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Corticomotor plasticity underlying the priming effects of motor imagery on strength performance

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INTRODUCTION:

Neural plasticity is capacity of neurons to reorganize their connectivity in response to cognitive and behavioral demands. Neural plasticity allows short-term modulations of strength through improved cortical gain over motor units. There is now convincing evidence that covert motor strategies such as motor imagery (MI) and action observation (AO) contribute to limit strength loss and prevent maladaptive plasticity [1][2][3]. The main objective of this study is to extend current understandings of the neurophysiological processes underlying strength gains as a result of embedded MI or AO within strength training sessions.

METHODS:

In a counterbalanced design involving three experimental conditions (AO, MI, Control) we recorded the total force, the integrated electromyograms of agonist and antagonist muscles (biceps brachii, triceps brachii and anterior deltoideus) and the ongoing electrical brain activity in 20 participants who performed 10 maximal isometric contractions of elbow flexor muscles against a force platform (10 s duration). During the one-minute inter-trial rest periods, participants then randomly performed: i) AO of the same strength task performed by an expert athlete, ii) MI of the task using first-person visual and kinesthetic imagery, and iii) AO of a video documentary about basketball shooting (Control).

RESULTS:

The linear mixed effect analysis carried on total force data revealed a CONDITION x MUSCLE interaction ($p= 0.0384$). Irrespective of the trial number, total force values were higher during MI compared to the Control condition ($p= 0.005$). Electromyograms showed a reduced relationship between triceps brachii activity and total force during MI. Also, the relationship between agonist electromyograms and the total force was greater during MI. Eventually, corticomotor connectivity, indexed from coherence between electric brain potentials and the electromyograms of the agonists within the alpha frequency range (8-12 Hz), was greater during MI compared to AO and Control. The topographical distribution of corticomotor connectivity values emphasized sensors located within the central and pre-central regions.

CONCLUSION:

Present findings provide new insights to the neurophysiological processes underlying strength gains in response to motor stimulation. The data showed improved strength performance only when MI was performed during the inter-trial recovery periods of strength training sessions. Topographical patterns of corticomotor connectivity provide evidence, for the first time, of the effect of MI training on corticomotor plasticity. Short-term corticomotor plasticity might thus represent an important underlying mechanism to the priming effects of mental training on strength performance.

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