The modulation of corticomuscular coherence reflects alteration of the central-peripheral network after stroke.

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Résumé

Introduction:
Stroke affects brain tissue, which leads to multiple rearrangements of both the central nervous and the peripheral neuromuscular systems, resulting in an alteration of motor network connectivity. By studying correlations between brain and muscle electrophysiological signals (EEG and EMG, respectively), also called corticomuscular coherence (CMC), previous studies have provided evidence of modifications in the brain-muscle communication for post-stroke subjects compared to controls. However, to date there is no consensus on whether CMC is lower or higher in stroke versus control subjects. Moreover, despite CMC provides information on the nature of the central command sent to synergistic muscles and reflects central-peripheral network alteration, it remains to investigate the functional role of CMC with respect to the loss of motor function after stroke. In view of increased spastic co-contraction occurring in post-stroke spastic paresis syndrome, this study investigates the effect of stroke on CMC computed between EEG and EMG from both agonist and antagonist muscles, concomitantly with the loss of motor function during active elbow extension. An alteration of CMC especially with antagonist muscles was expected in stroke subjects. Such finding would provide evidence for the involvement of CMC in the functional reorganization of the central-peripheral network, and could reflect an alteration of the central-peripheral network after stroke.

Method:

10 chronic stroke subjects (Stroke: 8 M/2 F) and 9 healthy subjects (Control: 4 M/5 F) participated in this study. Kinematic, EEG and EMG data were recorded during 20 active elbow extension movements performed at a self-spontaneous speed with the paretic upper limb for Stroke and non-dominant for Control, to quantify i) the active range of motion, ii) spastic co-contraction and iii) CMC with agonist and antagonist muscle groups in the 'beta' frequency band (β, 13-31 Hz) (Fig 1). For statistical comparisons, effect size (ES) was calculated for each variable using Hedges'g. A significant difference was considered as an ES

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with confidence interval not including zero.

Results:

Compared to Control, Stroke showed a lower active range of motion (62.5 vs. 95.5 degrees, ES = 2.75 [2.10:4.48]) and a higher level of spastic co-contraction (10.9 vs. 2.4%, ES = -1.62 [-3.33:-1.19]). β-CMC with both the agonist and antagonist muscles was higher in Stroke than in Control (respectively: 0.56 vs. 0.18, ES = -0.91[0.17:1.92] and 0.52 vs. 0.19, ES = -1.05 [0.38:2.03]) (Fig 2).

Conclusions:
This study highlighted an increased β-CMC during active elbow extension after stroke, suggesting a greater share of direct central drive sent to the synergistic muscles. This finding could reflect the functional reorganization of the central-peripheral network which contributes to the upper limb motor control. Contrary to our hypothesis, the observed modulation of CMC in stroke subjects occurred for both agonist and antagonist muscles. This may reflect a global reorganization of this central-peripheral network contributing to the muscular overactivity and to the loss of muscular selectivity after stroke. This work provides additional evidences of motor network reorganization after stroke, reinforcing the functional importance of CMC in the regulation of motor function.

Mots-Clés: Brain Injury, Electromyography, Electroencephalography, Motor Control, Motor Network.