

Additional Q&As about Dr. Massimini's webinar:

Q: Do you see that EnTMS-EEG could be used in the future to guide rTMS depression therapy as you showed now the data for stroke?

Dr. Massimini's answer: In stroke there seems to be a EnTMS-EEG signature showing a specific alteration in cortical circuits: a sleep-like, simple response in the perilesional area (Sarasso et al., Brain 2020; Tsherpel et al., Brain 2020). I'm not aware of such a specific TMS-EEG signature for depression. Nonetheless, a reliable quantification of the immediate (first 20 milliseconds) EEG response to TMS would represent an invaluable guide for depression therapy. First, one could set the stimulation parameters of the treatment (intensity, angle of the coil) based on their actual impact on cortical neurons (which cannot be known a priori). Second, it would be possible to have a clear read-out of the actual impact that the treatment has on cortical excitability (by comparing early TEPs at the beginning of the session with those at the end).

Q: Are there any enTMS-EEG data on patients diagnosed with brain death, or "postmortem" data.

Dr. Massimini's answer: No, but the closest condition in which TMS-EEG data have been collected is the post-anoxic vegetative state, otherwise called "apallic syndrome". In this condition, in which most cortical neurons are dead (although some subcortical structures such as the brainstem are still functional), TEPs are completely flat (Gosseries et al., Brain Stim 2015); the same happens when we target TMS exactly on a focal structural lesion, such as a Stroke (Sarasso et al., Brain 2020). These results make sense biologically (no cortex under the stimulator -> no EEG response to TMS) and have important methodological implications: when appropriately controlled for, sensory-evoked responses do not significantly contribute to TEPs.