Support surface acceleration affects tibialis anterior onset latency during support surface translation perturbations

Kimberly C. Lang, B.S.,† Lena H. Ting, Ph.D.,‡ J. Lucas McKay, Ph.D.‡
†Neuroscience Graduate Program, Emory University, Atlanta, Georgia, USA
‡Wallace H. Coulter Department of Biomedical Engineering at Georgia Tech and Emory University, Atlanta, Georgia, USA

ID# 89769

Introduction

Electromyography (EMG) data is an important tool for assessing reactive balance in various populations, including older adults and individuals with Parkinson’s disease. EMG onset time often serves as an assessment metric, but may vary with different perturbation types and intensities that are used with participants of different balance abilities. EMG response amplitude has been related to perturbation velocity, displacement [1] and acceleration [2]. Perturbation acceleration also appears to influence muscle onset latencies, however this may be confounded by use of different balance strategies [3] and may depend on the perturbation types that differ across laboratories.

The objective was to characterize the effect of perturbation acceleration and velocity on EMG onset latency during support-surface ramp-and-hold translation perturbations.

We hypothesized that perturbation acceleration would have a greater effect on average EMG onset time than perturbation velocity. We based this hypothesis on previous work that showed perturbation acceleration and velocity affected EMG amplitude during the “initial burst” (IB) and “plateau region” (PR) (first two 150 ms periods after onset) respectively, in a manner explained by a neuromechanical model in which temporal patterns of muscle activity are formed by overlapping contributions of center-of-mass (CoM) acceleration, velocity, and displacement [2].

Results

1. Variations in acceleration affect EMG amplitude during the initial burst (IB) more than variations in velocity.

• TA onset latency increased by 4 ms as platform velocity increased from 25 to 40 cm/s.

• ANOVA and post-hoc tests identified a significant effect of acceleration (F(3,421)=4.2; p=0.006) but significant differences only between the two highest velocity levels.

• ANOVA identified a significant interaction effect between acceleration and velocity (F(6,421)=2.7; p=0.014)

• This minor impact on onset latency is consistent with previous findings indicating that velocity impacted the PR, but not the IB [2].

2. Average TA onset latency decreased by 19 ms as platform acceleration increased from 0.2 to 0.4 g.

• TA onset decreased from 127±16 ms (mean±SD; n=7) at 0.2g to 108±15 ms at 0.4g. This is consistent with previous findings that mean IB EMG amplitude scales linearly with peak acceleration [2].

• ANOVA and post-hoc tests identified a significant effect of acceleration (F(2,21)=153.7; p<0.001) and significant differences between all acceleration levels.

3. Average TA onset latency increased by 4 ms as platform velocity increased from 25 to 40 cm/s.

• TA onset latency increased from 115±15 ms at 25 cm/s to 119±16 ms at 40 cm/s.

• ANOVA and post-hoc tests identified a significant effect of velocity (F(3,421)=4.2; p=0.006) but significant differences only between the two highest velocity levels.

• ANOVA identified a significant interaction effect between acceleration and velocity (F(6,421)=2.7; p=0.014)

• This minor impact on onset latency is consistent with previous findings indicating that velocity impacted the PR, but not the IB [2].

4. A regression model that considered only acceleration accounted for most of the variance in onset latency.

\[ \text{Onset} = 147.5 - 97.17 \times \text{Peak Acceleration (g)} \]

• Adjusted R² = 0.72; F(9,430)=138.9; p<0.001

• Suggests that perturbation acceleration is primarily responsible for variations in onset latency.

• This model is likely device-dependent.

Discussion

These findings confirm our model’s prediction that the initial EMG burst is due mostly to perturbation acceleration and not velocity. Greater platform acceleration resulted in shorter muscle onset latencies, whereas platform velocity had little effect. This effect of acceleration was present across a range of feet-in-place strategies for maintaining balance (ankle to hip strategies), consistent with the findings of Nonnekes et al [3]. Thus, acceleration should be considered when identifying muscle onset latency, especially as many perturbation devices do not explicitly monitor or control acceleration in each condition and acceleration profiles can deviate substantially from idealized trajectories.

The observed changes in muscle onset latency were similar to previous studies which showed latency changes of approximately 26 ms when perturbations were applied during quiet standing or during standing with arm extended [5]. This limited change could be attributed to changes in sensorimotor pathway excitability via spatial or temporal summation. Additionally, our results may be near the absolute minimum onset latency. This work examines only one representative muscle. Future work will examine medial gastrocnemius and more proximal flexors and extensors.

References:

Supported by National Institutes of Health Grant HD-075612.

Contact: klang@emory.edu