Modulation of TMS-evoked transcallosal inhibition by voluntary hand movement

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Purpose

Interhemispheric connectivity plays a key role in behavioural deficits in stroke and neurodegenerative disease, but a major debate questions whether homologous regions excite or inhibit each other. Linking inhibitory phenomena in the motor cortex to similar interactions in other cortical regions is a research imperative, and may be possible through measurements of electrophysiological connectivity using EEG or MEG. One promising signal is the post-movement-beta-rebound (PMBR), which is thought to reflect local inhibitory processing. In this study, we evaluate potential direct evidence for increased transcallosal inhibition occurring in the same period as PMBR, immediately following a voluntary movement.

Methods

We used the transcranial magnetic stimulation (TMS) paradigm called ipsilateral silent period (iSP), in which a unilateral hand squeeze is temporarily interrupted by a magnetic pulse to ipsilateral motor cortex, reflecting transcallosal inhibition of the contralateral motor cortex. Twenty, right-handed, participants were asked to make a button-press with the non-squeezing hand, which then triggered the TMS to randomly stimulate at 8 different time points, meant to overlap with the time course of PMBR. Additionally, to measure the time course of the PMBR, six different participants were asked to make the same button-press while their neural activity was measured with MEG.

Results

The extent of the iSP was not significantly modulated after voluntary movement in either the dominant or non-dominant hemisphere. Motor evoked potentials, reflecting excitability of the cortex controlling the voluntary movement, were increased 10 ms after movement offset but not at any subsequent timepoints. MEG recordings confirmed that the expected pattern of beta desynchronization and rebound was present for the button press movement.

Conclusion

Ipsilateral silent period does not appear to be significantly modulated following voluntary movement, suggesting that it does not share a common mechanism with the beta rebound observed in EEG and MEG recordings.