effective therapy with either gold or D-penicillamine. This does not, however, exclude the possibility that change in selective lymphocyte populations or in lymphocyte function may be occurring.

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SIR, There are important differences between the study of Ishigami *et al* and our report¹ which may explain the apparently discordant findings. Ishigami's study included 58 patients treated with gold over a 12 month period. Although we are not told, one assumes that the gold preparation and the dose schedule used were similar in both studies. In Ishigami's study gold therapy was 'considered successful in controlling or reducing disease activity by both clinical and laboratory criteria'. The only data offered in support of this claim, however, are changes in erythrocyte sedimentation rate (ESR) (Westergren?) and peripheral blood haemoglobin levels. If the pretherapy ESR values in the two studies are compared (mean (SD): 48 (27) Ishigami *et al*; 59 (25) Hanly and Bresnihan) it would appear that the latter group of patients had more active disease. Furthermore, the six month post-therapy ESR in our study (mean (SD): 24 (11)) compared with the 12 month post-therapy value in the study of Ishigami *et al* (mean (SD): 32 (21)) indicates a greater reduction in disease activity in the former group. This may be important as, in our study, the greatest fall in peripheral blood lymphocytes occurred with the initial and most marked reduction in disease activity. Therefore, such a change may not be apparent in patients whose pretherapy disease activity is less marked and who have a less dramatic reduction in disease activity with gold therapy.

As Ishigami's study is a retrospective review, presumably detailed assessments of articular disease activity were not available on all patients. Such data are essential to allow accurate measurement of the response to therapy. Some patients clearly had a fall in lymphocyte counts while receiving gold therapy and one wonders if these patients also had the greatest reduction in disease activity.

We have seen our initial findings reproduced in additional patients currently entered in a prospective study of gold therapy in rheumatoid arthritis (unpublished observations, Rooney and Bresnihan). Total circulating lymphocyte counts, however, may be too insensitive to detect changes in all such patients. As Ishigami *et al* point out the absence of a fall in the total lymphocyte count does not preclude changes in lymphocyte subpopulations and lymphocyte function, as recently reported by Hassan *et al*.²

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Serum lactic dehydrogenase as a marker of joint damage in rheumatoid arthritis

SIR, The finding that raised serum lactic dehydrogenase (LDH) levels were linked with progressive joint damage in patients with rheumatoid arthritis (RA) reported by Dawes and colleagues underlines the potential usefulness of biochemical markers of tissue turnover.¹

Vessel *et al* reported that synovial fluid from patients with RA showed the polymorph LDH isoenzyme pattern.² We have found raised (>2 SD) serum LDH in 22% of 32 outpatients with RA, and in 12 paired samples the mean (SD) ratio of synovial fluid:serum LDH was 4.3 (3.4). There was close correlation between synovial fluid leucocyte count and LDH level ($r=0.93$).

These data support the synovial fluid polymorph as the source of raised serum LDH and suggest that, like cytidine deaminase,³ LDH is released from dead and damaged cells into the synovial fluid and diffuses, via the lymphatics, from all inflamed joints to the blood. Serum levels reflect overall polymorph turnover as a measure of joint inflammation.

The finding that such a measure might predict the progression of joint damage offers hope to the clinician and may shed new light on the relation between aspects of inflammation and joint destruction.

Cytidine deaminase, with higher cell:serum gradient and lower serum background than LDH³ may offer even greater sensitivity.

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