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How gravity and muscle action control mediolateral center of mass excursion during slow walking: A simulation study

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ABSTRACT

Maintaining mediolateral (ML) balance is very important to prevent falling during walking, especially at very slow speeds. The effect of walking speed on support and propulsion of the center of mass (COM) has been focus of previous studies. However, the influence of speed on ML COM control and the associated coupling with sagittal plane control remains unclear. Simulations of walking at very slow and normal speeds were generated for twelve healthy subjects. Our results show that gluteus medius (GMED) contributions to ML stability decrease, while its contributions to sagittal plane accelerations increase during very slow compared to normal walking. Simultaneously the destabilizing influence of gravity increases in ML direction at a very slow walking speed. This emphasizes the need for a tight balance between gravity and gluteus medius action to ensure ML stability. When walking speed increases, GMED has a unique role in controlling ML acceleration and therefore stabilizing ML COM excursion. Contributions of other muscles decrease in all directions during very slow speed. Increased contributions of these muscles are therefore required to provide for both stability and propulsion when walking speed increases.

1. Introduction

A high incidence of falls is an important problem in modern society. In addition to the high associated cost on health care, these falls also have a major impact on the quality of life [1]. Many of these falls occur during walking.

To maintain balance during walking, control of the mediolateral (ML) motion of the center of mass (COM) is crucial. Accordingly, deviations of the gait pattern in the ML direction are often suggested to be a valid predictor of falls. Previous studies suggested step width (SW) and especially SW variability to be related to fall risk [2]. During steady-state gait, older adults reduce the ML COM accelerations as a compensatory mechanism to improve ML stability [3].

These modifications in COM movement and therefore its control are likely to become important at very slow speeds. Den Otter et al. [4] found specific bursts of muscle activity at very slow speeds and argued that these might be attributed to increased demands on postural stability. The direct relation to ML COM control still needs to be further investigated. As several studies found indeed that slower walking in elderly is associated with an increased risk of falls [5], it is highly relevant to understand how slow walking affects the ML COM control. To date, the relation between the ML and sagittal plane COM control remains unclear. In the sagittal plane, support and progression are coupled and similar muscles contribute to both in parallel. Principle component analysis of experimental electromyography (EMG) patterns shows that a reduced set of muscle activation modules can generate both support and propulsion [6]. This is also confirmed using muscle driven simulations [7–9]. Furthermore muscle contributions to both progression and support generally decrease proportionally when walking speed decreases [10,11].

Extending these insights to the ML COM control is less clear. Some experimental studies on posture suggest that ML and anterior–posterior (AP) motions have a coupled control [12]. In

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contrast, other researchers believe that COM control in the sagittal plane and ML stabilization are independent of each other during quiet stance [13] and balance perturbations [14]. Proof also exists for an independent control during gait, with decreasing walking speed, local dynamic stability in AP and vertical direction is enhanced, but stability in ML direction reduced [15]. This indicates that separate control of stability is required in different directions. Likewise, analysis of passive walking models suggests that during walking ML stability is actively controlled, while passive stability is provided in the sagittal plane [16]. Simulation studies of steady-state walking [17] investigated how individual muscles control ML balance. They concluded that muscles responsible for sagittal plane accelerations also accelerated the COM laterally (i.e. away from the midline) and that COM stability in all directions is controlled by similar muscle groups. However, a recent study [18] showed that control of ML accelerations of the COM requires additional synergies to the ones controlling the COM in the sagittal plane. Hence, these studies do not allow to uniformly conclude on sagittal plane versus ML COM control. Previous simulation studies [10,11] mainly explored the effect of speed on the muscle contributions to COM control in the sagittal plane. The study of Pandy et al. [17] investigated ML COM stability, but only reported one single speed. Only recently, a study of John et al. [19] explored the effect of walking speed on muscle contributions to the control of ML body motion. However, slowest speeds reported in their study are still largely above the walking speeds previously reported in for instance stroke patients and the subjects involved were children. Therefore, when exploring the role of individual muscle contributions to ML and sagittal plane COM control it seems advantageous to investigate the trade-off between sagittal plane and ML control also at very slow walking speeds and in an adult population.

In the present study, the effect of walking speed on ML and sagittal plane COM control is investigated using simulations of walking at normal and very slow speed. More particularly, we investigate if a decrease in speed affects similarly the muscle contributions in ML, vertical and AP direction. If a common control exists for the different planes, one would expect that with decreasing speed the observed decreases in muscle action (EMG) would have similar effects on the muscle contributions to COM accelerations in the various planes. However, if changes in muscle contributions to COM acceleration are specific for a given plane this would argue for separate control. One important muscle to consider is the gluteus medius (GMED). This muscle decreases EMG activity with decreasing gait speed [20]. Based on a simulation study, Liu et al. [10] concluded that GMED contributions to support are 'relatively constant across walking speeds'. However, a significant difference is reported between free and slow walking speed, with higher GMED contributions for the slow speeds.

Based on the assumption that the functional demands for progression decrease whereas stability remains equal or also decreases when walking at very slow speeds, it is hypothesized that muscles that act mainly in the sagittal plane will decrease their contributions in this plane, but maintain their ML contribution. In contrast, muscles preferentially contributing to ML stability such as GMED mainly decrease their contribution to ML accelerations of COM but maintain their contributions in the sagittal plane. This will therefore confirm an uncoupling of the COM control in the ML and sagittal plane.

2. Methods

2.1. Experimental data

We collected three-dimensional kinematic and kinetic data of twelve healthy subjects (age: 25.8 ± 4.0 years, weight: 71.1 ± 8.9 kg, leg length: 0.9 ± 0.04 m) walking on treadmill at a speed of 1 km/h (very slow) and 4 km/h (normal). The speed of 1 km/h was chosen because it is at the low end of the range of preferred speeds in stroke patients [21].

All subjects gave their informed consent prior to data collection and the experimental protocol was approved by the local ethical committee. Marker (active infrared LEDs) trajectories were collected at 100 Hz using a two-beam camera system (Krypton, Nikon Metrology NV, Belgium). The marker protocol consisted of six technical clusters and 16 additional individual markers; this protocol was described more detailed in a previous paper [22]. Ground reaction forces (GRF) and torques were measured at 1000 Hz using a force-plate instrumented split-belt treadmill (Forcelink, The Netherlands). EMG data were collected bilaterally at 1000 Hz for tibialis anterior, gastrocnemius lateralis, soleus (SOL), vastus lateralis, rectus femoris (RF), biceps femoris and semitendinosus using a wireless EMG system (Zero-wire EMG, Aurion, Italy). As part of the post-processing, the raw EMG signal was band-pass filtered between 10 and 500 Hz using a fourth order digital Butterworth filter and RMS was calculated using a 50 ms time window.

2.2. Simulations

Subject-specific simulations of both walking speeds were generated using a dedicated workflow in OpenSim [23]. In a first step, data of a static trial was used to scale a generic musculoskeletal model [24] to match the anthropometry of the subject. This generic model consists of 27 degrees of freedom (Supplemental Table 1). The leg and trunk joints were actuated by 92 Hill-type muscle-tendon units and the arms were driven by torque actuators. An in house developed Kalman smoothing algorithm [25] calculated the joint angles that minimize the difference between experimental and model markers. A residual reduction algorithm [26] reduced dynamic inconsistencies between the model kinematics and the measured GRF (Supplemental Fig. 1). Computed Muscle Control computed the most optimal muscle excitations patterns required to track the experimental walking task [26]. The calculated muscle activations were compared to the subject's measured EMG and visually verified (Supplemental Fig. 2). Simulated kinematics and kinetics closely tracked experimental kinematics and kinetics (Supplemental Figs. 3 and 4).

2.3. Analysis muscle function

A perturbation analysis [8] computed the AP, vertical and ML COM accelerations induced by a specific muscle or gravity. The force of each muscle or gravity was subsequently increased with 1 N and the equations of motion were integrated forward over a time window of 0.03 s to evaluate the effect on the COM. To allow changes in the GRF and moments during the perturbation, linear and torsional springs were added between the model's feet and the floor. We verified the validity of the simulations by comparing the summed muscle contributions to the COM acceleration with the COM accelerations in the reference simulation (Supplemental Fig. 5).

Contributions of muscles and gravity were calculated over the entire gait cycle, which was further divided into subphases (Figs. 2 and 3). While most simulation studies [8,10,11,17] consider head, arms and trunk as a single rigid body, this study used a model that included separate arms segments. This allowed us to identify the contribution of the arm dynamics to the COM accelerations.

In a post processing step, positive and negative contributions were separately averaged over each subphase and subsequently over all subjects. To simplify data analysis, contributions of smaller muscles with similar function were summed.