a radioisotope into the knee joint, but the popliteal cyst must not be punctured directly. Due notice should be taken of contraindications.

Other reports disagree about the success rates of radiosynoviotherapy in treating psoriatic arthritis compared with rheumatoid arthritis.3,4 A few years ago, only patients aged over 40 were treated with radiosynovitis. Today, this treatment is used in an increasing number of younger patients. The success rate for radiosynoviotherapy of olecranon bursitis is between 50 and 80%, depending on the localisation and the amount of inflammatory activity.5,6 Up to now, no studies of the treatment of chronic inflammation of the bursa by radiosynoviotherapy have been reported. In our patient, neither the treatment with a non-steroidal anti-inflammatory drug (200 mg diclofenac daily) nor the local treatment with triamcinolone hexacetonide after decompression of the cyst failed. An alternative to surgical bursectomy, radiosynoviotherapy with rhenium-186 was performed. The patient improved quickly and started working again the following day. The follow up examination, after intervals of three and nine months, confirmed the continuing success.

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

References


Figures

Figure 1 Sonography of the left elbow (A) showing an olecranon bursitis (58.1 mm; 17.7 mm; 3.3 mm; 2.5 mm) and (B) three months after radiation synovectomy.
we describe the case of a young woman with 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. She had been a patient of the rheumatology section of the same hospital for SSc, and her condition remained stable with treatment with oral penicillamine 500 mg daily and methylprednisolone 2 mg daily.

Clinical examination showed an alert woman with normal vital signs and typical appearance of scleroderma—that is, tightness and atrophy of the skin of her face and hands with contractions of her fingers. Examination of the lungs, heart, and abdomen showed no abnormality. Fundoscopy disclosed temporal papillar bilateral. There was vertical nystagmus on upward gaze and diplopia on looking to the right, without apparent ophthalmoplegia. Deep tendon reflexes were brisk and abdominal reflexes were absent bilaterally. An extensor plantar response was seen on the right but no muscle weakness or sensory loss.

There was no evidence for keratoconjunctivitis sicca, as Schirmer's I, rose bengal, and fluorescein showed positive antinuclear antibodies at a titre of 1:640, of the fine speckled pattern, and positive anti-ScI70 antibodies. Antibodies to cardiolipin and the other extractable nuclear antigens, including Ro(SSA), La(SSB), Sm, and RNP, were absent, as they had been on several occasions in the past. Visual evoked potentials were abnormal bilaterally. Cerebrospinal fluid (CSF) analysis disclosed increased intrathecal IgG synthesis (IgG index 0.88, normal <0.66) and oligoclonal bands. Magnetic resonance imaging (MRI) studies showed several abnormalities of the brain and the cervical cord (fig 1).

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, the 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 focal neurological compromise 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

Figure 1
(A) T weighted coronal MRI scan of the brain showing bilateral areas of increased signal intensity (up to 5 mm) into the white matter of the parietal lobes, mainly on the left. (B) Sagittal MRI scan of the cervical cord showing an area of increased signal intensity, which extends from the C3 to C7 level, resulting in focal enlargement of the cord at that level.
CORRECTION

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.

Corrections printed in the journal also appear on the Annals website www.annrheumdis.com and are linked to the original publication.

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**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>
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</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.
FORTHCOMING EVENTS

3rd International Congress on Autoimmunity
20–24 Feb 2002; Geneva, Switzerland
Contact: Professor Yehuda Shoenfeld, 3rd International Congress on Autoimmunity, PO Box 50006, Tel Aviv 61500, Israel
Tel: 9723 514 0018
Fax: 9723 517 5674
Email: autoim02@kennes.com

22nd European Workshop for Rheumatology Research
28 Feb–3 Mar 2002; Leiden, The Netherlands
Contact: Professor F.C. Breedveld, Leiden University Medical Centre, Department of Rheumatology, PO Box 9600, 2300 RC Leiden, The Netherlands
Tel: +31 (0)71 526 3598
Fax: +31 (0)71 526 6752
Email: F.C.Breedveld@lumc.nl
Website: www.eewrr.org

Tenth Intensive Applied Epidemiology Course for Rheumatologists
11–15 Mar 2002; Manchester, UK
No previous experience in epidemiology is needed. The course is residential and limited to 25 places.
Contact: Ms Lisa McClair, ARC Epidemiology Unit, University of Manchester, Oxford Road, Manchester M13 9PT, UK
Tel: +44 (0)161 275 5993
Fax: +44 (0)161 275 5043
Email: Lisa@fst.ser.man.ac.uk

OMERACT VI
11–14 Apr 2002; Bali
Includes two modules: MRI and economics; and four workshops: patients’ perceptions, imaging (healing), progressive systemic sclerosis, minimally important clinical difference and osteoarthritis.
Contact: Conference Organisers Q20, 7 Swann Street, Old Isleworth, Middlesex TW7 6JR, UK; or Peter Brooks, Faculty of Health Sciences, Level 1, Edith Cavell Building, Royal Brisbane Hospital, Herston 4029, Australia
Fax: +61 2808569 9555
Email: tony@q2q.co.uk or p.brooks@mailbox.uq.edu.au

British Society for Rheumatology
XIXth AGM
23–26 Apr 2002; Brighton, UK
Contact: BSR, 41 Eagle Street, London WC1R 4TL, UK
Website: www.rheumatology.org.uk

4th EULAR Sonography Course
25–28 Apr 2002; Madrid, Spain
The course is entitled “Practical use of musculoskeletal ultrasonography”
Contact: Esperanzo Naredo
Email: enaredo@eresmas.com
Website: www.clevelandclinicemed.com/courses/Vascultis2002.asp

10th International Congress on Behcet’s Disease
27–29 June 2002; Berlin, Germany
Under the auspices of the International Society for Behcet’s Disease. Up to eight young investigator awards, each of $500, will be awarded on the basis of abstracts submitted.
Contact: Professor Ch. C Zouboulis, Department of Dermatology, University Medical Centre, Hansveld Kleyer 108, 4000 Liège, Belgium
Fax: +32 4 254 12 90
Email: zoubbere@zedat.fu-berlin.de

29th Scandinavian Congress of Rheumatology
15–18 Aug 2002; Tromso, Norway
Contact: Hans Nossent, Department of Rheumatology, University Hospital Tromso, Norway
Tel: 47 776 27294
Fax: 47 776 27258
Email: 29scrc2002@ritzo.no or revhan@ritzo.no

21–22 Sep 2002; Pavia, Italy
Contact: Organising secretariat: eventi S.R.L., Corso Cavour, 18/20 - 27100 Pavia, Italy
Email: trs@e20pr.com
Website: www.e20pr.com
Congress website: www.medicine.ucsd.edu/albani/2001 meeting

OsteoArthritis Research Society International (OARSI) World Congress
22–25 Sep 2002; Sydney, Australia
Contact: OsteoArthritis Research Society International (OARSI), 2025 M Street, NW, Suite 800, Washington DC 20036, USA
Tel: +1 202 367 1177
Fax: +1 202 367 2177
Email: oarsi@oarsi.org
Website: www.oarsi.org

10th International Congress on Antiphospholipid Antibodies
29–3 Sep 2002; Sicily, Italy
Deadline for abstracts 1 April 2002
Contact: Secretariat, 10th International Congress on Antiphospholipid Antibodies, c/o Kenes International, PO Box 50006, Tel Aviv 61500, Israel
Tel: 972 3 5140189
Fax: 972 3 5140077 or 972 3 5172484
Email: aps@kenes.com
Website: www.kenes.com/aps

7th International Conference on Eicosanoids and Other Bioactive Lipids in Cancer, Inflammation and Related Diseases
14–17 Oct 2002; Nashville, Tennessee, USA
Contact: Lawrence J. Marnett, Biochemistry Department, Vanderbilt University, School of Medicine, Nashville TN 37232-0146, USA
Tel: (615) 343 7329
Fax: (615) 343 7534
Website: www.eicosanoids.science.eaync.edu

66th American College of Rheumatology AGM
25–29 Oct 2002; New Orleans, USA
Contact: ACR, Ronald P Olejko, Director of Conferences and Meetings, 1800 Century Park East, Suite 230, Atlanta, Georgia 30045–4300, USA
Tel: +1 404 633 3777
Fax: +1 404 633 1870
Email: acr@rheumatology.org
Website: www.rheumatology.org

Third International Meeting on Social and Economic Aspects of Osteoporosis and Osteoarthritis
7–9 November, 2002; Barcelona, Spain
Contact: Yolande Piette Communication, Blvd. evard Kleyer 108, 4000 Liège, Belgium
Tel: 32 4 254 12 25
Fax: 32 4 254 12 90
Email: ysyc@compuserve.com

Certifying Examination in Pediatric Rheumatology
18 Nov 2002
Contact: American Board of Pediatrics, 111 Silver Cedar Court, Chapel Hill, NC 27514-1513, USA
Tel: 919 929 0461
Fax: 919 918 7114 or 919 929 9255
Website: www.abp.org

www.anrheumdis.com

OsteoArthritis Research Society International (OARSI) World Congress
22–25 Sep 2002; Sydney, Australia
Contact: OsteoArthritis Research Society International (OARSI), 2025 M Street, NW, Suite 800, Washington DC 20036, USA
Tel: +1 202 367 1177
Fax: +1 202 367 2177
Email: oarsi@oarsi.org
Website: www.oarsi.org

10th International Congress on Antiphospholipid Antibodies
29–3 Sep 2002; Sicily, Italy
Deadline for abstracts 1 April 2002
Contact: Secretariat, 10th International Congress on Antiphospholipid Antibodies, c/o Kenes International, PO Box 50006, Tel Aviv 61500, Israel
Tel: 972 3 5140189
Fax: 972 3 5140077 or 972 3 5172484
Email: aps@kenes.com
Website: www.kenes.com/aps

7th International Conference on Eicosanoids and Other Bioactive Lipids in Cancer, Inflammation and Related Diseases
14–17 Oct 2002; Nashville, Tennessee, USA
Contact: Lawrence J. Marnett, Biochemistry Department, Vanderbilt University, School of Medicine, Nashville TN 37232-0146, USA
Tel: (615) 343 7329
Fax: (615) 343 7534
Website: www.eicosanoids.science.eaync.edu

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