Estimating the Contribution of Musculoskeletal Impairments to Altered Gait in Patients with Cerebral Palsy Using Predictive Simulations of Walking

Bram Van Den Bosch[1], Lars D'Hondt[1], Ilse Jonkers[1], Kaat Desloovere[2,3], Anja Van Campenhout[3,4], Friedl De Groote[1]
[1] Department of Movement Sciences, KU Leuven, Leuven, Belgium
[2] Department of Rehabilitation Sciences, KU Leuven, Leuven, Belgium
[3] UZ Leuven, Leuven, Belgium
[4] Department of Development and Regeneration, KU Leuven, Leuven, Belgium
Email: bram.vandenbosch@kuleuven.be

Summary

Surgery outcome of multilevel orthopedic surgery in cerebral palsy is unpredictable and highly variable due to our limited knowledge of how musculoskeletal impairments contribute to gait deficits. Here, we used predictive simulation to identify the contribution of musculoskeletal (MSK) impairments to altered gait in eight patients before surgery. We found that the contribution of MSK impairments to gait deficits was up to 42% and highly variable across patients in line with variable treatment outcomes.

Introduction

Cerebral palsy (CP) leads to impaired motor control, bone deformities, muscle contractures and weakness resulting in altered gait. Multilevel orthopedic surgery (MLS) targets musculoskeletal (MSK) impairments but treatment outcome prediction is hard due to complex interactions between motor control and musculoskeletal impairments leading to unpredictable and highly variable outcomes.

Here, we used model-based simulations of walking to isolate the contribution of MSK impairments to gait deficits in patients with CP scheduled for MLS.

Methods

We included eight patients (age: 16 ± 3.3 y) with spastic CP planned for MLS. All patients had a clinical exam and 3D gait analysis followed by an MRI scan of the legs and pelvis.

For each patient we made four MSK models with different levels of personalization. Models have 31 degrees of freedom and are actuated by 88 muscles based on [1] with bilateral quadratus femoris and gemellus removed (modeling deformities caused unrealistic operating ranges, removing in GEN barely affected simulations). **GEN** was obtained by scaling the generic model [1] to the patient's anthropometry. **GENMUS** was obtained by additionally modeling muscle contractures and weakness based on the clinical exam. Optimal fiber length was scaled such that the passive joint torque was 15 Nm at end range of motion (from clinical exam). Active muscle force in the Hill-type model was reduced based on the manual testing scores. **GEO** was obtained by personalizing hip and knee joint locations and muscle-tendon paths for hip and knee actuators based on MRI [2]. **GEOMUS** was obtained by adding muscle impairments to **GEO**.

We performed simulations of walking based on each model using PredSim [3,4]. Simulations were obtained by minimizing a movement related cost without relying on experimental movement data. We calculated Root Mean Square Differences (RMSD) and Cross-Correlations (CC) between simulated and measured sagittal plane kinematics. Differences in RMSD and CC between **GEN** and more personalized models were used as a measure for the contribution of the modeled impairment to the gait deficits.

Results and Discussion

Modeling impairments improved the agreement between simulated and measured sagittal plane kinematics (hip, knee and ankle) but improvements were variable across subjects and the modeled impairments never explained more than 42% of alterations in kinematics (Figure 1).

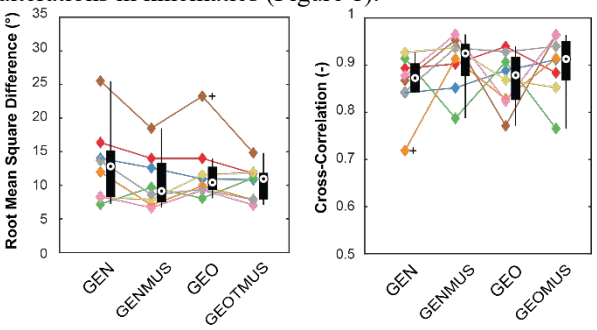

Figure 1: Simulations compared with experimental kinematics. Diamonds are individual patients, bars are boxplots.

Modeling muscle impairments (**GENMUS**) had a larger effect than modeling altered musculoskeletal geometry (**GEO**) of hip and knee.

Conclusions

Our simulations suggest that MSK impairments only explain up to 42% of gait deficits, suggesting a large contribution from motor control impairments.

Acknowledgments

This research was supported by funding from KU Leuven (C24M/19/064) and FWO (G0B4222N, 1SF1824N)

References

[1] D'Hondt L. et al. (2024). *PLOS Comput. Biol.*, **20**:e1012219.
[2] Scheys L. et al. (2006). *Biomed. Simul.*,
[3] Falisse A. et al. (2019). *J. R. Soc. Interface*, **16**:20190402.
[4] D'Hondt L. et al. (2024). *2024 10th IEEE RASEMBS Int. Conf. Biomed. Robot. Biomechatronics BioRob*,