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 aetiological factor in exacerbations of acute 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 aetiological factor.

Our results suggest caution in attributing intestinal carriage of *Klebsiella pneumoniae* to be an aetiologic 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

Reference


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 thrombocytopenia associated with repeated thrombosis. This is also seen in some patients with myeloproliferative disorders like polycythaemia 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...
consecutive occasions by 5-day courses of intravenously administered cytosine arabinoside 100 mg/day

This patient was followed by us for a further period of 22 months. During that time, while the haematological picture remained unchanged, he suffered from eleven episodes of excruciating bone pain, always responsive to the same treatment. Repeated skeletal x-rays and bone scans showed no osteolytic lesions. In December 1977 a fatal blastic crisis occurred. Permission for necropsy was not granted.

These data provide further evidence of the beneficial effect of cytosine arabinoside in the symptomatic treatment of patients with this condition.

J. Asherov, Y. Shoensfeld, and J. Pinkhas
Department of Medicine 'D', Beilinson Medical Center, Petah Tikva, Israel.

Book reviews


This revised edition makes its appearance only 2 years after the printing of the second edition. The editor in his preface explains that the decision to revise the text rather than merely reprinting was taken so that the book could be updated by integrating more than 100 key references representing significant advances or new review articles which had been published up to July 1976. This has been achieved by inserting appropriate sentences or paragraphs in the supplementary chapters which were added for the second edition. The overall layout of book, in terms of text, tables, and illustrations has not therefore significantly altered.

This book remains a tour de force; all aspects of SLE are dealt with, covering its history, laboratory animal models, pathology, current immunological status, and clinical aspects of the disease. Each chapter has been written by acknowledged experts within this field and the total list of references exceeds 3000. Certain statements, such as the LE cell test is the mainstay for the diagnosis of SLE, would not now be accepted by all rheumatologists, and some imbalance has occurred by retaining unaltered the original chapters of the 1965 edition, such as that on biological false-positive tests for syphilis, particularly as the Wassermann reaction is now rarely used in routine serological testing.

The book would become more readable if the original chapters and the supplementary chapters added for the second edition were integrated, but it remains an essential reference work for any clinician or laboratory worker interested in this field. Those already possessing the 1974 second edition need not feel compelled to purchase the revised version because of the relatively minor additions, but the editor must be congratulated for attempting to keep this textbook as up to date as possible.

IAN GRIFFITHS


This fairly small volume provides a concise and highly readable account of this complex group of diseases. It combines a clear and comprehensive review of their clinical manifestations with discussion of possible pathogenetic mechanisms. Systemic lupus erythematosus, as might be expected, receives the greatest attention in view of the clinical diversity of this condition, the widespread immunological abnormalities, and the findings in animal models. The possibility of a viral aetiology and the propensity of patients with inherited complement deficiencies to develop systemic lupus erythematosus are both important discussion points. A similar format covering epidemiology, pathology, and clinical findings is used in most subsequent sections to deal with the spectrum of diseases from rheumatoid arthritis and polymyositis to the various vasculitides. The author points out that a detailed description of rheumatoid arthritis is beyond the scope of this volume. The text is complemented by the liberal use of tables to summarise the main points of discussion. Short case histories have been included frequently and in general these are very effective in illustrating clinical problems of diagnosis or management.

This book must be considered as essential reading for postgraduate students studying for higher medical degrees. However, the last two sections, which provide a brief outline of immune complex disease and immunological tests in the rheumatic diseases, might, for such readers be better placed as introductory chapters, since they are closely connected with the discussion of aetiological factors. There are a number of minor printing errors, particularly in the first half of the book, but the comprehensive lists of up-to-date references at the end of each chapter further recommends this text as an important reference for all working in the field of the rheumatic diseases.

J. D. PIGGOTT


This book comprises a collection of 31 papers given at a conference commemorating the 25th anniversary of the