The Effect of Visual Stimuli on Postural Control in Standing Humans

by

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Abstract

Balance is crucial for upright stance and mobility, with the control of balance being fundamental to most daily activities including rising from bed and walking. Upright bipedal stance is inherently unstable as the majority of the body's mass is elevated high above the feet, creating instability and giving rise to a natural sway pattern. Balance control is a complex process involving sensorimotor systems, including the visual, vestibular, and somatosensory systems. Specifically, visual feedback has been demonstrated to be a potent sensory cue as the absence of vision leads to increased postural sway, while motion of the visual surround induces compensatory sway. We hypothesized that a rapid, transient, visual stimulus, akin to the motion parallax relayed during a push or slip, would evoke a rapid balance corrective reaction in the direction of the visual stimulus. Moreover, we hypothesized that the reaction would habituate with repeated exposure and provision of a stable light touch reference would stabilize or mitigate the balance reactions. Forty participants received 10 small, transient, visual displacements using the "moving room" paradigm. Visual stimuli were 2.5 cm in amplitude with a peak velocity of 124 mm/s. Twenty participants received forward visual displacements and the other twenty participants received backward visual displacement. In addition, half the participants received a stable light touch reference. Following the first visual stimulus 39 out of the 40 participants leaned in the direction of the visual stimulus, as demonstrated by a shift in the position of the center of pressure. However, the latency of the response was slow and unlikely to be a rapid balance reaction. Upon repeated exposure to the stimulus participants continued to lean in the direction the stimulus. The participants that received a stable light touch reference during the visual displacements also demonstrated a lean in the direction of the visual stimulus, although with smaller amplitudes. These results suggest that the slow latency postural leans exhibited by the participants are not balance reactions, but rather likely represent a postural reorientation of the body to realign with the new position of the visual surround. We suggest that this postural reorientation serves to reposition the head within the relocated spatial frame of reference of the visual surround. Further, the continued repositioning behaviour with repeated exposure and with the provision of light touch suggests that vision is a potent sensory cue in maintaining postural orientation.

Preface

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CHAPTER 1

Introduction

Balance is crucial to upright stance and mobility. Control of balance is fundamental to most daily activities including rising from bed and walking. However, balance control is compromised as a result of many injury and disease states, as well as the normal aging process (Anson & Jeka, 2010; Sundermier et al., 1996; Wallmann, 2009). Loss of balance can lead to falls, which are a leading cause of injury and hospitalization with the risk of falling increasing with age (Smith et al., 2021; Soomar & Dhalla, 2023; Wallmann, 2009). Upright bipedal stance is inherently unstable, as a majority of the body's mass is elevated high above the feet, this creates instability and gives rise to a natural sway pattern as gravity and other internal and external forces act upon the mass of the body. Efforts to reduce the number and severity of falls in balance compromised populations require foundational understanding of how the nervous system controls the motion of the body to maintain upright stance.

Maintaining an upright posture is a complex process involving sensorimotor systems (Peterka, 2002; Urbaniak-Olejnik et al., 2022). The neural control of balance integrates sensory information from multiple systems including visual, vestibular, and somatosensory feedback (Anson & Jeka, 2010; Mackinnon, 2018; Witkin & Wapner, 1950). Neurological damage affecting the function of one or more sensory systems can lead to impaired balance control or falls. It is widely established that when a single sensory system is impaired, other systems can compensate and contribute to maintaining balance. For example, vision is relied on more by individuals with vestibular damage, in order to remain stable (Winter, 1995). Overall, complex sensory systems integrate to provide the nervous system with the necessary information for the control of balance.

Visual feedback is a potent sensory cue in balance control, as evident by the decreased stability observed when the eyes are closed (Edwards, 1946; Lee & Lishman, 1975; Sundermier et al., 1996). Standing with the eyes closed, thereby eliminating visual input, results in an increased postural sway (Edwards, 1946). Moreover, individuals with congenital or acquired visual deficits demonstrate imbalance and increased postural sway during upright stance, compared with vision-intact individuals (Ray et al., 2008; Rutkowska et al., 2015; Tomomitsu et al., 2013). In fact, Rogge et al. (2021) observed that visually impaired adolescents have larger postural sway than the sway observed with unimpaired, but blindfolded adolescents. Rogge et al. (2021) argue that individuals who are blind have decreased balance function, compared to blindfolded sighted individuals, despite evidence indicating that the vestibular and somatosensory contributions to balance control are enhanced, suggesting that visual feedback is critical to normal balance control. Visual feedback is critically involved in balance control, with the elimination of visual input increasing postural sway and thus decreasing stability.

Visual feedback is suggested to inform balance control by detecting the motion of the head relative to the surroundings. This was demonstrated by Lishman & Lee (1973) who showed that participants standing on a moving trolley detected the motion of the trolley when visual feedback was not available, but reported the trolley was stationary if the visual surround moved synchronously with the trolley. Moreover, if a visual surround is oscillated slowly, the sway of the standing participant becomes entrained to the motion of the visual surround (Lee & Lishman, 1975). Extending this finding, Lestienne et al. (1977) demonstrated that a continuous, linear visual motion, such as what might be experienced when standing within a moving vehicle, resulted in a persistent lean in the direction of the visual motion. Similarly, Wolsley et al. (1996) demonstrated that participants viewing a rotating disc would lean laterally, in the direction of the disc's rotation. Together, these findings indicate that visual feedback provides cues of the relative motion of the head in space, assuming that the surrounding environment is stationary.

Extending the work of Lestienne et al. (1977), Bronstein (1986) demonstrated that postural responses evoked by episodic displacements of a visual surround habituated to repeated exposures to the stimulus. This suggests that the potent influence of visual input to balance control is plastic and can be adapted based on context and prior experience. However, Bronstein used a visual displacement with an amplitude of 30 cm lasting over the course of 12 s, whereas the postural response evoked by the stimulus occurred within the first 600 ms, when the visual surround had moved 1 cm or less. This suggests that events very early in the visual stimulus are involved in triggering the corrective reactions observed. Moreover, the response onset of 600 ms is quite slow, much slower than the rapid balance corrective responses observed following disturbances to the support surface (Diener, Dichgans, Bootz, et al., 1984; Diener et al., 1988; Rasman et al., 2018) or touch references (Misiaszek et al., 2016; Misiaszek & Vander Meulen, 2017). Therefore, it remains unclear if visual feedback is capable of initiating a balance correction that would protect against a fall. Consequently, the studies in the present thesis aim to determine if small, brief displacements of the visual surround, consistent with displacements that might be experienced with unexpected balance disturbances, are capable of initiating corrective reactions of sufficiently short latency (within 200 ms, Misiaszek et al., 2016; Misiaszek & Vander Meulen, 2017) to be compatible with fall prevention. It is also possible that the extinction of the response observed by Bronstein (1986) was the consequence of the excessively large stimulus that persisted long after the participant began to react to the unnatural stimulus parameters. Therefore, a second aim of this study is to assess if any responses that might be observed habituate with repeated exposure, when the stimulus is more ecological. Previously, it has been demonstrated that light touch of < 1N is capable of mitigating the impacts of reduced visual feedback (Jeka & Lackner, 1994; Kotecha et al., 2016). In addition, light touch has been shown to suppress visually-evoked sway and lean (Kozhina et al., 2015; Meyer et al., 2013; Peterka & Benolken, 1995). However, visually-evoked sway and lean are not eliminated by the presence of a touch reference, suggesting that the nervous system is incapable of completely ignoring sensory information, even if inaccurate (Kozhina et al., 2015). Therefore, a third aim of this thesis will be to assess whether corrective responses evoked by a small, brief visual stimulus, if present, will also persist when balance is stabilized by a light touch reference.

Previously, it was demonstrated that a push at the waist of walking participants resulted in balance corrections with an onset latency of about 100 ms (Forero & Misiaszek, 2013). Motion analysis estimated that the body center of mass had been displaced 1.25 cm by the time of the response onset. In a subsequent study, a 1.25 cm displacement (peak velocity of 124 mm/s) of a touch reference during standing, based on these parameters, also resulted in rapid (approximately 100 ms onset) balance corrective reactions (Misiaszek et al. 2016). It is argued that disturbances that cause the physical displacement of the body, as indicated by the movement of the torso or finger, will also likely displace the head, and induce associated visual feedback. To begin exploring how a visual perturbation effects balance, we decided to use twice the displacement of the body to ensure that we deliver a visual perturbation that sufficiently induces a postural reaction. Therefore, for this study, a visual field displacement of 2.5 cm (peak velocity of 124 mm/s) with a duration of 250 ms was used.

This thesis aims to understand the role of vision in balance reactions. Specifically, I will ask if a small, transient visual stimulus, is capable of evoking a response sufficiently rapid to be functionally relevant for fall prevention. In the subsequent chapter, I will review the literature to provide additional background information addressing the biomechanical challenges of the control of upright stance, and the role of visual, vestibular, and somatosensory systems in balance control. Chapter 3 will provide details on the methods used to address the research questions. Chapter 4 will then provide the findings of the current study and Chapter 5 will discuss the findings of the study in context of the existing literature, address the limitations of this study, and provide insights into future studies arising from these findings.

CHAPTER 2

Review of Literature

The biomechanical challenge of bipedal balance

Human upright stance is inherently unstable and can be mechanically challenging. This is because a majority of the body's mass is elevated high above the feet (Winter, 1995). The most common bipedal stance in humans is with the feet side-by-side, where the surface area created by the two feet in contact with the ground makes up the base of support (BOS) (Winter, 1995). When humans are in bipedal stance, the centre of mass (COM) is located high above the BOS, making the COM susceptible to gravitational forces that can pull the COM beyond the BOS leading to a fall. Therefore, balance can be defined as the ability of a person to maintain a static position of the body in space which keeps the COM within the BOS (Carini et al., 2017; Pollock et al., 2016; Winter, 1995). A common model of balance control is the inverted pendulum model, which postulates that the body oscillates in the anterior-posterior direction with the ankle joint as the axis of rotation. The inverted pendulum model suggests that muscles acting across the ankle joint counteract the destabilization caused by gravitational forces to maintain balance (Morasso et al., 2019). In turn, the motion of the body created by this correction, and further acted upon by gravitational forces, must itself be reversed, leading to an oscillation of the body's mass referred to as postural sway. However, if the natural sway of the COM is not adequately controlled, or if there is an unexpected disturbance that pushes the COM beyond the limits of the BOS, gravity will accelerate the mass of the body creating a fall. Given that the head is positioned at the top of this mechanical structure, the pull of gravity on the mass of the body will accelerate the head towards the ground creating a high risk for a head injury. Therefore, it is essential to understand how the nervous system controls the actions of the muscles to prevent this catastrophic event and mitigate the chance of serious injury.

Falls can occur when the body COM moves beyond the boundaries of the BOS. During quiet standing, displacement of the COM outside the boundaries of the BOS can result from an unexpected change in the configuration of the BOS, such as the sudden loss of the support surface, or from internal and external perturbations acting upon the COM. Rogers & Mille (2018) describe internal perturbations as forces generated by the muscles in the execution of motor tasks, such as the raising of an arm, which can displace the COM. In contrast, external perturbations arise when extrinsic forces, such as a push, are applied to the body resulting in displacement of the COM. In either case, preventing a fall requires maintaining the COM within the limits of the BOS. This is achieved by a combination of proactive and reactive control strategies. For example, the internal perturbation to the COM that is created by raising the arm is typically counteracted proactively by activating postural muscles prior to initiating the prime movers of the focal movement, often referred to as an anticipatory postural control strategy (Papa et al., 2015; Rogers & Mille, 2018). In contrast, unexpected external perturbations can only be counteracted after the disturbance has been detected by sensory feedback, requiring a balance response that lags the perturbation and is reactive (Rogers & Mille, 2018). Reactive balance corrections can occur quickly, as early as 70 – 90 ms after the onset of perturbation (Rogers & Mille, 2018). Correcting for a disturbance in the relationship between the COM and the BOS, either proactively or reactively, can be achieved by applying forces through a stable BOS, often referred to as a “fixed-support” strategy, or by adjusting the construct of the BOS, such as by taking a step or grasping a rail, often referred to as a “change-in-support” strategy

(Maki & McIlroy, 1997). Moreover, the broad category of “fixed-support” strategies can be achieved by generating torques about the ankle, the “ankle strategy”, or hip, the “hip strategy”, or by raising the arms, depending upon the context and environmental constraints at the time (Maki & McIlroy, 1997). Therefore, maintaining the mechanical relationship between the position of the COM within the limits of the BOS can be achieved by many different strategies, that must be decided upon and executed quickly to prevent a fall following a perturbation.

Sensory systems and standing balance

The control of balance and the postural responses that are produced are informed by the complex sensory systems of the nervous system, including the visual, vestibular, and somatosensory systems. Each of these sensory systems continuously provides functionally relevant feedback while interacting with the motor systems to maintain balance control (Mackinnon, 2018). Indeed, it has been demonstrated that reduced or impaired visual, vestibular, or somatosensory feedback can adversely affect postural sway during quiet standing (Peterka & Black, 1989; Samuel & Solomon, 2015). Multisensory information relayed by these systems integrate to plan, execute, and modulate relevant postural responses as needed by providing proprioceptive and kinesthetic input (Mackinnon, 2018). Proprioception is the unconscious sensory feedback that informs limb, head and body position in space and during motion, while kinesthesia is the conscious awareness of the position and motion of the body and body segments (Mackinnon, 2018). Critically, the sensory systems provide information about the location, position and motion of the COM, as well as the structure, position, and boundaries of the BOS. In other words, the integrated sensory information is essential for informing the mechanical relationship between the COM and BOS. Disturbances to this relationship, whether created by internal or external perturbations, are then detected through this sensory feedback allowing for the rapid, corrective reactions required to prevent a fall. For example, the motion of the head might be detected by visual and vestibular inputs (Casale et al., 2023; Cullen, 2012; Rokszin et al., 2010), along with the feedback about the posture, orientation and motion of the limbs through muscle stretch receptors (Fitz-Ritson, 1982; Mackinnon, 2018; Proske & Gandevia, 2012). Combined, this constellation of sensory input would indicate the location and motion of the COM. Whereas, muscle proprioceptors, combined with cutaneous feedback from the feet would indicate the location and structure of the BOS (Inglis et al., 2002; Kuling et al., 2016; Mackinnon, 2018). If the COM was to sway beyond the limits of the BOS, the appropriate response can be generated to reposition the COM or change the structure of the BOS. Therefore, understanding the role of these individual sensory systems, and how they interact, is essential to understanding how balance is maintained and how balance corrective responses are generated to prevent falls.

The objective of my thesis is to understand the role of vision in reactive balance control, and more specifically, the potential contribution of visual feedback in generating corrective postural responses. However, as indicated above, the visual system does not contribute to balance control in isolation, but rather, is one component of an integrated, multisensory system. It is therefore pertinent to view the results of my thesis within this framework. Therefore, the following subsections of my literature review will review key contributions of each sensory system as they relate to their role in reactive balance control.

Function and anatomy of the visual system

Vision relays feedback regarding the external environment, providing context and spatial reference. From this, potential threats to stability, options for balance strategies (e.g. reaching or grasping), and constraints to balance control (e.g. size and shape of a support surface) can be determined (Patla, 1997). In addition, motion of the head, such as when a person sways or is moving through an environment can be determined from visual inputs (Bronstein, 2016; Mackinnon, 2018; Patla, 1997). Consequently, visual inputs contribute critically to many aspects of balance control, both proactively in preventing a disturbance, and reactively in detecting and correcting disturbances once they occur (Patla, 1997).

Visual information is processed by both cortical and subcortical pathways. In the cortical pathway, visual input is received first by the primary visual cortex (V1) then further processed via two streams. The ventral stream is associated with object identification and may play a role in cognitive processes related to balance control, such as identifying potential threats and solutions. The dorsal stream is associated with processing spatial, form, and motion information and is considered to be critical in the visual system's role in maintaining and correcting balance. The dorsal stream projects to the superior and inferior parietal cortex (Mackinnon, 2018). The superior parietal cortex is involved in object motion and self-motion recognition and rapid limb correction when avoiding obstacles. This information is relayed to the dorsal pre-motor cortex (selection and planning of goal-directed movements), then the primary motor cortex (movement execution), and finally to spinal motor networks, via the corticospinal tract, to execute motor responses (Mackinnon, 2018; Nakayama et al., 2022; Reader & Holmes, 2018; Yip & Lui, 2022). The inferior parietal region is involved in spatial recognition of stationary and moving objects in the environment. From the inferior parietal region there are projections to the ventral pre-motor cortex which is involved in target directed grasping during change-in-support strategies (Mackinnon, 2018; Maki & McIlroy, 1997; Reader & Holmes, 2018). These cortical pathways are involved in processing visual information to identify relevant cues in the environment and to assist in executing appropriate motor behaviour.

The subcortical pathways, such as the reticulospinal tract and vestibulospinal tract, receive input directly from the retina and are hypothesized to be involved in more rapid postural responses to visual disturbances (Mackinnon, 2018). Retinal input to the superior colliculus, which contributes to neck and eye movements that orient the head towards or away from stimuli (Zubricky & Das, 2024), is relayed to the pontine reticular formation where it then projects (via the reticulospinal tract) predominantly to the axial and proximal muscles for muscle tone regulation and suppression of different muscles (Mackinnon, 2018; Rothwell, 2004). The pontine nuclei also project to the cerebellum, which receives proprioceptive input, and then to the vestibular nuclei. The efferents of the vestibular nuclei project to spinal motor neurons, via the vestibulospinal tract, and ultimately axial and proximal muscles to control postural equilibrium by mediating the level of postural tone (Mackinnon, 2018; Rothwell, 2004; Takakusaki, 2017). Therefore, subcortical pathways, such as the reticulospinal tract and vestibulospinal tract, are hypothesized to play a role in mediation of rapid postural responses that have a faster latency than voluntary responses (Mackinnon, 2018). The processing of visual information in the intricate connections between the retina and both cortical and subcortical pathways, and the effects of these pathways, highlight the critical role of vision in postural control.

Contribution of visual feedback in postural sway regulation

It is well established that vision plays a crucial role in balance control and stability (Robert et al., 2016). Seminal work by Edwards (1946) demonstrated that the natural sway of the body increases when an individual stands with their eyes closed. The increase in sway with the absence of visual input is due to the decreased input from a single sensory modality (Lishman & Lee, 1973; Oie et al., 2002). The contribution of the visual system to postural sway is further highlighted in individuals with congenital or acquired visual deficits, such as low visual acuity, who experience increased postural sway during both static and dynamic balance tasks, compared to participants with normal vision (Tomomitsu et al., 2013). Further, Rogge et al. (2021) showed increased postural sway in adolescents who were blind or visually impaired when compared with blindfolded adolescents with unimpaired vision. It is logical to assume that adolescents with visual impairments may have decreased sway as they are accustomed to the lack of visual feedback and thus other sensory systems have compensated for the loss of vision. However, as this was not the case, an explanation for the increase postural sway in blind adolescents (Rogge et al., 2021) is the exploratory role of postural sway (Carpenter et al., 2010). Carpenter et al. (2010) suggested that increased postural sway is a mechanism employed to increase the sensory feedback relayed to the nervous system. Therefore, during mechanically challenging tasks, the sway is increased as an exploratory mechanism to increase sensory feedback in order to compensate for limited input from another sensory source, i.e. vision (Carpenter et al., 2010). Nonetheless, these findings demonstrate that absent or impaired vision leads to increased sway during standing and highlights that visual feedback contributes to postural sway regulation.

Having a stable visual reference improves the ability of maintaining upright balance. Focusing on a nearby visual target while standing can reduce sway, compared to standing without a visual target (Simeonov et al., 2009). Proper visual clarity of the stable visual cues within the surrounding environment can allow for postural stability. However, reduction of this visual clarity, such as dim lighting, suggests that individuals with poor postural stability might be more impacted by reduced visual acuity (Brooke-Wavell et al., 2002). Moreover, having a stable visual reference can mitigate the increase of postural sway that occurs during mechanically challenging standing conditions (Simeonov & Hsiao, 2001; Tomomitsu et al., 2013). Simeonov & Hsiao (2001) tested the effect of standing at high elevation while the participant's visual field was limited to only seeing distant visual references. As expected, standing at a height on its own was challenging, but limiting the visual field further increased postural sway (Simeonov & Hsiao, 2001). Sway is stabilized when people are provided with stable visual anchors in the visual field, therefore it is clear visual feedback is involved in regulating postural sway.

Movement of the body, in relation to the surround, or the motion of objects in the environment in relation to the body can create what is called motion parallax (Bronstein, 2016; Kim et al., 2016). Situations such as an individual standing on a stationary train, while the train adjacent to it pulls forward can create the illusion that the original train is moving, due to motion parallax. Motion parallax provides feedback on the relative movement of the head within the environment and therefore is related to postural sway (Bronstein & Guerraz, 1999). The importance of motion parallax to balance regulation was highlighted by the work of Lee & Lishman (1975), who demonstrated that postural sway was entrained to the subliminal motion of a swinging room within which participants stood. Studies inducing an entrainment of postural sway to the movement of an oscillating room have demonstrated that the frequency of the postural sway was at similar frequencies as the motion of the room, with lower frequencies of oscillation being more closely coupled with sway (Lee & Lishman, 1975; Schmuckler, 2017).

Similar behaviour of induced postural sway in the direction of a visual stimulus has been repeated in virtual reality (VR) environments (Luo et al., 2018). Moreover, Lishman & Lee (1973) demonstrated that the perception of movement created by standing on a moving cart was negated when the visual surround moved in concert with the cart, thereby eliminating motion parallax. Lishman & Lee (1973) argue that the potent influence of motion parallax on the perception of self-motion indicates that visual feedback is a kinesthetic sense, important for detecting the motion of the body during balance tasks. Nashner and Berthoz (1978) corroborated the findings of Lishman and Lee when participants received balance disturbances with the absence of visual feedback resulting in an impaired balance stabilizing strategy (Nashner & Berthoz, 1978). Motion parallax, the relative motion between the head and environment, can be an indicator of the movement of the body and COM, and can entrain postural sway indicating that visual feedback is involved in postural regulation.

The visual system is a potent sensory source that has been demonstrated to influence postural sway. This is seen when the lack of visual input while standing is destabilizing, while addition of a stable visual reference can increase stability, and manipulations of the visual surround are potent enough to alter the sway pattern altogether (Brooke-Wavell et al., 2002; Edwards, 1946; Lee & Lishman, 1975; Lishman & Lee, 1973; Rogge et al., 2021; Simeonov et al., 2009; Simeonov & Hsiao, 2001; Tomomitsu et al., 2013). It is clear visual input, whether stable or oscillating, is critically involved in regulating postural sway in standing balance.

The role of the visual system in postural responses

Sustained, unidirectional visual input can induce a postural response, such as a lean in the direction of the visual stimulus (Bronstein & Buckwell, 1997; Guerraz et al., 2001; Lee & Lishman, 1975; Lestienne et al., 1977; L. Nashner & Berthoz, 1978; Wolsley et al., 1996). Lestienne et al., (1977) induced postural leans with the use of a persistent high velocity image. Thus, false sustained motion parallax input was sufficient to influence posture, suggesting that the processes involved in sustained responses, such as a lean, are similar to responses demonstrated in the studies with subliminal visual oscillations. Wolsley et al. (1996) were able to induce persistent leans in individuals viewing a rotating disc. After the stimulus onset, the individuals had a lateral lean until the disc rotation was terminated, and participants reset to their original positions. When participants were viewing the same stimulus but with their body deviated 90°, and their head turned such that the line of sight was deviated 30° from the center of the disc, a similar outcome was observed however the movement was in the anterior direction (Wolsley et al., 1996). Wolsley and colleagues concluded that postural muscles are influenced by motion parallax, including detection of visual cues (Wolsley et al., 1996). Guerraz et al. (2001) observed a similar outcome with the use of rotating discs where they observed postural readjustments in the medial-lateral axis in both neurologically intact participants and participants with visual vertigo. In each of the above studies, participants leaned as a response to a false persistent visual stimulus, but they did not fall. This strongly suggests that despite the persistent erroneous signal from the visual system, the balance control system prevents the body from leaning past the point that would induce a fall and presumably relies on the non-visual sensory systems. It is clear that postural leans are created with persistent motion parallax, indicating that visual feedback is potentially involved in balance control.

Postural reactions have been evoked using large, transient visual stimuli. With the use of a moving room, Bronstein (1986) delivered large unidirectional visual displacements that were 30 cm in length and 12 s in duration. Following these large, transient stimuli, participants responded with postural corrections in the direction of the motion of the visual stimulus (Bronstein, 1986). Bronstein (1986) termed these responses visually evoked postural responses, or VEPRs. With repeated displacements of the visual stimulus, under the same parameters, the VEPRs attenuated as early as the second exposure to the stimulus (Bronstein, 1986). More recently, studies utilizing virtual reality have been able to replicate the findings seen in real environments (Phillips et al., 2022). Postural reactions are evoked following episodic, transient motion parallax stimuli, however these responses habituate with repeated trials, demonstrating that the reliance on visual feedback, while standing, is modifiable.

It is clear from the potent effects of visual feedback on postural control, sway regulation and postural responses seen in the literature, that the visual system has a critical role in balance control. Given the head is in an elevated position relative to the ground, and motion parallax not only indicates the motion of the head during movement, but can also regulate sway and posture, it is reasonable to speculate that visual feedback is critical in maintaining posture to avoid falls. My thesis will extend the literature by utilizing unexpected small, rapid stimuli, consistent with a postural disturbance, such as a push to the body, to better understand if visual feedback potentially plays a role in rapid balance corrections to prevent falls, as opposed to the regulation of sway and posture confirmed by the existing literature.

The role of the vestibular system in the control of balance

Upright stance is prone to the effect of gravitational forces, with gravity being a frame of reference for the body's orientation in space (Jamon, 2014). The vestibular apparatus, or labyrinth, within the inner ear, provides continuous feedback regarding the movement and orientation of the head in relation to gravity. The vestibular labyrinth includes the otolith organs, which detect linear acceleration, and the semicircular canals, which detect angular acceleration. Through the integration of changes in these accelerations, the vestibular system is able to encode whether the head is stationary, accelerating or moving at a constant velocity, while also encoding the orientation of the head, relative to gravity (Day & Fitzpatrick, 2005; Sidebotham, 1988). The changes in acceleration are detected when fluid within the otoliths and semicircular canals cause hair cells to bend with the direction of flow. The resulting action potentials are delivered by primary afferents to the vestibular nuclei located within the medulla oblongata, before also projecting to the cerebellum and brainstem motor nuclei of the extraocular muscles. Outputs from the vestibular nuclei also descend to the spinal cord, via the vestibulospinal tract, projecting to motor nuclei of the antigravity muscles (O'Reilly et al., 2011). The main functions of the vestibular system are 1) stabilization of gaze during head movements, 2) stabilizing the head during movements, 3) providing the sense of self-motion, and 4) activating and modulating postural reflexes that help with maintaining balance and upright stance (Mackinnon, 2018).

The importance of the vestibular system in supporting balance control is evident in individuals with vestibular impairments (Majeed et al., 2023). As early as 1958, Carpenter et al. (1958) reported that bilateral lesions to the vestibular labyrinth in cats resulted in disruption to equilibrium and coordination of the body, as highlighted by staggering and falling. Similar impairments are observed in humans suffering damage to the vestibular apparatus which can present as instability during walking or standing, and an increased risk of falling (Majeed et al., 2023; Strupp et al., 2016). Experimentally, the feedback from the vestibular apparatus can be temporarily manipulated through techniques such as galvanic vestibular stimulation (GVS). For example, Woll et al. (2019) demonstrated that GVS stimulation above perceptual threshold resulted in a degradation of postural control, while imperceptible GVS stimulation resulted in increased postural stability, emphasizing the interaction of vestibular afferent inputs with generalized balance control (Woll et al., 2019). Furthermore, altering the activity of vestibular afferents through GVS results in a whole body lean towards one side, with related changes in muscle activity apparent as early as 40 ms in the arms or 55-65 ms in the legs (Fitzpatrick & Day, 2004). The speed with which the GVS activation of the vestibulospinal system induces a postural response suggests that the vestibular system is sufficiently fast to stabilize posture to unexpected balance disturbances and prevent a fall from occurring (MacKinnon 2018). It is clear that the vestibular system is involved in postural control, as demonstrated by studies involving patients with vestibular impairments and use of GVS application

Experimental studies using lesioned animals highlight the specific contribution of vestibular feedback during automatic postural responses following balance disturbances. Macpherson & Inglis (1993) observed that, following labyrinthectomy in cats, only moderate changes were seen to quiet standing, and overall, the latency and pattern of postural responses to translational perturbations to the support surfaces remained unchanged. However, there was hypermetria of the evoked postural response, in the 8–10 day range post-lesion, as highlighted by the over-response in the amplitude of the postural reaction. Thus, Macpherson and Inglis concluded that the vestibular input is likely not required for the initiation of postural responses and is involved in the scaling of the responses instead (Macpherson & Inglis, 1993). More specifically, Inglis & Macpherson (1995) further concluded that vestibular feedback is only involved in the scaling of evoked postural responses and does not contribute to the shaping or selection of the responses. More recently, Murray et al. (2018) demonstrated that ablation of the lateral vestibular nuclei (LVN) impaired balance corrective responses to a lateral translation of the support surface in standing rats due to significantly reduced amplitude of extensor muscle activation. This finding suggests that vestibular inputs were not likely responsible for initiating the balance corrective responses as the rats did not fall from the support beam and a balance corrective response was still observed. Rather, this result indicates the vestibular system contributes to the scaling of the response magnitude. Regardless, it is clear that the loss of LVN inputs had a profound impact on the corrective reactions of the animals that was not evident during open-field locomotion or undisturbed postural measures. Together these studies suggest that other sensory systems, such as the somatosensory system, likely triggers automatic postural responses to disturbances that the vestibular system scales relative to the context of the disturbance (Inglis & MacPherson, 1995; Macpherson & Inglis, 1993; Murray et al., 2018).

In humans, impairments to the vestibular system do not seem to affect initiation of postural responses and the ability to maintain stability, but rather influence the scaling of these responses. Nashner et al. (1982) demonstrated that, under normal sensory conditions, patients with vestibular impairments were able to stabilize their posture effectively during translation of the support surface, similar to control participants. These findings suggest that the vestibular system is not directly involved in triggering the responses required to arrest postural sway (Nashner et al., 1982). Runge et al. (1998) later demonstrated similar findings when patients with bilateral vestibular loss (BVL) were able to initiate a hip strategy to maintain balance following backward support surface perturbations. Although impairments to the vestibular system affect control of posture, it is not directly responsible for evoking a postural response (Nashner et al., 1982; Runge et al., 1998). This was further suggested by Horak et al. (1990) when they observed patients with vestibular impairments maintaining stability in normal standing environments but responding ineffectively on altered support surfaces. These findings led to the interpretation that the vestibular loss affects the advance selection of appropriate postural strategies, but not the initiation of postural responses during disturbances (Horak et al., 1990). However, as the previously mentioned animal studies suggest, the vestibular system does affect the amplitude of postural responses. Allum et al. (1994) delivered rotational and translational support surface perturbations and observed that the onset latencies of the muscle responses were similar in patients with bilateral peripheral vestibular deficit compared to control participants. However, the amplitude of the responses evoked in the leg muscles were altered in the patient group, thus demonstrating that the vestibular system is likely involved in scaling muscle responses in humans (Allum et al., 1994; Allum & Honeggar, 2019). Additionally, BVL patients receiving tilt perturbations to the support surface, had normal response onset, but the level of muscle activation in these responses were affected, further suggesting that the vestibular system influences the amplitude of the evoked responses (Carpenter et al., 2001). These studies together highlight the role of the vestibular system in scaling postural reaction as seen by the change in response amplitudes with vestibular impairments.

It is clear the vestibular system is important for balance and provides feedback on motion and orientation of the head. Vestibular input is critical in postural control as highlighted by changes to posture, such as lean, during impairments to this system. The current literature indicates that the vestibular system does not play a role in initiating postural responses, as is highlighted by the ability of human and animal subjects to maintain balance during support surface perturbations (Allum et al. 1994; Allum & Honegger, 2019; Carpenter et al., 2001; Inglis & MacPherson, 1995; Macpherson & Inglis, 1993; Mbongo et al., 2009; Nashner et al., 1982; Runge et al., 1998). However, the vestibular system likely assists postural control by scaling the postural reactions that are triggered by other sensory sources. Therefore, it is likely that somatosensory feedback triggers and shapes these responses, while the vestibular system is involved in scaling the postural responses.

Receptors and functions of the somatosensory system

The somatosensory system detects proprioceptive, touch, nociceptive, and thermal stimuli from receptors located in the skin, muscles, and joints. (Watson, 2012). Proprioceptive receptors detect the position of the body and limbs in space. Cutaneous mechanoreceptors detect various forms of mechanical deformations of the skin related to pressure, stretch, and vibration. Nociceptors and thermoreceptors are nerve endings found in the skin, skeletal muscles, joints and in the viscera that detect pain and temperature respectively (Purves, 2004; Watson, 2012).Together the sensory receptors of the somatosensory system relay information regarding the internal and external environments of the body. The following subsections of the literature review will focus on proprioceptors and cutaneous receptors which have direct effects on balance control.

Muscle and joint mechanoreceptors in balance control

Proprioception is the unconscious processing of sensory input regarding the location of the body and limbs (Mackinnon, 2018). Proprioceptive input to the somatosensory system is detected by three main types of receptors: 1) muscle spindles, 2) Golgi tendon organs, and 3) joint receptors (Mackinnon, 2018; Stein, 2017; Watson, 2012). Muscle spindles are located within skeletal muscles and are aligned parallel with the muscle fibres. This arrangement allows the spindles to detect changes in length of the muscle fibres as well as the rate of change (Mackinnon, 2018; Stein, 2017). Golgi tendon organs (GTOs) are located in series with the muscles within the tendon, which sense muscle tension and the forces exerted on a tendon, including detection of the force of gravity acting on the body. Joint receptors, located within joint capsules, respond to changes in pressure of the synovial fluid and are highly activated during the extremes of joint range of motion (Mackinnon, 2018). Together these receptors provide constant unconscious relay of information pertaining to the orientation and position of the limbs relative to the body.

Sensory input that is detected by muscle proprioceptors are relayed to various levels of the central nervous system that inform the control of balance. Following a balance disturbance, the movement of a limb stretches muscle spindles. The resulting action potentials are delivered to the spinal cord via fast conducting group Ia and II afferents. This input can monosynaptically activate motor neurons within the spinal cord and initiate a rapid stretch reflex of the originating muscle, or via one or more interneurons, activate motor neurons distributed throughout the spinal cord to initiate complex intermuscular, interlimb and intersegmental responses (Mackinnon 2018). These short latency spinal reflex circuits can be involved in rapid postural reactions that aim to counteract the balance disturbance and maintain stability (Mackinnon 2018). Additionally, ascending input from the muscle and joint mechanoreceptors also travels to cortical and subcortical regions, such as the cerebellum, somatosensory cortices, and brainstem regions (Mackinnon, 2018). The control of balance often is seen as subconscious as we are partially aware of the mechanisms employed to maintain upright stance, however, both cortical and subcortical processes are critical in maintaining upright stance (Forbes et al., 2018). Subcortical processes at the brainstem level are said to be the most dominant in the control of balance while it is suggested that the cortex finely tunes the motor outputs during balance control mechanisms (Boebinger et al., 2024; Forbes et al., 2018). Together, the sensory input relayed from the muscle and joint mechanoreceptors inform the spinal, subcortical, and cortical processes that are involved the control of balance and posture, including balance reactions and sway regulation (Carpenter et al., 2010; Mackinnon, 2018).

Given that the control of balance requires sensory input that detects the position and motion of the body COM to generate corrective forces to maintain stability, it is logical to suggest that the input from proprioceptors is critical in balance control (Rasman et al., 2018). The sense of limb and head position relative to the body, which can be relayed by muscle spindles, GTOs and joint receptors, is critical for the control of movement with respect to gravity to maintain stability (Horak, 2006; Huxham et al., 2001). Fukuoka et al. (2001) demonstrated that when using an ankle strategy, somatosensory feedback alone was sufficient to maintain an upright stance. Further, reduction of afferent input from the lower limbs, via ischemic block, increased postural sway (Mauritz & Dietz, 1980). Mauritz & Dietz (1980) suggested this increased instability is due to unreliable proprioceptive input. Populations with impaired proprioceptive sense, such as patients with diabetic neuropathy or anterior cerebellar lobe atrophy, have decreased stability, as highlighted by a prominent anterior-posterior sway (Diener, Dichgans, Bacher, et al., 1984; Simoneau et al., 1995), thus demonstrating the critical role of proprioception in postural regulation. It is clear that proprioceptive feedback is involved in regulating postural sway and provides adequate input for stabilizing upright stance (Rasman et al., 2018).

Proprioceptive information from muscles and joints relays the necessary sensory afference that is required for orienting the COM within BOS to maintain an upright stance and is likely involved in postural responses to perturbations (Riemann & Lephart, 2002; Wallmann, 2009). For example, during an external perturbation such as a push, the detection of the COM being displaced can be relayed by muscle spindles in the muscles stretched by the imposed movement (Wallmann, 2009). Consequently, this stretching response produces action potentials that relay input to trigger a postural response to maintain the COM within the BOS. It has been demonstrated that the immediate stretch of leg muscles, such as gastrocnemius, via toes-up rotation of the ankle joint, can evoke a stretch reflex response within 120 ms, in order to maintain the COM within the BOS (Nashner, 1976, 1977). Moreover, during support surface perturbations, the proprioceptive input from the stretch of ankle joint muscles is capable of evoking automatic postural responses in individuals (Horak et al. 1990; Nashner, 1982). Inglis et al. (1994) suggests that somatosensory feedback from the lower leg during support surface translation is involved in triggering short-latency balance reactions, as highlighted by the delayed response onset in patients with diabetic peripheral neuropathy (Inglis et al., 1994; Van Deursen & Simoneau, 1999). Inglis et al. (1994) also indicate that somatosensory cues are involved in scaling these responses as well. The persistence of automatic postural responses with impaired vestibular feedback, further supports that the somatosensory system, specifically proprioceptors in the ankle, contribute to triggering balance reactions in response to external disturbances

(Allum et al., 1994; Macpherson & Inglis, 1993; Murray et al., 2018; Runge et al., 1998). It is clear that while balance control is influenced by the integration of information from all the sensory systems, the proprioceptive feedback from muscle spindles is critical in initiating rapid balance corrective responses and regulating postural sway to maintain upright stability.

Cutaneous input in balance control

Balance is the ability of a person to maintain a static position of the body in space which keeps the COM within the BOS (Carini et al., 2017; Pollock et al., 2016; Winter, 1995). During bipedal stance, the feet make up the BOS while the interface between the soles of the feet and the ground provides a rich source of cutaneous input regarding the BOS (Inglis et al., 2002). Cutaneous mechanoreceptors in the glabrous skin respond to a range of highly sensitive stimuli. The glabrous skin contains four main types of mechanoreceptors that respond to mechanical stimuli, 1) Merkel discs, 2) Meissner corpuscles, 3) Pacinian corpuscles, and 4) Ruffini endings (Mackinnon, 2018; Watson, 2012). Balance can be compromised when the feedback from the interface between the feet and external environments is altered via cooling or anaesthesia (Felicetti et al., 2021; Strzalkowski et al., 2018). Moreover, there is the potential for cutaneous receptor feedback associated with the stretch of skin across the respective joints to contribute to proprioception (Collins et al., 2000). Recently, with the use of an artificial foot, skin stretch at the ankle joint has been shown to relay proprioceptive information of joint position (Kent, 2023), which would imply a functional role to balance control. It is clear that cutaneous input from the sole of the foot and skin across the joints can provide sensory information that informs balance control.

During bipedal stance, the BOS is not limited to the feet and can include the hands as well. More specifically, grasping of a cane or handrail to provide additional stability changes the BOS. The interface between the hands and objects that are used to maintain stability provides yet another interface where potent cutaneous information can originate. Cutaneous input from the hand, even without the mechanical stability it provides, can help stabilize balance. It is well documented that light touch of a single fingertip with a force of less than 1N, which is not mechanically stabilizing, can reduce postural sway (Holden et al., 1994; Jeka, 1997; Jeka & Lackner, 1994). The provision of a stable light touch reference is known to reduce the increased sway that occurs when a person stands with the eyes closed, demonstrating that tactile feedback alone can provide stability (Holden et al., 1994). Sensory feedback from a non-rigid source, such as a piece of fabric, was demonstrated to be stabilizing (Riley et al., 1999), while passive sensory feedback from other skin surfaces, such as the shoulder or leg, were also stabilizing to posture (Rogers et al., 2001). Application of stochastic noise, to enhance tactile feedback, further reduced sway (Kimura et al., 2012), and an ischemic block of the distal arm to block cutaneous feedback eliminated the touch-induced sway reduction (Kouzaki & Masani, 2008). Taken together these findings demonstrate the stabilizing effects of cutaneous feedback in balance control. Holden et al. (1994) demonstrated a potent interaction between tactile input and vision in postural regulation. Therefore, my thesis includes the addition of a stable light touch to evaluate if the provision of supplemental cutaneous feedback affects the expression of visually evoked postural responses that I hypothesize follow an unexpected small, transient perturbation of the visual surround.

CHAPTER 3

Methodology

Participants

Forty naïve participants (ages 18-25; 26 females, 14 males) participated in this study. All participants reported no history of neurological or musculoskeletal injuries, back pain, or concussions in the last 6 months. Participants consented to the present study which was approved by the University of Alberta Human Research Ethics Board (Pro00086777) and in accordance with the Declaration of Helsinki. Participants were recruited from the student population at the University of Alberta and members of the local community.

Set-up and apparatus

Participants stood barefoot on an ethylene-vinyl acetate foam pad (5 cm thick) with their feet shoulder-width apart, or what they deemed to be comfortable, atop a 6-component force plate (AMTI OR6-7-OP-2K-STT; A-Tech instruments LTD., Watertown, MA, USA) that was flush with the floor (Figure 1). An electrogoniometer (SG100A, Biometrics LTD, Newport, UK) was placed across the right ankle to record joint angle. Electromyographic (EMG) activity was recorded from the right tibialis anterior (TA), soleus (SOL), sternocleidomastoid (SCM), and orbicularis oculi (OO) using pairs of Ag/AgCl surface electrodes (NeuroPlus A10040, Vermed, Bellows Falls, VT, USA) positioned approximately 2 cm apart on the skin over the center of the belly of the muscle, oriented in the predicted direction of the muscle fibres. A ground electrode was placed on the skin over the olecranon process. All electrode sites were shaved and cleaned with alcohol to ensure impedance was below 20 kΩ (Grass F-EZM5, Astro-Med, Inc., West Warwick, Rhode Island, USA). Participants listened to white noise through a pair of over the ear headphones (Beats Solo by Dr. Dre, Beats Electronics LLC, Culver City, CA, USA) during all experimental testing to mask any background noises and the sound of the stepper motor used to deliver the visual displacements (see below). EMG signals were amplified and band-pass filtered (10 Hz - 1 kHz, 60 Hz notch filter, Grass P511 amplifiers, AstroMed Inc, West Warwick, RI, USA). All signals were digitized using a 12-bit data acquisition card (PCI-MI0-16E-4, National Instruments, Austin, TX, USA) with a sampling rate of 2000Hz and then stored on a hard drive using custom-written LabView programs (LabView Version 18, 64-bit, National Instruments).

Visual stimuli were introduced by the unexpected displacement of an enclosure around the participant. The enclosure (Figure 1) consisted of 3 walls (leaving the back of the enclosure open) and a ceiling, constructed from corrugated cardboard secured to a frame made from 80/20 T-slot extruded aluminum. The sides were approximately 0.5m from the participant's shoulders and extended 0.5m behind the participant. The distance from the front wall to the participant's chest was approximately 1.5m. The ceiling was approximately 0.5m above the participant's head. A shelf located within the enclosure was included to prevent the participant from seeing the floor. The shelf was located above the height of the participant's elbow and extended from the front of the enclosure to approximately 15 cm in front of the participant's torso. The structure of the enclosure was not adjustable and therefore the specific distances relative to the participant varied with the size of the participant. Small LED lights were placed inside the enclosure to reduce darkness and provide visual contrast. The enclosure was secured to a square-rail acme screw drive positioning stage (LinTech 130 Series; LinTech Monrovia, CA, USA), which allowed the entire apparatus to be displaced in the anterior-posterior direction. Movement of the enclosure was delivered by a stepper motor (Applied Motion Products Inc., Morgan Hill, CA, USA) powered by a PhidgetStepper (Phidgets, Inc, Calgary, AB, Canada) controller using a custom-written LabView program. The anterior-posterior position of the apparatus was measured using a linear displacement sensor (Penny & Giles SLS130; Curtiss-Wright Corporation Davidson, NC, USA).

Some participants were asked to touch a stable reference during testing (see Protocol). The touch reference was located beneath the shelf that obscured the floor, thereby obscuring the touch reference as well. The touch reference consisted of a 3D-printed cylinder (3cm x 6cm) with a small, raised bump at the centre of the surface to provide a distinct point of reference. The height of the touch reference was adjusted so that the elbow was flexed at 90°, with the forearm parallel to the floor (Figure 1). The touch reference sat atop a second force plate (AMTI OR6-7-OP-2K-STT) to allow the measurement of vertical force applied to the touch reference. The vertical force applied by the participant during testing was monitored online by an experimenter who provided verbal cueing to the participant if the force exceeded 1 N.

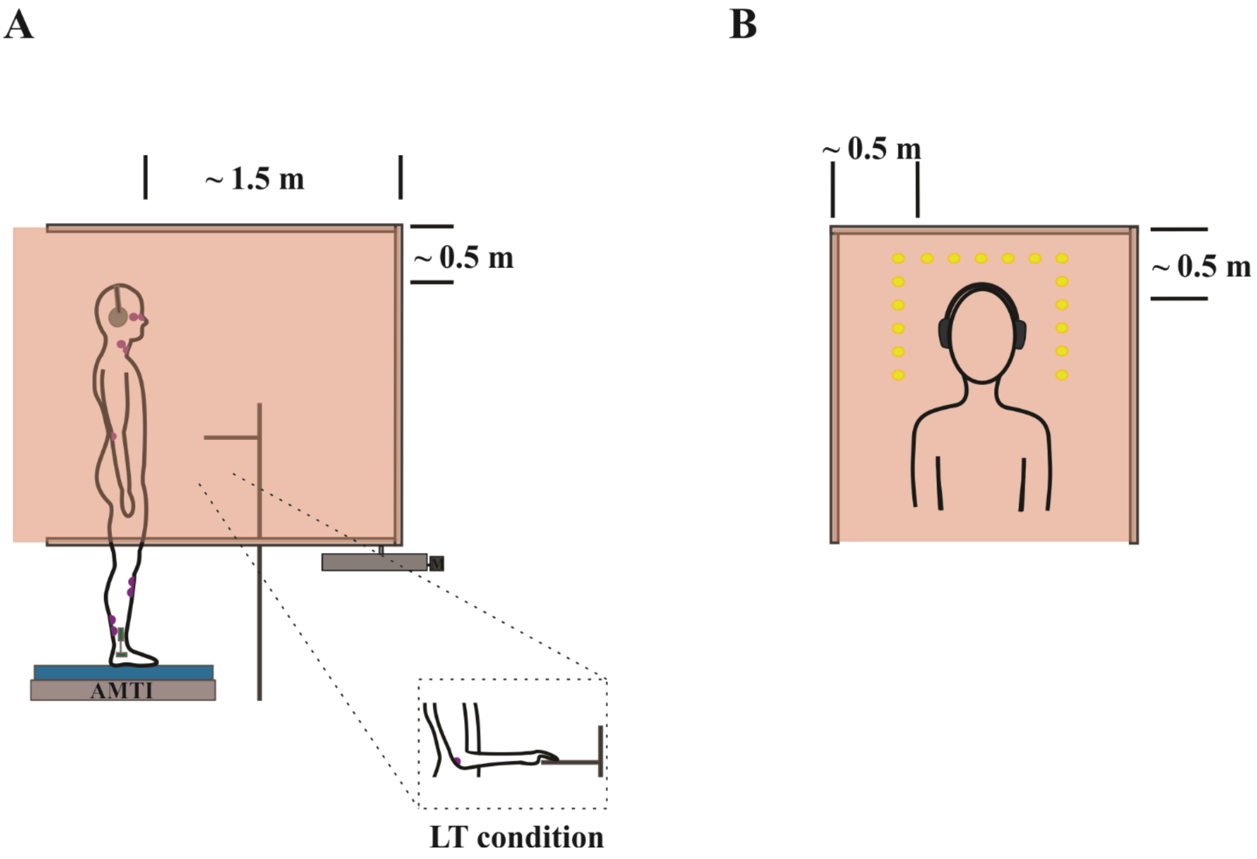

Figure 1 – Schematic representation of experimental set-up and apparatus
Participants stood on a foam pad (in blue) atop a 6-axis force plate (AMTI) while listening to white-noise through a pair of headphones. An electrogoniometer (in green) was placed on the right ankle joint, and pairs of surface EMG electrodes were placed on the belly of 4 different muscles 1) tibialis anterior (TA), 2) soleus (SOL), 3) sternocleidomastoid (SCM) and 4) orbicularis oculi (OO). **(A)** Shows a side-view of the set-up of the 'no-touch' conditions where participants have both arms relaxed at their sides. **(B)** Shows a side-view of the set-up of participants in the 'light touch' conditions, where the left arm is relaxed at the side while the participants right arm is flexed such that the elbow is bent at 90° and the pad of the right index finger is in light contact with a touch reference. The touch reference protrudes from a vertical structure that is in front of the participant. **(C)** Schematic of the posterior-view of the experimental set-up.

Protocol

Participants were pseudo-randomly allocated to one of four groups based on the direction of the displacement of the visual surround (forward, F; backward, B) and the presence of a stable touch reference (no touch, N; touch, T). Note that a forward displacement of the visual surround indicates the front wall moved away from the participant. Participants in the FN and BN groups completed 3 standing conditions: 1) standing within the stationary enclosure; 2) standing within the enclosure that was unexpectedly displaced; and 3) standing within the stationary enclosure. Participants in the FN and BN groups were asked to stand with their hands comfortably by their side during each of the condition. Participants in the FT and BT groups completed 4 standing conditions: 1) standing within the stationary enclosure; 2) standing within the stationary enclosure while touching a stable reference; 3) standing within the enclosure that was unexpectedly displaced while touch a stable reference; and 4) standing within the stationary enclosure. During the conditions with touch, participants were asked to lightly touch (<1 N of vertical load) the stable reference with the pad of their second digit (index finger) of their right hand with the other fingers curled into the palm. The left hand was comfortably by their side. All participants were asked to look at a visual reference located on the front wall of the enclosure during all trials. Participants sat between each condition for a minimum of 2 min.

Data for each of the quiet standing conditions (i.e. the conditions without visual stimuli) was recorded for 90 s. The primary purpose of the quiet standing conditions was to deceive the participants into believing the enclosure was stationary. As such, the order of the conditions was not randomized. During the visual displacement conditions, participants stood quietly for 10 s to 15 s within the enclosure before the visual surround was first unexpectedly displaced 2.5 cm, with a peak velocity of 124 mm/s, for a total movement duration of 250 ms. Subsequently, the enclosure was slowly returned to the starting position at an average velocity of 0.64 mm/s, which was imperceptible. The next stimulus was delivered after the enclosure had returned to the starting position for at least 2 s, with a random interval of up to 15 s. Ten such stimuli were delivered, initiated manually by a researcher. The visual displacement conditions lasted up to 6 min to complete. Participants were not informed that the visual surround would move prior to testing.

Once all experimental conditions were completed, maximum voluntary contractions (MVCs) were obtained for each muscle. To do so, isometric contractions of the target muscles were performed against resistance, with verbal encouragement, and monitored online. The electrogoniometers were calibrated *in situ*. Following data collection, participants were debriefed on the true nature of the study, including the deception, and given the opportunity to withdraw their consent.

Data analysis

Post-hoc analysis of the mechanical data was performed using custom-written LabView programs. Evoked reactions to the visual stimuli in the mechanical data were analyzed from a 1200 ms segment of data extracted from the continuous data stream and low-pass filtered (20Hz, 2nd order zero-lag Butterworth filter). Center of pressure in the anterior-posterior direction (COP_{AP}) was calculated from the ground reaction forces and moments, recorded by the force plate, using formula (1)

$$COP_{AP} = \frac{M_x - \left(Z_{off} \times F_y\right)}{F_z} \tag{1}$$

The COP_{AP} and ankle goniometer traces were corrected for the ongoing, background sway by removing the slope of the trace estimated from a linear regression calculated for 100 ms centered at the time of the stimulus onset (corrected and uncorrected COP_{AP} is demonstrated in Figure 2). The first displacement of the visual surround typically resulted in a biphasic sway pattern, with an initial, smaller component in the direction opposite the visual displacement and a second, larger component in the direction of the visual displacement (Figure 2). Consequently, the amplitude of the first deflection (D1) was calculated by measuring the distance of the first peak, relative to the position at time zero, while the amplitude of the second deflection (D2) was calculated by measuring the distance between the first and second peaks. The D1 and D2 amplitudes were normalized to the root-mean-square of the COP_{AP} recorded over 60 s of the first quiet standing condition for each participant. Responses to the subsequent visual stimuli often lacked the distinct, overt deflections observed following the first stimulus. Therefore, D1 and D2 for the second through tenth stimuli were calculated using the same time points for the peaks identified from the first trial. This was done to utilize a standardized time to observe the changes in the responses across trials, relative to the first response. The same procedure was applied to the ankle goniometer data, but the ankle data were not normalized to quiet standing and recorded in degrees.

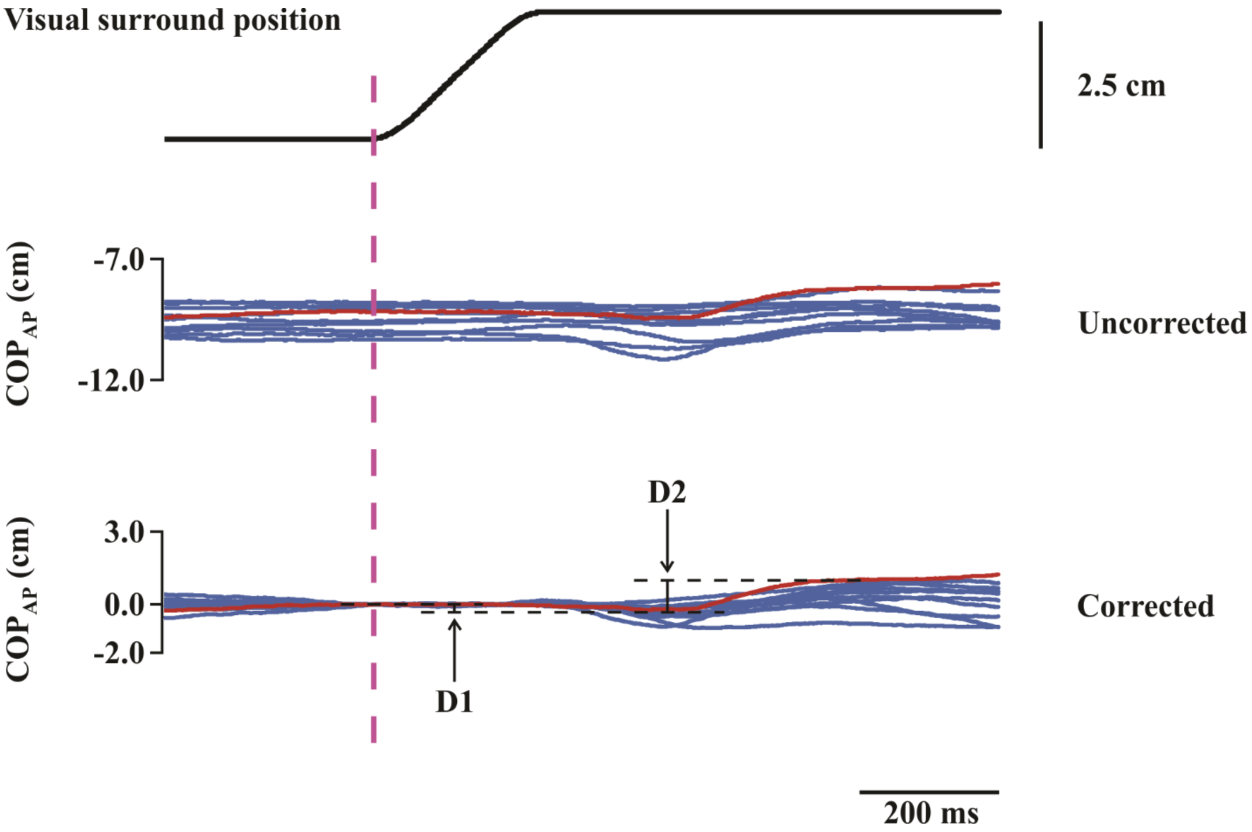

Figure 2 – Representation of centre of pressure analysis
Sample data traces from one participant that received a forward visual displacement. The top trace indicates the position of the visual surround. The middle traces depict the uncorrected anterior-posterior centre of pressure traces for each of the 10 trials, with the first trial depicted as the red trace. The bottom traces show the corrected COP_{AP} traces adjusting for the background sway and aligning the traces to the onset of the visual displacement (vertical dashed line). The amplitude calculations for D1 and D2 are indicated for the first trial.

Post-hoc analysis of the EMG data was performed using custom written MATLAB programs. Two approaches to analyzing the EMG data were attempted. In the first approach, the EMG trace extracted for each trial was full-wave rectified and then low-pass filtered (50 Hz, 4th order zero-lag Butterworth filter) to create a smoothed envelope (Figure 3A). A 95% confidence interval (CI) was calculated around the average background EMG activity occurring for the 300 ms prior to stimulus onset. A significant change in EMG activity was identified when the rectified and smoothed EMG trace exceeded the 95% CI for a minimum of 20 ms continuously, with an onset latency of at least 40 ms and no longer than 200 ms. No significant EMG responses were identified using this approach. However, visual inspection suggested that in some participants the ongoing tonic background activity in SOL was suppressed but had not reached significance because of a floor effect wherein the rectified EMG reached zero, but the lower limit of the 95% CI was less than zero. Consequently, a second approach, using wavelet analysis was attempted.

In the wavelet analysis approach, wavelets, small finite waves, of various frequencies were scaled along the time domain to decompose the raw continuous EMG data and stacked to create a 3D plot of a 1500 ms segment of data. The 3D plot (Figure 3B) had an x-axis of time, y-axis as frequency and the z-axis as power. This is visualized as a contour plot, or wavelet scalogram, with high and low values representing the power. The power of the individual peaks in the wavelet scalogram, were summed for each time point, resulting in a two-dimensional scale-averaged wavelet spectrum, with x-axis being time and the y-axis being cumulative power (red trace in Figure 3C). The scale-averaged wavelet was also inversed to create a secondary trace (blue trace in Figure 3C), to amplify the inhibitory periods of the wavelet scalogram. A 95% CI was calculated around the background activity in the scale-averaged wavelet spectrum, and its inverse, in the first 500 ms prior to stimulus onset (red and blue horizontal lines in Figure 3C). A significant change in EMG activity was identified when one of the scale-averaged wavelet traces (original or inversed) exceeded the 95% CI for a minimum of 20 ms continuously, with an onset latency of at least 40 ms and no longer than 200 ms. Whichever trace (red or blue) trace exceeded the 95% CI first, with the given criteria, was captured as a burst or silence in the EMG activity. If neither trace exceeded the confidence band with the specific criteria, then no burst or silence was identified for that trial.

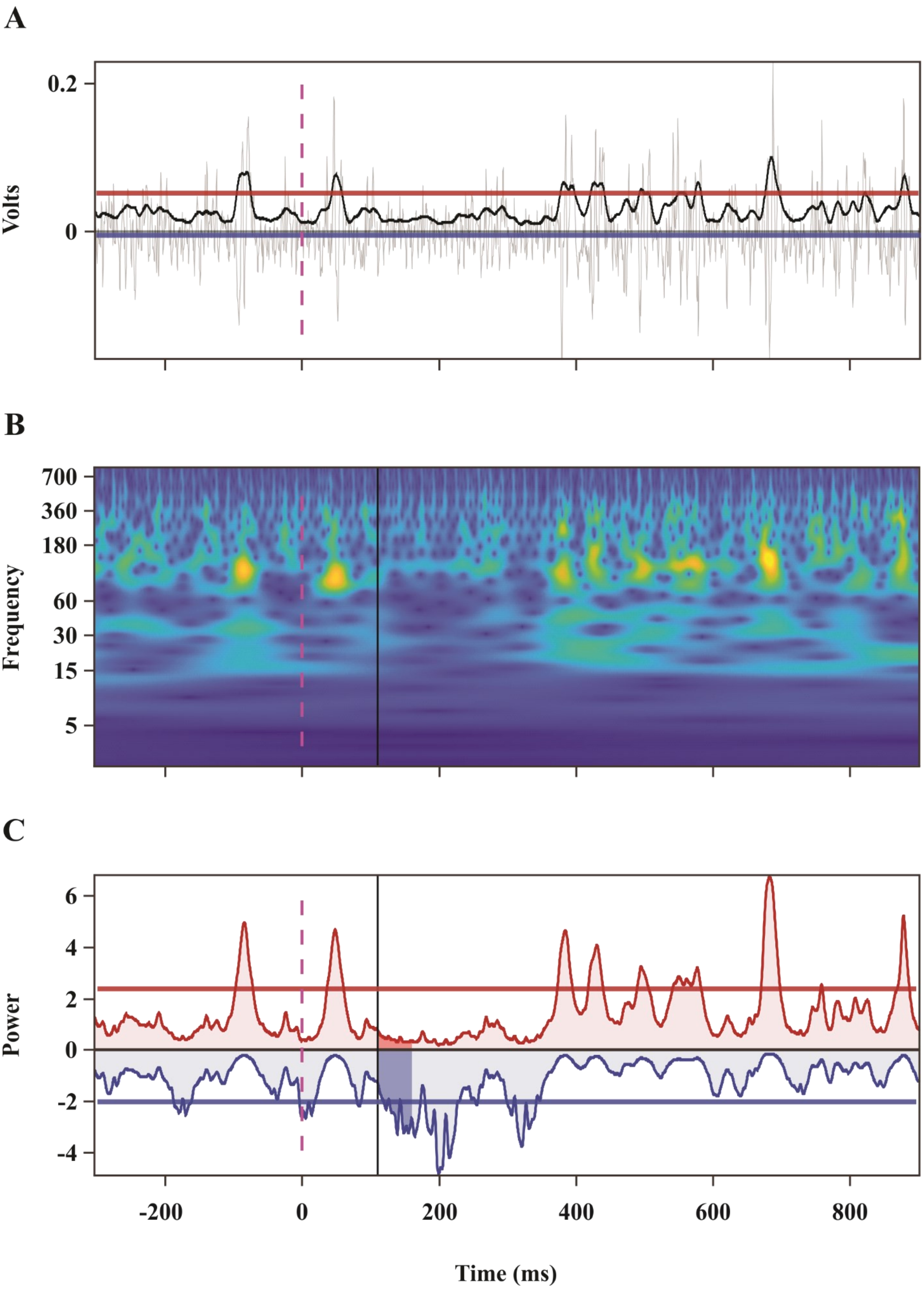
A
0.2
0
Volts
B
700
360
180
60
30
15
5
Frequency
C
6
4
2
0
-2
-4
Power
-200
0
200
400
600
800
Time (ms)

Figure 3 – EMG analysis approach

Sample electromyographic (EMG) activity from SOL of a single participant for a single trial of a forward visual displacement. (**A**) The raw EMG (thin grey grace) is overlaid by the full-wave rectified and filtered (50 Hz, 4th order zero-lag Butterworth) trace (black). The red and blue horizontal lines indicate the limits of the 95% confidence band constructed for the 300 ms pre-stimulus period. The vertical dashed line indicates the point of visual displacement onset. (**B**) The same data as in (A) are depicted as a 3-dimensional wavelet scalogram with the warm colours (yellow, orange, white) indicating higher power for that frequency. The cumulative sum of the powers for each time point is shown in (**C**), with the original (red) and inverted (blue) traces shown. The red and blue horizontal lines indicate the 95% confidence band. The vertical dashed line indicates the onset of the visual displacement, and the black vertical line indicates the onset of an identified inhibitory event.

Statistical analysis

The amplitude of the D1 and D2 deflections observed in the COP_{AP} and ankle data were analyzed separately for each direction of visual displacement. To evaluate the effect of repeated exposure to the visual stimulus (i.e. habituation), the amplitude of the outcome measure of the first trial was compared with the amplitude from the average of trials eight through ten, for each participant. Separate mixed two-way repeated measures analyses of variance (ANOVA) were performed for each outcome measure, to compare the effects of Touch (2 levels, T and NT) and Trial (2 levels, 1st trial and trials 8-10), where Touch is the independent factor and Trial is the repeated factor. Significant interaction terms were followed up with Tukey's Honestly Significant Difference (HSD) tests to identify differences of interest. Descriptive data are reported as means with standard deviations. Significance was set at $\alpha<0.05$. All statistical analysis was performed using the Real Statistics Resource Pack software (Release 8.9.1; Copyright (2013-2023) Charles Zaiontz).

CHAPTER 4

Results

Unexpected displacement of the visual surround evoked biphasic sway responses observed in the COP_{AP} and ankle angle traces (Figure 4 and 6) following the first displacement in a majority of the participants, with 39 of 40 participants exhibiting such a response. These biphasic responses were characterized by an initial, smaller deflection (D1) of the COP_{AP} in the direction opposite the visual displacement, with a second, larger deflection (D2) in the same direction as the visual displacement. The pattern observed in the COP_{AP} trace was closely mirrored in the pattern of motion observed in the ankle angle. These consistent observations in the COP_{AP} and ankle angle data were not reflected in the EMG data, with few examples where evoked changes in muscle activity in either TA or SOL were identified. Group average data are presented as: mean ± standard error.

Responses to forward visual perturbations

All 10 participants in the FN group and 9 out of 10 participants in the FT group showed a biphasic response in the COP_{AP} and ankle angle motion resulting in a forward sway. Figure 4 shows the data traces from one participant from the FN group, with the response from the first visual displacement (trial) depicted as the red traces. As can be seen, the first deviation in the COP_{AP} trace is a brief, backwards deflection, before the COP_{AP} then moves forward. This pattern of behaviour in the COP_{AP} is closely mirrored by an initial plantarflexion, with a subsequent dorsiflexion, at the ankle. The biphasic sway pattern in the COP_{AP} is less apparent following subsequent displacements of the visual surround (blue traces) in this participant. The EMG records for this participant are also depicted in Figure 4 and it is clear that the visual stimulus did not evoke distinct changes in muscle activity in any of the recorded muscles.

In Figure 5A and 5C, the group average amplitudes for the D1 and D2 deflections in the COP_{AP} are presented. The dark blue bars in Figure 5 depict the average amplitudes for the 10 participants in the FN group and demonstrate that the biphasic sway pattern described above for the individual participant's data was evident across participants. The initial deflection (D1) displayed a backward shift in the COP_{AP} (-86.2 ± 27.6 %RMS), while the second deflection (D2) displayed a more forwards position (144 ± 59.0 %RMS). It is also shown that the amplitudes of both D1 and D2 for trials 8 through 10 were generally smaller across participants, with average amplitudes of -39.5 ± 13.3 %RMS and 117.1 ± 21.8 %RMS, respectively. Also plotted are the group average data for the FT group in the light blue bars. Generally, the amplitudes of the D1 and D2 deflections were smaller when the participants were touching, for both the first trial and for the average of trials 8 through 10. The average amplitude of D1 was -36.5 ± 18.7 %RMS and -6.8 ± 5.6 %RMS for the first trial and trial 8 through 10, respectively. The average amplitude of D2 was 67.7 ± 26.3 %RMS and 52.9 ± 15.9 %RMS, for the first trial and for the average of trials 8 through 10, respectively. A mixed two-way repeated measures ANOVA of the D1 data revealed significant main effects for both Touch ($F_{(1,18)}=4.53$, $p=0.047$, $\eta^2_P = 0.20$) and Trial ($F_{(1,18)}=5.02$, $p=0.038$, $\eta^2_P = 0.22$). A mixed two-way repeated measures ANOVA of the D2 data revealed a significant main effect of Touch ($F_{(1,18)}=4.40$, $p=0.050$, $\eta^2_P = 0.20$), but not for Trial ($F_{(1,18)}=0.33$, $p=0.57$, $\eta^2_P = 0.018$). A significant interaction was not observed for either ANOVA ($p>0.05$).

The comparable ankle angle data are shown in Figure 5B and 5D. The ankle angle data reflected the changes in D1 and D2 described for the COP_{AP} across both the FN and FT groups.

The FN group demonstrated an initial plantarflexion (D1) of -0.434 ± 0.085° with a subsequent dorsiflexion (D2) of 0.927 ± 0.361° on average for the first trial. The amplitudes of the D1 and D2 deflections were smaller for the average of trials 8 through 10, with average amplitudes of -0.213 ± 0.119° and 0.255 ± 0.097°, respectively. The amplitudes of both D1 and D2 were reduced for the FT group, both for the first trial and for the average of trials 8 through 10. The average amplitude of D1 was -0.192 ± 0.049° and 0.004 ± 0.028° for the first trial and trial 8 through 10, respectively. The average amplitude of D2 was 0.170 ± 0.115° and 0.148 ± 0.077° for the first trial and trial 8 through 10, respectively. A mixed two-way repeated measures ANOVA of the D1 data revealed significant main effects for both Touch ($F_{(1,18)}$=11.58, p=0.003, η^2_P=0.39) and Trial ($F_{(1,18)}$=5.65, p=0.029, η^2_P=0.24). A mixed two-way repeated measures ANOVA of the D2 data revealed a significant main effect of Touch ($F_{(1,18)}$=5.65, p=0.029, η^2_P=0.24), but not for Trial ($F_{(1,18)}$=2.59, p=0.125, η^2_P=0.126). Significant interaction was not observed for either ANOVA (p>0.05).

The EMG data yielded few identifiable responses using either of the analysis approaches detailed in the Methods. There was no evidence that the forward visual field displacement induced a generalized startle response as no participants in either the FN or FT groups exhibited a response in SCM and an evoked response in OO was observed from a single trial from 1 FN participant and 1 FT participant. Moreover, only 1 FN participant demonstrated an initial evoked burst of activity in TA with forward visual field displacements on the first trial, whereas 2 FN participants demonstrated an initial evoked period of inhibition in SOL on the first trial. Similarly, initial evoked activity in TA was not observed in any FT participants and observed only twice in SOL. Given the very low number of identified responses across and within the FN and FT participants, it was not feasible to analyze the EMG data further.

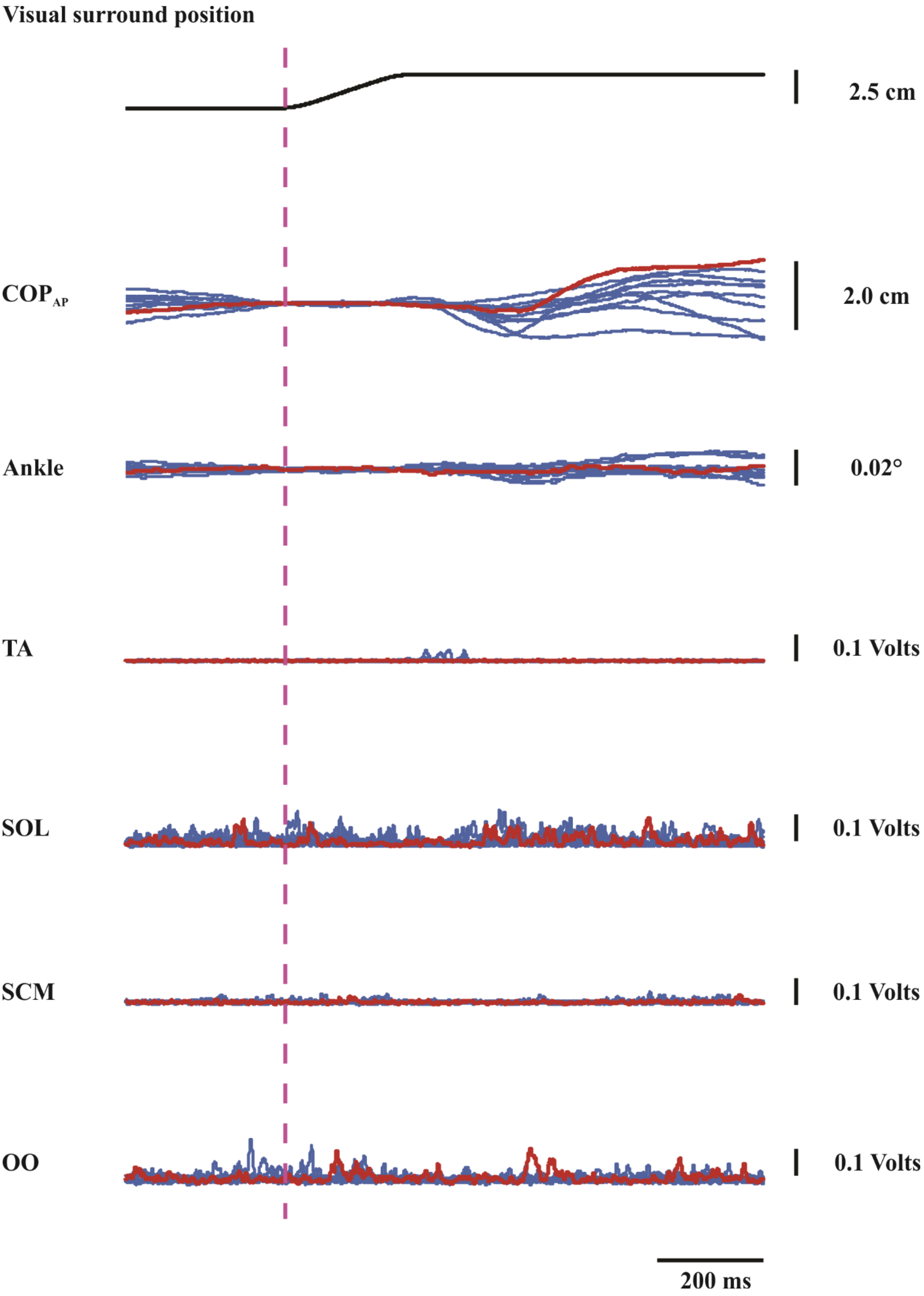
Visual surround position
2.5 cm
COP_{AP}
2.0 cm
Ankle
0.02°
TA
0.1 Volts
SOL
0.1 Volts
SCM
0.1 Volts
OO
0.1 Volts
200 ms

Figure 4. Complete series of data for a single participant receiving a forward visual displacement with no-touch (FN condition). The first trace is a schematic of the visual displacement , the subsequent sets of traces are the centre of pressure anterior-posterior (COP_{AP}) and ankle joint angle, respectively. All traces are 1200 ms (300 ms pre- and 900 ms post-stimulus onset) windows of data. The vertical dashed line is aligned with the onset of the visual stimulus. The red trace represents the response to the very first visual stimulus and the blue traces represent the responses to all subsequent exposures to the visual stimulus. On the left are the labels for each set of traces, and on the right are the vertical axis with units for each trace. Positive deflections represent a forward direction in the COP_{AP} trace and dorsiflexion in the ankle trace. COP_{AP}, center of pressure anterior-posterior; TA, tibialis anterior; SOL, soleus; SCM, sternocleidomastoid; OO, orbicularis oculi.

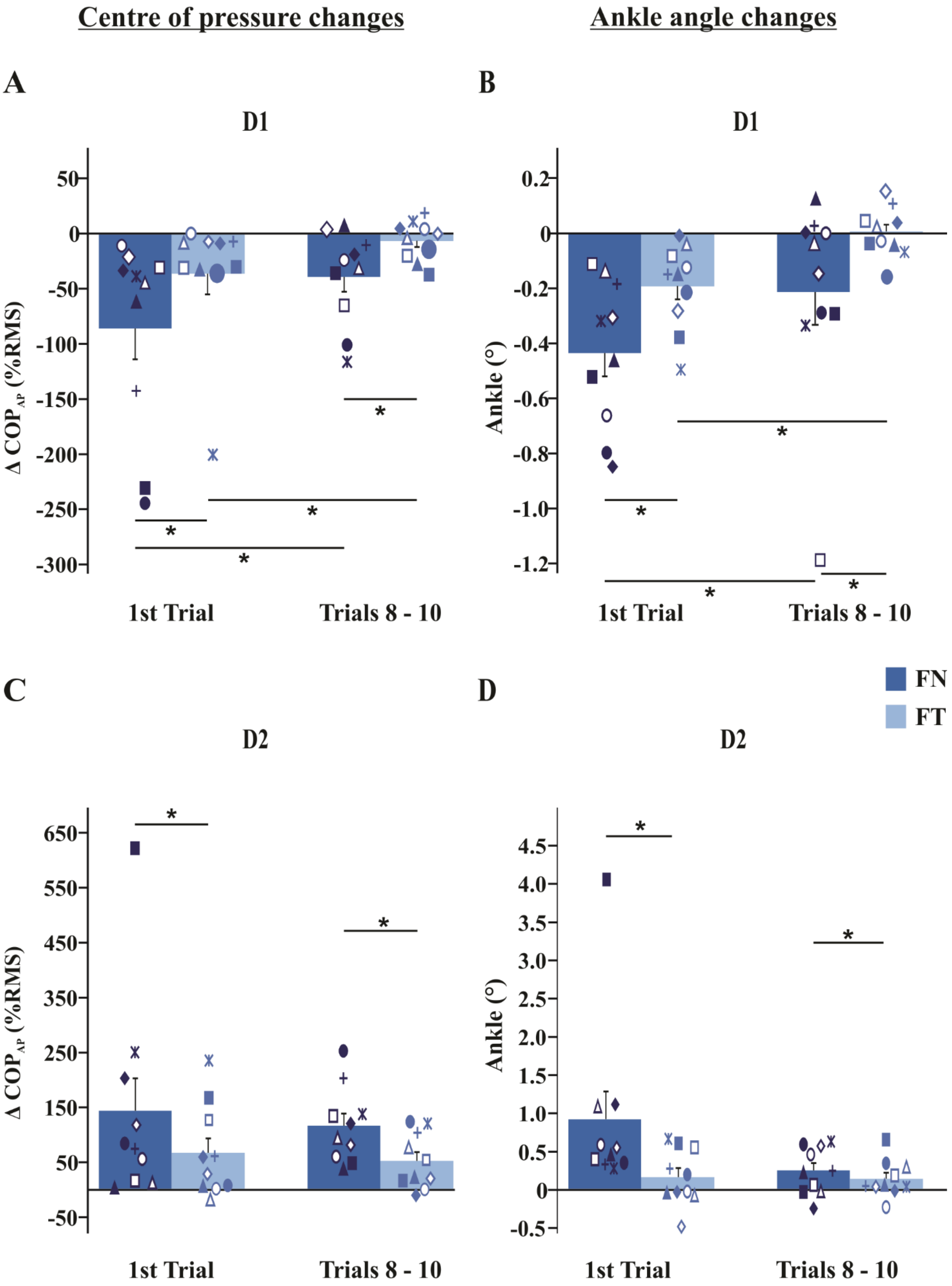
Centre of pressure changes
Ankle angle changes
A
D1
50
0
-50
-100
-150
-200
-250
-300
Δ COP_AP (%RMS)
*
1st Trial
Trials 8 - 10
B
D1
0.2
0
-0.2
-0.4
-0.6
-0.8
-1.0
-1.2
Ankle (°)
C
D2
650
550
450
350
250
150
50
-50
D
D2
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
0.5
-0.5
FN
FT

Figure 5. **Group average responses of participants receiving a forward visual perturbation.** Group averages of participants in the FN group are in dark blue; the group averages of participants in the FT group are in light blue. The left set of data in each subgraph are the averages of the 1st trial responses; on the right is the average of all participants' averaged trials 8 – 10. (**A**) Group average of the D1 in the COP_{AP} traces, in % RMS. (**B**) Group average amplitudes of D1 in the ankle traces, in degrees. (**C**) Group average of the D2 in the COP_{AP} trace, in % RMS. (**D**) Group average of D2 in the ankle trace, in degrees. Error bars indicate one standard error. Each individual marker indicates the response amplitude of a single participant in a specific group for the respective trial. Dark blue markers are participants in the FN group and light blue markers are participants in the FT group.

Responses to backward visual perturbations

All 20 participants that received a backward visual perturbation (BN and BT conditions) showed a biphasic response in the COP_{AP} and ankle angle motion that resulted in a backward sway. Figure 6 shows data traces from one participant from the BN group for all 10 trials of backward visual field displacement. The first trial is depicted by the red data trace in each panel. As can be seen, the first deviation in the COP_{AP} trace is a brief, forwards deflection, before the COP_{AP} then moves backward. This pattern of behaviour in the COP_{AP} is closely mirrored by an initial dorsiflexion, with a subsequent plantarflexion, at the ankle. The biphasic sway pattern in the COP_{AP} is largely absent following subsequent displacements of the visual surround (green traces in this participant), with either a general backward motion of the COP_{AP} present, or very little sway evident. The EMG traces for this participant are also shown in Figure 6 with no evidence of responses evoked in these muscles by the visual displacement.

Figure 7A and 7C show the group average amplitudes for the D1 and D2 deflections for the COP_{AP}. The dark green bars depict the average amplitudes for the 10 participants in the BN group. Following the first visual field displacement trial the initial deflection (D1) was characterized as a forward motion with an average displacement of 89.0 ± 19.6 %RMS across participants, while the second deflection (D2) was seen as backward motion with an average displacement of -172.6 ± 35.2 %RMS across participants. The amplitudes of both D1 and D2 were generally smaller for trials 8 through 10 across participants, with average amplitudes of 22.0 ± 12.1 %RMS and -7.1 ± 22.7 %RMS, respectively. As shown by the light green bars, the amplitudes of both D1 and D2 were reduced when participants of the BT group were touching a stable reference. The average amplitude of D1 was 15.6 ± 19.9 %RMS and 6.7 ± 12.4 %RMS, for the first trial and average of trials 8 through 10, respectively. The average amplitude of D2 was -70.1 ± 33.7 %RMS and -27.4 ± 23.0 %RMS, for the first trial and average of trials 8 through 10, respectively. A mixed two-way repeated measures ANOVA of the D1 data revealed significant interaction between Touch and Trial ($F_{(1,18)}$=5.88, p=0.026). Tukey's HSD tests indicated that D1 was smaller for the first trial responses of BT, compared with BN, and that trials 8-10 of BN were smaller than the first trial responses of BN. A mixed two-way repeated measures ANOVA of the D2 data revealed a significant interaction between Touch and Trial ($F_{(1, 18)}$=7.87, p=0.012). Tukey's HSD tests indicated that D2 was smaller for trials 8 through 10, compared with the first trial for the BN group, and that D2 for the first trial was smaller for the BT group than the BN group. However, the first trial and trials 8 through 10 were not different for the BT group and there was no difference between the BN and BT groups for trials 8 through 10.

The ankle angle data for the backward visual displacements are shown in Figure 7B and 7D. As can be seen, the changes in ankle angle closely resemble the changes in COP_{AP} for both the BN and BT groups. For the BN group, following the first trial, the average amplitude of D1 was 0.66 ± 0.16° of dorsiflexion, while the average amplitude of D2 was -1.37 ± 0.42° of plantarflexion. The amplitudes of D1 and D2 were smaller for the average of trials 8 through 10, with average amplitudes of 0.097 ± 0.037° and 0.000 ± 0.061°, respectively. For the BT group, the average amplitudes for D1 were 0.174 ± 0.138° and 0.003 ± 0.061° for the first trial and trials 8 through 10, respectively, while the average amplitudes for D2 were -0.179 ± 0.068° and -0.065 ± 0.079°, respectively. A mixed two-way repeated measures ANOVA of the D1 data revealed significant main effects for both Touch ($F_{(1,18)}$=6.05, p=0.024, η^2_P=0.25) and Trial ($F_{(1,18)}$=12.79, p=0.002, η^2_P=0.42), without a significant interaction (p>0.05). A mixed two-way repeated measures ANOVA of the D2 data identified a significant interaction between Touch and Trial

($F_{(1,18)}$=9.49, p=0.006). Tukey's HSD tests identified that D2 of the first trial was smaller for the BT group than the BN group and that trials 8 through 10 of the BN group were of smaller amplitude than the first trial of the BN group.

The analysis of the EMG data identified very few evoked responses with the backward visual displacements. None of the participants exhibited responses in either SCM or OO for any of the trials or conditions. Identifiable SOL bursts were observed for the first trial of 3 BN participants with an average onset latency of 134.0 ± 17.4 ms, whereas none of the BT participants exhibited evoked activity in either SOL or TA. Given the very low number of identified responses across and within the BN and the BT participants, it was not feasible to analyze the EMG data further.

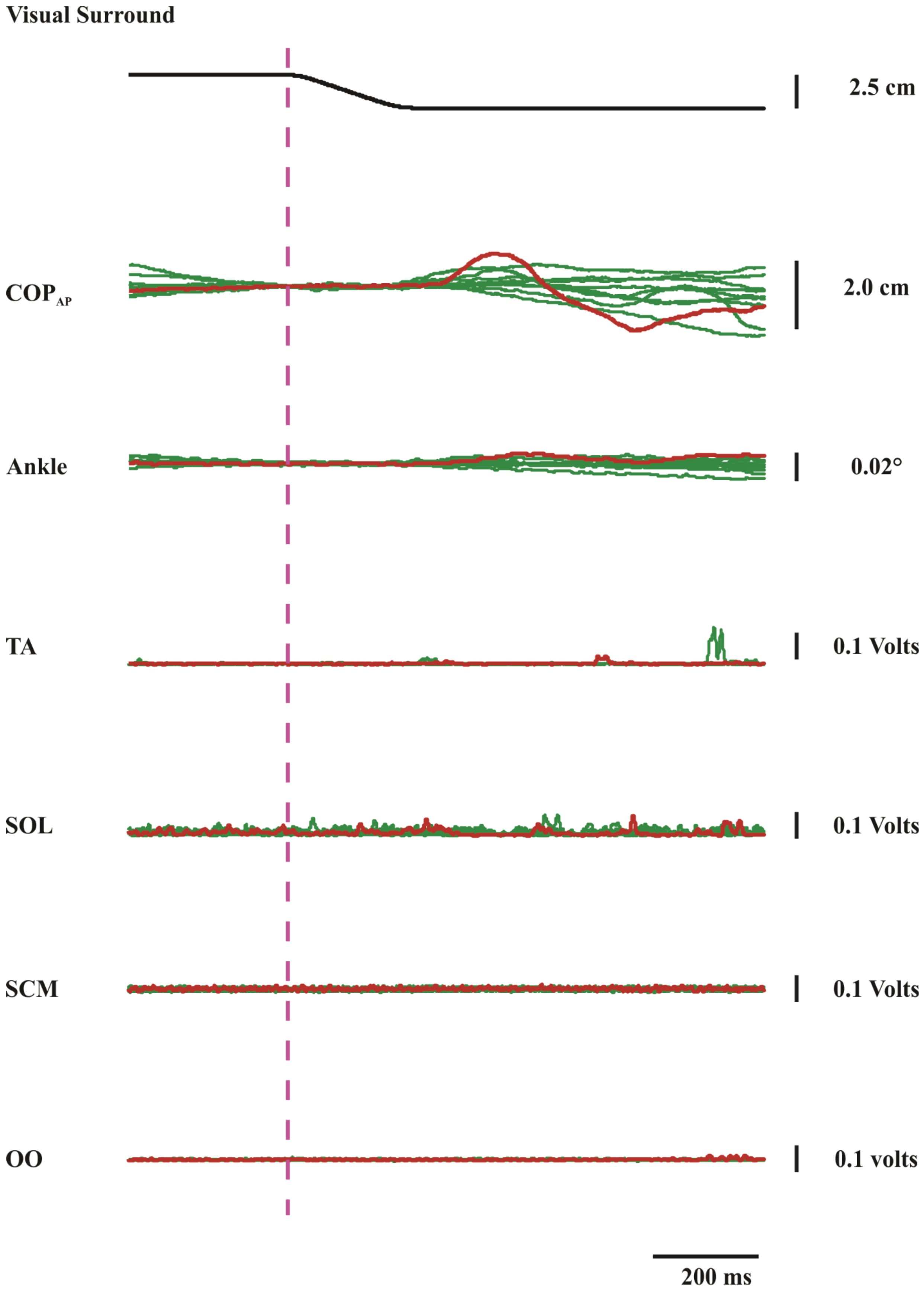
Visual Surround
2.5 cm
COP_{AP}
2.0 cm
Ankle
0.02°
TA
0.1 Volts
SOL
0.1 Volts
SCM
0.1 Volts
OO
0.1 volts
200 ms

Figure 6. Complete series of data for a single participant receiving a backward visual displacement with no-touch (BN group). The first trace is a schematic of the visual displacement, the subsequent sets of traces are the centre of pressure anterior-posterior (COP_{AP}) and ankle joint angle, respectively. All traces are 1200 ms (300 ms pre- and 900 ms post-stimulus onset) windows of data. The vertical dashed line is aligned with the onset of the visual stimulus. The red trace represents the response to the very first visual stimulus and the green traces represent the responses to all subsequent exposures to the visual stimulus. On the left are the labels for each set of traces, and on the right are the vertical axis with units for each trace. Negative deflections represent a backwards direction in the COP_{AP} trace and plantarflexion in the ankle trace. COP_{AP}, center of pressure anterior-posterior; TA, tibialis anterior; SOL, soleus; SCM, sternocleidomastoid; OO, orbicularis oculi.

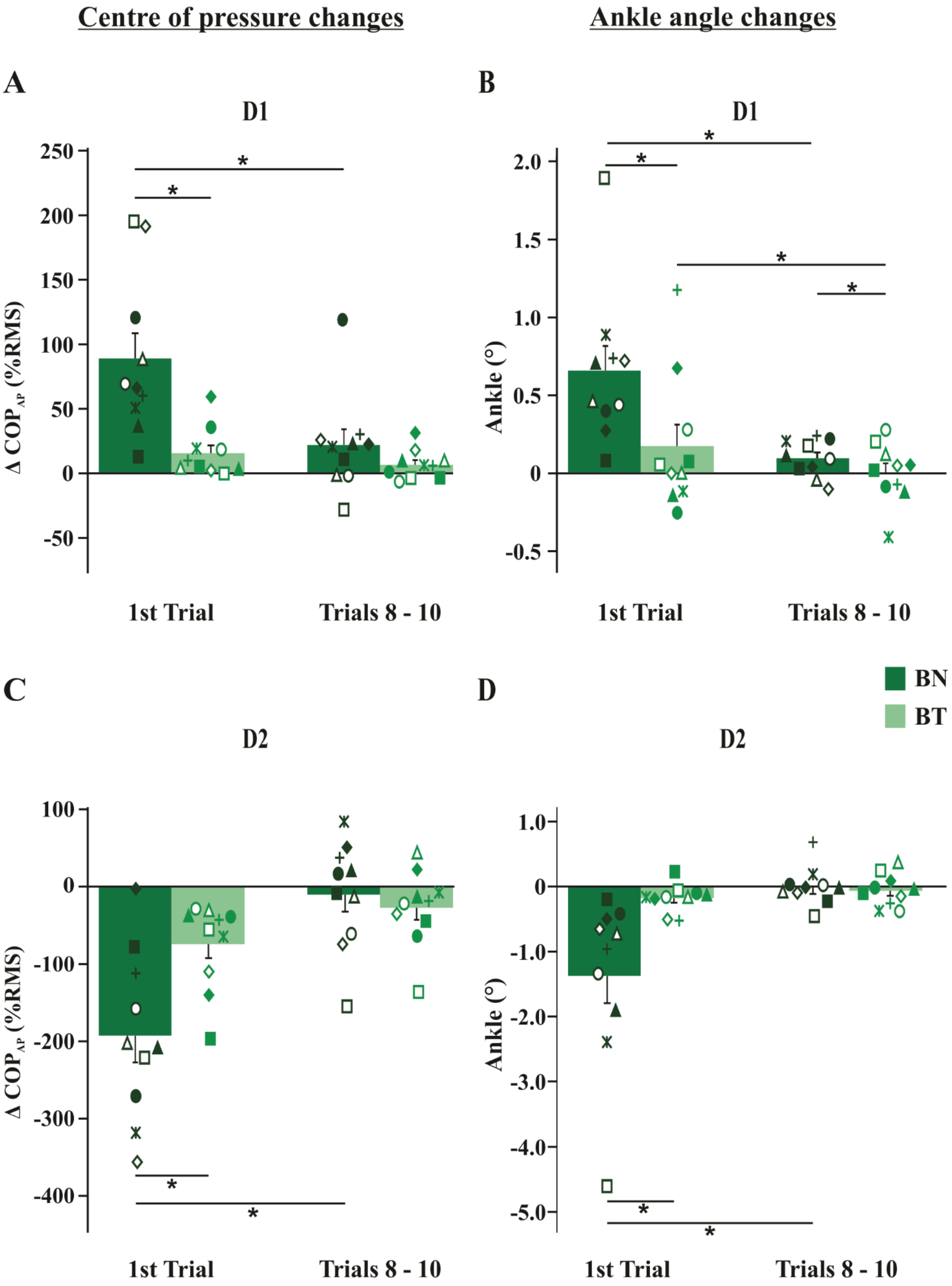
Centre of pressure changes
Ankle angle changes
A
D1
250
200
150
100
50
0
-50
Δ COP$_{AP}$ (%RMS)
*
*
1st Trial
Trials 8 - 10
B
D1
2.0
1.5
1.0
0.5
0
-0.5
Ankle (°)
*
*
*
*
1st Trial
Trials 8 - 10
C
D2
100
0
-100
-200
-300
-400
Δ COP$_{AP}$ (%RMS)
*
*
1st Trial
Trials 8 - 10
D
D2
BN
BT
1.0
0
-1.0
-2.0
-3.0
-4.0
-5.0
Ankle (°)
*
*
1st Trial
Trials 8 - 10

Figure 7. Group average responses of participants receiving a backward visual perturbation. Group averages of participants in the BN group are in dark green' the group averages of participants in the BT group are in light green. The left set of data in each subgraph are the averages of the 1st trial responses; on the right is the average of all participants' averaged trials 8 – 10. (**A**) Group average of the D1 in the COP_{AP} traces, in % RMS. (**B**) Group average amplitudes of D1 in the ankle traces, in degrees. (**C**) Group average of D2 in the COP_{AP} trace, in % RMS. (**D**) Group average of D2 in the ankle trace, in degrees. Error bars indicate one standard error. Each individual marker indicates the response amplitude of a single participant in a specific group for the respective trial. Dark green markers are participants in the BN group and light green markers are participants in the BT group.

CHAPTER 5

Discussion and Conclusions

I hypothesized that an unexpected, small, transient visual stimulus would evoke a balance corrective reaction in the direction of the visual stimulus. However, this hypothesis was not fully supported. Rather, I will argue that participants reacted to the visual stimulus by leaning and thus repositioning their body and head above a stationary base of support. I will further argue that this response indicates that visual feedback has an important role in controlling postural orientation but is not likely involved in initiating rapid balance corrective reactions. I also demonstrated that although the amplitude of the response to the visual stimulus generally decreased with repeated exposures, the response was never fully abolished. Similarly, I demonstrated that the provision of a light touch reference suppressed the response but did not completely negate it. I will argue that these two observations indicate that visual feedback is a potent cue in the control of postural orientation that is not easily ignored, despite the apparent conflict with other sensory systems.

Visual system regulates postural orientation

The small, transient, visual stimulus that was delivered in the current study evoked a biphasic shift in the COP_{AP} that ultimately led to a change in the COP_{AP} in the direction of the stimulus. This displacement in the COP_{AP} was accompanied by ankle angle changes that corroborate the movements in the COP_{AP}. In other words, participants receiving a forward visual displacement demonstrated a change in the COP_{AP} that led to a shift towards the anterior direction, which was generally accompanied with a slight dorsiflexion of the ankle joint. This forward displacement of the COP_{AP} , paired with a concomitant dorsiflexion of the ankle, suggests that the mass of the body was repositioned in a more forward direction, consistent with a forward lean. Similarly, participants that received a backward visual displacement leaned backward in response, as indicated by the repositioning of the COP_{AP} posteriorly with a concomitant ankle plantarflexion. Previous literature, that utilized visual stimuli, also demonstrated forward pitch of the body, or sway, in the direction of visual perturbation that was best characterized as leaning in the direction of the visual stimulus (Bronstein, 1986; Lee & Lishman, 1975; Lestienne et al., 1977).

The changes in the COP_{AP}, exhibited by the participants, were slow to develop, with visible deflection of the COP_{AP} beginning at around 300 – 400 ms. The slow-to-develop nature of the postural lean suggests that these responses were not sufficiently fast to be corrective balance reactions. Studies delivering mechanical perturbations in order to directly challenge balance demonstrated rapid balance reactions that were preceded by early and clear activation of relevant muscles at around 100 – 150 ms following the disturbance (Cresswell et al., 1994; Diener, Dichgans, Bootz, et al., 1984). Moreover, mechanical disturbances to balance that are expected to directly activate muscle stretch receptors of balance-related muscles, such as gastrocnemius, SOL or TA, generate responses even faster, with onset latencies of less than 100 ms (Diener et al., 1988; Nashner, 1976, 1977). This suggests that corrective balance reactions involve short-latency activation of relevant muscles to initiate forces directed at stabilizing the COM within the BOS. It is expected that there will be a lag from the appearance of a response in the EMG, the subsequent generation of torque at the joint, and the resulting displacement of the COP. Indeed, Misiaszek demonstrated that presumptive corrective balance reactions following unexpected displacement of a tactile reference resulted in short-latency EMG responses ranging from 75 – 120 ms in TA, with subsequent visible changes in the $\mathrm{COP}_{\mathrm{AP}}$ occurring at around 200 ms (Misiaszek et al., 2016; Misiaszek & Vander Meulen, 2017). Taken together, this suggests that the responses observed presently, with changes in the $\mathrm{COP}_{\mathrm{AP}}$ not visible until 300 - 400 ms, are too slow to be considered fast, corrective balance reactions by comparison. Rather, the responses observed following the small, transient visual displacements employed in this study are consistent with a lean or postural reorientation similar to that described previously for other forms of visual motion stimuli.

An unexpected finding from the current study was the biphasic nature of the $\mathrm{COP}_{\mathrm{AP}}$ displacements following the visual stimulus. This biphasic displacement in the $\mathrm{COP}_{\mathrm{AP}}$ was characterized by an initial small deflection in the opposite direction of the stimulus, followed by the larger deflection in the direction of the stimulus that is consistent with a reorienting postural lean. This early, small deflection in the $\mathrm{COP}_{\mathrm{AP}}$ likely represents a propulsive-like event related to the subsequent realigning of the body mass. That is, in response to a forward visual displacement the resulting forward motion of the body will require propulsive forces applied about the ankle and through the feet. Those propulsive forces would be in the direction opposite the intended direction of the focal movement. However, what is also interesting is that the ankle angles also demonstrated a small deflection in the direction opposite the focal movement, suggesting that the deflection in the $\mathrm{COP}_{\mathrm{AP}}$ was not only related to the application of propulsive force. McIlroy and Maki (1993b) demonstrated compensatory backwards stepping following a support surface translation was often preceded by a weight transfer to the stepping foot prior to the initiation of the step. The timing of the lateral weight shift observed by McIlroy and Maki (1993b) was about 300 ms, consistent with the timing of the propulsive-like event in the present data. It is also important to note that McIlroy and Maki (1993b) also reported that their stimulus induced a rapid, corrective balance reaction in TA that was insufficient to stabilize the COM within the BOS, necessitating the slower-to-develop compensatory step. However, McIlroy and Maki (1993a) also demonstrated that the weight transfer in anticipation of the compensatory step was not always expressed, and the magnitude of the weight transfer varied considerably from trial to trial. These observations, coupled with the long onset latency of the compensatory step, suggest that this more complex balance reaction involves a higher degree of processing than the shorter latency corrective balance reactions seen in TA, that were insufficient to maintain stability. The similarity between the response profiles observed by McIlroy and Maki (1993b) and following the visual displacements used in the present study are intriguing and suggest that postural reorientation described here requires a more complex processing mechanism than would be expected for a rapid balance corrective response, such as via cortical systems (Bolton, 2015).

The biphasic COP_{AP} displacement seen in the present study was not described previously in the literature for responses following visual displacements (Bronstein, 1986; Lestienne et al., 1977; Phillips et al., 2022). In part, this is likely related to the focus of those studies on the final postural orientation of the participants, rather than a careful analysis of the preceding events. (Bronstein, 1986; Lestienne et al., 1977). Moreover, Bronstein et al. (1986) delivered visual stimuli that had a gradual onset over a prolonged period of 30 s that evoked a commensurate gradual change in posture, unlike the present study which delivered a rapid, but brief visual stimulus. However, in a subsequent study, detailed in the Appendix, a smaller 1.25 cm visual displacement was explored and demonstrated a monophasic displacement of the COP_{AP} in the direction of the visual stimulus, lacking the propulsive-like component evident with the 2.5 cm displacement. This suggests that the smaller displacement used in the study of the Appendix resulted in a postural reorientation that did not require the same propulsive-like events initially.

This could be similar to the gradual forward lean induced by the gradual onset visual displacement employed by Bronstein et al. (1986). It is possible that the absence of a propulsive-like period prior to the focal postural reorientation is related to less need to generate large propulsive torques quickly with the smaller (Appendix) or more gradual (Bronstein et al., 1986) responses. Interestingly, McIlroy and Maki (1993a) demonstrated that compensatory stepping was not always preceded by a weight shift for which they argued the variability in how the compensatory step was generated was evidence for a complex interaction of cortical and cognitive influences in generating the responses they observed.

Day et al. (2016) proposed that visual information is processed in two distinct processes, after observing two separate and pronounced phases in the responses following rotation of a visual field in the frontal plane. The early phase of the response was suggested to be part of the quick acting balance control system that processes visual feedback to detect unintended self-motion and stabilize the body. The secondary, or late-phase, component of the response was suggested to be part of the postural control system that uses visual feedback to assist in aligning the body with gravity (Day et al., 2016). The postural control system, as described by Day et al. (2016), involves the slow processing of visual feedback alongside vestibular information to yield a gravitational reference for postural alignment. The postural realigning behaviour exhibited by the participants in the present study in response to the movement of the visual spatial reference (the visual stimulus) are consistent with the late-phase component of postural response proposed by Day et al. (2016). If so, then the visual stimulus applied in the present study was insufficient to trigger the quick acting reaction to stabilize the body. This would suggest that small, rapid and transient visual stimuli that might arise from a bump in a crowd are not sufficient to induce a balance corrective reaction, refuting the primary hypothesis of this thesis. Rather, we suggest that the visual stimulus used relayed a sense of self-motion and influenced the postural control system's estimation of the gravitational reference resulting in participants readjusting their posture to realign the body to the new visual spatial reference (Day et al., 2016).

Visual feedback is a potent sensory cue

In the present study, response amplitudes tended to habituate after ten exposures to the stimulus. However, measurable responses were still identified in some participants, particularly in the Forward No Touch group, indicating that the responses were not completely abolished This suggests that the visual stimulus is potent and difficult to ignore, even with repeated exposure and the participants becoming aware that the visual surround was unreliable. In our later study (see Appendix), that utilized a smaller 1.25 cm amplitude visual displacement, the postural realignment behaviour was also observed with repeated stimuli in some participants. As with the larger 2.5 cm stimuli, the responses to the 1.25 cm stimuli decreased in amplitude with repeated visual displacements. However, after ten trials a measurable shift in the COP_{AP} remained present in some participants. Thus, this much smaller and habituated visual stimulus continued to influence the control of the gravitational frame of reference, highlighting the potent influence of visual feedback in postural control.

This potency of the visual stimulus in inducing a response was further highlighted when the provision of light touch also failed to abolish the response in all participants, even after ten exposures to the visual displacement. The findings of the current study demonstrate that touch does mitigate postural responses, however the behaviour was not eliminated entirely. The stabilizing effects of light touch in balance control have been well established with touch capable of restoring postural sway of those standing with their eyes closed to magnitudes consistent to standing with eyes open (Jeka & Lackner, 1994). However, Kozhina et al. (2015) demonstrated that light touch stabilized sway when participants were exposed to a continuously oscillating virtual moving room stimulus, reducing the magnitude of sway evoked by the visual stimulus, but not abolishing the effect even after several seconds of exposure to the stimulus. Our findings are consistent with those of Kozhina et al. (2015) and indicate that the visual cues associated with establishing a gravitational frame of reference (Day et al. 2016) are difficult to ignore, despite the absence of corroborating sensory feedback from other sources, including augmented spatial cues provided through tactile feedback or the conflicting cues arising from the vestibular apparatus and muscle proprioceptors.

Future Directions

The primary finding of this study suggests that vision is involved in controlling postural alignment to the visual spatial reference that informs a gravitational frame of reference, and that visual feedback exerts such control despite conflicting sensory feedback from other posture-related sensory inputs. However, it remains unclear as to what the limits of this potent impact of visual feedback on postural orientation might be. For example, Lestienne et al. (1977) used a sustained stimulus that created motion parallax over a period of seconds to minutes, inducing a forward lean. However, they did not report participants fell or took a compensatory step. Presumably, the sensory signals from the vestibular apparatus and muscle proprioceptors, in conflict with the powerful feedback from the visual system, prevented the induced lean from displacing the COM beyond the BOS. Further studying how visual inputs that conflict with other sensory cues in the control of postural orientation will lead to better understanding of multisensory integration for balance control in complex tasks and environments.

One potential study would be to determine whether participants who lean forward following a small, transient visual displacement, such as the one used in the present study, remain in this new postural alignment over an extended period of time if the visual frame of reference remains at the new, forward position. If the participants continue to lean forward indefinitely, then this would indicate that the visual frame reference powerfully influences the establishment of the gravitational frame of reference and overrides the contributions from other sensory cues. However, if participants eventually realign to vertical, then this would indicate that the error in postural orientation detected by the other sensory cues assists with establishing an updated visual frame of reference. A second potential study would be to "stack" the visual displacements such that a second stimulus is delivered at about the time participant stabilizes at the new, forward position of their COP_{AP}. From the present study, we would expect that the response to this second stimulus would be decreased in amplitude. However, one might anticipate that the decrease in response amplitude would be greater for the "stacked" stimulus as the disagreement between the visual spatial frame of reference and the feedback from the other sensory cues would be more pronounced. This would further support the role of error in postural orientation, detected by the other sensory sources, in assisting with updating the visual spatial frame of reference that informs a gravitational frame of reference. If instead, participants continue to lean further forward with the "stacked" stimulus, then this would suggest that the processes involved in updating the visual spatial frame of reference require a long time, and presumably a high level of cortical processing. It would then be interesting to see if a series of "stacked" stimuli are capable pushing participants to instability and a loss of balance, or a compensatory step.

Limitations

A limitation of the current study was the small sample sizes used for each of the conditions. Post-hoc power analysis revealed the power for comparing within subject effect of trial was 0.40, and between subject effect of touch was 0.51 for the forwards conditions. Post-hoc power analysis for the backwards conditions revealed the within subject effect of trial had a power of 1, while the between subject effect of touch yielded a power of 0.47. Power calculations indicate that we were likely underpowered in some of the comparisons. Nevertheless, we were able to identify significant differences in some comparisons and the findings, in general, were consistent across participants suggesting that our interpretation of the findings is reasonable. Another limitation was the calculation of the D2 amplitude from the position of the peak of D1, rather than from time zero. The current calculation assumed that D1 and D2 are part of a singular response, therefore if these are two separate events our assumption may exaggerate the amplitude of D2. Additionally, the lack of information arising from the EMG analysis is a possible limitation. At the outset of the study, it was anticipated that the rapid visual displacements would generate fast corrective balance reactions, comparable to the responses evoked by a similar rapid displacement of a touch reference (Misiaszek et al. 2016). Misiaszek et al. (2016) were able to identify distinct EMG bursts in single trials (i.e. in response to individual stimuli) using the approach wherein the full-wave rectified trace exceeds the 95% confidence band for the EMG immediately prior to the response. In the present study, this same approach did not reveal any identifiable responses in the single trials. Consequently, we developed an approach employing wavelet transformations of the EMG to enhance the signal. Visual inspection of the resulting scalograms appeared to suggest that relevant changes in EMG activity were present in several instances. However, applying objective criteria to the analysis failed to consistently identify changes in the EMG traces. Therefore, it was not possible to estimate onset latencies of any changes or to confidently evaluate EMG amplitudes. Given that the responses observed in the COP_{AP} and ankle angle data suggested the behavioural response was slow to develop and relatively small in magnitude, it is possible that changes in EMG activity were present, but too small to emerge from the background activity with these techniques. It is also possible that the changes in COP_{AP} position were generated by muscles other than TA and SOL. However, given the concomitant movements of the motion of the COP_{AP} and the ankle angle, it is possible that the postural response was mediated via an ankle strategy which would suggest that TA and SOL should likely be involved, at least in part.

Conclusions

It is well established that vision is functionally relevant to balance and postural sway. Visual feedback indicates the motion of the head within the visual spatial frame of reference through motional parallax. Our findings suggest that creating the illusion of head-motion through a rapid, small, and transient displacement of a visual surround evoked a postural realignment, or repositioning of the head, to the new location of the visual spatial frame of reference. We further observed that these responses were not completely abolished with repeated exposure to the false visual stimulus. The participants that were given a stable light touch reference demonstrated the postural realigning behaviour, albeit at smaller amplitudes, despite the stable cutaneous feedback indicating the body did not move. These two outcomes suggest that visual feedback is a potent sensory cue that is difficult to ignore. Together, the findings suggest that visual feedback from the short, transient visual displacements were likely not involved in initiating rapid, corrective balance reactions to prevent falls. Instead, we suggest vision is involved in the alignment of posture to the visual spatial frame of reference and integrates with other sensory systems, such as the vestibular system and muscle proprioceptors, to align the body within a gravitational frame of reference. This study highlights potentially how potent visual feedback is in postural alignment, as seen by the continued postural readjustments by participants despite other sensory sources indicating their posture is appropriately aligned. We further suggest that understanding sensorimotor integration, including the role of vision, in postural regulation highlights the effects of false stimuli, or degradation of a sensory system with age or injury, in balance control and maintaining stability.

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APPENDIX

Introduction

Maintaining upright stance is critical for most daily activities, including walking, and standing. Upright stance is inherently unstable and is prone to be further challenged by many injury and disease states, as well as the normal aging process (Anson & Jeka, 2010; Sundermier et al., 1996; Wallmann, 2009. Inherent instability of upright stance is caused by the majority of the body's mass being elevated high above the feet, creating a natural sway pattern from gravity and other internal and external forces acting upon the mass of the body. Balance control is a complex process involving sensorimotor systems. Neural control of balance integrates sensory information from multiple systems including visual, vestibular, and somatosensory feedback. During standing, stable balance occurs when the body centre of mass (COM) is maintained within the limits of the base of support (BOS), made up of the feet. If the COM moves beyond the limits of the BOS the body becomes unstable and a fall can occur. The onset and progression of a fall will be encoded by the various sensory systems of the body. For example, motion about the hip and ankles created by the fall will stretch muscles about the joints and activate proprioceptors, while changes in pressure under the feet and stretching of the skin across the joints will activate cutaneous receptors. Moreover, head-based sensory systems, such as the vestibular and visual systems will encode the movement of the head relative to gravity and to the spatial reference, as gravity accelerates the head towards the ground (Day & Fitzpatrick, 2005; Mackinnon, 2018)

It is well established that visual feedback influences balance. Seminal work by Edwards (1946) demonstrated decreased stability when the eyes were closed (Edwards, 1946). In addition, slow oscillations of a visual surround entrains postural sway to oscillate at a similar frequency (Lee & Lishman, 1975) Moreover, findings from Lestienne et al., (1977) and Bronstein (1986) demonstrated that unidirectional displacement of a visual stimulus evoked a postural response in the direction of the stimulus. This was seen as forward body pitch or lean following a forward visual field displacement (Bronstein, 1986; Lestienne et al., 1977). Bronstein (1986) also demonstrated that the influence of visual input to balance control is plastic and can be adapted based on context and prior experience, as seen by the habituation of postural responses following repeated exposure to a visual stimulus (Bronstein, 1986).

In the main study of my thesis, I demonstrated that a small, rapid, transient visual field displacement of 2.5 cm, with a peak velocity of 124 mm/s, resulted in a postural correction consistent with the findings of Bronstein (1986), despite the much smaller and briefer stimulus. The postural corrections I observed habituated with repeated trials but were still expressed even after ten trials. Moreover, provision of light touch reduced, but did not abolish the responses, even with repeated exposures. This suggests that the visual displacement of 2.5 cm was difficult to ignore, and that visual feedback is a potent cue in balance control. The choice of using a 2.5 cm visual displacement was derived from the work of Forero & Misiaszek (2013) who delivered mechanical pulls at the waist during treadmill walking and observed rapid onset (<100 ms) postural corrections. In that study, the mechanical pulls displaced the waist 1.25 cm by the time the balance reaction was detected. Assuming the body is a rigid lever and rotates around the ankle joint, it was estimated that the head may move approximately twice as far as the waist with the pulls to the waist, thus 2.5 cm. However, this assumption is simplistic as a force applied at the waist likely results in a complex disturbance to the mechanical arrangement of the body segments, and likely includes piking or bending at the waist. Therefore, the disturbance to the position of the head with such a stimulus is likely less than the estimate of 2.5 cm. In the current study, a 1.25 cm visual displacement will be employed to mimic the estimated displacement following a pull to the waist. The peak velocity will remain at 124 mm/s, thereby reducing the duration, as well as the amplitude, of the stimulus. I hypothesize that participants will respond to this smaller, briefer visual stimulus with a postural correction similar to that observed with the 2.5 cm stimulus. Moreover, I hypothesize that the evoked postural reactions will persist with repeated trials and in the presence of a stabilizing light touch, thereby confirming the potent influence of visual feedback in regulating postural orientation. .

Methods

Participants

Twenty naïve participants (ages 18-39; 10 females, 10 males) participated in this study. All participants reported no history of neurological or musculoskeletal injuries, back pain, or concussions in the last 6 months. Participants consented to the present study which was approved by the University of Alberta Human Research Ethics Board (Pro00086777) and in accordance with the Declaration of Helsinki. Participants were recruited from the student population at the University of Alberta and members of the local community.

Set-up and apparatus

Participants stood barefoot on an ethylene-vinyl acetate foam pad (5 cm thick) with their feet shoulder-width apart, in a comfortable stance, atop a 6-component force plate (AMTI OR6-7-OP-2K-STT; A-Tech instruments LTD., Watertown, MA, USA) that was flush with the floor. An electrogoniometer (SG100A, Biometrics LTD, Newport, UK) was placed across the right ankle to record joint angle. Electromyographic (EMG) activity was recorded from the right tibialis anterior (TA), soleus (SOL), rectus femoris (RF), and sternocleidomastoid (SCM) using pairs of Ag/AgCl surface electrodes (NeuroPlus A10040, Vermed, Bellows Falls, VT, USA) positioned approximately 2 cm apart on the skin over the center of the belly of the muscle, oriented in the predicted direction of the muscle fibres. A ground electrode was placed on the skin over the olecranon process. All electrode sites were shaved and cleaned with alcohol to ensure impedance was below 20 kΩ (Grass F-EZM5 , Astro-Med, Inc., West Warwick, Rhode

Island, USA). Participants listened to white noise through a pair of over the ear headphones (Beats Solo by Dr. Dre, Beats Electronics LLC, Culver City, CA, USA) during all experimental testing to mask any background noises and the sound of the stepper motor, attached to the enclosure, used to deliver the visual displacements (see Chapter 3: Set Up and Apparatus for details on the moving room enclosure). EMG signals were amplified and band-pass filtered (10 Hz - 1 kHz, 60 Hz notch filter, Grass P511 amplifiers, AstroMed Inc, West Warwick, RI, USA). All signals were digitized using a 16-bit data acquisition card (PCI-MI0-16E-4, National Instruments, Austin, TX, USA) with a sampling rate of 2000Hz and then stored on a hard drive using custom-written LabView programs (LabView Version 18, 64-bit, National Instruments).

Protocol

Participants were pseudo-randomly allocated to one of four groups based on the direction of the displacement of the visual surround (forward, F; backward, B) and the presence of a stable touch reference (no touch, N; touch, T), thereby resulting in five participants per group. Note that a forward displacement of the visual surround indicates the front wall moved away from the participant. Participants in the FN and BN groups completed 3 standing conditions: 1) standing within the stationary enclosure; 2) standing within the enclosure that was unexpectedly displaced; and 3) standing within the stationary enclosure. Participants in the FN and BN groups were asked to stand with their hands comfortably by their side during each condition. Participants in the FT and BT groups completed 4 standing trials: 1) standing within the stationary enclosure; 2) standing within the stationary enclosure while touching a stable reference; 3) standing within the enclosure that was unexpectedly displaced while touching a stable reference; and 4) standing within the stationary enclosure. During the touch conditions, participants were asked to lightly touch (<1 N of vertical load) the stable reference with the pad of the second digit (index finger) of their right hand with the other fingers curled into the palm. The left hand was relaxed at their side. All participants were asked to look at a visual reference located on the front wall of the enclosure during all trials. Participants sat between each condition for a minimum of 2 min.

Data for each of the quiet standing conditions (i.e. the conditions without visual stimuli) was recorded for 90 s. The primary purpose of the quiet standing conditions was to deceive the participants into believing the enclosure was stationary. As such, the order of the conditions was not randomized. During the visual displacement conditions, participants stood quietly for 10 s to 15 s within the enclosure before the visual surround was first unexpectedly displaced 1.25 cm, with a peak velocity of 124 mm/s, for a total stimulus duration of 125 ms. Subsequently, the enclosure was slowly returned to the starting position at an average velocity of 0.64 mm/s, which was imperceptible. The next stimulus was delivered after the enclosure had returned to the starting position for at least 2 s, with a random interval of up to 15 s. Ten such stimuli were delivered, initiated manually by a researcher. The visual displacement conditions lasted up to 6 min to complete. Participants were not informed that the visual surround would move prior to testing.

Once all experimental conditions were completed, maximum voluntary contractions (MVCs) were obtained for each muscle. To do so, isometric contractions of the target muscles were performed against resistance, with verbal encouragement, and monitored online. The electrogoniometers were calibrated *in situ*. Following data collection, participants were debriefed on the true nature of the study, including the deception, and given the opportunity to withdraw their consent.

Data and statistical analysis

Post-hoc analysis of the mechanical data was conducted using custom written LabView programs. Evoked reactions to the visual stimuli in the force plates and ankle goniometer were analyzed from a 1200 ms segment of data extracted from the continuous data stream and low-pass filtered (20 Hz, 2nd order zero-lag Butterworth filter). Center of pressure in the anterior-posterior direction (COP_{AP}) was calculated from the ground reaction forces and moments recorded by the force plate, using formula (1).

$$COP_{AP} = \frac{M_x - \left(Z_{off} \times F_y\right)}{F_z} \tag{1}$$

The COP_{AP} and ankle goniometer traces were corrected for the ongoing, background sway by removing the slope of the trace estimated from a linear regression calculated for 100 ms centered at the time of the stimulus onset (see Figure 2, Chapter 3: Data Analysis). The first displacement typically resulted in a deflection of the COP_{AP} in the direction of the stimulus. The amplitude of the induced sway was calculated as the peak deflection in COP_{AP}, relative to the position at time zero. This amplitude was normalized to the root-mean-square of the COP_{AP} recorded over the 60 s of the first quiet standing condition for each participant. The induced sway amplitude of the second through tenth stimuli were calculated using the same time point of the peak identified in the first trial, for each participant. The same procedure was applied to the ankle goniometer data, but the ankle data were not normalized to quiet standing and recorded in degrees. Analysis of the EMG data was performed using the wavelet analysis procedure described in Chapter 3. Briefly, a change in EMG activity was identified when the scale-averaged wavelet traces exceeded the 95% confidence interval of the background EMG for a minimum of 20 ms continuously, with an onset latency of at least 40 ms and no later than 200 ms.

The amplitude of the deflections observed in the COP_{AP} and ankle data were analyzed separately for each direction of visual displacement. To evaluate the effect of repeated exposure to the visual stimulus (i.e. habituation), the amplitude of the outcome measure of the first trial was compared with the amplitude from the average of trials eight through ten, for each participant. Separate mixed model two-way repeated measures analyses of variance (ANOVA) were performed for the COP_{AP} and ankle amplitudes, to compare the effects of Touch (2 levels, T and NT) and Trial (2 levels, 1st trial and trials 8-10), where Touch is the independent factor and Trial is the repeated factor. Descriptive data are reported as means with standard deviations. Significance was set at $\alpha<0.05$. All statistical analysis was performed using the Real Statistics Resource Pack software (Release 8.9.1; Copyright (2013-2023) Charles Zaiontz).

Results

The first, unexpected displacement of the visual surround evoked a sway response in the direction of the visual stimulus in 9 out of 10, as observed in the COP_{AP} and ankle angle traces (Figure A.1 and A.3~~.~~ The response observed in the COP_{AP} was characterized by a unidirectional sway in the direction of the visual stimulus. Given the gradual development of the sway, the onset of the response is ambiguous, however, the position of the COP_{AP} has clearly shifted by 600 ms following the stimulus onset. The EMG analysis did not yield clearly identifiable responses to the visual stimulus in the muscles recorded and will therefore not be addressed further.

Responses to forward visual perturbations

Figure A.1 shows the data traces from a single participant in the FN group, with the response to the first visual displacement depicted in red. It is evident that this participant responded to the first stimulus by swaying forward, as shown by the progressive forward displacement of the COP_{AP}. The forward sway appears to be gradual and occur over a period of several milliseconds, the exact onset of the response is unclear. On subsequent trials, depicted by the blue traces, the same general response can be seen in a few trials, while some later trials show much flatter COP_{AP} trajectories or even backwards motion. This movement is reflected in the ankle angle motion as well.

All five participants in the FN condition expressed a forward sway in their COP_{AP} data with the first exposure to the visual stimulus. This consistent result in the FN group is expressed by the data for the first trial response amplitude depicted in Figure A.2A, averaged across all five participants (168 ± 84.3 %RMS). In contrast, the responses for trials 8-10 did not show a consistent forward sway across participants, with some participants responding with forward sway, backward sway, or very little response in these later trials, resulting in a mean COP_{AP} displacement of -1.94 ± 92.0 %RMS. Also shown in Figure A.2A are the group average data for the response amplitudes from the FT group. As can be seen, provision of light touch markedly altered the response to the first visual field displacement, with three participants exhibiting a forward sway and two participants swaying backwards, resulting in a group average response of -4.74 ± 37.7 %RMS. In contrast, participants typically swayed forward in response to trials 8-10 with the provision of touch, resulting in an average response amplitude of 36.9 ± 12.9 %RMS. However, a mixed model two-way ANOVA revealed no significant main effects of Trial ($F_{(1,8)}$=0.597, p=0.462, η^2_P=0.069) or Touch ($F_{(1,8)}$=2.63, p=0.143, η^2_P =0.25), and no significant interaction ($F_{(1,8)}$=1.63, p=0.0.238, η^2_P=0.17).

As shown in Figure A.2B, the ankle data showed a consistent dorsiflexion following the first visual field displacement across participants in the FN group, with an average dorsiflexion of 1.19 ± 0.90° and inconsistent behaviour in response to trials 8-10, resulting in a group average plantarflexion of 0.48 ± 0.48°. The response at the ankle to the first visual field displacement was less consistent in the FT group resulting in a group average plantarflexion of 0.108 ± 0.77°, whereas there was little evidence of ankle motion following trials 8-10 with an average plantarflexion of 0.054 ± 0.065°. A mixed model two-way ANOVA revealed no significant main effects of Trial ($F_{(1,8)}$=1.37, p=0.276, η^2_P =0.146) or Touch ($F_{(1,8)}$=3.75, p=0.089, η^2_P =0.319), and no significant interaction ($F_{(1,8)}$=1.55, p=0.247, η^2_P=0.16).

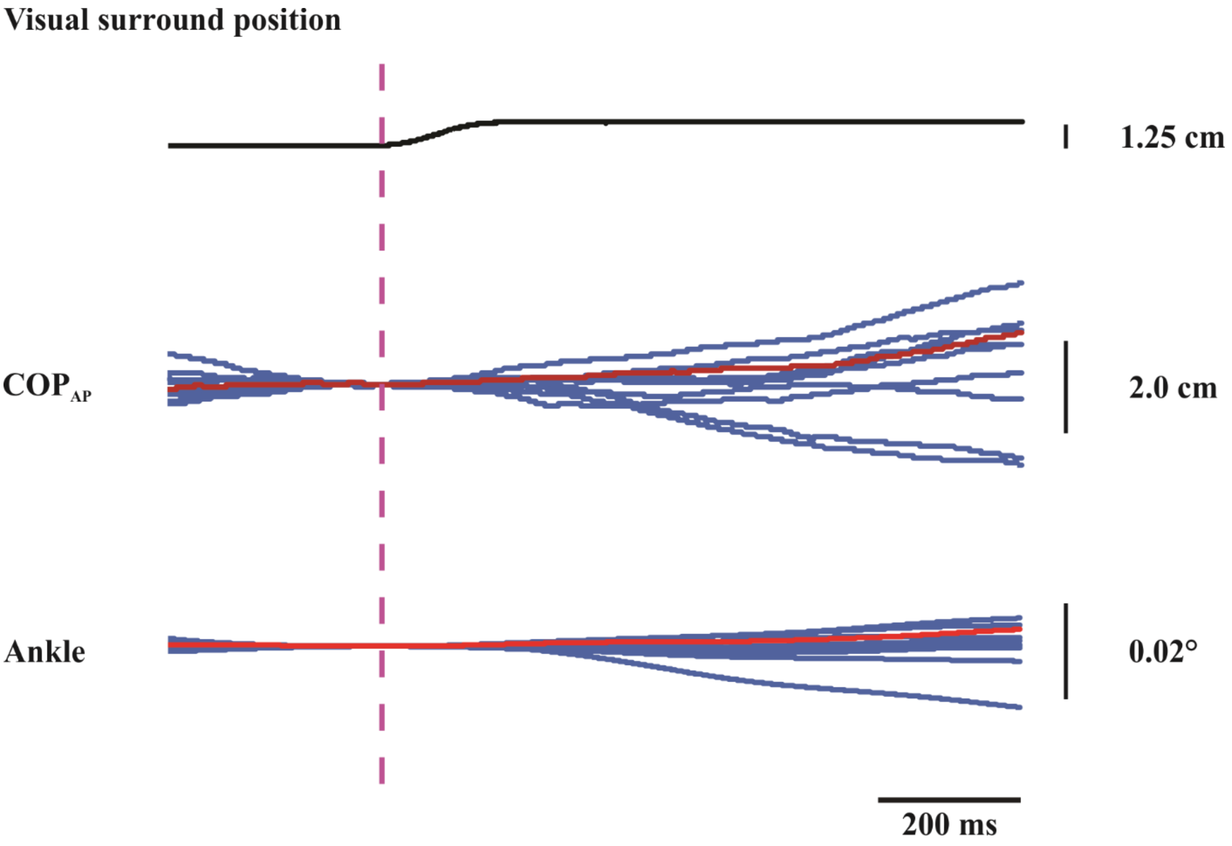

Figure A.1 Complete series of data for a single participant receiving a forward visual displacement with no-touch (FN condition). The first trace is a schematic of the visual displacement, the subsequent sets of traces are the centre of pressure anterior-posterior (COP_{AP}) and ankle joint angle, respectively. All traces are 1200 ms (300 ms pre- and 900 ms post-stimulus onset) windows of data. The vertical dashed line is aligned with the onset of the visual stimulus. The red trace represents the response to the very first visual stimulus and the blue traces represent the responses to all subsequent exposures to the visual stimulus. On the left are the labels for each set of traces, and on the right are the vertical axis with units for each trace. Positive deflections represent a forward direction in the COP_{AP} trace and dorsiflexion in the ankle trace.

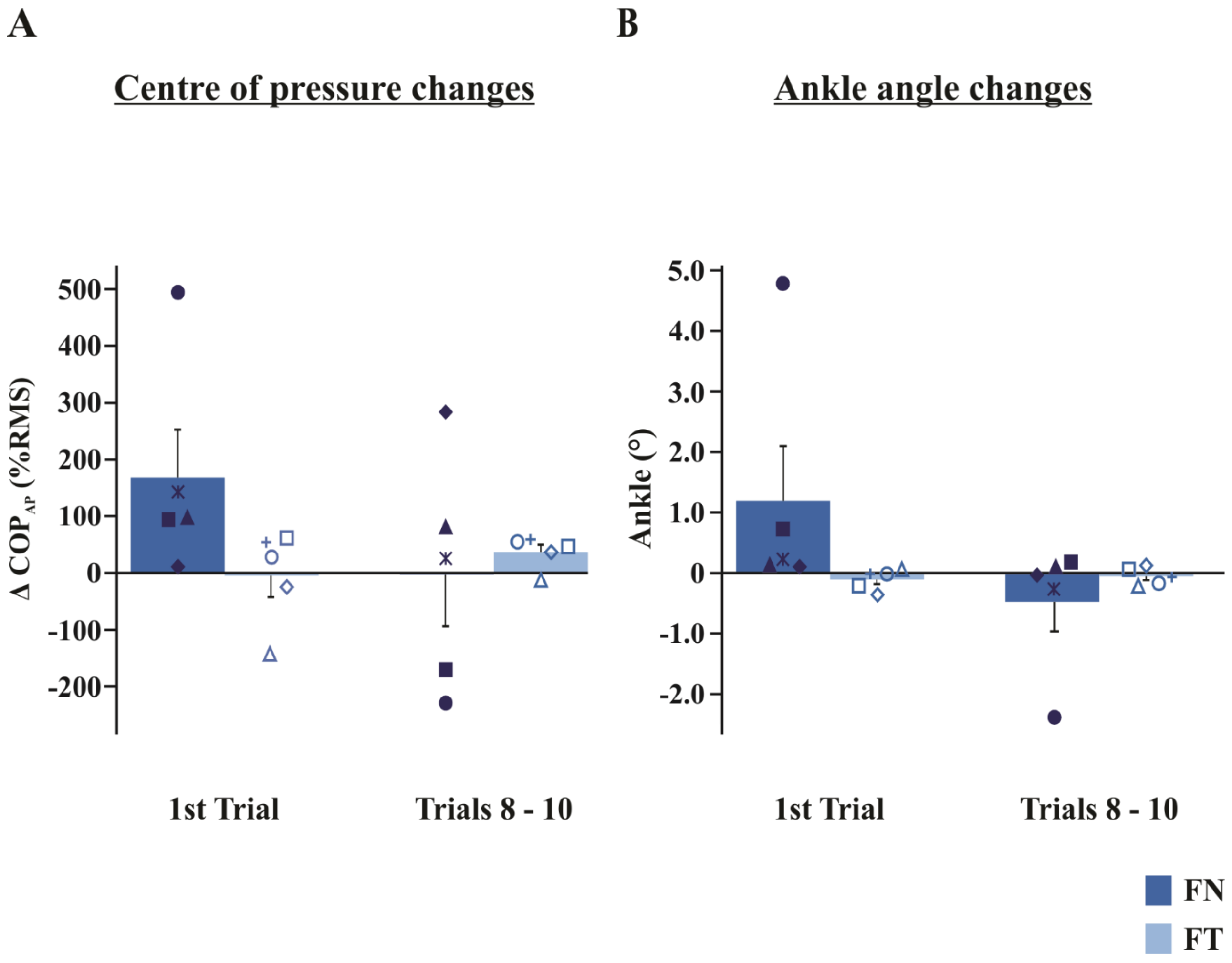

Figure A.2 Group average responses of participants receiving forward visual perturbations. Group averages of participants in the FN condition are in dark blue; the group averages of participants in the FT condition are in light blue. The left set of data in each subgraph are the averages of the first trial responses; on the right is the average of all participants' averaged trials 8 – 10. (**A**) Group average amplitude for the change in the COP$_{AP}$ trace, in % RMS. (**B**) Group average amplitudes for the change in ankle angle, in degrees. Error bars indicate one standard error. Each individual marker indicates the response amplitude of a single participant in a specific group for the respective trial. Dark blue markers are participants in the FN group and light blue markers are participants in the FT group.

Responses to backward visual perturbations

The data traces from a single participant in the BN condition are shown in Figure A.3, with the responses to the first visual displacement depicted in red. The data traces demonstrate this participant swayed backward, as shown by the gradual displacement of the COP_{AP} in the posterior direction. The backward sway occurs gradually over a period of several hundred milliseconds. Subsequent trials are depicted in green with a few of the traces demonstrating a similar backward sway. However, it is evident that in the later trials this participant begins to sway forward. The movement in the COP_{AP} is reflected in the ankle motion.

Four out of five the participants in the BN conditions exhibited a backward sway in their COP_{AP} data following the first exposure to the visual stimulus. Figure A.4A depicts this first trial result in the dark green bars which represents the BN group average response amplitudes, for all five participants (-60.5 ± 61.8 %RMS). Similarly, the responses for trials 8-10 also show a general backward sway across four out of five participants, resulting in a mean COP_{AP} displacement of -80.4 ± 61.2 %RMS. Also shown in Figure A.4A are the group average data for the response amplitudes from the five participants in the BT group. It is evident that the provision of a stable light touch reference generally reduced the average response amplitude to the first visual stimulus (-14.13 ± 15.8 %RMS), with four of five participants swaying backward and one swaying forward, similar to the BN condition. In contrast, in trials 8-10 three out of five participants in the BT condition swayed forward, and two swayed backward, yielding an average response amplitude in the forward direction (24.4 ± 24.9 %RMS). However, a mixed model two-way ANOVA for the COP_{AP} data revealed no significant main effects for Trial ($F_{(1,8)}=0.032$, $p=0.862$, $\eta^2_P=0.004$) or Touch ($F_{(1,8)}=3.75$, $p=0.089$, $\eta^2_P=0.319$), and no significant interaction ($F_{(1,8)}=0.318$, $p=0.588$, $\eta^2_P=0.038$).

Figure A.4B shows the ankle data of the BN group, in dark green, following the first visual displacement to be inconsistent with a backward sway, as indicated by the general dorsiflexion across 4 out of 5 participants (0.167 ± 0.07°). These same participants seem to show little evidence of ankle motion following trials 8-10, with an average dorsiflexion of 0.019 ± 0.106°. Participants in the BT condition (light green bars) appear to yield a different outcome with all five participants exhibiting plantarflexion following the first visual displacement, yielding a group average of -0.286 ± 0.176°. However, following trials 8-10, four of the five participants demonstrate dorsiflexion, yielding a group average of 0.425 ± 0.267°. A mixed model two-way ANOVA revealed no significant main effects of Trial ($F_{(1,8)}$=1.60, p=0.241, η^2_P =0.167) or Touch ($F_{(1,8)}$=0.057, p=0.817, η^2_P=0.007), and no significant interaction ($F_{(1,8)}$=3.73, p=0.090, η^2_P=0.318).

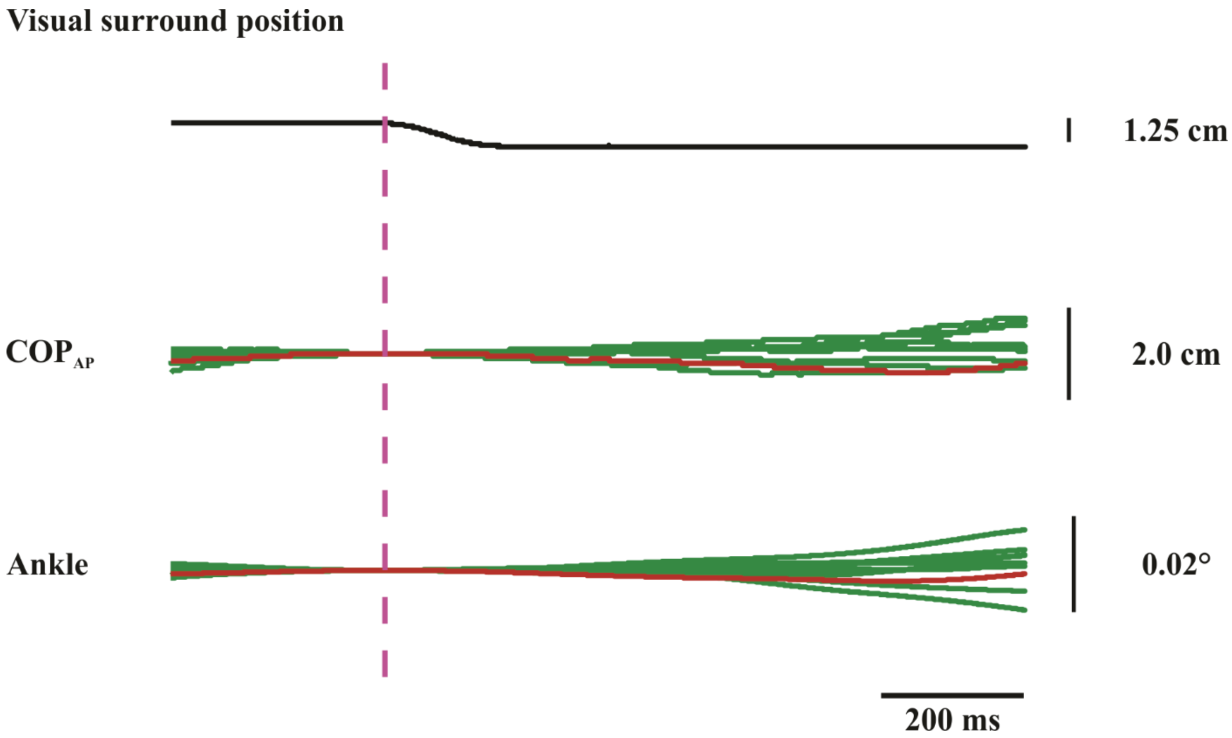

Figure A.3 Complete series of data for a single participant receiving a backward visual displacement with no-touch (BN condition). The first trace is a schematic of the visual displacement, the subsequent sets of traces are the centre of pressure anterior-posterior (COP$_{AP}$) and ankle joint angle, respectively. All traces are 1200 ms (300 ms pre- and 900 ms post-stimulus onset) windows of data. The vertical dashed line is aligned with the onset of the visual stimulus. The red trace represents the response to the very first visual stimulus and the green traces represent the responses to all subsequent exposures to the visual stimulus. On the left are the labels for each trace, and on the right are the vertical axis with units for each trace. Negative deflections represent a backwards direction in the COP$_{AP}$ trace and plantarflexion in the ankle trace.

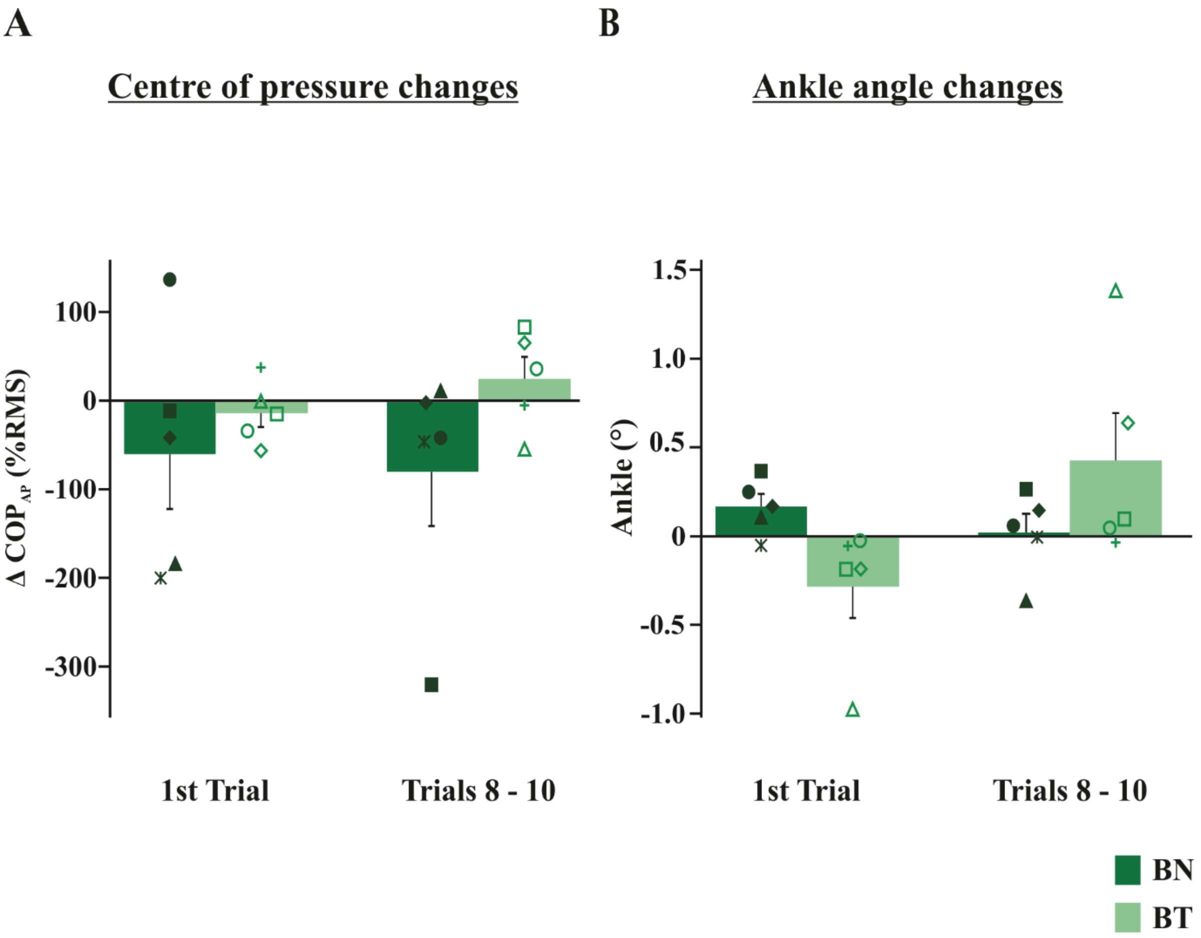

Figure A.4 Group average responses of participants receiving a backward visual perturbation. Group averages of participants in the BN condition are in dark green' the group averages of participants in the BT condition are in light green. The left set of data in each subgraph are the averages of the first trial responses; on the right is the average of all participants' averaged trials 8 – 10. (**A**) Group average amplitude for the change in the COP_{AP} trace, in % RMS. (**B**) Group average amplitude for the change in ankle angle, in degrees. Each individual marker indicates the response amplitude of a single participant in a specific group for the respective trial. Dark blue markers are participants in the FN group and light blue markers are participants in the FT group.

Discussion and Conclusions

Small, transient displacements of a visual surround, with an amplitude of 1.25 cm and peak velocity of 124 mm/s, resulted in a shift in the position of the COP_{AP} in the same direction as the stimulus. In general, this result is consistent with the findings of the main thesis, supporting the initial hypothesis. However, unlike the response to the larger 2.5 cm displacement, which showed a biphasic motion of the COP_{AP}, the response to the smaller 1.25 cm displacement lacked the early, small deflection in the COP. Instead, participants of the current study demonstrated a slow to develop, gradual repositioning of the COP_{AP}. I will argue that this suggests the induced response served to realign the head within the newly repositioned spatial frame of reference but was not likely a response that would serve to prevent or mitigate a fall. In addition, repeated exposure to the 1.25 cm stimulus and provision of a stabilizing light touch had inconsistent effects on the induced response, with some participants no longer reacting to the stimulus, while others reacted with sways in the opposite direction. I will argue that these results combined suggest the smaller visual stimulus was less effective in influencing postural orientation within the context of conflicting sensory information arising from sources indicating the body remained stable.

Visual stimuli influence the postural control system

In the present study, the small, transient, visual stimulus induced a slow to develop, and gradual monophasic shift in the COP_{AP} in the direction of the visual stimulus. The change in the COP_{AP} was paired with a concomitant change in the ankle joint. This suggests that the sway behaviour exhibited by the participants is likely a response that serves to reposition the head to align with the new position of the visual spatial reference. The gradual nature of the lean suggests the response was not likely a balance reaction aimed at preventing or mitigating a fall as pronounced displacement of the COP_{AP} required several hundred milliseconds to develop. This is largely reflective of the existing literature that demonstrates a forward pitch of the body following a forward visual displacement (Bronstein, 1986; Lee & Lishman, 1975; Lestienne et al., 1977). In particular, the small, but rapid visual stimulus used in this study seemed to replicate the behaviour demonstrated by Bronstein (1986) who demonstrated a similar slow to develop (onset of approximately 600 ms), gradual forward pitch of the body in response to a larger 30 cm visual displacement delivered over 12 s with a peak velocity of 30 mm/s . This suggests that the smaller amplitude, transient stimulus used in the present study was sufficient to activate similar postural control mechanisms as the larger stimuli used by Bronstein (1986) or in the main thesis, at least for the first trial. We suggest that it is unlikely that these postural responses are balance corrective responses. Balance reactions triggered by platform perturbations or pushes are more abrupt and faster (Diener et al., 1988; Nashner, 1976, 1977). A balance corrective reaction has clear early activation of relevant muscles around 100-150 ms following a perturbation (Cresswell et al., 1994; Diener et al., 1984). More specifically, Misiaszek demonstrated that the displacement of a light touch reference at the fingertip yields short-latency activation of the TA muscle around 75 – 120 ms, with subsequent visible changes in the COP_{AP} around 200 ms post stimulus (Misiaszek et al., 2016; Misiaszek & Vander Meulen, 2017). This further suggests that the gradual, visually evoked changes in the position of the COP_{AP} in the present study are unlikely to be balance stabilizing responses. Instead, the latency of postural responses is similar to the 192 ms and 206 ms latency of postural leans seen in Mohebbi et al. (2022) and Peterka (2002), respectively. Together with the existing literature, we suggest that the slow to develop postural leans observed in the present study are too slow to be balance correcting reactions that are involved in preventing a fall; it is more likely a postural reorientation of the body to realign with the visual spatial reference.

A key finding of the present study, compared with the findings of the main thesis, is the lack of an early propulsive-like component in the COP_{AP} displacement. The larger 2.5 cm displacement in the main thesis evoked a biphasic response in the position of the COP_{AP}, with a smaller deflection of the $COP_{AP,}$ in the opposite direction to the stimulus preceding the larger sway response. This unique component is absent in the postural responses evoked by the 1.25 cm visual displacements of the present study, which resulted in a unidirectional shift in the COP_{AP}. We suggest that the absence of this smaller deflection in the present study is due to the smaller visual displacement, which imparted a smaller motion parallax. Thus, it is likely the perceived speed and magnitude of the required postural realignment is decreased, and therefore a propulsive ground reaction force was not required. Rather, participants were likely able to subtly regulate the ongoing tension in the tonically active soleus muscle, or other leg muscles, to produce a forward lean that was gradual. This pattern of postural lean was similar to the monophasic forward lean evoked in Bronstein (1986). In other words, this was a smaller visual stimulus that required a subtle small shift in the COP_{AP}, thus a slow gradual change in posture was sufficient to realign the body.

Sensory reweighting alters visually evoked responses

In the present study, the first visual stimulus was sufficient to influence the postural control system and evoke a postural realignment in almost all the participants. However, on subsequent trials the responses were less consistent. With repeated exposure to the stimulus some participants continued to sway in the same direction, others swayed in the opposite direction, while some had little to no response. The less consistent behaviour across participants on subsequent trials suggests that the sway recorded was likely the stochastic pattern of the natural sway and not a postural response as seen on the first trial. With the larger 2.5 cm visual stimuli, participants continued to realign the posture on subsequent trials, albeit with lower amplitudes. The persistent responses from the larger stimuli in the main thesis, and the variation in the responses in the present study, suggest that the smaller visual stimulus was more easily disregarded as a false stimulus. Similarly, provision of a stable touch reference produced inconsistent responses in the present study. Participants displayed sway behaviour in both directions when touching, which we suggest is also reflective of the natural stochastic sway pattern, rather than being a result of the visual stimulus. Thus, it is likely that the addition of a stable sensory cue altered the response, and that touch was able to suppress the postural realigning behaviour following a 1.25 cm visual displacement. Together, the less consistent responses following repeated exposure and provision of touch suggest that the relevance of visual information in maintaining postural orientation has been decreased, consistent with the concept of sensory reweighting in postural control (Nashner & Berthoz, 1978; Oie et al., 2002).

Integration of sensory input is critical for postural control, with contributions arising from multiple sensory sources (Peterka, 2002). Peterka (2002) argues that the contribution of individual sensory channels is regulated, through a process known as sensory reweighting, to account for variability in the reliability and sensitivity of the sensory inputs. Oie et al., (2002) demonstrated that with simultaneous oscillations of both visual and tactile feedback, postural sway was dependent on the amplitude of the stimuli and the reweighting between the two sensory sources were non-linear. Moreover, the influence of the visual stimuli was dependent on the amplitude of the touch stimuli. It is clear that the postural control system reweights the dependence of one or more sensory systems as changes to the sensory information occur. We suggest that the lack of responses seen in the current study, when a light touch reference is provided is likely due to sensory reweighting; where dependence on the visual system is decreased when the conflicting and more reliable feedback from the somatosensory system is present. In the groups that did not receive light touch, the visual feedback from the stimulus sufficiently influenced the estimation of the gravitational reference. However, upon repeated exposure it is likely that the other sensory systems such as vestibular and proprioceptors dominated as there was a decreased weight on the visual system. A similar outcome is demonstrated by Mahboobin et al. (2005) where random visual oscillations presented between two discrete visual perturbations reduced the reliance on vision and decreased the postural response to the second visual perturbation. Mahboobin et al. (2005) suggest that sensory reweighting led to the habituation of the postural response, however similar to the main thesis, these responses were not entirely eliminated, only reduced. Thus, the literature and the present studies suggest that visual feedback is still quite potent and dependent on the amplitude of the stimulus. In the present study, sensory reweighting and a decreased reliance on visual feedback likely resulted in a habituation of the responses on repeated exposure. We suggest that the smaller amplitude visual stimuli in the present study were sufficient to influence posture, but were more easily disregarded in the presence of more reliable and conflicting sensory input from other sources.

Conclusion

In the main thesis, we had suggested that creating a false sense of head-motion through a rapid, small, and transient displacement of a visual surround evokes a postural realignment, or repositioning of the head, to the new location of the visual spatial frame of reference. The present study aimed to further investigate the influence of vision on postural control by delivering an even smaller, transient visual stimulus with an amplitude of 1.25 cm, to create a sense of self-motion. We found that this smaller stimulus evoked a more gradual and subtle lean that we suggest was a similar postural reorientation of the body that aimed to realign the posture to the new position of the visual spatial reference. However, a key finding from the 1.25 cm visual stimuli was the lack of a propulsive-like component preceding the lean, which was seen in the main thesis. We suggest that the smaller amplitude visual stimuli required a less abrupt and more gradual change in the positioning of the body, and participants likely released the tension in the lower leg muscles to shift their posture to realign with the spatial reference. We also demonstrated that with subsequent exposure to the small visual stimulus, and addition of a stable light touch reference, evoked a less consistent response with increased randomness. We suggest that due to sensory reweighting there was an increased reliance on the conflicting and more reliable sensory systems, which allowed those sensory inputs to dominate over the effect of the visual stimulus. The present study provided further evidence that allows us to more confidently suggest that vision is involved in the alignment of posture and that visual feedback can influence the estimation of the gravitational reference. Additionally, this study provides further insight into how potent visual stimuli can be on posture and the role of sensory reweighting on the postural control system. As individuals age, or acquire injuries, sensory degradation can exist which can affect their ability to reweight their reliance between sensory systems, ultimately affecting the control of balance and posture. Thus, it is important to understand how visual feedback, and the sensory systems as a whole, integrate to inform the postural and balance control systems to reduce the risk of falls for individuals with visual or sensory impairments

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