Analysis and design of a passive assistive device for patients with Duchenne Muscular Dystrophy

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Introduction

Duchenne Muscular Dystrophy (DMD) is the most common muscular dystrophy diagnosed during childhood and is characterized by progressive muscle weakness and loss of muscle mass. DMD is a genetic disease that causes alterations in the dystrophin protein, which protects muscle fibers. Without dystrophin, muscles are broken down by enzymes, which leads to the death of muscle cells and tissue, and causes degeneration and muscle weakness. Because DMD is inherited in an X-linked recessive pattern, it manifests mainly in males (1 in 5000). The mutation of the gene is usually transmitted from mother to child, but it may also occur by spontaneous mutations.[1]

The first symptoms of DMD usually appear in children between two and three years who generally need to use a wheelchair by the age of twelve or thirteen. The current orthopaedic treatment for people with DMD varies according to the stage of the disease. In the late-ambulant stage, when walking becomes very hard or almost impossible, Knee-Ankle-Foot Orthoses (KAFOs) are recommended in order to help control the joint stiffness, prolong ambulation and delay the onset of scoliosis.

Our goal is to design a passive device to assist the gait of patients with DMD in the ambulatory stage. To obtain the optimal device parameters, we analysed the motion data of a DMD patient and generated a musculoskeletal model of the patient. The device assistance joint torque was determined by solving an optimal control problem and minimizing muscle activity down to a healthy level.

Methods

The musculoskeletal model used is *Gait10dof18musc*, a lower limb model available in OpenSim simulation software. The model has a total of 10 degrees of freedom, 18 muscles, and 12 rigid bodies: torso, pelvis, and two legs (with femur, tibia, talus and calcaneus). Some joints in the model were modified to add more degrees of freedom (such as the hip abduction), which allowed the model to better fit the experimental data.

The maximum isometric force in the muscles of children with DMD is generally lower than in healthy children. Experimental data were used to quantify this difference and capture muscular dystrophy in the musculoskeletal model. A force scale factor was calculated from the muscle strength test using a hand-held dynamometer, which measured the passive muscle strength of the muscular groups responsible for the flexion/extension of the hip, knee, and ankle joints. Since the models and data used in this study are specific for two patients, the values of the dynamometer measurements are also specific for each patient.

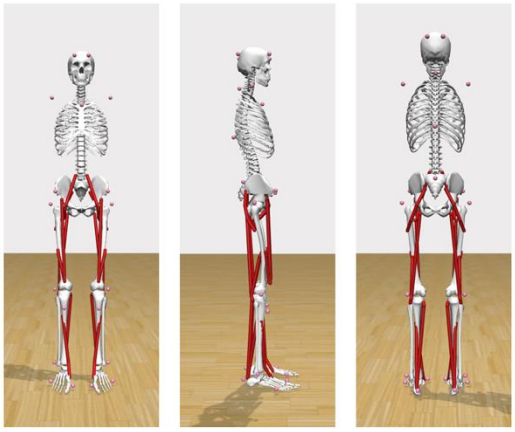

Figure 1: Musculoskeletal model in OpenSim

Experimental data from two subjects during gait were used (a 9-year-old DMD patient and a control subject). The model dimensions and mass properties were scaled to match that of the subjects. Then, the model coordinates for the gait motion were obtained from the marker position data by solving the inverse kinematics problem, which were used as a reference motion in the optimal control problem.

In order to determine the muscle forces from the motion data, an optimal control problem was solved. The cost function that was minimized included muscle activation and the error between experimental and computed coordinate values. Additional terms that minimize joint torque actuators and the residual forces on the pelvis were also included to help solver convergence. These joint actuators were later used to determine the device assistance needed.

Another optimization was carried out with a limited muscle activation of 0.5, which is the value obtained with the control subject model. The value of the joint actuators needed to assist the DMD subject model during the healthy gait motion were used to identify the passive device stiffness. Three different device stiffness values were identified for the right hip joint according to different phases of the gait cycle: the first one from the

complete cycle, the second one by removing the midstance and terminal stance, and the third one as the average of the other two. Finally, additional optimal control problems were solved with each of the three devices to assess the muscle activity of the assisted DMD subject model.

Results

Figure 2 shows the average muscle activation for each muscle during gait. The DMD subject presents larger muscle activity, which is mainly due to a reduced maximum isometric force of the muscles.[2] In addition, the pathological motion relies on compensatory movements, which may require more muscle activity than the control subject.[3]

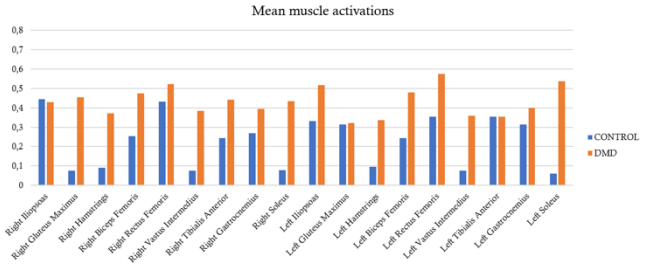

Figure 2: Mean muscle activation in a gait cycle for a DMD patient and a CONTROL subject.

Figure 3 shows the muscle activity of the DMD subject model with the three right-hip-joint passive devices. The implementation of the devices reduced the muscle hyper-activations compared to the pathological DMD gait. All device parameters produced similar results. The device that reduced more the activation of the muscles is the SPRING 2, the parameters of which were obtained by removing the hip joint assistive torque from the midstance and terminal stance. However, the device has the opposite effect on the left biceps femoris and hamstrings, which generate a left hip extensor torque.

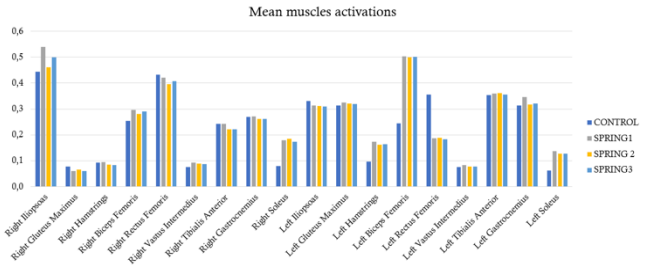

Figure 3: Mean muscle activation in a gait cycle for a model with DMD using a hip joint passive device with 3 stiffness values.

Conclusions

The parameters of a passive assistive device for the right hip joint of DMD patients were identified by solving an optimal control problem with experimental data tracking. The use of the device significantly reduced the overall muscle activity of the DMD patient model down to a healthy level. Further analysis would be needed to assess the use of devices in other joints, which can assist DMD patients not only during gait, but also during other daily life activities.

Acknowledgements

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