LETTERS TO THE EDITOR

Pulsed methylprednisolone therapy in rheumatoid arthritis

Sir: In a recent issue of this journal Smith, Ahern, and Roberts-Thomson discussed the value of pulsed methylprednisolone therapy in rheumatoid arthritis. We agree with their view that pulsed methylprednisolone is a useful adjunct to other treatments in this disease.¹

We have also tried pulsed methylprednisolone therapy in severe ankylosing spondylitis. Ten subjects (eight men and two women, aged 45 years, mean disease duration 17.5 years) were selected for study. All were judged on clinical grounds to have active disease and had persistent daytime and nocturnal pain despite maximum tolerated doses of non-steroidal anti-inflammatory drugs.

A double blind, crossover design was used. A detailed clinical examination was performed in the outpatient clinic. This included a subjective assessment of disease activity by the patient (pain score and duration of morning stiffness), as well as measurement of spinal and cervical flexion, erythrocyte sedimentation rate, total serum and salivary IgA. To estimate salivary IgA an antibody directed against secretory component was used.

After this assessment all 10 patients were admitted to the regional rheumatology centre and subdivided into two treatment groups, A and B, using a random number code. Group A were given three 1 g pulses of methylprednisolone on alternate days during the first week of admission and in the subsequent two weeks received intensive physiotherapy. Group B received identical physiotherapy but were given placebo infusions (of normal saline). The physiotherapy was based on our standard regimen for ankylosing spondylitis and included chest, back, and neck exercises as well as hydrotherapy.

Regular outpatient assessments were made during the subsequent four months. All 10 patients were then readmitted for physiotherapy. This time group A received placebo infusions and group B, pulsed methylprednisolone.

As in rheumatoid arthritis we found that pulsed methylprednisolone was well tolerated. Only one patient withdrew from the trial (at his own request) because of adverse effects (palpitations and flushing). Another patient withdrew at 20 weeks for personal reasons. The remaining eight patients noticed only minor problems (headache—methylprednisolone 1 instance, placebo 3; nausea—methylprednisolone 2, placebo 1; odd taste—methylprednisolone 3) and completed the trial.

The absence of serious adverse effects in our small study and the low incidence (10 out of 480 patients with rheumatoid arthritis) reported by Smith *et al*¹ is reassuring. In contrast with our previous findings in rheumatoid arthritis,^{2,3} pulsed methylprednisolone therapy was disappointing in ankylosing spondylitis. Duration of morning stiffness diminished in seven out of eight instances with pulsed methylprednisolone therapy, and in only four out of 10 instances after placebo infusions. When visual analogue pain scores

before admission were compared with data obtained at the first outpatient visit two weeks after completion of hospital treatment we found a significant improvement after placebo infusions plus physiotherapy ($p < 0.01$, Wilcoxon paired rank sum test), but after pulsed methylprednisolone plus physiotherapy in five out of eight instances patients actually recorded a deterioration.

Adding pulsed methylprednisolone therapy to conventional treatment also failed to improve objective response to physiotherapy. Thus we were able to show significant improvements ($p < 0.05$) of similar magnitude in lateral lumbar flexion immediately after physiotherapy, irrespective of whether pulsed methylprednisolone or placebo had been given. Anterior lumbar and cervical flexion only improved significantly after placebo infusions. We were unable to detect a significant change in erythrocyte sedimentation rate after either treatment. Only the immunological data, serum and salivary IgA, showed greater change with pulsed methylprednisolone than with placebo.

Finally, in the 10 subjects with severe ankylosing spondylitis whom we studied the response to intensive physiotherapy was short lived. Anterior lumbar flexion was the only clinical measurement to remain significantly improved at four months after treatment in either group.

Our data indicate that pulsed methylprednisolone therapy is effective in severe longstanding ankylosing spondylitis, a finding which suggests that inflammatory processes are less important than other factors in determining pain and stiffness in such patients. Pulsed methylprednisolone might be considered for severe ankylosing spondylitis of recent onset where inflammation may play a major part, but in our view is inappropriate for other categories of patients with ankylosing spondylitis. Most patients can, of course, be managed satisfactorily with a combination of non-steroidal anti-inflammatory drugs and physiotherapy.

Like others,⁴ we have shown that intensive courses of inpatient physiotherapy can produce measurable improvements in spinal mobility, even in those with longstanding disease. We found the duration of response to be short, however. Alternative treatment regimens, such as a daily home exercise programme reinforced by regular outpatient physiotherapy, may achieve better long term results and should be evaluated in such patients.

F EVARD
V NEUMANN
R HOPKINS
V WRIGHT

Rheumatology and Rehabilitation Research Unit
University Department of Medicine
36 Clarendon Road
Leeds, LS2 9

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Brown's syndrome: an unusual ocular complication of rheumatoid arthritis

Sir: We were interested in the case reported recently in the *Annals* of Brown's syndrome: an unusual ocular complication of rheumatoid arthritis.¹ We share the authors' surprise that this syndrome is not more frequently encountered, but would point out that we reported just such a case in 1980.² That patient had bilateral superior oblique tendon sheath problems, which resolved dramatically with local injections of corticosteroid. Our patient shared many features in common with the case reported by Cooper *et al*; particularly striking were the systemic features of rheumatoid disease. Our patient also had the rather curious phenomenon of 'early morning stiffness of the eyes', whereby his diplopia was much worse in the early morning and gradually resolved during the day.

Since reporting our case we have of course been alerted to this particular complication of rheumatoid arthritis, and one of us (PH) has seen four well recorded cases since our original report. We too are curious as to why 'trigger eye' is not as common as trigger finger in rheumatoid disease, as the pathology is identical. It may be that external trauma is the extra ingredient making trigger finger a much more common phenomenon.

P HICKLING
MARILYN BECK
Mount Gould Hospital
Mount Gould Road
Plymouth
PL4 7QD

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Magnetic resonance imaging

Sir: I would like to comment on an article published recently in the *Annals*.¹ Firstly, in fig 1, which describes magnetic resonance imaging of a normal left shoulder, items 7 and 8 are incorrectly labelled—item 7 should be the subscapular muscle and item 8 the infraspinatus muscle.

Secondly, I think it is important to point out that magnetic resonance imaging is still too expensive for common use in studying pathological processes that might be followed by other much less expensive ways, such as arthrography. In addition, reproducible sensitive results using magnetic resonance imaging are dependent on the radiologist, and in many cases the sensitivity of this study is certainly not surpassed by modalities already in common use.

RALPH J CURTIS
Orthopaedic Surgery Associates of San Antonio
Suite 1128
414 Navarro Street
San Antonio
Texas 78205
USA

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