Advancing Cortico-muscular Coherence Analysis in Stroke: EEG Coupled with Motor Neuron Spike Train Data

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Summary

Understanding corticomuscular connectivity is crucial for assessing motor control and recovery after neurological injury. Conventional Cortico-muscular Coherence (CMC) analysis using surface EMG and EEG is limited by signal contamination. To address this, we applied HDEMG decomposition to extract motor neuron (MN) spike trains and compute MN-based CMC in stroke and control participants. We further classified MNs into coherent and non-coherent groups based on CMC topology. In stroke, the paretic side showed lower and more dispersed CMC peaks compared to the unaffected side and controls. Coherent MNs correlated with EMG CMC peaks, while non-coherent MNs had lower or atypically located peaks. This novel approach enables non-invasive, *in vivo* CMC assessment at the MN level.

Introduction

CMC reflects motor cortex-muscle connectivity, offering insights into neuromuscular control. Surface EMG-based CMC is limited by peripheral contamination, whereas HDEMG decomposition enables direct neural drive measurement via extracting MN spike trains. EEG-MN coherence may reflect monosynaptic corticospinal connections [1], but its role in lower-limb,primarily polysynaptic pathways, and stroke remains unclear. This study advances non-invasive corticospinal assessments, providing new insights into recovery mechanisms.

Methods

Twenty persons with stroke (12 female, age 59.1 $\pm$ 8.7 years) and 20 controls (18 female; 62.0 $\pm$ 10.7 years) participated in the study. The protocol included clinical assessments (dorsiflexion strength, walking independence, spasticity, and motor recovery; stroke group only) and isometric dorsiflexion tasks. Participants performed three maximal voluntary contraction (MVC) trials (three repetitions, 60s rest in between) followed by five 20s submaximal holds at 20% MVC with visual feedback. HD-EMG was recorded from tibialis anterior (TA) synchronously with EEG from 21 electrodes covering sensorimotor cortex. EMG processing included filtering (Butterworth, 20–500 Hz), MN spike train decomposition, cummulative spike train (CST) computation, and bipolar EMG derivation. EEG preprocessing involved filtering (Butterworth, 5–100 Hz), rereferencing, and artifact removal. CMC was computed in the β-band (15–30 Hz) for bipolar EMG, MN spike trains, and CSTs, with z-transform normalization. CST-CST coherence in the δ-band (0.5–4 Hz) assessed common synaptic input within MN groups. Coherent MNs were identified based on significant CMC, considering peak coherence location for controls only. Ethical approval was obtained from the Human Subjects Ethics Sub-Committee of the Hong Kong Polytechnic University (HSEARS20210320003) and the Joint Chinese University of Hong Kong - New Territories East Cluster Clinical Research Ethics Committee (2022.309-T).

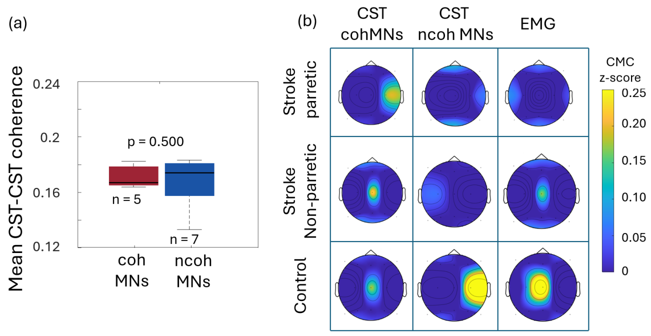

Figure 1: (a) Mean δ band CST-CST coherence for coherent (cohMNs) and non-coherent (ncohMNs) motor neuron (MN) groups for n control subjects (n = right-side recordings with >3 decomposed MNs). The p value determined by Wilcoxon signed rank test. Here n corresponds to all the control subjects recorded on the right side, with more than 3 decomposed MNs. (b) Z-transformed CMC topographies for one stroke participant (paretic: right, non-paretic: left) and one control (right side).

Results and Discussion

We found no significant difference in CST-CST coherence between coherent and non-coherent MN groups (fig.1a), suggesting both receive a similar proportion of common synaptic input. This indicates that coherence differences at the MN-EEG level may arise from selective cortical contributions rather than peripheral drive differences. Topographic plots of one stroke and one control participant (fig.1b) show peak CMC around central (Cz) electrode, consistent with previous findings on sensorimotor connectivity in TA [2]. CMC distributions on the non-paretic side resembled controls, while lower coherence on the paretic side suggests disrupted corticospinal drive, reduced cortical excitability, and potential motor network reorganization, possibly involving compensatory ipsilateral cortical activity.

Conclusions

TA MN-driven CMC provides insight into the coupling between motor cortex activity and MN firing in both stroke and controls. This approach may help in characterizing post-stroke neuroplasticity by detecting corticospinal connectivity changes at the individual MN groups level.

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