



# Short-Term Immobilization Promotes a Rapid Loss of Motor Evoked Potentials and Strength That Is Not Rescued by rTMS Treatment



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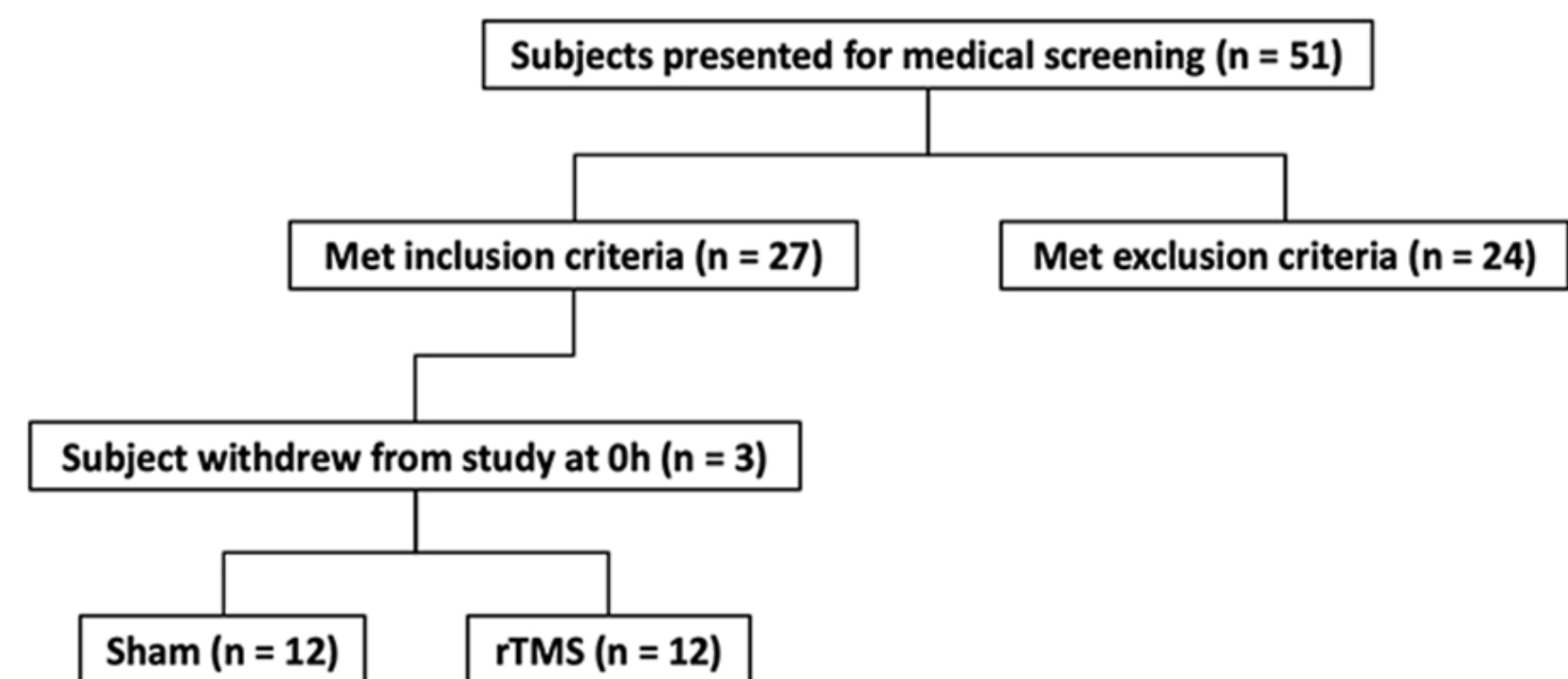
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## Background

- Immobilization of a limb prevents movement from injury, promoting loss of muscle strength and mass [1].
- Limb immobilization causes a decrease in excitability of motor cortex (M1) after 8 h: a concern if bed-bound, or older with reduced mobility [2].
- Limb immobilization promotes a decrease in the cortical thickness of the left M1 and reduces fractional anisotropy of white matter tracts associated with the right hemisphere M1, suggesting a reorganization of motor systems in the brain with immobilization [3].
- 20 Hz rTMS can significantly increase excitability in the motor pathway to the hand by increasing motor evoked potential (MEP) amplitudes [4].

**Aim: To determine the neurophysiologic basis of immobilization-induced skeletal muscle decline, and if 20 Hz rTMS to M1 can protect against it and facilitate cortical excitability.**

## Subjects



- 24 recreationally active young males participated. Subjects were (mean ± SEM) 20.7 ± 0.5 year, 69.1 ± 1.8 kg body mass, and had a BMI of 22.1 ± 0.5 kg/m<sup>2</sup>.
- Subjects were randomized into either a Sham or an rTMS group prior to data collection, as seen in the flow chart above.

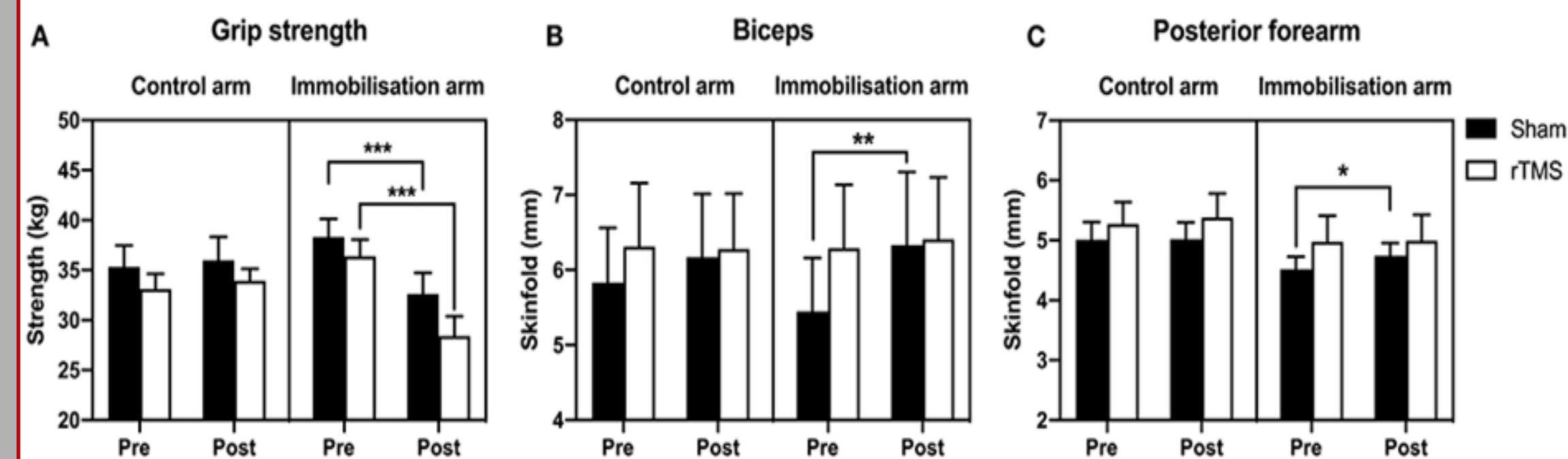
## Hypotheses

- Rapid declines in strength with immobilization will be underpinned by a loss of excitability within the motor pathway to the hand, indexed as a reduction in magnitude of MEPs.
- Stimulating M1 using 20 Hz rTMS would attenuate the decline of motor excitability and decline of skeletal muscle, which would have significant implications for prehabilitation or rehabilitation.

## Procedure

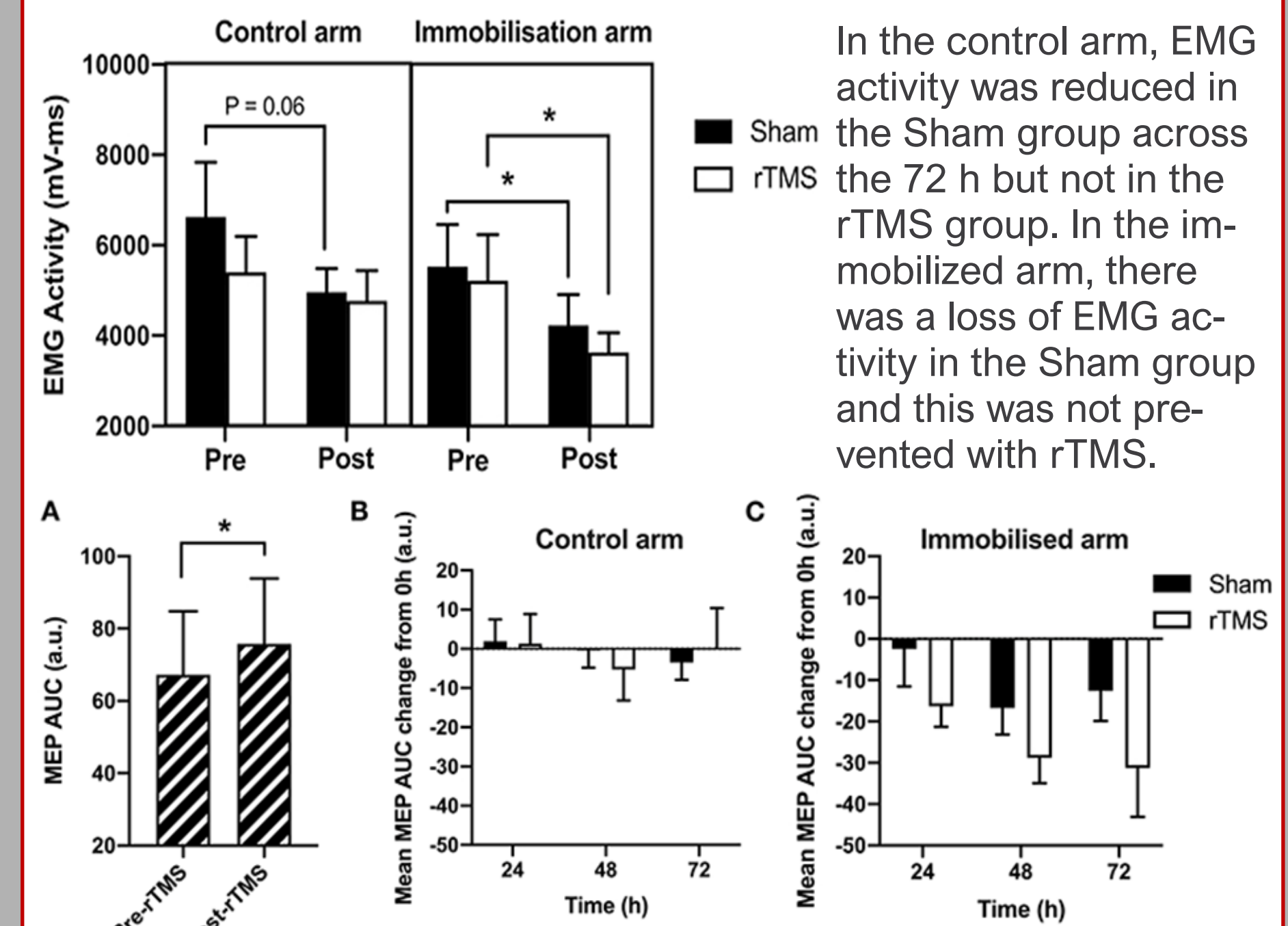
- Parallel design (Sham vs. rTMS groups) immobilizing the dominant arm using a shoulder sling for 72 hours. Before and after immobilization, maximal grip strength, volume-displacement plethysmography, skinfold-callipers, and circumference measurements were taken.
- Electromyography (EMG) of the FDI was taken with maximal grip strength. MEPs were recorded from left and right resting FDI via single-pulse TMS before immobilization, and at 24, 48, and 72 hours after immobilization.
- rTMS group received 6 × 1.5 s 30-pulse trains of 20 Hz biphasic rTMS with inter-train-intervals of 60s to the hand area of left M1 before immobilization, and at 24, 48, and 72 hours to promote cortical plasticity during immobilization.
- Sham group received an identical rTMS protocol, but the coil was held 3–4 cm away from the head. Cortical excitability was evaluated using MEPs from the FDI elicited by single-pulse TMS, at 0, 24, 48, and 72 hours.

## Effects of rTMS on Strength and Arm Composition



- Arm immobilization induced a significant decrease in grip strength in both the Sham group (10% loss) and rTMS group (22% loss) (A).
- In the Sham group, there was an increase in biceps skinfold (B;  $p < 0.01$ ) and posterior forearm skinfold (C;  $p < 0.05$ ) of the immobilized arm. Such changes in arm composition were not observed in the rTMS group.

## Effects of rTMS on EMG and MEP Activity



- There was a significant effect of rTMS on MEPs at 0 h (A).
- In the control arm, there was no significant change in MEP size across time or between Sham and rTMS (B).
- In the immobilized arm, there was a decrease in MEPs across time, which did not differ between Sham and rTMS (C).

## Conclusion

- Significant reduction in MEPs within 48 hours of immobilization.
- Whilst it enhanced motor excitability at baseline, 20Hz rTMS did not protect against immobilization-induced loss of motor excitability, loss of EMG activity, or maximal grip strength.
- rTMS may have modulated factors such as fluid retention or fat accumulation during immobilization, as there was no increase in skinfold thickness at the biceps and posterior forearm following rTMS.

### References:

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