(r_s=0·088; NS). There was, however, a significant negative correlation between change in haemoglobin concentration and change in the erythrocyte sedimentation rate (ESR) between the two occasions (r_s=0·534; $p<0·05$). There was no such correlation with C reactive protein (CRP), but there was a significant correlation between CRP changes and changes in serum ferritin levels in the patients as a whole (r_s=0·677; $p<0·05$).

This study therefore showed that any improvement in haemoglobin concentration in patients with rheumatoid arthritis who have a serum ferritin concentration of >15 µg/l is likely to be the result of the amelioration of the disease, in so far as that is reflected by the ESR. Iron therapy was not shown to have had any effect. It should be remembered, however, that any erythropoietic response to disease improvement will place extra demands upon the iron stores. If these are insufficient to meet the new requirements for increased haemoglobin synthesis, this response may be limited. In practice it might be reasonable to expect there to be stores equivalent to a serum ferritin concentration of 20 µg/l for every 10 g/l haemoglobin deficit.[2] Patients with iron stores less than this may in the future develop an iron imbalance, but simply having a serum ferritin of <15–60 µg/l cannot be considered an indication of the present need for iron therapy.

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SIR, Serum ferritin acts as an acute phase reactant which is increased in inflammatory diseases like rheumatoid arthritis. It is therefore inappropriate to use 15 µg/l as the lower normal limit in rheumatoid arthritis. We[1] and others[2–4] have previously shown that a lower limit of about 60 µg/l can discriminate between the absence and presence of stainable iron in bone marrow.

Of the anaemic patients in our study, an increase in haemoglobin concentration was found in 5/6 patients with serum ferritin below 15 µg/l, in 13/17 patients with serum ferritin between 15 and 60 µg/l, and in 6/12 patients with serum ferritin above 60 µg/l. If only an increase in haemoglobin concentration of more than 8 g/l is accepted, the trend is the same (4/6, 10/17, and 4/12 respectively).

If one accepts a decrease of some magnitude in ESR and CRP as an indicator of decreased disease activity, this could be shown in, respectively 1/3, 1/11, and 3/4 patients from the three groups who showed an increase in haemoglobin. If anything, this supports our conclusion, since three out of four patients in the last group (the 'false positives'), who showed an increased haemoglobin, probably did so because of decreased disease activity, whereas that was found in only one patient in each of the other two groups.

Serum ferritin rose in almost all patients given three months' treatment with iron. Since serum ferritin also acts as an acute phase reactant we are not surprised that serum ferritin levels did not reflect haemoglobin concentrations. The ultimate test would, of course, have been a bone marrow examination for iron. Short of this, it should be considered whether a decrease in serum transferrin can be taken as evidence of improved iron status. If this is accepted, it is of interest that serum transferrin decreased in 5/5 and 10/13 patients in the first two groups but in only 1/6 in the group with serum ferritin >60 µg/l.

We did not conclude that all patients with serum ferritin <60 µg/l should be treated with iron, but we suggest that one should look for iron deficiency in anaemic patients with rheumatoid arthritis when serum ferritin is <60 µg/l. This is in agreement with the study of others.[2–4] Serum ferritin <60 µg/l is a better indicator of iron deficiency than usual blood tests like mean cell volume, mean corpuscular haemoglobin concentration, serum iron, and serum transferrin.

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Observations on the effects of phenylbutazone

SIR, In a recent letter in the *Annals* Drs Moens and Moens described their 16 year experience in treating 15 patients suffering from severe osteoarthritis of the knee with intra-articular injections of phenylbutazone.[1] The investigators conclude with the statement that they were unable to find any previous report on this subject.

We reported our observations on the use and clinical effects of intra-articular phenylbutazone in the *Journal of Laboratory and Clinical Medicine*.[2] We administered 83 intra-articular injections of phenylbutazone in a series of 33 patients, including 18 with rheumatoid arthritis, 11 with osteoarthritis, and four with various allied conditions. A 20% solution of 1 g phenylbutazone with 1% lidocaine was employed. The average injection dose varied from 3 ml to 5 ml of the solution. The interval between injections ranged from one to six weeks, with a usual period of two

weeks. All patients were given intra-articular injections of saline or 1% procaine solution, or both, for control observations, before receiving phenylbutazone.

Of the 11 patients with osteoarthritis who received 34 intra-articular injections, one derived major improvement, seven derived slight to moderate improvement, and three failed to benefit. Of 18 patients with rheumatoid arthritis, given 45 intra-articular injections, seven obtained major benefit, seven slight to moderate improvement, and four failed to respond.

The only adverse effect was transient local discomfort described as a 'burning' sensation that occurred during and after the injection in approximately half of the patients.

Synovial fluid showed a significant decrease in the white blood cell count in five of 10 fluids examined one to two weeks after injection. When synovial fluid was removed shortly after the phenylbutazone injection, however, examination disclosed severe disintegration of the white cells, making a differential count impossible.

To summarise, although intra-articular injections of phenylbutazone may produce a beneficial response, the drug causes local irritation and offers no therapeutic advantage compared with corticosteroids.

Incidentally, if the authors had recommended a strict postinjection rest regimen, the use of this technique might have facilitated a beneficial response, especially in some of their patients reportedly unresponsive to conventional steroid joint injections.[3]

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Systemic lupus erythematosus presenting as polymyalgia rheumatica

SIR, I read with interest your recently published report of systemic lupus erythematosus presenting as polymyalgia rheumatica in the elderly,[1] in which three cases were described.

Three hundred and fifty six cases of polymyalgia rheumatica/giant cell arteritis have been recorded at this hospital since 1968, with long term follow up.

In 1973 a 76 year old Caucasian woman presented with a history of pain and stiffness of the shoulder and hip girdles developing over a four week period. She had also felt feverish and complained of general malaise. She had had a radical mastectomy for carcinoma of the breast two years previously, and had a longstanding ulcer over one shin. She had otherwise been in good health. Investigations showed a haemoglobin of 10·1 g/dl (101 g/l) and a markedly raised erythrocyte sedimentation rate (ESR) of 135 mm/h. Electrolytes, liver function tests, and creatine phosphokinase were all normal, and a temporal artery biopsy was negative. An initial diagnosis of polymyalgia rheumatica was therefore made, and she was started on prednisolone 10 mg daily. There was clinical improvement on this dose with an associated fall of the ESR to 41 mm/h. Autoimmune profile then showed a positive antinuclear antibody titre at 1/40 960 and a positive latex test and deoxyribonucleic acid (DNA) binding. She continued to require between 12·5 mg and 20 mg of prednisolone daily long term and on this dose there was also rapid healing of her leg ulcer.

In view of the markedly raised antinuclear antibodies (ANA) and raised DNA binding the diagnosis was changed to systemic lupus erythematosus (SLE), though she did not develop any other features of the disease; in particular at no time was there evidence of muscle disease. She died in 1976 of a myocardial infarction.

Of 356 cases of polymyalgia rheumatica/giant cell arteritis followed up over an 18 year period, this is our only recorded case of SLE. Weakly positive ANA titres of up to 1/40 have been recorded in a larger number but with normal DNA binding. Although we accept that SLE may have an atypical presentation in the elderly, it is clearly extremely uncommon for it to present with a polymyalgia-like syndrome.

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Small joint involvement: systematic roentgenographic study in rheumatoid arthritis

SIR, Halla, Fallahi, and Hardin reported in this journal their observations on radiological involvement of small joints in rheumatoid arthritis (RA).[1] They studied 200 consecutively hospitalised patients with definite or classical RA and concluded that radiological asymmetry was usual, unilateral involvement common, and absolute symmetry uncommon. We would like to report our findings in a similar group of patients.

Eighty three consecutive patients with definite or classical RA attending an outpatients clinic were studied. The disease duration was variable, and radiographs of the patients' hands were taken in the standard posteroanterior position.