MATTERS ARISING

Bimanual motor performance in controls and patients

Brown, Jahanshahi, and Marsden[1] conclude their study of bimanual movements in Parkinson's, Huntington's, and cerebellar disease by suggesting that: "Further research should focus on the precise mechanisms underlying the difficulties experienced by patients with motor disorders in performing bimanual movements." These difficulties are, however, experienced by many of the normal population. This became evident when we were piloting a replication of a bead and tapper task.[2] Some of us had great difficulty with this bimanual task, whereas those of us with specialised, bimanual motor skills—for example, typing or playing the piano—had relatively little dual-task interference of tapping when transferring beads with the other hand. A study of bimanual task performance, using the bead and tapper test, in controls and subjects with depression or parkinsonism confirmed this preliminary observation.[3] Across all subjects, those with specialised bimanual motor skills showed significantly less dual task interference on the bead and tapper test than did those subjects without such skills, and the size of this effect of bimanual motor skill was as large as the effect of parkinsonism. Future studies of bimanual movements, across groups of patients, should therefore control for the presence of specialised, bimanual motor skills in the different groups.

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Soluble interleukin-2 receptor levels in serum of patients with demyelinating polyneuropathy associated with monoclonal gammopathy.

I read with interest the report by Dr Vrethem and colleagues[1] of elevated serum levels of soluble interleukin-2 receptors (sIL-2R) in some patients with demyelinating neuropathy associated with monoclonal gammopathy, suggesting a possible role for T cell activation in the pathogenesis of this condition. The insidious onset and chronic nature of paraprotein-associated demyelinating neuropathy make it difficult to interpret the pathogenetic significance of point measurements of sIL-2R, but it is interesting to note that patients with acute idiopathic demyelinating neuropathy (Guillain-Barré syndrome) have been reported to have elevated sIL-2R levels at disease onset which fall with clinical recovery.[2]

Most patients with peripheral neuropathy in the report[1] had an IgM monoclonal gammopathy of undetermined significance (MGUS). IgM MGUS-associated peripheral neuropathy with anti-myelin-associated glycoprotein (anti MAG) antibodies has been reported to be relatively unresponsive to plasma exchange, unlike IgA and IgG MGUS-related neuropathies,[3] and hence may possibly represent a separate nosological entity.[4] Furthermore, a recent report documented paraprotein-associated demyelinating neuropathy responding solely to cyclosporin, an immunosuppressive agent that selectively inhibits T lymphocyte responses, despite the continued presence of paraprotein.[5] It is therefore possible that demyelinating neuropathy associated with monoclonal gammopathy is pathogenetically heterogeneous, a subset of patients having a T cell-related, rather than humoral, immunopathogenesis. An analogous situation may exist for Guillain-Barré syndrome.[6]

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NOTICES

The winter meeting of the British Neuropsychiatric Association, on **Brain repair and rehabilitation** will take place at the Royal Society of Medicine (London) on 21 January 1994.

The IVth meeting of the **European Neurological Society** (ENS'4) will be held in Barcelona, Spain on 25–29 June 1994 (teaching courses 25–26 June, scientific programme 27–29 June). Deadline for submission of abstracts: 15 January 1994. Further information from Support Services, c. Paris 150, 08036 Barcelona, Spain. Tel: +34-3-322-65-54; fax: +34-3-410-97-42.

The **XIIth International Congress of Neuropathology** will be held in Toronto, Ontario, Canada from 18–23 September 1994. This meeting will be conjoint with the American Association of Neuropathologists Annual Meeting and the Canadian Association of Neuropathologists Annual Meeting. For further information please contact Dr J J Gilbert, Victoria Hospital Research Institute, 375 South Street, London, Ontario N6A 4G5, Canada. Tel: +1-519-667-6649; fax: +1-519-432-7367.

Third International Congress of Movement Disorders

The congress will be held on 8–12 November 1994 in Lake Buena Vista (Orlando), Florida, USA. The deadline for abstract submission is 1 April 1994. For further information contact: Central Headquarters Office, The Movement Disorder Society, PO Box 6, Clarastrasse 57, CH-4005 Basel, Switzerland. Tel: ++41 61 691 51 11; Fax: ++41 61 691 81 89.

Correction

In the July 1993 issue of the journal, in Matters arising, the following errors appeared on p.834 in the article by P H Ellaway, N J Davey, and D W Maskill. The title should have been "Inhibition of motor unit discharge in humans evoked by transcranial magnetic stimulation." In the third paragraph, the fifth sentence should be: "A concentric needle electrode was inserted percutaneously into the first dorsal interosseous muscle to record the discharges of single motor units." The figure legend should have read: "A: Averaged surface, rectified EMG from the first weak voluntary contraction, in response to 50 magnetic stimuli at a strength (37% of output) below threshold for direct excitation. B: The peristimulus time histogram (below) and cusum (above) of the discharges of a single motor unit in the first dorsal interosseous during 100 magnetic stimuli at 37% output under identical conditions to those in A."