events at a young age (range 23–32 years), two having livedo reticularis, and all having raised IgG anticardiolipin antibodies (ACA).¹ One of these had cerebral haemorrhage and although she had suffered pre-eclampsia during an earlier pregnancy, was normotensive at the time of the cerebral event. In addition, hypertension would not account for her systemic illness and accompanying severe ulnar nerve palsy. I assume the patients referred to by Dr Baguley *et al*² had cerebral haemorrhage discounted radiologically.

Although we advocated no particular treatment either for the acute cerebral event or for prophylaxis, all four of our patients responded (i.e., systemic illness abated and cerebral status improved) after pulse methylprednisolone and cyclophosphamide. Immunosuppressive treatment is surely not irrational for 'antibody mediated' mechanisms. We have now had the opportunity of studying ACA levels longitudinally in these and other patients. ACA were raised in all four patients before their cerebral events. Falling levels may in fact herald these and other clinical events (e.g., pregnancy loss, renal crisis), though the effects of treatment versus antibody deposition are difficult to distinguish and need resolving.

I entirely support evaluation of the place of anticoagulation or antiplatelet agents, or both, in the prophylaxis of arterial and venous thrombosis in association with ACA, although clear guidelines will eventually be needed for when to start, how much to give, and for how long to treat what may be a young population at risk. Consideration should also be given to the risk of cerebral haemorrhage in those patients with prior cerebral events, and the additional benefit of immunosuppression for the acute crisis cannot be dismissed.

Royal National Hospital for
Rheumatic Diseases, Bath

N J McHUGH

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stress to the collagen fibrils without the need for any further linkage.^{2,3} The fibrils are oriented such that the swelling pressure of the tissue, which enables it to withstand applied compression, then places them under tension so that they provide the necessary reinforcement.^{3,4}

Department of Medical Biophysics,
University of Manchester,
Stopford Building,
Manchester M13 9PT

D W L HUKINS

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SIR, Dr Hukins' comments are most welcome and underline the importance of contributions from many disciplines in the attempt to elaborate hypotheses about joints. It would, however, be doing less than justice to colleagues at the meeting if we did not add that physicochemical aspects of the matrix were remarked upon by some; comments so diluted and deflected by a preponderance of biological and biochemical arguments that they did not register in a compressed report. We are compiling a dossier of propositions on which to draw in the formulation of extended hypotheses. It is our hope that not only will Dr Hukins let us have his, but also that knowledgeable colleagues will respond to his ideas. Meetings are highly stimulating, but continued application is required to generate and to garner ideas, especially the less familiar. We are appreciative of Dr Hukins' initiative in writing.

Department of Experimental Pathology,
Institute of Orthopaedics,
Stanmore, Middlesex HA7 4LP

ROBERT BROWN
PAUL BYERS

Adhesion in articular cartilage

SIR, A recent conference report, on the pathogenesis of osteoarthritis,¹ includes a discussion of 'the nature of the adhesion or glue' necessary to maintain the structural integrity of the network of collagen fibrils in articular cartilage. The implication of the report is that there must be some bridging molecules (or ions) which bind to specific sites on the surfaces of collagen fibrils if this tissue is to be mechanically stable. Calculations based on the theory of fibre reinforced composite materials, however, of which articular cartilage is a biological example, indicate that the viscosity of the proteoglycan gel and the shear strength of its interface with collagen are adequate to transfer tensile

Pulmonary hypertension in Sjögren's syndrome

SIR, The case report on pulmonary hypertension (PHT) in primary Sjögren's syndrome (SS) was of great interest.¹ We have previously reported its occurrence in a patient with 'secondary' SS.² Our patient, a woman with a multisystem illness going back several years, initially presented with nephritis, was diagnosed as having systemic lupus erythematosus (SLE), and long term steroid treatment was started. She presented again seven years later with the lupus inactive clinically and serologically. She