

Biophysical muscle model captures movement history dependence of joint hyper-resistance

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Summary

Joint hyper-resistance – also known as spasticity – is a common symptom in neurological disorders such as cerebral palsy (CP). Joint hyper-resistance may in part originate from an increased muscle stiffness, considering that both joint hyper-resistance and muscle stiffness increase with muscle activation and decrease after movement. Computer simulations based on neuromusculoskeletal models may help identify muscle stiffness contributions to joint hyper-resistance. Unfortunately, current simulations rely on phenomenological “Hill-type” models, which poorly capture muscle stretch responses. Biophysical muscle models capture muscle stiffness and its history dependence in isolated preparations, but have not been employed to joint hyper-resistance. We therefore interfaced a biophysical model with skeletal dynamics to simulate a “pendulum test” of joint hyper-resistance. Simulations reproduced both joint hyper-resistance and its decrease after movement, as observed in CP. Simulations attribute joint hyper-resistance to attached muscle cross-bridges, which increase in number with greater muscle activation and decrease in number during and after movement.

Introduction

The pendulum test is a clinical test to assess joint hyper-resistance, during which the lower leg of a relaxed patient is first held horizontally, and then released to swing under the force of gravity. The first swing excursion (FSE) reflects the amount of joint hyper-resistance, with smaller FSEs indicating greater resistance. When the lower leg is moved down and up before releasing it, FSE increases (Δ FSE). This observation is consistent with the history-dependent reduction in muscle’s resistance to stretch, which may originate from a temporary reduction in the number of attached ‘cross-bridges’ [1]. Here, we hypothesize that a considerable portion of joint hyper-resistance and its history dependence can be explained by the biophysical dynamics of cross-bridge cycling. We test this hypothesis by employing a biophysical muscle model in musculoskeletal simulations of the pendulum test, and comparing its output to empirical data on children with CP.

Methods

We modified existing neuromusculoskeletal simulations of the pendulum test [2] to incorporate biophysical muscle models. Biophysical models incorporated dynamics for the cyclic attachment and detachment of cross-bridges, and for cooperative activation of thick and thin filament [3]. These dynamics were interfaced with elastic tendon and skeleton dynamics, as in [4]. Muscle activation and movement were expected to increase and decrease the number of attached cross-bridges respectively, thereby increasing and decreasing

resistance to stretch, respectively. We performed model simulations of the pendulum test at different muscle activation levels, and compared them to existing data on both children with CP, and typically developing (TD) peers [2]. We predicted that a considerable portion of the difference in (Δ) FSE between CP and TD can be explained by activation.

Results and Discussion

Neuromusculoskeletal simulations of the pendulum test incorporating biophysical muscle models qualitatively explained differences in joint hyper-resistance between typical example cases of TD and CP (Fig. 1A), solely from differences in muscle activation (Fig. 1C). Simulations at higher muscle activation yielded smaller FSE and larger Δ FSE (Fig. 1D), as observed in CP (Fig. 1B). However, modelled FSEs were considerably greater than those empirically measured, which might be due to some unmodelled origins of joint hyper-resistance such as reflex hyper-excitability.

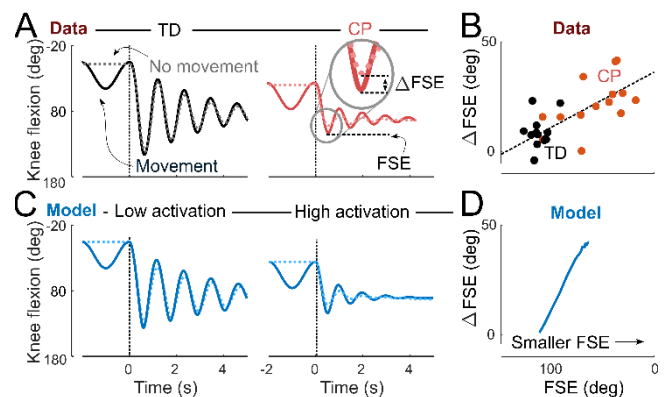


Figure 1: Musculoskeletal simulations with a biophysical muscle model predict joint hyper-resistance in cerebral palsy (CP).

Conclusions

Joint hyper-resistance in CP may arise from increased muscle stiffness that accompanies increases in muscle activation. Biophysical muscle models enable the simulation of history dependence in clinical tests of joint hyper-resistance.

Acknowledgments

This project was funded by the National Institute of Health of the United States of America (NIH HD90642).

References

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