Treatment with prednisolone 80 mg per day and indomethacin 100 mg per day was begun. Within three weeks the patient had defervesced. Lymph nodes diminished in size and synovitis resolved. After six months of treatment the patient was asymptomatic.

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Bacterial confirmation of gonococcal arthritis

SIR, The case report 'Moraxella infectious arthritis: first report in an adult' by Rosenbaum *et al.*¹ describes the unexpected culture of a species of *Moraxella* from the synovial fluid of a 42-year-old woman who was thought on clinical grounds to be suffering from gonococcal septic arthritis. However, the criteria used for identification were not sufficiently stringent to exclude the possibilities that the organism might in fact have been a strain of *Neisseria gonorrhoeae* or of *Branhamella catarrhalis*.

The isolate was described repeatedly as being a Gram-negative diplococcus. Lautrop² notes that the short, plump Gram-negative rods of *Moraxella* may approach a coccoid form, but stresses nevertheless that the rod-like shape of the genus is an important characteristic distinguishing it from the Gram-negative cocci within the family *Neisseriaceae*. The apparent absence of any rod-like forms in Gram-stained smears of the isolate is indicative of *Neisseria* or *Branhamella*, not *Moraxella*.

The isolate was reported as not utilising any sugars. We assume that the sugars tested were incorporated into the cysteine (*sic*) trypticase agar mentioned in the report, and that they included glucose, maltose, lactose, and sucrose. *B. catarrhalis* does not utilise sugars, and further tests may have indicated that the isolate belonged to this species. However, a more probable explanation is that the isolate was a strain of *N. gonorrhoeae* that failed to utilise glucose in cystine trypticase agar, similar to strains described by White and Kellogg.³

Perhaps the most disturbing aspect of the bacteriological investigations was the use of the API 20E system, designed primarily for the identification of members of the *Enterobacteriaceae*, for the characterisation of a fastidious Gram-negative coccus. In our laboratory known strains of *N. gonorrhoeae* (including a WHO reference strain) were identified as *Moraxella* sp. by this system. Other procedures for identification of *N. gonorrhoeae*, such as the use of specific fluorescent antibody or coagglutination tests, were apparently not carried out on the isolate.

In their introductory paragraph Rosenbaum *et al.* note the similarities between *Neisseria gonococcus* (*sic*) and *Moraxella* species, and they conclude their report with a comment on the need for bacterial culture to confirm a clinical diagnosis of suspected gonococcal arthritis. We emphasise that the identification procedures must include methods that are appropriate to the class of organism suspected, and that, if any doubt exists, confirmation of identity by a suitable reference laboratory should be obtained.

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Immune deposits at the dermoepidermal junction in patients with rheumatoid arthritis

SIR, The deposition of immunoglobulin and complement components at the dermoepidermal junction in normal skin is well described in systemic lupus erythematosus (SLE).¹ Studies in rheumatoid arthritis (RA) have yielded conflicting results with a frequency of 0-50% reported.²⁻⁶ casting doubt on the diagnostic specificity of the 'lupus band' test. Reasons for such variation are not clear from inspection of the studies.

We have determined the prevalence of immune deposits at the dermal junction in 45 patients with RA. The study was designed to establish factors which might influence the development of deposits. Thus skin was sampled from the forearm of all patients, and from 34 an additional biopsy was taken from the leg to determine regional variation. Patients were studied as a group and by subdivision into those with articular disease alone and those with extra-articular manifestations. The influence of serological factors and of drug therapy was also examined. For comparison biopsy specimens were also taken from the arm and the leg of 14 patients with SLE and a miscellaneous group of 22 control subjects and patients with other rheumatological disorders. Sections of skin were processed for routine histological examination and by a direct immunofluorescent technique using rabbit antisera to human IgG, IgM, IgA,

C3, C1Q, and fibrin obtained from Behring Diagnostics Ltd. Blood was withdrawn for estimation of ESR, rheumatoid factor, and antinuclear factor at the time of biopsy. The results are summarised in Table 1.

Six patients with rheumatoid arthritis had continuous granular deposits of IgM at the dermal junction and all were seropositive for antinuclear factor in a titre $>1/128$. The deposits were indistinguishable from those present in some patients with SLE. There was no relationship with extra-articular disease and no regional variation. At the time of the study all patients were receiving a nonsteroidal anti-inflammatory drug and 13 D-penicillamine. We found no evidence of a general association between dermal immune deposits and therapy with D-penicillamine, and there were no deposits in four patients who developed significant proteinuria. However, deposits of IgM were detected in one patient who may have developed a drug induced lupus syndrome similar to that described by Kirby *et al.*⁷ This patient with classical RA had received treatment with D-penicillamine for four years. Initially strongly seropositive for rheumatoid factor, she had lost the rheumatoid factor and developed antinuclear antibody (titre $\geq 1/640$). DNA binding was negative. She had no nephropathy or skin rash. Her main complaint was of increased joint pain. There was no joint swelling and the rheumatoid disease appeared inactive. Cessation of treatment with D-penicillamine was associated with loss of arthralgia and reappearance of rheumatoid inflammation. Lack of diagnostic specificity of IgM was emphasised by its presence at the dermal junction of a patient with chronic active hepatitis. In our experience, therefore, the presence of IgM alone at the dermal junction has limited diagnostic significance and will not distinguish SLE from RA. On the other hand the presence of combined deposits of immunoglobulin and complement seems to be

specific for SLE, confirming the diagnostic role of the 'lupus band' test.⁸

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Table 1 Immunoglobulin and complements deposits at dermal junction in patients with RA, SLE and other connective tissue disorders

Disease	Number	ANA-positive	Number with immune deposits	Type of deposit
Systemic lupus erythematosus	14	14	10	3 IgG+C3 2 IgM+C3 4 IgM 1 IgG 4 negative
Rheumatoid arthritis	45	17	6	IgM
Systemic sclerosis	5	4	0	
Chronic active hepatitis	2	2	1	IgM
Normal (6) Degenerative (6) Polymyalgia (2) Polyarteritis (1)	15	0	0	0

HLA types and palindromic rheumatism

SIR, We read with interest the report by A. Pines *et al.*¹ of the association of HLA types with palindromic rheumatism. However, we think that their claim, 'this study, the first to display the HLA antigens of an entire family that suffered from benign PR', is not quite accurate. We reported² the association of HLA types with disease considered to be palindromic rheumatism by clinical and other criteria at the XVth International Congress of Rheumatology in Paris in June 1981. As shown in Fig. 1 the affected members of the family described by us had

Fig. 1 HLA typing of family.