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The relationship between the anteroposterior and mediolateral margins of stability in able-bodied human walking

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ABSTRACT

Background: Control of dynamic balance in human walking is essential to remain stable and can be parameterized by the margins of stability. While frontal and sagittal plane margins of stability are often studied in parallel, they may covary, where increased stability in one plane could lead to decreased stability in the other. Hypothetically, this negative covariation may lead to critically low lateral stability during step lengthening.

Research question: Is there a relationship between frontal and sagittal plane margins of stability in able-bodied humans, during normal walking and imposed step lengthening?

Methods: Fifteen able-bodied adults walked on an instrumented treadmill in a normal walking and a step lengthening condition. During step lengthening, stepping targets were projected onto the treadmill in front of the participant to impose longer step lengths. Covariation between frontal and sagittal plane margins of stability was assessed with linear mixed-effects models for normal walking and step lengthening separately.

Results: We found a negative covariation between frontal and sagittal plane margins of stability during normal walking, but not during step lengthening.

Significance: These results indicate that while a decrease in anterior instability may lead to a decrease in lateral stability during normal walking, able-bodied humans can prevent lateral instability due to this covariation in critical situations, such as step lengthening. These findings improve our understanding of adaptive dynamic balance control during walking in able-bodied humans and may be utilized in further research on gait stability in pathological and aging populations.

1. Introduction

Human walking requires control of dynamic balance to stay upright while moving forward [1,2]. The walking human can be modeled as an inverted pendulum, representing the stance leg with the body's center of mass (CoM) on top. In this model, dynamic balance can be described by the relationship between the body's base of support (BoS) and the body's extrapolated CoM (XCoM) [3]. The mediolateral (ML) or anteroposterior (AP) distance (m) between the BoS and XCoM represents the margin of stability (MoS) [3]. The walker consistently progresses forwards when the BoS is placed behind and outside of the XCoM at foot-strike, i.e. when ML MoS is positive and AP MoS negative [4]. If the ML XCoM exceeds the ML BoS, a corrective step, counter-rotation strategy, or application of external forces would be necessary to prevent a fall [5]. Research on the MoS has provided us with knowledge on balance strategies in able-bodied [6,7] and pathological walking [5,8]. However, while multiple studies report both the AP and ML MoS in parallel, the relationship between the two remains largely unexplored.

First, it should be noted that during walking the AP XCoM should be in front of the anterior BoS to optimally exploit the pendulum-like properties of the moving body and progress forwards efficiently [4]. Therefore, during walking humans are often mechanically unstable in the anterior direction. Then, hypothetically, the natural variation in step length during walking may lead to variation in AP MoS. For instance, an increase in step length would lead to forward displacement of the AP BoS, which in turn would lead to a less negative AP MoS, thereby decreasing anterior instability. Then, the increased step length may coincide with an increase in stance time of the contralateral leg, as it takes longer to swing the leg forward when one increases step length [9]. The resulting increase in stance time is known to lead to a larger lateral excursion of the CoM, which leads to a decrease in ML MoS if step width remains constant, thereby decreasing lateral stability [10]. Therefore, a

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covariation between AP and ML MoS could exist in human walking. Indeed, results from several studies suggest that AP and ML MoS covary. When people post-stroke increase ML MoS in response to a sideward perturbation during treadmill walking, AP MoS decreases simultaneously [11]. Furthermore, when participants were perturbed by an outward translation of the treadmill, step width increased while AP XCoM moved more in front of the leading foot, i.e. ML MoS increased while AP MoS decreased [12]. Recently, it was shown that an increase in post-stroke paretic AP MoS due to step lengthening in response to a forward perturbation during treadmill walking, coincided with a decrease in the paretic ML MoS [13]. In these studies, the relationship between AP and ML MoS was assessed post-factum in perturbation experiments. However, this relationship has not been assessed directly in unperturbed human walking, while knowledge on this covariation may be important for understanding dynamic balance control during human walking. Therefore, we study whether there is covariation between AP and ML MoS during normal walking in able-bodied adults.

While covariation between AP and ML MoS may occur during normal walking, we often modify step length during daily walking activities, for instance to avoid obstacles [14,15]. Step lengthening leads to decreased anterior instability. However, the increased step length would come with increased single support time (SST), which would lead to a decrease in ML MoS [10]. Therefore, the hypothesized covariation between AP and ML MoS could lead to critically low lateral stability during step lengthening. However, since able-bodied humans remain upright during step lengthening, we expect that corrections in SST or step width will occur to modify the relationship between AP and ML MoS to prevent a fall. Therefore, to assess whether the hypothesized covariation between AP and ML MoS could lead to critically low lateral stability, we also investigate the relationship between AP and ML MoS during intentional step lengthening.

Here, we investigate the relationship between AP and ML MoS in able-bodied normal walking and step lengthening. First, we assess differences in AP MoS, ML MoS, and spatiotemporal parameters during step lengthening compared to normal walking, to gain insight into stepping behavior during step lengthening. Then, we investigate the relationship between AP and ML MoS within the normal walking and step lengthening condition separately. Step lengthening will be induced by stepping onto targets that are projected on the treadmill. Participants will be paced during step lengthening to control for altered stepping strategies, e.g. step shortening or lengthening, in anticipation of the lengthened steps [16,17].

2. Methods

2.1. Participants

Fifteen able-bodied adults (eight females, mean ± SD age: 23 ± 2 years, height: 1.74 ± 0.07 m, weight: 68 ± 9 kg) participated in this study. Participants were included if they had no prior experience with dual-belt treadmill walking, to ensure a more homogeneous sample, and no known impairments that affect balance, gait, hearing, or sight. All procedures were in line with the Declaration of Helsinki [18] and approved by the local ethics committee of the Department of Human Movement Sciences of the University Medical Center Groningen (201900583). Every participant provided written informed consent before the experiment.

2.2. Protocol

Participants walked on an instrumented dual-belt treadmill in tied-belt mode (Motek, Amsterdam, NL). The belt speed was normalized to leg length by multiplying the nominal treadmill belt speed (v) of 1.0 m s^{-1} with the square root of each participant's leg length (l) [19]. Participants were not allowed to hold on to handrails during the experiment to prevent effects of external stabilization [20], but wore a safety harness which did not constrain movement or provide body weight support. The experimental setup is visualized in Fig. 1.

Participants familiarized themselves with dual-belt treadmill walking for 5 min [21], referred to as the normal walking condition. During the fourth minute of normal walking, the participant's preferred walking cadence was recorded. From the fifth minute, participants were instructed to pace their steps to a metronome, set to their preferred cadence. Both the left and right steps were paced.

After familiarization, the text 'GO' was projected in front of the participant to indicate the start of the step lengthening condition. Participants continued to walk at the normalized walking speed and paced their steps to the metronome. After 10 strides the text 'Left' or 'Right' was projected onto the treadmill, indicating whether the upcoming target should be stepped on with the left or right foot. Below the 'left'/'right' text a number was projected, indicating the number of steps until the target would appear, starting from 3, counting down to 1. After the countdown, the target was projected on the treadmill as a white bar over the width of the treadmill with a length of 15 cm. The 'left'/'right' text, the countdown number, and the target all appeared or counted down on subsequent ipsilateral steps. For example, the text 'Right' and '3' would appear on the first right step, the '3' would count down to '2' on the second right step, the '2' would count down to '1' on the third right step, the target would appear on the fourth right step and the participant would step on the target with the fifth right step. When the target appeared, both the 'left'/'right' text and the countdown disappeared. A video of the step lengthening condition is available in Supplementary Video 1.

Participants were instructed not to approach the target in the preceding steps. The targets were projected at 40, 60 or 80 cm, normalized to leg length by multiplying the nominal distance by each participant's leg length (l) [19], in front of the participant's center of pressure (CoP) under the leading foot at foot-strike, to control for participants' varying AP positions on the treadmill. Target distances were based on pilot testing, to induce a large variation in step length while remaining feasible. Participants received 15 targets at each distance and on each side, to a total of 15 targets x 3 distances (40, 60 and 80 cm) x 2 sides (left and right) = 90 targets, in randomized order. The target disappeared when the participant made the target step, regardless of whether the participant hit the target or not. After the target step, a new 'left'/'right' text with countdown would be projected after 13 or 14 steps (depending on whether it was a left or right target), so that a new target step would be made every 21 or 22 steps.

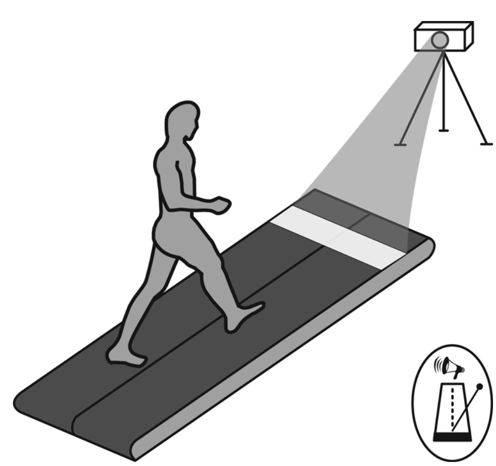

Fig. 1. Visualization of the experimental setup. For a video example of the step lengthening condition see Supplementary Video 1. Target projection and metronome pacing occurred only during the step lengthening condition.

2.3. Data analysis

The treadmill's embedded force plates measured 3D ground reaction forces (GRF (N)) and 2D CoP positions (m). Data were recorded at 1000 Hz and stored on an encrypted drive for offline analysis. AP CoP was analyzed online to determine the position of the targets on the treadmill.

All data and statistical analyses were performed in MATLAB (r2020a, the MathWorks Inc., Natick, MA, USA). CoP and GRF data were low-pass filtered at 15 Hz with a 2nd order Butterworth filter. Gait events were detected by finding the point at which the vertical GRF crossed the threshold of 50 N. SST (s) was defined as the period between contralateral toe-off and contralateral heel-strike. Step width (m) was calculated as the difference between the left and right ML CoP at toe-off. Step length (m) was calculated as the difference between the left and right AP CoP at heel-strike.

AP and ML CoM (m) was calculated by combining the 1) twice integrated and high-pass filtered CoM acceleration (m s^{-2}), acquired by dividing the respective GRF by the participant's weight (kg), and 2) the low-pass filtered CoP [6,22,23]. For both filters a 2nd order Butterworth filter with 0.2 Hz cut-off was used. AP and ML XCoM (m) were calculated with Eq. (1) [23], where g is the gravitational acceleration (9.81 m s^{-2}), l is leg length (m) measured from trochanter major to the floor multiplied by 1.2 [23] and vCoM is CoM velocity (m s^{-1}). In the AP direction, the average treadmill belt speed was added to vCoM [24]. AP and ML MoS (m) were defined as the distance between AP or ML CoP and AP or ML XCoM at contralateral toe-off, and calculated for the left and right leg independently [3,10]. A positive ML MoS indicates that the XCoM is medial of the BoS and a negative ML MoS indicates that the XCoM is lateral of the BoS. A positive AP MoS indicates that the XCoM is posterior to the BoS and a negative AP MoS indicates that the XCoM is anterior to the BoS.

$$XCoM = CoM + \frac{vCoM}{\sqrt{g/l}} \tag{1}$$

2.4. Statistical analysis

To assess normal walking, the last 35 left and 35 right steps of unpaced normal walking were selected for each participant. To assess step lengthening, 35 largest left step lengths and 35 largest right step lengths in the step lengthening condition were selected for each participant, which means that 10 left and 10 right target steps were not selected to exclude steps in which the target was projected outside of the treadmill, e.g. when the participant walked too far to the front of the treadmill. To assess whether pacing affected the normal steps in the step lengthening condition, we selected the ipsilateral steps preceding the lengthened steps, from here on referred to as pre-lengthening, resulting in 35 left and 35 right pre-lengthening steps. First, we conducted paired t-tests to compare AP MoS, ML MoS, SST, step width, and step length between 1) normal walking and pre-lengthening, and 2) normal walking

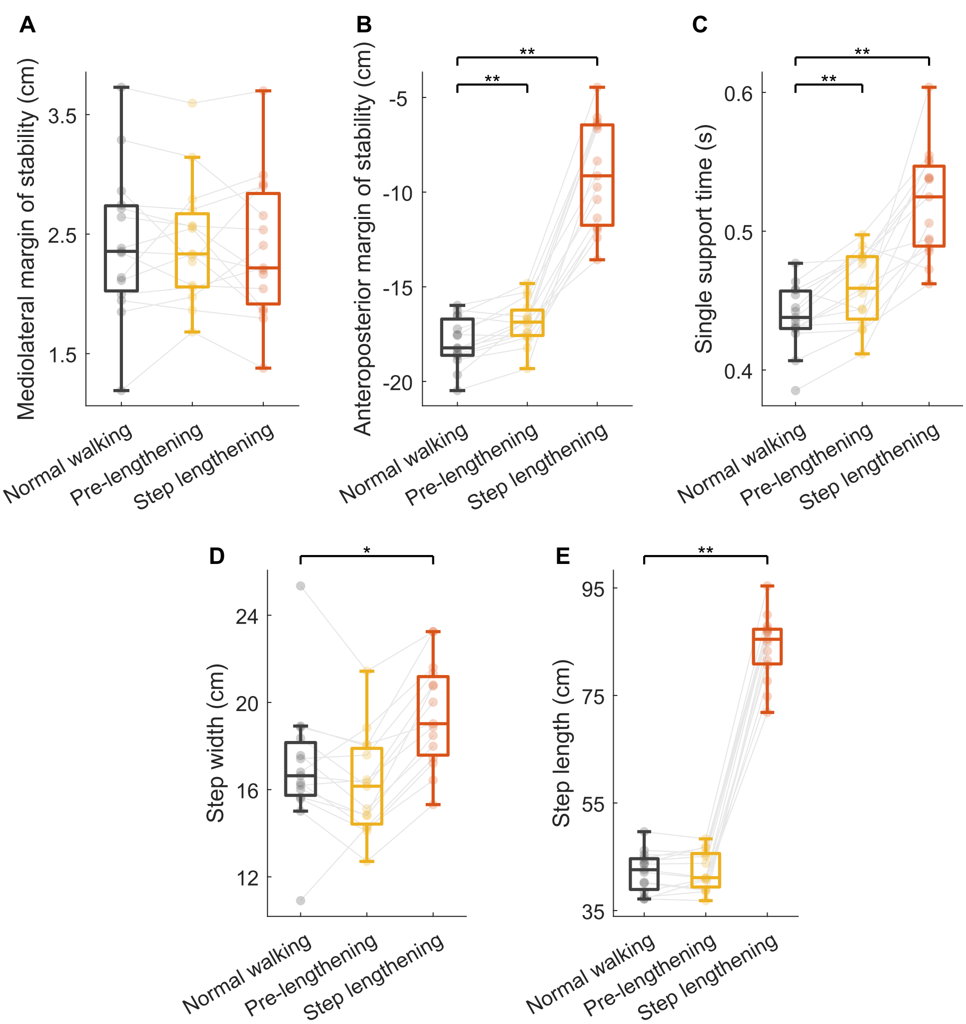

Fig. 2. Group distribution (N = 15) of dynamic balance and spatiotemporal parameters during the normal walking, pre-lengthening and step lengthening conditions. The panels show (A) mediolateral margins of stability, (B) anteroposterior margins of stability, (C) single support time, (D) step width, and (E) step length. Asterisks indicate significant differences between conditions (*$p < 0.05$, ** $p < 0.001$). Dots connected by grey lines represent individual participants.

and step lengthening. The average value of the 35 left and 35 right steps was calculated for each parameter, condition, and participant for the paired t-tests. Second, we fit two linear mixed-effects models to assess the relationship between the predictor AP MoS and the dependent variable ML MoS, in 1) normal walking and 2) step lengthening. For the linear mixed-effects models, the steps were not averaged, but the individual 35 left and 35 right steps from normal walking and step lengthening were included. These models included a main effect for AP MoS and a random intercept for each participant. Statistical significance was set a Holm-Bonferroni corrected alpha of 0.05 [25].

3. Results

Boxplots are shown in Fig. 2. The paired t-tests showed no significant differences between normal walking and pre-lengthening in ML MoS ($p = 0.8682$), step width ($p = 0.0962$), and step length ($p = 0.4122$), and small significant differences between normal walking and pre-lengthening in AP MoS (normal: -0.1791 m $\pm$ 0.0129, pre-lengthening: -0.1688 m $\pm$ 0.0117, $t(14)= -4.4122$, $p < 0.001$) and SST (normal: 0.439 s $\pm$ 0.023, pre-lengthening: 0.460 s $\pm$ 0.027, $t(14)= -4.8089$, $p < 0.001$), which indicates negligible effects (respectively 1.03 cm and 21 ms) of pacing on pre-lengthening. Therefore, we do not expect that pacing altered gait strategies during the step lengthening condition. This indicates that metronome pacing reduced anticipatory stepping strategies during the step-lengthening condition.

The other paired t-tests showed significant differences between normal walking and step lengthening in AP MoS (normal: -0.1791 m $\pm$ 0.0129, lengthening: -0.0901 m $\pm$ 0.0284, $t(14)= -14.018$, $p < 0.001$), SST (normal: 0.439 s $\pm$ 0.023, lengthening: 0.520 s $\pm$ 0.038, $t(14)= -9.532$, $p < 0.001$), step width (normal: 0.1704 m $\pm$ 0.0300, lengthening: 0.1945 m $\pm$ 0.0238, $t(14)= -3.9293$, $p = 0.0015$), and step length (normal: 0.4220 m $\pm$ 0.0370, lengthening: 0.8410 m $\pm$ 0.0603, $t(14)= -43.0661$, $p < 0.001$), but no significant difference in ML MoS ($p = 0.6930$).

To investigate the relationship between AP and ML MoS we fit two linear mixed-effects models (Fig. 3). The first model (normal walking) showed a significant main effect of AP MoS on ML MoS ($\beta=-0.1409$, $F(1,1048) = 78.853$, $p < 0.001$), which indicates a negative relationship between AP and ML MoS during normal walking. The second model (step lengthening) showed no significant effect of AP MoS on ML MoS ($\beta = 0.0172$, $F(1,1048) = 2.234$, $p = 0.1353$), which indicates there is no relationship between AP and ML MoS during step lengthening.

4. Discussion

Here, we investigated the relationship between AP and ML MoS in able-bodied adults during normal walking and step lengthening. During step lengthening, ML MoS did not change, but step length, SST, step width increased, and AP MoS became less negative. The latter is in line with previous research, where the authors found an increase in AP MoS in extremely long steps after an AP treadmill belt translation [26] and an increase in AP MoS when increasing step length during paced treadmill walking [27]. Furthermore, we found covariation between AP and ML MoS during normal walking, but not during step lengthening. This implies that frontal plane stability negatively covaries with sagittal plane stability in human walking, but that able-bodied humans may compensate for this covariation during step lengthening, by increasing step width when lateral stability may become critically low.

Although we found covariation between AP and ML MoS during normal walking, and step lengthening resulted in a less negative and large variation in AP MoS, ML MoS did not decrease during step lengthening compared to normal walking. This is in line with a previous study [27], where increasing step length during treadmill walking led to an increase in AP MoS, but not ML MoS. One would expect a smaller ML MoS during step lengthening because SST increased, which is known to decrease ML MoS [4,10]. However, the ML MoS is also determined by foot placement [5,28], as an increase in step width leads to an increase in BoS and thereby an increase in ML MoS. The results showed an increase in step width in step lengthening compared to normal walking. Therefore, the expected decrease in ML MoS as a result of increased SST during step lengthening may have been canceled out by an increase in step width. This indicates that able-bodied humans may increase their step width during step lengthening to maintain lateral stability.

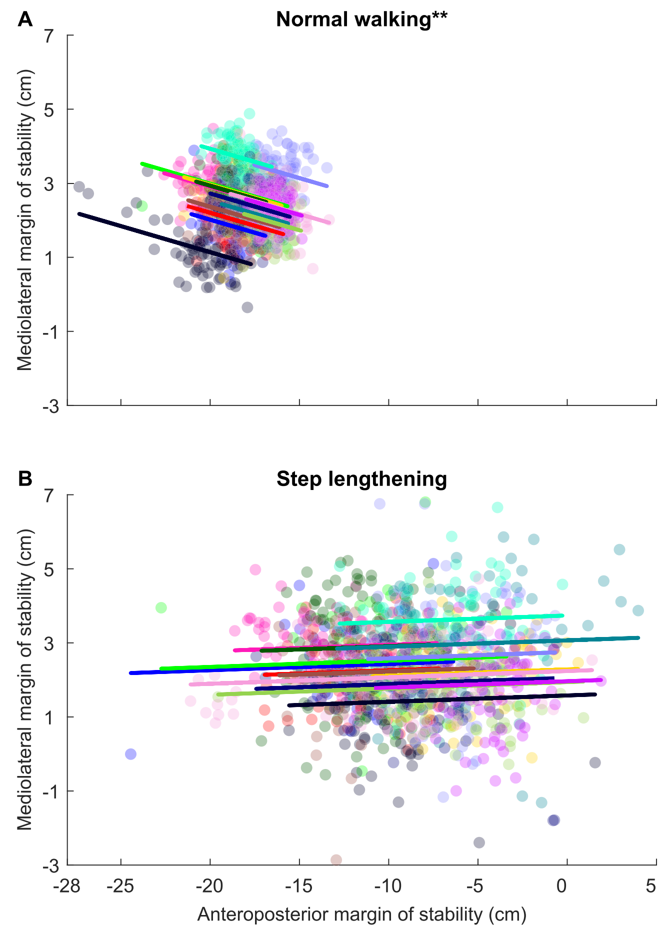

Fig. 3. The relationship between the anteroposterior and mediolateral margins of stability during (A) the normal walking condition and (B) the step lengthening condition. Every color represents an individual participant (N = 15), every dot represents a single step. Every line represents a single participant's fit, due to the use of random intercepts for participants. Only the relationship in (A) was statistically significant, as indicated by the double-asterisk ($p < 0.001$).

We found a negative covariation between AP and ML MoS during normal walking, where participants showed a negative AP MoS and a positive ML MoS. This indicates that the AP XCoM is in front of the AP BoS, which is anteriorly unstable, and that the ML XCoM is medial of the ML BoS, which is laterally stable. A less negative AP MoS, therefore, decreases anterior instability but coincides with a decrease in ML MoS, thereby reducing lateral stability, which indicates that increased sagittal plane stability may come at the cost of reduced frontal stability. At that point, alternative strategies, such as increasing step width, are necessary to maintain frontal plane stability.

We found no relationship between AP and ML MoS during step lengthening. As suggested, the hypothesized lateral instability during step lengthening may have been prevented by increasing step width. Additionally, one could question whether the inverted pendulum model is still valid in the context of instantaneous step lengthening. Additional mechanical work is necessary to accelerate the CoM forwards during step lengthening, while added mechanical work is not necessary in the inverted pendulum model [29]. During step lengthening, AP foot placement is then no longer the result of passive dynamics [2], but is instead controlled actively. Therefore, the forward-directed propulsion of the CoM may have prevented the CoM from falling sideward during the stance phase, as it would have in a passive inverted pendulum motion, which could explain why ML MoS did not change during step lengthening compared to normal walking.

Previous research showed a negative covariation between AP and ML MoS during walking in people post-stroke, when the paretic step was lengthened in response to rapid accelerations of the treadmill's belt [13]. In contrast, no covariation was found during step lengthening in able-bodied walking here. We propose two reasons for this discrepancy. First, in the former study participants increased step length in response to an external perturbation, in which a time-critical reactive stepping mechanism was utilized to prevent a fall. Whereas here, participants used proactive control of stepping to voluntarily step onto a target and increase step length. Therefore, one could question whether lateral foot placement strategies as found here, could still be utilized to increase lateral stability under time-critical conditions during reactive stepping in pathological populations. Second, in the former study [13], increased reliance on pelvic rotation to increase paretic step length was suggested as a mechanism underlying the covariation. However, the contribution of pelvic rotation to step length is quite small [30]. Alternatively, the negative covariation in reactive stepping post-stroke could be the result of abnormal coordination patterns, such as abnormal torque coupling, leading to the inability to combine hip flexion and abduction [31] and may thereby induce maladaptive paretic coupling [13].

While this study brings novel information on planar covariation in dynamic balance control during walking, it has a few limitations. The hypotheses of this study were predominantly based on the effect of changes in AP BoS on AP MoS, which would come with changes in SST and then coincide with changes in ML MoS. One could argue that step lengthening not only leads to a more anterior AP BoS, and thereby less negative AP MoS, but also a more anterior CoM position and higher CoM velocity, which would lead to a more anterior XCoM and thereby more negative AP MoS. However, the results support our hypotheses as an increase in step length led to a less negative AP MoS. Nonetheless, the AP MoS is also affected by AP CoM position and velocity and one should be aware that the AP MoS does not have to scale linearly with the AP BoS per se. Furthermore, the here presented results on step lengthening cannot be generalized to step shortening without considering the fact that step shortening might require other gait adaptations to remain upright. For instance, sudden step shortening might require a reduction in walking speed to slow down the CoM and prevent the AP XCoM from coming too far in front of the anterior BoS, which would otherwise lead to a forward fall.

Here, we found negative covariation between AP and ML MoS during normal walking, but not step lengthening. This indicates that an increase in anterior stability may come with a decrease in lateral stability, but that able-bodied humans can prevent lateral instability by altering foot placement. These findings improve our understanding of dynamic balance control and may be utilized to improve control of dynamic balance in pathological and aging populations, as these often prefer to lengthen rather than shorten their steps in obstacle avoidance [14,32], which could make them more vulnerable to lateral instabilities.

CRediT authorship contribution statement

Tom J.W. Buurke: Conceptualization, Methodology, Software, Formal analysis, Investigation, Writing - original draft, Visualization. **Rob den Otter:** Conceptualization, Methodology, Software, Formal analysis, Writing - review & editing, Supervision.

Declaration of Competing Interest

Tom Buurke was supported by a KU Leuven Internal Funds Postdoctoral Mandate. The sponsor had no involvement in the design, data collection, or writing of the manuscript.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.gaitpost.2021.08.008.

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