IgA serum levels and disease activity in ankylosing spondylitis

SIR, I read with considerable interest the report by Franssen and associates of a prospective study of IgA serum levels and disease activity in ankylosing spondylitis (AS).¹ In their comparative trial of phenylbutazone and diflunisal, serum IgA, erythrocyte sedimentation rate, and disease activity all decreased during regular drug treatment, suggesting a disease modifying effect of the non-steroidal anti-inflammatory drugs (NSAIDs) in AS.

That NSAIDs affect favourably the natural course of AS is not widely appreciated.² In fact this concept first became clear to me after completion of a five year trial of indomethacin in 28 patients that dates back to 1968.³ As judged by several criteria, including articular pain, duration of morning stiffness, onset of fatigue, and joint mobility, the overall response to indomethacin was rated as good in 21 patients, fair in five, and poor in two. After receiving indomethacin for an average period of 33 months, 21 of the 28 patients were in the American Rheumatism Association functional class I.⁴ Only one patient had been so classified before the drug trial. The average Westergren erythrocyte sedimentation rate values for all 28 patients decreased during the drug trial from 39 to 26 mm/h ($p < 0.01$, Student's t test). Indomethacin was discontinued in three patients—in two because of a poor response to the drug and in one because of an adverse reaction. Clearly, the results of this trial suggest that indomethacin favourably alters the course of AS.

In 1981, 18 years after the first patients were entered into this trial, it was possible to locate and reassess 14 of the remaining 25 patients.⁵ Of the 14, four had achieved remission and required no further drug therapy. Indomethacin had been discontinued in four patients—in two because of gastrointestinal side effects and in two because of a more favourable effect from another NSAID. The remaining six patients continue to receive indomethacin at an average daily dosage of 100 mg (range 75–100 mg) and continue to benefit from its long term use.

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References

- 1 Franssen M J A M, Van De Putte L B A, Gribnau F W J. IgA serum levels and disease activity in ankylosing spondylitis: a prospective study. *Ann Rheum Dis* 1985; **44**: 766–71.
- 2 Calabro J J. Sustained-release indomethacin in the management of ankylosing spondylitis. *Am J Med* 1985; **79** (suppl 4C): 39–51.
- 3 Calabro J J, Amante C M. Indomethacin in ankylosing spondylitis. *Arthritis Rheum* 1968; **11**: 56–64.
- 4 Steinbrocker O, Traeger C H, Batterman R C. Therapeutic criteria in rheumatoid arthritis. *JAMA* 1949; **140**: 659–62.
- 5 Calabro J J. Appraisal of efficacy and tolerability of Indocin (indomethacin, MSD) in acute gout and moderate to severe ankylosing spondylitis. *Semin Arthritis Rheum* 1982; **12** (suppl 1): 112–6.

Idiopathic haemarthrosis with chondrocalcinosis

SIR, We read with interest the article by Woolf *et al* about the idiopathic haemorrhagic rupture of the shoulder in destructive disease of the elderly.¹

In our experience haemarthrosis may also be associated with articular chondrocalcinosis and joint destruction.^{2,3}

We have reviewed 28 patients with haemarthrosis in 32 joints (20 knees and 12 shoulders).⁴ Periarticular ecchymosis was found in only one case relating to the knee but in four cases relating to the shoulder. There were calcium pyrophosphate dihydrate microcrystals in the shoulder fluid in eight cases but typical calcification of the joint in only three cases. Degenerative radiological changes with rupture of the rotator cuff were common, but in three cases the presentation was that of a neuropathic joint.

Patients who did not improve with rest, aspiration of the effusion, and intra-articular injection of steroids were treated by radioisotopic synoviorthese.⁵ In these patients, after injection of 2–3 mCi of gold-198 (four cases) or rhenium-186 (nine cases), the shoulder effusions disappeared in 11 cases (mean follow up of four years).

Persistence of pain after treatment in most cases was thought to be due to degenerative lesions.

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References

- 1 Woolf A D, Cawston T E, Dieppe P A. Idiopathic haemorrhagic rupture of the shoulder in destructive disease of the elderly. *Ann Rheum Dis* 1986; **45**: 498–501.
- 2 Menkès C J, Simon F, Delrieu F, *et al*. Destructive arthropathy in chondrocalcinosis articularis. *Arthritis Rheum*, 1976; **19** (suppl): 329–48.
- 3 Villiaume J, Galle P, Amoureux J. Arthropathies lytiques et chondrocalcinose articulaire. *Semin Hôp (Paris)* 1974; **50**: 3175–90.
- 4 Cayla J, Huchet B, Rondier J, Menkès C J. Hémarthroses de la chondrocalcinose articulaire. *Rev Rhum Mal Osteoartic* 1982; **49**: 281–5.
- 5 Menkès C J. Is there a place for chemical or radiation synovectomy in rheumatic diseases? *Rheumatol Rehabil* 1979; **2**: 65–77.

Connective tissue deposits in MRL/1 mice

SIR, The article on connective tissue abnormalities in MRL/1 mice published by Edwards *et al* in the *Annals*¹ is very interesting. The description of morphological changes in the connective tissue confirms our observations on arthritis in the MRL/1 mouse that we published in 1985.²

In our study of 58 animals of different ages and sex we