second eye, and when a similar regimen was used to treat giant cell arteritis without ocular involvement, all patients were protected from developing ischaemic optic neuropathy.

Furthermore, giant cell arteritis can actually be precipitated by inadequate treatment of polymyalgia rheumatica.⁴ It is our standard practice to treat patients with giant cell arteritis with an initial dose of 4 mg of dexamethasone intravenously immediately, followed by 4 mg dexamethasone intramuscularly four times a day for two days, and at the same time to start treatment with 80 mg prednisolone a day orally, gradually reducing the dose of oral prednisolone as the symptoms resolve. We would wholeheartedly concur with the view expressed by Kyle, Cawston, and Hazleman in the follow paper⁵ that neither erythrocyte sedimentation rate nor C reactive protein concentration is a reliable monitor of the state of the disease, and we would base dose reductions on the clinical features and symptoms. It may well be that giant cell arteritis leading to ocular involvement represents a different spectrum of disease than that represented in their paper. We would recommend, however, that any potential visual involvement be treated aggressively.

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Sir: We agree that threatened visual loss should be treated aggressively. The results of our study indicate that a high initial dose of prednisolone is necessary in treating giant cell arteritis followed by a slow carefully monitored reduction to achieve the lowest dose consistent with controlled symptoms and avoidance of side effects. Four of the 20 patients on the high dose regimen relapsed on reduction and

required a higher dose of prednisolone. It was the rate of reduction that was too rapid rather than the initial dose of prednisolone that was too low. Seventy per cent of relapses occurred when the dose was reduced to 20 mg prednisolone a day in the first month or to 20 mg prednisolone a day or less in the second month.

Six of the 39 patients with polymyalgia rheumatica developed giant cell arteritis during the first two months of treatment, but provided that patients are monitored it should not be necessary to use high doses of prednisolone for all patients with polymyalgia rheumatica. Frequent and careful follow up is necessary.

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Synovial fluid fibronectin concentrations in chronic arthritis: influence of intra-articular steroids or oral non-steroidal anti-inflammatory drugs

Sir: Goebel and Storck showed that the intra-articular administration of the antioxidant enzyme oryctein significantly reduced the synovial fluid fibronectin concentration in patients with rheumatoid arthritis.¹ Do other treatments influence synovial fluid fibronectin concentrations?

Fibronectin concentrations are significantly increased in synovial fluid from patients with rheumatoid arthritis, but, characteristically, plasma fibronectin concentrations do not vary greatly.² In contrast, it has been shown that plasma concentrations correlate with disease activity in systemic lupus erythematosus.³ Fibronectin also provides a useful biochemical marker in experimental arthritis and concentrations reflect the inflammatory activity.⁴ As an extracellular product of mesenchymal cells, fibronectin is a marker for proliferation of synovial connective tissue, a process which may be central to the pathogenesis of chronic synovitis.^{5,6} An increase in fibronectin concentration in synovial fluid may reflect increased connective tissue proliferation; similarly, a decrease in fibronectin may be a response by local tissue to treatment.

To study the hypothesis further we measured fibronectin concentrations in synovial fluid before and after treatment of chronic arthritis of the knee with either oral non-steroidal anti-inflammatory drugs or intra-articular steroids. Sixteen patients with chronic arthritis of the knee were admitted to the study, which was

approved by the local ethical committee. Ten were treated with oral azapropazone (600 mg twice daily) and six with one intra-articular injection of triamcinolone (25-50 mg). Synovial fluid was aspirated from the affected knee joint of patients before treatment and four weeks later. The fibronectin concentration in the synovial fluid was measured by an enzyme linked immunosorbent assay (ELISA) method.⁷

Mean initial fibronectin concentrations in synovial fluid were 0.64 (SD 0.11) µg/ml for the azapropazone group and 0.55 (0.12) µg/ml for the steroid group. Neither drug had a significant effect on the synovial fluid fibronectin concentration measured four weeks later: azapropazone 0.59 (0.16) µg/ml; triamcinolone 0.55 (0.11) µg/ml.

Although our results were negative, they are important in relation to previous studies which showed a significant reduction in synovial fluid fibronectin concentrations after treatment.¹ Our data indicate that the short term treatment of arthritis by steroids or non-steroidal anti-inflammatory drugs does not cause significant changes in fibronectin concentrations in synovial fluid. Our results imply that local steroids and oral non-steroidal anti-inflammatory drugs do not affect synovial connective tissue proliferation and therefore have no influence on proliferative disease processes.

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