Correspondence

Pain and rheumatoid arthritis

Sir, Gibson and Clark¹ have indicated that rheumatologists may underestimate their patients’ desire for pain relief. In their survey 47% of a random sample of patients with rheumatoid arthritis ranked pain relief as the most desirable objective of treatment. We also have been impressed by the knowledge that pain is the major symptom of our patients. We surveyed 250 patients and asked them to rank the importance of treatment related to the relief of four symptoms: pain, stiffness, joint swelling, and disability. The order in which the symptoms were presented to the patients was varied. One hundred and twenty two patients were suffering from rheumatoid arthritis, 53 from degenerative joint disease, and the remainder from a variety of other rheumatic diseases. 66% of the rheumatoid patients and 75% of the patients with degenerative joint disease ranked pain as the most important symptom to be treated. Only 22% of rheumatoid patients and 15% of patients with degenerative joint disease ranked disability as the most important symptom. Stiffness was ranked third by both groups, with 12% and 6% respectively. The patients with rheumatoid arthritis who considered disability to be all important were, in general, patients with end stage disease who had rheumatoid deformities without active synovitis. One wonders therefore whether the relatively small proportion of patients in Gibson and Clark’s survey who ranked pain relief important may have included a large proportion with end stage disease. Although the patients with degenerative joint disease considered similar priorities of treatment, they tended to have greater difficulty in differentiating the symptoms and appeared less dogmatic.

The message however remains clear: the success of treatment in the rheumatic diseases needs to be assessed by the relief of the patients’ most important symptom – pain.

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Frozen shoulder

Sir, I read with considerable interest the recent article by Binder and associates¹ in which patients with a frozen shoulder were assessed by arthrography and radionuclide studies. I have several questions and comments regarding the application of the first of these two modalities.

The authors report that their results disagreed with those contained in previous articles in that only half of the patients with adhesive capsulitis showed characteristic arthrographic abnormalities. These data are of concern to those of us who commonly perform arthrography in an attempt to diagnose and categorise the severity of capsular fibrosis. Perhaps an explanation for the seemingly low incidence of positive findings is related to arthrographic technique. Binder and associates state that a double-contrast examination was used, accomplished by injecting a combination of 5 ml of positive contrast material (Conray 280) and 5–15 ml of air. The usefulness of the double-contrast technique has been well described in previous articles and is most applicable to the evaluation of patients with suspected rotator cuff disruption.²⁻³ It is not the procedure of choice in the diagnosis of adhesive capsulitis, as early leakage of air and contrast material from the subacapsularis bursa and biceps tendon sheath occurs, a phenomenon that obscures the characteristic arthrographic alterations of adhesive capsulitis. In fact even those who are the most enthusiastic advocates of the double-contrast shoulder arthrogram indicate that adhesive capsulitis is better evaluated by a single positive-contrast examination.³ Furthermore, with this latter type of examination, slow distension of the glenohumeral joint with a mixture of contrast material and saline at the time of arthrography, after the diagnosis of adhesive capsulitis has been made, may provide excellent, although temporary, relief of the clinical manifestations of the frozen shoulder.⁴

These comments would indicate that meaningful data about the use of arthrography in the patient with adhesive capsulitis cannot be obtained if double-contrast arthrography of the shoulder has been employed. However, although Binder and associates state that such a technique was used in their study Figs 1 and 2 in their article appear to illustrate the single-contrast examination, as no air can be identified in the images. Perhaps, the authors could explain this discrepancy.

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References

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Str., A double-contrast arthrographic technique was used in the first six frozen shoulder patients studied, but no advantage was found over the single-contrast technique, and the latter technique was used in the remaining patients. These details were unfortunately omitted from the final draft of the paper. However, the procedures were performed under x-ray control, and adequate joint outline was obtained with both methods. There was no difference in the rate or extent of recovery of pain or range of passive movement and no difference in the frequency or severity of side effects with either technique. We therefore stand by our observation which is, as Wright and Haq suggested, that arthrographic features are not consistent in patients with painful stiff shoulders.

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Still’s disease and haemophagocytic syndrome

Str., We were very interested in the reports by Heaton et al., and by Morris et al. on two patients with Still’s disease and virus-associated haemophagocytic syndrome (VAHS). Although no research of haemophagocytic syndrome had ever been made in previous reports, it is striking to observe that acute episodes with similar clinical and biological features had already been reported and considered either as Reye’s syndrome or as consumption coagulopathy in systemic juvenile chronic arthritis.

For many years we have been intrigued by this life threatening complication and reported it in 1979 as a consequence of either virus infection, gold therapy, or other recent modifications in drug administration. We recently published a comprehensive study of seven patients, in whom we observed the association of features of consumption coagulopathy, pancytopenia, liver function alterations of various degree, and metabolic disturbances with changes suggestive of proteolysis. In our view, macrophage or other accessory cells such as Kupffer’s cells or endothelial cells might be the main cells responsible for this syndrome. Indeed, we had observed in histological material from our patients features of macrophage activation with phagocytosed material. The role of these cells is also suggested by a comparison of the VAHS occurring in Still’s disease with the main symptoms observed in two other rare and severe conditions: the accelerated phase of the Chédiak-Higashi syndrome and the familial erythrophagocytic lymphohistiocytosis.6 In the two latter clinical manifestations include lethargy, fever, hepatosplenomegaly, pancytopenia, and profuse bleeding with laboratory evidence of liver dysfunction, coagulation anomalies of complex origin, with a fibrinolytic process, and possible intravascular coagulation. These two syndromes are known to be associated with haemophagocytosis, and macrophages show in-vitro evidence of hyperactivation. Thus VAHS or drug induced HS in JCA and HS in Chédiak-Higashi syndrome and in familial lymphohistiocytosis share common features of a probable systemic macrophage activation.

The question remains why systemic JCA patients are more susceptible to virus or drug induced HS. The answer underlines the vulnerability of patients with systemic JCA and great caution must be taken when treating with high dose aspirin, gold salts, when adding another non-steroidal anti-inflammatory drug, or when a virus infection occurs. Most of the patients seem to survive only if they are rapidly treated with high dose steroid.

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References