A Model of Reduced Reciprocal Inhibition Induced by Rapid External Stretches in Children with Cerebral Palsy

Ellis Van Can[1], Jente Willaert[1], Kaat Desloovere[2], Anja Van Campenhout[3] Friedl De Groote[1]

[1.] Department of Movement Sciences, KU Leuven, Leuven, Belgium, [2.] Department of Rehabilitation Sciences, KU Leuven–UZ Leuven, Leuven, Belgium, [3.] Department of Development and Regeneration, KU Leuven–UZ Leuven, Leuven, Belgium

Summary

Joint hyper-resistance is common in children with cerebral palsy but it remains unclear how reduced reciprocal inhibition contributes to joint hyper-resistance. Here, we model activity of the antagonistic muscle upon stretch of the agonist based on sensory feedback from the agonist and show that our model can explain muscle activity during clinical tests of joint hyper-resistance in a child with CP. We will use this model to evaluate the contributions of reduced reciprocal inhibition to joint hyper-resistance and altered coordination during walking.

Introduction

Joint hyper-resistance, an increased resistance to imposed joint movements, is a common symptom in many neurological disorders including cerebral palsy (CP). A consensus study identified increased background muscle activity and hyperreflexia as the neural origins of joint hyper-resistance [1]. However, reciprocal inhibition, inhibition of antagonists upon stretch of agonists is also reduced in children with CP [2]. Here, we evaluated whether antagonistic muscle activation in response to stretch during a clinical assessment of joint hyper-resistance could be explained based on sensory information from the stretched muscle. This is a first step towards identifying reduced reciprocal inhibition and its contribution to functional impairments in CP.

Methods

Joint hyper-resistance was assessed by manually dorsiflexing the ankle joint of a relaxed patient as fast as possible. Joint kinematics, EMG, and interaction forces were recorded. Here, we present data of one child (Boy, 11.13 years, diplegic CP, GMFCS 2) but we will extend the analysis to 85 children based on existing data.

We modeled the reflex activity of the plantar flexors (medial gastrocnemius and soleus) by delayed linear feedback of muscle force and yank as previously has been shown that this model outperforms models based on length and velocity [3]. Similarly, we modeled the activity of the antagonistic m. tibialis anterior by delayed linear feedback of m. soleus force and yank. Computed reflex activity and constant background activity, recorded 0.5 s before stretch onset, were summed to construct the total muscle activity. We estimated muscle force and yank feedback gains based on experimental data.

To this aim, we scaled a musculoskeletal model to the dimensions of the child [4], used the measured joint kinematics to compute muscle-tendon length, and simulated the muscle's force and yank based on a Hill-type muscle model with the measured EMG and muscle-tendon length as inputs. We then estimated feedback gains by minimizing the difference between simulated by minimizing the difference between reconstructed muscle excitation, based on the previously described model with simulated force and yank as inputs, and experimental (EMG) muscle activation.

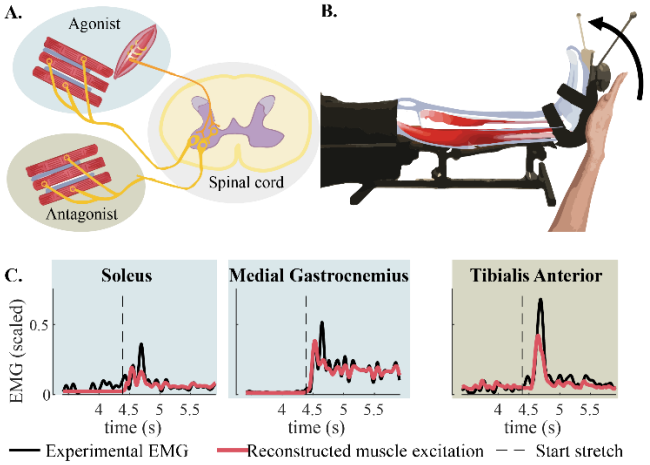

Figure 1 A. The mechanism of reciprocal inhibition, figure adapted from [5]; B. Set up of the instrumented joint hyper-resistance test [6]; C. Reconstructed muscle excitations

Results and Discussion

Our model largely captures the response of both plantarflexors and tibialis anterior to muscle stretch of the plantarflexors (Fig. 1C). This suggests that the activity of the antagonistic tibialis anterior is indeed driven by sensory information from the stretched agonistic soleus. The model underestimates the magnitude of the initial response to stretch. This can be due to Hill models not accounting for the effect of short-range stiffness [7], i.e. the steep increase in muscle force upon stretch of a muscle that has been held isometric. A model that accounts for short-range stiffness will predict a larger initial increase in force and yank and thus larger reflex and antagonistic activity.

Conclusions

Our preliminary data suggests that activity of the antagonistic muscle can be explained by sensory information from the stretched muscle during an instrumented clinical test of spasticity. Estimated feedback gains can be used as a measure of reduced reciprocal inhibition.

Acknowledgments

Funded by Research Foundation Flanders project G0B4222N.

References

[1] van den Noort J. C. et al. (2017). *Eur. J. Neurol.*, **24**:981-e38. [2] Gupta S. et al. (2024). *Cureus*, [3] Falisse A. et al. (2018). *PLoS ONE*, **13**:. [4] D'Hondt L. et al. (2024). *PLOS Comput. Biol.*, **20**:e1012219. [5] Miller F. (2020). *Cerebral Palsy*, [6] Bar-On L. et al. (2013). *Gait Posture*, **38**:141–147. [7] Blum K. P. et al. (2017). *PLOS Comput. Biol.*, **13**:e1005767.