ment as part of the overall care of patients with arthritis. This research was supported by a grant from the ARC (no. P0503).

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Co-existent Coeliac Disease and Scleroderma

SIR—In their case report, Marguerie et al. [1] stated that they had found only three other reports of patients with co-existent coeliac disease and scleroderma. They overlooked a case which we reported in this journal in 1993 [2] of a young woman with multiple autoimmune diseases, including well-documented coeliac disease and systemic sclerosis. As in Marguerie’s two patients, it was assumed that her malabsorption was due to sclerodermatous small bowel involvement, until a biopsy unexpectedly revealed the typical subtotal villous atrophy of coeliac disease, which responded to gluten exclusion. She was also found to have chronic pancreatic exocrine insufficiency which was contributing to her malabsorption and this was treated with pancrease.

Since malabsorption can give rise to unpleasant and embarrassing symptoms, as well as being associated with a poor prognosis in scleroderma, every effort should be made to identify treatable causes. In addition to aspiration and biopsy of the small bowel, investigation should include estimation of the serum trypsin and a pancreolauryl test to check pancreatic exocrine function.

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Massive Pericardial Effusion in Scleroderma

SIR—We read the recent paper on the above topic [1] with great interest. However, the prognosis may not be as poor as described. We would like to report the case of a patient with scleroderma, alive and at present reasonably well, almost 11 yr after presentation with cardiac tamponade.

A 33-yr-old secretary presented on 26 December 1984 with a 4 day history of palpitations, shortness of breath and gripping chest pain. At the beginning of December, she had had a 2 week course of prednisolone 20 mg daily, from her general practitioner, for painful swollen joints in her hands. She had first reported arthralgia in July 1984, and had been seen at that time by both of us and no abnormality was found on clinical examination or ESR. Autoantibody screen (ANF, RhF, DNA binding) was negative. ENA screen was not performed at this stage.

On admission on 26 December 1984, she had the clinical features of diffuse scleroderma affecting her hands, arms, face, legs and trunk. She had cardiac...
tamponade clinically and confirmed by echo-cardiogram. Her clinical condition was poor and she required pericardial aspiration; 300 ml straw-coloured fluid, initially under high pressure, were removed. Microscopy showed scanty leucocytes and biochemistry a protein content of 13 g/l.

After aspiration, she had no respiratory or oesophageal symptoms, and barium swallow showed normal peristalsis and a small sliding hiatus hernia. At the time of admission in December 1984, ANF, ENA screen and RhF were negative, and ESR was 2 mm/h.

She was treated with prednisolone 60 mg daily, reducing to 10 mg daily over the next month, and has remained on prednisolone 2.5-10 mg daily since. Penicillamine 250 mg daily was also started, and continued long-term. She had no clinical recurrence of her pericardial effusion, although echocardiography detected a small anterior effusion in January 1986. At this time, she complained of dysphagia of 2 months' duration. Barium swallow showed a slow primary stripping wave which faded away in the lower oesophagus. There was no reflux or dilatation. Pulmonary function tests performed for the first time showed a pure restrictive defect with a degree of alveolar capillary block. She had developed calcinosis at the wrist. Repeat autoantibody studies showed ANF positive for the first time (titre 1:50 speckled), with Sm, RNP, Ro, La and ScL70 antibodies negative. ESR was 4 mm/h.

Between January and April 1986, she received plasmapheresis five times in the hope of improving her skin disease and dysphagia. There was some subjective and objective improvement. She reported severe Raynaud's phenomenon in 1988, but was otherwise reasonably well until an episode of severe hypertension [blood pressure (BP) 240/120] in July 1992, despite pre-treatment with enalapril 5 mg daily. This responded rapidly to an increase in enalapril and the addition of nifedipine, and she remained well and free of arthritic symptoms, which returned 1 week after discharge on warfarin.

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As we are not involved in the treatment of rheumatoid arthritis, we felt that we should offer the idea for consideration by rheumatologists as a safer alternative to the use of immunosuppressive agents. Our experience with ulcerative colitis suggests that heparin is more effective when combined with sulphasalazine, but the use of non-steroidal anti-inflammatory agents with heparin is obviously contraindicated.

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