von Willebrand factor, exercise, and ischaemia/reperfusion injury

Sir: Farrell et al1 suggest that exercise induced release of von Willebrand factor (vWF) evidence for hypoxic reperfusion microvascular injury in rheumatoid arthritis (RA). They found that increases of plasma vWF in patients with RA were greater than those in age matched controls without RA who performed the comparable exercise. One interpretation for this greater increase is excess release from synovial endothelial cells as a result of ischaemia/reperfusion injury. However, Farrell et al may have overlooked another possible mechanism. One would presume that blood pressure and pulse rate would be increased in both patients and controls owing to the exercise. Increased systolic and diastolic blood pressure is associated with increased vWF, and a short (10 min) monotonous venous occlusion stress to the lower arm will also increase vWF.2 It may be, therefore, that some of the increased vWF in the patients with RA might have been due to release from more fragile endothelial cells in this haemodynamic force, even if the exercise was apparently within the patient’s capacity.

Nevertheless, we have been using a similar exercise regimen (where haemodynamic forces are controlled) to study the effects of ischaemia-reperfusion in patients with intermittent claudication but without evidence of connective tissue disease.4 Our experience is that five minutes of treadmill exercise leads a burst of thromboxane peaking five minutes after exercise has stopped, and a peak in vWF an hour after the cessation of exercise, only in patients. Our interpretation includes the possibility that vWF release from the lower limb endothelium is due to ischaemia-reperfusion. Thus both our data2,4 and those of Farrell et al support the growing hypothesis that increased vWF reflects endothelial injury. The remaining debate seems to be of which cells, and by what mechanism.

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Elusive ‘α-δ’ sleep in fibromyalgia and osteoarthritis

Sir: Association between disturbed sleep and primary fibromyalgia syndrome (PFS) is well

Patient characteristics. Mean (range) is shown

<table>
<thead>
<tr>
<th></th>
<th>PFS* (n=10)</th>
<th>OA* (n=10)</th>
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</thead>
<tbody>
<tr>
<td>Female:male</td>
<td>10:0</td>
<td>10:0</td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>53 (35-62)</td>
<td>40 (60-61)</td>
</tr>
<tr>
<td>Mean symptom duration (months)</td>
<td>18 (8-36)</td>
<td>38 (16-48)</td>
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</tbody>
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*PFS-primary fibromyalgia syndrome; OA-osteoarthritis.

unreported mini-arousal. No period of α-δ sleep was identified in any patient. No qualitative differences in recordings between groups were noted.

Our failure to detect α-δ intrusion in NREM sleep by visual and automated assessments most likely reflects differences in EEG interpretation rather than patient selection or technical differences. We used the strict criteria of Hauri and Hawkins who coined this term in a study of sleep disturbance in a heterogeneous, predominantly psychiatric, patient group.6 We commonly detected α waves, but mainly in relation to light (not NREM) sleep and frequent arousal. Poor sleep and frequent arousal alone could account for α-δ sleep in PFS when only two EEG channels were used7 without additional EEG channels to facilitate recognition of arousal. This could also explain non-specific α-δ sleep in patients without PFS and indeed normal subjects8 with poor sleep.

Similarity of sleeping EEGs in PFS and OA suggests that factors other than poor sleep alone are important for development of PFS. Although our study did not reproduce the assertion that a characteristic non-restorative sleep pattern is central to development of PFS,9,10 the importance of sleep to symptoms and disability in subjects with locomotor pain is not necessarily undermined. We suggest that factors other than sleep are required in this difficult area, and that full multiple EEG channel recording is necessary to allow adequate assessment and interpretation of electrophysiological findings.

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