Research Topics

Current state of postural research - Moving beyond the balance platform

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Current state of postural research - Moving beyond the balance platform

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Table of contents

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Editorial: Current state of postural research - Moving beyond the balance platform

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Editorial on the Research Topic
Current state of postural research - Moving beyond the balance platform

Assessment of postural control with a dynamic Balance Platform was a disruptive technology at its time (1). Findings emerging from research with the dynamic platform significantly altered not only the approach to studying control of posture, but also shaped theories of postural control over subsequent decades. In this Research Topic, Keshner et al. argue that the most dominant paradigm shift since adoption of the posture platform was that reactive postural control is not simply a reflexive process but one that is highly dependent on the demands of the specific task as well as the state of the performer. Hall et al. review evidence for a strong linkage between emotional state and the somatic nervous system in the control of posture. Our prior Research Topic (2) focused on postural research that challenged the view of postural control as a reflexive mechanism. The aim of this Research Topic was to solicit articles that focused on the concept of posture control as a complex and adaptable motor act and were not necessarily reliant upon the dynamic balance platform as an experimental protocol.

The contribution of cortical processing to posture control is an emergent theme in several of the papers in this Research Topic. Although underlying neural mechanisms that regulate postural stability are not yet well delineated, Fischer et al. hypothesized that shifts in attention contribute to inconsistencies in motor behavior by demonstrating increases in sample entropy as a measure of postural sway regularity under threatening circumstances. Through simultaneous EEG and EMG analyses, Stokkermans et al. identified distinct gain changes in the theta, alpha, beta, and low/high-gamma frequencies during the reactive balance responses for all leg muscles. Kannan et al. employed fMRI technology to identify significant associations between reactive balance control and cortico-subcortical regions even in individuals with mild cognitive impairment.

The finding of downstream control of postural behaviors does not in any way negate the importance of the sensory signals guiding postural responses. Sutter et al. demonstrated that the signals emerging from skin deformations generated by forces and pressures exerted between the foot skin and the standing surface increased sensory flow to the somatosensory cortex and improved balance control. Additionally, sensory flow to somatosensory cortex increased as did gamma activity over centroparietal regions during the preparation phase of the body weight transfers. Cleworth et al. looked specifically at perceptual thresholds at the ankle when facing a height-induced threat to balance and found raised perceptual thresholds and perceptions of increased motion of the foot suggesting cortical modulation of sensory feedback during threat induced instability.

Although training of automatic postural reactions has not proven robust in the treatment of instability and falls (3), findings that support the participation of cortical processes in planning and preparation for postural responses warrant attention in promoting experience-dependent neuroplasticity (4). Fadillioglu et al. provide evidence of the potential for training to improve postural reactions. Lockhart et al. found that training on a split-belt treadmill during gait and static postural stability tasks improved balance and stability of individuals with Parkinson's Disease. They conclude that these participants used their experiences with perturbation training to integrate and adapt feedforward and feedback behaviors to reduce falls, thereby illustrating that postural reactions practiced within a functional context can be learned.

Wang et al. explored a substantial component of cortically controlled behavior known as perceptual motor style. They quantified the variability that emerges both within and across individual performers and identified both static and dynamic markers that could specify the perceptual motor style. Significant heterogeneity was found across individuals performing locomotion in a height threatening virtual environment. Other markers of asymmetry in gait were explored in the lateral plane of motion, Dusane et al. showed that the ability to control lateral COM motion during walking could be a contributing factor to imbalance in people with incomplete spinal cord injury. Peterka et al. identified four major gait asymmetries (involving step width, ankle torque, stance duration and swing duration) as determinants of a separate mechanism in the control of mediolateral stability in gait. The variability of these asymmetry measures is shown to better distinguish between young and old performers than conventional measures associated with poor balance and fall risk.

Results from the research presented in this issue provide further evidence to support a significant contribution of both cortical and subcortical processing, attention control networks, and sensory-motor perceptual systems to the control of posture and balance under both perturbed and unperturbed (static and dynamic) conditions. The work done initially by Nashner and colleagues has, in fact, laid the foundation to a better understanding of control of posture and gait, using newly emerging technologies (e.g., mobile imaging and wearable sensors) and commercially available equipment that mimics the dynamic platform paradigm (i.e., motorized treadmills). The adoption of these approaches to postural control research in no way negates the continued value of the dynamic platform as a crucial paradigm that could be used both in stance and gait to examine cortical control (Keshner et al.). Instead, the evidence expands the importance of the study of reactive balance control by considering the contribution of cortical networks to posture and balance.

Author contributions

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References

1. Nashner LM. Fixed patterns of rapid postural responses among leg muscles during stance. *Exp Brain Res.* (1977) 30:13–24. doi: 10.1007/BF00237855

2. Keshner EA, Fung J. Editorial: Current state of postural research – beyond automatic behavior. *Front Neurol.* (2019) 10:1160. doi: 10.3389/fneur.2019.01160

3. Shapiro A, Melzer I. Balance perturbation system to improve balance compensatory responses during walking in old persons. *J Neuroeng Rehabil.* (2010) 7:32. doi: 10.1186/1743-0003-7-32

4. Kleim JA, Jones TA. Principles of experience-dependent neural plasticity: implications for rehabilitation after brain damage. *JSLHR.* (2008) 51:S225–39. doi: 10.1044/1092-4388(2008/018)

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Evolution of postural control assessment: From dynamic posturography to virtual reality

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During the early years of spaceflight it was documented that astronauts were impaired and incapacitated upon return to earth. Computerized Dynamic Posturography (CDP) was devised to investigate and quantify this deficit, and eventually progressed into a clinical assessment tool. The current sprouting of virtual reality (VR) technologies has allowed for the development of an alternative approach that could be more informative. Many low-cost VR systems (including desktop gaming programs designed for rehabilitation) are now available. Continued improvements in this technology indicate a high probability that VR will become an integral component of posturography by replacing present mechanical CDP techniques. We researched the relevant literature to evaluate the strengths and weaknesses of CDP using the Equitest (Neurocom International; Clackamas USA), and the added benefits of incorporating VR to help clinicians assess the complex task of balance maintenance. VR is capable of manipulating task and environmental demands in order to assess functional postural behavior. VR is also a useful tool for clinical testing of postural disorders resulting from sensory mismatch. Although posturography is still a useful clinical tool, VR provides an inherent conflict between the visual and vestibular senses and can elevate the effectiveness of CDP for both assessment and intervention. We conclude that, when initially developed, CDP was innovative and ahead of its time. However, with the advent of VR, we have a chance to modernize CDP and enhance its value as a clinical instrument.

KEYWORDS

balance, dizziness, vestibular rehabilitation, otoliths, diagnosis, intervention

Introduction

The European Society for the Clinical Evaluation of Balance Disorders (ESCEBD), based in Nancy, France, has been meeting yearly since 2005 to discuss themes related to balance and equilibrium that are not yet clearly defined or standardized. One of our latest discussions was with regard to the continued development of Computerized

Dynamic Posturography® (CDP) as a research and clinical tool in the clinical setting. Unfortunately, this method of testing is non-portable and expensive. We acknowledged that virtual reality (VR) has begun to emerge as a replacement for CDP from both the diagnostic and therapeutic points of view. The following is a brief synopsis of our discussion relating to the use of CDP in the clinical setting and how it has helped VR evolve as a clinical tool.

Control of posture and balance

Posture can be defined as the control of neural circuitry over mechanics of the skeletal-motor system to maintain orientation in space, in response to momentary demands of a task in a dynamic environment. The progression of knowledge in this field of research has closely followed our understanding of central nervous system (CNS) processing and the dynamic interplay between the organism, the task, and the environment. The origin of research on the control of posture stems from the descriptions and categorizing of the reflexes that emerged when animals would orient themselves in space. Early research into the reflex control of posture from more than a century ago was based on studies of decerebrate and decorticate animals and focused primarily on the reflexes that positioned the body segments in space with respect to gravity (1).

A major theoretical shift occurred in the 1970's with the development of CDP, which enabled postural behaviors to be quantified during dynamic disturbances at the base of support. Attempts to model the mechanisms of posture control were done by simplifying the biomechanics of the behavior. Posture was primarily modeled as an inverted pendulum with the principal motion occurring around the ankle joint (2). Although the complexity of the human multisegmental and multimodal system makes it understandable that accurate modeling requires a reduction of variables, we need to acknowledge that a simplification strategy such as this may not be as robust or generalizable to natural motion as we might hope. Indeed, more complex models allowing for a greater number of variables have been developed that suggests that the system is more adaptive and variable than early controlled studies might suggest (3).

The balance system involves complex, adaptive interactions among multiple sensory and motor components. When pathology strikes one of these components, the balance system attempts to adapt by changing how other components contribute to the motor outcome. This adaptive process (i.e., "compensation") can be difficult to untangle when several components have gradually modified through aging. Compensation is a multifactorial process that does not always reach a functional level. By the time a patient sees a medical professional for a balance problem, attempting to define or locate the site of pathology can be challenging. Even when the site of injury is identified, symptoms and impairments may vary widely across patients (4).

Balance and mobility deficits arise not only from the motor or sensory impairment but also from the inability to select and properly weight pertinent sensory information (5). If we accept that the human performer does not act as an inverted pendulum but rather as a multisegmental processor of the full array of incoming signals (6), then the additive approach to sensory signals that underlie a standard CDP system cannot produce functional assessments of balance. If we acknowledge that vestibular signs and symptoms can emerge from poorly weighted or conflicting multimodal signals (7, 8), then we must appreciate that VR presents more promise for a functional approach to treatment than a standard CDP system.

Evolution of Computerized Dynamic Posturography

During the early years of spaceflight, NASA researchers reported that astronauts were unstable and nauseated upon their return to earth. It had originally been predicted that "… the symptoms [of motion sickness] are the same whether they result from the movement of ships, aircraft or cars" (9) and would also occur in space (10). Otoliths were thought to play a major role in space motion sickness (11) and the balance and autonomic/visceral control centers, traditionally viewed as separated, began to be viewed as one functional entity (12). Disrupted processing of otolith inputs upon return from orbital flight was thought to be the source of postural instability of astronauts. Impairments in returning astronauts were discussed and summarized as being of vestibular origin by Black et al. (13) and it has been proposed [e.g., (14)] that astronauts were impaired in the same manner as "vestibular patients."

In order to investigate and quantify the vestibular deficit in returning astronauts, CDP was devised (15). This technology was commercialized as Equitest® (Neurocom International; Clackamas USA) in the mid-1980's and eventually, CDP was introduced as a clinical tool (16). CDP was the first diagnostic tool for the balance system that had been developed subsequent to the generally accepted vestibular tests (e.g., calorics which were described by Barany about 70 years previously). At the time CDP was developed, it was advanced technology in the clinical setting. Although expensive, systems were acquired by research laboratories in the field of balance and dizziness for both diagnostic and therapeutic purposes. Comparison of CDP investigations from one location to another made direct research between institutions possible; however, the prohibitive cost of CDP also led to a reliance on "home-made" force plate systems (e.g., "foam and dome") (17). The drawback of these systems was that they were not reliable (foam degrades over time) and not standardized for their specific properties (e.g., compliance) across clinical institutions.

CDP allows us to assess postural performance when challenged with the task of maintaining balance under situations which are orientationally disruptive. This allows for measurement of how *well* the patient is compensating, but also *how* they are doing so. The whole process of compensation can itself be a challenge, as performers have been shown to select information based on individual sensory preferences (6). CDP is very helpful at documenting subtle pathology, especially in the atypical patient (18).

Benefits of CDP to vestibular rehabilitation

Although a sensitive and standardized assessment technique, CDP is neither site-specific nor side specific. Unfortunately, the cost for CDP remains high and is prohibitive in terms of general availability. As time has passed and newer tests have developed, the role of CDP in the diagnostic toolbox has become less secure.

Despite these limitations, CDP should be recognized for its role in assessing patients with vestibular dysfunction. Only 10 to 20% of patients with dizziness suggestive of vestibular dysfunction have abnormalities on caloric testing as the low frequency caloric stimulus does not sufficiently challenge the vestibular system (19). Patients may exhibit symptoms of unexplained motion sickness that is not related to semicircular canal pathology and caloric testing is only capable of assessing the lateral semicircular canals. With CDP, abnormalities have been measured in about 50% of these patients (20). Thus, CDP results are a more robust indicator of a vestibular deficit.

Complaints of dizziness which are sometimes regarded as "nontraditional" in nature are strongly suggestive of otolithic pathology, and there is evidence that CDP effectively identifies impairment of otolithic structures (21, 22). Astronauts with similar impairments post flight also exhibit abnormal posturography and autonomic symptoms. These abnormalities are physiologic and suggest pathology of the balance system, quite possibly related to a disruptive effect on otolithic inputs (13, 14). The specificity for detection of "otolith disorders" is unclear (22), however. It has been stressed that otolithic disorders affect posture, but postural tests themselves do not specifically measure otolithic function. This has become important as otolithic disorders can occur in an isolated fashion without other pathology (23). Individuals who have suffered head and neck trauma can also exhibit otolithic symptoms and it has been shown that the results of assessments in two different populations (i.e., vestibular dysfunction as a result of trauma and without trauma) are the same (4, 21, 24). A wide range of pharmacotherapeutics is available to manage symptoms of dizziness (18), but there are none that relieve the underlying cause. Cognitive behavioral therapy combined with traditional vestibular physical therapy has offered some relief to those experiencing anxiety-related dizziness, but no lasting effect has been demonstrated (25).

The vestibular system may also generate symptoms of dizziness in some earth-bound individuals similar to those of returning astronauts even in the absence of balance difficulty. These individuals are often categorized as being visually sensitive or having visual dependence (2, 19, 21). This symptom complex has been recently grouped under the term persistent postural-perceptual dizziness or PPPD (25). A significant obstacle to the development of a standard treatment protocol for individuals with PPPD is that they do not always demonstrate vestibular dysfunction with standard clinical testing.

Individuals who have been given a diagnosis of PPPD for their symptom complex often state after a CDP assessment that the assessment protocol has reproduced their symptom set. This report from a patient is very helpful diagnostically, as it suggests the symptoms are of vestibular origin (24, 26). In addition to helping localize the pathology, it is also often a great relief to the individual sufferer, who may be concerned that their complaints will be dismissed. It is not uncommon for patients to hear that "since all the assessments are normal there probably isn't anything wrong with you." This leads to wrongly categorizing their complaints as psychiatric in origin (18). No other assessment techniques have been shown to simulate and characterize this symptom set, thus CDP remains a valuable clinical tool.

Benefits of VR to vestibular rehabilitation

Vision is a powerful input to the balance system, and its role as a sensory input in balance maintenance is well assessed by CDP. Disturbances of posture and spatial orientation via visual motion and complexity are, however, due to perceptual as well as vestibular influences (27–29) which can not be fully controlled with CDP. Several studies have demonstrated that posture could be functionally changed after repeated exposures to intense optokinetic stimulation (30, 31) or visually busy virtual environments (32, 33). The limiting factor to this approach has been the tolerance of an individual to the treatment which often exacerbates negative symptoms of nausea and dizziness (27, 33).

In order to assess the role played by the vision in everyday environmental situations, it is useful to expose the patient to a wide variety of visual conditions relevant to daily life activities. Although this is not an option provided by a standard CDP system, the tremendous advances in computer graphics technology and the development of VR now afford us the ability to re-create specific visual environments and also change the properties of a visual scene to reduce or enhance object "clutter," alter positions of objects, and change colors and contrast. These are all properties that influence motion perception within the visual environment. A visual scene can also be programmed to move in-phase or out-of-phase with the natural movements of the patient. All these capabilities can be of value when the patient is being assessed to objectively define subjective reports of dizziness. Once the instrumental disturbance is identified, the individual can be trained to cope with existing deficits. Still to be explored is whether it is more effective to gradually expose the system to disturbing stimuli in order to provoke neural adaptation (34), or to disrupt the system with intense stimulation (19, 20, 22, 35, 36) to provoke desensitization and habituation. Both approaches can be addressed with VR.

Neither visual or vestibular signals are exclusively responsible for perceptual disturbances of balance and spatial orientation. Indeed, the ability to separate self-motion from motion of the world is dependent on the confluence of visual-vestibular information. Spatial disorientation is evident during an illusion of self-motion and even during self-initiated motion when a full field of view visual motion does not match actual or imagined physical motion (37). Positron emission tomography (PET) and magnetic resonance imaging (MRI) studies indicate that when both retinal and vestibular inputs are processed, there are changes in the medial parieto-occipital visual area and parieto-insular vestibular cortex (38, 39) as well as the cerebellar nodulus (28, 34) suggesting the nervous system deactivates the structures that process object-motion when there is a perception of physical motion.

VR is an excellent tool for the clinical assessment and intervention of postural disorders resulting from sensory mismatch. As stated above, the value of VR in assessment lies in its flexible presentation of complex and demanding visual environments. A recent study (40) has demonstrated that the presence or absence of recognizable objects and verticality cues in the visual world influences the attainment of postural control and spatial orientation in individuals diagnosed with visual-vestibular mismatch disorders.

VR can portray unexpected circumstances, such as a tilting room, producing a mismatch between the world and physical motion. This produces a sensory conflict resulting in the inability to distinguish between visual motion and self-motion (41, 42). A virtual optic flow field not matched to the performer's head motion produces disparity between visual and vestibular inputs that results in a perception of self-motion called vection (32). Extensive work in the literature, ranging from behavioral studies (31, 33, 43) to neuroimaging studies (27, 34, 38) has demonstrated that vestibular and visual inputs converge at the brainstem and cortical levels during vection to contribute to the illusion of self-motion. Current approaches to the treatment of perceptual-vestibular complaints rely on desensitization to such erroneous or conflicting visual cues that occur in VR (30, 35, 36, 44).

But it also needs to be acknowledged that VR alone is not sufficient for assessment of postural dysfunction. CDP is a reliable measure of postural instability but VR requires additional technologies (e.g., motion analysis and EMG systems) to measure meaningful change in motor control mechanisms (45, 46). The weakness of VR as an independent measure of postural stability was revealed in an intervention protocol performed by Drs. Keshner, Longridge and Mallinson at Vancouver General Hospital (unpublished data). Using standardized clinical tools, they attempted to chart changes in postural control and disorientation in four individuals (37–60 yrs) who had complained of chronic symptoms of dizziness and nausea. Each participant stood on dense foam while wearing an Oculus Rift (https://www.oculus.com) and viewing a virtual environment with no cues to vertical once a week for four weeks. The visual field was rotated sinusoidally in pitch and roll and anterior-posterior translated at 0.25 Hz for 60 s, 3 times in each direction. The field of view (FOV) was initially presented as central visual field stimulation of +/- 10 deg horizontal and gradually increased over the four weeks. Outcome measures were taken following a pre-intervention and post-intervention trial with a more complexly textured environment.

Outcome measures suggested that exposure to the virtual environment had a positive impact on postural control, but the investigators were disheartened by the variability of results in patients with apparently similar symptoms. Participants fell on 75% of the pre-test trials whereas only one fall occurred during post-testing, but this was not reflected on the Falls Efficacy Scale results (Figure 1). Three of four subjects reported increased balance confidence and reductions in subjective dizziness. However, these results relied on subjective reporting and could have been due to practice or individualized attention. There was no way to verify that gradually increasing exposure to conflicting visual and self-motion feedback produced adaptation or habituation. To do that, a more objective measurement technology, such as CDP, would need to be incorporated into this protocol (49).

Reinventing CDP in the 21st century

Initial attempts to measure and quantify balance maintenance through CDP revealed automatic postural reactions that exhibited shorter latencies than voluntary behaviors but were also adaptive and modifiable (16) depending on the parameters of the postural disturbance and the particular abilities of the individual. Thus, posture maintenance was considered a process that was learned and became automatic through repeated use, but also depended primarily upon the nature of the task at hand. It was not thought to be influenced by any pre-planning or attentional resources of the performer. Scientific and clinical interest focused primarily on the sensory pathways controlling and generating postural reactions (2, 3) and how the loss of these pathways would alter postural behavior.

The greatest value of CDP as a diagnostic tool is that it quantifies how a person is using their balance system to

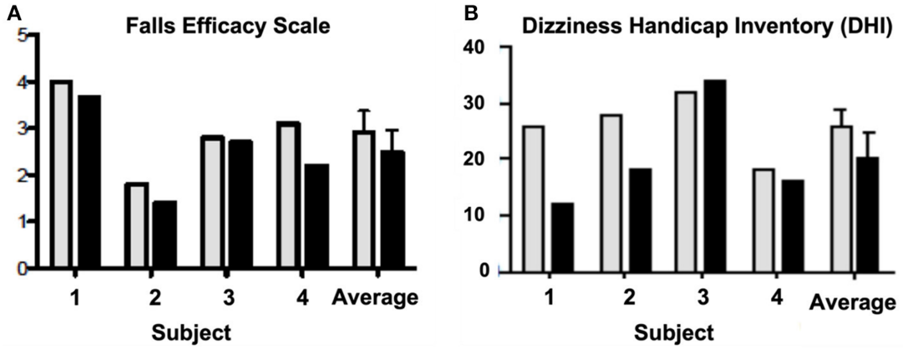

FIGURE 1
Pre-test (gray) and post-test (black) scores on the **(A)** Falls Efficacy Scale (47) and the **(B)** Dizziness Handicap Inventory (DHI) (48) for each subject. The last two columns on each graph are the average scores + SD across all four subjects for the pre- and post-test.

cope with different sensory inputs even though the role played by the vestibular system in balance maintenance, and what happens when something goes wrong with the system, is still not well documented. The development of CDP supplied the tools for the clinician to document and quantify abnormalities in patients who up until this point were diagnosed based on their subjectively reported symptomatic history alone. CDP could be used to measure static sway and also increase the overall diagnostic sensitivity of vestibular function testing. CDP can also be used to design a balance training program to help address a motor behavior deficit and monitor the progression of clinical recovery. The current absence of reliable inter-institutional comparisons might be assuaged by incorporating an inexpensive, reliability tested hard CDP like standard force plate which could potentially reduce the years of contradictory studies necessary to find the most effective therapeutic protocols.

When initially developed, CDP was innovative and ahead of its time. However, as new technologies and better computerization have developed, the advent of VR gives an opportunity to modernize CDP so that it continues to be a valuable clinical tool. The principle behind VR is its ability to produce a disturbance in spatial orientation through perception of the visual environment, rather than mechanical instability at the base of support. Thus, incorporating VR as a tool for assessing and treating balance disorders may shift the focus of clinical assessment from the maintenance of biomechanical stability to the assessment of whole body balance and orientation behaviors (46). Currently, a number of low-cost VR systems designed for and targeting rehabilitation have become available. These include a variety of rehabilitation-oriented desktop gaming programs that implement VR properties (e.g., feedback, documentation, motivation). The increasing accessibility of embedded ambient technologies (e.g., inexpensive cameras, proximity sensors, wearable computing) that support the monitoring of motor and cognitive functioning under real-world conditions has extended VR-based interventions beyond the clinical setting (50). Evidence is also accumulating that learning in the virtual environment will transfer to the physical world (29, 51, 52). Through employing the principles of motor learning (53, 54) and adding tools such as robots, treadmills, and dynamic platforms into the virtual environment, we can manipulate task and environmental demands in order to assess meaningful postural behavior. Continued improvements in this technology indicate a high probability that VR will become an integral component of (and possibly supplant) CDP (50) to provide valued diagnostic and therapeutic information to the clinician.

Discussion

The broad range of postural control and motor abilities among both healthy and impaired individuals necessitates that test conditions be adapted to individual abilities. This would require the development of various test conditions and comparison of the performance within each individual, which can be achieved with VR. Similar to the combination of optokinetic stimulation and VR, combining CDP with VR should assist with rehabilitation by inducing symptoms and encouraging “habituation.” CDP and VR are also helpful in documenting and reproducing patient symptoms, which will aid the clinician to understand the presenting complaints and perform, through the use of these tools, optimal rehabilitation. We maintain that combining CDP with VR provides the strongest available approach to assessment and intervention for postural and orientation disorders.

Data availability statement

The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author.

Ethics statement

The studies involving human participants were reviewed and approved by University of Vancouver IRB. The patients/participants provided their written informed consent to participate in this study.

Author contributions

EK, AM, and PP were responsible for the content of the paper. NL, SS, and HP were responsible for editing and approving the content. All authors participated as speakers at the Clinical Evaluation of Balance Disorders (ESCEBD) meeting in Nancy, France and contributed to the conceptualization of this paper.

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References

1. Magnus R, de Kleijn A. The influence of the position of the head on the tone of the muscles of the extremities. *Pflugers Arch.* (1912) 145:455–548. doi: 10.1007/BF01681127

2. Mergner T, Maurer C, Peterka RJ, A multisensory posture control model of human upright stance. *Prog Brain Res.* (2003) 142:189–201. doi: 10.1016/S0079-6123(03)42014-1

3. Peterka RJ. Sensory motor integration in human postural control. *J Neurophysiol.* (2002) 88:1097–118. doi: 10.1152/jn.2002.88.3.1097

4. Mallinson A, Maire R, Beyaert C, Vibert D, Coffinet L, Longridge N, et al. Understanding and managing trauma induced vestibular deficits. *J Internat Adv Otol.* (2021) 17:559–65. doi: 10.5152/iao.2021.21258

5. Lamontagne A, Keshner EA, Bugnariu N, Fung J. "Virtual reality reveals mechanisms of balance and locomotor impairments". In Weiss PL (Tamar), Keshner EA, Levin MF (editors). *Virtual Reality for Physical and Motor Rehabilitation.* New York: Springer (2014), p. 169-202. doi: 10.1007/978-1-4939-0968-1_9

6. Keshner EA, Kenyon RV, Langston J. Postural responses exhibit multisensory dependencies with discordant visual and support surface motion. *J Vestib Res.* (2004) 14:307–19. doi: 10.3233/VES-2004-14401

7. Benson AJ, Barnes GR. Vision during angular oscillation: the dynamic interaction of visual and vestibular mechanisms. *Aviat Space Environ Med.* (1978) 49:340–5.

8. Oman CM. Are evolutionary hypotheses for motion sickness "just-so" stories? *J Vestib Res.* (2012) 22:117–27. doi: 10.3233/VES-2011-0432

9. Preber L. Vegetative reactions in caloric and rotatory tests. A clinical study with special reference to motion sickness. *Acta Otolaryngol Suppl (Stockh).* (1958) 144:1–119.

10. Oman CM, Lichtenberg BK, Money KM, McCoy RK. MIT/Canadian vestibular experiments on the Spacelab-1 mission: 4 Space motion sickness: symptoms, stimuli and predictability. *Exp Br Res.* (1986) 64:316–34. doi: 10.1007/BF00237749

11. Parker DE. The relative roles of the otolith organs and semicircular canals in producing space motion sickness. *J Vestib Res.* (1998) 8:57–9. doi: 10.3233/VES-1998-8108

12. Oman CM. Sensory conflict theory and space sickness: our changing perspective. *J Vestib Res.* (1998) 8:51–6. doi: 10.3233/VES-1998-8107

13. Black FO, Paloski WH, Reschke MF, Igarashi M, Guedry F, Anderson DJ. Disruption of postural readaptation by inertial stimuli following space flight. *J Vestib Res.* (1999) 9:369–78. doi: 10.3233/VES-1999-9506

14. Mallinson AI. *Visual-Vestibular Mismatch: A Poorly Understood Presentation of Balance System Disease.* [dissertation thesis]. Maastricht: University of Maastricht (2011).

15. Nashner LM. *Sensory Feedback in Human Posture Control.* [dissertation thesis]. Boston, MA: Massachusetts Institute of Technology (1970).

16. Peters JF, Nashner LM. Dynamic posturography in the diagnosis and management of dizziness and balance disorders. *Neurol Clin.* (1990) 8:331–49. doi: 10.1016/S0733-8619(18)30359-1

17. Horak FB, Henry SM, Shumway-Cook A. Postural perturbations: New insights for treatment of balance disorders. *Phys Ther.* (1997) 77:517–33. doi: 10.1093/ptj/77.5.517

18. Perrin P, Mallinson A, Van Nechel C, Peultier-Celli L, Petersen H, Magnusson M, et al. Defining clinical-posturographic and intra-posturographic discordances: what do these two concepts mean? *J Int Adv Otol.* (2018) 14:127–9. doi: 10.5152/iao.2018.4349

19. Mallinson AI, Longridge NS. Caloric response does not decline with age. *J Vestib Res.* (2004) 14:393–6. doi: 10.3233/VES-2004-14505

20. Lipp M, Longridge NS. Computerized dynamic posturography: its place in the evaluation of patients with dizziness and imbalance. *J Otolaryngol.* (1994) 23:177–83.

21. Mallinson AI, Longridge NS. Specific vocalized complaints in whiplash and minor head injury patients. *Am J Otol.* (1998) 20:809–13.

22. Basta D, Clarke A, Ernst A, Todt I. Stance performance under different sensorimotor conditions in patients with post-traumatic otolith disorders. *J Vestib Res.* (2007) 17:25–31. doi: 10.3233/VES-2007-17103

23. Lee JD, Park MK, Lee BD, Park JY, Lee TK, Sung KB. Otolith function in patients with head trauma. *Eur Arch Otorhinolaryngol.* (2011) 268:1427–30. doi: 10.1007/s00405-010-1426-5

24. Mallinson AI, Longridge NS. Visual vestibular mismatch in work related vestibular injury. *Otol Neurotol.* (2005) 26:691–4. doi: 10.1097/01.mao.0000169637.71064.c6

25. Staab JP, Eckhardt-Henn A, Horii A, Jacob R, Strupp M, Brandt Th, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the committee for the classification of vestibular disorders of the Bárány Society. *J Vestib Res.* (2017) 27:191–208. doi: 10.3233/VES-170622

26. Mallinson AI, Kuijpers AC, Van Zwieten G, Kakal J, Mullings W, Longridge NS. Computerized dynamic posturography does not detect measured cVEMP and oVEMP abnormalities. *Gait Posture.* (2019) 67:248–50. doi: 10.1016/j.gaitpost.2018.10.019

27. Wiest G, Amorim MA, Mayer D, Schick S, Deecke L, Lang W. Cortical responses to object-motion and visually-induced self-motion perception. *Cog Brain Res.* (2001) 12:167–70. doi: 10.1016/S0006-8993(01)02457-X

28. Dieterich M, Brandt T. Brain activation studies on visual-vestibular and ocular motor interaction. *Curr Opin Neurol.* (2000) 13:13–8. doi: 10.1097/00019052-200002000-00004

29. Levin MF, Deutsch JE, Kafri M, Liebermann DG. "Validity of virtual reality environments for sensorimotor rehabilitation". In Weiss PL (Tamar), Keshner EA, Levin MF (editors). *Virtual Reality for Physical and Motor Rehabilitation*. New York: Springer (2014), p. 95-118. doi: 10.1007/978-1-4939-0968-1_6

30. Gallagher M, Choi R, Ferrè ER. Multisensory interactions in virtual reality: Optic flow reduces vestibular sensitivity, but only for congruent planes of motion. *Multisens Res.* (2020) 33:625–44. doi: 10.1163/22134808-20201487

31. Slobounov S, Tutwiler R, Sebastianelli W, Slobounov E. Alteration of postural responses to visual field motion in mild traumatic brain injury. *Neurosurg.* (2006) 59:134–9. doi: 10.1227/01.NEU.0000219197.33182.3F

32. Keshner EA, Kenyon RV. The influence of an immersive virtual environment on the segmental organization of postural stabilizing responses. *J Vestib Res.* (2000) 10:207–19. doi: 10.3233/VES-2000-104-505

33. Thurrell AE, Bronstein AM. Vection increases the magnitude and accuracy of visually evoked postural responses. *Exp Brain Res.* (2002) 147:558–60. doi: 10.1007/s00221-002-1296-1

34. Kleinschmidt A, Thilo KV, Buchel C, Gresty MA, Bronstein AM, Frackowiak RS. Neural correlates of visual-motion perception as object- or self-motion. *Neuroimage.* (2002) 16:873–82. doi: 10.1006/nimg.2002.1181

35. Pavlou M, Bronstein AM, Davies RA. Randomized trial of supervised versus unsupervised optokinetic exercise in persons with peripheral vestibular disorders. *Neurorehabil Neural Repair.* (2013) 27:208–18. doi: 10.1177/1545968312461715

36. Bronstein AM, Pavlou M. Balance. *Handbook Clin Neurol.* (2013) 110:189–208. doi: 10.1016/B978-0-444-52901-5.00016-2

37. Wright WG, Agah MR, Darvish K, Keshner EA. Head stabilization shows visual and inertial dependence during passive stimulation: implications for virtual rehabilitation. *IEEE TNSRE.* (2013) 21:191–7. doi: 10.1109/TNSRE.2012.2237040

38. Brandt T, Glassauer S, Stephan T, Bense S, Yousry TA, Deutschlander A, et al. Visual-vestibular and visuovisual cortical interaction: new insights from fMRI and PET. *Ann NY Acad Sci.* (2002) 956:230–41. doi: 10.1111/j.1749-6632.2002.tb02822.x

39. Brandt T, Bartenstein P, Janek A, Dieterich M. Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. *Brain.* (1998) 121:1749–58. doi: 10.1093/brain/121.9.1749

40. Al-Sharif DS, Tucker CA, Coffman DL, Keshner EA. The effects of visual context on visual-vestibular mismatch revealed by electrodermal and postural response measures. *J Neuroeng Rehabil.* (2022) 19:113. doi: 10.1186/s12984-022-01093-5

41. Lackner JR, DiZio P. Visual stimulation affects the perception of voluntary leg movements during walking. *Perception.* (1988) 17:71–80. doi: 10.1068/p170071

42. Lambrey S, Berthoz A. Combination of conflicting visual and non-visual information for estimating actively performed body turns in virtual reality. *Int J Psychophysiol.* (2003) 50:101–15. doi: 10.1016/S0167-8760(03)00127-2

43. Dichgans J, Brandt T, Held R. The role of vision in gravitational orientation. *Fortschr Zool.* (1975) 23:255–63.

44. Akiduki H, Nishiike S, Watanabe H, Matsuoka K, Kubo T, Takeda N. Visual-vestibular conflict induced by virtual reality in humans. *Neurosci Lett.* (2003) 340:197–200. doi: 10.1016/S0304-3940(03)00098-3

45. Merians AS, Tunik E, Fluet GG, Qiu Q, Adamovich SV. Innovative approaches to the rehabilitation of upper extremity hemiparesis using virtual environments. *Eur J Phys Rehabil Med.* (2009) 45:123–33.

46. Keshner E, Lamontagne A. The untapped potential of virtual reality in rehabilitation of balance and gait in neurological disorders. *Front Virtual Real.* (2021) 2:641–50. doi: 10.3389/frvir.2021.641650

47. Yardley L, Beyer N, Hauer K, Kempen G, Piot-Ziegler C, Todd C. Development and initial validation of the Falls Efficacy Scale-International (FES-I). *Age Ageing.* (2005) 34:614–9. doi: 10.1093/ageing/afi196

48. Formeister EJ, Krauter R, Kirk L, Zhu TR, Rizk HG, Sharon JD. Understanding the Dizziness Handicap Inventory (DHI): aA cross sectional analysis of symptom factors that contribute to DHI variance. *Otol Neurotol.* (2020) 41:86–93. doi: 10.1097/MAO.0000000000002438

49. San Pedro Murillo E, Bancroft MJ, Koohi N, Castro P, Kaski D. Postural misperception: a biomarker for persistent postural perceptual dizziness. *J Neurol Neurosurg Psychiatry*. (2022) 3:9321. doi: 10.1136/jnnp-2022-329321

50. Weiss PL (Tamar), Keshner EA, Levin MF. "Current and future trends for VR and motor rehabilitation". In Weiss PL (Tamar), Keshner EA, Levin MF (editors). *Virtual Reality for Physical and Motor Rehabilitation*. New York: Springer (2014), p. 217-226. doi: 10.1007/978-1-4939-0968-1_11

51. Joyner JS, Vaughn-Cooke M, Benz HL. Comparison of dexterous task performance in virtual reality and real-world environments. *Front Virtual Real.* (2021) 2:599–274. doi: 10.3389/frvir.2021.599274

52. Mirelman A, Deutsch JE, Hausdorff JM. "Virtual reality augmented training for improving walking and reducing fall risk in patients with neurodegenerative disease". In Weiss PL (Tamar), Keshner EA, Levin MF (editors). *Virtual Reality for Physical and Motor Rehabilitation*. New York: Springer (2014), p. 145-168. doi: 10.1007/978-1-4939-0968-1_8

53. Krakauer JW. Motor learning: its relevance to stroke recovery and neurorehabilitation. *Curr Opin Neurol.* (2006) 19:84–90. doi: 10.1097/01.wco.0000200544.29915.cc

54. Levac DE, Huber ME, Sternad DJ. Learning and transfer of complex motor skills in virtual reality: a perspective review. *J NeuroEng Rehab.* (2019) 16:121 doi: 10.1186/s12984-019-0587-8

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Distinct cortico-muscular coupling between step and stance leg during reactive stepping responses

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Balance recovery often relies on successful stepping responses, which presumably require precise and rapid interactions between the cerebral cortex and the leg muscles. Yet, little is known about how cortico-muscular coupling (CMC) supports the execution of reactive stepping. We conducted an exploratory analysis investigating time-dependent CMC with specific leg muscles in a reactive stepping task. We analyzed high density EEG, EMG, and kinematics of 18 healthy young participants while exposing them to balance perturbations at different intensities, in the forward and backward directions. Participants were instructed to maintain their feet in place, unless stepping was unavoidable. Muscle-specific Granger causality analysis was conducted on single step- and stance-leg muscles over 13 EEG electrodes with a midfrontal scalp distribution. Time-frequency Granger causality analysis was used to identify CMC from cortex to muscles around perturbation onset, foot-off and foot strike events. We hypothesized that CMC would increase compared to baseline. In addition, we expected to observe different CMC between step and stance leg because of their functional role during the step response. In particular, we expected that CMC would be most evident for the agonist muscles while stepping, and that CMC would precede upregulation in EMG activity in these muscles. We observed distinct Granger gain dynamics over theta, alpha, beta, and low/high-gamma frequencies during the reactive balance response for all leg muscles in each step direction. Interestingly, between-leg differences in Granger gain were almost exclusively observed following the divergence of EMG activity. Our results demonstrate cortical involvement in the reactive balance response and provide insights into its temporal and spectral characteristics. Overall, our findings suggest that higher levels of CMC do not facilitate leg-specific EMG activity. Our work is relevant for clinical populations with impaired balance control, where CMC analysis may elucidate the underlying pathophysiological mechanisms.

KEYWORDS

balance control, corticomuscular coupling, EEG, reactive stepping, motor control

1. Introduction

Performing daily activities (e.g., standing or walking) constantly challenges postural balance. With different postures and activities, continuous adaptation through contraction and relaxation of specific muscles allows the control of balance and the execution of corrective steps whenever these are needed. In the past decade, many studies using mobile EEG have provided evidence that the cortex plays an important role in postural control (1–3). This notion is in line with studies investigating postural control in people with cortical lesions (e.g., stroke), which reported deficient recruitment of the muscles involved in perturbation-evoked responses, including delayed onset latencies, lower response amplitudes, and aberrant coordination patterns across muscles (4, 5).

Postural perturbations are known to elicit several event-related potentials, suggesting cortical involvement in the ensuing balance recovery responses. The initial P1 response (30–90 ms) is thought to represent proprioceptive sensory afferents (6), followed by the N1 (90–200 ms), which is suggested to reflect monitoring of postural stability (1, 7–11). The N1 perturbation-related response is accompanied by a transient power increase of the theta (3–8 Hz) rhythm (12). Interestingly, the N1 and theta rhythm have been shown to predict the response outcome to a balance perturbation with stronger cortical dynamics for stepping responses compared to feet-in-place responses at similar perturbation intensities (12). Yet, little is known about the temporal evolution of cortical interaction with specific leg muscles that contribute to the generation of the balance correcting response (either directly or indirectly through cortico-cortical coupling with the motor areas).

Cortico-muscular coupling (CMC) is a powerful analysis tool to investigate functional connectivity between the cerebral cortex and muscles across the body. CMC is known to take place with the Tibialis Anterior (TA) and Soleus (SO) muscles at beta (15–25 Hz) frequencies during isometric contraction and gamma (40–80 Hz) frequencies during isotonic contraction (13, 14). In addition, several studies reported beta frequency coupling with the medial Gastrocnemius (MG) and TA muscles during cyclic ankle movements (15). These findings suggest that multiple cortical frequency bands may couple to similar muscles during different muscle exercises. Until today, only a few studies have investigated CMC of dynamic human behavior related to postural control and gait. Moreover, studies investigating gait reported an increase in theta, alpha, and beta band CMC after foot strike with TA, Vastus Medialis (VM), Biceps Femoris (BF), Peroneus longus (PL), MG and SO muscles (16–18). Cortical involvement in the balance response is thought to occur during later phases of the balance recovery response (19, 20), which suggests that reactive step responses may coincide with strong CMC time locked to perturbation onset. Only few studies investigated CMC during walking and standing balance (17, 21). Muscle synergies showed strong coherence with cortical activity over the Piper rhythm (∼40 Hz) during lateral balance perturbation of unipedal stance that led to feet-in-place responses (21). In addition, strong theta and alpha (8–13 Hz) band CMC dynamics were observed from cortex to muscle with MG, TA and PL muscles during physical standing perturbations leading to feet-in-place responses compared to visual perturbations while standing (17). This indicates that physical perturbations require more cortical control. Yet, their experimental setup only focused on the theta and alpha frequency bands averaged over a window of 1 s, thus lacking information on the temporal evolution of cortical interaction with the muscles during the balance response. In addition, beta and gamma frequency bands are motor task related rhythms, which may also be involved in the recruitment of balance responses.

The aim of this study was to investigate CMC from cortex to muscle through spectral Granger causality coupled to key events of the stepping response in balance. We hypothesized that CMC would occur over multiple frequency bands during stepping responses. In addition, we expected CMC to differ between the step and stance leg according to their differential muscle recruitment patterns inherent in executing the step response. In particular, we expected that stronger CMC would be most evident for the muscles involved in generating the greatest biomechanical contribution for the stepping movement (depending on the step and stance leg), and that an increase in CMC would precede changes in EMG activity in these muscles relative to foot off and foot strike event. Therefore, we conducted separate CMC analysis time-locked to perturbation onset, foot-off and foot strike event.

2. Materials and methods

Twenty young healthy adults participated in this study. We analyzed a total of 18 datasets (8 female; age mean 23.9 years, sd 3.6 years) due to technical issues in two other datasets. All participants received ample information about the experiment and signed an informed consent document prior to the start of the experiment. Participants were financially compensated after completion of the study. None of the participants had a history of neuromuscular disease or any other impairment that could affect their performance in the experiment. The experimental procedure was approved by the Research Ethics Committee of the Radboud University Medical Center (Nijmegen, The Netherlands; Dossier 2018-4970). The experiments were conducted in line with the Declaration of Helsinki.

2.1. Experimental paradigm

Data used in this study were derived from a protocol to investigate theta power modulations related to balance monitoring by imposing leaning angles prior to perturbation (22) (Figure 1A). Participants were familiarized with the balance platform through a series of 28 forward and backward perturbations with increasing acceleration, delivered by the Radboud Falls Simulator (2, 12, 23, 24). Participants stood barefoot on the movable platform with their feet at shoulder width and their arms crossed in front of the body and had to maintain three different initial leaning postures prior to a balance perturbation. At the beginning of each sequence, the participants were instructed about which leaning posture to maintain throughout the series of perturbations. Participants were instructed and encouraged to respond with feet-in-place responses following balance perturbations. A real-time 3D-motion data stream monitored the participants' posture and performance (Vicon motion systems, United Kingdom), such as

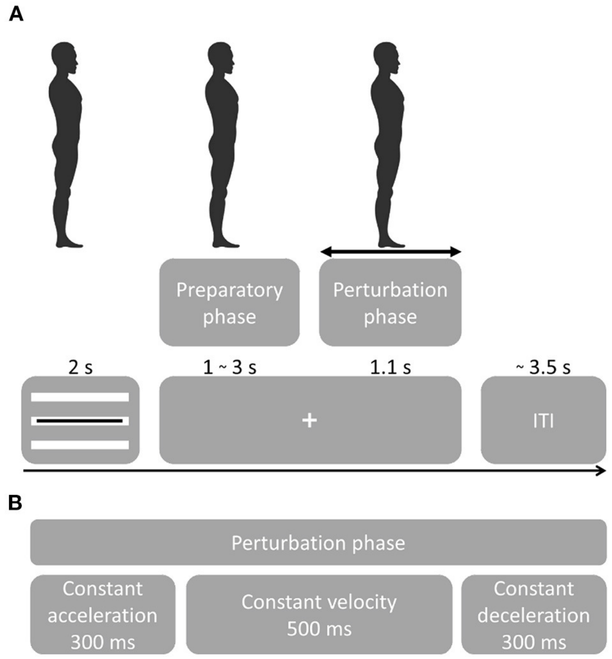

FIGURE 1
Experimental procedure. **(A)** Participants were instructed to maintain a straight posture during the sequence of perturbations. Perturbation onset times were randomized as well as direction and acceleration. A visual cue tracking the participant's posture was presented for 2 s, followed by a fixation cross. Platform onset was randomized from 1–3 s followed by a perturbation that lasted 1.1 s. Following a perturbation, the platform returned to the initial position. At platform return, the visual feedback of the leaning posture was presented. Feedback for the leaning posture [important for the experimental setup in (22)] was presented through three white bars representing forward leaning (top bar), neutral stance (middle bar) and backward leaning (bottom bar). The black bar presented on top of the white bars, indicated the participant's real-time leaning angle. Participants were instructed to maintain the black bar on the white bar corresponding to the instructed leaning posture. The initial leaning posture had to be maintained while the fixation cross was presented ensuring that postural stability was controlled at platform perturbation. **(B)** Platform perturbation profiles.

maintaining leaning angle, excessive knee flexion and changes in leg weight bearing (which may indicate whether specific strategies to counteract balance perturbations were used).

Balance perturbation profiles consisted of 300 ms platform acceleration, 500 ms constant velocity and 300 ms deceleration (Figure 1B). Platform accelerations were randomized and ranged from 0.25 to 1.9 m/s^2 with a higher resolution at lower accelerations in both forward and backward sway direction (0.25, 0.4, 0.7, 1.0, 1.3, 1.6, 1.9 m/s^2). The initial experiment contained 15 sequences with 29 balance perturbations each (435 total). The first perturbation of a sequence always consisted of a low-intensity dummy trial and was not included in the analyses. For the analysis of the present study, we only considered the neutral stance conditions with forward and backward perturbation directions (i.e., 140 trials per participant).

Participants took a small break of 5 min after three consecutive sequences and were seated on a chair to prevent fatigue. After nine perturbation sequences, participants were given a 20-min resting break. The active experiment time was 2.5 h and the preparation time was 2.5 h, the complete lab visit lasted a maximum of 6 h (including resting breaks).

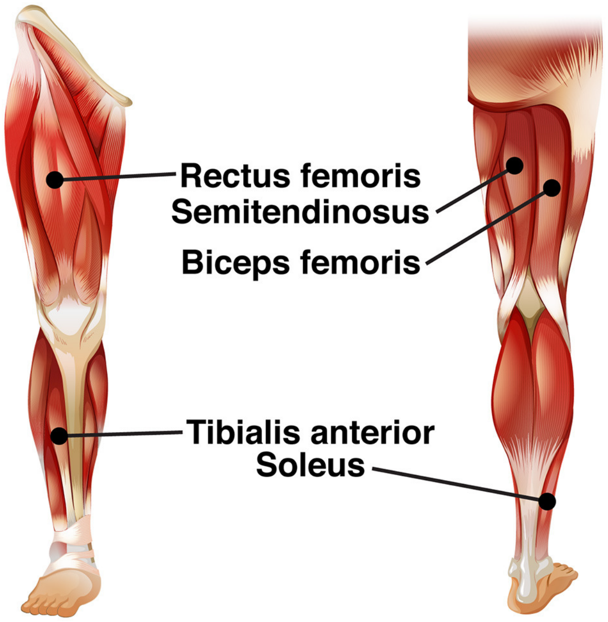

FIGURE 2
EMG electrode leads.

2.2. Data acquisition

We recorded high-density EEG using a cap with 126 Ag-AgCl electrodes (WaveGuard, ANT Neuro, The Netherlands). The electrodes were fixed in the cap and distributed across the scalp according to the five percent electrode system (25). The EEG data were referenced to the common average during acquisition. The ground electrode was placed on the left mastoid. A biosignal amplifier (REFA System, TMSi, The Netherlands) recorded the EEG at 2,048 Hz without any filters, except for a hardware low-pass filter at 552 Hz. To monitor physiological activity that could present artifacts in the EEG, we also recorded electrical activity of the left eye in the vertical and horizontal direction (electrooculogram, EOG) using adhesive Ag-AgCl electrodes. The EOG was recorded from electrodes placed slightly under the left eye (vertical eye movement) and at the outer canthus of the left eye (horizontal eye movement).

We recorded electrical activity bilaterally from five leg muscles [see Figure 2; soleus (SO), tibialis anterior (TA), rectus femoris (RF), biceps femoris (BF), semitendinosus (ST)], using surface EMG electrodes (Mini Wave, Cometa systems, Italy). Muscle sites were shaved to remove hair, and the skin was scrubbed with skin preparation gel (Nuprep, MedCat) to improve skin conduction and cleaned with alcohol. The EMG amplifier (Wave plus wireless, Cometa, Italy) recorded muscle activity at 2,000 Hz. The EMG signal of the muscles was carefully checked before the start of the experimental paradigm.

Body movement was recorded using an 8-camera 3D motion analysis system (Vicon motion systems, United Kingdom) at a sample rate of 100 Hz. For this purpose, a total of 23 reflective markers (PlugInGait Full-body AI model excluding the head and arm markers; Vicon Nexus software 2.7.1) were attached to anatomical landmarks on the participants' body.

Ground reaction forces were recorded from two force plates (AMTI Custom 6 axis composite force platform, USA; size: 60 × 180 cm each; sampling rate: 2,000 Hz) embedded in the moveable platform. Trials were recorded from−2 to +5 s relative to the platform perturbation. Synchronization triggers were generated by the platform controller and recorded for *post-hoc* alignment of EMG, EEG and motion data.

2.3. EMG processing

The EMG signal was preprocessed in MATLAB using low-pass filtering with the 'filtfilt.m' function (125 Hz low-pass filtered 5th order Butterworth IIR filters, zero-phase shift), and downsampling to 250 Hz. EMG was separately preprocessed for EMG envelope visualization and Granger causality.

For EMG envelope visualization, the data were band-pass filtered (20–120 Hz band-pass 5th order Butterworth IIR filters, zero-phase shift), full-wave rectified, low-pass filtered (40 Hz low-pass 5th order Butterworth IIR filters, zero-phase shift), and normalized per muscle per subject to the maximum muscle activation at 0.7 m/s^2 for feet-in-place responses. This normalization allowed us to evaluate the difference in activation between step and stance legs during stepping trials since both legs contributed equally in the feet-in-place trials. The acceleration of 0.7 m/s^2 was the maximum platform acceleration where all participants were able to respond with feet-in-place in either direction.

Prior to Granger causality computation, we downsampled and normalized (z-score) the EMG activity. Then we subtracted the ensemble average EMG from the single-trial EMG data per participant and muscle. This procedure is necessary for Granger causality analyses involving event related potential to facilitate model-fitting and reduce non-stationarities [(26); see Figure 3C for an example on removing non-stationarities from EEG data].

2.4. EEG processing

For the preprocessing of EEG and EOG data, MATLAB functions of the EEGLAB toolbox were used (27). Continuous data were epoched into intervals of −2 to +3 s relative to perturbation onset. Data were bandpass filtered using the 'filtfilt.m' function (2–200 Hz, consecutive high-pass and low-pass 5th order Butterworth IIR filters, zero-phase shift) and common average re-referenced. Noisy channels were flagged for rejection based on a kurtosis >3 and a variance >3 and rejected based on visual inspection. In addition, epochs were visually inspected for noise. Independent components analysis (Infomax ICA) with a minimum of 90 and maximum of 125 principal components (depending on the rank of the EEG data) was run, and independent components were rejected based on being excessively noisy and of non-brain origin (mean = 91, sd = 16 rejected components; two data sets showed excessive noise resulting in a large mean). Back-projection of the retained independent components resulted in artifact-reduced EEG data. Noise rejected channels were interpolated.

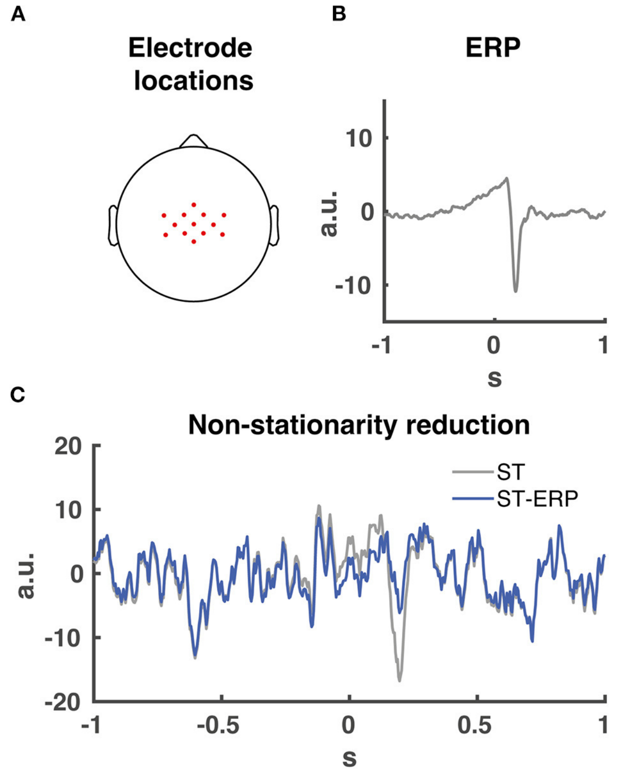

FIGURE 3
EEG electrode locations and ERP signal removal. **(A)** 13 midfrontal electrode scalp locations averaged together for the Granger causality analysis. **(B)** Participant EEG average event related potential (ERP) from midcentral electrode location CZ time-locked to perturbation onset. **(C)** signal non-stationarity reduction. Time series of single trial (ST) EEG data from midcentral electrode location CZ time-locked to perturbation onset indicated in grey. Time series of ERP subtraction from single trial time series data (ST-ERP) in blue.

Our and other studies indicated that midfrontal cortical activity plays a major role during the initial phase of the balance response regarding the monitoring and cortical control of the balance response (2, 12, 17, 22). In addition, cortical (pre-)motor regions are located around the midfrontal head location. Therefore we chose a substantial amount of midrontal electrodes, resulting in the 13 selected electrodes (FCz, Cz, CPz, C1, C2, CCP3h, CCP1h, CCP2h, CCP4h, FCC3h, FCC1h, FCC2h and FCC4h; see Figure 3A for topographical locations) centered over the midfrontal scalp location for CMC analysis. Prior to the Granger causality computation, the ERP (Figure 3B) was subtracted from the single-trial data for both EMG and EEG. This process maintains spectral information of the data and reduces the non-stationarities in the signal (Figure 3C).

2.5 Data inclusion

We collected a total of 2,491 trials and rejected 149 trials, based on flat lines and artifacts in EEG and EMG data. Of the remaining 2,342 trials (mean = 130, sd = 19 trials per subject), 1,177 were forward and 1,165 were backward perturbations. In the forward direction there were 675 feet-in-place responses vs. 502 step responses. In the backward direction, we recorded 565 feet-in-place responses and 600 step responses.

2.6 Granger causality analysis

We applied Granger causality analysis to compute the directional coupling from the cerebral cortex to the muscles between individual EEG channels and EMG data over 1 to 100 Hz with a resolution of 0.05 Hz. For the analysis we used the spectral Granger causality Matlab MVGC toolbox (28, 29). We applied a sliding window of 400 ms to predict EMG activity from the EEG signal (using a smaller window would collapse the frequency interpretation of lower frequencies, whereas a larger window would diminish temporal accuracy). We used a model order of 100 ms, meaning we predicted muscle activity up to 100 ms ahead of the current EEG sample. This 100 ms model order was visually determined through time domain Granger causality tests of multiple participants, identifying a clear Granger causality increase relative to baseline (−1.4 to −0.75 s relative to perturbation onset) and a stationary baseline Granger causality. The time domain Granger causality analysis determined the Granger gain of cortical interaction averaged over all muscles. According to these parameters, Granger causality temporal data should be interpreted as a prediction of EMG from EEG over the past 400 ms window where EEG signal of sample t_0 predicts EMG activity up to sample t_{100} (up to 100 ms ahead).

Granger Causality analysis benefits from large amount of data. To optimize the Granger causality outcome we conducted the analysis over a time series of 4 s with a consistent amount of data for all frequency bands and individual muscles. Second, we included all step trials per participant, which resulted in a substantial amount of 500 (forward) and 600 (backward) trials (see 2.5 Data inclusion). In addition, we removed the ERP from both the EMG and EEG data to improve the signal stationarity (see 2.4 EEG processing). The sliding window approach of the Granger causality analysis provided by the MVGC toolbox (28, 29) also improves analysis of non-stationary signals. The sliding window was sufficiently large enough to interpret low frequency (3–8 Hz) data similar to the Peterson study (17). Lastly, the data was averaged over 13 midfrontal electrodes (see 2.4 EEG processing and Figure 3C), which further increased the midfrontal Granger causality signal to noise ratio.

We conducted spectral Granger causality relative to three time-locking events to get a good alignment with the EMG activity. Due to the temporal sliding window of the Granger causality analysis, between-signal comparisons can only be made at specific time locked events. Therefore, we time-locked to three specific events at which we can temporally accurately compare EMG with Granger causality at time 0 s. First, perturbation onset time-locked Granger causality analysis was done to investigate coupling with muscles immediately after perturbation. Secondly, Granger causality time-locked to foot-off event was computed to investigate CMC prior to step initiation and during stepping response. Finally, Granger causality time-locked to foot strike was done to investigate CMC prior to and after foot landing. For each time-locking event we averaged the Granger causality data over all 13 EEG electrodes, meaning that we analyzed one average cortical Granger causality time-frequency map for each muscle.

2.7. Statistical analysis

For EMG and CMC time series data, statistical tests were done over all time windows of the time-locked events. Interquartile range latencies were used to determine the upper temporal boundaries for statistical testing. Participant average foot off event latencies did not significantly differ between directions (Ranksum = 391, Z = 1.8, $p = 0.07$; $\text{median}_{forward} = 451$ ms, $\text{IQR}_{forward} = 210$ ms, $\text{median}_{backward} = 400$ ms, $\text{IQR}_{backward} = 202$ ms). In addition, foot strikes latencies did not differ for step direction (Ranksum= 362, Z=0.9, p=0.23; $\text{median}_{forward} = 641$ ms, $\text{IQR}_{forward} = 214$ ms, $\text{median}_{backward} = 629$ ms, $\text{IQR}_{backward} = 225$ ms). For convenience, we rounded the 75th percentile values up to foot off = 600 ms, foot strike = 800 ms. This resulted in the following analysis time windows; perturbation onset to 600 ms, −200 to 250 ms relative to foot off and−250 to 200 ms relative to foot strike. Please note that there is temporal overlap between these time windows. To determine whether significant EMG activity and CMC occurred relative to baseline activity in response to balance perturbations, multiple sample-wise t-tests were conducted time-locked to perturbation onset for each muscle per step and stance leg separately. Given the multiple tests computed in the time domain, the corresponding p-values were corrected for false discovery rate [FDR; (30)]. Statistical significance was assessed for critical $\alpha = 0.05$. For EMG activity, an activity duration threshold of 100 ms and mean baseline activity +1SD (−500 to 0 ms relative to perturbation onset) was used to eliminate premature false positive significance results.

To determine differences between step and stance leg, significant differences between muscle specific step and stance leg EMG activity were computed using a sample-wise t-test. Differences in Granger gain for muscle-specific step and stance legs were computed with sample-wise t-tests ($p < 0.05$, FDR correction). Temporal windows for significance testing were determined by foot-off and foot strike latencies.

3. Results

3.1. Corticomuscular coupling

Time-frequency analysis averaged over all 13 midfrontal EEG electrodes and muscles in the forward and backward direction revealed increases in Granger causality over multiple frequency bands in response to perturbation onset (Figure 4). The frequencies of interest for further analysis were in the theta (θ: 3–8 Hz), alpha (α: 10–13 Hz), beta (β: 15–22 Hz), low gamma (γ_{lo}: 25–40 Hz)

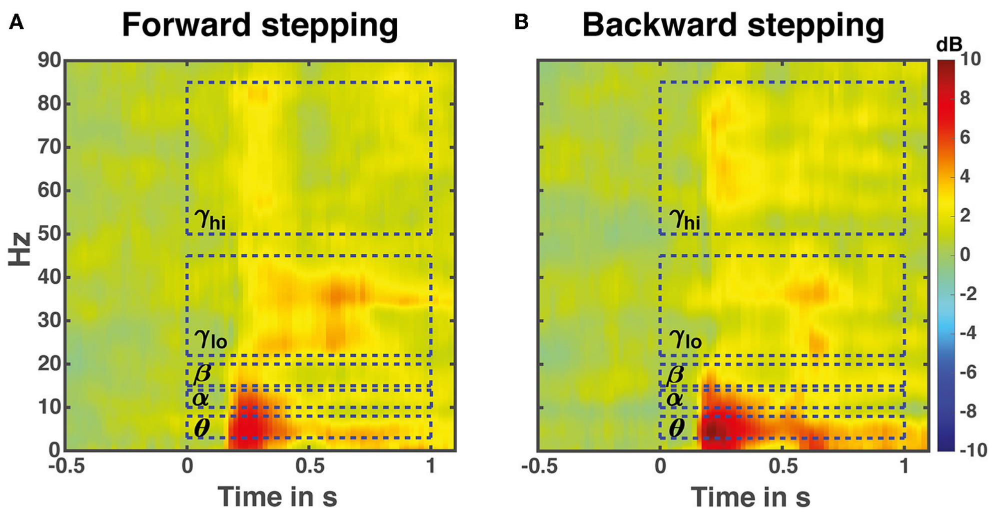

FIGURE 4
Perturbation time-locked time frequency granger gain. Illustrated data are averaged over all leg muscles in either the forward **(A)** or backward **(B)** stepping direction. The dashed boxes indicate frequency bands of interest for the time course analyses. Note that the temporal boundaries are illustrative; the actual time windows for analysis differed based on stepping times and time-locking events.

and high gamma (γ_{hi}: 50–85 Hz) ranges, and are indicated with dashed boxes.

3.2. EMG envelopes and frequency-specific CMC

Overall, EMG activity significantly increased compared to baseline in both perturbation directions (forward average EMG onset latency = 152 ms, SD = 37 ms; backward average EMG onset latency = 145 ms, SD = 30 ms; seen in Table 1), indicating that all leg muscles were actively engaged during the stepping responses. All muscles exhibited significant increases in CMC relative to baseline during the reactive step task, though the specific frequency band dynamics varied over time-locking events and muscles (forward average CMC onset latency = 186 ms, SD = 85 ms; backward average CMC onset latency = 171 ms, SD = 83 ms; seen in Table 1). In addition, the onset of significant CMC dynamics following perturbation onset lagged the transient increase in EMG activity. Note that the end of the perturbation onset time window may include some foot-off-related activity. The next sections separately describe for both perturbation directions whether a muscle functions as agonist or antagonist during the specific time locked events. In addition, the next section reports simultaneous muscle frequency specific CMC dynamics per time locked event. For an illustrative summary of the EMG and CMC data and observed significance values, please see the Supplementary Figures S1, S2.

3.3. Forward reactive stepping

Time-locked to perturbation onset, all muscles showed bilateral increase of EMG activity in the initial 150 ms following perturbation onset as steps have not yet been initiated (i.e., both step and stance leg showed similar increased activity; Figure 5). A similar significant increase of CMC was observed across different muscles and over different frequencies. In the theta range, CMC increase was observed in all muscles. Alpha band CMC dynamics were most abundant in both SO,TA,ST and RF muscles, though not significant for the step leg TA. In addition, we observed symmetrically increased beta coupling for all but RF muscles (i.e., similar CMC dynamics between step and stance leg). In the low-gamma frequency band, a gradually and symmetrically increased CMC was observable in the ST and BF muscles. In the high-gamma band CMC increased symmetrically but not significantly across muscles.

Time-locked to foot off, all muscles showed increased EMG activity relative to baseline. Prior to foot off, symmetric EMG activity was observed in both ST and BF muscles. Asymmetric activity was observed in SO (larger in stance leg), TA and RF (larger in stepping leg), consistent with their differential roles in the stance and stepping leg. Observed CMC dynamics did not follow similar patterns to EMG activity within time locked events. Theta dynamics were mainly increased for stance leg muscles and the lower leg muscles of the step leg. Alpha band CMC only increased bilaterally in the SO and ST muscles. In the beta frequency, increased CMC compared to baseline was observed in the step leg SO and ST. Increased CMC in the low-gamma frequency band is observed around the foot off event for all but the TA muscles. The high-gamma band dynamics are overall symmetrical for all muscles and mostly not significantly increased relative to baseline.

Time-locked to the foot strike event, all muscles showed elevated muscle activity relative to baseline. We observed greater EMG activity in step leg TA compared to the stance-leg TA muscle and the other stance leg muscles show increased EMG activity relative to the step-leg.

Although significant CMC dynamics are observed, these did not follow similar activity patterns from the EMG activity.

TABLE 1 Onset of significant EMG and CMC dynamics following perturbation onset relative to baseline. Presented data are in ms.

Forward muscles												
	SO		TA		ST		BF		RF			
	Step	Stance	Step	Stance	Step	Stance	Step	Stance	Step	Stance	M	SD
EMG	112	116	152	160	144	144	172	176	184	172	152	26
CMC												
Theta	160	180	160	180	160	180	220	200	280	180	190	37
Alpha	180	80		180	220	200	160		260	200	185	52
Beta	160	180	180		20					260	160	87
Low gamma	300				200		0	260	260		204	119
High gamma	0	160	180	220		300		240		240	191	96
								CMC onset			186	78
Backward muscles												
	SO		TA		ST		BF		RF			
	Step	Stance	Step	Stance	Step	Stance	Step	Stance	Step	Stance	M	SD
EMG	164	184	96	100	176	156	148	156	136	136	145	30
CMC												
Theta	180	180	160	160	220	180	240	160		180	184	30
Alpha						240				220	207	57
Beta		180									180	
Low gamma		80	200	200	340		0	140			163	118
High gamma	100	0	120	200			320	220		140	157	111
								CMC onset			178	79

Significant increase in theta CMC was observed prior to foot strike in the step-leg SO and following foot strike event in the stance-leg ST and BF. Alpha band CMC dynamics were limited to both SO muscles. In the beta frequency, step-leg SO and both ST leg muscles showed increased CMC prior to foot strike. In addition, all but the BF muscles showed asymmetrical beta CMC increase following the foot strike event. Low-gamma CMC dynamics were mostly increased symmetrically prior to foot strike in the posterior leg muscles. In addition, both low and high gamma showed asymmetric activation patterns for all except the TA muscles following foot strike.

3.4. Backward reactive stepping

In the backward perturbation direction, EMG activity of all muscles was increased relative to baseline, yet only modestly so in SO. This increased EMG activity is sustained throughout all time-locked events (Figure 6).

Time-locked to perturbation onset, EMG activity transiently and symmetrically increased for all muscles over the initial 200 ms. Similar transient and symmetrical CMC dynamics are observed in the theta range for all muscles. Only stance leg ST and RF showed significant Alpha CMC increase following perturbation onset. Beta CMC dynamics followed a corresponding transient and symmetrical pattern in the lower leg muscles which only resulted in a significant increase for the stance-leg SO. Gradual and modest increase of low-gamma dynamics are observed in posterior leg muscles, albeit not always reaching significance. High-gamma band dynamics overall show similar symmetrical transient cortical interaction patterns as the theta band in all muscles.

Time-locked to the foot off event, all muscles showed increased EMG activity relative to baseline. Prior to foot off, a symmetrical increase in muscle activity was observed in all lower leg muscles. However, upper leg muscles show greater EMG activity in the step leg hamstring and stance leg RF muscles, in line with their agonist roles in backward step initiation. Overall, CMC theta dynamics follow similar symmetrical patterns for the lower leg muscles in comparison to the EMG activity. Yet, dissimilar theta band CMC dynamics compared to EMG were observed in the upper leg muscles. Only increased alpha CMC was observed in the stance leg TA. Please note that the alpha CMC seems elevated for the RF throughout the foot off event while a significant effect remains absent. However, this is caused by strongly increased CMC in one participant specific for this muscle and frequency band. Removal of this participant's data did not influence the observation of additional significant difference effects. Little significant CMC dynamics were observed in the beta band. In the low-gamma band, almost all leg muscles, except for the RF, showed a gradual increase in CMC activity. In the high-gamma frequency band, all lower leg muscles show similar CMC patterns compared to the EMG activity.

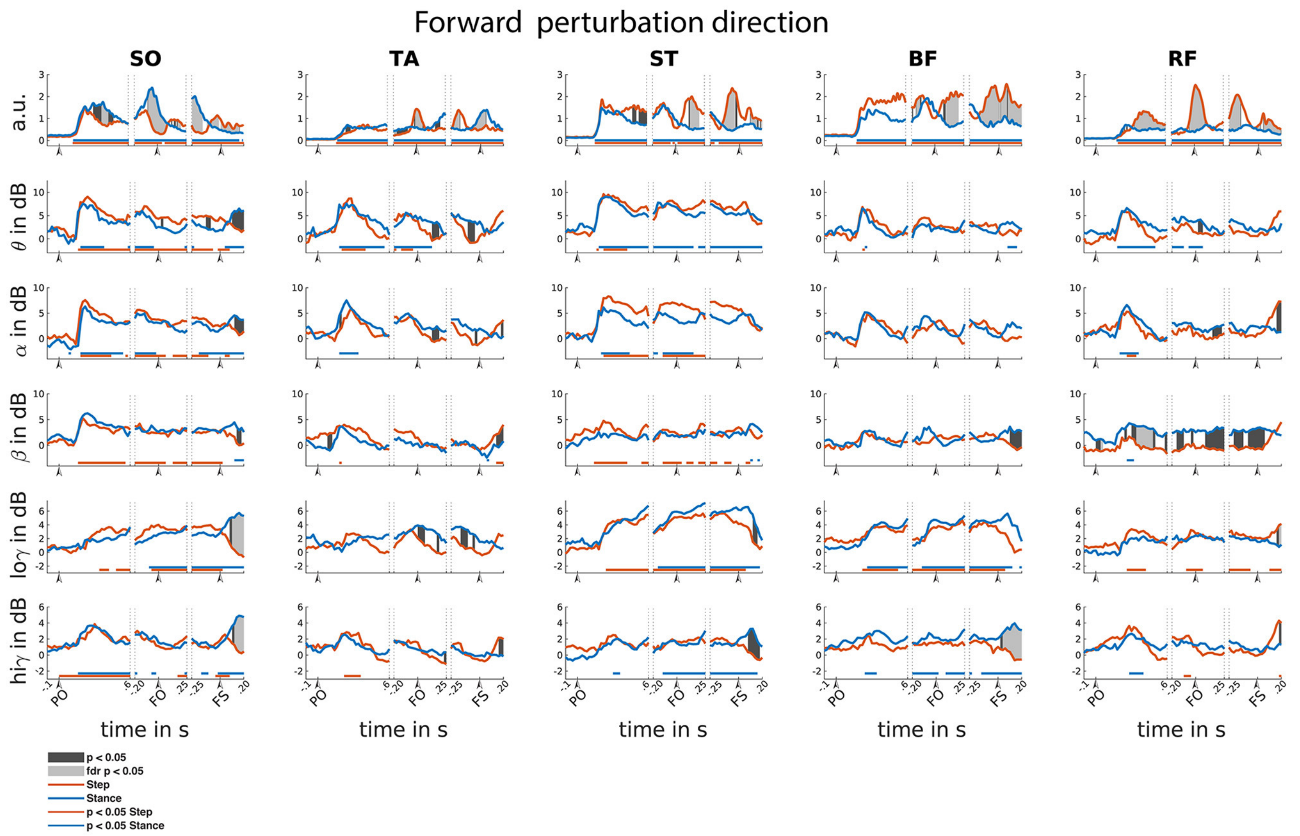

FIGURE 5
Forward reactive stepping muscle specific EMG and frequency specific CMC. Top row columns contain normalized EMG muscle activity, below are frequency-specific CMC dynamics. Figure columns are leg muscles Soleus (SO), Tibialis anterior (TA), Semitendinosus (ST), Biceps Femoris (BF), Rectus Femoris (RF). Statistically significant differences relative to baseline are indicated using horizontal lines, and differences between step and stance are indicated with grey shaded patches between the time courses. Note that there is temporal overlap between the different temporal windows of interest. Perturbation onset (PO), Foot off (FO), Foot Strike (FS). Foot off latency: Median = 451 ms, IQR = 210ms. Foot strike latency: median = 641 ms, IQR = 214 ms.

However, the upper leg muscles overall show little dynamics in comparison to the EMG activity during foot-off event with only slight high-gamma band CMC increases over stance-leg RF and BF muscles.

Time-locked to the foot strike event, all muscles show increased EMG activity and step leg muscles show greater muscle activation. In addition, all muscles showed CMC over multiple frequency bands, yet the observed asymmetrical EMG activation patterns are not mirrored in the CMC patterns in the various frequency bands. All stance leg muscles except the ST muscle show increased CMC in the theta frequency. In addition, the step leg ST and BF showed increased theta CMC following foot strike. Alpha band CMC dynamics were limited to the stance leg SO and the upper leg muscles that showed increased EMG activity prior to the foot strike event. CMC dynamics in the beta band were primarily observed following foot strike for the upper leg muscles that showed increased EMG activity prior to foot strike. Overall, relatively similar CMC dynamics were observed over both the low and high gamma bands.

3.5. Differences between step and stance leg

Overall, asymmetrical activation patterns in EMG and CMC between step and stance leg were observed time locked to foot off and foot strike events in both stepping directions. Following a brief symmetrical recruitment of the various muscles shortly after perturbation onset (consistent with an automatic postural response), the activation patterns of stance- and stepping leg muscles started to differ depending on their differential functional roles. Time locked to foot strike, all muscles show difference in CMC activity primarily over beta and gamma frequency bands. Only the RF muscles show similar asymmetrical activity over beta and gamma frequencies compared to the EMG activity following foot strike.

In the backward perturbation direction (Figure 6), the lower leg muscles showed significant EMG activity increases in the step leg during the foot strike time-locked phase of the step response. Whereas, in the upper leg muscles the posterior step leg muscles and stance leg anterior muscles showed consistent increases

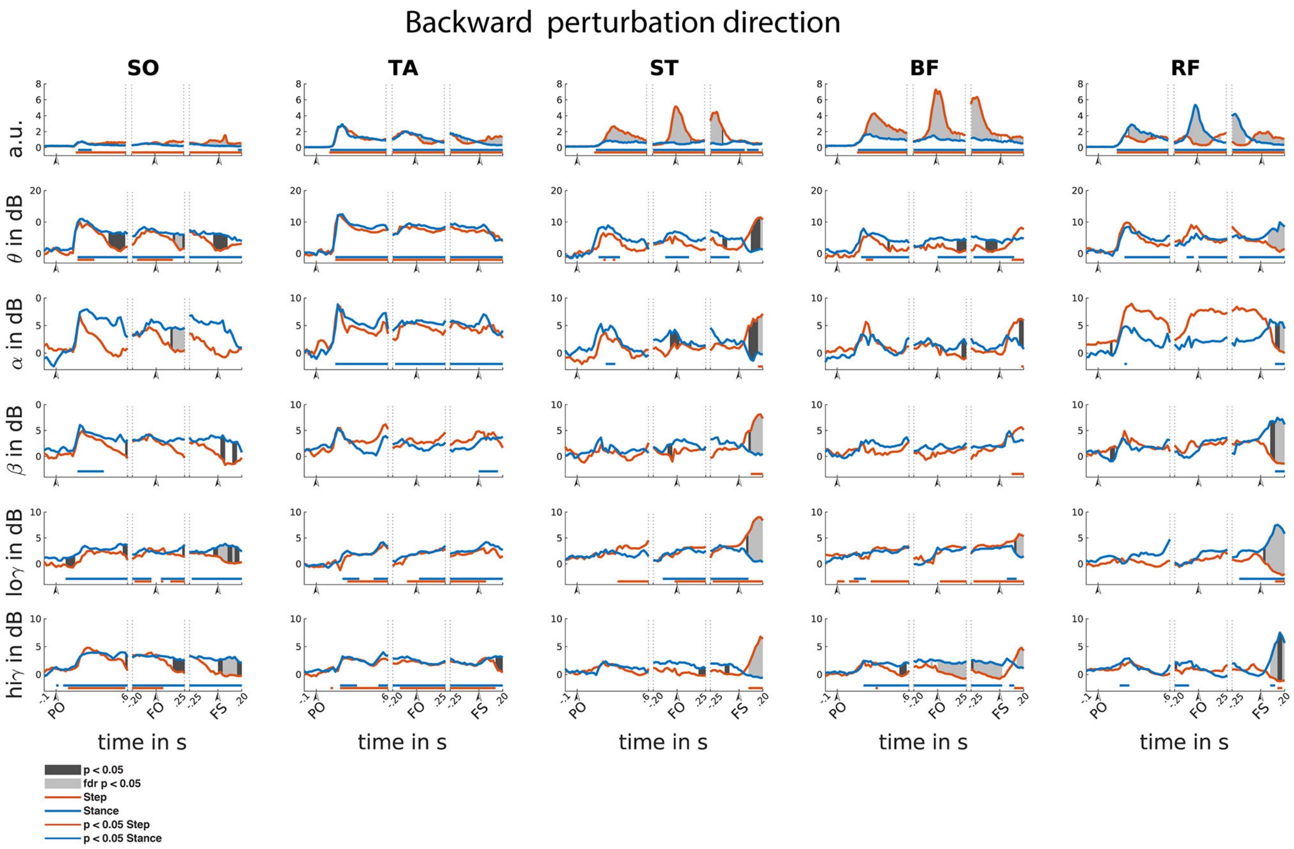

FIGURE 6
Backward reactive stepping muscle specific EMG and frequency specific CMC. Top row columns contain normalized EMG muscle activity, below are frequency band specific CMC dynamics. Figure columns are leg muscles Soleus (SO), Tibialis anterior (TA), Semitendinosus (ST), Biceps Femoris (BF), Rectus Femoris (RF). Differences relative to baseline are indicated using horizontal lines, and differences between step and stance are indicated with grey shaded patches between the time courses. Note that there is temporal overlap between the different time-locking events. Perturbation onset (PO), Foot off (FO), Foot Strike (FS). Foot off latency: median = 400 ms, IQR = 202 ms. Foot strike latency: median = 629 ms, IQR = 225 ms.

throughout all three time-locked events of the step response. Prior to foot strike time-locked event, EMG activity in anterior stance leg muscles was lower compared to the step leg EMG activity. Yet following foot strike, EMG activity in these anterior step leg muscles increases relative to the stance leg. Following foot strike, all but the TA muscle show greater EMG activity in the step leg. With respect to the CMC, all but the TA muscle showed distinct differences over all frequency bands following the foot strike event.

4. Discussion

Our aim was to identify cortical interactions with various leg muscles through spectral Granger causality during a reactive balance task. We hypothesized that CMC would occur during reactive stepping responses, indicating the contributions of the cerebral cortex to the execution of these responses. In line with our hypothesis, our results illustrated significant CMC increase relative to baseline over multiple muscles and frequency bands following balance perturbations in both directions. In addition, we expected CMC dynamics to differ between step and stance leg. In particular, we expected that stronger CMC would be most evident for the agonist muscles involved in generating the stepping movements, and that an increase in CMC would precede an upregulation of EMG activity in these muscles. Contrary to our expectations, our data illustrated that step and stance leg CMC generally did not show similarity with EMG data (i.e., increases in CMC did not align with increases in EMG activity). Our findings shed new light on cortical involvement of dynamic postural responses with implications for future studies involving clinical populations with deficient CMC (such as stroke).

4.1. Increase in CMC during reactive balance stepping

Granger causality analysis during reactive step responses revealed cortical interaction over multiple frequency bands for all muscles in both stepping directions, showing that on average the cortex becomes actively involved in the execution of reactive stepping response at ∼180 ms post perturbation. Overall, we observed significant CMC increases in the theta, beta and both gamma frequency bands relative to baseline for individual muscles in either perturbation direction and throughout the three time locked events. In the backward direction, notable broadband interaction was present in upper leg muscles following the foot strike event. Although such broadband CMC following foot strike may suggest that observed dynamics were caused by an artifact, increased broadband CMC in the stance leg muscles during forward stepping and the absence of similar broadband CMC in other step leg muscles (the leg that receives most movement impact that may cause artifacts) support the interpretation of a causal cortical interaction that spanned multiple canonical frequency bands. We propose that the broadband interaction with specific muscles may emphasize the importance of these respective muscles in maintaining a stable posture following the stepping response and we will elaborate on our arguments below. As multiple studies investigated distinct functional roles of cortical rhythms, we will separately discuss their potential roles during the reactive stepping response.

4.1.1. Theta band coupling

Strong transient cortico-muscular interactions in the theta frequency band were observed following perturbation onset in both perturbation directions for almost all leg muscles, indicating that theta dynamics may play a general role in the initial reactive stepping response. Interestingly, after the foot strike event in the backward stepping direction CMC in the theta frequency band mostly involved the muscles that showed increased muscle activity around foot off (i.e., stance-leg RF and step-leg BF), suggesting that the leg muscles that primarily showed increased EMG activation also require relatively more cortical interaction in the later phase of the response. Our findings are in agreement with an increase in CMC in the theta frequency band observed in lateral pull perturbations while standing, which was speculated to facilitate muscle recruitment of the feet-in-place balance response (17). Thus, the increase of CMC in the theta frequency band in the current study may emphasize the importance of the theta rhythm in muscle control following the perturbation onset and termination of the stepping response.

4.1.2. Alpha band coupling

Increased dynamics in the alpha band has been coupled to sensorimotor processes and motor readiness, and it may facilitate a similar role during the reactive balance response. Several studies reported increased alpha coupling during standing compared to walking (17, 31, 32). Although not all muscles showed increased CMC in the alpha band, most dynamics were primarily observed following perturbation onset and time locked to foot off event. These events require specific muscle activation to initially respond to the balance perturbation and initiate the stepping response. Therefore, our results may hint that the alpha frequency signals for muscle readiness in the reactive balance response.

4.1.3. Beta band coupling

The cortical beta frequency band is known for its close relation with voluntary muscle recruitment in a wide variety of tasks (33) and we reasoned that it may play a similar role during the muscle recruitment to facilitate the reactive stepping response. Although relatively few muscles showed significant beta coupling, beta CMC dynamics were most evident following perturbation onset and after foot strike. During these phases, muscle activity was elevated compared to baseline and yet relatively little changes in EMG dynamics occurred, suggesting a rather isometric (semi-static) muscle contraction. Previous studies investigated isometric leg muscle contractions, observing increased CMC in the beta frequency band (14, 34, 35), suggesting that beta dynamics play a role in leg muscle control. In addition, CMC in the beta band has been demonstrated during quiet standing postural control (36) and during feet-in-place balance responses following small lateral perturbations in unipedal stance (21). Interestingly, *increase* in beta band CMC is linked to increase in muscle activity, while a simultaneous power *decrease* of the cortical beta rhythm (note: cortical beta power should not be confused with cortical beta coupling) is observed during sustained muscle contraction (37). Yet, although a decrease in beta power has been reported following perturbation onset for either feet-in-place and step response (2, 12, 17, 22), no consistent increase in beta CMC dynamics was observed in the present study and that of Peterson (17). As beta CMC has mainly been observed during muscle activity in (semi-)static postures, we speculate that the general lack of significant beta CMC may be due to the dynamic nature of the reactive stepping response, with beta CMC becoming somewhat more evident when participants maintained a (semi-)static posture following foot strike. Therefore, we propose that the observed beta CMC in our study may relate to relatively isometric muscle recruitment facilitating stability in this phase of the reactive step response.

4.1.4. Gamma band coupling

Gamma coupling dynamics scale with increased muscle contraction and coordination (14, 38), and may facilitate a similar increased muscle recruiting role during reactive balance stepping. The cortical gamma rhythm covers a wide frequency range and although we analyzed two distinct frequency bands, cortical coupling in either band occurred mostly between foot off and foot strike. Therefore, we will discuss these results as general gamma dynamics [for an extensive review on gamma oscillations and CMC during the control of movements see Ulloa (38)]. Interestingly, we observed gamma rather than beta coupling throughout the reactive step response. Relative increase over CMC dynamics in the gamma band compared to beta band coupling has been linked to an increase in force and muscle coordination during isotonic compared to isometric muscle contraction of ankle and knee joints (14). Therefore, we speculate that the greater need for coordinated muscle recruitment during the step response may result in greater gamma CMC dynamics.

4.2. No evidence that lateralized EMG activity is driven by lateralized cortical coupling

Distinct cortical interactions with step or stance leg mainly occurred with respect to the foot off and foot strike events in either direction, suggesting that although the cortex was initially engaged following perturbation onset (as evidenced by the increased coupling relative to baseline; see Figures 5, 6), surprisingly little leg-specific interactions were observed under the hypothesis that transcortical loops are involved in facilitating the reactive stepping response (20, 39). Interestingly, distinct CMC between step and stance leg in both stepping directions were also not mirrored in similar asymmetric EMG activity, contrasting with our expectation that distinct cortical interaction would precede distinct increase in EMG activity. Previous studies did find a correlation between CMC and EMG (13, 15, 34), but these studies concerned voluntary movements and thus involved intentional top-down regulation of muscle activity. In contrast, the stepping responses in the present study were executed in response to an unexpected balance perturbation and were thus *reactive* in nature. We speculate that the difference between feedforward and feedback control may explain these discrepant findings regarding the relationships between CMC and EMG patterns. While the initial phase of the balance recovery response (i.e., automatic postural response) is known to be mediated by subcortical circuits (20), the present study may hint at these circuits also playing a greater-than-expected role during leg specific muscle activations of the stepping response.

4.3. Clinical implications

Our results demonstrate CMC throughout the reactive stepping response, indicating that impairments in any of the cortico-muscular communication circuitry may lead to dysfunctional cortico-muscular interaction underlying the impaired balance response. Indeed, impaired stepping responses have been reported for a variety of conditions of the central nervous system (e.g., stroke and Parkinson's disease (40, 41); which may in turn contribute to their elevated risk of falling (42, 43). Yet, the specific mechanisms underlying reactive stepping impairments are still poorly understood. Our work indicates that the application of Granger causality analysis for studying muscle and frequency specific deviations may help provide valuable insights in the underlying pathophysiology of impaired balance response in these clinical populations.

4.4. Limitations and strengths

The current study is exploratory and there are some limitations to consider. An important limitation is the sliding window approach used in the Granger causality analysis, meaning that observed effects are smeared over time. Yet, while peak activity is not systematically biased by the windowing, temporal comparison between EMG and CMC timeseries needs to be done with care, as events time locked to foot-off and foot strike contain a signal mixture related to other events due to the large sliding window and the heterogeneity of response latencies.

The experiment involved balance perturbations of different intensities resulting in stepping responses. As perturbation intensity is known to affect the amplitude of the perturbation evoked potential, the ERP contains the average of all perturbation intensities (i.e., low and high intensities and thus small and large amplitudes). While subtracting the ERP from the single trial data, we may have induced minor non-stationarities. Therefore, we recommend future studies to include perturbation intensities of a single intensity that is strong enough to elicit step responses.

On the other hand, our study shows novelty and strengths in several aspects. First of all, CMC at key events throughout the stepping response elicited temporal evolution of distinct spectral dynamics with respect to EMG. Future studies may further investigate the specific role of the spectral dynamics with respective muscles throughout the balance response. In specific, it is of interest to investigate the role of each frequency band with respect to the functional muscular role during the reactive step response. In addition, the current study is the first to conduct separate analysis of step- and stance-leg CMC in comparison to the temporal evolution of EMG activity. The surprising dissimilarity that we observed in CMC and EMG patterns between the legs indicates that the cortex does not appear to facilitate EMG recruitment for executing the balance recovery step in a refined leg- and muscle specific manner.

5. Conclusion

We conclude that reactive balance responses require direct interactions from the cortex with the individual muscles, yet without substantial leg-specific differences in CMC patterns. Our work is relevant for clinical populations with impaired balance control, where CMC analysis may elucidate the underlying pathophysiological mechanisms.

Data availability statement

The original contributions presented in the study are publicly available. This data can be found here: The Donders Repository (https://data.donders.ru.nl/) via di.dccn.DSC_4220000.06_843, 'EEG of human balance control'.

Ethics statement

The studies involving human participants were reviewed and approved by Research Ethics Committee of the Radboud University Medical Center (Nijmegen, The Netherlands; Dossier 2018-4970). The patients/participants provided their written informed consent to participate in this study.

Author contributions

MS: designed research, analyzed data, and wrote paper. TS-E, MC, and VW: supervised MS throughout the study and provided feedback. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fneur.2023.1124773/full#supplementary-material

References

1. Sipp AR, Gwin JT, Makeig S, Ferris DP. Loss of balance during balance beam walking elicits a multifocal theta band electrocortical response. *J Neurophysiol.* (2013) 110:2050–60. doi: 10.1152/jn.00744.2012

2. Solis-Escalante T, van der Cruijsen J, de Kam D, van Kordelaar J, Weerdesteyn V, Schouten AC. Cortical dynamics during preparation and execution of reactive balance responses with distinct postural demands. *Neuroimage.* (2019) 188:557–71. doi: 10.1016/j.neuroimage.2018.12.045

3. Varghese JP, McIlroy RE, Barnett-Cowan M. Perturbation-evoked potentials: Significance and application in balance control research. *Neurosci Biobehav Rev.* (2017) 83:267–80. doi: 10.1016/j.neubiorev.2017.10.022

4. Mansfield A, Inness EL, Wong JS, Fraser JE, McIlroy WE. Is impaired control of reactive stepping related to falls during inpatient stroke rehabilitation? *Neurorehabil Neural Repair.* (2013) 27:526–33. doi: 10.1177/1545968313478486

5. de Kam D, Geurts AC, Weerdesteyn V, Torres-Oviedo G. Direction-specific instability post-stroke is associated with deficient motor modules for balance control. *Neurorehabil Neural Repair.* (2018) 32:655–66. doi: 10.1177/1545968318783884

6. Dietz V, Quintern J, Berger W, Schenck E. Cerebral potentials and leg muscle e.m.g. responses associated with stance perturbation. *Exp Brain Res.* (1985) 57:348–54. doi: 10.1007/BF00236540

7. Slobounov S, Cao C, Jaiswal N, Newell KM. Neural basis of postural instability identified by VTC and EEG. *Exp Brain Res.* (2009) 199:1–16. doi: 10.1007/s00221-009-1956-5

8. Payne AM, Ting LH. Worse balance is associated with larger perturbation-evoked cortical responses in healthy young adults. *Gait Posture.* (2020) 80:324–30. doi: 10.1016/j.gaitpost.2020.06.018

9. Hülsdünker T, Mierau A, Strüder HK. Higher balance task demands are associated with an increase in individual alpha peak frequency. *Front Hum Neurosci.* (2015) 9:695. doi: 10.3389/fnhum.2015.00695

10. Varghese JP, Staines WR, McIlroy WE. Activity in functional cortical networks temporally associated with postural instability. *Neuroscience.* (2019) 401:43–58. doi: 10.1016/j.neuroscience.2019.01.008

11. Varghese JP, Marlin A, Beyer KB, Staines WR, Mochizuki G, McIlroy WE. Frequency characteristics of cortical activity associated with perturbations to upright stability. *Neurosci Lett.* (2014) 578:33–8. doi: 10.1016/j.neulet.2014.06.017

12. Solis-Escalante T, Stokkermans M, Cohen MX, Weerdesteyn V. Cortical responses to whole-body balance perturbations index perturbation magnitude and predict reactive stepping behavior. *Eur J Neurosci.* (2020) 54:8120–38. doi: 10.1111/ejn.14972

13. Glories D, Soulhol M, Amarantini D, Duclay J. Specific modulation of corticomuscular coherence during submaximal voluntary isometric, shortening and lengthening contractions. *Sci Rep.* (2021) 11:6322. doi: 10.1038/s41598-021-85851-w

14. Gwin JT, Ferris DP. Beta- and gamma-range human lower limb corticomuscular coherence. *Front Hum Neurosci.* (2012) 6:258. doi: 10.3389/fnhum.2012.00258

15. Yoshida T, Masani K, Zabjek K, Chen R, Popovic MR. Dynamic increase in corticomuscular coherence during bilateral, cyclical ankle movements. *Front Hum Neurosci.* (2017) 11:155. doi: 10.3389/fnhum.2017.00155

16. Artoni F, Fanciullacci C, Bertolucci F, Panarese A, Makeig S, Micera S, et al. Unidirectional brain to muscle connectivity reveals motor cortex control of leg muscles during stereotyped walking. *Neuroimage.* (2017) 159:403–16. doi: 10.1016/j.neuroimage.2017.07.013

17. Peterson SM, Ferris DP. Group-level cortical and muscular connectivity during perturbations to walking and standing balance. *Neuroimage.* (2019 S) 198:93–103. doi: 10.1016/j.neuroimage.2019.05.038

18. Roeder L, Boonstra TW, Kerr GK. Corticomuscular control of walking in older people and people with Parkinson's disease. *Sci Rep.* (2020) 10:2980. doi: 10.1038/s41598-020-59810-w

19. Taube W, Schubert M, Gruber M, Beck S, Faist M, Gollhofer A. Direct corticospinal pathways contribute to neuromuscular control of perturbed stance. *J Appl Physiol.* (2006) 101:420–9. doi: 10.1152/japplphysiol.01447.2005

20. Jacobs JV, Horak FB. Cortical control of postural responses. J *Neural Transm.* (2007) 114:1339–48. doi: 10.1007/s00702-007-0657-0

21. Zandvoort CS, van Dieën JH, Dominici N, Daffertshofer A. The human sensorimotor cortex fosters muscle synergies through cortico-synergy coherence. *Neuroimage.* (2019) 199:30–7. doi: 10.1016/j.neuroimage.2019.05.041

22. Stokkermans M, Solis-Escalante T, Cohen MX, Weerdesteyn V. Midfrontal theta dynamics index the monitoring of postural stability. *Cereb Cortex.* (2022). doi: 10.1093/cercor/bhac283

23. Nonnekes J, de Kam D, Geurts ACH, Weerdesteyn V, Bloem BR. Unraveling the mechanisms underlying postural instability in Parkinson's disease using dynamic posturography. *Expert Rev Neurother.* (2013) 13:1303–8. doi: 10.1586/14737175.2013.839231

24. Nonnekes J, Scotti A, Oude Nijhuis LB, Smulders K, Queralt A, Geurts ACH, et al. Are postural responses to backward and forward perturbations processed by different neural circuits? *Neuroscience.* (2013) 245:109–20. doi: 10.1016/j.neuroscience.2013.04.036

25. Oostenveld R, Praamstra P. The five percent electrode system for high-resolution EEG and ERP measurements. *Clin Neurophysiol.* (2001) 112:713–9. doi: 10.1016/S1388-2457(00)00527-7

26. Wang X, Chen Y, Ding M. Estimating Granger causality after stimulus onset: A cautionary note. *Neuroimage.* (2008) 41:767–76. doi: 10.1016/j.neuroimage.2008.03.025

27. Delorme A, Makeig S, EEGLAB. an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods.* (2004) 134:9–21. doi: 10.1016/j.jneumeth.2003.10.009

28. Barnett L, Seth AK. The MVGC multivariate Granger causality toolbox: a new approach to Granger-causal inference. *J Neurosci Methods.* (2014) 223:50–68. doi: 10.1016/j.jneumeth.2013.10.018

29. Barnett L, Seth AK. Granger causality for state-space models. *Phys Rev E Stat Nonlin Soft Matter Phys.* (2015) 91:040101. doi: 10.1103/PhysRevE.91.040101

30. Benjamini Y, Yekutieli D. The control of the false discovery rate in multiple testing under dependency. *Ann Statist.* (2001) 29:1165–88. doi: 10.1214/aos/1013699998

31. Lau TM, Gwin JT, Ferris DP. Walking reduces sensorimotor network connectivity compared to standing. *J Neuroeng Rehabil.* (2014) 11:14. doi: 10.1186/1743-0003-11-14

32. Wagner J, Solis-Escalante T, Neuper C, Scherer R, Müller-Putz G. Robot assisted walking affects the synchrony between premotor and somatosensory areas. *Biomed Tech.* (2013) 58(Suppl 1):4434. doi: 10.1515/bmt-2013-4434

33. Khanna P, Carmena JM. Neural oscillations: beta band activity across motor networks. *Curr Opin Neurobiol.* (2015) 32:60–7. doi: 10.1016/j.conb.2014.11.010

34. Kilner JM, Baker SN, Salenius S, Hari R, Lemon RN. Human cortical muscle coherence is directly related to specific motor parameters. *J Neurosci.* (2000) 20:8838–45. doi: 10.1523/JNEUROSCI.20-23-08838.2000

35. Dal Maso F, Longcamp M, Cremoux S, Amarantini D. Effect of training status on beta-range corticomuscular coherence in agonist vs. antagonist muscles during isometric knee contractions. *Exp Brain Res.* (2017) 235:3023–31. doi: 10.1007/s00221-017-5035-z

36. Jacobs JV, Wu G, Kelly KM. Evidence for beta corticomuscular coherence during human standing balance: Effects of stance width, vision, and support surface. *Neuroscience.* (2015) 298:1–11. doi: 10.1016/j.neuroscience.2015.04.009

37. Echeverria-Altuna I, Quinn AJ, Zokaei N, Woolrich MW, Nobre AC, van Ede F. Transient beta activity and cortico-muscular connectivity during sustained motor behaviour. *Prog Neurobiol.* (2022) 3:102281. doi: 10.1016/j.pneurobio.2022.102281

38. Ulloa JL. The control of movements via motor gamma oscillations. *Front Hum Neurosci.* (2021) 15:787157. doi: 10.3389/fnhum.2021.787157

39. Bolton DAE. The role of the cerebral cortex in postural responses to externally induced perturbations. *Neurosci Biobehav Rev.* (2015) 57:142–55. doi: 10.1016/j.neubiorev.2015.08.014

40. de Kam D, Nonnekes J, Oude Nijhuis LB, Geurts ACH, Bloem BR, Weerdesteyn V. Dopaminergic medication does not improve stepping responses following backward and forward balance perturbations in patients with Parkinson's disease. *J Neurol.* (2014) 261:2330–7. doi: 10.1007/s00415-014-7496-3

41. de Kam D, Roelofs JMB, Bruijnes AKBD, Geurts ACH, Weerdesteyn V. The next step in understanding impaired reactive balance control in people with stroke: the role of defective early automatic postural responses. *Neurorehabil Neural Repair.* (2017) 31:708–16. doi: 10.1177/1545968317718267

42. Mansfield A, Wong JS, McIlroy WE, Biasin L, Brunton K, Bayley M, et al. Do measures of reactive balance control predict falls in people with stroke returning to the community? *Physiotherapy.* (2015) 101:373–80. doi: 10.1016/j.physio.2015.01.009

43. Paul SS, Allen NE, Sherrington C, Heller G, Fung VSC, Close JCT, et al. Risk factors for frequent falls in people with Parkinson's disease. *J Parkinsons Dis.* (2014) 4:699–703. doi: 10.3233/JPD-140438

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Cerebello-cortical functional connectivity may regulate reactive balance control in older adults with mild cognitive impairment

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Background: Older adults with mild cognitive impairment (OAwMCI) experience a two-fold increased risk of falling compared to their cognitively intact counterparts. This increased risk could be attributed to impairments in balance control mechanisms (both volitional and reactive), however, the exact neural substrates contributing to the balance impairments remain unclear. While changes in functional connectivity (FC) networks in volitional balance control tasks have been well highlighted, the relationship between these changes and reactive balance control has not been examined. Therefore, this study aims to explore the relationship between FC networks of the brain obtained during resting state fMRI (no visualization or active task performed) and behavioral measures on a reactive balance task in OAwMCI.

Methods: Eleven OAwMCI ($<$ 25/30 on MoCA, $>$ 55 years) underwent fMRI and were exposed to slip-like perturbations on the Activestep treadmill. Postural stability, i.e., dynamic center of mass motion state (i.e., its position and velocity) was computed to determine reactive balance control performance. The relationship between reactive stability and FC networks was explored using the CONN software.

Results: OAwMCI with greater FC in default mode network-cerebellum ($r^2 = 0.43$, $p < 0.05$), and sensorimotor-cerebellum ($r^2 = 0.41$, $p < 0.05$) network exhibited lower reactive stability. Further, people with lower FC in middle frontal gyrus-cerebellum ($r^2 = 0.37$, $p < 0.05$), frontoparietal-cerebellum ($r^2 = 0.79$, $p < 0.05$) and cerebellar network-brainstem ($r^2 = 0.49$, $p < 0.05$) exhibited lower reactive stability.

Conclusion: Older adults with mild cognitive impairment demonstrate significant associations between reactive balance control and cortico-subcortical regions involved in cognitive-motor control. Results indicate that the cerebellum and its communications with higher cortical centers could be potential substrates contributing to impaired reactive responses in OAwMCI.

KEYWORDS

fall risk, functional connectivity, cognitive decline, reactive balance responses, higher cortical cognitive function, cerebello-cortical networks

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Introduction

Mild cognitive impairment (MCI) is a state of memory decline affecting about 3 to 42% of older adults above 65 years of age in the United States with at least 32% of them progressing to dementia within 5 years (1, 2). These older adults with mild cognitive impairment (OAwMCI) experience subtle balance control and gait deficits resulting in a two-fold increased risk of falls compared to their cognitively intact older adults (CIOA). Additionally, OAwMCI fallers are five times more likely to be admitted to the hospital compared to non-fallers. The consequences of such falls (like fractures, head injuries etc.) significantly affects their quality of life and independent functioning that increases financial and psychosocial burdens (3, 4). Considering the high incidence of falls among OAwMCI, a thorough comprehension of the underlying causative mechanisms of falls is imperative for designing effective fall prevention interventions.

There is enough evidence that OAwMCI exhibit significant alterations in structural (i.e., physical areas/tracts that connect brain/spinal cord regions) brain connectivity compared to CIOA (5–11). Such alterations are postulated to affect their physical function during activities of daily living like poor balance control and decreased walking function. Specifically, OAwMCI with lower gray matter volume in the temporal horn, frontal lobe, and caudate exhibited postural instability. People with medial temporal atrophy, reduced volume in hippocampus, middle frontal gyrus and superior frontal gyrus showed an increased fall risk. Furthermore, OAwMCI with increased number of white matter lesions in the frontal, parietal, and temporal cortices; thalamus, basal ganglia, and brainstem exhibited postural instability. People with reduced fractional anisotropy in corpus callosum, forceps minor, and left inferior fronto-occipital fasciculus had decreased gait speed when performed with a serial subtraction task. In the same study, reduced fractional anisotropy in the above-mentioned areas were also associated with increased number of falls (5–11).

Additionally, evidence shows disrupted functional connectivity (i.e., strength to which an activity between 2 or more cortical/subcortical signals correlates over time, FC) between networks that regulate motor and attentional demands in OAwMCI (12–14). Specifically, alterations in FC, within default mode network, frontoparietal network, supplementary motor area, dorsal attention network, sensorimotor network, executive control network, and salience network, have been established among OAwMCI (12–14). Studies demonstrate increased FC within default mode network during resting state in OAwMCI compared to healthy counterparts. The default mode network consists of medial prefrontal cortex, posterior cingulate cortex/precuneus, and inferior parietal lobe and the inability to deactivate this network during task performance is often associated with impaired cognitive function that could interfere with performance of goal-directed activities (15). A recent study showed that an increased default mode network activity at rest was associated with decreased dual task performance (simultaneous performance of motor and cognitive task) (12). Additionally, increased default mode network – supplementary motor area FC at rest was associated with decreased gait speed and increased postural sway (12). Similarly, OAwMCI fallers showed significantly lower default mode-sensorimotor network FC which was also associated with increased postural sway. Apart from this, studies show decreased FC within frontoparietal regions associated with decreased executive function (including motor planning and execution) and information processing (13, 14). While tasks in above-mentioned studies involve volitional activities, no study has examined the relationship of these networks with reactive balance control, activated to maintain or recover balance upon unexpected perturbations to the body.

Reactive balance responses are motor responses triggered upon an external perturbation to reestablish the relationship between the center of mass (COM) and base of support (BOS). These responses involve eliciting different strategies and further adjust the response based on the perturbation magnitude. Small magnitude perturbations elicit in-place strategies (using ankle or hip) in an attempt to restore the COM position within base of support. Large magnitude perturbations involve change in support strategies (i.e., stepping or reaching out) to reestablish larger BOS such that the displaced COM falls within it. Studies mainly use support surface movable platforms or motorized treadmills to deliver external perturbations for inducing balance loss in a safe environment. Pathologies related to neurocognitive disorders, such as MCI, when combined with age-related changes can significantly accelerate reactive balance control deficits (16, 17). For example, one study reported that performance on reactive balance control performance, measured using the BESTest, was significantly reduced among OAwMCI compared to CIOA (17). Our recent study demonstrated that upon exposure to stance forward-slip like perturbation, OAwMCI showed significantly decreased compensatory stepping responses compared to young adults and CIOA (16). This was demonstrated by OAwMCI having the lowest postural stability, shortest step length, slowest step initiation, and execution times (16). Further, they were unable to modulate (parametrize muscle and joint forces) their responses based on the perturbation magnitude (16). It should be noted that our study only examined and reported behavioral findings. Despite the high fall risk incidence among OAwMCI, studies have understudied the neural contributions to balance recovery that may be crucial for understanding the increased fall risk.

A neural control of compensatory stepping responses in reactive balance control is postulated to be triggered from subcortical balance control centers *via* the brainstem neural loop with cortical involvement only in the later phase of balance recovery *via* transcortical loop (18, 19). Higher cortical centers may play a critical role in the success of this response by contributing to modulate it based on prior experience and current task/environmental demands (20–22). Our recent findings indicate that structural brain integrity negatively influences reactive balance control such that OAwMCI with decreased fractional anisotropy in corticospinal, and frontopontine tracts showed reduced postural stability (23). Further, OAwMCI with reduced gray matter volume in brainstem and cerebellum exhibited reduced postural stability (23). Given such potential impact of the pathology associated structural brain changes in OAwMCI of reactive balance control, understanding how such changes affect the FC within/between cortico-subcortical regions may provide a better mechanistic understanding on the increased risk of falls in OAwMCI. Therefore, no hypotheses were formulated, and this study was entirely exploratory that examined the relationship between FC networks of the brain (i.e., between/within cortical and subcortical areas commonly associated with cognitive-motor functions) obtained during resting state fMRI while lying down in a scanner (no visualization or active task performed), and behavioral measures on a reactive balance control task in OAwMCI.

Methods

Participants

The study included eleven OAwMCI above the age of 55 years after obtaining a written informed consent. This study was approved by the University of Illinois at Chicago (UIC) institutional review board (Protocol #2018-1257). Participants were recruited by posting flyers at geriatric centers, nearby independent living senior centers, bus stops, train stations, and grocery stores.

Participants' eligibility

To be included, participants must receive a score between 18 and 24/30 on the Montreal Assessment on Cognitive Assessment (MoCA). Participants with uncontrolled cardiovascular disease, presence of any neurological condition (e.g., Parkinson's disease, Alzheimer's disease), and/or severe musculoskeletal diseases that may interfere with the ability to undergo balance control testing were excluded. People with the inability to walk independently for more than 10 meters (to verify ambulatory status among community dwelling older adults) and with heel bone density T-score of < -2.0 (measured using Lunar Achilles Insight) indicating risk of osteoporosis were also excluded for safety of participants during the reactive balance test.

Magnetic resonance imaging data acquisition

All imaging was acquired at the UIC Center for Magnetic Resonance Research using a 3 Tesla GE Discovery MR750 System (Milwaukee, WI) with a 32-channel head coil.

Anatomic MRI

High resolution 1 mm isotropic voxel resolution T1-weighted (T1w) images were obtained using a 3D axial FSPGR BRAVO imaging sequence with the following parameters: slice thickness = 1 mm, in-plane resolution = 1 mm, repetition time (TR) = 9.3 ms, echo time (TE) = 3.8 ms, inversion time (TI) = 450 ms, flip angle = 13°, field of view (FOV) = 220 × 220 mm. This was used as structural volumes for preprocessing.

Resting state functional MRI

T2 weighted images were obtained for determining functional whole-brain blood-oxygen-level dependent (BOLD) and optimized to reduced susceptibility artifacts with TR = 2000 ms, TE = 25 ms, flip angle = 82°, FOV = 220 × 220 mm, acquisition matrix 64 × 64, slice thickness = 3 mm, gap = 0 mm, 44 axial slices, 182 volumes per run. High resolution T1w structural scan was performed for anatomical localization. Participants were made to lie in the scanner and were asked to focus on an "X" with open eyes on the screen for 8 min without thinking anything. Resting state fMRI data preprocessing and analysis was conducted using CONN toolbox (24) which uses statistical parametric mapping software (SPM 12). Realignment was performed to correct for motion, corrected for errors in slice timing, outlier detection was subjected, and co-registered to the anatomical image. These images were spatially transformed to standard MNI space using transformation, resampled to 2-mm voxels, and smoothed with an 8 mm FWHM Gaussian kernel prior to analysis as detailed in Nieto-Castanon (2020).

Reactive balance control task

The Active step (Simbex, Lebanon, NH) motorized treadmill was used to induce a stance perturbation. The full-body kinematics were recorded *via* Cortex software using an eight-camera motion capture system (Motion Analysis, Corporation, Santa Rosa, CA) with a sampling rate of 120 Hz. A safety harness attached to an overhead metal arch prevented participants' knees to contact the belt surface in case of a fall. Twenty-nine Helen Hayes markers were placed bilaterally on bony landmarks to compute the center of mass (COM), and one marker was placed on the treadmill belt to identify the perturbation onset (i.e., sudden forward treadmill belt acceleration). Participants attained a comfortable stance position with their feet shoulder-width apart. They were instructed to execute a natural response to regain their balance by taking a step upon a sudden forward movement of the belt (slip-like perturbation, prevalent type of accidental falls). A familiarization trial was provided before the actual test and participants were unknown to perturbation onset (acceleration = 21.5 m/s^2, speed = 0.86 m/s, distance = 0.02m, duration = 40 ms). The reactive stability was computed using a custom-written algorithm in MATLAB version 2014b (The MathWorks Inc., Nactick, MA).

Reactive stability is calculated as the shortest distance of the relative COM state (i.e., its position and velocity) to the dynamic feasible theoretical boundary for backward loss of balance (25). The relative COM position was derived by expressing the absolute COM position relative to the rear edge of the base of support (BOS) normalized to each individual's foot length. Similarly, the COM velocity was expressed relative to the velocity of the rear BOS normalized by the factor $\sqrt{g} \times bh$, where g is the gravitational acceleration and bh is the individual's body height (Note × indicates multiplication) (26). Higher values indicate greater stability.

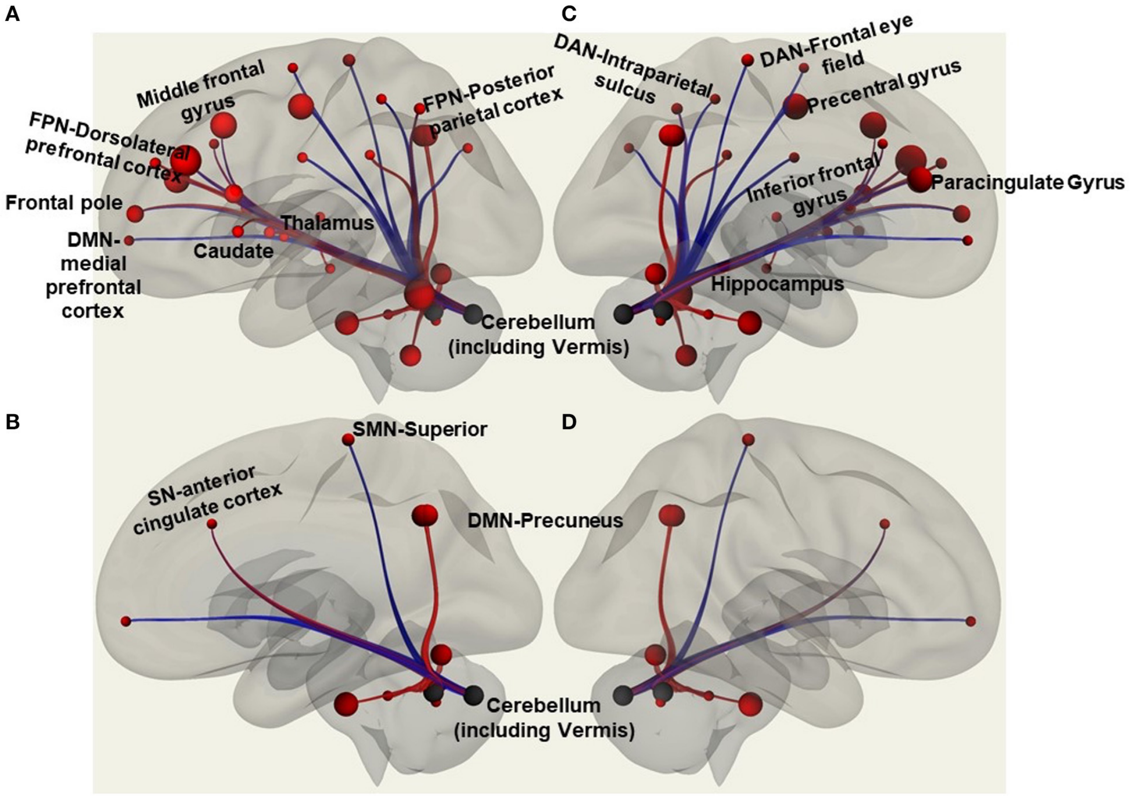

FIGURE 1
Regions of interest in the CONN software included functional connectivity analyses of atlas-atlas, altas-network, and network-network associations with red lines to indicate increased functional connectivity strength and blue lines to show decreased functional connectivity strength. The figure is a representation of some of those regions with **(A)** showing atlas regions involving frontal pole, middle frontal gyrus, caudate, thalamus, cerebellum; and network regions like frontoparietal network (FPN) that includes dorsolateral prefrontal cortex and posterior parietal cortex. **(B)** Shows atlas region involving the cerebellum; and anterior cingulate cortex of salience network (SN), superior of sensorimotor network (SMN), and precuneus of default mode network (DMN). **(C)** Shows inferior frontal gyrus, precentral gyrus, paracingulate gyrus, cerebellum and vermis, hippocampus; and frontal eye field, and intraparietal sulcus of dorsal attention network (DAN). **(D)** Shows precuneus of DMN and the cerebellum and vermis.

Statistical analyses

Regions of interest

Functional connectivity (FC) is the statistical relationships between cortical/subcortical signals over time that defines the strength to which activity between cortical/subcortical correlates. Atlas-Atlas (FSL Harvard-Oxford atlas cortical and subcortical areas), Network-Network (Yeo Parcellation) and Atlas-Network resting state FC were explored for brain regions associated with balance control (Figure 1) for the resting state fMRI data collected. The atlas areas included Frontal pole (cognitive function), middle frontal gyrus, Precentral gyrus (balance control), Inferior frontal gyrus (motor inhibition, attention and working memory cognitive function), Paracingulate gyrus (motor function), Precuneus (balance control), Caudate (cognitive function), Putamen (motor learning and control), Pallidum (cognitive and motor processing), hippocampus (memory), Cerebellum (balance control), Vermis (balance control), and Brainstem (balance control). The networks included default mode (medial prefrontal cortex, posterior cingulate cortex/precuneus, and inferior parietal lobe), sensorimotor (primary motor cortex, cingulate cortex, premotor cortex, and supplementary motor area), salience (anterior cingulate cortex, anterior insula, right rostral prefrontal cortex), dorsal attention (visual motion area, frontal eye fields, superior parietal lobule, intraparietal sulcus, ventral premotor cortex), frontoparietal (dorsolateral prefrontal cortex, posterior parietal cortex), and cerebellar.

Resting state functional connectivity associations with behavioral measures

Reactive stability was inputted as a covariate into the CONN software to run a regression analysis for examining the resting state FC within and between regions of interest. A priori significance threshold of p *False discovery rate* $= 0.2$ was set to correct for multiple comparisons and these values are reported in the results.

We performed planned bivariate Pearson's correlation of all resting state FC networks (default mode, sensorimotor, salience, dorsal attention, frontoparietal, and cerebellar) with cerebellum, vermis, and brainstem to determine their relationship with reactive stability. For this, we used individual z-scores from CONN software exported after first level analyses. Further, to validate the robustness of our results, we performed a multiple comparisons test – False

TABLE 1 Demographics and clinical characteristics of older adults with mild cognitive impairment (MCI).

	OAwMCI
Age [Means (SD)]	62.2 (5.9)
Range in years	56-74
Sex (M/F)	8/3
Arm Dominance (R/L)	11/0
Height (cm) [Means (SD)]	173.73 (8.95)
Range in cm	152.4–181.61
Weight (lbs) [Means (SD)]	162.62 (30.56)
Range in lbs	116–211
BBS Out of 56 [Means (SD)] Pre	54.18 (2.08)
Range	49–56
MoCA Out of 30 [Means (SD)]	21.63 (1.8)
Range	19–23
Reactive stability [Means (SD)]	−0.11 (0.39)
Range	−0.84–0.266

BBS, Berg Balance Scale; MoCA, Montreal Cognitive Assessment.

discovery rate (by comparing all the p-values) using the Benjamini and Hochberg (27) procedure.

Results

Demographics

A total of 11 participants included in the study analysis had a MoCA score ranging from 19 to 25 and their age, gender, height, and weight are provided in Table 1.

Effect of stability on resting state Atlas-Atlas functional connectivity

We observed a significant decrease in FC at resting state between frontal pole left and middle frontal gyrus ($\beta = -0.46$, $p = 0.02$), middle frontal gyrus right and caudate ($\beta = -0.23$, $p = 0.04$), middle frontal gyrus and putamen ($\beta = -0.24$, $p = 0.03$) (Figures 2, 3). Additionally, we observed a significant increase in FC between middle frontal gyrus and cerebellum ($\beta = 0.33$, $p = 0.04$), middle frontal gyrus and Vermis ($\beta = 0.38$, $p = 0.09$), cerebellum and vermis ($\beta = 0.48$, $p = 0.05$) (Figure 2).

Effect of stability on resting state network-network functional connectivity

We observed a significant decrease in FC between dorsal attention Frontal eye field left and dorsal attention intraparietal sulcus ($\beta = -0.25$, $p = 0.02$). Additionally, we observed a significant increase in FC between salience network and sensorimotor lateral network right ($\beta = 0.38$, $p = 0.02$) and left ($\beta = 0.47$, $p = 0.02$), dorsal attention Frontal eye field left and default mode lateral parietal ($\beta = 0.28$, $p = 0.06$).

Effect of stability on resting state atlas-network functional connectivity

We observed a significant decrease in FC between cerebellum and sensorimotor network ($\beta = -0.39$, $p = 0.2$), vermis and sensorimotor network ($\beta = -0.41$, $p = 0.006$), vermis and default mode network medial prefrontal cortex ($\beta = -0.29$, $p = 0.07$). Additionally, we observed that there was a significant increase in FC between cerebellum and frontoparietal network (posterior parietal cortex) ($\beta = 0.39$, $p = 0.04$), cerebellum and frontoparietal network (lateral prefrontal cortex) ($\beta = 0.37$, $p = 0.09$), cerebellum and cerebellar anterior network ($\beta = 0.44$, $p = 0.1$), cerebellum and default mode network (posterior cingulate cortex) ($\beta = 0.31$, $p = 0.07$), accumbens and frontoparietal network (lateral prefrontal cortex) ($\beta = 0.31$, $p = 0.13$), vermis and cerebellar posterior network ($\beta = 0.39$, $p = 0.18$) (Figure 3).

Planned associations between reactive stability and functional connectivity

Default mode network

A significant decrease in FC was observed between DMN-Cerebellum [r^2 (10) =0.52, $p = 0.01$], and DMN-Vermis [r^2 (10) =75, $p = 0.001$] such that people with higher reactive stability had lower FC between these regions (Figure 3A).

Sensorimotor network

A significant decrease in FC was observed between SMN and Cerebellum [r^2 (10) = 0.41, $p = 0.050$], SMN and Vermis [r^2 (10) = 0.57, $p = 0.007$] such that people with higher reactive stability had lower FC (Figure 3B).

Frontoparietal network

A significant increase in FC between FPN and cerebellum [r^2 (10) = 0.75, $p = 0.001$] was observed such that people with higher reactive stability had higher FC (Figure 3C).

Cerebellar network

A significant increase in FC between CN-Cerebellum [r^2 (10) = 0.78, $p < 0.001$], CN-Vermis [r^2 (10) = 0.61, $p = 0.004$], CN-Brainstem [r^2 (10) = 0.49, $p = 0.01$] (Figure 3D), was observed such that people with higher reactive stability had higher FC.

Multiple comparisons

False discovery rate for planned comparisons revealed that of the eight cases reported above, all of them were true for reactive balance control, thus validating the robustness of our findings.

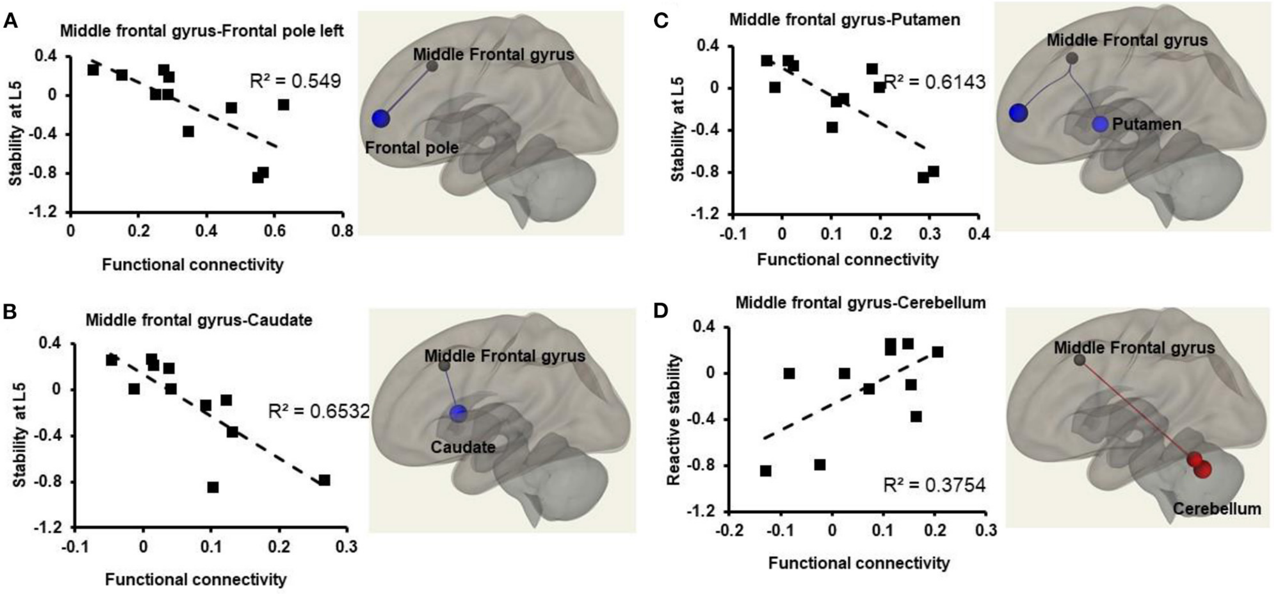

FIGURE 2
Relationship between atlas-atlas network (represented in z-scores on the x-axis) and reactive stability (represented on the y-axis) in older adults with mild cognitive impairment. Weakened connectivity in the brain is indicated via blue lines and increased functional connectivity with red lines. **(A)** Weakened functional connectivity between middle frontal gyrus and frontal pole left in the brain such that older adults with mild cognitive impairment with higher reactive stability exhibited lower functional connectivity strength during resting state. **(B)** Weakened functional connectivity between middle frontal gyrus and caudate in the brain such that older adults with mild cognitive impairment with higher reactive stability exhibited lower functional connectivity strength during resting state. **(C)** Weakened functional connectivity between middle frontal gyrus and putamen in the brain such that older adults with mild cognitive impairment with higher reactive stability exhibited lower functional connectivity strength during resting state. **(D)** Increased functional connectivity between middle frontal gyrus and cerebellum such that older adults with mild cognitive impairment with higher reactive stability exhibited higher functional connectivity strength during resting state.

Discussion

This pilot based preliminary exploratory study was conducted to understand the association of reactive stability with FC between/within cortical and subcortical areas commonly associated with cognitive-motor functions among OAwMCI. Our study provides preliminary findings that altered (increase or decrease) FC of cognitive-motor related cortico-subcortical regions (observed during resting state fMRI) may contribute to the reduced reactive stability predisposing OAwMCI to increased fall risk. These results are specific to OAwMCI and future studies involving CIOA for a comparative analysis may yield better understanding of the involvement of the cortico-subcortical regions involved in reactive balance control.

Atlas-atlas

Our results showed that the middle frontal gyrus located in the frontal lobe of the brain had weakened FC with caudate and putamen at rest such that OAwMCI with lower FC in these regions was associated with higher reactive stability. Based on MRI studies in young adults, activation of the middle frontal gyrus during tasks such as imagining or observing a slipping state, or observing a perturbed stance on a wobble, it is postulated that the middle frontal gyrus could be involved in planning and initiating movements when attention is reallocated to an unexpected stimulus (28–31). Caudate and putamen are parts of the basal ganglia, a key controller of motor sequence and modification of motor strategies and involved with higher-order cognitive aspects of motor control primarily regulating motor planning and execution (32–35). Specifically, during an external perturbation, it acts as a mediator between the motor cortex and brainstem *via* the basal ganglia-cortical loop (1 of 2 main central loops for postural response) for selecting a brainstem response during the initial state of balance loss (32–35). During an unpredicted balance loss, resources from the middle frontal gyrus and the basal ganglia could be recruited to relay and optimize pre-selected/triggered motor responses, i.e., compensatory stepping response. Therefore, it is possible that OAwMCI with higher FC in our study could not deactivate these areas at rest subsequently hindering the ability to trigger an appropriate response when the condition demands resources. Further, the ability to reallocate attentional resources (required for motor planning, step initiation, and execution to recover from balance loss), could have been inhibited among OAwMCI. It should be noted that this postulation is based on results yielded during a resting state condition that are subjected to change when a task-based fMRI is introduced. While task specific fMRI may be difficult to incorporate for reactive balance control, a mental imagery-based fMRI may provide a comprehensive understanding.

The middle frontal gyrus showed higher FC with the cerebellum, and the vermis (area that connects the 2 cerebellar

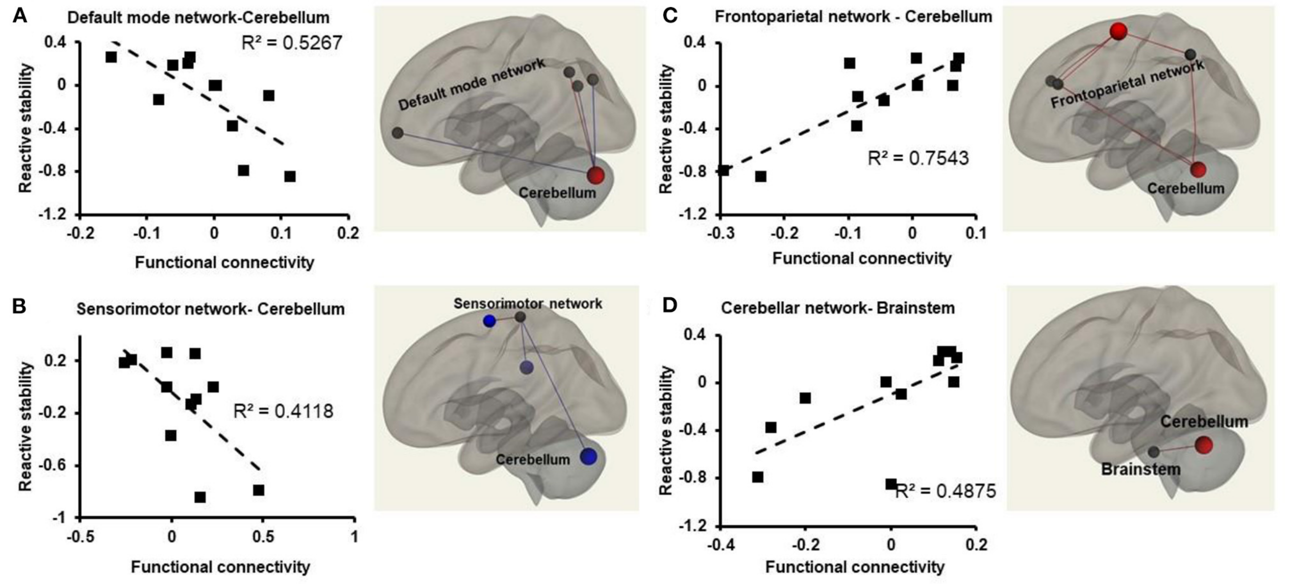

FIGURE 3
Relationships between atlas-network (represented in z-scores on the x-axis) and reactive stability (represented on the y-axis) in older adults with mild cognitive impairment. Weakened connectivity in the brain is indicated via blue lines and increased functional connectivity with red lines. **(A)** Weakened functional connectivity between default mode network and the cerebellum in the brain such that older adults with mild cognitive impairment with higher reactive stability exhibited lower functional connectivity strength during resting state; **(B)** weakened functional connectivity observed between the sensorimotor network and the cerebellum in the brain such that older adults with mild cognitive impairment with higher reactive stability exhibited lower functional connectivity strength during resting state; **(C)** increased functional connectivity strength observed between the frontoparietal network and the cerebellum in the brain such that older adults with mild cognitive impairment with higher reactive stability exhibited higher functional connectivity strength during resting state; **(D)** increased functional connectivity strength observed between the cerebellar network and the brainstem in the brain such that older adults with mild cognitive impairment with higher reactive stability exhibited higher functional connectivity strength during resting state.

hemispheres) such that people with higher FC exhibited higher reactive stability. It is postulated that the cerebellum could be involved in identifying movement error, online planning a stepping response based on the environment, and modulating motor responses to match the intended movement (28, 36–38). Such involvement relates to cerebellum interaction with the middle frontal gyrus *via* the cerebellar-cortical loop (2 of 2 main central loops for postural response) for processing sensory information to modulate motor programs based on prior experience. Another study has shown a reduced FC between the middle frontal gyrus and cerebellum during resting state in people with Alzheimer's disease compared to CIOA (39). It is possible that OAwMCI with higher FC were able to better process the sensory information relayed to the cerebellum and convey to middle frontal gyrus (supplementary motor area) for modulating the motor reactive response.

Network-network

The frontal eye field and intraparietal sulcus nodes of the dorsal attention network showed lower FC such that people with lower FC within this network exhibited higher reactive stability. Both the nodes are primarily recruited when there is a need for voluntary shift of spatial attention and flexibility to allocate attentional resources for effective cognitive processing (40–42). In our study, OAwMCI who could deactivate this network during resting state exhibited higher reactive stability. Contrastingly, a strong FC between the frontal eye field and the default mode network was observed such that OAwMCI with higher FC exhibited higher reactive stability. It is known that default mode network activation uses attentional resources required for one's introspection at rest or when not actively engaged in a task (43, 44). When intending to perform a task, the default mode network deactivates, freeing up resources for the task at hand. Previous studies have shown efficient working memory were associated with strong anticorrelation between dorsal attention network and default mode network (i.e., increased dorsal attention network and decreased default mode network FC) during resting state (45–47). However, OAwMCI and people with Alzheimer's disease showed the opposite (48) where there was a weak anticorrelation strength between these regions, i.e., decreased dorsal attention network and increased default mode network FC, which is in line with our results. Another study reported that executive function may be involved in motor planning for selecting the stepping strategy in response to an external perturbation (49). Therefore, the associations between dorsal attention network and default mode network observed in our study could suggest that cognitive state may influence reactive responses.

The results also showed a higher FC between the salience and sensorimotor networks such that OAwMCI with higher FC showed higher reactive stability. Regions of the salience network include anterior cingulate cortex, anterior insula and prefrontal cortex that is postulated to play a significant role in reactive

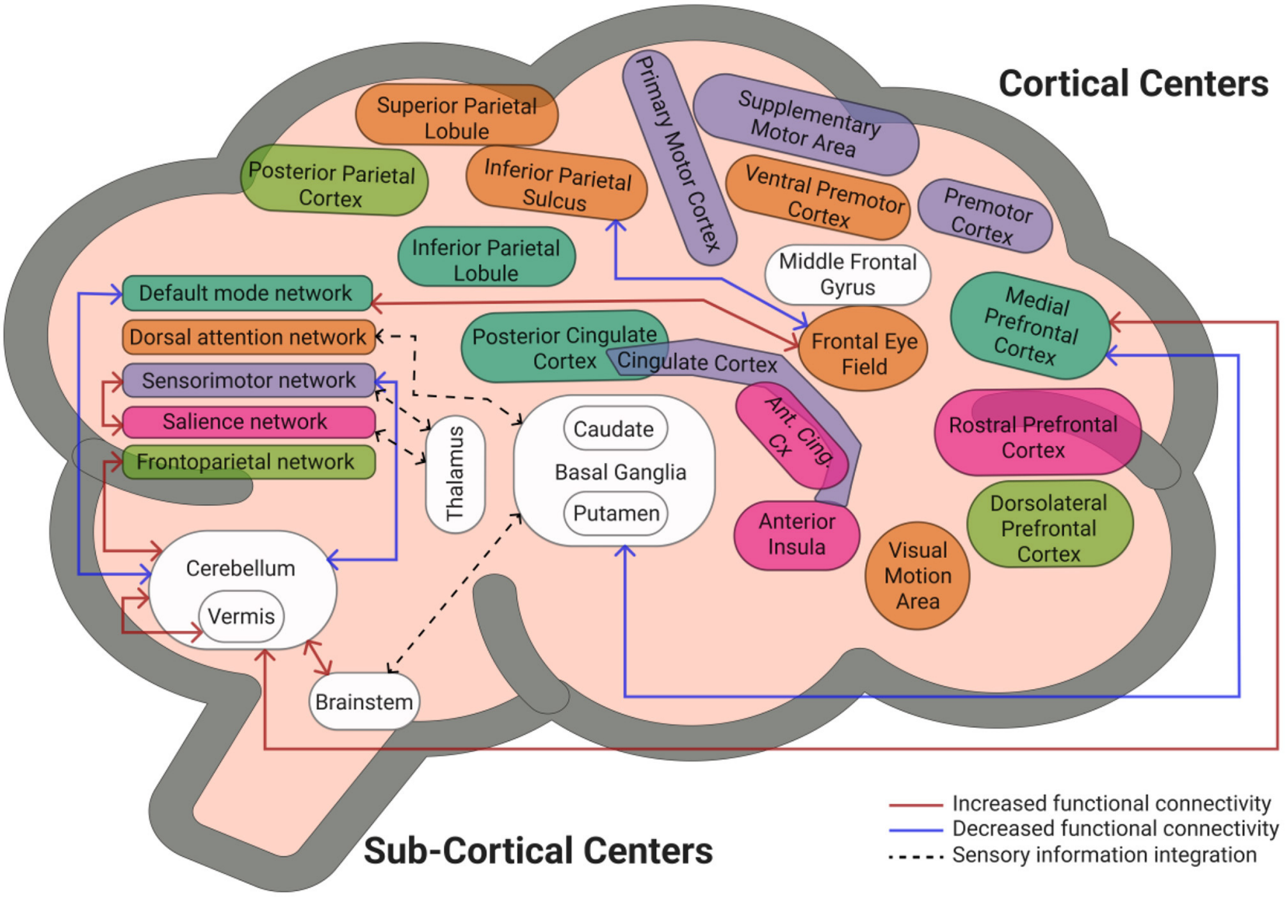

FIGURE 4
Summary model representing functional connectivity patterns across brain regions and that observed within/between the cerebellum and higher cortical networks/regions. The sensorimotor and salience network along with the brainstem communicate with the thalamus and basal ganglia to integrate sensory information perceived (denoted by black dotted lines). Our results indicate that the cerebellum could be the common center for relaying this sensory information. However, at a state of cognitive pathology like mild cognitive impairment causing uninterrupted functional activation/deactivation (denoted by red, increased functional connectivity strength; blue line, decreased/weakened functional connectivity), there is limited resources available for generating an "error signal" to even identify/perceive a balance loss and delay information processing.

balance control. Specifically, an earlier study showed that the anterior cingulate cortex and prefrontal cortex were activated during imagined slipping (28) and are thought to be involved in monitoring error during task performance and identifying balance loss. Additionally, the prefrontal cortex might help modulate balance recovery response based on the threat perceived in relation to the current body state (by anterior insula). On the other hand, the sensorimotor network comprises of primary motor cortex, cingulate cortex, premotor cortex, and supplementary motor area that are involved in modifying the ongoing movement. However, the relationship between salience network and sensorimotor network among OAwMCI compared to CIOA are not well known.

Atlas-network

Cerebellum and default mode network

In line with previous findings, our study demonstrated that OAwMCI had reduced FC between the cerebellum and default mode network at rest (50). Additionally, it was observed that OAwMCI with lower FC between the cerebellum (including the Vermis) and default mode network exhibited higher reactive stability. This is the first study to explore the relationship of reactive balance control with default mode network and cerebellum. The cerebellum consists of three functional areas namely the spinocerebellum (proprioceptive information), the cerebrocerebellum (movement planning), and the vestibulocerebellum (orientation of head in relation to the body). The spinocerebellar tract provides online proprioceptive information about body segments to the cerebrocerebellum. It is postulated that these areas are involved in generating an "error signal" between the expected and the actual postural status, which is then conveyed to the task-specific internal models (gait vs. stability regulation) to formulate and initiate the motor action – such as triggering the compensatory stepping response (20–22, 51). While these functions relate more toward spinocerebellum and cerebrocerebellum, the specific involvement of vestibulocerebellum in reactive responses is not much highlighted. Recent findings indicate that proprioception and vestibular systems are impaired in OAwMCI, however, the severity is less compared to people with Alzheimer's disease (52). Additionally, people with vestibular loss had 3 to 4 times increased likelihood of having cognitive impairment compared to healthy controls (53). However, participants involved in our study were not assessed for vestibular function to understand its contribution in reactive responses.

Cerebellum and sensorimotor network

The precentral gyrus (motor) and postcentral gyrus (proprioception) and supplementary motor area (complex motor planning) of the sensorimotor network showed lower FC with the cerebellum (including vermis). Regardless of the older adults' cognitive state, a previous study observed that fallers demonstrate a higher FC within the sensorimotor network (54). Such increased FC was attributed to the scaffolding theory of aging and cognition (55), where an adaptive compensatory mechanism is enabled to counteract the age-associated disruption in the sensorimotor networks so that existing motor and/or cognitive processes can still be functional (54). When such adaptive compensatory mechanisms are operating at their full capacity for maintaining all daily living functioning, they cannot be recruited for flight or fright responses that are encountered suddenly such as a perturbation, resulting in a balance loss or fall. In line with this, our previous study showed that OAwMCI exhibited significantly deteriorated ability to initiate a compensatory stepping response when an external perturbation was induced (16). Therefore, it could be that OAwMCI who suffer significant brain atrophy may not have the ability to counteract the disrupted networks to generate cortico-subcortical networks required for effective reactive responses. This could have resulted in lower FC in the cerebellum and sensorimotor network (54).

Cerebellum and frontoparietal network

Our study observed a higher FC between the cerebellum and frontoparietal network, that includes dorsolateral prefrontal cortex (regulates cognitive function like executive function) and posterior parietal cortex (spatial attention). Additionally, OAwMCI with higher FC between these regions exhibited higher reactive stability. It has been postulated that the cerebellum could be involved in identification of balance loss and recovery (20). In line with this, our recent findings showed that reduced gray matter volume to be associated with reduced reactive balance control against backward loss of balance in OAwMCI (23). It could be that the termination of motor pathways resides in parts of cerebellum making it a critical area responsible for relaying balance recovery information (23). Additionally, studies have shown its role in regulating executive function based on anatomical links with sensorimotor, prefrontal, and parietal cortices that serve higher cognitive functions (56–58). Specifically, the dorsolateral prefrontal cortex along with the cerebellum is shown to be coactivated under non-motor working memory tasks (59). Such coactivation increases as the load of the cognitive task increases, thus increasing information processing. Additionally, because of anatomical cerebellar-cortical links, cognitive function is facilitated by learning from previous experiences (*via* the cerebellum role in adaptation) to accurately predict the occurrence of an upcoming threat and updates the sensitivity to perceive the threat for better prediction in the future. Thus, in the occurrence of a balance threat, which could be like the type of threat already stored in the memory by the dorsolateral prefrontal cortex (28, 60), would be required to retrieve the previously learned motor memory for triggering a response. However, the activation of dorsolateral prefrontal cortex is reduced during memory retrieval in OAwMCI (61). In line with this, our study showed that people with lower FC within the cerebellum, and between the cerebellum and frontoparietal network exhibited lower reactive stability.

Overall, our results support the existing evidence on FC patterns within/between brain regions in OAwMCI with preliminary evidence that these FC pattern may negatively influence balance recovery responses against unexpected external perturbations. It can be postulated that the cerebellum could be the common center for relaying perturbation specific information (acceleration, displacement, and velocity) to the higher cortical centers (regions involved in frontoparietal, sensorimotor, and default mode networks). Therefore, it could be that people with relatively preserved FC patterns may deactivate or activate corresponding networks online subsequently allocating and recruiting attentional resources to trigger an appropriate and effective response (Figure 4). However, it can be speculated that when uninterrupted functional activation/deactivation is ongoing due to a cognitive pathology, there is limited resources available for generating an "error signal" to even identify/perceive a balance loss and delay information processing. Such delay incapacitates the ability to simultaneously retrieve the motor memory (if any stored) further delaying triggering a compensatory response against an external perturbation while the COM at this time may already have moved farther from the BOS resulting the individual experiencing a fall. While this is just a postulation specific to OAwMCI, future studies should focus on mental imagery-based brain imaging along with a control group to yield a profound understanding of FC pattern associated with reactive responses.

The results demonstrating an association of the strength of resting state functional brain networks with reactive stability during stance perturbations could provide preliminary understanding of brain regions involved in balance recovery. However, the study findings must be interpreted in the light of its limitations. This study was a preliminary exploratory study and hence had a sample size of $N = 11$. Higher sample size may yield better conclusive findings. It should also be noted that OAwMCI included in our study were not assessed for their fall-risk and the inferences made from the results of this study might not be disease specific and could be markers of general characteristics of people with increased risk of falling. Secondly, the study included only OAwMCI, and inclusion of a CIOA may help interpret the increased/decreased FC in specific regions to provide better understanding of FC associated with reactive balance control. Future studies that include a control group could also use these FC patterns as preliminary source of networks to advance the understanding of reactive balance control. Additionally, the current study was conducted during resting state and analyzing FC pattern during a reactive balance control mental imagery task-based fMRI may yield a comprehensive understanding of the neural networks involved during reactive balance control. Thirdly, the included OAwMCI were not assessed for vestibular function to rule out its contribution that could affect reactive stability during an external perturbation. Lastly, given the probable conversion from mild cognitive impairment to Alzheimer's disease, future studies must consider type of MCI as a subgroup or covariate to determine if they affect the associated FC patterns.

Conclusion

The results demonstrated statistically significant associations between measures of reactive measures and the cortico-subcortical regions contributing to cognitive and motor functions in OAwMCI. Specifically, higher reactive stability was associated with decreased FC in default mode and sensorimotor networks, and increased FC in cerebellar, and frontoparietal networks, thus, indicating higher fall risk. The study findings indicate that the cerebellum and its communications with higher cortical centers could be potential substrates contributing to impaired reactive responses in OAwMCI.

Data availability statement

Data sharing requests will be furnished with a data-sharing agreement approved by the University of Illinois at Chicago institutional review board that contains commitments to 1) Using the data for research purposes only (no commercial use of the data), 2) not attempting to re-identify any participant, 3) securing the data using appropriate technology, and 4) destroying or returning the data after analyses. Other stipulations may be added to the data-sharing agreement if deemed necessary. Data may be shared as a complete or partial dataset depending on the request. Requests to access the datasets should be directed to the corresponding author.

Ethics statement

The studies involving human participants were reviewed and approved by the Institutional Review Board at University of Illinois at Chicago. The patients/participants provided their written informed consent to participate in this study.

Author contributions

LK: conceptualization, formal analysis, investigation, writing—original draft, writing–review and editing, visualization, and project administration. TB: conceptualization, writing—original draft, writing—review and editing, supervision, resources, and funding acquisition. OA: formal analysis and writing—review and editing. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

References

1. Ward A, Tardiff S, Dye C, Arrighi HM. Rate of conversion from prodromal Alzheimer's disease to Alzheimer's dementia: a systematic review of the literature. *Dement Geriatr Cogn Dis Extra.* (2013) 3:320–32. doi: 10.1159/000354370

2. Owens DK, Davidson KW, Krist AH, Barry MJ, Cabana M, Caughey AB, et al. Screening for cognitive impairment in older adults: US Preventive Services Task Force recommendation statement. *JAMA.* (2020) 323:757–63. doi: 10.1001/jama.2020.0435

3. Delbaere K, Kochan NA, Close JC, Menant JC, Sturnieks DL, Brodaty H, et al. Mild cognitive impairment as a predictor of falls in community-dwelling older people. *Am. J. Geriatri. Psychiatr.* (2012) 20:845–53. doi: 10.1097/JGP.0b013e31824afbc4

4. Lindbergh CA, Dishman RK, Miller LS. Functional disability in mild cognitive impairment: a systematic review and meta-analysis. *Neuropsychol Rev.* (2016) 26:129–59. doi: 10.1007/s11065-016-9321-5

5. Snir JA, Bartha R, Montero-Odasso M. White matter integrity is associated with gait impairment and falls in mild cognitive impairment. *Results Gait Brain Study NeuroImage Cli.* (2019) 24:101975. doi: 10.1016/j.nicl.2019.101975

6. Ogama N, Sakurai T, Shimizu A, Toba K. Regional white matter lesions predict falls in patients with amnestic mild cognitive impairment and Alzheimer's disease. *J Am Med Dir Assoc.* (2014) 15:36–41. doi: 10.1016/j.jamda.2013.11.004

7. Makizako H, Shimada H, Doi T, Park H, Yoshida D, Uemura K, et al. Poor balance and lower gray matter volume predict falls in older adults with mild cognitive impairment. *BMC Neurol.* (2013) 13:1–8. doi: 10.1186/1471-2377-13-102

8. Kose Y, Ikenaga M, Yamada Y, Morimura K, Takeda N, Ouma S, et al. Timed Up and Go test, atrophy of medial temporal areas and cognitive functions in community-dwelling older adults with normal cognition and mild cognitive impairment. *Exp Gerontol.* (2016) 85:81–7. doi: 10.1016/j.exger.2016.09.019

9. Kido T, Tabara Y, Igase M, Ochi N, Uetani E, Nagai T, et al. Postural instability is associated with brain atrophy and cognitive impairment in the elderly: the J-SHIPP study. *Dement Geriatr Cogn Disord.* (2010) 29:379–87. doi: 10.1159/000255106

10. Brueggen K, Dyrba M, Cardenas-Blanco A, Schneider A, Fliessbach K, Buerger K, et al. Structural integrity in subjective cognitive decline, mild cognitive impairment and Alzheimer's disease based on multicenter diffusion

tensor imaging. *J Neurol.* (2019) 266:2465–74. doi: 10.1007/s00415-019-09429-3

11. Allali G, Annweiler C, Predovan D, Bherer L, Beauchet O. Brain volume changes in gait control in patients with mild cognitive impairment compared to cognitively healthy individuals; GAIT study results. *Exp Gerontol.* (2016) 76:72–9. doi: 10.1016/j.exger.2015.12.007

12. Crockett RA, Hsu CL, Best JR, Liu-Ambrose T. Resting state default mode network connectivity, dual task performance, gait speed, and postural sway in older adults with mild cognitive impairment. *Front Aging Neurosci.* (2017) 9:423. doi: 10.3389/fnagi.2017.00423

13. Mao Y, Liao Z, Liu X, Li T, Hu J, Le D, et al. Disrupted balance of long and short-range functional connectivity density in Alzheimer's disease (AD) and mild cognitive impairment (MCI) patients: a resting-state fMRI study. *Annal Transl Med.* (2021) 9:7019. doi: 10.21037/atm-20-7019

14. Xue J, Guo H, Gao Y, Wang X, Cui H, Chen Z, et al. Altered directed functional connectivity of the hippocampus in mild cognitive impairment and Alzheimer's disease: a resting-state fMRI study. *Front Aging Neurosci.* (2019) 11:326. doi: 10.3389/fnagi.2019.00326

15. Eichele T, Debener S, Calhoun VD, Specht K, Engel AK, Hugdahl K, et al. Prediction of human errors by maladaptive changes in event-related brain networks. *Proc Nat Acad Sci.* (2008) 105:6173–8. doi: 10.1073/pnas.0708965105

16. Kannan LN, Bhatt TS. Perturbation-based balance assessment: Examining reactive balance control in older adults with mild cognitive impairments. *Physiol. Int.* (2021) 108:353–70. doi: 10.1556/2060.2021.00181

17. Tangen GG, Engedal K, Bergland A, Moger TA, Mengshoel AM. Relationships between balance and cognition in patients with subjective cognitive impairment, mild cognitive impairment, and Alzheimer disease. *Phys Ther.* (2014) 94:1123–34. doi: 10.2522/ptj.20130298

18. Magnus R. Some results of studies in the physiology of posture. *Lancet.* (1926) 2:531–6. doi: 10.1016/S0140-6736(01)27826-X

19. Sherrington CS. Flexion-reflex of the limb, crossed extension-reflex, and reflex stepping and standing. *J Physiol.* (1910) 40:28–121. doi: 10.1113/jphysiol.1910.sp001362

20. Jacobs J, Horak F. Cortical control of postural responses. *J Neural Transm.* (2007) 114:1339–48. doi: 10.1007/s00702-007-0657-0

21. Payne AM, Ting LH. Worse balance is associated with larger perturbation-evoked cortical responses in healthy young adults. *Gait Posture.* (2020) 80:324–30. doi: 10.1016/j.gaitpost.2020.06.018

22. Solis-Escalante T, Stokkermans M, Cohen MX, Weerdesteyn V. Cortical responses to whole-body balance perturbations index perturbation magnitude and predict reactive stepping behavior. *Eur J Neurosci.* (2020) 52:8120–32. doi: 10.1111/ejn.14972

23. Kannan L, Bhatt T, Zhang A, Ajilore O. Association of balance control mechanisms with brain structural integrity in older adults with mild cognitive impairment. *Neurosci Lett.* (2022) 783:136699. doi: 10.1016/j.neulet.2022.136699

24. Whitfield-Gabrieli S, Nieto-Castanon A. Conn: a functional connectivity toolbox for correlated and anticorrelated brain networks. *Brain Connect.* (2012) 2:125–41. doi: 10.1089/brain.2012.0073

25. Bhatt T, Pai Y-C. Long-term retention of gait stability improvements. *J Neurophysiol.* (2005) 94:1971–9. doi: 10.1152/jn.00266.2005

26. McMahon TA. *Muscles, Reflexes, and Locomotion.* Princeton, NJ: Princeton University Press (1984).

27. Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J Royal Stat Soc.* (1995) 57:289–300. doi: 10.1111/j.2517-6161.1995.tb02031.x

28. Bhatt T, Patel P, Dusane S, DelDonno SR, Langenecker SA. Neural mechanisms involved in mental imagery of slip-perturbation while walking: a preliminary fMRI study. *Front Behav Neurosci.* (2018) 12:203. doi: 10.3389/fnbeh.2018.00203

29. Doricchi F, Macci E, Silvetti M, Macaluso E. Neural correlates of the spatial and expectancy components of endogenous and stimulus-driven orienting of attention in the Posner task. *Cerebral Cortex.* (2010) 20:1574–85. doi: 10.1093/cercor/bhp215

30. Shulman GL, Astafiev SV, Franke D, Pope DL, Snyder AZ, McAvoy MP, et al. Interaction of stimulus-driven reorienting and expectation in ventral and dorsal frontoparietal and basal ganglia-cortical networks. *J Neurosci.* (2009) 29:4392–407. doi: 10.1523/JNEUROSCI.5609-08.2009

31. Taube W, Mouthon M, Leukel C, Hoogewoud H-M, Annoni J-M, Keller M. Brain activity during observation and motor imagery of different balance tasks: an fMRI study. *Cortex.* (2015) 64:102–14. doi: 10.1016/j.cortex.2014.09.022

32. Leisman G, Braun-Benjamin O, Melillo R. Cognitive-motor interactions of the basal ganglia in development. *Front Syst Neurosci.* (2014) 8:16. doi: 10.3389/fnsys.2014.00016

33. Lehéricy S, Bardinet E, Tremblay L, Van de Moortele P-F, Pochon J-B, Dormont D, et al. Motor control in basal ganglia circuits using fMRI and brain atlas approaches. *Cerebral Cortex.* (2006) 16:149–61. doi: 10.1093/cercor/bhi089

34. Jueptner M, Weiller C, A. review of differences between basal ganglia and cerebellar control of movements as revealed by functional imaging studies. *J Neurol.* (1998) 121:1437–49. doi: 10.1093/brain/121.8.1437

35. Boecker H, Dagher A, Ceballos-Baumann A, Passingham R, Samuel M, Friston K, et al. Role of the human rostral supplementary motor area and the basal ganglia in motor sequence control: investigations with H2 15O PET. *J Neurophysiol.* (1998) 79:1070–80. doi: 10.1152/jn.1998.79.2.1070

36. Bakker M, Allum JH, Visser JE, Grüneberg C, van de Warrenburg BP, Kremer BH, et al. Postural responses to multidirectional stance perturbations in cerebellar ataxia. *Exp Neurol.* (2006) 202:21–35. doi: 10.1016/j.expneurol.2006.05.008

37. Horstmann G, Dietz V. The contribution of vestibular input to the stabilization of human posture: a new experimental approach. *Neurosci Lett.* (1988) 95:179–84. doi: 10.1016/0304-3940(88)90653-2

38. Pisotta I, Molinari M. Cerebellar contribution to feedforward control of locomotion. *Front Hum Neurosci.* (2014) 8:475. doi: 10.3389/fnhum.2014.00475

39. Tang F, Zhu D, Ma W, Yao Q, Li Q, Shi J. Differences changes in cerebellar functional connectivity between mild cognitive impairment and Alzheimer's disease: a seed-based approach. *Front Neurol.* (2021) 12:987. doi: 10.3389/fneur.2021.645171

40. Corbetta M, Kincade JM, Ollinger JM, McAvoy MP, Shulman GL. Voluntary orienting is dissociated from target detection in human posterior parietal cortex. *Nat Neurosci.* (2000) 3:292–7. doi: 10.1038/73009

41. Kelly AC, Uddin LQ, Biswal BB, Castellanos FX, Milham MP. Competition between functional brain networks mediates behavioral variability. *Neuroimage.* (2008) 39:527–37. doi: 10.1016/j.neuroimage.2007.08.008

42. De Pisapia N, Turatto M, Lin P, Jovicich J, Caramazza A. Unconscious priming instructions modulate activity in default and executive networks of the human brain. *Cereb Cortex.* (2012) 22:639–49. doi: 10.1093/cercor/bhr146

43. Sonuga-Barke EJ, Castellanos FX. Spontaneous attentional fluctuations in impaired states and pathological conditions: a neurobiological hypothesis. *Neurosci Biobehav Rev.* (2007) 31:977–86. doi: 10.1016/j.neubiorev.2007.02.005

44. Whitfield-Gabrieli S, Ford JM. Default mode network activity and connectivity in psychopathology. *Annu Rev Clin Psychol.* (2012) 8:49–76. doi: 10.1146/annurev-clinpsy-032511-143049

45. Hampson M, Driesen N, Roth JK, Gore JC, Constable RT. Functional connectivity between task-positive and task-negative brain areas and its relation to working memory performance. *Magn Reson Imaging.* (2010) 28:1051–7. doi: 10.1016/j.mri.2010.03.021

46. Keller JB, Hedden T, Thompson TW, Anteraper SA, Gabrieli JD, Whitfield-Gabrieli S. Resting-state anticorrelations between medial and lateral prefrontal cortex: association with working memory, aging, and individual differences. *Cortex.* (2015) 64:271–80. doi: 10.1016/j.cortex.2014.12.001

47. Sala-Llonch R, Pena-Gomez C, Arenaza-Urquijo EM, Vidal-Piñeiro D, Bargallo N, Junque C, et al. Brain connectivity during resting state and subsequent working memory task predicts behavioural performance. *Cortex.* (2012) 48:1187–96. doi: 10.1016/j.cortex.2011.07.006

48. Wang J, Liu J, Wang Z, Sun P, Li K, Liang P. Dysfunctional interactions between the default mode network and the dorsal attention network in subtypes of amnestic mild cognitive impairment. *Aging.* (2019) 11:9147. doi: 10.18632/aging.102380

49. Sturnieks DL, Menant J, Vanrenterghem J, Delbaere K, Fitzpatrick RC, Lord SR. Sensorimotor and neuropsychological correlates of force perturbations that induce stepping in older adults. *Gait Posture.* (2012) 36:356–60. doi: 10.1016/j.gaitpost.2012.03.007

50. Eyler LT, Elman JA, Hatton SN, Gough S, Mischel AK, Hagler DJ, et al. Resting state abnormalities of the default mode network in mild cognitive impairment: a systematic review and meta-analysis. *J Alzh Dis.* (2019) 70:107–20. doi: 10.3233/JAD-180847

51. Solis-Escalante T, van der Cruijsen J, de Kam D, van Kordelaar J, Weerdesteyn V, Schouten AC. Cortical dynamics during preparation and execution of reactive balance responses with distinct postural demands. *Neuroimage.* (2019) 188:557–71. doi: 10.1016/j.neuroimage.2018.12.045

52. Ide R, Ota M, Hada Y, Watanabe S, Takahashi T, Tamura M, et al. Dynamic balance deficit and the neural network in Alzheimer's disease and mild cognitive impairment. *Gait Posture.* (2022) 93:252–8. doi: 10.1016/j.gaitpost.2022.01.018

53. Wei EX, Oh ES, Harun A, Ehrenburg M, Xue Q-L, Simonsick E, et al. Increased prevalence of vestibular loss in mild cognitive impairment and Alzheimer's disease. *Curr Alzheimer Res.* (2019) 16:1143–50. doi: 10.2174/1567205016666190816114838

54. Maidan I, Droby A, Jacob Y, Giladi N, Hausdorff JM, Mirelman A. The neural correlates of falls: Alterations in large-scale resting-state networks in elderly fallers. *Gait Posture.* (2020) 80:56–61. doi: 10.1016/j.gaitpost.2020.05.023

55. Park DC, Reuter-Lorenz P. The adaptive brain: aging and neurocognitive scaffolding. *Annu Rev Psychol.* (2009) 60:173–96. doi: 10.1146/annurev.psych.59.103006.093656

56. Schmahmann JD. The cerebellum and cognition. *Neurosci Lett.* (2019) 688:62–75. doi: 10.1016/j.neulet.2018.07.005

57. Kim HJ. Cheong En, Jo S, Lee S, Shim WH, Kwon M, et al. The cerebellum could serve as a potential imaging biomarker of dementia conversion in patients with amyloid-negative amnestic mild cognitive impairment. *Eur Jo Neurol.* (2021) 28:1520–7. doi: 10.1111/ene.14770

58. Stoodley CJ. The cerebellum and cognition: evidence from functional imaging studies. *Cerebellum.* (2012) 11:352–65. doi: 10.1007/s12311-011-0260-7

59. Brissenden JA, Somers DC. Cortico–cerebellar networks for visual attention and working memory. *Curr Opin Psychol.* (2019) 29:239–47. doi: 10.1016/j.copsyc.2019.05.003

60. Gold AL, Morey RA, McCarthy G. Amygdala–prefrontal cortex functional connectivity during threat-induced anxiety and goal distraction. *Biol Psychiatry.* (2015) 77:394–403. doi: 10.1016/j.biopsych.2014.03.030

61. Uemura K, Shimada H, Doi T, Makizako H, Tsutsumimoto K, Park H, et al. Reduced prefrontal oxygenation in mild cognitive impairment during memory retrieval. *Int J Geriatr Psychiatry.* (2016) 31:583–91. doi: 10.1002/gps.4363

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Asymmetry measures for quantification of mechanisms contributing to dynamic stability during stepping-in-place gait

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The goal of this study is to introduce and to motivate the use of new quantitative methods to improve our understanding of mechanisms that contribute to the control of dynamic balance during gait. Dynamic balance refers to the ability to maintain a continuous, oscillating center-of-mass (CoM) motion of the body during gait even though the CoM frequently moves outside of the base of support. We focus on dynamic balance control in the frontal plane or medial–lateral (ML) direction because it is known that active, neurally-mediated control mechanisms are necessary to maintain ML stability. Mechanisms that regulate foot placement on each step and that generate corrective ankle torque during the stance phase of gait are both known to contribute to the generation of corrective actions that contribute to ML stability. Less appreciated is the potential role played by adjustments in step timing when the duration of the stance and/or swing phases of gait can be shortened or lengthened to allow torque due to gravity to act on the body CoM over a shorter or longer time to generate corrective actions. We introduce and define four asymmetry measures that provide normalized indications of the contribution of these different mechanisms to gait stability. These measures are 'step width asymmetry', 'ankle torque asymmetry', 'stance duration asymmetry', and 'swing duration asymmetry'. Asymmetry values are calculated by comparing corresponding biomechanical or temporal gait parameters from adjacent steps. A time of occurrence is assigned to each asymmetry value. An indication that a mechanism is contributing to ML control is obtained by comparing asymmetry values to the ML body motion (CoM angular position and velocity) at the time points associated with the asymmetry measures. Example results are demonstrated with measures obtained during a stepping-in-place (SiP) gait performed on a stance surface that either remained fixed and level or was pseudorandomly tilted to disturb balance in the ML direction. We also demonstrate that the variability of asymmetry measures obtained from 40 individuals during unperturbed, self-paced SiP were highly correlated with corresponding coefficient of variation measures that have previously been shown to be associated with poor balance and fall risk.

KEYWORDS

balance, gait, stability, gait asymmetry, foot placement, step timing, ankle torque, stepping-in-place

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1. Introduction

This is a methods-focused study whose goal is to introduce and to motivate the use of new quantitative measures to improve our understanding of mechanisms that contribute to dynamic balance control in the frontal plane during gait. We use the term dynamic balance to refer to the maintenance of stability during a gait task where body center-of-mass (CoM) frequently moves outside the base of support during movement (1, 2). This contrasts with static balance during stance where stability is maintained by ensuring that the CoM remains within the base of support.

While passive mechanical properties of oscillatory leg motions in the sagittal plane likely contribute to the maintenance of stable forward motion (3, 4) it is well accepted that active, neurally-mediated control is necessary to maintain dynamic balance in the frontal plane or medial-lateral (ML) direction (4–6). During successive foot placements in a walking gait, the CoM in the ML direction oscillates back and forth between the feet (2, 6, 7). To maintain dynamic balance during gait, the nervous system must execute control actions via mechanisms that ensure a stable oscillating pattern of ML body motion (8, 9).

Justification for focusing on ML balance includes evidence that ML balance during gait is disturbed to a much greater extent by ML visual motion stimuli than is AP balance by AP stimuli suggesting a dominant contribution of active mechanisms for stabilizing ML balance compared to AP balance during gait (4). Additional justification comes from studies in older adults demonstrating greater difficulty with ML than AP balance (10–14) and tasks challenging ML balance were better predictors of falls in older subjects than tasks challenging AP balance (15).

This study illustrates new measures for characterizing dynamic balance control by making use of data collected using a stepping-in-place (SiP) paradigm. The primary use of a SiP paradigm has been to document the effect of vestibular dysfunction on heading direction (16). Similarly, Agathos et al. (17) used SiP to investigate the influence of optic flow on self-motion perception. There is only a small literature that has used SiP to investigate the mechanics of gait. Brenière (18, 19) considered that the pattern of ML CoM and CoP motions during SiP closely resembled those that occur during a walking gait such that experimental results from SiP could be used to draw conclusions about "the relationships between body parameters and gravity and the central programming of locomotor parameters." Garcia et al. (20) made comparisons of stepping parameters obtained using both SiP and forward walking in healthy adults and adults with hemiplegia. The similar results from both paradigms led to the overall conclusion that the results "may provide solid evidence that stepping-in-place and gait are inherently related." We made use of the similarity in ML motion in SiP and walking gait and the need for dynamic balance control in both gaits to develop new measures that can characterize the contributions of mechanisms that control dynamic balance.

There are multiple mechanisms that can potentially contribute to ML dynamic balance control. Many studies considered that a mechanism based on "step-width regulation" is the primary mechanism for controlling dynamic balance (5, 21–29) with a theoretical study showing that step-width regulation is an efficient mechanism for controlling ML balance (5). Step-width regulation involves the subject placing a foot further from or closer to the body's midline on each step, to generate a gravity-induced corrective torque that is appropriate to maintain dynamic balance (with the corrective torque proportional to the distance between the body CoM and the stance foot position, and with sequential foot placements determined in relation to sensory-detected deviations from the desired body movement).

Ankle torque and reaction torque (latter from the coordinated motion of the upper body relative to the lower body and also called counter-rotation mechanism) have also been recognized as contributors to ML balance control in humans (7, 21, 30–37). Additionally, variations in actions affecting mainly forward walking progression can also contribute to ML control to the extent that there is crosstalk between sagittal and frontal control mechanisms. These include modulation of push-off torque (4, 8, 35, 38) and alteration in the direction of travel (steering control) (39).

Largely unrecognized in the physiological literature is the potential contribution of step timing mechanisms for controlling ML dynamic balance. In studies of balance in cats (40) and humans (21, 41), researchers found that changes in single-leg support time were observed in response to sudden ML body displacements caused by impulsive perturbations. Additionally, a recent study identified that an ML margin of stability measure, assumed to be indicative of the quality of balance control, was found to be closely related to adjustments in single-leg support times during various treadmill walking manipulations while the modulation of step width showed less co-variation (42). Significantly, in a robotics study of gait control, in both model-simulations and actual implementations in a cat-sized quadruped robot, researchers developed an algorithm that relied entirely on leg loading and unloading to alter step timing to control ML dynamic balance during forward walking (43, 44) and, thus, demonstrated the ability of this mechanism alone to achieve ML dynamic balance control.

The potential for step-timing to contribute to ML dynamic balance control can be understood by considering the physics of body motion during the single-leg stance phase of a gait cycle. At the beginning of each single-leg stance phase the body CoM is moving toward the stance leg, but the CoM is medial to the stance foot (7). The location of the body CoM relative to the foot provides a gravity-induced frontal plane component of torque that initially slows, then stops, and finally reverses the ML progression of the CoM. By extending/shortening the duration of the single-leg stance phase, torque acts over a longer/shorter time interval, imparting greater/lesser corrective action to control motion in the ML direction and facilitating stability of ML dynamic balance. Furthermore, the swing phase duration for each leg, the time from toe off to the next heel contact, could potentially be separately regulated to alter the duration over which ML corrective torque is available during the previous or subsequent stance phases.

To quantify the relationship between deviations of ML CoM motion from the normal step cycle to the modulation of step parameters contributing to dynamic balance control, we developed four unitless asymmetry measures that compared normalized step-to-step changes in step parameters. We refer to these measures as Step Width Asymmetry (SWA), Ankle Torque Asymmetry (ATA), Stance Duration Asymmetry (StDA), and Swing Duration Asymmetry (SwDA). All of these asymmetry calculations were formulated with the property that their signs are indicative of step-to-step changes that contribute to compensation for directional deviations in ML CoM body motion from a stable ML gait cycle. In this methods-focused paper we use results from SiP tests to illustrate the calculation of these four asymmetry measures which are applicable to the analysis of both SiP and forward walking gaits. For forward walking gait, the potential exists that asymmetry measures that characterize step-to-step changes in push-off torque (4, 8, 35, 38) and steering control (39) could be developed.

While studies evaluating unperturbed variations in CoM motion in relation to compensatory actions (45, 46) have contributed to understanding balance control during gait, the application of ML balance perturbing stimuli have also been used to provide evidence of control actions that contribute to balance corrections. Perturbing stimuli have included physically moving the subject by pushing or pulling (41, 47–49), application of galvanic stimulation (34, 36, 50), perturbing the visual environment (35, 51–54), and moving both the visual scene and walking surface (55). We demonstrate the application of our asymmetry measures in both unperturbed and perturbed conditions.

In unperturbed conditions the variability of asymmetry time series would likely be related to traditional variability measures (i.e., coefficient of variation of step width, step time, swing time, stride time). Since traditional gait variability measures have been linked to gait stability deficits and falls (56–58), the demonstration of a high correlation between asymmetry variability and traditional gait variability measures would suggest that asymmetry variability could provide a similar indicator of gait deficits. Furthermore, a demonstration that asymmetry measures are associated with mechanisms that correct for deviations of ML CoM motion could additionally link traditional gait variability measures with mechanisms regulating dynamic balance control.

The purpose of this paper is to define the algorithms to calculate these new asymmetry measures, to show how biomechanical measures can be processed in order to relate both stimulus-evoked and spontaneous variation in CoM motion to the asymmetry measures, and to give examples of these methods. Additionally, we show that the variability of asymmetry measures calculated in conditions where no stimulus perturbation is applied are highly correlated with step variability measures that have previously been shown to relate to the quality of balance control during gait and to disability (56, 57, 59–62). Application of our new measures may provide a bridge between studies using differing methodologies (with and without perturbations, with and without special subject populations) by offering a method which can easily be employed to facilitate understanding mechanisms contributing to ML dynamic balance control in a variety of settings and in different patient populations.

2. Materials and methods

2.1. Participants

Example data were drawn from healthy participants who were all Veterans with no reported balance deficits. Participants included a 'young' age group ($N = 20$, age range 25–43 years, mean 32 years, 16 male) and an 'old' age group ($N = 20$, age range 65–82 years, mean 72 years, 19 male). All participants gave written informed consent in the study whose recruitment procedures and experimental protocols were approved by the Veterans Affairs Portland Health Care System Institutional Review Board.

Subjects were screened using a questionnaire to rule out current and past conditions that could be contributors to balance dysfunction (neurological deficits, concussion, numbness, heart disease, fainting, arthritis and joint pain, motion limitations, diabetes, meningitis, sensory dysfunction including hearing, vision, vestibular/balance disorders) and to query their ability to stand and walk for 20 min. VA medical records were accessed to verify responses to the subject questionnaire and to identify whether subjects were taking medications that could affect balance. Subjects were not included if disorders were present, fitness was low, or medications affecting balance were used.

2.2. Data collection, protocols, and gait measures

Tests were performed on a custom balance platform (see Supplementary Figure S1) consisting of dual force plates (each 25 × 50 cm) that measured vertical forces from each foot at the corners of each force plate. Subjects stood or stepped-in-place with one foot on each force plate and faced a semicircular visual surround with a high contrast scene. The platform and visual-surround could tilt side-to-side to perturb ML balance with tilt angles determined by servo-controlled motors. The rotation axes of platform and visual surround were perpendicular to the subject's frontal plane, located at ankle joint height, and aligned with the middle of the platform. A custom real-time LabVIEW data collection program generated stimuli and collected experimental data (LabVIEW version 2015; NI PXI-8115 controller; NI PXI 6259 multifunction module, National Instruments, Austin, TX, United States)[1]. The program generated (1) the waveform used for the surface-tilt or visual-tilt stimulus and, on some trials, (2) a metronome pacing signal. The program recorded (1) vertical forces from eight force transducers (MLP-150 load cells, Transducer Techniques, Temecula, CA)[2] located near the corners of each force plate, (2) signals from four optical distance sensors (Sharp Corp., Sakai, Osaka, Japan)[3] used to measure ML foot locations on the force plate surfaces, (3) signals from two potentiometers (model CP-2UT, Midori America Corp., Irvine, CA, United States)[4] used to measure ML body motion at hip and shoulder levels, (4) measures of actual surface-tilt or visual-tilt angles, and (5) the metronome pacing signal. The sampling rate for all signals was 200/s.

Surface-tilt and visual-tilt stimuli were defined by a waveform based on a 5-stage pseudorandom ternary maximal length sequence [PRTS; (63)] that was mathematically integrated and scaled to give 36.3-s duration stimulus profiles with peak-to-peak tilt amplitudes of 0° (fixed surface or visual tilt angle), 1°, 3°, or 6°. Six continuous PRTS cycles were presented per trial. The ML angular tilt of the stance surface was controlled by a servomotor (Model 4102DCM000B1CCF006, Cleveland Motion Controls, Billerica, MA)[5] with custom gear reduction using Amacoil/Uhing linear drive nuts

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[1] https://ni.com

[2] https://www.transducertechniques.com/

[3] https://global.sharp/

[4] https://www.midoriamerica.com/

[5] https://www.cmccontrols.com/

(Amacoil, Inc., Aston, PA, United States)[6] and controller (Model BL20-40, BL Linear Amplifier, Aerotech Inc., Pittsburgh, PA, United States)[7]. The visual-surround was controlled by the same model servomotor, also with custom gear reduction, and controller (Model SD2-720-40-1 amplifier, Servo Dynamics Corp., Chatsworth, CA, United States)[8]. The metronome rate (~92.56 beats per minute) was chosen to give 56 steps per 36.3-s PRTS cycle duration.

Subjects wore a harness with attachments to overhead beams to prevent falls with the attachment straps adjusted so they did not provide support during testing. Tests were performed without shoes or socks to ensure availability of tactile information (see next paragraph). Headphones were used to mask ambient sounds and to deliver metronome beats to control step timing on most trials. On trials without metronome pacing, audio book recordings were played to maintain subject alertness.

T-shaped foam strips (6 mm height) were taped to the platform surface with the vertical portion of the T (9.5 cm width) located between the feet, and the horizontal portion of the T (8 cm width) located at the front of the platform. Subjects were instructed to maintain toe contact on each step with the horizontal foam strip to minimize forward or backward drift during SiP. The vertical foam strip provided feedback to reduce drift in the ML direction. The width of the vertical foam strip was less than the distance between the feet during SiP such that subjects typically did not make contact with the strip during SiP. Subjects were instructed to be aware that if they did make contact with the vertical strip then they were to move laterally on subsequent steps to avoid continued contact with the vertical strip.

Each subject performed 27 tests with each test having a duration of 258 s: 11 were SiP tests performed with metronome pacing, 11 were stance tests, and 5 were SiP tests performed without metronome pacing. The 11 SiP tests with metronome and the 11 stance tests both included tests in 3 conditions (visual tilt, surface tilt with eyes open and closed) each with 1°, 3°, or 6° stimulus amplitudes. There were two additional eyes open and closed tests on a fixed and level surface. The non-metronome-paced SiP tests included tests performed eye open and closed on a fixed surface and 3 eyes closed tests performed with 1°, 3°, or 6° surface-tilt stimuli. All tests were performed in randomized order in 2 or 3 sessions on separate days. Five-minute rest periods were provided every 4th test or as needed. Subjects were given practice trials with metronome pacing prior to testing. Not every metronome-paced test was performed with perfect compliance throughout each test, but perfect compliance was not necessary for the calculation of our asymmetry measures.

2.2.1. Foot placement measures

Two optical distance sensors were placed on the outer edges of the platform (Sharp Corp. model GPY0A41SK0F with 4–30 cm range) to measure the distance from the platform edge to the outer edge of each foot (at approximate location of the ankle joint). Two additional sensors (Sharp Corp. GPY0A51SK0F with 2–15 cm range) were placed between the feet to measure the distance from the midpoint of the platform to the inner edges of each foot. Data from these sensors were used to calculate the ML foot center of each foot during the stance phase of SiP gait and later used to calculate the 'step width' defined as the ML distance between right and left foot centers at time points that were at the approximate midpoint of the double support phase of the gait cycle.

2.2.2. Gait timing measures

Recordings of the time courses of total vertical force under each foot were used to calculate foot-on and foot-off contact times for each foot and each step following the methods related to those described by Hausdorff et al. (64). Briefly, an initial force threshold, whose value was about 1% of total body weight, was used to identify approximate foot-on contact times (when the force first rose above the threshold) and foot-off contact times (when the force first fell below the threshold). The contact times were refined by calculating the rate-of-change of the force signals (force velocity) and searching in the vicinity of the previously identified contact times for the time point where the force velocity fell below a velocity threshold value that was close to zero. The identified foot-on and foot-off contact times were used to calculate the duration of each foot's contact with the surface (i.e., stance duration) and the duration when each foot was not in contact with the surface (i.e., swing duration).

2.2.3. Vertical force and center-of-pressure (CoP) measures

Measures from the force plate transducers were used to calculate the total vertical force under each foot, the ML CoP displacement under each foot when the foot was in contact with the force plate, and the whole-body ML CoP displacement.

2.2.4. Medial–lateral body motion measures

Medial–lateral body displacements at hip and shoulder heights were recorded using 'sway rods' that consisted of Earth-fixed potentiometers (Midori America Corp.) to which were attached the sway rods (aluminum arrow shafts) with the distal ends of the rods placed in hooks attached to the subject a hip and shoulder levels. The potentiometer voltages were recorded and processed, taking into account trigonometric relationships, to calculate ML displacements. At the beginning of test sessions subjects performed a calibration trial where they were instructed to sway very slowly side-to-side using a variety of upper and lower body orientations. The calibration data were analyzed to determine the linear regression factors that defined the relationship between ML hip and shoulder displacements and the ML center-of-mass (CoM) displacement relative to the center of the platform using the assumption that the CoP displacement will be vertically aligned with CoM displacement for very slow motions [see (65)]. However, because a subject did not remain perfectly centered on the platform during SiP, adjustments were made to the measured ML hip and shoulder displacements so that these displacements represented displacements relative to the subject's 'path' defined as the trajectory over time of the ML location of a point midway between the feet (see next section). The regression factors from the calibration trial were then applied to the path-corrected hip and shoulder displacement to calculate the CoM displacement relative to the path. Finally, using the path-corrected CoM displacement and an estimate of a subject's CoM height above the ankle joint based on body segment measures (66), the subject's ML CoM tilt angle was calculated. This ML CoM tilt angle was the primary output variable used in later stages of analysis.

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[6] https://www.amacoil.com/

[7] https://www.aerotech.com/

[8] https://www.servodynamics.com/

2.2.5. Medial–lateral path analysis

The path trajectory was estimated using the procedures illustrated in Figure 1. The path trajectory was used to compensate for any potential ML drift during SiP, allowing CoM displacements to be referenced to the ML location of the calculated path which defined the time-varying location midway between the feet throughout the test. The vertical force measures from the R and L force plates were used to detect the time segments when only one foot was in contact with the surface (Figure 1A, thick bars). During these foot-contact segments the optical distance sensors provided measures used to calculate the ML location of the foot center for each foot to give a set of discontinuous ML foot locations (Figure 1B, thick bars). Separately for each foot, a moving window average with window width 1.5 s was applied to the discontinuous foot location segments. The averaging was applied to all ML foot displacement values during contact segments that were within the 1.5 s window as the window center point incremented through time resulting in a continuous time series that ramped toward each successive foot location (Figure 1B, dotted lines). A phaseless lowpass filter (4 order 0.5 Hz cutoff using Matlab filtfilt function; The MathWorks, Inc., Natick, MA; Matlab, 2021a) was then applied to the windowed time series for each foot (Figure 1B, thin solid lines) to calculate the trajectory of the ML displacement of each foot. Then the R and L foot trajectories were averaged to give the overall path trajectory (Figure 1B, thick solid line). Finally, the computed path trajectory was used to provide a path-corrected CoP and CoM displacement trajectories (Figure 1C) and the path-corrected CoM displacement was used to compute the CoM tilt angle about an origin defined by the path and located at ankle height.

We chose to quantify the dynamic state of the body using the angular tilt of the CoM. Asymmetry measures could have been calculated using the horizontal CoM displacement relative to the path with similar results. An advantage of using CoM angle is that this provides a normalization that accounts for subjects with different CoM heights. It is important to recognize that our CoM angular measure represents the state of the body orientation relative to Earth vertical and is not a measure proportional to the gravitational torque exerted on the body during the single leg stance phases of gait which depends on the distance between the CoM and foot position.

2.3. Gait asymmetry measures

Four gait asymmetry measures were defined by comparing gait measures across adjacent pairs of steps over the course of an experimental trial.

2.3.1. Step width asymmetry

The *SWA* measure compares adjacent pairs of ML (frontal plane) step widths (SW_i, SW_{i+1}) calculated from foot placement measures. Specifically, *SW* is the ML coordinate of the right-foot center location minus the ML coordinate of the adjacent left-foot center location. The *SWA* is normalized by the sum of the absolute values of the two step widths to give a unitless measure indicating the relative change in step width and the direction of lateral motion resulting from the combination of two adjacent steps. The equation for calculating *SWA* depends on which foot stepped first. When the first step is by the right (R) foot (as in Figure 2), the equations are:

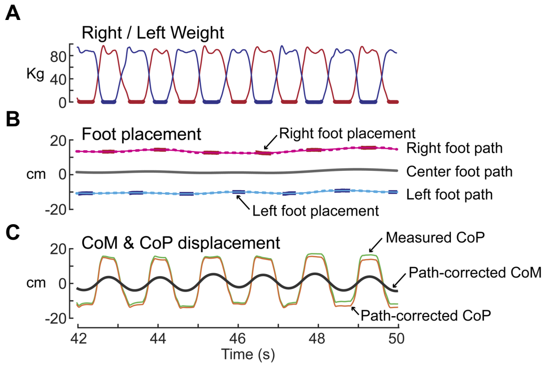

FIGURE 1
Path analysis of stepping-in-place data for the purpose of estimating the trajectory of the midpoint between the right and left foot locations over time. **(A)** Shows time courses of weight changes measured by the right (red) and left (blue) force plates. Thick lines indicate single-leg stance segments of gait. **(B)** Shows the processing of right and left foot placement data (red and blue bars) to calculate the right and left foot paths (thin solid lines) and then averaging of these paths to calculate the center foot path (thick black line). **(C)** The center foot path was used to derive path-corrected measures of the whole-body medial-lateral center-of-pressure (CoP) and center-of-mass (CoM) displacements.

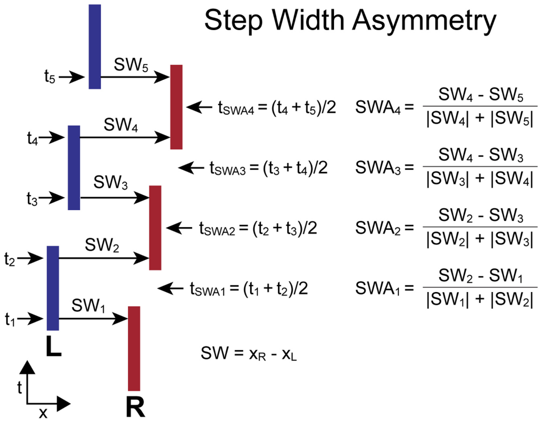

FIGURE 2
Step Width Asymmetry (*SWA*) calculation parameters and formula. Times t_i are the times at the midpoints of the double-support phases at which the step widths, SW_i, are measured. The *SWA* measures are based on a normalized comparison of adjacent pairs of *SW* measures. The time point, t_{SWAi}, associated with each *SWA* measure is the average of the mid-double-support times used for the *SWA* calculation. The calculations are shown for the condition where the first step is with the right (R) foot and differs from the calculation when the first step is with the left (L) foot so that positive *SWA* measures are always indicative of a movement toward the R independent of whether the first step was with the R or L foot.

$$SWA_i = \left(SW_{i+1} - SW_i\right) / \left(|SW_i| + |SW_{i+1}|\right) \quad \text{for } i = \text{odd (i.e., R-to-L steps)}$$
$$SWA_i = \left(SW_i - SW_{i+1}\right) / \left(|SW_i| + |SW_{i+1}|\right) \quad \text{for } i = \text{even (i.e., L-to-R steps)}$$

When the first step is by the left (L) foot, the equations are:

$$SWA_i = \left(SW_i - SW_{i+1}\right) / \left(|SW_i| + |SW_{i+1}|\right) \quad \text{for } i = \text{odd (i.e., L-to-R steps)}$$
$$SWA_i = \left(SW_{i+1} - SW_i\right) / \left(|SW_i| + |SW_{i+1}|\right) \quad \text{for } i = \text{even (i.e., R-to-L steps)}$$

For both calculations the R-to-L step width is subtracted from the adjacent L-to-R step width. Therefore, the *SWA* asymmetry measure has a positive value when the adjacent steps produced a net movement to the R, and a negative value when the net movement is to the left. Individual *SW* values can be negative in the case of crossover steps.

The time (t_i) assigned to the *i*th step width measure (SW_i) is defined as the time at the approximate midpoint of the double-support phase of the gait cycle which was calculated by taking the mean of the time points associated with adjacent R and L stance periods. The time assigned to the *i*th *SWA* measure (t_{SWAi}) is defined as the average of the two adjacent t_i times used in the *SWA* calculation. Thus, t_{SWAi} is approximately at the midpoint of the single-leg stance phase of the gait cycle.

Figure 2 illustrates the calculation of *SWA* for a stepping pattern that was continuously progressing to the R and began with a R step. In this case, all *SW* measures with even indexes are L-to-R steps, and these steps are larger than all *SW* measures with odd indexes (R-to-L steps). The SWA_i measures will have positive signs for all indexes. The assignment of a time to each *SWA* measure allows for the comparison of body motion variables measured at the times associated with the *SWA* measures.

2.3.2. Ankle torque asymmetry

The *ATA* measure compares values related to ankle torque (AT_i, AT_{i+1}) measured at the midpoint of single leg stance phases of adjacent R and L steps. An ankle everting or inverting torque produces a ML shift in the location of the CoP under a foot. This CoP shift moves the effective point of force application either closer to or farther away from the body CoM causing a decrease or increase, respectively, in the net torque that affects the ML acceleration of the body. The *AT* for each foot is calculated by subtracting the recorded ML foot placement (*FP* – a measure of the ML location of the foot center) from the ML *CoP* value at the midpoint of the single leg stance phase ($AT_i = CoP_i - FP_i$). The *ATA* is normalized by dividing by foot width (*FW*). The equation for calculating *ATA*, which does not depend on which foot stepped first, is:

$$ATA_i = \frac{\left(AT_i + AT_{i+1}\right)}{FW}.$$

The time (t_i) assigned to the *i*th *AT* measure (AT_i) is defined as the time at the midpoint of single-leg contact time with the surface. The time assigned to the *i*th *ATA* measure (t_{ATAi}) is defined as the average of the two adjacent t_i times used in the *ATA* calculation. Thus, t_{ATAi} is approximately at the midpoint of the double-leg stance phase of the gait cycle.

Figure 3 illustrates the calculation of *ATA* for a stepping pattern where the CoP is shifted toward the right relative to the foot placement for both R and L feet. In this example, the rightward shift in the R foot is caused by ankle inversion and in the L foot is caused by ankle eversion. Thus, *AT* values for both R and L feet have positive values and *ATA* measures at all indexes have positive values. This pattern provides a corrective torque appropriate to compensate for a rightward bias in body lean during gait. The assignment of a time to each *ATA* measure allows for the comparison of body motion variables measured at the times associated with the *ATA* measures.

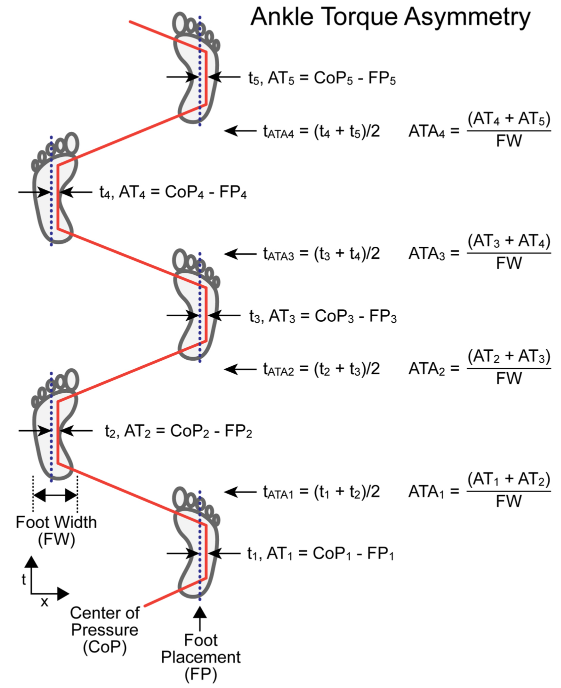

FIGURE 3
Ankle Torque Asymmetry (*ATA*) calculation parameters and formula. Times t_i are the times at the midpoints of the single-leg support phases at which the lateral distance between the center-of-pressure under the foot (CoP_i) and the center line of the foot placement (FP_i) were used to calculate a value proportional to ankle torque (AT_i) but with units of displacement (e.g., cm). The *AT* measures from adjacent steps were added and then divided by the foot width (*FW*) to give a unitless *ATA* measure associated with the paired steps at a time, t_{ATAi}, that occurs at approximately the mid-point of the double-leg support phase. The example shows a condition where the CoP under both the right and left feet during the single-leg support phases are always displaced to the right relative to the foot center.

2.3.3. Stance duration asymmetry

The *StDA* measure compares adjacent R and L leg stance durations (StD_i, StD_{i+1}). *StD* for each foot is the duration from the time of first contact with the surface (typically at heel-contact during walking but usually at ball of foot contact during SiP) to the time the foot leaves the surface (typically at toe-off during walking). The *StDA* is normalized by the sum of the two stance durations used in its calculation to give a unitless measure indicating the relative change in duration of stance phases and the leg that had the longest contact duration. The equation for calculating *StDA* depends on which foot stepped first. When the first step is by the R foot, the equations are:

$$StDA_i = \frac{\left(StD_i - StD_{i+1}\right)}{\left(StD_i + StD_{i+1}\right)} \qquad \text{for } i = \text{odd}.$$

$$StDA_i = \frac{\left(StD_{i+1} - StD_i\right)}{\left(StD_i + StD_{i+1}\right)} \qquad \text{for } i = \text{even}.$$

When the first step is by the L foot, the equations are:

$$StDA_i = \frac{\left(StD_{i+1} - StD_i\right)}{\left(StD_i + StD_{i+1}\right)} \qquad \text{for } i = \text{odd.}$$

$$StDA_i = \frac{\left(StD_i - StD_{i+1}\right)}{\left(StD_i + StD_{i+1}\right)} \qquad \text{for } i = \text{even.}$$

For both calculations the L foot stance duration is subtracted from the adjacent R foot stance duration. Therefore, the *StDA* asymmetry measure has a positive value when the R foot remained on the surface longer than the L foot on adjacent steps, and a negative value when the L foot remained on the surface longer than the R foot.

The time (t_i) assigned to the *i*th stance duration measure (StD_i) is defined as the time at the midpoint of a foot's contact time with the surface. The time assigned to the i^{th} *StDA* measure (t_{StDAi}) is defined as the average of the two adjacent t_i times used in the *StDA* calculation. Thus, t_{StDAi} is approximately at the midpoint of the double-leg stance phase of the gait cycle.

Figure 4 illustrates the calculation of *StDA* for a stepping pattern that began with a R step. All R foot stance durations are slightly longer than L foot stance durations. Thus, the *StDA* measures at all indexes will have positive values. The assignment of a time to each *StDA* measure allows for the comparison of body motion variables measured at the times associated with the *StDA* measures.

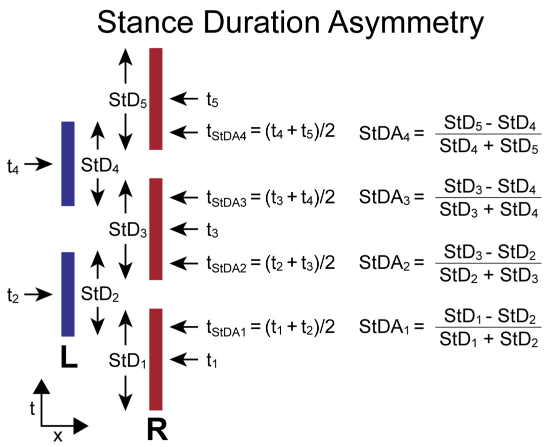

FIGURE 4
Stance Duration Asymmetry (*StDA*) calculation parameters and formula. Times t_i are the times at the midpoint in time when a foot is in contact with the surface defined as the stance durations (StD_i). The difference between the values of *StD* on adjacent steps divided by their sum determines the value of *StDA*. The time point (t_{StDAi}) associated with each *StDA* measure is the average of the midpoints in time of the right and left stance phases used for the *StDA* calculation and occurs approximately at the midpoint of the double-leg support phase of gait. The calculations are shown for the condition where the first step is with the right (R) foot and differs from the calculation when the first step is with the left (L) foot so that a positive *StDA* measure is always indicative of a pattern of stepping where the R foot contact time is greater than the L foot contact time.

2.3.4. Swing duration asymmetry

The *SwDA* measure compares adjacent R and L leg swing durations (SwD_i, SwD_{i+1}). *SwD* for each foot is the duration from the time when the foot leaves the surface (typically toe-off time during walking) to the following time of foot contact with the surface (typically the heel-contact time during walking). The *SwDA* is normalized by the sum of the two swing durations used in its calculation to give a unitless measure indicating the relative change in swing-leg duration and the leg that had the longest swing duration. The equation for calculating *SwDA* depends on which foot stepped first. When the first step is by the R foot, the equations are:

$$SwDA_i = \frac{\left(SwD_{i+1} - SwD_i\right)}{\left(SwD_i + SwD_{i+1}\right)} \qquad \text{for } i = \text{odd,}$$

$$SwDA_i = \frac{\left(SwD_i - SwD_{i+1}\right)}{\left(SwD_i + SwD_{i+1}\right)} \qquad \text{for } i = \text{even.}$$

When the first step is by the L foot, the equations are:

$$SwDA_i = \frac{\left(SwD_i - SwD_{i+1}\right)}{\left(SwD_i + SwD_{i+1}\right)} \qquad \text{for } i = \text{odd,}$$

$$SwDA_i = \frac{\left(SwD_{i+1} - SwD_i\right)}{\left(SwD_i + SwD_{i+1}\right)} \qquad \text{for } i = \text{even.}$$

For both calculations the R foot swing duration is subtracted from the adjacent L foot swing duration. Therefore, the *SwDA* asymmetry measure has a positive value when the R foot swing duration is shorter than the L foot swing duration on adjacent steps, and a negative value when the R foot swing duration is longer than the L foot swing duration.

The time (t_i) assigned to the *i*th swing duration measure (SwD_i) is defined as the time at the midpoint of a leg's swing phase. The time assigned to the *i*th *SwDA* measure (t_{SwDAi}) is defined as the average of the two adjacent t_i times used in the *SwDA* calculation. Thus, t_{SwDAi} is approximately at the midpoint of the double-leg stance phase of the gait cycle.

Figure 5 illustrates the calculation of *SwDA* for a stepping pattern that began with a R step. All R foot swing durations are slightly shorter than L foot swing durations. Thus, the *SwDA* measures at all indexes will have positive values. The assignment of a time to each *SwDA* measure allows for the comparison of body motion variables measured at the times associated with the *SwDA* measures.

2.4. Relating asymmetry measures to CoM deviation

To the extent that modulations of step width, ankle torque, and stance and swing durations contributed to the maintenance of dynamic balance we anticipated the asymmetry measures would be correlated with deviations from the normal symmetric oscillatory pattern of ML CoM angular motion about earth-vertical expected

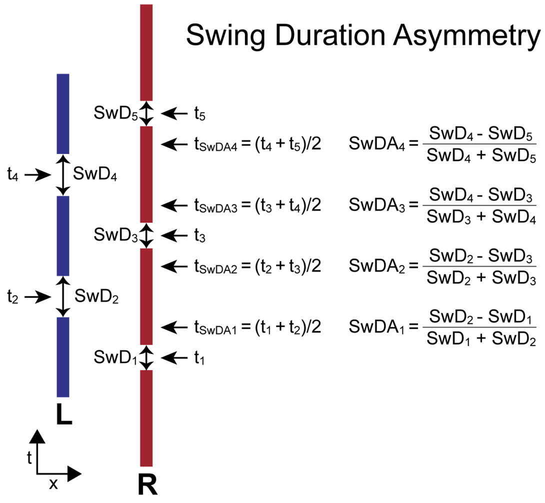

FIGURE 5
Swing Duration Asymmetry (*SwDA*) parameters and formula. Times t_i are the times at the midpoint in time of the swing phase of gait when a foot is not in contact with the surface defined as the swing durations (*SwD*$_i$). The difference between the values of *SwD* on adjacent steps divided by their sum determines the value of *SwDA*. The time point (t_{SwDAi}) associated with each *SwDA* measure is the average of the midpoints in time of the right and left swing phases used for the *SwDA* calculation and occurs approximately at the midpoint of the double-leg support phase of gait. The calculations are shown for the condition where the first step is with the right (R) foot and differs from the calculation when the first step is with the left (L) foot so that a positive *StDA* measure is always indicative of a pattern of stepping where the R foot *SwD* is shorter than the L foot *SwD*.

during gait on a level surface. CoM angular deviations from the normal pattern can occur due to normal step-to-step variability caused by imperfect control of gait and from application of stimuli that either directly evoke body sway (an external push) or stimuli that indirectly, via sensory integration mechanisms, bias the mean body orientation away from an oscillatory pattern about earth vertical.

To measure the CoM deviation from upright we applied a phaseless 4-order Butterworth lowpass filter with a cutoff frequency of 0.5 Hz to the measured CoM motion. This cutoff frequency was below the metronome-paced stepping frequency of about 0.77 Hz and thus eliminated most of the oscillatory sway pattern while preserving the longer-term deviations of CoM from upright. The effects of different choices of the cutoff frequency were investigated (see Section 3.3).

A central difference calculation applied to the filtered CoM was used to obtain a measure of CoM velocity. We refer to the filtered CoM and CoM velocity time series as deviation signals ΔCoM and $\Delta\text{CoM}_{\text{vel}}$, respectively. The values of ΔCoM and $\Delta\text{CoM}_{\text{vel}}$ were sampled at the corresponding asymmetry time points and then regression analysis was applied to explore the relationship between body motion and asymmetry:

$$Asym = offset + P * \Delta\text{CoM} + V * \Delta\text{CoM}_{\text{vel}}.$$

Where *P* is the regression position factor, *V* the regression velocity factor, *offset* a constant component, ΔCoM is the deviation from upright orientation, and $\Delta\text{CoM}_{\text{vel}}$ is the deviation from zero velocity. An R^2 value was calculated for each regression to measure the amount of variance of the data accounted for by the regression equation. The *P* and *V* factors quantify the extent to which step width, ankle torque, stance duration, and swing duration asymmetry measures were modulated by CoM motion. This method was applicable on trials with or without external stimuli and with or without metronome pacing. On metronome-paced trials it was not necessary that the subject remained fully in sync with the metronome across the entire trial.

When there was no evidence of habituation or adaptation across a trial, cycle-to-cycle variability of the ΔCoM and $\Delta\text{CoM}_{\text{vel}}$ signals and the asymmetry measures can be reduced by averaging across stimulus cycles. For the across-stimulus-cycle averaging of asymmetry measures, each 36.3-s duration stimulus cycle was divided into 56 bins (2 bins per gait cycle for metronome-paced trials). Asymmetry measures that fell within each bin and the time of occurrences of those asymmetry measures were accumulated across all the stimulus cycles. Then the asymmetry values within each bin and their times of occurrence were averaged and these cycle-averaged data were used in the regression analysis. The reduced variability from cycle averaging provided clearer visualization of the regression results and comparisons of the predicted to the measured asymmetry values on trials where ML sway was evoked by external stimulation. The cycle-averaged comparisons were applicable on trials whether or not the subject was able to maintain perfect metronome-paced stepping and on non-metronome paced trials.

To explore how asymmetry variability related to traditional gait variability measures, the variability of asymmetry measures, expressed as the standard deviation (SD) of the step-to-step asymmetry measures obtained from non-metronome paced SiP tests with no applied stimulus were compared with coefficient of variation (CV) measures of step width, stance time, and swing time measures.

3. Results

We first present data to demonstrate the various data processing steps involved in constructing and analyzing our asymmetry variables. Data from non-perturbed trials are then presented with comparisons made between the variability of our asymmetry measures and conventional gait variability measures utilized in the current literature.

3.1. Example results

Example results are shown from a subject performing metronome-paced SiP with eyes closed on a stance surface that tilted side-to-side during 6 cycles of a PRTS stimulus with 3° peak-to-peak amplitude (Figures 6A,B) and during an eyes closed SiP trial with no surface-tilt stimulus (Figure 6C). Similar to results from stance control tests (67), subjects tend to adjust their body orientation away from upright and towards alignment with the tilting surface. Deviation of the mean body orientation away from upright during SiP adds a bias to the destabilizing force due to gravity such that the mechanisms involved in maintaining dynamic stability must make appropriate adjustments. These adjustments are represented by the modulation of the four asymmetry measures that we have defined.

To the extent that a subject makes use of a particular mechanism, we expect the asymmetry measure associated with that mechanism will be modulated in relation to the deviation of body orientation from upright. Since all of the asymmetry measures were defined such that

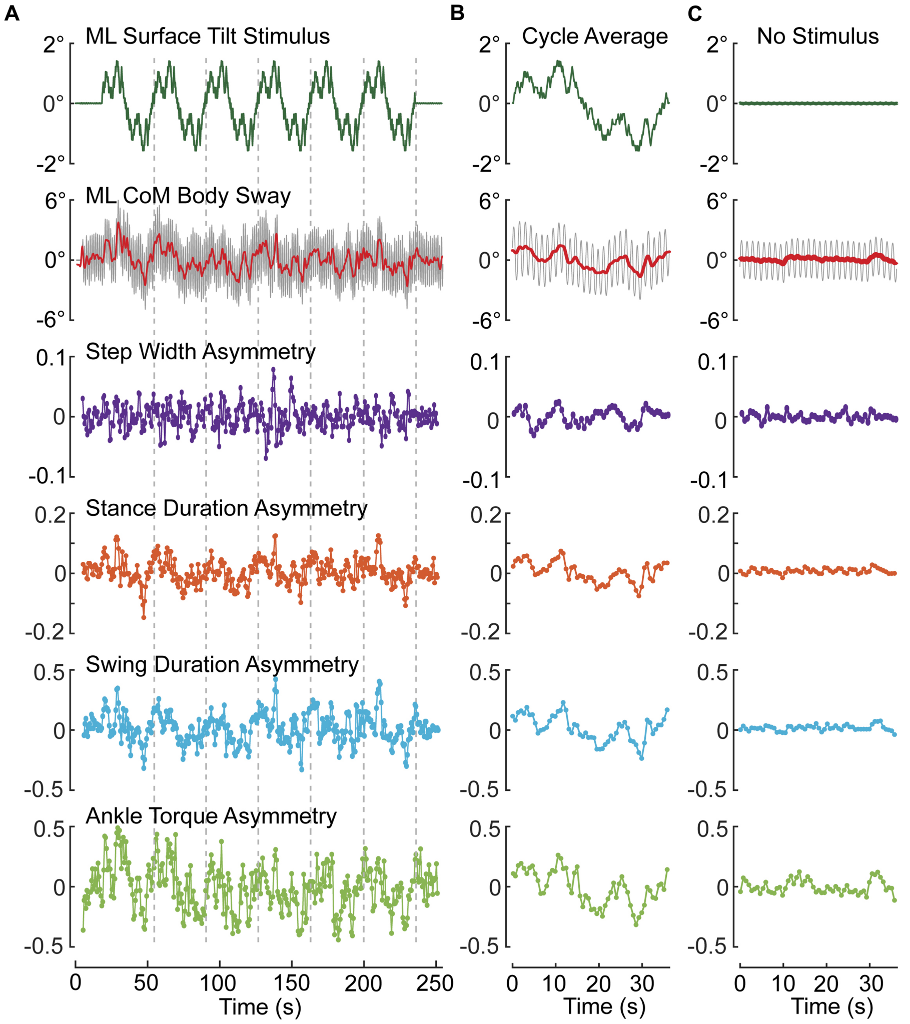

FIGURE 6
Example data from one subject showing modulation of ML CoM sway and asymmetry measures during an eyes-closed stepping-in-place trial on a tilting surface that rotated laterally during application of six cycles of a pseudorandom stimulus with 3° peak-to-peak amplitude **(A,B)** and average results from an eyes closed trial on a fixed, unmoving surface **(C)**. **(A)** Shows the entire time series of stimulus, path-corrected center-of-mass (CoM) sway and the four asymmetry measures. **(B)** Shows measures averaged over the last five 36.3-s duration cycles of the pseudorandom stimulus. **(C)** Shows cycle-averaged results for an eyes-closed stepping-in-place trial on a fixed surface demonstrating the lack of modulation of asymmetry measures compared to perturbed conditions. Stepping during both test conditions was metronome paced to give 56 steps per 36.3s pseudorandom cycle (92.56 beats/min). The red line through the center of oscillating CoM motion (gray) is the 0.5-Hz lowpass filtered CoM sway.

a positive (negative) asymmetry value is associated with a step-to-step action appropriate to correct for a rightward (leftward) bias in body orientation and motion, then we expect to see positive and negative modulation of the asymmetry values as the surface-tilt stimulus evokes rightward and leftward body leans away from upright. The step-to-step asymmetry measures in Figure 6A across the ~250-s duration of the SiP trials showed considerable variation making it difficult to appreciate the relation of the asymmetry measures to deviation in CoM sway (ΔCoM) from upright represented by the solid red line through the oscillating SiP sway. Averaging the stimulus, CoM sway, and asymmetry measures across the last 5 cycles of the PRTS stimulus reveals clear qualitative relationships between ΔCoM and all four asymmetry measures showing a continuous modulation in relation to the stimulus-evoked CoM sway.

When there was no surface-tilt stimulus, on average the body sway oscillations occurred about an upright orientation and there were minimal average deviations of CoM body orientation from 0° in comparison to the PRTS stimulus trial (Figure 6C vs. Figure 6B). Nevertheless, the average sway showed some small deviations from 0° and there was corresponding modulation in the asymmetry measures that were most evident in *ATA* and *SwDA*.

3.2. Regression analysis example

To quantify the relationship between the stimulus-induced deviation of CoM orientation and the asymmetry measures, a regression analysis was performed that characterizes the extent to which a cycle-averaged asymmetry measure could be predicted based on the cycle-averaged ΔCoM orientation angles and angular velocity measures derived from the 0.5 Hz phaseless low-pass filtering of the recorded CoM sway angle. Figure 7 shows an example *StDA* regression analysis for one subject on one trial (same subject and trial as in Figure 6) demonstrating that a large proportion of the variance in the

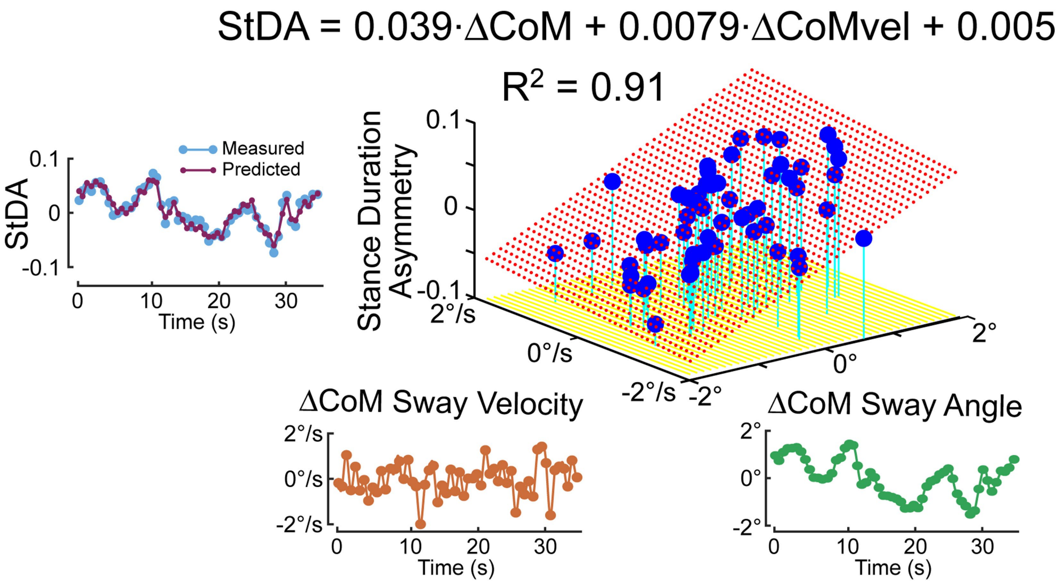

FIGURE 7
Example of regression analysis relating cycle-averaged Stance Duration Asymmetry (*StDA*) measures to average ΔCoM sway angle and ΔCoM angular velocity at time points corresponding to the mean times of occurrence of the *StDA* measures across the 56 time points 36.3-s duration of the pseudorandom PRTS surface tilt stimulus that perturbed ML balance during a stepping-in-place trial. The left plot shows that the measured cycle-averaged *StDA* is reliably predicted by results from the regression analysis. Red dotted surface is the regression fit.

data was accounted for by the regression and consistent with an accurate prediction of the cycle-averaged *StDA* measures based on the regression equation.

While cycle averaging reduces variability and reveals average behavior, the individual step-to-step asymmetry measures can also be regressed against their associated ΔCoM orientation angles and angular velocity measures. The regression results shown in Figure 8A, using the same data set as in Figure 7 analysis, show similar regression coefficients. Additionally, the individual regression results could potentially reveal more detailed step-to-step behaviors. Although all four of the asymmetry measures are consistent with step changes that compensate for deviations from desired motion, corresponding changes may not occur in all measures on a given step. For example, in Figure 6A near the 220-s time point, the subject's sway deviated to the right and was accompanied by positive deviations in *StDA* and *SwDA* but no clear changes in *SWA* and *ATA* were evident.

The results in Figure 6C showed some modulation in asymmetry measures in a no-stimulus condition even after averaging across cycles suggesting that there is sufficient step-to-step variability to estimate regression coefficient without the need to deliberately perturb balance. Results in Figure 8B support that view. Additionally, results demonstrated so far have been from metronome-paced trials where the subject maintained accurate pacing throughout the trials. Figure 8C shows regression results from the same subject from a self-paced zero-stimulus trial that shows similar regression coefficients to those obtained in the metronome-paced trial.

3.3. Effects of CoM cutoff frequency on regression analysis

A 0.5 Hz cutoff frequency was used to separate changes in CoM orientation from the dynamic oscillation that occurs during SiP under the assumption that the lower frequency changes in body orientation are the main determinates of step-to-step modulation of mechanisms controlling dynamic stability. But this choice was a heuristic one. To investigate the consequences of this choice of cutoff frequency, we took advantage of the odd symmetric properties of the PRTS stimulus (the second half of a PRTS cycle is the inverse of the first half) and that the metronome pacing was an even symmetric signal (within a PRTS cycle the second half of the metronome signal was the same as the first half). For subjects who stayed in phase with the metronome pacing, these signal properties allowed for a separation of the PRTS evoked response from the dynamic oscillation without the use of filtering.

Specifically, subtracting the second half of the recorded CoM sway from the first half, and dividing by two, cancels out the dynamic oscillation and leaves the sway response to the PRTS. Then adding the second half of a PRTS-length cycle of the recorded CoM to the first half, and dividing by two, cancels the sway response to the PRTS stimulus and leaves the dynamic oscillation. Figure 9 shows an example of the separation process applied to the sway data shown in Figure 6A.

The separated PRTS CoM response waveform was used to reconstruct a full cycle of the PRTS CoM response and this full cycle was repeated to produce a 6 cycle CoM response to the PRTS. Finally, this 6-cycle CoM signal was processed using different cutoff frequencies and analyzed to calculate regression coefficients for the asymmetry measures to determine how varying the cutoff frequency affects these coefficients. In this analysis the asymmetry time points determined in the original analysis were used to sample the reconstructed CoM response angle and angular velocity waveforms that were used in the calculation of regression factors.

Figure 10 shows examples from two subjects illustrating how regression analysis position and velocity factors changed as the cutoff frequency applied to the reconstructed CoM was varied from 0.1 to

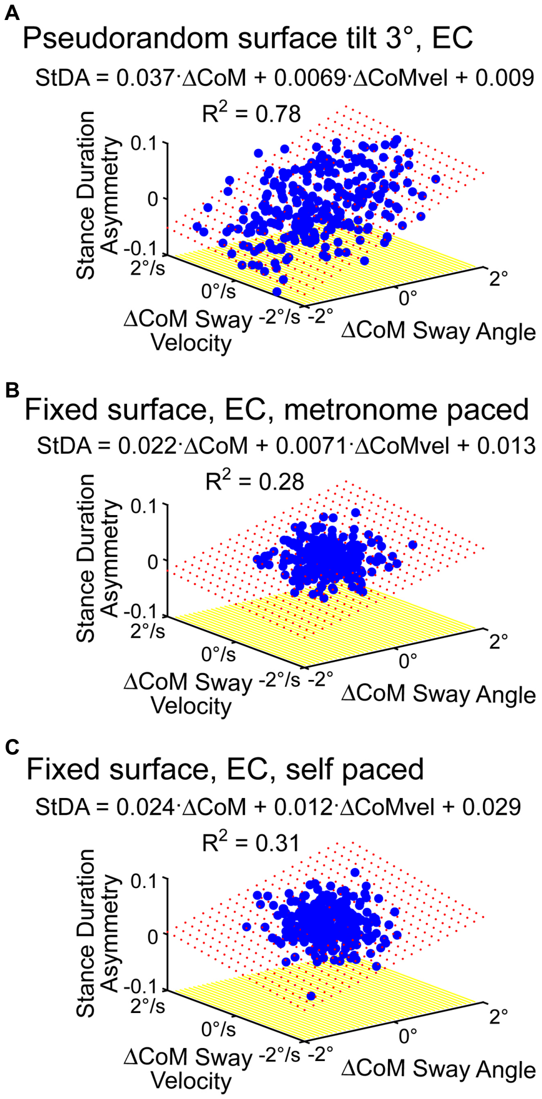

FIGURE 8
Examples of regression analysis applied to metronome-paced **(A,B)** and non-metronome-paced trials **(C)** using time series of individual asymmetry measures. **(A)** Shows regression analysis results of *StDA* using data from the same subject and trial as shown in Figure 7. **(B)** Shows *StDA* regression analysis results from a metronome-paced eyes-closed stepping-in-place trial performed on a fixed, unmoving surface. **(C)** Shows results from the same subject and same conditions as in **(B)** except with self-pacing rather than metronome pacing. Red dotted surface represents the regression fit.

5 Hz. The position and velocity factors for all four asymmetry measures were essentially unchanged for cutoff frequencies greater than about 1 Hz leading to a possible interpretation that the factors measured in this region provide unbiased measures of the position and velocity factors that relate stimulus-evoked sway to asymmetry changes. However, these results can only be obtained under conditions of metronome-paced stepping with specific properties of the perturbing stimulus (odd symmetry) in relation to the metronome signal (even symmetry). The example results in Figure 10 demonstrate that the potential for bias in regression factor measures exists when lower cutoff frequencies are used. Specifically, as the cutoff frequency was lowered the position factors remained relatively stable (constant for *SWA* and *StDA* and slightly increasing for *ATA* and *SwDA*). Velocity factors showed larger changes and the pattern of changes differed in the two examples. The filled symbols at 0.5 Hz show the values of the regression factors as determine by the original analysis (without reconstruction). The 0.5 Hz cutoff frequency was selected to be below the 0.77 Hz stepping frequency to eliminate most of the ML CoM stepping oscillation while preserving most of the stimulus-evoked CoM sway. Of note is that the variance accounted for by the CoM position and velocity fits to the asymmetry data tended to peak at cutoff frequencies just below the stepping frequency suggesting that there was a precision/accuracy tradeoff in estimating regression factors. That is, the higher R^2 values suggest regression factor estimates would have lower variance but with possible biases that made them less accurate.

3.4. Asymmetry variability relationships with conventional gait variability measures

Gait variability, quantified by calculation of coefficient of variation (CV) of step width and gait timing measures, has been used as an indicator of the quality of balance control during gait with larger CVs indicative of reduced balance control and greater likelihood of falls in older individuals and those with neurological deficits (68–72). Step-to-step variability was also present in our asymmetry measures obtained in conditions with no applied stimulus. The extent to which conventional CV measures are correlated with step-to-step variability in our asymmetry measures could indicate that future studies using asymmetry variability measures could also identify gait disorders indicative of fall propensity and neurological decline.

Step-to-step variability in our asymmetry measures, quantified by calculating the SD of the asymmetry measures, was observed in conditions of both metronome-paced and non-metronome-paced conditions when no perturbing stimulus was presented. Supplementary Figure S2 illustrates results from non-metronome-paced SiP performed with eyes open and closed showing that CV measures of gait variability were well correlated with variability measures based on the SD of *StDA*, *SwDA*, and *SWA* values across all step cycles. Data used in Supplementary Figure S2 are given in Supplementary materials in addition to showing data for *ATA* SD measures (Supplementary Tables S1–S4). CV measures based on ankle torque measures are not practical since mean ankle torque values can be close to zero. Tables 1, 2 show results from comparisons between young and old subjects for the various asymmetry SD and conventional CV measures of variability. In eyes open conditions (Table 1) only the *ATA* SD measures showed significant differences between young and old subjects. In eyes closed conditions (Table 2) three of the four asymmetry SD measures showed significant differences between young and old while one of three CV measures was significant.

4. Discussion

The main contribution of this paper was to define and demonstrate the potential utility of new asymmetry measures for quantifying the step-to-step changes that are contributing to dynamic balance control. In experiments designed to investigate sensory integration during SiP gait we observed systematic deviations of CoM ML body orientation

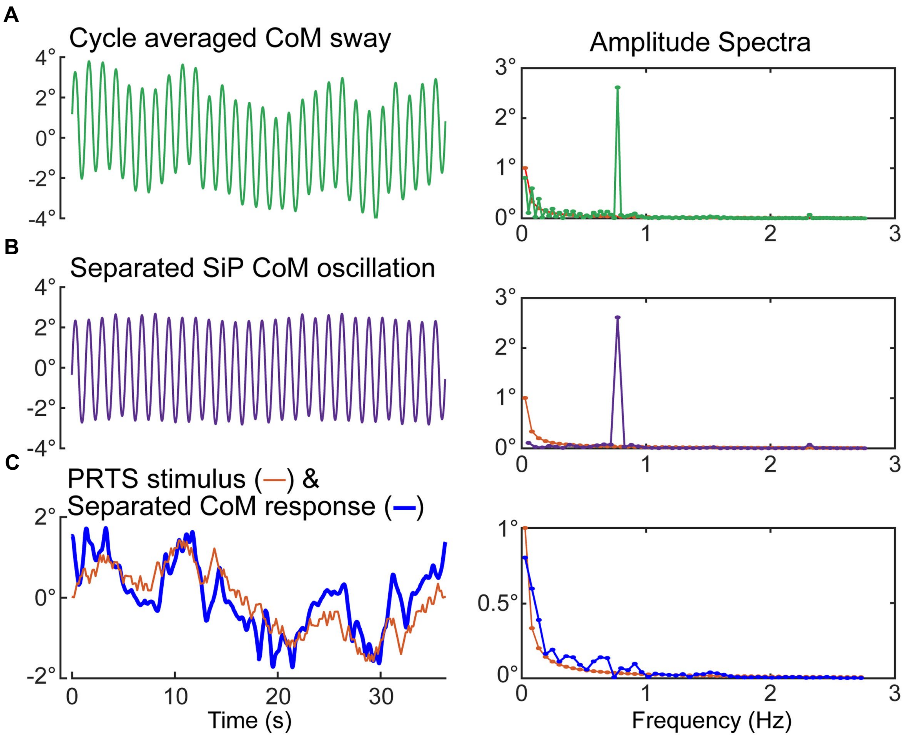

FIGURE 9
Separation of medial–lateral (ML) oscillation of the CoM during SiP from the CoM sway evoked by the ML pseudorandom PRTS surface-tilt stimulus. Metronome-paced trials with an even number of steps per stimulus cycle can be separated into the stimulus-evoked sway component and the oscillatory sway component. Left side plots show the cycle-average sway **(A)**, the separated oscillatory component at the stepping frequency **(B)**, and the separated stimulus-evoked CoM sway and the PRTS time course **(C)**. Corresponding amplitude spectra are shown in the right-side plots. The amplitude spectrum of the stimulus is shown in all right-side plots with only those harmonic components of the stimulus that have non-zero stimulus energy shown.

from earth vertical during SiP on a surface that continuously rotated side-to-side or while viewing a visual surround that rotated side-to-side. We noted that changes in ML body orientation were accompanied by modulations in step width, ankle torque, and in the duration of stance and swing phases in a manner consistent with their contributing to the maintenance of dynamic stability. We formulated four asymmetry measures from these gait parameters such that the positive or negative sign of all measures indicated the direction of corrective action afforded by the step-to-step changes in each gait parameter. We demonstrated that regression analysis could relate the change in an asymmetry measure to a deviation of ML CoM angular displacement and velocity.

We additionally demonstrated that the asymmetry measures could be used to quantify step-to-step changes in trials with no perturbing stimulus. We showed that the variability of our asymmetry measures was correlated with conventional measures of gait variability but may possibly have advantages over conventional measures in that they (1) relate directly to control mechanisms; and (2) they were better able to identify differences between younger and older healthy adults.

4.1. Control mechanisms

For control based on foot placement, if there was a rightward bias in body orientation and if the next step was with the right foot then placing that foot further to the right would result in the CoM being further to the left of the right foot position during the right leg stance phase resulting in greater gravity induced torque toward the left that corrects for the rightward bias in body orientation. If the next step is with the left foot, then a more medial placement of that foot would reduce the magnitude of the rightward gravity induced torque and would also contribute to correcting for a rightward bias in body orientation. The regulation of step placement is understood to be an important contributor to dynamic balance control during gait (5, 7, 41, 45, 48, 73). Figure 6 shows a change in *SWA* in response to ML body orientation deviations produced by the PRTS perturbation where the cycle-averaged *SWA* magnitude moved complementary with the cycle-averaged ΔCoM body sway. For example, at the time point around 30 s the ΔCoM body sway had a negative amplitude (demonstrating an average tilt of the body to the left) and the SWA at that time point showed a corresponding negative amplitude consistent with a change in step width that provided compensation for the stimulus-evoked leftward ML body tilt.

For dynamic balance control based on ankle torque, an ankle inversion of the right foot and eversion of the left foot during stance phases would generate a corrective torque the compensates for a rightward bias in body orientation. Contribution of ankle torque is recognized as a contributor to dynamic balance control in the frontal plane (8, 34). Figure 6 shows a change in cycle-averaged *ATA* in response to PRTS perturbation-induced body tilt whose amplitude

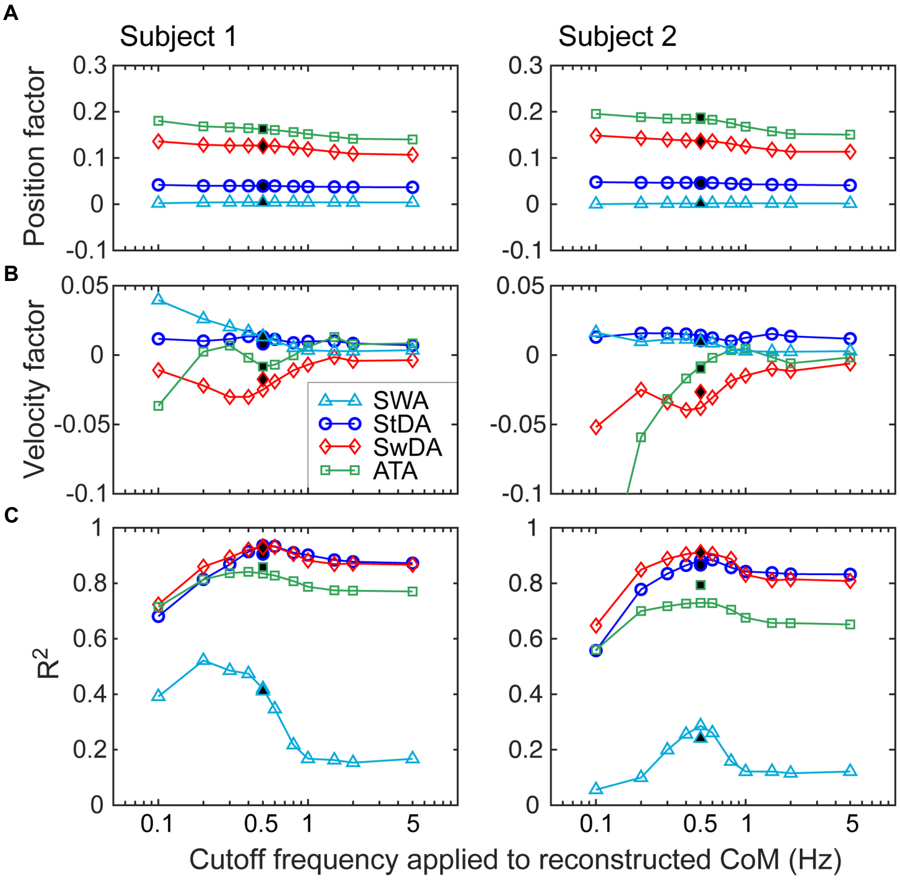

FIGURE 10
Investigation of the effects of the choice of the cutoff frequency of a lowpass filter applied to CoM stimulus-evoked motion on the calculation of position **(A)** and velocity **(B)** regression factors that relate CoM motion to asymmetry measures. Data from two different subjects are shown from eyes-closed, metronome-paced tests with 3° peak-to-peak surface-tilt stimuli. Lowpass filtering with cutoff frequencies ranging from 0.1 to 5Hz was applied to the separated stimulus-evoked CoM sway (see Figure 9) before performing regression analysis to calculate CoM angular position and velocity factors. **(C)** R^2 values indicating the variance accounted for by the regression analysis varied with the cutoff frequency. Filled points at 0.5Hz are from the normal analysis that did not use the separated stimulus-evoked CoM sway.

was in a similar direction to the cycle-averaged ΔCoM body sway response to the perturbation consistent with a step-to-step change in ankle torque that compensated for the stimulus-evoked body lean.

Less well appreciated is the potential for step timing regulation to contribute to dynamic balance control. Studies of quadrupedal robots have demonstrated that ML balance during gait can be achieved entirely by regulating step timing regulated by limb loading and unloading (43, 44). Step timing changes have been noted in experimental studies in cats and humans (21, 40, 41). Control can be achieved by regulating the duration of the stance phase and/or the swing phase of gait. For a rightward bias in body orientation increasing the duration of the right leg stance phase will lengthen the time over which a gravity induced torque toward the left is present. Reducing the duration of the left leg stance phase will shorten the time over which a rightward gravity torque is present. Both of these actions would compensate for a rightward bias in body orientation. Figure 6 shows a change in *StDA* in response to the PRTS perturbation-induced body tilt demonstrating this corrective action. For example, around time 10 s the cycle-averaged ΔCoM body sway had a positive deviation (indicating body tilt toward the right) and the *StDA* had a corresponding positive amplitude indicating a longer right leg stance duration compared to the left. The longer right leg stance duration provided a longer duration over which a leftward directed gravity torque could act to compensate for the rightward lean.

A shorter right leg swing duration would be consistent with either extending the duration of the previous right leg stance phase or ensuring an earlier foot contact time on the next right leg stance phase. Both scenarios would extend the time over which a leftward gravity induced torque was present during right leg stance. Lengthening the left leg swing duration would similarly contribute to reducing the left leg stance phase. Thus, a combination of a shorter right leg and longer left leg swing phases would contribute to compensation for a rightward biased body orientation during gait. Figure 6 shows modulation of cycle-averaged *SwDA* consistent with changes in swing duration that contributed to compensation for changes in the stimulus-evoked ΔCoM.

One might consider that metronome paced stepping would result in a perfect correlation between *StDA* and *SwDA* measures such that they would be completely redundant. But even if a subject's heel strikes were perfectly synchronized with the metronome, different segments of the stepping cycle contribute to the *StDA* and *SwDA* calculations that correspond to approximately the same time point. For example,

TABLE 1 Comparison of standard deviation (SD) of asymmetry measures and coefficient of variation (CV) of gait measures in young versus old subjects on self-paced and non-perturbed stepping-in-place tests performed with eyes open.

	Young mean (SD)	Old mean (SD)	*t* Test value	*p* Value
SWA SD	0.0172 (0.0038)	0.0178 (0.00326)	−0.532	0.598
Step Width CV	0.0503 (0.0189)	0.0468 (0.00940)	0.741	0.463
StDA SD	0.0188 (0.00402)	0.0202 (0.00333)	−1.17	0.249
Stance CV	0.0424 (0.0120)	0.0411 (0.00726)	0.428	0.671
SwDA SD	0.0599 (0.0202)	0.0635 (0.0160)	−0.618	0.540
Swing CV	0.119 (0.0530)	0.119 (0.0345)	0.041	0.968
ATA SD	0.0876 (0.0248)	0.123 (0.0722)	−2.08	**0.044**

SWA: Step Width Asymmetry, StDA: Stance Duration Asymmetry, SwDA: Swing Duration Asymmetry, ATA: Ankle Torque Asymmetry. Measures with bolded *p* values are significant at $p < 0.05$.

TABLE 2 Comparison of standard deviation (SD) of asymmetry measures and coefficient of variation (CV) of gait measures in young versus old subjects on self-paced and non-perturbed stepping-in-place tests performed with eyes closed.

	Young mean (SD)	Old mean (SD)	*t* Test value	*p* Value
SWA SD	0.0202 (0.00538)	0.021 (0.00409)	−0.871	0.389
Step width CV	0.0537 (0.0126)	0.0543 (0.00931)	−0.161	0.873
StDA SD	0.0239 (0.00469)	0.0322 (0.0131)	−2.66	**0.012**
Stance CV	0.0478 (0.00884)	0.0521 (0.0139)	−1.18	0.245
SwDA SD	0.0693 (0.0161)	0.0990 (0.0315)	−3.74	**<0.0001**
Swing CV	0.123 (0.0333)	0.153 (0.0466)	−2.36	**0.024**
ATA SD	0.130 (0.0398)	0.193 (0.112)	−2.38	**0.022**

SWA: Step Width Asymmetry, StDA: Stance Duration Asymmetry, SwDA: Swing Duration Asymmetry, ATA: Ankle Torque Asymmetry. Measures with bolded *p* values are significant at $p < 0.05$.

times t_{StDA2} in Figure 4 and t_{SwDA1} in Figure 5 both occur at approximately the middle of the double support phase near the beginning of the second right leg stance segment. The $StDA_2$ value depends on the duration of the second right and the first left leg stance durations (StD_3 and StD_2). But, assuming heel strike synchronization with the metronome, the $SwDA_1$ value depends on the swing duration associated with the first right leg gait segment (i.e., SwD_1 which depends on the first right leg stance duration StD_1) and the first left leg gait segment (i.e., SwD_2 which depends on the first left leg stance duration StD_2). Thus, the *StDA* and *SwDA* measures will not be fully redundant since they are a function of different gait segments. Additionally, the possibility exists that stance and swing duration could be driven by separate control mechanisms particularly under conditions where there is no metronome pacing constraining segments of the gait cycle.

4.2. Asymmetry measures

The above descriptions of the four dynamic balance control mechanisms we considered emphasize the complementary nature of actions of the two legs which suggested that asymmetry measures which compared metrics from two adjacent steps could provide useful quantification of the contribution of these mechanisms to dynamic balance control. Various asymmetry measures are widely employed in gait studies (74–77). A common measure, referred to as a symmetry index (SI) measure (78), compares the difference between two gait measures to their mean value. Our *SWA*, *StDA*, and *SwDA* measures are of this form except that we chose to divide the difference by the sum rather than the mean since this affords some desirable features (74). Our *ATA* measure appears to differ from this form since the numerator is the sum of two measures proportional to ankle torque. However, while the two numerator terms have the same sign, one represents an eversion torque and the other an inversion torque. Therefore, in terms of ankle-referenced torque measures their signs are opposite.

While our asymmetry measures could have been calculated without regard to their having any relationship to body motion, our calculations were formulated with the desired property that the sign of the asymmetry measures would be indicative of step-to-step changes that compensated for deviations in ML CoM body orientation during gait. With the exception of the *ATA* calculation, it was necessary to consider which foot made the first step. With our definitions all of the asymmetry measures had positive (negative) signs when the balance control mechanisms they represented were compatible with generating corrective actions that compensated for rightward (leftward) deviations of ML body orientation during gait.

It is important to point out that the asymmetry measures do not provide a measure of the magnitude of the corrective action (torque) because (1) they are normalized measures and (2) they are based on measures associated with a single point in time with that time being the average of two discrete time points. However, for a particular asymmetry measure, a larger value indicates that a greater balance correction was made by the mechanism that that asymmetry measure represents. We consider that the regression analysis will be most useful in providing insight into dynamic balance control variations among people, between different subject groups, and as a function of environmental conditions. For example, constraining changes in step width or the ability to generate ankle torque would be expected to result in increased contributions from mechanisms that were not constrained (33, 73). The increased contributions would be represented by larger regression coefficient values that relate CoM motion to asymmetry measures for those measures that substituted for the constrained mechanisms.

The example data shown in Figure 6 from a metronome-paced trial demonstrated that an ML surface-tilt perturbation evoked deviation from an upright orientation during SiP. All four asymmetry measures demonstrated that step width, stance duration, swing duration, and ankle torque mechanisms were all contributing to dynamic balance control. Furthermore, the cyclic nature of the applied pseudorandom stimuli allowed for across-cycle averaging of CoM sway and asymmetry measures (Figure 6B). These averaged results could be further analyzed to calculate regression factors relating CoM sway angle and angular velocity to the asymmetry measure (Figure 7) to give insight into what dynamic aspects of CoM sway (i.e., position and/or velocity sensitivity) were associated with changes in a balance control mechanism. The application of cyclic stimuli to evoke changes in body orientation had the advantage that the analysis could average measures across cycles to reduce variability. However, the similar regression results obtained without averaging (Figure 8A) suggested that reliable results could be obtained without averaging.

Cycle averaging and CoM regression analysis can also be applied to data obtained in non-metronome paced trials when care is taken to select an appropriate lowpass cutoff frequency for filtering the CoM signal but with the recognition that the cutoff frequency has some effect on the calculated asymmetry values (Figure 10).

Additionally, with metronome pacing with an even integer number of step cycles per PRTS stimulus cycle (or other stimuli with odd-harmonic properties where the second half of the stimulus is the inverse of the first half), it is possible to separate the stimulus-evoked sway from the oscillatory component at the stepping frequency if the subject is able to keep pace with the metronome (Figure 9). This separation would allow a detailed investigation of the stimulus-evoked sway at frequencies both below and above the stepping frequency. Such an analysis would not be possible with non-metronome paced tests.

4.3. Asymmetry variability in non-perturbed conditions

Good correlations between the SD of asymmetry and conventional CV measures were observed during unperturbed SiP (Supplementary Figure S2). If variability measures during walking show similar correlations between CV measures and asymmetry SD measures, then asymmetry SD measures could provide an equally informative indicator of fall risk and balance degradation due to aging and neurological deficits as demonstrated in numerous studies (24, 68–72, 79–83). If asymmetry SD measures during SiP were correlated with CV measures during walking gait, then this could support using a SiP protocol to evaluate fall risk and balance degradation since a SiP paradigm is relatively simple to implement.

The demonstration that our asymmetry measures were modulated by CoM motion in both perturbed and unperturbed SiP (Figures 6–8) provides evidence that these asymmetry measures are likely indicative of the contribution of various mechanisms to balance control during gait. Thus, covariation of conventional CV and asymmetry SD measures could provide a bridge that links CV measures to specific balance control mechanisms rather than just being an indicator of the quality of balance control. In this way our asymmetry analyses may provide additional insights into what specific deficits in balance control mechanisms are producing increased gait variability.

By analogy to standing balance control, body sway variability can arise from sensory, motor, and central processing noise sources but is also influenced by the dynamic properties of the overall control system (84, 85). While spontaneous sway variability during stance provides some indication of overall balance system behavior, the application of external perturbations and the use of system identification methods that are appropriate for control systems where feedback plays a major role (86) can enhance our ability to identify which components of the system are responsible for balance deficits (87). While some aspects of standing balance control are likely relevant to gait, the existence of multiple mechanisms contributing to balance during gait, the necessary coordination between these mechanisms, and the shifting of control action from one leg to the other poses a challenge to fully account for balance control during gait. Additional considerations regarding the role of feedforward control, as used in visually-guided step placement (88), are needed for a more complete understanding of balance control during gait.

4.4. Limitations

In this primarily methods-based report we defined asymmetry measures to characterize the contributions of four different mechanisms to dynamic balance control in the frontal plane. The measures were developed to quantify results from SiP tests that were obtained in a group of 20 younger and 20 older adults tested under a variety of conditions. The limited example data we presented to illustrate our methods show representative results but final conclusions require publication of the full study which is in preparation.

The literature using a SiP paradigm to investigate frontal plane balance control is limited but suggests there are similarities between SiP and forward walking gait (18–20). However, SiP is not walking and there are balance mechanisms that contribute to frontal plane balance control that involve forward walking [AP/ML crosstalk associated with push-off torque (4, 8, 35, 38) and steering control (39)]. Therefore, even if our asymmetry measures provide an excellent characterization of mechanisms contributing to frontal plane balance during SiP they will be incomplete as regards the full set of mechanisms contributing to frontal plane balance during walking. Additionally, a comparison of asymmetry measures from SiP and walking will be necessary to determine the extent to which SiP results are similar to or different from walking. A preliminary analysis of data we have collected during forward walking gait indicates that the methods developed for SiP analysis also apply to walking gait but a detailed comparison requires a full analysis.

Subjects performing SiP also needed to control their sagittal plane balance. But none of our asymmetry measures provided any information about sagittal plane balance control. The absence of forward motion during SiP reduces the likelihood that any characterization of sagittal plane balance during SiP would be relevant to mechanisms contributing to sagittal plane balance during walking.

We demonstrated that metronome pacing facilitates some types of analysis (Figures 6, 7, 9) but is an unnatural condition that may affect results. It is currently unknown the extent to which metronome pacing influences results from SiP tests.

4.5. Future directions

We suggest that use of a SiP protocol is under-explored and may be a useful paradigm for investigating the contributions of mechanisms that are known to be important contributors to frontal plane balance during gait (i.e., step width and ankle torque) and less well understood contributions from variations in step timing. The experimental equipment needed for SiP tests is less complex than needed for overground walking and for treadmill walking especially if perturbations such as tilting of the surface are found to be informative. SiP tests can readily be performed with eyes closed to provide a focus on the contributions of proprioceptive and vestibular cues for balance control. Anecdotal reports suggest that eyes closed walking is uncomfortable for subjects and not possible with treadmill walking. However, the utility of a SiP protocol needs to be demonstrated in comparison with better-known paradigms, such as treadmill walking, in experiments that similarly explore balance control mechanisms under a variety of conditions (varying pace, varying mean step width, application of perturbations such as visual motion and externally applied forces, galvanic vestibular stimulation). If SiP results correspond well with results from walking gait, the SiP protocol could have an important future role in clinical evaluations.

We demonstrated good correlations between traditional variability measures and variability of our asymmetry measures from non-metronome paced SiP tests performed with no perturbation. Additionally, our asymmetry variability measures were better able to distinguish between younger and older adults than traditional measures (Tables 1, 2). Because traditional measures based on walking gait have previously been shown to relate to aging, falls, and various neurological disorders as described in the Introduction, it would be important to know if (1) quantification of walking gait variability using asymmetry measures rather than traditional measures would be better at distinguishing between groups; and (2) if asymmetry variability from SiP tests could be equally or possibly more effective in distinguishing between groups than variability measures from walking gait. The latter result would support the clinical use of SiP testing and quantification based on the variability of easily measured asymmetry variables to screen patients for gait-related balance deficits.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary material, further inquiries can be directed to the corresponding author/s.

Author contributions

RP contributed the original conception and design of the study. RP, AG-F, and PH contributed to algorithm development. RP and AG-F contributed to collection, analysis, and interpretation of the data. RP wrote the first draft. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

Supplementary material

The Supplementary material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fneur.2023.1145283/full#supplementary-material

References

1. Neptune RR, Vistamehr A. Dynamic balance during human movement: measurement and control mechanisms. *J Biomech Eng.* (2019) 141:0708011–07080110. doi: 10.1115/1.4042170

2. Winter DA. Human balance and posture control during standing and walking. *Gait Posture.* (1995) 3:193–214. doi: 10.1016/0966-6362(96)82849-9

3. McGeer T. Passive dynamic walking. *Int J Robot Res.* (1990) 9:62–82. doi: 10.1177/027836499000900206

4. O'Connor SM, Kuo AD. Direction-dependent control of balance during walking and standing. *J Neurophysiol.* (2009) 102:1411–9. doi: 10.1152/jn.00131.2009

5. Kuo AD. Stabilization of lateral motion in passive dynamic walking. *Int J Robot Res.* (1999) 18:917–30. doi: 10.1177/02783649922066655

6. Hof AL, van Bockel RM, Schoppen T, Postema K. Control of lateral balance in walking. Experimental findings in Normal subjects and above-knee amputees. *Gait Posture.* (2007) 25:250–8. doi: 10.1016/j.gaitpost.2006.04.013

7. MacKinnon CD, Winter DA. Control of whole body balance in the frontal plane during human walking. *J Biomech.* (1993) 26:633–44. doi: 10.1016/0021-9290(93)90027-C

8. Fettrow T, Reimann H, Grenet D, Thompson E, Crenshaw J, Higginson J, et al. Interdependence of balance mechanisms during bipedal locomotion. *PLoS One.* (2019) 14:e0225902. doi: 10.1371/journal.pone.0225902

9. Hof AL. The 'Extrapolated Center of Mass' concept suggests a simple control of balance in walking. *Hum Mov Sci.* (2008) 27:112–25. doi: 10.1016/j.humov.2007.08.003

10. Lord SR, Rogers MW, Howland A, Fitzpatrick R. Lateral stability, sensorimotor function and falls in older people. *J Am Geriatr Soc*. (1999) 47:1077–81. doi: 10.1111/j.1532-5415.1999.tb05230.x

11. Maki BE, Edmondstone MA, McIlroy WE. Age-related differences in laterally directed compensatory stepping behavior. *J Gerontol A Biol Sci Med Sci*. (2000) 55A:M270–7.

12. Williams HG, McClenaghan BA, Dickerson J. Spectral characteristics of postural control in elderly individuals. *Arch Phys Med Rehabil*. (1997) 78:737–44. doi: 10.1016/S0003-9993(97)90082-4

13. Maki BE, McIlroy WE. Postural control in the older adult. *Clin Geriatr Med*. (1996) 12:635–58. doi: 10.1093/gerona/55.5.m270

14. Maki BE, McIlroy WE, Perry SD. Influence of lateral destabilization on compensatory stepping responses. *J Biomech*. (1996) 29:343–53. doi: 10.1016/0021-9290(95)00053-4

15. Hilliard MJ, Martinez KM, Janssen I, Edwards B, Mille M-L, Zhang Y, et al. Lateral balance factors predict future falls in community-living older adults. *Arch Phys Med Rehabil*. (2008) 89:1708–13. doi: 10.1016/j.apmr.2008.01.023

16. Zhang YB, Wang WQ. Reliability of the Fukuda stepping test to determine the side of vestibular dysfunction. *J Int Med Res*. (2011) 39:1432–7. doi: 10.1177/147323001103900431

17. Agathos CP, Bernardin D, Baranton K, Assaiante C, Isableu B. Drifiting while stepping in place in old adults: Association of Self-Motion Perception with reference frame reliance and ground optic flow sensitivity. *Neuroscience*. (2017) 347:134–47. doi: 10.1016/j.neuroscience.2017.01.044

18. Brenière Y. Why we walk the way we do. *J Mot Behav*. (1996) 28:291–8. doi: 10.1080/00222895.1996.10544598

19. Breniere Y, Ribreau C. A double-inverted pendulum model for studying the adaptability of postural control to frequency during human stepping in place. *Biol Cybern*. (1998) 79:337–45. doi: 10.1007/s004220050483

20. Garcia RK, Nelson AJ, Ling W, Van Olden C. Comparing stepping-in-place and gait ability in adults with and without hemiplegia. *Arch Phys Med Rehabil*. (2001) 82:36–42. doi: 10.1053/apmr.2001.19012

21. Hof AL, Vermerris SM, Gjaltema WA. Balance responses to lateral perturbations in human treadmill walking. *J Exp Biol*. (2010) 213:2655–64. doi: 10.1242/jeb.042572

22. Townsend MA. Biped gait stabilization via foot placement. *J Biomech*. (1985) 18:21–38. doi: 10.1016/0021-9290(85)90042-9

23. Bauby CE, Kuo AD. Active control of lateral balance in human walking. *J Biomech*. (2000) 33:1433–40. doi: 10.1016/S0021-9290(00)00101-9

24. Dean JC, Alexander NB, Kuo AD. The effect of lateral stabilization on walking in young and old adults. *IEEE Trans Biomed Eng*. (2007) 54:1919–26. doi: 10.1109/TBME.2007.901031

25. Donelan JM, Shipman DW, Kram R, Kuo AD. Mechanical and metabolic requirements for active lateral stabilization in human walking. *J Biomech*. (2004) 37:827–35. doi: 10.1016/j.jbiomech.2003.06.002

26. Redfern MS, Furman JM. Portural sway of patients with vestibular disorders during optic flow. *J Vestib Res*. (1994) 4:221–30.

27. van Leeuwen AM, van Dieen JH, Daffertshofer A, Bruijn SM. Active foot placement control ensures stable gait: effect of constraints on foot placement and ankle moments. *PLoS One*. (2020) 15:e0242215. doi: 10.1371/journal.pone.0242215

28. Bruijn SM, van Dieën JH. Control of human gait stability through foot placement. *J R Soc Interface*. (2018) 15:20170816. doi: 10.1098/rsif.2017.0816

29. Arvin M, Hoozemans MJM, Pijnappels M, Duysens J, Verschueren SM, van Dieën JH. Where to step? Contribution of stance leg muscle spindle afference to planning mediolateral foot placement for balance control in young and old adults. *Front Physiol*. (2018) 9:1134. doi: 10.3389/fphys.2018.01134

30. Matsusaka N. Control of the medial-lateral balance in walking. *Acta Orthop Scand*. (1986) 57:555–9. doi: 10.3109/17453678609014793

31. Pandy MG, Lin Y-C, Kim HJ. Muscle coordination of mediolateral balance in Normal walking. *J Biomech*. (2010) 43:2055–64. doi: 10.1016/j.jbiomech.2010.04.010

32. Hof AL, Duysens J. Responses of human ankle muscles to mediolateral balance perturbations during walking. *Hum Mov Sci*. (2018) 57:69–82. doi: 10.1016/j.humov.2017.11.009

33. van den Bogaart M, Bruijn SM, Spildooren J, van Dieen JH. The effect of constraining mediolateral ankle moments and foot placement on the use of the counter-rotation mechanism during walking. *J Biomech*. (2022) 136:111073. doi: 10.1016/j.jbiomech.2022.111073

34. Reimann H, Fettrow TD, Thompson ED, Agada P, McFadyen BJ, Jeka JJ. Complementary mechanisms for upright balance during walking. *PLoS One*. (2017) 12:e0172215. doi: 10.1371/journal.pone.0172215

35. Reimann H, Fettrow T, Thompson ED, Jeka JJ. Neural control of balance during walking. *Front Physiol*. (2018) 9:1271. doi: 10.3389/fphys.2018.01271

36. Reimann H, Fettrow T, Grenet D, Thompson ED, Jeka JJ. Phase-dependency of medial-lateral balance responses to sensory perturbations during walking. *Front Sports Act Living*. (2019) 1:25. doi: 10.3389/fspor.2019.0025

37. Fettrow T, DiBianca S, Vanderlinde dos Santos F, Reimann H, Jeka J. Flexible recruitment of balance mechanisms to environmental constraints during walking. *Front Virtual Real*. (2020) 1:5. doi: 10.3389/frvir.2020.00005

38. Kim M, Collins SH. Once-per-step control of ankle-foot prosthesis push-off work reduces effort associated with balance during walking. *J Neuroeng Rehabil*. (2015) 12:43. doi: 10.1186/s12984-015-0027-3

39. Rebula JR, Ojeda LV, Adamczyk PG, Kuo AD. The stabilizing properties of foot yaw in human walking. *J Biomech*. (2017) 53:1–8. doi: 10.1016/j.jbiomech.2016.11.059

40. Karayannidou A, Zelenin PV, Orlovsky GN, Sirota MG, Beloozerova IN, Deliagina TG. Maintenance of lateral stability during standing and walking in cats. *J Neurophysiol*. (2009) 101:8–19. doi: 10.1152/jn.90934.2008

41. Matjačić Z, Zadravec M, Olenšek A. An effective balancing response to lateral perturbations at pelvis level during slow walking requires control in all Thress planes of motion. *J Biomech*. (2017) 60:79–90. doi: 10.1016/j.jbiomech.2017.06.020

42. Buurke TJW, Lamoth CJC, van der Woude LHV, Hof AL, den Otter R. Bilateral temporal control determines Mediolatera margins of stability in symmetric and asymmetric human walking. *Sci Rep*. (2019) 9:1. doi: 10.1038/s41598-019-49033-z

43. Maufroy C, Kimura H, Takase K. Integration of posture and rhythmic motion controls in quadrupedal dynamic walking using phase modulations based on leg loading/unloading. *Auton Robot*. (2010) 28:331–53. doi: 10.1007/s10514-009-9172-5

44. Maufroy C, Kimura H, Nishikawa T. Stable dynamic Walkiing of the quadruped "Kotetsu" using phase modulations based on leg loading/unloading against lateral perturbations. In *IEEE International Conference on Robotics and Automation* (2012): pp. 1883–1888.

45. Wang Y, Srinivasan M. Stepping in the direction of the fall: the next foot placement can be predicted from the current upper body state in steady-state walking. *Biol Lett*. (2014) 10:20140405. doi: 10.1098/rsbl.2014.0405

46. Hurt CP, Rosenblatt N, Crenshaw JR, Grabiner MD. Variation in trunk kinematics influences variation in step width during treadmill walking by older and younger adults. *Gait Posture*. (2010) 31:461–4. doi: 10.1016/j.gaitpost.2010.02.001

47. Vlutters M, van Asseldonk EHF, van der Kooij H. Center of Mass Velocity-Based Predictions in balance recovery following pelvis perturbations during human walking. *J Exp Biol*. (2016) 219:1514–23. doi: 10.1242/jeb.129338

48. Joshi V, Srinivasan M. A controller for walking derived from how humans recover from perturbations. *J R Soc Interface*. (2019) 16:20190027. doi: 10.1098/rsif.2019.0027

49. Matjačić Z, Zadravec M, Olenšek A. Influence of treadmill speed and perturbation intensity on selection of balancing strategies during slow walking perturbed in the frontal plane. *Appl Bionics Biomech*. (2019) 2019:1046459:1–14. doi: 10.1155/2019/1046459

50. Fettrow T, Reimann H, Grenet D, Crenshaw J, Higginson J, Jeka J. Walking cadence affects the recruitment of the medial-lateral balance mechanisms. *Front Sports Act Living*. (2019) 1:40. doi: 10.3389/fspor.2019.00040

51. Franz JR, Francis CA, Allen MS, O'Connor SM, Thelen DG. Advanced age brings a greater reliance on visual feedback to maintain balance during walking. *Hum Mov Sci*. (2015) 40:381–92. doi: 10.1016/j.humov.2015.01.012

52. Franz JR, Francis CA, Allen MS, Thelen DG. Visuomotor entrainment and the frequency-dependent response of walking balance to perturbations. *IEEE Trans Neural Syst Rehabil Eng*. (2017) 25:1135–42. doi: 10.1109/TNSRE.2016.2603340

53. Qiao M, Feld JA, Franz JR. Aging effects on leg joint variability during walking with balance perturbations. *Gait Posture*. (2018) 62:27–33. doi: 10.1016/j.gaitpost.2018.02.020

54. Warren WH, Kay BA, Yilmaz EH. Visual control of posture during walking: functional specificity. *J Exp Psychol Hum Percept Perform*. (1996) 22:818–38. doi: 10.1037/0096-1523.22.4.818

55. McAndrew PM, Dingwell JB, Wilken JM. Walking variability during continuous pseudo-random oscillations of the support surface and visual field. *J Biomech*. (2010) 43:1470–5. doi: 10.1016/j.jbiomech.2010.02.003

56. Hausdorff JM, Cudkowicz ME, Firtion R, Wei JY, Goldberger AL. Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. *Mov Disord*. (1998) 13:428–37.

57. Hausdorff JM, Edelberg HK, Mitchell SL, Goldberger AL, Wei JY. Increased gait unsteadiness in community-dwelling elderly fallers. *Arch Phys Med Rehabil*. (1997) 78:278–83. doi: 10.1016/S0003-9993(97)90034-4

58. Brach JS, Berlin JE, VanSwearingen JM, Newman AB, Studenski SA. Too much or too little step width variability is associated with a fall history in older persons who walk at or near Normal gait speed. *J Neuroeng Rehabil*. (2005) 2:21. doi: 10.1186/1743-0003-2-21

59. Hausdorff JM, Nelson ME, Kaliton D, Layne JE, Bernstein MJ, Nuernberger A, et al. Etiology and modification of gait instability in older adults: a randomized controlled trial of exercise. *J Appl Physiol*. (2001) 90:2117–29. doi: 10.1152/jappl.2001.90.6.2117

60. Hausdorff JM. Gait variability: methods, modeling and meaning. *J Neuroeng Rehabil*. (2005) 2:19. doi: 10.1186/1743-0003-2-19

61. Hausdorff JM, Lowenthal J, Herman T, Gruendlinger L, Peretz C, Giladi N. Rhythmic auditory stimulation modulates gait variability in Parkinson's disease. *Eur J Neurosci*. (2007) 26:2369–75. doi: 10.1111/j.1460-9568.2007.05810.x

62. Hausdorff JM, Rios DA, Edelberg HK. Gait variability and fall risk in community-living older adults: a 1-year prospective study. *Arch Phys Med Rehabil*. (2001) 82:1050–6. doi: 10.1053/apmr.2001.24893

63. Davies WDT. *System Identification for Self-adaptive Control*. London: Wiley-Interscience (1970).

64. Hausdorff JM. Footswitch system for measurement of the temporal parameters of gait. *J Biomech*. (1995) 28:347–51. doi: 10.1016/0021-9290(94)00074-E

65. Peterka RJ, Murchison CF, Parrington L, Fino PC, King LA. Implementation of a central sensorimotor integration test for characterization of human balance control during stance. *Front Neurol*. (2018) 9:1045. doi: 10.3389/fneur.2018.01045

66. Winter DA. *Biomechanics and Motor Control of Human Movement*. *3rd* ed. New York, NY: John Wiley & Sons, Inc. (2005).

67. Goodworth AD, Peterka RJ. Sensorimotor integration for multisegmental frontal plane balance control in humans. *J Neurophysiol*. (2012) 107:12–28. doi: 10.1152/jn.00670.2010

68. Brach JS, Studenski S, Perera S, VanSwearingen JM, Newman AB. Stance time and step width variability have unique contributing impairments in older persons. *Gait Posture*. (2008) 27:431–9. doi: 10.1016/j.gaitpost.2007.05.016

69. Brach JS, Studenski SA, Perera S, VanSwearingen JM, Newman AB. Gait variability and the risk of incident mobility disability in community-dwelling older adults. *J Gerontol Med Sci*. (2007) 62A:983–8. doi: 10.1093/gerona/62.9.983

70. Owings TM, Grabiner MD. Step width variability, but not step length variability or step time variability, discriminates gait of healthy young and older adults during treadmill locomotion. *J Biomech*. (2004) 37:935–8. doi: 10.1016/j.jbiomech.2003.11.012

71. Francis CA, Franz JR, O'Connor SM, Thelen DG. Gait variability in healthy old adults is more affected by a visual Perturbatin than by a cognitive or narrow step placement demand. *Gait Posture*. (2015) 42:380–5. doi: 10.1016/j.gaitpost.2015.07.006

72. Wuehr M, Schniepp R, Schlick C, Huth S, Pradhan C, Dieterich M, et al. Sensory loss and walking speed related factors for gait alterations in patients with peripheral neuropathy. *Gait Posture*. (2014) 39:852–8. doi: 10.1016/j.gaitpost.2013.11.013

73. Perry JA, Srinivasan M. Walking with wider steps changes foot placement control, increases kinematic variability and does not improve linear stability. *R Soc Open Sci*. (2017) 4:160627. doi: 10.1098/rsos.160627

74. Alves SA, Ehrig RM, Raffalt PC, Bender A, Duda GN, Agres AN. Quantifying asymmetry in gait: the weighted universal symmetry index to evaluate 3d ground reaction forces. *Front Bioeng Biotechnol*. (2020) 8:579511. doi: 10.3389/fbioe.2020.579511

75. Plotnik M, Wagner JM, Adusumilli G, Gottlieb A, Naismith RT. Gait asymmetry and bilateral coordination of gait during a six-minute walk test in persons with multiple sclerosis. *Sci Rep*. (2020) 10:12382. doi: 10.1038/s41598-020-68263-0

76. Tabor P, Iwanska D, Grabowska O, Karczewska-Lindinger M, Popieluch A, Mastalerz A. Evaluation of selected indices of gait asymmetry for the assessment of running asymmetry. *Gait Posture*. (2021) 86:1–6. doi: 10.1016/j.gaitpost.2021.02.019

77. Blazkiewicz M, Wiszomirska I, Wit A. Comparison of four methods of calculating the symmetry of spatial-temporal parameters of gait. *Acta Bioeng Biomech*. (2014) 16:29–35. doi: 10.5277/abb140104

78. Robinson RO, Herzog W, Nigg BM. Use of force platform variables to quantify the effects of chiropractic manipulation on gait symmetry. *J Manip Physiol Ther*. (1987) 10:172–6.

79. Plotnik M, Giladi N, Balash Y, Peretz C, Hausdorff JM. Is freezing of gait in Parkinson's disease related to asymmetric motor function? *Ann Neurol*. (2005) 57:656–63. doi: 10.1002/ana.20452

80. Plotnik M, Giladi N, Hausdorff JM. A new measure for quantifying the bilateral coordination of human gait: effects of aging and Parkinson's disease. *Exp Brain Res*. (2007) 181:561–70. doi: 10.1007/s00221-007-0955-7

81. Hausdorff JM. Gait dynamics in Parkinson's disease: common and distinct behavior among stride length, gait variability, and fractal-like scaling. *Chaos*. (2009) 19:026113. doi: 10.1063/1.3147408

82. Pieruccini-Faria F, Black SE, Masellis M, Smith EE, Almeida QJ, Li KZH, et al. Gait variability across neurodegenerative and cognitive disorders: results from the Canadian consortium of neurodegeneration in aging (Ccna) and the gait and brain study. *Alzheimers Dement*. (2021) 17:1317–28. doi: 10.1002/alz.12298

83. Lord S, Howe T, Greenland J, Simpson L, Rochester L. Gait variability in older adults: a structured review of testing protocol and Clinimetric properties. *Gait Posture*. (2011) 34:443–50. doi: 10.1016/j.gaitpost.2011.07.010

84. van der Kooij H, Peterka RJ. Non-linear stimulus-response behavior of the human stance control system is predicted by optimization of a system with sensory and motor noise. *J Comput Neurosci*. (2011) 30:759–78. doi: 10.1007/s10827-010-0291-y

85. Campbell KR, King LA, Parrington L, Fino PC, Prokopios A, Peterka RJ. Central sensorimotor integration assessment reveals deficits in standing balance control in people with chronic mild traumatic brain injury. *Front Neurol*. (2022) 13:897454. doi: 10.3389/fneur.2022.897454

86. van der Kooij H, van Der Helm FCT. Observations from unperturbed closed loop systems cannot indicate causality. *J Physiol*. (2005) 569:705–6. doi: 10.1113/jphysiol.2005.569001

87. Pasma JH, Engelhart D, Schouten AC, van der Kooij H, Maier AB, Meskers CGM. Impaired standing balance: the clinical need for closing the loop. *Neuroscience*. (2014) 267:157–65. doi: 10.1016/j.neuroscience.2014.02.030

88. Matthis JS, Yates JL, Hayhoe MM. Gaze and the control of foot placement when walking in natural terrain. *Curr Biol*. (2018) 28:1224–1233.e5. doi: 10.1016/j.cub.2018.03.008

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Control of center of mass motion during walking correlates with gait and balance in people with incomplete spinal cord injury

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Background: There is evidence that ambulatory people with incomplete spinal cord injury (iSCI) have an impaired ability to control lateral motion of their whole-body center of mass (COM) during walking. This impairment is believed to contribute to functional deficits in gait and balance, however that relationship is unclear. Thus, this cross-sectional study examines the relationship between the ability to control lateral COM motion during walking and functional measures of gait and balance in people with iSCI.

Methods: We assessed the ability to control lateral COM motion during walking and conducted clinical gait and balance outcome measures on 20 ambulatory adults with chronic iSCI (C1-T10 injury, American Spinal Injury Association Impairment Scale C or D). To assess their ability to control lateral COM motion, participants performed three treadmill walking trials. During each trial, real-time lateral COM position and a target lane were projected on the treadmill. Participants were instructed to keep their lateral COM position within the lane. If successful, an automated control algorithm progressively reduced the lane width, making the task more challenging. If unsuccessful, the lane width increased. The adaptive lane width was designed to challenge each participant's maximum capacity to control lateral COM motion during walking. To quantify control of lateral COM motion, we calculated lateral COM excursion during each gait cycle and then identified the minimum lateral COM excursion occurring during five consecutive gait cycles. Our clinical outcome measures were Berg Balance Scale (BBS), Timed Up and Go test (TUG), 10-Meter Walk Test (10MWT) and Functional Gait Assessment (FGA). We used a Spearman correlation analysis (ρ) to examine the relationship between minimum lateral COM excursion and clinical measures.

Results: Minimum lateral COM excursion had significant moderate correlations with BBS (ρ=−0.54, p=0.014), TUG (ρ=0.59, p=0.007), FGA (ρ=−0.59, p=0.007), 10MWT-preferred (ρ=−0.59, p=0.006) and 10MWT-fast (ρ=−0.68, p=0.001).

Conclusion: Control of lateral COM motion during walking is associated with a wide range of clinical gait and balance measures in people with iSCI. This finding suggests the ability to control lateral COM motion during walking could be a contributing factor to gait and balance in people with iSCI.

KEYWORDS

spinal cord injury, clinical balance measures, locomotion, lateral stability, stability

Introduction

The requirements of the nervous system to actively control mediolateral motion of the whole-body center of mass (COM) during walking are substantial in comparison to fore-aft plane of motion that benefit from stabilizing body mechanics (1–3). In particular, the challenges of controlling this mediolateral motion are considerable during the single limb support phase of the gait cycle. Beginning at toe-off, the lateral velocity of the COM is relatively large and directed towards the stance limb. To maintain a straight-ahead walking trajectory, this lateral velocity must be reduced to zero (typically occurring around midstance as the COM position reaches its most lateral excursion and is positioned above the supporting limb), and then redirected towards the midline. Failure to arrest the lateral momentum of the COM will result in motion beyond the lateral base of support border (determined by the stance limb medio-lateral foot-placement). COM travelling beyond the lateral base of support border will require a corrective step(s) to restore the desired forward walking trajectory and prevent a fall. External moments acting in the frontal-plane about the ankle joint of the stance limb work collectively to arrest and then redirect the COM lateral velocity (3). The nervous system will use a combination of anticipatory and reactive control mechanisms to modulate these moments. Thus, the capacity to control lateral COM motion is likely a fundamental component of effective forward walking.

Growing evidence suggests that ambulatory people with incomplete spinal cord injury (iSCI) have considerable challenges controlling their lateral COM motions during walking. This includes difficulty arresting lateral motion after a walking maneuver (4, 5), impaired mediolateral foot placement (6), limited ability to increase lateral margins of stability following a perturbation (7), and sizeable metabolic energy cost to stabilize lateral motion during walking (8). Perhaps as a consequence of these challenges, ambulatory people with iSCI who walk without assistive devices demonstrate a substantially larger lateral COM excursion during walking at preferred and fast speeds when compared to age matched adults without iSCI (5). As such, many of the cautious gait patterns observed in people with iSCI [e.g., slower walking speeds, wider steps, shorter steps, more time in double support (6, 9–12)] have been suggested as compensatory mechanisms that proactively aid in COM control during walking (9, 10, 12).

Studies in populations without neurologic injuries have found that the control of mediolateral COM motion is critical for maintaining dynamic balance (13) and creating walking stability during directional changes (14). Almost all activities of daily living involve directional changes or turning maneuvers that require reorientation of the body in the anticipated direction of travel (15). Therefore, the ability to control lateral COM motion during walking may be a skill fundamental to functional gait and balance. If this relationship is supported, interventions that directly aim to improve the ability to control lateral COM motion may translate to improvements in gait and balance. The first step is to identify if the ability to control lateral COM motion during walking is related to functional gait and balance in people with iSCI.

The ability to control lateral COM motion during walking could be related to several functional measures of gait and balance. We selected four clinical outcome measures, the Functional Gait Assessment (FGA), the Timed Up and Go test (TUG), the 10-meter walk test (10MWT), and the Berg Balance Scale (BBS), that collectively would provide insights into the relationship between the ability to control lateral COM motion during walking and walking balance, walking speed, and postural balance, respectively. People with iSCI have impaired abilities to control lateral motion during walking (4–7), which may reduce functional walking balance on tasks requiring turns and change of gait speed. In the current study, we used the TUG (16–18) and the FGA (19–21) to examine walking balance. Both these tests include turns and changes in walking speed. People with iSCI often select cautious gait patterns, including walking at slower speeds (9, 10, 12), that reduces COM velocity. Slower COM motions are believed to enhance gait stability by decreasing perturbation intensities (9, 12). Thus, it seems likely that there could also be a relationship between the ability to control COM motion during walking and walking speed. We conducted the 10MWT, a widely used and recommended measure to assess gait speed (22–25), and analyzed relationship of preferred and fast 10MWT speeds with the ability to control lateral COM motion. Finally, we used the BBS to evaluate postural balance. The BBS has strong correlations with walking ability in people with iSCI (26–29), suggesting that similar mechanisms may be responsible for controlling COM dynamics during walking and standing. In the current study, we used the BBS to examine if the ability to control lateral COM motion during walking is related to postural balance. While these measures provide an overview of functional gait and balance, it should be noted that they do not provide a direct method to assess the ability to control lateral COM motion during walking.

We are not aware of any studies that have previously aimed to quantify people's capacity to control (minimize) lateral COM motion during walking, however two studies have successfully used visual feedback to encourage people without neurologic injuries to minimize vertical oscillations of the COM during walking (30, 31). Based on these prior studies, to quantify the ability to control lateral COM motion during walking, we developed a treadmill-based assessment that used visual feedback to encourage people to minimize lateral COM motion during forward walking. During this assessment, a target walking lane and the real-time mediolateral position of the participant's COM are projected on the walking surface of an oversized treadmill. Participants are instructed to maintain their mediolateral COM position within the projected lane during walking. If participants maintain their COM within the target lane, the lane width is progressively decreased, increasing the challenge of the walking task. This external visual feedback encouraged participants to try their best to control their COM motion during forward walking. To quantify the participants' capacity to control their lateral COM motion, we performed a post-hoc kinematic analysis to identify the minimum lateral COM excursion occurring during five consecutive gait cycles.

The purpose of this study was to evaluate the relationship between the ability to control lateral COM motion during walking and validated clinical gait and balance measures commonly used to assess ambulatory people with iSCI. We hypothesized that the ability to control lateral COM motion during walking would correlate with clinical gait and balance outcome measure scores.

Materials and methods

Participants

Twenty adults with chronic incomplete spinal cord injury participated in this cross-sectional study. All participants had spinal cord injuries between C1-T10 and were classified as C or D on the American Spinal Injury Association Impairment Scale (AIS). Our inclusion criteria were the following: age between 18 to 80 years, more than 6 months post-incomplete spinal cord injury, medically stable, and able to ambulate 10 m without physical assistance or use of assistive devices. Our exclusion criteria were the following: excessive spasticity in the lower limbs (>3 on the Modified Ashworth Scale), unable to tolerate 10 min of standing, presence of severe cardiovascular and pulmonary disease, unhealed decubiti or other skin compromise, history of recurrent fractures or known orthopedic problems in the lower extremities, concomitant central or peripheral neurological injury, unable to provide informed consent due to cognitive impairments, enrolled in concurrent physical therapy or research involving locomotor training, and use of braces/orthotics crossing the knee joint. The criteria for participation in this study, in particular the ability to walk without assistive devices, will inherently result in a participant sample that is considered high functioning. As our objective was to assess the relationship between the ability of the individual to control lateral COM motion and functional gait and balance, we restricted the use of assistive devices, because devices like canes and walkers will aid in the control of COM motion. We believe the relationship between the ability to control COM motion and functional gait and balance will be greatest in those who walk without assistive devices. Thus, in the current study we limited our study population to individuals who were able to walk without assistive devices.

This study was conducted at the Human Agility Laboratory, Physical Therapy and Human Movement Sciences, Northwestern University Feinberg School of Medicine. The study protocol was approved by the Institutional Review Boards at Northwestern University and the Edward Hines Jr. Veterans Affairs Hospital. All participants provided informed written consent prior to enrollment in the study.

Experimental setup

To assess participants' ability to control their lateral COM motion during walking, we recorded kinematic data as participants walked on an oversized treadmill, walking surface 2.6 × 1.4 m, (TuffTread, Willis, TX) while receiving visual feedback about their lateral COM position. For safety, participants wore a trunk harness attached to passive overhead support that did not provide bodyweight support (Aretech, Ashburn, VA). The harness straps were adjusted to allow participants unrestricted lateral travel across the treadmill. During treadmill walking, participants were not allowed to use any assistive devices (canes, walkers, handrails) except for any passive ankle-foot orthoses they would typically wear during community ambulation. In the event of a loss of balance, a physical therapist providing standby assistance would give manual support as necessary to allow the participant to regain balance and continue walking. During the treadmill-based assessment of participants' ability to control their lateral COM motion, participants were not allowed to use any assistive devices (canes, walkers, handrails) except for any passive ankle-foot orthoses. In addition, we only analyzed walking periods when no manual assistance was provided.

During treadmill walking, we used a 12-camera motion capture system (Qualisys, Gothenburg Sweden) to collect 3D coordinates of 19 reflective markers placed on the pelvis and lower limbs at 100 Hz. Markers were placed at the following locations: S2 vertebrae and bilaterally on each sacroiliac joint, greater trochanter, anterior superior iliac spine, highest point of the iliac crest, lateral malleolus, calcaneus, and the 2nd, 3rd, and 5th metatarsals.

We evaluated participant's capacity to control their lateral COM motion during three treadmill walking trials that were each 21 m in length. During the trials participants were given visual feedback of their lateral COM position to challenge them to minimize their lateral motion during walking (Supplementary video S1). Specifically, their real-time mediolateral COM position was represented by a white line projected along the length of the treadmill surface using a short throw projector mounted on the wall alongside the treadmill (Hitachi, Tokyo, Japan). The lateral COM position was calculated using real-time 3D locations of the pelvis markers that were streamed to a custom-programmed control algorithm (LabVIEW, National Instruments, Austin, TX). The control algorithm calculated mediolateral COM position as the midpoint between the two greater trochanter markers (32) and transformed the data into the treadmill coordinate system for display.

Additionally, lateral boundary targets for COM position ("target lane") were projected on the treadmill (Figure 1). To maximally challenge participants to minimize their lateral COM motion during walking, the control algorithm systematically adjusted the width of the target lane based on how successful the participant was at maintaining their lateral COM position within the green target lane (Figure 1). During walking, if the COM moved outside the target lane, that area outside the target turned red to provide an immediate visual cue to return to the green target lane. At the beginning of the first walking trial, the initial lane width was set to 200 mm. Our prior research found that people with iSCI who were able to ambulate with no assistive devices or manual assistance had an average lateral COM excursion per stride of 80 mm (5). Thus, we selected a starting width of 200 mm with the goal that the lane would be sufficiently wide that all participants in this study would be successful at the start of the trial. Once the assessment began, the control algorithm made a 10 mm step change in the lane width based on the following logic: if the participant maintained their lateral COM position in the lane for 1.5 consecutive meters of forward walking, the lane width was decreased by 10 mm. If the participant walked for 3 meters without maintaining COM position within the lane for at least 1.5 consecutive meters, the lane width increased by 10 mm. We selected to make iterative changes in the lane width of 10 mm based on two competing factors. First, we wanted the iterative changes to be large enough that the task would converge on an optimally challenging width for each participant by the participant's second 21 m trial. Second, we wanted the iterative changes to be small enough that the task would adjust gradually with sufficient resolution to capture each participant's optimal ability. Based on these criteria and extensive pilot testing we selected 10 mm iterative changes. A minimum lane width was set at 5 mm based on the resolution of the lines projected on the treadmill. Consecutive walking assessments started at the lane width achieved at the end of the previous trial (e.g., the starting lane width for the second assessment was equal to the ending lane width of the first assessment). The algorithm thresholds were established in pilot testing prior to the current study to minimize walking time yet converge on the smallest lane width participants could maintain.

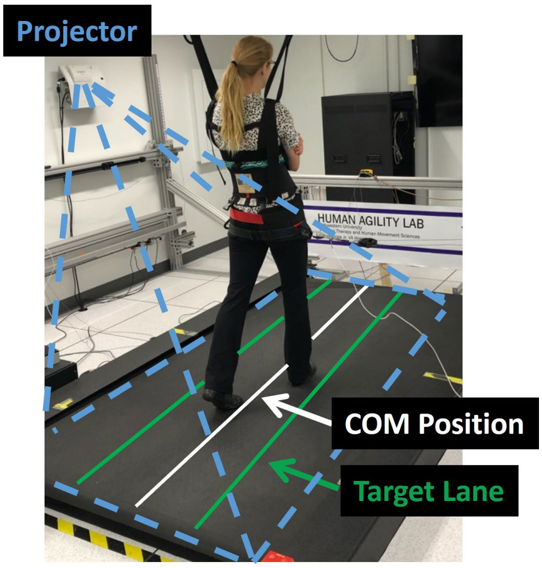

FIGURE 1
Experimental set-up. The laboratory balance assessment is performed three times on an oversized treadmill to test an individual's lateral center of mass (COM) control. The real-time mediolateral COM position of the participant is projected on the treadmill through a white line. Participants are instructed to walk at their preferred treadmill speed and to do their best to keep the white line within the green target lane. If successful, the target lane width is progressively decreased. If not, the area outside of the target lane (to either the left or right) changes to red, providing an immediate visual cue that the participant has made an error and should try to return the white line to the projected increased lane.

We selected 21 m as the distance for each individual treadmill trial length for two reasons. First, we wanted to select a distance that our participants would likely be able to walk continuously without requiring a rest break. Given that the inclusion criteria for the study was the ability to walk 10 m without assistance, we felt that 21 m should be accomplishable for most participants. Second, we wanted to select a distance that was equally divisible by both 1.5 m and 3 m, the distances the algorithm used to evaluate performance and then iteratively update the width of the feedback lane.

We used a fixed walking distance for each trial (21 m) and updated the lane width at fixed intervals of distance traveled (either 1.5 m or 3.0 m) that was consistent across all participants. The purpose of the visual feedback was specifically to encourage participants to reduce their COM excursion. We updated the lane based on the distance traveled to ensure that the number of times the target lane width updated within a trial was consistent across all participants independent of how fast they walked or their stride length.

Clinical outcome measures

Timed Up and Go test (TUG): The TUG is a valid and reliable measure for functional mobility, balance, and fall-risk in people with iSCI (16, 17). It is a widely used and recommended screening tool for prediction of fall-risk (33–35). The participant is asked to rise from the chair, walk 3 meters safely, turn around, walk back, and sit down on the chair. The TUG score is the time recorded from when participants rise from the chair until they sit down again.

Functional Gait Assessment (FGA): The FGA is a valid and reliable test that measures balance and gait functions in people with iSCI (20, 21). It is a modification of the Dynamic Gait Index (19) consisting of 10 items scored on a four-point ordinal scale ranging from 0 to 3, such that 0 indicates severe impairment and 3 indicates normal/no impairment with a total score of 30. The clinical practice guideline recommends usage of FGA as a core outcome for patients with neurological conditions (36).

Berg Balance Scale (BBS): The BBS is a 14-item scale, widely used, valid, and reliable measure for balance assessment of people with iSCI during predetermined tasks performed in daily living (27–29). Each item is scored on a five-point ordinal scale ranging from 0 to 4, such that 0 signifies the lowest level of function and 4 signifies the highest level of function. The maximum total score is 56, with higher total scores indicating better balance (37). The BBS has also been useful as a screening tool to predict risk of falls beyond cut-off scores (38–40).

10-Meter Walk Test (10MWT): The 10MWT has been found to be a valid and reliable test to measure overground walking speed, both preferred and fast, in people with iSCI (17, 41–43). It is a recommended measure for assessment of gait speed among neurological populations (24, 25).

For the clinical measures assessment, participants were allowed to use assistive devices such as cane, rolling walker and/or ankle foot orthosis they would typically wear during community ambulation.

Protocol

Participants in the current study were part of a larger clinical trial investigating a high intensity gait training intervention. As such, all participants first underwent an extensive clinical assessment prior to future enrollment in the intervention (any participation in the gait training intervention occurred 1-week after completing all testing described in the current study). A licensed physical therapist collected demographic information (age, gender, date of birth), date of spinal cord injury, level of spinal cord injury, cause of spinal cord injury, current, and past medical history, current medications (to screen for use of beta blockers which could potentially affect heart rate during the gait training intervention and to evaluate if any medications could have resulted in balance deficits as a side effect), current ambulatory ability in the home and community (including the use of any assistive devices), and self-reported number of falls in the past year. The physical therapist then collected four clinical outcome measures: BBS, TUG, FGA, and 10MWT at preferred and fast speeds.

Next, participants performed the treadmill walking portion of the experiment that was used to assess their ability to control their lateral COM motion. The participant's preferred treadmill walking speed was identified through a staircase method of increasing and decreasing the treadmill speed until the participant's desired speed is confirmed through verbal feedback. Participants were given several minutes to accommodate to walking on the treadmill at this preferred speed.

With the treadmill stopped, participants were then given detailed instructions about the assessment to be performed. The projector used to display the participant's real-time lateral COM position and a target lane was turned on. Participants were instructed to make some small movements to their left and right so that they understood that the side-to-side movement of their body controlled the position of the white line being projected on the treadmill. Participants were asked to perform three walking trials of 21 m each. Participants were instructed to do their best to maintain the white line representing the midline of their body within the target lane. They were also told that if they were successful, the width of the target lane would be progressively reduced. Once the participant understood the instructions, the treadmill was started, and participants were given time to reach steady-state before the assessment began. At the end of the 21 m assessment, the treadmill was stopped, and participants were given time to rest as needed. Then two more 21 m assessments, separated by a rest break, were performed. Consecutive walking assessments started at the lane width achieved at the end of the previous trial (e.g., the starting lane width for the second assessment was equal to the ending lane width of the first assessment).

Data analysis and processing

Data from all treadmill walking assessments were examined and used to estimate participants' ability to control their lateral COM motion during treadmill walking. Kinematic marker data was processed using Visual3D (C-Motion, Germantown, MD) and a custom MATLAB (Mathworks, Natick, MA) program. Marker data was gap-filled and low-pass filtered (Butterworth, 6 Hz cut-off frequency). Time of initial foot contact (IC) and toe-off (TO) events were identified for each step based on maximum and minimum fore-aft positions of the calcaneus and 2nd metatarsal markers, respectively. Mediolateral COM position was calculated in Visual3D as the center of the Visual3D model's pelvis. We then calculated the lateral COM excursion for each gait cycle. Finally, from all gait cycles, we identified the five consecutive gait cycles that produced the smallest average lateral COM excursion. This value, the minimum lateral COM excursion over five consecutive gait cycles, was used to represent each participant's ability to control their lateral COM motion during forward walking.

Statistical analysis

Descriptive variables, scores of clinical outcome measures, and minimum lateral COM excursion were reported as mean [standard deviation (SD)]. The Shapiro–Wilk test for normality was used to confirm the assumption of normality of clinical balance measures and minimum lateral COM excursion. To evaluate the relationship between the ability to control lateral center of mass motion during walking and clinical outcome measures, a Spearman correlation analysis was performed between minimum lateral COM excursion and the following clinical outcome measures: TUG, FGA, BBS and preferred and fast 10MWT. Spearman's correlation coefficients (ρ) used for all the measures was interpreted as follows: >0.70 as strong, 0.50–0.70 as moderate, 0.30–0.50 as weak (44). A valuable property of the Spearman coefficient is that it is relatively robust against outliers because it quantifies a strictly monotonic relationship between two variables (45). All statistical analyses were performed using SPSS (Version 24, SPSS Inc., Chicago, IL, United States) with $\alpha = 0.05$. A Bonferroni correction was applied to account for multiple correlations with level of significance of 0.01.

Results

Twenty people with iSCI participated in our study (mean age of 52.9 ± 18.2 years). Participant characteristics are reported in Table 1. Participants mean scores of clinical and laboratory outcomes are reported in Table 2. All participants were able to successfully complete the walking assessments. The participants found the laboratory-based assessment to be challenging yet engaging and enjoyable. Participants were able to use the visual feedback to make changes in the way they walked.

Minimum lateral COM excursion significantly correlated with all clinical outcome measures we examined. There was a moderate, positive correlation between minimum lateral COM excursion and TUG time ($\rho = 0.59$, $p = 0.007$) (Figure 2A). There was a moderate, negative correlation between minimum lateral COM excursion and FGA score ($\rho = -0.59$, $p = 0.007$) (Figure 2B) and BBS score ($\rho = -0.54$, $p = 0.014$) (Figure 2C). The minimum lateral COM excursion had a moderate, negative correlation with fast 10MWT speed ($\rho = -0.68$; $p = 0.001$) (Figure 3A) and with preferred 10MWT speed ($\rho = -0.59$; $p = 0.006$) (Figure 3B).

TABLE 1 Participant characteristics.

Demographics (*n* =20)	Results
Age in years, mean (SD)	52.9 (18.06)
Gender	
Male	*n* = 14; 70%
Female	*n* = 6; 30%
Height in meters, mean (SD)	1.73 (0.07)
Years post SCI, mean (SD)	7.95 (9.06)
Level of injury, number, and percent of participants	
Cervical	*n* = 13; 65%
Thoracic	*n* = 7; 35%
Mechanism of injury, number, and percent of participants	
Traumatic	*n* = 15; 75%
Non-traumatic	*n* = 5; 25%
Self-selected Walking Index for Spinal Cord Injury (WISCI II), mean (SD)	17.65 (2.74)
Lower Extremity Motor Score (LEMS), mean (SD)	43.85 (4.69)
Walking device, number and percent of participants	
Rolling walker	*n* = 1; 5%
Cane	*n* = 10; 50%
None	*n* = 9; 45%
Brace usage, number and percent of participants	
Using brace	*n* = 4; 20%
Not using brace	*n* = 16; 80%
Falls, number and percent of participants	
No falls	*n* = 7; 35%
1 fall	*n* = 4; 20%
2 or more falls	*n* = 9; 45%

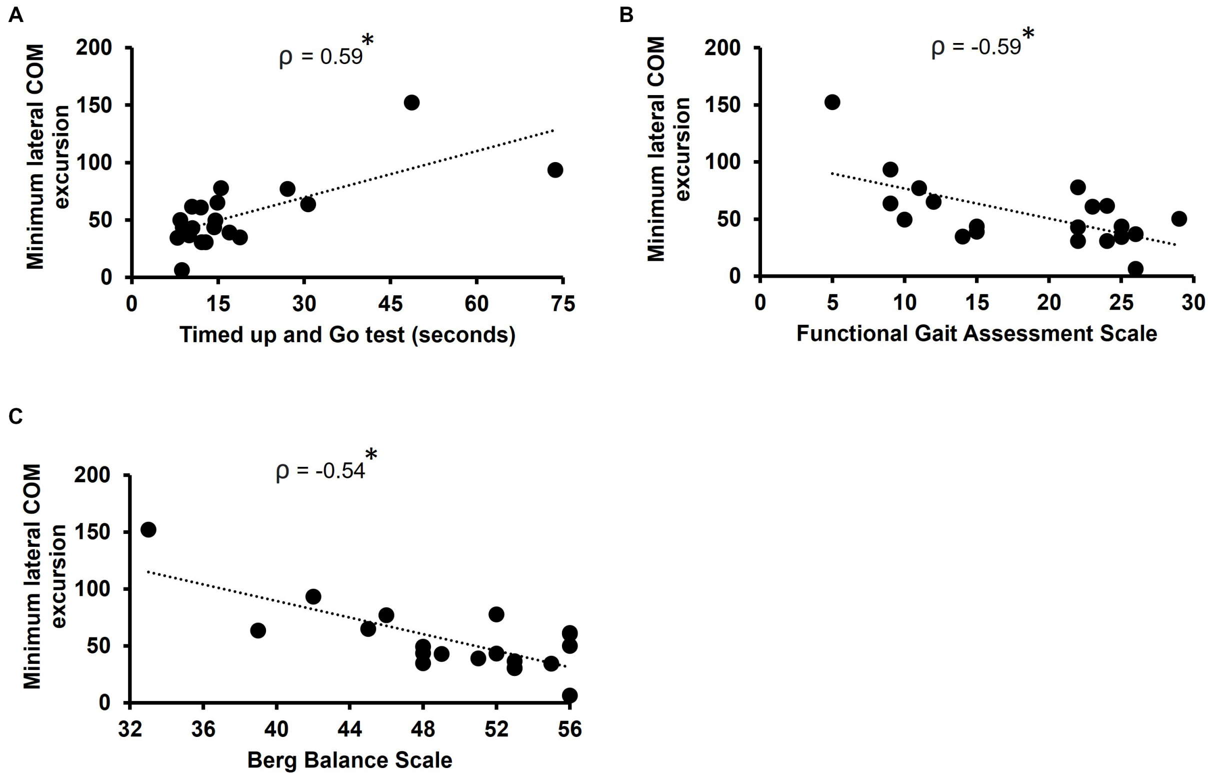

FIGURE 2
Scatterplots of minimum lateral COM excursion with **(A)** Timed Up and Go test, **(B)** Functional gait assessment scale, and **(C)** Berg balance scale. Each circle represents one participant. *p <0.01.

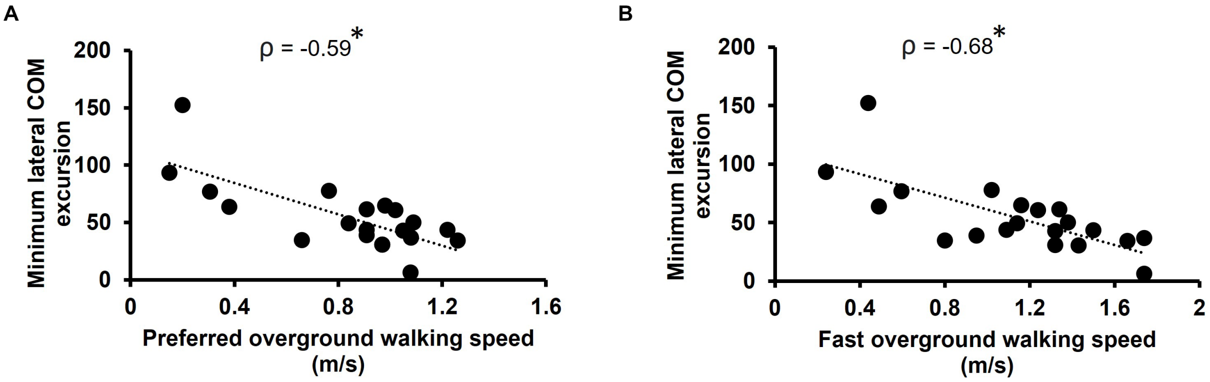

FIGURE 3
Scatterplots of minimum lateral COM excursion with **(A)** preferred overground walking speed as measured by the 10MWT and **(B)** fast overground walking speed as measured by the 10MWT. Each circle represents one participant. *p <0.01.

Discussion

In support of our hypothesis, we found that for ambulatory adults with iSCI, the ability to control their lateral COM motion during walking moderately correlated with clinical outcome measures related to walking balance, walking speed, and postural balance.

The ability to control lateral COM motion during walking was found to be moderately correlated with our two clinical measures of walking balance, the TUG and FGA. Smaller minimum lateral COM excursions were associated with shorter TUG times and higher scores on the FGA, both indicating greater walking ability. A moderate correlation indicated that both (clinical measure and lateral COM excursion) measure a similar construct, i.e., walking balance. Both the TUG and FGA involve walking and maneuvering with a pivot turn that challenges functional walking balance. Specifically, the FGA provides a range of gait activities (change in gait speed, head turns, narrow base of support walking) that require complex stabilizing strategies and postural adjustments. Whereas the TUG requires motor

TABLE 2 Clinical and laboratory outcome measures.

Outcome measure	Mean score (SD)
Timed Up and Go test (TUG) (seconds)	18.86 (16.13)
Functional Gait Assessment (FGA)	18.40 (7.26)
Berg Balance Scale (BBS)	49.55 (6.19)
10meter walk test (10MWT)	
Preferred overground walking speed (meter/s)	0.84 (0.33)
Fast overground walking speed (meter/s)	1.13 (0.43)
Minimum lateral center of mass (COM) excursion (mm)	54.67 (30.45)

planning and capacity to anticipate transitioning from one motor task to another in a particular sequence (46). Due to their sensorimotor deficits, reduced muscle strength, impaired proprioception and deficits in balance and coordination, people with iSCI have difficulty with safely performing complex maneuvers during walking. These impairments challenge dynamic balance and their ability to make anticipatory changes during walking. Our finding that individuals with the poorest ability to control their lateral COM excursion also had the lowest scores of FGA and took the longest time to perform TUG is built on previous findings indicating that people with iSCI have impaired control of lateral motion during walking (4–7).

Similar to the TUG and FGA, minimum lateral COM excursion showed a moderate, negative correlation with BBS score. Specifically, individuals with greater BBS scores, which indicate greater postural control and balance, demonstrated a better ability to control their lateral COM excursion during walking. The BBS is often referred to as a "gold standard" because it is one of the most widely used, valid, and reliable clinical measures for assessing balance and postural control (28, 29, 38, 47, 48). Since scoring for BBS is based on how well the participant performs a series of balance challenging tasks that are performed in daily life, it is a good indicator of functional balance (including static and dynamic) and is used for assessment of fall-risk in several populations (49–52). Previous studies examining the relationship between balance measured by BBS and walking ability in neurological population found that BBS score is a strong predictor of walking ability (home and community ambulation) among people with stroke (53–55). BBS score is also found to correlate with walking performance in people with SCI (26) demonstrating a strong relationship between walking function and balance. Our results support this relationship such that people with iSCI who had the best ability to control their lateral COM excursion during walking had the highest scores on the BBS. Because our study participants were relatively high functioning there was a possibility that the BBS may be susceptible to a ceiling effect. In our study, 4 of our 20 participants scored a 56, the highest score on the BBS. Thus, majority of our participants did not experience a ceiling effect. Despite the potential for ceiling effects, we felt it was important to include this measure of balance in the study because of its extensive use and its recommendation by the Spinal Cord Injury EDGE Task Force as one of the measure of balance (56).

Our results also demonstrated a correlation between better lateral COM control during walking and walking speed. This relationship was moderate for both preferred and fast walking speed. These results could be attributed to an association between changes in COM excursion with walking speed. Studies in healthy adults reported that lateral COM excursion decreases with increase in walking speed (57) and restrictions to COM excursion lead to increased walking speeds in order to ensure dynamic stability (58). Thus, the ability to control lateral COM excursion could provide stability that enables individuals to walk faster.

We developed a novel task to assess participants' maximum capacity to control their lateral COM motion during forward walking. We provided participants with visual feedback by projecting a target lane on the treadmill to encourage them to try and minimize their lateral motion during walking. We then assessed their best performance during five consecutive gait cycles occurring at any time during three trials of the 21 m walking task. We used five consecutive gait cycles to evaluate performance as this brief, but sustained period is likely to have minimized the likelihood that this top performance was by chance. While this task was engaging and challenging for participants, the task itself may have some limitations as a method of evaluating the ability to control lateral COM motion. For example, to receive visual feedback, participants had to look down at the treadmill walking surface. It is possible that this requirement may have affected participants' normal walking pattern. However, in developing this protocol we have found that participants with iSCI strongly prefer visual feedback to be provided on the treadmill rather than on a monitor mounted at eye-level in front of the treadmill that require participants to "look up." This is likely because people with iSCI often use vision for foot placement during walking (59, 60). In addition, this task is only able to evaluate one component of walking – maximum ability to control lateral COM motion during forward walking. However, as the correlations between the control of lateral COM motion and our functional gait and balance measures demonstrated moderate strength of association, it is clear that there are other underlying skills that will contribute to performance. For example, in the current trial we evaluated performance during consecutive gait cycles during treadmill walking in a relatively consistent environment. During real-world walking and balance tasks, people may be required to make regular step-to-step changes to continually adjust their foot placement in order to react to small changes in their COM state (position and velocity). Indeed, in our prior work we have found that increases in step-to-step variability of lateral foot placement when walking at fast speeds may be a method that people with iSCI use to maintain stability of lateral COM dynamics during walking (5).

Our findings have important clinical implications for balance assessment and training. Our study indicates that the ability to control lateral COM motion during walking is closely related to several functional measures of gait and balance in people with iSCI. We believe this relationship may be because control of lateral COM motion during walking and the gait and balance measures we examined are all dependent on the capacity of the nervous system to both anticipate and react to ongoing COM dynamics. People with iSCI have an impaired ability to anticipate COM dynamics due to sensory (61) and motor (7) dysfunction. Additionally, research has found deficits in reactive balance responses used to control lateral COM motion in this population (62). Thus, limitations in the ability to accurately anticipate and react to ongoing COM dynamics may reduce their ability to control lateral COM motion during walking and to perform functional measures of gait and balance that require these fundamental skills. This relationship could motivate clinical interventions that directly target the ability to control lateral COM motion during walking to improve functional balance and gait in people with iSCI. The clinical interventions could focus more on traditional balance training tasks, like narrow base of support walking, rapid maneuvers during walking, or manual perturbations to improve lateral balance control. Alternately, a novel approach to train lateral COM control in people with iSCI could be to perform gait training in a movement amplification field that applies proportional forces in the same direction as the real-time lateral velocity of the participant (63, 64).

There are a few limitations associated with this study. While our findings suggest that lateral COM motion during walking is associated with clinical gait and balance outcomes, it is not clear if this relationship is causative. Future research is recommended to investigate if there is a causal relationship between these measures. Our current study has a small sample size and relatively high functioning (community dwelling), ambulatory people with iSCI were included. Thus, findings from our study cannot be generalized to all individuals with iSCI but should be important for those who walk with limited or no assistive devices.

Conclusion

This study finds that the ability to control lateral COM motion during walking correlates with previously validated clinical gait and balance measures and may be a contributing factor to gait and balance outcomes in people with iSCI. Further research should explore if interventions designed to improve control of lateral COM motion during walking translate to improvements in functional gait and balance in people with iSCI.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving human participants were reviewed and approved by Institutional Review Boards at Northwestern University and the Edward Hines Jr. Veterans Affairs Hospital. The patients/participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

KG, WO, AS, TC, and SD designed the study. KG, AS, HH, and SD collected the data. AS, SD, K-YK, and KG analyzed the data. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

Supplementary material

The Supplementary material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fneur.2023.1146094/full#supplementary-material

SUPPLEMENTARY VIDEO

The video demonstrates the treadmill-based task used to evaluate participant's capacity to control their lateral COM motion during walking. The video shows a participant walking on an oversized treadmill under the supervision of two physical therapists. The participant receives visual feedback of their real-time mediolateral COM position, represented by a white line projected along the length of the treadmill, and a target lane, represented by two green lines. The participant is instructed to try and maintain the white line within the target lane. During the task, if the participant's COM moves outside the target lane the area outside the target turns red providing the participant with an immediate visual cue to try and move their COM within the target lane. To challenge each participant's capacity to control their lateral COM motion, the target lane progressively narrows or widens based on their performance.

References

1. Bauby CE, Kuo AD. Active control of lateral balance in human walking. *J Biomech*. (2000) 33:1433–40. doi: 10.1016/S0021-9290(00)00101-9

2. Kuo AD. Stabilization of lateral motion in passive dynamic walking. *Int J Robot Res*. (1999) 18:917–30. doi: 10.1177/02783649922066655

3. MacKinnon CD, Winter DA. Control of whole body balance in the frontal plane during human walking. *J Biomech*. (1993) 26:633–44. doi: 10.1016/0021-9290(93)90027-C

4. Ochs WL, Woodward J, Cornwell T, Gordon KE. Meaningful measurements of maneuvers: people with incomplete spinal cord injury 'step up' to the challenges of altered stability requirements. *J Neuroeng Rehabil*. (2021) 18:46. doi: 10.1186/s12984-021-00840-4

5. Cornwell T, Woodward J, Ochs W, Gordon KE. Stabilization strategies for fast walking in challenging environments with incomplete spinal cord injury. *Front Rehabil Sci*. (2021) 2:22. doi: 10.3389/fresc.2021.709420

6. Zwijgers E, van Asseldonk EH, Vos-van der Hulst M, Geurts AC, Keijsers NL. Impaired foot placement strategy during walking in people with incomplete spinal cord injury. *J Neuroeng Rehabil*. (2022) 19:1–9. doi: 10.1186/s12984-022-01117-0

7. Arora T, Musselman KE, Lanovaz JL, Linassi G, Arnold C, Milosavljevic S, et al. Reactive balance responses to an unexpected slip perturbation in individuals with incomplete spinal cord injury. *Clin Biomech*. (2020) 78:105099. doi: 10.1016/j.clinbiomech.2020.105099

8. Matsubara JH, Wu M, Gordon KE. Metabolic cost of lateral stabilization during walking in people with incomplete spinal cord injury. *Gait Posture*. (2015) 41:646–51. doi: 10.1016/j.gaitpost.2015.01.015

9. Lemay J-F, Duclos C, Nadeau S, Gagnon D, Desrosiers É. Postural and dynamic balance while walking in adults with incomplete spinal cord injury. *J Electromyogr Kinesiol*. (2014) 24:739–46. doi: 10.1016/j.jelekin.2014.04.013

10. Bone MD, Arora T, Musselman KE, Lanovaz JL, Linassi GA, Oates AR. Investigating proactive balance control in individuals with incomplete spinal cord injury while walking on a known slippery surface. *Neurosci Lett*. (2021) 749:135744. doi: 10.1016/j.neulet.2021.135744

11. Wu MNM, Brown G, Gordon KE. Control of locomotor stability in stabilizing and destabilizing environments. *Gait Posture*. (2017) 55:191–8. doi: 10.1016/j.gaitpost.2017.04.021

12. Arora T, Musselman KE, Lanovaz JL, Linassi G, Arnold C, Milosavljevic S, et al. Walking stability during normal walking and its association with slip intensity among individuals with incomplete spinal cord injury. *PM R*. (2019) 11:270–7. doi: 10.1016/j.pmrj.2018.07.012

13. Schrager MA, Kelly VE, Price R, Ferrucci L, Shumway-Cook A. The effects of age on medio-lateral stability during normal and narrow base walking. *Gait Posture*. (2008) 28:466–71. doi: 10.1016/j.gaitpost.2008.02.009

14. Patla AE, Adkin A, Ballard T. Online steering: coordination and control of body center of mass, head, and body reorientation. *Exp Brain Res*. (1999) 129:0629–34. doi: 10.1007/s002210050932

15. Glaister BC, Bernatz GC, Klute GK, Orendurff MS. Video task analysis of turning during activities of daily living. *Gait Posture*. (2007) 25:289–94. doi: 10.1016/j.gaitpost.2006.04.003

16. Poncumhak P, Saengsuwan J, Kamruecha W, Amatachaya S. Reliability and validity of three functional tests in ambulatory patients with spinal cord injury. *Spinal Cord*. (2013) 51:214–7. doi: 10.1038/sc.2012.126

17. van Hedel HJ, Wirz M, Dietz V. Assessing walking ability in subjects with spinal cord injury: validity and reliability of 3 walking tests. *Arch Phys Med Rehabil*. (2005) 86:190–6. doi: 10.1016/j.apmr.2004.02.010

18. Podsiadlo D, Richardson S. The timed "up & go": a test of basic functional mobility for frail elderly persons. *J Am Geriatr Soc*. (1991) 39:142–8. doi: 10.1111/j.1532-5415.1991.tb01616.x

19. Wrisley DM, Marchetti GF, Kuharsky DK, Whitney SL. Reliability, internal consistency, and validity of data obtained with the functional gait assessment. *Phys Ther*. (2004) 84:906–18. doi: 10.1093/ptj/84.10.906

20. Gordon K, Kahn J, Ferro S, Frank L, Klashman L, Nachbi R, et al. Reliability and validity of the functional gait assessment in spinal cord injury. *Arch Phys Med Rehabil*. (2016) 97:e87. doi: 10.1016/j.apmr.2016.08.266

21. Kahn JH, Ohlendorf A, Olsen A, Gordon KE. Reliability and validity of the functional gait assessment in incomplete spinal cord injury. *Top Spinal Cord Inj Rehabil*. (2020) 26:268–74. doi: 10.46292/sci19-00069

22. Graham JE, Ostir GV, Fisher SR, Ottenbacher KJ. Assessing walking speed in clinical research: a systematic review. *J Eval Clin Pract*. (2008) 14:552–62. doi: 10.1111/j.1365-2753.2007.00917.x

23. Bohannon RW. Comfortable and maximum walking speed of adults aged 20—79 years: reference values and determinants. *Age Ageing*. (1997) 26:15–9. doi: 10.1093/ageing/26.1.15

24. Lindholm B, Nilsson MH, Hansson O, Hagell P. The clinical significance of 10-m walk test standardizations in Parkinson's disease. *J Neurol*. (2018) 265:1829–35. doi: 10.1007/s00415-018-8921-9

25. Bahrami F, Dehkordi SN, Dadgoo M. Inter and intra rater reliability of the 10 meter walk test in the community dweller adults with spastic cerebral palsy. *Iran J Child Neurol*. (2017) 11:57–64.

26. Forrest GF, Lorenz DJ, Hutchinson K, VanHiel LR, Basso DM, Datta S, et al. Ambulation and balance outcomes measure different aspects of recovery in individuals with chronic, incomplete spinal cord injury. *Arch Phys Med Rehabil*. (2012) 93:1553–64. doi: 10.1016/j.apmr.2011.08.051

27. Lemay J, Nadeau S. Standing balance assessment in ASIA D paraplegic and tetraplegic participants: concurrent validity of the Berg balance scale. *Spinal Cord*. (2010) 48:245–50. doi: 10.1038/sc.2009.119

28. Wirz M, Müller R, Bastiaenen C. Falls in persons with spinal cord injury: validity and reliability of the Berg balance scale. *Neurorehabil Neural Repair*. (2010) 24:70–7. doi: 10.1177/1545968309341059

29. Jørgensen V, Opheim A, Halvarsson A, Franzén E, Roaldsen KS. Comparison of the Berg balance scale and the Mini-BESTest for assessing balance in ambulatory people with spinal cord injury: validation study. *Phys Ther*. (2017) 97:677–87. doi: 10.1093/ptj/pzx030

30. Gordon KE, Ferris DP, Kuo AD. Metabolic and mechanical energy costs of reducing vertical center of mass movement during gait. *Arch Phys Med Rehabil*. (2009) 90:136–44. doi: 10.1016/j.apmr.2008.07.014

31. Ortega JD, Farley CT. Minimizing center of mass vertical movement increases metabolic cost in walking. *J Appl Physiol*. (2005) 99:2099–107. doi: 10.1152/japplphysiol.00103.2005

32. Havens KL, Mukherjee T, Finley JM. Analysis of biases in dynamic margins of stability introduced by the use of simplified center of mass estimates during walking and turning. *Gait Posture*. (2018) 59:162–7. doi: 10.1016/j.gaitpost.2017.10.002

33. Shumway-Cook A, Brauer S, Woollacott M. Predicting the probability for falls in community-dwelling older adults using the timed up & go test. *Phys Ther*. (2000) 80:896–903. doi: 10.1093/ptj/80.9.896

34. Kojima G, Masud T, Kendrick D, Morris R, Gawler S, Treml J, et al. Does the timed up and go test predict future falls among British community-dwelling older people? Prospective cohort study nested within a randomised controlled trial. *BMC Geriatr*. (2015) 15:1–7. doi: 10.1186/s12877-015-0039-7

35. Panel on Prevention of Falls in Older Persons, A.G.S. and B.G. Society. Summary of the updated American Geriatrics society/British Geriatrics society clinical practice guideline for prevention of falls in older persons. *J Am Geriatr Soc*. (2011) 59:148–57. doi: 10.1111/j.1532-5415.2010.03234.x

36. Moore JL, Potter K, Blankshain K, Kaplan SL, O'Dwyer LC, Sullivan JE. A core set of outcome measures for adults with neurologic conditions undergoing rehabilitation: a clinical practice guideline. *J Neurol Phys Ther*. (2018) 42:174–220. doi: 10.1097/NPT.0000000000000229

37. Blum L, Korner-Bitensky N. Usefulness of the Berg balance scale in stroke rehabilitation: a systematic review. *Phys Ther*. (2008) 88:559–66. doi: 10.2522/ptj.20070205

38. Berg KO, Wood-Dauphinee SL, Williams JI, Maki B. Measuring balance in the elderly: validation of an instrument. *Can J Public Health*. (1992) 83:S7–11.

39. Berg K, Wood-Dauphine S, Williams J, Gayton D. Measuring balance in the elderly: preliminary development of an instrument. *Physiother Can*. (1989) 41:304–11. doi: 10.3138/ptc.41.6.304

40. Berg KO, Maki BE, Williams JI, Holliday PJ, Wood-Dauphinee SL. Clinical and laboratory measures of postural balance in an elderly population. *Arch Phys Med Rehabil*. (1992) 73:1073–80.

41. Kahn J, Tefertiller C. Measurement characteristics and clinical utility of the 10-meter walk test among individuals with spinal cord injury. *Arch Phys Med Rehabil*. (2014) 95:1011–2. doi: 10.1016/j.apmr.2014.01.021

42. Scivoletto G, Tamburella F, Laurenza L, Foti C, Ditunno J, Molinari M. Validity and reliability of the 10-m walk test and the 6-min walk test in spinal cord injury patients. *Spinal Cord*. (2011) 49:736–40. doi: 10.1038/sc.2010.180

43. Lam T, Noonan VK, Eng JJ. A systematic review of functional ambulation outcome measures in spinal cord injury. *Spinal Cord*. (2008) 46:246–54. doi: 10.1038/sj.sc.3102134

44. Hinkle DE, Wiersma W, Jurs SG. *Applied statistics for the behavioral sciences, vol. 663 Houghton Mifflin College Division*. New York: Houghton Mifflin Company, (2003).

45. Schober P, Boer C, Schwarte LA. Correlation coefficients: appropriate use and interpretation. *Anesth Analg*. (2018) 126:1763–8. doi: 10.1213/ANE.0000000000002864

46. Carmeli E, Bar-Chad S, Lotan M, Merrick J, Coleman R. Five clinical tests to assess balance following ball exercises and treadmill training in adult persons with intellectual disability. *J Gerontol Ser A Biol Med Sci*. (2003) 58:M767–72. doi: 10.1093/gerona/58.8.M767

47. La Porta F, Caselli S, Susassi S, Cavallini P, Tennant A, Franceschini M. Is the Berg balance scale an internally valid and reliable measure of balance across different etiologies in neurorehabilitation? A revisited Rasch analysis study. *Arch Phys Med Rehabil*. (2012) 93:1209–16. doi: 10.1016/j.apmr.2012.02.020

48. Freixes O, Passuni DA, Buffetti E, Elizalde M, Lastiri F. Berg balance scale: inter-rater and intra-rater reliability of the Spanish version with incomplete spinal cord injured subjects. *Spinal Cord Ser Cases*. (2020) 6:1–5. doi: 10.1038/s41394-020-0278-5

49. Lima C, Ricci N, Nogueira E, Perracini MR. The Berg balance scale as a clinical screening tool to predict fall risk in older adults: a systematic review. *Physiotherapy*. (2018) 104:383–94. doi: 10.1016/j.physio.2018.02.002

50. Maeda N, Kato J, Shimada T. Predicting the probability for fall incidence in stroke patients using the Berg balance scale. *J Int Med Res*. (2009) 37:697–704. doi: 10.1177/147323000903700313

51. Park S-H, Lee Y-S. The diagnostic accuracy of the berg balance scale in predicting falls. *West J Nurs Res*. (2017) 39:1502–25. doi: 10.1177/0193945916670894

52. Schlenstedt C, Brombacher S, Hartwigsen G, Weisser B, Möller B, Deuschl G. Comparison of the Fullerton advanced balance scale, mini-BESTest, and berg balance scale to predict falls in Parkinson disease. *Phys Ther*. (2016) 96:494–501. doi: 10.2522/ptj.20150249

53. Liao W-L, Chang C-W, Sung P-Y, Hsu W-N, Lai M-W, Tsai S-W. The Berg balance scale at admission can predict community ambulation at discharge in patients with stroke. *Medicina*. (2021) 57:556. doi: 10.3390/medicina57060556

54. Louie DR, Eng JJ. Berg balance scale score at admission can predict walking suitable for community ambulation at discharge from inpatient stroke rehabilitation. *J Rehabil Med*. (2018) 50:37–44. doi: 10.2340/16501977-2280

55. Fulk GD, He Y, Boyne P, Dunning K. Predicting home and community walking activity poststroke. *Stroke*. (2017) 48:406–11. doi: 10.1161/STROKEAHA.116.015309

56. Kahn JH, Tappan R, Newman CP, Palma P, Romney W, Tseng Stultz E, et al. Outcome measure recommendations from the spinal cord injury EDGE task force. *Phys Ther*. (2016) 96:1832–42. doi: 10.2522/ptj.20150453

57. Orendurff MS, Segal AD, Klute GK, Berge JS, Rohr ES, Kadel NJ. The effect of walking speed on center of mass displacement. *J Rehabil Res Dev*. (2004) 41:829. doi: 10.1682/JRRD.2003.10.0150

58. Tesio L, Rota V. The motion of body center of mass during walking: a review oriented to clinical applications. *Front Neurol*. (2019) 10:999. doi: 10.3389/fneur.2019.00999

59. Malik RN, Cote R, Lam T. Sensorimotor integration of vision and proprioception for obstacle crossing in ambulatory individuals with spinal cord injury. *J Neurophysiol*. (2017) 117:36–46. doi: 10.1152/jn.00169.2016

60. Lemay J-F, Gagnon D, Duclos C, Grangeon M, Gauthier C, Nadeau S. Influence of visual inputs on quasi-static standing postural steadiness in individuals with spinal cord injury. *Gait Posture*. (2013) 38:357–60. doi: 10.1016/j.gaitpost.2012.11.029

61. Wirz M, van Hedel HJ. Balance, gait, and falls in spinal cord injury. *Handb Clin Neurol*. (2018) 159:367–84. doi: 10.1016/B978-0-444-63916-5.00024-0

62. Kuo AD. The relative roles of feedforward and feedback in the control of rhythmic movements. *Mot Control*. (2002) 6:129–45. doi: 10.1123/mcj.6.2.129

63. Brown G., Wu M., Huang F.C., Gordon K.E., *Movement augmentation to evaluate human control of Locomotor stability.* 2017 39th Annual International Conference of the Ieee Engineering in Medicine and Biology Society (EMBC), (2017). p. 66–69.

64. Wu MM, Brown GL, Woodward JL, Bruijn SM, Gordon KE. A novel movement amplification environment reveals effects of controlling lateral centre of mass motion on gait stability and metabolic cost. *R Soc Open Sci*. (2020) 7:190889. doi: 10.1098/rsos.190889

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Postural threat increases sample entropy of postural control

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Introduction: Postural threat elicits modifications to standing balance. However, the underlying neural mechanism(s) responsible remain unclear. Shifts in attention focus including directing more attention to balance when threatened may contribute to the balance changes. Sample entropy, a measure of postural sway regularity with lower values reflecting less automatic and more conscious control of balance, may support attention to balance as a mechanism to explain threat-induced balance changes. The main objectives were to investigate the effects of postural threat on sample entropy, and the relationships between threat-induced changes in physiological arousal, perceived anxiety, attention focus, sample entropy, and traditional balance measures. A secondary objective was to explore if biological sex influenced these relationships.

Methods: Healthy young adults (63 females, 42 males) stood quietly on a force plate without (No Threat) and with (Threat) the expectation of receiving a postural perturbation (i.e., forward/backward support surface translation). Mean electrodermal activity and anterior–posterior centre of pressure (COP) sample entropy, mean position, root mean square, mean power frequency, and power within low (0–0.05Hz), medium (0.5–1.8Hz), and high-frequency (1.8–5Hz) components were calculated for each trial. Perceived anxiety and attention focus to balance, task objectives, threat-related stimuli, self-regulatory strategies, and task-irrelevant information were rated after each trial.

Results and Discussion: Significant threat effects were observed for all measures, except low-frequency sway. Participants were more physiologically aroused, more anxious, and directed more attention to balance, task objectives, threat-related stimuli, and self-regulatory strategies, and less to task-irrelevant information in the Threat compared to No Threat condition. Participants also increased sample entropy, leaned further forward, and increased the amplitude and frequency of COP displacements, including medium and high-frequency sway, when threatened. Males and females responded in the same way when threatened, except males had significantly larger threat-induced increases in attention to balance and high-frequency sway. A combination of sex and threat-induced changes in physiological arousal, perceived anxiety, and attention focus accounted for threat-induced changes in specific traditional balance measures, but not sample entropy. Increased sample entropy when threatened may reflect a shift to more automatic control. Directing more conscious control to balance when threatened may act to constrain these threat-induced automatic changes to balance.

KEYWORDS

postural control, balance, postural threat, sample entropy, attention focus, perceived anxiety, physiological arousal

1. Introduction

Postural threat manipulations have been used to investigate the effects of emotions, such as fear and anxiety, on balance control (1). One common manipulation of postural threat involves altering the height of the support surface on which individuals stand. When tasked to quietly stand at or near the edge of an elevated platform, healthy young adults typically lean further away from the edge and adopt a balance strategy characterized by decreased amplitude and increased frequency of centre of pressure (COP) displacements in the anterior–posterior (A-P) direction (2–12). Postural threat has also been manipulated by having individuals quietly stand without or with the expectation of receiving an unexpected postural perturbation. When standing in anticipation of an A-P support surface translation, healthy young adults typically lean further forward and have increased amplitude and increased frequency of COP displacements in the A-P direction (13, 14). This research shows that threat-induced changes in leaning and sway amplitude vary with the threat context while increases in sway frequency specifically higher frequency components (>0.5 Hz) are consistent across these different types of postural threat (11, 12, 14), as well as other conditions of increased arousal and anxiety (15, 16).

Although threat-induced changes in balance are now well established, the underlying mechanism(s) that contribute to these changes remain poorly understood (1). Threat-induced changes in attention (e.g., directing more attention to balance) is one factor that may be responsible for the observed threat-induced balance changes. For example, healthy young adults report more conscious control of balance when standing on an elevated platform (7, 9) and direct more attention focus to balance, task objectives, threat-related stimuli, self-regulatory or coping strategies, and less attention focus to task irrelevant information in response to both surface height and postural perturbation threats (11–14, 17, 18). Concomitant reductions in attention to balance and high-frequency sway are observed when healthy young adults are repeatedly exposed to a surface height threat (11) and reductions in attention to self-regulatory/coping strategies and high-frequency sway are observed when standing in anticipation of a postural perturbation threat while performing a cognitive distractor task (14). Observed relationships between specific threat-induced changes in attention focus and balance further support attention as a mechanism underlying threat-induced balance changes. For example, healthy young adults who reported more conscious control of balance when standing on an elevated platform leaned further away from the edge (7). Furthermore, healthy young adults who had larger increases in attention to balance when threatened were more likely to show greater increases in sway frequency at height (17) and lean further forward and have a larger increase in sway amplitude in anticipation of a postural perturbation (13). Individuals who had a larger increase in attention to self-regulatory/coping strategies when threatened with a postural perturbation were more likely to have a larger increase in sway frequency (13).

Most studies examining the effects of postural threat on balance control have used traditional balance measures (e.g., amplitude- or frequency-based) to summarize the COP time-series. To further explore the possibility of attention underlying threat-induced changes in balance, the use of non-linear balance measures like sample entropy may provide novel insight into the balance strategy adopted in threatening conditions. Sample entropy assesses the regularity or predictability of COP time-series data and informs about the temporal dynamics or structure of the COP (19). The probability of a particular sequence of data points repeating itself in time is calculated; higher sample entropy values indicate a more irregular and unpredictable COP time-series (i.e., greater probability of observing different sequences in the data) while lower sample entropy values indicate a more regular and predictable COP time-series (i.e., greater probability of observing repeated sequences in the data) (20). Although a shift in sample entropy values (lower or higher) on a continuum may be interpreted differently (21), critical to the current study, these shifts are thought, by some, to reflect the attentional involvement in balance control (22, 23). In this case, higher sample entropy values are thought to reflect less attention to balance and a more automatic balance control; in contrast, lower sample entropy values are thought to reflect more attention to balance and a less effective control of balance. This perspective is supported by research that has shown lower sample entropy values in individuals with stroke compared to controls with sample entropy values shifting higher during stroke recovery as less attention to balance is needed (22). Lower sample entropy values have also been reported in individuals with vestibular deficits compared to controls (24). Higher sample entropy values have been reported in dance experts compared to non-dancers with the experts presumably needing to devote less attention to balance (25). A shift to lower sample entropy values has been shown when standing compared to sitting (23), when standing with eyes closed compared to eyes open (26), when standing with changes in the visual complexity of the environment (24), and when standing on a compliant compared to normal support surface (24, 27) with the more challenging task conditions thought to require a higher degree of attentional involvement in balance. In contrast, a shift to higher sample entropy values has been reported when standing and using external attention focus instructions (28, 29) or performing a concurrent cognitive task (30, 31) with these task constraints acting to direct attention away from standing promoting a more automatic control of balance (32). Research has also shown that individuals who report a greater tendency to consciously control movement have lower sample entropy values during a quiet standing task (33). In addition, studies that have directly manipulated conscious control of balance have revealed lower sample entropy values when individuals received movement monitoring instructions compared to when they were distracted from focusing on their balance (34, 35).

Based on this work, sample entropy or COP regularity is thought to reflect the amount of attention invested in postural control. Roerdink and colleagues suggested that postural threat would shift sample entropy values lower reflecting more attention to balance and less automatic behaviour (23). This view seems plausible as postural threat increases attention to balance (7, 9, 11–14, 17, 18). However, Stins and colleagues have reported no change in sample entropy when healthy young adults stood on a high compared to low platform (36). More recently, Ellmers and colleagues showed an increase in conscious motor processing that was accompanied by an increase in sample entropy suggesting a more automatic control when older adults stood on a high compared to low platform (37, 38), opposite to what would have been theoretically expected (23). Directing more conscious control to balance may constrain threat-induced automatic changes to balance; threat-induced increases in sample entropy were amplified when older adults were distracted in this threatening condition (37).

Given these discrepancies, it is important to confirm how sample entropy changes in healthy young adults when threatened, and if these changes vary for a different type of postural threat manipulation.

The main objectives of this study were (1) to investigate the effects of postural threat on sample entropy, and (2) to explore the relationships between threat-related changes in physiological arousal, perceived anxiety, attention focus, sample entropy, and traditional balance control measures. Given prior observations of sex-dependent changes in balance when standing on an elevated surface (2), sex differences in autonomic responses to stress and anxiety (39–41), and the influence of personality traits on threat-induced changes in balance (9), a secondary objective of this study was to explore how biological sex may influence threat-related changes in physiological, psychological, attention focus, and balance responses. Understanding how other individual factors like biological sex influence these responses may have important implications for interpreting and addressing threat-induced changes in balance. Postural threat was manipulated by having healthy young adults stand with or without the expectation of receiving a postural perturbation allowing for a comparison between No Threat and Threat conditions. Data were taken from two published studies (13, 14) and combined to address these objectives. Apart from the larger data set that was created by combining the studies, a novel component of the current study was the investigation of sample entropy changes in response to the threat of a postural perturbation, which had not been examined in the previously published work.

As individuals report directing more attention to balance when threatened (7, 9, 11–14, 17, 18), a significant decrease in sample entropy was theoretically expected in the Threat compared to No Threat condition (23). It was also expected that a combination of biological sex and threat-induced changes in physiological, psychological, and attention focus measures would significantly predict threat-induced changes in sample entropy, with threat-induced changes in attention focus to balance emerging as the strongest predictor. For example, it was anticipated that larger increases in attention to balance would significantly account for larger decreases in sample entropy.

2. Materials and methods

Data was combined from two published studies that quantified threat-induced changes in physiological arousal, perceived anxiety, attention focus, and balance control measures (13, 14). The two studies used the same postural threat, standing with or without the expectation of receiving a postural perturbation. Although certain procedures differed between the two studies, there was always a no threat (i.e., one trial performed prior to any threat/perturbation experience) and threat (i.e., one trial performed after experience with the threat/perturbation) condition that formed the basis of the current analyses.

2.1. Participants

One-hundred and five healthy young adults (63 females, 42 males) were included in this study. Descriptive statistics for participant characteristics including trait measures of anxiety (State–Trait Anxiety Inventory) (42), movement reinvestment (Movement Specific Reinvestment Scale) (43, 44), and physical risk-taking (Domain-Specific Risk-Taking Scale, Recreational Domain) (45) are presented in Table 1. Details of these measures can be found in (9). Exclusion criteria were any self-reported neurological or musculoskeletal conditions that could influence balance control. All experimental procedures were performed in accordance with the Declaration of Helsinki and were approved by the Brock University Bioscience Research Ethics Board. Each participant provided written informed consent prior to the start of any experimental procedures.

2.2. Procedure

2.2.1. Postural threat manipulation

Participants stood on a force plate (OR6-7, AMTI, Watertown, MA, United States) that was surrounded by a wooden platform (0.9 m x 1.6 m) fitted flush with its surface. The force plate and platform were secured to a motorized 4.3-m linear positioning stage (H2W Technologies Inc., Valencia, CA, United States). Participants were instructed to stand quietly with bare-feet, in a stance width equal to their foot length, with arms at their side, and their gaze fixed on an eye-level target located on the wall 4-m away. Stance width was kept consistent across all conditions by outlining with tape the position of the feet on the force plate. Throughout the experiment, a spotter was positioned beside the platform and participants wore a harness that was attached to a track secured to the ceiling.

Participants stood with no expectation of receiving a postural perturbation (No Threat) or with the expectation of receiving a postural perturbation (Threat). The perturbation was a temporally and directionally unpredictable support surface translation in the anterior or posterior direction (displacement = 0.25 m, peak velocity = 0.9 m/s, peak acceleration = 1.7 m/s^2). No restrictions were placed on the use of balance recovery strategies.

TABLE 1 Mean and standard deviation (SD) values for participant characteristics.

	Females (n=63) Mean (SD)	Males (n=42) Mean (SD)	value of p
Age (years)	21.32 (2.60)	22.62 (3.05)	**0.021**
STAI (20–80)	37.71 (10.10)	35.21 (7.19)	0.169
MSRS-CMP (5–30)	19.14 (4.35)	19.07 (4.86)	0.937
MSRS-MSC (5–30)	16.59 (5.46)	14.81 (5.30)	0.101
DOSPERT (6–42)	23.24 (8.57)	26.45 (8.79)	0.065

Value of *p* from independent samples t-tests between females and males (bold font identifies a significant difference); STAI, state–trait anxiety inventory (20 items, 4-point Likert scale, range 20–80 with higher scores representing more trait anxiety) (42); MSRS, movement-specific reinvestment scale (43, 44); CMP, conscious motor processing subscale (5 items, 6-point Likert scale, range 5–30 with higher scores representing more trait CMP); MSC, movement self-consciousness subscale (5 items, 6-point Likert scale, range 5–30 with higher scores representing more MSC); DOSPERT, domain specific risk-taking scale (recreational domain only, 6 items, 7-point Likert scale, range 6–42 with higher scores representing more risk-tasking behaviour) (45).

2.2.2. Experimental protocol

The following is the common protocol that participants experienced in the two studies (13, 14). First, participants completed one No Threat trial which served as a practice trial to address first trial effects on balance control (3, 46) and to prime the anxiety and attention focus questionnaires. Next, participants completed a second No Threat trial before continuing with the Threat trials. In each No Threat trial, participants stood with no expectation of receiving a postural perturbation. In each Threat trial, participants stood with the expectation of receiving a postural perturbation. The quiet stance duration prior to the delivery of the perturbation was varied to ensure the temporal unpredictability of the perturbation. The stance duration for one of these trials matched the stance duration for the No Threat trial. As such, No Threat (i.e., one trial performed prior to any threat/perturbation experience) and Threat (i.e., one trial performed after experience with the threat/perturbation) conditions with equal stance durations were used for comparison. The other Threat conditions were excluded from the analyses as they were only completed to give participants experience with the perturbation and to ensure the temporal unpredictability of the perturbation.

Table 2 shows the relevant No Threat and Threat conditions that 80 participants completed in the initial study (13) and 25 participants completed in the second study (14). In each study, participants experienced the same number of perturbations prior to the Threat condition that was used for comparison. Of note, a second No Threat trial was completed after the Threat trials in the initial study which confirmed the absence of any order effects (13). There were two additional blocks of No Threat and Threat trials completed while performing a secondary cognitive task in the second study (14). However, these blocks of trials always followed the first block of trials performed without the secondary cognitive task. The one noted difference between the studies was the quiet standing duration of the No Threat and Threat trials used for comparison (i.e., 30-s or 60-s). As stance duration can influence balance measures including sample entropy (47, 48), the decision was made to combine the data sets from the two studies and compare traditional balance measures and sample entropy between No Threat and Threat trials calculated over 30-s durations. Thus, only the first 30-s of the 60-s trials completed in the second study were used (14). To address any concern that changes in sample entropy may be due to a shorter time-series, sample entropy was also examined in 25 participants who completed 60-s of quiet standing in the No Threat and Threat trials (14). The same threat-induced increase in sample entropy was observed in this subset of participants. The results of this analysis are presented as Supplementary material Table 1. It should be noted that questionnaires for perceived anxiety and attention focus (described below) are, consequently, based on different durations of standing trials, but are unlikely to influence the outcomes of these measures.

2.3. Dependent measures

2.3.1. Physiological arousal

To estimate changes in physiological arousal and confirm that the perturbation threat generated a significant emotional response, electrodermal activity (EDA) was recorded using a constant voltage of 0.5 V to two silver–silver chloride (Ag/AgCl) electrodes (EL-507, BIOPAC Systems Inc., United States) placed on thenar and hypothenar eminences of the non-dominant hand (49). Prior to electrode placement, a skin preparation gel was applied to the palmar recording sites (NuPrep, Weaver and Company, United States). Electrodermal activity was A/D sampled at 1000 Hz (13) and down sampled to 100 Hz or sampled at 100 Hz ((14); Micro1401, CED, Cambridge, United Kingdom) and recorded using Spike2 software (CED, Cambridge, UK). A custom script that calculated mean EDA for the 30-s trial was used (MATLAB R2020a, MathWorks, United States).

2.3.2. Perceived anxiety

Perceptions of anxiety were recorded from a self-report questionnaire to establish that the perturbation threat altered emotional state. The questionnaire was administered to evaluate worry-related and somatic anxiety. In the Johnson and colleagues (2019) study (13), responses were rated on a scale ranging from 0 ("I was not at all worried") to 100 ("I was very worried") on how respondents generally felt from the start to the end of the standing trial (or the time prior to platform translation). Responses to the

TABLE 2 Experimental conditions used for the combined data set in the current study.

Threat condition	Expectation of perturbation	Quiet standing duration
Johnson et al. (13)		
No Threat	**No**	**30-s**
Threat*	Yes	30-s
Threat	Yes	10-s
Threat	Yes	15-s
Threat	**Yes**	**30-s**
No Threat	No	30-s
Johnson et al. (14)		
No Threat	**No**	**60-s**
Threat	Yes	5-s
Threat	Yes	30-s
Threat	**Yes**	**60-s**

No Threat and Threat conditions used for comparison in the current study are in bold font. These specific Threat conditions were selected as participants had previous experience with the perturbation. *Reflects a Threat trial in which no perturbation was delivered after the quiet standing period. Only the first 30-s of the 60-s quiet standing trials from (14) were used to standardize trial duration. Eighty participants were involved in (13) and 25 participants were involved in (14).

question "How physically anxious did you feel when performing the balance task?" were rated on a scale ranging from 0 ("I did not feel anxious at all") to 100 ("I felt very anxious") to represent somatic anxiety. In the Johnson and colleagues (2020) study (14), these responses were rated on scales ranging from 1 to 9 with the same anchors. Thus, worry-related and somatic anxiety scores were converted to a percent of maximum possible score (50). Perceived anxiety was then calculated by averaging the scores of the worry-related and somatic anxiety questions.

2.3.3. Attention focus

A questionnaire was administered to evaluate attention focus with the following statement preceding each question, "While completing the balance task, you may have directed your attention toward different information. Please indicate the extent to which you thought about or paid attention to: (1) movement processes (balance), (2) task objectives, (3) threat-related stimuli, (4) self-regulatory strategies, and (5) task-irrelevant information (17). Responses were rated on a 9-point Likert scale ranging from 1 ("Not at all") to 9 ("Very much so") on how respondents directed their attention from the start to the end of the standing trial, or the time prior to platform translation. This information was obtained to determine if there were broad changes in attention focus, and more specifically whether individuals reported more conscious control of balance when threatened.

2.3.4. Sample entropy and traditional balance measures

Ground reaction forces and moments from the force plate were either sampled at 1000 Hz (13) and down sampled to 100 Hz or sampled at 100 Hz (14). All data was low-pass filtered offline using a second order Butterworth filter with a cut-off frequency of 10 Hz and used to calculate COP measures in the A-P direction (aligned with the direction of the postural threat). The COP measures from each trial were used to calculate summary measures of sample entropy and traditional balance measures, including mean position (COP-MPOS), root-mean-square (COP-RMS) amplitude, mean power frequency (COP-MPF) and the average power contained within specific frequency bands.

Sample entropy is the negative natural logarithm of the conditional probability that two similar sequences with the same amount of data points remain similar when another data point is added (19). Sample entropy in the A-P direction was calculated from customized MATLAB scripts [Mathworks, United States] presented by Richman and Moorman (19):

$$\text{Sample Entropy}\left(m,r,N\right)=-\log\left(\frac{\text{A}}{\text{B}}\right)$$

where, m is the length of the sequences to be compared, r is the tolerance value for accepting matches, N is the length of the data, and A/B are defined as follows:

$$A=\left\{\frac{\left(n-m-1\right)\left(n-m\right)}{2}\right\}A^{m}\left(r\right)$$

, and

$$B=\left\{\frac{\left(n-m-1\right)\left(n-m\right)}{2}\right\}B^{m}\left(r\right)$$

where, $\text{A}^{\text{m}}(\text{r})$ is the probability that sequences match for $\text{m}+1$ points, and $\text{B}^{\text{m}}(\text{r})$ is the probability that sequences match for m points. Parameter values were set to $m=2$ and $r=0.15$*SD. Although there is no established consensus on parameter selection, parameter settings for balance control studies are commonly set to $m=2$ or 3, and r between 0.1 and 0.25*SD (19). Separate analyses calculated sample entropy in combinations of $m=(2, 3)$ and $r=(0.15, 0.25)$; sample entropy was consistent using these different parameter value combinations. Of note, sample entropy was calculated on the filtered COP data. As filtering has been shown to influence the calculation of sample entropy (51), sample entropy was also calculated on the unfiltered COP data to determine if the processing approach altered the effect of threat on this measure. While the absolute sample entropy values were higher when calculated from the unfiltered data, the directional effect of threat on sample entropy was the same. The results of these analyses are presented as Supplementary material Table 2.

Traditional balance measures, including mean position (COP-MPOS), root-mean-square (COP-RMS) amplitude, mean power frequency (COP-MPF) and the average power contained within specific frequency bands were also calculated from COP data. COP-MPOS was calculated to provide an estimate of leaning when referenced to participants' ankle joints. COP-MPOS was subtracted from the COP signal to remove bias prior to calculating amplitude and frequency measures (52, 53). COP-RMS was used to provide a description of the COP time series magnitude. As a comprehensive assessment of postural control should involve several descriptors of the COP, including measures in both the time and frequency domains (54, 55), frequency-based measures were also calculated to provide information about the spectral properties of the COP which can inform about different processes involved in maintaining quiet stance. The Fast Fourier Transform was performed on equal length, non-overlapping data segments and converted to power spectra (56). Power spectrum analysis was used to estimate the average frequency contained within a power spectrum (COP-MPF) and the average power contained within specific frequency bands: 0–0.05 Hz (low frequency; COP-Freq_{LOW}), 0.5–1.8 Hz (medium frequency; COP-Freq_{MED}), and 1.8–5 Hz (high frequency; COP-$\text{Freq}_{\text{HIGH}}$) (11, 57). All analyses were performed using MATLAB 2020a (Mathworks, United States).

2.4. Statistical analysis

2.4.1. Repeated measures ANOVAs

Separate repeated measures analysis of variance (RM ANOVA) procedures with between-subject (biological sex; female, male) and within-subject (threat; No Threat, Threat) factors were performed for physiological arousal, perceived anxiety, attention focus, sample entropy, and traditional balance measures. The assumption of normality was confirmed prior to the statistical analysis. Non-normal variables (EDA, COP-RMS, COP-MPF, COP-Freq_{LOW}, COP-Freq_{MED}, COP-$\text{Freq}_{\text{HIGH}}$) were corrected using logarithmic transformations, which calculated the base 10 logarithm of each value of the non-normal dependent variable. Significant biological sex by threat interaction effects were explored using Bonferroni-corrected *post hoc* tests. Significance level was set at $p<0.05$.

2.4.2. Multiple linear regressions

Change scores between Threat and No Threat conditions were calculated for each dependent variable. Multiple linear regressions were then conducted to determine if a combination of biological sex and threat-induced changes in physiological arousal, perceived anxiety, and attention focus measures contributed to explaining threat-induced changes in sample entropy and traditional balance measures. Bivariate correlations between biological sex, physiological, psychological, and attention focus change scores did not detect any significant collinearity; no variables were considered highly related ($r > 0.80$) and each of these variables were included as independent variables in the regressions. Seven multiple linear regressions were conducted with biological sex, physiological arousal, perceived anxiety, and attention to movement processes, task objectives, threat-related stimuli, self-regulatory strategies, and task-irrelevant information as the predictor variables and sample entropy, COP-MPOS, COP-RMS, COP-MPF, COP-Freq$_{\mathrm{LOW}}$, COP-Freq$_{\mathrm{MED}}$, and COP-Freq$_{\mathrm{HIGH}}$ as the dependent variables. Significance level was set at $p < 0.05$.

3. Results

3.1. Repeated measures ANOVAs

Descriptive statistics for physiological arousal, perceived anxiety, attention focus, sample entropy, and traditional balance measures for No Threat and Threat conditions for all participants and separately for females and males are presented in Table 3.

3.1.1. Threat effects

A significant main effect of threat was observed for EDA ($F_{(1,103)} = 87.93$, $p < 0.001$) and perceived anxiety ($F_{(1,103)} = 251.71$, $p < 0.001$). Electrodermal activity was significantly greater, and participants reported more anxiety in the Threat compared to No Threat condition (Table 3; Figure 1). A significant main effect of threat was observed for attention to movement processes ($F_{(1,103)} = 79.99$, $p < 0.001$), task objectives ($F_{(1,103)} = 5.99$, $p = 0.016$), threat-related stimuli ($F_{(1,103)} = 228.16$, $p < 0.001$), self-regulatory strategies ($F_{(1,103)} = 44.98$, $p < 0.001$), and task-irrelevant information ($F_{(1,103)} = 37.54$, $p < 0.001$). Participants reported directing significantly more attention to movement processes, task objectives, threat-related stimuli, and self-regulatory strategies, and significantly less attention to task-irrelevant information (Table 3; Figure 1).

A significant main effect of threat was observed for sample entropy ($F_{(1,103)} = 39.86$, $p < 0.001$). Sample entropy was significantly higher in the Threat compared to No Threat condition (Table 3; Figure 2). A significant main effect of threat was also observed for COP-MPOS ($F_{(1,103)} = 50.82$, $p < 0.001$), COP-RMS ($F_{(1,103)} = 14.05$, $p < 0.001$), and COP-MPF ($F_{(1,103)} = 68.91$, $p < 0.001$). Participants leaned significantly further forward and had significantly higher amplitude and frequency of COP displacements in the Threat compared to No Threat condition (Table 3; Figure 2). There was no

TABLE 3 Mean and standard error (SE) values for physiological, psychological, attention focus, sample entropy, and traditional balance measures for No Threat and Threat Conditions for all participants, females, and males, and RM ANOVA results.

	All Participants (n =105)		Females (n =63)		Males (n =42)		RM ANOVA		
	No Threat Mean (SE)	Threat Mean (SE)	No Threat Mean (SE)	Threat Mean (SE)	No Threat Mean (SE)	Threat Mean (SE)	Sex value of p	Threat value of p	Interaction value of p
EDA (μS)	15.78 (0.63)	18.93 (0.71)	16.46 (0.86)	19.89 (0.98)	14.76 (1.04)	17.49 (1.15)	0.124	**<0.001**	0.296
ANX (%)	14.26 (1.84)	56.11 (2.59)	14.85 (2.55)	55.14 (3.49)	13.36 (2.59)	57.56 (3.85)	0.901	**<0.001**	0.465
AF-MP (1–9)	4.79 (0.24)	6.73 (0.20)	4.97 (0.30)	6.54 (0.27)	4.52 (0.40)	7.02 (0.29)	0.959	**<0.001**	**0.044**
AF-TO (1–9)	5.40 (0.23)	5.91 (0.22)	5.65 (0.29)	6.13 (0.30)	5.02 (0.36)	5.60 (0.31)	0.150	**0.016**	0.824
AF-TRS (1–9)	2.17 (0.14)	5.42 (0.23)	2.24 (0.19)	5.56 (0.29)	2.07 (0.21)	5.21 (0.35)	0.432	**<0.001**	0.684
AF-SRS (1–9)	3.56 (0.22)	4.77 (0.22)	3.87 (0.28)	5.44 (0.28)	3.10 (0.34)	3.76 (0.32)	0.113	**<0.001**	0.443
AF-TII (1–9)	3.60 (0.21)	2.29 (0.17)	3.75 (0.27)	2.51 (0.23)	3.38 (0.34)	1.95 (0.22)	0.156	**<0.001**	0.663
SampEn	0.093 (0.004)	0.131 (0.005)	0.088 (0.004)	0.124 (0.006)	0.100 (0.006)	0.142 (0.009)	**0.022**	**<0.001**	0.619
COP-MPOS (mm)	40.61 (1.97)	49.98 (2.09)	36.19 (2.54)	45.39 (2.77)	47.25 (2.84)	56.85 (2.86)	**0.004**	**<0.001**	0.881
COP-RMS (mm)	4.55 (0.17)	5.41 (0.20)	4.49 (0.22)	5.31 (0.26)	4.63 (0.29)	5.55 (0.32)	0.542	**<0.001**	0.823
COP-MPF (Hz)	0.26 (0.01)	0.41 (0.02)	0.25 (0.01)	0.38 (0.02)	0.28 (0.01)	0.45 (0.03)	**0.007**	**<0.001**	0.247
COP-Freq$_{\mathrm{LOW}}$ (mm^2/bin)	99.61 (9.92)	114.76 (11.50)	98.93 (12.31)	111.98 (14.36)	100.64 (16.74)	118.94 (19.22)	0.795	0.280	0.856
COP-Freq$_{\mathrm{MED}}$ (mm^2/bin)	0.65 (0.03)	2.09 (0.15)	0.62 (0.05)	1.89 (0.18)	0.69 (0.05)	2.39 (0.28)	0.096	**<0.001**	0.148
COP-Freq$_{\mathrm{HIGH}}$ (mm^2/bin)	0.023 (0.002)	0.085 (0.008)	0.020 (0.002)	0.068 (0.007)	0.027 (0.003)	0.111 (0.015)	**0.003**	**<0.001**	**0.016**

Bold font value of *p*s identify significant differences. EDA, electrodermal activity; ANX, perceived anxiety; AF, attention focus; MP, movement processes; TO, task objectives; TRS, threat-related stimuli; SRS, self-regulatory strategies; TII, task-irrelevant information; SampEn, sample entropy; COP, centre of pressure; MPOS, mean position; RMS, root mean square; MPF, mean power frequency; Freq$_{\mathrm{LOW}}$, low frequency (0–0.05 Hz); Freq$_{\mathrm{MED}}$, medium frequency (0.5–1.8 Hz); Freq$_{\mathrm{HIGH}}$, high frequency (1.8–5 Hz).

significant main effect of threat for COP-Freq$_{\mathrm{LOW}}$ ($F_{(1,103)}$ = 1.18, p = 0.280). However, a significant main effect of threat was observed for COP-Freq$_{\mathrm{MED}}$ ($F_{(1,103)}$ = 104.67, p < 0.001) and COP-Freq$_{\mathrm{HIGH}}$ ($F_{(1,103)}$ = 82.59, p < 0.001). COP-Freq$_{\mathrm{MED}}$ and COP-Freq$_{\mathrm{HIGH}}$ were significantly higher in the Threat compared to No Threat condition (Table 3; Figure 2).

3.1.2. Biological sex effects

No main effects of biological sex were observed for EDA, perceived anxiety, or any of the five attention focus measures (Table 3). There were significant main effects of biological sex observed for sample entropy ($F_{(1,103)}$ = 5.39, p = 0.022), COP-MPOS ($F_{(1,103)}$ = 8.86, p = 0.004), COP-MPF ($F_{(1,103)}$ = 7.66, p = 0.007) and COP-Freq$_{\mathrm{HIGH}}$ ($F_{(1,103)}$ = 9.04, p = 0.003). Independent of threat, males compared to females had significantly higher sample entropy values (males, mean ± SE = 0.121 ± 0.008; females, mean ± SE = 0.106 ± 0.006), leaned further forward (males, mean ± SE = 52.05 ± 2.93 mm; females, mean ± SE = 40.79 ± 2.71 mm), had higher COP-MPF (males, mean ± SE = 0.36 ± 0.03 Hz; females, mean ± SE = 0.32 ± 0.02 Hz), and displayed higher COP-Freq$_{\mathrm{HIGH}}$ (males, mean ± SE = 0.069 ± 0.008 mm^2/bin; females, mean ± SE = 0.044 ± 0.004 mm^2/bin).

3.1.3. Biological sex by threat interaction effects

There was a significant biological sex by threat interaction effect observed for attention to movement processes ($F_{(1,103)}$ = 4.16, p = 0.044) that supersedes the main effect of threat observed for this measure. Both females and males directed significantly more attention to movement processes in the Threat compared to the No Threat condition (p < 0.001, for both groups) with the threat-induced change appearing to be larger for males (Table 3). However, there were no significant differences between females and males observed in the No Threat (p = 0.365) or Threat (p = 0.232) conditions.

There was also a significant biological sex by threat interaction effect observed for COP-Freq$_{\mathrm{HIGH}}$ ($F_{(1,103)}$ = 6.01, p = 0.016) that supersedes the main effects of threat and biological sex observed for this measure. Follow-up comparisons revealed that COP-Freq$_{\mathrm{HIGH}}$ was significantly higher in the Threat compared to the No Threat condition for both females (p < 0.001) and males (p < 0.001) with the threat-induced change appearing to be larger for males (Table 3). There were also differences between females and males in the No Threat (p = 0.039) and Threat (p = 0.006) conditions.

No other significant biological sex by threat interaction effects were observed.

3.2. Multiple linear regressions

The multiple linear regression analyses revealed that a combination of biological sex and threat-induced changes in physiological arousal, perceived anxiety, and attention focus significantly accounted for changes in COP-RMS (R^2 = 0.235, $F_{(8,96)}$ = 3.69, p < 0.001), COP-Freq$_{\mathrm{LOW}}$ (R^2 = 0.158, $F_{(8,96)}$ = 2.25, p = 0.030), COP-Freq$_{\mathrm{MED}}$ (R^2 = 0.227, $F_{(8,96)}$ = 3.53, p = 0.001), and COP-Freq$_{\mathrm{HIGH}}$ (R^2 = 0.294, $F_{(8,96)}$ = 4.99, p < 0.001), but not sample entropy (R^2 = 0.071, $F_{(8,96)}$ = 0.920, p = 0.504), COP-MPOS (R^2 = 0.101, $F_{(8,96)}$ = 1.35, p = 0.228) or COP-MPF (R^2 = 0.099, $F_{(8,96)}$ = 1.32, p = 0.245; Table 4). Significant predictors were biological sex, EDA, attention to movement processes, and attention to task-irrelevant information. Being male was associated with a larger increase in COP-Freq$_{\mathrm{HIGH}}$ (β = −0.217, p = 0.021) between Threat and No Threat conditions. A larger increase in EDA between Threat and No Threat conditions was significantly associated with a larger increase in COP-Freq$_{\mathrm{MED}}$ (β = 0.325, p < 0.001), and COP-Freq$_{\mathrm{HIGH}}$ (β = 0.298, p = 0.001). A larger increase in attention to movement processes between Threat and No Threat conditions was significantly associated with a larger increase in COP-RMS (β = 0.320, p = 0.005), COP-Freq$_{\mathrm{LOW}}$ (β = 0.304, p = 0.011) and COP-Freq$_{\mathrm{HIGH}}$ (β = 0.309, p = 0.005). A larger decrease in attention to task-irrelevant information was significantly associated with a larger increase in COP-Freq$_{\mathrm{MED}}$ (β = −0.209, p = 0.027).

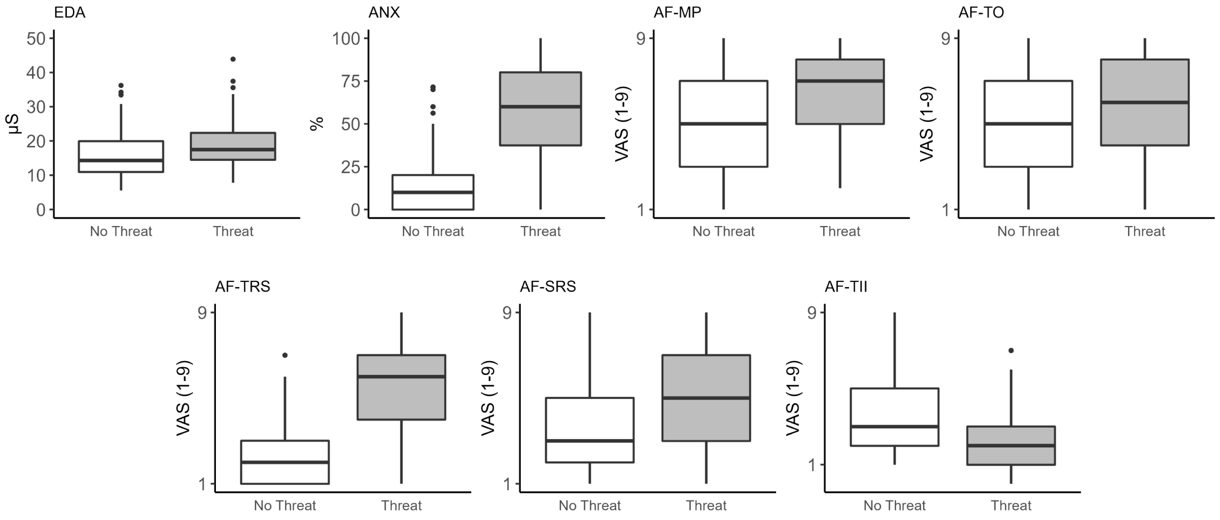

FIGURE 1
Box plots of the effects of threat on physiological, psychological, and attention focus measures. Significant differences were observed for all measures between No Threat and Threat conditions. EDA, electrodermal activity; ANX, perceived anxiety; AF, attention focus; MP, movement processes; TO, task objectives; TRS, threat-related stimuli; SRS, self-regulatory strategies; TII, task-irrelevant information.

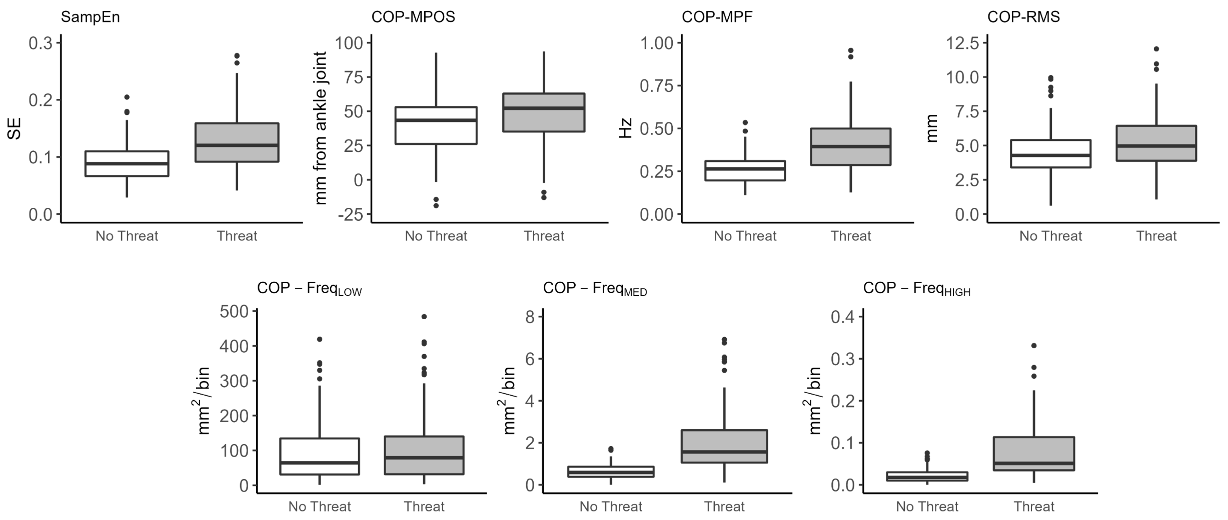

FIGURE 2
Box plots of the effects of threat on sample entropy and traditional balance measures. Significant differences were observed for all measures except for COP- Freq$_{LOW}$ between No Threat and Threat conditions. SampEn, sample entropy; COP, centre of pressure; MPOS, mean position; RMS, root mean square; MPF, mean power frequency; Freq$_{LOW}$, low frequency (0–0.05Hz); Freq$_{MED}$, medium frequency (0.5–1.8Hz); Freq$_{HIGH}$, high frequency (1.8–5Hz).

TABLE 4 Multiple correlations (R^2) and standardized beta weights for regressions between biological sex and threat-induced changes in physiological, psychological, and attention focus measures, and threat-induced changes in sample entropy and traditional balance measures.

	Δ SampEn	Δ COP-MPOS	Δ COP-RMS	Δ COP-MPF	Δ COP-Freq$_{LOW}$	Δ COP-Freq$_{MED}$	Δ COP-Freq$_{HIGH}$
Sex	−0.106	0.000	0.044	−0.164	0.041	−0.152	**−0.217**
Δ EDA	0.109	0.102	0.075	0.230	0.006	**0.325**	**0.298**
Δ ANX	0.073	−0.115	0.132	−0.034	0.084	0.003	0.037
Δ AF-MP	−0.174	0.278	**0.320**	0.037	**0.304**	0.199	**0.309**
Δ AF-TO	−0.116	−0.024	0.110	−0.174	0.089	−0.072	−0.138
Δ AF-TRS	−0.153	−0.088	0.024	−0.009	−0.048	−0.019	0.018
Δ AF-SRS	0.110	0.144	0.020	0.155	0.067	0.130	0.091
Δ AF-TII	−0.003	−0.063	−0.073	−0.041	−0.061	**−0.209**	−0.145
R^2	0.071	0.101	**0.235**	0.099	**0.158**	**0.227**	**0.294**
value of *p*	0.504	0.228	**<0.001**	0.245	**0.030**	**0.001**	**<0.001**

Δ, measures represent change scores between Threat and No Threat conditions; EDA, electrodermal activity; ANX, perceived anxiety; AF, attention focus MP, movement processes; TO, task objectives; TRS, threat-related stimuli; SRS, self-regulatory strategies; TII, task-irrelevant information; SampEn, sample entropy; COP, centre of pressure; MPOS, mean position; RMS, root mean square; MPF, mean power frequency; Freq$_{LOW}$, low frequency (0–0.05 Hz); Freq$_{MED}$, medium frequency (0.5–1.8 Hz); Freq$_{HIGH}$, high frequency (1.8–5 Hz). Bold font indicates a significant model or beta value.

4. Discussion

4.1. Effects of postural threat on physiological, psychological, and attention focus measures

The threat of a postural perturbation significantly altered physiological and psychological state in this group of healthy young adults. As anticipated, physiological arousal and perceptions of anxiety significantly increased when standing with the expectation of receiving a postural perturbation further revealing the efficacy of using this type of threat manipulation to study the effect of emotions on balance control (13, 14, 18, 58, 59). The threat of a postural perturbation also generated broad changes in attention focus. When threatened, participants increased attention towards balance, task objectives, threat-related stimuli, and self-regulatory or coping strategies, and decreased attention to task-irrelevant information. Similar changes in this threat-induced pattern of attention focus have been observed for surface height (11, 12, 17) and other postural perturbation threat (e.g., medial-lateral support surface translations) manipulations (18).

4.2. Effects of postural threat on sample entropy, and its relationships with physiological, psychological, and attention focus measures

A novel objective of this study was to examine the effects of postural threat on sample entropy, a measure of the regularity of the

COP signal used to indicate the attentional involvement in balance control. A decrease in sample entropy is thought to reflect a shift to more attention needed to control balance and less automatic control, while an increase in sample entropy corresponds to less attention required for balance and more automatic control (22, 23). Given this interpretation, coupled with research that shows more attention directed to balance under conditions of postural threat (7, 9, 11–14, 17, 18), it was hypothesized that sample entropy would decrease when threatened. The results of the study revealed that postural threat did have a significant effect on sample entropy. However, this effect was opposite to that expected as an increase in sample entropy was observed when standing with compared to without the expectation of receiving a support surface perturbation. Interpreting this based on the assumptions underlying the sample entropy, attention, and automaticity relationship (22, 23), higher sample entropy values would suggest the use of a more automatic balance control strategy when threatened. Although this result is opposite to that theorized and incongruent with participants reporting more attention directed to their balance when threatened in this study, and other research that reveals greater cortical involvement in balance when threatened (60, 61), it does align with recent work that showed an increase in sample entropy in combination with increased conscious processing of balance when older adults experienced a surface height threat (37, 38). In this work, the authors suggested that the threat-induced increase in sample entropy occurs regardless of the threat-induced increase in conscious processing of balance. The increase in conscious processing of balance may serve to constrain automatic threat-induced balance changes acting as a strategy to limit the irregularity or unpredictability of the balance control system. Supporting this view, when participants were threatened and distracted from attending to their balance, sample entropy values continued to increase (37).

A second original aspect of the current study was exploring whether threat-induced alterations in physiological state, psychological state, and attention focus, including attention to balance, contributed to explaining threat-induced changes in sample entropy. The results of the multiple linear regression analysis showed that a combination of biological sex and threat-induced changes in physiological state, psychological state and attention focus did not predict threat-induced changes in sample entropy. These results did not confirm the hypothesis that attention focus and in particular attention to balance would be a significant predictor of changes in sample entropy and suggest that sample entropy may not be as susceptible to change through this mechanism under conditions of postural threat.

4.3. Interpretation of postural threat-induced changes in sample entropy

Despite the results of this study not supporting the theoretical assumptions of the sample entropy, attention and automaticity relationship, other possible explanations for the increase in sample entropy when standing with the expectation of receiving a postural perturbation need to be considered. It has been suggested that an increase or decrease in sample entropy can be interpreted in different ways (21). One explanation for the increase in sample entropy when threatened is that it reflects a heightened level of alertness and a shift to a more vigilant control of balance that prepares the system to deal with the threat of the unexpected postural perturbation. As physiological arousal and vigilance, although considered independent constructs, often vary together (62), it would be expected that changes in physiological arousal should be related to changes in sample entropy to support this interpretation. However, although physiological arousal (i.e., EDA) and sample entropy increased when threatened, threat-induced changes in physiological arousal were not associated with threat-induced changes in sample entropy.

A second explanation for the increase in sample entropy when threatened is that it may reveal an inability to use effective attention strategies. This interpretation may be supported by the threat-induced changes in attention focus to multiple sources beyond simply directing more attention to balance when threatened. This broad impairment in attention control (e.g., to balance, task objectives, threat-related stimuli, self-regulatory/coping strategies) may have produced greater interference when threatened, leading to the increase in sample entropy. Past research has used specific external and internal attention focus instructions or concurrent cognitive tasks to distract attention from balance to support the sample entropy, attention and automaticity relationship (28–31, 34, 35). If attention to balance had been the only change in attention reported by healthy young adults when threatened, the expected decrease in sample entropy may have been observed.

A third interpretation of the increase in sample entropy when threatened is that it may reflect greater noise present in the balance control system. This view may be supported by research that has revealed increased sensory gain in multiple sensory systems (e.g., proprioceptive, vestibular) (59, 63–68) and increased cortical excitability (69, 70) in response to a surface height threat.

At this point, the results of the current study are not able to definitively support one of these interpretations. As research has shown that threat-induced changes in balance are not always aligned with changes in attention or perceptions of sway (10), it is likely that a combination of attentional and neurophysiological mechanisms combine to influence the postural control strategy used when threatened (1).

4.4. Effects of postural threat on traditional balance measures

The balance strategy as described using traditional COP summary measures was also significantly different when standing with compared to without the expectation of receiving a postural perturbation. Healthy young adults leaned further forward and demonstrated increased amplitude and frequency of COP displacements, specifically in the higher frequency bands (i.e., > 0.5 Hz) when threatened. Increased amplitude and frequency of COP displacements in the medial-lateral direction have also been observed in response to the threat of a medial-lateral support surface translation (18). The findings from the current study reinforce but also expand upon the results reported in the two published studies from which the data set for this study was derived (13, 14). For example, inconsistencies in threat-induced changes in amplitude of COP displacements (i.e., increased or no change) between studies were resolved and threat-induced increases in the higher frequency components of sway which contribute to the increase in MPF across studies were confirmed in the larger sample used in the present study.

Past research has revealed inconsistent relationships between threat-induced changes in physiological state, psychological state, attention focus, and traditional balance measures (7, 13, 17). Therefore, it was important to examine these relationships in a larger sample to potentially inform about the mechanisms underlying threat-induced changes in balance. Combining the data from two previously published studies revealed that although biological sex, physiological state, psychological state, and attention focus measures were not related to sample entropy, they were related to specific traditional balance measures. The multiple linear regressions showed that a combination of these measures could predict amplitude of sway, and the low, medium, and high-frequency components of sway. Physiological arousal and attention to balance emerged as the most common significant predictors. A larger increase in attention to balance was associated with leaning further forward and a larger increase in low and high-frequency sway, while a larger increase in EDA was associated with a larger increase in medium and high-frequency components of sway. These relationships are different from these reported by Johnson and colleagues who reported larger increases in attention to balance being associated with leaning further forward and having larger increases in amplitude of sway, while larger increases in attention to self-regulatory/coping strategies were associated with larger increases in sway frequency (i.e., MPF) (13). Taken together, these results partially support the work of Ellmers and colleagues who showed parallel increases in conscious motor processing, sample entropy and higher frequency components of sway when older adults faced a surface height threat manipulation (37).

4.5. Sex differences in threat-induced behaviour

A secondary aim of this study was to explore how biological sex interacted with threat-induced changes in, and associations between, psychological, physiological, attention focus and balance responses. Females and males responded in much the same way when standing with the expectation of a receiving a postural perturbation. Threat-induced changes in physiological arousal and perceived anxiety were not influenced by sex. Although previous research has revealed sex-differences in autonomic responses to stress and anxiety (39–41), these sex differences did not emerge in the current study. Although there were some sex differences in balance control that were found independent of threat, only attention focus to balance and high-frequency sway measures revealed a significant interaction between sex and threat, and sex only emerged as a significant predictor along with attention to balance and physiological arousal for high-frequency sway. In general, it appears that only the magnitude but not the direction of the threat-induced change was different between females and males, with males having a larger change in attention to balance and high-frequency sway when threatened. Previous sex differences in balance control in response to a surface height threat have been observed. However, these findings were different from the current results as females compared to males demonstrated a larger increase in MPF when standing on a high compared to low surface height (2).

Despite sex not significantly interacting with many threat-related changes in psychological, physiological, attention focus and balance responses and not emerging as a significant predictor for these threat-related changes, future work should be directed to identify if a combination of sex and personality traits (e.g., trait anxiety, movement reinvestment, and risk taking), although not significantly different between males and females in this study, can explain threat-induced behaviour.

4.6. Limitations

The results of this study are only generalizable to healthy young adults experiencing a postural perturbation threat. It is unknown if changes in sample entropy and relationships between sample entropy and other physiological, psychological, attention focus, and balance measures may differ under different threat contexts or in different populations (e.g., older adults reporting a fear of falling). The unbalanced number of females and males in this study may have also been a limitation for observing sex differences. Another possible limitation was that the COP time-series data from the Johnson and colleagues (2020) study (14) was shortened to 30-s so as to combine the data with that from the Johnson and colleagues (2019) study (13). Although this allowed for consistency in time-series length when comparing traditional balance measures and sample entropy, it has been recommended to use at least time-series of 60-s for calculating traditional balance measures (47) and sample entropy (48).

5. Conclusion

A robust emotional response, as evidenced by increases in physiological arousal and perceived anxiety, and more conscious control of balance, were observed when standing with compared to without the threat of a postural perturbation. Sample entropy and high-frequency postural sway increased when threatened suggesting a shift to a more automatic control strategy. Given the theoretical assumptions underlying the interpretation of sample entropy, higher sample entropy values are typically associated with less attention to balance (22, 23). Although the current findings are incongruent with this expected relationship, directing more conscious control to balance when threatened may act to constrain these threat-induced automatic changes to balance (37). However, given the evidence of broad threat-related changes in attention focus (i.e., shifts in attention focus to multiple sources), this increase in sample entropy may also be interpreted as an inability to employ effective attention control in this threatening context (21). As past research has also revealed changes in sensory and cortical processing when threatened (1), an increase in sample entropy may reflect increased noise in the balance control system. It is likely that the effects of threat on balance control rely on a complex interaction between changes in attentional and neurophysiological processes (1). Future work should be directed to investigating complementary traditional and non-linear balance measures to inform about the potential mechanisms underlying changes in balance under different threat scenarios.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving human participants were reviewed and approved by Brock University Bioscience Research Ethics Board. The patients/participants provided their written informed consent to participate in this study.

Author contributions

OF, KM, CT, MC, and AA contributed to the conception and design of the study. OF and KM organized and analyzed the data. AA and MC contributed to the first draft of the manuscript. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

Supplementary material

The Supplementary material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fneur.2023.1179237/full#supplementary-material

References

1. Adkin AL, Carpenter MG. New insights on emotional contributions to human postural control. *Front Neurol.* (2018) 9:789. doi: 10.3389/fneur.2018.00789

2. Carpenter MG, Frank JS, Silcher CP. Surface height effects on postural control: a hypothesis for a stiffness strategy for stance. *J Vestib Res.* (1999) 9:277–86. doi: 10.3233/VES-1999-9405

3. Adkin AL, Frank JS, Carpenter MG, Peysar GW. Postural control is scaled to level of postural threat. *Gait Posture.* (2000) 12:87–93. doi: 10.1016/s0966-6362(00)00057-6

4. Carpenter MG, Adkin AL, Brawley LR, Frank JS. Postural, physiological and psychological reactions to challenging balance: does age make a difference? *Age Ageing.* (2006) 35:298–303. doi: 10.1093/ageing/afl002

5. Hauck LJ, Carpenter MG, Frank JS. Task-specific measures of balance efficacy, anxiety, and stability and their relationship to clinical balance performance. *Gait Posture.* (2008) 27:676–82. doi: 10.1016/j.gaitpost.2007.09.002

6. Davis JR, Campbell AD, Adkin AL, Carpenter MG. The relationship between fear of falling and human postural control. *Gait Posture.* (2009) 29:275–9. doi: 10.1016/j.gaitpost.2008.09.006

7. Huffman JL, Horslen BC, Carpenter MG, Adkin AL. Does increased postural threat lead to more conscious control of posture? *Gait Posture.* (2009) 30:528–32. doi: 10.1016/j.gaitpost.2009.08.001

8. Cleworth TW, Horslen BC, Carpenter MG. Influence of real and virtual heights on standing balance. *Gait Posture.* (2012) 36:172–6. doi: 10.1016/j.gaitpost.2012.02.010

9. Zaback M, Cleworth TW, Carpenter MG, Adkin AL. Personality traits and individual differences predict threat-induced changes in postural control. *Hum Mov Sci.* (2015) 40:393–409. doi: 10.1016/j.humov.2015.01.015

10. Cleworth TW, Carpenter MG. Postural threat influences conscious perception of postural sway. *Neurosci Lett.* (2016) 620:127–31. doi: 10.1016/j.neulet.2016.03.032

11. Zaback M, Adkin AL, Carpenter MG. Adaptation of emotional state and standing balance parameters following repeated exposure to height-induced postural threat. *Sci Rep.* (2019) 9:12449. doi: 10.1038/s41598-019-48722-z

12. Zaback M, Luu MJ, Adkin AL, Carpenter MG. Selective preservation of changes to standing balance control despite psychological and autonomic habituation to a postural threat. *Sci Rep.* (2021) 11:384. doi: 10.1038/s41598-020-79417-5

13. Johnson KJ, Zaback M, Tokuno CD, Carpenter MG, Adkin AL. Exploring the relationship between threat-related changes in anxiety, attention focus, and postural control. *Psychol Res.* (2019) 83:445–58. doi: 10.1007/s00426-017-0940-0

14. Johnson KJ, Watson AM, Tokuno CD, Carpenter MG, Adkin AL. The effects of distraction on threat-related changes in standing balance control. *Neurosci Lett.* (2020) 716:134635. doi: 10.1016/j.neulet.2019.134635

15. Geh CL, Beauchamp MR, Crocker PR, Carpenter MG. Assessed and distressed: white-coat effects on clinical balance performance. *J Psychosom Res.* (2011) 70:45–51. doi: 10.1016/j.jpsychores.2010.09.008

16. Horslen BC, Carpenter MG. Arousal, valence and their relative effects on postural control. *Exp Brain Res.* (2011) 215:27–34. doi: 10.1007/s00221-011-2867-9

17. Zaback M, Carpenter MG, Adkin AL. Threat-induced changes in attention during tests of static and anticipatory postural control. *Gait Posture.* (2016) 45:19–24. doi: 10.1016/j.gaitpost.2015.12.033

18. Johnson KJ, Zaback M, Tokuno CD, Carpenter MG, Adkin AL. Repeated exposure to the threat of perturbation induces emotional, cognitive, and postural adaptations in young and older adults. *Exp Gerontol.* (2019) 122:109–15. doi: 10.1016/j.exger.2019.04.015

19. Richman JS, Moorman JR. Physiological time-series analysis using approximate entropy and sample entropy. *Am J Physiol Heart Circ Physiol.* (2000) 278:H2039–49. doi: 10.1152/ajpheart.2000.278.6.H2039

20. Kędziorek J, Błażkiewicz M. Nonlinear measures to evaluate upright postural stability: a systematic review. *Entropy (Basel).* (2020) 22:1357. doi: 10.3390/e22121357

21. Borg FG, Laxåback G. Entropy of balance--some recent results. *J Neuroeng Rehabil.* (2010) 7:38. doi: 10.1186/1743-0003-7-38

22. Roerdink M, De Haart M, Daffertshofer A, Donker SF, Geurts ACH, Beek PJ. Dynamical structure of center-of-pressure trajectories in patients recovering from stroke. *Exp Brain Res.* (2006) 174:256–69. doi: 10.1007/s00221-006-0441-7

23. Roerdink M, Hlavackova P, Vuillerme N. Center-of-pressure regularity as a marker for attentional investment in postural control: a comparison between sitting and standing postures. *Hum Mov Sci.* (2011) 30:203–12. doi: 10.1016/j.humov.2010.04.005

24. Lubetzky AV, Hujsak BD, Kelly JL, Fu G, Perlin K. Control mechanisms of static and dynamic balance in adults with and without vestibular dysfunction in oculus virtual environments. *PM&R.* (2018) 10:1223–1236.e2. doi: 10.1016/j.pmrj.2018.07.001

25. Stins JF, Michielsen ME, Roerdink M, Beek PJ. Sway regularity reflects attentional involvement in postural control: effects of expertise, vision and cognition. *Gait Posture.* (2009) 30:106–9. doi: 10.1016/j.gaitpost.2009.04.001

26. Donker SF, Roerdink M, Greven AJ, Beek PJ. Regularity of center-of-pressure trajectories depends on the amount of attention invested in postural control. *Exp Brain Res.* (2007) 181:1–11. doi: 10.1007/s00221-007-0905-4

27. Franco C, Fleury A, Diot B, Vuillerme N. Applying entropy to human Center of Foot Pressure Data to assess attention Investment in Balance Control. *Annu Int Conf IEEE Eng Med Biol Soc.* (2018) 2018:5586–9. doi: 10.1109/EMBC.2018.8513533

28. Rhea CK, Diekfuss JA, Fairbrother JT, Raisbeck LD. Postural control entropy is increased when adopting an external focus of attention. *Mot Control.* (2019) 23:230–42. doi: 10.1123/mc.2017-0089

29. Becker KA, Hung CJ. Attentional focus influences sample entropy in a balancing task. *Hum Mov Sci.* (2020) 72:102631. doi: 10.1016/j.humov.2020.102631

30. Potvin-Desrochers A, Richer N, Lajoie Y. Cognitive tasks promote automatization of postural control in young and older adults. *Gait Posture.* (2017) 57:40–5. doi: 10.1016/j.gaitpost.2017.05.019

31. Richer N, Lajoie Y. Automaticity of postural control while dual-tasking revealed in Young and older adults. *Exp Aging Res.* (2020) 46:1–21. doi: 10.1080/0361073X.2019.1693044

32. Wulf G. Attentional focus and motor learning: a review of 15 years. *Int Rev Sport Exerc Psychol.* (2013) 6:77–104. doi: 10.1080/1750984X.2012.723728

33. Uiga L, Capio CM, Ryu D, Wilson MR, Masters RSW. The role of conscious control in maintaining stable posture. *Hum Mov Sci.* (2018) 57:442–50. doi: 10.1016/j.humov.2017.10.008

34. Kal EC, Young WR, Ellmers TJ. Balance capacity influences the effects of conscious movement processing on postural control in older adults. *Hum Mov Sci.* (2022) 82:102933. doi: 10.1016/j.humov.2022.102933

35. Jie LJ, Kal E, Ellmers TJ, Rosier J, Meijer K, Boonstra TW. The effects of conscious movement processing on the neuromuscular control of posture. *Neuroscience.* (2023) 509:63–73. doi: 10.1016/j.neuroscience.2022.11.010

36. Stins JF, Roerdink M, Beek PJ. To freeze or not to freeze? Affective and cognitive perturbations have markedly different effects on postural control. *Hum Mov Sci.* (2011) 30:190–202. doi: 10.1016/j.humov.2010.05.013

37. Ellmers TJ, Kal EC, Young WR. Consciously processing balance leads to distorted perceptions of instability in older adults. *J Neurol.* (2021) 268:1374–84. doi: 10.1007/s00415-020-10288-6

38. Ellmers TJ, Wilson MR, Kal EC, Young WR. Standing up to threats: translating the two-system model of fear to balance control in older adults. *Exp Gerontol.* (2022) 158:111647. doi: 10.1016/j.exger.2021.111647

39. Wilhelm FH, Rattel JA, Wegerer M, Liedlgruber M, Schweighofer S, Kreibig SD, et al. Attend or defend? Sex differences in behavioral, autonomic, and respiratory response patterns to emotion–eliciting films. *Biol Psychol.* (2017) 130:30–40. doi: 10.1016/j.biopsycho.2017.10.006

40. Velasco ER, Florido A, Milad MR, Andero R. Sex differences in fear extinction. *Neurosci Biobehav Rev.* (2019) 103:81–108. doi: 10.1016/j.neubiorev.2019.05.020

41. Liu Q, Zhang W. Sex differences in stress reactivity to the trier social stress test in virtual reality. *Psychol Res Behav Manag.* (2020) 13:859–69. doi: 10.2147/PRBM.S268039

42. Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, Jacobs GA. *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press (1983).

43. Masters RSW, Eves FF, Maxwell JP. Development of a movement specific reinvestment scale. *Presented at the 11th World Congress of Sport Psychology*, Sydney, Australia (2005)

44. Masters R, Maxwell J. The theory of reinvestment. *Int Rev Sport Exerc Psychol.* (2008) 1:160–83. doi: 10.1080/17509840802287218

45. Blais AR, Weber EU. A domain-specific risk-taking (DOSPERT) scale for adult populations. *Judgment Decis Making.* (2006) 1:33–47. doi: 10.1017/S1930297500000334

46. Zaback M, Reiter ER, Adkin AL, Carpenter MG. Initial experience of balance assessment introduces 'first trial' effects on emotional state and postural control. *Gait Posture.* (2021) 88:116–21. doi: 10.1016/j.gaitpost.2021.05.013

47. Carpenter MG, Frank JS, Winter DA, Peysar GW. Sampling duration effects on Centre of pressure summary measures. *Gait Posture.* (2001) 13:35–40. doi: 10.1016/s0966-6362(00)00093-x

48. Montesinos L, Castaldo R, Pecchia L. On the use of approximate entropy and sample entropy with Centre of pressure time-series. *J Neuroeng Rehabil.* (2018) 15:116. doi: 10.1186/s12984-018-0465-9

49. Boucsein W. *Electrodermal Activity*. *2nd* ed. New York: Springer (2012).

50. Cohen P, Cohen J, Aiken LS, West SG. The problem of units and the circumstances for POMP. *Multivar Behav Res.* (1999) 34:315–46. doi: 10.1207/S15327906MBR3403_2

51. Lubetzky AV, Harel D, Lubetzky E. On the effects of signal processing on sample entropy for postural control. *PLoS One.* (2018) 13:e0193460. doi: 10.1371/journal.pone.0193460

52. Duarte M, Freitas SMSF. Review of posturography based on force platform for balance evaluation. *Rev Bras Fisioter.* (2010) 14:183–92. doi: 10.1590/S1413-35552010000300003

53. Palmieri RM, Ingersoll CD, Stone MB, Krause BA. Center-of-pressure parameters used in the assessment of postural control. *J Sport Rehabil.* (2002) 11:51–66. doi: 10.1123/jsr.11.1.51

54. Schubert P, Kirchner M, Schmidtbleicher D, Haas C. About the structure of posturography: sampling duration, parametrization, focus of attention (part I). *J Biomed Sci Eng.* (2012) 5:496–507. doi: 10.4236/jbise.2012.59062

55. Quijoux F, Nicolaï A, Chairi I, Bargiotas I, Ricard D, Yelnik A, et al. A review of center of pressure (COP) variables to quantify standing balance in elderly people: algorithms and open-access code. *Physiol Rep.* (2021) 9:e15067. doi: 10.14814/phy2.15067

56. Reynolds RF. The ability to voluntarily control sway reflects the difficulty of the standing task. *Gait Posture.* (2010) 31:78–81. doi: 10.1016/j.gaitpost.2009.09.001

57. Beckham G, Suchomel T, Mizuguchi S. Force plate use in performance monitoring and sport science testing. *New Stud Athl.* (2014) 29:25–37.

58. Shaw JA, Stefanyk LE, Frank JS, Jog MS, Adkin AL. Effects of age and pathology on stance modifications in response to increased postural threat. *Gait Posture.* (2012) 35:658–61. doi: 10.1016/j.gaitpost.2011.12.020

59. Horslen BC, Murnaghan CD, Inglis JT, Chua R, Carpenter MG. Effects of postural threat on spinal stretch reflexes: evidence for increased muscle spindle sensitivity? *J Neurophysiol.* (2013) 110:899–906. doi: 10.1152/jn.00065.2013

60. Zaback M, Adkin AL, Chua R, Inglis JT, Carpenter MG. Facilitation and habituation of cortical and subcortical control of standing balance following repeated exposure to a height-related postural threat. *Neuroscience.* (2022) 487:8–25. doi: 10.1016/j.neuroscience.2022.01.012

61. Zaback M, Missen KJ, Adkin AL, Chua R, Inglis JT, Carpenter MG. Cortical potentials time-locked to discrete postural events during quiet standing are facilitated during postural threat exposure. *J Physiol.* (2023). doi: 10.1113/JP284055

62. Oken BS, Salinsky MC, Elsas S. Vigilance, alertness, or sustained attention: physiological basis and measurement. *Clin Neurophysiol.* (2006) 117:1885–901. doi: 10.1016/j.clinph.2006.01.017

63. Davis JR, Horslen BC, Nishikawa K, Fukushima K, Chua R, Inglis JT, et al. Human proprioceptive adaptations during states of height-induced fear and anxiety. *J Neurophysiol.* (2011) 106:3082–90. doi: 10.1152/jn.01030.2010

64. Horslen BC, Dakin CJ, Inglis JT, Blouin JS, Carpenter MG. Modulation of human vestibular reflexes with increased postural threat. *J Physiol.* (2014) 592:3671–85. doi: 10.1113/jphysiol.2014.270744

65. Naranjo EN, Allum JHJ, Inglis JT, Carpenter MG. Increased gain of vestibulospinal potentials evoked in neck and leg muscles when standing under height-induced postural threat. *Neuroscience.* (2015) 293:45–54. doi: 10.1016/j.neuroscience.2015.02.026

66. Naranjo EN, Cleworth TW, Allum JHJ, Inglis JT, Lea J, Westerberg BD, et al. Vestibulo-spinal and vestibulo-ocular reflexes are modulated when standing with increased postural threat. *J Neurophysiol.* (2016) 115:833–42. doi: 10.1152/jn.00626.2015

67. Lim SB, Cleworth TW, Horslen BC, Blouin JS, Inglis JT, Carpenter MG. Postural threat influences vestibular-evoked muscular responses. *J Neurophysiol.* (2016) 117:604–11. doi: 10.1152/jn.00712.2016

68. Horslen BC, Zaback M, Inglis JT, Blouin JS, Carpenter MG. Increased human stretch reflex dynamic sensitivity with height-induced postural threat: increased stretch reflex dynamic sensitivity with postural threat. *J Physiol.* (2018) 596:5251–65. doi: 10.1113/JP276459

69. Adkin AL, Campbell AD, Chua R, Carpenter MG. The influence of postural threat on the cortical response to unpredictable and predictable postural perturbations. *Neurosci Lett.* (2008) 435:120–5. doi: 10.1016/j.neulet.2008.02.018

70. Sibley KM, Mochizuki G, Frank JS, McIlroy WE. The relationship between physiological arousal and cortical and autonomic responses to postural instability. *Exp Brain Res.* (2010) 203:533–40. doi: 10.1007/s00221-010-2257-8

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Cortical facilitation of tactile afferents during the preparation of a body weight transfer when standing on a biomimetic surface

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Self-generated movement shapes tactile perception, but few studies have investigated the brain mechanisms involved in the processing of the mechanical signals related to the static and transient skin deformations generated by forces and pressures exerted between the foot skin and the standing surface. We recently found that standing on a biomimetic surface (i.e., inspired by the characteristics of mechanoreceptors and skin dermatoglyphics), that magnified skin–surface interaction, increased the sensory flow to the somatosensory cortex and improved balance control compared to standing on control (e.g., smooth) surfaces. In this study, we tested whether the well-known sensory suppression that occurs during movements is alleviated when the tactile afferent signal becomes relevant with the use of a biomimetic surface. Eyes-closed participants ($n = 25$) self-stimulated their foot cutaneous receptors by shifting their body weight toward one of their legs while standing on either a biomimetic or a control (smooth) surface. In a control task, similar forces were exerted on the surfaces (i.e., similar skin–surface interaction) by passive translations of the surfaces. Sensory gating was assessed by measuring the amplitude of the somatosensory-evoked potential over the vertex (SEP, recorded by EEG). Significantly larger and shorter SEPs were found when participants stood on the biomimetic surface. This was observed whether the forces exerted on the surface were self-generated or passively generated. Contrary to our prediction, we found that the sensory attenuation related to the self-generated movement did not significantly differ between the biomimetic and control surfaces. However, we observed an increase in gamma activity (30–50 Hz) over centroparietal regions during the preparation phase of the weight shift only when participants stood on the biomimetic surface. This result might suggest that gamma-band oscillations play an important functional role in processing behaviorally relevant stimuli during the early stages of body weight transfer.

KEYWORDS

plantar sole afferents, voluntary movement, sensory gating, biomimetic surface, balance, EEG, somatosensory evoked potentials (SEPs), gamma oscillations

Introduction

Inputs from the foot cutaneous receptors provide information on the body's position in space while standing or walking. Our brain has the ability to control the amount of these cutaneous cues that will be processed by filtering out or increasing their transmission. Since the seminal study of Chapin and Woodward (1) in rats, and later ones in humans (2–5), the transmission of cutaneous inputs from the periphery to the cortical level has been probed by assessing the brain areas' sensitivity to electric skin stimulation. Sensory inputs can undergo the so-called movement-related sensory gating (6, 7). This suppression or attenuation of sensory inputs corresponds to a top-down filtering of the afferent information and is hypothesized to be linked to motor prediction: an efferent signal from motor areas canceling out the predicted sensory reafferences that arise during voluntary movements (8, 9). It has been proposed that this sensory gating's role is to differentiate between sensations created by one's own movements and sensations resulting from external stimuli thereby assigning greater weight to less predictable external sensory inputs (9, 10). While the movement is accompanied by sensory gating, it is also generally acknowledged that when sensory information is relevant to behavior, the movement-related sensory gating can be partially alleviated (11–13). For example, using electric stimulation of the tibial nerve containing mainly afferents from the sole of the foot, Duysens et al. (3) demonstrated that during locomotion, there was a phase-dependent modulation of the sensory suppression. The gating was partly alleviated before footfall, that is likely to anticipate the need for cutaneous information for the forthcoming foot placement. The use of electrical stimulation ensures that stimulation remains rather constant throughout the movement, but this has a drawback since the stimulation is without informational contents and non-selective for the types of cutaneous afferents activated. Hence, the richness of information provided by the four functionally distinct types of tactile sensors encoding skin mechanical deformation is lost (i.e., information on the time course, magnitude, direction, and spatial distribution of contact forces, skin stretches, and the friction between contacted surfaces and the skin; see [(14, 15) for reviews].

On the basis of previous behavioral and electrophysiological studies which show that movement-related gating can be partly alleviated when sensory information is relevant to the task (3, 13), we tested whether the enhancement of relevant information for balance control enabled by specific skin-biomimetic surface contact interaction would help counteract the movement-related sensory suppression. To test this hypothesis, we recorded the cortical responses to skin/surface interaction (i.e., somatosensory-evoked potential, SEP) because it represents the amount of sensory transmission and the early sensory processes (16–18). This was done when standing participants voluntarily shifted their body weight laterally toward one foot (hereafter named "active task"). The body weight shift is known to be initiated by exerting forces onto the supporting surface [e.g., (19)], which in turn stimulates the cutaneous receptors of the plantar sole. We compared the SEP recorded when the participants stood either on a biomimetic or a smooth surface (i.e., surfaces enhancing or impoverishing relevant tactile information, respectively). Indeed, a recent study showed faster and greater responses of the somatosensory cortex when the participants were resting upright on a translating biomimetic surface than on a smooth surface (20). We also measured the SEP in a passive task, where the participants were standing motionless and similar forces as in the active task were passively generated by translating the surfaces under the participants' feet (i.e., similar skin–surface contact interaction as in the active task). Since there was no voluntary movement of the participant that could have induced sensory suppression (at the time of the SEP measurement), this passive task allowed us to normalize, for both types of surfaces, the amount of sensory gating during the self-generated movement. In most of the studies, the SEP was found to be reduced by ∼55 to 70% with respect to the SEP measured in a "resting condition" [e.g., (3, 5, 7, 21)]. We expected that executing the voluntary body weight transfer on a biomimetic surface providing relevant tactile cues might suppress or at least lessen the sensory gating observed during movement execution.

Moreover, to move the whole body safely while standing, the brain must be informed about the body's position relative to the support surface prior to body motion (22–25). The foot sole's cutaneous receptors are thought to contribute to this information because, while the body is motionless, the foot sole undergoes pressure variations due to postural sways that stimulate the cutaneous receptors (26). For instance, Mouchnino et al. (5) [see also (27)] observed that the transmission of foot cutaneous inputs to the cortex (following electric stimulation of the foot sole) was facilitated during the preparation stage of a step movement (∼700 ms before motor execution) compared to a standing condition without step preparation. This sensory facilitation during the preparation of the upcoming stepping movement could contribute to building up an accurate representation of the body's position in space. We hypothesized that the biomimetic surface that enhances the stimulation of foot mechanoreceptors should increase the efficiency of the sensory processing during the preparation of the body weight shift. To test this hypothesis, we analyzed gamma event-related synchronization (ERS) during the preparation phase preceding the body weight transfer toward the supporting leg. Indeed, previous studies show that movement preparation is accompanied by an increased synchronization of cyclical fluctuations in neuronal excitability across populations of neurons over the sensorimotor cortex in the gamma bandwidth (30–50 Hz), before the start of the movement (28, 29). The current knowledge on the functional significance of gamma event-related synchronization (ERS) is largely related to the preparation of the movement characteristics [e.g., increased ERS was scaled with movement distance and peak velocity (29)]. However, converging lines of observations point to the critical role of gamma ERS to increase the efficiency of sensory processing during the movement preparation period. Indeed, Tatti et al. (29) found greater synchronization over the posterior regions known to integrate motor signals with proprioceptive and visual information (30–32), and Palmer et al.'s study (33) showed a relationship between increased gamma ERS and enhanced perception for the forthcoming force reproduction (i.e., fewer errors). Based on the abovementioned studies, we expected that participants who are preparing to shift their body weight to one leg will show greater gamma activity in the somatosensory and parietal cortices when they are standing on a biomimetic surface compared to a smooth surface.

Materials and methods

Participants and task

Twenty-five participants (13 women) without any known neurological and motor disorders participated in the experiment (mean age 23 ± 2 years, mean weight 68 ± 12 kg). All participants gave their written informed consent to take part in this study, which conformed to the ethical standards set out in the Declaration of Helsinki and which was approved by the research ethics committee CERSTAPS (IRB00012476-2021-09-12-140).

Participants were requested to stand barefoot with their feet at a natural distance apart on different types of surfaces (see below), fixed in the middle of a movable force platform. They wore a safety harness attached to the ceiling. The feet's position was kept constant across the experimental session. We used a setup employed in previous studies for stimulating foot tactile afferents (34). The platform was positioned on two guide rails (Bosh Rexroth) with a ball-bearing system to reduce friction. The platform was held stationary by an electromagnet and could be translated to the right by deactivating the electromagnet. A cable attached to this platform (at the opposite side of the electromagnet) was connected at the other end to a pulley system with a load fixed to its extremity (Figure 1A). The load was adapted to the participants' weight, such that switching off the electromagnet allowed the platform to accelerate to the participants' right, without endangering their balance.

At the start of a trial, the participants looked at a fixation point which was positioned at the eye level, 2 m directly in front of them. They were asked to close their eyes upon receiving verbal instructions on the upcoming task and to remain still. The instruction indicated one of these three tasks: active (40 trials), passive (40 trials), and stationary (10 trials). The experimental session, therefore, included 90 trials which were randomly distributed.

During the active task, participants had to shift their body weight toward their right foot, while keeping both feet on the surface. This body weight shift was initiated by propulsive forces (vigorous pressure onto the ground) in the leftward direction (Figure 1B). The participants were instructed to perform their body movement ∼2 s after receiving the instruction on the upcoming task. For the passive task, the electromagnet was deactivated ∼2 s after this instruction. This released the platform which translated to the right of the participants, thereby triggering passive shear forces in the leftward direction. The participants were asked to remain upright during the translations. The stationary trials were used to analyze vibration and EEG signals (see below). During these trials, the participants had to remain still during 20 s.

To make the shear forces comparable between the active and passive tasks (Figure 1B), before the experimental session, we measured for each participant the lateral forces that were passively elicited by the platform translation in three trials. Afterward, we asked the participants to shift their body weight in a rapid and accurate manner to reproduce the same forces as those produced in these "passive" trials. The experimenter controlled for the potential difference (e.g., forces rise time and amplitude) between these forces. Verbal corrective instructions were provided to the participant if necessary. The participants needed ∼3 to 10 trials to match the initial passive forces (i.e., those evoking the SEPs) in a satisfactory manner. During these trials, the participants were standing on the surface (i.e., biomimetic or smooth, see below for their description) on which they were standing at the start of their experimental session (see below). Note that this training session, which lasted ∼3 min, unlikely altered the sensory gating [see (35), for evidence of unaltered SEP gating during repetitive practice]. The analyses showed that, for both types of surfaces, the amplitude of the shear forces exerted by participants on the platform in the active task was fairly similar to those recorded in the passive task (see Figure 2A). An ANOVA indicated that the peak shear forces

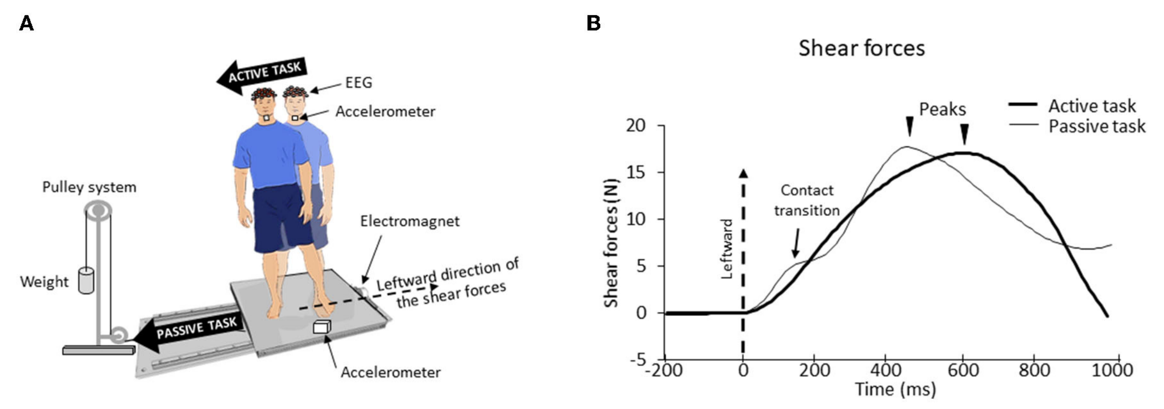

FIGURE 1
(A) Experimental setup. The participant stood barefoot on a force platform which, on deactivation of the electromagnet, would undergo a translation due to gravity loading. **(B)** Mean lateral forces for one representative participant for both surfaces (biomimetic, smooth) during active and passive tasks. The time 0 (vertical dotted line) corresponds to the onset of the shear forces change. For the passive task, the first peak force (here smoothed due to the average) corresponds to the maximal extensibility of the skin under the feet until the frictional force (i.e., shear force) can no longer resist the sliding leading to transient variations of the local strain distribution ("skin–surface contact transitions"). Afterward, a second force peak occurred, corresponding to a voluntary postural reaction.

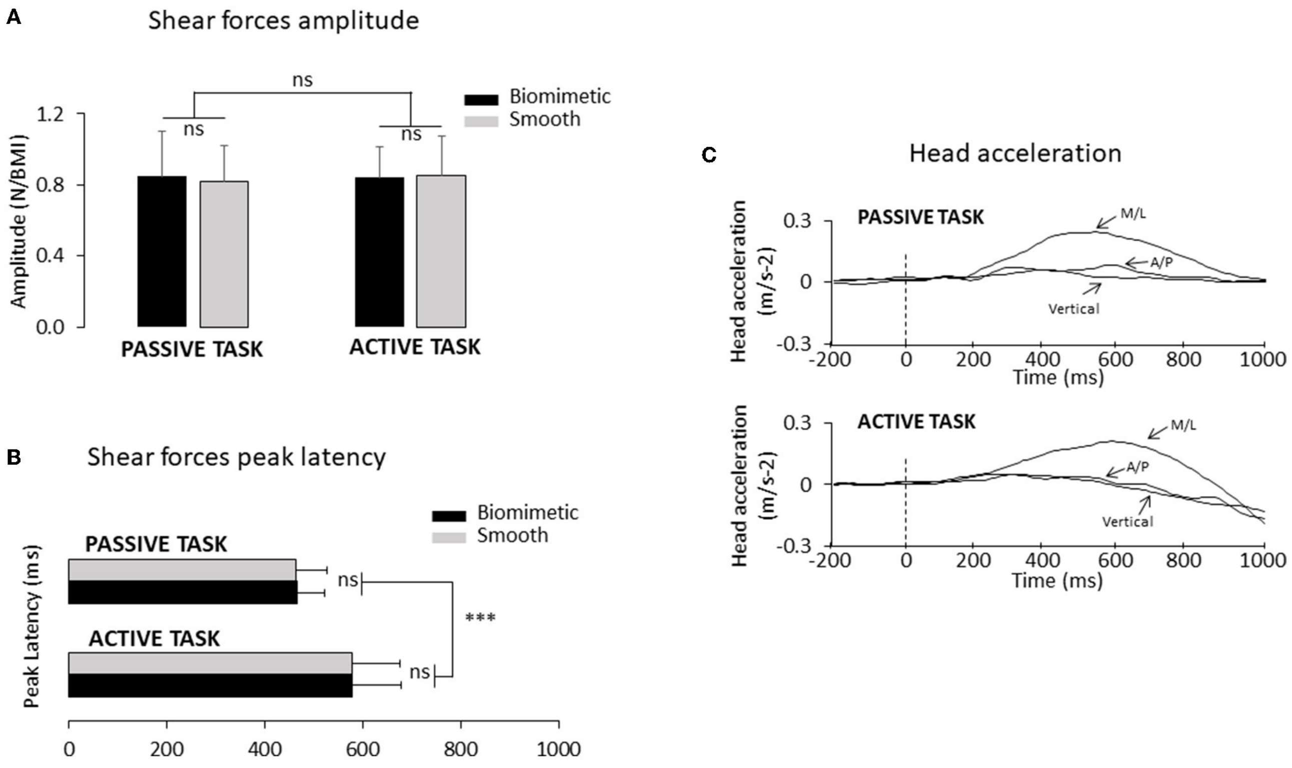

FIGURE 2
(A) Mean mediolateral forces amplitude normalized to the BMI for all participants ($n = 25$). Error bars represent standard deviation across participants; ns, not significant. **(B)** Mean time to peak duration for all participants (***$p < 0.001$). **(C)** Mean head acceleration computed for one representative participant using the biomimetic and the smooth surfaces, in the passive task (top panel) and in the active task (bottom panel). *Three* axes of the head acceleration: M/L corresponds to the mediolateral axis, A/P corresponds to the anteroposterior axis, and vertical corresponds to the vertical axis. The time 0 (vertical dotted line) corresponds to the onset of the shear forces change.

were not significantly affected by the tasks ($F_{1,24} = 0.74$; $p = 0.40$) although the peak force was reached later for the active task (578 ± 98 ms) than for the passive task (465 ± 60 ms) (significant task effect, $F_{1,24} = 28.77$; $p = 1.7{*}10^{-5}$, Figure 2B). Finally, neither the peak amplitudes ($F_{1,24} = 0.21$; $p = 0.65$) nor their latencies ($F_{1,24} = 1.19{*}10^{-5}$; $p = 0.99$) were significantly affected by the type of surface on which the participants were standing.

Surfaces

Participants stood on two different surfaces which were glued onto the platform: a biomimetic surface and a smooth surface. These surfaces were created with a 3D printer (Ultimaker 2+) using biopolymer thermoplastic (polylactic acid, PLA). Three characteristics were selected to build the biomimetic surface: shape, spatial period, and depth of the ridges. Circular shapes were inspired by both the shape of the tactile receptors' fields that demonstrate a preferential skin strain axis and the orientation of this axis, which was not the same for all units (36, 37) and the circular forms of the dermatoglyphics (38). For instance, Scheibert et al. (39) showed that when scanning a surface with a fake finger, friction-induced vibrations (FIVs), whose characteristics depend not only on the surface texture but also on the fingerprints (40–42), were amplified when the fake finger had the main geometrical characteristics of human fingerprints compared to when it had a smooth surface (i.e., no ridges). The power spectrum issued from the finger exploration showed an amplification of the signal around the frequency pertaining to the optimal sensitivity range of the Pacinian receptors [i.e., ranging between 100 and 300 Hz (43)].

The spatial period of the biomimetic surface corresponded to the distance between the center of adjacent receptive fields of the mechanoreceptors (43). Finally, the depth of the valley between the ridges was computed from what we know based on finger surface exploration (44, 45) and balance maintenance literature (22). A smooth surface, also printed in PLA but without any designed patterns, was used as a control surface.

Behavioral recordings and analyses

The ground reaction forces and moments were recorded with an AMTI force platform (60 × 120 cm, Advanced Mechanical Technology Inc., USA) at a sampling rate of 1,024 Hz and low pass filter (Butterworth 4th-order, 10 Hz cutoff frequency). In our study, the shear forces were analyzed only along the mediolateral (ML) axis as they were in the same direction as the platform translation and body weight transfer. The shear forces were normalized to each participant's body mass index (BMI) which takes into account both their height and their weight.

Head acceleration was recorded at a frequency of 1,024 Hz using a triaxial accelerometer (4630 Model: Measurement Specialties, USA) placed on the participants' chin. We measured the delay between the shear forces change onset and lateral head acceleration to verify if vestibular stimulation occurred well after the mechanoreceptor's stimulation. The vestibular stimulation threshold was defined when the lateral head acceleration exceeded $0.048\,\mathrm{m.s^{-2}}$ [i.e., threshold for vestibular stimulation (46)]. We paid particular attention to the two other axes (i.e., vertical and anteroposterior axis) before the platform displacement to be sure that no head tilt occurred along these axes (Figure 2C).

Another accelerometer (PCB 352A24, PCB Piezotronics, Inc.) was glued with wax onto the participant's right first toenail to measure the friction-induced vibrations (FIV), i.e., the vibrations induced by the transient local phenomena (local sticking, sliding, and detachments) occurring at the skin–surface contact interface. Eight out of twenty-five participants were excluded from the accelerometric analyses due to noisy accelerometer signals. Providing information related to the frequencies and intensities of the foot vibration, this highly sensitive monoaxial accelerometer (sensitivity of 100 mV/g and measurement range of ±50 g peak) allows us to get an estimate of the stimulation of the tactile receptors of the foot sole. From the measurement of these FIVs, we computed the power spectral density (PSD). These power spectra describe the distribution of power into frequency components composing the signal and give information on the frequencies excited during the frictional contact interaction when the foot skin interacts with the surface. The sampling frequency of the acquisition system was 1,024 Hz with an average analysis time window of 183 ± 72 ms. One hundred twenty-eight samples were used for the calculation of the NFFT (i.e., a type of Fourier transform). The period considered was defined from the shear forces onset to the breakdown in the curve observed during the passive task (i.e., contact transition) as this period was considered to be at the origin of the response of the somatosensory cortex to the stimulation of the cutaneous receptors of the foot sole [see Figure 1B and (20)]. The same period was used for the active task even though no such breakdown was observed.

Data transmitted by the accelerometer fixed on the toenail could also include noise (e.g., electromagnet activation/deactivation) or contain signals that arose from vibrations that were not linked to the skin–surface interaction (friction of the platform on the ball-bearing system). To test for such possibilities, we ran a series of trials in which the participants stood on either the biomimetic or the smooth surface while the accelerometer was glued on either of the surfaces or directly on the platform. The signals were recorded when the platform was held stationary with the electromagnet and when it translated sideways. To translate the platform, we either used the activation/deactivation of the magnet (as in the main experiment) or manually held the platform (rather than using the electromagnet) before releasing it. This series of tests allowed us to isolate the vibrations that are generated by the experimental setup from those generated by skin–surface interaction. These tests showed that the ball-bearing system produced very negligible vibrations and that the vibrations recorded at the level of the biomimetic and smooth surfaces were very similar. However, a clear peak emerged at ∼100 Hz when using the deactivation of the electromagnet to trigger the platform translation (Figure 5C). This peak was absent during the stationary trials and when the translations were manually triggered. We conducted a t-test on the power of the peak (which is evoked only by the deactivation of the electromagnet) against a reference value of 100 Hz. This test revealed that the emerging peak of nearly 100 Hz is too closely related to the deactivation of the electromagnet because no significant difference appeared from the reference value ($t_{13} = 1.29$; $p = 0.22$). As a result, this peak has not been treated as a characteristic vibrational signature of the smooth surface. For each surface, we calculated a mean PSD of the 15 test trials. The time window for this analysis was similar to the one mentioned above, and it is the one that corresponds to the initial skin–surface interaction (20). Because the low frequencies, under 20 Hz, are mainly due to the macroscopic vibrations (overall motion) of the mechanical system composed by the platform and the participant's body, and because these low frequencies are far from the sensitive range of cutaneous receptors, we considered for the analyses the (20–500 Hz) bandwidth frequency vibrations propagating through tissues [see (14) for a review]. For example, Pacini endings (fast-adapting type II) are extremely sensitive to mechanical transient high-frequency vibrations (∼40–400 Hz), and Meissner endings (fast-adapting type I) are sensitive to dynamic skin deformation with a peak of response at ∼30–40 Hz.

For the FIV analyses, we normalized (with subtraction) the PSD measured in both the active and passive tasks with respect to the PSD measured in the stationary condition (i.e., no movement during 20 s). Furthermore, for the passive task, to eliminate the noise associated with the deactivation of the electromagnet, we subtracted, for each surface, the mean PSD which was calculated in the 15 test trials (see above).

Electrophysiological recordings and analyses

Electroencephalographic (EEG) activity was continuously recorded from 64 Ag/AgCl surface electrodes embedded in an elastic cap (BioSemi ActiveTwo system: BioSemi). Specific to the BioSemi system, "ground" electrodes were replaced by common mode sense (CMS) active and driven right leg (DRL) passive electrodes. The signals were pre-amplified at the electrode sites, post-amplified with DC amplifiers, and digitized at a sampling rate of 1,024 Hz (ActiView acquisition program). The signals of each electrode were referenced to the mean signal of all electrodes. Four Ag/AgCl electrodes placed near the outer canthus of each eye and under/over the left eye orbit allowed us to control blinks and horizontal and vertical eye movements. We primarily based our analyses on the P_1N_1 wave extracted from the SEP evoked by the tactile stimulation induced by the weight transfer (active task) or the platform translation (passive task). Consistent with studies on cortical potentials evoked by lower limb stimulation (23, 47), the SEPs were found to be maximal over the vertex (Cz electrode). The cortical SEP (P_1N_1) was obtained by averaging, for each participant, all synchronized epochs relative to the onset of the mechanical stimulus (i.e., shear force). For both the active and passive tasks, the stimulus onset was identified at the onset of the increase of the lateral shear forces (see Figure 1B). The average amplitude computed 50 ms prior to this onset served as the baseline. The amplitude of P_1N_1 was measured from peak to peak, and its latency was assessed by measuring the P_1 latency.

The cortical sources were reconstructed using Brainstorm software [(48), freely available at http://neuroimage.usc.edu/brainstorm]. We employed the minimum-norm technique to resolve the inverse problem with unconstrained dipole orientations. The forward models were computed using a boundary element method [BEM, (49)] on the anatomical MRI Colin 27 high-resolution brain template (306,716 vertices) provided by the Montreal Neurological Institute (MNI). We opted for a model with three realistic layers (scalp, inner skull, and outer skull) which yields more accurate solutions compared to a simple three concentric sphere model (50). We used the trials of a stationary task in which the participants stood still as a baseline to compute the co-variance matrices.

Single-trial EEG data were transformed in the time–frequency domain using Morlet wavelet transforms. We used a 1 Hz central frequency [full width at half maximum (FWHM) $t_c = 3$ s], which offers a good compromise between temporal and spectral resolutions (51). The analyses of the time–frequency distribution were performed in the source space. For both types of surface, we computed the mean amplitude envelope (i.e., power) of gamma (mean 30–50 Hz, step: 1 Hz) bandwidth computed between -1 and 0 s [i.e., the planning phase of the body shift (52)]. The power was normalized with respect to the motionless baseline period (-4 to -2 s) and then averaged for each task. To control whether a change in gamma power observed between the biomimetic and the smooth surface conditions was related to the planning of the body weight transfer movement, the same analyses were performed in the passive task.

Statistics

The behavioral and EEG data were submitted to separate analyses of variance (ANOVA). A 2 × 2 ANOVA was used for mean comparisons of shear forces and SEP with the support surface (biomimetic, smooth) and task (active or passive) as intra-participant factors. Significant effects (statistical threshold of $p < 0.05$) were further analyzed using Tukey *post-hoc* tests. A paired *t*-test was used when necessary. We assessed the effect of the type of surface on the topography of the normalized gamma-band power (30–50 Hz) computed during the preparation phase of the body weight transfer [i.e., by contrasting the source of gamma-band power estimated in the biomimetic and smooth surface conditions (significance threshold $p < 0.05$, Bonferroni correction test for multiple corrections)].

Results

Effect of the surface on the preparation phase of the body weight transfer

Since the body weight transfer is prepared well in advance of its execution (52), we analyzed the cortical processing of sensory information over the 1,000-ms period preceding shear force production (Figure 3). The statistical cortical maps (Figure 3A) revealed significantly greater power (warm color) in gamma-band oscillations (30–50 Hz) in the primary somatosensory cortex (SI) and superior parietal lobule (SPL) when standing on the biomimetic than on the smooth surface. Greater activity of the extrastriate body area (EBA) in the occipital cortex (BA19, cold color) was observed when participants stood on the smooth surface. For comparison, the topography of gamma power was compared between the biomimetic and the smooth surface conditions in the passive task, which did not involve preparation for a body weight shift. For these analyses, we used the same time window as that used in the active task with respect to the onset of the shear forces, which were passively generated by the platform's translation in the passive task. The statistical source map did not reveal any significant difference between the gamma power computed in the biomimetic and smooth surfaces (Figure 3B).

Somatosensory potentials evoked by the feet/surface interaction

To assess whether the surface and/or the task altered the response of the somatosensory areas to plantar sole cutaneous stimulation, we compared the amplitude of the P1N1 between surfaces (i.e., biomimetic and smooth) and tasks (i.e., active and passive, Figure 4A). The ANOVA showed significant surface ($F_{1,24} = 67.47$, $p = 1.98{*}10^{-8}$) and task ($F_{1,24} = 15.78$, $p = 5.7{*}10^{-4}$) effects with no significant interaction between the surface and the task ($F_{1,24} = 1.83$, $p = 0.19$). The amplitude of the P_1N_1 was greater when standing on a biomimetic than on a smooth surface and greater in the passive than in the active tasks (Figure 4B). However, contrary to our predictions (i.e., less movement-related sensory gating for the biomimetic surface in the active task), the percentage of decrease of the SEP amplitude (i.e., gating) between the passive and the active tasks did not differ between the surfaces ($t_{24} = -1.76$; $p = 0.09$). On average, the overall movement-related sensory suppression was 61.4 ± 18%. The surface ($F_{1,24} = 19.86$, $p = 1.65{*}10^{-4}$) and the task ($F_{1,24} = 24.16$, $p = 5.2{*}10^{-5}$) had significant effects on P1 latency, but the ANOVA did not reveal significant task × surface interaction ($F_{1,24} = 0.002$, $p = 0.96$). The P1 latency was shorter when participants were standing on a biomimetic (125 ± 21 ms and 150 ± 18 ms for active and passive tasks, respectively) than when they were standing on a smooth surface (respectively, 135 ± 26 ms; 160 ± 18 ms for active and passive tasks) (see Figure 4C).

We compared P1 latency and the time at which head acceleration reached the vestibular threshold during the translation to determine whether the vestibular inputs evoked by head acceleration could have been at the origin of early changes in brain activity (i.e., P1). Paired *t*-tests showed that P_1 latencies significantly preceded vestibular stimulation onset for both surfaces in the active and passive tasks (see Table 1). This indicates that the SEP was more likely evoked by tactile inputs originating from the skin–surface interaction than by vestibular inputs. Note that the participants complied with the instruction to remain still before the task-induced changes in the shear forces as attested by head accelerometric analyses (see Figure 2C).

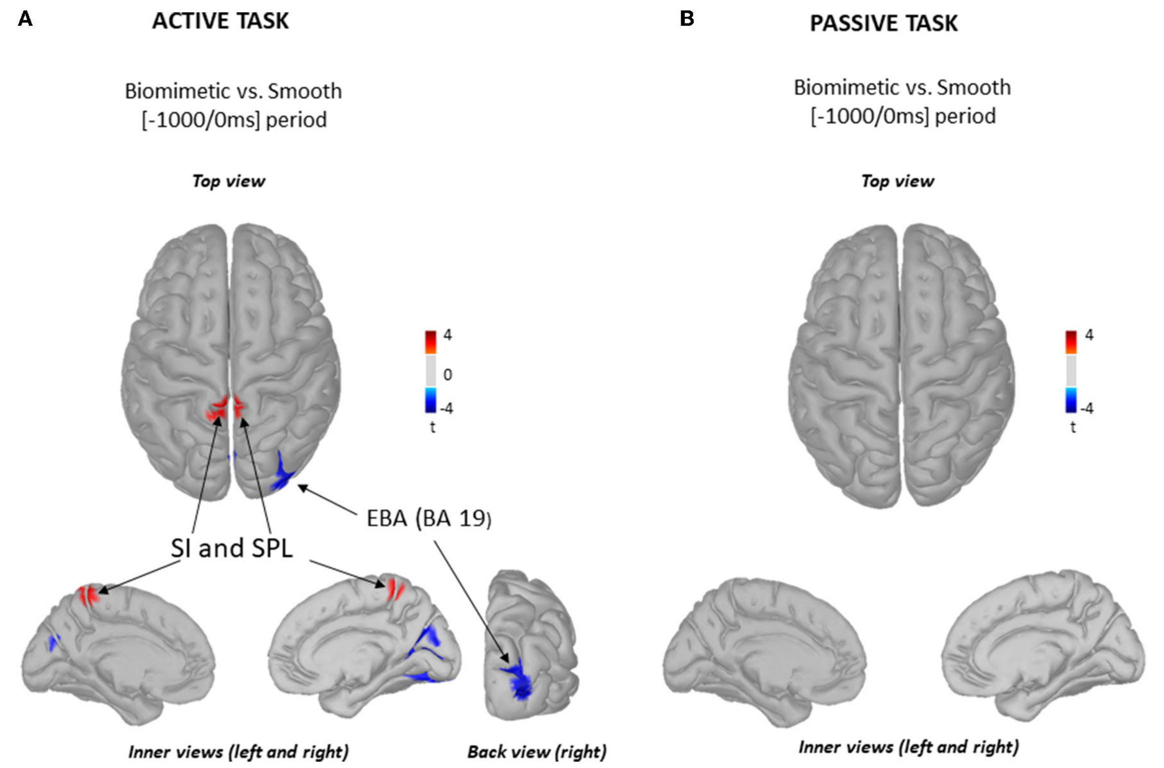

FIGURE 3
Source localization of gamma (30–50 Hz) frequency band oscillations. Statistical source localization maps for biomimetic vs. smooth contrast in the active task **(A)** and in the passive task **(B)**. Significant *t*-values ($p < 0.05$, $n = 25$) of the source localization were shown for the primary somatosensory areas (SI), superior parietal lobule (SPL), and extrastriate body area (EBA) during the time window from −1,000 ms to the onset of the mediolateral shear forces. Sources are projected on a cortical template (MNI's Colin 27). For each contrast, we display the top, left, and right inner cortical views. The back view of the right hemisphere for biomimetic vs. smooth contrast is displayed solely for the active task **(A)**.

In addition to the movement-related sensory gating, the smaller SEP amplitude observed for the active task relative to the passive task could partly stem from the weaker feet-surface interaction. The impulse (i.e., the integral of the shear force over a time interval) was analyzed over a short 100-ms period (starting from the onset of the lateral shear forces change) prior to P1 SEP for all participants, surfaces, and tasks. The results showed that it was significantly more vigorous for the passive task ($F_{1,24} = 244$; $p = 4.43{*}10^{-14}$) than for the active task, but the ANOVA did not reveal a significant surface effect ($F_{1,24} = 1.47$; $p = 0.23$). The ANOVA revealed that the interaction task x surface just reached the conventional level of significance ($F_{1,24} = 4.57$; $p = 0.04$). However, Tukey's pairwise comparisons failed to explain this marginal global reciprocal influence of task and surface factors [see (53) for a discussion on this issue].

The vibrations arising from the feet–surface interaction were also investigated to get an estimate of the cutaneous stimulation during the active and passive tasks, in both the biomimetic and smooth surface conditions. In the active task, no characteristic vibration signature emerged, the mean PSD being close to 0 in all frequencies for both tested surfaces (Figure 5A). In the passive task, however, clear and pronounced PSD peaks were observed at ∼200 and ∼300 Hz for most participants with the biomimetic surface (see Figure 5B). Several participants also showed PSD peaks at these frequencies with the smooth surface, but with markedly less power (see Figure 5C). These results suggest greater stimulation of vibratory-sensitive mechanoreceptors in the passive than in the active task. To determine whether the PSD significantly differed, in the passive task, between the biomimetic and smooth surfaces, we submitted to separate *t*-tests the maximal PSD values computed for each participant and surface, between 190 and 230 Hz (referred as 200 Hz frequency) and between 290 and 330 Hz (referred as 300 Hz frequency). The results from these *t*-tests showed that the PSD was significantly greater for the biomimetic than for the smooth surface conditions at both 200 Hz ($t_{16} = 3.06$; $p = 0.007$) and 300 Hz ($t_{16} = 3.46$; $p = 0.003$).

Discussion

The aim of this study was to determine whether the well-known movement-related sensory suppression during self-generated motor actions is partly alleviated when foot cutaneous stimulation is shaped to be relevant for balance control [e.g., FIV that enhances subcutaneous stress vibrations in a way that facilitates their processing by the Pacinian corpuscles channel (54)]. Designed with a texture inspired by the characteristics of human mechanoreceptors and dermatoglyphics (i.e., toeprints), the biomimetic surface used in the present study has been found to enhance the cortical processing of foot cutaneous information and to decrease standing task difficulty as compared to a smooth surface

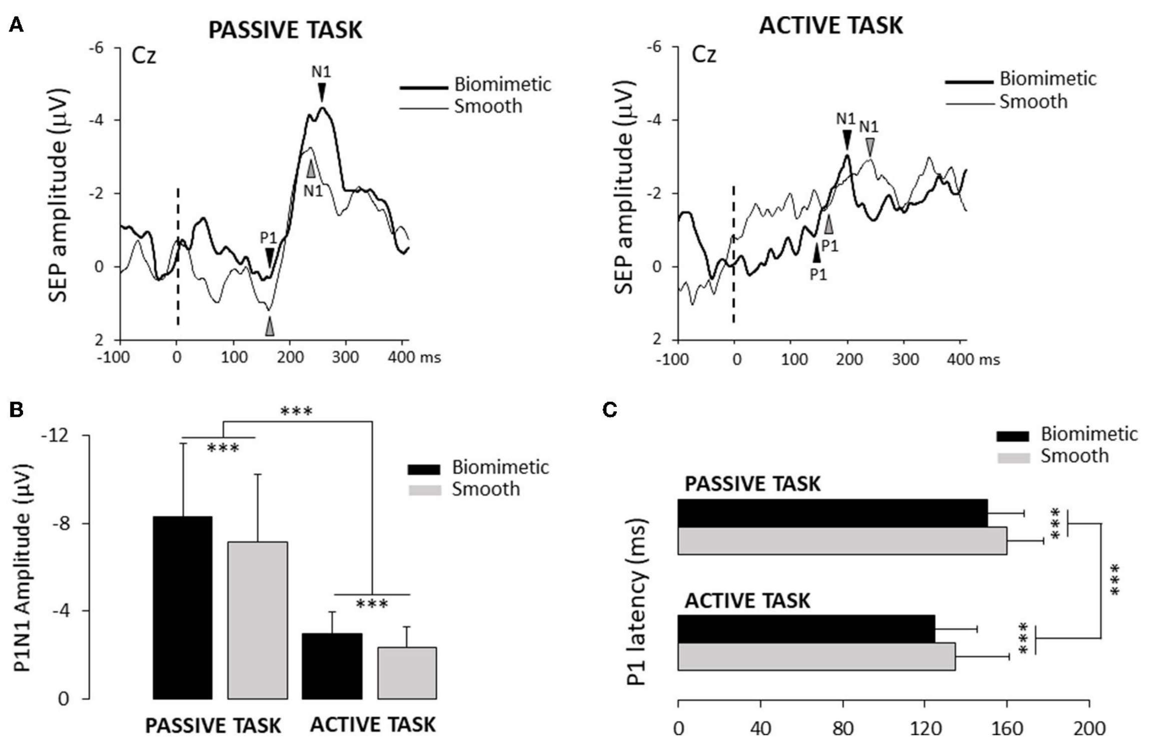

FIGURE 4
(A) Average of one participant of the SEP recorded over the Cz electrode for the two surfaces (biomimetic and smooth) and two tasks (passive and active). The broken line indicated the onset of the leftwards shear force. **(B)** Mean amplitude of the average P1N1 SEP on the two surfaces (biomimetic and smooth) and two tasks (passive and active). Error bars represent standard deviation across participants, ***$p < 0.001$. **(C)** Mean P1 latency for all participants. Error bars represent standard deviation across participants, ***$p < 0.001$.

TABLE 1 Mean latencies of all participants ($n = 25$) and standard deviation across participants (SD) for P_1, N_1 and the time when the head reached the vestibular threshold as a function of the tasks and surfaces on which participants were standing.

Biomimetic				Smooth			
Vestibular threshold							
Passive task		Active task		Passive task		Active task	
248 ms (±34)		167 ms (±59)		233 ms (±23)		163 ms (±58)	
P1	**N1**	**P1**	**N1**	**P1**	**N1**	**P1**	**N1**
150 ± 18 ms	211 ± 24 ms	125 ± 21 ms	168 ± 33 ms	160 ± 18 ms	215 ± 22 ms	135 ± 26 ms	167 ± 33 ms
T-test							
$t = 17.75$; $p < 0.001$	$t = 5.14$; $p < 0.001$	$t = 3.37$; $p = 0.003$	$t = -0.09$; $p = 0.92$	$t = 15.45$; $p < 0.001$	$t = 4.94$; $p < 0.001$	$t = 2.07$; $p = 0.04$	$t = -0.28$; $p = 0.78$

The paired *t*-test corresponds to the comparison between P_1 or N_1 and the vestibular threshold.

(20). In Sutter et al.'s study (20), foot receptors were stimulated by translating the supporting surface on which the participants were standing motionless. The same task was used here as a baseline for controlling for the movement-related sensory suppression when the cutaneous stimulation was evoked by a voluntary body weight shift (i.e., self-generated motor action) while the participants were standing on either a smooth or a biomimetic surface.

Contrary to our predictions, when executing a body weight transfer on a biomimetic surface, the well-known movement-related gating of somatosensory information was not alleviated as shown in other studies when information is relevant to the behavior (12, 55–58). For both surfaces, the sensory suppression led to a 60% reduction when compared to the SEP amplitude measured in the passive task. This sensory gating is then comparable to the sensory gating observed during electric stimulation of the tibial nerve during walking [as compared to standing, e.g., 64–69% in (3)].

Although the amount of sensory suppression was similar between the surface textures when participants performed a voluntary weight shift, the SEP amplitude remained greater when standing on the biomimetic surface in both active and passive tasks. The greater amplitude of the SEP suggests augmented stimulus intensities for activating somatosensory cortical responses. Unexpectedly, the biomimetic surface resulted in greater FIV only in the passive task wherein two pronounced

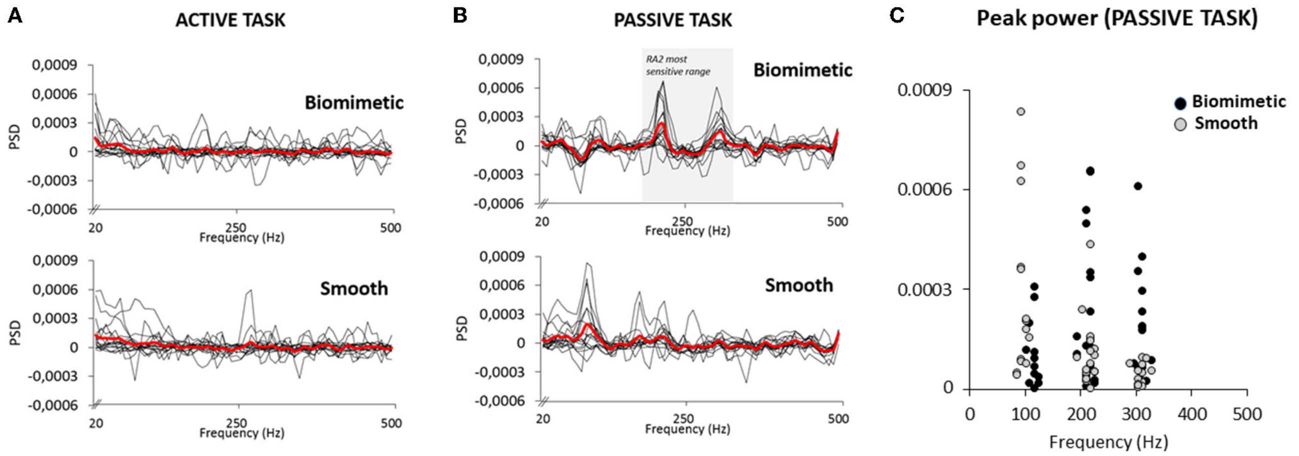

FIGURE 5
Power spectral density (PSD) of the accelerometer signal for 17 participants in active task **(A)** and the passive task **(B)**. The thick red lines represent the averaged PSD of all participants. The shaded area (**B** top panel) corresponds to the RA 2 (i.e., Pacinian corpuscles) most sensitive range. **(C)** Peak PSD for all participants exhibited at their own frequency in the passive task. Note that the peak observed around 100 Hz was likely due to the deactivation of the electromagnet to trigger the platform translation.

PSD peaks were observed at ∼200 and ∼300 Hz. These frequency peaks have been observed by Scheibert et al. (39) when sliding a fake finger with ridges on a surface, but not when sliding a smooth fake finger. Based on the microneurography literature, these frequencies targeted the preferred frequencies of Pacini skin mechanoreceptors responses. Indeed, the Pacini corpuscles (RA2) respond to mechanical transitions and vibrations with the highest sensitivity around 300 Hz (59) and respond to very small skin motion (≈10 nanometers) at 200 Hz (60).

While the greater SEP observed in the active task when standing on a biomimetic surface could not be explained by an increase in skin vibrations, the augmented contribution of other peripheral stimuli with this surface cannot be dismissed [e.g., lateral skin stretch stimulating slow-adapting type II, (41)]. Indeed, the biomimetic surface with circular patterns, similar to finger- or toeprints, could enhance the encoding of variations in the skin strain distributions, thanks to the multidirectional deformation of the skin in contact with the ridges of the biomimetic surface. This has been observed by Prevost et al. (54) when the fingerprints are oriented perpendicular to the scanning direction. A spatially variable distribution of the stress–strain fields in the skin, following the surface ridges along the overall spatial directions, could also increase the detectability of its variation and provide additional information on the directionality of the causes (e.g., direction of platform/body motion). In the passive task, the shear stresses between skin and surface were directly generated by the tangential rigid motion of the platform, inducing local sliding (i.e., relative motion) between skin and surface asperities, which are at the origin of FIV. Inversely, in the active task, the load was applied by the weight redistribution operated by the participant, without a direct tangential relative displacement between the foot sole and the platform (i.e., lower impulse for the shear force), explaining the FIV's low-frequency content.

The fact that the sensory gating was not alleviated in the active task may also suggest that the biomimetic surface enhanced the sensory processing at an earlier stage, i.e., during the preparation period of the body weight shift. Indeed, previous studies have shown that somatosensory information from the lower limb is crucial for shaping, before their execution, the postural adjustments (i.e., shear forces) responsible for initiating the body weight shift (22, 24, 25). This sensory information is thought to have less importance after the postural adjustment onset (24). In line with this interpretation, we observed increased gamma rhythms in somatosensory and SPL areas during the preparation period of the body shift when the participants stood on the biomimetic surface. An increase in gamma power reflects cortical activation (61, 62). Our spectral analyses of the EEG activity, therefore, corroborate the results of previous studies supporting the idea that gamma oscillations correlate better with movement preparation than with movement execution (61, 63–65). This was also confirmed in the present study by the absence of surface-modulation of gamma activity in the passive task, wherein no movement was prepared. It should be noted that the translation of the supporting surface evoked a postural reaction (on average 126 ± 15 ms after the platform started to move) that helped the participants keeping their center of mass within their base of support (66, 67). The fact that we did not observe increased gamma power when the participants stood on the biomimetic surface in the passive task despite the presence of self-generated movements (as in the active task), albeit without a preparation period, rules out the possibility that the gamma modulation observed in the biomimetic/active condition was linked to movement execution *per se*.

A striking result of the present study is that the amplitude of the gamma activity was spatially modulated as a function of the supporting surface during the preparation period. A topographical gamma expression over centroparietal regions (SI and SPL) was observed when standing on a biomimetic surface, whereas it was observed over occipitotemporal areas (EBA) with the smooth surface. Overall, these results suggest that gamma-band oscillations play an important functional role in differentially processing tactile stimuli from the foot soles. The increased power of gamma oscillations in SI and SPL for the biomimetic surface is consistent with the hypothesis that gamma may modulate the gain of incoming tactile information in preparation for the upcoming execution of the body weight transfer. Supporting this interpretation, Palmer et al. (33) showed a high correlation between an increase gamma ERS during the preparation period and increased accuracy of upcoming tactile perception during a force-matching paradigm. Interestingly, this interpretation is also supported by studies showing that gamma-band synchronization can promote the transfer of relevant information between different regions of the cortex (68, 69). The increased gamma activity observed over S1 and SPL cortical areas accounts for this possibility as both cortical areas receive tactile afferents. Indeed, the somatosensory nature of these two areas has been suggested by neuroanatomical studies (70–72). Interestingly, the different nuclei of the "somatosensory thalamus", conveying tactile afferents from the periphery to cortical areas, project in different combinations and with different densities directly to the postcentral somatosensory areas 3b, 1, or 5 (part of the SPL) which respond to cutaneous stimuli (72). Area 5, which is traditionally considered to be a high-order area positioned after the information process in areas 3b and 1, was identified by Impieri et al. (71) to have direct projections from nuclei composing the "sensory thalamus" and to receive strong afferences from the distal part of the limb (in particular the legs). Based on electrophysiological and neuroanatomical studies, gamma activity in both S1 and SPL could constitute the neural underpinning of the facilitation process of relevant tactile information on foot skin–surface interaction during the preparation period of motor execution. By contrast, when the relevance of tactile cues decreases as when participants stood on the smooth surface, the EBA was engaged, likely to estimate the current state of the body in space, a necessary requirement for specifying a motor plan (73). The localization of gamma activity over the EBA points to a visual representation of body and limb position in space (74, 75) probably used when there is low confidence in relying on sensory cues (here, irrelevant cutaneous inputs).

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving human participants were reviewed and approved by Research Ethics Committee CERSTAPS (IRB00012476-2021-09-12-140). The patients/participants provided their written informed consent to participate in this study.

Author contributions

CS conceived the idea, designed the method, performed experiments, analyzed the data, and wrote the manuscript. AM conceived the idea, designed the method, and contributed to data acquisition. LF, FM, and JB contributed to the analysis, interpretation of data, and the writing and critical revision of the study. LM conceived the idea and contributed to data acquisition, the analysis, interpretation of data, and the writing and critical revision of the study. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

References

1. Chapin JK, Woodward DJ. Somatic sensory transmission to the cortex during movement: gating of single cell responses to touch. *Exp Neurol.* (1982) 78:654–69. doi: 10.1016/0014-4886(82)90082-6

2. Bolton DAE, McIlroy WE, Richard Staines W. The impact of light fingertip touch on haptic cortical processing during a standing balance task. *Exp Brain Res.* (2011) 212:279–91. doi: 10.1007/s00221-011-2728-6

3. Duysens J, Tax AAM, Nawijn S, Berger W, Prokop T, Altenmüller E. Gating of sensation and evoked potentials following foot stimulation during human gait. *Exp Brain Res.* (1995) 105:423–31. doi: 10.1007/BF00233042

4. Fabre M, Antoine M, Robitaille MG, Ribot-Ciscar E, Ackerley R, Aimonetti J-M, et al. Large postural sways prevent foot tactile information from fading: neurophysiological evidence. *Cereb Cortex Commun.* (2021) 2:1–10. doi: 10.1093/texcom/tgaa094

5. Mouchnino L, Fontan A, Tandonnet C, Perrier J, Saradjian AH, Blouin J, et al. Facilitation of cutaneous inputs during the planning phase of gait initiation. *J Neurophysiol.* (2015) 114:301–8. doi: 10.1152/jn.00668.2014

6. Chapman CE, Bushnell MC, Miron D, Duncan GH, Lund JP. Sensory perception during movement in man. *Exp Brain Res.* (1987) 68:516–24. doi: 10.1007/BF00249795

7. Seki K, Perlmutter SI, Fetz EE. Sensory input to primate spinal cord is presynaptically inhibited during voluntary movement. *Nat Neurosci.* (2003) 6:1309–16. doi: 10.1038/nn1154

8. Haggard P, Whitford B. Supplementary motor area provides an efferent signal for sensory suppression. *Cogn Brain Res.* (2004) 19:52–8. doi: 10.1016/j.cogbrainres.2003.10.018

9. Wolpert DM, Ghahramani Z, Jordan MI. An internal model for sensorimotor integration. *Science.* (1995) 269:1880–2. doi: 10.1126/science.7569931

10. Miall RC, Wolpert DM. Forward models for physiological motor control. *Neural Netw.* (1996) 9:1265–79. doi: 10.1016/S0893-6080(96)00035-4

11. Blouin J, Saradjian AH, Lebar N, Guillaume A, Mouchnino L. Opposed optimal strategies of weighting somatosensory inputs for planning reaching movements toward visual and proprioceptive targets. *J Neurophysiol.* (2014) 112:2290–301. doi: 10.1152/jn.00857.2013

12. Confais J, Kim G, Tomatsu S, Takei T, Seki K. Nerve-specific input modulation to spinal neurons during a motor task in the monkey. *J Neurosci.* (2017) 37:2612–26. doi: 10.1523/JNEUROSCI.2561-16.2017

13. Cybulska-Klosowicz A, Meftah E-M, Raby M, Lemieux M-L, Chapman CE. A critical speed for gating of tactile detection during voluntary movement. *Exp Brain Res.* (2011) 210:291–301. doi: 10.1007/s00221-011-2632-0

14. Johansson RS, Flanagan JR. Coding and use of tactile signals from the fingertips in object manipulation tasks. *Nat Rev Neurosci.* (2009) 10:345–59. doi: 10.1038/nrn2621

15. Lederman SJ, Klatzky RL. Haptic perception: a tutorial. *Atten Percept Psychophys.* (2009) 71:1439–59. doi: 10.3758/APP.71.7.1439

16. Desmedt JE, Robertson D. Differential enhancement of early and late components of the cerebral somatosensory evoked potentials during forced-paced cognitive tasks in man. *J Physiol.* (1977) 271:761–82. doi: 10.1113/jphysiol.1977.sp012025

17. Hämäläinen H, Kekoni J, Sams M, Reinikainen K, Näätänen R. Human somatosensory evoked potentials to mechanical pulses and vibration: contributions of SI and SII somatosensory cortices to P50 and P100 components. *Electroencephalogr Clin Neurophysiol.* (1990) 75:13–21. doi: 10.1016/0013-4694(90)90148-D

18. Lin Y-Y, Shih Y-H, Chen J-T, Hsieh J-C, Yeh T-C, Liao K-K, et al. Differential effects of stimulus intensity on peripheral and neuromagnetic cortical responses to median nerve stimulation. *Neuroimage.* (2003) 20:909–17. doi: 10.1016/S1053-8119(03)00387-2

19. Mouchnino L, Aurenty R, Massion J, Pedotti A. Coordination between equilibrium and head-trunk orientation during leg movement: a new strategy build up by training. *J Neurophysiol.* (1992) 67:1587–98. doi: 10.1152/jn.1992.67.6.1587

20. Sutter C, Fabre M, Massi F, Blouin J, Mouchnino L. When mechanical engineering inspired from physiology improves postural-related somatosensory processes. *bioRxiv* (2022). doi: 10.1101/2022.07.20.500443

21. Bernier P-M, Burle B, Vidal F, Hasbroucq T, Blouin J. Direct evidence for cortical suppression of somatosensory afferents during visuomotor adaptation. *Cerebral Cortex.* (2009) 19:2106–13. doi: 10.1093/cercor/bhn233

22. Mouchnino L, Blouin J. When standing on a moving support, cutaneous inputs provide sufficient information to plan the anticipatory postural adjustments for gait initiation. *PLoS ONE.* (2013) 8:e0055081. doi: 10.1371/journal.pone.0055081

23. Saradjian AH, Tremblay L, Perrier J, Blouin J, Mouchnino L. Cortical facilitation of proprioceptive inputs related to gravitational balance constraints during step preparation. *J Neurophysiol.* (2013) 110:397–407. doi: 10.1152/jn.00905.2012

24. Timmann D, Horak F. Perturbed step initiation in cerebellar subjects: 2. Modification of anticipatory postural adjustments. *Exp Brain Res.* (2001) 141:110–20. doi: 10.1007/s002210100858

25. Timmann D, Horak FB. Perturbed step initiation in cerebellar subjects. *Experimental Brain Research.* (1998) 119:73–84. doi: 10.1007/s002210050321

26. Morasso PG, Schieppati M. Can muscle stiffness alone stabilize upright standing? *J Neurophysiol.* (1999) 82:1622–6. doi: 10.1152/jn.1999.82.3.1622

27. Fabre M, Chavet P, Fornerone T, Juan B, Abossolo O, Pardo F, et al. Somatosensory cortical facilitation during step preparation restored by an improved body representation in obese patients. *Gait Post.* (2020) 80:246–52. doi: 10.1016/j.gaitpost.2020.06.002

28. Gunduz A, Brunner P, Sharma M, Leuthardt EC, Ritaccio AL, Pesaran B, et al. Differential roles of high gamma and local motor potentials for movement preparation and execution. *Brain Comp Interfaces.* (2016) 3:88–102. doi: 10.1080/2326263X.2016.1179087

29. Tatti E, Ferraioli F, Cacciola A, Chan C, Quartarone A, Ghilardi MF. Modulation of gamma spectral amplitude and connectivity during reaching predicts peak velocity and movement duration. *Front Neurosci.* (2022) 16:836703. doi: 10.3389/fnins.2022.836703

30. Bernier P-M, Grafton ST. Human posterior parietal cortex flexibly determines reference frames for reaching based on sensory context. *Neuron.* (2010) 68:776–88. doi: 10.1016/j.neuron.2010.11.002

31. Mountcastle VB, Lynch JC, Georgopoulos A, Sakata H, Acuna C. Posterior parietal association cortex of the monkey: command functions for operations within extrapersonal space. *J Neurophysiol.* (1975) 38:871–908. doi: 10.1152/jn.1975.38.4.871

32. Wolpert DM, Goodbody SJ, Husain M. Maintaining internal representations: the role of the human superior parietal lobe. *Nat Neurosci.* (1998) 1:529–33. doi: 10.1038/2245

33. Palmer CE, Davare M, Kilner JM. Physiological and perceptual sensory attenuation have different underlying neurophysiological correlates. *J Neurosci.* (2016) 36:10803–12. doi: 10.1523/JNEUROSCI.1694-16.2016

34. Saradjian AH, Teasdale N, Blouin J, Mouchnino L. Independent early and late sensory processes for proprioceptive integration when planning a step. *Cerebral Cortex.* (2019) 29:2353–65. doi: 10.1093/cercor/bhy104

35. Akaiwa M, Matsuda Y, Saito H, Shibata E, Sasaki T, Sugawara K. Effects of repetitive practice of motor tasks on somatosensory gating. *Front Hum Neurosci.* (2023) 17:1131986. doi: 10.3389/fnhum.2023.1131986

36. Kennedy PM, Inglis JT. Distribution and behaviour of glabrous cutaneous receptors in the human foot sole. *J Physiol.* (2002) 538:995–1002. doi: 10.1113/jphysiol.2001.013087

37. Valbo AB, Olausson H, Wessberg J, Kakuda N. Receptive field characteristics of tactile units with myelinated afferents in hairy skin of human subjects. *J Physiol.* (1995) 483:783–95. doi: 10.1113/jphysiol.1995.sp020622

38. Cummins H, Midlo C. Palmar and plantar epidermal ridge configurations (dermatoglyphics) in European-Americans. *Am J Phys Anthropol.* (1926) 9:471–502. doi: 10.1002/ajpa.1330090422

39. Scheibert J, Leurent S, Prevost A, Debregeas G. The role of fingerprints in the coding of tactile information probed with a biomimetic sensor. *Science.* (2009) 323:1503–6. doi: 10.1126/science.1166467

40. Dacleu Ndengue J, Cesini I, Faucheu J, Chatelet E, Zahouani H, Delafosse D, et al. Tactile perception and friction-induced vibrations: discrimination of similarly patterned wood-like surfaces. *IEEE Trans Haptics.* (2017) 10:409–17. doi: 10.1109/TOH.2016.2643662

41. Fagiani R, Massi F, Chatelet E, Berthier Y, Sestieri A. Experimental analysis of friction-induced vibrations at the finger contact surface. *Proc Inst Mech Eng Part J.* (2010) 224:1027–35. doi: 10.1243/13506501JET722

42. Felicetti L, Chatelet E, Latour A, Cornuault P-H, Massi F. Tactile rendering of textures by an electro-active polymer piezoelectric device: mimicking friction-induced vibrations. *Biotribology.* (2022) 31:100211. doi: 10.1016/j.biotri.2022.100211

43. Johansson RS, Vallbo ÅB. Tactile sensory coding in the glabrous skin of the human hand. *Trends Neurosci.* (1983) 6:27–32. doi: 10.1016/0166-2236(83)90011-5

44. Camillieri B, Bueno M-A, Juan B, Lemaire-Semail B, Mouchnino L, Fabre M. From finger friction and induced vibrations to brain activation: Tactile comparison between real and virtual textile fabrics. *Tribol Int.* (2018) 126:283–96. doi: 10.1016/j.triboint.2018.05.031

45. Peyre K, Tourlonias M, Bueno M-A, Rossi R. Conception d'un doigt artificiel à fibre optique polymérique pour application du toucher de surface textile. *23eme Congres Francais de Mecanique* (2017).

46. Gianna C, Heimbrand S, Gresty M. Thresholds for detection of motion direction during passive lateral whole-body acceleration in normal subjects and patients with bilateral loss of labyrinthine function. *Brain Res Bull.* (1996) 40:443–7. doi: 10.1016/0361-9230(96)00140-2

47. Altenmüller E, Berger W, Prokop T, Trippel M, Dietz V. Modulation of sural nerve somatosensory evoked potentials during stance and different phases of the step-cycle. *Electroencephalogr Clin Neurophysiol.* (1995) 96:516–25. doi: 10.1016/0013-4694(95)00093-E

48. Tadel F, Baillet S, Mosher JC, Pantazis D, Leahy RM. Brainstorm: a user-friendly application for MEG/EEG Analysis. *Comput Intell Neurosci.* (2011) 2011:1–13. doi: 10.1155/2011/879716

49. Gramfort A, Papadopoulo T, Olivi E, Clerc M. OpenMEEG: opensource software for quasistatic bioelectromagnetics. *BioMed Eng OnLine.* (2010) 9:45. doi: 10.1186/1475-925X-9-45

50. Sohrabpour A, Yunfeng L, Bin H. Estimating underlying neuronal activity from EEG using an iterative sparse technique. *Annu Int Conf IEEE Eng Med Biol Soc* (2015) 634–637. doi: 10.1109/EMBC.2015.7318442

51. Allen DP, MacKinnon CD. Time-frequency analysis of movement-related spectral power in EEG during repetitive movements: a comparison of methods. *J Neurosci Methods.* (2010) 186:107–15. doi: 10.1016/j.jneumeth.2009.10.022

52. MacKinnon CD, Bissig D, Chiusano J, Miller E, Rudnick L, Jager C, et al. Preparation of anticipatory postural adjustments prior to stepping. *J Neurophysiol.* (2007) 97:4368–79. doi: 10.1152/jn.01136.2006

53. Garofalo S, Giovagnoli S, Orsoni M, Starita F, Benassi M. Interaction effect: are you doing the right thing? *PLoS ONE.* (2022) 17:1–19. doi: 10.1371/journal.pone.0271668

54. Prevost A, Scheibert J, Debrégeas G. Effect of fingerprints orientation on skin vibrations during tactile exploration of textured surfaces. *Commun Integr Biol.* (2009) 2:422–4. doi: 10.4161/cib.2.5.9052

55. Fink AJP, Croce KR, Huang ZJ, Abbott LF, Jessell TM, Azim E. Presynaptic inhibition of spinal sensory feedback ensures smooth movement. *Nature.* (2014) 509:43–8. doi: 10.1038/nature13276

56. Gilbert CD, Sigman M. Brain states: top-down influences in sensory processing. *Neuron.* (2007) 54:677–96. doi: 10.1016/j.neuron.2007.05.019

57. Lee S, Carvell GE, Simons DJ. Motor modulation of afferent somatosensory circuits. *Nat Neurosci.* (2008) 11:1430–8. doi: 10.1038/nn.2227

58. Staines WR, Brooke JD, McIlroy WE. Task-relevant selective modulation of somatosensory afferent paths from the lower limb. *NeuroReport.* (2000) 11:1713–9. doi: 10.1097/00001756-200006050-00024

59. Bolanowski SJ, Gescheider GA, Verrillo RT, Checkosky CM. Four channels mediate the mechanical aspects of touch. *J Acoust Soc Am.* (1988) 84:1680–94. doi: 10.1121/1.397184

60. Brisben AJ, Hsiao SS, Johnson KO. Detection of vibration transmitted through an object grasped in the hand. *J Neurophysiol.* (1999) 81:1548–58. doi: 10.1152/jn.1999.81.4.1548

61. Pfurtscheller G, Neuper C, Kalcher J. 40-Hz oscillations during motor behavior in man. *Neurosci Lett.* (1993) 164:179–82. doi: 10.1016/0304-3940(93)90886-P

62. Spydell JD, Ford MR, Sheer DE. Task dependent cerebral lateralization of the 40 hertz EEG rhythm. *Psychophysiology.* (1979) 16:347–50. doi: 10.1111/j.1469-8986.1979.tb01474.x

63. Donoghue JP, Sanes JN, Hatsopoulos NG, Gaál G. Neural discharge and local field potential oscillations in primate motor cortex during voluntary movements. *J Neurophysiol.* (1998) 79:159–73. doi: 10.1152/jn.1998.79.1.159

64. Pfurtscheller G, Neuper C. Simultaneous EEG 10 Hz desynchronization and 40 Hz synchronization during finger movements. *Neuroreport.* (1992) 3:1057–60. doi: 10.1097/00001756-199212000-00006

65. Sanes JN, Donoghue JP. Oscillations in local field potentials of the primate motor cortex during voluntary movement. *Proc Natl Acad Sci USA.* (1993) 90:4470–4. doi: 10.1073/pnas.90.10.4470

66. Horak FB, Henry SM, Shumway-Cook A. Postural perturbations: new insights for treatment of balance disorders. *Phys Ther.* (1997) 77:517–33. doi: 10.1093/ptj/77.5.517

67. Nashner LM, Cordo PJ. Relation of automatic postural responses and reaction-time voluntary movements of human leg muscles. *Exp Brain Res.* (1981) 43:395–405. doi: 10.1007/BF00238382

68. Engel AK, Senkowski D, Schneider TR. Multisensory integration through neural coherence. In: Murray MM, Wallace MT, editors. *The Neural Bases of Multisensory Processes*. Boca Raton, FL: CRC Press/Taylor & Francis (2012). Available online at: http://www.ncbi.nlm.nih.gov/books/NBK92855/ (accessed February 13, 2023).

69. Tallon-Baudry C, Bertrand O. Oscillatory gamma activity in humans and its role in object representation Catherine Tallon-Baudry and Olivier Bertrand. *Trends Cogn Sci.* (1999) 3:151–62. doi: 10.1016/S1364-6613(99)01299-1

70. Chung JM, Lee KH, Surmeier DJ, Sorkin LS, Kim J, Willis WD. Response characteristics of neurons in the ventral posterior lateral nucleus of the monkey thalamus. *J Neurophysiol.* (1986) 56:370–90. doi: 10.1152/jn.1986.56.2.370

71. Impieri D, Gamberini M, Passarelli L, Rosa MGP, Galletti C. Thalamo-cortical projections to the macaque superior parietal lobule areas PEc and PE. *J Comp Neurol.* (2018) 526:1041–56. doi: 10.1002/cne.24389

72. Padberg J, Cerkevich C, Engle J, Rajan AT, Recanzone G, Kaas J, et al. Thalamocortical connections of parietal somatosensory cortical fields in Macaque monkeys are highly divergent and convergent. *Cerebral Cortex.* (2009) 19:2038–64. doi: 10.1093/cercor/bhn229

73. Shadmehr R, Krakauer JW. A computational neuroanatomy for motor control. *Exp Brain Res.* (2008) 185:359–81. doi: 10.1007/s00221-008-1280-5

74. Astafiev SV, Stanley CM, Shulman GL, Corbetta M. Extrastriate body area in human occipital cortex responds to the performance of motor actions. *Nat Neurosci.* (2004) 7:542–8. doi: 10.1038/nn1241

75. Urgesi C, Candidi M, Ionta S, Aglioti SM. Representation of body identity and body actions in extrastriate body area and ventral premotor cortex. *Nat Neurosci.* (2007) 10:30–1. doi: 10.1038/nn1815

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Effects of jaw clenching on dynamic reactive balance task performance after 1-week of jaw clenching training

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Introduction: Good balance is essential for human daily life as it may help to improve the quality of life and reduce the risk of falls and associated injuries. The influence of jaw clenching on balance control has been shown under static and dynamic conditions. Nevertheless, it has not yet been investigated whether the effects are mainly associated with the dual-task situation or are caused by jaw clenching itself. Therefore, this study investigated the effects of jaw clenching on dynamic reactive balance task performance prior to and after 1 week of jaw clenching training. It was hypothesized that jaw clenching has stabilizing effects resulting in a better dynamic reactive balance performance, and these effects are not related to dual-task benefits.

Methods: A total of 48 physically active and healthy adults (20 women and 28 men) were distributed into three groups, one habitual control group (HAB) and two jaw clenching groups (JAW and INT) that had to clench their jaws during the balance tasks at T1 and T2. One of those two groups, the INT group, additionally practiced the jaw clenching task for 1 week, making it familiar and implicit at T2. The HAB group did not receive any instruction regarding jaw clenching condition. Dynamic reactive balance was assessed using an oscillating platform perturbed in one of four directions in a randomized order. Kinematic and electromyographic (EMG) data were collected using a 3D motion capture system and a wireless EMG system, respectively. Dynamic reactive balance was operationalized by the damping ratio. Furthermore, the range of motion of the center of mass (CoM) in perturbation direction (RoM_{CoM_AP} or RoM_{CoM_ML}), as well as the velocity of CoM (V_{CoM}) in 3D, were analyzed. The mean activity of the muscles relevant to the perturbation direction was calculated to investigate reflex activities.

Results: The results revealed that jaw clenching had no significant effects on dynamic reactive balance performance or CoM kinematics in any of these three groups, and the automation of jaw clenching in the INT group did not result in a significant change either. However, high learning effects, as revealed by the higher damping ratio values and lower V_{CoM} at T2, were detected for the dynamic reactive balance task even without any deliberate balance training in the intervention phase. In the case of backward perturbation of the platform, the soleus activity in a short latency response phase increased for the JAW group, whereas it decreased for HAB and INT after the intervention. In the case of forward acceleration of the platform, JAW and INT showed a higher tibialis anterior muscle activity level in the medium latency response phase compared to HAB at T1.

Discussion: Based on these findings, it can be suggested that jaw clenching may lead to some changes in reflex activities. However, the effects are limited to anterior–posterior perturbations of the platform. Nevertheless, high learning effects may have overall overweighed the effects related to jaw clenching. Further studies with balance tasks leading to less learning effects are needed to understand the altered adaptations to a dynamic reactive balance task related to simultaneous jaw clenching. Analysis of muscle coordination (e.g., muscle synergies), instead of individual muscles, as well as other experimental designs in which the information from other sources are reduced (e.g., closed eyes), may also help to reveal jaw clenching effects.

KEYWORDS

stomatognathic activity, postural control, temporomandibular joint, Posturomed, reflex phases, dual task

Introduction

Balance is one of the essential aspects of postural control and is crucial to accomplish daily routine activities, such as unassisted standing and walking. Impaired balance control may lead to an increased risk of falls and a reduced quality of life (1, 2). From a mechanical point of view, balance involves controlling the center of mass (CoM) with respect to the base of support (1). During standing, the CoM sways steadily within the body's base of support (i.e., static steady balance), whereas during perturbations, stability needs to be recovered to bring the CoM back to the allowed limits necessary for maintaining posture (i.e., dynamic reactive balance); (3). Given the importance of balance (1), it is valuable to improve its control mechanisms through balance training. This is recommended for performance enhancement in sports (4) to prevent injuries (5) and to decrease falls in at-risk groups (6, 7).

An important prerequisite for balance is the sensory input that derives from the somatosensory, visual, and vestibular systems and provides the central nervous system (CNS) with information regarding the state of the body and the environment. This sensory information is weighted in a task-dependent manner (8). For example, when the support surface is rapidly displaced (i.e., the dynamic reactive balance control is challenged), the CNS mostly relies on somatosensory inputs since these enable faster reactions than other systems of sensory input (1). Given the importance of somatosensory information for dynamic reactive balance control, any alteration that improves dynamic stability may be relevant for fall prevention, especially in unexpected external perturbations (2, 9).

A growing body of literature suggests that there is a close relationship between the stomatognathic system and balance (10–18). The underlying mechanisms have not yet been fully understood; however, in various studies (19–22), it was shown that jaw clenching like the Jendrassik maneuver (23) may lead to increased motor excitability and increased H-reflex responses. In addition, co-contraction behavior of the masticatory and neck muscles occurring as a result of complex neurophysiological interactions (24) may also contribute to improved postural control, for example, via a more stable head or gaze position (25–27). These results are neuroanatomically supported by findings in animal models which found neuronal links of the trigeminal nerve to numerous brainstem nuclei and all levels of the spinal cord (28).

Although jaw clenching has been shown to affect balance performance under both static (12, 29, 30) and dynamic conditions (10, 14), it is still unknown whether these effects are associated with the dual-task situation [i.e., influences of simultaneously performed additional motor tasks (31, 32)] or those specifically connected to jaw clenching. In general, when two tasks are performed simultaneously, performance decreases in one or both tasks (33), which can be explained by the limited capacity of attention (34). However, with respect to balance control, previous studies showed that combining a secondary task with a balance task may actually improve performance compared to a single-task condition (32). This phenomenon can be explained by altered attention and increased automatization of balance control processes (31, 35). Therefore, one might argue that stabilizing effects on balance control could be caused by the secondary task of jaw clenching.

To sum up, the acute positive effects of jaw clenching have been shown in various studies (10, 12, 14, 29, 30); however, it has not yet been evaluated if these effects are associated with dual-task benefits or specifically based on neurophysiological effects caused by jaw clenching. Therefore, this study established an intervention group (INT) that trained jaw clenching, so that it becomes an implicit task. The comparison with a group (JAW) that was only instructed in jaw clenching shortly before T1 and T2 and with a group without any training as well as instruction (HAB) should help to draw a firm conclusion about the abovementioned dual-task issue. It was hypothesized that jaw clenching has an effect on dynamic reactive balance, and this effect is not related to dual-task benefits, which would be indicated by the missing differences in dynamic reactive balance performance between the INT and JAW groups at T2.

Methods

The study design comprised two measurement times (T1 and T2, separated by 1 week) and three groups (INT: intervention, JAW: jaw clenching, and HAB: habitual), whose details can be found in the following sections. The data of two groups (JAW and HAB) at T1 were partially presented in previously published studies (10, 36). An *a priori* power analysis was performed based on the study by Ringhof et al. (30) who analyzed the effects of submaximal jaw clenching on postural stability. The results revealed that 16 participants per group would be sufficient to reach a power of >0.8.

Participants

A total of 48 physically active adults (20 women and 28 men; age: 23.2 ± 2.4 years; height: 1.74 ± 0.09 m; and body mass: 69.4 ± 10.4 kg) participated in this study. All participants gave written informed consent prior to the study, confirmed that they were participating in any kind of sports regularly at least three times per week, and were naive to the balance task instrument. They had no muscular or neurological diseases, showed no signs or symptoms of temporomandibular disorders [based on the Research Diagnostic Criteria for Temporomandibular Disorders (37)], and presented with full dentition (except for third molars) in neutral occlusion. The study was approved by the Ethics Committee of the Karlsruhe Institute of Technology.

Study design

To investigate whether the stabilizing effects of jaw clenching are merely a result of dual-task effects, the principal idea of our three-armed intervention study was that one of the groups, namely, INT, repeatedly practiced jaw clenching to make it a familiar and implicit task. The details of the three different groups (INT, JAW, and HAB) are shown in Figure 1.

Dynamic reactive balance performance was assessed using a commercially available oscillating platform (Posturomed, Haider-Bioswing, Weiden, Germany), which has previously been used to systematically investigate dynamic reactive balance performance after perturbations in many other studies (38–40). It is a rigid platform (12 kg, 60 × 60 cm) connected to a metal frame with eight steel springs (15 cm) of identical strength and can swing along the horizontal plane in all directions freely. A custom-made release system was used to apply mechanical perturbations in one of the four possible directions, back (*B*), front (*F*), left (*L*), and right (*R*), in a randomized order (10). Before the trials began, the participants were familiarized with the Posturomed with two trials without and two trials with perturbation. Afterward, a baseline measurement with a perturbation was conducted in the habitual stomatognathic motor condition to determine initial balance performance (10). Before each trial, participants were asked to stand on the platform on their dominant leg, hands at hips, eyes focusing on a fixed point at the eye level horizontally 4 m away from the center of the platform, and to compensate for the perturbation as quickly as possible. Their dominant leg was determined based on self-reports or, in case of uncertainty, by testing on the Posturomed (10, 41). In each trial, the platform was perturbed by the release system unpredictably in one of the four possible directions in a randomized order. The release system was used to release the platform from its maximum displaced position along the perturbation axis. After the perturbation, no external resistance forces were applied, and the participants had to dampen the perturbation by bringing the platform into its central position as soon as possible.

Both INT and JAW were jaw clenching groups and were instructed to clench their jaws during the balancing task. INT additionally trained in the jaw clenching task between T1 and T2, which were separated by 1 week. The purpose of this intervention was to make the novel jaw clenching task more automated, such that focused attention is reduced at T2. Groups were assigned considering the subjects' gender as well as their initial balance performance to ensure even distribution across the three groups. It was statistically confirmed that there were no baseline performance differences between the three groups (one-way ANOVA, $p = 0.920$).

During the balance task, INT and JAW were asked to clench their jaws with a force of 75 N. To familiarize themselves with this task, participants were trained for 5 min just before the measurements with a RehaBite® (Plastyle GmbH, Uttenreuth, Germany), a medical training device consisting of liquid-filled plastic pads, to get used to applying this level of force (10, 42). During the measurements, the EMG activity of the masseter muscle corresponding to 75 N was used as a reference, and the participants in these two groups bit down on an Aqualizer® intra-oral splint (medium volume; Dentrade International, Cologne, Germany). The HAB did not receive any instructions regarding the stomatognathic motor condition or an Aqualizer®. In the 1-week intervention phase between T1 and T2, INT trained three times a day for 10 min (10 repetitions of three sets, applying force for 10 s, stretching the jaw muscles, and resting for 10 s). For this purpose, the participants received a RehaBite® and a diary to record the training sessions.

Measurements

A total of 22 anthropometric measures were manually taken from each participant, and 42 reflective markers were placed on the participants' skin in accordance with the Advanced Lagrangian Solver in Kinetic Analysis modeling system [ALASKA, INSYS GmbH, Chemnitz, Germany (43)] to capture full body kinematics. Four reflective markers were attached to the upper surface of the Posturomed platform (10), and their displacements were captured using a 3D motion capture system (Vicon Motion Systems; Oxford Metrics Group, Oxford, UK; 10 Vantage V8 and 6 Vero V2.2 cameras; 200 Hz).

The activity of nine muscles [peroneus longus (PL), soleus (SOL), tibialis anterior (TA), rectus femoris (RF), semitendinosus (SM), rectus abdominis (AB), internal oblique (IO), erector spinae (ES), and masseter (MA)] was recorded using a wireless EMG system (Noraxon, Scottsdale, USA; 2,000 Hz) at the standing leg side. Before the measurements, the skin over the relevant muscles was shaved, abraded, and rinsed with alcohol. Bipolar Ag/AgCl surface electrodes (diameter 14 mm, center-to-center distance 20 mm; Noraxon Dual Electrodes, Noraxon, Scottsdale, USA) were attached in accordance with the European Recommendations for Surface EMG (44). Afterward, maximum voluntary contraction (MVC) tests were performed for normalization. At T1, the positions of EMG electrodes were marked with a temporary tattoo ink, so they could be placed in the same positions at T2.

A total of 12 valid trials (three per each of the four perturbation directions in a randomized order, each lasting 30 s) were recorded.

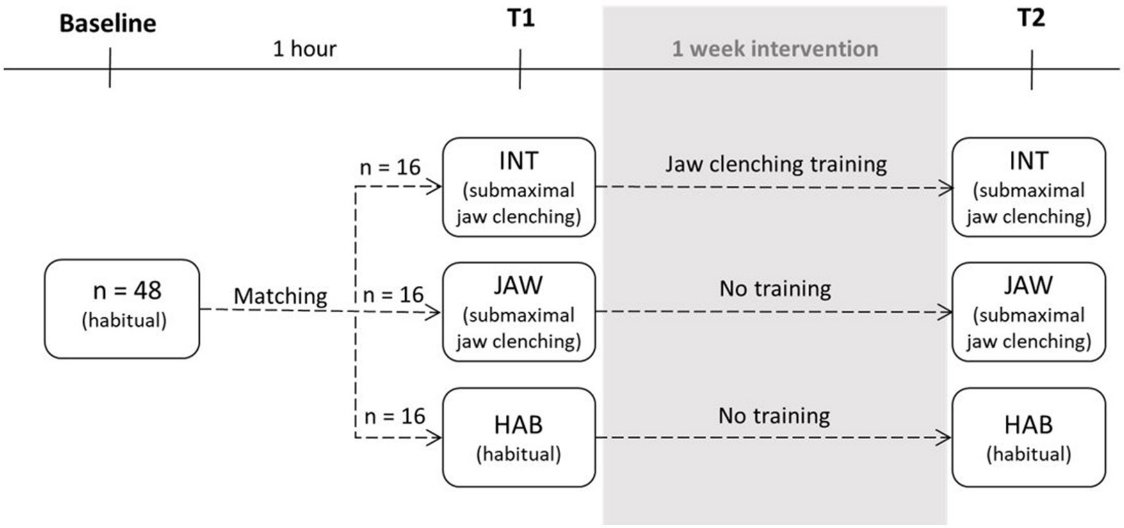

FIGURE 1
Study design (INT, intervention; JAW, jaw clenching; HAB, habitual; T1 and T2 are the measurement times).

Trials were invalid if participants did not apply enough force with their jaws (for INT and JAW), touched the ground with the non-standing foot, moved their standing foot, or released their hands from the hip. The success rate was high (i.e., only 1–2 invalid trials per participant) and did not differ between the groups. At T1 and T2, the same measurement process was followed.

Data analysis

All data were recorded in Vicon Nexus 2.10 and processed with MATLAB R2021b (MathWorks). Kinematic data were filtered by a fourth-order Butterworth low-pass filter (10 Hz) and EMG data with a fourth-order Butterworth band-pass filter (10–500 Hz). The filtered EMG data were rectified and normalized to the MVC amplitudes (29). *R* and *L* directions were re-sorted into ipsilateral (*I*) and contralateral (*C*) according to the standing leg of the participants.

To operationalize dynamic reactive balance performance, the damping ratio (10, 38) was calculated based on the movement of the Posturomed using the data of the markers attached to the platform (Equation 1, Figure 2). Larger damping ratio values represent better compensation of the perturbation and, therefore, better dynamic reactive balance and *vice versa*. With respect to the EMG data, three main latency responses were considered after the onset of perturbation: short (SLR, 30 to 60 ms), medium (MLR, 60 to 85 ms), and long (LLR, 85–120 ms) (40, 45). Two further time windows were considered: 100 ms before the onset of perturbation (PRE, −100–0 ms) and after the reflex phases until the end of the individual damping ratio (DRP, 120–1,136 ± 131 ms). Mean activities of the relevant muscles (directions *B*: PL and SOL; *F*: TA and AB; *I*: SM and IO, and *C*: RF and ES (46); MA for all directions) were calculated for the five phases, that is, PRE, SLR, MLR, LLR, and DRP.

$$Damping\ ratio_i = \frac{\wedge}{\sqrt{\wedge_i^2 + 4\pi^2}} \quad ;$$
$$\wedge_i = \frac{1}{3} \ln \frac{K_0}{K_i}\ ,\ \ K_i:\ i^{th} positive\ amplitude.\ (Equation\ 1) \qquad (1)$$

The marker trajectories in 3D were used to estimate the CoM trajectories with the full-body Dynamicus model [ALASKA, INSYS GmbH, Chemnitz, Germany (43)]. The COM displacement (47) was calculated as the range of motion of CoM along the perturbation axis (RoM_{CoM_AP} for *B* and *F*, and RoM_{CoM_ML} for *I* and *C*). Furthermore, the three-dimensional velocity of the CoM (V_{CoM}) (48) was calculated for each trial and averaged for the whole damping ratio time window (0 ms until the end of the individual damping ratio).

Statistics

Statistical calculations were done using IBM SPSS Statistics 25.0 (IBM Corporation, Armonk, NY, United States). For all dependent parameters (damping ratio, RoM_{CoM_AP}, RoM_{CoM_ML}, V_{CoM}, and mean muscle activities), the three trials within each of the four perturbation directions were averaged. The normality of the data distributions was confirmed by Kolmogorov–Smirnov tests. The statistical assumptions were met to perform the repeated measures ANOVA (rmANOVA). The four perturbation directions were analyzed separately (10) since it was suggested that the direction of surface translation influences the sensation, central processing, and output of the postural responses differently (49, 50). For each dependent parameter, direction, and phase, a rmANOVA was calculated with the factors group (INT, JAW, and HAB) and time (T1 and T2). The significance level was set *a priori* to a p-value of < 0.05. In case of significant differences, *post hoc* tests or *t*-tests were performed for pairwise comparisons. Partial eta-squared and Cohen's d were calculated to quantify the effect sizes for rmANOVA

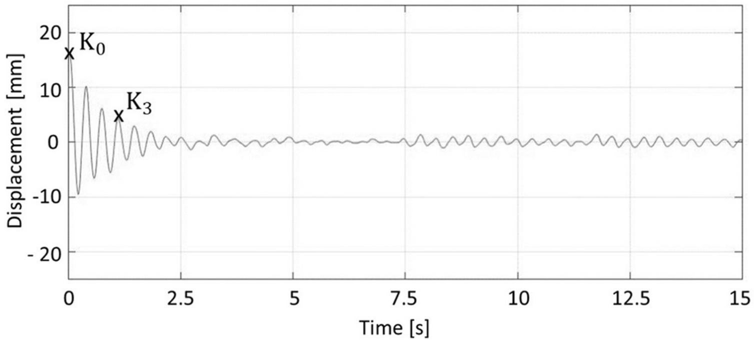

FIGURE 2
Calculation of damping ratio. The initial maximum displacement (K$_0$) and the third positive amplitude (K$_3$) were used for Eq.1.

and *post hoc* tests, respectively [small effect: $\eta_p^2 < 0.06$, $d < 0.50$; medium effect: $0.06 < \eta_p^2 < 0.14$, $0.5 < d < 0.8$; large effect: $\eta_p^2 > 0.14$, $d > 0.8$; (51)]. The Bonferroni–Holm method was applied to correct the results for multiple comparisons (52).

Results

Dynamic reactive balance performance

The results regarding the damping ratio for the four directions are illustrated in Figure 3. For the factor time, rmANOVA results revealed significant improvements in the directions *B*, *F*, and *C* with high effect sizes (*B*: $p = 0.042$, $\eta_p^2 = 0.168$; *F*: $p = 0.015$, $\eta_p^2 = 0.206$; *C*: $p < 0.001$, $\eta_p^2 = 0.356$). However, there were no significant effects for the factor group as well as no interaction effects between the factors time and group. Accordingly, jaw clenching had no effect on dynamic reactive balance performance. In addition, the training of jaw clenching in the INT group did not show any effects on dynamic reactive balance performance. Independent of the groups, the dynamic reactive performance was better at T2 compared to T1.

Center of mass kinematics

The $\mathrm{RoM}_{\mathrm{CoM\_AP}}$, $\mathrm{RoM}_{\mathrm{CoM\_ML}}$, and V_{CoM} results for the four directions are represented in Figures 4A, B. $\mathrm{RoM}_{\mathrm{CoM\_AP}}$ and $\mathrm{RoM}_{\mathrm{CoM\_ML}}$ did not show any significant effects. V_{CoM} had significant differences for the factor time in the directions *B*, *F*, *I*, and *C* with high effect sizes (*B*: $p < 0.001$, $\eta_p^2 = 0.869$; *F*: $p = 0.004$, $\eta_p^2 = 0.289$; *I*: $p = 0.027$, $\eta_p^2 = 0.230$; and *C*: $p = 0.037$, $\eta_p^2 = 0.220$). No significant effects for the factor group as well as no interaction effects between the factors time and group were detected. The results revealed that jaw clenching or its training had no significant effects on the center of mass kinematics. Across the groups, the V_{CoM} decreased at T2.

Jaw clenching task controlled by masseter activity

The mean activities of the muscle MA for each phase are shown in Table 1. MA showed significant effects in all of the five phases for the factor time with medium effect sizes (PRE: $p < 0.001$, $\eta_p^2 = 0.133$; SLR: $p < 0.001$, $\eta_p^2 = 0.116$; MLR: $p < 0.001$, $\eta_p^2 = 0.106$; LLR: $p < 0.001$, $\eta_p^2 = 0.113$; and DRP: $p < 0.001$, $\eta_p^2 = 0.121$) and for the factor group with high effect sizes (PRE: $p < 0.001$, $\eta_p^2 = 0.362$; SLR: $p < 0.001$, $\eta_p^2 = 0.364$; MLR: $p < 0.001$, $\eta_p^2 = 0.356$; LLR: $p < 0.001$, $\eta_p^2 = 0.351$; and DRP: $p < 0.001$, $\eta_p^2 = 0.340$). In two of the five phases, there were significant interaction effects of the factors group and time with medium effect sizes (PRE: $p = 0.002$, $\eta_p^2 = 0.066$; and SLR: $p = 0.003$, $\eta_p^2 = 0.061$). *Post hoc* results showed that HAB had significantly lower MA activity with high effect sizes in all of the five phases in comparison to INT (PRE: $p < 0.001$, $d = 1.268$; SLR: $p < 0.001$, $d = 1.260$; MLR: $p < 0.001$, $d = 1.240$; LLR: $p < 0.001$, $d = 1.229$; DRP: $p < 0.001$, $d = 1.225$) as well as in comparison to JAW (PRE: $p < 0.001$, $d = 1.674$; SLR: $p < 0.001$, $d = 1.681$; MLR: $p < 0.001$, $d = 1.641$; LLR: $p < 0.001$, $d = 1.621$; and DRP: $p < 0.001$, $d = 1.599$).

These results indicated, first, that the MA activity at T1 was higher than at T2 independent of the group. Second, the group HAB had significantly lower MA activity compared to both jaw clenching groups, INT and JAW, independent of the measurement time. Third, the reduction in MA activity level from T1 to T2 was partly higher for the jaw clenching group, INT, that trained for the task between two measurement times compared to JAW and HAB.

Muscle activities in the critical phases for reflexes

The mean activities of the analyzed muscles for each phase are shown in Tables 2, 3 for anterio-posterior and medio-lateral perturbations, respectively. The significant effects are highlighted in the tables. The corresponding *p*-values and effect sizes are reported in the following paragraphs.

For direction *B*, the muscle SOL showed significant interaction effects between the factors time and group with high effect sizes in

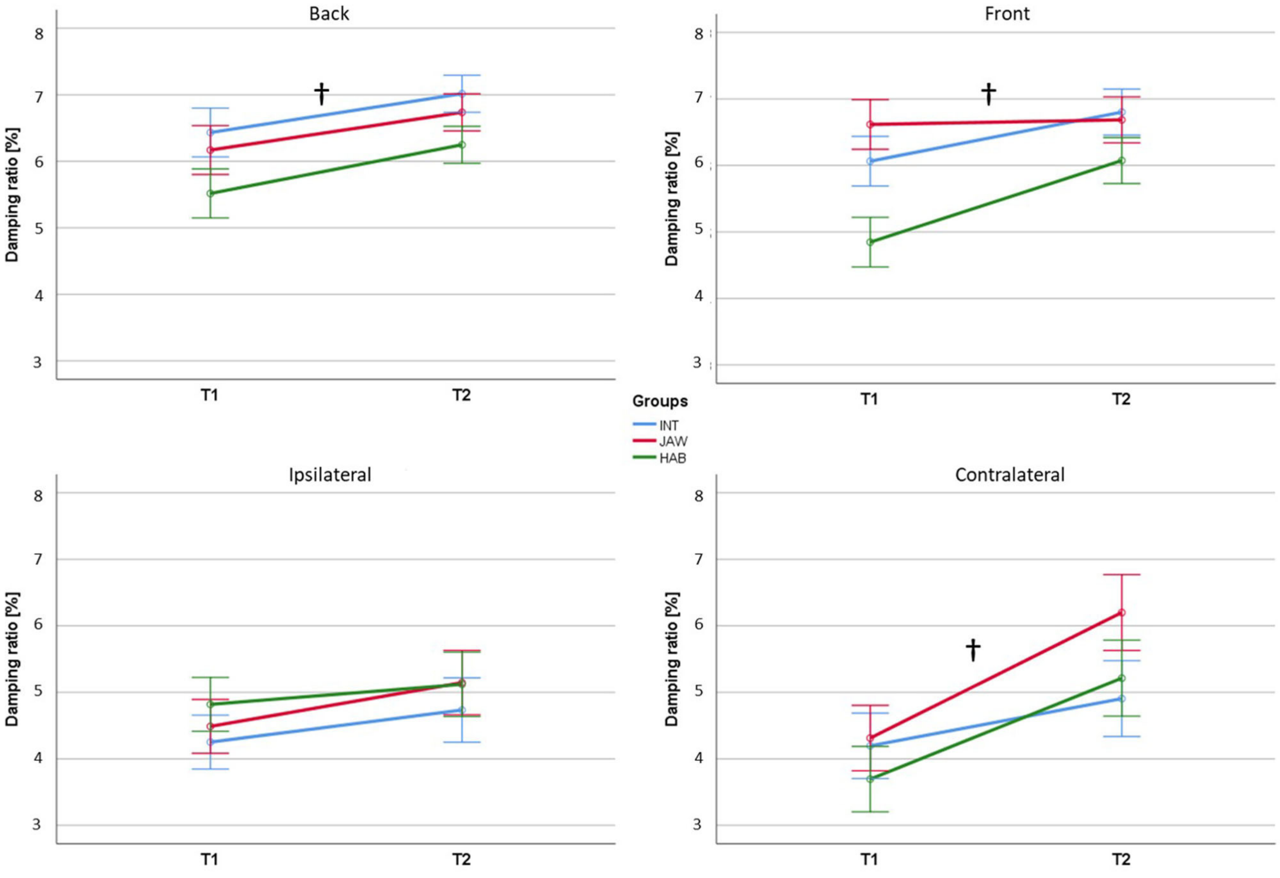

FIGURE 3
Damping ratio results. INT, intervention, JAW, jaw clenching, HAB, habitual. † signifies significant effects for the factor time. Significance level was set at $p < 0.05$.

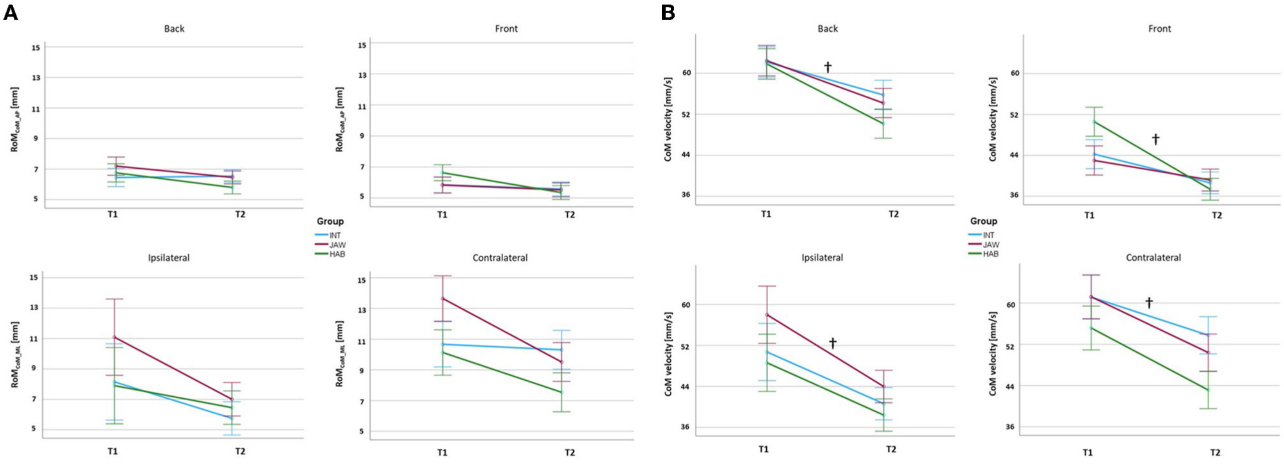

FIGURE 4
(A) RoM$_{CoM\_AP}$ and RoM$_{CoM\_ML}$ results **(B)** V_{CoM} results. INT, intervention; JAW, jaw clenching; HAB, habitual. † signifies significant effects for the factor time. The significance level was set at $p < 0.05$.

SLR ($p = 0.002$, $\eta_p^2 = 0.240$). At T2, the group JAW had an increased level of SOL activity compared to T1, whereas the other two groups had a decreased level. For the direction *F*, the muscle TA showed significant effects with high effect sizes for the factor time in three of the five phases (PRE: $p < 0.001$, $\eta_p^2 = 0.269$; SLR: $p = 0.003$, $\eta_p^2 = 0.177$; and DRP: $p < 0.001$, $\eta_p^2 = 0.333$) as well as for the factor group in one phase (MLR: $p < 0.001$, $\eta_p^2 = 0.306$). Across all groups, the level of TA activity was decreased at T2 compared to

TABLE 1 Mean muscle activities for Masseter in the five phases for all perturbation directions.

All directions		PRE		SLR		MLR		LLR		DRP	
		T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
Masseter	INT	**7.37 ± 9.47**$^{*\dagger\#}$	**4.80 ± 4.95**$^{*\dagger\#}$	**7.36 ± 9.84**$^{*\dagger\#}$	**4.50 ± 4.55**$^{*\dagger\#}$	**7.65 ± 9.48***†	**4.76 ± 4.95***†	**7.02 ± 8.77***†	**4.85 ± 5.29***†	**7.15 ± 8.74***†	**4.65 ± 4.94***†
	JAW	**5.10 ± 3.25**$^{*\dagger\#}$	**5.36 ± 3.14**$^{*\dagger\#}$	**5.19 ± 3.33**$^{*\dagger\#}$	**5.38 ± 3.37**$^{*\dagger\#}$	**5.09 ± 3.09***†	**5.32 ± 3.26***†	**5.12 ± 3.29***†	**5.26 ± 3.07***†	**4.88 ± 2.96***†	**5.11 ± 2.93***†
	HAB	**1.47 ± 2.90**$^{*\dagger\#}$	**0.45 ± 0.68**$^{*\dagger\#}$	**1.51 ± 2.98**$^{*\dagger\#}$	**0.48 ± 0.93**$^{*\dagger\#}$	**1.53 ± 2.95***†	**0.42 ± 0.69***†	**1.49 ± 2.84***†	**0.44 ± 0.72***†	**1.51 ± 2.77***†	**0.54 ± 0.99***†

Significant effects are highlighted in bold. † signifies significant effects for the factor time, * for the factor group, and # the interaction effects. The significance level was set at $p < 0.05$.

T1 in PRE, SLR, and DPR. Furthermore, across the measurement times, the JAW and INT groups had a higher level of TA activity compared to HAB in MLR. The *post hoc t*-test results revealed that these differences were valid at T1 but not at T2. For directions *C* and *I*, no significant effects were found.

In summary, the results showed that the reflex activity changes were limited to anterior–posterior directions (*B* and *F*). In the case of backward acceleration of the platform, the JAW group showed increases in SOL activity at T2, whereas the other two groups revealed decreases. In the case of forward acceleration of the platform, the TA activity was lower at T2 compared with T1 in three reflex phases, independent of the groups. Furthermore, the two jaw clenching groups (JAW and INT) had higher TA activity compared to HAB in the MLR phase at T1.

Discussion

This study aimed to investigate the effects of jaw clenching training on a dynamic reactive balance task performance after 1 week of jaw clenching training. It was hypothesized that jaw clenching has stabilizing effects resulting in better dynamic reactive balance performance, and these effects persist at T2 after the intervention. This would mean that these improvements are not a result of the dual-task effect but are specifically associated with jaw clenching. The results indicated that neither jaw clenching nor its automation through training resulted in significant dynamic reactive balance performance differences. However, independent of the groups, the dynamic reactive balance performance was better at T2 compared to T1. As there was not any deliberate balance training in the intervention phase, this result is indicative of high learning effects. Furthermore, jaw clenching may lead to some changes in reflex activities, but they are limited to anterior–posterior perturbation of the platform.

Effects of jaw clenching on dynamic reactive balance performance and CoM kinematics

Dynamic reactive balance performance was operationalized by the damping ratio as in other studies (10, 38). In addition, the RoM of CoM along the perturbation axis, as well as V_{CoM}, were calculated. In all of the directions, no significant effects due to jaw clenching were observed. Previous studies showed that jaw clenching may affect balance performance under static steady-state conditions (12, 29, 30, 53) as well as under dynamic conditions (10, 14). However, the nature of these effects is still unknown and could be associated with the dual-task situation. To the best of our knowledge, research so far has not addressed this point explicitly. This study investigated the effects of jaw clenching on dynamic reactive balance performance after 1 week of jaw clenching training to determine whether the effects are a result of the general dual-task situation or specifically due to the neurophysiological effects of jaw clenching. At T1 and T2, both INT and JAW groups were instructed to do the same dual task.

These two groups differed only in the intervention: INT trained in the jaw clenching task, whereas JAW did not. It was assumed that after 1 week of training (18 training sessions with 10 min of practice), the participants of INT would be able to fulfill the jaw clenching task in an automated manner. Therefore, it was hypothesized that the INT group would have reduced focused attention on the secondary jaw clenching task (31) and, therefore, a worse balance performance than JAW at T2. However, the results did not reveal any significant performance differences between the groups. Based on this, it can be concluded that the jaw clenching task did not have any observable effects on dynamic reactive balance performance, which was operationalized by the damping ratio and CoM kinematics. Furthermore, its automation also did not result in any significant changes. On the other hand, another explanation might be that the response of the motor system to the complexity of the present balance task possibly masked the effects of jaw clenching, which were identified in previous experiments with static balance tasks (29, 30). In addition, in a previous study by Tardieu et al. (54), the effects of dental occlusion on postural control were investigated both in eyes open and closed conditions. They reported that the sensory information associated with dental occlusion becomes more important when the other sensory cues become scarce (e.g., eyes closed). Based on this, it can be suggested that a jaw clenching task might potentially be beneficial once sensory information from other sources reduces. Nevertheless, in this study, the balance task was performed with open eyes since the Posturomed task was too difficult to be handled with closed eyes.

High learning effects even without training between sessions

In three of four directions (*B*, *F*, and *C*), dynamic reactive balance performance was improved at T2 even though the participants did not perform any balance training between T1 and T2. Furthermore, in all directions, V_{CoM} decreased significantly at T2, whereas the $\mathrm{RoM}_{\mathrm{CoM\_AP}}$ and $\mathrm{RoM}_{\mathrm{CoM\_ML}}$ were not affected. It should be noted that the participants performed familiarization trials before the real measurements as in similar studies (40, 55). Furthermore, within the individual measurement session, there were no systematic performance improvements in terms of dynamic reactive balance. These results indicate that learning effects occurred without deliberate balance training for this specific task. Subsequently, the question arose whether the learning effects were so large that they outweighed the possible effects of jaw clenching. With this study design, this question cannot be answered, and further studies are needed. From the findings of this study, it can be concluded that the balance task used here shows high learning effects and is rather unsuitable for studies in which low intervention effects on balance performance are expected. In the present case as well as in similar cases, care should therefore be taken to select a balance task that shows only low learning effects or a longer intervention period should be scheduled between T1 and T2 to mitigate the unwanted learning effects.

The results also revealed that the velocity of the CoM changed, but its RoM in the perturbation direction did not change at T2. This may be explained by the decreased CoM movement in case of the better damping of the platform by the participants since the CoM is one of the controlled variables as suggested in postural studies (56, 57). On the other hand, $\mathrm{RoM}_{\mathrm{CoM\_AP}}$ and $\mathrm{RoM}_{\mathrm{CoM\_ML}}$ depended for the most part on the initial maximum displacement of the platform, which was identical at T1 and T2. Therefore, the RoM did not change at T2.

Changed muscular activity levels at T2

The results regarding muscle activities in reflex phases revealed that in the case of the backward perturbation of the platform (*B*), the SOL activity of JAW increased, whereas that of the other two groups decreased at T2 in the SLR phase. It is important to add that SOL is one of the most important muscles that help to restore equilibrium in response to posterior translations (46). This result may be interpreted as a difference between INT and JAW groups, and it can be suggested that the jaw clenching task resulted in increased muscle activity in SOL at T2, but these effects were not visible when the jaw clenching task became an implicit task and therefore lost its novelty (e.g., for the group INT). In addition, in the case of forward acceleration of the platform, TA activity of both jaw clenching groups (INT and JAW) was higher overall compared to that of HAB in the MLR phase at T1. This finding contradicts the initial hypothesis that the jaw clenching task results in changes in reflex activities, and these effects persist after 1 week of jaw clenching training. Nevertheless, these results are only limited to this perturbation direction and to this specific reflex phase. Furthermore, these changes did not cause any effects on dynamic reactive balance performance (i.e., damping ratio results).

In response to anterior surface translations, TA contracts to counteract the torques at the ankle and, therefore, helps to restore equilibrium (46). The TA activity decreased at T2 in three phases (PRE, SLR, and DRP) across the groups parallel to dynamic balance performance improvements. These results indicate that in the case of forward acceleration of the platform, better performance at T2 is possibly related to a decreased TA activity. In general, significant changes were detected only for the anterior–posterior perturbation directions. Based on these results, it can be suggested that the jaw clenching task may result in changed muscle activity patterns, as observed with the alterations in certain muscle activities in the reflex phases, but changes seem to be direction-dependent as well as muscle dependent. This task specificity can be explained by the different postural responses to different perturbation directions (49, 50, 58).

Furthermore, it should be noted that the muscle activity changes, and the dynamic balance performance differences did not show a common pattern for all directions (e.g., no changes in muscle activity levels in perturbation direction *C*, despite the improvements in dynamic reactive balance performance at T2). This may also possibly have been caused by the selection of the posture relevant muscles. Posture and its control are the

TABLE 2 Mean muscle activities for perturbation-relevant muscles in the five phases for anterio-posterior perturbations.

Back		PRE		SLR		MLR		LLR		DRP	
		T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
Peroneus longus	INT	14.41 ± 10.92	13.74 ± 9.68	16.82 ± 16.77	17.68 ± 17.08	17.17 ± 12.68	18.54 ± 18.13	14.81 ± 10.70	19.46 ± 15.65	22.63 ± 11.40	22.56 ± 14.41
	JAW	21.08 ± 11.67	12.75 ± 6.80	22.94 ± 15.97	16.16 ± 11.00	21.04 ± 11.88	14.39 ± 9.14	20.13 ± 11.78	17.16 ± 12.68	26.51 ± 10.56	21.06 ± 10.22
	HAB	37.55 ± 74.72	19.41 ± 18.60	26.26 ± 35.13	20.87 ± 16.98	44.48 ± 99.47	23.17 ± 30.02	45.94 ± 103.20	22.14 ± 23.77	37.21 ± 41.44	25.42 ± 18.58
Soleus	INT	19.62 ± 12.34	15.31 ± 5.02	**21.22 ± 11.71#**	**13.75 ± 5.59#**	20.60 ± 24.14	14.83 ± 11.05	21.81 ± 19.00	18.38 ± 12.32	26.36 ± 13.56	24.26 ± 15.23
	JAW	14.63 ± 6.71	18.32 ± 7.38	**12.81 ± 6.19#**	**18.32 ± 9.54#**	15.43 ± 12.10	14.31 ± 7.70	18.42 ± 12.82	16.77 ± 10.09	21.77 ± 10.08	23.57 ± 11.87
	HAB	16.23 ± 10.66	12.95 ± 7.97	**16.95 ± 13.18#**	**11.41 ± 5.91#**	15.54 ± 16.53	10.19 ± 4.82	13.01 ± 9.51	12.92 ± 7.06	20.83 ± 11.57	19.56 ± 9.38
Front		PRE		SLR		MLR		LLR		DRP	
		T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
Tibialis anterior	INT	**11.96 ± 13.00**†	**5.93 ± 7.86**†	**12.68 ± 15.92**†	**5.30 ± 5.83**†	**11.79 ± 11.29***	**4.42 ± 3.71***	9.57 ± 8.28	6.28 ± 5.06	**14.23 ± 5.83**†	**10.34 ± 6.42**†
	JAW	**9.63 ± 5.85**†	**6.05 ± 2.86**†	**10.59 ± 9.50**†	**5.17 ± 3.02**†	**10.86 ± 7.11***	**5.38 ± 2.94***	9.60 ± 6.94	5.82 ± 5.20	**15.40 ± 8.23**†	**9.66 ± 5.14**†
	HAB	**7.09 ± 5.28**†	**5.79 ± 2.78**†	**5.50 ± 4.01**†	**5.78 ± 3.81**†	**6.39 ± 4.15***	**6.50 ± 5.71***	6.19 ± 3.92	7.03 ± 4.45	**13.41 ± 7.28**†	**11.49 ± 5.93**†
Rectus abdominis	INT	1.45 ± 2.13	0.91 ± 1.38	1.46 ± 2.51	0.94 ± 1.46	1.43 ± 2.05	0.84 ± 1.15	1.37 ± 1.70	0.81 ± 1.24	1.45 ± 1.59	1.08 ± 1.63
	JAW	0.86 ± 0.57	0.90 ± 0.68	0.86 ± 0.77	0.81 ± 0.74	0.75 ± 0.43	0.82 ± 0.61	0.96 ± 0.75	0.86 ± 0.90	1.08 ± 0.72	0.91 ± 0.68
	HAB	1.14 ± 1.12	1.27 ± 1.02	1.16 ± 1.12	1.07 ± 0.96	1.08 ± 1.07	1.18 ± 0.95	0.90 ± 0.76	1.31 ± 1.04	1.43 ± 1.17	1.26 ± 0.85

Significant effects are highlighted in bold. † signifies significant effects for the factor time, *for the factor group, and # the interaction effects. The significance level was set at $p < 0.05$.

TABLE 3 Mean muscle activities for perturbation-relevant muscles in the five phases for medio-lateral perturbations.

Ipsilateral		PRE		SLR		MLR		LLR		DRP	
		T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
Semitendinosus	INT	5.04 ± 5.50	4.11 ± 6.86	7.17 ± 9.62	3.86 ± 5.55	6.28 ± 9.69	4.48 ± 7.11	6.12 ± 8.20	4.80 ± 8.75	7.95 ± 7.73	6.53 ± 8.51
	JAW	3.88 ± 3.41	3.11 ± 2.39	3.48 ± 3.53	3.04 ± 3.05	4.46 ± 4.06	3.65 ± 3.13	4.10 ± 3.23	3.76 ± 3.19	5.85 ± 4.86	4.06 ± 2.50
	HAB	4.99 ± 4.43	3.87 ± 5.10	4.65 ± 4.00	4.00 ± 5.46	4.69 ± 4.35	4.57 ± 5.82	5.20 ± 4.87	4.30 ± 5.79	6.75 ± 5.87	5.66 ± 6.99
Internal oblique	INT	2.85 ± 2.66	1.95 ± 1.77	3.33 ± 3.26	2.58 ± 1.96	2.95 ± 3.11	2.66 ± 2.42	3.16 ± 3.47	2.75 ± 2.89	3.72 ± 3.06	3.28 ± 2.34
	JAW	2.68 ± 1.54	1.87 ± 1.61	2.69 ± 1.69	2.52 ± 2.20	2.54 ± 1.29	2.65 ± 2.64	2.64 ± 1.39	2.80 ± 2.47	3.50 ± 1.55	3.30 ± 2.13
	HAB	2.21 ± 1.19	2.08 ± 1.41	2.24 ± 1.31	1.82 ± 1.46	2.22 ± 1.30	1.88 ± 1.17	2.09 ± 1.02	2.01 ± 1.31	3.91 ± 3.13	2.85 ± 2.09
Contralateral		PRE		SLR		MLR		LLR		DRP	
		T1	T2	T1	T2	T1	T2	T1	T2	T1	T2
Rectus femoris	INT	2.27 ± 4.32	3.05 ± 3.04	3.78 ± 2.77	3.29 ± 3.28	4.81 ± 8.34	3.13 ± 3.11	4.55 ± 7.35	3.33 ± 3.57	5.31 ± 3.93	5.23 ± 5.59
	JAW	3.82 ± 2.27	3.37 ± 3.30	5.42 ± 4.40	3.27 ± 3.06	3.61 ± 2.55	3.63 ± 3.76	4.74 ± 2.91	3.89 ± 4.22	6.23 ± 3.87	5.02 ± 3.88
	HAB	2.71 ± 2.69	2.62 ± 1.96	2.51 ± 1.98	2.60 ± 2.19	2.88 ± 2.91	2.45 ± 1.81	2.87 ± 2.62	2.60 ± 1.60	4.78 ± 4.87	4.23 ± 2.67
Erector spinae	INT	5.13 ± 3.17	7.00 ± 6.42	5.24 ± 3.06	5.74 ± 3.94	6.84 ± 5.59	7.25 ± 7.71	7.20 ± 6.47	6.80 ± 6.92	7.85 ± 5.10	10.22 ± 8.10
	JAW	4.57 ± 4.93	4.44 ± 3.56	4.06 ± 4.32	4.09 ± 3.08	4.73 ± 4.75	4.50 ± 4.05	4.84 ± 3.76	4.76 ± 4.73	8.13 ± 7.91	6.16 ± 4.51
	HAB	4.26 ± 3.45	4.79 ± 3.42	4.74 ± 4.01	4.58 ± 3.84	5.69 ± 7.52	4.16 ± 2.96	4.32 ± 3.30	4.41 ± 3.37	8.15 ± 7.11	6.98 ± 5.17

product of inter-muscular coordination patterns. Determining the activity of individual muscles might be the limiting factor in the analysis presented here. In light of these aspects, the question arises whether mean muscle activities for the critical phases were sensitive enough to reveal changes on a muscular level. Nevertheless, these parameters were used in similar studies [e.g., iEMG in Freyler et al. (40) and Pfusterschmied et al. (39)]. In the present study, mean muscle activity was preferred since DRP was not the same length for each trial or participant. It was expected that an increased level of reflex activities would be manifested by an increased level of muscle activities (59). However, potentially jaw clenching effects are seen less in a changed level of individual muscle activities and more in a changed interplay of different muscles. Therefore, in future studies, the coordination of different muscles should be analyzed in addition to the analysis of the activity of individual muscles. Coordination models, such as muscle synergies, are particularly suitable for this purpose (60, 61).

Jaw clenching task controlled by masseter activity

The EMG results indicate that the activity of the MA was higher for the groups, INT and JAW compared to HAB. This suggests that the majority of the subjects in HAB, who did not receive instructions regarding the activity of the stomatognathic system, had their jaws in the physiologically expected resting position (lips closed, teeth out of contact). It should be noted that the participants of JAW and INT trained immediately before starting the balancing task measurements with the Rehabite® device so they can apply a force at a level of 75 N consistently without feedback. The higher reduction in MA activity between T1 and T2 in the INT group compared with the other groups can be attributed to the training during the intervention phase. Similar effects were also shown in a previous study (62), in which short-term force-controlled biting on a hydrostatic system caused long-term training effects.

A force of 75 N is easy to achieve for the stomatognathic system as normal masticatory activities are in the range of this force level. The RehaBite®-training in the group INT between T1 and T2 was used to turn a novel, unfamiliar task (biting on a hydrostatic system is not part of the common functional repertoire of the stomatognathic system) into an implicit behavior so that it would not require additional attention during the balancing task. Therefore, RehaBite® training between T1 and T2 in INT was not used to train the masticatory muscles but to address a potential dual-task effect during the balance task. It should also be noted that the jaw clenching task in this study is a different stomatognathic activity than the daily chewing activity occurring when eating (62). During the submaximum jaw clenching task, a force of 75 N was applied continuously, whereas, during chewing, an alternating force is applied. Based on this, it can be assumed that the deliberate jaw clenching task was novel to the participants at the first measurements. Furthermore, it was also shown that the chewing task had no significant effects on body sway reduction during upright standing, whereas the feedback-controlled jaw clenching task had significant effects (53). This also supports that the submaximum jaw clenching and the chewing tasks are not the same task, and they may lead to different neurophysiological effects.

Limitations

This study has some limitations. First, even though the participants did not train for the balance task, learning effects occurred in three of the four directions independent of the group. These high learning effects may have outweighed the potential effects of jaw clenching. For future studies, more care should be taken to minimize possible learning effects. Second, all the participants were physically active and healthy adults, therefore potentially good at balancing. The same results may not be seen in groups with compromised postural control such as the elderly (63) or people with neurological disorders (64). In future studies, the participants with poorer postural control might reveal the effects of jaw clenching. Third, the onset of the reflex phases was defined based on Posturomed movement but not on muscle activity peaks (45) or ankle movements since there were no clear peaks in the EMG or kinematics data. Finally, the group HAB did not receive any instructions regarding stomatognathic activities. Self-administrative questionnaires regarding the clenching habit would have been useful to collect habitual status.

Conclusion

This study investigated the effects of jaw clenching on dynamic reactive balance task performance after 1 week of jaw clenching training to examine whether the effects are a result of a dual-task situation. Both jaw clenching and automation of the jaw clenching task seemed not to have any observable effects on dynamic reactive balance performance, but jaw clenching seemed to be related to some changes in reflex activities. However, these effects were limited to anterior–posterior perturbations. Further studies containing other balance tasks with less learning effects as well as with longer intervention periods are needed. Analysis of muscle coordination, as well as other experimental designs with reduced sensory information from other sources (e.g., closed eyes), may also help to reveal jaw clenching effects.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving human participants were reviewed and approved by Ethics Committee of the Karlsruhe Institute of Technology. The participants provided their written informed consent to participate in this study.

Author contributions

CF and LK conducted the experiment. CF carried out data analysis and took the lead in writing the manuscript. All authors were involved in the interpretation and discussion of the results, provided critical feedback, contributed to the article, and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

References

1. Shumway-Cook A, Woollacott MH. *Motor Control, Translating Research into Clinical Practice*. Philadelphia: Wolters Kluwer. (2017).

2. Rubenstein LZ. Falls in older people: epidemiology, risk factors and strategies for prevention. *Age Ageing.* (2006) 35:37–41. doi: 10.1093/ageing/afl084

3. Hof AL, Gazendam MGJ, Sinke WE. The condition for dynamic stability. *J Biomech.* (2005) 38:1–8. doi: 10.1016/j.jbiomech.2004.03.025

4. Hrysomallis C. Balance ability and athletic performance. *Sports Med.* (2011) 41:221–32. doi: 10.2165/11538560-000000000-00000

5. Hrysomallis C. Relationship between balance ability, training and sports injury risk. *Sports Med.* (2007) 37:547–56. doi: 10.2165/00007256-200737060-00007

6. Sherrington C, Whitney JC, Lord SR, Herbert RD, Cumming RG, Close JCT. Effective exercise for the prevention of falls: a systematic review and meta-analysis. *J Am Geriatr Soc.* (2008) 56:2234–43. doi: 10.1111/j.1532-5415.2008.02014.x

7. Sherrington C, Fairhall NJ, Wallbank GK, Tiedemann A, Michaleff ZA, Howard K, et al. New Cochrane review assesses the benefits and harms of exercise for preventing fall s in older people living in the community. *Saudi Med J.* (2019) 40:CD012424. doi: 10.1002/14651858.CD012424.pub2

8. Peterka RJ. Sensorimotor integration in human postural control. *J Neurophysiol.* (2002) 88:1097–118. doi: 10.1152/jn.2002.88.3.1097

9. Winter DA. Human balance and posture standing and walking control during. *Gait Posture.* (1995) 3:193–214. doi: 10.1016/0966-6362(96)82849-9

10. Fadillioglu C, Kanus L, Möhler F, Ringhof S, Schindler HJ, Stein T, et al. Influence of controlled masticatory muscle activity on dynamic reactive balance. *J Oral Rehabil.* (2022) 49:327–336. doi: 10.1111/joor.13284

11. Julià-Sánchez S, Álvarez-Herms J, Gatterer H, Burtscher M, Pagès T, Viscor G. Dental occlusion influences the standing balance on an unstable platform. *Motor Control.* (2015) 19:341–54. doi: 10.1123/mc.2014-0018

12. Ringhof S, Leibold T, Hellmann D, Stein T. Postural stability and the influence of concurrent muscle activation - beneficial effects of jaw and fist clenching. *Gait and Posture.* (2015) 42:598–600. doi: 10.1016/j.gaitpost.2015.09.002

13. Allen CR, Fu YC, Cazas-Moreno V, Valliant MW, Gdovin JR, Williams CC, et al. Effects of jaw clenching and jaw alignment mouthpiece use on force production during vertical jump and isometric clean pull. *J Strength Cond.* (2018) 32:237–43. doi: 10.1519/JSC.0000000000002172

14. Tomita Y, Tanaka Y, Sako K, Ono Y, Tanaka M. Effect of jaw clenching on postural adjustments to a predictable external perturbation. *J Electromyogr Kinesiol.* (2021) 57:102512. doi: 10.1016/j.jelekin.2020.102512

15. Ohlendorf D, Seebach K, Hoerzer S, Nigg S, Kopp S. The effects of a temporarily manipulated dental occlusion on the position of the spine: a comparison during standing and walking. *Spine J.* (2014) 14:2384–91. doi: 10.1016/j.spinee.2014.01.045

16. Alghadir A, Zafar H, Whitney SL, Iqbal Z. Effect of chewing on postural stability during quiet standing in healthy young males. *Somatosens Motor Res.* (2015) 32:72–6. doi: 10.3109/08990220.2014.969837

17. Zafar H, Alghadir AH, Iqbal ZA, Iqbal A, Anwer S, Alnahdi AH. Influence of different jaw positions on dynamic balance using Y-balance test. *Brain Behav.* (2020) 10:1–6. doi: 10.1002/brb3.1507

18. Alghadir AH, Zafar H, Iqbal ZA. Effect of tongue position on postural stability during quiet standing in healthy young males. *Somatosens Motor Res.* (2015) 32:183–6. doi: 10.3109/08990220.2015.1043120

19. Boroojerdi B, Battaglia F, Muellbacher W, Cohen LG. Voluntary teeth clenching facilitates human motor system excitability. *Clini Neurophysiol.* (2000) 111:988–93. doi: 10.1016/S1388-2457(00)00279-0

20. Miyahara T, Hagiya N, Ohyama T, Nakamura Y. Modulation of human soleus H reflex in association with voluntary clenching of the teeth. *J Neurophysiol.* (1996) 76:2033–41. doi: 10.1152/jn.1996.76.3.2033

21. Tuncer M, Tucker KJ, Türker KS. Influence of tooth clench on the soleus H-reflex. *Arch Oral Biol.* (2007) 52:374–6. doi: 10.1016/j.archoralbio.2006.12.011

22. Takada Y. Miyahara, Taniguchi H, Ohyama T, Nakamura Y. Modulation of H reflex of pretibial and soleus muscles during mastication in humans. *Muscle and Nerve.* (2001) 24:1142–8. doi: 10.1002/mus.1125

23. Jendrassik E. Zur untersuchung des kniephaenomens. *Neur Zbl.* (1885) 4:412–5.

24. Giannakopoulos NN, Schindler HJ, Hellmann D. Co-contraction behaviour of masticatory and neck muscles during tooth grinding. *J Oral Rehabil.* (2018) 45:504–11. doi: 10.1111/joor.12646

25. Gangloff P, Louis JP, Perrin PP. Dental occlusion modifies gaze and posture stabilization in human subjects. *Neurosci Lett.* (2000) 293:203–6. doi: 10.1016/S0304-3940(00)01528-7

26. Abrahams VC. The physiology of neck muscles; their role in head movement and maintenance of posture. *Can J Physiol Pharmacol.* (1977) 55:332–8. doi: 10.1139/y77-047

27. Tanaka S, Hirai T, Koshino H, Yokoyama Y, Ishijima T. Influence of teeth clenching on the bodily equilibrium against striking weight impact. *Prosthodontic Res Pract.* (2006) 5:143–9. doi: 10.2186/prp.5.143

28. Ruggiero DA, Ross CA, Reis DJ. Projections from the spinal trigeminal nucleus to the entire length of the spinal cord in the rat. *Brain Res.* (1981) 225:225–33. doi: 10.1016/0006-8993(81)90832-5

29. Hellmann D, Stein T, Potthast W, Rammelsberg P, Schindler HJ, Ringhof S. The effect of force-controlled biting on human posture control. *Hum Mov Sci.* (2015) 43:125–37. doi: 10.1016/j.humov.2015.08.009

30. Ringhof S, Stein T, Potthast W, Schindler H, Hellmann D. Force-controlled biting alters postural control in bipedal and unipedal stance. *J Oral Rehabil.* (2015) 42:173–84. doi: 10.1111/joor.12247

31. Andersson G, Hagman J, Talianzadeh R, Svedberg A, Larsen HC. Effect of cognitive load on postural control. *Brain Res Bull.* (2002) 58:135–9. doi: 10.1016/S0361-9230(02)00770-0

32. Broglio SP, Tomporowski PD, Ferrara MS. Balance performance with a cognitive task: A dual-task testing paradigm. *Med Sci Sports Exerc.* (2005) 37:689–95. doi: 10.1249/01.MSS.0000159019.14919.09

33. Schmidt RA, Lee TD, Winstein C, Wulf G, Zelaznik HN. *Motor Control and Learning: A Behavioral Emphasis*. Champaign, IL: Human Kinetics (2018).

34. Woollacott M, Shumway-Cook A. Attention and the control of posture and gait: a review of an emerging area of research. *Gait Posture.* (2002) 16:1–14. doi: 10.1016/S0966-6362(01)00156-4

35. Wachholz F, Tiribello F, Promsri A, Federolf P. Should the minimal intervention principle be considered when investigating dual-tasking effects on postural control? *Brain Sci.* (2020) 10:1–13. doi: 10.3390/brainsci10010001

36. Fadillioglu C, Kanus L, Möhler F, Ringhof S, Hellmann D, Stein T. Influence of controlled stomatognathic motor activity on sway, control and stability of the center of mass during dynamic steady-state balance—an uncontrolled manifold analysis. *Front Human Neurosci.* (2022) 16:868828. doi: 10.3389/fnhum.2022.868828

37. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Orofac Pain.* (1992) 6:301–55.

38. Kiss RM, A. new parameter for characterizing balancing ability on an unstable oscillatory platform. *Med Eng Physics.* (2011) 33:1160–6. doi: 10.1016/j.medengphy.2011.04.017

39. Pfusterschmied J, Stöggl T, Buchecker M, Lindinger S, Wagner H, Müller E. Effects of 4-week slackline training on lower limb joint motion and muscle activation. *J Sci Med Sport.* (2013) 16:562–6. doi: 10.1016/j.jsams.2012.12.006

40. Freyler K, Gollhofer A, Colin R, Brüderlin U, Ritzmann R. Reactive balance control in response to perturbation in unilateral stance: interaction effects of direction, displacement and velocity on compensatory neuromuscular and kinematic responses. *PLoS ONE.* (2015) 10:1–18. doi: 10.1371/journal.pone.0144529

41. Ringhof S, Stein T. Biomechanical assessment of dynamic balance: specificity of different balance tests. *Hum Mov Sci.* (2018) 58:140–7. doi: 10.1016/j.humov.2018.02.004

42. Giannakopoulos NN, Rauer AK, Hellmann D, Hugger S, Schmitter M, Hugger A. Comparison of device-supported sensorimotor training and splint intervention for myofascial temporomandibular disorder pain patients. *J Oral Rehabil.* (2018) 45:669–76. doi: 10.1111/joor.12662

43. Härtel T, Hermsdorf H. Biomechanical modelling and simulation of human body by means of DYNAMICUS. *J Biomech.* (2006) 39:549. doi: 10.1016/S0021-9290(06)85262-0

44. Hermens H, Freriks B, Merletti R, Stegeman D, Blok J, Rau G, et al. *European Recommendations for Surface Electromyography: Results of the SENIAM Project*. Enschede: Roessingh Research and Development (1999).

45. Taube W, Schubert M, Gruber M, Beck S, Faist M, Gollhofer A. Direct corticospinal pathways contribute to neuromuscular control of perturbed stance. *J Appl Physiol.* (2006) 101:420–9. doi: 10.1152/japplphysiol.01447.2005

46. Henry SM, Fung J, Horak FB, EMG. responses to maintain stance during multidirectional surface translations. *J Neurophysiol.* (1998) 80:1939–50. doi: 10.1152/jn.1998.80.4.1939

47. Pohl T, Brauner T, Wearing S, Horstmann T. Limb movement, coordination and muscle activity during a cross-coordination movement on a stable and unstable surface. *Gait and Posture.* (2020) 81:131–7. doi: 10.1016/j.gaitpost.2020.07.019

48. Alizadehsaravi L, Bruijn SM, Maas H, van Dieën JH. Modulation of soleus muscle H-reflexes and ankle muscle co-contraction with surface compliance during unipedal balancing in young and older adults. *Exp Brain Res.* (2020) 238:1371–83. doi: 10.1007/s00221-020-05784-0

49. Nonnekes J, Scotti A, Oude Nijhuis LB, Smulders K, Queralt A, Geurts ACH, et al. Are postural responses to backward and forward perturbations processed by different neural circuits? *Neuroscience.* (2013) 245:109–20. doi: 10.1016/j.neuroscience.2013.04.036

50. Chen CL, Lou SZ, Wu HW, Wu SK, Yeung KT, Su FC. Effects of the type and direction of support surface perturbation on postural responses. *J Neuroeng Rehabil.* (2014) 11:1–12. doi: 10.1186/1743-0003-11-50

51. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. Mahwah, NJ: Lawrence Erlbaum Associates (1988) p. 20–27

52. Holm S, A. Simple Sequentially Rejective Multiple Test Procedure Author (s): Sture Holm Published by: Wiley on behalf of Board of the Foundation of the Scandinavian Journal of Statistics Stable U. *Scand J Statist.* (1979) 6:65–70.

53. Hellmann D, Giannakopoulos NN, Blaser R, Eberhard L, Schindler HJ. The effect of various jaw motor tasks on body sway. *J Oral Rehabil.* (2011) 38:729–36. doi: 10.1111/j.1365-2842.2011.02211.x

54. Tardieu C, Dumitrescu M, Giraudeau A, Blanc JL, Cheynet F, Borel L. Dental occlusion and postural control in adults. *Neurosci Lett.* (2009) 450:221–4. doi: 10.1016/j.neulet.2008.12.005

55. Petró B, T Nagy J, Kiss RM. Effectiveness and recovery action of a perturbation balance test–a comparison of single-leg and bipedal stances. *Comput Methods Biomech Biomed Engin.* (2018) 21:593–600. doi: 10.1080/10255842.2018.1502278

56. Winter DA, Patla AE, Prince F, Ishac M, Gielo-perczak K. Stiffness control of balance in quiet standing. *J Neurophysiol.* (1998) 80:1211–21. doi: 10.1152/jn.1998.80.3.1211

57. Richmond SB, Fling BW, Lee H, Peterson DS. The assessment of center of mass and center of pressure during quiet stance: current applications and future directions. *J Biomech.* (2021) 123:110485. doi: 10.1016/j.jbiomech.2021.110485

58. Kiss RM. Do lateral dominance, body mass, body height and direction of perturbation influence the lehr's damping ratio, which characterizes the balancing ability on an unstable oscillatory platform? *WIT Trans Built Environ.* (2011) 15:373–82. doi: 10.2495/EHR110321

59. Ertuglu LA, Karacan I, Yilmaz G, Türker KS. Standardization of the Jendrassik maneuver in Achilles tendon tap reflex. *Clin Neurophysiol Pract.* (2018) 3:1–5. doi: 10.1016/j.cnp.2017.10.003

60. Ting LH, Macpherson JM. A limited set of muscle synergies for force control during a postural task. *J Neurophysiol.* (2005) 93:609–13. doi: 10.1152/jn.00681.2004

61. Munoz-Martel V, Santuz A, Bohm S, Arampatzis A. Proactive modulation in the spatiotemporal structure of muscle synergies minimizes reactive responses in perturbed landings. *Front Bioeng Biotechnol.* (2021) 9:761766. doi: 10.3389/fbioe.2021.761766

62. Hellmann D, Giannakopoulos NN, Blaser R, Eberhard L, Rues S, Schindler HJ. Long-term training effects on masticatory muscles. *J Oral Rehabil.* (2011) 38:912–20. doi: 10.1111/j.1365-2842.2011.02227.x

63. Henry M, Baudry S. Age-related changes in leg proprioception: implications for postural control. *J Neurophysiol.* (2019) 122:525–38. doi: 10.1152/jn.00067.2019

64. Xia R, Mao ZH. Progression of motor symptoms in Parkinson's disease. *Neurosci Bull.* (2012) 28:39–48. doi: 10.1007/s12264-012-1050-z

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Effects of postural threat on perceptions of lower leg somatosensory stimuli during standing

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Height-induced postural threat affects emotional state and standing balance behaviour during static, voluntary, and dynamic tasks. Facing a threat to balance also affects sensory and cortical processes during balance tasks. As sensory and cognitive functions are crucial in forming perceptions of movement, balance-related changes during threatening conditions might be associated with changes in conscious perceptions. Therefore, the purpose of this study was to examine the changes and potential mechanisms underlying conscious perceptions of balance-relevant information during height-induced postural threat. A combination of three experimental procedures utilized height-induced postural threat to manipulate emotional state, balance behavior, and/or conscious perceptions of balance-related stimuli. Experiment 1 assessed conscious perception of foot position during stance. During continuous antero-posterior pseudorandom support surface rotations, perceived foot movement was larger while actual foot movement did not change in the High (3.2 m, at the edge) compared to Low (1.1 m, away from edge) height conditions. Experiment 2 and 3 assessed somatosensory perceptual thresholds during upright stance. Perceptual thresholds for ankle rotations were elevated while foot sole vibrations thresholds remained unchanged in the High compared to Low condition. This study furthers our understanding of the relationship between emotional state, sensory perception, and balance performance. While threat can influence the perceived amplitude of above threshold ankle rotations, there is a reduction in the sensitivity of an ankle rotation without any change to foot sole sensitivity. These results highlight the effect of postural threat on neurophysiological and cognitive components of balance control and provide insight into balance assessment and intervention.

KEYWORDS

postural threat, balance, perception, somatosensory, fear, anxiety

1. Introduction

Postural threat associated with standing on elevated surfaces, or anticipation of a balance perturbation affects emotional state and standing balance behavior during static, voluntary, and dynamic tasks (Adkin and Carpenter, 2018). Facing a threat to balance also affects sensory and cortical processes during balance tasks (Adkin et al., 2008; Davis et al., 2011; Horslen et al., 2013;

Naranjo et al., 2016). Fear and anxiety responses to actual or perceived threats can be accompanied by changes in perception of individual sensory inputs, including auditory (Borsky, 1979; Siegel and Stefanucci, 2011; Asutay and Västfjäll, 2012; Gagnon et al., 2013), visual (Stefanucci et al., 2008; Teachman et al., 2008; Clerkin et al., 2009; Vasey et al., 2012) and tactile stimuli (Shi et al., 2012). Multi-sensory perceptions, such as those related to whole-body postural sway (Fitzpatrick and McCloskey, 1994; Mergner and Rosemeier, 1998), have also been shown to be amplified under threatening conditions during quiet standing (Cleworth and Carpenter, 2016), voluntary leaning (Cleworth et al., 2018), and dynamic stance tasks (Cleworth et al., 2019). However, the mechanisms through which fear and anxiety affect these perceptions of whole-body movement are currently not known.

Changes in the amplitude of perceived movements may be mediated by (a) a decrease in the detectable threshold (stimulus/contrast gain), (b) an amplification of the response proportional to stimulus intensity (response gain), or (c) a combination of the two (Horak and Diener, 1994; Lim et al., 2014). Decreasing the threshold for detectable movements will allow smaller movements to be perceived, increasing somatosensory acuity. Alternatively, the response to a detectable stimulus may be modified by a gain factor resulting in an increased response strength proportional to stimulus intensity, independent of changes in perceptual thresholds.

Both stimulus gain and response gain may rely on peripheral sensory receptors and central processing of the sensory information (Dannenbaum and Jones, 1993; Simoneau et al., 1995), both of which have been shown to be affected by a height-induced postural threat (Adkin et al., 2008; Sibley et al., 2010; Davis et al., 2011; Horslen et al., 2013; Naranjo et al., 2016). Whole-body sway is thought to be heavily reliant on afferent information from somatosensory receptors in the ankle (Thelen et al., 1998), as the majority of sway occurs about the ankle joint in the sagittal plane (Gage et al., 2004). Likewise, movement detection thresholds are lowest for somatosensory-related movements compared to visual or vestibular systems (Fitzpatrick and McCloskey, 1994). It is therefore important to consider the somatosensory system as a potential contributor to height-related effects on perception.

The aim of this study was to investigate the effect of height-induced postural threat on lower leg somatosensory acuity. Since somatosensory acuity relies on different classes of somatosensory receptors including muscle spindles and cutaneous mechanoreceptors (Refshauge and Fitzpatrick, 1995), multiple experiments were needed to determine how different classes of somatosensory receptors contribute to any height-related changes. Three unique experiments were performed to examine how threat influences conscious perception of passive ankle rotation (Experiment 1), and perceptual thresholds for passive ankle rotation and cutaneous foot sole stimulation (Experiments 2 and 3 respectively) in a fully loaded leg during upright stance. Uni-lateral stimuli (ankle rotations and foot-sole stimulation) were applied with subjects braced to minimize balance perturbations, and isolate perception of somatosensory changes from other potential sensory inputs (ie vestibular) that would be involved in whole-body sway. Based on prior work, we hypothesized that conscious perception and perceptual thresholds of passive ankle joint rotation would be larger under postural threat and independent of rotation direction (Adkin et al., 2008; Davis et al., 2011; Horslen et al., 2013, 2018; Cleworth and Carpenter, 2016; Cleworth et al., 2019). Likewise, we hypothesized that height-induced threat would decrease perceptual thresholds of foot-sole vibrations independent of frequency, while higher frequency vibrations would be more easily perceived than low frequency across height conditions (Strzalkowski et al., 2015).

2. Materials and methods

A total of 45 young healthy adults volunteered to participate in this study across 3 experiments; Experiment 1: $n = 14$ (9 Female), mean age 25.4 ± 5.0 years; Experiment 2: $n = 15$ (9 Female), mean age 26.5 ± 3.8 years; Experiment 3: $n = 18$ (10 Female), mean age 27.2 ± 5.2 years. All participants self-reported having no known neurological, orthopedic, or cognitive disorders that may affect their balance performance, perception of ankle rotations or foot sole vibrations. The University of British Columbia Clinical Research Ethics Board approved the experimental procedures. All methods were performed in accordance with the relevant guidelines and regulations at UBC, and in accordance with the Declaration of Helsinki. All participants gave informed written consent prior to participation.

2.1. Common methods for all 3 experiments:

In all experiments, participants stood under different conditions of threat manipulated by adjusting the surface height on which they stood using a hydraulic lift (M419-207B01H01D, Pentalift, Canada). In the Low threat condition, the top of the standing support surface was 1.1 m above the ground. An extension was added to the right of the participant 60 cm beyond the edge of the platform to position the participants further away from the edge of the support surface, providing a stable surface in all directions, and further reduce threat in this condition (Figure 1A; Carpenter et al., 2001). In the High threat condition, the support surface was raised to 3.2 m above the ground, with no additional support surface to the right of the participant to maximize threat effects (Figure 1B; Carpenter et al., 2001).

Emotional responses to the threat manipulation were confirmed using self-reported questionnaires, and physiological measures of arousal. Prior to each experimental condition, seated participants rated how confident they felt they could remain upright and avoid a fall on a scale from 0 (not confident at all) to 100 (completely confident). After each condition, participants provided a subjective rating of fear of falling (0 = no fear, 100 = fearful), perceived stability (0 = not stable, 100 = very stable), and state anxiety using a 16 item, 9-point Likert scale assessing elements of somatic, worry and concentrations that were summed to a total anxiety score (Hauck et al., 2008). Electrodermal activity (EDA) was collected from the thenar and hypothenar eminences of the left hand (100 Hz, Skin Conductance Module 2502, Cambridge Electronic Design, UK) as a physiological measure of sympathetic arousal (Critchley, 2002).

In all experiments, participants stood with their eyes open, and gaze fixated on a visual target located approximately 3 meters away at eye level. Feet were positioned side-by-side with a maximum distance of 40 cm. In Experiment 1 & 2, the left foot was positioned on a AMTI force plate (model OR6-7-1000, AMTI, United States), while in Experiment 3, the left foot was positioned on a custom force plate

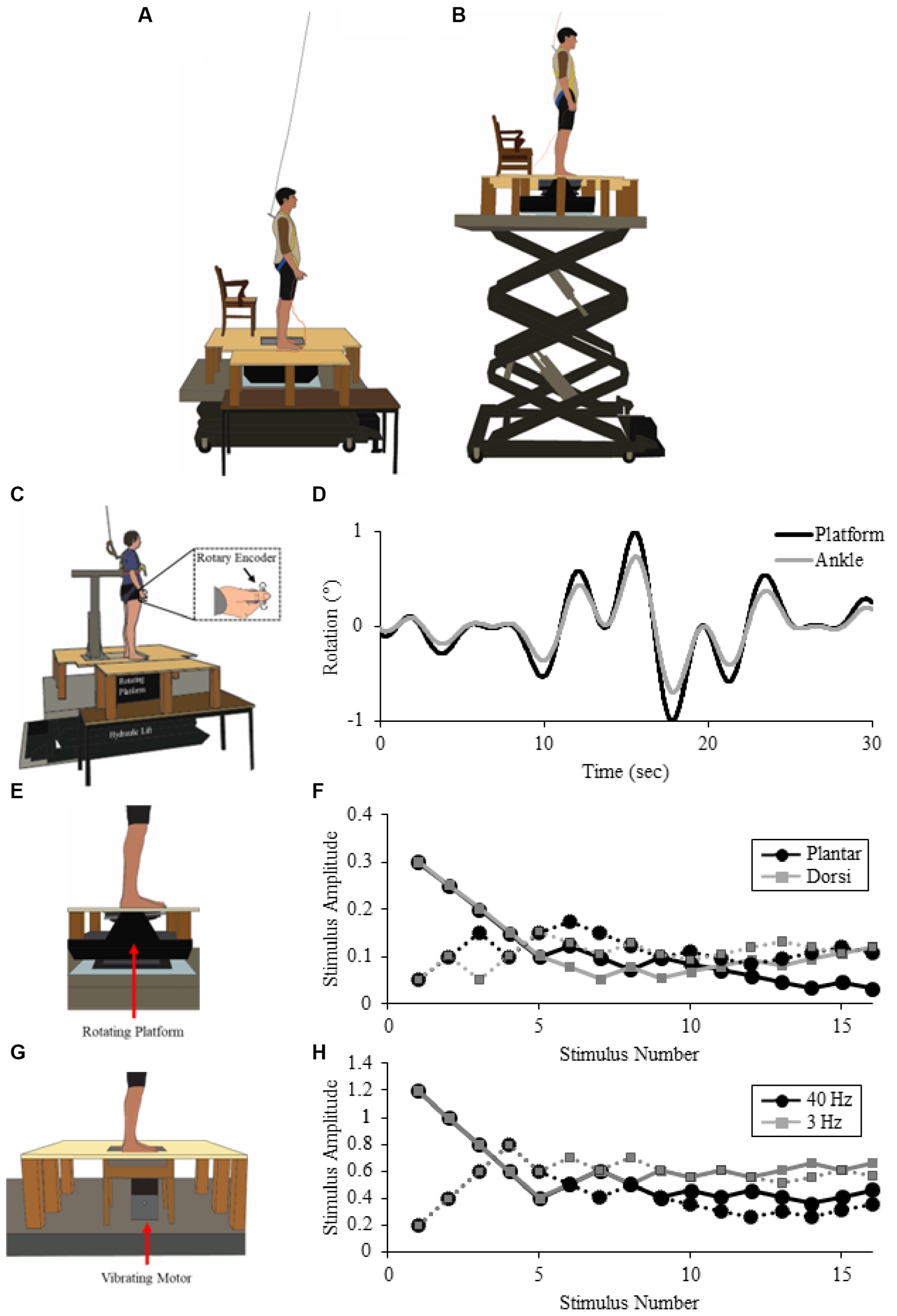

FIGURE 1
Experimental setup. Illustration of experimental setup for Low **(A)** and High **(B)** conditions (from Experiment 2). Also, illustration of experimental setup for the Low condition in Experiment 1 **(C)**, Experiment 2 **(E)** and Experiment 3 **(G)**. Participants stood on a force plate on top of a rotating platform **(C,E)**, Experiment 1 or 2, respectively or force platform with a vibrating probe protruding through the support surface [**(G)**, Experiment 3]. Representative data for Experiment 1 [**(D)**; platform (black) and Ankle (gray) displacement], Experiment 2 [**(F)**; ascending (dotted line) and descending (solid line) staircase for plantar- (black) and dorsi-flexion (gray) rotations], and Experiment 3 [**(H)**; ascending (dotted line) and descending (solid line) staircase for 40 Hz (black) and 3 Hz (gray) vibrations].

embedded with 4 vertical load cells (Experiment 3; SSB-250 with BSC4A-C14, Interface Advanced Force Measurement, United States). Vertical ground reaction forces were sampled at 100 Hz. Prior to the first experimental condition, participants stood quietly for 20 s to establish pre-stimulus baseline (neutral) measures. Baseline vertical force level was monitored throughout experimental trials and used to provide verbal feedback from the experimenter if a neutral position was deviated from by two standard deviations. For Experiment 2 and 3, feedback was delivered when necessary, only immediately after a stimulus was perceived to minimize the possibility of shifting attention away from the psychophysical task.

3D motion capture data were collected to estimate platform (Experiment 1 and 2) and lower limb position (all experiments, 250 Hz, accuracy: 0.1 mm, resolution: 0.01 mm, Optotrak, Northern

Digital Inc.). Infrared emitting diodes were placed at the front and back of the force plate's top surface, left base of the fifth metatarsal, lateral malleolus, and fibular head. Two-dimensional filtered coordinates defining the foot (toe to lateral malleolus) and shank (malleolus to fibular head) were used to calculate ankle angle.

Electromyography (EMG) was recorded using a bipolar arrangement of 2 surface electrodes placed 2 cm apart over the muscle bellies of the left soleus (SOL) and tibialis anterior (TA). EMG was band-pass filtered between 10 and 500 Hz (Telemyo, 2400R, Noraxon, United States), and sampled at 2000 Hz. Offline, EMG data was low-pass filtered at 100 Hz using a dual-pass Butterworth filter, bias corrected, and full-wave rectified. Mean background EMG activity, vertical force, and foot and ankle position were calculated offline from the entire trial (Experiment 1) or from 1 s prior to stimulus onset (Experiment 2 and 3) in each height condition.

2.2. Experiment-specific methods and procedures

2.2.1. Experiment 1

Participants stood barefoot while strapped to a rigid structure (see Figure 1C) limiting the amount of anteroposterior (AP) sway (Lackner, 2021). In addition, reducing the amount of AP sway allowed for a consistent stimulus across height conditions as postural threat can change sway amplitude and leaning (Adkin and Carpenter, 2018) which could otherwise influence the amount of ankle rotation. The force plate under the left foot was mounted to a custom-built single axis servo-controlled tilting platform; the right foot was positioned on an adjacent stable surface. The feet were positioned so that the axis of rotation of the tilting platform was aligned with the participant's ankle joints. Foot position was marked on the force plate and kept constant across trials to ensure a consistent rotation of the ankle. During experimental conditions, participants were instructed to remain upright and avoid using the brace as support during continuous AP support surface oscillations of the left ankle. During continuous support surface rotations used to induce small and large movements (pseudorandom oscillations <0.5 Hz, ± 1°, see Figure 1C and Figure 1D), participants were instructed to track accurately their ankle position in real-time using a hand-held rotary encoder (1/4″ shaft, model E14102402302, Dynapar, United States). Two weak springs, one on either side of the point of contact with the thumb were used to limit the amount of drift that could occur using the tracking device (Cleworth and Carpenter, 2016; Cleworth et al., 2019), and provided minimal feedback of the encoder's neutral (vertical) position. Participants performed a minimum of two practice trials, lasting 30 s each, to become familiar with the ankle movements and to practice using the device. Participants completed the practice trials first with eyes open and then eyes closed. The experimenter ensured proper use of the device during these trials by comparing ankle angular displacement, platform angular displacement, and tracked position (rotary encoder voltage). If ankle rotation and tracked position were congruent in amplitude and direction (visual inspection for similar patterns), and participants reported ease of use, the experiment continued (all participants correctly performed the task within two eyes open and one eyes closed condition). Participants then performed two seven-minute trials in the Low condition (to control for first trial effects, while the second trial was used in the analysis), and one in the High condition with their eyes open.

2.2.1.1. Analysis

The rotary encoder voltages and ground reaction forces and moments were collected and exported at 2000 Hz (Power 1401 with Spike2 software, CED, UK). Tracked sway was determined from the voltage of the rotary encoder and was band-pass filtered using a 0.005 Hz to 2 Hz dual-pass Butterworth filter (Cleworth et al., 2019). For ankle and tracked data, the mean position was calculated and subtracted from each respective trace to remove any bias. Due to the tracked sway data having a 'unitless' quantity, data were normalized to the Low condition and expressed as a percentage of this movement (Cleworth et al., 2019). Normalization was calculated by dividing each data point by the maximum value in the Low condition. Both the Low and High condition data were normalized to the maximum amplitude from the Low condition.

Root mean square (RMS) were calculated from platform, ankle and tracked data in the AP direction from the unbiased normalized signal to quantify the amplitude of actual and perceived movement. A quotient (QRMS) was then calculated between perceived and actual movement (tracked RMS was divided by ankle RMS) to signify the relative changes in perceived movement related to actual movement within a condition. Three participants were removed prior to analysis due to an inability to perform the task correctly (difficulty in tracking, an inability to remain static during continuous rotations, or could not complete the task).

Cross correlation analyses were performed to quantify the participants' ability to track ankle rotation. The time (lag) and amplitude of the maximum cross-correlation coefficient with a maximum lag of 1.5 s were calculated from the ankle and tracked data in the AP direction from the unbiased normalized signal for both height conditions.

2.2.2. Experiment 2

Similar to Experiment 1, participants stood barefoot with the left foot on a force plate mounted to a rotating motorized platform, right foot on an adjacent stable surface, and both ankle joints aligned with the platform's axis of rotation (Figure 1E). While standing in the two postural threat conditions, participants performed an ankle rotation discrimination task. The left ankle was rotated in the pitch plane in a dorsi-flexion or plantar-flexion direction. Platform rotation speed was kept constant at 0.25°/s while amplitude was varied. Potentiometer-based feedback from the platform and the signal supplying the motor was recorded (sampled at 2000 Hz, Power 1401 with Spike2 software, CED, UK). Participants were asked to indicate when they felt either an ankle rotation by pushing a right hand-held bidirectional toggle switch either up for dorsi-flexion or down for plantar-flexion. The correct detection of an ankle rotation was only accepted if the switch was correctly pushed within 2 s after platform movement offset. To reduce the likelihood of false-positive responses, participants were specifically instructed to indicate a movement direction only when they were sure they had felt a movement, and to not respond if no movement or an unidentifiable movement was perceived.

Prior to any experimental conditions, participants completed two practice trials to become familiar with the experimental procedures, and to remove any first trial effects. At Low height, participants were asked to identify which of two large amplitude ankle rotations they felt (using the toggle switch) followed by a verbal report. A minimum of five randomly ordered stimuli were administered using a constant amplitude (all presumed suprathreshold; 1° rotation). This trial was used to ensure proper use of experimental equipment, and that each participant could detect large amplitude stimuli. The second practice trial consisted of 16 stimuli of each direction, randomly presented to ensure participants could sufficiently perform the task.

When assessing the sensitivity within the proprioceptive system, there are a number of methods that can be used to determine a threshold, including method of limits, constant stimuli, and adaptive staircase methods. Due to the time limitations for maintaining emotional changes with height, an adaptive staircase method was selected because of its relatively short duration (5 min) compared to approximately 20 min needed for a method of limits approach (Berquin et al., 2010). Between stimuli, the peak-to-peak amplitude of the voltage command was adjusted using an adapted staircase method (4–2-1-step, modified from Dyck et al., 1993). Each staircase starts with 0.1 V (approximately 0.2° which is above thresholds previously determined, Fitzpatrick and McCloskey, 1994) steps in stimulus intensity. Steps were halved to 0.05 V after four steps. Stimuli were halved again to 0.025 V after another four steps and kept constant for seven steps. This method allowed for stimuli to remain within the motion capture systems ability to detect stimuli amplitudes, and provided more efficient (fewer trials) thresholds in pilot data than the reduction in step size based on reversals as previously used (Dyck et al., 1993; Peters et al., 2016, where a reversal point, when the participant goes from responding "yes" to responding "no," or vice versa, reduced the step size by half until the next reversal point, reducing by half again until all trials are given). To accommodate the discrimination technique, two staircases were interlaced, one for each of the two stimuli (Figure 1F). Furthermore, to accommodate an ascending and descending staircase method (typically seen in a method of limits technique), a new staircase was implemented halfway through each experimental condition. Staircase-direction was counterbalanced across participants such that stimulus 17 for each of the movement directions was set at a low or high amplitude depending on whether the start of the experiment started with a high or low amplitude, respectively. The 4–2-1-step algorithm was then reset and continued until the end of the block of trials. Stimulus-direction was randomly presented to avoid any anticipation bias.

2.2.2.1. Analysis

To compute ankle rotation discrimination thresholds, data were reanalyzed offline using the peak displacement of the foot calculated from motion capture (see limitations). In accordance with previous work (Peters et al., 2016), this method accounted for trial-to-trial variability and inter-individual differences in biomechanical properties of the ankle and/or foot. Thresholds were then calculated as the mean of the smallest step-amplitude reversal points. If only one reversal point for a given direction was observed in the smallest step-amplitudes, the last two stimuli were averaged and used to calculate a mean. False detection rates were calculated from all 2 s periods where no platform movement was given within a trial and reported as a percentage of the number of times a stimulus was perceived during these periods (number of blank stimuli detected divided by total number of 2 s periods with no stimulus). Participants were removed from further analysis if a false detection rate in the Low condition exceeded 20% (Berquin et al., 2010). As a result, one participant was removed due to large false detection rates. One additional outlier was removed due to higher than normal (two times higher than any other participant) thresholds within the Low condition.

2.2.3. Experiment 3

A 6 mm probe protruded through a 7 mm opening in the customized force plate and contacted the left foot sole (Figure 1G). The left foot was positioned to align to the probe with a location approximately 80% of maximum width from the lateral border near the ball of the foot, and 80% of maximum length from the tip of the big toe to the back of the heel. If the probe aligned with the space between the metatarsal and phalange, where there is little to no skin contact with the support surface, the foot was moved anteriorly until sufficient force (approximately 2 N) on the probe was obtained (less than 5 mm). Corresponding with previous studies (Peters et al., 2016), this location corresponded to the skin over the anterior aspect of the metatarsal head, which has been reported to be more tightly coupled to balance relative to more posterior areas of skin on the sole of the foot in the elderly (Cruz-Almeida et al., 2014). Foot position was marked on the force plate and kept constant across trials to ensure a consistent contact force of the probe onto the skin surface (Table 1). The probe was attached to a linear motor (model MT-160; Labworks) in series with a force transducer (model 31; Honeywell). An accelerometer (model 2220–010; X Tronics) was also secured to the back of the motor piston. Acceleration and force from the single force transducer were differentially amplified (×1 and ×100, respectively) and online low-pass filtered at 600 Hz (Brownlee model 440; AutoMate Scientific), and sampled at 5 kHz (Power 1401 with Spike2 software, CED, UK).

During upright stance, vibrations were applied at random intervals (3 to 5 s) perpendicular to the skin. Vibration stimuli were applied for 1 s in duration. Participants were asked to indicate when they felt either a 3 Hz vibration by pushing a button in one hand, or a 40 Hz vibration by pushing a button the other hand (participants were able to choose which hand held the 3 and 40 Hz buttons, and this orientation was kept constant across height conditions). The correct detection of a vibration was only accepted if the button was pushed between vibration onset and 1 s after vibration offset (2 s total). To reduce the likelihood of false-positive responses, participants were specifically instructed to push a button (indicating 3 Hz or 40 Hz felt) only when they were sure they had felt the given vibration, and to not respond if no vibration or unidentifiable vibration was felt.

Prior to any experimental conditions, participants completed two practice trials to familiarize with the experimental procedures, and to remove any first trial effects. At Low height, participants were asked to identify which of two foot sole vibrations they felt (using the correct input device for each stimulus) followed by a verbal report. A minimum of five randomly ordered stimuli were administered using a constant amplitude (all presumed suprathreshold; 3 Hz >1 N and 40 Hz >0.5 N). This trial was used to ensure proper use of experimental equipment, and that each participant could detect large amplitude stimuli. The second practice trial consisted of 16 stimuli of each frequency, randomly presented to ensure participants could sufficiently perform the task.

Similar to Experiment 2, during the experimental trials, the peak-to-peak amplitude of the voltage command was adjusted from stimulus-to-stimulus using an adapted staircase method (4–2-1-step, modified from Dyck et al., 1993), and staircases (Figure 1H) for both

TABLE 1 Summary of statistical test results for emotional state and baseline results.

	Experiment 1				Experiment 2				Experiment 3			
	Low x̄ SD	High x̄ SD	t(11) p	d	Low x̄ SD	High x̄ SD	t(12) p	d	Low x̄ SD	High x̄ SD	t(16) p	*d*
Emotional state measures												
Balance confidence	**96.36** **6.74**	**80.91** **20.83**	**2.974** **0.013**	**0.86**	**99.23** **2.77**	**80.23** **19.77**	**3.672** **0.003**	**1.02**	**95.56** **7.05**	**80.56** **19.62**	**3.112** **0.007**	**0.75**
Stability	**90.45** **11.30**	**66.64** **30.06**	**2.965** **0.013**	**0.86**	**88.31** **12.89**	**75.38** **20.86**	**2.403** **0.033**	**0.66**	**90.83** **11.01**	**71.94** **21.22**	**4.261** **0.001**	**1.03**
Fear	**3.45** **5.13**	**21.00** **21.29**	**3.060** **0.011**	**0.88**	**1.62** **3.73**	**22.31** **17.27**	**4.341** **0.001**	**1.20**	**4.72** **7.57**	**29.17** **24.15**	**5.088** **<0.001**	**1.23**
Anxiety	**33.91** **15.24**	**47.36** **21.01**	**3.001** **0.012**	**0.87**	**24.23** **10.61**	**38.46** **21.92**	**2.710** **0.019**	**0.75**	**24.39** **6.27**	**42.83** **17.78**	**5.270** **<0.001**	**1.28**
EDA	**11.85** **7.93**	**14.89** **7.73**	**3.604** **0.004**	**1.04**	**20.70** **4.21**	**23.82** **4.20**	**3.420** **0.005**	**0.95**	**19.84** **10.73**	**27.09** **15.47**	**2.959** **0.009**	**0.72**
Baseline measures												
Vertical force	**1.31** **0.28**	**1.37** **0.29**	**2.541** **0.028**	**0.73**	**1.10** **0.41**	**1.14** **0.43**	**3.467** **0.004**	**0.99**	**2.63** **0.55**	**2.72** **0.58**	**3.722** **0.002**	**0.90**
SOL BGD	7.8 4.1	8.1 4.4	1.54 0.152	0.44	6.7 4.2	7.1 4.6	0.638 0.535	0.18	8.7 3.3	8.8 3.0	0.233 0.818	0.06
TA BGD	**2.6** **2.7**	**2.7** **2.8**	**2.237** **0.047**	**0.65**	1.3 1.0	1.7 1.4	1.141 0.275	0.32	1.5 1.5	5.0 8.9	1.863 0.081	0.45
Pre-stim foot POS					0.02 0.07	0.02 0.32	0.389 0.704	0.11	<0.001 0.014	0.002 0.005	0.511 0.617	0.12
Pre-stim ankle angle					100.9 34.54	100.6 34.45	0.965 0.352	0.27	62.74 2.83	63.12 2.79	1.705 0.108	0.41
Pre-stim probe force									5.35 0.96	5.41 1.04	0.833 0.417	0.20

Significant effects are in bold. Gray filled areas indicate variables not measured for the experiment.
x̄ = mean; SD = standard deviation; t(#) = t statistic with degrees of freedom; *p* = *p* value; *d* = Cohen's d; EDA = electrodermal activity; SOL = Soleus; BGD = background TA = tibialis anterior; POS = position; pre-stim = pre-stimulus.

frequencies were interlaced. Descending staircases were used prior to ascending for all participants to ensure suprathreshold stimuli were used first. Stimulus frequency was randomly presented to avoid any anticipation bias.

2.2.3.1. Analysis

A similar method to Experiment 2 was used to compute discrimination thresholds for foot sole vibrotactile sensation using peak-to-peak force amplitude from the force transducer. No participants were removed due to large false detection rates, but one outlier was removed due to higher than normal (two times higher than any other participant) thresholds within the Low condition.

2.3. Statistical analysis

Paired sample t-tests were used to examine the effects of threat on EDA and self-reported measures of fear, anxiety, confidence and stability. In Experiment 1, paired sample *t*-tests were also used to compare RMS quotient and cross-correlation measures of actual and perceived movement. In cases where data were not normally distributed as determined by the Shapiro Wilks test, a non-parametric Wilcoxon Signed Ranks Test was used to compare height effects. In Experiment 2 and 3, a 2 (height) x 2 (stimulus characteristic) x 2 (staircase-direction) repeated measures ANOVA was used to test the effects of height (Low, High), stimulus characteristic (Experiment 2: dorsi-flexion and plantar-flexion; Experiment 3: 3 Hz and 40 Hz), and staircase-direction (ascending, descending) for calculated thresholds. If a significant stimulus characteristic effect was observed, separate 2 (height) by 2 (staircase-direction) repeated measures ANOVA's were used to examine each stimulus characteristic independently. All dependent measures were analyzed separately (similar to previous research; Cleworth et al., 2019). The criteria for a significant result was set at $p \leq 0.05$ with trends identified when $p \leq 0.1$, and effect sizes reported using Cohen's d for t-tests and partial eta squared (n_p^2) for ANOVAs.

3. Results

3.1. Effect of height

3.1.1. Emotional state

Postural threat had a significant effect on all psychological variables and EDA that was consistent across all three experiments

(Table 1). Balance confidence and perceived stability decreased, while fear, anxiety and EDA increased when standing in the High compared to Low threat condition (Table 1).

3.1.2. Baseline measures

There was a significant increase in vertical force at height under the stimulated (left) foot in all three experiments (< 5%, Table 1). In contrast, there was no evidence in either experiment of any effect of height on the background activity for SOL (Table 1). Similarly, there was no effect of height on the background activity for TA in Experiment 2; however, there was a significant increase at height in Experiment 1 and a non-significant trend for larger TA activity at height in Experiment 3 (Table 1). There was no effect of height on the pre-stimulus location of the foot segment or ankle angle in Experiment 2 and 3, or pre-stimulus probe force in Experiment 3 (Table 1; note, no pre-stimulus positions were calculated for Experiment 1 due to continuous perturbations used).

3.1.3. Effect of height on tracking (Experiment 1)

Participants were accurate in tracking their ankle movements for both Low and High threat conditions (mean r ± SD: 0.52 ± 0.15 Low and 0.51 ± 0.14 High), which were tightly coupled to the rotations of the support surface (Figure 2A). Cross-correlations revealed no change in r ($t(10) = 0.248$, $p = 0.809$, $d = 0.14$) or lag (0.136 s ± 0.125 s Low and 0.183 s ± 0.148 s High; $t(10) = 1.868$, $p = 0.091$, $d = 0.083$) across height conditions. Similar patterns, changes in amplitude and frequencies were observed between the platform displacement, foot angular displacement and tracked movement across participants (Figure 2A).

QRMS was significantly influenced by height; QRMS increased in the High (1.36 ± 0.38) compared to Low (1.04 ± 0.24) condition, indicating more movement was perceived at height ($t(10) = 2.543$, $p = 0.029$, $d = 0.77$; Figure 2C). There were no differences in platform RMS across height conditions (Figure 2B).

3.1.4. Effect of height on discriminatory thresholds (Experiment 2 and 3)

In Experiment 2, there was a significant effect of height on perceptual thresholds calculated for ankle rotations (Figure 3C; $F(1,12) = 7.285$, $p = 0.018$, $n_p^2 = 0.38$). Specifically, higher perceptual thresholds across stimulus and staircase-direction were observed in the High (0.103° ± 0.041°) compared to Low condition (0.081° ± 0.031°). Nine of thirteen participants had an average increase in threshold amplitude across stimulus-direction by staircase-direction conditions. Twelve of thirteen participants had a higher threshold at height within at least two of the four stimulus-direction by staircase-direction conditions. There were no significant stimulus-direction or staircase-direction by threat interactions, or three-way interaction effects for ankle rotation perceptual thresholds. Detection thresholds for ankle rotations were on average 0.09° ± 0.03°, similar to previous work when taking into account the velocity used in the current study (Fitzpatrick and McCloskey, 1994).

In Experiment 3, there were no significant height effects on the thresholds calculated for 3 Hz ($F(1,16) = 1.369$, $p = 0.259$, $n_p^2 = 0.08$) or 40 Hz ($F(1,16) = 0.137$ $p = 0.716$, $n_p^2 < 0.01$) foot sole vibrations (Figure 3F). There were no significant staircase-direction by threat interactions for foot sole vibrations.

3.2. Effect of staircase-direction and stimulus in Experiment 2 and 3

In Experiment 2, a negative hysteresis effect was visually observed for half the trials, where the first reversal point in ascending data was smaller than the first reversal point in descending data. Participants could perceive platform-triggered ankle rotations as small as 0.03°, on average (averages ranged from 0.03° to 0.17° for ankle dorsi-flexion and plantar-flexion across heights, Figure 3A, while individual thresholds ranged from 0.024° to 0.368°). There was a significant effect of staircase-direction ($F(1,12) = 9.289$, $p = 0.01$, $n_p^2 = 0.44$), where ascending stimuli (0.079° ± 0.03°) were significantly smaller than descending stimuli (0.105° ± 0.04°) further supporting a negative hysteresis effect (Figure 3B). The counterbalance of ascending or descending stimuli resulted in no differences between first block and second block of staircase delivery ($F(1,12) = 0.042$, $p = 0.840$, $n_p^2 < 0.01$). There was no significant difference ($F(1,12) = 3.902$, $p = 0.07$, $n_p^2 = 0.25$; Figure 3A) in amplitude of detection thresholds for dorsi-flexion (0.079° ± 0.03°) compared to plantar-flexion (0.105° ± 0.05°, Figure 3A).

In Experiment 3, a perceptual hysteresis effect was visually observed, where the first reversal point in ascending data was larger than the first reversal point in descending data. There was no main effect of staircase-direction (3 Hz: $F(1,16) = 1.378$, $p = 0.258$, $n_p^2 = 0.08$; 40 Hz: $F(1,16) = 0.155$, $p = 0.699$, $n_p^2 < 0.01$) nor were there any interactions (Figure 3E). There were significant differences between the 3 Hz and 40 Hz stimuli ($F(1,16) = 62.986$, $p < 0.001$, $n_p^2 = 0.80$) with smaller amplitude vibrations perceived for the 40 Hz compared to the 3 Hz vibration (Figure 3D).

4. Discussion

The aim of this study was to determine the effect of postural threat on conscious perceptions and detection thresholds for somatosensory stimuli of the lower leg during standing. When young healthy adults stood at the edge of an elevated support surface, the amplitude of tracked ankle rotation was significantly increased (Experiment 1), despite similar ankle movements. Height-induced postural threat increased perceived ankle movement by approximately 1.3 times. This observation corroborates with prior reports of increased perception of whole-body sway in static and dynamics task compared to actual movements (Cleworth and Carpenter, 2016; Cleworth et al., 2018, 2019) and suggests sensory information from the ankle joint could contribute to threat-related changes in single-joint and whole-body movement perceptions.

To determine if the threat-related changes in perceived ankle rotation were due to changes in perceptual thresholds, we examined just-noticeable differences at the level of detection in passive ankle rotation in Experiment 2. Contrary to our hypotheses, the ability to perceive and discriminate ankle rotation direction was significantly reduced with threat, where increased perceptual thresholds were observed in the High compared to Low height condition.

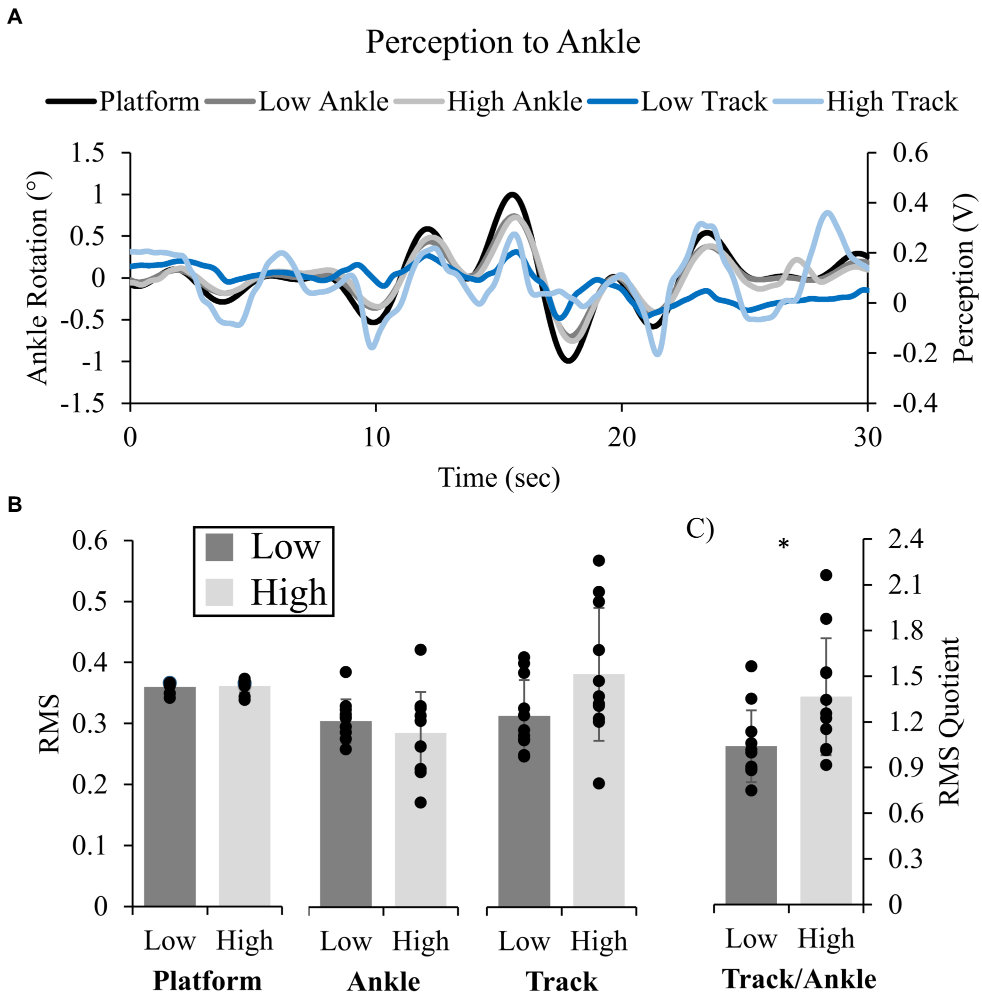

FIGURE 2
Representative traces and group mean data for Experiment 1. Platform (black), Ankle displacement (gray), and tracked displacement (blue) for Low (darker shades) and High (lighter shades) conditions **(A)**. Group mean (standard deviation) for amplitude of platform, ankle, and tracked (Track) displacements **(B)**, and calculated quotients (QRMS) between perceived and ankle motion [**(C)**; Low: black, High: gray]. Statistics were performed on platform RMS and RMS quotient only. * indicates a significant difference ($p < 0.05$).

Since perception of ankle rotations likely rely on muscle spindles and cutaneous receptors, we sought to isolate cutaneous threshold detection in Experiment 3. However, contrary to our hypotheses, detection thresholds for foot sole vibrations did not significantly change between Low and High height (Figure 3F). These observations are consistent with prior observations of unchanged cutaneous reflexes when standing at height (Horslen, 2016).

In the absence of evidence for decreased perceptual thresholds, the observed threat-related changes in perceptual gain during whole-body movements (Cleworth and Carpenter, 2016; Cleworth et al., 2018, 2019) and single limb rotations (Experiment 1) are likely mediated by changes in sensory response gain (Horak and Diener, 1994; Lim et al., 2014) in proprioceptive pathways, either at the spinal and/or supraspinal level. With postural threat, there is strong evidence of increased muscle spindle sensitivity, based on augmented amplitude and velocity-scaling responses to rapid ankle stretch (Horslen et al., 2018), and constant (Horslen et al., 2013) or decreased Hoffman reflexes (Sibley et al., 2007). Threat-related changes have also been observed for 1b reflexes which originate from Golgi tendon organs and have largely inhibitory effects on anti-gravity muscle activity (Horslen et al., 2017), although their role in controlling static balance is less clear. Although there is the potential for cutaneous reflexes to also be amplified by arousal during gait when modulation of cutaneous input may be critical (Zaback et al., 2018), there is less evidence for cutaneous reflex gain during stance (Experiment 3 and Horslen, 2016) when its role may be less crucial. Comparing foot sole vibrations in this study to previous work is difficult given differences in probe diameter, stimulus location, postural orientation and dependent variables used (displacement or force); however, the observations of lower detection thresholds for 40 Hz (0.154 N) vibrations compared to 3 Hz (0.395 N) vibrations matches previous reports (Strzalkowski et al., 2015).

The increased gain in movement perception observed in Experiment 1 and previous studies (Cleworth and Carpenter, 2016; Cleworth et al., 2018, 2019) may also be related to a change in sensory gating, where threat increases the amount of somatosensory

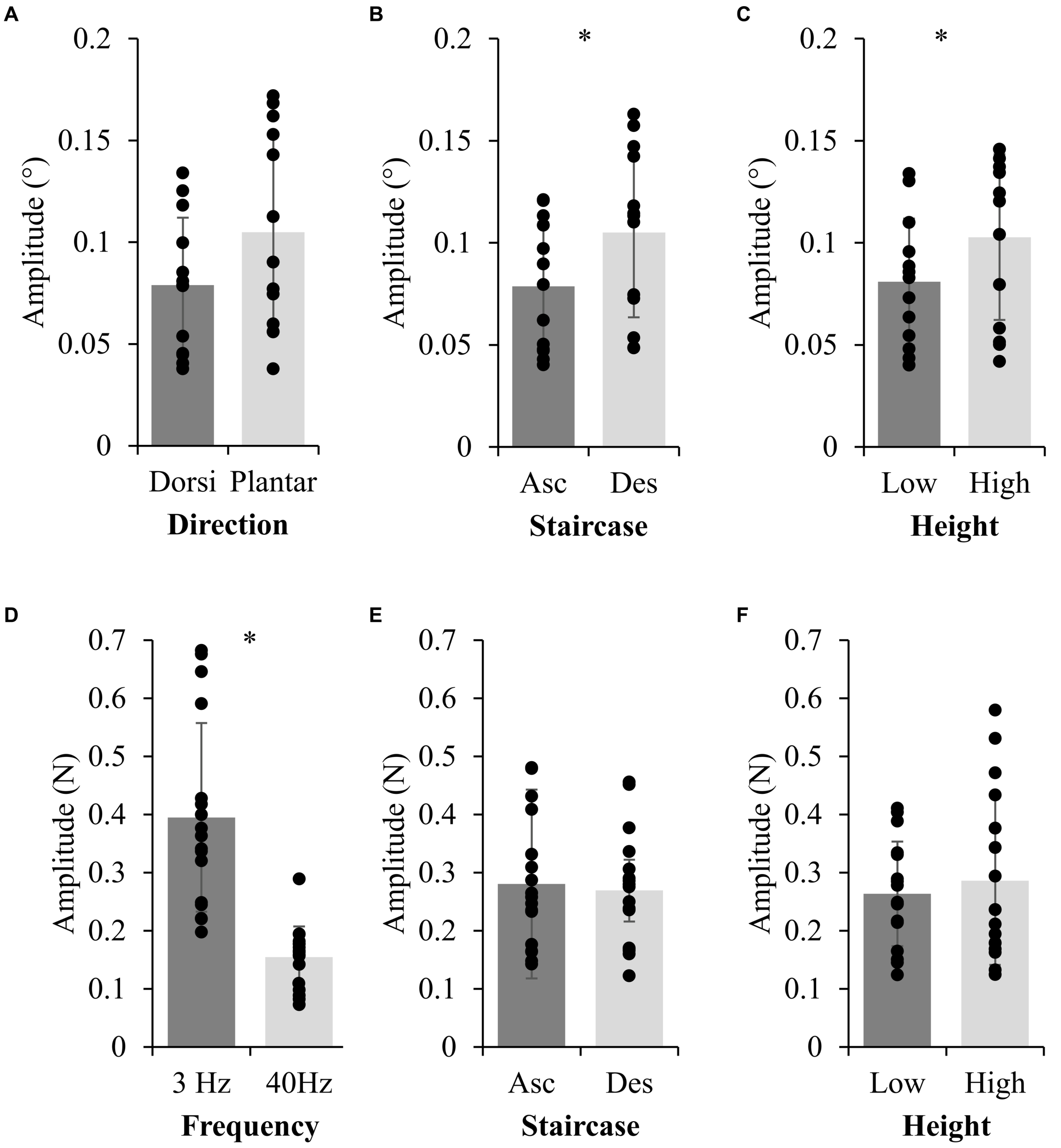

FIGURE 3
Group data for main effects on ankle rotation and foot-sole vibration thresholds. Group mean (standard deviation) from Experiment 2 (top row) and 3 (bottom row) for stimulus effects (left column), staircase direction (middle column; Asc=Ascending; Des=Descending), and height conditions (right column). * indicates a significant difference ($p < 0.05$).

information for large amplitude movements. Cortical areas receiving information pertaining to somatosensory stimuli can be modulated across tasks and by threatening conditions (Staines et al., 1997; McIlroy et al., 2003; Davis et al., 2011). The amount and direction of modulation has been shown to be reliant on the somatosensory information required for the task (Staines et al., 1997). Sensory gating occurs during passive movement (Staines et al., 1997) where faster movements increase gating (Rauch et al., 1985). Sensory gating also occurs during balance tasks (McIlroy et al., 2003) and when standing quietly in a threatening condition (Davis et al., 2011). Facilitation of initial cortical activity occur with kinaesthetic task demands (Staines et al., 1997), and with responses to destabilizing perturbations when balancing in threatening conditions (McIlroy et al., 2003; Adkin et al., 2008; Sibley et al., 2010), while later cortical processing is affected by height-induced threat (Horslen, 2016).

Selective attention is another possible mechanism for a threat-related change in perceived movement amplitude. Allocation of attentional resources to secondary tasks can directly affect performance during tactile (Lloyd et al., 1999), auditory (Haykin and Chen, 2005), visual (Verghese, 2001) and ankle-related perceptual tasks (Yasuda et al., 2014). Previous reports have indicated changes in attention can occur with height-induced threat. During static and anticipatory postural control tasks, height-induced threat increases attention toward movement-related processes, threat-related stimuli, and self-regulatory strategies while decreasing attention toward task-irrelevant information (Zaback et al., 2016). Threat-related changes in attention have also been linked to an increased conscious control and monitoring of movement (Huffman et al., 2009). These attentional changes were linked to a focus of attention during postural tasks independent of secondary tasks. Therefore, when threatened, attentional resources may be redirected to the movement perception task increasing the response gain and modifying the amplitude of perceived movements.

Based on signal detection theory (Green and Swets, 1989), the ability to detect ankle rotations or foot sole vibrations depends on the stimulus signal exceeding the current level of noise within the respective sensory modality. Since the threshold amplitude of perceived ankle rotations from the rotating platform (maximum across participants is 0.36°) were well within the ankle angular displacement from quiet standing postural tasks (1–1.5°, Gatev et al., 1999; Gage et al., 2004), height-related increases in ankle thresholds could be due to increased sensory noise created by increases in frequency of sway (Carpenter et al., 2001; Davis et al., 2009; Cleworth et al., 2012) and increased activity of proprioceptive receptors, such as muscle spindles (Horslen et al., 2013, 2018). Further evidence can be drawn from the increased false detection rates observed in Experiment 2, which has been shown to vary with the level of noise within the system (Ferrè et al., 2016).

Alternatively, the observed increase in proprioceptive detection thresholds could be related to changes in cortical involvement. Threat has the potential to influence synchronous spiking activity within and across specific cortical regions which has been associated with increased performance in consciously detecting stimuli (Melloni et al., 2007). Gamma band (40–80 Hz) oscillations are associated with attending to relevant stimuli, while alpha band (8–14 Hz) oscillations are related to the suppression of distracting stimuli (Foxe and Snyder, 2011). However, recent evidence has shown that increased anxiety increased levels of alpha band EEG (Knyazev et al., 2004) mediated through changes in movement reinvestment (Ellmers et al., 2016), and a shift from predominantly beta band to gamma band frequencies when standing under postural threat (Zaback et al., 2022). Together these changes would predict a decrease in detection thresholds (Garcia-Garcia et al., 2010), in contrast to the increased or unchanged thresholds observed in Experiment 2 and 3, respectively. However, the potential effect of attention cannot be overlooked, as simultaneous performance of a postural task (remain upright) and a perceptual task with similar stimuli to the balance task (ankle rotation) could contribute to elevated perceptual thresholds (Experiment 2).

Changes in muscle activity may also potentially contribute to altered movement-related thresholds (Taylor and McCloskey, 1992; Peters et al., 2017). There was an increase in TA activity in Experiment 1 as reported previously in studies where subjects stood facing the edge of the platform (Carpenter et al., 2001; Zaback et al., 2021). However, there were no significant changes in muscle activity observed in Experiment 2 and 3, likely due to the fact the subject was oriented perpendicular to the platform edge to control for potential confounding effects of leaning Naranjo et al., 2016).

4.1. Limitations

Due to the use of uncalibrated units for the tracking data, the accuracy of the amplitude of perceived movements with respect to actual movements for a given participant is difficult to identify in the current study, i.e., participants may have been underestimating ankle rotation when in the Low condition. However, given a within-subject design was used, it is clear there was a change in amplitude of perceived movement in Experiment 1, which supports previous work, indicating postural threat amplifies the perceived movement associated with ankle rotations and whole-body motion.

A change in vertical force or an increase in SOL and TA co-contraction at height may influence the detection of ankle rotations and foot sole vibrations. Vertical force on the stimulated leg did increase with height; however, this relatively small (< 5%) and potentially functionally irrelevant change is unlikely to explain the observed results, given that previous reports have illustrated no change in ankle angular displacement thresholds (Refshauge and Fitzpatrick, 1995) or foot sole vibrations (especially with 3 Hz and 40 Hz vibrations, Germano et al., 2016; Mildren et al., 2016) when standing (full body weight, large vertical force acting on foot sole) compared to lying (no vertical force acting on foot sole).

Hysteresis, judgment uncertainty, and task specificity may explain some of the results of the current study. Judgment uncertainty, which arises when participants cannot clearly separate two perceptual alternatives (Hock and Schöner, 2010), may explain the 'negative hysteresis' (Lopresti-Goodman et al., 2013) observed in Experiment 2. During a postural task, it may be difficult to perceive a passive ankle rotation, but not an externally produced foot sole vibration. Therefore, the negative hysteresis observed in Experiment 2 is likely mediated by uncertainty between dorsi– and plantar-flexion rotations intermixed with natural sway movements.

Finally, while upright quiet stance typically involves bilateral ankle rotations, unilateral support surface rotations were used to reduce the likelihood of evoking balance perturbations or corrective responses (Corna et al., 1996; Horslen et al., 2018), which would potentially confound the ankle somatosensory perceptual task. In addition, given these results are in line with previous work using whole body movements (Cleworth and Carpenter, 2016; Cleworth et al., 2018, 2019), these results are functionally relevant to postural control and would suggest similar results when using single vs. bilateral stimuli. However, further work is needed to address this effect.

4.2. Conclusion

In conclusion, perceived ankle rotation increased in amplitude, detectable thresholds for ankle rotations also increased, whereas thresholds for foot sole vibrations remained unaffected during threatening conditions. Changes in sensory receptors, afferent inflow and cortical activity are possible mechanisms to explain these results. Since perceptual thresholds in balance-relevant somatosensory systems remain unchanged or increase with postural threat, the height-related changes in perceptual gain of whole-body movements are likely attributed to stimulus gain of other sensory systems not tested in this study (e.g., vestibular or visual), or increases in response gain (increased response strength proportional to stimulus intensity; Horak and Diener, 1994; Lim et al., 2014) of all, or select balance-related sensory stimuli.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving human participants were reviewed and approved by University of British Columbia Clinical Research Ethics Board. The patients/participants provided their written informed consent to participate in this study.

Author contributions

TC and MC contributed to conception and design of the study. TC collected and analyzed the data and wrote the first draft of the manuscript. TC and MC performed the statistical analysis. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

References

Adkin, A. L., Campbell, A. D., Chua, R., and Carpenter, M. G. (2008). The influence of postural threat on the cortical response to unpredictable and predictable postural perturbations. *Neurosci. Lett.* 435, 120–125. doi: 10.1016/j.neulet.2008.02.018

Adkin, A. L., and Carpenter, M. G. (2018). New insights on emotional contributions to human postural control. *Front. Neurol.* 9:789. doi: 10.3389/fneur.2018.00789

Asutay, E., and Västfjäll, D. (2012). Perception of loudness is influenced by emotion. *PLoS One* 7:e38660. doi: 10.1371/journal.pone.0038660

Berquin, A. D., Lijesevic, V., Blond, S., and Plaghki, L. (2010). An adaptive procedure for routine measurement of light-touch sensitivity threshold. *Muscle Nerve* 42, 328–338. doi: 10.1002/mus.21689

Borsky, P. N. (1979). Sociopsychological factors affecting the human response to noise exposure. *Otolaryngol. Clin.* 12, 521–535. doi: 10.1016/S0030-6665(20)32441-5

Carpenter, M. G., Frank, J. S., Silcher, C. P., and Peysar, G. W. (2001). The influence of postural threat on the control of upright stance. *Exp. Brain Res.* 138, 210–218. doi: 10.1007/s002210100681

Clerkin, E. M., Cody, M. W., Stefanucci, J. K., Proffitt, D. R., and Teachman, B. A. (2009). Imagery and fear influence height perception. *J. Anxiety Disord.* 23, 381–386. doi: 10.1016/j.janxdis.2008.12.002

Cleworth, T. W., Adkin, A. L., Allum, J. H. J., Inglis, J. T., Chua, R., and Carpenter, M. G. (2019). Postural threat modulates perceptions of balance-related movement during support surface rotations. *Neurosci.* 404, 413–422. doi: 10.1016/j.neuroscience.2019.02.011

Cleworth, T. W., and Carpenter, M. G. (2016). Postural threat influences conscious perception of postural sway. *Neurosci. Lett.* 620, 127–131. doi: 10.1016/j.neulet.2016.03.032

Cleworth, T. W., Horslen, B. C., and Carpenter, M. G. (2012). Influence of real and virtual heights on standing balance. *Gait Posture* 36, 172–176. doi: 10.1016/j.gaitpost.2012.02.010

Cleworth, T. W., Inglis, J. T., and Carpenter, M. G. (2018). Postural threat influences the conscious perception of body position during voluntary leaning. *Gait Posture* 66, 21–25. doi: 10.1016/j.gaitpost.2018.08.003

Corna, S., Galante, M., Grasso, M., Nardone, A., and Schieppati, M. (1996). Unilateral displacement of lower limb evokes bilateral EMG responses in leg and foot muscles in standing humans. *Exp. Brain Res.* 109, 83–91.

Critchley, H. D. (2002). Review: Electrodermal responses: what happens in the brain. *Neuroscientist* 8, 132–142. doi: 10.1177/107385840200800209

Cruz-Almeida, Y., Black, M. L., Christou, E. A., and Clark, D. J. (2014). Site-specific differences in the association between plantar tactile perception and mobility function in older adults. *Front. Aging Neurosci.* 6, 68.

Dannenbaum, R. M., and Jones, L. A. (1993). The assessment and treatment of patients who have sensory loss following cortical lesions. *J. Hand Ther.* 6, 130–138. doi: 10.1016/S0894-1130(12)80294-8

Davis, J. R., Campbell, A. D., Adkin, A. L., and Carpenter, M. G. (2009). The relationship between fear of falling and human postural control. *Gait Posture* 29, 275–279. doi: 10.1016/j.gaitpost.2008.09.006

Davis, J. R., Horslen, B. C., Nishikawa, K., Fukushima, K., Chua, R., Inglis, J. T., et al. (2011). Human proprioceptive adaptations during states of height-induced fear and anxiety. *J. Neurophysiol.* 106, 3082–3090. doi: 10.1152/jn.01030.2010

Dyck, P. J., O'Brien, P. C., Kosanke, J. L., Gillen, D. A., and Karnes, J. L. (1993). A 4, 2, and 1 stepping algorithm for quick and accurate estimation of cutaneous sensation threshold. *Neurology* 43, 1508–1512. doi: 10.1212/WNL.43.8.1508

Ellmers, T. J., Machado, G., Wong, T. W. L., Zhu, F., Williams, A. M., and Young, W. R. (2016). A validation of neural co-activation as a measure of attentional focus in a postural task. *Gait Posture* 50, 229–231. doi: 10.1016/j.gaitpost.2016.09.001

Ferrè, E. R., Sahani, M., and Haggard, P. (2016). Subliminal stimulation and somatosensory signal detection. *Acta Psychol.* 170, 103–111. doi: 10.1016/j.actpsy.2016.06.009

Fitzpatrick, R., and McCloskey, D. I. (1994). Proprioceptive, visual and vestibular thresholds for the perception of sway during standing in humans. *J. Physiol.* 478, 173–186. doi: 10.1113/jphysiol.1994.sp020240

Foxe, J. J., and Snyder, A. C. (2011). The role of alpha-band brain oscillations as a sensory suppression mechanism during selective attention. *Front. Psychol.* 2:154. doi: 10.3389/fpsyg.2011.00

Gage, W. H., Winter, D. A., Frank, J. S., and Adkin, A. L. (2004). Kinematic and kinetic validity of the inverted pendulum model in quiet standing. *Gait Posture* 19, 124–132. doi: 10.1016/S0966-6362(03)00037-7

Gagnon, K. T., Guess, M. N., and Stefanucci, J. K. (2013). Fear influences perceived reaching to targets in audition, but not vision. *Evo. Hum. Behav.* 34, 49–54. doi: 10.1016/j.evolhumbehav.2012.09.002

Garcia-Garcia, M., Yordanova, J., Kolev, V., Domínguez-Borràs, J., and Escera, C. (2010). Tuning the brain for novelty detection under emotional threat: the role of increasing gamma phase-synchronization. *NeuroImage* 49, 1038–1044. doi: 10.1016/j.neuroimage.2009.07.059

Gatev, P., Thomas, S., Kepple, T., and Hallett, M. (1999). Feedforward ankle strategy of balance during quiet stance in adults. *J. Physiol.* 514, 915–928. doi: 10.1111/j.1469-7793.1999.915ad.x

Germano, A. M., Schmidt, D., Schlee, G., and Milani, T. L. (2016). Plantar sensory vibration thresholds are not influenced by body position. *Cogent. Med.* 3:1238600. doi: 10.1080/2331205X.2016.1238600

Green, D. M., and Swets, J. A. *Signal detection theory and psychophysics*. John Wiley, (1989). New Jersey

Hauck, L. J., Carpenter, M. G., and Frank, J. S. (2008). Task-specific measures of balance efficacy, anxiety, and stability and their relationship to clinical balance performance. *Gait Posture* 27, 676–682. doi: 10.1016/j.gaitpost.2007.09.002

Haykin, S., and Chen, Z. (2005). The cocktail party problem. *Neural Comput.* 17, 1875–1902. doi: 10.1162/0899766054322964

Hock, H. S., and Schöner, G. (2010). Measuring perceptual hysteresis with the modified method of limits: dynamics at the threshold. *Seeing Perceiving* 23, 173–195. doi: 10.1163/187847510X503597

Horak, F. B., and Diener, H. C. (1994). Cerebellar control of postural scaling and central set in stance. *J. Neurophysiol.* 72, 479–493. doi: 10.1152/jn.1994.72.2.479

Horslen, B. C. (2016) *Vestibular, proprioceptive, and cutaneous reflex modulation explored through a height-induced postural threat.* Dissertation thesis. Vancouver (ON, Canada): University of British Columbia.

Horslen, B. C., Inglis, J. T., Blouin, J. S., and Carpenter, M. G. (2017). Both standing and postural threat decrease Achilles' tendon reflex inhibition from tendon electrical stimulation. *J. Physiol.* 595, 4493–4506. doi: 10.1113/JP273935

Horslen, B. C., Murnaghan, C. D., Inglis, J. T., Chua, R., and Carpenter, M. G. (2013). Effects of postural threat on spinal stretch reflexes: evidence for increased muscle spindle sensitivity? *J. Neurophysiol.* 110, 899–906. doi: 10.1152/jn.00065.2013

Horslen, B. C., Zaback, M., Inglis, J. T., Blouin, J. S., and Carpenter, M. G. (2018). Increased human stretch reflex dynamic sensitivity with height-induced postural threat. *J. Physiol.* 596, 5251–5265. doi: 10.1113/JP276459

Huffman, J. L., Horslen, B. C., Carpenter, M. G., and Adkin, A. L. (2009). Does increased postural threat lead to more conscious control of posture? *Gait Posture* 30, 528–532. doi: 10.1016/j.gaitpost.2009.08.001

Knyazev, G. G., Savostyanov, A. N., and Levin, E. A. (2004). Alpha oscillations as a correlate of trait anxiety. *Int. J. Psychophysiol.* 53, 147–160. doi: 10.1016/j.ijpsycho.2004.03.001

Lackner, J. R. (2021). The importance of being in touch. *Frontiers in neurology*.12, 646640.

Lim, S. L., Bruce, A. S., and Aupperle, R. L. (2014). The influence of a working memory task on affective perception of facial expressions. *PLoS One* 9:e111074. doi: 10.1371/journal.pone.0111074

Lloyd, D. M., Bolanowski, S. J., Howard, L., and McGlone, F. (1999). Mechanisms of attention in touch. *Somatosens. Mot. Res.* 16, 3–10. doi: 10.1080/08990229970609

Lopresti-Goodman, S. M., Turvey, M. T., and Frank, T. D. (2013). Negative hysteresis in the behavioral dynamics of the affordance "graspable". *Atten. Percept. Psychophys.* 75, 1075–1091. doi: 10.3758/s13414-013-0437-x

McIlroy, W. E., Bishop, D. C., Staines, W. R., Nelson, A. J., Maki, B. E., and Brooke, J. D. (2003). Modulation of afferent inflow during the control of balancing tasks using the lower limbs. *Brain Res.* 961, 73–80. doi: 10.1016/S0006-8993(02)03845-3

Melloni, L., Molina, C., Pena, M., Torres, D., Singer, W., and Rodriguez, E. (2007). Synchronization of neural activity across cortical areas correlates with conscious perception. *J. Neurosci.* 27, 2858–2865. doi: 10.1523/JNEUROSCI.4623-06.2007

Mergner, T., and Rosemeier, T. (1998). Interaction of vestibular, somatosensory and visual signals for postural control and motion perception under terrestrial and microgravity conditions – a conceptual model. *Brain Res. Rev.* 28, 118–135. doi: 10.1016/S0165-0173(98)00032-0

Mildren, R. L., Strzalkowski, N. D., and Bent, L. R. (2016). Foot sole skin vibration perceptual thresholds are elevated in a standing posture compared to sitting. *Gait Posture* 43, 87–92. doi: 10.1016/j.gaitpost.2015.10.027

Naranjo, E. N., Cleworth, T. W., Allum, J. H. J., Inglis, J. T., Lea, J., Westerberg, B. D., et al. (2016). Vestibulo-spinal and vestibulo-ocular reflexes are modulated when standing with increased postural threat. *J. Neurophysiol.* 115, 833–842. doi: 10.1152/jn.00626.2015

Peters, R. M., Dalton, B. H., Blouin, J. S., and Inglis, J. T. (2017). Precise coding of ankle angle and velocity by human calf muscle spindles. *Neurosci.* 349, 98–105. doi: 10.1016/j.neuroscience.2017.02.034

Peters, R. M., McKeown, M. D., Carpenter, M. G., and Inglis, J. T. (2016). Losing touch: age-related changes in plantar skin sensitivity, lower limb cutaneous reflex strength, and postural stability in older adults. *J. Neurophysiol.* 116, 1848–1858. doi: 10.1152/jn.00339.2016

Rauch, R., Angel, R. W., and Boylls, C. C. (1985). Velocity-dependent suppression of somatosensory evoked potentials during movement. *Electroencephalogr. Clin. Neurophysiol.* 62, 421–425. doi: 10.1016/0168-5597(85)90051-6

Refshauge, K. M., and Fitzpatrick, R. C. (1995). Perception of movement at the human ankle: effects of leg position. *J. Physiol.* 488, 243–248. doi: 10.1113/jphysiol.1995.sp020962

Shi, Z., Jia, L., and Müller, H. J. (2012). Modulation of tactile duration judgments by emotional pictures. *Front. Integr. Neurosci.* 6, 1–9. doi: 10.3389/fnint.2012.00024

Sibley, K. M., Carpenter, M. G., Perry, J. C., and Frank, J. S. (2007). Effects of postural anxiety on the soleus H-reflex. *Hum. Mov. Sci.* 26, 103–112. doi: 10.1016/j.humov.2006.09.004

Sibley, K. M., Mochizuki, G., Frank, J. S., and McIlroy, W. E. (2010). The relationship between physiological arousal and cortical and autonomic responses to postural instability. *Exp. Brain Res.* 203, 533–540. doi: 10.1007/s00221-010-2257-8

Siegel, E. H., and Stefanucci, J. K. (2011). A little bit louder now: negative affect increases perceived loudness. *Emotion* 11, 1006–1011. doi: 10.1037/a0024590

Simoneau, G. G., Ulbrecht, J. S., Derr, J. A., and Cavanagh, P. R. (1995). Role of somatosensory input in the control of human posture. *Gait Posture* 3, 115–122. doi: 10.1016/0966-6362(95)99061-O

Staines, W. R., Brooke, J. D., Cheng, J., Misiaszek, J. E., and MacKay, W. A. (1997). Movement-induced gain modulation of somatosensory potentials and soleus H-reflexes evoked from the leg I. Kinaesthetic task demands. *Exp. Brain Res.* 115, 147–155. doi: 10.1007/PL00005674

Stefanucci, J. K., Proffitt, D. R., Clore, G. L., and Parekh, N. (2008). Skating down a steeper slope: fear influences the perception of geographical slant. *Perception* 37, 321–323. doi: 10.1068/p5796

Strzalkowski, N. D., Triano, J. J., Lam, C. K., Templeton, C. A., and Bent, L. R. (2015). Thresholds of skin sensitivity are partially influenced by mechanical properties of the skin on the foot sole. *Physiol. Rep.* 3:e12425. doi: 10.14814/phy2.12425

Taylor, J. L., and McCloskey, D. I. (1992). Detection of slow movements imposed at the elbow during active flexion in man. *J. Physiol.* 457, 503–513. doi: 10.1113/jphysiol.1992.sp019390

Teachman, B. A., Stefanucci, J. K., Clerkin, E. M., Cody, M. W., and Proffitt, D. R. (2008). A new mode of fear expression: perceptual bias in height fear. *Emotion* 8, 296–301. doi: 10.1037/1528-3542.8.2.296

Thelen, D. G., Brockmiller, C., Ashton-Miller, J. A., Schultz, A. B., and Alexander, N. B. (1998). Thresholds for sensing foot dorsi– and plantarflexion during upright stance: effects of age and velocity. *J. Gerontol. A Biol. Sci.* 53A, M33–M38. doi: 10.1093/gerona/53A.1.M33

Vasey, M. W., Vilensky, M. R., Heath, J. H., Harbaugh, C. N., Buffington, A. G., and Fazio, R. H. (2012). It was as big as my head, I swear!: biased spider size estimation in spider phobia. *J. Anxiety Disord.* 26, 20–24. doi: 10.1016/j.janxdis.2011.08.009

Verghese, P. (2001). Visual search and attention: a signal detection theory approach. *Neuron* 31, 523–535. doi: 10.1016/S0896-6273(01)00392-0

Yasuda, K., Sato, Y., Iimura, N., and Iwata, H. (2014). Allocation of attentional resources toward a secondary cognitive task leads to compromised ankle proprioceptive performance in healthy young adults. *Rehabil. Res. Pract.* 2014:170304. doi: 10.1155/2014/170304

Zaback, M., Adkin, A. L., Chua, R., Inglis, J. T., and Carpenter, M. G. (2022). Facilitation and habituation of cortical and subcortical control of standing balance following repeated exposure to a height-related postural threat. *Neurosci* 487, 8–25. doi: 10.1016/j.neuroscience.2022.01.012

Zaback, M., Carpenter, M. G., and Adkin, A. L. (2016). Threat-induced changes in attention during tests of static and anticipatory postural control. *Gait Posture* 45, 19–24. doi: 10.1016/j.gaitpost.2015.12.033

Zaback, M., Horslen, B. C., Cleworth, T. W., Collings, L., Langlet, C., Inglis, J. T., et al. (2018). Influence of emotional stimuli on lower limb cutaneous reflexes during human gait. *Neurosci. Lett.* 664, 123–127. doi: 10.1016/j.neulet.2017.11.013

Zaback, M., Luu, M. J., Adkin, A. L., and Carpenter, M. G. (2021). Selective preservation of changes to standing balance control despite psychological and autonomic habituation to a postural threat. *Sci. Rep.* 11, 1–15. doi: 10.1038/s41598-020-79417-5

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Emotional state as a modulator of autonomic and somatic nervous system activity in postural control: a review

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Advances in our understanding of postural control have highlighted the need to examine the influence of higher brain centers in the modulation of this complex function. There is strong evidence of a link between emotional state, autonomic nervous system (ANS) activity and somatic nervous system (somatic NS) activity in postural control. For example, relationships have been demonstrated between postural threat, anxiety, fear of falling, balance confidence, and physiological arousal. Behaviorally, increased arousal has been associated with changes in velocity and amplitude of postural sway during quiet standing. The potential links between ANS and somatic NS, observed in control of posture, are associated with shared neuroanatomical connections within the central nervous system (CNS). The influence of emotional state on postural control likely reflects the important influence the limbic system has on these ANS/somatic NS control networks. This narrative review will highlight several examples of behaviors which routinely require coordination between the ANS and somatic NS, highlighting the importance of the neurofunctional link between these systems. Furthermore, we will extend beyond the more historical focus on threat models and examine how disordered/altered emotional state and ANS processing may influence postural control and assessment. Finally, this paper will discuss studies that have been important in uncovering the modulatory effect of emotional state on postural control including links that may inform our understanding of disordered control, such as that observed in individuals living with Parkinson's disease and discuss methodological tools that have the potential to advance understanding of this complex relationship.

KEYWORDS

emotional state, emotional response, emotion, feeling, autonomic nervous system, somatic nervous system, limbic system, postural control

1. Introduction

Postural control requires the centre of mass (COM) of an individual to be maintained within the limits of the base of support (BOS). It is a complex function involving peripheral, spinal and supraspinal signaling and processing as well as finely tuned and precisely executed motor output. This maintenance of posture requires homeostasis of many different systems of the body and the integration of multi-level nervous system inputs and processing. Research has shown that the motor system is influenced by sensory, cognitive, and behavioral state inputs (1) and thus the state of an individual's nervous system at the time of the executed movement or postural response influences the motor output of the system. Emotional states (e.g., anxiety, fear) can be considered the product of an emotional response and the associated conscious feeling. Individuals' emotional state influences their perception of sensory stimuli, with more threatening perceptions of sensory inputs evoked during fearful states (2, 3) (see Table 1 for definitions of relevant terms). Historically, the most common emotional states probed in the postural control literature have been fear and anxiety (4–8). There is strong evidence of links between autonomic nervous system (ANS) activity and somatic nervous system (somatic NS) activity during postural control activities (4, 5, 9–12). The ANS is made up of the parasympathetic and sympathetic divisions (PNS and SNS) and is responsible for automatic bodily processes such as breathing, heart rate and digestion. Research has demonstrated that the ANS and somatic NS share key "relay stations" in the brainstem, cerebral cortex, and basal ganglia (13–15) and these regions are connected to and influenced by a distributed network of brain regions responsible for emotional processing, the limbic system. There are many different examples of tasks which routinely require the coordinated efforts of the ANS and somatic NS that help to shed light on the importance of the neurofunctional link between these systems. Postural stability requires a dynamic interplay between emotional state, self-awareness, attention allocation, autonomic activity and sensorimotor processing, even under conditions which may be considered automatic (16). As such, it is imperative that balance assessment in both research and clinical settings consider an individual's emotional state.

To explore how emotional states affect standing balance control, researchers have taken various approaches, from experimentally manipulating fear or anxiety using a threat model to studying postural sway in individuals with varying levels of anxiety or fear. Studies have also revealed the importance of emotional state on dynamic tasks such as gait initiation (17, 18). Due to the challenge in distinguishing the influence of emotional state on postural control versus voluntary movement initiation in gait initiation, the current review focusses primarily on studies that evaluate control of postural equilibrium in spontaneous and/or perturbed standing balance tasks. Furthermore, in an effort to maintain this focus, we do not discuss anticipatory postural adjustments in this paper. This area of study is broadly relevant for understanding postural control in order to effectively identify, address and prevent instability that may contribute to increased risk of falls and/or reduced mobility. The objectives of this paper are to:

1. Outline a conceptual framework of common terms as they pertain to the effects of emotional states on postural control.
2. Briefly review neuroanatomical links between emotion processing, and ANS and somatic NS control of posture.
3. Review physiological and behavioural evidence of the association between emotional state, ANS activity, somatic NS activity and posture control.
4. Explore how disordered/altered emotional or ANS processing may influence posture control and assessment.

2. Conceptual framework for studying the influence of emotional state on postural control

The term emotion is commonly used in two distinct ways, either being referred to as a physiological response to stimuli or as the conscious experiences accompanying these bodily responses (often thought of as "feelings"). Furthermore, it is often left undefined in many studies, adding to the confusion. One important aim of this paper is to propose a framework for discussing emotion with regards to postural control (see Table 1 for summary of definitions). Here, the term *emotion* and *emotional responses* is used to refer to the set of physiological responses that occur when the central nervous system (CNS) detects certain negatively or positively valanced stimuli. These are automatic physiological responses that occur both within the cortex and subcortical regions and are expressed in different systems within the body. In cortical and subcortical areas, these emotions involve changes in arousal levels and in cognitive functions such as attention, memory processing and behavioural strategies (19). Within the body, these emotions involve endocrine, autonomic, and musculoskeletal responses (19). LeDoux (20) stated that feelings are "accounts our brain creates to represent the physiological phenomena generated by our emotions." The term "*feeling*" will be used when referring to the conscious experience of these somatic and cognitive responses. The term "*mood*" can be defined as a persistent emotional state, or in other words, the internalized state of emotions and their associated feelings. The term "*affect*" is the external expression of emotion, often observed in facial or bodily reactions and speech. Conceptually, and for example, fear and anxiety have been regarded as emotional states that are caused by external or internal stimuli and that underlie a specific set of measurable behavioural, physiological, hormonal, and autonomic reactions (21). Fear and anxiety can be either transient or persistent emotional states depending on chronicity, and the outward expression of these emotional states are the individual's affect. "*Cognition*" is defined as the mental action or process of acquiring knowledge and understanding through thought, experience, and the senses. Cognition includes such mental processes as sensation, attention and perception, and complex operations such as memory, learning, language use, problem solving, decision making, reasoning and intelligence. The concept "*cognitive-emotional state*" encompasses both the emotion and the associated conscious feeling, as well as the associated cognitive contributions to individuals' state of being. In a review of the relationship between emotion and cognition, Pessoa (22) states that "behaviour is a product of the orchestration of many brain areas; the aggregate function of these brain areas leads to emotion and cognition." Brain regions viewed as "affective"/emotional are also involved in cognition (and regions viewed as "cognitive" are also involved in emotion), and it is known that cognition and emotion are integrated in the brain. Thus, it is important to reflect this relationship in the conceptual framework we use in this field of study. "*Central set*," a term used often within postural control research is defined as the state of readiness of the nervous system for an impending act, based on prior experiences and perception of external conditions. However, it is proposed here that central set be redefined as the state of readiness for an impending act, based on the *cognitive-emotional state* of an individual's nervous system at any given instant in time. This would expand on the already established cognitive contributions to nervous system state to include emotion/emotional response and the associated conscious feeling/experience, as well as the associated cognitive contributions to individuals' state of being (including sensation, perception, attention, memory of past experience, etc.). It also reflects the physiological responses that occur within cortical and subcortical areas at any instant in time (19). For example, when a loss of balance occurs, an individual's nervous system is processing/evaluating: (1) somatic

TABLE 1 Definitions of relevant terms.

Autonomic Nervous System (ANS)	Consists of parasympathetic and sympathetic divisions (PNS and SNS). Responsible for bodily processes such as breathing, heart rate and digestion. Regulates "fight-or-flight" and "rest and digest" responses.
Somatic Nervous System (somatic NS)	A component of the peripheral nervous system. Plays a role in voluntary movements and sensory processing.
Emotion/Emotional Response	The set of physiological responses that occur when the central nervous system (CNS) detects certain negatively or positively valanced stimuli. Responses are coordinated through neural substrates that comprise the limbic system and ANS. Responses include changes in arousal level in the CNS, cognitive function, and autonomic, endocrine, and musculoskeletal responses.
Feeling	The conscious experience of emotions/emotional responses.
Cognition	The mental action or process of acquiring knowledge and understanding through thought, experience, and the senses (includes processes such as attention, perception, sensation).
Emotional State	Emotion/emotional response and the associated conscious feeling.
Cognitive-Emotional State	Encompasses both the emotional state and the associated cognitive contributions to the individual's state of being (i.e., sensation, perception, attention).
Central Set	The state of readiness of the nervous system for an impending act, based on prior experiences and perception of external conditions.
	Proposed definition: The state of a readiness for an impending act based on the *cognitive-emotional* state of an individual's nervous system at any given instant in time.
Affect	The external expression of emotion, often expressed in facial or bodily reactions and speech.
Mood	A persistent emotional state, or in other words, the internalized state of emotions and their associated feelings.
Threatening Perception	Sensory inputs that an individual consciously or subconsciously perceives as threatening.
Anxiety	Emotional state caused by external or internal stimuli and that underlies a specific set of measurable behavioural, physiological, hormonal, and autonomic reactions, as well as a consciously perceived feeling/experience (subjective feeling of anxiety/ worry, nervousness, or unease). Can be transient or persistent.
Fear	Emotional state caused by external or internal stimuli that an individual has perceived as threatening or dangerous (subjective feeling of fear) and that underlies a specific set of measurable behavioural, physiological, hormonal, and autonomic reactions. Can be transient or persistent.

motor responses (i.e., processing sensory information during a loss of balance triggers subsequent motor and autonomic reactions aimed at recovering balance), (2) emotional responses (internal and external sensory stimuli trigger emotional responses, including ANS responses and feelings prior to, and during, the loss of balance), and (3) cognitive responses (sensory information results in allocation of attention, elicits retrieval or storage of context specific memories, etc.). All of these processes/responses come together to result in a behavioural response; an action driven by the present sensory information and the individual's internal state (22).

In the present paper, focus is often directed to the construct of "*emotional state*" (emotion and its associated conscious feeling) as a subset of "*cognitive-emotional state*." This is done, in part, to integrate historical frameworks/conceptualization of emotion/emotional state as an independent process from cognition. Specifically, the modulatory effects of emotional state on ANS and somatic NS activity during postural control are examined. The potential association between cognitive-emotional state and postural control can be considered in both indirect and direct ways. An indirect association is the influence of system responses to changes in cognitive-emotional state that do not expressly influence the somatic NS processing to control balance. As an example of an indirect influence, increased ventilation that can be associated with increased arousal may influence measures of postural sway. Alternatively, direct influences would reflect neuromodulatory effects and interactions between somatic NS control of sensorimotor processes for postural control, and cortical and subcortical changes in activity linked to change in cognitive-emotional state. To understand the potential for direct influences on postural control, the following section briefly reviews neural control of emotional responses, including the neural substrates involved and neuroanatomical associations to somatic control systems.

3. Associations between neuroanatomical networks for emotion processing, ANS and somatic NS control of posture

Researchers have identified neural links between regions of the brain that are involved in emotional states and those that are responsible for balance control (23, 24). The ANS is thought to be one of the most important mediators between the mind and body/viscera (25). Studies show that feelings of fear and anxiety trigger sympathetic nervous system drive and this manifests as physical symptoms/characteristics (26). Homeostasis is mediated via the ANS by fine tuning the balance between sympathetic, parasympathetic and various hormonal systems (27) and is influenced by a number of cortical regions as well as regions in the brain stem, and peripheral and visceral system inputs. We will briefly review key shared "relay stations" in neuroanatomical networks for emotion processing, ANS and somatic NS below.

The reticular formation (RF) is a network of nuclei in the brainstem that plays a key integrative role in the relationship between emotional state and ANS and somatic NS activity (Figure 1). The RF has complex

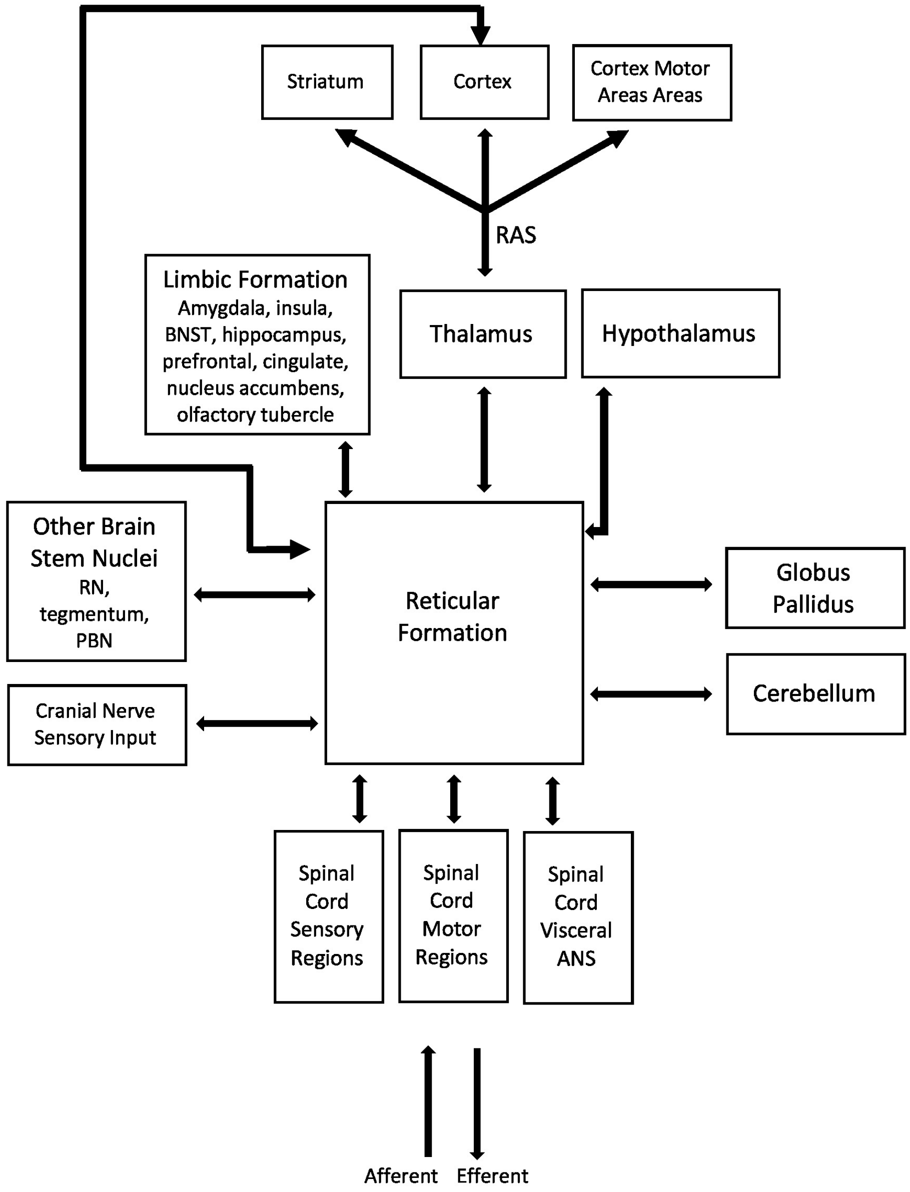

FIGURE 1
Simplified hypothetical model of the shared neural substrates between the limbic system, autonomic and somatic nervous systems. These shared neural networks and pathways drive the experience of emotional states, autonomic activity and postural control. The reticular formation (RF) has complex connections to multiple regions in the CNS and is a site of convergence, divergence and overlap (filtering input and regulating output), with projections to most nuclei within the brainstem, cortical and subcortical regions including limbic regions and the spinal cord. It is well positioned to be one of the main shared neural substrates for interaction and modulation of emotional state, autonomic and somatic nervous system activity. The RF and reticulospinal tracts main functions are (1) autonomic control, (2) respiration and (3) tone of postural muscles. The limbic system is involved in emotional processing, motivation, learning and memory and operates by influencing the endocrine and autonomic systems. Neural substrates that make up the limbic system have bidirectional connections to the RF and it is likely through these connections that incoming sensory/visceral information can modulate our emotional states and perceived feelings, and how feelings and emotional states can influence altered output through somatic and autonomic nervous systems.

connections to multiple regions in the CNS and is a site of convergence, divergence and overlap (filtering input and regulating output), with projections to most nuclei within the brainstem, cortical and subcortical regions including limbic regions and the spinal cord. The three main functions of the RF are (1) autonomic control; (2) respiration and; (3) postural muscle tone. When a stimulus is detected by RF nuclei, system wide lowering of membrane threshold potentials occurs which results in an increase in general nervous system excitability. This has an impact on factors such as reaction time and is likely behind the short latencies we see with balance reactions (28). The reticular activating system (RAS) consists of ascending fibres from the RF which contribute to vital activities in relation to the vigilance state of animals, including humans (29). The RAS receives collaterals from specific ascending pathways. Most ascending fibres of RAS relay in midline and intralaminar nuclei in the thalamus and spread diffusely into various parts of the cerebral cortex, modulating cortical activities via thalamocortical networks (30). The RAS therefore diffusely stimulates the cerebral cortex and along with the thalamus, is responsible for our level of arousal and attention, and modifies postural muscle tone (31). Furthermore, brainstem projections and those descending to the spinal cord contribute to innate motor functions such as eye-head coordination (32, 33) and control of posture and locomotion (34–41) among other functions. It should be noted that much of this research comes from animal models, although there are case studies in individuals with lesions to sections of their RF. Takakusaki et al. (29) report that in most vertebrates, reticulospinal neurons contribute to various types of locomotor movements such as swimming in fishes, crawling in reptiles, flying in birds, quadrupedal locomotion in higher mammals and bipedal gait in higher primates (37). Reticulospinal neurons activate neuronal circuits in the spinal cord that generate locomotor rhythm (central pattern generator) (37, 38, 40–42). Studies have shown the presence of functional topographical organizations with respect to the regulation of postural muscle tone and locomotion in both the mesopontine tegmentum and the pontomedullary reticulospinal system (29). These organizations are modified by neurotransmitter systems, particularly the cholinergic pedunculopontine tegmental nucleus projections to the pontine reticular formation. The author states that because efferents from the forebrain structures as well as the cerebellum converge to the mesencephalic and pontomedullary reticular formation, changes in these organizations may be involved in the appropriate regulation of posture-gait synergy depending on the behavioral context. Furthermore, it is through bidirectional communication between structures of the limbic system and the RF that feelings and emotional states modulate activity of the emotional response, including changes in autonomic activity, breathing rate and postural muscle tone, among other things.

The limbic system is involved in emotion, motivation, learning and memory and operates by influencing the endocrine system and ANS (26, 43). The limbic system consists of six main neural substrates in the brain: (1) the thalamus, which acts as a primary sensory relay station (26, 44); (2) the hypothalamus, which plays a key role in the regulation of many important functions of the body including circadian rhythm and regulation of the ANS; (3) the hippocampus, which plays a key role in forming new memories; (4) the amygdala which rates the emotional importance of the situation, plays a key role in processing emotions and is largely tied to anxiety and fear; (5) the cingulate cortex which plays a role in affect regulation to assist in learning from experience (45, 46); and (6) the basal ganglia which plays a role in habitual behaviour, emotion and cognition (47, 48). In addition, the prefrontal cortex is heavily connected to structures of the limbic system and has been shown to influence both emotive control and movement (49–51). Neural substrates that make up the limbic formation have bidirectional connections to the RF and sharing of information through these connections is one mechanism by which incoming sensory/visceral information can modulate our feelings and emotional states and how our feelings and emotional states can influence altered output through our somatic NS and ANS (muscle tone, heart rate, breathing, etc.). Of important note when discussing the fight or flight response is that the hypothalamus, which integrates a range of sensory inputs and coordinates autonomic, endocrine and behavioural responses aimed at maintaining body setpoints (homeostasis) or overcoming stressors (allostasis), has bidirectional connections to the RF and the thalamus, as well as connections to various other neural substrates of the limbic system (52). As mentioned in Section 2, there are physiological responses that occur when the CNS detects negatively or positively valanced stimuli, and these are mediated in large part by the RF/RAS and hypothalamus. In cortical and subcortical areas, these responses involve changes in arousal levels and in cognitive functions such as attention, memory processing and behavioural strategies. Within the body, these responses involve endocrine, autonomic, and musculoskeletal responses (19). Furthermore, autonomic activity, such as increased respiration or heart rate, has a direct modulatory effect on the activity of the thalamic nuclei (i.e., changing their firing rate) and this may have implications for the way sensory and motor information is processed and/or relayed by the thalamus to other regions of the cortex and subcortical areas during emotional responses (53). This has direct implications for postural control because if the gain of somatosensory processing has been altered, it will result in a change in response to a given stimulus, such as loss of balance.

In summary, research has demonstrated that the ANS and somatic NS share key "relay stations" in the brainstem, cerebral cortex and basal ganglia (13–15) and these regions are part of, or connected to, the limbic system. The coordination/integration of these systems seems to be a key component in postural control and learning through experience, involving the coordination of higher centres, the ANS and the somatic NS. These shared neuroanatomical networks between the limbic system and somatic NS point to the important associations between ANS activity, emotional state and somatic control, and inform the selection of our methodical strategies to concurrently measure emotional state and ANS activity during postural control studies.

4. Physiological and behavioural links between emotional state, ANS and somatic NS activity, and postural control

Evidence of the association between emotion, ANS and postural control comes primarily from studies that have measured postural control in response to a change in emotional state. The most common approach is to use a postural threat model, in which individuals are placed at height. In 2018, Adkin and Carpenter provided a review of the evidence which supports the efficacy of using height-induced threat to study the effects of arousal, anxiety, and fear of falling on postural control (54). Manipulating the height of the support surface has been shown to increase perceived consequences of instability and results in changes in postural control proposed to be evidence of threat-related changes in emotional state on postural control (54). Beyond the use of height-induced threat models, researchers have used models such as threat of perturbation (i.e., threat of translating balance platform) and social evaluation threat (i.e., presence of an observer/examiner). These previous studies have been invaluable in paving the way for our understanding of the neural mechanisms of threat-related changes in balance control in healthy individuals. This review will consider these studies along with other evidence of the possible linkages between emotional state, ANS and somatic NS activity and postural control. This includes a focus on different emotional states and the impact of pathology on emotion processing and/or the ANS, and how these states influence postural control. To do so, this section will focus on evidence from various associations between emotion, ANS activity and postural control including:

1. Evidence of integrated ANS and somatic NS activity.
2. Direct and indirect associations between emotional state, ANS activity and postural control.
3. Disordered emotional state, ANS function and links to postural control.

4.1. Integrated ANS and somatic NS activity

In the daily life of an individual, ANS activity is routinely coordinated and integrated with somatic motor activity and/or neuroendocrine regulation. This happens in response to some combination of external environmental cues, internal physiological conditions, or centrally generated emotional and cognitive states (55–57). This connectivity between systems is apparent when looking at a select few of the many physiological changes that occur in the body during the "fight or flight" sympathetic response. During this response, changes in firing rate of muscles involved in breathing are seen as respiration rate increases (58) and increases in the cell membrane depolarization rate of the sinoatrial node leads to heart rate increases (59). In addition, there is an increase in both smooth and skeletal muscle tone that occurs in times of increased arousal (60, 61). This mechanism is also responsible for the general increase in muscle tension during emotional states such as anxiety, fear and stress with the intensity and duration of sympathetic system responses varying in response to the triggering stimuli. The intensity and duration of the fight or flight response involves a complex interplay between our emotional state, our experience with a given situation, and the state of our sensory and autonomic nervous systems at the time of the event.

4.2. Indirect and direct associations between emotional state, ANS and somatic NS activity and postural control

As mentioned above, the potential association between cognitive-emotional state and postural control can be considered in both indirect and direct ways. An indirect association is the influence of system responses to changes in cognitive-emotional state that do not expressly influence the somatic NS processing to control balance. Alternatively, direct influences would reflect neuro-modulatory effects and interactions between somatic NS control of sensorimotor processes for postural control, and cortical and subcortical changes in activity linked to change in cognitive-emotional state. We can draw from the background knowledge of neural control of emotional responses, including the neural substrates involved and neuroanatomical associations to somatic control systems to aid in our understanding of the potential for direct influences on postural control.

By example, postural control can be indirectly altered by emotional state influences on cardiovascular and respiratory outputs that would impact activation of core musculature, as well as movement of the centre of mass as the rib cage expands and retracts with heavy breathing. Furthermore, various emotional states can result in changes to the attentional resources available for allocation to postural control. This can indirectly result in altered balance and posture responses, which have been reflected in changes in amplitude and frequency of center of pressure displacement, as well as measures such as sway area and sway variability (62–65). In contrast, there are proposed direct links between ANS and somatic NS activity during the control of balance, specifically during reactive balance control most commonly by exploring the impact on postural control associated with changes in fear, anxiety through changes in postural threat (4–8). Fear influences the perception of several sensory stimuli, with more threatening perceptions evoked during fearful states (2, 3). In the context of postural control, anxiety has been shown to have a modulatory effect on both ANS and somatic NS activity during quiet stance and during balance reactions (4–6, 9–12, 66–69). It is well established that emotional responses involve changes in sympathetic and parasympathetic activity (58) and there is also strong evidence for ANS activity modulation in balance reaction and postural control (4, 9–12). Researchers have approached this topic by looking into various emotional states and the response elicited by the motor system. Specifically, studies have examined postural threat scenarios where individuals are perturbed while on an elevated platform, and quiet standing and balance tasks in individuals with high versus low trait anxiety. These studies are reviewed below.

Furthermore, evidence suggests the context of the postural threat greatly affects the demonstrated behaviours that are observed. Several studies have examined the impact of context on postural responses to altered emotional states, including the influence of anxiety or threat on core muscle activation, spinal reflex excitability, conscious perceptions of sway as well as others looking at the influence of threat on allocation of attention. These studies have been important in furthering our understanding of the relationship between emotional state, ANS activity and postural control, through the lens of the impact of arousal on postural control outcome measures. However, it is important to build on these findings and embrace a "*cognitive-emotional state*" framework when interpreting the results of these studies, and to help synthesize future methodological approaches to probe these research questions.

If we are to use this research to inform both laboratory studies, and clinical assessment and treatment approaches in the future, it is important to take both an open focus (looking at the overall cognitive-emotional state of an individual and how altered states of being may impact observable behavioural outcomes) as well as a "spotlight" focus on these various specific direct and indirect mechanisms (i.e., looking at the association of cognitive-emotional state on specific processes, such as altered attentional focus and postural control, for example) by which cognitive-emotional state can modulate postural control. As discussed earlier in the paper, brain regions viewed as emotional are also involved in cognition (and regions viewed as cognitive are also involved in emotion), and it is known that cognition and emotion are integrated in the brain (22). The cognitive contributions to an individuals' state of being (including sensation, perception, attention, memory of past experience, etc.) impact the response of that individuals' nervous system to a given stimulus (such as a loss of balance/challenge to balance) and stimulus driven changes to cognition (which are integrated with emotional responses), including both altered sensory processing and attentional focus for example (not one exclusively as is often interpreted), can help to clarify many of the findings in this body of literature. When a threat (or an altered emotional state such as anxiety) is present, RF/RAS, thalamus and hypothalamus mediated changes in emotional responses (including endocrine and ANS activity changes) and cognitive processes occur which ultimately manifest as changes in behavioural outcome measures, including altered COP sway and frequency, for example. That is, the interaction between emotional and cognitive responses (including perception of threat) ultimately leads to the context specific behavioural outcome. The next several sub-sections will discuss some of these direct and indirect associations between cognitive-emotional state, ANS activity and postural control in various behavioural studies.

4.2.1. Indirect and direct associations between emotional state and breathing rate and core muscle activation and in quiet stance

4.2.1.1. Association between anxiety, breathing rate and core muscle activation

Studies linking emotional states such as anxiety and fear with autonomic, respiratory and cardiac activity have helped to clarify the neurophysiological relationship between feelings, emotional states and their physical manifestation. It has been demonstrated that stress and anxiety have a modulatory effect on ANS activity and on breathing rate (70–72). Furthermore, irregular breathing involving shallow and/or deep rapid breathing patterns and periods of apnea, has been shown to modulate the ANS leading to excitation of the nervous system (70, 73, 74). With regards to motor control, research has shown that breathing pattern can alter the effectiveness of core musculature used for functional tasks (75–77). This is likely due to the diaphragm, a key breathing muscle, also acting as a key core muscle regulator (78–80) with an associated postural function (81). The diaphragm has been found to contract prior to initiation of upper extremity movement (82, 83) independently of respiration phase (83). When respiration becomes quick and shallow, the diaphragmatic muscle activity is altered, and this is likely to have an impact on how effectively it, and the other core muscles, stabilize the trunk during functional movement (84). In these instances, postural muscles are affected by emotional states indirectly, whereby emotional state and increased sympathetic activity impacts breathing rate, which in turn affects core muscle function. The relationship between pattern of diaphragmatic breathing and core stability has been linked to static and dynamic balance and it has been shown that the amplitude of COP displacement increases with increasing respiration (25, 27, 84–87). While it is possible to argue for a stabilizing benefit of increased trunk stiffness, the more transient impact of changes in ventilation can result in an increase in measured postural sway due to trunk motion (88, 89).

4.2.1.2. Associations between anxiety, stress, and postural sway in quiet stance

Evidence of the modulating influence of arousal during quiet stance was first demonstrated in a 1996 study by Maki et al. (90). Since then, studies looking at postural sway have found that high anxiety modifies the location and frequency of centre of pressure (COP) in the antero-posterior (AP) axis (5, 6). A study by Wada et al. (6) demonstrated increased postural sway in the AP axis in a high versus low trait anxious group. In another study, Bolmont et al. (5), demonstrated that a deterioration in mood state, measured by the profile of mood state (POMS) questionnaire, affects balance performance and participants' ability to use input from either the somatosensory, visual or vestibular system to maintain balance during quiet stance (5). A study by Hainaut et al. (66), found that when vision was absent, there was an increase in postural sway during static balance control when comparing individuals with high versus low trait anxiety. These authors also suggest that state anxiety could modify the processing of various sensory inputs involved in balance control regardless of the individual's trait anxiety. Furthermore, a study by Coco et al. (67), revealed a significant positive correlation between cortisol awakening response (marker of chronic stress) and perceived stress, and that these factors influenced postural stability, which manifested as increased COP excursion (67–69). This study also demonstrated a stronger influence of stress when no visual information was present (67). These studies highlight the relationship between emotional state and postural control in quiet stance that may be amplified in more challenging conditions such as reduced vision. One explanation for the altered sensory utilization could be due to the fact that the chronic increased sympathetic activation that is present in those with higher trait relative anxiety, as well as during state anxiety (i.e., situational specific "stress-response"/increased arousal), both mediated by the RF/RAS, thalamus and hypothalamus, leads to changes in sensory processing and subsequent changes in sensory-motor responses (53). As mentioned earlier in this review, this has direct implications for postural control because if the gain of somatosensory processing has been altered, it will result in a change in response to a given stimulus (i.e., loss of balance or challenge to the balance system).

4.2.2. Indirect and direct associations between postural threat models, emotional state and postural control

4.2.2.1. Postural threat studies to increase fear, anxiety and arousal

To gain insight into the relationship between anxiety, ANS and somatic NS activity and postural control/reactions, researchers have experimentally manipulated fear or anxiety by eliciting arousal scenarios with "threats" to individuals, often accomplished by having the participants stand on elevated platforms. Research investigating the effect of height-induced postural threat has shown increased anxiety and fear of falling, lower balance confidence, elevated physiological arousal (as measured by electrodermal activity—EDA) and changes in blood pressure (9–11, 91–94). Researchers have also shown that state anxiety caused by actual threat of a fall could lead healthy participants to develop a postural stiffening strategy characterised by smaller amplitude and higher frequency of COP displacement (4, 7, 95, 96). These and other studies have shown that this postural threat modifies the speed and amplitude of postural sway in quiet standing (4, 7, 9–12). It could be argued that this type of postural anxiety/threat scenario could reflect the possible arousal that a fearful or anxious individual may experience with a loss of balance in real-world settings, and thus has been frequently used as an experimental model to look at the effect of arousal on human balance and related measures of interest. It remains important to keep in mind that this experimental design has context specific consequences to the behavioural outcome (for example, in the elevated platform model participants lean away from the edge, and this impacts COP trajectories). Thus, the context specific changes to behavioural outcomes always needs to be top of mind when interpreting results. As mentioned, it is important to probe these specific relationships to gain an understanding of the indirect and direct associations with emotional state and postural control, however, it remains important to frame these study findings within the lens of a cognitive-emotional state framework. That is, to keep in mind that the interaction between both emotional and cognitive processes and responses ultimately leads to the context specific measurable behavioural outcomes.

4.2.2.2. Postural threat studies (to increase fear, anxiety and arousal) and spinal reflexes

Some studies using the elevated height model have explored the potential influence of postural threat, when standing at height, on spinal reflex excitability of the plantar flexors. It has been proposed that the gain of these reflexes may have an influence on control of standing balance, with higher gain resulting in tighter control. Sibley et al. (97) explored this using the electrically evoked H-reflex and Horslen et al. (98) evaluated both the H-reflex and the mechanically evoked tendon reflex (T-reflex). More recently Hodgson et al. (99) compared H-reflexes during standing at different visual heights, using virtual reality. The results of these studies were mixed, with respect to the H-reflex gain. Sibley et al. (97) and Hodgson et al. (99) revealed a lower gain and Horslen revealed no change in H-reflex standing at height, but instead revealed a higher T-reflex gain which they proposed was linked to higher fusimotor drive. The challenge comparing such studies is the numerous factors, unrelated to postural threat, that may be associated with changes in reflex gain. For example, the evoked reflexes are themselves destabilizing, serving as a perturbation and the observation of reduced gain, while potentially linked to increased threat, could have reflected the CNS attempt to minimize the destabilizing influence of the evoked reflex. In addition, H reflexes are attenuated in more challenging task conditions, unrelated to threat, that is proposed to be linked to reducing sensory inputs to optimize information processing. The speculation of increased fusimotor drive, that maybe linked to threat and the desire to tighten control and reduce sway, is consistent with studies revealing task related differences in fusimotor drive during challenging locomotor tasks. While such reflex studies do afford the potential to reveal insight into the underlying neurophysiological changes that may be linked to emotional state, they will require far greater attention the factors that impact reflex gain including attention to time-varying changes in state that occur during continuous standing.

4.2.2.3. Associations between emotional state, sensory processing and postural control

Two studies by Cleworth and colleagues have probed the question of whether threat has an impact on sensory perception during balance tasks, further looking into a direct association between emotional state, ANS activity and postural control. In one study by Cleworth et al. (100), examining how changes in threat influenced conscious perceptions of postural sway in the antero-posterior plane, participants reported an increased level of fear, anxiety, and arousal, and a decreased level of balance confidence when standing at height (100). These researchers also found that sway amplitude is reduced, while sway perception appears to remain unchanged. They argue that as threat is increased, sensory gain may be increased to compensate for postural strategies that reduce sway (i.e., stiffening strategy), allowing availability of sufficient afferent information to maintain, or even increase the conscious perception of postural sway (100). In another study by Cleworth et al. (101) examining the impact of height-related threat on voluntary balance control (i.e., leaning towards targets) in healthy individuals, it was found that an elevated platform height resulted in significantly increased EDA, fear and anxiety, and decreased balance confidence and that the psychological state of an individual can significantly affect perceived body position during postural tasks (101). Although sensory gain is one mechanism by which threat modulates postural strategies, conscious perception of postural sway, and perceived body position during postural tasks, it is important to consider other possible mechanisms for these results, such as altered attentional focus that would occur when an individual is asked to lean towards targets while a threat is present.

4.2.2.4. Associations between emotional state, attentional allocation and postural control

There is considerable evidence demonstrating the attentional resources required for processing emotional stimuli (102, 103), though it is not clear the specific impact of the emotional state or the underlying cognitive processing. Emotional state is well recognized as having a significant influence on cognitive processes and is specifically influential on attention (102). The "emotional attention" that can be required under different states can compete with other demands on attentional processing. This raises the possibility that changes in postural control associated with changes in emotional state could be linked to altered attentional demands, in some ways paralleling dual-task effects. In such a case, a demanding postural task (i.e., significant need for executive resources), would be more likely to be impacted by emotion processing. Researchers have proposed that changes in allocation of attention may influence the relationship between postural threat (eliciting anxiety) and balance control. The presence of perceived threat can alter allocation of attention, either directing attention toward or away from an individual's posture, depending on the nature of the threat.

Huffman et al. (104) demonstrated that with height-induced threat, individuals have a greater tendency to consciously control and monitor their posture. This increase in conscious control was shown to be related to leaning further away from the platform edge, independent of any changes in amplitude or frequency of COP displacements. In various studies, when standing in conditions of increased threat, individuals self-report broad shifts in attentional focus such as directing more attention to the mechanics of movement, threat-related stimuli, and strategies to improve confidence and/or reduce anxiety (104–107). These shifts in attention have been associated with, and may indirectly contribute to, specific threat-related changes in standing balance (104–107). For example, recent work revealed that changes in ankle muscle co-contraction and high-frequency COP displacements were the only changes in standing balance to adapt following repeated exposure to height-related threat, with changes in attention to movement as the strongest predictor of changes in high-frequency COP displacements (106). These findings align with research suggesting a relationship between conscious movement control and high-frequency COP displacements in patients with anxiety-related balance disorders (62–64). A study by Johnson et al. (108), found that when participants were threatened, they were more anxious and reported directing more attention to movement processes, threat-related stimuli, and self-regulatory strategies, and less to task-irrelevant information. It was also found that postural sway amplitude and frequency increased with threat. Furthermore, greater increases in frequency and smaller increases in amplitude of COP were observed with perturbation experience. In instances with no experience, it was found that threat-related changes in postural control could be accounted for by changes in anxiety, with larger changes in anxiety related to larger changes in sway amplitude. The authors state that with perturbation experience, threat-related postural control changes were accounted for by changes in attentional focus, with increases in attention to movement processes being related to greater forward leaning and increases in sway amplitude. It was also found that increases in attention to self-regulatory strategies were related to greater increases in sway frequency. These findings suggest relationships between threat-related changes in anxiety, attentional focus, and postural control depend on the context associated with the threat.

Although changes in attentional focus is one mechanism by which threat modulates postural strategies, changes in sensory processing/ sensory gain of information that is relevant to the balance task plays a role in shifts in attentional focus that may occur. For example, recent research in healthy older adults has shown that conscious balance processing may drive behaviours that are opposite to postural stiffening responses (i.e., reduced sway frequency and increased sway amplitude) (109, 110). In young healthy adults, conscious movement processing (CMP) has been shown to increase sway amplitude during relatively simple static balance tasks (111, 112). However, as task difficulty increases, the effect of CMP on postural sway may change, with some evidence to suggest that CMP may help enhance balance performance (110, 113). This could be due in part to increased sensory gain to task-relevant sensory information and it can be reasoned that sensory gain changes (i.e., increased processing of internal sensory information pertaining to control of movement) the more an individual shifts attentional focus into conscious control of their movement. Jie et al. (114) argues that compared to a low-CMP condition, high CMP (as often seen in anxious individuals and clinical populations) leads to increased postural sway, which is often interpreted as worse performance in an easy, very stable (solid surface) task condition. However, these effects may be less pronounced or even reversed during more challenging balance conditions in which individuals are standing on more unstable surfaces, such as foam (114). The challenge of standing on foam, depending on the compliance, is an alteration in the sensory input that arises from plantar surface contact and but also change in the transfer of muscle force to the support surface. This will impact both the required sensorimotor transformation and the attentional focus which may account for differences compared to stable support surface conditions.

4.2.2.5. Association between repeated exposure to height induced threat, emotional state and postural control

In a study by Zaback et al. (115), individuals were repeatedly exposed to height-induced postural threat to determine if reducing the emotional response to threat influences standing balance control. Following repeated threat exposure, participant emotional responses to the threat were attenuated, however, the threat-induced changes in standing balance were still present. When initially exposed to the threat, individuals leaned backward (a context specific direct association) and demonstrated smaller amplitude and higher frequency of COP adjustments and these balance outcome measures did not change following repeated threat exposure. High frequency COP oscillations (>1.8 Hz) and ankle muscle co-contraction showed adaptation to repeated exposure to the threat, with behavioural adaptations being accounted for by a combination of emotional and cognitive state changes. The findings suggest that some threat-induced standing balance changes are more closely linked with the arousal response to the threat than others and therefore, are altered by the repeated exposure to the threat. The arousal response is one aspect of an emotional response, in addition to the RF/RAS, thalamus and hypothalamus mediated changes in cognitive processing. As cognitive processes and emotional processes in the brain are integrative, it is the interaction between emotional (including arousal) and cognitive processes will result in the observed behavioural outcomes. Other findings reflect the context specific conditions that the task imposes, which ultimately also contribute to the observed behavioural outcome. In a follow up study, Zaback et al. (116) used a prolonged threat exposure protocol to manipulate emotional state within a threatening context to determine if any threat-induced standing behaviours are employed independent of emotional state. With the initial threat exposure, individuals leaned backward, showed reduced low-frequency COP power, and increased high-frequency COP power as well as plantar/dorsifexor coactivation. Following repeated exposure, the psychological and autonomic responses to the threat were decreased and high-frequency COP power and plantar/ dorsifexor coactivation habituated. In this study, individuals were re-exposed after 2–4 weeks and demonstrated a partial recovery of the emotional response to the threat with few standing balance adaptations retained. The authors suggested that some threat-induced standing behaviours are coupled with the psychological and autonomic state changes induced by the presence of threat, while others may reflect context-appropriate adaptations resistant to habituation.

In another study examining the effects of initial and repeated postural threat exposure on emotional, cognitive, and postural responses, Johnson et al. (117) had young and older adults stand on a force plate fixed to a translating platform and manipulated threat through expectation of temporally and directionally (left or right) unpredictable platform perturbations. Postural threat elicited similar emotional, cognitive, and postural changes in young and older adults. With initial threat exposure, participants reported increases in self-reported anxiety and physiological arousal, as well as broad changes in attentional focus. Participants also significantly increased COP amplitude and frequency, and COP power within medium and high frequencies. With repeated threat exposure, anxiety, arousal, and some threat-induced changes in attentional focus significantly adapted. The authors reported that changes were accompanied by significant reductions in COP frequency and COP power within medium frequencies. Some emotional and cognitive outcomes returned to no threat levels while postural outcomes did not. The authors concluded, similarly to the conclusions made by Zaback et al. (115), that their findings suggest that some threat-related changes in standing postural control may be closely linked with one's emotional response to threat, while others may be context-dependent. When participants experienced the feeling of anxiety and physiological arousal, there is an internal shifting of attentional focus, potentially through mechanisms associated with RAS/RF mediated changes in cognitive processes. The associated altered COP frequencies may reflect both the RF/ANS meditated changes in attentional focus, postural tone and altered sensory perception gain with changes in cognitive-emotional state.

4.2.2.6. Associations between threat, conscious experience (feeling) of fear and postural control

Ellmers et al. (118), has proposed that the mechanisms responsible for behavioural and physiological responses to threat may be distinct from those underpinning the conscious emotional experience itself (a feeling). To examine this, the researchers had older adults stand on the edge of a raised platform and were stratified based on whether they reported fear in response to this postural threat. Behaviours indicative of postural "stiffening" during the threat condition were observed regardless of whether participants reported fear or not. The authors state that self-reports indicated the participants cognitively monitored these changes in balance, and fear of falling was experienced in those who interpreted these behaviours to imply that harm was likely to occur. Fearful participants also showed changes in balance, including increased movement complexity and altered utilisation of sensory feedback. The authors concluded that these behaviours were influenced by attempts to consciously control balance. As can be seen from this work, the interaction between emotional and cognitive responses (including conscious perception of fear) ultimately leads to the behavioural outcome. Those individuals who experienced the feeling of fear of falling during the study had a corresponding change to their motor control as well as alterations in sensory feedback. Autonomic responsivity (e.g., increased respiration or heart rate) that may occur in individuals who experience fear when standing at the edge of an elevated platform, can have a direct modulatory effect on the activity of the thalamic nuclei and this has implications for the way sensory information is processed and/or relayed by the thalamus to other regions of the cortex and subcortical areas (53). From this perspective, it is possible that participants in the Ellmers study who interpreted the behaviours indicative of "postural stiffening" as potentially harmful, also had more pronounced shifts in utilisation of sensory feedback, resulting in altered interpretation of the incoming sensory information (demonstrating how cognitive-emotional state impacts behavioural outcomes). This has the potential to result in high vigilance and over-sensitivity to environmental signals which are reflected in inappropriate emotional responses and ANS dynamics.

4.2.2.7. Association between social evaluation threat, emotional state and postural control

Furthermore, social evaluation threats (such as the presence of an expert evaluator) have been used to elicit changes in emotional states. Doumas et al. (119) examined whether similar stress-related changes in postural sway can be observed using stress induced by social evaluative threat while performing arithmetic tasks under a time pressure. Postural sway amplitude was greater and reaction times faster when performing arithmetic task under a time pressure, but not under other task challenges. In this example, there are likely direct (i.e., arousal mediated changes, altered sensory gain, context specific influences on ones sensorimotor processing, etc.) and indirect (i.e., altered attentional allocation) mechanisms occurring which ultimately lead to the observed behavioural outcomes.

4.2.3. Affect, arousal and postural control

In addition to manipulations of postural threat, studies have also explored the relationship between mood, affect and the control of posture. A common approach in neuropsychosociological research is to provoke changes in arousal and affect using picture/images. There exists, as example, an International Affective Picture System (IAPS) that is used to provide visual images for assessing emotional outcomes and these images are categorized based on the valence (negative to positive) and impact on arousal (calm to exciting) (120). The link between postural control and visual stimuli had been reviewed in detail by Lelard et al. (121). Lelard et al. (121) summarized that the majority of studies have focused on differences in valence (positive versus negative) and linked postural changes to approach and withdrawal behavior. There have been some studies who have also reported freezing in response to the most negative/arousing stimuli. As an example, Stins et al. (122) used pictures to provoke state changes in affect with images linked to positive or negative valance and measured the impact on balance control. While they noted little difference in postural sway measures across visual stimuli that were intended to provoke different emotional states, they did reveal two key outcomes. The first was a reduction in sway path when viewing pictures of mutilation that they hypothesized was fear induced bradykinesia. Second, this influence was only evident in the more challenging postural task condition (unipedal stance versus standard stance). Similarly, Roelofs et al. (123) reported significant decrease in the sway in response to the angry expressions as opposed to happy and neutral expressions and interpreted the reduced body sway as "freezing behaviour." They suggested that freezing behaviour enables an individual to first detect relevant information, then to mobilize the whole body, and ultimately to trigger "fight or fight" behaviour (124). The challenge in such work is distinguishing the potential impact of valance versus arousal on postural control, since such strong stimuli with negative valance are also highly arousing. Horslen and Carpenter (125), sought to distinguish the effects of arousal and valance on balance control since differences associated with altered affect (positive or negative) may be attributable to changes in arousal/anxiety. Measures from both balance (COP) and ANS reactivity (EDA) were influenced by the changes in arousal independent of evoked changes in valence. Consistent with studies elevating arousal through changes in threat, the authors concluded that arousal may be the underlying mediator of emotional state related changes in postural control. While there appears to be evidence of the impact of emotional visual stimuli on postural control and that the influence of arousal state may be most important with effects of valance being inconsistent or absent. The observation that some stimuli, such as those with high negative valence evoke change in postural control may arise from indirect influences such as altered body position or even changes in muscle tone, though the latter is rarely measured. Continuing to disentangle the impact of valence versus arousal and to establish the specific mechanisms of modulation of posture control will be important to better understand the insight that can be gain from these paradigms.

4.3. Disorders of anxiety and emotion processing and potential links to postural control

The majority of research in the area of emotional state and postural control has been conducted in healthy individuals whose cognitive-emotional state is influenced transiently by varying task conditions. An alternative approach is to study postural control in individuals with cognitive-emotional state disorders. Unfortunately, by comparison to work with healthy adults, there is considerably less research that has explicitly examined the impact of disorders of emotional processing on balance control. In addition, a significant focus in studies on healthy individuals is directed towards states of heightened arousal where states of low arousal can be a frequent consequence of specific neurologic disorders. Furthermore, several disorders are characterised by muted autonomic or emotional reactivity which may have implications to the control of balance. Below is a brief discussion of some of the evidence of the links between the control of postural equilibrium and disorders of anxiety and emotion processing.

Balaban and Thayer (24) presented a detailed framework for neuroanatomical underpinnings of the links between anxiety and the control of balance highlighting support for a bidirectional influence. Evidence in support of these foundations has been reinforced through studies focussed on balance control among individuals with anxiety disorders (126–128) and findings have shown evidence of altered balance control among those diagnosed with anxiety disorders [e.g., generalized anxiety disorder (GAD), social anxiety disorder, or other specific phobias]. For example, Redfern et al. (129) highlighted increased sway among individuals with GAD in response to visual motion. Findings were attributed to the inability of GAD patients to disregard misleading visual information. Evidence of increased postural sway has also been observed in highly anxious children relative to children without an anxiety disorder (129). However, the relationship between anxiety and control of balance is complex as there is also evidence, in support of the experiments performed at height, that anxious individuals may adopt a defensive stiffening strategy and be characterised by lower levels of measured postural sway (130). The commonly reported increase in muscle tone that is linked to anxiety disorders such as GAD (131) is a potential indirect influence on the control of balance, as could be attentional focus. Evidence suggests shared neuroanatomical and neurochemical links as the foundation for comorbidity of balance and anxiety disorders (126).

Emotion processing deficits are often linked to challenges in regulating emotions and attentional biases that impact perception and impaired recognition of emotions, among others, and diagnosed individuals are often highly co-morbid (132). For example, persons diagnosed with alexithymia have difficulty identifying and describing feelings however it is common to be reported alongside other co-morbid conditions including neurological disease such as Parkinson's disease (133). There exist many neurologic diseases/injuries that present with deficits in emotion processing and with balance control. Examples include Parkinson's diseases (134), Alzheimer's disease (135), post-traumatic stress (136) and traumatic brain injury (137). However, all are commonly characterized by other sensorimotor and/or executive processing symptoms that may also impact postural control. As a result, it is often difficult to isolate an independent association to postural control and emotion processing, and we are not aware of specific studies that have isolated a specific and independent link between emotion processing deficits and the control of balance.

While there are certainly a range of potential factors that might link these disorders to balance control there is some commonality among these disorders which may tie directly to the control of balance. There appears to be a common characteristic of altered autonomic activity among those with disorders of anxiety and emotional processing. As examples, electrodermal hypoactivity appears to be a reliable feature of depression with skin conductance measures being described as low or flat, reflecting both low tonic levels and a muted reaction to stimuli (138, 139). Anxiety disorders have been associated with muted ANS reactivity which has been described as decreased autonomic flexibility. Diamond and Fisher (140) revealed evidence of decreased ANS flexibility among those with GAD during structured interviews. It should be qualified that there are mixed findings in terms of the direction/amplitude of these effects among depressed/anxious patients which might be dependent on the type of stressor used to provoke the ANS reactivity (141). Muted ANS reactivity to arousing stimuli has also been observed among those with alexithymia (142). Among a study of those with emotional dysregulation associated with borderline personality disorder, there was a muted skin conductance and heart rate responses to emotional stimuli (143). Autonomic dysfunction is commonly reported among older adults and those with neurodegenerative disease (144). It is specifically common among those with Parkinson's disease (145) who are also characterized by blunted ANS reactivity to emotional stimuli (127). There is of course a strong direct connection between autonomic dysfunction and balance control when considering changes in vagal tone during postural transitions associated with orthostatic hypotension (146). Such observations of attenuated resting vagal modulation and vagal reactivity in response to postural maneuvers has been observed in people with GAD (147). Given the coupled activation of ANS and somatic NS in balance control, highlighted earlier in this review, it raises the possibility that muted ANS activity, linked emotion processing disorders, may have an impact on postural control.

There is also the question of the potential association role between emotional state and vestibular disorders, the latter often considered classically as balance disorders. There is a longstanding recognition of the association between outcomes such as anxiety, fear and avoidance with dizziness and vestibular disorders (148, 149). While anxiety can be expressed among those with non-vestibular causes of balance control deficits, there is a unique relationship between vestibular function and emotional state. As reviewed by Rajagopalan et al. (150) the vestibular nuclei project widely to many centres including those involved in emotion processing and as a result, vestibular stimulation can directly influence emotional state and has even been used to treat some cognitive-emotional disorders. The direct evidence of vestibular stimulation impacting emotional state is suggested to result from shared neural networks between vestibular and emotion processing (151). The vestibular system is also a critical modulator of autonomic activity through vestibular-autonomic networks (152) which could impact not only vestibular-autonomic reflexes such as those for orthostatic regulation but also emotional responsiveness. Given the important influence of vestibular activity has across the CNS impacting cognitive, emotion and autonomic processing, there is a need to consider the consequence of vestibular dysfunction as more than simply a loss of the sensory contributions to orientation and stability control (153).

5. Recommendations for future studies assessing balance control

5.1. The need for assessment of cognitive-emotional state

As is evident through this paper, there are many research studies that demonstrate a link between emotional states, autonomic nervous system ANS activity and somatic NS activity during movement/postural control tasks. It is argued by some that the underlying association between arousal and ANS reactivity may be largely mediated by the link between emotional state and the control of balance. While we would support the idea that measuring the level of arousal and autonomic reactivity is critical to understanding balance control, this review has also highlighted links between feelings, emotional states, and postural control that may be considered independent of the direct state of arousal or ANS activity. Cognitive-emotional state, in addition to arousal, remains an important consideration within the field. The evidence of the important modulatory influence of cognitive-emotional state on balance control does raise the general concern of the need to provide such context when interpreting balance reactions and/or the outcomes of balance control studies. Many have argued for the importance of quantitative measures of balance control including kinetic and/or kinematic measures. Such fidelity is important, but the general lack of concurrent attention to the state of the individual during the test of balance, either through complementary measures and/or task controls, limit the value of such detailed measures of motor behaviour.

5.2. The need for novel, standardized methods to address cognitive-emotional state

Moving forward, efforts must be made to determine/standardize methods of assessing or controlling for cognitive-emotional state during balance testing. This includes addressing indirect factors such as body posture, ventilation and focus of attention, as well as situations and personal characteristics that have the potential to influence cognitive-emotional state and ultimately impact postural or balance assessment. Factors such as what the participant was doing prior to arriving for postural or balance assessment, whether the individual has had their balance/posture assessed previously, caffeine intake (and other substances that alter ANS activity), medical history that may impact emotional state, social support, etc., also need to be considered. It is also important to consider the concurrent impact of the relative task difficulty on both balance control and emotional state, and the possible interaction with somatic control. Given the information this review has presented, on both the association of emotional state, arousal and postural control and balance, and the fact that these factors are influenced by having an observer/evaluator present, it is imperative that these factors be taken into consideration during both research and clinical balance assessments.

5.3. The use of self-reported indices of affect and mood as insight into cognitive-emotional state

It is considered important to advance the use of tools that can provide insight into an individual's emotional state and level of arousal during postural control tasks. There are self-reported indices of affect or mood that have been used such as the positive and negative affect schedule (PANAS), brief mood introspection scale, multiple affect adjective checklist revised version (MAACL-R), profile of mood states (POMS), and discrete emotions questionnaire (DEQ) (154). The concern is that such measures may not be sensitive enough or do not measure context during the balance task. They may also be prone to errors/biases as they are based on self-reporting. Alternatively, there is a strong case to be made for assessing the physiological responses that may be linked to cognitive-emotional state as a continuous time-varying signal that can be associated with the balance control task. There are several possible candidate signals including electrodermal activity (EDA), heart rate variability (HRV), and electroencephalography (EEG), which will be briefly discussed below.

5.4. The use of EDA to probe arousal and gain insight into the relationship between cognitive-emotional state and postural control

Electrodermal recordings have been the more popular research tool to use as a proxy of sympathetic activity due to their utility in representing a continuous variable that can be temporally coupled with the onset of relevant events, such as instability or perturbation (93). There are some limitations related to the temporal lag and other factors that can impact skin conductance (e.g., body temperature) but the methodology and analytics are standardized and could be more broadly introduced. However, use of EDA alone, as a proxy for arousal, does not meet the need for considering an individual's cognitive-emotional state prior to and at the time of testing.

5.5. The use of HRV to provide insight into the relationship between cognitive-emotional state and postural control

HRV is an index of sympathetic and parasympathetic tone which has been used to a limited extent to examine the relationship between emotional state, autonomic and somatic activity, and postural control (155, 156). Among other outcomes, HRV has been found to correlate with cognition, attention, reaction time, memory, emotional regulation, postural control and executive function (155–157). It has also been found that different emotional states are reflected in individuals' state-specific patterns in heart rhythms (158), independent of the amount of HRV, although state-specific changes in the magnitude of HRV are also important. Having high heart rate variability (HRV) is associated with higher emotional well-being, including being correlated with lower levels of worry and rumination, lower anxiety, and generally more regulated emotional responding (159–164). Thus, individuals with higher HRV appear to be better at regulating their emotions. Furthermore, cardiac coherence has been found to be related to emotional states (158). Using a 24 h index of HRV to help inform the emotional regulation of each individual and a 5 min HRV measurement prior to and following research collection, as well as during collection, to quantify the interaction between situation-specific changes and potential balance perturbation (i.e., stimuli coming from the environment). These measures, along with others introduced here, would enable future studies to examine whether individuals who are better able to regulate their emotional state are also less affected by an emotionally triggering stimulus in the environment (such as postural threat) and to assess the impact the stimulus on balance and postural outcomes. Given correlations of HRV and emotional state, and that it is an index of ANS activity, HRV could be a useful tool to utilize when examining the relationship between emotional states, ANS activity and postural control.

5.6. The use of EEG to provide insight into the relationship between cognitive-emotional state and postural control

In addition to HRV, EEG recordings would be beneficial to examining this relationship [please see reference (165) for a review of EEG computation methods for emotional state]. It has been found that changes in the demand to balance control are associated with changes in activity of the cortex as measured by frequency content of EEG recordings (166, 167). Many studies have shown cortical contributions to the recovery of balance following a perturbation in humans (168–171). Event-related potentials (ERP) are known to reflect underlying emotional responses when emotional stimuli are present (i.e., a threat or other emotional stimuli) (165). Furthermore, analysis of cortical cardiac coherence (as well as cortical coherence) could prove to be important when examining this relationship, when considering that 85%–90% of the fibers making up the vagus nerve are afferent, and cardiovascular related afferent neural signal transmission significantly affects activity in most higher brain centers, as well as cognitive processes and emotional state (158, 172, 173).

5.7. Future studies

Finally, future studies should explore these relationships among individuals with ANS, mood and emotional processing disorders for their potential links to postural control, as well as in individuals with neurologic disorders (e.g., Parkinson's disease) where pathology can impact cognitive-emotional processing and autonomic centres. Experimental models that look at the impact of disordered emotion and ANS control on balance reactions should extend beyond the effects of fear/anxiety and threat to include a wider breadth of emotional states and their influence on postural control. Doing so will help to gain understanding of the complex interplay between central and peripheral components that create the conscious perception of our emotional state and how this relates to postural control.

6. Conclusion

- There are strong links between cognitive-emotional state, ANS activity and somatic NS activity in postural control supported by shared neuroanatomical networks including integrated involvement of the limbic system.
- Support for the interaction between cognitive-emotional state and balance control emerges primarily from behavioural studies exploring the impact of evoked changes in emotional state, most often using threatening states, on measures of postural control such as postural sway.
- However, there remains a need to increase a focus on these important associations among those with disordered/altered emotional state, ANS processing and vestibular function.
- It is important to distinguish whether postural behaviours resulting from changes in cognitive-emotional state are from direct or indirect sources to better advance understanding of the specific associations between emotional state, ANS and somatic NS activity and postural control.
- There is a need for development of standardized methods to assess cognitive-emotional state in postural control studies to advance fundamental understanding and to inform interpretation of posture control behaviour. The most promising are likely based on biophysical signals such as EDA, ECG and/or EEG.
- It remains important to further our understanding of the influence of cognitive-emotional states on postural control in a clinical setting for both assessment and treatment of postural and balance dysfunction.

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KH wrote the first draft of the review paper. WM and KO wrote sections of the manuscript. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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References

1. Swanson LW. Quest for the basic plan of nervous system circuitry. *Brain Res Rev*. (2007) 55:356–72. doi: 10.1016/j.brainresrev.2006.12.006

2. Zadra JR, Clore GL. Emotion and perception: the role of affective information. *Wiley Interdiscip Rev Cogn Sci*. (2011) 2:676–85. doi: 10.1002/wcs.147

3. MacLeod C, Mathews A. Anxiety and the allocation of attention to threat. *Q J Exp Psychol A*. (1988) 40:653–70. doi: 10.1080/14640748808402292

4. Carpenter M, Frank J, Silcher C, Peysar G. The influence of postural threat on the control of upright stance. *Exp Brain Res*. (2001) 138:210–8. doi: 10.1007/s002210100681

5. Bolmont B, Gangloff P, Vouriot A, Perrin PP. Mood states and anxiety influence abilities to maintain balance control in healthy human subjects. *Neurosci Lett*. (2002) 329:96–100. doi: 10.1016/S0304-3940(02)00578-5

6. Wada M, Sunaga N, Nagai M. Anxiety affects the postural sway of the antero-posterior axis in college students. *Neurosci Lett*. (2001) 302:157–9. doi: 10.1016/S0304-3940(01)01662-7

7. Adkin AL, Frank JS, Carpenter MG, Peysar GW. Postural control is scaled to level of postural threat. *Gait Posture*. (2000) 12:87–93. doi: 10.1016/S0966-6362(00)00057-6

8. Ohno H, Wada M, Saitoh J, Sunaga N, Nagai M. The effect of anxiety on postural control in humans depends on visual information processing. *Neurosci Lett*. (2004) 364:37–9. doi: 10.1016/j.neulet.2004.04.014

9. Cleworth TW, Horslen BC, Carpenter MG. Influence of real and virtual heights on standing balance. *Gait Posture*. (2012) 36:172–6. doi: 10.1016/j.gaitpost.2012.02.010

10. Adkin AL, Frank JS, Carpenter MG, Peysar GW. Fear of falling modifies anticipatory postural control. *Exp Brain Res*. (2002) 143:160–70. doi: 10.1007/s00221-001-0974-8

11. Hauck LJ, Carpenter MG, Frank JS. Task-specific measures of balance efficacy, anxiety, and stability and their relationship to clinical balance performance. *Gait Posture*. (2008) 27:676–82. doi: 10.1016/j.gaitpost.2007.09.002

12. Yiou E, Deroche T, Do MC, Woodman T. Influence of fear of falling on anticipatory postural control of medio-lateral stability during rapid leg flexion. *Eur J Appl Physiol*. (2011) 111:611–20. doi: 10.1007/s00421-010-1680-7

13. Critchley HD. Review: electrodermal responses: what happens in the brain. *Neuroscientist*. (2002) 8:132–42. doi: 10.1177/107385840200800209

14. Berridge CW, Waterhouse BD. The locus coeruleus-noradrenergic system: modulation of behavioral state and state-dependent cognitive processes. *Brain Res Rev*. (2003) 42:33–84. doi: 10.1016/S0165-0173(03)00143-7

15. Jacobs JV, Horak FB. Cortical control of postural responses. *J Neural Transm*. (2007) 114:1339–48. doi: 10.1007/s00702-007-0657-0

16. Elboim-Gabyzon M, Pitluk M, Shuper EE. The correlation between physical and emotional stabilities: a cross-sectional observational preliminary study. *Ann Med*. (2022) 54:1678–85. doi: 10.1080/07853890.2022.2056241

17. Naugle K, Hass C, Bowers D, Janelle C. Emotional state affects gait initiation in individuals with Parkinson's disease. *Cogn Affect Behav Neurosci*. (2012) 12:207–19. doi: 10.3758/s13415-011-0071-9

18. Naugle K, Hass C, Joyner J, Coombes J, Janelle C. Emotional state effects initiation of gait. *Emotion*. (2011) 11:267–77. doi: 10.1037/a0022577

19. Kandel ER, Schwartz JH, Jessell TM, Siegelbaum SA, Hudspeth AJ, Mack S. *Principles of neural science. 5*. United States of America: McGraw-Hill; (2013). 1079–1093.

20. LeDoux JE. Feelings: what are they and how does the brain make them? *Proc Am Acad Arts Sci*. (2015) 144:96–111. doi: 10.1162/DAED_a_00319

21. Tevote P, Fadok JP, Luthi A. Neural circuits for fear and anxiety. *Nat Rev*. (2015) 16:317–31. doi: 10.1038/nrn3945

22. Pessoa L. On the relationship between emotion and cognition. *Nat Rev Neurosci*. (2008) 9:148–58. doi: 10.1038/nrn2317

23. Nakano K. Neural circuits and topographic organization of the basal ganglia and related regions. *Brain Dev*. (2000) 22:5–16. doi: 10.1016/S0387-7604(00)00139-X

24. Balaban CD, Thayer JF. Neurological bases for balance-anxiety links. *J Anxiety Disord*. (2001) 15:53–79. doi: 10.1016/S0887-6185(00)00042-6

25. Tancer ME, Lewis MH, Stein MB. Biological aspects In: MB Stein, editor. *Social phobia, clinical and research perspectives*. Washington, DC: American Psychiatric Press (1995). 229–57.

26. Kaplan HI, Sadock BJ. *The brain and behaviour: neuroanatomy. Synopsis of psychiatry*. Baltimore, MD: Williams and Wilkins; (1998). 76–97

27. Mosqueda-Garcia R In: D Robertson, I Biaggioni, G Burnstock, PA Low and JFR Paton, editors. *Central autonomic regulation: primer on the autonomic nervous system*. 3rd ed. San Diego, CA: Academic Press (1996)

28. Lakhani B, Van Ooteghem K, Miyasike-daSilva V, Akram S, Mansfield A, McIlroy WE. Does the movement matter? Determinants of the latency of temporally urgent motor reactions. *Brain Res*. (2011) 1416:35–43. doi: 10.1016/j.brainres.2011.08.013

29. Takakusaki K, Chiba R, Nozu T, Okumura T. Brainstem control of locomotion and muscle tone with special reference to the role of the mesopontine tegmentum and medullary reticulospinal systems. *J Neural Transm*. (2016) 7:695–725. doi: 10.1007/s00702-015-1475-4

30. Steriade M, McCormick DA, Sejnowski TJ. Thalamocortical oscillations in the sleeping and aroused brain, science. *Science*. (1993) 262:679–85. doi: 10.1126/science.8235588

31. Portas CM, Rees G, Howseman AM, Josephs O, Turner R, Frith CD. A specific role for the thalamus in mediating the interaction of attention and arousal in humans. *J Neurosci*. (1998) 18:8979–89. doi: 10.1523/JNEUROSCI.18-21-08979.1998

32. Grantyn ABA. Reticulo-spinal neurons participating in the control of synergic eye and head movements during orienting in the cat I, behavioral properties. *Exp Brain Res*. (1987) 66:339–54. doi: 10.1007/BF00243309

33. Grantyn A, Ong-Meang V, Berthoz A. Reticulo-spinal neurons participating in the control of synergic eye and head movements during orienting in the cat. II, morphological properties as revealed by intra-axonal injections of horseradish peroxidase. *Exp Brain Res*. (1987) 66:355–77. doi: 10.1007/BF00243310

34. Drew T, Dubuc R, Rossignol S. Discharge patterns of reticulospinal and other reticular neurons in chronic, unrestrained cats walking on a treadmill. *J Neurophysiol*. (1986) 55:375–401. doi: 10.1152/jn.1986.55.2.375

35. Drew T, Rossignol S. Functional organization within the medullary reticular formation of intact unanesthetized cat. I. Movements evoked by microstimulation. *J Neurophysiol*. (1990) 64:767–81. doi: 10.1152/jn.1990.64.3.767

36. Garcia-Rill E. The basal ganglia and the locomotor regions. *Brain Res*. (1986) 11:47–63. doi: 10.1016/0165-0173(86)90009-3

37. Grillner S. Control of locomotion in bipeds, tetrapods, and fish In: VB Brooks, editor. *The nervous system II*. Bethesda: American Physiological Society Press (1981). 1179–236.

38. Mori S. Integration of posture and locomotion in acute decerebrate cats and in awake, free moving cats. *Prog Neurobiol*. (1987) 28:161–96. doi: 10.1016/0301-0082(87)90010-4

39. Prentice S, Drew T. Contributions of the reticulospinal system to the postural adjustments occurring during voluntary gait modifications. *J Neurophysiol*. (2001) 85:679–98. doi: 10.1152/jn.2001.85.2.679

40. Rossignol S In: LB Rowell, editor. *Neural control of stereotypic limb movements*. New York, NY: Oxford University Press (1996). 173–216.

41. Takakusaki K. Neurophysiology of gait: from the spinal cord to the frontal lobe. *Mov Disord*. (2013) 28:1483–91. doi: 10.1002/mds.25669

42. Grillner S, Georgopoulos AP, Jordan LM. Selection and initiation of motor behavior In: S PSG, editor. *Neurons, networks, and motor behavior*. Cambridge, MA: MIT Press (1999). 3–19.

43. Nagahama Y, Okada T, Katsumi Y, Hayashi T, Yamauchi H, Oyanagi C, et al. Dissociable mechanisms of attentional control within the human prefrontal cortex. *Cereb Cortex*. (2001) 11:85–92. doi: 10.1093/cercor/11.1.85

44. Morgane P, Galler J, Mokler D. A review of systems and networks of the limbic forebrain/limbic midbrain. *Prog Neurobiol*. (2005) 75:143–60. doi: 10.1016/j.pneurobio.2005.01.001

45. Carter CS, Braver TS, Barch DM, Botvinick MM, Noll D, Cohen JD. Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*. (1998) 280:747–9. doi: 10.1126/science.280.5364.747

46. Falkenstein M, Hoormann J, Christ S, Hohnsbein J. ERP components on reaction errors and their functional significance: a tutorial. *Biol Psychol*. (2000) 51:87–107. doi: 10.1016/S0301-0511(99)00031-9

47. Florio TM, Scarnati E, Rosa I, Di Censo D, Ranieri B, Cimini A, et al. The basal ganglia: more than just a switching device. *CNS Neurosci Ther*. (2018) 24:677–84. doi: 10.1111/cns.12987

48. Graybiel AM. Basal ganglia: habit In: LR Squire, editor. *Encyclopedia of neuroscience. 9th* ed: Amsterdam, The Netherlands: Academic Press (2009). 93–6.

49. Dixon ML, Christoff K. The lateral prefrontal cortex and complex value-based learning and decision making. *Neurosci Biobehav Rev*. (2014) 45:9–18. doi: 10.1016/j.neubiorev.2014.04.011

50. Etkin A, Büchel C, Gross JJ. The neural bases of emotion regulation. *Nat Rev Neurosci*. (2015) 16:693–700. doi: 10.1038/nrn4044

51. Mihara M, Miyai I, Hatakenaka M, Kubota K, Sakoda S. Role of the prefrontal cortex in human balance control. *NeuroImage*. (2008) 43:329–36. doi: 10.1016/j.neuroimage.2008.07.029

52. Mangold SA, Das M. *Neuroanatomy, reticular formation*. Treasure Island, Florida: StatPearls Publishing (2022).

53. Falco ED, Solca M, Bernasconi F, Rabo-Rebelo M, Young N, Sammartino F, et al. Single neurons in thalamus and subthalamic nucleus process cardiac and respiratory signals in humans. *bioRxiv* (2022);1–39, doi: 10.1101/2022.07.25.501133. [Epub ahead of preprint]

54. Adkin AL, Carpenter MG. New insights on emotional contributions to human postural control. *Front Neurol*. (2018) 9:9. doi: 10.3389/fneur.2018.00789

55. Blessing WW. *The lower brainstem and bodily homeostasis*. Oxford: Oxford University Press (1997).

56. Saper CB. The central autonomic nervous system: conscious visceral perception and autonomic pattern generation. *Annu Rev Neurosci*. (2002) 25:433–69. doi: 10.1146/annurev.neuro.25.032502.111311

57. Jänig W. *Integrative action of the autonomic nervous system*. Cambridge: Cambridge University Press (2006).

58. Jerath R, Crawford MW, Barnes VA, Harden K. Self-regulation of breathing as a primary treatment for anxiety. *Appl Psychophysiol Biofeedback*. (2015) 40:107–15. doi: 10.1007/s10484-015-9279-8

59. Wu Y, Gao Z, Chen B, Koval OM, Singh MV, Guan X, et al. Calmodulin kinase II is required for fight or flight sinoatrial node physiology. *Proc Natl Acad Sci U S A*. (2009) 106:5972–7. doi: 10.1073/pnas.0806422106

60. Liu QH, Zheng YM, Korde AS, Yadav VR, Rathore R, Wess J, et al. Membrane depolarization causes a direct activation of G protein-coupled receptors leading to local Ca^{2+} release in smooth muscle. *Proc Natl Acad Sci U S A*. (2009) 106:11418–23. doi: 10.1073/pnas.0813307106

61. Chu B, Marwaha K, Sanvictores T, Ayers D. *Physiology, stress response*. Treasure Island, Florida: StatPearls Publishing (2022).

62. Young WR, Mark Williams A. How fear of falling can increase fall-risk in older adults: applying psychological theory to practical observations. *Gait Posture*. (2015) 41:7–12. doi: 10.1016/j.gaitpost.2014.09.006

63. Hadjistavropoulos T, Delbaere K, Fitzgerald TD. Reconceptualizing the role of fear of falling and balance confidence in fall risk. *J Aging Health*. (2011) 23:3–23. doi: 10.1177/0898264310378039

64. Staab JP. Threat assessment and locomotion: clinical applications of an integrated model of anxiety and postural control. *Semin Neurol*. (2013) 33:297–306. doi: 10.1055/s-0033-1356462

65. Polskaia N, Richer N, Dionne E, Lajoie Y. Continuous cognitive task promotes greater postural stability than an internal or external focus of attention. *Gait Posture*. (2015) 41:454–85. doi: 10.1016/j.gaitpost.2014.11.009

66. Hainaut JP, Caillet G, Lestienne FG, Bolmont B. The role of trait anxiety on static balance performance in control and anxiogenic situations. *Gait Posture*. (2011) 33:604–8. doi: 10.1016/j.gaitpost.2011.01.017

67. Coco M, As F, Perciavalle V, Maci T, Mc P, Perciavalle V. Stress exposure and postural control in young females. *Mol Med Rep*. (2015) 11:2135–40. doi: 10.3892/mmr.2014.2898

68. Miller GE, Chen E, Zhou ES. If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychol Bull*. (2007) 133:25–45. doi: 10.1037/0033-2909.133.1.25

69. Fries E, Dettenborn L, Kirschbaum C. The cortisol awakening response (CAR): facts and future directions. *Int J Psychophysiol*. (2009) 72:67–73. doi: 10.1016/j.ijpsycho.2008.03.014

70. Storm H, Myre K, Rostrup M, Stokland O, Lien MD, Raeder JC. Skin conductance correlates with perioperative stress. *Acta Anaesthesiol Scand*. (2002) 46:887–95. doi: 10.1034/j.1399-6576.2002.460721.x

71. Gomez P, Stahel WA, Danuser B. Respiratory responses during affective picture viewing. *Biol Psychol*. (2004) 67:359–73. doi: 10.1016/j.biopsycho.2004.03.013

72. Masaoka Y, Homma I. The effect of anticipatory anxiety on breathing and metabolism in humans. *Respir Physiol*. (2001) 128:171–7. doi: 10.1016/S0034-5687(01)00278-X

73. Leung RST, Floras JS, Bradley TD. Respiratory modulation of the autonomic nervous system during Cheyne–Stokes respiration. *Can J Physiol Pharmacol*. (2006) 84:61–6. doi: 10.1139/Y05-145

74. Stephens RJ, Haas M, Moore WL, Emmil JR, Sipress JA, Williams A. Effects of diaphragmatic breathing patterns on balance: a preliminary clinical trial. *J Manip Physiol Ther*. (2017) 40:169–75. doi: 10.1016/j.jmpt.2017.01.005

75. Kim SH, Park SY. Effect of hip position and breathing pattern on abdominal muscle activation during curl-up variations. *J Exerc Rehabil*. (2018) 14:445–50. doi: 10.12965/jer.1836170.085

76. Ishida H, Watanabe S. Maximum expiration activates the abdominal muscles during side bridge exercise. *J Back Musculoskelet Rehabil*. (2014) 27:481–4. doi: 10.3233/BMR-140469

77. Yoon T, Kim KS, Cynn HS. Slow expiration reduces sternocleidomastoid activity and increases transversus abdominis and internal oblique muscle activity during abdominal curl-up. *J Electromyogr Kinesiol*. (2014) 24:228–32. doi: 10.1016/j.jelekin.2013.10.003

78. Iizuka M. Respiration-related control of abdominal motoneurons. *Respir Physiol Neurobiol*. (2011) 179:80–8. doi: 10.1016/j.resp.2011.01.003

79. Iwasawa T, Kagei S, Gotoh T, Yoshiike Y, Matsushita K, Kurihara H, et al. Magnetic resonance analysis of abnormal diaphragmatic motion in patients with emphysema. *Eur Respir J*. (2002) 19:225–31. doi: 10.1183/09031936.02.00044602

80. Barr KP, Griggs M, Cadby T. Lumbar stabilization. *Am J Phys Med Rehabil*. (2005) 84:473–80. doi: 10.1097/01.phm.0000163709.70471.42

81. Pirker W, Asenbaum S, Hauk M, Kandlhofer S, Tauscher J, Willeit M, et al. Imaging serotonin and dopamine transporters with 123I-beta-CIT SPECT: binding kinetics and effects of normal aging. *J Nucl Med*. (2000) 41:36–44.

82. Kuikka JT, Bergstrom KA, Telaranta T. Serotonin transporter density after trans-thoracic sympathetic block in patients with phobic avoidance episodes: preliminary findings. *Eur J Nucl Med*. (2000) 27:1119.

83. File SE, Kenny PJ, Cheeta S. The role of the dorsal hippocampal serotonergic and cholinergic systems in the modulation of anxiety. *Pharmacol Biochem Behav*. (2000) 66:65–72. doi: 10.1016/S0091-3057(00)00198-2

84. Hodges PW, Gandevia SC, Richardson CA. Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers. *J Appl Physiol*. (1997) 83:753–60. doi: 10.1152/jappl.1997.83.3.753

85. Malliani A. The visceral nervous system as a mediator of psychosocial risk factors: therapeutic implications of its specific organization. *Act Nerv Super*. (1982) 3:504–12.

86. Brooks CM. Newer concepts of the autonomic system's role derived from reductionist and behavioral studies of various animal species. *J Auton Nerv Syst*. (1983) 7:199–212. doi: 10.1016/0165-1838(83)90073-5

87. Hugdahl K. Cognitive influences on human autonomic nervous system function. *Curr Opin Neurobiol*. (1996) 6:252–8. doi: 10.1016/S0959-4388(96)80080-8

88. Caron O, Fontanari P, Cremieux J, Joulia F. Effects of ventilation on body sway during human standing. *Neurosci Lett*. (2004) 366:6–9. doi: 10.1016/j.neulet.2004.04.085

89. Malakhov MV, Melnikov AA. Postural performance at different breathing strategies in patients with limitation of mobility. *Fiziol Cheloveka*. (2017) 43:47–53. doi: 10.1134/S0362119716060116

90. Maki BE, McIlroy WE. Influence of arousal and attention on the control of postural sway. *J Vestib Res*. (1996) 6:53–9. doi: 10.3233/VES-1996-6107

91. Stapley PJ, Drew T. The pontomedullary reticular formation contributes to the compensatory postural responses observed following removal of the support surface in the standing cat. *J Neurophysiol*. (2009) 101:1334–50. doi: 10.1152/jn.91013.2008

92. Sibley KM, Mochizuki G, Lakhani B, McIlroy WE. Autonomic contributions in postural control: a review of the evidence. *Rev Neurosci*. (2014) 25:687–97. doi: 10.1515/revneuro-2014-0011

93. Boucsein W. *Electrodermal activity*. New York: Spinger (2012).

94. Sara SJ, Bouret S. Orienting and reorienting: the locus coeruleus mediates cognition through arousal. *Neuron*. (2012) 76:130–41. doi: 10.1016/j.neuron.2012.09.011

95. Carpenter MG, Frank JS, Silcher CP. Surface height effects on postural control: a hypothesis for a stiffness strategy for stance. *J Vestib Res*. (1999) 9:277–86. doi: 10.3233/VES-1999-9405

96. Brown LA, Polych MA, Doan JB. The effect of anxiety on the regulation of upright standing among younger and older adults. *Gait Posture*. (2006) 24:397–405. doi: 10.1016/j.gaitpost.2005.04.013

97. Sibley KM, Carpenter MG, Perry JC, Frank JS. Effects of postural anxiety on the soleus H-reflex. *Hum Mov Sci*. (2007) 26:103–12. doi: 10.1016/j.humov.2006.09.004

98. Horslen BC, Murnaghan CD, Inglis JT, Chua R, Carpenter M. Effects of postural threat on spinal stretch reflexes: evidence for increased muscle spindle sensitivity? *J Neurophysiol*. (2013) 110:899–906. doi: 10.1152/jn.00065.2013

99. Hodgson DD, Darici O, Dalton BH, Cleworth TW, Cluff T, Peters RM, et al. Visual feedback-dependent modulation of arousal, postural control, and muscle stretch reflexes assessed in real and virtual environments. *Front Hum Neurosci*. (2023) 17:17. doi: 10.3389/fnhum.2023.1128548

100. Cleworth TW, Carpenter MG. Postural threat influences conscious perception of postural sway. *Neurosci Lett*. (2016) 620:127–31. doi: 10.1016/j.neulet.2016.03.032

101. Cleworth TW, Inglis JT, Carpenter MG. Postural threat influences the conscious perception of body position during voluntary leaning. *Gait Posture*. (2018) 66:21–5. doi: 10.1016/j.gaitpost.2018.08.003

102. Tyng CM, Amin HU, Saad MNM, Malik AS. The influences of emotion on learning and memory. *Front Psychol*. (2017) 8:1454. doi: 10.3389/fpsyg.2017.01454

103. Vuilleumier P. How brains beware: neural mechanisms of emotional attention. *Trends Cogn Sci*. (2005) 9:585–94. doi: 10.1016/j.tics.2005.10.011

104. Huffman JL, Horslen BC, Carpenter MG, Adkin AL. Does increased postural threat lead to more conscious control of posture? *Gait Posture*. (2009) 30:528–32. doi: 10.1016/j.gaitpost.2009.08.001

105. Zaback M, Cleworth TW, Carpenter MG, Adkin AL. Personality traits and individual differences predict threat-induced changes in postural control. *Hum Mov Sci*. (2015) 40:393–409. doi: 10.1016/j.humov.2015.01.015

106. Johnson K, Watson A, Tokuno C, Carpenter M. The effects of distraction on threat-related changes in standing balance control. *Neurosci Lett*. (2020) 716:134635. doi: 10.1016/j.neulet.2019.134635

107. Zaback M. Threat-induced changes in attention during tests of static and anticipatory postural control. *Gait Posture*. (2016) 45:19–24. doi: 10.1016/j.gaitpost.2015.12.033

108. Johnson K, Zaback M, Tokuno C, Carpenter M. Exploring the relationship between threat-related changes in anxiety, attention focus, and postural control. *Psychol Res*. (2019) 83:445. doi: 10.1007/s00426-017-0940-0

109. Ellemers TJ, Kal EC, Young WR. Consciously processing balance leads to distorted perceptions of instability in older adults. *J Neurol*. (2021) 268:1374–84. doi: 10.1007/s00415-020-10288-6

110. Kal EC, Young WR, Ellmers TJ. Balance capacity influences the effects of conscious movement processing on postural control. *Hum Mov Sci*. (2022) 82:102933–11. doi: 10.1016/j.humov.2022.102933

111. Boisgontier M, Beets IAM, Duysens J, Nieuwboer A, Krampe RT, Swinnen SP. Age-related differences in attentional cost associated with postural dual tasks: increased recruitment of generic cognitive resources in older adults. *Biobehav Rev*. (2013) 37:1824–37. doi: 10.1016/j.neubiorev.2013.07.014

112. Chow V, Ellmers T, Young W, Mak T, Wong T. Revisiting the relationship between internal focus and balance control in young and older adults. *Front Neurol*. (2019) 9:1131. doi: 10.3389/fneur.2018.01131

113. Leung T, Mak T, Wong T. Real-time conscious postural control is not affected when balancing on compliant surface by young adults. *J Mot Behav*. (2022) 54:37–43. doi: 10.1080/00222895.2021.1879724

114. Jie L, Kal E, Ellmers TJ, Rosier J, Meijer K, Boonstra TW. The effects of conscious movement processing on the neuromuscular control of posture. *Neuroscience*. (2022) 509:63–73. doi: 10.1016/j.neuroscience.2022.11.010

115. Zaback M, Adkin AL, Carpenter M. Adaptation of emotional state and standing balance parameters following repeated exposure to height-induced postural threat. *Sci Rep*. (2019) 9:12449. doi: 10.1038/s41598-019-48722-z

116. Zaback M, Reiter E, Adkin AL, Carpenter M. Initial experience of balance assessment introduces 'first trial' effects on emotional state and postural control. *Gait Posture*. (2021) 88:116–21. doi: 10.1016/j.gaitpost.2021.05.013

117. Johnson K, Zaback M, Tokuno C, Carpenter M. Repeated exposure to the threat of perturbation induces emotional, cognitive, and postural adaptations in young and older adults. *Exp Gerontol*. (2019) 122:109–15. doi: 10.1016/j.exger.2019.04.015

118. Ellmers T, Wilson W, Kal W, Young W. Standing up to threats: translating the two-system model of fear to balance control in older adults. *Exp Gerontol*. (2022) 158:111647. doi: 10.1016/j.exger.2021.111647

119. Doumas M, Morsanyi K, Young W. Cognitively and socially induced stressed affects postural control. *Exp Brain Res*. (2018) 236:305–14. doi: 10.1007/s00221-017-5128-8

120. Lang PJ, Bradley MM, Cuthbert BN. *International Affective Picture System (IAPS): affective ratings of pictures and instruction manual. Technical Report A-8*. Gainesville, FL: University of Florid (2008).

121. Lelard T, Stins J, Mouras H. Postural responses to emotional visual stimuli. *Neurophysiol Clin*. (2019) 49:109–14. doi: 10.1016/j.neucli.2019.01.005

122. Stins JF, Beek PJ. Effects of affective picture viewing on postural control. *BMC Neurosci*. (2007) 8:83. doi: 10.1186/1471-2202-8-83

123. Roelofs K, Hagenaars MA, Stins J. Facing freeze: social threat induces bodily freeze in humans. *Psychol Sci*. (2010) 21:1575–81. doi: 10.1177/0956797610384746

124. Lang PJ, Bradley MM. Emotion and the motivational brain. *Biol Psychol*. (2010) 84:437–50. doi: 10.1016/j.biopsycho.2009.10.007

125. Horslen BC, Carpenter MG. Arousal, valence and their relative effects on postural control. *Exp Brain Res*. (2011) 215:27–34. doi: 10.1007/s00221-011-2867-9

126. Balaban C. Neural substrates linking balance control and anxiety. *Physiol Behav*. (2002) 77:469–75. doi: 10.1016/S0031-9384(02)00935-6

127. Balaban CD, Jacob RG, Furman JM. Neurologic bases for comorbidity of balance disorders, anxiety disorders and migraine: neurotherapeutic implications. *Expert Rev Neurother*. (2011) 11:379–94. doi: 10.1586/ern.11.19

128. Feldman R, Schreiber S, Pick C, Been E. Gait, balance and posture in major mental illnesses: depression, anxiety and schizophrenia. *Austin Med Sci*. (2020) 5:1039.

129. Redfern MS, Furman JM, Jacob RG. Visually induced postural sway in anxiety disorders. *J Anxiety Disord*. (2007) 21:704–16. doi: 10.1016/j.janxdis.2006.09.002

130. Levitan MN, Crippa JA, Bruno LM, Pastore DL, Freire RC, Arrais KC, et al. Postural balance in patients with social anxiety disorder. *Braz J Med Biol Res*. (2012) 45:38–42. doi: 10.1590/S0100-879X2011007500155

131. Pluess M, Conrad A, Wilhelm FH. Muscle tension in generalized anxiety disorder: a critical review of the literature. *J Anxiety Disord*. (2009) 23:1–11. doi: 10.1016/j.janxdis.2008.03.016

132. Kret ME, Ploeger A. Emotion processing deficits: a liability spectrum providing insight into comorbidity of mental disorders. *Neurosci Biobehav Rev*. (2015) 52:153–71. doi: 10.1016/j.neubiorev.2015.02.011

133. Ricciardi L, Demartini B, Fotopoulou A, Edwards MJ. Alexithymia in neurological disease: a review. *J Neuropsychiatry Clin Neurosci*. (2015) 27:179–87. doi: 10.1176/appi.neuropsych.14070169

134. Avanzino L, Lagravinese G, Abbruzzese G, Pelosin E. Relationships between gait and emotion in Parkinson's disease: a narrative review. *Gait Posture*. (2018) 65:57–64. doi: 10.1016/j.gaitpost.2018.06.171

135. Chaudhary S, Zhornitsky S, Chao HH, van Dyck CH, Li CSR. Emotion processing dysfunction in Alzheimer's disease: an overview of behavioral findings, systems neural correlates, and underlying neural biology. *Am J Alzheimers Dis Other Dement*. (2022) 37:15333175221082834. doi: 10.1177/15333175221082834

136. Kearney BE, Lanius RA. The brain-body disconnect: a somatic sensory basis for trauma-related disorders. *Front Neurosci*. (2022) 21:1015749. doi: 10.3389/fnins.2022.1015749

137. Bornhofen C, Mcdonald S. Emotion perception deficits following traumatic brain injury: a review of the evidence and rationale for intervention. *J Int Neuropsychol Soc*. (2008) 14:511–25. doi: 10.1017/S1355617708080703

138. Vahey R, Becerra R. Galvanic skin response in mood disorders: a critical review. *Int J Psychol. Psychol Ther*. (2015) 15:275–304.

139. Sarchiapone M, Gramaglia C, Iosue M, Carli V, Mandelli L, Serretti A, et al. The association between electrodermal activity (EDA), depression and suicidal behaviour: a systematic review and narrative synthesis. *BMC Psychiatry*. (2018) 18:22. doi: 10.1186/s12888-017-1551-4

140. Diamond AE, Fisher AJ. Comparative autonomic responses to diagnostic interviewing between individuals with GAD, MDD, SAD and healthy controls. *Front Hum Neurosci*. (2017) 10:677. doi: 10.3389/fnhum.2016.00677

141. Hu MX, Lamers F, de Geus EJC, Penninx BWJH. Differential autonomic nervous system reactivity in depression and anxiety during stress depending on type of stressor. *Psychosom Med*. (2016) 78:562–72. doi: 10.1097/PSY.0000000000000313

142. Martínez-Velázquez ES, Honoré J, de Zorzi L, Ramos-Loyo J, Sequeira H. Autonomic reactivity to arousing stimuli with social and non-social relevance in alexithymia. *Front Psychol*. (2017) 8:361. doi: 10.3389/fpsyg.2017.00361

143. Baschnagel JS, Coffey SF, Hawk LW, Schumacher JA, Holloman G. Psychophysiological assessment of emotional processing in patients with borderline personality disorder with and without comorbid substance use. *Personal Disord Theory Res Treat*. (2013) 4:203–13. doi: 10.1037/a0029777

144. Low PA. Prevalence of orthostatic hypotension. *Clin Auton Res*. (2008) 18:8–13. doi: 10.1007/s10286-007-1001-3

145. Palma J, Kaufmann H. Treatment of autonomic dysfunction in Parkinson disease and other synucleinopathies. *Mov Disord*. (2018) 33:372–90. doi: 10.1002/mds.27344

146. Zalewski P, Słomko J, Zawadka-Kunikowska M. Autonomic dysfunction and chronic disease. *Br Med Bull*. (2018) 128:61–74. doi: 10.1093/bmb/ldy036

147. Chang HA, Fang WH, Wan FJ, Tzeng NS, Liu YP, Shyu JF, et al. Attenuated vagally-mediated heart rate variability at rest and in response to postural maneuvers in patients with generalized anxiety disorder. *Psychol Med*. (2020) 50:1433–41. doi: 10.1017/S0033291719001302

148. Hallam RS, Stephens SDG. Vestibular disorder and emotional distress. *J Psychosom Res*. (1985) 29:407–13. doi: 10.1016/0022-3999(85)90026-1

149. Rolf J, Furman JM. Psychiatric consequences of vestibular dysfunction. *Curr Opin Neurol*. (2001) 14:41–6. doi: 10.1097/00019052-200102000-00007

150. Rajagopalan A, Jinu KV, Sailesh KS, Mishra S, Reddy UK, Mukkadan JK. Understanding the links between vestibular and limbic systems regulating emotions. *J Nat Sci Biol Med*. (2017) 8:11–5. doi: 10.4103/0976-9668.198350

151. Preuss N, Hasler G, Mast FW. Caloric vestibular stimulation modulates affective control and mood. *Brain Stimul*. (2014) 7:133–40. doi: 10.1016/j.brs.2013.09.003

152. Yates BJ, Bronstein AM. The effects of vestibular system lesions on autonomic regulation: observations, mechanisms, and clinical implications. *J Vestib Res*. (2005) 15:119–29. doi: 10.3233/VES-2005-15301

153. Mast FW, Preuss N, Hartmann M, Grabherr L. Spatial cognition, body representation and affective processes: the role of vestibular information beyond ocular reflexes and control of posture. *Front Integr Neurosci*. (2014) 8:44. doi: 10.3389/fnint.2014.00044

154. Harmon-Jones C, Bastian B, Harmon-Jones E. The discrete emotions questionnaire: a new tool for measuring state self-reported emotions. *PLoS One*. (2016) 11:e0159915. doi: 10.1371/journal.pone.0159915

155. Giggey K, Tope R. Poor postural control may signal autonomic imbalances: a cross sectional pilot study. *J Chiropr Edu*. (2008):11.

156. You S, Kim H, Lee H. Association of postural instability with autonomic dysfunction in early Parkinson's disease. *J Clin Med*. (2020) 9:3786. doi: 10.3390/jcm9113786

157. McCraty R, Zayas MA. Cardiac coherence, self-regulation, autonomic stability, and psychosocial well-being. *Front Psychol*. (2014) 5:1–13. doi: 10.3389/fpsyg.2014.01090

158. McCraty R, Atkinson M, Tomasino D, Bradley RT. The coherent heart: heart-brain interactions, psychophysiological coherence, and the emergence of system-wide order. *Integr Rev*. (2009) 5:10–115.

159. Beauchaine TP, Thayer JF. Heart rate variability as a transdiagnostic biomarker of psychopathology. *Int J Psychophysiol*. (2015) 98:338–50. doi: 10.1016/j.ijpsycho.2015.08.004

160. Kemp AH, Quintana DS. The relationship between mental and physical health: insights from the study of heart rate variability. *Int J Psychophysiol*. (2013) 89:288–96. doi: 10.1016/j.ijpsycho.2013.06.018

161. Shaffer F, McCraty R, Zerr CL. A healthy heart is not a metronome: an integrative review of the heart's anatomy and heart rate variability. *Front Psychol*. (2014) 5:5. doi: 10.3389/fpsyg.2014.01040

162. Ottaviani C, Thayer JF, Verkuil B, Lonigro A, Medea B, Couyoumdjian A, et al. Physiological concomitants of perseverative cognition: a systematic review and meta-analysis. *Psychol Bull*. (2016) 142:231–59. doi: 10.1037/bul0000036

163. Chalmers JA, Quintana DS, Abbott MJ, Kemp AH. Anxiety disorders are associated with reduced heart rate variability: a meta-analysis. *Front Psych*. (2014) 5:5. doi: 10.3389/fpsyt.2014.00080

164. Appelhans BM, Luecken LJ. Heart rate variability as an index of regulated emotional responding. *Rev Gen Psychol*. (2006) 10:229. doi: 10.1037/1089-2680.10.3.229

165. Kim M, Kim M, Oh E, Kim S. A review on the computational methods for emotional state estimation from the human EEG. *Comput Math Methods Med*. (2013) 2013:1–13. doi: 10.1155/2013/573734

166. Bruijn SM, Van Dieën JH, Daffertshofer A. Beta activity in the premotor cortex is increased during stabilized as compared to normal walking. *Front Hum Neurosci*. (2015) 9:593. doi: 10.3389/fnhum.2015.00593

167. Sipp AR, Gwin JT, Makeig S, Ferris DP. Loss of balance during balance beam walking elicits a multifocal theta band electrocortical response. *J Neurophysiol*. (2013) 110:2050–60. doi: 10.1152/jn.00744.2012

168. Bolton DAE. The role of the cerebral cortex in postural responses to externally induced perturbations. *Neurosci Biobehav Rev*. (2015) 57:142–55. doi: 10.1016/j.neubiorev.2015.08.014

169. Payne AM, Hajcak G, Ting LH. Dissociation of muscle and cortical response scaling to balance perturbation acceleration. *J Neurophysiol*. (2019) 121:867–80. doi: 10.1152/jn.00237.2018

170. Payne AM, Ting LH, Hajcak G. Do sensorimotor perturbations to standing balance elicit an error-related negativity? *Psychophysiology*. (2019) 56:e13359. doi: 10.1111/psyp.13359

171. Varghese JP, McIlroy RE, Barnett-Cowan M. Perturbation-evoked potentials: significance and application in balance control research. *Neurosci Biobehav Rev*. (2017) 83:267–80. doi: 10.1016/j.neubiorev.2017.10.022

172. Cameron OG. *Isceral sensory neuroscience: interoception*. New York, NY: Oxford University Press (2001).

173. Kim DK, Rhee JH, Kang SW. Reorganization of the brain and heart rhythm during autogenic meditation. *Front Integr Neurosci*. (2014) 7:109. doi: 10.3389/fnint.2013.00109

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Effects of protective step training on proactive and reactive motor adaptations in Parkinson's disease patients

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The aim of this study was to investigate to what extent PD affects the ability to walk, respond to balance perturbations in a single training session, and produce acute short-term effects to improve compensatory reactions and control of unperturbed walking stability. Understanding the mechanism of compensation and neuroplasticity to unexpected step perturbation training during walking and static stance can inform treatment of PD by helping to design effective training regimens that remediate fall risk. Current rehabilitation therapies are inadequate at reducing falls in people with Parkinson's disease (PD). While pharmacologic and surgical treatments have proved largely ineffective in treating postural instability and gait dysfunction in people with PD, studies have demonstrated that therapy specifically focusing on posture, gait, and balance may significantly improve these factors and reduce falls. The primary goal of this study was to assess the effectiveness of a novel and promising intervention therapy (protective step training – i.e., PST) to improve balance and reduce falls in people with PD. A secondary goal was to understand the effects of PST on proactive and reactive feedback responses during stance and gait tasks. Multiple-baseline, repeated measures analyses were performed on the multitude of proactive and reactive performance measures to assess the effects of PST on gait and postural stability parameters. In general, the results indicate that participants with PD were able to use experiences with perturbation training to integrate and adapt feedforward and feedback behaviors to reduce falls. The ability of the participants with PD to adapt to changes in task demands suggests that individuals with PD could benefit from the protective step training to facilitate balance control during rehabilitation.

KEYWORDS

Parkinson's disease, accidental falls, protective step training, motor learning, gait and balance, motor adaptability, feedforward and feedback, physical therapy

1. Introduction

Parkinson's disease (PD) is a neurological disorder characterized by bradykinesia, tremor, rigidity, postural instability and, affects an estimated 1 million individuals in the US. Postural Instability and Gait Dysfunction (PIGD), a subset of PD symptoms describing impaired standing posture and balance, bradykinetic gait features, freezing of gait (FOG), and falls, is a disabling condition that, unlike other cardinal features of PD, is often not adequately treated by dopaminergic medications. Fall incidence rates among the PD population are estimated to range as high as 50–70%, with many individuals suffering recurrent falls, and these falls are a major cause of injury and disability. It is estimated that healthcare expenditures related to these falls exceeded $27 billion in 2013 (1). As the population of older adults (>65 years old) in the US increases over the coming decades, reaching a projected 98 million by 2060 (2), the rates of PD and associated falls are expected to rise dramatically. Although modern medicine and new medical technologies offer enormous potential to improve the diagnosis and treatment of many symptoms, falls still represent a major and largely untreated problem for PD patients. While pharmacologic and surgical treatments have proved largely ineffective in treating PIGD thus far (3–9), studies have demonstrated that therapy specifically focusing on posture, gait, and balance may significantly improve these factors and reduce falls (10–13).

Perturbation-based balance training, or protective step training (PST), defined as balance training using repeated, external perturbations, is one such method of therapy that has demonstrated improvements in balance and fall recovery in multiple populations (14). Several studies have observed decreased fall rate and an increased ability to recover from a fall upon repeated exposure to a perturbation in healthy controls (15, 16). Investigators have reported that adaptations to avoid falling can be modulated *via* both feedforward (predictive) and feedback (reactive) mechanisms (17). Predictive mechanisms of recovery involve changes to gait parameters such as base of support, trunk angle, and velocity, that may reduce the magnitude of the required balance recovery response upon delivery of a perturbation. Reactive mechanisms of improvement may involve earlier detection of perturbation, likely to require recovery response, and improved motor responses triggering increased relevant muscle action and fewer maladaptive movements following perturbation (17). Studies suggest that reactive balance may not be entirely intact in PD, but that learning is still possible (3–9), making it important to study the effects of PST in this population.

Recent literature for PST in populations with PD is represented in Table 1. Studies indicate that PST may be useful in improving gait and postural control that precipitate future falls (23), however, there is a lack of consistency regarding the specific improvements and whether those improvements can effectively transfer from a rehabilitation setting to activities of daily living. Studies have shown that training is more effective if it is specific to the skill to be improved (23), and while perturbation-training in PD is ongoing, the majority of research is not task-specific and only a few studies have attempted to replicate common causes of falls (e.g., slips and trips) (19–21, 24). To this end, the specificity of PST is in need of further study to determine what types of perturbation are most effective at inducing adaptive response and what intensity, frequency, and duration of perturbation training sessions are required for these results to be retained.

The primary goal of this study was to assess the effectiveness of two kinds of PST (anterior translations of a split-belt forceplate during (1) forward gait and (2) static postural stability tasks) to improve balance and stability in people with PD by better understanding the effects of PST on proactive and reactive feedback responses during stance and gait. We hypothesized that PD patients will be able to learn in an explicit, feedforward manner, adjusting base of support prior throughout walking trials to prepare for unexpected perturbation, but may be unable to improve reactive response variables such as reaction time and strategy. This work may enhance the clinician's ability to treat balance/gait disturbances leading to falls in people with PD utilizing protective step training.

2. Materials and methods

2.1. Participants

Twelve participants diagnosed with PD by a movement disorders neurologist were recruited for this study (age = 62 ± 7.1; 9 males, 3 females). Participants were included in the study if they were able to ambulate without assistance, had no known neurologic, cardiovascular, or orthopedic deficit that could significantly impact cognition and functional performance (Mini-Mental Status Examination <25), and had a Hoehn & Yahr (H&Y) score between II-III. Subjects were excluded if they exhibited functionally disabling dyskinesia or dystonia, orthostatic hypotension, neurosurgical intervention (deep brain stimulation), and any significant musculoskeletal or metabolic disorders. All subjects were examined during the "on" dopaminergic medication state, having taken their last dose approximately 1–1.5 h prior to testing. Disease severity and clinical scales of symptoms were tested in the "on" state utilizing the H&Y scale (25) and the motor subscale of the International Parkinson and Movement Disorder Society-Sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS Part III). Subjects in the present study had an average H&Y score of 2.7 and an average disease duration of approximately 3.5 ± 3.1 years. Prior to testing, subjects were randomly assigned to two groups. One group started with postural perturbation (PP) training (during stance), while the other group began with gait perturbation (GP) training before crossing over (during walking). During this onboarding period, self-reported and observed leg dominance in bilateral mobilizing was utilized to determine the dominant leg that will be perturbed in the walking trials. Investigators described a scenario for the participant in which they were asked which leg they would use to kick a ball over the ground. This study (experimental procedures and design) was approved by the Institutional Review Board at Arizona State University and performed according to the declaration of Helsinki. All participants provided written informed consent prior to data collection.

2.2. Study design

Prior to baseline testing, two 3-min walking sessions were given to the participants to familiarize themselves with the treadmill and the laboratory environment. The first session was primarily for familiarizing and adapting to the treadmill. Following this session, the participant was given a rest period and encouraged to ask questions or bring up any concerns with the task. The second session was used to standardize the participant's preferred walking speed (PWS) that they would be using throughout the trials. In this session, walking speed was increased incrementally until participants indicated that the speed was consistent with their normal walking speed. The walking speed was then increased in 0.1 m/s increments until the participant expressed discomfort or reported the speed to be inconsistent with their normal walking

TABLE 1 Characteristics of Gait Perturbation Studies in PD.

Publication	Sample size	Perturbation Training	Protocol	Perturbation Type	Outcomes
Oates et al. (18)	$n = 8$ PD $n = 10$ age-matched controls	Overground walk with slip perturbation	Single session 15 walk trials 1 unexpected slippery stop 5 planned stops 5 cued stops	Unexpected slip perturbation during GT[1]	PD showed slower, wider steps and less stability Feedforward adaptations: shorter, wider step, modified MOS[2]. Feedback adaptations: longer, wider step.
Steib et al. (19)	$N = 38$ PD	Treadmill walking	3 months 16 sessions total 8 weeks of treadmill walking for 30 min	Three-dimensional tilting movements to the treadmill	No effect with perturbation training on gait and balance in PD patients.
Klamroth et al. (20)	$N = 39$ PD	Treadmill walking with tilting	1 session, 20 min of treadmill walking and 10 min	Three-dimensional tilting movements to the treadmill floor	Increased walking speed (overground) in PBT[3] group compared to control group. Gait variability during treadmill walking significantly decreased after walking with PBT
Martelli et al. (21)	$N = 18$ subjects - PD subjects ($n = 9$) - HOA[4] ($n = 9$)	Treadmill walking with cable perturbations	Single session, 30 minutes Treadmill walking: 9 blocks of 8 AP or ML perturbations by cables	AP & ML push or pull perturbations	Reduced stability in AP direction and proactive adaptations. Reported short-term after-effects of increased gait stability.
Hulzinga et al. (22)	$N = 52$ PD subjects - FOG[5] ($n = 22$) - Non-FOG ($n = 30$)	Treadmill training: SBT[6] & TBT[7]	*Training:* *For 4 weeks, 3x per week.* *Three 1-min walking trials: (1) TBT (baseline); (2) SBT (to assess early and late adaptation); and (3) TBT (to assess early and late de-adaptation)* Tests: 1 week prior (pre), 1 week after (post) and 4-weeks (post).	*Asymmetrical gait-speed perturbations: SBT had 50% reduction in speed on one side.*	SBT-training improved gait adaptation more than TBT, effects that were sustained at follow-up and during dual tasking. Gait speed and step length improved with SBT & TBT. Gait adaptation did not transfer to over-ground turning speed.

Gait termination[1]; margin of stability[2]; perturbation training[3]; healthy older adults[4]; freezing of gait[5]; split-belt treadmill training[6]; tied-belt treadmill training[7].

speed. Following the familiarization period, the protocol began with a series of baseline tests: 2-min of overground walking (OG$_1$), Timed Up-and-Go Test (TUG$_1$), Short Physical Performance Battery Test (SPPB$_1$), postural stability in both the eyes-open and eyes-closed conditions (PSEO$_1$, PSEC$_1$), and 2-min of treadmill walking (TW$_1$). Following baseline trials, participants in the first group commenced postural perturbation (PP) training during stance while the second group commenced gait perturbation (GP) training during walking. Both groups were instructed to maintain their balance and avoid a fall when introduced to unexpected perturbations. Upon completion of the respective training paradigms, a 'washout' period was introduced in which the groups performed a second 2-min treadmill walking trial (TW$_{2)}$ to observe any after-effects from baseline. Following this period, the two groups crossed over and commenced the alternate perturbation training paradigms. Finally, both groups performed a final 2-min treadmill walking trial (TW$_3$) along with the post-training tests performed during baseline testing: OG$_2$, TUG$_2$, SPPB$_2$, PSEO$_2$, and PSEC$_2$. All perturbations occurred on each participant's dominant leg. A schematic of the study design is presented in Figure 1. Figure 2 illustrates comparisons made in this study.

2.3. A protective step training during stance and gait

Perturbation training protocol was based on previous study on young and older adults without PD (26–29). The protective step training protocol during walking, i.e., gait perturbation (GP) training, consisted of continuously walking on a treadmill with 12 blocks (GP$_{1\text{-}12}$) of unexpected perturbations. Participants began walking unperturbed for a baseline period of approximately 15 s. The subsequent 10 s following the baseline period are designated as the perturbation window: a 10-s window in which the subject is given an unexpected anterior translation of the right treadmill belt (acceleration of 10 m/s^2; duration of 0.2 s) at the instant of right heel contact, i.e., the perturbed step. This translation resulted in the displacement of the subject's COM, in which participants were instructed to restore their balance. Following the perturbed step and the subsequent recovery duration, participants walked unperturbed until they were able to match their preferred walking speed again.

The protective step training protocol during stance, i.e., postural perturbation (PP) training, evaluated standing balance maintenance given 12 unexpected perturbation blocks (PP$_{1\text{-}12}$). Participants were instructed to stand upright with their arms by their sides and look

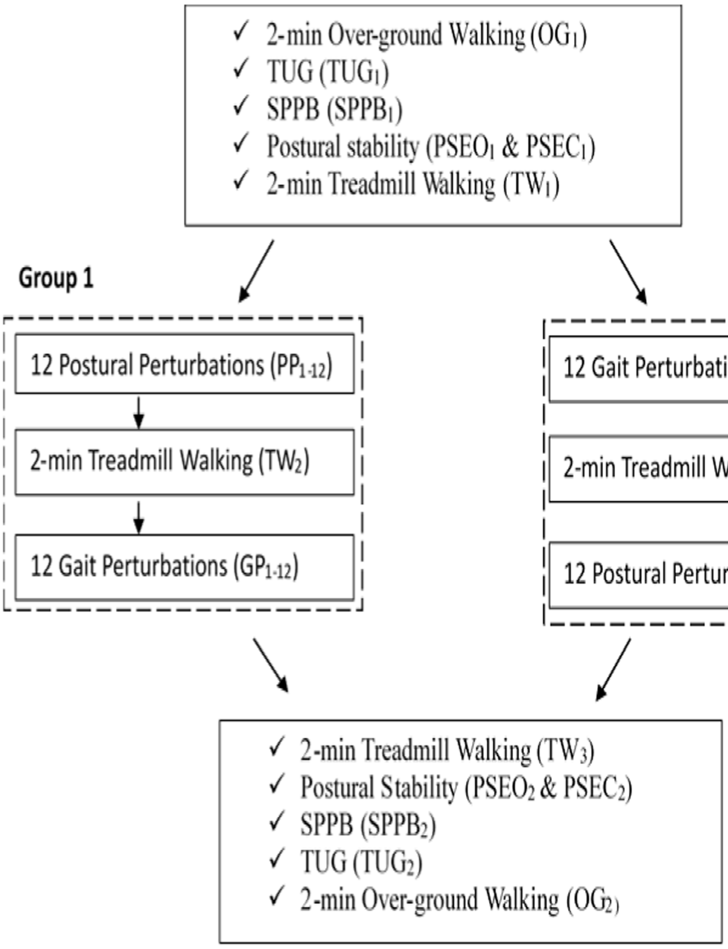

FIGURE 1
Schematic of study design.

straight ahead. The training began with participants in quiet stance for approximately 15 s. Following this static period, a simultaneous anterior translation of both treadmill belts was induced with an acceleration of 8 m/s^2 and duration of 0.1 s. Akin to the GP training, the resulting platform translation resulted in the displacement of the subject's COM, in which participants were instructed to restore their balance.

2.4. Experimental setup

GRAIL system (Gait Real-time Analysis Interactive Lab, Motek Medical, Amsterdam, the Netherlands) was utilized to simulate both types of (i.e., during standing and walking) perturbations. GRAIL consisted of a dual-belt instrumented treadmill equipped with dual embedded force plates in a speed-matched virtual environment projected on a semi-cylindrical screen (during the experiment, the virtual environment was turned off and a blank wall was projected to avoid any visual perturbation effects) (Figure 3). Subjects were equipped with standardized footwear to minimize experimental confounds, as well as a full-body harness tethered to an instrumented safety system that supported their full weight. The dual force plates embedded in the treadmill belts were utilized to collect data during postural stability trials. Lower body kinematics was recorded using 12 Vicon cameras (100 Hz; Vicon Bonita, Vicon, United States) with a modified Helen-Hayes marker set, including 25 reflective markers, which were placed in accordance with the lower body Vicon full-body Plug-in-Gait model. Motion capture data was filtered using a fourth-order low-pass Butterworth filter and a cut-off frequency of 6 Hz. Accordingly, force plate data was filtered using a fourth-order-low-pass Butterworth filter with a cut-off frequency of 10 Hz, to eliminate extraneous measurement noise. Nonlinear measures – applied during TW_1, TW_2, and TW_3 – were implemented to estimate the structure of variability, e.g., the scaling behavior (scaling exponent α) of stride intervals and the signal regularity (MSE) of center of mass (COM) velocities – were unfiltered during the analysis. All analysis was performed using custom Matlab routines (The Mathworks, Version 2016a).

2.5. Data analyses

Feedforward responses from gait were analyzed from the 15 s of unperturbed walking prior to each perturbation. Spatiotemporal parameters and gait variability were extracted from the 10 steps prior to the perturbation, while dynamic stability was examined at the final heel contact before the perturbation (pert_{hc}). Adaptive behavior was

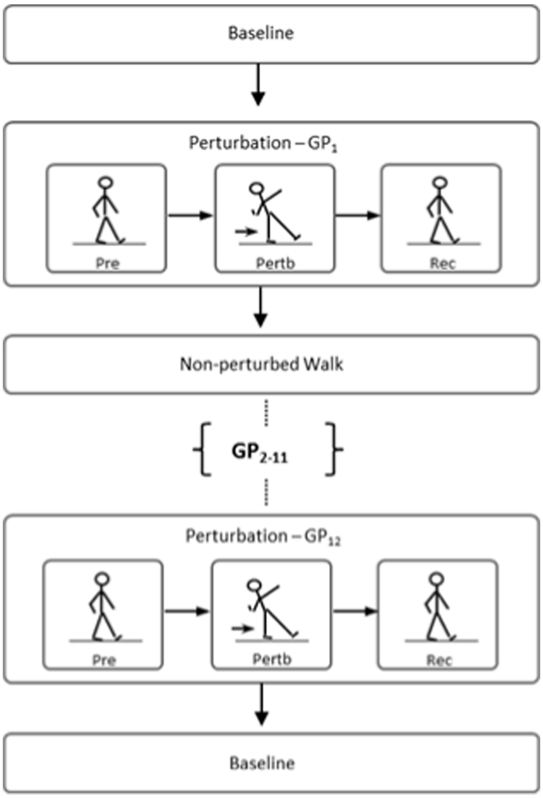

FIGURE 2
Illustration of comparison made between novel recovery from the first perturbation (i.e., GP_1 or PP_1) vs. trained recovery from the last two perturbations (i.e., $GP_{11,12}$ or $PP_{11,12}$).

determined by comparing responses from GP_{11} and GP_{12}, with GP_1. Regarding PP training, predictive postural adjustments were assessed from the 15 s of quiet standing prior to the perturbation. The whole-body center-of-mass (COM) dynamics, base of support (BOS), and angular kinematics of the trunk, knee, and ankle in the sagittal plane delineated any changes to feedforward control prior to a perturbation. Adaptive behavior was assessed by comparing responses from the last two perturbation blocks (PP_{11} and PP_{12})-Post-test-with the initial block (PP_1) – Pre-test. Joint angles were measured from $pert_{hc}$, while joint angle range of motion (ROM) was standardized from the minimum and maximum angles within the dominant leg's normalized gait cycle just prior to $pert_{hc}$. Angles were calculated by the segmental method for determining 2D joint angles by the means of cardan sequences and a 6 degrees of freedom model. Relative angle was determined between the local coordinate systems of each proximal and distal segment. The angles chosen for sagittal plane analysis were trunk flexion/extension (measured as the angle between a vertical line, perpendicular to the ground, bisecting the sacrum and a line bisecting the thoracic spine), knee flexion/extension (defined by the long axis of the tibia with respect to the long axis of the femur), and ankle plantar and dorsiflexion determined by the shank and foot segments. For knee joint angles, full extension was defined as zero degrees and movement into flexion being positive. Regarding ankle angles, zero was set at 90° to delineate plantarflexion and dorsiflexion. Plantarflexion was set as the negative degrees. In the frontal plane, lateral trunk flexion was measured as the angle between a vertical line bisecting the contralateral ASIS (perpendicular to the ground) and a line from the ASIS to the AC joint marker. Table 2 provides further operational definitions of the feedforward parameters.

Reactive feedback responses from GP training were examined by identifying the contralateral recovery step (rec_{hc}) immediately following the perturbation (Figure 4A). Dynamic stability of the first recovery step from perturbation blocks (GP_{11} and GP_{12}) was compared with the dynamic stability of the initial block (GP_1), to evaluate adaptive feedback control. For PP training, reactive modifications from the first recovery step after the perturbation (rec_{hc}) was identified as the initial recovery mechanism. The end of the recovery period was identified as the zero-cross point of the COM velocity in the anteroposterior direction (Figure 4B). Table 2 outlines the feedback parameters used to evaluate subject performance.

Dynamic stability was calculated by the margin of stability (MOS), which measures the movement of the COM relative to the base of support (30). Specifically, MOS in the anteroposterior (AP) direction, was determined by the distance between the anterior boundary of the BOS at heel contact (toe marker of the leading foot) and the extrapolated COM (30); MOS in the mediolateral (ML) direction was calculated as the difference between the lateral boundary of the BOS at heel contact (heel marker of the leading foot) and the extrapolated COM. An increased MOS indicates the COM is further within the BOS, while decreased MOS indicates COM is nearer to the limits of the BOS.

Combined after-effects of the perturbation training were evaluated from continuous gait on the treadmill, both before and after testing (TW_1 and TW_3). Measures of gait variability, complexity, and smoothness were employed to determine the sensitivity of the pre-and post-training effects. Variability was assessed by the RMS of COM accelerations (AP, ML, and V) along with statistical measures of variability from spatiotemporal gait parameters: Standard deviation (SD) and coefficient of variation (CV). CV denotes the variability of a specific gait parameter normalized to its mean value; it is represented as a percentage (CV=SD/mean × 100). Gait complexity was measured by multiscale entropy (MSE). MSE is a regularity measure developed by Costa et al. (31) that quantifies the information content of the gait signal (COM velocities in the AP, ML, and V directions) over a range of physiologically relevant time scales while sample entropy is computed for every consecutive coarse-grained time series. The entropy values are then plotted as a function of the time scales in which the area under the curve reveals the signal's complexity index (CI). A complex signal is associated with a time evolution with a rich structure on multiple scales. The first 10 s and the last 10 s – initiation and termination of 2-min treadmill walking ($TW_{1\text{-}3}$) – were excluded from the analysis. The local average and the local SD of each time series were computed for each spatiotemporal parameter. Table 2 provides further details.

2.6. Statistical analyses

For all statistical comparisons, assumptions of ANOVA (e.g., homogeneity of variance and normal distribution) were tested using the normality and Leven's tests. Correction for multiple follow-up comparisons was done using the Bonferroni correction. All other univariate analyses uses one-way split-plot repeated measures ANOVA with Greenhouse–Geisser correction for sphericity (i.e., between subject effect, training group, is the whole plot effect of a split-plot design). The Subject effect is nested within the Group effect which was specified as random.

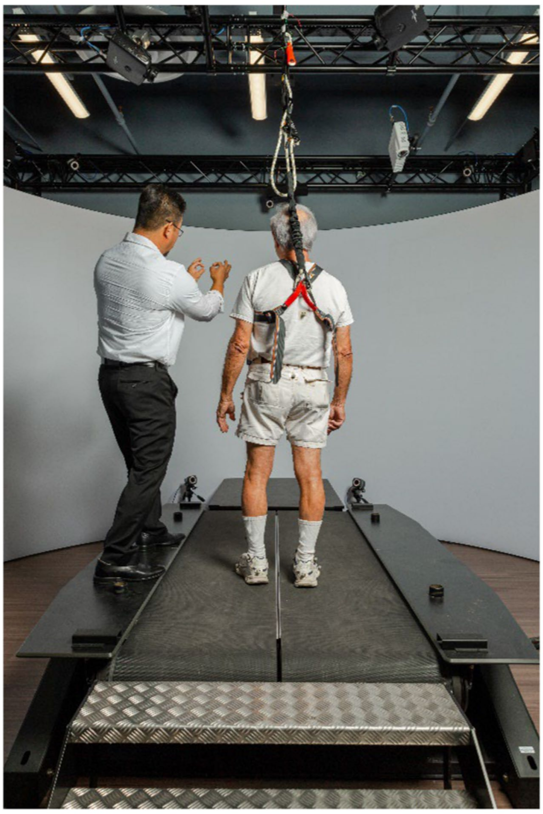

FIGURE 3
GRAIL system with dualbelt instrumented treadmill.

The generalizability of two types of training programs (PST during stance and dynamic gait) and Pre-test and Post-test differences on dependent measures in Table 2 were ascertained using a linear mixed effect model on all gait and posture parameters using the above repeated measures ANOVA. The statical analyses were processed using the JMP Pro 16, 2021, SAS Institute.

3. Results

3.1. Effects of PST during stance on feedforward or proactive adaptation and, associated group effect (generalizability of two types of PST-stance and walking)

The results of the univariate repeated measures ANOVAs on all dependent measures associated with feedforward variables in Table 2 indicated a significant differences on only one of the feedforward variables – Knee flexion angle during pre and post-trial period ($F_{1,10} = 5.662$, $p = 0.0386$, effect size 0.259) (Figure 5). No significant differences were observed for the group comparison or generalizability of two types of training ($F_{1,10} = 0.0102$, $p = 0.92$) on the knee flexion angle or all other feedforward dependent measures in Table 2. In general, knee flexion angles were significantly different indicating a proactive response using the knee strategy (i.e., bending the knee to lower the whole body COM) to maintain stability (Figure 6).

TABLE 2 Definition of parameters.

Parameters	Definition
Feedback parameters	
Recovery period ($Time_{Rec}$)	Time elapsed from perturbation onset until the zero-cross of the AP COM velocity (recovery point).
Latency ($Time_{Latency}$)	Time elapsed between perturbation onset and the initial reactive response from the AP force plate [ms].
Recovery step time ($Time_{1stStep}$)	Time elapsed between perturbation onset and the first recovery step of the contralateral foot [ms] – Step calculated by the zero-cross of AP heel marker velocity.
Path length (PL)	The total length of the COP trajectory in the AP & ML directions [mm].
Normalized path length (nPL)	Path length normalized by its variance. Measures the coordinative structure of the COP (AP & ML); reflects the number of times there is a change in direction.
Velocity	Velocity of COP calculated by Path Length over $Time_{Rec}$ [mm/s].
Margin of stability (MOS_{ML})	Distance between the lateral boundary of the BOS at heel contact (heel marker of the leading foot) and the extrapolated COM.
Margin of stability (MOS_{AP})	Distance between the anterior boundary of the BOS at heel contact (toe marker of the leading foot) and the extrapolated COM.
Root mean square (RMS)	Statistical measure of COP magnitude in the AP & ML directions.
Feedforward parameters	
Trunk angle	Trunk flexion/extension from the sagittal plane (positive = flexion; negative = extension) [deg].
Knee flexion angle	Knee flexion angle from the sagittal plane (positive = flexion; negative = extension) [deg] from the dominant leg.
Ankle angle	Dorsiflexion/plantarflexion of the ankle joint in the sagittal plane (positive = plantarflexion; negative = dorsiflexion) [deg] from the dominant leg.
Base of support (BOS_{ML})	Horizontal stride width during the double-support phase of gait. Stance width during standing perturbations.
Base of support (BOS_{AP})	Step length during the double-support phase of gait. Stance width during static standing.
Margin of stability (MOS_{ML})	Distance between the lateral boundary of the BOS at heel contact (heel marker of the leading foot) and the extrapolated COM.
Margin of stability (MOS_{AP})	Distance between the anterior boundary of the BOS at heel contact (toe marker of the leading foot) and the extrapolated COM.
Heel contact velocity (HCV)	Instantaneous AP heel velocity calculated utilizing AP heel velocities at the foot displacement 1/100 s (Δt) before and after $pert_{hc}$ [m/s] (Lockhart et al., 2003) from the dominant leg. $vpert_{hc} = [x(i+1) - x(i-1)]/2\Delta t$

(Continued)

TABLE 2 (Continued)

Parameters	Definition
Gait parameters	
Gait cycle time	Time elapsed between two consecutive heel contacts of ipsilateral foot.
Step time	Time elapsed from the heel contact of one foot to heel contact of the subsequent contralateral foot.
Stance time (RST & LST)	Time elapsed from the heel contact to the toe-off of a single footfall [s]. Calculate left (LST) & right (RST).
Double support time (DST)	Time elapsed from the heel contact of one foot to the toe-off of the contralateral foot. The sum of two periods of double support in the gait cycle [s].
Root mean square (RMS)	Statistical measure of the COM magnitude in the AP, ML, or V direction compared to the total trunk acceleration magnitude.
Coefficient of variation (CV)	Measure of variability normalized to the mean of a specific parameter [%]. CV = (SD/Mean) × 100

3.2. Effects of PST during stance on feedback or reactive adaptations

Compared to the initial unexpected perturbation block, reaction time ($\text{Time}_{\text{Latency}}$) showed significant improvement ($F_{1,10} = 4.94$, $p = 0.050$, effect size =0.352) (Figure 7). This suggests that reactive adaption utilizing feedforward mechanisms is still active and may be trained and directed toward improving fall safety (Figure 8). No significant group (generalizability of two types of training) effect ($F_{1,10} = 0.012$, $p = 0.916$) was observed for the reactive adaptation variables in Table 2.

3.3. Effects of PST during walking on feedforward or proactive adaptations and generalizability of training groups

Proactive adaptations during gait were assessed during the 10–15 steps before the initial perturbation block (GP_1) and the 10–15 steps preceding the last perturbation block (GP_{12}). Flexion angles of the trunk, hip, and knee of the perturbed limb (pertb_{hc}) were significantly greater from GP_1 to GP_{12}, revealing feedforward adaptations in anticipation of the perturbation. These biomechanical modifications are characterized by the varying trunk ($F_{1,10} = 11.311$, $p = 0.007$, effect size = 0.08) (Figures 9–11) and hip flexion angles ($F_{1,10} = 5.709$, $p = 0.038$, effect size = 0.05) (Figures 12, 13) and, adopting vigilant gait marked by higher heel contact velocity ($F_{1,10} = 6.503$, $p = 0.029$, effect size = 0.33) (Figures 14, 15). Group effects (generalizability of training) were not statistically significant for these variables (i.e., trunk and hip flexion angles and heel contact velocity).

3.4. Effects of PST during walking on feedback or reactive adaptations

No significant differences were observed in any of the reactive parameters associated with recovery from a slip.

4. Discussion

The primary goal of this study was to assess how PD affects the ability to respond to slip specific perturbations and if one session of protective step training can produce short-term adaptations to improve walking and static balance control. A secondary goal was to determine the generalizability of two types of training programs (PST during stance and dynamic gait) and to what extent the perturbation-based balance training are specific and transferable to the nature of the perturbations experienced. At present, little is known regarding the effect of PD on the ability to react and adapt to standing and walking perturbations. For example, previous studies have shown improved adaptive capacity with perturbation responses in the mediolateral direction (21, 32). However, the specific nature of the perturbations in the current study were to simulate realistic slip perturbations without any walking aid, which were performed by anterior translations during standing and walking. We hypothesized that participants with PD would experience difficulty adapting reactive feedback-based strategies, but would adapt to planned feedforward strategies to standing and dynamic walking perturbation programs. The results generally support this hypothesis, showing that participants with PD were able to use experiences with perturbation training to integrate and adapt proactive feedforward strategies. Reactive, feedback strategies were less frequently improved through practice in the current cohort. Notably, there was little generalization between in-place and walking practice. The ability of the participants with PD to adapt to changes in task demands, particularly proactive behavior, suggests that individuals with PD could benefit from a specific training paradigm to facilitate specific balance control during rehabilitation (13, 33).

The results in this study are consistent with previous studies regarding early PD, which reported proactive adjustments during postural stability and locomotor perturbation tasks (6, 7, 20). This was evident during the walking perturbation program when comparing the effects of walking behavior prior to the initial walking perturbation block (GP_1) and the final perturbation block (GP_{12}). Particpants demonstrated significant feedforward adaptations in anticipation of the unexpected perturbation by significantly increasing trunk and hip flexions during walking along with higher heel contact velocity to veer away from untimely balance perturbations and adopting a more cautious gait to increase stability. However, this proactive effort may not have been fully realized in the current study as we found no significant differences in any of the reactive measures for these patients.

Predictive control is associated with supraspinal structures (8) involving cognitive processes like attention and memory (9) that may not be impaired in the early stages of the disease (8, 9). Predictive responses are important components for safe locomotion (33, 34) because they reduce the consequences of expected perturbations (12) and ultimately reduce the risk of falls. Thus, the increased risk of falls in early PD patients may be associated with deficits in reactive motor control. Understanding the ability of someone with PD to adapt to changes in task-specific demands will be useful in therapeutic intervention strategies.

Studies have closely linked the striatal system to motor learning, (5, 35, 36), suggesting that individuals with deficiencies in this system, such as those with Parkinson's disease, would, in addition to the degradation of their movement patterns at baseline, have difficulty acquiring movement schema that would allow them to

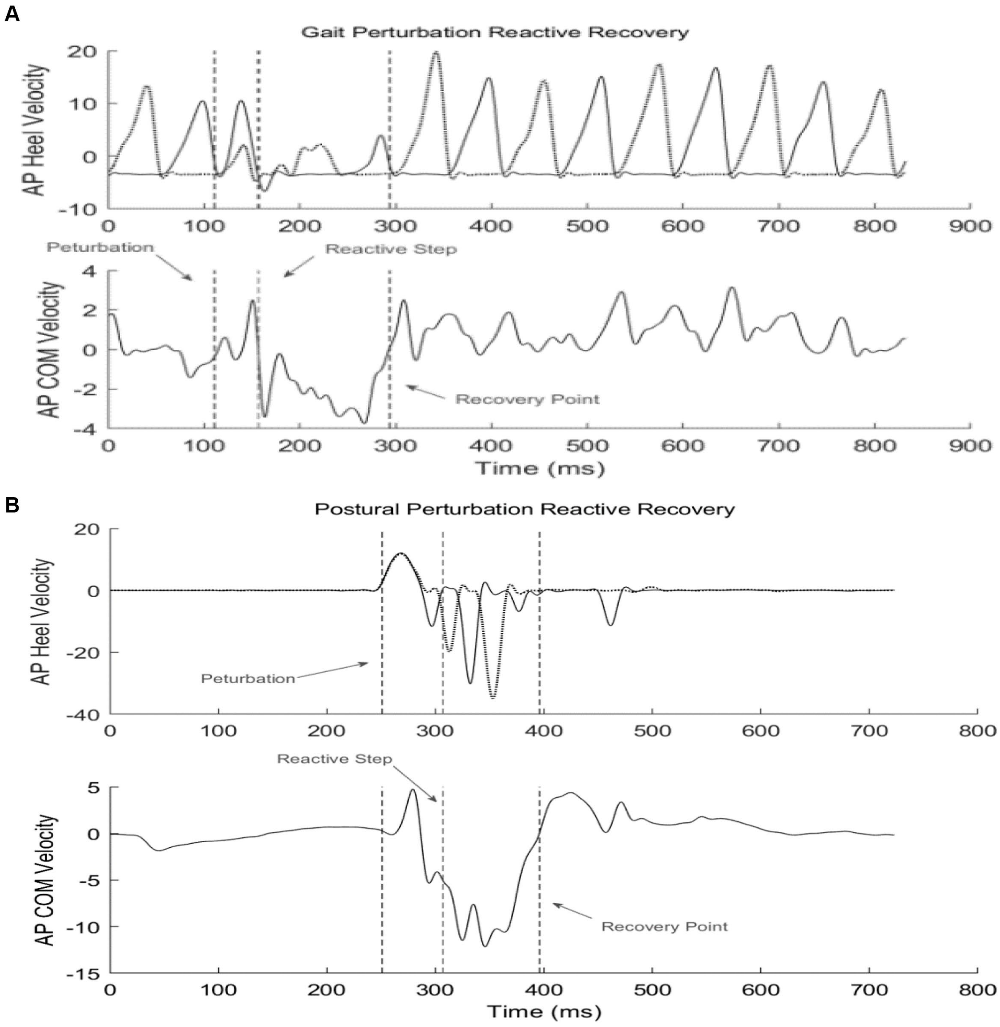

FIGURE 4
Example of reactive recovery dynamics given a perturbation during stance or gait. The 1st dotted line identifies perturbation onset; the 2nd line identifies the reactive recovery step (rechc); the 3rd line marks the termination of the recovery period. **(A)** Gait perturbation example; **(B)** Postural perturbation example.

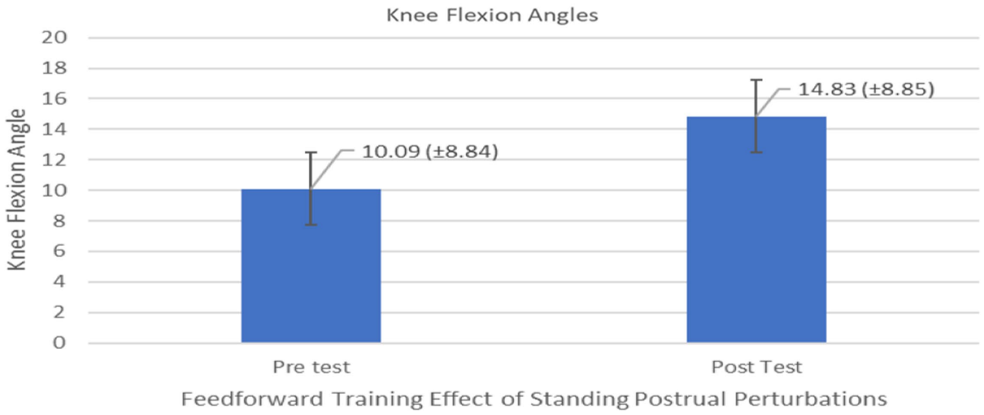

FIGURE 5
Feedforward Effects of PST during stance on the knee flexion angles with the means and standard deviations shown.

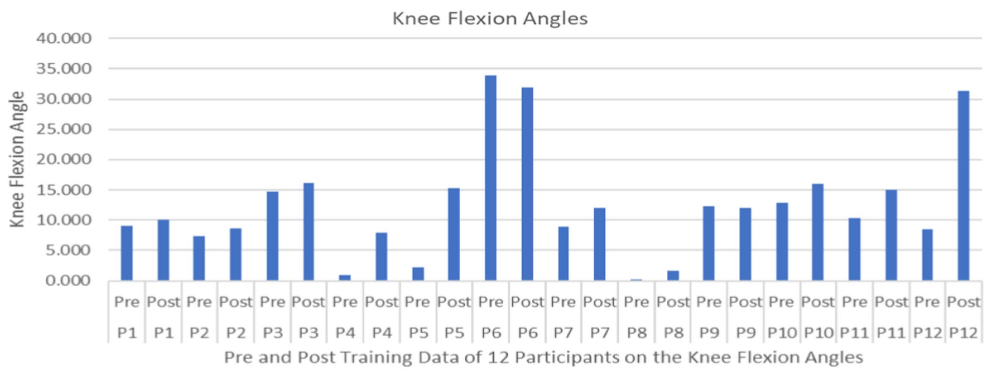

FIGURE 6
Feedforward Effects of PST during stance on the knee flexion angles of all participants.

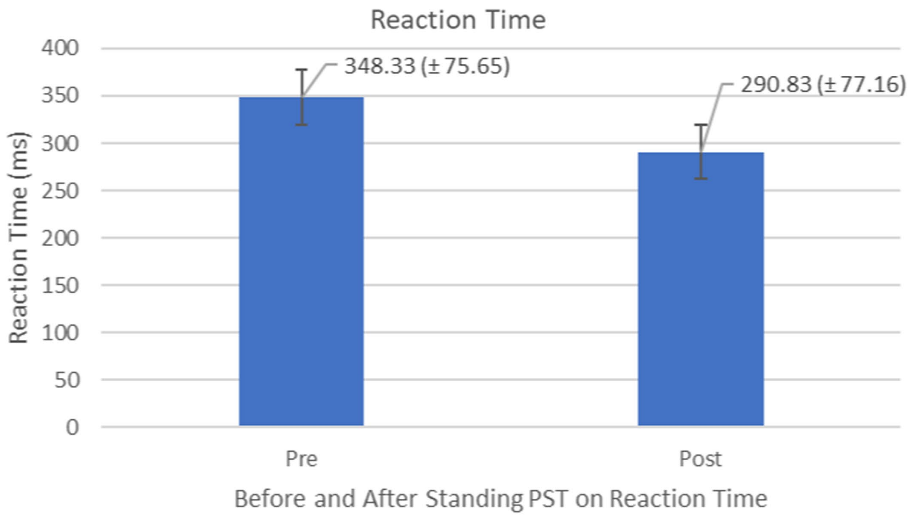

FIGURE 7
Feedback Effects of PST during stance on reaction time of the stance foot with the mean reaction times and standard deviations shown.

learn tasks quickly and accurately. However, studies examining the ability of patients with PD to learn and adapt to motor tasks have been relatively inconsistent (10–13, 37–45). While these studies indicate that PD patients are able to learn motor tasks, there is disagreement about the amount and type of improvement. A possible reason for this is that conflicting studies utilized different types of learning (implicit and explicit), considering specific aspects of learning are more severely impacted by PD, especially in the early stages of the disease. It has been reported that PD patients are able to learn specific tasks, however, they may require more practice than healthy controls do. Furthermore, the learned skills are not easily generalizable to other tasks, even if those tasks are similar (5, 40–45). The slower rate of learning and lack of transference may imply that PD patients are still able to learn in an explicit, feedforward manner – they may "pre-program" specific techniques and tasks quite capably - i.e., proactive mechanisms – but they may be unable to easily adjust to changes requiring the use of automatic or reactive mechanisms, making ready adaptation to changing conditions or simultaneous completion of multiple tasks, both of which are often required for balance and gait, quite difficult.

4.1. Predictive motor adaptations of PST

Learning integration for both proactive (feedforward) and reactive (feedback) adaptations were analyzed from the gait and postural PST paradigm, Figure 1. Feedforward responses from gait were extracted from the 15 s of unperturbed walking prior to each perturbation. Trunk flexion and hip flexion of the perturbed limb (pertb_{hc}) were significantly greater compared to baseline, revealing feedforward adaptations in anticipation of the perturbation. Furthermore, heel contact velocity was increased in an effort to regain balance given a perturbation. The gait modification demonstrates the adoption of a more considered and vigilant gait to increase stability.

4.2. Reactive motor adaptations of PST

However, even after a significant effort of the feedforward system, the reactive responses to gait PST were not robustly impacted through a single session of 12 perturbations. Regarding walking slips, recovery step time ($\text{Time}_{\text{1stStep}}$) – the time elapsed between perturbation onset and

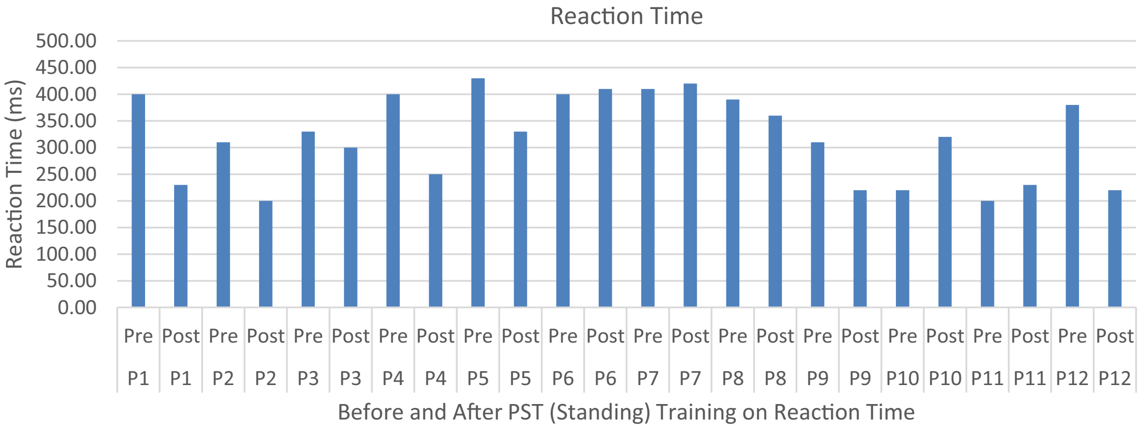

FIGURE 8
Reaction time or Time$_{Latency}$ (ms) associated with postural perturbation training during the first and the last perturbations. Participants were able to produce quicker responses to postural perturbations after the PST.

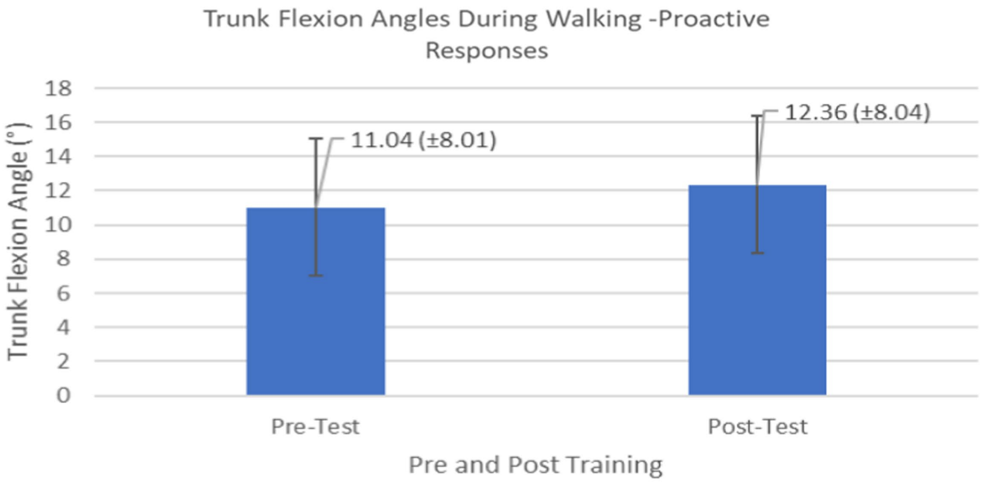

FIGURE 9
Feedforward Effects of gait perturbation training with the mean reaction times and standard deviations shown.

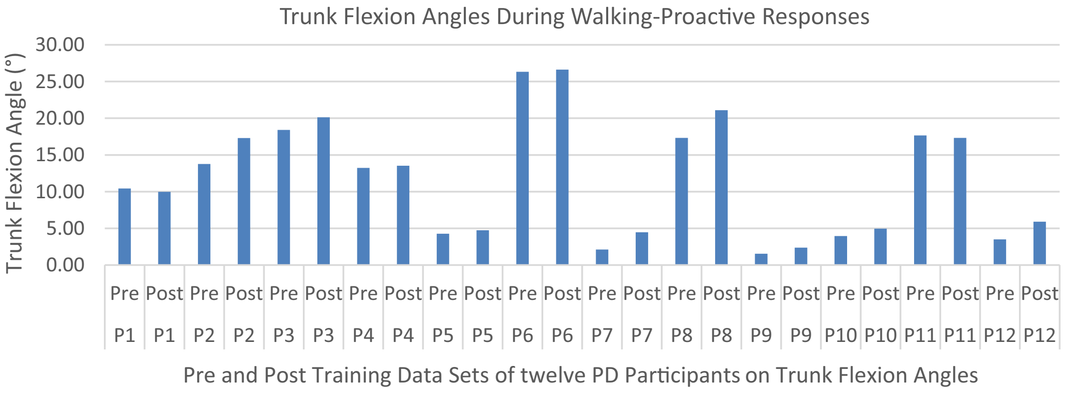

FIGURE 10
Individual effects of gait perturbation training on feedforward/proactive adaptation of trunk flexion.

the first recovery step of the contralateral foot – showed no significant difference between baseline and the last perturbation trial. Similarly, the recovery period ($Time_{Rec}$) – time elapsed from perturbation onset until the zero-cross of the anteroposterior COM velocity – also did not show improvement in their reactive recovery time.

Like gait PST, reactive feedback control for static stance PST was not robustly improved through practice. This is in contrast with some previous results (46, 47). Alternatively, it is also possible that feedforward modifications may not have generated sufficient stability improvements post-perturbation adaptations to the stability margins. Finally, it is possible that 12 perturbations was too little of a dose to produce meaningful improvements as were observed in previous studies.

4.3. Perturbation-specific transference

The secondary goal of this study was to determine the generalizability of PST during stance and PST during dynamic gait, and to what extent the specific type of the perturbations may transfer to perturbations in everyday situations. Dopaminergic treatments has been shown to be ineffective or unsatisfactory at treating postural instability and gait dysfunction in idiopathic PD, however, studies have demonstrated that therapy explicitly focusing on posture, gait, and balance may significantly improve these factors (18, 32). It is hypothesized that therapy specifically modeling situations in which individuals with PD are likely to fall (e.g., slipping due to shuffled steps/reduced executive function) may be more beneficial to prevent future falls than more generalized physical therapy. Further, repeated perturbation training has been shown in previous studies to improve features of postural stability and gait in PD patients. However, most of this preceding work has focused primarily on ascertaining the effects of training during static stance, either through training of center of pressure shifts toward a patient's limits of stability (teaching the patient to weight-shift and lean safely) (48, 49) or through the training of adaptive responses (either postural adjustments or compensatory stepping) to regain balance following an external perturbation (32, 46, 50–52). These studies suggest that PD patients are able to learn to adapt to perturbations, that these adaptations may persist for several months, and that PST may enhance balance confidence. However, the generalizability of response improvements to other types of perturbation, such as during standing and walking, is uncertain (40–45). Results from the current study suggested that there may be limited generalizability across two types of training programs (PST during stance and dynamic gait). Albeit, healthy older adults were able to genderized their training (slips/trips) (53), in this study with PD patients, the specific training (either postural or gait perturbation training) did not improve balance in the untrained task. Thus, more personalized and specific training program is required to improve balance maintenance in PD patients.

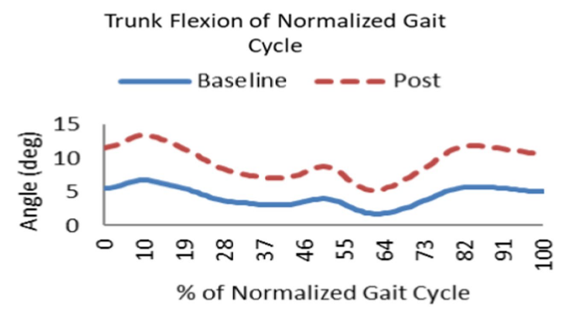

FIGURE 11
Following repeated gait perturbation training a subject's trunk flexion is greater throughout the gait cycle exhibiting feedforward/proactive adaptation.

5. Limitations

Several limitation to this study should be considered when interpreting the findings. First, this was a preliminary study to elucidate the optimal dose and frequency, as well as the therapeutic

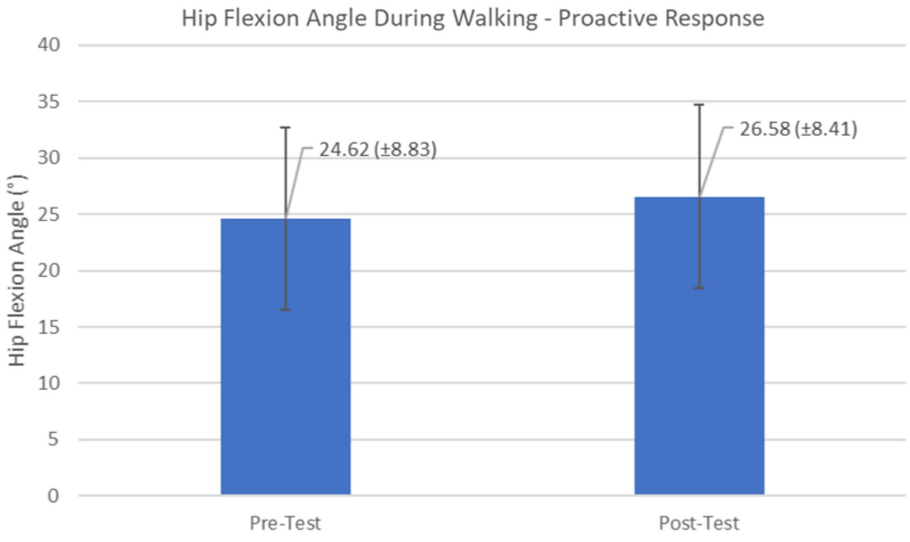

FIGURE 12
Feedforward Effects of gait perturbation training with the mean hip flexion angles and standard deviations shown.

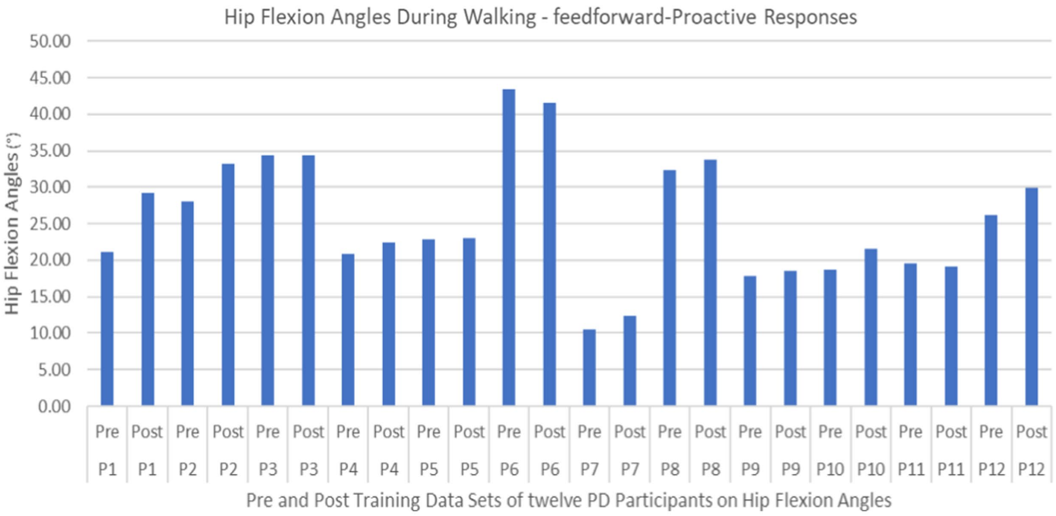

FIGURE 13
Individual effects of gait perturbation training on feedforward/proactive adaptation of hip flexion.

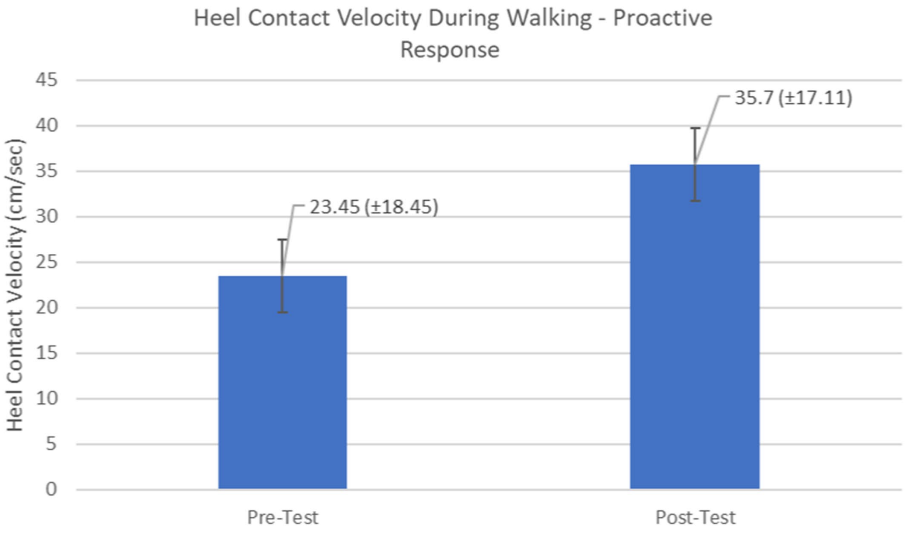

FIGURE 14
Feedforward Effects of gait perturbation training on heel contact velocity.

index, to determine maximal efficacy for a PST dose–response relationship in PD populations. The present study applies only a single perturbation acceleration of 10 m/s^2 in the anterior direction to elicit a slip-specific perturbation effect. We also did not use a younger or age-matched control group to be compared. This perturbation was chosen to represent a worst-case scenario condition for PD and as an incipient marker for a perturbation scenario and population that is not well researched in the literature. Given the preliminary nature of this study, our sample size was relatively small (post effect sizes were from 0.03 to 0.352) and may affect the strength of our conclusions. Given the heterogeneity of PD, further studies involving a larger number and a wider range of PD participants is warranted. Further, the present finding only looked at acute after-effects of compensatory and adaptive behavior modifications, directly after the intervention. The authors did not perform repeated measurements over a period of time to determine the efficacy of the adaptive responses and do not expect the after-effects to imprint over a longitudinal period. Further, observed feedback and feedforward behavior may be dependent on the specific type of the perturbations experienced and may not show transfer to other forms of perturbations. Future studies will investigate the longevity of the acute after-effects produced in the present study. Finally, these effects were associated with PD patients' average H&Y score of 2.7, and further study linking these two assessments to create a personalized treatment program is highly recommended.

6. Conclusion

PST is an efficient and effective way to discern reactive and proactive responses to therapeutic intervention. It has been suggested that this task-specific training approach may present a paradigm shift in fall prevention. While PD patients are still able to improve

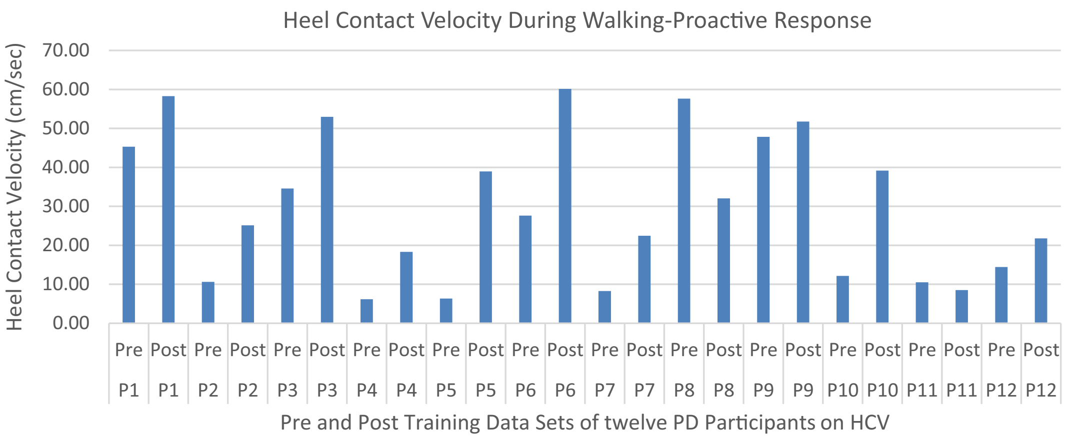

FIGURE 15
Individual effects of gait perturbation training on feedforward/proactive adaptation of heel contact velocity.

performance with practice, particularly in feedforward aspects of postural responses, reactive aspects of postural responses were not uniformly improved through practice. Because of this, patients with PD may require more training to achieve and retain motor learning and may require additional sensory information or motor guidance in order to facilitate this learning. These shortcomings in motor learning in PD could contribute to the degeneration in gait and balance often seen in the disease, as patients are unable to adapt to the gradual sensory and motor degradation. Research has shown that physical and exercise therapy can help PD patients to adapt new feedforward strategies to partially counteract these symptoms. In particular, balance, treadmill, resistance, and repeated perturbation training (PST) therapies have been shown to improve motor patterns in PD. However, much research is still needed to determine which of these therapies best alleviates which symptoms of PIGD, the needed dose and intensity of these therapies, the long-term retention effects, and the benefits of such technologies as augmented feedback, motorized perturbations, virtual reality, and weight-bearing assistance.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving humans were approved by Arizona State University Institutional Review Board. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study.

Author contributions

TL was responsible for compiling the sources used within this study and writing the drafted manuscript. TL, MO, and AL provided additional articles, discussed possible implications, and revised the manuscript. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher’s note

References

1. Shulman JM, De Jager PL, Feany MB. Parkinson's disease: genetics and pathogenesis. *Annu Rev Pathol Mech Dis*. (2011) 6:193–22. doi: 10.1146/annurev-pathol-011110-130242

2. Roemmich RT, Nocera JR, Stegemoller EL, Hassan A, Okun MS, Hass CJ. Locomotor adaptation and locomotor adaptive learning in Parkinson's disease and normal aging. *Clin Neurophysiol*. (2014) 125:313–9. doi: 10.1016/j.clinph.2013.07.003

3. Wilkinson L, Khan Z, Jahanshahi M. The role of the basal ganglia and its cortical connections in sequence learning: evidence from implicit and explicit sequence learning in Parkinson's disease. *Neuropsychologia*. (2009) 47:2564–73. doi: 10.1016/j.neuropsychologia.2009.05.003

4. Frith CD, Bloxham CA, Carpenter KN. Impairments in the learning and performance of a new manual skill in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry*. (1986) 49:661–8. doi: 10.1136/jnnp.49.6.661

5. Redgrave P, Rodriguez M, Smith Y, Rodriguez-Oroz MC, Lehericy S, Bergman H, et al. Goal-directed and habitual control in the basal ganglia: implications for Parkinson's disease. *Nat Rev Neurosci*. (2010) 11:760–72. doi: 10.1038/nrn2915

6. Pascual-Leone A, Grafman J, Clark K, Stewart M, Massaquoi S, Lou JS, et al. Procedural learning in Parkinson's disease and cerebellar degeneration. *Ann Neurol*. (1993) 34:594–602. doi: 10.1002/ana.410340414

7. Muslimović D, Post B, Speelman JD, Schmand B. Motor procedural learning in Parkinson's disease. *Brain*. (2007) 130:2887–97. doi: 10.1093/brain/awm211

8. Brichta L, Greengard P, Flajolet M. Advances in the pharmacological treatment of Parkinson's disease: targeting neurotransmitter systems. *Trends Neurosci*. (2013) 36:543–54. doi: 10.1016/j.tins.2013.06.003

9. Calabresi P, Picconi B, Parnetti L, Di Filippo M. A convergent model for cognitive dysfunctions in Parkinson's disease: the critical dopamine-acetylcholine synaptic balance. *Lancet Neurol*. (2006) 5:974–83. doi: 10.1016/S1474-4422(06)70600-7

10. Yitayeh A, Teshome A. The effectiveness of physiotherapy treatment on balance dysfunction and postural instability in persons with Parkinson's disease: a systematic review and meta-analysis. *BMC Sports Sci Med Rehabil*. (2016) 8:1–10. doi: 10.1186/s13102-016-0042-0

11. Smania N, Corato E, Tinazzi M, Stanzani C, Fiaschi A, Girardi P, et al. Effect of balance training on postural instability in patients with idiopathic Parkinson's disease. *Neurorehabil Neural Repair*. (2010) 24:826–34. doi: 10.1177/1545968310376057

12. Pendt LK, Reuter I, Muller H. Motor skill learning, retention, and control deficits in Parkinsons disease. *PLoS One*. (2011) 6. doi: 10.1371/journal.pone.0021669

13. Nieuwboer A, Rochester L, Müncks L, Swinnen SP. Motor learning in Parkinson's disease: limitations and potential for rehabilitation. *Park Relat Disord*. (2009) 15:53–8. doi: 10.1016/S1353-8020(09)70781-3

14. Olson M, Lockhart TE, Lieberman A. Motor learning deficits in Parkinson's disease (PD) and their effect on training response in gait and balance: a narrative review. *Front Neurol*. (2019) 10:62. doi: 10.3389/fneur.2019.00062

15. Gerards MHG, McCrum C, Mansfield A, Meijer K. Perturbation-based balance training for falls reduction among older adults: current evidence and implications for clinical practice. *Geriatr Gerontol Int*. (2017) 17:2294–303. doi: 10.1111/ggi.13082

16. McCrum C, Gerards MHG, Karamanidis K, Zijlstra W, Meijer K. A systematic review of gait perturbation paradigms for improving reactive stepping responses and falls risk among healthy older adults. *Euro Rev Aging Phys*. (2017) 14:3. doi: 10.1186/s11556-017-0173-7

17. McCrum C, Bhatt TS, Gerards MHG, Karamanidis K, Rogers MW, Lord SR, et al. Perturbation-based balance training: principles, mechanisms and implementation in clinical practice. *Front Sports Act Living*. (2022) 4:1015394. doi: 10.3389/fspor.2022.1015394

18. Oates AR, van Ooteghem K, Frank JS, Patla AE, Horak FB. Adaptation of gait termination on a slippery surface in Parkinson's disease. *Gait Posture*. (2013) 37:516–20. doi: 10.1016/j.gaitpost.2012.09.002

19. Steib S, Klamroth S, Gaßner H, Pasluosta C, Eskofier B, Winkler J, et al. Perturbation during treadmill training improves dynamic balance and gait in Parkinson's disease: a single-blind randomized controlled pilot trial. *Neurorehabil Neural Repair*. (2017) 31:758–68. doi: 10.1177/1545968317721976

20. Klamroth S, Steib S, Gaßner H, Goßler J, Winkler J, Eskofier B, et al. Immediate effects of perturbation treadmill training on gait and postural control in patients with Parkinson's disease. *Gait Posture*. (2016) 50:102–8. doi: 10.1016/j.gaitpost.2016.08.020

21. Martelli D, Luo L, Kang J, Kang UJ, Fahn S, Agrawal SK. Adaptation of stability during perturbed walking in Parkinson's disease. *Sci Rep*. (2017) 7:17875. doi: 10.1038/s41598-017-18075-6

22. Hulzinga F, Seuthe J, D'Cruz N, Ginis P, Nieuwboer A, Schlenstedt C. Split-Belt treadmill training to improve gait adaptation in Parkinson's disease. *Move Disord Off J Move Disord Soc*. (2023) 38:92–03. doi: 10.1002/mds.29238

23. Coelho DB, de Oliveira CEN, Guimarães MVC, Ribeiro de Souza C, Dos Santos ML, de Lima-Pardini AC. A systematic review on the effectiveness of perturbation-based balance training in postural control and gait in Parkinson's disease. *Physiotherapy*. (2022) 116:58–71. doi: 10.1016/j.physio.2022.02.005

24. Hulzinga F, de Rond V, Vandendoorent B, Gilat M, Ginis P, D'Cruz N, et al. Repeated gait perturbation training in Parkinson's disease and healthy older adults: a systematic review and Meta-analysis. *Front Hum Neurosci*. (2021) 15:732648. doi: 10.3389/fnhum.2021.732648

25. Goetz CG, Poewe W, Rascol O, Sampaio C, Stebbins GT, Counsell C, et al. Movement Disorder Society task force report on the Hoehn and Yahr staging scale: status and recommendations. *Mov Disord*. (2004) 19:1020–8. doi: 10.1002/mds.20213

26. Parijat P, Lockhart TE. Effects of moveable platform training in preventing slip-induced falls in older adults. *Ann Biomed Eng*. (2012) 40:1111–21. doi: 10.1007/s10439-011-0477-0

27. Parijat P, Lockhart TE, Liu J. Effects of perturbation-based slip training using a virtual reality environment on slip-induced falls. *Ann Biomed Eng*. (2015) 43:958–67. doi: 10.1007/s10439-014-1128-z

28. Parijat P, Lockhart TE, Liu J. EMG and kinematic responses to unexpected slips after slip training in virtual reality. *IEEE Trans Biomed Eng*. (2015) 62:593–9. doi: 10.1109/TBME.2014.2361324

29. Liu J, Lockhart TE, Parijat P, McIntosh JD, Chiu YP. Comparison of slip training in VR environment and on moveable platform. *Biomed Sci Instrum*. (2015) 51:189–97.

30. Hof AL, Gazendam MGJ, Sinke WE. The condition for dynamic stability. *J Biomech*. (2005) 38:1–8. doi: 10.1016/j.jbiomech.2004.03.025

31. Costa M, Goldberger AL, Peng CK. Multiscale entropy analysis of complex physiologic time series. *Phys Rev Lett*. (2002) 89:6–9. doi: 10.1103/PhysRevLett.89.068102

32. Ribeiro de Souza C, Ávila de Oliveira J, Takazono PS, da Silva Rezende L, Silva-Batista C, Coelho DB, et al. Perturbation-based balance training leads to improved reactive postural responses in individuals with Parkinson's disease and freezing of gait. *Eur J Neurosci*. (2023) 57:2174–86. doi: 10.1111/ejn.16039

33. Hausdorff JM, Gruendlinger L, Scollins L, O'Herron S, Tarsy D. Deep brain stimulation effects on gait variability in Parkinson s disease. *Mov Disord*. (2009) 24:1688–92. doi: 10.1002/mds.22554

34. Pötter-Nerger M, Volkmann J. Deep brain stimulation for gait and postural symptoms in Parkinson's disease. *Mov Disord*. (2013) 28:1609–15. doi: 10.1002/mds.25677

35. Hikosaka O. Role of basal ganglia in control of innate movements, learned behavior and cognition—a hypothesis. *Adv Behav Biol Basal Ganglia*. (1994) 4:589–96. doi: 10.1007/978-1-4613-0485-2_61

36. Hikosaka O. Basal ganglia - possible role in motor coordination and learning. *Curr Opin Neurobiol*. (1991) 1:638–43. doi: 10.1016/S0959-4388(05)80042-X

37. Shen X, Mak MKY. Technology-assisted balance and gait training reduces falls in patients with Parkinson's disease: a randomized controlled trial with 12-month follow-up. *Neurorehabil Neural Repair*. (2015) 29:103–11. doi: 10.1177/1545968314537559

38. Protas EJ, Mitchell K, Williams A, Qureshy H, Caroline K, Lai EC. Gait and step training to reduce falls in Parkinson's disease. *Neuro Rehab*. (2005) 20:183–90.

39. Jöbges M, Heuschkel G, Pretzel C, Illhardt C, Renner C, Hummelsheim H. Repetitive training of compensatory steps: a therapeutic approach for postural instability in Parkinson's disease. *J Neurol Neurosurg Psychiatry*. (2004) 75:1682–7. doi: 10.1136/jnnp.2003.016550

40. Agostino R, Sanes JN, Hallett M. Motor skill learning in Parkinson's disease. *J Neurol Sci*. (1996) 139:218–26. doi: 10.1016/0022-510X(96)00060-3

41. Soliveri P, Brown RG, Jahanshahi M, Marsden CD. Effect of practice on performance of a skilled motor task in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry*. (1992) 55:454–60. doi: 10.1136/jnnp.55.6.454

42. Worringham CJ, Stelmach GE. Practice effects on the Preprogramming of discrete movements in Parkinsons disease. *J Neurol Neurosurg Psychiatry*. (1990) 53:702–4. doi: 10.1136/jnnp.53.8.702

43. Behrman AL, Cauraugh JH, Light KE. Practice as an intervention to improve speeded motor performance and motor learning in Parkinson's disease. *J Neurol Sci*. (2000) 174:127–36. doi: 10.1016/S0022-510X(00)00267-7

44. Jessop RT, Horowicz C, Dibble LE. Motor learning and Parkinson disease: refinement of movement velocity and endpoint excursion in a limits of stability balance task. *Neurorehabil Neural Repair*. (2006) 20:459–67. doi: 10.1177/1545968306287107

45. Swinnen SP, Steyvers M, Van Den Bergh L, Stelmach GE. Motor learning and Parkinson's disease: refinement of within-limb and between-limb coordination as a result of practice. *Behav Brain Res*. (2000) 111:45–59. doi: 10.1016/S0166-4328(00)00144-3

46. Peterson DS, Dijkstra BW, Horak FB. Postural motor learning in people with Parkinson's disease. *J Neurol*. (2016) 263:1518–29. doi: 10.1007/s00415-016-8158-4

47. Monaghan AS, Finley JM, Mehta SH, Peterson DS. Assessing the impact of dual-task reactive step practice in people with Parkinson's disease: a feasibility study. *Hum Mov Sci*. (2021) 80:102876. doi: 10.1016/j.humov.2021.102876

48. Jankovic J. Parkinson's disease: clinical features and diagnosis. *J Neurol Neurosurg Psychiatry*. (2008) 79:368–76. doi: 10.1136/jnnp.2007.131045

49. Sveinbjornsdottir S. The clinical symptoms of Parkinson's disease. *J Neurochem*. (2016) 139:318–24. doi: 10.1111/jnc.13691

50. Barajas JS, Peterson DS. First-trial protective step performance before and after short-term perturbation practice in people with Parkinson's disease. *J Neurol*. (2018) 265:1138–44. doi: 10.1007/s00415-018-8821-z

51. Smulders K, Esselink RA, de Swart BJ, Geurts AC, Bloem BR, Weerdesteyn V. Postural inflexibility in PD: does it affect compensatory stepping? *Gait Posture*. (2014) 39:700–6. doi: 10.1016/j.gaitpost.2013.10.003

52. de Kam D, Nonnekes J, Oude Nijhuis LB, Geurts ACH, Bloem BR, Weerdesteyn V. Dopaminergic medication does not improve stepping responses following backward and forward balance perturbations in patients with Parkinson's disease. *J Neurol*. (2014) 261:2330–7. doi: 10.1007/s00415-014-7496-3

53. Bhatt T, Wang Y, Wang S, Kannan L. Perturbation training for fall-risk reduction in healthy older adults: interference and generalization to opposing novel perturbations Post intervention. *Front Sports Act Living*. (2021) 3:697169. doi: 10.3389/fspor.2021.697169

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Heterogeneities of the perceptual-motor style during locomotion at height

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In a recent review, we summarized the characteristics of perceptual-motor style in humans. Style can vary from individual to individual, task to task and pathology to pathology, as sensorimotor transformations demonstrate considerable adaptability and plasticity. Although the behavioral evidence for individual styles is substantial, much remains to be done to understand the neural and mechanical substrates of inter-individual differences in sensorimotor performance. In this study, we aimed to investigate the modulation of perceptual-motor style during locomotion at height in 16 persons with no history of fear of heights or acrophobia. We used an inexpensive virtual reality (VR) video game. In this VR game, Richie's Plank, the person progresses on a narrow plank placed between two buildings at the height of the 30th floor. Our first finding was that the static markers (head, trunk and limb configurations relative to the gravitational vertical) and some dynamic markers (jerk, root mean square, sample entropy and two-thirds power law at head, trunk and limb level) we had previously identified to define perceptual motor style during locomotion could account for fear modulation during VR play. Our second surprising result was the heterogeneity of this modulation in the 16 young, healthy individuals exposed to moving at a height. Finally, 56% of participants showed a persistent change in at least one variable of their skeletal configuration and 61% in one variable of their dynamic control during ground locomotion after exposure to height.

KEYWORDS

motor style, sensorimotor, locomotion, virtual reality, perceptive

1 Introduction

In a recent review (Mantilla et al., 2020), we summarized the characteristics of perceptual-motor styles in humans. Style can vary from individual to individual, task to task, and pathology to pathology, as sensorimotor transformations demonstrate considerable adaptability and plasticity. Although the behavioral evidence for individual styles is substantial, much remains to be done to understand the neural and mechanical substrates of inter-individual differences in sensorimotor performance. Perceptual-motor style may change during intensive physical activity or during the course of an illness, but this is no guarantee that it will be to the benefit of the individual, the athlete, or the patient. Numerous studies also show that perceptual-motor styles can evolve with proactive learning. So, whether training athletes, patients, or soldiers, the problem would be similar: identifying and longitudinally monitoring a person's perceptual-motor style would help considerably in revealing the onset of a pathological process. Ultimately, this would make it possible to personalize training and/or treatment and to decide when they need to be readjusted to maintain optimal motor control.

To track a person's perceptual-motor style, reliable markers of motor behavior need to be identified. To answer this question, we quantified motor behavior at rest, during walking, and during running at maximum speed (Vidal and Lacquaniti, 2021). We verified that motor control could be conveniently decomposed into static (stable head, trunk, and limb configurations relative to the gravitational vertical) and dynamic [jerks, root mean square (RMS), sample entropy, and the two-thirds power law quantifying head, trunk, and limb movements] components. We then postulated that markers identifying low intra-individual variability and high inter-individual variability were adequate to define the perceptual-motor style of individuals who otherwise exhibited high inter-individual variability.

Once these factors have been defined, one can tackle the question of how cognitive factors modulate motor perceptual style (Lelard et al., 2019; Williams et al., 2020). In the present study, we investigated locomotion during exposure to height. Studies have described three types of responses to height exposure (Brandt and Huppert, 2014; Brandt et al., 2015). The first response is a physiological height imbalance resulting from impaired visual control of balance: the distance from the stationary visual scene becomes too great to detect and counteract body movements. As a result, visual cues come into conflict with vestibular and proprioceptive cues. In addition, changes in attention can lead to threat-related postural changes (Huffman et al., 2009). In the second response, visual height intolerance induces a more or less pronounced apprehension of losing balance or falling. In the third response, acrophobia presents the same symptoms with such intensity that it can be considered a specific phobia, leading to panic attacks. Several types of symptoms can be observed during height exposure: anxiety, weak knees, and inner restlessness. Neurovegetative symptoms (accelerated heart rate, sweating, drowsiness, and tremors) are predominant. At the motor level, static postural control is impaired due to the co-contraction of the antigravity muscles, which stiffens the whole body. Oculomotor and head movements in all three dimensions are reduced and consist mainly of gaze fixation on the horizon. Individuals tend to walk slowly, stride length is reduced, and double-support phases are increased. All these symptoms are reinforced by anxiety and increased height. Postural symptoms saturate at approximately 20 m above ground and anxiety at approximately 40 m in non-acrophobic patients and 70 m in acrophobic participants (Teggi et al., 2019; Huppert et al., 2020).

Most studies of the threats posed by height to human postural control have focused on static balance. When locomotion was studied, the number of markers was limited. Moreover, these parameters were averaged across participants (see Section 4). This is why we have undertaken a quantitative analysis of perceptual-motor style and its inter-individual heterogeneity during height locomotion. To answer this question, we used the markers we had identified to characterize perceptual-motor style during locomotion at ground level (Vidal and Lacquaniti, 2021). The study involved 16 young individuals with no history of fear of heights or acrophobia. For height exposure, we used an inexpensive virtual reality (VR) video game, with the idea of later using the same protocol clinically to study how emotion modulates perceptual-motor style, including acrophobia and fear of falling. In this VR game, Richie's Plank, the player must progress on a narrow plank placed between two buildings at the height of the 30th floor.

2 Materials and methods

2.1 Participants

We included 16 volunteers (12 men), with a mean age of 24 ± 2 years, 64 ± 16 kg, and 176 ± 6 cm tall. The data of the sixteen volunteers are available. Their body mass index corresponded to a normal range (*WHO | The World Health Report 2006—Working together for health*). *A priori* approval was obtained from the university's research board (IRB CER no. 2021-12-WANGVIDAL, 6 April 2021), and written informed consent was obtained from all participants. Before the experiment, all participants were asked whether they had blood pressure or heart problems, whether they participated in regular physical activity, and whether they were afraid of heights. We excluded all participants with agoraphobia, acrophobia, and fear of heights.

2.2 Experimental protocol

All data collection took place at the Plateforme d'Etude de la Sensorimotricité at the Université de Paris Cité, Paris. We used a headset, the HTC Vive, to immerse the subjects in VR. To increase the anxiety level during locomotion, we used the Richie's Plank video game: the person progressed on the ground and on a narrow plank placed between two buildings at the height of the 30th floor.

The recordings of the locomotion episodes were performed with the Codamotion 3D Analysis System (Charnwood Dynamics, Leicestershire, United Kingdom) (Figure 1A). Infrared light signals generated by the markers placed on the anatomical landmarks were captured by the Coda sensor module at 100 Hz. The data were processed with the Codamotion ODIN software on a personal computer with a Microsoft Windows-based operating system. A total of 24 Coda active markers were placed on the body in four segments (head, trunk, legs, and feet), with a minimum of three markers placed for each segment (Figure 1A). Markers were detected by four Coda CX1 units placed in the working space in the laboratory to cover the running range.

- Four markers were placed on the headset (one on the forehead was placed on the Xsens sensor, one each on the left temporal bone and right temporal bone, and one on the external occipital protuberance).
- Two markers were placed at the level of the left and right acromion.
- Two markers were placed at the left and right wrists and one marker at the left and right elbows.

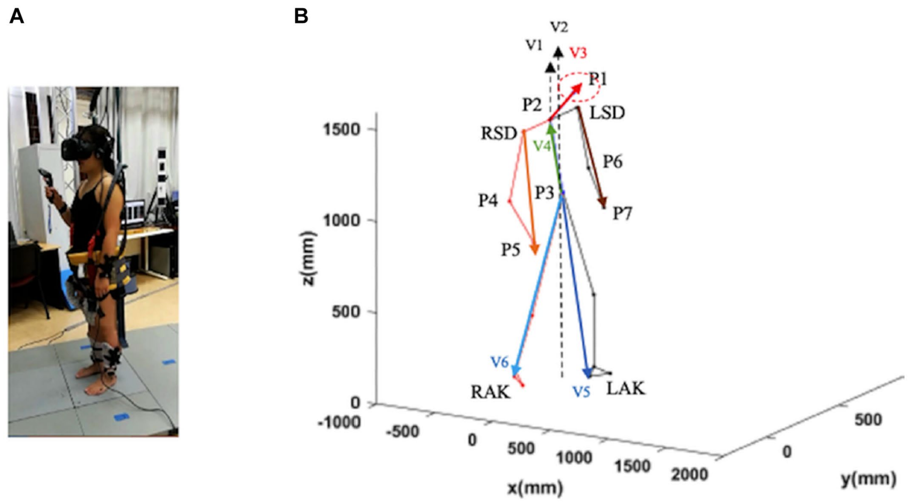


FIGURE 1
Schematic representation of a subject in a static position. Trunk inclination was defined as the angle between the gravity vector passing through P3 and the vector P3–P2 (V4; green vector). Head inclination was defined as the angle between the gravity through P2 and the vector P2–P1 (V3; red vector). Leg inclination (left and right) was defined as the angle between the gravity vector passing through P3 and the vector P3–LAK (V5; blue vector). and the vector P3–RAK (V6; blue vector). Arm inclination (left and right) was defined as the angle between the gravity vector passing through P2 and the vector LSD-P7 and the vector RSD-P5. LSD, left acromion; LAK, left ankle; P1–P7, virtual points; RSD, right acromion; RAK, right heel (see text for further details).

- Two markers were placed at the level of the left and right hips.
- Two 4-marker clusters were installed below the right and left lateral condyles of the tibia.
- Six markers were placed on the feet (one marker each on the left and right heel elbow, on the left and right heel at the low level, and on the right and left fifth metatarsal-phalangeal joint).

The participant wearing the headset displaying the Richie's Plank video game was barefoot and protected with a harness when walking. The protocol consisted of three scenarios.

1) The VR game started: the individual was in the elevator. The person decided it was time to start walking on the ground (G1 from now on) in a virtual landscape depicting a street. They were then asked to "stop and come back after 4,000 mm of progression."
2) The individual was in the elevator and selected the "plank" with a VR handle. The elevator door opened when the subject "arrived" at the 30th floor (H from now on). A 200 × 4,000 mm virtual suspended board was in front of the subject. The subject was asked to walk on the suspended board and decided for himself when to start walking. The subject was asked to stop and then return to the elevator when he reached the end of the board. Then, the subject selected "ground" with a VR handle in the elevator and descended to ground level.
3) Scenario 1 was repeated (G2 from now on).

2.3 Data processing

2.3.1 Static marker extraction

We defined seven virtual points based on the collected location data of markers for each part (P) (Figure 1B): P1: the midpoint of the four sensors on the head; P2: the midpoint of the line connecting the two shoulders; P3: the midpoint of the line connecting the two waist points; P4 and P6: the midpoint of the two sensors on the elbows; P5 and P7: the midpoint of the two sensors on the wrists.

To determine the skeletal configuration of each participant in the sagittal, frontal, and transversal planes, we computed four inclination angles:

1) Head inclination angle was calculated as the angle between the vector of P2-P1 (V2) and the vertical axis in the sagittal and frontal planes and the angle between the vector of P2-P1 (V2) and the anterior–posterior axis in the transverse plane.
2) Trunk inclination: the angle between the vector of P3-P2 and the vertical axis (V2) calculated in the sagittal and frontal planes, and the angle between the vector of P3-P2 (V4) and the anterior–posterior axis calculated in the cross-sectional plane.
3) Leg inclination: the angle between the vector of P3-left ankle (P3-LAK; V5) or P3-right ankle (P3-RAK; V6) and the vertical axis calculated in the sagittal plane and the frontal plane, and only the leg inclination of the forward step calculated. The angle between the vector of P3-LAK (V5) or P3-RAK (V6) and the anterior–posterior axis was calculated in the transversal plane.
4) Arm inclination: the angle between the vector right acromion-P4 (RSD-P5) or left acromion (LSD-P7) and the vertical axis V1 in the sagittal and frontal planes. The angle between the vectors RSD-P5 or LSD-P7 and the anterior–posterior axis was calculated in the transversal plane.

2.3.2 Gait variable

We used six gait parameters. Of these, step width, step length, step time, and step height were calculated directly from the gait cycle using the markers mounted on each foot. We also calculated the velocity and acceleration of six body segments: head, trunk, arm, thigh, calf, and feet.

2.3.3 Jerk

Jerk is the third derivative of position. It is a measure of the rate at which each part of the body is accelerated. First, the positional data for each marker were smoothed using a fourth-order, zero-lag Butterworth filter as described by Hreljac (1993). Second, the second derivative (acceleration) of the positional data involved using finite difference equations. Finally, the acceleration data were smoothed, and the first derivative of the acceleration $\frac{d(Acc)}{dt}$ (jerk) was calculated.

For the transversal plane, jerk was calculated as follows:

$$JERK = \frac{1}{2}\int_0^t \left(\frac{dAccX}{dt}\right)^2 + \left(\frac{dAccY}{dt}\right)^2$$

where $AccX$ corresponds to the obtained acceleration in the medio-lateral axis and $AccY$ corresponds to the obtained acceleration in the anterior–posterior axis. We calculate Jerk in the other planes by combining the acceleration in the other directions: $AccX$ and $AccZ$ for the frontal plane and $AccY$ and $AccZ$ for the sagittal plane.

2.3.4 Root mean square (RMS)

The RMS of trunk acceleration is frequently used in gait analysis. For marker displacement, the RMS amplitude represents the standard deviation of the displacement of the marker. This parameter measures the average absolute displacement around the mean marker and is often used. For example, a decrease in the RMS amplitude of the center of pressure represents an increased ability to preserve an upright stance. An increased RMS value suggests a decreased ability to maintain postural control.

2.3.5 Sample entropy

Sample entropy is a variation of the approximate entropy method. Sample entropy is the foundation for determining the complexity of both stationary and non-stationary signals. Sample entropy is the negative natural logarithm of the probability that two sequences will be similar for $m+1$ data points divided by the probability that two sequences will be similar from data points. For two data points to match, they need to be within a range of tolerance of ± r, which is from 10% to 20% of the standard deviation of the original dataset. The match of the template sequence to itself is excluded from this calculation. The elimination of this self-matching is the distinction between sample entropy and approximate entropy. The equation for calculating the sample entropy of a set of time series data is as follows:

$$SampEnt = \ln\left(\frac{\sum_{i=1}^{N-m} n_i'^{m}}{\sum_{i=1}^{N-m} n_i'^{m+1}}\right)$$

where N is the number of points in the dataset, $n_i'^{m}$ is the number of vector matches for vectors with length m, and $n_i'^{m+1}$ is the number of vector matches for vectors with length $m+1$.

2.3.6 Two-thirds power law

A mathematical equation known as the two-thirds power law, proposed by Lacquaniti et al. (1983), shows that the kinematics of many different human movements obey an identical relationship between the tangential velocity and the curvature of the motor trajectories. This law states that $v = \gamma\kappa^{-\beta}$ represents a robust local relationship between the geometrical and temporal aspects of human movement, represented by curvature κ and speed v, with a piecewise constant γ and exponent value $\beta = ⅓$. This law has been partially studied for the trajectory of the center of mass of the human body during walking (Tesio et al., 2011). The trajectory of the center of mass was segmented into high- and low-curvature segments. The β coefficient was close to the expected one-third value if the complete trajectory was considered. However, with high-curvature segments, the β coefficient is markedly higher ($\beta = 0.486$). By contrast, with low-curvature segments, the β coefficient is markedly lower ($\beta = 0.185$). In an equivalent form, let ω be the angular or curvilinear speed, r_c the radius of curvature, $C = 1/r_c$ the curvature, and k a constant. In its simplest form, the law predicts that $\omega = kC^{2/3}$, where k is a constant. Hence, the popular term is the two-thirds power law.

2.4 Statistical analysis

Statistical analysis of individual gait and its dynamic parameters followed three different scenarios: 16 participants on ground (G1), aerial (H), and secondary ground (G2). For all comparisons, a p-value of ≤0.05 was considered statistically significant. Statistical analyses and variable computation involved using MATLAB 2019.

The current statistical procedure begins with the examination of multiple variables through a rigorous process. To assess whether there are statistically significant differences within each variable across our three conditions (G1, H, and G2), each variable underwent testing using a univariate repeated measures analysis of variance (rm-ANOVA), and their p-values were adjusted by Bonferroni correction (Table 1). This correction method divides the desired significance level (here $a = 0.05$) by the number of comparisons to ensure that the family-wise error rate is controlled at the desired level. If the univariate rm-ANOVA yields a significant result, indicating that there are indeed differences between the scenarios, a post-hoc analysis is conducted to delve deeper into the nature of these differences. We performed a typical post-hoc analysis, which involves pairwise comparisons through paired t-tests with Bonferroni adjustment.

Concerning the intra-individual differences in different scenarios, the variables that are computed per step (e.g., step length per step, step height per step, and angles per step) are examined through a systematic approach to maintain the overall Type I error rate while conducting multiple hypothesis tests.

In the first step of this procedure, an analysis of variance (ANOVA) is performed for each variable of interest, and the corresponding p-values are adjusted by Bonferroni.

For those variables that remain statistically significant, post-hoc analyses are conducted through pairwise t-tests. Importantly, the Bonferroni correction is once again applied at this stage, ensuring that the significance level for each pairwise comparison is appropriately adjusted to control the overall Type I error rate. By adhering to this two-step procedure, we can confidently explore multiple hypotheses without inflating the risk of Type I errors. The Bonferroni correction serves as a critical safeguard in the process, enabling rigorous statistical control throughout the multiple testing process, both in the initial ANOVA analyses and in subsequent post-hoc pairwise comparisons.

TABLE 1 **Outcomes of significance subsequent to the application of multiple univariate repeated measures analysis of variance (rm-ANOVA) with Bonferroni adjustment, indicated by a + sign.**

	Head			Trunk			Arm			Thigh			Calf			Foot		
	Tr	Sg	Fr	Tr	Sg	Fr	Tr	Sg	Fr	Tr	Sg	Fr	Tr	Sg	Fr	Tr	Sg	Fr
ß factor										+	+		+					
Jerk																		
Entropy							+	+	+									
RMS																		
Angle																		
Speed	+			+			+			+			+			+		
Acc																		
	Step length			Step time			Step height			Step width								
Gait	+						+											

It is crucial to highlight that, for parameters retaining significance following the aforementioned (strong indeed) p-value adjustment, post-hoc pairwise analyses revealed significant differences across all pairs of conditions (G1, H, and G2).

For those comparisons that remain significant after the above steps, the Cohen's D effect size is calculated.

Cohen's D categorizes the significant changes in three standard effect size classes: small ($0.2 \leq D < 0.5$), medium ($0.5 \leq D < 0.8$), and large ($0.8 \leq D$). For each of these classes, we assign an arbitrary weight through the number of stars (*), * for small, ** for medium, and *** for large effect sizes, which will be used to weigh these differences appropriately.

Using the stars mentioned above, we finally define a score for each participant that indicates the overall statistical change that we were able to observe during locomotion at the level of the head, trunk, and limbs when comparing the characteristics during G1 vs. H, H vs. G2, and G1 vs. G2.

We have 16 types of parameters: Gait, 3D Angles, 3D ß factor, 3D Jerk, 3D RMS, and 3D entropy. For every individual, we checked for significant changes for every parameter (p), in every body segment (bs) in all three comparisons c (G1-H, H-G2, and G1-G2) in all three planes (ax). As was mentioned previously, every parameter was attributed by a number of stars s (no star for non-significant, * for small, ** for medium, and *** for large effect sizes).

Considering that the maximum number score (MNS) that someone can take per type is given by $MNS_i = p * bs * c * s * ax$, we calculate the 16 relative scores per individual as:

$$S_i = \frac{Score_i}{MNS_\mathrm{i}}$$

where $Score_i$ is the summation of an individual's stars per type. The final score SC_j of every individual j is given by:

$$SC_j = \sum_{i=1}^{21} S_i$$

In this study, we considered six parameters for Gait in one plane, angles were calculated in four segments, and three planes and ß factors, Jerk, RMS, and entropy were calculated in six segments and three planes.

Let us give some examples of how the MNS is calculated:

$$MNS_{Gait} = 4(p)*1(bs)*3(s)*3(c)*1(ax) \\ +2(p)*6(bs)*3(s)*3(c)*1(ax) = 144.$$

$$MNS_{Angle} = 1(p)*4(bs)*3(s)*3(c)*3(ax) = 108.$$

$$MNS_{Beta} = 1(p)*6(bs)*3(s)*3(c)*3(ax) = 162.$$

$$MNS_{Jerk} = 1(p)*6(bs)*3(s)*3(c)*3(ax) = 162.$$

$$MNS_{Entropy} = 1(p)*6(bs)*3(s)*3(c)*3(ax) = 162.$$

$$MNS_{RMS} = 1(p)*6(bs)*3(s)*3(c)*1(ax) = 162.$$

We calculated the propensity to assess percentages using the maximum total number of scores for the 16 subjects as a base.

We used changes in assessment scores to measure subjects' tendency to change in response to environmental changes. In this study, instead of calculating the evaluated value, we use the number of times the variable changes from ground to height or from height to ground.

Let us look at the total value of the various maximal changes VMC.

We have already calculated the maximal evaluated value of each variable per subject, and based on this, multiplied by the total number of subjects, is the maximal incidence. $VMC = 16 * MNS$.

$$VMC_{Gait} = NomberSubject * MNS_{Gait} = 16 * 144 = 2304$$

$$VMC_{angle} = NomberSubject * MNS_{angle} = 16 * 108 = 1728$$

$$VMC_{beta} = NomberSubject * MNS_{beta} = 16 * 162 = 2592$$

$$VMC_{jerk} = NomberSubject * MNS_{jerk} = 16 * 162 = 2592$$

$$VMC_{entropy} = NomberSubject * MNS_{entropy} = 16 * 162 = 2592$$

$$VMC_{RMS} = NomberSubject * MNS_{RMS} = 16 * 162 = 2592$$

We computed the score increased or reduced percentage using the following equation.

$$Changed\ Score\ Percentage = \frac{\begin{matrix}Number\ of\ variable\\ score\ changed\end{matrix}}{VMC}\%$$

3 Results

3.1 Average modulation of the markers of the perceptual-motor style by height exposure

For each participant within every condition (G1, H, and G2), the aforementioned assessment was conducted, involving the calculation of a total of 100 parameters. The outcomes are presented in Table 1, wherein parameters retaining significance following the adjusted repeated measures analysis of variance (rm-ANOVA) are denoted with a + sign. It is noteworthy that, owing to the application of multiple univariate rm-ANOVAs, a notably low alpha level (Bonferroni correction) was established. Consequently, the presence of the + sign signifies a compelling indication of pronounced change across the conditions.

Table 2 shows the percentage change in each marker of the perceptual-motor style defined in the methods section when the subject walks from ground to height (G1-H), from height to ground (H-G2), and for ground locomotion episodes before and after height exposure (G1-G2).

The first three rows show the percentages of change in these six markers averaged over the 16 subjects and for the three planes of space (frontal, sagittal, and transverse). Please note one exception: gait markers (length, height, width, duration, step speed, and acceleration) were only calculated in the sagittal plane. The last three rows show the percentage change in score for all subjects in each of the three planes of space (Fr-frontal, Sg-sagittal, and Tr-transverse).

Altogether, Table 2 shows the presence of significant changes in locomotion control in subjects exposed to height, not only while walking at height but also when returning to the ground. On the other hand, there was no significant change in the variables recorded according to the different planes of space, except for entropy. Finally, these averages should not mask the vast heterogeneity of the modulation of locomotion in subjects exposed to height, as detailed below.

TABLE 2 Modulation of the markers of the perceptual-motor style by height exposure.

	Gait (%)	Angle (%)	ß factor (%)	Jerk (%)	Entropy (%)	RMS (%)
G1-H	16	5	13	15	7	8
H-G2	25	7	15	19	10	20
G1-G2	25	7	14	17	10	19
Fr	–	6	12	17	11	12
Sg	–	8	16	18	10	18
Tr	–	5	15	16	7	18

3.2 Study of the skeletal configuration

We studied the skeletal configuration of the participants during locomotion, first on the ground (G1), then at height (H), and again on the ground (G2). That is, we measured the inclination of the head, trunk, forearms, thigh, and leg relative to the gravity vertical in the three planes of space (Figure 2).

3.2.1 In the sagittal plane

- From the ground upwards (G1-H), 52% of the participants modified their posture at the level of the head, little at the upper limbs (8%) and lower limbs (8%), and not at all at the trunk. That is, most participants tilted their heads forward.
- When returning to the ground (H-G2), 56% of participants modified their posture at the level of the head, less at the trunk (20%), and at the upper limbs (20%), and not at all at the lower limbs. Hence, most of the changes were head-straightening.
- There were some changes in the skeletal configuration when comparing the first episode of locomotion on the ground with the second one following the height test (G1-G2). A total of 56% of the participants modified their posture at the level of the head after 10 min at the height. Fewer modifications were observed at the trunk (32%) and upper limbs (32%) levels, and none at the lower limbs level. The changes were head-straightening, and trunk and upper limb positioning varied among persons.

3.2.2 In the frontal plane

- From the ground to height, 24% of the participants changed the posture of the upper limbs, and much less at the head (12%), trunk (12%), and lower limbs (8%). These changes consisted of abduction (24%) of the forearms, a slight inclination of the head

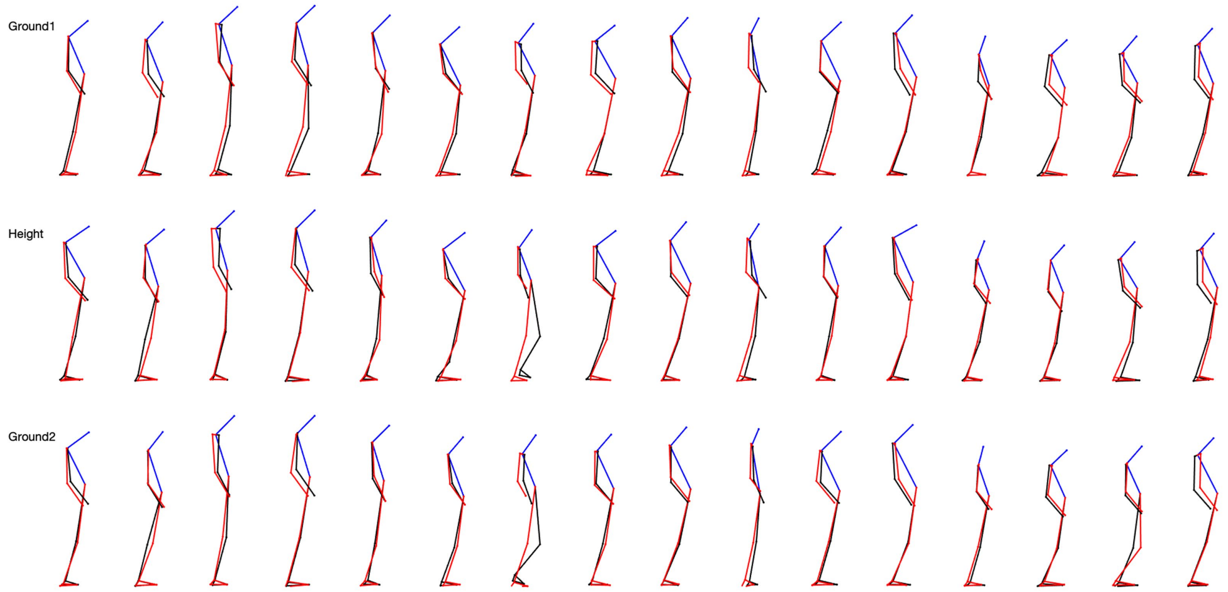


FIGURE 2
5-step overlapping skeletal posture configuration in sagittal view for 16 subjects. The top row is the situation on the ground, the middle row is the situation at height, and the last row is the situation from height back to the ground.

in one direction or the other, a straightening of the trunk, and a reduction of the support polygon.

- When returning to the ground, 52% of the participants modified their posture at the upper limbs, less at the lower limbs (20% of participants), at the head (12%), and at the trunk (12%). These changes consisted of a widening of the support polygon and an adduction or abduction of the forearms.
- When comparing the first episode of locomotion on the ground with the second one following the height test, 44% of the participants modified their posture at the upper limbs (abduction or adduction), 24% at the lower limb, and 8% at the head and trunk.

3.2.3 In the transverse plane

- From the ground to the height, 20% of the participants modified their posture at the level of the head and less at the trunk (12%), and upper and lower limbs separately (8%). The rotations could take place to the right or the left.
- When returning to the ground, 24% of the participants modified their posture at the upper limbs, at the level of the head (20%), and much less at the trunk (12%) and lower limbs (12%). The modification consisted of a decrease in the rotation of the head and trunk.
- When comparing the first episode of locomotion on the ground with the second one following the height test, changes in the skeletal configuration were mainly at the upper limbs (32%), and less at the lower limbs (12%), the head, and the trunk (8%).

3.2.4 Summary

A surprising finding was the very large variability of the skeletal configuration we observed in the participants when exposed to height in every experimental condition. It mainly concerned the position of the head in the sagittal plane and the configuration of the forelimbs in the frontal plane.

3.3 Study of the modifications of gait characteristics among experimental conditions

When participants changed from walking on the ground to walking at height (G1-H), some characteristics of their steps were modified. It was the case for their step duration (67% of participants), step width (60%), height (27% of participants), and length (33%). The body velocity was changed at the head (60%) and all other body segments (40%). The body acceleration was affected in a decreasing order at the arm (67%), the feet (60%), the trunk (60%), the lower limbs (50%), and the head (33%).

When participants walked again on the ground after having progressed at height (H-G2), some characteristics of their steps were also modified. This was the case for their step duration (47% of participants), step width (53%), height (60% of participants), and length (80%). The body velocity was changed at all body segments in more than 87% of the cases. The body acceleration was affected in a decreasing order at the arm (93%), the trunk (67%), the head (60%), the feet (53%), and the lower limbs (53%).

The characteristics of walking on the ground before and after height exposure (G1-G2) were also modified. This was the case for the step duration (73% of participants), step width (87%), height (67% of participants), and length (87%). The body velocity was changed at all body segments in more than 80% of the cases. The body acceleration was affected in a decreasing order at the arm (87%), the lower limbs (73%), the trunk (67%), the feet (67%), and the head (40%).

TABLE 3 Summary of each variable across the three conditions.

		G1-H						H-G2						G1-G2					
Variable	Plane	Head	Trunk	Arm	Thigh	Calf	Foot	Head	Trunk	Arm	Thigh	Calf	Foot	Head	Trunk	Arm	Thigh	Calf	Foot
ß factor	Fr	18	48	42	48	30	48	24	30	30	48	36	36	24	18	24	30	30	42
	Sg	42	42	42	54	48	12	36	36	48	78	48	54	24	30	60	60	60	42
	Tr	18	36	42	42	42	30	42	54	42	66	48	36	30	30	60	42	54	48
Jerk	Fr	6	24	18	30	42	12	12	24	24	42	48	60	12	12	30	48	48	54
	Sg	6	6	48	30	12	24	42	24	42	30	12	36	30	48	36	30	12	42
	Tr	30	24	48	36	36	60	48	42	84	48	60	36	24	54	60	42	42	48
Entropy	Fr	6	24	18	30	42	12	12	24	24	42	48	60	12	12	30	48	48	54
	Sg	6	6	48	30	12	24	42	24	42	30	12	36	30	48	36	30	12	42
	Tr	6	18	6	18	30	36	6	6	12	24	42	42	12	12	6	18	30	48
RMS	Fr	12	0	12	24	30	36	18	24	30	30	54	78	18	36	42	48	66	66
	Sg	24	24	18	24	24	36	66	66	78	66	78	78	60	60	66	60	60	60
	Tr	24	24	18	24	24	36	66	66	78	66	78	78	60	60	66	60	60	60

In summary, most of the characteristics of the gait were affected in most participants, but here again we observed a large disparity among subjects.

3.4 Study of the dynamic characteristics of locomotion

We studied the dynamic characteristics of locomotion first on the ground (G1), then at height (H), and again on the ground (G2). That is, we measured the ß factor, jerk, RMS, and entropy for the head, trunk, forearms, the two segments of the leg, and the foot in the three planes of space (sagittal, frontal, and transversal planes) for each condition (G1, H, and G2). Then we studied whether these variables changed across conditions, that is, when the participants changed from walking on the ground to walking at height (G1-H) and from walking at height to the ground (H-G2), and we compared the dynamic characteristics of locomotion during the first and second episodes of walking on the ground (G1-G2).

Table 3 summarizes our results. It illustrates the percentage of subjects who exhibited statistically significant modifications in the dynamic characteristics of their locomotion and which variables are concerned during the (G1-H) transition, the (H-G2) transition, and the (G1-G2) condition.

3.5 Summary

Some characteristics of the change in the dynamics of locomotion emerged. First, it was largely affected among participants across the three conditions. Second, the changes in the dynamics of locomotion followed a gradient, with more modification taking place at the leg level, then at the trunk level, and finally at the head level. Third, the ß factor emerges as a prominent marker of the influence of height on locomotion during the transition from ground to height (G1-H) and from height to ground (H-G2).

3.6 Stake percentage of change

Figure 3A illustrates the markers that helped to differentiate the perceptual-motor style of the participants during locomotion at a height. One can make three observations. First, it confirmed that the characteristics of locomotion varied considerably among the 16 subjects exposed to height. Second, their control of locomotion, despite this large variability, varied smoothly; that is, the participants could not be divided into distinct subpopulations. Third, exposure to height affected all variables characterizing locomotion, although their modulation was very different across subjects.

4 Discussion

Among the markers we previously used to describe perceptual-motor style during ground locomotion, we identified those that could account for its changes during and immediately after locomotion at height. A surprising result was the great heterogeneity of these changes in the 16 individuals we tested. Static markers (stable head, trunk, and limb configurations relative to the gravitational vertical) and dynamic markers (jerks, entropy, RMS, gate, and two-thirds power law) were modulated by height exposure.

4.1 Comparison with previous results

Static balance has been intensively studied during height exposure, but much less so during locomotion (Adkin and Carpenter, 2018). Peterson et al. (2018) studied the effects of VR during beam walking on physiological stress and cognitive load and found no significant difference in walking speed for low and high VR (Parsons et al., 2017). Schniepp et al. (2014), using a 6.7 m-long pressure-sensitive mat, found that when exposed to height, individuals sensitive to visual height intolerance walked more slowly, with reduced cadence, a shorter stride length, and increased double-support phase. Their locomotion was described as a slow, cautious, broad-based gait with small steps. They walked with flat foot contact and less dynamic vertical oscillations of body and head. This gait was not considered specific but rather similar to the cautious gait observed in visually deprived children and adults. Finally, in a study parallel to our own, Zhu et al. (2023) investigated the effects of virtual and physical elevation on physiological stress during height exposure. In this study, plates were placed at the lower ends of the platform, causing slight instability during walking. In addition, a surface layer of foam was introduced to add further postural instability when walking on the raised platform. These instability factors created difficulties that had the effect of increasing the individual's anxiety level when walking. The platform's walking space was 2.4 m long and 0.3 m high, while ours was 4 m long and 0.2 m high. Averaging the values recorded in their participants, Zhu et al. (2023) found that increased threat perception (height) prompted the individual to use a cautious walking style with reduced step length and increased steps taken and trial time. Overall, our results are consistent with these earlier findings. They are also consistent with the concept of cautious walking, coined by gerontologists. However, these earlier studies did not aim to detail the motor control of each individual tested at height and therefore did not describe the heterogeneities of their perceptual-motor style.

4.2 Exposure to height modulates locomotion. What could be the etiological factors?

As mentioned above, among the markers we previously used to describe perceptual-motor style during ground locomotion, we identified those that could account for its alterations during height locomotion. However, our protocol failed to uncover the causes of the surprisingly large behavioral differences we found between individuals when exposed to height. Presumably, a range of factors were at play, as described by Adkin and Carpenter (2018) in their review.

Fear and anxiety could play a role in modulating the response to height exposure. In the study by Nakahara et al. (2000), recognition that participants were standing at height had two effects: non-acrophobic participants had less body sway at rest if their eyes were uncovered and open, while acrophobic participants showed the opposite response in subsequent trials. In addition to their height intolerance, acrophobic individuals had poorer postural performance in static and dynamic balance tasks (Boffino et al., 2009). Personality traits measured by questionnaires on anxiety and willingness to take physical risks corroborated alterations in static postural control when standing on the edge of a raised platform (Zaback et al., 2019) or during locomotion on the edge of a raised platform (Zhu et al., 2023). In other words, acrophobic people have a fear of falling, and some of the participants we tested exhibited similar symptoms, suggesting that they may share common anxiogenic traits.

The modulation of the perceptual-motor style by exposure to height could also be linked to a greater or lesser visual dependence for the control of balance and locomotion: the distance from the stationary visual scene becomes too great to be detected in order to counteract body movements (Hüweler et al., 2009; Brandt and Huppert, 2014). This leads to impaired motor control, described by Brandt et al. (2015) as a "fear of heights," resulting in tonic immobility. Fear of falling from a height triggers antigravity muscle co-contractions that increase the sensitivity of sensorimotor balance reflexes. This scenario represents an atavistic motor response resembling death feinting, a primitive behavior widespread in the animal world. The resulting rigid regulation of body sway in turn aggravates the subjective and objective imbalance and initial anxiety, initiating a vicious circle.

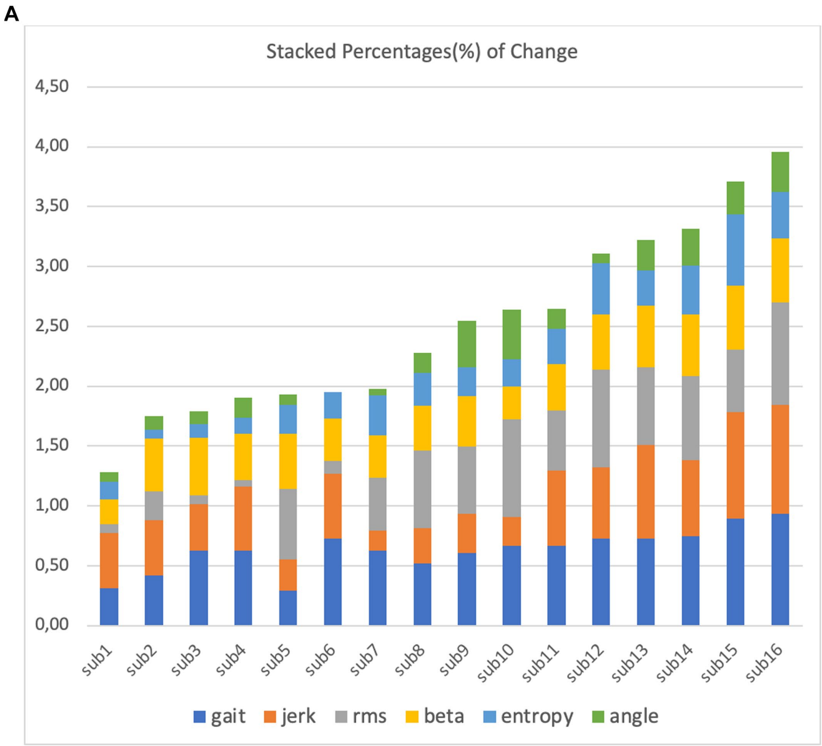
Height exposure also modulates postural and locomotor control by influencing how attentional resources are allocated. Several studies have shown changes in attentional focus and alterations in static postural control (see Adkin and Carpenter, 2018 for a review), suggesting that they are causally linked. When threatened, individuals tend to control their posture more consciously (Huffman et al., 2009; Peterson et al., 2018). This may have contributed to changes in some of the markers we used to monitor locomotion at height and, to a variable extent, among the individuals we tested.

Finally, neural networks involved in emotional control have been shown to modulate motor control in animal models, and evidence has also been established in humans. Notably, threat increased muscle spindle sensitivity, ib reflex gain, vestibular control of balance and gaze, and oculomotor control (Adkin and Carpenter, 2018 for a review).

4.3 Variability in perceptual-motor style between participants

The high variability we observed among participants for both static and dynamic features of height locomotion and its persistence on the ground is the result of the variability of each perceptual-motor style marker we recorded. This contrasts with the results of our previous study (Vidal and Lacquaniti, 2021) concerning the perceptual-motor style of individuals when walking and running on a treadmill at ground level. These are illustrated in Figure 3B for comparison. In this case, a few markers are enough to differentiate the individuals.

This is reminiscent of a previous study (Bonan et al., 2013) we undertook on control and hemiplegic patients to test their sensitivity to proprioceptive, vestibular, and visual stimuli with regard to postural control (Bonan et al., 2013). Once again, we found that controls and stroke patients showed significant inter-individual variation in response to all three types of sensory stimulation. The control group could be divided into two subgroups according to whether their resting postural control was unaffected or affected by the sensory stimuli. However, none of the hemiparetic patients were insensitive to sensory stimulation. Not only were they excessively



B

	FRONTAL				SAGITTAL				TRANSVERSAL			
	HEAD	TRUNK	LEGS	FEET	HEAD	TRUNK	LEGS	FEET	HEAD	TRUNK	LEGS	FEET
Inclination at rest					+	+						
Inclination while locomotion					+	+						
JERK		+	+									
RMS									+	+		
ENTROPY			+	+	+	+			+	+		
β factor	+	+	+	+	+	+	+	+	+	+	+	+

FIGURE 3
(A) Stacked percentage of change from every variable: gait, jerk, RMS, beta, entropy and angle **(B)** to characterize the perceptual motor style of individual participants when walking and running on a treadmill at ground level.

dependent on visual information to control their posture, but they were also more sensitive to vestibular and proprioceptive information than the controls. Overall, for both static postural control and locomotion, modulation of perceptual-motor style seems relatively straightforward to characterize in healthy subjects, both in terms of markers and taxonomy. In contrast, when pathologies occur or challenging conditions are tested, the perceptual-motor styles become more heterogeneous and affect more markers of motor control.

4.4 A gradient in terms of timing and variability during locomotion at a height

In a previous study, we tested subjects with multiple balance perturbations provided by unpredictable translations of the supporting surface in different directions and speeds (Le Goic et al., 2018). Our data showed that there is little time to adjust the way one falls from a standing position. During the initial part of a fall, the observed trajectory results from the interaction between the destabilizing external force and the body; the intrinsic inertial properties of joints, ligaments, and the musculotendinous system then have a major contribution. This passive phase is then followed by an active phase, which consists of a corrective response to the postural perturbation. The motor synergies at play followed a temporal gradient from the limb to the neck muscles. Furthermore, our results revealed that visual and vestibular information could not detect the fall at its onset because the head remained stable with respect to space. That is the participants are prepared for the impact on the basis of the proprioceptive information. Similarly, in the present study, the variability of the movements of the body segments during the locomotion on the plank followed an ascending order from legs to head. In order to control the cautious gait on the plank, visual information was also of no use, being the distance to the ground; vestibular information contributed little, being the low-frequency content of the head movement. That is, proprioceptive afferences were also the main source of information to control locomotion. Therefore, it would be the emphasis on proprioceptive information during fall and the cautious gait that would explain the leg-to-arms-to gradient we observed both in terms of timing and variability.

4.5 Limitations

Our study had several limitations. Our sample size was small. Although the participants were of approximately the same age and cultural background and had no pathology, a larger panel might have revealed an age dependency and/or established a partition in the three types of populations, as illustrated in Figure 3A. Furthermore, we did not record participants' visual dependence, neurovegetative responses, or attention allocation strategies during postural and locomotor control, nor did we test their psychological traits using suitable questionnaires. This could have explained the underlying causes of heterogeneity in participants' perceptual-motor style when exposed to height. Another limitation of this study was the extensive multiple testing procedures and the application of the Bonferroni correction of *p*-values, which, while controlling for Type I errors, may have increased the likelihood of Type II errors by overly conservative adjustment, potentially leading to the non-detection of true effects. Nevertheless, this limitation can be interpreted as substantiating the robustness of the results presented in this study. It suggests the possibility that less pronounced changes, yet to be identified, may exist and warrant investigation in future research.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Ethics statement

The studies involving humans were approved by CER U-Paris N° 2021-12-WANGVIDAL Présidente: Jacqueline Fagard. PROTOCOLE: Influence of fear stimuli under virtual reality to the posture control. Noms du/des chercheur(s): DW and P-PV. Email pour la correspondance: danping.wang@parisdescartes.fr; pierre-paul.vidal@parisdescartes.fr. Labo/Service: Plateforme d'Etude de la Sensorimotricité; Université de Paris, Évalué à la séance du 6 avril 2021. AVIS: favorable. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

DW: experience, signal processing, data analysis, paper writing. IB and NV: data analysis. JC and LO: signal processing. P-PV: data analysis and paper writing. All authors contributed to the article and approved the submitted version.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

References

Adkin, A. L., and Carpenter, M. G. (2018). New insights on emotional contributions to human postural control. *Front. Neurol.* 9:789. doi: 10.3389/fneur.2018.00789

Boffino, C. C., de Sá, C. S., Gorenstein, C., Brown, R. G., Basile, L. F., and Ramos, R. T. (2009). Fear of heights: cognitive performance and postural control. *Eur. Arch. Psychiatry Clin. Neurosci.* 259, 114–119. doi: 10.1007/s00406-008-0843-6

Bonan, I. V., Marquer, A., Eskiizmirliler, S., Yelnik, A. P., and Vidal, P. P. (2013). Sensory reweighting in controls and stroke patients. *Clin. Neurophysiol.* 124, 713–722. doi: 10.1016/j.clinph.2012.09.019

Brandt, T., and Huppert, D. (2014). Fear of heights and visual height intolerance. *Curr. Opin. Neurol.* 27, 111–117. doi: 10.1097/WCO.0000000000000057

Brandt, T., Kugler, G., Schniepp, R., Wuehr, M., and Huppert, D. (2015). Acrophobia impairs visual exploration and balance during standing and walking. *Ann. N. Y. Acad. Sci.* 1343, 37–48. doi: 10.1111/nyas.12692

Hreljac, A. (1993). Preferred and energetically optimal gait transition speeds in human locomotion. *Med. Sci. Sports Exerc.* 25, 1158–1162. doi: 10.1249/00005768-199310000-00012

Huffman, J. L., Horslen, B. C., Carpenter, M. G., and Adkin, A. L. (2009). Does increased postural threat lead to more conscious control of posture? *Gait Posture* 30, 528–532. doi: 10.1016/j.gaitpost.2009.08.00

Huppert, D., Wuehr, M., and Brandt, T. (2020). Acrophobia and visual height intolerance: advances in epidemiology and mechanisms. *J. Neurol.* 267, 231–240. doi: 10.1007/s00415-020-09805-4

Hüweler, R., Kandil, F. I., Alpers, G. W., and Gerlach, A. L. (2009). The impact of visual flow stimulation on anxiety, dizziness, and body sway in individuals with and without fear of heights. *Behav. Res. Ther.* 47, 345–352. doi: 10.1016/j.brat.2009.01.011

Lacquaniti, F., Terzuolo, C., and Viviani, P. (1983). The law relating the kinematic and figural aspects of drawing movements. *Acta Psychol.* 54, 115–130. doi: 10.1016/0001-6918(83)90027-6

Le Goic, M., Wang, D., Vidal, C., Chiarovano, E., Lecompte, J., Laporte, S., et al. (2018). An initial passive phase that limits the time to recover and emphasizes the role of proprioceptive information. *Front. Neurol.* 9:986. doi: 10.3389/fneur.2018.00986.;10:118

Lelard, T., Stins, J., and Mouras, H. (2019). Postural responses to emotional visual stimuli. *Neurophysiol. Clin.* 49, 109–114. doi: 10.1016/j.neucli.2019.01.005

Mantilla, J., Wang, D., Bargiotas, I., Wang, J., Cao, J., Oudre, L., et al. (2020). Motor style at rest and during locomotion in human. *J. Neurophysiol.* 123, 2269–2284. doi: 10.1152/jn.00019.2019

Nakahara, H., Takemori, S., and Tsuruoka, H. (2000). Influence of height on the spatial orientation and equilibrium of the body. *Otolaryngol. Head Neck Surg.* 123, 501–504. doi: 10.1067/mhn.2000.107316

Parsons, T. D., Gaggioli, A., and Riva, G. (2017). Virtual reality for research in social neuroscience. *Brain Sci.* 7:42. doi: 10.3390/brainsci7040042

Peterson, S. M., Furuichi, E., and Ferris, D. P. (2018). Effects of virtual reality high heights exposure during beam-walking on physiological stress and cognitive loading. *PLoS One* 13:e0200306. doi: 10.1371/journal.pone.0200306

Schniepp, R., Kugler, G., Wuehr, M., Eckl, M., Huppert, D., Huth, S., et al. (2014). Quantification of gait changes in subjects with visual height intolerance when exposed to heights. *Front. Hum. Neurosci.* 8:963. doi: 10.3389/fnhum.2014.00963

Teggi, R., Comacchio, F., Fornasari, F., and Mira, E. (2019). Height intolerance between physiological mechanisms and psychological distress: a review of literature and our experience. *Acta Otorhinolaryngol. Ital.* 39, 263–268. doi: 10.14639/0392-100X-2190

Tesio, L., Rota, V., and Perucca, L. (2011). The 3D trajectory of the body centre of mass during adult human walking: evidence for a speed-curvature power law. *J. Biomech.* 44, 732–740. doi: 10.1016/j.jbiomech.2010.10.035

Vidal, P. P., and Lacquaniti, F. (2021). Perceptual-motor styles. *Exp. Brain Res.* 239, 1359–1380. doi: 10.1007/s00221-021-06049-0

Williams, J. H. G., Huggins, C. F., Zupan, B., Willis, M., Van Rheenen, T. E., Sato, W., et al. (2020). A sensorimotor control framework for understanding emotional communication and regulation. *Neurosci. Biobehav. Rev.* 112, 503–518. doi: 10.1016/j.neubiorev.2020.02.014

Zaback, M., Adkin, A. L., and Carpenter, M. G. (2019). Adaptation of emotional state and standing balance parameters following repeated exposure to height-induced postural threat. *Sci. Rep.* 9:12449. doi: 10.1038/s41598-019-48722-z

Zhu, H. Y., Chen, H. T., and Lin, C. T. (2023). The effects of virtual and physical elevation on physiological stress during virtual reality height exposure. *IEEE Trans. Vis. Comput. Graph.* 29, 1937–1950. doi: 10.1109/TVCG.2021.3134412

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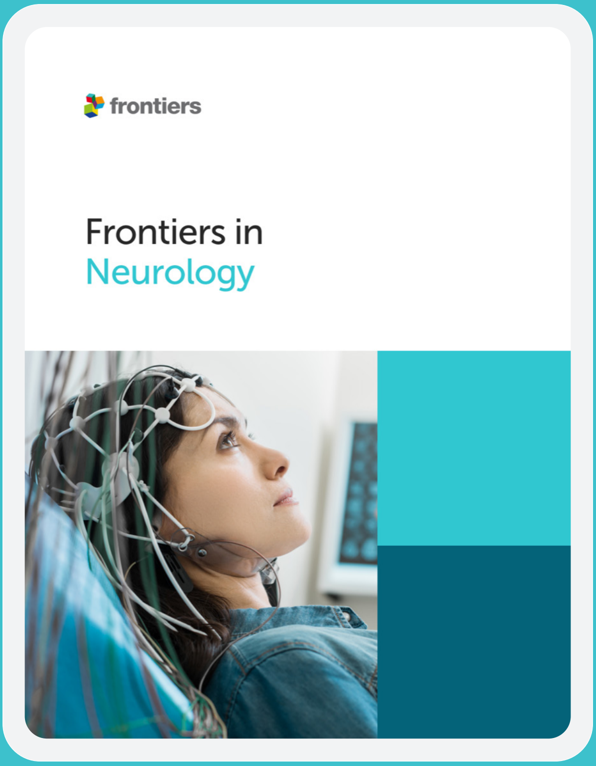
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