suspected the organism may be identified more rapidly by analysis of the usually foul smelling pus by GLC.

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Calciferol levels in RA and OA

SIR, We were interested to read the report of Bird *et al.* demonstrating no significant difference in 1, 25-dihydroxycholecalciferol levels ($1, 25(\text{OH})_2\text{D}_3$) in a group of patients with rheumatoid arthritis (RA) compared with a similar group with osteoarthritis (OA).¹

We have demonstrated a general increase in bone turnover as measured by whole body retention (WBR)² of ^{99m} technetium methylene diphosphonate in rheumatoid patients compared with matched controls.³ Serum levels of vitamin D metabolites including $1, 25(\text{OH})_2\text{D}_3$ were normal in 8 patients whose WBR was elevated. This lends additional support to the concept of a generalised as well as a periarthral abnormality of bone turnover in RA which occurs as part of the inflammatory disease process.

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Streptococci and reactive arthritis

SIR, We were interested to read the case report of Hubbard and Hughes.¹ There are some similarities between their patient and the one we described in 1980.² A 43-year-old salesman developed, 3 weeks after a throat infection due to beta-haemolytic streptococci, predominant lower limbs arthritis, low back pain, and severe talalgia.

In the presence of subcutaneous nodules, pericardial effusion, and a previous history of rheumatic fever at the age of 19 an acute attack of rheumatic fever was diagnosed. The ASO titre was 1600 U/ml. In addition to these classical signs of rheumatic fever the patient sustained not only sacroiliac pain for 2 weeks but also a painful swelling of the right big toe and severe bilateral talalgia lasting more than 1 year, which evolved to calcaneal erosions. There were no yersinia agglutinins, and HLA B27 was positive.

It is interesting to note that both rheumatic fever and a reactive arthritis (considered as an incomplete form of Reiter's syndrome in our report) followed a streptococcal throat infection in this patient. The association of both conditions could be fortuitous. However, these data suggest, as in the case reported by our colleagues, that streptococci might be considered as one of the infectious agents capable of precipitating a reactive arthritis in an HLA B27-positive individual.

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Histocompatibility antigens in patients with ectopic ossification due to fibrodysplasia ossificans progressiva

SIR, Sharpiro *et al.*¹ have suggested that HLA B27 might have the effect of increasing liability to new bone formation. The basis for this was a claimed association between B27 and ankylosing hyperostosis, but, although the evidence is that there is no such association, we have taken the opportunity to study the HLA antigen frequencies in 23 patients from the UK with fibrodysplasia ossificans

Table 1 Blood groups and HLA tissue types in 23 patients with fibrodysplasia ossificans progressiva

No.	Sex	Year of Birth	ABO group	Rh type	HLA A locus	HLA B locus
1	F	1955	A	Pos	3, 11	7, 18
2	F	1952	A	Pos	2, 29	12
3	F	1920	A	Pos	2, 3	12, 27
4	F	1922	B	Pos	2, 3	7, 15
5	M	1949	O	Pos	2, 29	8, 12
6	M	1965	O	Pos	3, 10	7, 12
7	M	1960	O	Pos	11, 29	12, (21)
8	M	1972	ND	ND	1, 11	8, W6
9	M	1963	O	Pos	1, 9	8, 17
10	M	1953	A	Pos	1, 3	8, 12
11	F	1951	AB	Pos	1, 28	8, 12
12	F	1947	A	Pos	1	5, 8
13	F	1950	A	Pos	1	8
14	M	1940	A	Pos	1, 2	7, 17
15	M	1952	A	Pos	1, 10	8
16	F	1947	O	Pos	28, 32	14
17	M	1960	O	Pos	28	17, 37
18	M	1949	O	Pos	2, 29	12, 27
19	F	1910	A	Pos	1, 28	8, 15
20	M	1971	A	Pos	2, 3	18, 21
21	M	1965	AB	Pos	1, 2	8, 12
22	F	1944	A	Neg	3, 32	14
23	F	1956	O	Pos	2, 10	27

progressiva (FOP, synmyositis ossificans progressiva), a rare autosomal dominant disorder in which there is progressive ossification in the soft tissues. The clinical features of these patients have been described elsewhere.² The HLA, ABO, and Rh phenotypes are given in Table 1.

Three patients were B27-positive (13.0%) compared with 50 of 550 controls (9.1%). None of the other HLA antigens were significantly different from the controls. Furthermore, the severity of ectopic ossification was not associated with any HLA antigen.

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Book reviews

Articular Synovium: Anatomy, Physiology, Pathology, Pharmacology and Therapy. International Symposium on Articular Synovium, Bruges, October 1981. Ed. P. Franchimont. Pp.184. SFr.114. S. Karger: Basel.1982.

I have read this little book with interest and profit. The text comprises the substance of 15 papers given at a symposium on synovial tissue held in October 1981 under the combined auspices of Cilag AG and McNeil Pharmaceutical.

The reason why this book excites interest is that the majority of the authors have taken the opportunity to give individual accounts of their views and often of their techniques. This is not so with chapter 1 (Bassleer *et al.*), which is a long, conventional, and rather dated survey of synovial histology. Chapter 2 (Faure *et al.*) by contrast is a clear brief description of the analysis of rheumatoid (RA) synovial cell populations by monoclonal antibodies. Swann (chapter 4) fully explains *lubricin* (lubricating glycoprotein-I), and Chayen and Bitensky (chapter 5) give one of their best short accounts of normal and abnormal synovial cellular biochemistry. Russell and his colleagues (chapter 6) tell us of their work on the regulation of synovial proteinases and give a valuable short review of the background to this work. E. D. Harris (chapter 8) offers a philosophy of joint destruction in RA, though many may prefer his compelling chapter

in Kelley's textbook; and Gysen *et al.* (chapter 10) taken us into the relatively unexplored field of synovial fluid N-terminal procollagen type III peptide in relation to synovitis and tissue injury.

This is an excellent companion for a long railway journey and a valuable guide to the thoughts of a number of experts in the experimental study of synovial joint disease.

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People With Arthritis Need Help and Support. A report on services available for rheumatic sufferers. By P. H. N. Wood and E. M. Bradley. Pp. 20. 50p. ARC: London, 1981.

This pamphlet, from the ARC Rheumatology Research Unit, is the third in the series with which rheumatologists will be familiar. As such it provides a very useful compendium of facts for the rheumatologist and for those interested in the delivery of health care. The style and format will be familiar to those who have read Dr Wood's previous publications and particularly interesting for those who understand the ICD Rubrics.

The statistics are of course dependent on the Department of Health's statistics and SH3 returns, and those of us who