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 Blood was withdrawn for estimation of ESR, rheumatoid factor, and antinuclear factor at the time of biopsy.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 ≥ 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]

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

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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 reported[2] 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

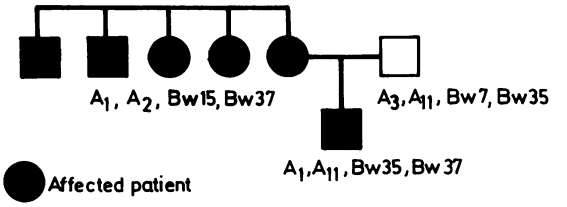

Fig. 1 *HLA typing of family.*

HLA-A, Bw37 which appears to be different from that found in the family described by Pines *et al.*

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Spontaneous rupture of the spleen in RA

SIR, We read with interest the article by Haskard *et al.*[1] describing two cases of rheumatoid arthritis (RA) in which spontaneous rupture of the spleen occurred. We should like to report two additional cases: one of which was classical RA without splenomegaly or Felty's syndrome, the second presenting a somewhat unusual Felty's syndrome.

The first case was that of a 63-year-old man with an 11 year history of classical seropositive RA. Apart from subcutaneous nodes no other extra-articular manifestations were observed, nor was there evidence of vasculitis, splenomegaly, or Felty's syndrome. In January 1983 he complained of sudden pain in the left hypocondrium and developed tachycardia and falling blood pressure. At laparotomy a splenic subcapsular haematoma with rupture into the peritoneum was found. He denied any history of precipitating factors. Splenectomy was performed and the patient made a good recovery. Pathological examination of the spleen showed no significant abnormality, nor was there any evidence of granulomas, vasculitis, or capsular fibrosis.

Our second case was a 33-year-old women referred to us in September 1981 because of massive splenomegaly and pancytopenia. Haematocrit was 32%, white cell count 1×10^9/l, and platelets 73×10^9/l. She gave a one-year history of assymetrical and erratic articular pain in shoulders and knees, without tenderness, morning stiffness, or subcutaneous nodules. Three latex tests proved negative, an ANA test was weakly positive (1:80) with homogeneous staining, and fluorescent antigranulocyte antibodies were positive at low titre (1:20). Other serological tests were negative. A ^{99m}Tc uptake scan, upper gastrointestinal *x*-ray, ultrasound examination, coeliac angiography, and bone marrow biopsy were non-contributory. Four weeks after admission she complained of sudden abdominal pain, and hypovolaemic shock followed. At laparotomy a splenic rupture was found. Pathological examination showed a spleen 30 cm in diameter and 2950 g in weight. The splenic pulp showed no significant abnormality. Following splenectomy, granulocyte and platelet counts returned to normal and to date have continued stable. Two months later a clinical picture of RA evolved with positive rheumatoid factor (1:1280) and aggressive articular involvement two years after follow-up.

In the first of these two cases splenic rupture appeared as a belated complication in a classical seropositive RA of 11 years' standing. We were unable to find the splenic capsular involvement described by Haskard *et al.*,[1] although this was specifically looked for. This therefore appeared to be a spontaneous rupture. The second case commenced as a massive splenomegaly and pancytopenia. Felty's syndrome appeared unlikely because neutropenia and splenomegaly very rarely precede the stage of joint involvement,[2] which generally tends to be severe.[3] Splenic size in Felty's syndrome is moderate in 90% of cases, with an average weight of 710 g.[3] The degree of splenomegaly in this case exceeded previously described limits and appeared to be the cause of the spontaneous rupture. This occurrence in Felty's syndrome, which is not uncommon in other types of massive splenomegaly, has only once been reported.[4] On the evidence of this case we believe that Felty's syndrome should be considered in the differential diagnosis of pancytopenia and splenomegaly with spontaneous rupture even in the absence of rheumatoid factor and articular disease.

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