MATTERS ARISING

Different concepts of musculoskeletal pain

In his leading article, Dr Awerbuch1 purports to address the problem for the clinician of patients who present with pain but in whom there is no abnormality detected on physical examination. Whereas in former times these same patients may have been labelled 'hysters, hypochondriacs or malingerers',2 Dr Awerbuch suggests that they now be categorised as 'somatising patients'. Whilst this formulation might allow some clinicians to then address underlying causation. However, his warning on the clinical misinterpretation of abnormalities is frequently found in various imaging studies is timely. In the context of chronic spinal pain, the term 'somatic dysfunction' is synonymous with the phenomenon of myofascial pain syndrome (MPS) and of 'fibromyalgia syndrome' (FS). His preferred therapeutic approach is to acknowledge the 'legitimacy' of the pain experience of these patients and to then address underlying causation. However, Dr Awerbuch provides no insights into causation of MPS and FS (apart from a suggestion of iatrogenesis), and resiles from the theme of his article by failing to discuss whether they are in reality part of the syndrome of somatisation or further examples of non-disease.

The danger that clinicians can be seduced by a 'special test' into making an incorrect diagnosis is highlighted by Dr Awerbuch. He gives as an example of this phenomenon the use of the upper limb or brachial plexus tension test (BPTT) in the diagnosis of the pain syndrome known as Repetition Strain Injury (RSI), a pain syndrome which Dr Awerbuch has declared to be a 'unique Aussie disease', perhaps to be remembered under the eponym 'Kangaroo Paw'.3 His claim that the BPTT was developed before the formulation of the RSI concept is true. The BPTT was originally devised by Elvey, an Australian manipulative therapist, specifically to assist examiners in the differential diagnosis of shoulder pain.4 A recent independent study suggests that the test does have discriminative validity when used in this clinical context.5

In so far as it appears that the BPTT does give an indication of the mechanosenstivity of those neural tissues which span the neck and wrist, it is a test which is similar to the straight-leg-raising test.6 It was not until 1986 that the BPTT was reported to be useful in the diagnosis of a group of patients with RSI,7 some two years after the Australian RSI epidemic had started.8 A significant neurogenic contribution to the symptomatology of RSI was already being recognised as long before the responses to the BPTT in these patients became widely known.9 Other authors using different examination techniques have commented upon the increased mechanosenstivity of upper limb neural tissues present in many of these patients.10 A close reading of the paper by Cohen et al11 fails to reveal any mention of the BPTT. These authors produced cogent evidence in support of a 'neuropathic' basis for clinical features of what they prefer to call 'Refractory Cervicobrachial Pain Syndrome'. For a reviewer to invoke non-disease and somatisation in the face of this evidence borders on the solicitipuis. Whether or not this particular pain syndrome is 'neurogenic', as has been proposed,12 remains unresolved. Dr Awerbuch's assertion that physical examination remains the bedrock of clinical rheumatology is a truism. An explosion of knowledge in the area of pain pathophysiology13 has presented those of us engaged in neuromusculoskeletal medicine with a formidable challenge. We will serve long patients well if we accept this challenge and, in so doing, we will enlarge our own comfort zones.

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AUTHORS' reply: Dr Quintner raises a number of issues. Somatisation disorder, while not a rheumatological disorder is none the less a 'proper diagnosis' worthy of treat¬ment. Fortunately, most cases resolve spontaneously or after appropriate explana¬tion. The undertaking of excessive or unnecessary investigations and failure appropriately to reassure may serve to alarm the patient, reinforce illness conviction, and perpetuate illness behaviour.

Whatever the genesis of pain in myofascial pain syndrome and fibromyalgia syndrome, data published subsequently to the preparation of my manuscript suggest that emotional trauma and somatisation play a significant aetiological role.1-3

The Australian repetitive strain injury (RSI) epidemic was unique in a number of respects, not least of which was the sheer size of the epidemic. Whereas epidemiological data from other parts of the world indicated an incidence of neck and arm pain in the workplace of about 25%,4,8 in Australia many workers had already experienced arm pain which transcended age, occupation, nature and duration of activity, and pattern of arm usage. A new name was needed. The term 'RSI' was born in 1985, the first appearing in a National Health and Medical Research Council booklet entitled Approved occupational health guide repetition strain injuries. In 1985 one could not have envisaged that RSI would one day prove to be one of Australia's more successful exports. Happily for Australian workers, not to mention the Australian economy, the epidemic died out in the workplace of about 80% by 1993, as those surrounding its birth. The cause was never found and by 1987 researchers were unable to find sufficient new cases of RSI to undertake further studies. As late as 1992, Dr Quintner were appearing in the Medical Journal of Australia under the heading 'Hypothesis' in an attempt to explain the phenomenon. However, the neuropathic pain hypothesis which was proposed was based on epidemiological grounds alone. As long as the pathogenesis of so called RSI remains hypothetical, it is unlikely that any clinical tests will be embraced by serious scholars.

Dr Quintner undermines his own argument on the BPTT by citing the paper of which he was a co-author.10 This study (with no controls), consisted of 60 self selected patients, 33 of whom attended the practice of one of the authors (a general practitioner) and 27 of whom attended Dr Quintner's practice. The diagnosis of RSI was a fair accomplish before the study started; indeed, it was a prerequisite.6 Patients who complained predominantly of arm and hand symptoms (pain, aching, burning, heaviness, tingling, numbness) and of neck, upper back and shoulder symptoms who were receiving workplace or medical care were agreed to take part in the study. Little wonder then that 59 of the 60 patients had a notionally positive BPTT.

In another paper cited by Dr Quintner there was also no control group. This was a retrospective study consisting of 100 self selected RSI patients: 'criteria for inclusion were persistent (greater than 6 months) pain in the upper limb even overuse at work and for which a worker's
LETTER TO THE EDITOR

Activated protein C resistance caused by factor V Arg 506→Gln mutation has no role in thrombotic manifestations of Behçet’s disease

Vascular involvement is found in approximately 25% of patients with Behçet’s disease and includes venous occlusions (88%), arterial aneurysms, or arterial occlusions (12%), but the mechanism of thrombosis remains unexplained. Recently, a genetically determined defect in anticoagulation characterised by resistance to activated protein C (APC) was frequently found in patients with venous thromboembolism. APC resistance is highly linked to a single factor V gene mutation, Arg 506→Gln. To see whether APC resistance could explain the thrombotic events observed in Behçet’s disease we looked for the factor V gene mutation in 15 unrelated patients suffering from the condition. The patients were 11 men and four women (mean age 39 years (range 15–58). All fulfilled the International Study Group criteria for Behçet’s disease. Superficial thrombophlebitis and retinal occlusions were not considered. All patients had a history of thrombosis, affecting veins in 12 and arteries in six. Recurrent vascular events were noted in nine patients: venous followed by venous in five, venous then arterial in three, and arterial-arterial in one. The factor V gene mutation was identified using the polymerase chain reaction and denaturing gradient gel electrophoresis, as described previously. The plasma APC resistance test could not be performed because the majority of patients were receiving anticoagulants at the time of the study. No patient had the Arg 506→Gln mutation.

We conclude that APC resistance linked to the Arg 506→Gln mutation is not a cause of thrombotic manifestations in Behçet’s disease.

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