events at a young age (range 23–32 years), two having livedo reticularis, and all having raised IgG anticardiolipin antibodies (ACA). 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 ulcerative nerve palsy. I assume the patients referred to by Dr Baguley et al 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.

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References

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**Adhesion in articular cartilage**

Sir. A recent conference report, on the pathogenesis of osteoarthritis, 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 stress to the collagen fibrils without the need for any further linkage. 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.

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**References**

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.

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**Pulmonary hypertension in Sjögren’s syndrome**

Sir. The case report on pulmonary hypertension (PHT) in primary Sjögren’s syndrome (SS) was of great interest. We have previously reported its occurrence in a patient with ‘secondary’ SS. 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 started. She presented again seven years later with the lupus inactive clinically and serologically. She
had, however, features of SS accompanied by marked swelling of both parotid and submandibular glands and severe PHT later complicated by right heart failure. She died suddenly several months later. In addition to the references cited in the case report, we were able to identify a further patient with PHT and SS.

PHT in association with connective tissue disease occurs most commonly with progressive systemic sclerosis (particularly the CREST variant (calcinosis, Raynaud’s phenomenon, oesophageal dysmotility, sclerodactyly, telangiectasia)), in SLE, and in mixed connective tissue disease. In SLE it usually manifests as PHT indistinguishable from the ‘primary’ idiopathic type and is rarely of the thromboembolic variety. It is considerably rarer in association with other connective tissue diseases, such as rheumatoid arthritis, when it may be more frequently associated with pulmonary vasculitis. It is excessively rare in conditions such as dermatomyositis and discoid LE. Its occurrence in primary SS may perhaps provide clues to its pathogenesis. An interesting hypothesis concerning the relation between hypothyroidism and PHT has recently been postulated by Chin and Fisher. Several factors may contribute.

Clotting factors are typically raised in hypothyroidism and may contribute to a relatively hypercoagulable state, resulting in situ microvascular thrombosis. More importantly, norepinephrine, which may cause systemic and pulmonary vasoconstriction, is increased in hypothyroidism. This may result from a diminution in the number of alpha and beta receptors. Increases of pulmonary artery norepinephrine concentrations have also been found in patients with increased pulmonary venous and arterial pressures. Chin and Fisher found that third out of 25 of their patients with unexplained PHT had evidence of hypothyroidism. This prevalence of 12% is far higher than that of hypothyroidism in the general population, which ranges from 0·4 to 5·9%. Additionally, they found a further seven patients with connective tissue disorders and PHT who also showed evidence of hypothyroidism.

There is also an association between Raynaud’s phenomenon, common in patients with PHT, and hypothyroidism, as well as evidence, recently confirmed, of the efficacy of triiodothyronine in patients with Raynaud’s phenomenon, relieving vasospasm both subjectively and objectively.

The frequent occurrence of organ specific antibodies to constituents of thyroid as well as clinical Hashimoto’s thyroiditis in patients with SS leads one perhaps to speculate on a common link between these conditions.

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References
8 Christensen N J. Increased levels of plasma noradrenaline in hypothyroidism. J Clin Endocrinol Metab 1972; 35: 559-63.

Note
Volvo awards for low back pain research 1989

The Volvo Company of Göteborg, Sweden will again this year sponsor three prizes of US $8000 each. Original reports within the following three areas: clinical studies, biomechanical studies, other basic science areas, must reach the address below by 15 November 1988. Further information from Professor A Nachemson, Department of Orthopaedics, Sahlgren Hospital, S-413 45 Göteborg, Sweden.