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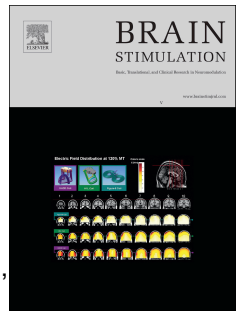

Modulation of human corticospinal excitability by paired associative stimulation in patients with amyotrophic lateral sclerosis and effects of Riluzole

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ACCEPTED MANUSCRIPT

Modulation of human corticospinal excitability by paired associative stimulation in patients with amyotrophic lateral sclerosis and effects of Riluzole

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Abstract:

BACKGROUND: Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disease that causes an impairment in both the upper and lower motor neurons. The recent description of numerous non-motor signs points to an involvement of the neocortex networks that is more complex than was previously believed. Paired associative stimulation (PAS), a combination of transcranial magnetic stimulation (TMS) and peripheral nerve stimulation, can enhance motor output in the contralateral hand through an NMDA-mediated sensorimotor mechanism.

OBJECTIVE: To describe the effects of PAS on ALS patients before and after Riluzole intake compared with healthy subjects.

METHODS: PAS was used to detect differences between 24 newly-diagnosed ALS patients and 25 age-matched healthy controls. MEP amplitude from the abductor pollicis brevis was considered before PAS, immediately after (T0) and after 10 (T10), 20 (T20), 30 (T30) and 60 (T60) minutes. Statistical significance was calculated using RM-ANOVA.

RESULTS: In healthy controls, PAS significantly increased MEP amplitude at T10, T20 and T30 ($p<0.05$). In ALS patients, a significant increase in MEP amplitude was also observed after 60 minutes ($p<0.05$), thus demonstrating NMDA-mediated enhanced facilitatory plasticity. After two weeks of riluzole intake, no MEP amplitude increase was evident after PAS at any time point. In three monomelic-onset ALS patients, sensorimotor facilitation was evident only in the hemisphere corresponding to the affected side and appeared in the opposite hemisphere when the patients manifested contralateral symptoms.

CONCLUSIONS: PAS may be considered a useful tool when investigating NMDA-mediated neocortical networks in ALS patients and the modulation of such networks after anti-glutamatergic drug intake.

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