Matters arising

Reflex path length and clonus frequency

Sir: Iansek, in a recent article on the effects of reflex path length on clonus, noted that Walsh proposed the existence of a central pacemaker as the determinant of clonus frequency. However, we did not use the term "pacemaker" as he stated, nor did we imply such a mechanism. We proposed that clonus is due to the repeated activation of the muscle stretch receptors, and we observed consequent central refractory and excitatory periods. As Iansek noted, the refractory period would not restrict the lower frequency, but only the upper frequency. We disagree with the author's statement that the lower frequency is determined by the reflex delay (alone), which would imply that for any muscle, the lower frequency could not be changed. Although we were unable to lower the frequency of clonus, Rack has demonstrated that it is possible to do so using a different experimental design. The lower frequency depends upon the muscle contraction and relaxation times which are influenced by the inertial load on the muscle, as well as the reflex delay time. We measured the refractory period only in the triceps surae muscle (90–100 ms). This period may differ for other muscle groups with different central stretch reflex organisations, thereby resulting in different maximum clonus frequencies. In order to reach an understanding of clonus, it is essential to consider not only reflex path length but also muscle contraction and relaxation times, muscle load, muscle spindle activity and central excitability, all of which play a role in clonus. The author's conclusion that clonus frequency in spastic muscles is a direct consequence of path length is an oversimplification. The reflex latency time is only one of the peripheral contributing factors which must be integrated with alteration of the stretch reflex refractory and excitatory phases in order for clonus to be manifest.

To summarise, Iansek has not accounted for our findings of refractoriness. It is true that different muscles may have different rates of clonus, but this in no way changes our conclusion that both central and peripheral elements must play a role.

PETER NATHAN
National Hospital for Nervous Diseases,
Queen Square, London WC1N 3BG, UK

MILAN R DIMITRIJEVIC
ARTHUR M SHERWOOD
Baylor College of Medicine,
1333 Moursund Avenue,
Houston, Texas 77030 USA

References