NEUROMUSCULAR ADAPTATIONS IN GAIT WITH AGE AND MUSCULOSKELETAL PATHOLOGY

[1] Chris A. McGibbon, and [2] David E. Krebs
[1]Institute of Biomedical Engineering, University of New Brunswick, Fredericton NB, Canada,
[2]Massachusetts General Hospital, Biomotion Laboratory, Boston MA, USA; email: cmcgibb@unb.ca

INTRODUCTION

Arthritis and other chronic joint conditions are the leading cause of disability in older adults. Changes in locomotor patterns with age [1-3] and disability [4] may be caused by neuromuscular adaptations, which alter segmental kinematics and kinetics through reorganization of muscle firing patterns to compensate for primary mobility impairments. However, the underlying neuromuscular adaptations that arise from idiopathic, age-related impairment and musculoskeletal pathology are not well understood. The purpose of this study was to explore the underlying mechanisms of gait disorder among older adults, and determine which kinematic and kinetic variables best discriminate between young and old healthy adults, and between healthy and disabled elders.

METHODS

Ankle, knee and hip peak angles, moments and powers in the sagittal plane were acquired during gait in 120 subjects: 45 healthy young (HY), 37 healthy elders (HE), and 38 disabled elders (DE) with functional limitations due to lower-extremity musculoskeletal pathology (primarily arthritis). MANCOVA with discriminate analysis, statistically controlled for gait speed, identified the variables that discriminate between young and old healthy adults, and between healthy and disabled elders. Correlation analysis was used to explore interrelationships among these variables within each group to identify possible mechanisms underlying gait dysfunction among older adults.

RESULTS AND DISCUSSION

Eight variables strongly discriminated among groups (Figure 1). HE subjects were discriminated (sensitivity 76%, specificity 82%) from HY subjects via decreased late-stance ankle plantar flexion angle (AR2), and increased late-stance knee power absorption (KP4) and early-stance hip extensor power generation (HP1). DE subjects were discriminated (sensitivity 74%, specificity 73%) from HE subjects via decreased late-stance ankle plantar-flexor moment (AM2) and ankle plantar-flexor power generation (AP2), and increased early-stance ankle dorsi-flexor moment (AM1), late-stance hip flexor moment (HM2), and late-stance hip flexor power absorption (HP2). Among the eight variables, the number of significant ($p<.005$) correlations increased with age and disability: there were 5 for HY, 7 for HE, and 13 for DE groups. For the DE group, late-stance hip flexor power absorption and hip flexor moment were more tightly coupled to other kinematic and kinetic variables than for healthy groups.

The data suggest that gait changes caused by lower extremity impairment, manifest differently than do gait changes from aging alone. Beyond age-related changes, elders with lower-extremity dysfunction rely excessively on passive hip flexion torque to provide propulsion in late-stance, probably to assist with advancement of the swing leg, and ankle dorsi-flexors of the contralateral limb, probably to enhance trunk stability. This compensatory strategy probably increases hip joint stresses, leading to increase OA incidence [5] and promoting hip/trunk muscle overuse and thus fatigue. Furthermore, weak or fatigued ankle muscles could jeopardize control of the body center of mass, and increase risk of falling. Relationships among the biomechanical variables showed a higher degree of coupling for the DE subjects compared to the HY and HE subjects, suggesting reduced flexibility to alter motor strategies.

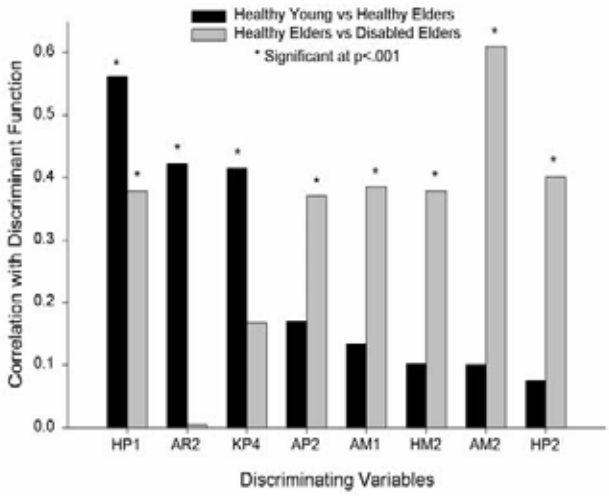

Figure 1. Strength of discriminate functions.

CONCLUSIONS

This work supports a growing body of evidence that pathological gait changes from age and disability have a neuromuscular basis, which may be informative in a motor control framework for physical therapy interventions. Study of physical therapies, such as optimal functional training, ie, practicing more healthy and younger gait strategies, aimed at reducing the dynamic coupling among neuromuscular control parameters is warranted.

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