Microcirculation in ankylosing spondylitis

It was interesting to read the paper by Beauvais *et al* reporting two cases of ankylosing spondylitis (AS) with cutaneous vasculitis and IgA nephropathy¹ emphasising the possibility of vascular involvement in AS. We wish to emphasise this aspect by the evaluation of microcirculation in AS using nailfold capillaroscopy.²

Forty six patients were enrolled in this prospective study, divided into 32 AS patients (fulfilling the revised New York criteria,³ mean age 38 years. Twenty eight were HLA B27 positive, and there were 14 control patients (disc herniation) mean age 34.6 years.

Capillaroscopic findings evaluated by the same investigator (JCR) (unaware of the diagnosis in most of the cases) were classified into five groups:⁴ normal; minor dystrophies (characterised by more than 15% tortuosity); oedema (recognised by fuzziness of pericapillary environment); microangiopathy (this pattern associates—a qualitative element represented by major dystrophies like megacapillaries with irregular diameter, tortuous meandering or bushy capillaries—and quantitative element (reduction of loop number in the nailfold distal row less than 9 per mm), and stasis (characterised by a dark blood flow, sometimes granular, with low speed and regular enlargement of the two branches).

Statistical analysis used Fisher's exact test for normal and minor dystrophies on the one hand, and oedema and microangiopathy on the other.

The results, summarised in the table, show more frequent capillaroscopic abnormalities in the AS group compared with controls, for the oedema and microangiopathy patterns ($p = 0.01$), whereas there was no difference for minor dystrophies. No differences were found in terms of age, disease duration rheumatological and extra articular manifestations (skin, kidney, gut) or biological parameters (CRP, serum IgA) between AS patients with microangiopathy ($n = 5$) and AS patients with a normal capillaroscopy ($n = 9$).

Nailfold capillaroscopy is a simple, non invasive and reproducible technique.² In this study minor dystrophies are seen with the same prevalence in both groups. A specific capillaroscopic pattern of AS does not seem to exist. Conversely, this study shows an increase of abnormalities like pericapillary fuzziness (oedema) (due to an inflammatory reaction), and microangiopathy. These findings are in accordance with the reports of clinical vasculitis associated with AS, such as cutaneous microscopic vasculitis^{1 5 6} with renal or gut involvement, or large vessel vasculitis, Takayasu's arteritis^{7 8} or polyarteritis nodosa.^{9 10} Histological studies have also revealed the possibility of vascular in-

volvement in AS, as well as in the skin,^{11 12} with immune deposits^{12 13} as in the kidney.^{14 15}

The significance of these capillaroscopic modifications remains to be clarified (none of our patients with capillaroscopic microangiopathy displayed extra articular manifestation) but the mechanism of such a microvascular involvement may be consistent with an immune complex disease¹⁶ in AS.

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Cervical neuropathology in rheumatoid arthritis

I read with interest the article on the neuropathology of the brainstem and spinal cord in long standing, severe, rheumatoid arthritis¹ and the authors' assessment of the pathological mechanisms involved in the neural damage.

They conclude that the major mechanism of damage is pressure on the anterior aspect of the cord by the skeletal elements making up the neural canal due to the subluxation deformity of the neck. However, they seemed to remain uncertain as to why the major neural damage was seen in the posterior part of the spinal cord.

In 1982 we reported a series of patients with manubrio-sternal joint subluxation due to rheumatoid arthritis, and noted that this deformity was closely associated with major deformities in the cervical spine.²

We postulated that both deformities resulted from chronic forward flexion of the head on the trunk giving rise to both anterior (manubrio-sternal) and posterior (cervical spine) joint subluxation. This would agree with the hypothesis put forward by Henderson *et al* that the damage to the cord is due to forced flexion of the cord over the deformed cervical spine, leading to anterior compression and more seriously to chronic stretching and fissuring of the posterior part of the cord.

In both Henderson's paper and in ours the straight position of the resected cervical spine specimens is almost certainly a post mortem artefact, the in vivo position being chronic, severe, anterior flexion. It is likely that in this flexed position the severe narrowing of the spinal canal that is present in the illustrated specimens, would be greatly lessened.

Perhaps the results of surgery would be enhanced if efforts were made to stabilise the neck (as well as the anterior elements of the chest) without attempting to reduce the forward flexion so typical of these patients.

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- Henderson F C, Geddes J F, Crockard H A. Neuropathology of the brainstem and spinal cord in end-stage rheumatoid arthritis: implications for treatment. *Ann Rheum Dis* 1993; 52: 629–37.
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AUTHOR'S REPLY: Dr Rooney's observation of patients with rheumatoid arthritis that manubrio-sternal subluxation is associated with chronic flexion of the neck helps to explain our unexpected histological findings in nine patients who came to necropsy, which we reported in our recent article.¹ We concur with Dr Rooney² and Professor Breig³ that damage to the spinal cord results from flexion over a deforming mass, such as a subluxed odontoid process or pannus formation. The shear caused by the ventral deformation is greatest dorsally, and correspondingly results in dorsal cord injury.

The fixed neck flexion which Dr Rooney observed helps to explain why there was selective injury to the axons of the cuneate fasciculi. Several authors have suggested that mechanical injury to the brachial nerve roots may occur as they are repetitively pulled taut around the pedicles during flexion of the neck.^{4 5} We believe that chronic stretch injury

Capillaroscopic findings in ankylosing spondylitis (AS) patients and controls (C).

Number (%)	Normal	Minor dystrophies	Oedema	Micro angiopathy	Stasis
AS n = 32	9 28%	4 12%	13* 40%	5 15%	1
C n = 14	10 71%	3 21%	1 7%	0	0

*3 associated with dystrophy, and one with microhemorrhagia.

to the cervical roots occurs in rheumatoid arthritis and that this phenomenon is responsible for the diffuse axonal injury we observed in the cuneate fasciculi.

We therefore argue that patients with rheumatoid arthritis should be stabilised in a neutral or slightly extended position to lessen spinal cord stretch and to prevent flexion-deformation injury to the neuraxis over a subluxed dens or other deforming mass. Furthermore, the unstretched spinal cord in the extended posture is more resilient to deformation injury than the stretched spinal cord in the flexed posture.

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Fibromyalgia in the workplace

While not subscribing entirely to the views expressed by Dr Reilly in his leader

Fibromyalgia in the workplace: a "management" problem,¹ it seems to me that the criticisms by Champion *et al*² are ill-founded. As pointed out by Dr Reilly, the views of these authors expressed in their letter do not constitute the "popular view" of Australian rheumatologists on the subject. Given the admission by Champion *et al* that "no significant somatic abnormality has been identified in these cervico-brachial disorders", the umbrage which they appear to have taken to Dr Reilly's views appears churlish. These three authors have been vociferous campaigners in Australia of the concept of RSI as organic injury, entraining in support of their hypothesis inter alia a number of indirect observations, for example, notional disordered C-fibre function following topical Capsaicin administration and notional clinical observation, such as, the presence of allodynia and hyperalgesia,² but fail to acknowledge alternative explanations which might account for these observations. These authors make no attempt to explain the oft-found upper limb anaesthesia in an extended glove distribution in patients with so-called 'RSI'. Essentially, their hypothesis is that "altered central processing is maintained dynamically by peripheral nociceptive input", but have yet to identify such input, which appears at odds with their acknowledgement that there is "no significant somatic abnormality . . . in these cervico-brachial disorders". Dr Reilly is correct in pointing out that the concept of 'RSI' as organic injury flies in the face of overwhelming epidemiological evidence which suggests the very opposite. I would commend to these authors a reading of the small area analysis undertaken by Hadler on the USA West Communications Inc workforce which was located in a number of USA states.³ This study was able to identify no ergonomic variable to account for the variance of distribution of arm complaints in the different states in individuals doing the same work on the same equipment for the same duration.

What has emerged in Australia and is starting to emerge in the USA is that when normal discomforts associated with musculo-

skeletal activity are conceptualised as symptomatic of a potentially hazardous injury in the absence of prima facie evidence then the consequence is an epidemic of arm pain in the workplace.³⁻⁹

The British experience was prophesied by no less a personage than Auberon Waugh: "But in one point at least the Australians are ahead of us, and I bring news of this great innovation, called Repetitive Strain Injury (RSI) with some pride. Briefly RSI is the ache we all feel when we do something too often or too long . . . since it was invented as a disease about 6 years ago, it has spread like a bush fire throughout the whole spectrum of employment . . . I prophesy a tremendous future for this wankers' disease in Britain, as soon as a few more people learn about it. It will go through the country like a dose of salts".¹⁰

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