ment as part of the overall care of patients with arthritis.

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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 pancreas.

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