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Arm swing in human walking: What is their drive?

Marije Goudriaan [a], Ilse Jonkers [b], Jaap H. van Dieen [c,d], Sjoerd M. Bruijn [c,e,*]

[a] Research Group for Neuromotor Rehabilitation, Department of Rehabilitation Sciences, Biomedical Sciences Group, KU Leuven, Belgium
[b] Human Movement Biomechanics Research Group, Department of Kinesiology, Biomedical Sciences Group, KU Leuven, Belgium
[c] MOVE Research Institute Amsterdam, Faculty of Human Movement Sciences, VU University Amsterdam, Amsterdam, The Netherlands
[d] King Abdulaziz University, Jeddah, Saudi Arabia
[e] Department of Orthopedics, First Affiliated Hospital of Fujian Medical University, Fuzhou, Fujian, PR China

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ABSTRACT

Although previous research has studied arm swing during walking, to date, it remains unclear what the contribution of passive dynamics versus active muscle control to arm swing is.
In this study, we measured arm swing kinematics with 3D-motion analysis. We used a musculoskeletal model in OpenSim and generated dynamic simulations of walking with and without upper limb muscle excitations. We then compared arm swing amplitude and relative phase during both simulations to verify the extent to which passive dynamics contribute to arm swing.
The results confirm that passive dynamics are partly responsible for arm swing during walking. However, without muscle activity, passive swing amplitude and relative phase decrease significantly (both $p < 0.05$), the latter inducing a more in-phase swing pattern of the arms. Therefore, we conclude that muscle activity is needed to increase arm swing amplitude and modify relative phase during human walking to obtain an out-phase movement relative to the legs.

1. Introduction

During walking the arms swing out of phase relative to the legs, to minimize the body's angular momentum around the vertical axis, which reduces energy expenditure [1–5].

With respect to the effect of arm swing on gait stability there is less consensus; Ortega et al. [4] found an increase in stability due to arm swing, whereas Bruijn et al. [6] and Pijnappels et al. [7] found negative effects of arm swing on gait stability.

A number of studies have focused on the "how" of this movement pattern [3,5,8–12]. In these studies, the arms are often represented as pendula [5,8–11], or as mass dampers [11] that swing passively due to thorax movements [5,8–11]. However, experimental studies using surface EMG have shown that arm swing is at least in part controlled actively [3,8,9,11–13]. Fernandez-Ballesteros et al. [12] were the first to document muscle activity in arm muscles during walking. They showed that the posterior and middle parts of the deltoid were active at contralateral heel strike, when the arm changed direction. Barthelemy & Nielsen [13] and Kuhtz-Buschbeck & Jing [3] reported shoulder muscle activity during walking suggesting that muscle activity might be used to initiate direction changes of the arms, to keep them swinging out-of-phase with the legs. A recent review by Meyns et al. [14] concluded that it is still unclear to what extent muscle control or passive dynamics (e.g., accelerations of the thorax) determine arm swing. It could be that muscle activity merely amplifies arm swing, without changing the movement pattern qualitatively. Alternatively, muscle activity may be necessary to maintain the out of phase relationship between the arms [1,2,5,15].

Answering the question whether arm muscle activity is needed to maintain the out of phase relationship with the legs, requires analyzing how the arms would swing without muscle activity, but with passive muscle characteristics present. Modelling provides a platform that potentially can offer such insights, since it allows altering the excitations of the upper limb muscles to evaluate the effect on the kinematics. Indeed, Jackson et al. [8] and Kubo et al. [10] excluded all arm muscle activity from the gait simulations with their pendulum models, but still accounted for passive muscle characteristics. Jackson et al. [8] found a 'very small and ragged' arm movement when muscle activity was excluded from the simulation. Kubo et al. [10] did not focus on passive arm swing kinematics, but studied the transition from 2:1 to 1:1 with respect to arm to leg swing ratio. However, they hypothesized that muscle

* Corresponding author at: Research Institute MOVE, Faculty of Human Movement Sciences, VU University Amsterdam, Van der Boechorststraat 9, NL-1081 BT Amsterdam, The Netherlands. Tel.: +31 618844777.
E-mail address: s.m.bruijn@gmail.com (S.M. Bruijn).

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activity was needed to change passive arm swing amplitude and/or relative phase. In the previous studies [8,10], muscle parameters were based on mathematical equations and not on physiological values. Furthermore, these studies used simplified kinematics as input to their simulations (i.e., only the first harmonics, and thus, the accelerations at stride and step frequency). In the present study, we compare simulations with and without arm muscle excitations for normal (out-of-phase) walking using a complex musculoskeletal model that accounts for the physiological active and passive properties of all relevant muscular structures around the shoulder. Furthermore, we used experimentally collected 3D kinematics of whole body movement and ground reaction forces at different speeds as input for the simulations. These points are innovative compared to previous work and will allow us to further the current understanding on how arm swing is organized. In turn, a better insight in the organization of arm swing kinematics might improve rehabilitation techniques for patients with impairments in arm swing during walking (e.g., in stroke or cerebral palsy) [14].

2. Methods

2.1. Subjects

Five subjects (age 28.6 ± 2.61 (mean ± SD)) participated in this study, approved by the ethical advisory board of KU Leuven. All subjects gave written informed consent. All subjects were familiar with treadmill walking, had normal or corrected to normal vision and no known neuromuscular disorders and were naïve to the specific research question.

2.2. Measurement protocol

While walking on a treadmill (custom-built, Forcelink, Culemborg, The Netherlands) three different walking speeds were imposed (0.56 m/s, 1.11 m/s and 1.67 m/s). Each condition lasted approximately 60 s, with data being collected during the last 30 s.

The three walking speeds were randomized. 3D marker data were collected using a 10 camera Vicon system (Nexus 1.7.1, Vicon-UK, Oxford, UK) sampled at 100 samples/second. Reflective markers were attached over the bony landmarks according to the full body Plug-in-Gait model. Muscle activity of both left and right shoulder anteflexors (anterior deltoid, biceps brachii) and retroflexors (posterior deltoid, triceps and latissimus dorsi) was measured using surface EMG (Zerowire, Aurion, Milan, Italy) at 1000 samples/second. The EMG electrodes were applied according to Konrad [16]. Ground reaction force data under each foot was measured at 1000 samples/second, using force plates embedded in the treadmill.

2.3. EMG processing

The raw EMG data were corrected for offset and filtered with a dual-pass 4th order Butterworth band-stop filter between 49 and 51 Hz. Next, the data were filtered with a dual-pass 1st order high-pass filter at 20 Hz and rectified, followed by a dual-pass 4th order low-pass Butterworth filter at 10 Hz. For analyses, EMG's were time normalized to the gait cycle, and the mean of fifteen gait cycles was calculated.

2.4. Dynamic simulations

We generated muscle-driven simulations of walking in OpenSim based on experimental motion capture data. For each subject we generated one simulation per walking speed for the two conditions: with and without arm muscle activity, resulting in six simulations per subject. Initial marker data processing was performed in Nexus. Custom code in Matlab generated the appropriate file format for analysis in OpenSim.

We used the upper and lower body model from the ULB-project [17–22], but adjusted the model in order to decrease the simulation runtime. The final (adjusted) model contained 35 degrees of freedom as well as the description of the geometry and force generating capacity of 102 muscle-tendon actuators, 48 of which controlled the action of the upper limbs. For information regarding the DOF of the model we refer to the OpenSim website (https://simtk.org/home/ulb_project). In our model, we locked four DOF: the subtalar angle, the MTP angle, wrist flexion and wrist deviation. We ran muscle-generated simulations with and without arm muscle excitations according to the workflow described in Fig. 1. In a first simulation set, we calculated the muscle excitations that tracked the measured kinematics of all degrees of freedom in the model, including the arms, allowing muscle excitations to vary between 0.01 and 1. In these simulations (ACT), arm kinematics were therefore controlled by active muscle force. During the second set of simulations (PAS), upper limb kinematics were no longer tracked and the upper limb muscle excitations were limited to 0.02. The limit of 0.02, instead of 0, was introduced for numerical reasons. Although this can result in a very small level of muscle activity, it guarantees that mainly passive muscle structures influenced the arm kinematics that were induced passively through the accelerations of the neighboring segments.

The validity of the simulations was evaluated by visually comparing (1) the calculated muscle excitations during the first set

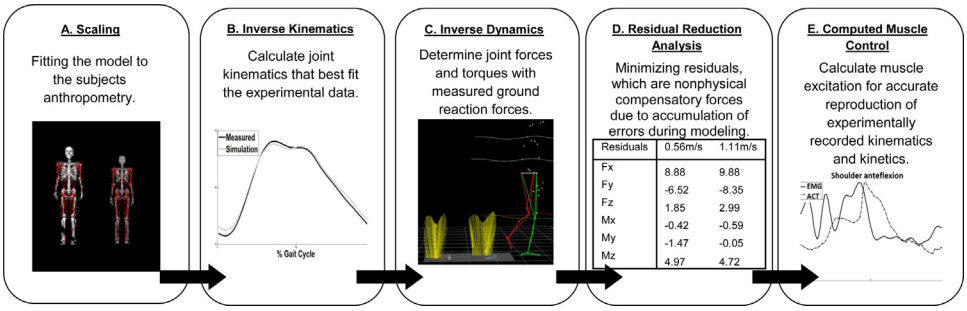

Fig. 1. Workflow of the simulation process.
First the model was scaled to the subject's anthropometry using experimentally measured marker positions (A). The Inverse Kinematics tool was then used to calculate joint kinematics that best fit the experimental marker data (B). The measured ground reaction forces were used to determine net forces and torques for each joint via the Inverse Dynamics tool (C). During the process of modelling, accumulation of errors can lead to nonphysical compensatory forces. The Residual Reduction Algorithm tool was used to minimize these residuals (D). Thereafter, the Computed Muscle Control tool (CMC) calculated muscle excitations of all muscle-tendon actuators in the model allowing accurate reproduction of the experimentally recorded kinematics and kinetics for all the experimentally measured ground reaction forces and marker data in the different conditions (E).