Effects of Artificially Induced Ischemia on Maximal Voluntary Plantar Flexion

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Summary

Artificially induced ischemia has been shown to inhibit Ia-afferents prior to motor axons. However, it is unclear how voluntary contractions are affected by the time of H-reflex inhibition. Participants performed maximal isometric contractions before, during, and after ischemia. By the time of reflex inhibition, torque and rate of torque development were reduced while M-wave remained unchanged. Discomfort during ischemia might have affected voluntary contractions.

Introduction

Artificially induced ischemia typically inhibits Ia-afferents before motor axons [1] making it an interesting tool to investigate motor function without short-latency stretch reflex activity. The aim of this study was to assess how maximal voluntary contractions are affected by ischemia at the time of reflex inhibition.

Methods

Eight healthy participants (30±5y, 172±6cm, 66.8±9.3kg, 4 male) were seated on a dynamometer with their knees extended, hip at 120°, and ankle in neutral position (90º). An inflatable cuff with automatic pressure regulation was placed on the distal thigh, centered 12.7cm above the patella. Personal torniquet pressure (PTP) was defined as 105% of individual limb occlusion pressure. Bipolar electromyography (EMG) of the Soleus muscle and electrical stimulation of the tibial nerve were used to elicit maximal H-reflex (H) and M-wave (M). Before occlusion, every minute during occlusion, and after reperfusion, participants underwent two stimulations (100% H and 120% of M) and discomfort rating from 0 (no discomfort) to 10 (maximal discomfort). Occlusion was sustained until H was inhibited. Peak-to-peak amplitudes of H and M before occlusion (PRE), immediately after inflating the cuff (I0), at 25% (I25), 50% (I50), 75% (I75), and 100% (I100) of individual time to reflex-inhibition, as well as at minute one (R1) and five (R5) of reperfusion were used for statistical analysis. Participants performed three maximal voluntary isometric contractions (MVIC) at PRE, I0, I100, and R5. The highest peak torque (pT) and peak rate of torque development (pRTD) of the three MVICs at each time point were used for analysis. Peak root-mean square soleus EMG during MVIC (rmsEMG) was calculated. Repeated measures ANOVAs were used to analyze differences within participants and simple contrasts were used for pairwise comparisons. Alpha-level was 0.05 and Bonferroni correction was used for contrasts. Data is presented as mean ± standard deviation.

Results and Discussion

H was inhibited after 18.5±1.9min at PTPs of 170±24mmHg. Compared to PRE, significant reductions in H were found at I25 (-54±36%), I50 (-41±30%), I100 (-98±2%), and R1 (-66±35%) and in M at R1 (-57±26%), and R5 (-52±30%). Compared to PRE, pT (87±25Nm) and pRTD (421±101Nm/s) were reduced to 68±19Nm and 303±140Nm/s at I100, respectively. rmsEMG was not significantly reduced at any timepoint. Discomfort was rated highest at I100 (6±3a.u.), but no pain was reported during ischemia. Following reperfusion, all participants reported a transient tingling sensation which was almost gone by R5 with two participants additionally reporting pins and needles in their foot and shank.

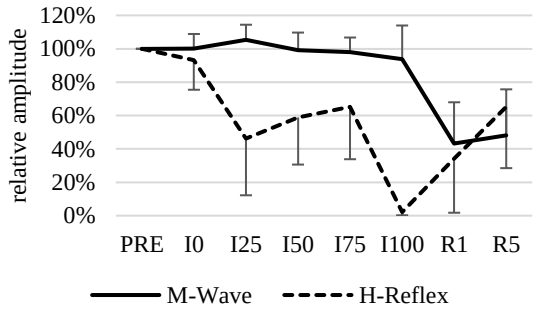

Figure 1: Mean ± SD of Soleus H-reflex and M-wave prior (PRE), during (I0 – I100), and after (R1 & R5) induced ischemia.

H experienced a first drop at I25 with subsequent rise until I75 before being inhibited at I100 (Figure 1). This behavior can be attributed to a rise in M responses at the lower stimulation intensities, indicating a shift of the recruitment curves (RC) to lower intensities during ischemia [2]. However, it is unclear if this shift was exacerbated by the MVIC at I0. While torque and EMG returned to baseline levels after reperfusion, significant reductions in M indicate a potentially insufficient stimulation intensity to elicit maximal M and therefore a shift of its recruitment curve to higher intensities possibly caused by muscle swelling.

Conclusions

Maximal isometric torque production was reduced at the time of reflex inhibition while direct motor nerve responses (M-wave) remained unaffected. Effects of ischemia and reperfusion on RC shifts should be further explored.

References

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