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 nephropathy emphasising the possibility of vascular involvement in AS. We wish to emphasise this aspect by the evaluation of microcirculation in AS using nailfold capillaroscopy.

Forty six patients were enrolled in this prospective study, divided into 32 AS patients (fulfilling the revised New York criteria), 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: normal; minor dystrophies (characterised by more than 15% tortuosity); occasional or frequent fuzzy capillaries—loss of peri-capillary environment; microangiopathy (this pattern associates—a qualitative element represented by major dystrophies like mega-capillaries with irregular diameter, tortuous meandering or bushy capillaries—and quantitutive 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 parameter (urokinase, CRP, serology ( IgA, 

<table>
<thead>
<tr>
<th>Number (%)</th>
<th>Normal</th>
<th>Minor dystrophies</th>
<th>Oedema</th>
<th>Microangiopathy</th>
<th>Stasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>AS n = 32</td>
<td>9</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>C n = 14</td>
<td>10</td>
<td>3</td>
<td>1</td>
<td>0</td>
<td>0</td>
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*3 associated with dystrophy, and one with microhemorrhagia.

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

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Cervical neuropathy in rheumatoid arthritis

I read with interest the article on the neuropathy of the brainstem and spinal cord in long standing, severe, rheumatoid arthritis and the authors' assessment of the pathological mechanisms involved in this neurological complication.

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 raise the possibility of a minor neurapraxia, or minor neural damage 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.

We postulated that both deformities resulted from chronic forward flexion of the head on the trunk giving rise to both (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 spinal canal on the cord, 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 severity of the 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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AUTHOR'S COMMENT. 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. I concur with Dr Rooney and Professor Bradford 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 deformity is directly 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 upper cervical 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. We believe that chronic stretch injury...