Development Of A Computer Model To Simulate The Effect Of Localized Muscle Weakness On Walking.

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Introduction

Musculoskeletal models can help clarify the effect of weakness on performance and help to evaluate hypotheses on the nature of the compensation response. The clinician often aims to identify the primary deficit when a patient presents with complicated gait deviation. But the patient naturally and automatically compensates for that weakness. Identification of the primary deficit is not always straightforward. Yet, the treatment plan is often ideally directed at the primary deficit. A musculoskeletal model that responds in a manner similar to a patient can clarify the deficit-dysfunction-disability relationship. The sensitivity analysis performed on the neuro-musculo-skeletal model¹ is useful to understand the model response to parameter changes.

A model has been implemented in which stable locomotion emerges through the global entrainment among the rhythmic activity of the neural system, the rhythmic movements of the musculo-skeletal system and the interaction with the changing environment. The neural system is represented of simple oscillatory circuits called central pattern generators (CPGs). Environmental information is represented by the sensory input given to the neural rhythm generator.

Statement of Clinical Significance

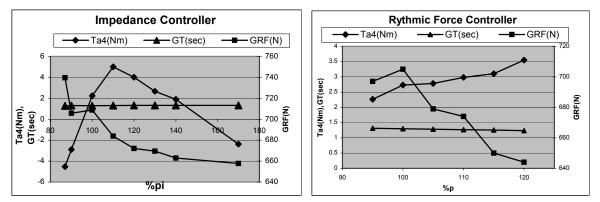
The development of a model that simulates the effect of muscle weakness could help the clinician better understand the deficit and develop strategies and treatments that maximize the capabilities of individuals with chronic weakness. Understanding how patients compensate for specific deficits is also critical to matching treatment recommendations with existing function.

Methodology

The model sensitivity was tested by varying some of the input parameters as a percentage of their original value. Model performance was compared to the baseline once stable walking was generated (usually after a few model steps or 3-4 seconds of simulation time). The following parameters were varied: strength of the sensory input, the impedance parameter, and the magnitude of the coefficient in the rhythmic force controller. The change of the right knee active muscle torque (Ta4), the gait cycle time (GT) and the peak toe-off vertical ground reaction force (GRF) were used to analyze the effect of the change of the above parameters on model performance. A limit in a sensitivity parameter was assumed when model became unstable and fell over.

Results

Strength of Sensory Input:(70% to 182%)With increase in the strength of the sensory input above base value there was an increase in the peak active flexive torque. The gait cycle time decreased and the toe-off GRF decreased too.



Impedance parameters: (87% to 170%). The active extensive torque occuring at the right knee during midstance was analyzed.

Magnitude of the coefficient of the rhythmic force controller: (70% to 110%). The active flexive torque occuring at the right knee during early swing phase was analyzed. The model was found to be most sensitive to the coefficient of the rhythmic force controller and was found to be stable for a large change in sensory input as expected.

Discussion

As expected we found the model to be more sensitive to decrease in sensory input rather than increase. A minimum level of sensory input is required to cross the threshold of the neurons in the CPG to produce the muscle torque. Though increasing the sensory input too much would make the model very sensitive and unstable thus explaining its fall. The increase in this parameter increases the touch sensitivity and decreased the gait cycle time, which was mainly due to a reduction in stance time.

The model was found to be more sensitive to decreases than to increases of the impedance parameter. The impedance controller does not affect bi-articular and flexor knee muscles. The impedance controller only acts upon extensor muscles and is responsible for maintaining a stable upright posture. Midstance knee flexion was observed when the parameter was reduced from the baseline value. This explains its loss of stability and hence the collapse. The coefficient for the rhythmic force controller may be representative of the number of muscle fibers employed to generate the muscle torque. The rhythmic force controller provides the torque necessary to move the body. As expected the flexor torque during early swing phase increased significantly with a corresponding increase in the parameter value. Sensitivity analysis gives us an insight into how the model would react to different pathological or environmental conditions. Further plans include studying the compensatory response to muscle weakness by implementing another control scheme in place or on top of the existing one. The new scheme will utilize the contributions of each joint moment as calculated by intersegmental dynamics analysis² or induced acceleration. This control scheme will explicitly incorporate the potential or ability of each muscle group to compensate for specific muscle weaknesses.

References

[1] Taga G. Biological Cybernetics 1995; 73:97-111.[2] Talaty M. Doctoral Dissertation 2002. Acknowledgments

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