COMPENSATORY STRATEGIES IN RESPONSE TO EXCESSIVE MUSCLE CO-CONTRACTION AT THE ANKLE DURING WALKING

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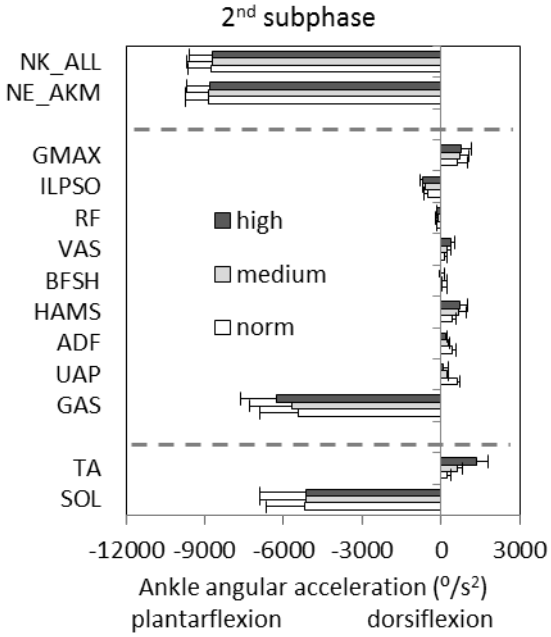
INTRODUCTION

Co-contraction, defines as the concurrent activation of agonist and antagonist muscles (antagonistic pairs) across the same joint, occurs in many activities including postural control, walking and running [1-3]. In normal gait, antagonistic muscle pairs in the lower extremities contract in an alternating pattern with short durations of concurrent activity to generate sufficient joint moment [4]. In some gait disorders, e.g. spastic gait, the temporal separation and magnitude differences of activations between agonist & antagonist muscles are frequently attenuated and motor control becomes poor [5]. The aims of this study were to analyze dynamic muscle functions during walking at a nominal speed and to identify the necessary compensatory mechanisms to overcome excessive co-contraction of the soleus-tibialis anterior pair and retain a normal walking pattern, using computed muscle control and induced acceleration analysis.

METHODS

Nine healthy adults (age: 30 ± 3yrs) were examined using a motion capture system (Vicon MX40, Oxford, UK). Ground reaction forces were obtained from two forceplates (Kistler, Winterthur, Switzerland). Surface EMG signals (Motion Laboratory System, Baton Rouge, LA) were recorded from the biceps femoris long head, rectus femoris, medial gastrocnemius, soleus (SOL) , and tibialis anterior (TA) bilaterally. Muscle-actuated simulations were performed in OpenSim, which consisted of scaling, inverse kinematics, residual reduction algorithm and computed muscle control (CMC) [6]. Induced acceleration analysis (IAA) was used to compute contributions of primary ankle dorsi/plantarflexors, knee flexor/extensors and hip flexor/extensors to the accelerations of ankle and knee joints. The co-contraction level was assessed based on the overlapping area of EMG activity from the antagonistic pairs, wherein larger degree of overlapping activity corresponded to a higher co-contraction. To maintain the dominant role of the muscles, three co-contraction levels (normal, medium and high) were simulated by increasing the activation of the muscle with less activation during normal gait. The response of other muscles to the excessive co-contraction of SOL-TA was computed by repeating CMC and IAA after constraining excitations of SOL-TA at each co-contraction level. Three sub-phases were identified according to the role of the dominant muscle in each subject.

RESULTS AND DISCUSSION



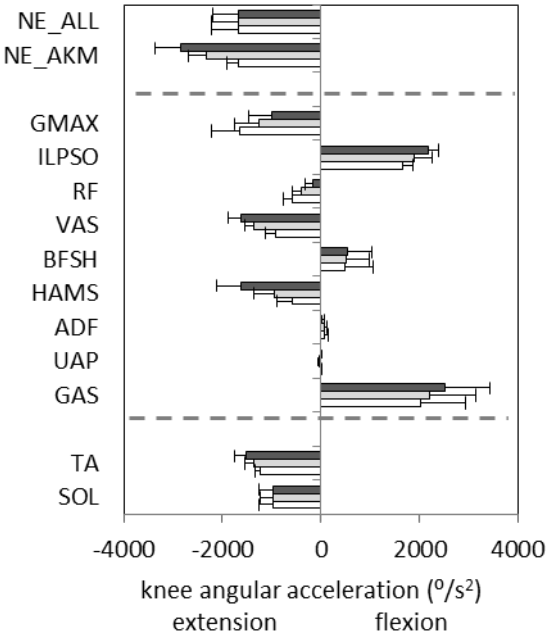


Figure 1: Contributions from primary muscles spanning ankle, knee and hip to ankle dorsiflexion accelerations and knee flexion acceleration in the 2nd sub-phase. Each bar represents the mean ± 1 S.D. of the 9 subjects at normal, medium and high co-contraction levels. The net effect of muscles' contribution were summed separately from muscles spanning ankle and knee only (NE_AKM) and from all the muscles (NE_ALL) (GAS: lateral gastrocnemius and medial gastrocnemius; UAP: the other uniarticular ankle plantarflexors: tibialis posterior and peroneus longus; ADF: other ankle dorsiflexors: extensor digitorum longus and extensor hallucis longus. HAMS: semimembranosus, semitendinosus and biceps femoris long head; BFSH: biceps femoris short head; VAS: vastus medialis, vastus intermedius, and vastus lateralis; RF: rectus femoris; ILPSO: iliacus and psoas combined; GMAX: gluteus maximums).

An example result of the 2nd subphase was illustrated in the Figure 1. At the ankle joint, when SOL-TA co-contraction was increased through increased excitation of TA, TA contributed more to decelerate plantarflexion. The primary compensation was increased excitation of GAS, which led to increased plantarflexion acceleration contribution. At the knee joint, when SOL-TA co-contraction was increased through increased excitation of TA, TA contributed more to accelerate knee extension. The compensation was primarily by muscles spanning the knee and hip, including GAS, HAMS, VAS, RF, ILPSO and GMAX.

Theoretically, the net joint accelerations provided by all the muscles should be approximately constant under different co-contraction levels, since joint angles/moments remain the same in the simulations. In our study, the net acceleration of the ankle and knee joints from all muscles (NE_ALL) was generally unchanged (Figure 1) when increased antagonistic muscle co-contraction was simulated. The accelerations from the muscles spanning only the ankle and knee (NE_AKM) were constant at the ankle, but varied at the knee joint. This indicates that ankle and knee muscles alone are able to compensate for increased co-contraction at the ankle joint and generate sufficient ankle moment. However, at the knee joint, hip muscles must also be involved, which agrees with recent findings that hip flexors also have important contributions to knee angular acceleration.

Understanding how individual muscles contribute to joint accelerations can help to clarify the neurological control strategies by means of muscle excitation patterns to overcome excessive muscle co-contraction. Results of this simulation indicate that with a high level of dorsiflexor/plantarflexor co-contraction, one can still perform normal walking through other means; the dynamic equations of motions can be fully satisfied under relative high levels of muscle co-contraction.

CONCLUSIONS

This study identified how redundancy in muscle contributions to ankle and knee angular accelerations during walking allows the nervous system to compensate for specific antagonistic muscle co-contraction. The results of this study can help to clarify how muscles can provide compensation to co-contraction at the ankle joint in patient populations with motion disorders affecting motor control of walking. It can also be informative for clinical interpretation of motion analyses in persons with motion disorders, when secondary muscle co-contraction or deficits may occur simultaneously.

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