

Neuromodulation of induced beta rhythms using transcutaneous spinal cord stimulation

Blanca Delgado Bonet, Juan Pablo Martínez Cortés, Jaime Ibáñez Pereda

BSiCoS, Instituto de Investigación en Ingeniería de Aragón (I3A), Universidad de Zaragoza, Mariano Esquillor s/n, 50018, Zaragoza, Spain.

Introduction

Spinal cord stimulation shows therapeutic potential in several neurological disorders [1,2], but its mechanisms of action remain poorly understood. In particular, little is known about how it interacts with endogenous neural

oscillations. Here, we use transcranial magnetic stimulation (TMS) to induce controlled beta-band activity (13-30 Hz) [3] and investigate whether and to what way transcutaneous spinal cord stimulation (tSCS) modulates these rhythms.

H₁: tSCS modulates endogenous neural rhythms when frequency-locked to the ongoing oscillatory activity, and its effects depend on the relative phase between the stimulation and the oscillation.

Methods

Six healthy participants (28±6 years; 2 females) performed mild isometric wrist-flexion contractions. Force, surface and intramuscular electromyography (sEMG and iEMG) of the flexor carpi radialis and ulnaris (FCR and FCU), and 63-channel electroencephalography (EEG) were recorded. Single-pulse TMS (90% motor threshold, monophasic, antero-posterior) was applied over the cortical representation of the FCR to induce beta-band oscillations. Cervical tSCS (90% motor threshold, 5kHz carrier frequency, 1 ms pulse width, 40 pulses) followed the TMS pulses, targetting four equidistant phases of the evoked beta.

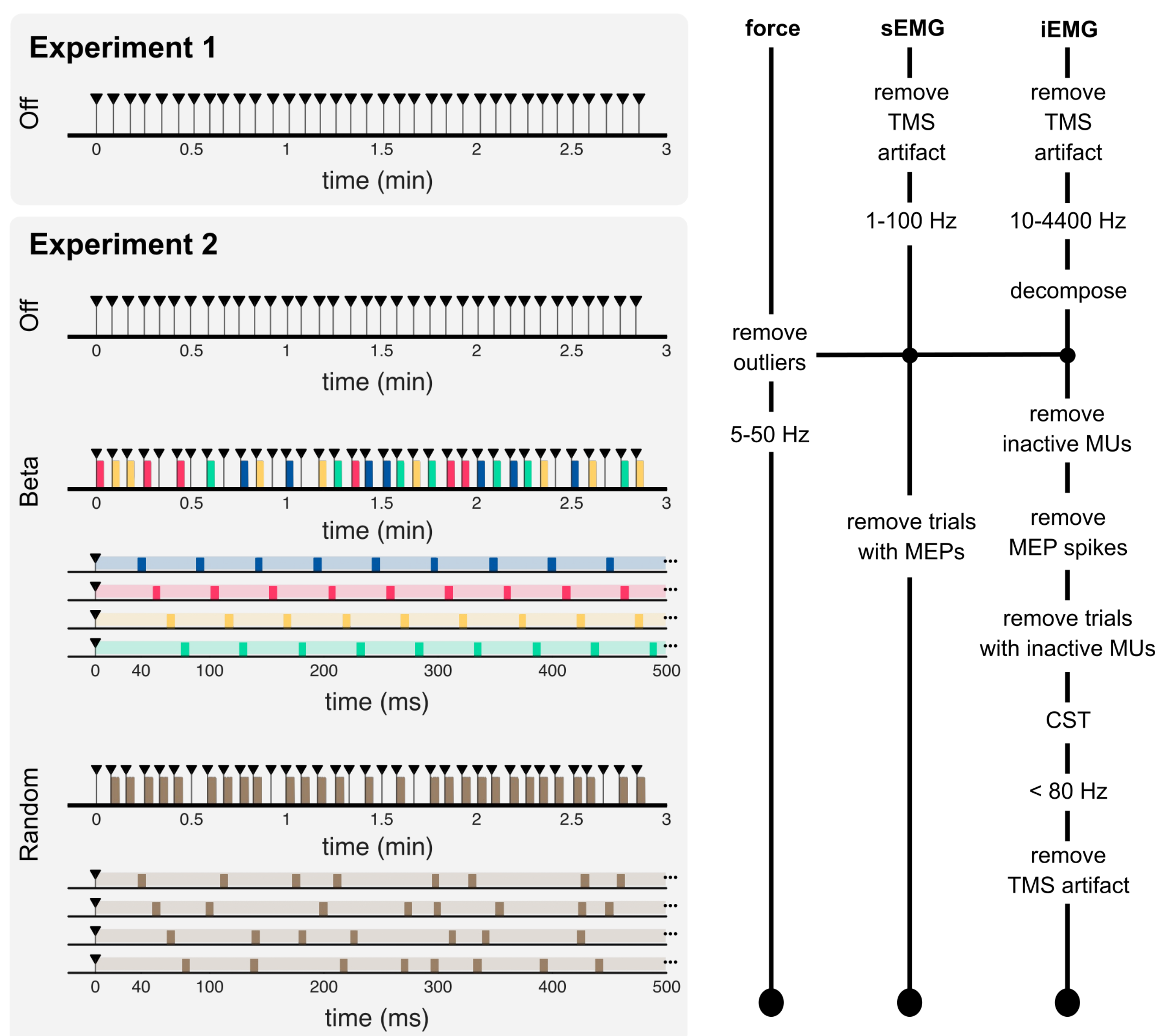


Figure 1. Transcranial magnetic stimulation (TMS) to evoke beta and transcutaneous spinal cord stimulation (tSCS) to modulate it. Top: participant performing the experiment. Bottom left: experimental protocol consisting of TMS pulses only ('Off') or paired with trains of tSCS ('Beta' and 'Random') targetting four phases. Bottom right: preprocessing pipeline.

Conclusions

1. TMS elicits **phase-locked beta oscillations**, further characterized by a consistent number of cycles **transmitted up to the force level**.
2. tSCS may enhance beta, possibly due to increased excitability [4].

Results

TMS pulses elicited transient beta-band activity peaking at 55-86 ms, 22-23 Hz, that were observable at three levels: motor unit discharge timings, muscle activity and force output. Beta responses were strongest in sEMG (likely reflecting the contribution of a larger motor unit population), reduced in cumulative spike trains (CST) derived from iEMG (probably due to its limited sampling of the active motor units, which enables analysis of individual neural firing patterns), and smallest in force (still detectable despite the low-pass filtering properties of muscle contraction).

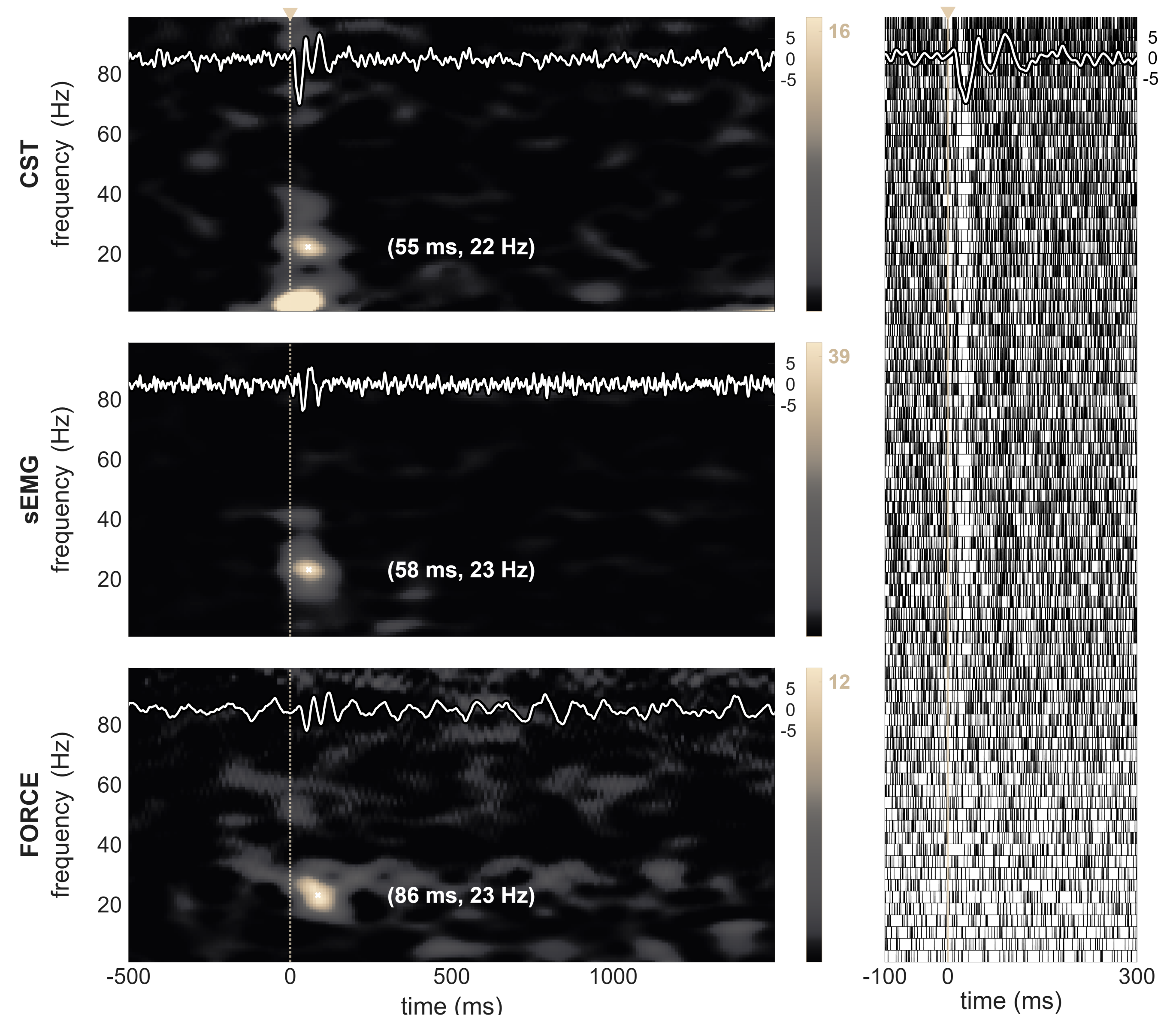


Figure 2. Elicited beta activity. Left: average spectrogram (normalized to pre-stimulus baseline; saturated to peak beta activity) and z-scored average time-domain responses for cumulative spike train (CST), surface electromyography (sEMG), and force. Right: zoom to CST response and raster plot (spikes pooled across trials for each motor unit).

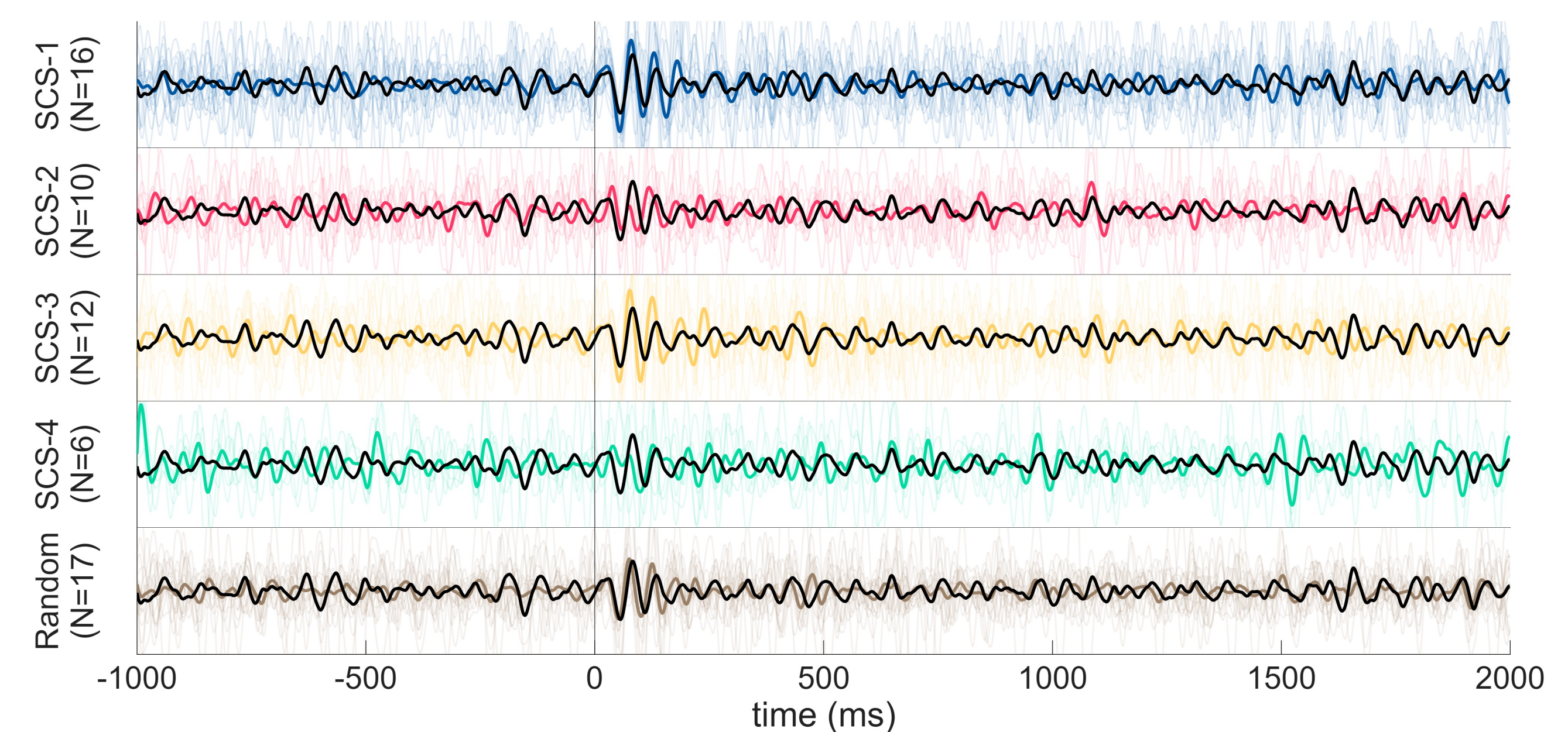


Figure 3. Modulation of beta activity. Averaged, z-scored, beta-filtered force from an example subject after transcranial magnetic stimulation alone (black, Experiment 1) or paired to a train of spinal cord stimulation delivered at subject-specific beta frequency – targetting four phases of the beta cycle – or random frequency (colors, Experiment 2).

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- [4]. AL'JOBOORI, Y.; et al. Frontiers in Neuroscience. 2021.

