



How does ankle mechanical stiffness change as a function of muscle activation in standing and during the late stance of walking?

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Abstract

Objective: Ankle joint stiffness is known to be modulated by co-contraction of the ankle muscles; however, it is unclear to what extent changes in agonist muscle activation alone affect ankle joint stiffness. This study tested the effects of varying levels of ankle muscle activation on ankle joint mechanical stiffness in standing and during the late stance phase of walking.

Methods: Dorsiflexion perturbations were applied at various levels of ankle muscle activation via a robotic platform in standing and walking conditions. In standing, muscle activation was modulated by having participants perform an EMG target matching task that required varying levels of plantarflexor activation. In walking, muscle activation was modulated by changing walking speeds through metronome-based auditory feedback. Ankle stiffness was evaluated by performing a Least-squares system identification using a parametric model consisting of stiffness, damping, and inertia. The association between ankle muscle activation and joint stiffness was evaluated using correlation analyses. Linear regression models were used to determine the extent to which muscle activation contributed to ankle stiffness. An inclusive statistical approach (both classical and Bayesian analyses) was adopted to measure the statistical significance (p-value) and Bayes Factor (BF_{10}).

Results: Results indicate that plantarflexor activity was positively correlated with ankle stiffness in both standing and walking ($p < 0.001$, $BF_{10} > 900$), whereas dorsiflexor activity was negatively

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correlated with ankle stiffness in walking ($p=0.014$, $BF_{10}=3.9$) but not in standing ($p=0.725$). Regression analyses indicated that ankle muscle activation predicted about 84% of the variation in ankle stiffness in standing and 45% in walking ($p<0.001$, $BF_{10}>100$).

Conclusion: Ankle muscle activation significantly contributes to ankle stiffness during standing and walking.

Significance: The results highlight the role of muscle activation on maintaining joint stiffness and underscore the importance of accounting for muscle activation when measuring ankle stiffness in healthy as well as patient populations.

Keywords

Impedance; rehabilitation; biomechanics; neural control; co-contraction

I. Introduction

Gait research on healthy human subjects suggests that the human nervous system tries to modulate the dynamic mechanical properties (i.e., the stiffness and viscosity of the limbs and joints) to achieve stability in static and dynamic tasks [1, 2]. Prior work has also shown that ankle joint stiffness is significantly altered after stroke and contributes significantly to functional limitations after stroke [3-5]. Collectively, these results suggest that ankle joint stiffness measurement may serve as a fundamental quantitative tool for assessing gait quality and stability.

Several factors (*e.g.*, age, gender, pathology, *etc.*) could affect the stiffness properties of a joint [4, 6-9]. Recent research on healthy human adults suggests that ankle stiffness during standing is linearly modulated based on the loading and co-contraction of the muscles spanning the ankle joint [7]. However, it is unclear to what extent changes in agonist muscle activation alone (*i.e.*, without much change in co-activation of antagonistic muscles) contributes to ankle stiffness. Further, the extent to which these results translate to dynamic tasks such as walking is also not known, as prior research has been only performed in static tasks [10].

An understanding of the effects of muscle activation on ankle stiffness will not only provide new insight into neural control during normal and pathological locomotion but may also new ideas to treat abnormal gait after a neurological injury, such as stroke [9]. However, unlike static tasks, modulating muscle activation volitionally during gait is not easily feasible without affecting normal mechanics of human gait. Thus, muscle activation has to be indirectly modulated to study its effect on ankle stiffness. One approach to altering ankle muscle activation during walking is by manipulating the walking speed, as ankle muscle activation is known to increase with increase in walking speed [11]. Here, we used controlled perturbations (such perturbations are necessary to quantify joint stiffness because human joints are actively actuated by the muscles [12]) in standing and during the late stance of walking to better understand how muscle activation affects the measurements of ankle stiffness. We hypothesized that ankle stiffness would increase with increasing plantarflexor

muscle activation and changes in muscle activation would significantly predict changes in stiffness both during standing and walking.

II. METHODS

A. Participants

Twelve (7 male, 5 female) participants (Age: 31.5 ± 14.2 yrs; Height: 1.75 ± 0.10 m; Mass: 71.0 ± 15.3 kg) with no history of neurological or orthopedic disorders participated in this study. All participants but two were right leg dominant, as established by their preferred leg to kick a ball [13, 14]. The experimental protocol was approved by the University of Michigan Institutional Review Board, and all participants provided written informed consent prior to participation. All experiments were performed on the same day to minimize any measurement noise from experimental or anatomical changes.

B. Experimental Protocol

For each participant, we measured overground walking speed, maximum plantarflexor (medial gastrocnemius and soleus) and dorsiflexor (tibialis anterior) muscle activation (M-wave), and ankle impedance on the dominant leg. Ankle impedance measurements were performed during two tasks – walking and quiet standing.

In the walking task, participants walked across a custom-designed mechatronic platform. This platform induced a small rotational perturbation (constant velocity ramp; direction: dorsiflexion; amplitude: 2 deg; duration: 75 ms; velocity: 45.8 deg s^{-1} ; max acceleration: 1800 deg s^{-2}) [15, 16] during the late stance of walking. One hundred walking bouts were recorded, for each participant, at each of three different walking speeds – their self-selected (measured during overground walking), 25% slower, and 50% slower. Walking speed was controlled by asking participants to match their heel strike with a loud audible tone produced using a LabVIEW-based digital metronome. The metronome frequency was changed between conditions to alter the walking speed.

In the standing task, participants stood with their dominant foot placed flat on the mechatronic platform, and non-dominant foot rested on a stationary surface while the platform induced perturbations with the above characteristics. Participants stood normally (quiet standing) or matched their plantarflexor muscle activation (smoothed [bandpass filtered between 20Hz and 500Hz and linear enveloped at 2Hz] average of soleus and medial gastrocnemius) to 5%, or 10% of their maximum M-wave response obtained using a constant current stimulator (DS7AH, Digitimer, Hartfordshire, UK). Feedback for the muscle activation was provided using a LabView based graphical display (Figure 1).

In both paradigms we used 12 motion capture cameras (Miquis M3, Qualisys, Göteborg, Sweden) to record the motion of 31 reflective markers (4 makers placed on each shank, 6 on each foot, 2 on ankle and knee anatomical landmarks, and 3 on the robotic platform). We recorded muscle activation using 6 surface EMG sensors (Trigno Avanti, Delsys, Natick, Massachusetts, USA) placed on the medial gastrocnemius, soleus, and tibialis anterior muscles of both legs. Finally, a six-axis force plate (Kistler Inc., Novi, Michigan, USA) recorded the ground reaction forces of the dominant leg when the subject stepped on the

robotic platform. In 50% of the recorded walking trials, the mechatronic platform elicited a small rotational perturbation of the stance foot about the ankle joint in the sagittal plane at about 55% of the stance phase of the gait [17]. In the standing trials, the platform performed the same rotational perturbation 10 times, with each sequential perturbation timed to occur randomly between 10 and 15 seconds after the previous perturbation.

C. Ankle Stiffness Estimation

Ankle stiffness was estimated by computing the participant-specific ensemble averages of changes in ankle angle and torque due to perturbation and fitting a 2nd order parametric model (Figure 1). Ankle angle was determined by calculating the 3-d transformation matrix for the foot and shank rigid bodies relative to their static poses [18], determining the Euler angle for the ankle axis of rotation and taking the difference between these two measured angles. Angular velocity and acceleration were determined numerically by differentiating the ankle angle data [19]. Ankle torque was determined by filtering out the inertial component for each channel of the force plate and using the combination of ground reaction forces and center of pressure to calculate the effective force-torque pair acting at the ankle joint center and about the ankle axis of rotation.

Ankle joint kinematics and kinetics were filtered using a 4th order, zero-phase, low-pass Butterworth filter with a 20Hz cutoff. These signals were used to determine the torque response and rotation of the ankle over a time window of ~ 100 ms after the perturbation was triggered [17].

As in our previous work [18], we fit a 2nd order parametric model to determine the impedance properties of the ankle joint:

$$T_p = I_{tot} \ddot{\theta}_p + b_a \dot{\theta}_p + k_a \theta_p, \quad (1)$$

where T_p is the torque response to perturbation, I_{tot} is the total inertia of the foot and other coupled body segments, k_a and b_a are the stiffness and viscosity of the ankle, θ_p is the angular perturbation displacement, and the dot operator denotes the time derivative. While we estimated all components of ankle impedance (stiffness, viscosity, and inertia), this work focused on the analysis of ankle joint stiffness, as prior work has shown that this component of ankle impedance is the most affected after a neurological injury [5].

D. Muscle Activation Analysis

EMG signals were band-pass filtered (20 to 500Hz) using a butterworth filter (zero-phase, 4th order), rectified and smoothed using a low-pass filter of the same type with a cut-off frequency of 6Hz. The EMG activity during the standing task was normalized to the maximum M-wave response of the corresponding muscles to mimic the normalization process for the target matching task performed during standing. The EMG activity during the walking task was normalized to the peak EMGs observed during the unperturbed trials at self-selected walking speed. For the standing condition, the mean normalized ankle muscle EMG during the entire target matching trial was used in the analysis. For the walking condition, since the participant was not matching a background EMG activity and the EMG activity changes across the entire gait cycle, the mean normalized ankle muscle EMG

observed over the same time window as the perturbation response (i.e., ~ 100 ms after the perturbation was triggered) was used in the analysis. An electromechanical delay of 80 ms was assumed for EMG calculations in the walking task [20].

E. Data Reduction and Analyses

The mean EMG signals of the gastrocnemius and soleus muscles were scaled and summed to compute the mean plantarflexor activation during the standing and the walking task. For the standing task, the relative change in ankle stiffness and mean EMG activity of the plantarflexor and dorsiflexor muscles from the quiet standing condition was used in the analysis. For the walking condition, the relative change in ankle stiffness and mean EMG activity of the plantarflexor and dorsiflexor muscles from the 25% slower condition was used in the analysis. Correlation analyses were used to determine the strength of association (0-0.19 = very weak, 0.2-0.39 = weak, 0.40-0.59 = moderate, 0.6-0.79 = strong, and 0.8-1 = very strong correlation) between the change in EMG activity of the ankle muscles and the change in ankle stiffness. To determine whether muscle activation contributes to ankle stiffness, the change in EMG activity of the plantarflexor and dorsiflexor muscles were regressed with the change in ankle stiffness. Because of the small sample size, we adopted an inclusive statistical approach where we performed both classical and Bayesian analyses [21-24]. More details regarding the Bayesian analysis and how to interpret the results of this analysis are provided in the JASP manual: (http://static.jasp-stats.org/Manuals/Bayesian_Guide_v0.12.2.pdf) and in the supplementary material document. All statistical analyses were performed in JASP version 0.14. The default prior in JASP was used for Bayesian correlation and regression analyses. The Bayes Factor Robustness Check analysis was performed for a wide range of prior distributions to examine the extent to which the results were affected by the prior specification. A significance level of $\alpha = 0.05$ was used for classical correlation and regression analyses.

III. Results

The mean plantarflexor and dorsiflexor activation during standing and walking are shown in Figure 2a and Figure 2b, respectively. In standing, participants modulated their plantarflexor activation without much change in dorsiflexor activation, indicating that they did not use co-contraction during target matching. In walking, increase in walking speed increased both the plantarflexor and dorsiflexor activation; however, at the time of perturbation, participants exhibited primarily an increase in plantarflexor activation (Figure 2c).

The mean impedance parameters (stiffness, viscosity, and inertia) estimated during standing and walking conditions are provided in Table I. The impedance measurements accounted for more than 95% of variation in the torque response due to perturbation in the standing ($95.4 \pm 4.6\%$) and walking ($97.9 \pm 2.2\%$) tasks, indicating that the 2nd order parametric model provided a good fit for the observed data (Supplementary Figures 1 and 2). The relationship between changes in ankle muscle EMG and ankle stiffness obtained from the standing and walking perturbations are shown in Figure 3a and Figure 3b, respectively. Ankle stiffness was found to strongly increase ($r > 0.6$) with increasing plantarflexor activation in both the standing and the walking task ($p < 0.001$). Additionally, ankle stiffness was found

to moderately decrease ($r>0.4$) with increasing dorsiflexor activation in the walking task ($p=0.014$) but no relationship was observed in the standing task ($p=0.725$).

Linear regression analyses indicated that both plantarflexor and dorsiflexor activation explained about 84% and 45% of variation in ankle plantarflexion stiffness in standing ($p<0.001$) and the late stance of walking ($p<0.001$), respectively. Bayesian statistical analysis of the likelihood of these linear models show that these models have strong or extremely strong support (Bayes Factor > 10) for the two-sided alternative hypothesis based on the experimental data and that the likelihood of these models is robust to changes in the prior distribution of the data (Figures 4a and 4b, Table 2).

IV. Discussion

This study was performed to evaluate the effect of muscle activation on measurements of ankle stiffness in standing and during the late stance of walking. To this end, we provided controlled perturbations at various levels of ankle plantarflexor activation during standing and at different gait speeds during walking. We then evaluated the relationship between changes in muscle activation and changes in stiffness measurements using correlation and linear regression models. The principal findings of this study were: 1) changes in plantarflexor muscle activation were linearly associated with changes in ankle stiffness during both standing and walking tasks, 2) changes in dorsiflexor muscle activation were moderately inversely associated with changes in ankle stiffness during the walking task, but not in the standing task, and 3) changes in plantarflexor and dorsiflexor muscle activation explained a large percent of the variation in ankle stiffness during standing and walking tasks. These results establish for the first time that ankle muscle activation during functional tasks such as standing and walking contributes to the ankle stiffness observed in these tasks. Our results also emphasize the importance of accounting for muscle activation when evaluating joint stiffness in static and dynamic conditions.

A. Relationship between joint stiffness and muscle activation

Our finding that ankle stiffness increases with increasing plantarflexor activation was consistent with previous studies performed in seated tasks [10, 25, 26]. This finding matches our intuition about the mechanisms involved in joint stiffness modulation, i.e., increasing muscle activation increases the short-range stiffness (i.e., stiffness from deformation of attached actin-myosin cross-bridges) of the ankle muscles, thus increasing joint stiffness [27-29]. The negative correlation between dorsiflexor activation and ankle stiffness in walking, however, does not match this intuition. While such a relationship has been observed before [30], the underlying mechanism for this observation is currently unclear.

One potential explanation for the observed negative correlation between dorsiflexor activity and ankle stiffness in the walking task could be the covariation in plantarflexor and dorsiflexor muscle activation. When evaluating the relationship between plantarflexor and dorsiflexor muscles during standing and walking, we found a significant negative relationship during walking but no relationship in standing (Supplementary Figure 3). It is to be noted that the ankle dorsiflexors acted as an antagonist to the plantar flexor muscles while performing the standing and walking tasks. Thus, the negative relationship between

changes in antagonistic dorsiflexor activation and ankle stiffness suggests that the results of prior studies reporting an increase in ankle stiffness with increasing co-contraction levels [7] could have been primarily mediated by the activation of agonist muscles performing the task. Further exploration of the effects of low and high levels of co-contraction on ankle stiffness (in conjunction with isolated effects of agonist activation) might help answer this question.

It is important to note that the variation in dorsiflexor activation in standing was much lower than during the late stance of walking, which might have contributed to the lack of correlation between ankle stiffness and dorsiflexor activation in standing. For the task being performed, subjects can maintain static equilibrium on the platform while increasing plantarflexor activation using increased dorsiflexor activation (i.e., by increasing co-contraction) or forward shifts of their center of pressure or a combination of both [31]. Unlike prior experiments [7], we intentionally chose to not control for the center of pressure location, as we wanted to study the isolated effects of plantarflexor activation on ankle stiffness and not the effects of co-contraction. In our experiment, participants primarily chose to shift their center of pressure forward while increasing their plantarflexor activation (Figure 5), which resulted in similar dorsiflexor activation between different standing conditions. Thus, it is likely that the lack of variation in dorsiflexor activation could have prevented us from establishing a relationship between dorsiflexor activation and ankle stiffness during standing. To further evaluate this issue, we performed a pilot evaluation ($n = 6$), where the perturbation was applied when participants were matching differing levels of dorsiflexor activation. The results of this experiment indicated that there was a weak positive correlation between changes in dorsiflexor activation and changes in ankle joint stiffness. However, the changes in plantarflexor activation still explained the majority of the variation in the changes in ankle joint stiffness (Supplementary Figure 4).

B. Implications for ankle impedance measurements

Our results for the standing task match the current understanding of the implications of increasing ankle activation on ankle joint stiffness in standing [7, 32] as well as results from measurements performed in seated tasks [33] and re-affirm the importance of controlling for muscle activation during such measurements. Prior studies investigating these relationships in walking have considered muscle activation and co-contraction as possible causes of ankle stiffness differences observed in healthy [30] as well as clinical populations [5]; however, no significant relationship has been reported. Alterations in walking speeds create a large and consistent change in muscle activation within subjects, allowing us to study the effects of these modulations with greater certainty. The resulting relationships highlight the importance of ankle activation changes on ankle stiffness and suggest that reductions in walking speed and maximum muscle activations might produce ankle stiffness changes observed in clinical populations.

C. Limitations

Stiffness measurements are sensitive to misalignments of the ankle joint rotation axis and the rotation axis of the Perturberator robot. Hence, some amount of measurement error would have been introduced in our observed stiffness values, as it is nearly impossible

to precisely align the robot and the ankle axes during experimental conditions. However, we believe that the impact of the axes alignment errors on the observed outcomes was minimal because these errors are random (and not systematic). Moreover, we observed an average misalignment error of < 1 cm, which translates to only about 5% of measurement error [15]. Another limitation is that these measurements assume that the dynamics of the ankle joint are well represented by a second-order system and that walking impedance is a piecewise constant function of stride percentage. The first of these assumptions is a reasonable simplification of dynamics, while the second is necessitated by the large amount of data required to measure impedance at any individual time-point over a stride (about 40 trials including unperturbed trials based on our sensitivity analysis; see Supplementary Figure 5). Even with the limitations caused by these assumptions, we found that impedance measurements accounted for more than 95% of the variation in the torque response after perturbation in the standing and walking tasks.

While this study provides a first insight into the isolated effects of ankle muscle activation on ankle stiffness in the late stance of walking, it is limited by small sample size. However, the large coefficient of variation and the robustness of the Bayes factor suggest that this limitation might not have a large effect on the results of the analysis. Another limitation was that we controlled the time at which the perturbation was provided but not the angle at which the perturbation occurred. As a result, some amount of changes in stiffness values could be due to the changes in the “operating point” on the force-length relationship. However, we note that the average differences in ankle joint angle between different conditions were about 2 degrees (Supplementary Figures 6-8), whereas the changes in stiffness values were 35% to 108%, indicating that most of the changes in ankle joint stiffness were due to changes in muscle activation and not due to alterations in contributions of the passive stiffness to the overall stiffness. We also note that the results are only applicable to the late stance phase of the gait, and further experiments are required to evaluate if the effect of muscle activation on ankle joint stiffness is similar across the gait cycle. Finally, it is unclear whether the observed relationship between muscle activation and ankle stiffness is specific to a muscle group/joint or a more generalized phenomenon. Though we have only measured the ankle stiffness for plantarflexion perturbations in the dominant leg of our subjects, based on our understanding of the mechanics of the ankle joint, this relationship is likely to be mirrored for dorsiflexion perturbations, i.e. stiffness increasing with increased activity of the dorsiflexors, and to remain unchanged bilaterally. However, future research is needed to verify this premise.

V. Conclusion

In conclusion, the results of the study indicate that ankle stiffness is significantly affected by ankle muscle activation in standing and during the late stance phase of walking. Specifically, ankle stiffness strongly increased with increasing levels of plantarflexor activation in both standing and walking tasks and moderately increased with decreasing levels of dorsiflexor activation in the walking task. More importantly, plantarflexor and dorsiflexor activation together explained more than 70% of variation in ankle stiffness during standing and walking tasks. These results highlight the importance of accounting for muscle activation when evaluating joint stiffness in static and dynamic conditions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgment

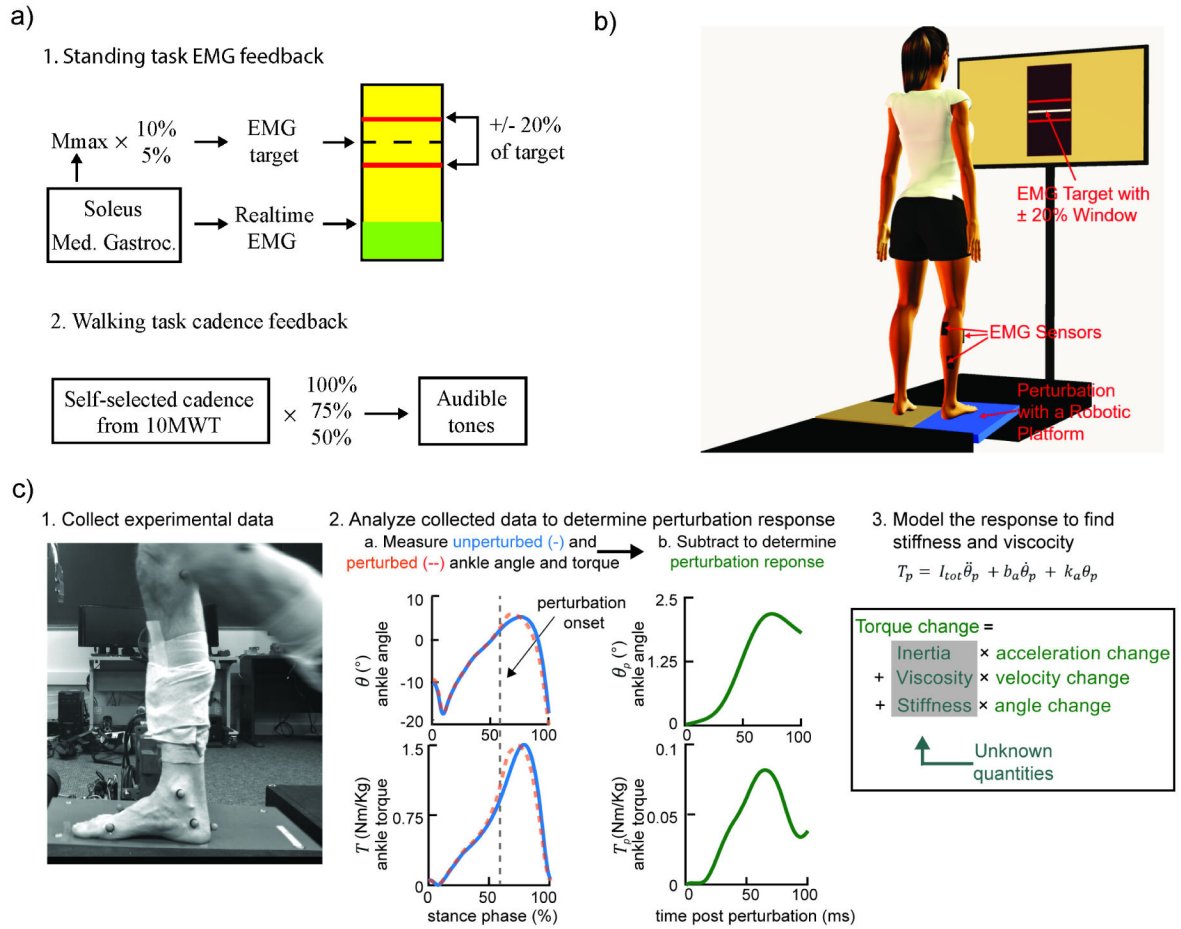
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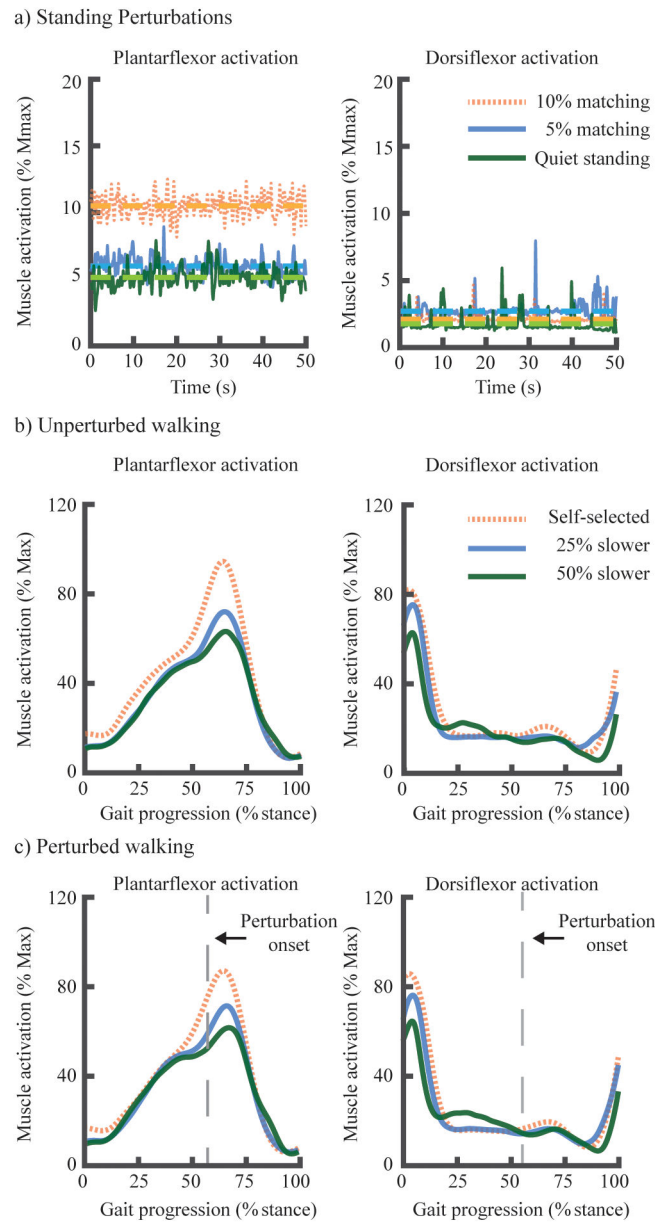
References

- [1]. Burdet E, Osu R, Franklin DW, Milner TE, and Kawato M, "The central nervous system stabilizes unstable dynamics by learning optimal impedance," *Nature*, vol. 414, no. 6862, pp. 446–9, Nov 22, 2001.
- [2]. Hogan N, "The mechanics of multi-joint posture and movement control," *Biol Cybern*, vol. 52, no. 5, pp. 315–31, 1985.
- [3]. Lamontagne A, Malouin F, Richards CL, and Dumas F, "Mechanisms of disturbed motor control in ankle weakness during gait after stroke," *Gait Posture*, vol. 15, no. 3, pp. 244–55, Jun, 2002.
- [4]. Roy A, Forrester LW, Macko RF, and Krebs HI, "Changes in passive ankle stiffness and its effects on gait function in people with chronic stroke," *J Rehabil Res Dev*, vol. 50, no. 4, pp. 555–72, 2013.
- [5]. Shorter AL, "Ankle Joint Mechanical Impedance during Locomotion - Implications for Prosthesis Control and Rehabilitation," *Biomedical Engineering*, Northwestern University, Chicago, 2020.
- [6]. Adjei E, Nalam V, and Lee H, "Sex Differences in Human Ankle Stiffness During Standing Balance," *Front Sports Act Living*, vol. 2, pp. 570449, 2020.
- [7]. Nalam V, Adjei E, and Lee H, "Quantification and Modeling of Ankle Stiffness during Standing Balance," *IEEE Trans Biomed Eng*, vol. PP, Sep 11, 2020.
- [8]. Shorter AL, Finucane S, and Rouse EJ, "Ankle Mechanical Impedance During Waling in Chronic Stroke: Preliminary Results," *IEEE Int Conf Rehabil Robot*, vol. 2019, pp. 246–251, Jun, 2019.
- [9]. Lee H, Rouse EJ, and Krebs HI, "Summary of Human Ankle Mechanical Impedance During Walking," *IEEE J Transl Eng Health Med*, Vol. 4, 2100407, pp. 1–7, Sep, 2016.
- [10]. Weiss PL, Hunter IW, and Kearney RE, "Human ankle joint stiffness over the full range of muscle activation levels," *J Biomech*, vol. 21, no. 7, pp. 539–44, 1988.
- [11]. Hof AL, Elzinga H, Grimmius W, and Halbertsma JP, "Speed dependence of averaged EMG profiles in walking," *Gait Posture*, vol. 16, no. 1, pp. 78–86, Aug, 2002.
- [12]. Rouse EJ, Gregg RD, Hargrove LJ, and Sensinger JW, "The difference between stiffness and quasi-stiffness in the context of biomechanical modeling," *IEEE Trans Biomed Eng*, vol. 60, no. 2, pp. 562–8, Feb, 2013.
- [13]. Krishnan C, Dharia AK, Augenstein TE, Washabaugh EP, Reid CE, Brown SR, and Ranganathan R, "Learning new gait patterns is enhanced by specificity of training rather than progression of task difficulty," *J Biomech*, vol. 88, pp. 33–37, May 9, 2019.
- [14]. Krishnan C, Ranganathan R, and Tatarbe M, "Interlimb transfer of motor skill learning during walking: No evidence for asymmetric transfer," *Gait Posture*, vol. 56, pp. 24–30, Jul, 2017.
- [15]. Rouse EJ, Hargrove LJ, Perreault EJ, Peshkin MA, and Kuiken TA, "Development of a mechatronic platform and validation of methods for estimating ankle stiffness during the stance phase of walking," *J Biomech Eng*, vol. 135, no. 8, pp. 081009-1–081009-8, Aug, 2013.
- [16]. Shorter AL, and Rouse EJ, "Mechanical Impedance of the Ankle During the Terminal Stance Phase of Walking," *IEEE Trans Neural Syst Rehabil Eng*, vol. 26, no. 1, pp. 135–143, Jan, 2018.

- [17]. Rouse EJ, Hargrove LJ, Perreault EJ, and Kuiken TA, "Estimation of human ankle impedance during the stance phase of walking," *IEEE Trans Neural Syst Rehabil Eng*, vol. 22, no. 4, pp. 870–8, Jul, 2014.
- [18]. Arun KS, Huang TS, and Blostein SD, "Least-squares fitting of two 3-d point sets," *IEEE Trans Pattern Anal Mach Intell*, vol. 9, no. 5, pp. 698–700, May, 1987.
- [19]. Scheid F, *Schaum's outline of theory and problems of numerical analysis*: McGraw-Hill, 1988.
- [20]. Inman VT, Ralston HJ, Saunders JB, Feinstein B, and Wright EW Jr., "Relation of human electromyogram to muscular tension," *Electroencephalogr Clin Neurophysiol*, vol. 4, no. 2, pp. 187–94, May, 1952.
- [21]. Krishnan C, "Effect of paired-pulse stimulus parameters on the two phases of short interval intracortical inhibition in the quadriceps muscle group," *Restor Neurol Neurosci*, vol. 37, no. 4, pp. 363–374, 2019.
- [22]. Ruiz-Ruano Garcia AM, and Lopez Puga J, "Deciding on Null Hypotheses using P-values or Bayesian alternatives: A simulation study," *Psicothema*, vol. 30, no. 1, pp. 110–115, Feb, 2018.
- [23]. Wagenmakers EJ, Love J, Marsman M, Jamil T, Ly A, Verhagen J, Selker R, Gronau QF, Dropmann D, Boutin B, Meerhoff F, Knight P, Raj A, van Kesteren EJ, van Doorn J, Smira M, Epskamp S, Etz A, Matzke D, de Jong T, van den Bergh D, Sarafoglou A, Steingroever H, Derks K, Rouder JN, and Morey RD, "Bayesian inference for psychology. Part II: Example applications with JASP," *Psychon Bull Rev*, vol. 25, no. 1, pp. 58–76, Feb, 2018.
- [24]. Wagenmakers EJ, Marsman M, Jamil T, Ly A, Verhagen J, Love J, Selker R, Gronau QF, Smira M, Epskamp S, Matzke D, Rouder JN, and Morey RD, "Bayesian inference for psychology. Part I: Theoretical advantages and practical ramifications," *Psychon Bull Rev*, vol. 25, no. 1, pp. 35–57, Feb, 2018.
- [25]. Galiana L, Fung J, and Kearney R, "Identification of intrinsic and reflex ankle stiffness components in stroke patients," *Exp Brain Res*, vol. 165, no. 4, pp. 422–34, Sep, 2005.
- [26]. Lee H, Krebs HI, and Hogan N, "Multivariable dynamic ankle mechanical impedance with active muscles," *IEEE Trans Neural Syst Rehabil Eng*, vol. 22, no. 5, pp. 971–81, Sep, 2014.
- [27]. Campbell KS, and Lakie M, "A cross-bridge mechanism can explain the thixotropic short-range elastic component of relaxed frog skeletal muscle," *J Physiol*, vol. 510 (Pt 3), pp. 941–62, Aug 1, 1998.
- [28]. De Groote F, Allen JL, and Ting LH, "Contribution of muscle short-range stiffness to initial changes in joint kinetics and kinematics during perturbations to standing balance: A simulation study," *J Biomech*, vol. 55, pp. 71–77, Apr 11, 2017.
- [29]. Rack PM, and Westbury DR, "The short range stiffness of active mammalian muscle and its effect on mechanical properties," *J Physiol*, vol. 240, no. 2, pp. 331–50, Jul, 1974.
- [30]. Nalam V, "Characterization of 2D Human Ankle Stiffness during Postural Balance and Walking for Robot-Aided Ankle Rehabilitation," *Arizona State University*, 2020.
- [31]. Kim D, and Hwang JM, "The center of pressure and ankle muscle co-contraction in response to anterior-posterior perturbations," *PLoS One*, vol. 13, no. 11, pp. e0207667, 2018.
- [32]. Kozasa K, Hoang PDH, Hirai H, Hori K, Niwa H, Fujihara R, Matsui K, Nishikawa A, and Krebs HI, "Electrical Stimulation to Modulate Human Ankle Impedance: Effects of Intervention on Balance Control in Quiet and Perturbed Stances." pp. 258–263.
- [33]. Sinkjaer T, Toft E, Andreassen S, and Hornemann BC, "Muscle stiffness in human ankle dorsiflexors: intrinsic and reflex components," *J Neurophysiol*, vol. 60, no. 3, pp. 1110–21, Sep, 1988.

**Fig. 1.**

(a) Schematic of the EMG and metronome feedback for the standing and walking tasks, respectively. (b) Set-up of the target matching experiment with EMG feedback during the standing perturbation task. (c) Experimental setup and methods for determining ankle impedance during late stance of walking. Participants walked across an instrumented mechatronic platform as kinematic and kinetic data were recorded. Data from perturbed and unperturbed walking conditions were combined to determine the perturbation response for the subject. A second order model of dynamics was then fit to this response and the impedance parameters: inertia, viscosity and stiffness are calculated.

**Fig. 2.**

Time-series plots of plantarflexor and dorsiflexor EMG for a) standing perturbations, b) unperturbed walking and c) perturbed walking. The dark lines show the ensemble average of EMG values across all subjects for the three walking speeds - self-selected (red), 25% slower (blue) and 50% slower (green)- as well as the three activation conditions - 10% matching (red), 5% matching (blue) and quiet standing (green). The vertical dashed line in the walking condition indicates the onset of the ankle perturbations. The horizontal dashed lines in the standing condition represent the mean of the muscle group activation for the given condition.

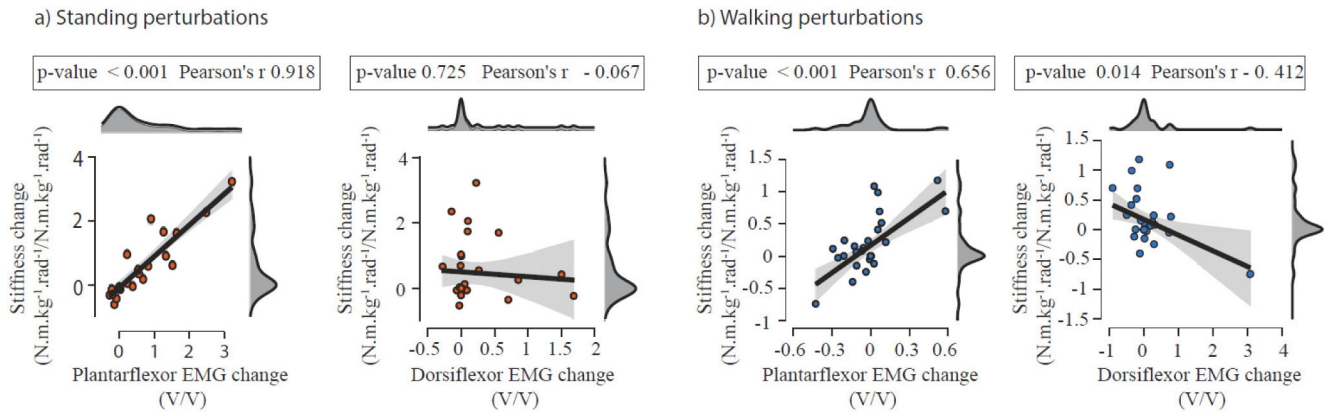
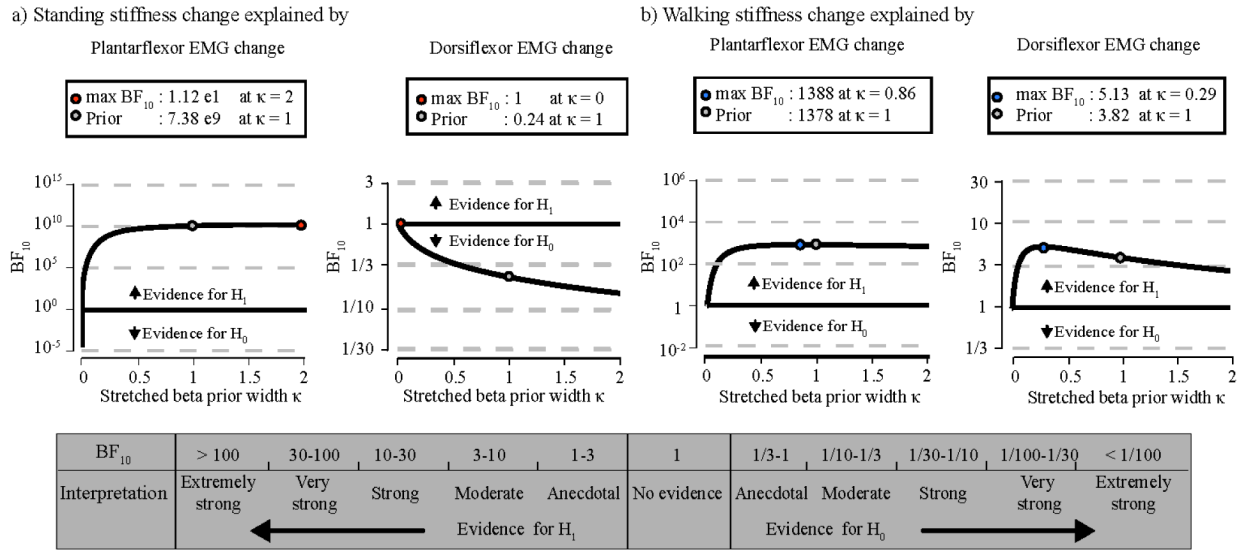
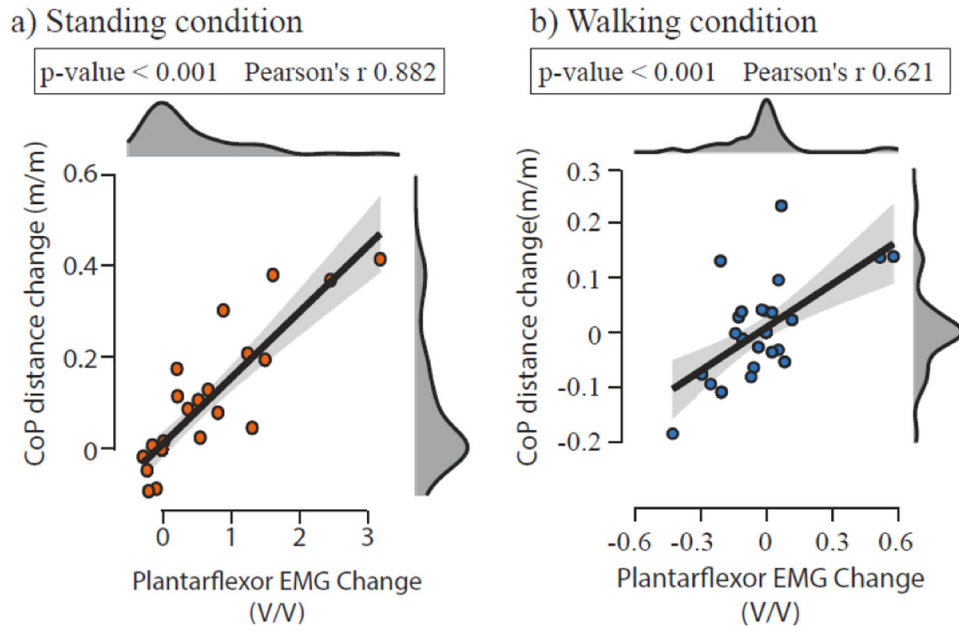


Fig. 3.

Scatter plots showing the relationship between changes in plantar and dorsiflexor muscle activation and changes in ankle stiffness for a) the different muscle activation conditions in the standing perturbation task and b) the different speed conditions in the walking perturbation task. The solid lines represent the linear regression lines and the gray bands surrounding the regression lines represent the 95% confidence intervals. The density plots showing the distribution of x- and y-values are shown on top and to the right of each plot, respectively. For each linear fit the p-value and correlation coefficients are shown on top. Note that all units in the figures are unitless due to the normalization process.

**Fig. 4.**

Plots showing the robustness of Bayes Factor (i.e., the relative likelihood of the alternative hypothesis to the null hypothesis) obtained from the Bayesian correlation analyses over a wide range of prior distributions (stretched beta prior width from 0 to 2) for a) the different plantarflexor activation conditions in the standing task and b) the different speed conditions in the walking task. The maximum Bayes factor and the Bayes factor for a uniformly distributed prior are shown on top of each correlation analyzed. The Bayes Factors were relatively consistent over a range of different prior specifications for the correlation between changes in plantarflexor activation and ankle stiffness during standing and for the correlation between changes in both plantar and dorsiflexor activation and ankle stiffness during walking.

**Fig. 5.**

Scatter plots showing the relationship between changes in plantarflexor muscle activation and changes in center of pressure location for a) standing perturbations and b) walking perturbations. CoP data for standing were normalized by computing the relative change in CoP from the quiet standing condition. CoP data for walking were normalized by computing the relative change in CoP from the 25% slower condition. The solid lines represent the linear regression lines and the gray bands surrounding the regression lines represent the 95% confidence intervals. The density plots showing the distribution of x- and y-values are shown on top and to the right of each plot, respectively. For each linear fit the p-value and correlation coefficients are shown on top. Note that all units in the figures are unitless, due to the normalization process.

TABLE I

Mean ($\pm$ SEM) values of the impedance parameters estimated during standing and walking conditions.

Condition	Stiffness	Viscosity	Inertia
Quiet Standing	1.48 ± 0.16	0.036 ± 0.003	0.0072 ± 0.002
$\uparrow 5\%$ Standing	1.77 ± 0.20	0.037 ± 0.004	0.0081 ± 0.002
$\uparrow 10\%$ Standing	3.08 ± 0.25	0.040 ± 0.004	0.0027 ± 0.002
Self-Selected Walking	3.36 ± 0.58	0.023 ± 0.003	0.0223 ± 0.005
$\downarrow 25\%$ Self-Selected	2.86 ± 0.49	0.024 ± 0.003	0.0186 ± 0.008
$\downarrow 50\%$ Self-Selected	2.46 ± 0.39	0.026 ± 0.002	0.0092 ± 0.002

Abbreviations: SEM = standard error of the mean. Units: Stiffness = $\text{N} \cdot \text{m} \cdot \text{Kg}^{-1} \cdot \text{rad}^{-1}$; Viscosity = $\text{N} \cdot \text{m} \cdot \text{s} \cdot \text{Kg}^{-1} \cdot \text{rad}^{-1}$; Inertia = $\text{N} \cdot \text{m} \cdot \text{s}^2 \cdot \text{rad}^{-1}$

TABLE II

Bayesian regression analysis of regression models for ankle stiffness change as a function of ankle muscle activation change.

Stiffness change in standing					
Models	P(M)	P(M data)	BF_M	BF₁₀	R²
Null model	0.333	3.3e-10	6.7e-10	1	0
PFChange + DFChange	0.333	0.186	0.458	5.6 e+8	0.844
PFChange	0.167	0.814	21.852	4.9 e+9	0.843
DFChange	0.167	6.0e-11	3.0e-10	0.361	0.005
Stiffness change during the late stance of walking					
Models	P(M)	P(M data)	BF_M	BF₁₀	R²
Null model	0.333	0.001	0.003	1	0
PFChange + DFChange	0.333	0.415	1.420	325.7	0.446
PFChange	0.167	0.582	6.954	914.1	0.431
DFChange	0.167	0.002	0.012	3.9	0.170

Abbreviations: P(M) = prior model probability; P(M|data) is the probability of the posterior distribution having taken into account the experimental data; BF_M shows how much the model has improved after seeing the data; BF₁₀ is the Bayes Factor in favor of alternative hypothesis (H₁) over null hypothesis (H₀) for each model against the null model; R² is the coefficient of determination.