LEADERS

Cytokines and eicosanoids p 207
It is now agreed that suppression of prostaglandin synthesis does not modify the course of inflammatory joint disease and attention has turned to the mode of action of the eicosanoids (seemingly like local hormones) and cytokines (regulatory factors acting locally but with a longer term of action). This group of substances lumped together and loosely called cytokines comprises a variety of factors but with different regulatory functions, including some that regulate eicosanoid production. How this happens is not yet clear. The subject is complex and the leader explores the current state of knowledge.

Pigmented villonodular synovitis p 210
What is pigmented villonodular synovitis? We are far from clear about it, not least because it is so rare, but there is a hint that it may be inflammatory in origin even though we have no satisfactory villain in view for the causative agent(s). We are not even sure how best to treat it, though thankfully we know that people do not die from it. It is timely to air our ignorance of the subject.

SCIENTIFIC PAPERS

Nephropathy and rheumatoid arthritis (RA) p 214
Patients with RA with and without nephropathy were compared in this Finnish study to see if there were any differences in levels of circulating immune complexes (CICs) between the two groups. No such differences were noted, but serum IgA and IgM concentrations were significantly higher in those with amyloid deposits and mesangial glomerulopathy. This is in contrast with the increase in CICs seen in most extra-articular complications in RA.

Rheumatoid arthritis, mild inflammatory arthritis, and genes. I p 219
It is difficult when an inflammatory arthritis begins to see whether this is mild and self limiting, or will progress to definite rheumatoid arthritis (RA). A study of the major histocompatibility complex may help to sort this out if a significant genetic predisposition to one or the other can be discovered. Such a difference between the two was indeed uncovered in this study: patients with definite RA had overrepresentation of DR4 and DR2 and associated extended haplotypes, whereas those with mild inflammatory arthritis tended only to have antigens of the A and B loci. This clearly had diagnostic implications.

Rheumatoid arthritis, mild inflammatory arthritis, and genes. II p 225
Further study of the genetic differences between patients with definite rheumatoid arthritis (RA) and those with mild inflammatory arthritis suggested that the frequency of the immunoglobulin heavy chain allotype Glm(2) on chromosome 14 was increased in RA: previous studies have been conflicting. Frequencies of the complement C3F allele were normal in RA but increased in mild inflammatory arthritis and perhaps it acts independently on major histocompatibility genes to protect or otherwise against disease. Mild inflammatory arthritis may be provoked by many infective agents and the response of the individual does seem at least in part to be genetically determined:

Rheumatoid arthritis (RA) and osteocalcin p 229
Osteocalcin is present in the synovial fluid in much greater quantities in patients with osteoarthritis (OA) than in those with RA. Furthermore, the osteocalcin binds to hydroxyapatite to only a limited extent in RA but almost wholly in OA. The authors conclude that active osteocalcin is produced in only limited amounts in RA, the inactive form being secreted in larger amounts than normal. Perhaps osteoblast function is abnormal in this disease.

Femoral head cartilage and crystals p 231
Crystals are not usually seen in the non-calcified layer of articular cartilage, though of course they are present in osteoarthritis and the crystal deposition diseases. In femoral head articular cartilage in elderly patients crystal deposition was found to be denser in the superficial layers of the cartilage, particularly where the matrix contained cell debris, than in the deeper layers. This crystal deposition did not seem to be related to chondrocyte activity in contrast with previously reported studies, and it may be associated with the chemical nature of the matrix cell debris or with changes in the matrix associated with cell death. Other explanations are of course possible as the authors intimate.

Diabetes mellitus, joint mobility, and youth p 236
There have been many reports associating insulin dependent diabetes and decreased joint mobility, and this report suggests that the incidence of this abnormality varies depending upon the method of assessment used. Limitation of joint mobility shows a trend towards increasing with age and duration of the diabetes but seems to have little relation to diabetic control and indeed may be seen in patients with recent onset of their diabetes. There is clearly a need for standardisation of the assessment methods and the significance of the finding is not yet understood.

Mouse interleukin 1 (IL1), joint inflammation, and patellar cartilage p 238
When mouse recombinant interleukin 1 was injected into mouse knees it had a definite action within 24 hours with a pronounced effect on proteoglycan breakdown; this quickly returned to normal after one injection, though proteoglycan synthesis took a little longer to recover. The degree of inflammation induced by one injection of IL1 was minimal, but repeated injections caused much more severe change. Interleukin 1 clearly has a profound effect on proteoglycan loss and inhibition of its synthesis.

Systemic lupus erythematosus (SLE), porphyria, and antinuclear antibodies p 246
A possible association between SLE and porphyria has been postulated many times and a common genetic predisposition to both has been looked for but never found. Both conditions share many clinical findings and positive antinuclear antibodies may be seen in either of them (over 50% in porphyria in this study). The presence of these antibodies does not necessarily imply the diagnosis of SLE, however, and the application of careful diagnostic criteria enables the two to be distinguished. Their association appears to be a chance finding.
SLE, neuropsychiatric disease, and lymphocytotoxic antibodies  p 249
Many types of circulating autoantibodies have been described in SLE and the association between neuropsychiatric disease and lymphocytotoxic antibodies in particular has been described. This paper re-evaluates this association and indicates that although the antibodies were only marginally more prevalent in patients with neuropsychiatric disease, they were significantly increased in those with cognitive impairment. There was no association seen though between their presence and other organ involvement or with disease activity, implying a specific association with this cognitive dysfunction.

CASE REPORTS
SLE and toxoplasmosis  p 254
These two conditions share many symptoms and signs and may at first be hard to distinguish. Just to make matters worse patients with SLE have antibodies to toxoplasmosis much more commonly than controls, presumably because they are immunosuppressed and more susceptible to infections. This report highlights some of the diagnostic problems involved.

Septic arthritis from Pseudomonas cepacia  p 258
This organism is normally regarded as having low pathogenicity in man but it is now increasingly recognised that it nevertheless may be responsible for septic arthritis. Furthermore, it may be difficult to get rid of owing to its antibiotic resistance. In this example the infection followed intra-articular injection of a corticosteroid, and this has been implicated in a similar report before.

NOW AND THEN
The Arthritis and Rheumatism Council Epidemiology Research Unit  p 260
The Arthritis and Rheumatism Council’s Epidemiology Research Unit and its predecessor has been in existence since 1956. The opportunity has been taken on the retirement of Professor Philip Wood to review its history and achievements coupled with the distinguished career of its late director. First with John Lawrence, next with Philip Wood, and now with Alan Silman the unit has gone from strength to strength and has had a profound effect in bringing the skills and importance of epidemiology to the attention of rheumatologists. Each director has had his own strengths: all have been important.

REVIEW
Acquired chondronecrosis  p 262
Many environmental factors have now been implicated or guessed at as having a necrotic effect on cartilage, and Kashi-Beck disease is the prototype for this as Professor Sokoloff points out. Study of these factors may give us some clues to help us understand how the chondrocyte may be adversely affected early in its life. The effects of the drugs we give to our patients may be better understood if we have a clearer knowledge of how chondrocyte metabolism may be affected by external agents.

VIEWPOINT
Pulse corticosteroid therapy and rheumatoid arthritis  p 265
The way we use corticosteroids in RA has been profoundly modified since the first uncritical enthusiasm with which they were at first applied. Our indications for their use are now much more restricted, though we cannot as yet do without them. Pulse therapy has been in use for 10 years or so with good effect on selected occasions, though it has clearly recognised dangers. Is its benefit unproved as some would maintain? The authors of this viewpoint article would contest this.

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