MOTOR EVOKED POTENTIALS TO MAGNETIC STIMULATION IN CHRONIC AND ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY

JOHANNES C. WÖHRLE, MD, THOMAS KAMMER, MD, WOLFGANG STEINKE, MD, and MICHAEL HENNERICI, MD

To date, there are few data on transcranial (TMS) and paravertebral (PMS) magnetic stimulation in inflammatory demyelinating polyneuropathies (IDP). Particularly, the significance of involvement of proximal nerve segments for the central motor conduction time (CMCT) has not been studied systematically. Since motor evoked potentials (MEP) may detect coexisting central and peripheral demyelinating disease, supporting a hypothesis of pyramidal tract dysfunction, we looked at the patterns of MEP latency abnormalities in 14 patients with IDP who presented without any clinical symptoms or signs of CNS involvement.

METHODS

In 9 patients with chronic (CIDP) and 5 with acute (AIDP) inflammatory demyelinating polyneuropathy, MEP were recorded from first dorsal interosseous (FDI) and anterior tibial muscles (TA). The patients fulfilled current clinical diagnostic criteria of CIDP and AIDP/Guillain–Barré syndrome, and they had no clinical or other evidence of central nervous system disease. Each gave their informed consent prior to the examination.

In the CIDP group, patients were between 36 and 75 years old; and their severity of disease ranged from 1 ("mild symptoms, no signs, or vice versa") to 4 ("requiring assistance with eating, dressing, etc.") according to the scale of McCombe et al. AIDP patients were 31–83 years of age and scored clinically between 2 ("mild motor or sensory symptoms or both, with signs") and 5 ("not ambulant").

Magnetic stimuli were applied to the cortex, over the cervical (C-7) and lumbar spine (L-5) using a 9-cm circular coil (Magstim 200, Madaus Medizin Elektronik GmbH & Co. KG, D-7803 Gundelfingen) by standard technique. We obtained four recordings for each test and superimposed at least two traces (Dantec Counterpoint EMG device, Dantec Medizinelektronik, D-76275 Ettlingen). We assessed MEP latencies and amplitudes (qualitatively) and calculated CMCT: CMCT = CML – PML (CMCT: central motor conduction time; CML: cortical motor latency; PML: peripheral motor latency). We compared the patient data with age-matched normal controls, as reported by Kloten et al. and as confirmed for our laboratory.

RESULTS

In the 9 patients with CIDP, CML was prolonged to FDI in 5 and to TA in 7, PML to FDI in 4 and to TA in 5 patients. CMCT was prolonged in 4 patients for FDI and in 4 patients for TA. However, for FDI in 2 and for TA in 3 patients, CMCT was abnormally short (Table 1).

In the 5 AIDP patients, CML was prolonged to FDI in 2. No cortical MEP was obtained from TA in 1 patient; the CML of all the other patients was normal. PML was prolonged to FDI in 3 and to TA in 1 patient. No TA potentials were elicited over the spine in 1 patient. CMCT was prolonged to FDI in 1, and to TA in 1 patient; and CMCT was abnormally short to FDI in 1, and to TA in 1 patient (Table 1).

Analyzing the combined data for CIDP and
AIDP patients, we observed the following patterns of latency abnormalities: (a) CML and PML were both prolonged (16 extremities); and the calculated CMCT was either normal (4 extremities), prolonged (8 extremities); or shortened (4 extremities). (b) A prolonged CML associated with a normal PML resulted in an abnormally long CMCT (4 extremities) or a CMCT approximately at the upper limit of normal. (c) A normal CML with a prolonged PML produced an abnormally short CMCT (3 extremities). (d) A shortened or prolonged CMCT also resulted from both a normal CML and PML (3 extremities).

The few patients in whom we were unable to elicit a MEP either cortically or peripherally belonged to the clinically more severely affected group. In the CIDP group, patient 9 revealed a severely prolonged CMCT (mean plus 8 standard deviations) to the right FDI with a failure to evoke a paraspinal MEP on the left in the presence of a similarly prolonged CML (Table 1).

## DISCUSSION

In patients with hereditary demyelinating motor and sensory neuropathies, magnetic stimulation generally showed a normal CMCT. However, Mano et al. noted a markedly prolonged CMCT in 2 of 13 patients without clinical signs of upper motor neuron involvement. In our patients with acquired IDP, we obtained MEP from FDI and TA muscles by TMS and PMS and observed several patterns of latency abnormalities. CML or PML, or both, were prolonged, and the calculated CMCT was either normal, prolonged, or even shortened, depending on the relationship between the measurements of CML and PML. Moreover, in some
patients, an abnormal CMCT was the only indicator for a conduction abnormality in an extremity. As magnetic stimulation over the spine results in activation of the spinal nerve in the intervertebral foramen, the CMCT includes conduction delay in nerve roots. Thus, a mildly prolonged CMCT most likely reflected abnormal conduction in the proximal nerve segments rather than in the upper motor neuron pathways. This proximal segment of the lower motor neuron is a known site of predilection in demyelinating disease.

A markedly prolonged CMCT may be due to additional CNS demyelination, but the amount of prolongation required is not clear if one considers the reduced excitability of the lower motor neuron in IDP as known from F-wave studies. In our CIDP group, we found only 1 patient (no. 9) with an extraordinary CMCT prolongation.

In a surprising number of patients, we encountered an abnormally shortened CMCT, indicating an excessively prolonged PML. This finding could be explained by a lower stimulation threshold of slowly conducting fibers associated with a relative refractoriness of faster fibers for direct magnetic stimuli over the spine, while these faster fibers still conducted transsynaptic impulses after cortical stimulation. The presumed suboptimal excitability of the proximal nerve would be pathological because in normal controls the fastest fibers are always stimulated first.

Transcranial and paravertebral magnetic stimulation is useful to demonstrate proximal conduction defects in inflammatory demyelinating polyneuropathies. Its clinical role for longitudinal studies remains to be determined. It may be helpful for identifying subgroups with a markedly prolonged CMCT, suggestive of CNS demyelination, who may follow a different clinical course.

REFERENCES


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