Desferrioxamine, a metal chelating agent with high affinity for iron, has been found to suppress tissue injury in animal models of inflammation.8 Few studies have been performed in humans, and these have given conflicting results. Giordano et al showed an improvement of clinical conditions and a significant increase of serum iron and haemoglobin with a marked progressive decrease of serum ferritin in patients with RA with hyposideraemic anaemia in a short desferrioxamine treatment.4 Fudman et al by intrarticular administration of desferrioxamine noted predominantly systemic effects with decreased serum ferritin and decreased serum concentrations of lipid peroxidation products.15 However, in other treated patients with RA, with larger doses of desferrioxamine and found serious side effects consisting of loss of consciousness and of pigmentary retinopathy.11 Pall et al observed ocular toxicity after a low dose of desferrioxamine.12 Polson et al found no significant changes in rheumatological indices or in immunological markers of disease activity of patients with RA refractory to conventional treatment and receiving desferrioxamine for six months.13 Recent studies have shown that desferrioxamine affects lymphocyte function. It can inhibit proliferation of human lymphocytes, promote T lymphocytes reductase and DNA synthesis.14 It is found that desferrioxamine treatment impairs the expression of interleukin 2 binding receptors on lymphoid cells in response to mitogen and markedly reduces interleukin 2 production by mitogen stimulated cells.15

In this study we noted a statistically significant improvement of clinical indices in the patients with RA at the 28th day. Patients had no side effects. A notable increase of serum ferritin and microglobulin concentrations was noted in all patients at the 14th day followed by a statistically significant decrease at the 28th day in patients with more active disease. This increase in ferritin and microglobulin at the end of desferrioxamine administration (14th day) without a worsening in clinical indices has not been explained. It may be due to a polyclonal B lymphocyte reaction in response to iron ingestion determined by desferrioxamine displacement of iron or other metals. Polyclonal B lymphocyte activators are mitogens that non-specifically stimulate lymphocytes to secrete immunoglobulins, and both groups of patients had significantly increased concentrations of immunoglobulins after desferrioxamine treatment.

High serum ferritin concentrations seem to correlate with the severity of arthritis involvement. In this study patients with more active RA had higher serum ferritin concentrations than patients with less active disease. On the other hand, low serum ferritin concentrations may be connected with iron deficiency and in this study patients with RA and low serum ferritin concentrations had lower serum iron and higher transferrin and iron binding capacity than patients with higher serum ferritin.

In patients with severe active RA the association of raised concentrations of ferritin and β2 microglobulin suggests that conspicuous iron deposits may play a part in the stimulation of lymphoid cells. Desferrioxamine seems to reduce lymphocyte activation and function, probably by inhibiting DNA synthesis and interleukin 2 action in these patients. In patients with less active RA and low or normal serum ferritin concentrations the desferrioxamine effects do not seem to influence lymphocyte activity.

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Azathioprine and warts
Sir: In 1963 we began treatment of patients with rheumatoid arthritis with azathioprine,1 and we are still following up six patients who have been treated with this drug for more than 25 years. At the start we used a dose of 2-5 mg/kg daily and now we use 50 mg a day. Aspirin was added for pain relief when necessary.

The course of their disease has been quite satisfactory with a minimum of discomfort and they have led a fairly active life. The treatment has not prevented the appearance of classical bone deformities, however.

During the past two years, four of our six patients, whose ages range from 60 to 83 years, developed skin lesions on hands and feet, which were diagnosed as warts. To confirm this diagnosis one patient was operated. The anatomical diagnosis was 'hyperkeratotic seborrhoeic wart' (Professor La Chapelle, University of Louvain).

Hyperkeratotic warts are fairly common among old people. It is impossible to draw any conclusion from a group of only six patients but we wonder if the high incidence of warts among our azathoprine treated patients is related to their treatment.

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