Simulating the pendulum test to understand mechanisms of Parkinsonian rigidity

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Introduction

- **The pendulum test** is a diagnostic method used to objectively quantify spasticity by evaluating the pattern of lower leg movement after release from the horizontal based on three key kinematics features: reduced first swing excursion, reduced number of oscillations, and non-vertical resting leg angle. Spasticity is clinically-defined as a velocity-dependent resistance to muscle stretch. However, our recent biomechanical simulation showed that muscle tone and short-range stiffness (SRS) also contribute to abnormal pendulum test kinematics in spastic cerebral palsy[1]
- Here we used this pendulum test and a **biomechanical model** to understand the possible mechanisms contributing to Parkinsonian rigidity. Rigidity is clinically defined as increased resistance to passive movement throughout the range of motion independent of velocity[2]. Prior studies suggest that increased muscle tone plays a primary role in abnormal pendulum test kinematics in Parkinson's disease (PD), but the models used are difficult to interpret physiologically[3,4]

Objectives

- In this preliminary study we asked the following questions:

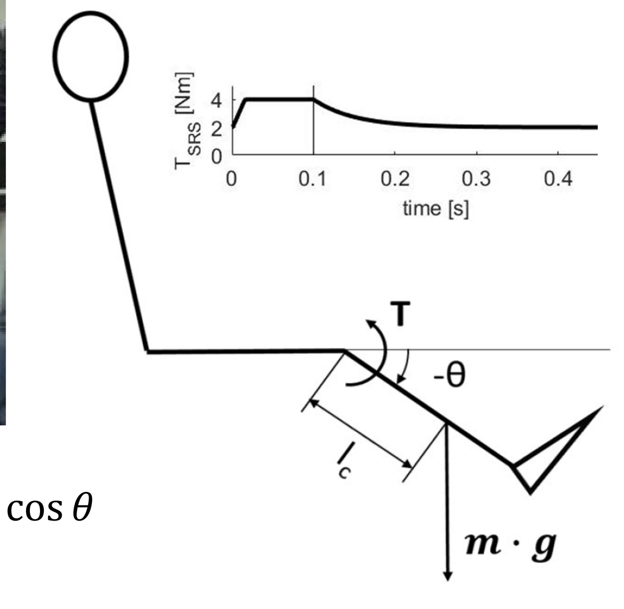
1. **We tested whether pendulum test kinematics outcomes are abnormal in PD.** (features found important in spasticity[5]) Specifically, whether the key kinematics features of reduced first swing excursion, reduced duration of oscillations, and non-vertical resting leg angle are associated with clinical scores of leg rigidity in the relaxed condition
2. **We then tested whether the abnormal pendulum test kinematics in a selected set of participants could be explained by abnormal muscle tone, short-range stiffness and reflexes**
   - In CP, the role of SRS proportional to muscle tone was critical for explaining the reducing first swing angle[1]
3. **Can the effect of increasing rigidity using an activation maneuver be explained by abnormal muscle tone, short-range stiffness and reflexes**
   - Clinically, rigidity is exacerbated by: finger tapping[6], fist clenching or Jendrassik maneuver (increasing spinal excitability)
   - It is not known whether activation maneuvers (AMs) increase tone or reflexes:
     - Prediction if tone: reduced duration of oscillation and resting angle
     - Prediction if reflex: reduced number of oscillation but no change in resting angle

Methods

- We recorded 15 participants with PD (11 males/4 female, 67±10years, MDS-UPRS-III 31.6±15.7) OFF medication and during 3 activation maneuvers (finger tapping, fist clenching, Jendrassik maneuver)

1. **We assessed the first swing excursion, the resting angle and the duration of oscillations using motion capture**
2. **We tested whether pendulum test kinematics can be explained by only varying two parameters in our model, T_b and k_T**

Biomechanical Model



$$J\ddot{\theta} + T + T_b + T_R = mgl_c\cos\theta$$

$$T = T_d + T_{SRS}$$

$$T_R = k_T T(t-\tau) + k_Y \dot{T}(t-\tau)$$

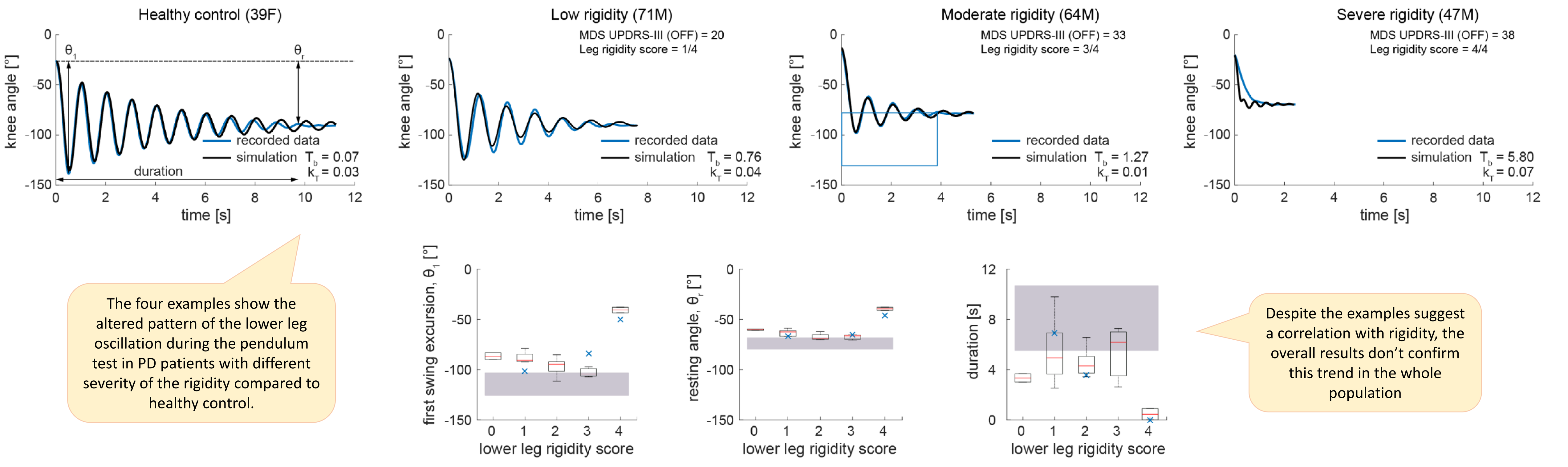
From De Groote et al., 2018

- The model simulates the lower leg as a torque-driven single-link pendulum[1]. Muscle tone is modeled as a constant baseline joint torque (T_b), and short-range stiffness torque (T_{SRS}) dependent on the level of muscle tone. The passive torque consists of a damper (T_d). Simulated reflex activity is a delayed sensory feedback torque (T_R) based on muscle force

3. **We tested the changes in pendulum test kinematics due to an activation maneuver**

Results

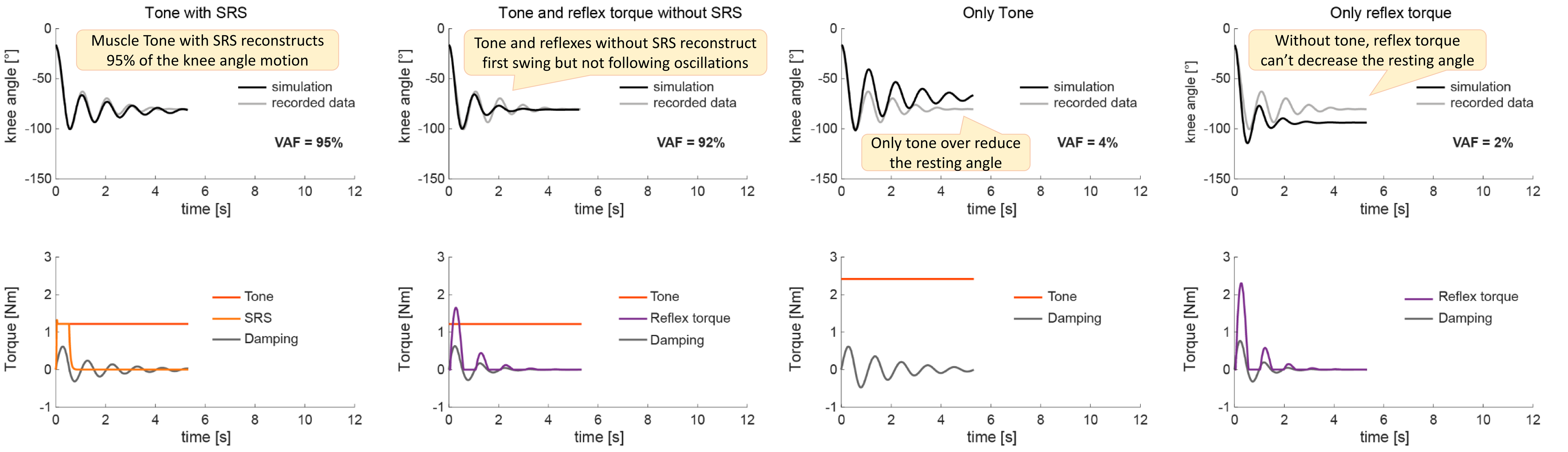
1. Pendulum test kinematics were abnormal in Parkinson's disease, but not clearly associated with rigidity



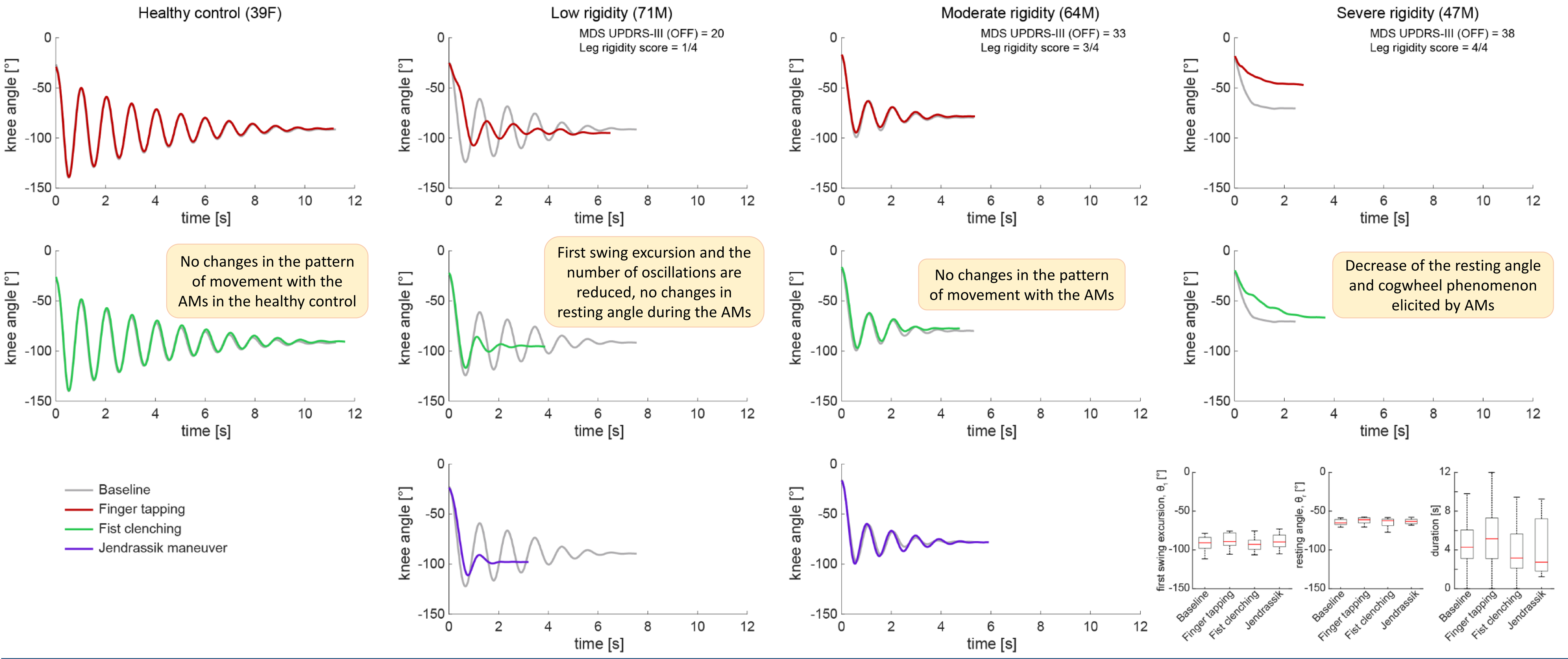
Grey bars indicate healthy control values taken from Stillmann and McMeeken, 1995

2a. Abnormal pendulum test kinematics can be accounted for by the model with an increase in muscle tone with SRS

2b. When muscle fibers are stretched after being held at a constant length for a period of time, SRS causes a rapid and transient increase in muscle force that depends on the level of muscle tone[8]. Both tone and SRS were necessary to simulate decreased initial swing excursion and non-vertical resting leg angle



3. The activation maneuvers lead to variable changes in the pendulum test outcomes between participants



Conclusions

1. **Outcomes of the pendulum test may provide an objective <u>quantitative measure</u> of lower limb rigidity in PD**

2a. Combined information from <u>experimental data and simulations</u> could reveal <u>mechanisms</u> of rigidity in PD

2b. Similar to CP[1], <u>SRS is critical</u> in reducing first swing excursion . This is not accounted for in most muscle models which affects conclusions based on modeling

3. **Activation maneuvers may involve <u>different mechanisms</u> affecting muscle activity. In some cases the reduction of the number of oscillation without changes in resting angle suggest the need for additional stiffness not accounted for by the current model**

<u>Future directions</u>

- **A model with a more accurate physiological representation may be needed to account for the observed changes in the pendulum test in PD. For example, a limitation of the current model is the absence of antagonist muscles, that could provide additional stiffness taking in account that rigidity involves increases in tone in the flexors and extensors**
- **Exploring the simulation of different mechanisms, for example the abnormal shortening response in PD or the presence of symptoms associated to different pathological phenotypes (i.e tremor, bradykinesia, freezing, cogwheel phenomenon, lead pipe), could clarify the physiological variables associated to rigidity in PD**

References

1. De Groote F, Blum KP, Horslen BC, Ting LH, PLoS ONE, 2018 2. Fung V and Thompson P, Rigidity and Spasticity, 2002 3. Brown RA et al., J Neurol Neurosurg Psychiatry, 1988 4. Le Cavorzin P et al., Movement Disorders, 2003 5. Fowler EG, Nwigwe AI, Ho TW, Dev Med Child Neurol, 2000 6. Hong M, Perlmutter JS, Earhart GM, Movement Disorders, 2007 7. Stillmann B, McMeeken J, Archives of Physical Medicine and Rehabilitation, 1995 8. Getz EB, Cooke R, Lehman L, Biophysical Journal, 1998

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Support:
NIH R01 HD046922-6
NIH K25 HD086276

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