Gait & Posture 68 (2019) 187–192

Contents lists available at ScienceDirect

Gait & Posture

journal homepage: www.elsevier.com/locate/gaitpost

Full length article

Voluntary control of forward leaning posture relates to low-frequency neural inputs to the medial gastrocnemius muscle

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ARTICLE INFO

Keywords:
Variability
Low-frequency oscillations
Center of pressure
Electromyogram

ABSTRACT

Background: Variability is an inherent feature of the motor output. Although low-frequency oscillations (< 0.5 Hz) are the most important contributor to the variability of force during single-joint isolated force tasks, it remains unclear whether they contribute to the variability of a more complex task, such as a voluntary postural task.

Research question: Do low-frequency oscillations contribute to postural sway (center of pressure (COP) variability) when participants attempt to voluntarily maintain posture in a forward leaning position?

Methods: Fourteen healthy young adults performed two tasks: 1) stand quietly (control condition); 2) leaned their body forward to 60% of their maximum lean distance by dorsiflexing the ankle joint. We recorded the COP and electromyographic (EMG) activity from the medial gastrocnemius (MG) and soleus (SL) muscles. We quantified the following: 1) COP variability as the standard deviation (SD) of anteroposterior COP displacements; 2) modulation of COP as the power in COP displacements from 0 to 2 Hz; 3) modulation of EMG bursting as the power in the rectified and smoothed EMG from 0 to 2 Hz; 4) modulation of the interference EMG as the power in the EMG from 10 to 35 and 35–60 Hz.

Results: The SD of COP displacements related to the COP oscillations < 0.5 Hz in both quiet standing and lean tasks. However, only for the lean task, the < 0.5 Hz COP oscillations related to the EMG burst oscillations < 0.5 Hz of the MG muscle. The EMG burst oscillations < 0.5 Hz of the MG muscle further related to the interference EMG oscillations from 35 to 60 Hz for the lean task.

Significance: Voluntary control of forward leaning posture relates to low-frequency neural inputs to the MG muscle.

1. Introduction

The motor output is intrinsically variable, as evidenced in the output of single-joint [1] and multi-joint [2] contractions. Numerous recent findings, based primarily on single-joint steady force contractions, suggest that low-frequency oscillations in force (< 0.5 Hz) contribute the most to force variability [1,3]. The underlying mechanism appears to be a low-frequency common input to the spinal motor neuron pool [4,5]. Although variability is evident during upright standing as postural sway [6–10], it is unclear whether it is related to low-frequency oscillations. Here, we examine whether low-frequency oscillations contribute to postural sway when participants attempt to voluntarily maintain posture in a forward leaning position.

The low-frequency oscillations in force during steady force contractions [1–3,11,12] are evident in single motor unit activity [4] and whole muscle activity as bursts in electromyogram (EMG) [1,3,13], likely as a consequence of the central drive oscillations to the motor neuron pool. The work by Farina and colleagues has demonstrated a strong association between the oscillations in force during steady contractions and the variability in estimated common input to motor neurons [4,5]. Specifically, they suggest that the common input to the motor neuron pool includes low-frequency oscillations [5], which are transmitted to force output [4]. At the whole muscle level, there is also evidence that oscillations in EMG bursting < 0.5 Hz contribute to low-frequency oscillations in force [1]. Furthermore, the oscillations of the interference EMG (raw EMG) from 10 to 60 Hz are associated with the

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https://doi.org/10.1016/j.gaitpost.2018.11.026
Received 3 May 2018; Received in revised form 18 October 2018; Accepted 18 November 2018

corticospinal projections [14,15], and the modulation of the interference EMG from 15 to 35 Hz relates to the low-frequency oscillations in force and EMG bursting [1]. Less 10–35 Hz power and more 35–60 Hz power in the interference EMG are associated with greater amplitude of low-frequency oscillations in the force output [1,12]. This indicates that low-frequency oscillations in force during isometric contractions likely represent the modulation of the motor neuron pool by the central nervous system (CNS).

The low-frequency oscillations are also evident in postural sway during upright standing. These oscillations are commonly quantified using the center of pressure (COP) displacements. For example, COP oscillations < 1 Hz increased after 20-day bed rest, resulting in an increase in the COP velocity [7]. Further, there is evidence that an increase in COP oscillations from 1 to 10 Hz relates to increased COP velocity [6]. These studies suggest that low-frequency oscillations can be detrimental to postural control; however, the findings are limited to quiet standing, which is controlled primarily (about 90%) by a passive ankle torque [16]. It remains unknown whether low-frequency oscillations contribute to postural sway during a postural task requiring continuous voluntary effort. In this study, therefore, we aim to determine whether COP is modulated at a low frequency (< 0.5 Hz) during a voluntary postural task and whether this COP modulation is evident in the modulation of the active muscles.

2. Methods

2.1. Participants

Fourteen healthy young adults (mean age ± SD = 22.4 ± 1.1 years, mean height ± SD = 165.9 ± 7.5 cm, 5 females) participated in this study. Participants had no history of orthopedic, neurological, or cognitive disorders that could affect the postural performance. They all had normal or corrected-to-normal vision and were right-foot dominant. The experimental procedure of this study was approved by the local ethics committee, and all participants provided a written informed consent prior to the data collection.

2.2. Experimental procedures

Participants performed two postural tasks (quiet standing and forward leaning) in a counterbalanced order. For the lean task, we determined the maximum forward postural lean distance for each participant using the following procedures: 1) The participants stood quietly on a force plate for 30 s with their feet parallel to each other and a heel-to-heel distance of 15 cm. To maintain the same feet positions throughout the experiment, we marked the feet positions on the force plate. We then quantified the mean anteroposterior COP position from the force signals during quiet standing. 2) We asked the participants to lean their body forward as much as they could (three trials). The lean movements were based on pure ankle dorsiflexion, which required them to maintain the rest of the body straight. 3) We quantified the maximum lean distance as the maximum anteroposterior COP position during the lean minus the mean anteroposterior COP position during quiet standing. We used the greatest lean distance out of three trials as the maximum forward postural lean distance.

2.2.1. Lean task

We asked the participants to lean forward using only ankle dorsiflexion and match a horizontal target line (green) displayed on a monitor (LCD-MF235XDBR, I–O Data, Japan) set at 1 m in front of the participant with the approximate visual angle of ~1° (Fig. 1). We used a customized LabVIEW program (National Instruments, Austin, TX, USA) to display target lines and COP line on the monitor. The target line was 60% of the maximum forward postural lean distance. The lean distance was determined so to obtain a significant amount of activation in the plantar flexor muscles and not to make the task extremely difficulty to perform. We also placed two yellow horizontal lines at ± 5% of the target line and displayed the COP as a red line. The COP line progressed from left to right and moved upward or downward as the participant leaned forward or backward. We instructed the participants to voluntarily control their postural sway and keep the COP on the target line as accurately and consistently as they could. The task duration was 40 s, and each participant performed two practice trials and three test trials with a break of at least 3 min between the trials to avoid fatigue.

2.2.2. Quiet standing task

We asked the participants to stand quietly on a force plate for 40 s while looking at a fixation sign on the monitor (three trials), which served as our control condition. Our dependent variables in each task represent the average from the three trials.

2.3. Apparatus

We collected EMG signals from the right medial gastrocnemius (MG) and soleus (SL) muscles using wireless EMG sensors (Trigno, Delsys, Boston, MA, USA), as these muscles are the primary contributors to the control of posture [8,17]. The EMG sensors were placed according to the SENIAM recommendations (http://www.seniam.org/). We amplified (×1000), filtered (20–450 Hz), and sampled (2000 Hz) the recorded EMG signal from each muscle, and collected force signals at a sampling rate of 1000 Hz using a force plate (Tech Gihan, Kyoto, Japan).

2.4. Data analysis

We analyzed the data for the middle 30 s of the collection period using a customized Matlab script (MathWorks, Natick, MA, USA). We quantified the following parameters.

2.4.1. COP variability and oscillations

We quantified COP variability as the SD of anteroposterior COP displacements (low-pass filtered at 15 Hz with a fourth-order zero phase lag Butterworth filter). We further quantified COP oscillations using a Fourier analysis and the resulting power spectrum density (PSD) [18]. For the Fourier analysis, we detrended and low-pass filtered the anteroposterior COP displacements at 2 Hz (a fourth-order zero phase lag Butterworth filter) (Fig. 2a) [1,3]. The window size was set at 30 s, to maximize the PSD resolution (0.033 Hz) for our data. For statistical comparisons, we divided the PSD of COP into four frequency bins (0-0.5, 0.5–1.0, 1.0–1.5, 1.5–2.0 Hz) [1,3]. The dependent variable was the absolute power in each frequency bin.

2.4.2. EMG burst oscillations

We quantified EMG burst oscillations using a Fourier analysis and the resulting PSD [18]. We detrended, rectified, and low-pass filtered at 2 Hz (a fourth-order zero phase lag Butterworth filter) the EMG signals from the MG and SL muscles (Fig. 2a) [3]. The window size was set at 30 s, to maximize the PSD resolution (0.033 Hz) for our data. For statistical comparison, we divided the PSD of the EMG bursting into four frequency bins (0-0.5, 0.5–1.0, 1.0–1.5, 1.5–2.0 Hz) [1,3]. The dependent variable was the absolute power in each frequency bin.

2.4.3. Oscillations of interference EMG signals

We quantified interference EMG oscillations using a Fourier analysis and the resulting PSD [18]. The window size was set at 30 s, to maximize the PSD resolution (0.033 Hz) for our data. For statistical comparison, we divided the PSD of the interference EMG into two frequency bins (10–35 and 35–60 Hz) [1,3]. This was performed because,

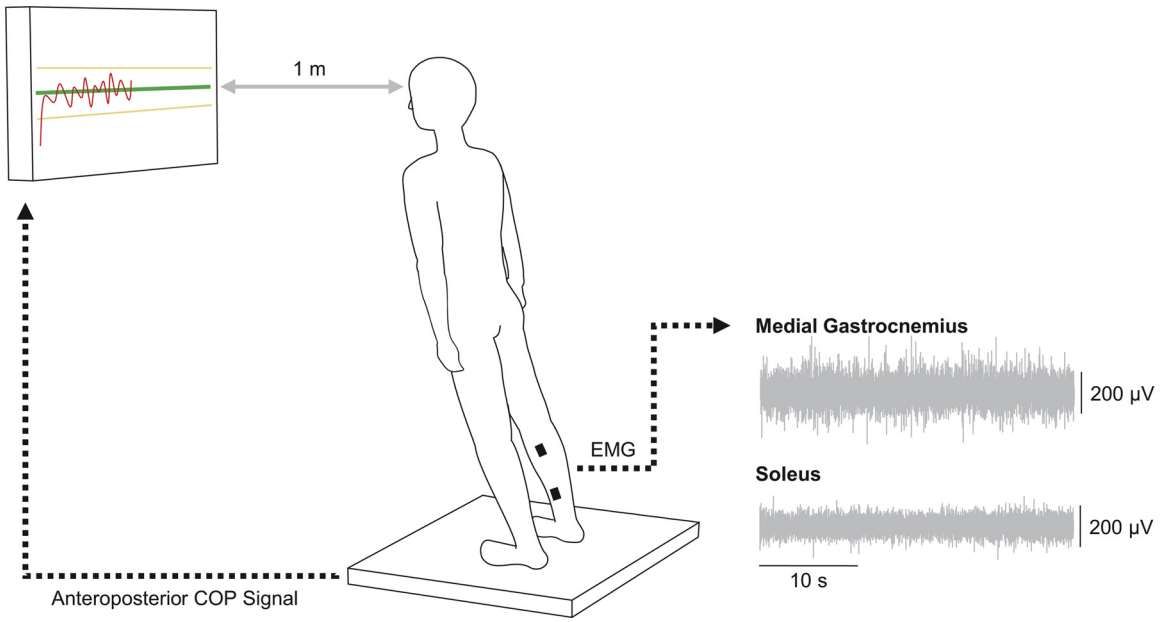

Fig. 1. Schematic of the experimental setup for the lean task. The participant stood on a force plate and was instructed to lean the body forward and voluntarily control the postural sway to keep the COP (red line) on the target line (green line) as accurately and consistently as possible. We recorded EMG from the right medial gastrocnemius and soleus muscles using wireless sensors. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article).

although both frequency bins are related to corticospinal projections [14,15], the 10–35 Hz is associated with the steady state of motor output [15], and the 35–60 Hz is associated with movement execution or dynamic force output [19]. The dependent variable was the absolute power in each bin.

2.5. Statistical analysis

We used stepwise multiple linear regression analysis to establish statistical models that predict the following: a) the COP variability from COP oscillations, b) the COP oscillations from EMG burst oscillations, and c) the EMG burst oscillations from interference EMG oscillations (10–35 and 35–60 Hz). We used the Durbin-Watson (DW) statistic to detect the presence of autocorrelation. We performed the statistical analyses using SPSS (IBM, Armonk, NY, USA) at alpha level of 0.05.

3. Results

3.1. COP oscillations

We found that most of the power in COP variability during the voluntary leaning task was from 0-0.5 Hz (58% of the total power from 0 to 2 Hz). Powers in other frequency bins were relatively small: 0.5–1.0 Hz (28%), 1.0–1.5 Hz (12%), and 1.5–2.0 Hz (2%) (Fig. 2b). We used a stepwise regression analysis to predict the COP variability from the COP oscillations for the lean task and revealed that the power from 0-0.5 Hz (part $r = 0.61$) and 0.5–1.0 Hz (part $r = 0.49$) of COP oscillations were independent predictors of the COP variability ($R^2 = 0.61$, DW = 1.71, $p = 0.024$). We also performed the stepwise regression analysis for the quiet standing task (control condition) and found that the 0-0.5 Hz COP oscillations was the single significant predictor of the COP variability ($R^2 = 0.53$, DW = 1.46, $p = 0.003$). These findings suggest that the greatest contributor to COP variability is COP oscillations from 0-0.5 Hz for both tasks.

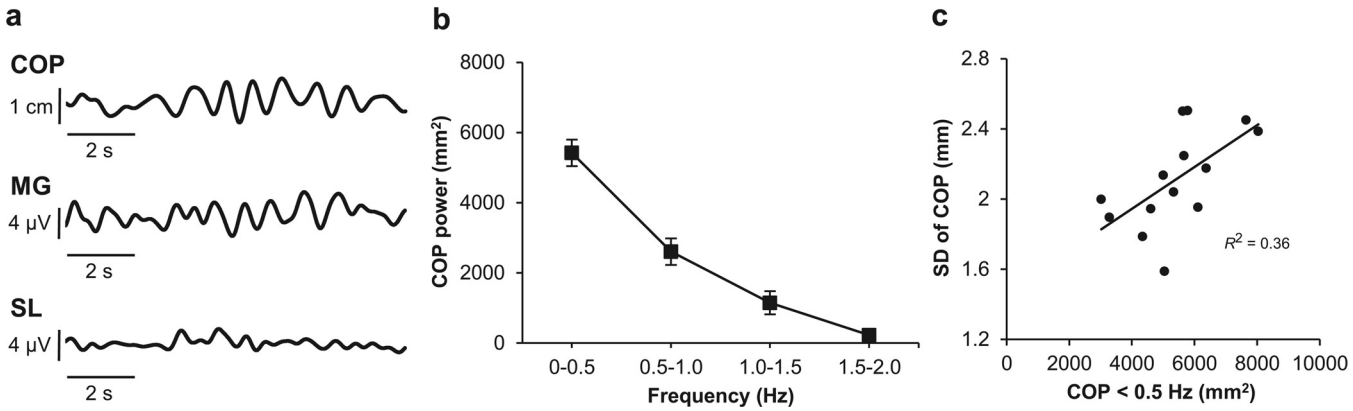

Fig. 2. Low-frequency COP and EMG oscillations during forward postural lean. Representative low-pass filtered (2 Hz) anteroposterior COP displacements and EMG activities of medial gastrocnemius (MG) and soleus (SL) muscles are shown from top to bottom (a). Absolute power in the anteroposterior COP oscillations (b) and association between standard deviation (SD) of COP displacements and COP oscillations < 0.5 Hz (c). Error bars show standard error of the mean.

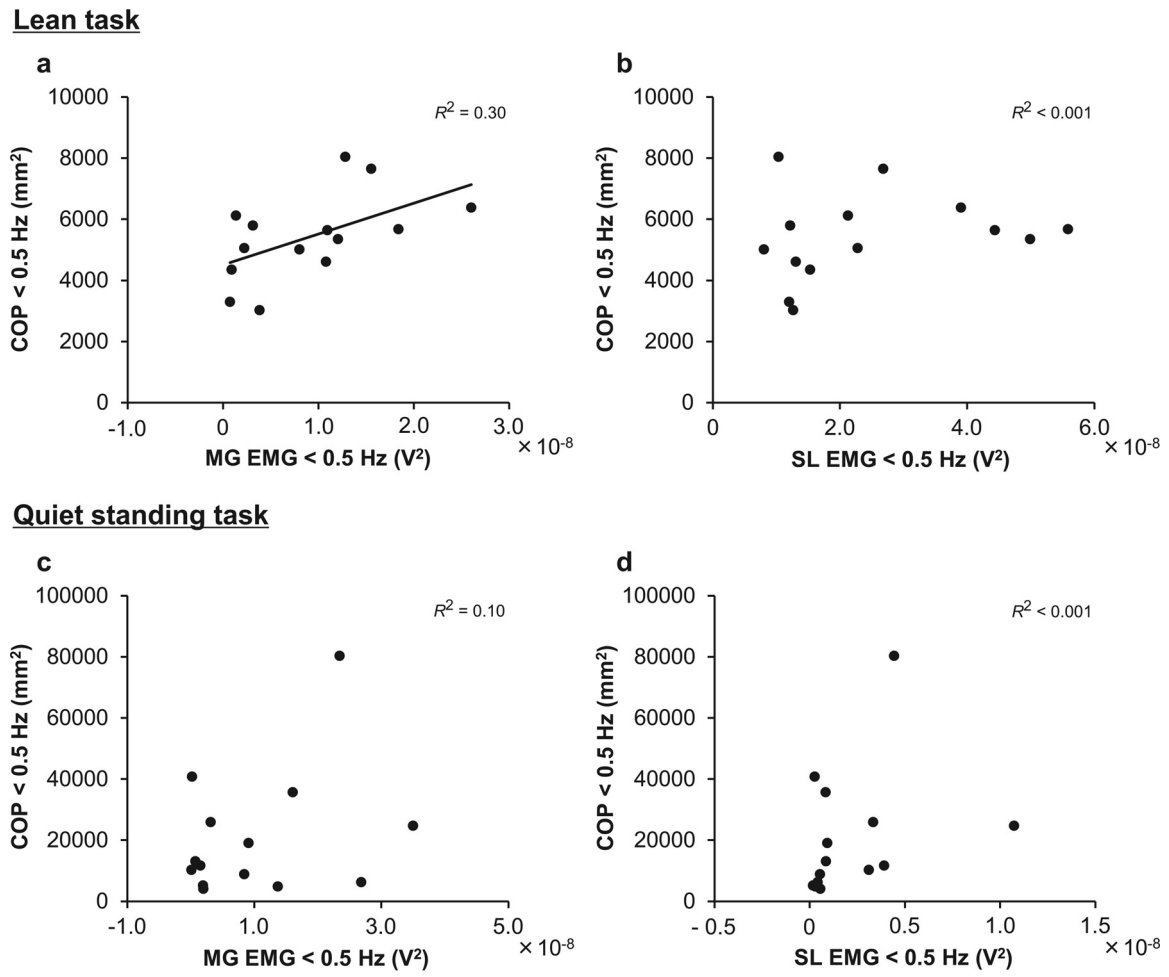

Fig. 3. Association between COP oscillations < 0.5 Hz and EMG burst oscillations < 0.5 Hz of the medial gastrocnemius (MG) and soleus (SL) muscles for the lean (a and b) and quiet standing tasks (c and d).

3.2. Oscillations in muscle activity

The 0-0.5 Hz COP oscillations were the strongest predictor of the COP variability. Therefore, we used a stepwise regression analysis to predict the power in the 0-0.5 Hz COP oscillations from the EMG burst oscillations. For the lean task, the 0-0.5 Hz EMG burst oscillations of the MG muscle was the single significant predictor of the 0-0.5 Hz COP oscillations (R^2 = 0.30, DW = 2.41, p = 0.044), and the modulation of the SL muscle was not statistically associated with the 0-0.5 Hz COP oscillations (Fig. 3a and b). For the quiet standing task, the modulation of the MG or SL muscle was not statistically related to the 0-0.5 Hz COP oscillations (Fig. 3c and d).

For the lean task, the 0-0.5 Hz EMG burst oscillations of the MG muscle were positively correlated with modulation of the interference EMG from 35 to 60 Hz (R^2 = 0.33, DW = 2.13, p = 0.033; Fig. 4). These findings suggest that the MG modulation is primarily responsible for the COP oscillations from 0-0.5 Hz during the voluntary forward lean task.

4. Discussion

In this study, we aimed to determine whether the COP and muscle activity are modulated at a low frequency (< 0.5 Hz) during a voluntary postural task. We found that low-frequency oscillations (< 0.5 Hz) contribute the most to COP variability during the forward postural lean and quiet standing tasks. This low-frequency modulation of the COP was evident in the modulation of the MG muscle burst activity (< 0.5 Hz), which was associated with greater power in interference EMG from 35 to 60 Hz, for the lean task but not for the quiet standing task. These findings indicate that, similar to single-joint steady force contractions [1,3], sway variability during a voluntary postural task is related to a low-frequency neural drive to the motor output.

Low-frequency oscillations are evident during steady isometric contractions [1–3,11]. For example, when participants attempt to maintain a steady force, the variability of force is strongly related to low-frequency oscillations in force (< 0.5 Hz) [1,3]. Further, greater low-frequency oscillations in the force output strongly relate to greater modulation of single motor unit activity [4], EMG bursting from 0-0.5 Hz [1,3], and interference EMG from 35 to 60 Hz [1,3]. Thus, the current belief is that the low-frequency oscillations in force likely represent the modulation of the motor neuron pool by the CNS [5,12]. This study's findings expand the current literature, which is based primarily on isolated single-joint contractions, to postural tasks. Specifically, when the postural task requires a voluntary input to maintain a steady posture, the COP modulation at frequencies < 0.5 Hz, which is directly related to the COP variability, relates to the modulation of EMG bursting < 0.5 Hz and modulation of the interference EMG from 35 to 60 Hz, similar to previous studies on single-joint contractions [1,3]. Therefore, the common findings in single-joint contractions and our postural task provide support to the hypothesis that low-frequency oscillations are of central origin at least partly, which modulate the motor neuron pool during voluntary contractions.

In addition to the direct inputs from the motor cortex, movements associated with respiration could have influenced the low-frequency

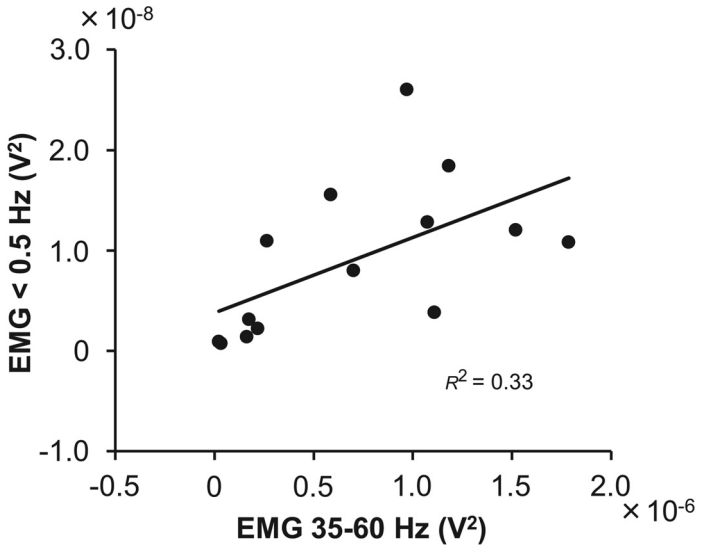

Fig. 4. Association between EMG burst oscillation < 0.5 Hz and interference EMG oscillations from 35 to 60 Hz of the medial gastrocnemius muscle for the lean task.

COP oscillations. It has been reported that respiration could increase the low-frequency oscillations in force output [20] as well as COP displacements [21]. Furthermore, one of peaks in the power of COP oscillations was found to be at the frequency of respiration (< 0.5 Hz) [22]. Therefore, it is likely that the < 0.5 Hz COP oscillations found in both quiet standing and lean tasks include the respiration-induced body movements to some extent. However, because the < 0.5 Hz COP oscillations statistically correlated with the muscle activity only during the lean task, we hypothesize that the oscillations in the voluntary drive primarily contribute to the < 0.5 Hz COP oscillations during the voluntary control of posture. Contrarily, the respiration could be a primary contributor to < 0.5 Hz COP oscillations during the quiet standing.

An interesting question is: "why does the modulation of EMG bursting < 0.5 Hz relate to the modulation of the interference EMG from 35 to 60 Hz?" The modulation of the interference EMG from 10 to 60 Hz likely relates to the modulation of cortical activity [14,15,19]. Inputs to the motor neuron pool from 10 to 35 Hz relate to a steady force output [15], whereas inputs to the motor neuron pool from 35 to 60 Hz relate to movement execution [19]. For isolated single-joint steady contractions, more power from 35 to 60 Hz results in greater amplitude of low-frequency oscillations in EMG bursting [1,3]. Here, we hypothesize that when a postural task requires more corrections because of continuous postural unsteadiness, the most efficient way to produce the corrective movements is a neural input from 35 to 60 Hz to the motor neuron pool. Although this neural input (35–60 Hz) is likely useful for producing fast corrective movements, it could increase the amplitude of low-frequency oscillations in the COP [12]. Thus, a neural input from 35 to 60 Hz to the MG was likely advantageous for maintaining the targeted posture with corrective movements of the ankle; however, the modulation at this particular frequency range likely resulted in greater amplitude of the low-frequency EMG bursting of the MG, which resulted in greater variability of the COP displacements.

Another interesting observation from this study was that the < 0.5 Hz COP oscillations correlated with the low-frequency EMG burst modulation of the MG muscle but not the SL muscle. Anatomically, the gastrocnemius muscle has about 50% of fast-twitch fibers, while the SL muscle consists about 90% of slow-twitch fibers [23]. Thus, the gastrocnemius muscle seems more appropriate for generating stronger and faster forces, whereas the SL muscle is more proper for continuous activation. It is possible, therefore, that during the lean task the SL muscle was continuously active, contributing relatively more to holding the body, whereas the MG activated intermittently to correct the anteroposterior postural sway.

The study is limited by the following: First, there is a potential risk of cross-talk between EMG recordings. As no procedures to eliminate the cross-talk are currently available [24], the results may need to be interpreted with caution. Second, the task was limited to 60% of maximum postural lean distance; therefore, results could be different with different leaning distances. Third, our subjects were healthy young adults, and thus we can't extrapolate to elderly adults and individuals with neurological disorders who could present different frequency characteristics [25]. Finally, it is possible that other postural muscles (e.g., hamstrings and lower back muscles) and antagonist muscles (e.g., tibialis anterior muscle) that we did not measure could have influenced the postural sway. Future studies should address these limitations by expanding our study to different leaning distances, other populations, and recording from other postural muscles.

In summary, we provide novel findings that low-frequency oscillations (< 0.5 Hz) contribute the most to COP variability during a forward postural lean task. This low-frequency modulation of the COP was evident in the modulation of the MG muscle. These findings indicate that, similar to single-joint steady force contractions [1,3], sway variability during a voluntary postural task is related to a low-frequency neural drive to the motor output. Therefore, decreasing low-frequency oscillations could be beneficial for improving voluntary control of posture.

Funding

This work is supported by a grant from the Japan Society for the Promotion of Science (a Grant-in-Aid for JSPS fellows [29-700]).

Declarations of interest

None.

References

[1] H. Moon, C. Kim, M. Kwon, Y.T. Chen, T. Onushko, N. Lodha, et al., Force control is related to low-frequency oscillations in force and surface EMG, PLoS One 9 (2014) e109202.
[2] N. Lodha, G. Misra, S.A. Coombes, E.A. Christou, J.H. Cauraugh, Increased force variability in chronic stroke: contributions of force modulation below 1 Hz, PLoS One 8 (2013) e83468.
[3] S.H. Park, A. Casamento-Moran, B. Yacoubi, E.A. Christou, Voluntary reduction of force variability via modulation of low-frequency oscillations, Exp. Brain Res. 235 (2017) 2717–2727.
[4] F. Negro, A. Holobar, D. Farina, Fluctuations in isometric muscle force can be

described by one linear projection of low-frequency components of motor unit discharge rates, J. Physiol. (Paris) 587 (2009) 5925–5938.
[5] D. Farina, F. Negro, Common synaptic input to motor neurons, motor unit synchronization, and force control, Exerc. Sport Sci. Rev. 43 (2015) 23–33.
[6] M. Kouzaki, K. Masani, Postural sway during quiet standing is related to physiological tremor and muscle volume in young and elderly adults, Gait Posture 35 (2012) 11–17.
[7] M. Kouzaki, K. Masani, H. Akima, H. Shirasawa, H. Fukuoka, H. Kanehisa, et al., Effects of 20-day bed rest with and without strength training on postural sway during quiet standing, Acta Physiol. Oxf. (Oxf) 189 (2007) 279–292.
[8] T. Watanabe, K. Saito, K. Ishida, S. Tanabe, I. Nojima, Coordination of plantar flexor muscles during bipedal and unipedal stances in young and elderly adults, Exp. Brain Res. 236 (5) (2018) 1229–1239.
[9] T. Watanabe, K. Saito, K. Ishida, S. Tanabe, I. Nojima, Age-related declines in the ability to modulate common input to bilateral and unilateral plantar flexors during forward postural lean, Front. Hum. Neurosci. 12 (2018).
[10] T. Watanabe, K. Saito, K. Ishida, S. Tanabe, I. Nojima, Fatigue-induced decline in low-frequency common input to bilateral and unilateral plantar flexors during quiet standing, Neurosci. Lett. 686 (2018) 193–197.
[11] E.J. Fox, H.S. Baweja, C. Kim, D.M. Kennedy, D.E. Vaillancourt, E.A. Christou, Modulation of force below 1 hz: age-associated differences and the effect of magnified visual feedback, PLoS One 8 (2013).
[12] N. Lodha, E.A. Christou, Low-frequency oscillations and control of the motor output, Front. Physiol. 8 (2017) 78.
[13] Y. Yoshitake, M. Shinohara, Oscillations in motor unit discharge are reflected in the low-frequency component of rectified surface EMG and the rate of change in force, Exp. Brain Res. 231 (2013) 267–276.
[14] T. Mima, M. Hallett, Corticomuscular coherence: a review, J. Clin. Neurophysiol. 16 (1999) 501–511.
[15] A.K. Engel, P. Fries, Beta-band oscillations–signalling the status quo? Curr. Opin. Neurobiol. 20 (2010) 156–165.
[16] I.D. Loram, M. Lakie, Direct measurement of human ankle stiffness during quiet standing: the intrinsic mechanical stiffness is insufficient for stability, J. Physiol. (Paris) 545 (2002) 1041–1053.
[17] K. Masani, M.R. Popovic, K. Nakazawa, M. Kouzaki, D. Nozaki, Importance of body sway velocity information in controlling ankle extensor activities during quiet stance, J. Neurophysiol. 90 (2003) 3774–3782.
[18] E.A. Christou, Visual feedback attenuates force fluctuations induced by a stressor, Med. Sci. Sports Exerc. 37 (2005) 2126–2133.
[19] W. Omlor, L. Patino, M.C. Hepp-Reymond, R. Kristeva, Gamma-range corticomuscular coherence during dynamic force output, Neuroimage 34 (2007) 1191–1198.
[20] H.S. Baweja, B.K. Patel, O.P. Neto, E.A. Christou, The interaction of respiration and visual feedback on the control of force and neural activation of the agonist muscle, Hum. Mov. Sci. 30 (2011) 1022–1038.
[21] O. Caron, P. Fontanari, J. Cremieux, F. Joulia, Effects of ventilation on body sway during human standing, Neurosci. Lett. 366 (2004) 6–9.
[22] P.W. Hodges, V.S. Gurfinkel, S. Brumagne, T.C. Smith, P.C. Cordo, Coexistence of stability and mobility in postural control: evidence from postural compensation for respiration, Exp. Brain Res. 144 (2002) 293–302.
[23] M.A. Johnson, J. Polgar, D. Weightman, D. Appleton, Data on the distribution of fibre types in thirty-six human muscles. An autopsy study, J. Neurol. Sci. 18 (1973) 111–129.
[24] D. Farina, R. Merletti, R.M. Enoka, The extraction of neural strategies from the surface EMG: an update, J. Appl. Physiol. 2014 (117) (1985) 1215–1230.
[25] M. Hallett, Overview of human tremor physiology, Mov. Disord. 13 (Suppl 3) (1998) 43–48.