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Motor Evoked Potentials Reveal Subclinical Upper Motor Neuron Dysfunction In Progressive Muscular Atrophy: Two Case Reports

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Abstract:
The diagnosis of Amyotrophic Lateral Sclerosis remains challenging in early disease stages, particularly in patients lacking overt clinical signs of upper motor neuron involvement. In this setting, motor evoked potentials (MEPs) may provide valuable neurophysiological evidence of subclinical corticospinal dysfunction. We report two patients initially presenting with progressive muscular atrophy (PMA), in whom MEP studies demonstrated central motor
pathway abnormalities suggestive of upper motor neuron involvement, thereby supporting early diagnostic reclassification toward ALS. These observations highlight the diagnostic contribution of MEPs in motor neuron disorders, especially when conventional clinical examination and neuroimaging are non-contributory.
Keywords: *Amyotrophic lateral sclerosis; motor evoked potentials; MEPs; progressive muscular atrophy; upper motor neuron; electroneuromyography.*

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I. Introduction:

Amyotrophic Lateral Sclerosis is a progressive neurodegenerative disorder characterized by combined degeneration of upper and lower motor neurons **[1]**. Diagnosis relies on clinical and electrophysiological evidence of both central and peripheral motor neuron involvement according to established international criteria.

However, some patients initially present with isolated lower motor neuron manifestations, corresponding to a phenotype of progressive muscular atrophy (PMA), without clinically detectable pyramidal signs. Such presentations may delay diagnosis, particularly when magnetic resonance imaging (MRI) findings are unremarkable during the early stages of the disease **[2]**.

Electroneuromyography (ENMG) is the cornerstone investigation for documenting lower motor neuron degeneration but provides limited information regarding corticospinal tract involvement. In contrast, motor evoked potentials (MEPs), obtained using transcranial magnetic stimulation, allow assessment of central motor conduction and may reveal subclinical upper motor neuron dysfunction through abnormalities in central motor conduction time **[3]**.

We report two clinical observations illustrating the usefulness of MEPs in detecting early corticospinal tract involvement in patients initially diagnosed with PMA.

Case Reports

Case 1

A 28-year-old man presented with progressive right-sided weakness evolving over several months. Neurological examination revealed right hemiparesis of predominantly peripheral pattern without objective pyramidal signs. Deep tendon reflexes were preserved, and neither Babinski sign nor spasticity was observed. Cranial nerve examination was normal.

Brain and spinal MRI findings were unremarkable.

ENMG demonstrated diffuse lower motor neuron involvement with fasciculations and active denervation changes consistent with diffuse neurogenic motor impairment. In the absence of clinical upper motor neuron signs, an initial diagnosis of progressive muscular atrophy was considered.

Three months later, the patient developed progressive weakness involving all four limbs with worsening functional disability. MEP assessment was subsequently performed.

MEP studies demonstrated prolonged central motor conduction times and increased transcallosal latencies, indicating corticospinal tract dysfunction. These findings provided objective evidence of subclinical upper motor neuron involvement and supported the diagnosis of Amyotrophic Lateral Sclerosis.

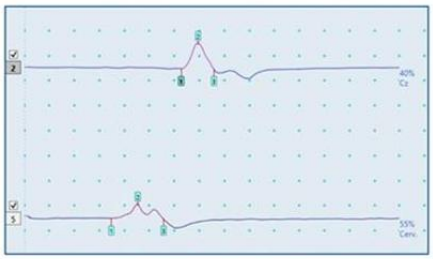
Case 2

A 64-year-old woman presented with progressive weakness of all four limbs evolving over six months and associated gait impairment. Her medical history was notable for cervical spondylotic myelopathy.

Neurological examination demonstrated tetraparesis predominantly affecting the upper limbs without definite pyramidal signs. No spasticity or Babinski sign was detected. Sensory examination was normal.

ENMG revealed diffuse neurogenic abnormalities with bulbar resting activity, consistent with diffuse lower motor neuron involvement. However, the absence of overt upper motor neuron signs complicated the diagnosis of ALS, particularly in the context of concomitant cervical spondylotic myelopathy, which represented a potential confounding factor.

MEP evaluation revealed prolonged central motor conduction times compatible with corticospinal tract involvement. These neurophysiological abnormalities strengthened the diagnostic hypothesis of Amyotrophic Lateral Sclerosis despite the paucity of clinical upper motor neuron findings.



Temps de conduction :

N	Stim. zone	Point de stimulation	Latence (ms)	Amplitude (mV)	Durée (ms)	Temps de conduction (ms)
Abducteur Vème doigt, ULNAIRE C8-T1, D						
2	Cortical	Cz	25,0	2,03	5,25	11,2
5	Médullaire	Cervical	13,9	1,11	8,3	

Figure 1: MEPs in a patient with progressive muscular atrophy (PMA) showing prolonged latencies consistent with subclinical upper motor neuron involvement (Case 1).

Figure 2: Technical demonstration of MEP recording from the abductor pollicis brevis muscle/median nerve (Case 2)

A. Cortical stimulation at the Cz point

B. Cervical spinal stimulation level

II. Discussion

Early diagnosis of Amyotrophic Lateral Sclerosis remains a major challenge, particularly in lower motor neuron-predominant phenotypes in which upper motor neuron signs may be absent or delayed **[4]**.

Patients presenting with PMA have historically been considered to exhibit isolated lower motor neuron degeneration. Nevertheless, accumulating evidence suggests that a substantial proportion of these patients subsequently develop clinical or electrophysiological features consistent with ALS, supporting the concept of a disease spectrum rather than distinct nosological entities **[5]**.

ENMG remains the reference neurophysiological investigation for identifying active and chronic denervation. However, it does not directly evaluate corticospinal tract integrity. MEPs therefore represent a valuable complementary diagnostic modality by enabling assessment of central motor conduction **[4]**.

Prolongation of central motor conduction times may reflect subclinical corticospinal dysfunction even before overt pyramidal signs become clinically apparent. In both of our cases, MEP abnormalities provided objective evidence of upper motor neuron involvement despite inconclusive clinical findings **[3]**.

In the first observation, MEP findings contributed to reclassification of PMA as ALS. In the second case, MEPs helped support the diagnosis in a clinically challenging context involving cervical spondylotic myelopathy.

The diagnostic contribution of MEPs appears particularly relevant in patients presenting with **[6]**:

- progressive pure motor syndromes;
- ENMG findings suggestive of lower motor neuron degeneration;
- absence of definite pyramidal signs;
- or coexisting conditions potentially obscuring corticospinal involvement.

Integration of MEPs into the neurophysiological workup of suspected motor neuron disease may therefore reduce diagnostic delay and facilitate earlier therapeutic intervention and multidisciplinary management.

III. Conclusion:

Motor evoked potentials constitute a valuable neurophysiological tool for detecting subclinical upper motor neuron involvement in patients presenting with progressive muscular atrophy **[2]**.

Through these two observations, MEPs provided important diagnostic evidence supporting early identification of Amyotrophic Lateral Sclerosis. Their use in selected clinical settings may improve diagnostic accuracy and optimize patient management during the early stages of disease progression.

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