

## Journal summary

### LEADERS

**Osteoporosis and its treatment** p 663  
Osteoporosis is a disorder of bone remodelling and by its treatment we aim at maintaining the bone mass and its mechanical strength. We can do this in two ways: we can try to prevent it happening in the first place—which is difficult—or we can treat the established disease and hope to minimise its effect. In the future we may be able to modulate local growth factors, but there are so many of them known now that this is looking less likely; we shall see.

**Isolated Raynaud's phenomenon and connective tissue disease** p 666  
Raynaud's phenomenon is due to the constriction of small, digital arteries on exposure mainly to cold and may occur in isolation or in relation to connective tissue disease. Is there any connection between the two varieties? Sadly the answer may be yes in a substantial number. Those who go on to develop significant disease (usually scleroderma) may already have signs at an early stage if we look carefully enough.

### SCIENTIFIC PAPERS

**Purine metabolites, synovial membrane, and the inflammatory arthritides** p 669  
RA damages joints and nucleotide catabolism increases during tissue injury—so does the increasing presence of purine metabolites reflect worsening damage to the joint? The correlation with laboratory measurements was alas only weak and there was no association with disease duration and radiological findings.

**Synovial membrane and predictors for the course of rheumatoid arthritis (RA)** p 673  
It is difficult to predict the progress of RA, and biopsy and subsequent histological examination of the membrane has not proved satisfactory because of the need to do serial examinations and the difficulty in obtaining representative tissue samples reliably. This study, however, showed a correlation over three years between various features and clinical status in mild but not active disease.

**Serum amyloid, C reactive protein, and inflammation** p 677  
C reactive protein and amyloid A protein are probably the best acute phase proteins with which to measure the inflammatory stimulus. This study from Bristol and Leeds using immunodiffusion and immunoassay to measure the effect on these proteins of intradermal injection of urate crystals showed that amyloid A was a more sensitive indicator of inflammation, though both increased in response. Unfortunately, its routine assay is impracticable and C reactive protein is an acceptable second best.

**Drug interactions in the arthritic patient** p 680  
The potential for drug interaction was present in over a half of 100 arthritic patients studied, but fortunately only a small proportion had any obvious clinical evidence of this. As so many patients need to take several drugs together to control their symptoms this is reassuring news, though it still remains a potential cause for concern.

**Salicylate ototoxicity and serum salicylate concentration** p 682  
It is interesting to know whether the presence of salicylate ototoxicity reflects the serum concentration of the drug. Apparently not. Many patients had no symptoms even when

their serum concentration was much higher than those with symptoms. The presence of symptoms affecting the ear was not a sensitive indicator.

**Sjögren's syndrome and neutrophil function** p 685  
Infections are a major cause of morbidity in Sjögren's syndrome, particularly those affecting the respiratory tract. It is important to know, therefore, whether neutrophil function is affected, but this Swedish report found little evidence for this. Neutrophil adherence is impaired, however, and this may possibly have a role. Tracheobronchial dryness is possibly also another factor.

**T cells and Sjögren's syndrome** p 691  
Subsets of T cells may well act as part of the trigger in this syndrome, and an immunohistochemical study of labial biopsy specimens showed that the predominant cell in the infiltrate bore the T cell helper/inducer phenotype. Most showed the  $\alpha/\beta$  T cell receptor and were of the CD4 subset, though a high proportion of cells within lesional tissue expressed CD5 and CD45 cell surface molecules. Their function in pathogenesis remains to be explored.

**A *Yersinia enterocolitica* epidemic and its late complications** p 694  
A follow up report 13 years on of an epidemic due to this organism showed that about half of the patients remained healthy but of those complaining of problems, most had musculoskeletal disorders. There was a close correlation between the HLA-B27 antigen and the presence of these disorders and this was also evident with a persistently raised yersinia antibody. Two of the patients developed ankylosing spondylitis.

**Candida arthritis** p 697  
We are seeing more and more of systemic candidiasis in immunocompromised patients, but we rarely see it in the joint fortunately. The ability of *Candida albicans* to cause lymphocyte blastogenesis is well known, and this effect was much more evident on synovial fluid lymphocytes than on those in the peripheral blood. The proportion of cells expressing class II antigens was also greatly increased in the synovial fluid, and synovial derived CD4 positive T lymphocyte clones with specificity for candida antigens were characterised.

**Systemic lupus erythematosus (SLE) in the elderly** p 702  
SLE tends to be a disease of younger women, though it is seen in older patients too. In this series nearly a fifth of the patients presented with the disease for the first time after the age of 50 years. In this older group arthritis was less common as were many other clinical features but, by contrast, a greater incidence of myositis was noted. The older patients also tended to have lower titres of antibodies to dsDNA and Ro.

**Disease activity and infection and their effects on interleukin 2 receptors in SLE** p 706  
Soluble interleukin 2 receptor concentration was found to be increased in SLE compared with controls even if the disease was inactive: it became higher still when the SLE became active. The presence of infection also raised it whether the SLE was in remission or not and this was particularly evident in chronic infective disease. Soluble interleukin 2 receptor concentration is likely to be helpful in distinguishing whether infection is present or not therefore.

**SLE and blood rheology** p 710  
Haemorrhheological abnormalities have been seen in several autoimmune diseases, and this investigation was designed to

look at the flow properties of blood in SLE as compared with controls. The investigators were able to show that plasma viscosity and red cell aggregation were significantly different, indicating that there was reduced blood fluidity in SLE, though there were no marked sex differences. This was particularly seen in those with the systemic form of the disease. The clinical significance of all this is as yet far from clear.

## CASE REPORTS

### Acquired C1 inhibitor deficiency and SLE p 713

Complement deficiency is sometimes associated with SLE and the genetic deficiency of C1 inhibitor is linked with hereditary angioneurotic oedema. A 22 year old woman is reported here with both SLE and acquired C1 inhibitor deficiency. Although she did not have angioedema, she was psychotic and had diffuse slow waves in her electroencephalogram. She had gross abnormalities of her complement system with virtually no C4; a very low C4 concentration may be associated with central nervous system abnormality.

### Pseudoseptic arthritis and neuropathic arthropathy p 717

A 49 year old man presented with bilateral shoulder swelling due to a neuropathic arthropathy associated with syringomyelia. The synovial fluid showed signs of inflammation so it was understandable that this was at first thought to be an example of a septic arthritis.

### Septic arthritis in osteoarthritis (OA) of the hip p 722

Here the infection in the hip joint was real but its development was insidious and it again caused diagnostic difficulty, as is so often the case. It is always a serious matter and one patient of the four died and two required joint excision. The presence of OA may perhaps be a predisposing factor, and the authors suggest that if a patient with OA develops new symptoms in a joint with systemic upset the diagnosis of infection should always be considered.

### Splenectomy, Felty's syndrome, and septic arthritis p 724

This time septic arthritis is reported as complicating Felty's syndrome in a patient who had earlier had a splenectomy for this condition. The infecting organism was *Streptococcus pneumoniae* and this led to complication. She had previously

been vaccinated with a multivalent pneumococcal vaccine, but the infecting serotype was not represented among them, unfortunately. The outcome was satisfactory, however, but the authors suggest that similar patients who have had a splenectomy should carry a card stating this. A sensible suggestion.

## MASTERCLASS

### Algodystrophy p 727

This was first particularised during the American Civil War, though the syndrome was incompletely recognised before. It usually occurs after trauma, though this is not always the case. The symptoms are accompanied often by vasomotor changes in the skin, and treatment may be difficult. It is the cause of occasional management headaches to the rheumatologist so it is timely to consider this afresh.

## REVIEW

### Immune functions, inflammatory reactions, and HLA-B27 p 731

As the authors point out, the association between the major histocompatibility antigen HLA-B27 and ankylosing spondylitis is the strongest one known between class I HLA and disease. Many are the tendencies that have been found to be associated with B27 but none of these is powerful enough to explain the disease links and there is not much in common between the bacterial structures we potentially associate with B27 linked diseases. There is much yet to unravel.

## HYPOTHESIS

### Heat shock proteins and RA and hormonal factors p 735

The idea of a working hypothesis is to induce some lateral thinking in us all and perhaps stimulate research in order to shed more light on a particular enigma that exercises the curiosity of the scientific community. The current article seeks to look at a possible link between heat shock proteins and hormonal and reproductive factors in RA. This is as yet an area in which productive research may be quite exciting.

EDITOR