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Differential patterns of TMS-evoked activation of the sensorimotor network following stroke: a case series

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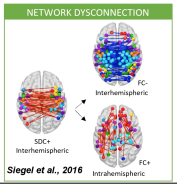
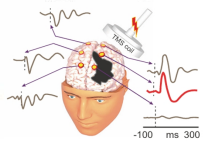
INTRODUCTION

Impairment after stroke arises from direct anatomical damage and remote structural and functional abnormalities in large-scale resting state networks **(Fig.1 left)**. A promising non-invasive technique to study brain connectivity with high temporal resolution is transcranial magnetic stimulation combined with electroencephalography (TMS-EEG). This methodology probes cortical reactivity through the registration of TMS-evoked cortico-cortical potentials (TEPs) **(Fig.1 right).**



Siegel et al., 2016

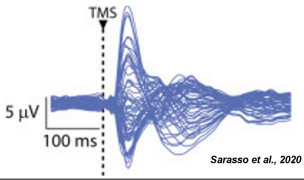


Massimini et al., 2023

AIM

A **sleep-like response** of functionally altered regions has been proposed as a marker of altered cortical dynamics with negative prognostic power. **(Fig. 2)**

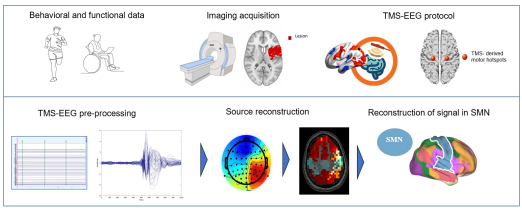
In this study, we investigated whether we could identify a sleep-like response in chronic stroke patients with severe residual motor impairment. Crucially, after stimulation of the motor cortex (M1), we assessed whole-brain TEPs and the evoked activation of the sensorimotor network (SMN).



Sarasso et al., 2020

METHODS

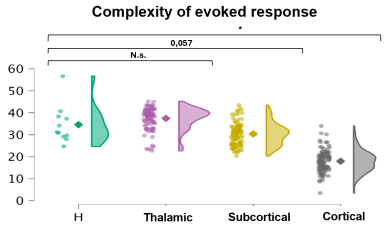
For all patients, TMS was applied over the ipsilesional and contralesional M1 while registering high-density EEG. Source reconstruction was used to estimate the distribution of activity which was mapped onto a functional parcellation atlas to **quantify the activation of the SMN (Fig.3)**. The results were compared with normative data from 12 healthy controls.



RESULTS

We recruited **three chronic stroke patients** with motor impairment. For each patient we collected demographic and clinical variables along with structural MRI scans **(Fig. 4)**.

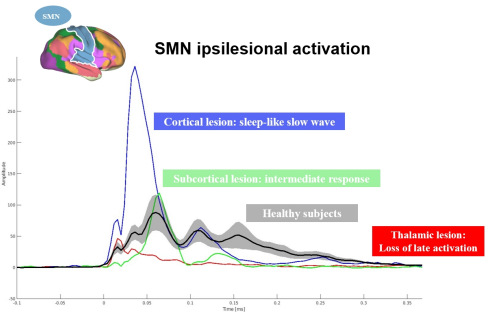
The first patient presented right hemiparesis after a left frontal lesion involving M1. The second patient showed left hemiplegia after a right corona radiata lesion. The last patient presented right hemiplegia after a left thalamic lesion.



Compared to healthy controls, ipsilesional **TMS-evoked potentials** revealed distinct activation profiles for patients with cortical and subcortical lesions, but **no clear differences were observed in the patient with thalamic damage (Fig.5).**

SMN profiles of activation were **altered in all patients compared to healthy controls.** SMN activation patterns varied according to lesion location.

Cortical damage determined an excessive early activation of the SMN, producing a local prominent **sleep-like cortical response**. Thalamic damage was associated with the loss of the physiological late activation of the SMN, possibly related to to sensorimotor integration. Finally, subcortical damage produced an intermediate pattern **(Fig. 6).**



DISCUSSION

Only **perilesional stimulation following extensive cortico-subcortical damage elicited a sleep-like response**, despite comparable behavioral deficits. **Network-level evaluation of TEPs** enabled a comprehensive characterization of regions potentially contributing to post-stroke impairment, including spatially distant yet functionally relevant areas. Our findings revealed **differential activation within the SMN**, possibly reflecting **lesion-specific effects**. Future studies could build on these results to explore mechanisms extending beyond the sensorimotor system.