PostScript

MATTERS ARISING

Ultrasound guided musculoskeletal injections

We read with interest the paper by Hall and Buchbinder,[1] which discussed the importance of accurate needle placement guided by imaging techniques in the therapeutic response to local corticosteroid injection (LCI) for musculoskeletal (MSK) conditions.

Certainly, as the authors state,[1] more studies providing evidence of short and long term benefit and cost effectiveness of imaging guided LCI versus blinded injection are needed. However, we would like to make some comments on a number of important points.

Firstly, Hall and Buchbinder[1] include in radiological guidance different imaging techniques such as radiography, computed tomography (CT), magnetic resonance imaging, and ultrasonography (US). We would like to point out that MSK US has considerable advantages over other imaging modalities as it has no secondary effects, is quick to perform, is low cost, can be repeated, and is well accepted by patients. In addition, MSK US is routinely used by an increasing number of rheumatologists from many European countries. The accuracy, safety, and simplicity of US for guiding interventional procedures in the MSK system have been widely described.[2–9]

Secondly, the authors mentioned the contradictory results of two papers comparing imaging guided versus blinded LCI in the shoulder.[8 10] We found a better clinical response to US guided than to blinded LCI,[8] whereas Shanahan *et al* reported a similar response to CT guided and blinded suprascapular nerve block.[10] Both studies were randomised, assessor blinded, and short term. Nevertheless, both interventional procedures are essentially different. In suprascapular nerve block the aim is to place the needle next to the suprascapular nerve at the suprascapular notch so that the steroid diffuses into the nerve. The use of anatomical landmarks by an experienced operator probably is enough to achieve successful placement of the LCI. On the contrary, rotator cuff, biceps tendon, and subacromial-subdeltoid bursa are located close together. Therefore accurately siting the needle in the target as well as avoiding damaging intra-tendon injection are difficult using external landmarks. In addition, CT is radioactive, expensive, and requires a radiologist, whereas US is non-invasive, available, cheap, and can be performed by a rheumatologist at the patient's bedside while accurately diagnosing shoulder lesions.

In conclusion, we would like to emphasise that US has become a powerful extension of MSK evaluation performed by many rheumatologists for improving diagnosis and interventional procedures.

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Authors' reply

We thank Dr Naredo and colleagues for their interest and observations.

Musculoskeletal ultrasound remains a safe, non-invasive, and (relatively) inexpensive form of imaging. It has been taken up widely by clinicians, particularly in Europe, though there has been less enthusiasm elsewhere.

However, there remains a hypothesis in need of more formal testing implicit in this communication. Naredo *et al* propose that some targets such as the suprascapular nerve can be identified by anatomical landmarks, whereas others require precise localisation through imaging to ensure therapeutic impact. Our editorial proposes that this assumption needs to be tested. Is it really mandatory to inject precisely into the subacromial-subdeltoid bursa as opposed to the rotator cuff or the biceps tendon in a patient with shoulder pain to guarantee a reduction in pain and improvement in function over the longer term?

Until there is sufficient evidence from both participant and outcome assessor blinded randomised trials documenting a real difference between image guided needle placement and the anatomical landmark approach over the longer term (sufficient to justify the extra cost), the requirement for precise localisation remains speculative. We welcome the results of such trials to see whether or not "the Emperor has no clothes", the fairy tale equivalent of a null hypothesis.

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Increased prevalence of ocular glaucomatous abnormalities in systemic sclerosis

Dr Allanore and team members have conducted an inspirational study into the prevalence of ocular glaucomatous abnormalities in systemic sclerosis. However, it seems that they have overlooked a major methodological flaw about the definition of glaucomatous change pertaining to normal tension glaucoma (NTG). Unless further clarification can be offered, we cannot concur with the conclusion that glaucomatous neuropathy consistent with the vascular pathogenic hypothesis for NTG was dramatically more prevalent in patients with systemic sclerosis.[1]

Lee *et al* have revised the definitions of NTG of several major studies.[2] Emphasis on maximal intraocular pressure (IOP) $\leqslant$21 mm Hg and the importance of recognising the characteristic glaucomatous optic disc change or visual field defect have been implicated.[2] Almost 89% of the studies/publications required the demonstration of a characteristic visual field defect on perimetry as a prerequisite for diagnosing NTG.[2] In the present study, the above-mentioned key features have also been adopted as the defining criteria. Moreover, defining a case with a

visual field mean deviation <−2 dB was arbitrary and differed significantly from our understanding of the neuropathological basis of visual field damage specific to NTG.

Araie and coworkers have indicated that NTG and the ordinary primary open angle glaucoma or high tension glaucoma (HTG) showed significantly different visual field damage.[3] Visual field defects in NTG are more localised and predominant in the lower hemifield, whereas HTG has significantly more diffuse visual field damage.[4–6] It has been demonstrated that mean deviation in perimetry is good measure for assessing the more diffuse visual field damage characteristic of HTG but not as good for pinpointing a localised defect such as that seen in NTG.[7 8] Instead, pattern standard deviation or corrected pattern standard deviation were suggested as alternative indicators in representing the focal visual field defect in NTG.[7 8] As a result, the authors' conclusion about the relationship between NTG and systemic sclerosis may be based on an erroneous visual field index (mean deviation), which is neither sensitive nor specific for NTG.

Moreover, it should be pointed out that Allanore *et al* have adopted another arbitrary means of defining the IOP of the subjects recruited, which again showed marked disparity from our usual practice. The authors did not explain why phasing of the IOP was not undertaken given the fact that IOP shows diurnal variation, especially prominent in glaucomatous subjects such as those with NTG.[9] Recording of only one IOP measurement may not be sufficient owing to the influence of this confounding factor.

Appropriate case definition lies at the heart of every epidemiological research on glaucoma and any deviation from the consensual definitions may inevitably skew or even imperil the validity of the data.[9] In the interest of readers, we would be most grateful if the authors can provide us with more information about the rationale for the methodology used.

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Authors' reply

We thank Drs Chan and Liu for their comments about our article evaluating ocular glaucomatous changes in systemic sclerosis (SSc).

High intraocular pressure (>21 mm Hg) is undoubtedly known to be the main risk factor associated with glaucoma[1]; however, substantial evidence was provided recently to support a key role of vascular abnormalities in the pathogenesis of glaucoma. In particular, patients with normal tension glaucoma, who do not have the main risk factor of developing glaucoma (increased intraocular pressure), may also develop optic neuropathy, and numerous recent studies support the hypothesis that these lesions are associated with vasculopathies.[2–4] These findings led us to investigate the prevalence of glaucomatous changes in SSc, a disease which is strikingly associated with generalised vascular involvement.

Although primary open angle glaucoma is well defined, normal tension glaucoma is more difficult to diagnose. Independently of intraocular pressure, glaucomatous changes are supported by optic disc cupping together with visual field defects.[1] Thus, for the purpose of our comparisons between groups, we had to define cut off values for these two variables. For optic disc cupping, we chose a cut off point based on reported data[5]; we defined mild abnormalities as a c/d >0.3 and severe involvement as a c/d >0.7. For visual field, we also chose a mean difference <−2 dB according to reported data. Thus, the significant differences between SSc and matched controls for these measures allow us to suggest that patients with SSc have glaucomatous abnormalities as compared with our controls. Although there is no consensual definition of NTG, these results clearly suggest that patients with SSc have glaucomatous propensity. The continuing prospective standardised follow up of our patients and other series will quantify the precise risk factor of SSc for normal tension glaucoma.

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CD68 is not a macrophage-specific antigen

The article of Kunisch *et al* discussing a cross reactivity of allegedly macrophage-specific anti-CD68 antibodies with fibroblasts and activated endothelial cells demonstrates amply that these antibodies should not be used for the identification of macrophages.[1] Yet they have been used for this purpose in nearly all medical disciplines, particularly in vascular diseases. In 1990 we observed that some neointimal cells in experimental transplantation atherosclerosis, human native atherosclerosis, and experimental native atherosclerosis had reacted with both presumptive macrophage-specific antibodies (RAM11, HAM56) and an antibody against muscle actin (HHF35).[2] In 1997, Andreeva *et al* demonstrated that the very same human intimal and neointimal cells were immunopositive, both with anti-macrophage (CD68, HAM56) and anti-muscle actin (asm-1, HHF35) antibodies.[3] On the basis of these findings, these authors formed a hypothesis that the macrophage markers involved in these reactions were not indicative of cell histogenesis but of phagocytosis. Neither our observation[2] nor the demonstration of Andreeva *et al*[3] had any influence on the practice of macrophage identification by the above mentioned antibodies.

Today, I share Kuhn's opinion[4] that the acceptance or rejection of new scientific ideas depends on their relationship to existing paradigms. If they are in agreement with them they are accepted, but if they contradict them they are usually ignored. When the immunohistochemical identification of macrophages was originally proposed there was no existing paradigm in this field and its authors presented their methods against no substantial opposition. My observation that an unreasonably high amount of macrophages had been identified with new monoclonal antibodies in comparison with previously used electron microscopy was disregarded.[5] Rare articles describing the reactivity of the above mentioned anti-macrophage antibodies with other cell phenotypes in other medical disciplines were also neglected.

Kuhn described the scientific process as a conflict, in which less satisfactory paradigms are replaced successively by better ones.[4] There is only one way which guarantees the correctness of individual paradigms: a strict observance of the facts. For example, an immunological injury induces an intimal thickening composed only of ''macrophages''