faeces were collected at home and returned direct to our laboratory by post.

*Klebsiella pneumoniae* was cultured from the faeces of 8 (33·3%) of the patients and 8 (30·8%) of the controls. This difference is not significant. When the results were analysed with regard to our classification of activity of their spondylitis, we found that 2 (33·3%), 5 (33·3%), and 1 (33·3%) of patients with definitely active, probably active, and inactive spondylitis respectively carried *Klebsiella* in their gastrointestinal tract. There are no differences between these three groups. 2 (25·0%) and 6 (37·5%) of the spondylitic patients with and without gastrointestinal *Klebsiella* respectively had inflammatory disease of a peripheral joint at the time of study. No patient had active anterior uveitis.

Two (25·0%) of the patients with ankylosing spondylitis and gastrointestinal carriage of *Klebsiella* had been in hospital within the preceding 7 months. This compares with 1 (6·3%) of patients who did not have gastrointestinal *Klebsiella* and with 4 (50·0%) and 8 (44·4%) of the controls with and without gastrointestinal *Klebsiella* respectively. The recent antibiotic usage in patients and controls with and without gastrointestinal *Klebsiella* was similar.

These results do not show the high gastrointestinal carriage rate of *Klebsiella* in patients with clinically active ankylosing spondylitis reported by Ebringer et al. None of our patients had active anterior uveitis. This could be the explanation for the difference between the two studies since the presence of active anterior uveitis was one of their criteria of active disease. It may be that *Klebsiella pneumoniae* is an aetiologial factor in exacerbations of active anterior uveitis, eradication of these microorganisms being followed by the complete remissions observed. Presence of active anterior uveitis, however, does not imply activity of ankylosing spondylitis since the ocular disease may occur in the absence of clinical activity of the spinal disease. In addition, ankylosing spondylitis is a chronic disease in which radiological progression may occur in the absence of severe symptoms. This suggests that episodic infection associated with clinical activity is unlikely to be an aetiologial factor.

Our results suggest caution in attributing intestinal carriage of *Klebsiella pneumoniae* to be an aetiologial factor in ankylosing spondylitis itself.

C. J. EASTMOND
Department of Rheumatology

E. M. COOKE
Department of Bacteriology

and V. WRIGHT
Department of Rheumatology
University of Leeds
Leeds LS2 9PJ

Thrombocytosis in rheumatoid arthritis

Sir,

I read with great interest the case report by Ehrenfeld et al. (Annals, 1977, 36, 579). The authors clearly show thrombocytosis and thrombocytaphaemia associated with repeated thrombosis. This is also seen in some patients with myeloproliferative disorders like polymythaemia vera. We recently managed a patient with primary polycythaemia who in spite of adequate anticoagulation developed cerebral thrombosis, and platelet functions showed marked exaggeration in aggregation. The patient was given dipyridamole (Persantin) 400 mg/day, which altered the platelet function. Unfortunately, the original cerebral thrombosis produced extensive brain stem lesions and hemiplegia.

In the case reported by Ehrenfeld et al., dipyridamole 225 mg was given. This may have been inadequate, as we found that a minimum blood level of 3·5 mmol/l was necessary to produce the desired antiplatelet effect (Rajah et al., 1977). If there are no facilities for estimating dipyridamole blood levels it may be necessary to give >300 mg/day and correlate with platelet function. Alternatively, especially in the case reported, a combination of dipyridamole and aspirin might have been effective. We have found an additive effect of these two drugs (unpublished data). Thus, 330 mg aspirin per day had been added to the 225 mg dipyridamole, the platelet function would have been altered and possibly the thrombosis prevented. This would of course be additional to the treatment of the primary disease with azathioprine and steroids, etc.

S. M. RAJAH
Department of Haematology,
Seacroft Hospital,
Leeds LS14 6UH.

Reference


Bone pains in chronic myelomonocytic leukaemia, treated by cytosine arabinoside

Sir,

A patient with chronic myelomonocytic leukaemia with severe generalised bone pains was reported by Douer et al. (Annals, 1977, 36, 192). Analgesics, anti-inflammatory agents, and corticosteroids were of no avail, but a dramatic relief of pain was achieved on three similar

Reference