PostScript

MATTERS ARISING

Treatment of shoulder pain

Hay and colleagues concluded, in their extended report of a trial of physiotherapy and injection for unilateral shoulder pain that physiotherapy and local steroid injection are of similar effectiveness.[1] They suggest that ''The high overall success rates… argue against the need for further exploratory trials in this condition''. I disagree. A large number of studies of shoulder pain have been bedevilled by diagnostic criteria that are not precise,[2] and this study must unfortunately join the others.

Unilateral shoulder pain has a number of different causes. The study by Hay excludes a few specific conditions—in particular, a ruptured rotator cuff, but must by definition include a heterogeneous group of problems that are in fact quite discrete. These include frozen shoulder (adhesive capsulitis), rotator cuff injuries without full rupture, subacromial joint arthritis (sometimes known as subacromial bursitis), bicipital tendinitis, acromioclavicular joint disease, and subdeltoid bursitis. It is barely credible to imagine that several of these could be successfully treated by a steroid injection into the subacromial joint. In particular, the subacromial joint does not communicate with the glenohumeral joint unless the rotator cuff is ruptured, so frozen shoulder cannot be treated with a subacromial injection. Thus any study of shoulder pain must separate the different causes into different groups. Others have done this and shown that the relative benefits of physiotherapy and injection may be different.[3]

Furthermore, it must be clear that any clinicians contributing to a trial are working to the same diagnostic criteria. Even experienced consultant rheumatologists cannot agree on exact diagnoses, as I and colleagues have shown previously,[4] and we concluded ''…recruitment of patients for studies of the treatment of shoulder lesions requires care to avoid selection of a heterogeneous group''. Given the variability of rheumatology training and experience in general practice it seems unlikely that diagnostic precision will be sufficient in that setting, and Hay's study does not conform to our recommendation.

For these reasons, far from suggesting that no further research is needed, this study underlines the need for clear and exact diagnostic criteria and further treatment trials for each of the specific causes of unilateral shoulder pain. Partly because clinical diagnosis may be difficult the use of magnetic resonance imaging scanning to define pathology may be an essential part of the investigation before treatment; I have certainly encountered many patients where a clear clinical picture is belied by a scan, particularly in the identification of rotator cuff pathology.

My own local audit of about 800 referrals of patients with shoulder pain suggested that some 40% had a frozen shoulder, with another 40% having abnormality in the rotator cuff/subacromial joint mechanism. Thus one could argue that a pair of steroid injections, one into the glenohumeral joint and one into the subacromial joint, might be expected to benefit about 80% of patients. However, such an approach, while practical, will not resolve in a scientific way the continuing uncertainty over the management of shoulder pain.

A N Bamji
Queen Mary's Hospital Frognal Avenue, Sidcup, UK

Correspondence to: Dr A N Bamji;
andrewbamji@lineone.net

References

1 **Hay EM**, Thomas E, Paterson SM, Dziedzic K, Croft PR. A pragmatic randomised controlled trial of local corticosteroid injection and physiotherapy for the treatment of new episodes of unilateral shoulder pain in primary care. *Ann Rheum Dis* 2003;**62**:394–9.
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Author's reply

We agree with Bamji that, although a number of ways of classifying shoulder problems have been proposed, none have been shown to be valid or reliable. In our large primary care trial[1] we adopted the ''red flag'' approach and are clear about its limitations in the paper. Although our trial was pragmatic, it examined a clearly stated question—''in patients presenting to general practitioners with a new episode of unilateral shoulder pain, and in whom specific ''red flag'' problems have been excluded, is a subacromial injection or a course of physiotherapy the best first choice?''.

Essentially, we were comparing two treatments commonly used by general practitioners. We were not investigating the relative merits of different types of injection, or indeed which components of the physiotherapy package had specific benefits. These are different questions, important in their own right, but not the ones we chose to answer in this particular study.

E M Hay
Staffordshire Rheumatology Centre, The Haywood, Burslem, Stoke-on-Trent, Staffordshire ST6 7AG, UK

Correspondence to: Dr E M Hay;
e.m.hay@keele.ac.uk

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Do etanercept-naive patients with rheumatoid arthritis respond better to infliximab than patients for whom etanercept has failed?

We read with interest the article by van Vollenhoven *et al*.[1] In short, they report on 31 patients with rheumatoid arthritis (RA), 18 of whom used etanercept (ETA) first and then switched to infliximab (IFX)in most part because of inefficacy, and 13 patients who used IFX first and changed to ETA mostly owing to adverse events. They suggested using the other tumour necrosis factor (TNF) inhibitor when one of them fails. Although, in general, agreeing with their findings, we would like to present our experience which is somewhat different from theirs and to discuss the possible reasons for this.

We set up an IFX registry at the Hospital for Special Surgery in February 2000, with the start of IFX infusions. The registry collected prospective data on all the patients with RA who started treatment with IFX, and followed up them every 2 months until May 2001. All patients completed questionnaires about their RA history, treatment, and functional disability (modified Health Assessment Questionnaire (mHAQ)) at baseline and every 2 months thereafter. A 42 joint count for tender and swollen joints was performed at each visit. Patients were telephoned 3–5 days after infusions and asked about reactions while at home.

The availability of ETA before the approval of IFX and the fact that use of ETA did not require concomitant methotrexate has resulted in the treatment of more patients with RA with ETA before trying IFX. However, after failure of ETA, several patients changed treatment to IFX. We compared response to treatment, adverse events, and discontinuation rates between patients for whom ETA had failed before IFX treatment (ETA-F) and patients who had not used ETA before—that is, etanercept-naive (ETA-N).

Eighty eight patients were treated with IFX between February 2000 and May 2001 (77 women, mean (SD) age 61 (12.1) years, mean (SD) RA duration 13.4 (9.8) years, failed DMARDs 2). In 37 (42%) patients ETA had failed before IFX was introduced. There was no difference in age, disease duration, and number of failed DMARDs between ETA-F and ETA-N patients. Sixteen ETA-F and 10

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