

EN: Alteration of cortical oscillatory activity contributes to abnormal movement patterns in post-stroke patients.

FR : L'altération de l'activité oscillatoire corticale participe à la détérioration des patterns du mouvement du membre supérieur chez le patient post-AVC.

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Abstract

Background: Spastic co-contraction is a motor disabling form of muscle over-activity occurring after stroke, contributing to the limitation of the active movement and motor impairment. To date, the cortical mechanisms underlying spastic co-contraction remain to be more fully elucidated. The cortical activity involved in a motor task is usually investigated by movement-related beta desynchronization, which has been linked to the cortical excitability [1]. Indeed, abnormalities in cortical oscillatory activity could be a key mechanism underlying motor impairment after stroke [2]. This study aimed to investigate the association between cortical oscillations and the alterations of the kinematic and muscle activation patterns after stroke.

Methods: Fifteen post-stroke patients and nine healthy controls were included. They performed active elbow extensions during which we assessed elbow kinematics, elbow flexors and extensor electromyographic-based muscles activation, and cortical oscillatory activity by computing the movement-related beta desynchronization from electroencephalography.

Results: In stroke patients, movement-related beta desynchronization was decreased by 28.6 ± 8.3 % during elbow extension movement ($t_{22} = 4.66$, $P < 0.05$, $g = 1.88$). We found an association between movement-related beta desynchronization and elbow flexors activation during the active elbow extension in stroke patients ($r = 0.6$, 95% CI [0.13;0.85]). Compared to

healthy subjects, stroke patients exhibited greater muscle activation level ($t_{22} = 4.46$, $P < 0.05$, $g = 1.8$) and significant alterations of elbow kinematics ($t_{22} = -7.62$, $P < 0.05$, $g = -3.07$).

Discussion: In line with earlier studies, our results showed significant differences in kinematic variables between post-stroke and healthy participant, and a pattern of muscle overactivity on both elbow flexors and extensor in post-stroke patients reflecting a considerable effort to perform an elbow extension [3,4]. Our results concur to reveal a specific encoding of antagonist muscles activity through beta cortical oscillations and reinforces the fact that an alteration of beta cortical oscillations could be a key mechanism contributing to spastic co-contraction in post-stroke. Our study pleads for EEG-based assessment of cortical oscillatory activity to further understand and characterize neuromuscular plasticity induced by therapeutic interventions.

Bibliography :

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