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Cortical contribution to postural control

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Statement of Originality

I declare that the work presented in this thesis is my own, with the exception of anything appropriately acknowledged and referenced.

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Abstract

More detailed knowledge of the cortical elements involved in postural control may allow us to understand complex balance disorders.

I first exposed older adults with so-called “idiopathic dizziness” to a dynamic postural task (Chapter 1) and found that postural sway was tightly linked to the subjective report of instability. Perceived instability was greater in patients compared to controls, implying a ‘distorted’ sense of imbalance.

Psychological variables have been linked to human postural control. To further explore the role of expectation in postural performance and the perception of stability, healthy young subjects were asked to complete a dynamic postural task after being primed about the upcoming perturbation (Chapter 2). Greater sway and/or higher perceived instability was observed, depending on the priming information given. These findings indicate that expectations could modulate postural control.

Subsequently, I sought to explore the potential role of the asymmetrical vestibular cortical network for postural control. Accordingly, healthy young subjects received cathodal transcranial Direct Current Stimulation (tDCS) to the left parietal cortex (Chapter 3). The degree of hemispheric dominance modulated postural control by reducing body sway proportionally to their individual dominance. These results indicate that vestibular parietal areas influence postural control and open research avenues for balance disorders.

Finally, I explored the changes in brain activity in patients with bilateral vestibular hypofunction (BVH) (Chapter 4) who had their electric brain activity measured using ElectroEncephaloGraphy (EEG) with eyes open and closed. Patients showed reduced alpha activity enhancement on eye closure, suggesting that the patients’ brains remained aroused even with their eyes closed. Further work will seek to investigate alpha reactivity as a measure of central vestibular compensation.

The body of work carried out for this thesis furthers our understanding of the underlying neural areas and processes involved in postural control and helps define the nature of neurological balance disorders.

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Introduction

1. Project description

The PhD project presented in this document is a compilation of four experimental chapters detailing investigations conducted to gather information regarding the contribution of the cerebral cortex and its processes to postural control. The role of cortical areas and elements such as emotion, expectations and cognition in objective and subjective instability are explored. Both healthy controls of different ages and patients with balance disorders were studied to examine how the elements mentioned above and possible disconnection between cortical areas contribute to balance maintenance.

The motivation for this project comes from a clinical finding regarding a group of older adults with permanent dizziness and imbalance without a known cause. The discovery of this group of patients highlighted the current gap in understanding regarding the processes behind objective and subjective postural control. Accordingly, Chapter 1 seeks to identify whether these older adults with permanent dizziness have distinctly perceived instability compared to age-matched healthy controls and patients with peripheral vestibular dysfunction. Additionally, the investigation detailed in this chapter intends to clarify the contribution of cerebral white matter disconnection, postural features, and cognitive and emotional elements to their perceived instability. Subsequently, Chapter 2 explains the investigation that was designed and executed to further explore the specific modulation that expectations and anxiety can have in subjects' perceived instability. In parallel, the investigation described in Chapter 3 intends to explain the role of cortical vestibular areas, and their lateralisation, in postural control by measuring objective and subjective instability in healthy young adults before and after the induction of cortical changes via brain stimulation. Finally, in Chapter 4, the brain activity of patients with bilateral vestibular dysfunction is studied to visualise possible changes that occur in the cerebral cortex to maintain balance when the vestibular organ input is compromised.

Although each of the chapters describes an independent investigation, they all contribute to the main aim of this PhD project, which is to further the current knowledge on the

contribution of the cerebral cortex and its processes to objective and subjective postural control.

2. General concepts

Postural control is an essential task previously thought to involve mainly automatic, lower-level motor reflexes. However, over time, evidence has shown that maintaining balance also requires the involvement of the cerebral cortex, including motor, sensitive, cognitive and emotional areas and the connections between them. Accordingly, the relevance of sensory inputs to postural control has been continuously evidenced (Alberts, Selen & Medendorp, 2019; Henry & Baudry, 2019).

Postural control requires the integration of multiple sensory inputs, including proprioceptive sensors, vision and the peripheral vestibular system. (Herdman, 2014). All of these inputs are then integrated at the cortical level.

The vestibular peripheral vestibular, including the otolith organs, Utricle and Saccule, and the three semi-circular canals (anterior, posterior and lateral) are responsible for detecting head movement in all planes (Herdman, 2014). The VIII cranial nerve then delivers the information to the central structures, where it generates postural responses accordingly after being integrated with other inputs (Herdman, 2014). The vestibular system experiences changes in its function with increased age, affecting postural control (Allen et al., 2016). Additionally, changes in the visual and proprioceptive systems and increased cognitive participation in posture and coactivation of leg antagonist pairs can lead to a poorer posture in older adults (Henry & Baudry, 2019).

3. Age-related dizziness and stability perception

Ageing comes with multi-system decline, including all of the sensory inputs mentioned above, but also muscles, reflexes and central mechanisms, all of which can impair postural control (Young & Mark Williams, 2015; Baudry, Lecoeuvre & Duchateau, 2012; Yeh et al., 2015; Allen et al., 2016) and increase the risk of falls (Sterling, O'connor & Bonadies, 2001; Ayoung-Chee et al., 2014; Tinetti, Speechley & Ginter, 1988).

Although vestibular and postural difficulties seem to be common in older adults, current testing has not been able to identify all of the functional disturbances seen in this population, particularly those affecting daily activities (Anson & Jeka, 2016).

Moreover, it is common to observe signs and symptoms of dizziness and instability in this group without a specific cause (Dros et al., 2011; Maarsingh et al., 2010; Ahmad et al., 2015; Ibitoye et al., 2021, 2022). Old people with such 'unexplained' dizziness usually lack a diagnosis and treatment options, which often leads to permanent disability (van Leeuwen & van der Zaag-Loonen, 2012).

We will define this group of patients as having "idiopathic dizziness", being a group with particular characteristics. These patients manifest permanent imbalance, dizziness, giddiness or light-headedness. Their symptoms onset is not acute, they start to notice them over a period of time, which usually add up to months or years before seeing a doctor as these are sometimes attributed to other difficulties like blood pressure or blood sugar variations. For doctors, it is difficult to get to a certain diagnosis as they usually do not show abnormalities in a typical neuro-otological assessment (clinical and instrumental). They do not experience vertigo, and their symptoms are not exacerbated by visual stimulation or changes in head position. These patients sometimes can be thought to have vestibular migraine (VM) or Permanent Postural Perceptual Dizziness (PPPD) as the cause for their problems due to the permanent characteristic of their symptoms and the normal vestibular assessment, however, they do not follow the criteria for these diagnose (Staab et al., 2017).

Due to the typical appearance of age-related white matter lesions, the dizziness and instability symptoms could be derived from the disconnection of cortical areas (Ahmad et al., 2015). Accordingly, Cerebral Small Vessel Disease (CSVD), is suggested as the basis for the dizziness and instability observed in older people.

The term 'CSVD' refers to a pathological process in small arteries, arterioles, venules, and capillaries of the brain (Pantoni, 2010). As with most vascular conditions, some of the risk factors though to be relevant in CSVD include hypertension, hypercholesterolaemia, smoking and diabetes mellitus (Shi & Wardlaw, 2016). The resulting syndrome includes clinical characteristics such as cognitive impairment such that CSVD is thought to be the

cause for more than 40% of dementia worldwide (Pantoni, 2010). This condition can be identified by particular imaging findings such as White Matter Hyperintensities (WMH).

The prevalence of WMHs has been observed at 25.9% in patients aged below 45 (Pantoni, 2010) number that raises to 50% in community-dwelling adults aged 44–48 (Wardlaw et al., 2013) and to 60–100% in subjects over 65 years old (Wardlaw, Smith & Dichgans, 2019; De Groot et al., 2002).

In the population with WMHs, the incidence of stroke, dementia, and mortality are about double of those of the normal population (Inzitari et al., 2009; Habes et al., 2016).

Evidence suggests that white matter hyperintensity load is linked to posture in older adults, especially when the sensory afferents are unreliable. SVD has also been associated with both gait impairments (Pasi et al., 2016; de Laat et al., 2010, 2011; Zhang, Yu & Wang, 2010) and cognitive decline (Pasi et al., 2016; Zhang, Yu & Wang, 2010; Pantoni & Garcia, 1995; Vermeer et al., 2003; De Groot et al., 2002) and recently has been linked with idiopathic dizziness (ID) affecting the ageing population (Ibitoye et al., 2021, 2022; Ahmad et al., 2015; Cerchiai et al., 2017). Objective postural disturbances and attendance at a healthcare centre due to dizziness have been linked to a higher SVD load (Ibitoye et al., 2021, 2022).

Previous work has linked SVD and unexplained dizziness experienced by a group of older adults; however, the specific mechanisms underpinning this relationship and whether white matter disconnection is related to distorted perceptions of instability are not entirely known.

4. Objective and subjective postural control

Subjective instability is the subject's perception of postural unsteadiness and can be measured in different situations. The cortical areas associated with subjective instability perception have not been fully identified, but it is not solely the sensory regions that are thought to be involved in the perception of body movement (Berlucchi & Aglioti, 1997).

Motor areas have been evidenced to participate in bodily consciousness, which suggests that the construction of postural instability -needed for postural control- involves a vast cortical network (Morita & Amemiya, 2016; Melzack, 1990). Additionally, integrative

cortical areas, such as the parietal lobes (Fogassi et al., 2005; Andersen et al., 1997), and in particular, the posterior parietal cortex (PPC) have been observed to be involved in corporal self-awareness and the construction of body representation (Berlucchi & Aglioti, 1997; Morita & Amemiya, 2016; Daprati, Sirigu & Nico, 2010), which is needed to subjectively rate our postural performance and generate postural adjustments. The participation of multiple cortical areas gives an idea of the complex process behind the rating of self-stability. Although objective body sway is a significant contributor to the construction of subjectively perceived instability (Castro et al., 2019; Schieppati et al., 1999a), more elements must be involved to fully understand balance disorders. When measured online, objective and subjective instability are tightly coupled, and this relationship is maintained at different ages and even in patients with balance difficulties (Castro et al., 2019b; Schieppati et al., 1999). Objective postural problems are usually easily observed in patients. However, instability perception is more challenging to evidence, especially when a vestibular disorder does not entirely explain it.

Previous studies have shown how anxiety can modulate objective instability (Adkin et al., 2008; Davis et al., 2009; Carpenter et al., 2004). Similarly, evidence indicates that instability perception will also change when a person experiences anxiety; this might be explained by an abnormal increase in sensory input gains driven by an anxious state (Cleworth & Carpenter, 2016). An alternative but not exclusive mechanism is that anxiety could also make subjects more conscious of balance processing, which in turn will result in an increased sense of instability (Ellmers, Kal & Young, 2021). Since the modulation of anxiety over postural control has been extensively evidenced (Carpenter et al., 2001; Cleworth & Carpenter, 2016; Davis et al., 2009), it is not surprising that anxiety contributes significantly to the construction of perceived instability. Accordingly, it becomes relevant to discover whether emotional and cognitive elements could also be involved in the distorted perception of instability that the previously mentioned group of older adults experience, which is a disabling (and key) symptom in this patient group.

5. The cognitive and emotional impact on objective and subjective postural control

Similar to anxiety, other cognitive elements can modify postural responses. A good example is dual-tasking, in which older subjects sway more when completing a cognitive task in addition to a postural one due to the deviation of cognitive resources from motor performance (Motealleh et al., 2021; Boisgontier et al., 2013). Another use of cognitive resources includes the information that subjects manage before facing a motor task, which will generate expectations and affect their performance. The motor plans to execute a task are usually generated using the common task characteristics and then modified according to the task's online challenges (Edwards, Fotopoulou & Pareés, 2013). Similarly, efficient balance maintenance depends on the adjustments that subjects make depending on the task requirements and their abilities (Horak, 2006). External information given before, during or after a specific performance can modify the subject's execution (Stoykov & Madhavan, 2015), evidencing the relevance of the data handled when generating motor plans, especially for complex tasks. It has been shown that subjects show reduced balance efficacy upon task repetition after being told their balance performance is low, while subjects who are informed that their balance performance is high will have increased perceived stability, despite the fact that no objective change to the actual task has actually occurred (Lamarche, Gammage & Adkin, 2011; Lamarche et al., 2009). If this is the case for motor responses, maybe the expectations and the comparison of these with actual performance can also modify the subjective perception of efficiency, i.e. stability when completing a postural task.

Expectations, however, can be created and modified using approaches other than verbal stimuli (Russell et al., 2022). Priming has been used in different modalities, all of which show the modulation of subjects' performance on specific motor tasks (Ginsberg, Rohmer & Louvet, 2012; Lamarche, Gammage & Adkin, 2011). Priming on postural control has not been extensively investigated. Studies on the effect of affective posture viewing on postural control have shown a modest difference in the displacement of the Centre of Pressure (COP) when subjects are primed with "unpleasant" pictures compared to "pleasant" ones (Stins & Beek, 2007). Interestingly, the authors found that priming with pictures of mutilation generated a shorter total sway path than other pictures, suggesting a stance immobility possibly derived from the perception of threat (Stins & Beek, 2007).

In another study, the authors observed that subliminal and supraliminal reward stimuli were effective in improving forward reach, but being the conscious reward stimuli more effective than the unconscious (Aoyama et al., 2017).

Although the effect of priming on subjectively perceived body stability and the relationship between objective and subjective measures has not yet been evidenced, the cortical processes involved in postural control and the construction of perceived subjective instability are likely to be influenced by previous information and the expectations generated by this. If patients' personal beliefs modulate their objective and subjective instability, these need to be considered when assessing, diagnosing and treating patients with balance disorders. Accordingly, it is vital to determine the extent to which emotional components modulate the faithful cortical subjective representation of objective postural cues and how this perception can be modified.

6. The cortical activity involved in postural control

Postural control and subjective perception of instability encompass essential involvement of the cerebral cortex. Hence, anything altering the cortical function could influence postural control and the conscious perception of balance.

Although postural control has been studied for years, the cortical areas associated with postural maintenance still need to be identified.

Imaging studies have identified that the parietal lobe is intimately involved in integrating vestibular, visual and proprioceptive information, three vital sensory inputs used in postural control (Pellijeff et al., 2006; Limanowski & Blankenburg, 2016). In parallel, stimulation of the left PPC with transcranial direct current stimulation (tDCS) has been shown to modulate subcortical vestibular responses (Arshad et al., 2015a; Arshad, 2017a; Bednarczuk et al., 2017), but its engagement in postural control has not been evidenced until now.

A study by Arshad et al in 2013, found an asymmetrical handedness-related down-regulation of the VOR after binocular rivalry, which was proposed to occur as a result of a disruption of the parietal hemispheric balance (Arshad, Nigmatullina & Bronstein, 2013).

Considering trans-cranial direct current stimulation (tDCS) has been observed to modulate cortical excitability through the generation of temporal changes in the local field polarity (Sparing et al., 2009; Cohen Kadosh et al., 2010), Arshad et al in 2014 used tDCS over the left and right parietal lobes to modulate excitability levels and to induce an hemispheric parietal lobe imbalance and describe the effect of this generated imbalance in the VOR (Arshad et al., 2014).

Bilateral right-anodal/left-cathodal and unilateral left-cathodal tDCS stimulation reduced the VOR slow phase velocity generated by the caloric irrigation. There was no such effect on caloric responses in the right-cathodal condition. Accordingly, this study proposed the cathodal stimulation over the left parietal cortex as the stimulation able to induce VOR modulation. The theoretical proposition is that left cathodal stimulation inhibits the left parietal lobe making it less able to process the vestibular signals and then leading to VOR suppression (Arshad et al., 2014).

The lack of modulation of the VOR after stimulation over the right parietal lobe is suggested to be due to the predominance it has over the vestibular processing (Dieterich et al., 2003), and then the increased ability to compensate the inhibition caused by tDCS.

Previous investigations using tDCS and vestibular stimulation have focused on identifying the characteristics and participation of the hemispheric difference observed in brain activity, which is proposed as the main characteristic of the brain representation of the vestibular information (Dieterich et al., 2003). By enhancing the right vestibular cortex dominance and seeing its effect on postural control, the role of the vestibular cortex in balance maintenance can be described, clarifying the cerebral cortex's participation in posture even further.

By understanding the brain mechanisms underpinning posture, the expected changes in balance performance in patients with white matter disconnection, i.e. poor cortical integration, could be illustrated. Additionally, brain stimulation and cortical enhancement could be used in the future diagnosis and treatment of patients with balance disorders and brain lesions.

7. Cortical representation of sensory afferents

Sensory afferents provide information about the body in space from the peripheral organs, which is needed to maintain balance. The proprioception, vision and vestibular input are the primary inputs on which the vestibular system relies. When a peripheral vestibular loss occurs, the peripheral information is unreliable, and balance maintenance is impaired. At this point, the central nervous system needs to reorganise to avoid permanent imbalances and falls. This is how the brain reweights the information given by the other sensory afferents (Curthoys, 2000). However, the abnormal integration of visual information could make patients more dependent on visual information (Bronstein, 2016), which becomes a relevant issue for patients with bilateral peripheral impairment.

This process still needs to be fully explained despite the significant impact of sensory reorganisation on postural control.

The study of the electrical activity in the brain has been used widely in neurology over the last decades. The analysis of these responses involves identifying waves of certain frequencies, as these have been linked with different cerebral processes. Starting from the lowest frequency range, we have Delta oscillations which have a frequency <4Hz. The delta activity has been linked to motivational processes and has also been seen to correlate positively with the amplitude of P300 component in Evoked Response Potentials (ERP) studies. It is suggested that increased delta activity could be linked with satisfaction of basic biological needs (Knyazev, 2007).

After Delta we observe Theta oscillations, with a frequency content from 4 to 7 Hz. His frequency has been related to memory and emotional regulation. Some studies have also noted this frequency related to encoding information during exploratory movements and spatial navigation (Aftanas et al., 2001).

Next, we can find alpha frequency band, with a frequency range usually placed between 7 to 12Hz. Alpha activity has been observed to be inversely related to cortical activation, and to increase significantly with eyes closed (Harmon-Jones & Gable, 2017).

Continuing with higher frequencies, there is the Beta band which frequency content ranges from 12 to 30 Hz, and has been more regularly studied in sensorimotor behaviour.

The beta band power has also been observed to decrease during voluntary movements, and bursts after the termination of the act (Nam et al., 2011).

Finally, Gamma band has its range between 30 and 50 Hz or even higher (Nam et al., 2011)r. This band has been linked to object representation. As Gamma power in increases in complex and attention-demanding tasks, it has been proposed as a neural substrate of cognitive processes (Tallon-Baudry & Bertrand, 1999).

Accordingly, electroencephalography could be a helpful tool to understand how the different brain areas work during balance tasks. The electrical activity of the brain and its changes during increasing postural difficulty has been shown to induce changes in the theta (Hülsdünker et al., 2015; Mierau et al., 2017) and alpha band power (Mierau et al., 2017; Hülsdünker, Mierau & Strüder, 2016; Edwards et al., 2018). Additionally, vision has a well-known impact on alpha band activity. Therefore, a possible interaction of this type of brain activity could occur in patients with no vestibular function and with increased reliance on vision to maintain balance. This is why understanding how cortical activity changes after a peripheral vestibular insult and the possible role of vision in this reorganisation becomes relevant as a potential diagnostic and therapeutic tool.

8. General aim

To help reduce the previously mentioned gaps in the knowledge regarding the contribution of cortical areas and processes in postural control, this PhD project aimed to characterise the cortical regions and processes involved in objective and subjective aspects of postural control.

The four experimental chapters described in this thesis provide insights into how cortical disconnection produced by Small Vessel Disease (SVD) can cause the balance disturbances and unexplained dizziness symptoms observed in older patients (Chapter 1). Additionally, the thesis provides information on the effect that expectations and emotional components have on objective and subjective instability (Chapter 2). Finally, this thesis aims to increase the understanding of how critical cortical areas related to the vestibular system and their hemispheric lateralisation contribute to postural control (Chapter 3) and what

cortical changes take place to maintain balance when the peripheral vestibular input is impaired (Chapter 4).

It is hoped that all of this information will contribute to developing better diagnostic and therapeutic tools to help clinicians and patients.

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Chapter 1

Objective and subjective instability in Idiopathic Dizziness

1. Overview

The content of this chapter is being prepared for submission to a scientific journal.

This chapter studies patients with permanent dizziness and imbalance, in whom a possible cause has not been found. In particular, it is of interest to identify whether they evidence a difference in their perceived instability compared to healthy individuals. Similarly, identifying what factors could be contributing to the construction of perceived instability in these patients also seems relevant. Accordingly, this study aimed to describe the relationship between objective and subjective instability in patients with idiopathic dizziness (ID) and to identify the influence of white matter load, emotional factors and postural control on this instability perception. Additionally, patients with peripheral vestibular hypofunction were tested in an attempt to define features specific to ID patients that could help with the diagnosis of these patients and eventually their management.

As expected, the subjective perception of instability has been shown to be tightly coupled with objective body sway. However, patients who experience constant imbalance, such as patients with ID, have a distorted sense of instability, suggesting that not all of the elements involved in this process are completely understood. Patients with ID and healthy controls completed a dynamic postural task on a moving platform and had their objective body sway and perceived instability recorded and analysed. Subjects also completed questionnaires and had an MRI scan to evidence their white matter load. These factors were included in the analysis of their postural performance and perceived instability in order to explain their contribution. Additionally, patients without peripheral vestibular function completed the postural task in order to specify which findings were specific to the ID patients and which ones could be present in other vestibular dysfunctions.

In summary, the present study will hopefully help the field to have a clearer idea of the process and factors modulating the subjective perception of instability in patients with ID, providing valuable information to develop diagnostic markers and effective therapeutic approaches in the future, and thereby helping clinicians and patients.

2. Abstract

Effective postural control involves multiple networks that perceive when a postural disturbance has occurred. Objective and self-reported postural stability are typically tightly coupled. However, a group of older adults perceive frequent dizziness and imbalance that does not match their objective (in)stability. This group will be referred to as having Idiopathic Dizziness (ID). The cause of this dizziness has not been established. A mix of subclinical postural abnormalities, anxiety, and cortical disconnection and their influence on this abnormal perceived instability is explored.

Thirty patients with ID and 30 age-matched healthy controls stood on a moving platform with their eyes covered. Sled oscillations lasting 30s were delivered at six different velocities. Their body sway was measured using wearable sensors, and subjective instability and anxiety ratings were obtained after each trial.

A subgroup of ID patients (n=28) and age-matched controls (n=24) had an MRI scan to determine their White Matter Hyperintensity (WMH) volume, number of lesions and Fractional Anisotropy.

The objective-subjective instability relationship had a logarithmic fit in all groups. The slope was significantly steeper for the ID group than for the controls, indicating a heightened perceived instability for the same body sway compared to the controls. Stepwise multiple regression showed that the objective-subjective instability slope was best explained by cortical disconnection, plus perceived (Falls Efficacy Scale-International) and objective functional balance (Short Physical Performance Battery). The stepping response analysis identified a slower stimulus velocity threshold to trigger a foot response in the ID patients than in the controls ($t=2.048$, $p<0.046$).

Patients with ID experience a distorted perception of instability, which could be related to subclinical abnormalities, such as an abnormal stepping response. Understanding the mechanisms behind their subjective performance would allow clinicians to understand this condition better and develop a more efficient treatment.

3. Introduction

Effective postural control involves multiple structures of the central nervous system (CNS), including cortical and subcortical networks (Peterka, 2002). A crucial role of these networks is to quickly and accurately detect when a disturbance to postural equilibrium has occurred, thus allowing for the appropriate postural responses to be triggered. In addition to fairly automatic postural adjustments, recent research also shows that such inputs are consciously processed and perceived (Liss et al., 2022).

Previous research has shown a tight coupling between objective markers of balance performance (e.g. postural sway amplitude) and self-reported postural stability in healthy controls and individuals with balance disorders, e.g., Parkinson's Disease (Schieppati et al., 1999; Castro et al., 2019b). This would indicate that humans are generally accurate at perceiving both the presence and magnitude of their unsteadiness. However, factors such as anxiety (Castro et al., 2019b; Ellmers, Kal & Young, 2021; Cleworth & Carpenter, 2016), the degree of attention directed towards consciously monitoring balance (Ellmers, Kal & Young, 2021), expectations about upcoming instability (Castro et al., 2022; Russell et al., 2022; San Pedro Murillo et al., 2022), and vestibular cortex dominance (Castro et al., 2019a) can alter (and de-couple) the relationship between objective and subjective instability. This can lead to 'distorted perceptions of instability' (Ellmers, Kal & Young, 2021), whereby individuals perceive themselves to be more unstable than they are.

Dizziness is common in older adults; around one-third of those aged 65 years and above are affected (Colledge et al., 1994). For many of these individuals, the dizziness cannot be readily attributed to neuro-otological dysfunction (Ahmad et al., 2015; Ibitoye et al., 2022, 2021); hence, they are often described as having 'Idiopathic Dizziness' (ID). Patients with ID have lower frontal white matter integrity, lower fractional anisotropy in the genu of the corpus callosum, and poorer structural connectivity in an extensive bihemispheric white matter network (Ibitoye et al., 2022). Additionally, increased desynchronisation and disruption of postural EEG networks have been observed in these patients (Ibitoye et al., 2021). Hence, brain abnormalities could disrupt the integration of incoming sensory information to maintain balance (Ibitoye et al., 2022, 2021). This reduced connectivity as a result of increased white matter hyperintensities (Ibitoye et al., 2022,

2021) might also lead to an increased or 'distorted' sense of instability compared to healthy individuals of the same age (Ibitoye et al., 2022). However, as patients with ID also tend to have increased anxiety and fear of falling (Ibitoye et al., 2022), these latter factors may also contribute to their distorted perception of instability.

It was hypothesised that (1) the ID patients could have a higher sense of perceived (but not actual) instability compared to the controls (i.e., a distorted perception of instability), (2) that this would be a consequence of subclinical postural abnormalities not captured by regular clinical neuro-otological testing, and 3) that small vessel disease may be a significant contributing factor, as these patients were also MRI scanned as part of a wider research programme into dizziness in old age. Given the paucity and non-specificity of the symptoms and signs in these patients, the relationship between objective sway and subjective instability was assessed to understand the mechanisms involved in the emergence of dizziness and imbalance in these individuals. Accordingly, the aims of this study were: i) to explore postural control in patients with ID and to determine whether they had distorted perceptions of instability during a dynamic balance task compared to healthy age-matched controls and young controls to assess the changes possibly related to age; ii) to explore the contribution of other factors such as general balance performance, white matter hyperintensity (WMH), and psychological factors (e.g., anxiety, fear of falling, expectations) to perceived instability; and, iii) to identify the factors unique to ID rather than those generalisable to dizziness caused by other issues by comparing the current results with a group of patients with a well-defined cause for their unsteadiness, i.e. bilateral vestibular hypofunction (BVH).

4. Material and Methods

This study was approved by the North East–York Research ethics committee and all of the subjects signed an informed consent form before any procedure.

4.1 Participants

In this study, 30 patients with ID (mean age: 77.6 ± 6.4) and 30 age-matched healthy controls (mean age: 76.4 ± 6.1) were recruited. Additionally, 12 patients with bilateral vestibular hypofunction (BVH) (mean age: 65.1 ± 14.2) with their corresponding age-matched controls (n=12; mean age: 64.8 ± 15.3) were recruited to assess how severe reduction of peripheral vestibular input influences perceptions of instability.

Patients were recruited from neuro-otology clinics, with "dizziness" as their primary concern for consultation. The ID patients were assessed by a neurologist specialist in neuro-otology who found no neurological or vestibular deficit in their clinical examination, normal peripheral vestibular function (determined by either a video head impulse test (vHIT) or Caloric testing) and no neurological disease in an MRI scan. Additionally, the patients did not meet the diagnostic criteria for persistent postural-perceptual dizziness (PPPD) (Staab et al., 2017). Healthy controls without a history of dizziness were recruited from older people groups and a community registry of older people. They were screened for neurological and vestibular abnormalities by the research team. (Castro et al., 2019b; Ibitoye et al., 2022, 2021).

4.2 Clinical testing

After the initial clinical examination, the patients with ID and their age-matched healthy controls completed the Short Physical Performance Battery (SPPB). This test measures gait speed, static balance and lower extremity function, giving a maximum score of 12 points (Guralnik et al., 1995). Low scores in this assessment are linked to poor balance performance and increased fall risk (Lauretani et al., 2019). Subsequently, participants completed a Time Up and Go test (TUG), which required them to stand from the seated position, walk 3 metres, turn around and then walk back to the chair and sit back down whilst being timed. Higher TUG times are linked to impaired balance function and increased fall risk (Browne & Nair, 2019).

Individuals also completed a "pull test" to evaluate their protective stepping responses (Luchies et al., 1994). Stepping responses following a pull test can be abnormal (i.e. more than two steps or fall) across a range of neurological disorders (Lee et al., 2013),

including SVD-related encephalopathy (Ibitoye et al., 2022). In this test, patients stand with their feet hip-width apart, and the examiner stands behind them, facing in the same direction. The evaluator pulls the patient's shoulders in a quick, strong movement to destabilise the patient. The number of steps taken after the perturbation is recorded, or "fall" is noted if the balance cannot be restored and the patient is caught to prevent a fall (Hunt & Sethi, 2006).

Vibration thresholds at the ankles and shanks were obtained as a measure of leg proprioception. The assessment was performed by a trained clinician using a calibrated 64 Hz tuning fork (Aesculap, Germany). This procedure involves subjects reporting when the tuning fork vibration is no longer felt after it has been placed at each leg's medial malleolus and tibial tuberosity. A mark on the tuning fork is read when the vibration is not perceived, which provides a vibratory perceptual threshold in amplitude arbitrary units (Bergin et al., 1995).

All subjects also completed four questionnaires to assess their symptoms (Vertigo Symptom Scale, VSS (Yardley et al., 1992)), their dizziness-related handicap (Dizziness Handicap Inventory, DHI (Jacobson & Newman, 1990)), their level of anxiety and depression (Hospital Anxiety and Depression Scale, HADS (Zigmond & Snaith, 1983)), and their concern about falling (Falls Efficacy Scale, FES-I (Yardley et al., 2005)).

4.3 Dynamic balance task

Subjects then completed the experimental dynamic balance task to test their objective and subjective (in)stability (Castro et al., 2019b, 2022). Subjects stood on a moving platform with their eyes covered whilst wearing noise-cancelling headphones. Each trial lasted 30 seconds and featured different velocities of oscillatory platform movement. The stimulus used to drive the platform was a complex waveform containing four sinewaves of different frequencies (0.18, 0.37, 0.69 and 0.9 Hz, Figure 1A), generated using custom-made software ("Arbwave", D. Buckwell). There were six different stimulus velocities: stationary (motor off), vibration (motor on, no movement), slight movement (peak velocity 0.01 m/s), small movement (peak velocity 0.05 m/s), medium movement (peak velocity 0.1 m/s) and large movement (peak velocity 0.2 m/s) (Table 1). Each of these six stimuli was repeated

twice for a total of 12 trials per subject, which were presented in a randomised order that was different for each subject. Subjects were not provided with any information about the upcoming platform oscillation velocity (Castro et al., 2019b).

Stimulus	Peak velocity
1. No movement (motor off)	0 m/s
2. No movement (motor on, vibration)	0m/s
3. Slight movement	0.01m/s
4. Small movement	0.05m/s
5. Medium movement	0.1m/s
6. Large movement	0.2m/s

Table 1

Peak velocities in m/sec for each of the six stimuli amplitudes used to drive the sled.

To measure their level of postural stability, a linear displacement electromagnetic sensor (Fastrak, Vermont, USA) was placed at the level of the C7 vertebra, which recorded the sway path (Hufschmidt et al., 1980) with respect to the platform. A gyroscope was placed on the right iliac crest to provide angular velocity postural information. Finally, copper contact plates were mounted to the soles of participants' shoes and used to detect foot lifts and steps (Figure 1C). The data was recorded using custom-made software ("Acquire", D. Buckwell) sampled at 250Hz.

Subjective instability and anxiety ratings were recorded after each of the 12 trials with a 0-10 visual analogue scale. For subjective instability, 0 was "very stable", and 10 was "so unstable I would fall", and for anxiety, the scale went from 0 ("not at all anxious") to 10 ("most anxious I can be") (Castro et al., 2019b; Schieppati et al., 1999) (Figure 1B).

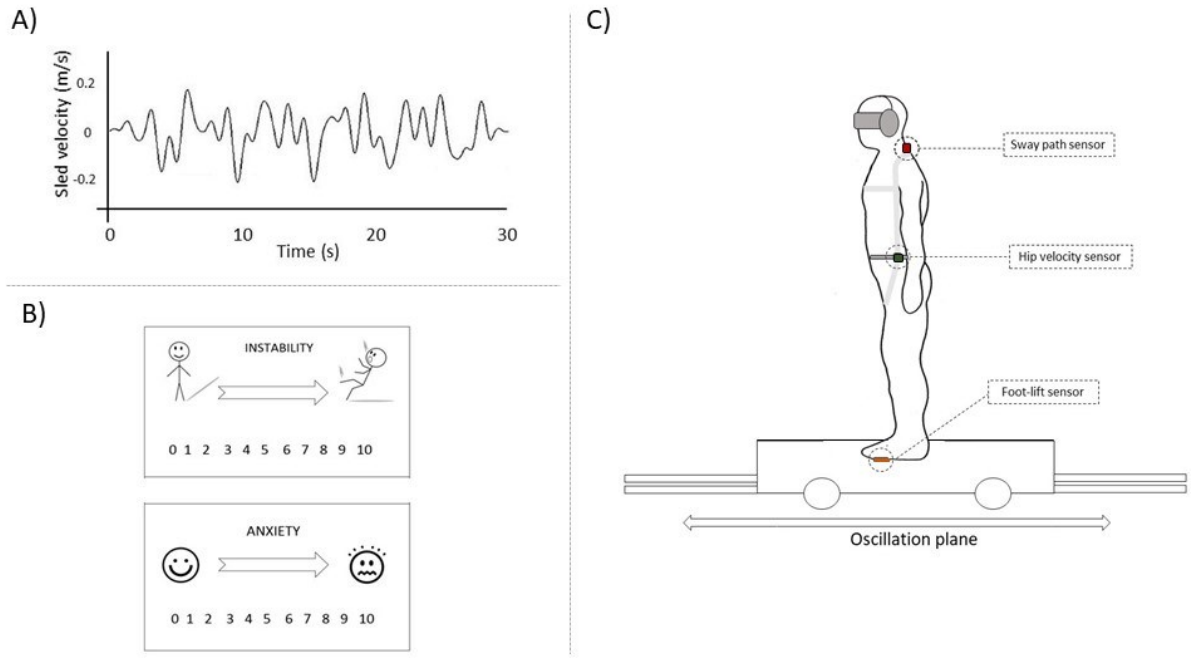

Figure 1

a) Stimulus profile used to drive the sled. The waveform was built with the combination of 4 sines of different frequencies (0.18, 0.37, 0.69 and 0.9 Hz). The stimulus represented is the large movement, with a peak velocity of 0.2 m/s and a duration of 30 sec.
b) Representation of the cartoon aid shown to subjects when they were asked to rate their subjective instability and anxiety after each trial.
c) Representation of the platform task. Subjects stood on a platform looking in the direction of the oscillation plane. Subjects wore a blindfold and earmuffs to avoid visual and auditory cues. Sensors were used to record body sway. A fast track was placed in the C7 vertebra, which provided the sway path information about the upper body with respect to the platform. The hip sensor provided the angular velocity of the hip oscillation. These two measures were correlated. Finally, copper sensors were placed in the shoe sole to detect foot lifts.

4.4 MRI testing

A subgroup of ID patients (n=28) and age-matched controls (n=24) had an MRI scan as part of a parallel study (Ibitoye et al., 2022) to determine their White Matter Hyperintensities (WMH) load and integrity.

4.4.1 MRI acquisition

Structural, FLAIR and diffusion-weighted data were acquired using a Siemens 3-T Verio scanner (Siemens® Healthcare) (Ibitoye et al., 2022). An independent consultant neuroradiologist reviewed the images to exclude other pathologies.

4.4.2 MRI analysis

Voxel-wise WMH probabilities were determined using the Lesion Prediction Algorithm, a MATLAB® toolbox applied to FLAIR images. A neurologist inspected the WMH masks (Ibitoye et al., 2022). Total WMH volume and number of lesions were used as variables.

Furthermore, fractional anisotropy (FA) data was produced from diffusion imaging data. Lower FA values in the white matter tracts suggest reduced white matter structural integrity (Ibitoye et al., 2022). The FA was used for further analysis in addition to the WMH volume and number of lesions.

4.5 Data analysis

The data analysis was performed using custom-made software ("Analysis", D. Buckwell). The sway path in cm was calculated as the total body displacement during each 30-sec trial (Hufschmidt et al., 1980). The Root Mean Square (RMS) of the hip velocity in deg/sec measured with the gyroscope was used to correlate and cross validate the sway path measurements. As in previous studies, this correlation was significant so only the sway path is reported (Castro et al., 2019b).

The contact sensors placed under the soles of participants' shoes were used to identify steps and foot lifts. Steps and foot lifts were first visually counted to compare the total number of foot responses across the different conditions and subject groups. The foot response recordings were also used to measure step generation thresholds: the four more prominent velocity peaks of the sled oscillation trace were identified and used for every subject to provide a standardised stimulus (Figure 2A). Once the peaks had been identified (6.240s, 9.900s, 15.600s and 22.480s), a dual time window was selected, starting from the

point where the stimulus velocity started, building up until the peak was reached (acceleration phase; Figure 2B) and from the peak until the stimulus velocity was 0 again (deceleration phase). If a foot response was identified on either the acceleration or deceleration phase during these windows, the onset of the foot-response was marked and the sled velocity at that point was recorded (Figure 2C).Sled oscillations backwards or forwards were separated depending on the direction of the acceleration or deceleration from each peak. Foot responses were later subdivided into “forward” and “backwards” based on the sled movement direction that induced them (Figure 2A).

For the step threshold analysis, and , previous datafrom young adult controls (mean age: 27.8 ± 5.02 years) was re-examined collected using the same methodological procedures (Castro et al., 2019b)was included. The data was anonymised and was analysed using the same methods described previously to ensure comparability between groups.

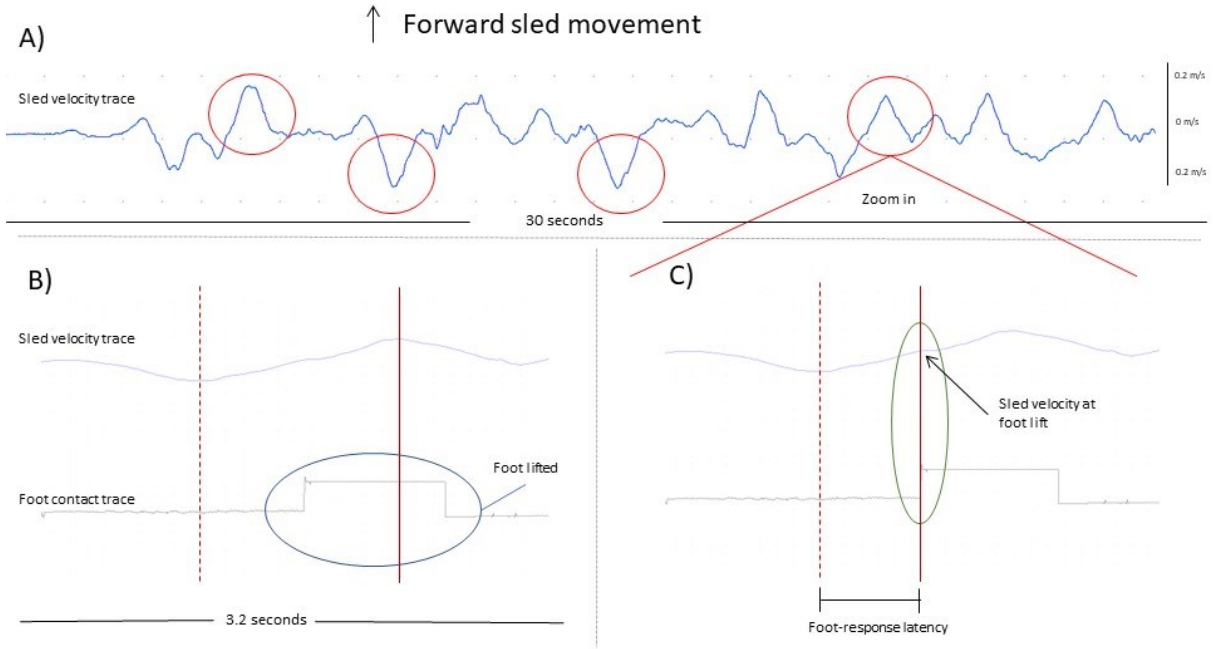

Figure 2

a) Identification of the main four stimulus peaks used to calculate the sled velocity needed to generate a foot response - either lift or step. The red circles mark the four main peaks, in which the max point was the centre of the window that was used to find the foot responses. The sled oscillation forward was seen in the acceleration phase for peaks one and four and in the deceleration phase for two and three. When separating the stimuli in forward and backwards oscillations, the foot responses found at the previously named stages were considered forward sled oscillation. Those occurring on the deceleration of peaks one and four and acceleration of peaks two and three were considered backwards oscillation occurring foot responses.
b) Pre-peak window to identify a foot response. The solid red line marks the stimulus peak; the dashed red line marks the point when the velocity began to increase before the peak. The circle shows a foot lift occurring during the established window and then being analysed.
c) The latency and sled velocity identification when the foot was lifted from the platform. The solid red line marks when the foot no longer made contact with the sled. The sled velocity at this point was recorded as the point where the foot response was generated (green circle). The time between the beginning of the velocity build-up (dashed line) and the foot response (solid line) is defined as the latency of the foot response generation.

4.6 Statistical analysis

Statistical analysis was performed using SPSS version 27. The significance for all tests was considered when the p-value was <0.05.

One-way ANOVA tests were used when comparing single values between more than two different subject groups. When comparing two groups only, independent t-tests were selected. Simple repeated measures ANOVA were selected when comparing multiple variables in the same subjects, and mixed ANOVA were selected when including "group" as a between-subjects variable. Pearson correlations were used to identify the relationship between variables. Multiple regression with curve estimation was selected when building the objective-subjective instability function curves. Later, data was separated for each subject and individual logarithmic regressions were generated to obtain a single slope value for each individual as a measure of their Objective-Subjective instability relationship. This individual slope value was used to calculate average and differences between groups and on the multiple variable analysis. Finally, stepwise linear multiple regression was chosen to identify the independent variables contributing to a dependent variable. The analysis used mean values for each group or the entire sample.

5. Results

5.1 Clinical data

The patients with ID performed significantly worse on the gait and balance related tasks (score on the SPPB) compared to the controls (t:4.854, p<0.001, Figure 3). They also had significantly higher (i.e. slower) TUG scores (t: -5.18, p<0.001) (Table 2). As expected, the ID patients also had significantly greater dizziness symptoms (VSS, t:-7.67, p<0.001), dizziness handicap (DHI, t:-10.1, p<0.001), concerns about falling (FES-I, t:-7.67, p<0.001) and generalised anxiety (HADS, t: -4.535, p<0.001) (Figure 4).

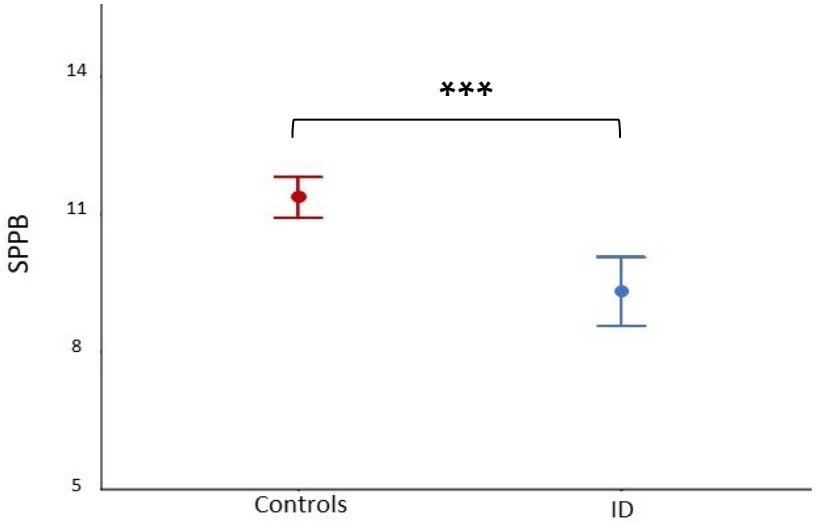

Figure 3

Mean SPPB score (0-12) for patients with ID and controls. Bars represent 95% CI. *** p<0.001.

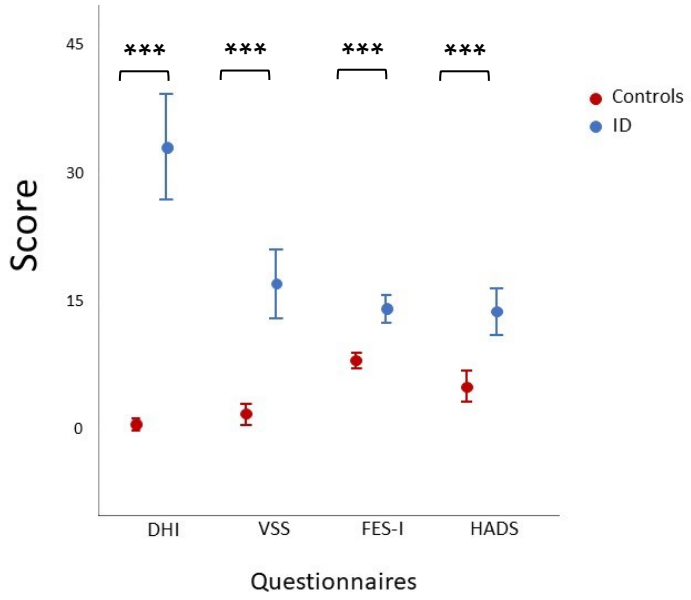

Figure 4

Mean scores for all four questionnaires, DHI, VSS, FES-I and HADS, for patients (blue) and controls (red). Bars represent 95% CI. *** p<0.001.

	Controls	Patients with ID	Significance (p-value)
Age (years)	76.38 (+/- 6.07)	77.66 (+/- 6.42)	0.427
Ankle proprioception threshold (arbitrary units)	4.6 (+/- 1.69)	3.44 (+/- 2.1)	0.024 *
Knee proprioception threshold (arbitrary units)	5.25 (+/- 1.24)	4.72 (+/-1.53)	0.151
Pull test steps (number of steps)	1.48 (+/- 0.93)	2.03 (+/- 0.88)	0.019 *
SPPB (score)	11.36 (+/- 1)	9.31 (+/- 1.96)	<0.001 ***
TUG (seconds)	9.66 (+/- 1.53)	13.77 (+/- 3.97)	<0.001 ***

Table 2

Mean and standard deviation of age, ankle and knee proprioceptive thresholds, pull test step number, SPPB score and TUG for patients with ID and controls. P-value: *<0.05, **<0.01, ***<0.001.

Subsequently, the questionnaires were correlated with the SPPB. A significant negative correlation was observed for all questionnaires, meaning that a lower SPPB, i.e. worse balance performance, was associated with higher questionnaire scores, namely more significant handicap, dizziness symptomatology, anxiety and fear of falling (DHI: r: -0.734, p<0.001; VSS: r: -0.498, p<0.001; FES-I: r: -0.772, p<0.001; HADS: r: -0.432, p<0.01. When separating the ID patients from controls, the correlation disappeared for the controls in all questionnaires. Still, it remained in the patient group only for DHI (r: -0.655, p<0.001, Figure 5A) and FES-I (r: -0.703, p<0.001, Figure 5B).

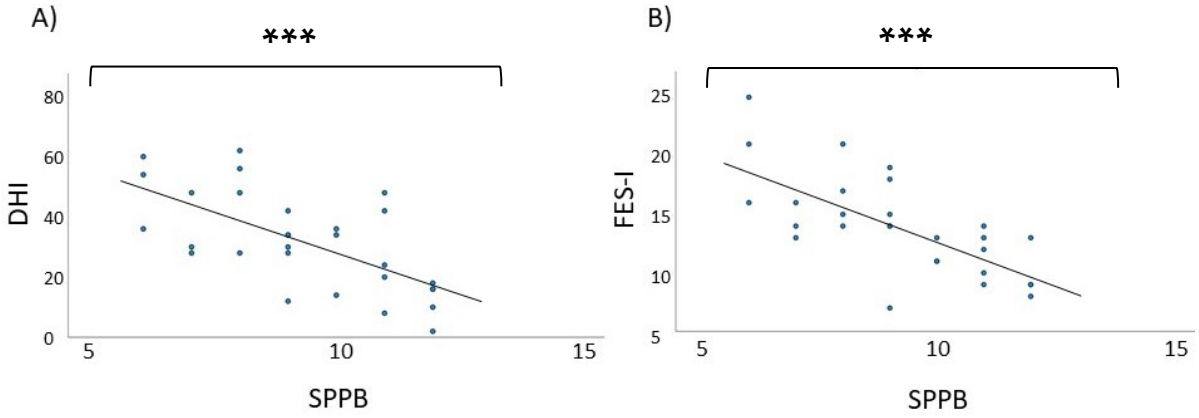

Figure 5

Correlation between (A) SPPB and DHI and (B) SPPB and FES-I, for ID patients. *** p<0.001.

The ankles and knee vibration thresholds were compared between the patients and controls. A significant difference was observed in the ankle vibration thresholds (t: 2.326, p<0.05) but not in the knees (t: 1.460, p=0.151) (Table 2).

The pull test for postural reactions was abnormal in 30% of the patients with ID and 13% of the controls. The mean number of steps taken during the pull-test (2.33 for ID and 1.48 for controls) was significantly larger for the patients with ID compared to the controls (t:-2.320, p<0.05)

5.2 Objective-Subjective instability

In the dynamic balance platform task, both objective (i.e. sway path, Figure 6) and subjective instability (i.e. visual analogue scale, Figure 7) increased significantly in all subject groups as a function of motion stimulus intensity ($F_{(5.260)}$=638.282, p<0.001 for sway path, and $F_{(5.260)}$=290.597, p<0.001 for subjective instability). However, the averaged sway path across all trials was not significantly different between the ID patients and the controls (t:-0.219, p=0.827, Figure 8).

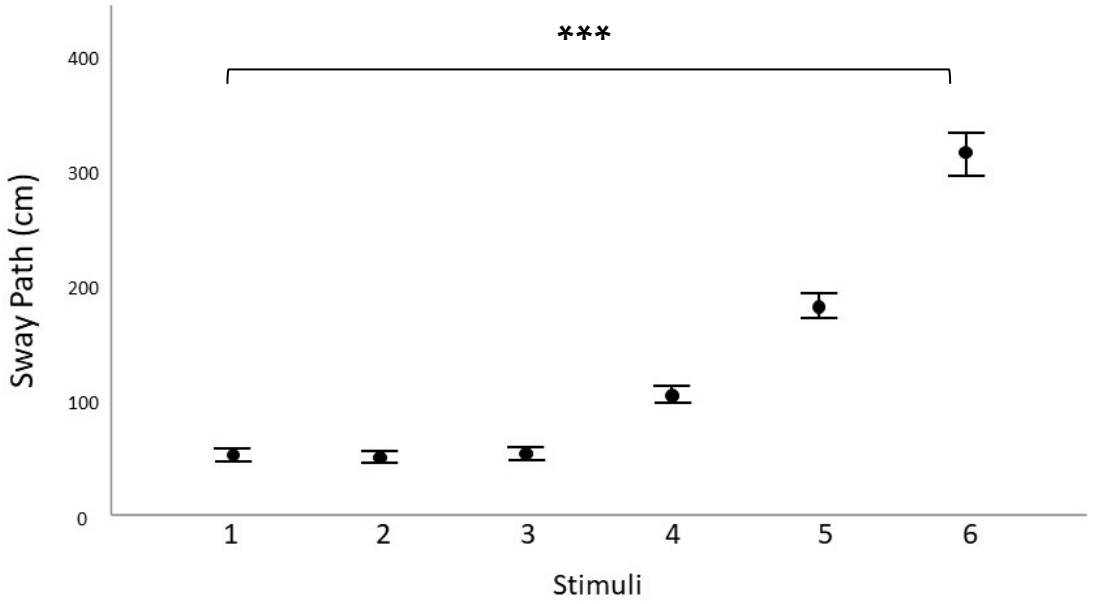

Figure 6

Mean SP (in cm) for all subjects in the different stimulus amplitudes. Bars represent 95% CI. *** p<0.001.

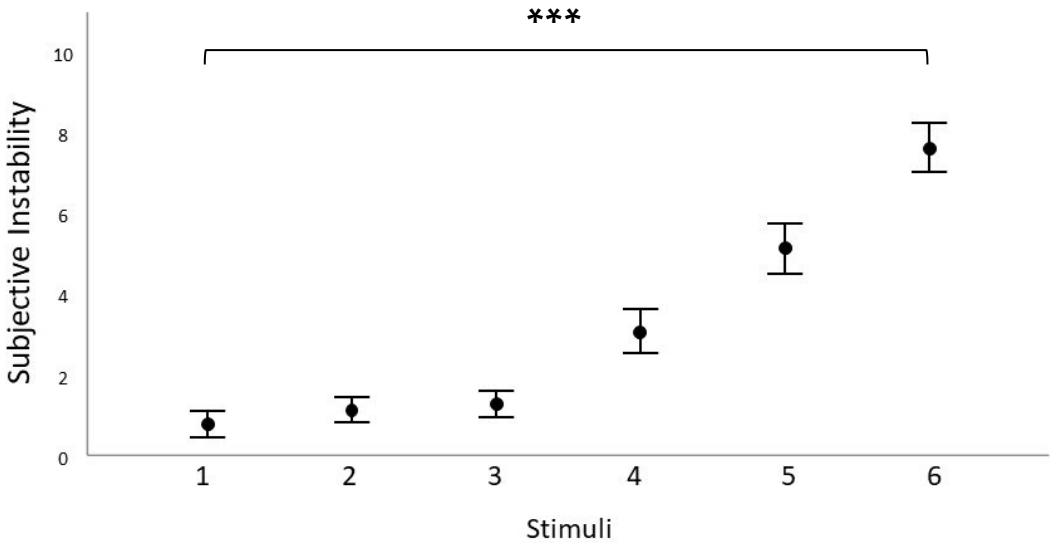

Figure 7

Mean SI score for all subjects in the different stimulus amplitudes. Bars represent 95% CI. *** p<0.001.

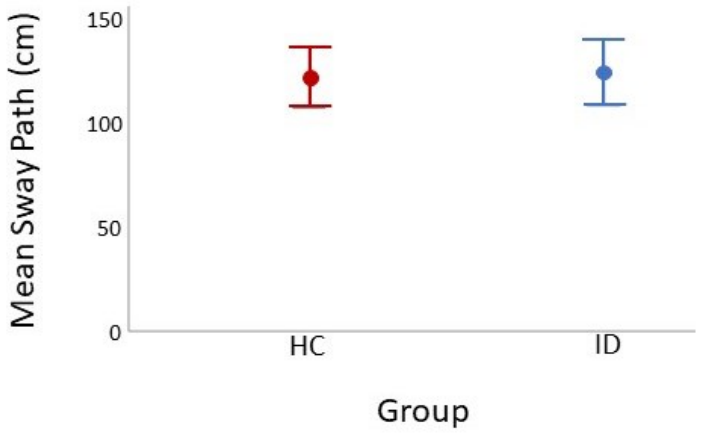

Figure 8

Mean SP (in cm) for patients with ID (blue) and controls (red). Bars represent 95% CI.

The objective-subjective instability relationship had a statistically significant logarithmic fit in the ID patients and controls (R2:608, $p<0.001$ for ID and R2:0.699, $p<0.001$ for controls, Figure 9). At this point, the slope of the objective-subjective instability curve for each subject was calculated to obtain an individual value for this relationship. The mean objective-subjective instability slope was significantly steeper for the ID group compared to the controls (t: -2.28, $p<0.05$, Figure 10), meaning that patients reported higher subjective instability when experiencing the same amount of objective instability (body sway) compared to the controls (Figure 9).

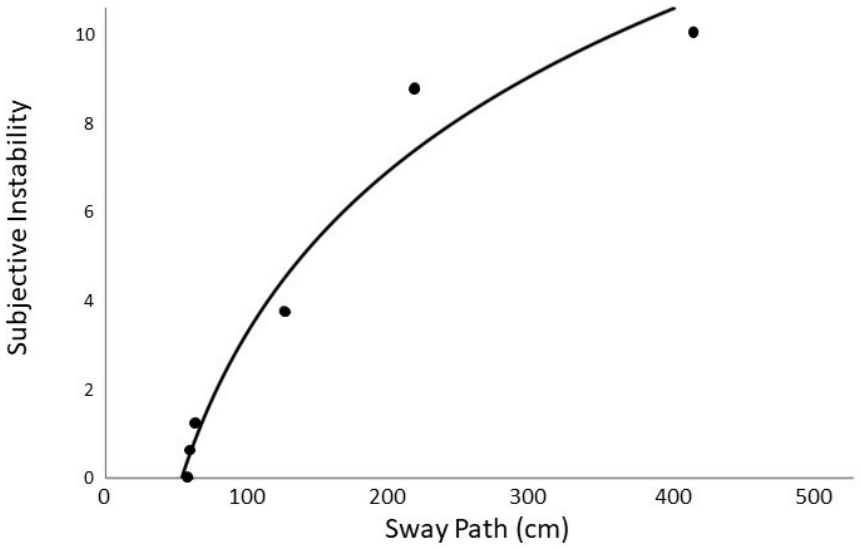

Figure 9

Representative trace of the bjective (SP in cm) versus Subjective (SI) instability relationship for one patientwith ID The best fit regression showed a logarithmic relationship.

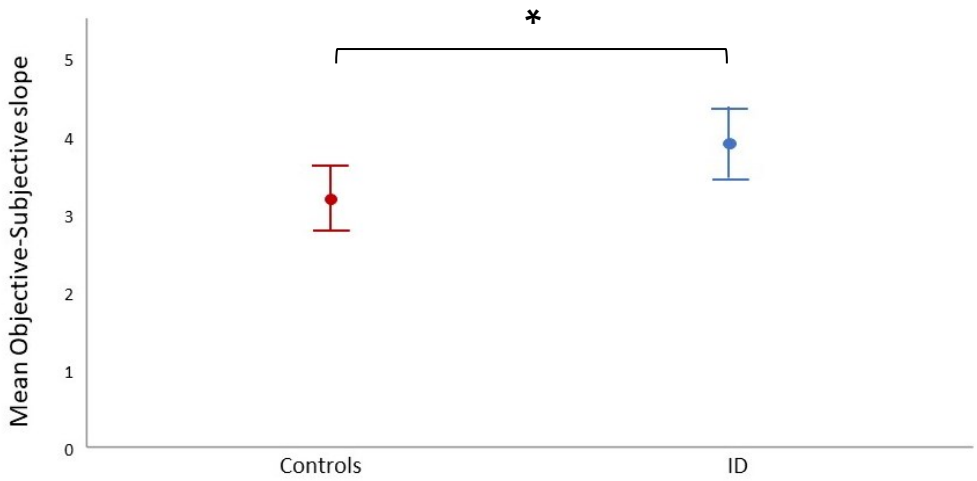

Figure 10

Mean Objective-Subjective relationship slope for patients (blue) and controls (red). The patients with ID have a higher slope compared to the controls, meaning that they reported more instability for the same amount of objective sway. Bars represent 95% CI. * $p<0.05$.

The focus was on the effect of low-velocity stimuli on balance perception because stability is least threatened during low-velocity stimuli, meaning that excessive responses

may be more likely to occur. To explore this, the stimuli were separated into two groups - the three faster and three slower platform oscillations (peak velocities (in m/s): group fast: 0.1-0.2-0.3; group slow: 0.1, 0 with vibration, 0 without vibration). Subsequently, the objective-subjective instability slope was calculated for each group on both slow and fast oscillations. Interestingly, the slope was still significantly higher for the ID group in the slow oscillation stimuli (t: -2.48, $p<0.05$) but not for the fast oscillation stimuli (t: -0.722, $p=0.237$), meaning that differences in perceived instability between the patients and controls occurred only in low-risk postural tasks (Figure 11). When looking at the mean objective-subjective instability slopes for the slow and fast oscillations, the slow slope was significantly lower than the fast slope in the controls (t: -2.150, $p<0.05$) but not in the patients (t: 0.245, $p=0.809$) (Figure 11). This suggests that the controls could modulate their instability rating based on the extent to which their balance was threatened, while the patients consistently reported high levels of instability for all stimuli, including those with low postural demands/threats to balance.

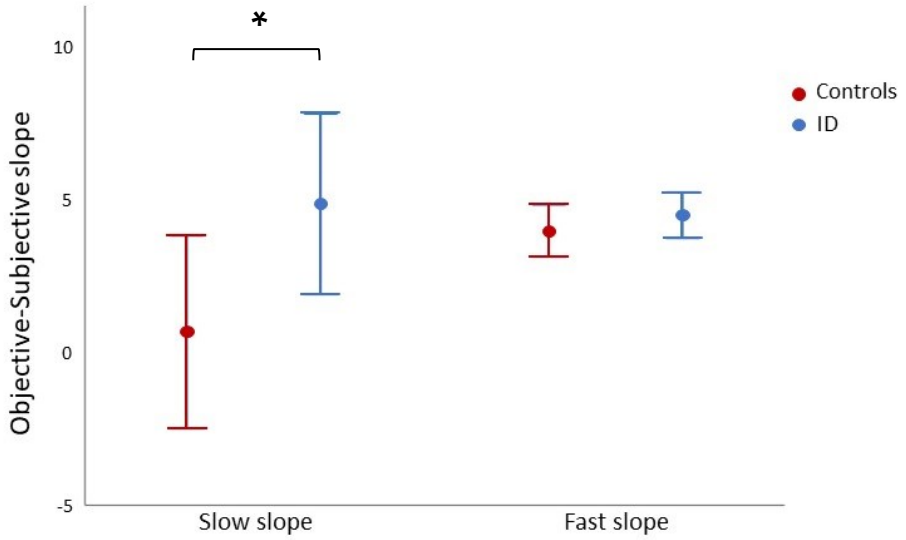

Figure 11

Mean Objective-Subjective relationship slope for the slow and fast velocity stimuli on both patients (blue) and controls (red). The controls show a reduced slope for the slow velocity stimuli compared to the high velocity stimuli, whilst the patients with ID have a high slope for both velocities despite posing a different postural challenge. Bars represent 95% CI. * $p<0.05$.

5.3 Stepping response

The stepping response analysis identified the stimulus velocity threshold that triggered a foot response (lift or step). A significantly lower sled velocity threshold was needed to induce a foot response in the ID patients than in the controls (t=2.048, p<0.046). When including the data from young healthy controls, it was possible to observe that slower velocity thresholds are needed to induce a foot response in old controls compared to young controls, and this was reduced even further in patients with ID ($F_{(2,69)}$= 8.200, p<0.001) (Figure 12).

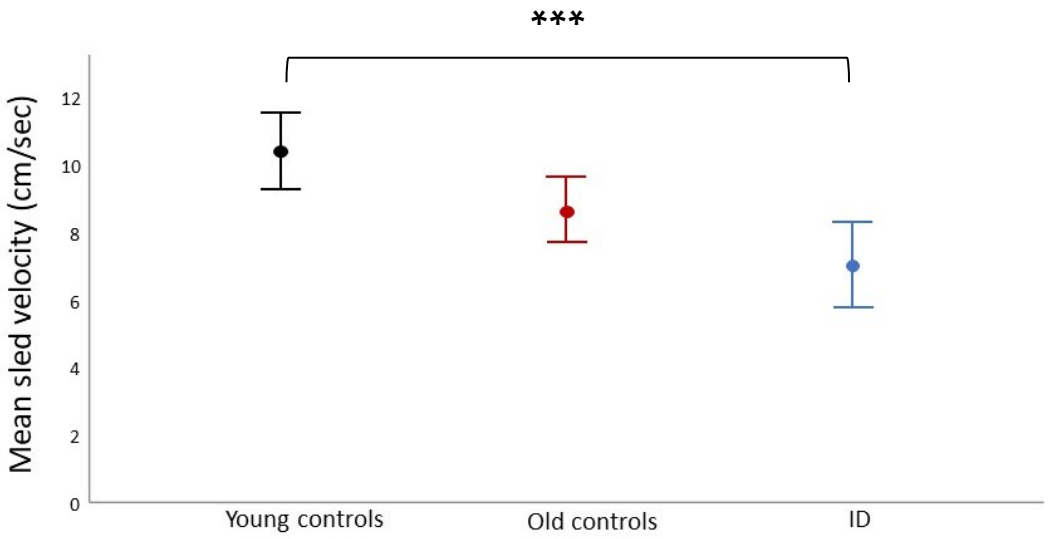

Figure 12

Mean sled velocity threshold (in cm/sec) needed to generate a foot response in patients with ID (blue), old controls (red) and young controls (black). Bars represent 95% CI. *** p<0.001.

The foot responses were later separated depending on whether they occurred during a “forward” or “backward” sled movement, which typically induce a step in the opposite direction. The significant differences between the subject groups remained only for the foot responses generated by a forward sled oscillation (t:2.266, p<0.05) and not for those generated by a backward sled movement (t:0.590, p=0.558) (Figure 13).

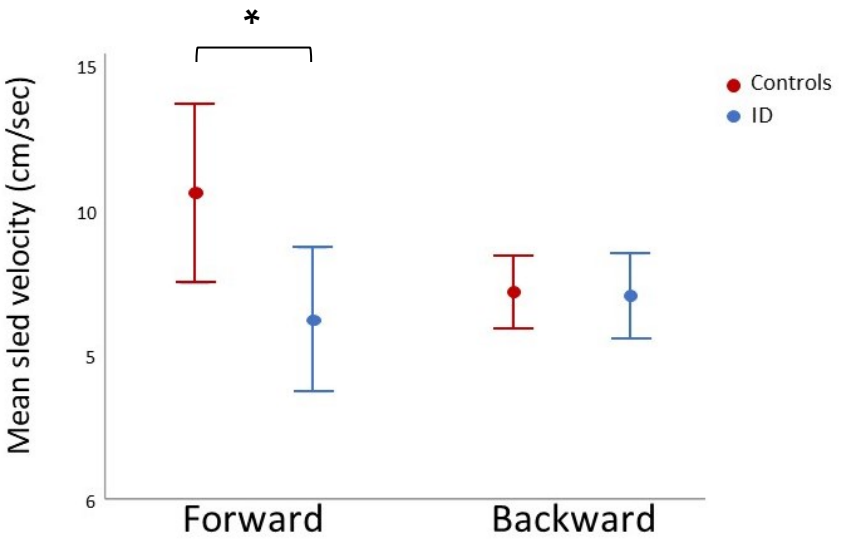

Figure 13

Mean sled velocity threshold (in cm/sec) needed to generate a foot response in patients with ID (blue) and controls (red) for forward and backward sled oscillation. Bars represent 95% CI. * $p<0.05$. The controls showed a slower velocity threshold for backward compared to forward sled oscillations, whilst patients with ID presented a reduced velocity for foot response generation in both sled oscillation directions.

The time taken to lift the feet (latency) was calculated from the onset of the acceleration or deceleration phase, according to when the foot response was generated. The foot response latency did not significantly differ between the ID patients and controls (t:-0.97,p=0.339).

The number of steps in the clinical pull test was correlated with the velocity needed to lift a foot. No significant correlation was seen between the two variables in any of the sled oscillation directions (r:-0.15, p=0.495 for forward sled oscillation, r:0.00, p=0.998 for backward sled oscillation).

5.4 Task-related anxiety

The average task-related anxiety, that is, during each oscillation trial, was significantly higher in the patients with ID than in the controls (t:-2.980, $p<0.01$, Figure 14). The subjective instability-anxiety slope was calculated for each subject to obtain an individual value for this variable. This slope was then averaged and compared between groups. There were no significant differences between the patients with ID and the controls in terms of this relationship slope (t:-0.242, p=0.810, Figure 15), meaning that although the

patients reported higher anxiety levels, these were in line with their increased subjective instability, similar to the healthy controls.

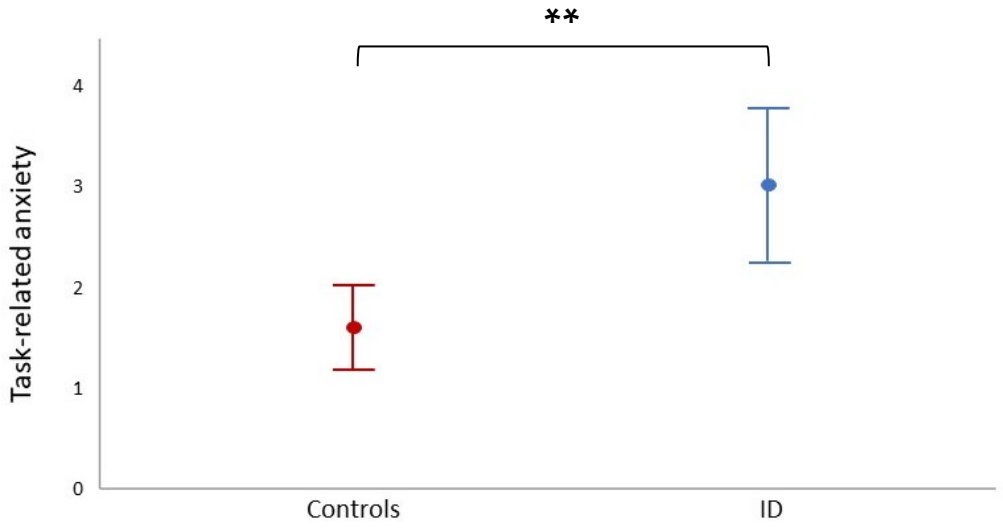

Figure 14

Mean task-related anxiety for patients (blue) and controls (red). Bars represent 95% CI. ** p<0.01.

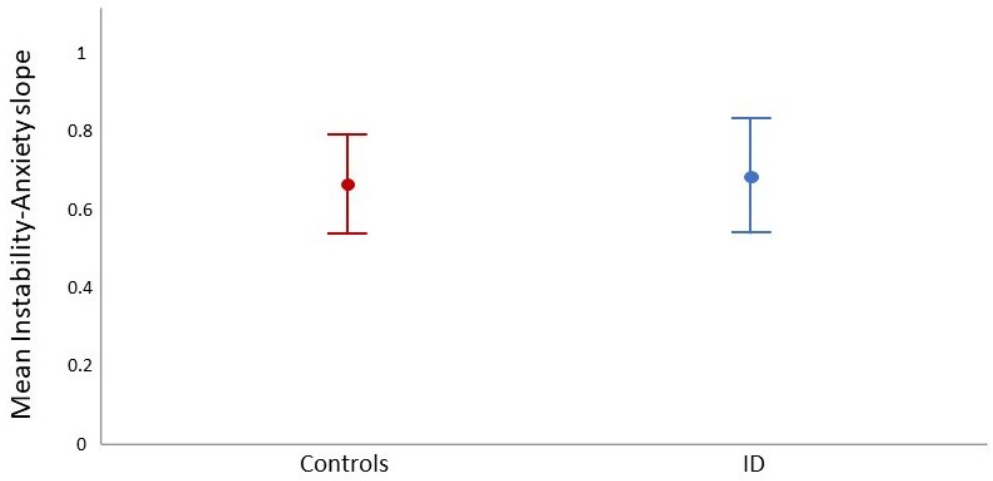

Figure 15

Subjective instability-anxiety relationship slope in patients (blue) and controls (red). Bars represent 95% CI.

5.5 Imaging data

The analysis of the number of lesions and WMH volume did not show differences between the patients and controls (t:-1.479, p=0.148 for WMH volume and t:0.195, p=0.846 for the number of WMH lesions). The difference between the patients and controls in mean FA, a measure of white matter integrity, was borderline significant (t:1.973, p=0.054). A correlation was performed between the foot response velocity threshold and the imaging data. No significant correlation was found between the stepping response (platform task) and WMH volume (r:-0.071, p=0.641), number of WMH lesions (r:0.265, p=0.072) or FA (r:-0.006, p=0.970).

5.6 Multiple variable analysis

To better understand the variables associated with the significantly higher objective-subjective instability slope observed in the ID patients, the individual patient slopes were correlated with the questionnaire scores, general balance measures and sled velocity threshold for foot response. There was a negative correlation between the overall objective-subjective instability slope and the threshold velocity for foot response only in the patient group, which was borderline significant (r: -0.356, p=0.068); this correlation was not significant in the control group (r:0.275, p=0.183). The slow and fast oscillation stimuli were separated to identify whether strong or soft postural disturbances drove this correlation. It was found that the correlation between the slope and the threshold remained only for the slow oscillation stimuli (r: -0.416, $p<0.05$, Figure 16) and disappeared in the fast oscillation (r: 0.22, p=0.38), meaning that subjects who gave a higher subjective instability score to their objective body sway in the task needed a lower sled velocity threshold to lift a foot.

A stepwise multiple regression was intended to understand which factors influenced the objective-subjective instability slope value. The assumptions to perform a multiple regression were met (i.e. the dependant variable using a continuous scale, the variables of interest having a linear relationship, homoscedasticity, discard multicollinearity, no spurious outliers, and normal distribution of residuals). After checking the assumptions, the model was build including FES-I scores, SPPB score, WMH load, sled velocity threshold for foot responses, TUG, DHI score, HADS score and WMH Fractional Anisotropy. The analysis

showed that the objective-subjective instability slope was best explained by concerns about falling (FES-I scores), SPPB and WMH volume (r2=0.475, p<0.001), suggesting that cortical disconnection, in addition to perceived (FES-I) and objective (SPPB) functional balance, are the best predictors of subjective instability during a dynamic balance task.

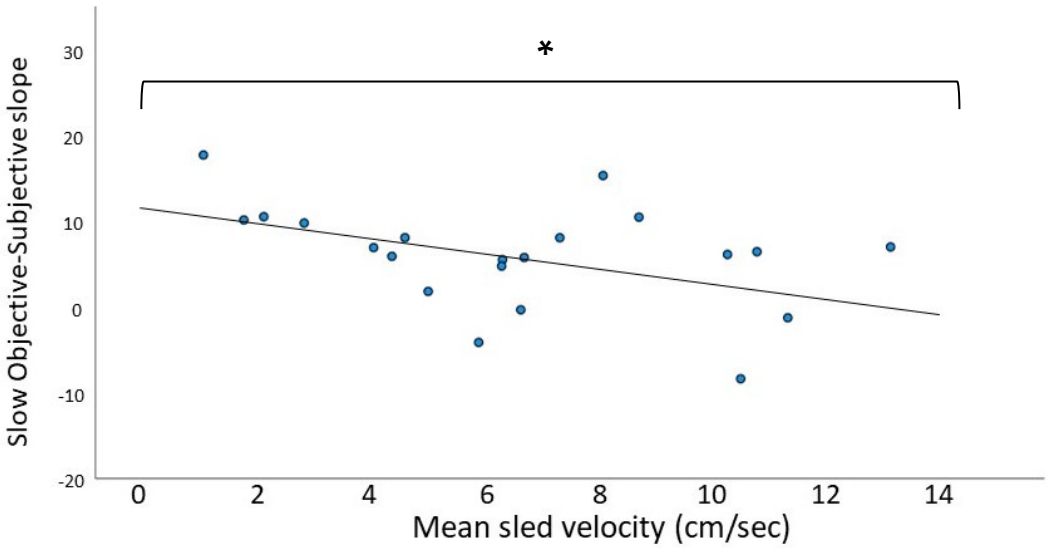

Figure 16

Correlation between objective-subjective relationship slope in the slow velocity stimuli and the sled velocity threshold needed to generate a foot lift in patients with ID. The negative correlation means that subjects with a higher instability perception during the slow velocity oscillations had a lower velocity threshold for foot response. * p<0.05.

The sled velocity threshold for foot response generation (i.e. stepping threshold) correlated negatively with all of the questionnaires (r:-0.426, p<0.001 for DHI; r:-0.366, p<0.01 for HADS; r:-0.305, p<0.05 for VSS; r:-0.286, p<0.05 for FES-I), but was most robust with DH (Figure 17), where a higher handicap was related to a reduced stimuli oscillation speed needed to generate a protective foot response. To understand the variables influencing the sled velocity thresholds, a stepwise multiple regression was run, including the objective-subjective instability slope, balance performance, imaging data and questionnaires. DHI was the only variable that was significantly associated with the velocity thresholds (r2=0.205, p<0.01), i.e. a higher dizziness handicap was associated with a lower foot response generation thresholds.

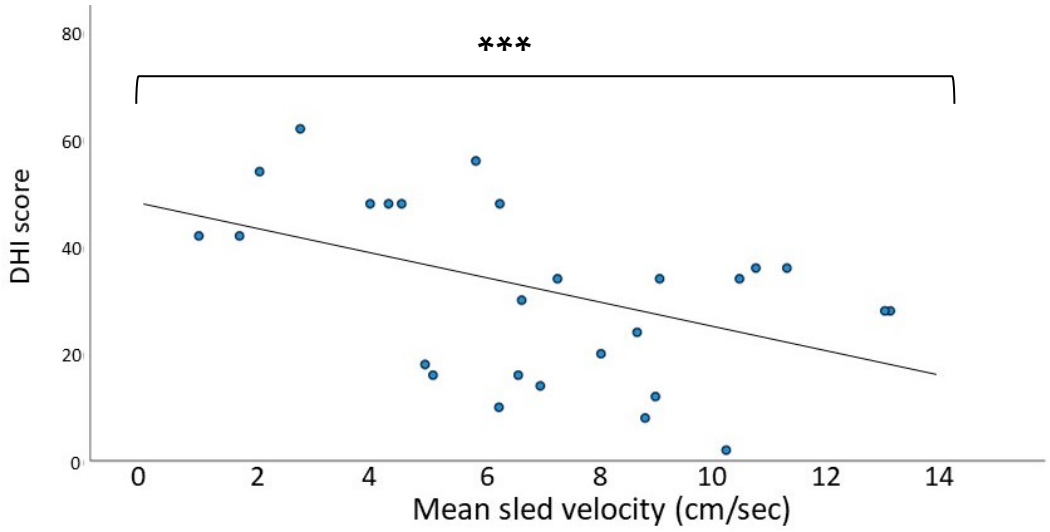

Figure 17

Correlation between DHI score and the sled velocity threshold needed to generate a foot lift in patients with ID. A negative correlation was observed, meaning that subjects with a higher perceived handicap due to their dizziness showed a lower velocity threshold for foot response. *** p<0.001.

5.7 BVH patients

To identify the factors unique to ID rather than those related to dizziness/unsteadiness caused by peripheral vestibular dysfunction, a group of patients with bilateral vestibular hypofunction (BVH) were included. The patients with BVH reported higher scores in all of the questionnaires compared to the controls, except for the HADS (DHI: t: -5.816, p<0.001; VSS: t:-3.17, p<0.01; FES-I: t:-4.788, p<0.001; HADS: t: -1.847, p=0.084).

In the platform task, the patients with BVH had a significantly larger mean sway path than the controls (t: -2.153, p<0.05, Figure 18). The objective-subjective relationship was also best fit by a logarithmic function for the BVH and the healthy control group (r2:0.627, p<0.001 for controls and r2:0.572, p<0.001 for patients with BVH). The objective-subjective instability slope was borderline significantly higher for the patients than for the controls (t: -2.082, p=0.053, Figure 19). However, upon separating the sled oscillations into two groups, slow and fast sled oscillations, the difference between the BVH patients and the controls disappeared (t: -0.871, p=0.399 for slow oscillations and t: -0.721, p=0.479 for fast oscillations).

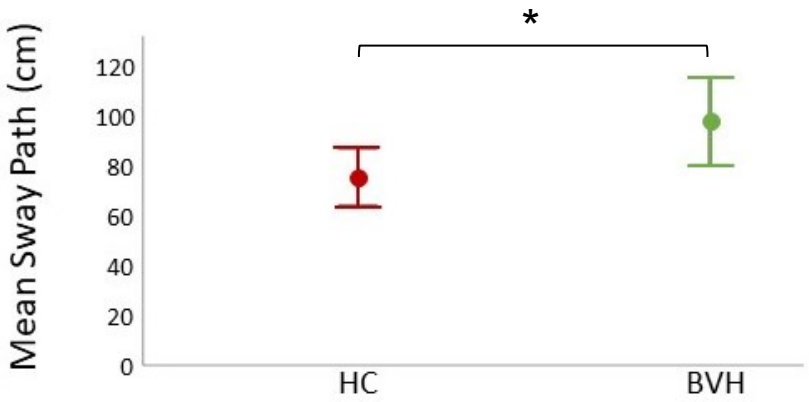

Figure 18

Mean SP (in cm) for patients with BVH (green) and controls (red). Bars represent 95% CI. * p<0.05.

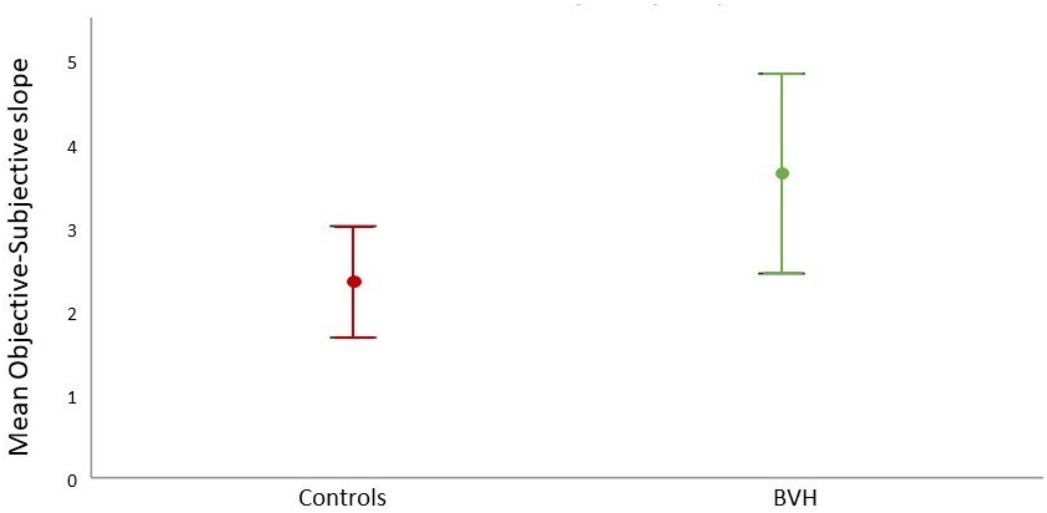

Figure 19

Mean Objective-Subjective relationship slope inpatients with BVH (green) and controls (red). The controls showed a borderline reduced slope compared to the patients with BVH. Bars represent 95% CI.

With regard to the step analysis, the sled velocity threshold needed to generate a foot response was not significantly different between the patients with BVH and the controls (t:-0.122, p=0.904, Figure 20).

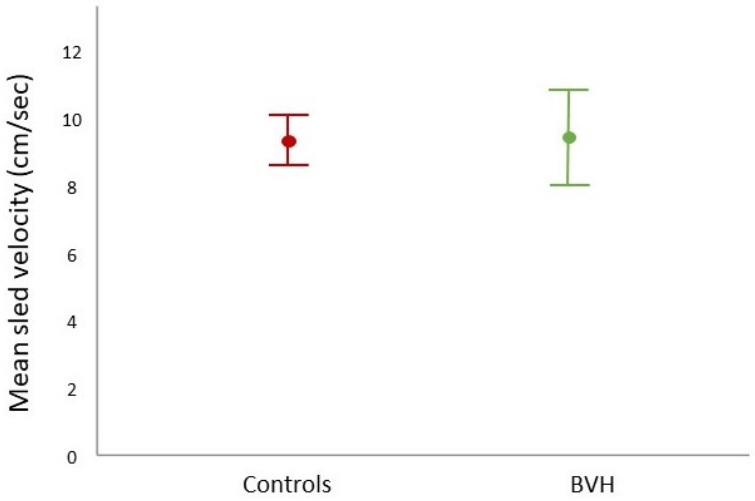

Figure 20

Mean sled velocity threshold (in cm/sec) needed to generate a foot response in patients with BVH (green) and controls (red). Bars represent 95% CI.

Additionally, this foot response generation threshold was not correlated with the objective-subjective instability slope either including all stimuli or when separating slow and fast sled oscillations (r:-0.267, p=0.427 for all stimuli; r:-0.502, p=0.139 for slow stimuli; r:-0.503 p=0.115 for the fast stimuli).

In summary, the BVH patients showed a higher sway path than the controls, unlike the ID patients. The BVH patients' objective-subjective slope was steeper than that of the controls for all stimuli and close to statistical significance. However, this difference was not maintained when the slow and fast stimuli were analysed separately, in contrast to that observed in the ID patients. Additionally, the patients with BVH did not exhibit a reduced foot response velocity threshold, in contrast to the ID patients.

6. Discussion

The present study aimed to determine whether patients with ID have distorted perceptions of instability during a dynamic balance task, and to explore how different factors contribute to such perceived instability. Additionally, it was relevant to identify the characteristics that are unique to ID that are not present in patients with peripheral vestibular dysfunction, like BVH.

The ID patients reported greater dizziness (VSS) and dizziness handicap (DHI) compared to the healthy controls, despite a lack of objective, readily-identifiable neuro-otological dysfunction. The patients also showed significantly lower ankle vibration thresholds, but exhibited comparable levels of objective imbalance (postural sway) during the dynamic balance task compared to the control group. However, the patients reported feeling more imbalanced during this task – demonstrating a decoupling between actual and perceived instability (i.e. a 'distorted' perception of instability). WMH volume (i.e. more cortical disconnection) was a factor predicting the objective-subjective instability relationship slope. This suggests that the effective integration of sensory information between different cortical areas would be required to accurately represent and subsequently perceive postural (in)stability. The reduced threshold for foot response observed in the ID patients suggests that these individuals exhibit abnormal anticipatory postural mechanisms, triggering premature and overly-cautious protective stepping responses.

6.1 Heightened instability perception

Despite complaining of persistent dizziness and imbalance, the patients with ID did not exhibit abnormal vestibular function. However, the patients with ID scored significantly higher than the controls on the questionnaires assessing symptom severity, dizziness handicap, concerns about falling, and anxiety, highlighting the need for subjective assessments in this patient group. This supports previous work that has reported general low associations between subjective complaints of dizziness and objective vestibular function tests (Herdman et al., 2020).

The objective-subjective instability relationship was strong in all of the groups studied, and followed a logarithmic function, as seen previously (Castro et al., 2019b; Schieppati et al., 1999). However, the patients with ID had significantly higher slopes in this relationship than the controls: they felt more imbalance when facing the same levels of postural threat and objective instability. However, this was true for the low – but not high–velocity/challenging stimulus. Whilst paradoxical, this finding is in line with the clinical picture in this patient group, namely persistent imbalance when standing or walking, with symptoms occurring even in the absence of a defined postural challenge. Nevertheless, if their balance is more impaired than normal subjects (e.g., worse SPPB scores), why does a challenging task not generate even higher perceived instability? One possible explanation would be a simple ceiling effect, as they reported high levels of instability with the slow oscillation stimuli. Another explanation could be that when faced with a threatening stimulus, normal subjects increase their task-related anxiety as they have a real chance of falling – much like the ID patients did during the low-challenge tasks. This could, in turn, alter (and perhaps 'distort') their perceived instability (Ellmers, Kal & Young, 2021; Castro et al., 2019b; Cleworth & Carpenter, 2016), potentially due to 'hypervigilant' monitoring of sensory input related to balance (Ellmers, Kal & Young, 2021). This is confirmed by the increased slope from the slow to fast oscillation trials observed in the controls. In this case, older subjects would raise their perception of instability and match the perception and reports provided by ID patients. In other words, ID patients adopt a constant 'high threat' assessment even during low challenging tasks, leading to anxiety and associated changes in attention and subsequent perceptions of high levels of postural instability (irrespective of the actual instability experienced). These outcomes are then similarly observed in healthy controls during conditions of high postural threat.

6.2 Reduced stimulus velocity threshold required for foot response generation

A key finding of this research is the reduced sled velocity threshold needed to generate a foot response in the ID patients compared to the controls. A relevant thing to note is that the stepping responses were identical when the platform moved forward (i.e., generated a backward step) – and were only different when the platform moved backwards

(i.e. generated a forward step). This importantly suggests that despite the ID patients having poorer functional balance (SPPB), they could display appropriate stepping responses during dynamic perturbations similar to the controls. Therefore, the early forward steps could be an overly cautious strategy, rather than purely a consequence of their physical balance.

This heightened sensitivity of the protective foot response generation system was not present in the BVH group, showing some specificity of this type of dizziness. The literature has extensively demonstrated how stepping thresholds are lower with increasing age (Rogers et al., 2003; Luchies et al., 1994). This would suggest that patients with ID have an "older" brain (i.e. poorer central integration) compared to healthy controls of the same age which could be related to WMH burden (Ahmad et al., 2015; Ibitoye et al., 2022, 2021). However, the fact that patients have a foot response with a slower stimulus does not necessarily mean that these patients will protect themselves from falls more efficiently. The abnormal response observed in the pull test supports this idea. Accordingly, patients will be more prone to lift their feet or take a step when facing postural disturbances (i.e. an 'overly-cautious' response), but these steps will not be sufficient to prevent them from falling. This supports existing work linking other overly-cautious gait behaviours to increased fall risk (Verghese et al., 2009; Herman et al., 2005).

It is proposed that these inefficient, overly-cautious postural responses observed in the present work could be a possible consequence of both poor sensory processing at the peripheral level (see between-group differences in proprioceptive threshold), as well as a potential impaired sensory integration at the cortical level (maybe derived from SVD). This would lead to a hypersensitivity towards postural instability that does not translate into an efficient postural adaptation (like a proper protective stepping response) and in turn makes patients more likely to lose balance and feel more unstable. The lack of a difference in the sled velocity needed to lift the feet when the body is moving backwards supports the idea of a disrupted (and overly cautious) "protective stepping mechanism", which would also explain the more significant proportion of abnormal stepping responses seen in these patients on the pull-test. Additionally, the patients in this study had more steps in the pull test than the controls. An increased number of steps in the pull test has also been seen in Parkinson's Disease (PD) patients compared to controls (Lee et al., 2013), indicating how a stepping response abnormality reflects a postural disturbance. Accordingly, the current

finding of the increased number of steps in the pull-test in the ID patients also supports the idea of a subclinical balance and gait difficulty in this group of patients.

However, is the lower velocity threshold for foot response generation the cause of the heightened instability perception, or is it the consequence? There is a possibility that patients with ID will generate a foot response with a reduced stimulus velocity due to their abnormally heightened perception of instability when facing postural tasks, even when they are not at risk. The fact that the sled velocity threshold was not one of the variables explaining the objective-subjective instability slope supports the direction of this process. Then, their perceived instability triggers an abnormal motor response and, considering how anxiety influences instability perception, high anxiety could modulate their postural processing. For instance, psychological factors such as a significant burden of dizziness and anxiety will lead to a more conscious (or 'hypervigilant') balance control strategy (Ellmers, Kal & Young, 2021), presumably shortening the decision time to stepping. This notion is supported by the fact that the main predictor of the stepping-response threshold was DHI.

6.3 Task-related anxiety

Whilst the average task-related anxiety was increased in patients with ID, the subjective instability-anxiety relationship slope was not. This means that ID patients show increased task-related anxiety but that such anxiety is linked to instability levels in both patient and control subjects. This finding suggests that subjective instability drives task-related anxiety and not vice versa, as anxiety followed the subjects' subjective instability rating. Could the raised anxiety levels detected during the test be a consequence of heightened perceived instability, maybe due to subclinical postural disturbances such as too early, inefficient stepping responses? Although the data points in this direction, the fact that the patients with ID had higher anxiety scores in the HADS and FES-I questionnaires is evidence of a baseline-raised trait of anxiety and fear of falling. However, the score questionnaires could also have been influenced by their postural performance, i.e. abnormal protective responses leading to a higher perceived instability and permanent dizziness sensation. Previous research has suggested that anxiety drives subjective instability (Ellmers,

Kal & Young, 2021; Castro et al., 2019b; Cleworth & Carpenter, 2016). However, the current findings imply that this relationship may be bidirectional in nature.

6.4 BVH patients

Unsurprisingly, the patients with BVH scored significantly higher in all of the questionnaires, showing how these tools gather essential information on patients' daily functions. However, the fact that the questionnaire scores were raised in both groups of patients also shows the non-specificity of self-report tools in recognising different disorders (Berge et al., 2020) and therefore the need to have specific markers to help with diagnosis and management.

The lack of a difference in the foot response velocity threshold in this group of patients compared to the controls suggests that the reduction in this threshold is not related to the peripheral vestibular organ but, rather, has a central origin. Additionally, as this velocity threshold is not related to the objective subjective instability slope in the patients with BVH, the subjective instability rating could be built with proper weighting and integration of the sensory inputs, as the instability score would reflect the increased sway that these patients presented. In contrast, the patients with ID and their permanent dizziness, which was hypothesised to be linked with WMH load, would not reflect the postural performance in their perceived instability but, in turn, would overestimate their imbalance.

6.5 Construction of perceived instability

What made these patients more prone to feeling more unstable than they were? Despite scoring lower on the SPPD, it seems unlikely that the overall level of instability experienced is driving these results, as (1) the patients were no more unstable than the controls during the task itself, and (2) their body sway levels did not correlate with the objective-subjective instability slope. Other mechanisms are likely to be at play. Higher levels of anxiety could increase the gain of sensory input, leading to the CNS having increased sensitivity for detecting minor postural disturbances (Cleworth & Carpenter,

2016). Increased anxiety could also lead to these directing more attention towards their balance, which has also been shown to contribute to distorted perceptions of instability in older adults (Ellmers, Kal & Young, 2021). WMH volume possibly contributes to the objective-subjective instability slope. This would suggest that the white matter disconnection may be contributing to the distorted perceptions experienced by the patient group, whereby the integration of the information needed to generate an accurate perception of stability would be disrupted. This idea is supported by previous work, which has identified reduced WMH volume as a key possible pathophysiological mechanism explaining ID symptoms (Ibitoye et al., 2022, 2021).

Relatedly, one possibility is that that ID patients have to rely on top-down expectations rather than bottom-up sensory input to generate perceptions of instability. In other words, if their sensory processing is impaired at both the peripheral and possibly (central) multi-sensory integration levels, these individuals would be less able to rely on incoming sensory data to generate a perception about how unstable they are. Instead, they would depend more on how stable they expect to be. Due to their high concerns about falling, these individuals are likely to have a strong expectation that they will be unstable. This results in them consistently perceiving high levels of instability regardless of how unstable they actually are – as observed in the present research and elsewhere (Ibitoye et al., 2021). This explanation may also account for the premature and overly-cautious stepping response: As these individuals would rely less on bottom-up sensory input, stepping responses could be triggered in any instance when they expect instability to occur rather than when they are actually required. This notion is in line with ideas presented in a recent framework that describes the mechanisms underpinning overly-cautious gait behaviours in older adults (Harris, Wilkinson & Ellmers, 2023) .

7. Conclusion

The patients with ID reported more subjective instability than the controls in general (questionnaires) and specific (postural task) situations. Although no evident vestibular deficits were present, patients could have had subclinical abnormalities that accounted for their symptoms, such as an abnormal stepping response, as seen in the pull-test and the sled velocity threshold for foot response. This velocity is linked to the patients' self-report; therefore, this abnormality could have been adding to the construction of their stability perception. Understanding the mechanisms behind their subjective performance would allow clinicians to understand this condition better and develop a more efficient treatment.

8. References

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Chapter 2

Cognitive and emotional influence on objective and subjective instability

1. Overview

The content of this chapter has been published in Gait and Posture (Castro, P., Papoutselou, E., Mahmoud, S., Hussain, S., Fuentealba Bassaletti, C., Kaski, D., Bronstein, A. & Arshad, Q. (2022) Priming overconfidence in belief systems reveals negative return on postural control mechanisms. Gait and Posture. 94, 1–8. doi:10.1016/J.GAITPOST.2022.02.015).

I declare that I completed most of the work presented in this chapter, including the design, data collection and data analysis, the generation of the manuscript for submission, and the revision of the final version for publication. The contribution of the co-authors to this article is as follows: Papoutselou, E. helped with the data collection, part of which was used in her Master's degree project. Mahmoud, S., Hussain, S., and Fuentealba Bassaletti, C., helped with the recruitment and data collection. Finally, Kaski, D., Bronstein, A. and Arshad, Q. are the supervisors of this PhD and in this project provided support with the conceptualisation, design, and revision of the manuscript for publication.

In the previous chapter, it was possible to observe how objective and subjective postural control is influenced by different elements, including emotional factors such as anxiety. Following this topic, the present chapter focuses on clarifying the influence of cognitive and emotional elements on postural control. In particular, the introduction of information to subjects prior to performing a postural task was used to prime participants' expectations about their upcoming balance performance. The aim of this study was to observe the effect of this priming on their objective and subjective instability.

The association between anxiety and postural control has been widely evidenced over the last decades, and therefore, the aim of this investigation was to assess whether, by managing prior expectations, it is possible to generate postural-related anxiety and consequently disrupt the well-established relationship between sway and subjective instability.

This work provides information on postural control, highlighting the importance of expectation and anxiety for the perception of self-stability.

2. Abstract

The modulation of postural control strategies and distorted perceptions of instability when exposed to postural threats illustrate the association between anxiety and postural control. This investigation aimed to test whether modulating prior expectations can engender postural-related anxiety, which, in turn, may impair postural control and dissociate the well-established relationship between sway and subjective instability. Accordingly, expectations of the difficulty posed by an upcoming dynamic postural task (moving platform) were modulated via priming. In the visual priming condition, participants watched a video of an actor performing the task with either a stable or unstable performance before they themselves proceeded with the postural task. In the verbal priming paradigm, participants were given erroneous verbal information regarding the amplitude of the forthcoming platform movement or no prior information. Following the visual priming, the normal relationship between trunk sway and subjective instability was preserved only in those individuals that viewed the stable actor. In contrast, subjects who had watched the video of the actor having an unstable performance swayed more during the second half of the task, which in turn uncoupled their objective and subjective instability. In the verbal priming experiment, an increase in subjective instability and anxiety was observed during the task performance in individuals who had been erroneously primed to believe that the sled amplitude would increase, when in fact it did not. The present findings show that people's subjective experiences of instability and anxiety during a balancing task are powerfully modulated by priming. The contextual provision of erroneous cognitive priors dissociates the normally 'hard wired' objective-subjective instability relationship. These results have potential clinical significance for the development of enhanced cognitive retraining in patients with balance disorders, e.g. via modifying expectations.

3. Introduction

The scientific foundations of the postural control system were built upon work in decerebrated animals at the turn of the 20th century (Stein et al., 1973). Later, work with patients with CNS and peripheral sensory disorders was also essential in understanding the input-output mechanisms governing postural control (Fitzpatrick, Rogers & McCloskey, 1994; Khan & Chang, 2013). Postural maintenance, however, is not solely dependent upon sensory-motor loops, as significant contributions are provided by cognitive and emotional inputs. For example, increasing the postural threat by raising the height of a standing surface generates anxiety due to fear of falling and results not only in associated changes in postural control strategies (i.e. stiffened stance) (Carpenter et al., 2004; Davis et al., 2011), but also in the modulation of perceptual ratings of instability (Cleworth & Carpenter, 2016; Davis et al., 2011).

Healthy individuals (Castro et al., 2019b; Schieppati et al., 1999) and patients with neurological conditions (Schieppati et al., 1999) are able to accurately rate their perceived postural stability (i.e. how unsteady am I?), as illustrated by the fact that they tally well with objective measures of body sway. The logarithmic function linking objective instability (sway) and subjective instability (self-ratings) is fairly consistent across groups of different ages and neurological diseases (Castro et al., 2019b; Schieppati et al., 1999).

Despite this, it is conceivable that this robust relationship between how unsteady we are and how unsteady we feel could be modulated by emotional and cognitive factors that are particular to the individual. Such modulation in healthy individuals could be attributed to the aforementioned fear of falling (Cleworth & Carpenter, 2016) or increased anxiety making balance processing conscious (Ellmers, Kal & Young, 2021). In patients with functional (psychogenic) balance disorders, however, it could be mediated by a dissociation between perceived body unsteadiness and actual (physical) body sway (Dieterich, Staab & Brandt, 2016; Staab, 2016). Such a dissociation is in line with the theoretical framework suggested in the functional movement disorder literature, whereby these patients manifest a contextually inappropriate expectation that interferes with their rational belief systems (Edwards, Fotopoulou & Pareés, 2013).

The investigation described in this chapter sought to identify whether the robust link between actual objective postural performance and subjective ratings of stability can be distorted in healthy individuals by introducing erroneous expectations (i.e. confidence) around the associated difficulty of an upcoming postural task. Accordingly, the aim was to identify whether providing erroneous prior cues via visual and verbal priming could lead to an expectational mismatch, which, in turn, would modulate postural control. Specifically, it was predicted that inducing an expectational mismatch prior to postural task performance would distort the normal objective-subjective instability relationship during random whole-body perturbations.

4. Material and methods

4.1 Participants

For the visual priming experiment (see below), 30 healthy participants (12 females, age range: 19-36yr) were recruited. A separate group of 30 individuals (17 females, age range: 18-35yr) took part in the verbal priming experiment. No subject had any current or past-history of vestibular, neurological, ophthalmological or psychiatric disorder, and this was ensured with a pre-screening questionnaire. All of the participants provided written informed consent as approved by the local ethics research committee.

4.2 Postural task

Participants stood on a linearly oscillating sled powered by two motors (Castro et al., 2019b) for 30 seconds whilst attempting to maintain steady postural control. The stimulus driving the sled was composed of 4 sine waves with different frequency content (0.18, 0.37, 0.69 and 0.9 Hz) and a velocity profile that ranged from 0 to 0.2m/s (Figure 1). The subjects were blindfolded and wore earmuffs during the 30s oscillation.

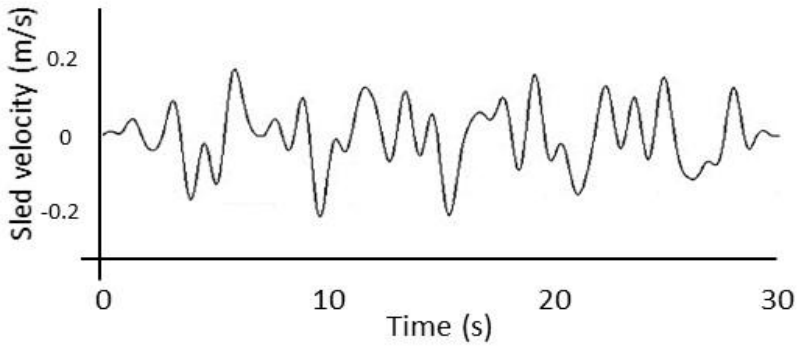

Figure 1

Stimulus profile used to drive the sled. The stimulus contained 4 sinewaves of 0.18, 0.37, 0.69 and 0.9 Hz combined with a duration of 30s resulting in pseudorandom perturbations with different maximum velocity. In this case, the trace corresponds to the stimulus used in Experiment 1, which matches the sled amplitude “Large” from Experiment 2.

4.3 Objective and subjective measures of body sway

During the task, objective measures of body sway were collected using three sensors: a Fastrak electromagnetic (Vermont, USA) sensor placed on the C7 vertebra to record linear displacement of the upper body with respect to the platform (Patel, Kaski & Bronstein, 2014; Reynolds & Bronstein, 2003); a gyroscope placed on the right iliac crest, which recorded the angular velocity of the hip movement, and contact sensors placed on each foot sole to signal foot lifts off the platform (Figure 2).

The subjective instability measure was collected immediately after the 30s oscillation with a Likert scale that ranged from 0 to 10, with 0 being “completely steady” and 10 corresponding to “so unsteady I am about to fall” (Castro et al., 2019b; Schieppati et al., 1999). Similarly, a task-related anxiety measure was obtained using a similar scale, for which 0 was “not anxious at all” and 10 was “as anxious as I could be” (Figure 3).

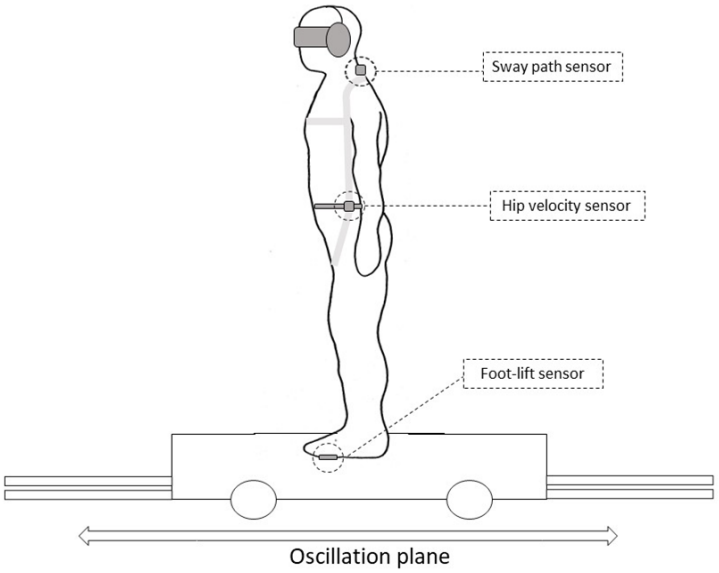

Figure 2

Schematic representation of the postural task. Subjects stood on the platform parallel to the rail i.e. the sled oscillations were perceived as back and forth. Subjects wore a blindfold and earmuffs to avoid distractions and a safety harness to prevent possible falls. Three body sensors were used to record body sway: a Fastrak on the C7 vertebra to record Sway Path, an Accelerometer on the right hip to record Hip Velocity, and copper strips on each foot sole to record foot lifts from the platform.

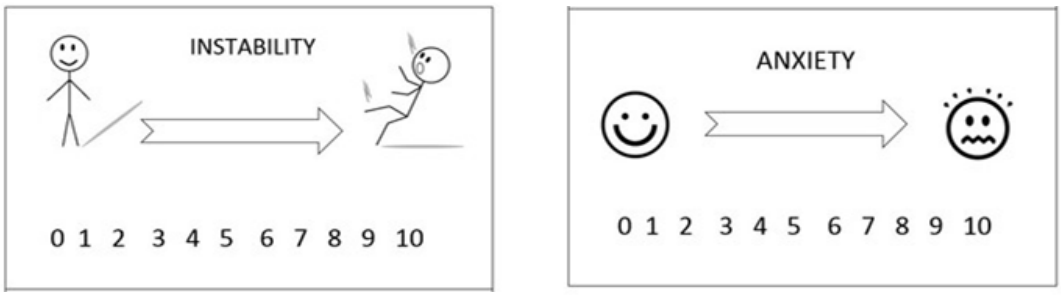

Figure 3

Cartoon aided scales to measure Instability and Anxiety. Both sheets were shown to subjects each time when asking their subjective rating.

4.4 Priming paradigm

4.4.1 Visual priming

Participants watched a video of an actor performing a postural task, which they subsequently performed (as described previously). Participants were randomly allocated to either the “SteadyVideo” group (participants watched a video of the actor performing the task with stability) or the “UnsteadyVideo” group (this group watched the same actor performing the task unsteadily). Both videos were identical in all other parameters apart from the actor’s postural performance (Figure 4).

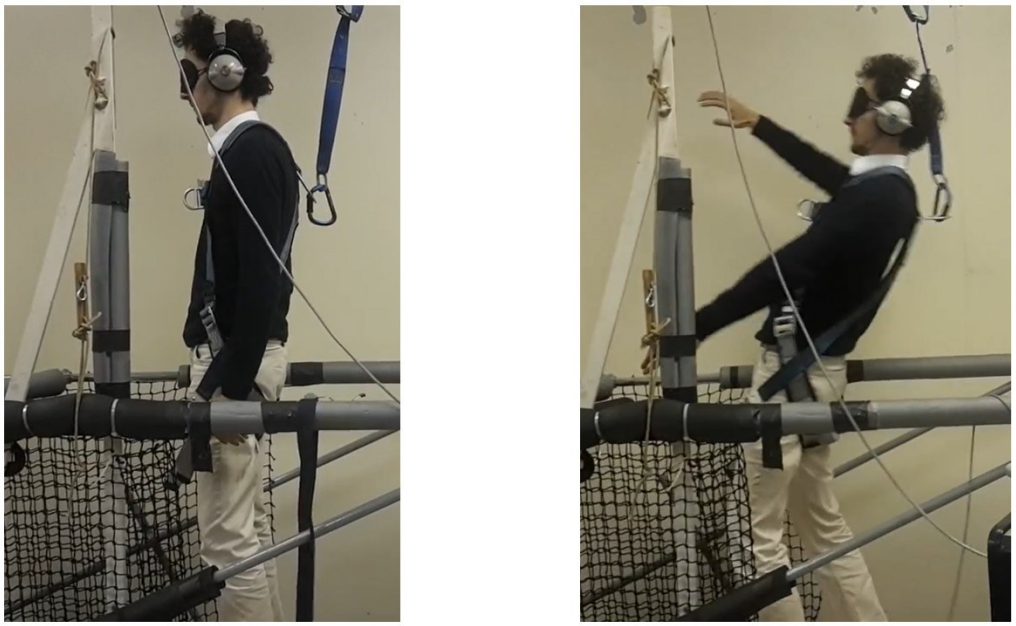

Figure 4

Screenshot of the videos shown to participants. The left image details the “Steady” video, where the actor performed the task with good stability. The right image details the “Unsteady” video, which showed the actor performing the task very unsteadily.

4.4.2 Verbal priming

In the case of the verbal priming experiment, participants were given either erroneous information or no information regarding the amplitude of the upcoming motion stimuli. The sled oscillations were grouped into two conditions: “with”, or “without” prior

information. In the “with information” condition, the information given to the subjects classified the stimuli amplitude as either “Small”, “Medium” or “Large” but, critically, all trials had the same objective amplitude (maximum velocity of 0.1 m/s). In the “without information” condition, subjects were not given any verbal cues about the trial, but critically this time the sled movements did actually have either a “small amplitude” (peak velocity of 0.05 m/s), a “medium amplitude” (peak velocity of 0.1 m/s) or a “large amplitude” (peak velocity of 0.2 m/s) perturbation. All of the conditions and stimuli were randomly presented to the subjects.

4.5 Questionnaires

In order to have a baseline measure of the subject’s anxiety, the Trait component of the State and Trait Anxiety Index was used (STAI), (Spielberger, Gorsuch & Lushene, 1970). This questionnaire contains 40 statements that assess an individual’s tendency towards anxious characteristics (e.g. “I feel nervous and restless”). Subjects are asked to rate each sentence on a scale of 1–4 (with 1 = never, 2 = sometimes, 3 = very often, 4 = always) giving a possible score range of 40-160 points.

4.6 Experimental protocols

The flowcharts for the two experimental protocols can be seen in figures 5 and 6.

For the visual priming experiment, all participants initially completed the STAI questionnaire and subsequently watched one of the two videos of the task they would be completing (Either StableVideo or UnstableVideo). After watching the video, all subjects were asked about their anxiety towards the task with a Likert scale from 0 to 10 (Figure 3). This anxiety measure aimed to identify the immediate effect of the video and was used as a pre-task anxiety measure. Subjects then performed the postural task (all subjects with the same motion stimulus amplitude, peak velocity of 0.2 m/s). After the postural task, their anxiety was measured again using the same Likert scale, corresponding to their post-task anxiety. Additionally, their perceived instability during the task was registered using a Likert scale (Figure 3)

For the verbal priming experiment, after completing the STAI questionnaire participants were informed that they were about to experience different magnitudes of sled oscillations and that prior information regarding the oscillation amplitude was going to be provided only for some of the trials. Subjects then completed 12 trials (6 trials x2) of the postural task with 3 different sled amplitudes- as explained previously and detailed in Figure 6. Finally, after each trial, the subjective instability and state anxiety measures were assessed using the same 0 to 10 Likert scales (Figure 3).

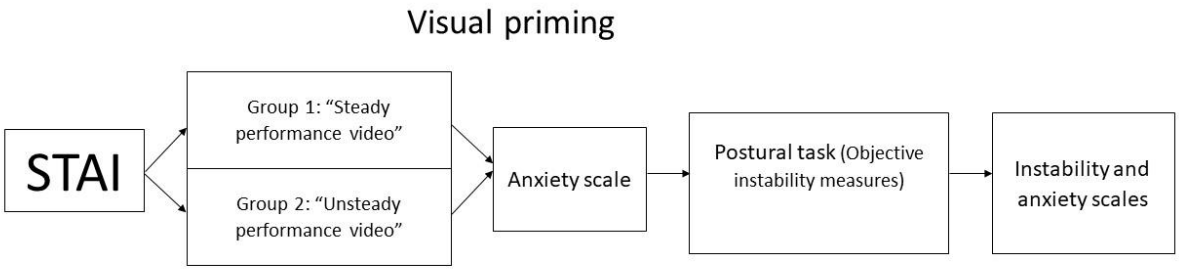

Figure 5

Flowchart of the Visual priming experimental protocol (Experiment 1).

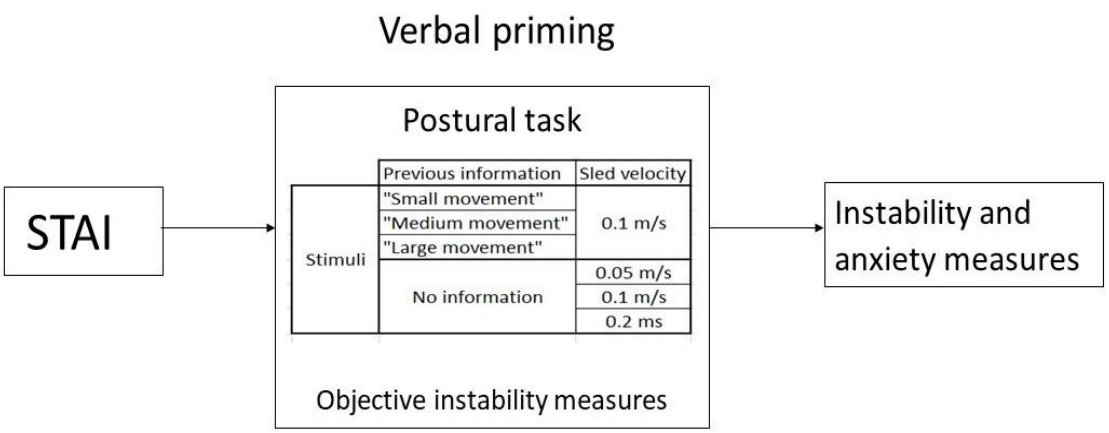

Figure 6

Flowchart showing the procedures for experiment 2, Verbal priming.

4.7 Data analysis

The variables used during the analysis to confirm the body movement were sway path, defined as the total body displacement with respect to the platform in cm, during each 30sec trial, hip velocity in deg/sec, and foot lift count in natural numbers. Anxiety and instability were recorded as a value between 0 and 10, with half points permitted.

4.8 Statistical analysis

Statistical analysis was performed using SPSS version 24. Spearman correlations were used to investigate the relationship between the variables, and regression models with curve estimation were used to analyse the objective-subjective instability function, as performed in previous studies (Castro et al., 2019b; Schieppati et al., 1999). To compare one measure between two independent groups, an independent t-test was used. For more than two groups, a one-way ANOVA was implemented. Finally, repeated measures ANOVA were used to compare paired data.

5. Results

5.1 Visual priming experiment

The subjective instability scores recorded after performing the task were similar for the SteadyVideo group and the UnsteadyVideo group (mean scores= 5.2 for SteadyVideo group and 4.7 for UnsteadyVideo group) (independent t-test,$p=0.43$). With respect to the state anxiety scores, after watching the video (pre-task), no differences between the two groups either before ($p=0.23$) or after the performance of the task were found (t-test $p=0.38$). However, a main effect of change in anxiety was seen between the pre and post task performance in a mixed ANOVA (main factor: anxiety score change; between subjects factor: video) ($F_{(1,28)}=32.5$, $p<0.001$), with the change being significantly larger for the SteadyVideo group ($F_{(1,28)}=5.1$, $p<0.05$, Figure 7). Hence watching the steady video before performing the task increased anxiety levels during the task, as reflected in the post-task measurement.

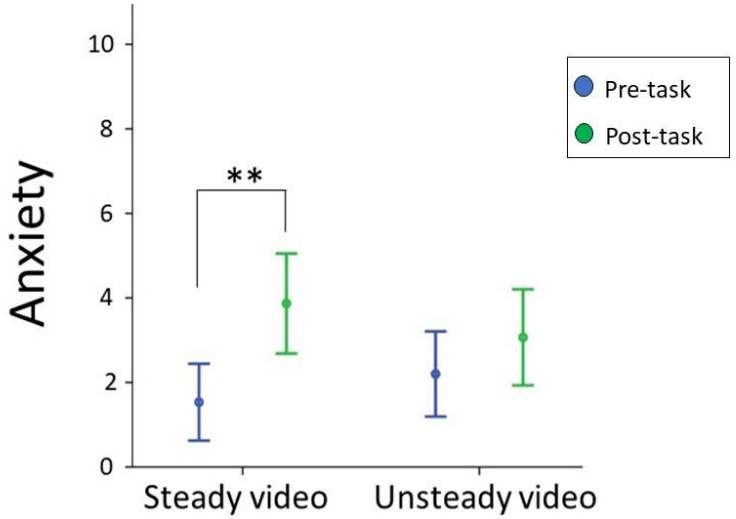

Figure 7

State anxiety measures before and after performing the postural task for subjects who watched the Steady and Unsteady videos. Blue represents the pre-task measurement (but after watching the video) and green denotes the anxiety ratings after experiencing the task. The circles represent the mean values and the bars represent the error. A significant increase from pre to post task anxiety is observed only in the group that watched the Unsteady video. Bars represent 95% CI. **p <0.01.

The objective measures of body sway (sway path, hip velocity and foot lift count) correlated strongly with each other (p<0.01 for all correlations), as expected. Accordingly, for all of the further analyses, the sway path was used as a single representative parameter, to ensure consistency with previous studies (Castro et al., 2019b).

Measures	Correlation coefficient (r)	p-value
Sway path – Hip velocity	0.63	<0.001
Sway path – Foot-lift count	0.63	<0.001
Hip velocity – Foot-lift count	0.50	<0.01

Table 1

Pearson correlations of the measures of body sway. All pairs are significantly correlated.

There was no significant difference in sway path between the two video groups (mean values= 283.02 cm for the SteadyVideo group and 318.83 cm for the UnsteadyVideo group, p=0.21, Figure 8A). However, visual inspection of the data indicated a sway effect as a function of time; therefore, the 30s recordings were separated into two epochs of 15s each (early and late). This revealed a significant difference between the early and late

epochs only for the UnsteadyVideo group; namely a sway increase during the last 15s of the task compared to the first 15s (Repeated measures ANOVA, sway path change as main effect ($F_{(1,28)}$=16.93, p<0.001)). The SteadyVideo group showed similar sway values during both epochs (Figure 8B). Hence, watching the unsteady video appeared to induce a late increase in sway during the task, as confirmed in the next paragraph.

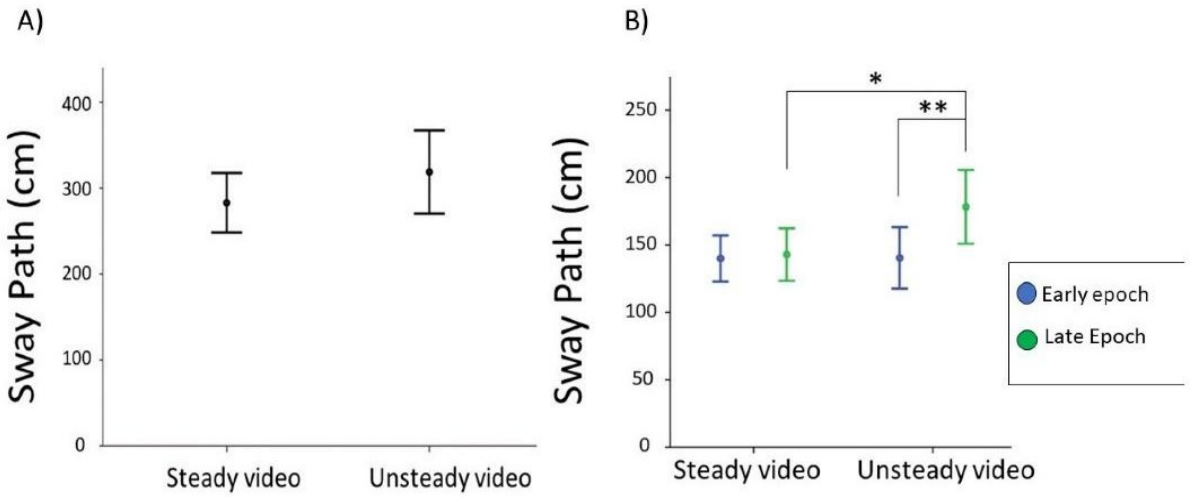

Figure 8

A) Sway path (in cm) recorded during the postural task for subjects in the Steady video and the Unsteady video groups. B) Sway path for early and late epochs (15s each) for both video groups. There was a significant increase in Sway Path from the early to the late epoch only in the group that watched the Unsteady video, making the late epoch in this group significantly different from the late epoch in the Steady video group. Bars represent 95% CI. ***p <0.001.

In order to identify whether the change in sway path between the two epochs was independent of priming, previous data (identical task, similar age) without visual or verbal priming was analysed (Castro et al., 2019b). A one-way ANOVA showed that the change observed in the UnsteadyVideo group was significantly different to that in the SteadyVideo group and indeed to subjects previously tested without any priming (p<0.01, Figure 9). This indicates that the observed difference in sway between the first and second epochs is attributable to viewing the video with the unsteady performance. Critically, participants in the SteadyVideo group were matched for baseline demographics (age, sex) or trait anxiety scores (mean score= 40.33/160for steady group and 40.27/160 for unsteady group) to participants in the UnsteadyVideo group.

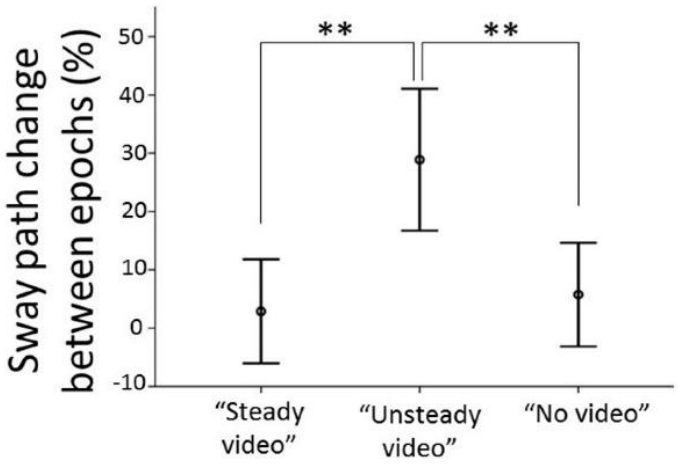

Figure 9

Sway path percentage change between first and second epochs in the groups that watched the Steady and Unsteady videos and a third group of subjects who did not watch a video. A significant increase from the early to the late epoch was observed in the group watching the Unsteady video compared to the other two groups. The circles represent the mean values, and the bars represent 95% CI. **p <0.01.

To investigate the objective-subjective instability relationship, which, as mentioned previously, is typically coupled (Castro et al., 2019b; Schieppati et al., 1999), the objective sway path measures were correlated with subjective instability scores in both video groups. The strong correlation normally present between objective and subjective instability measures was fully preserved in the SteadyVideo group (r:0.53, p<0.05, Figure 10A) but, critically, was absent in the UnsteadyVideo group (r:0.27, p=0.447, Figure 5B). Furthermore, as a strong relationship has also been observed between subjective instability and anxiety (Castro et al., 2019), whether this association was disrupted by video viewing was tested. A strong positive correlation in the SteadyVideo group (r:0.767, p<0.01, Figure 11A) was seen, but there was no such relationship in the UnsteadyVideo group (r:0.434, p=0.106, Figure 11B). Hence, watching the unsteady video disrupted the normal relationship between subjective instability vs objective instability and anxiety.

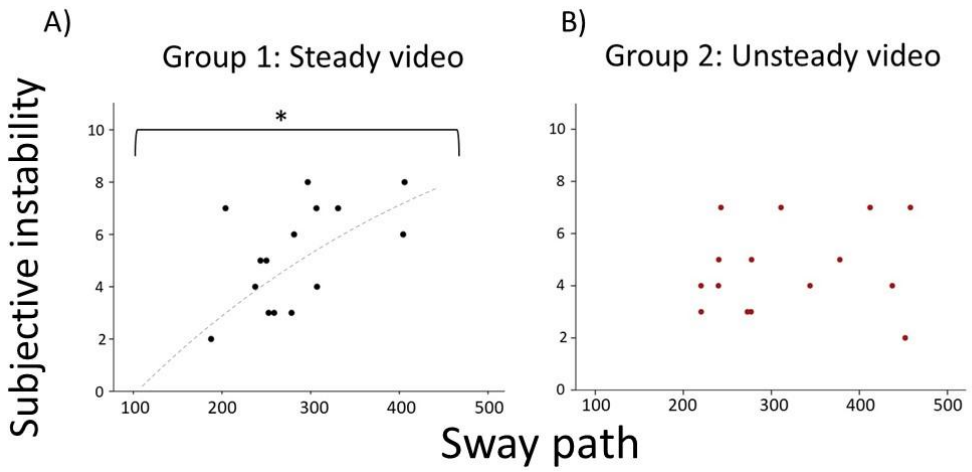

Figure 10

Relationship between Sway Path (objective) and Subjective instability for both groups. A significant relationship was observed only in the group that watched the Steady video (A); such a relationship was absent in the Unsteady video group (B). * p<0.05.

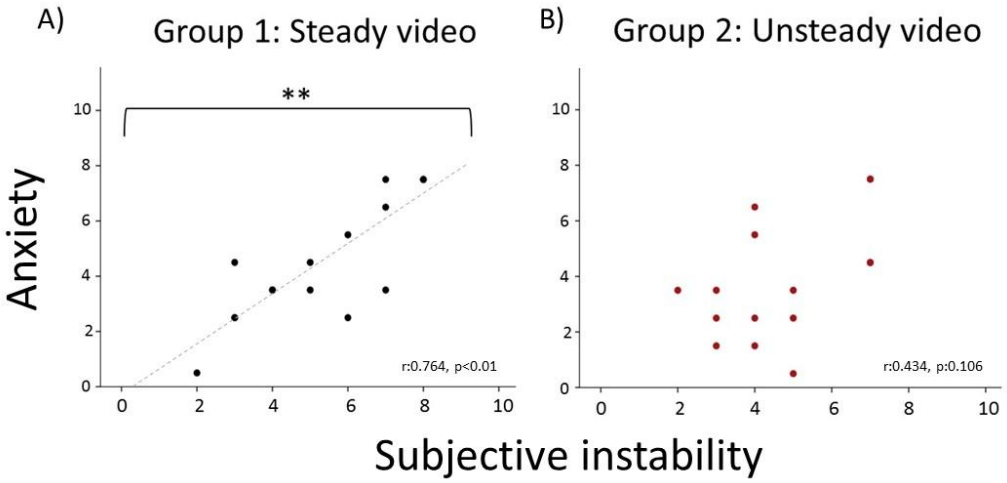

Figure 11

Relationship between Subjective instability and Anxiety for both groups. A significant relationship was observed only in the group that watched the Steady video (A); such a relationship was absent in the Unsteady video group (B). p<0.01.

5.2 Verbal priming Experiment

In the “no information” trials, the sway path was significantly different between the three amplitudes, as evidenced by a repeated measures ANOVA ($F_{(2,50)}$=226.565, p<0.001), hence confirming that the change in the stimuli amplitude generated a change in body sway (Figure 12A). In those trials where individuals received erroneous information about the forthcoming sled movement (but were actually exposed to the same sled velocity), they did not exhibit an increased sway path in relation to the magnitude of the priming (S:78.76cm, M:80.55cm and L:80.92cm; repeated measures ANOVA, p=0.314, Figure 12B). Hence, giving erroneous information about the upcoming task did not affect their objective postural sway.

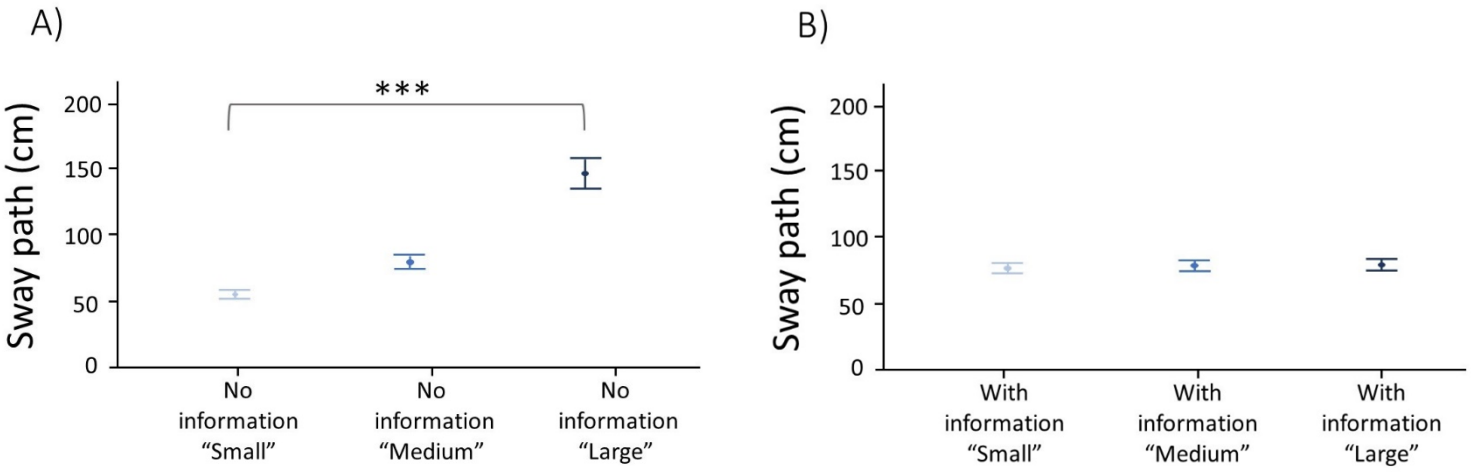

Figure 12

A) Sway path (in cm) recording from subjects during the three sled amplitudes without previous information and sled velocity change. The increase in body sway across the amplitudes was significant. B) Sway path (in cm) recordings from subjects when experiencing the three amplitudes with previous information but no real change in sled velocity. There is no significant difference in body sway on any of the amplitudes. Bars represent 95% CI. ***p <0.001.

Regarding the subjective measures, a repeated measures ANOVA demonstrated a significant increase in the perception of instability and anxiety with the change in sled amplitude ($F_{(2,50)}$=193.767,p<0.001 for instability and $F_{(2,50)}$=57.142, p<0.001 for anxiety,

Figure 13A) when no prior information was provided, in line with the observed change in sway path. Interestingly, in the trials with erroneous prior information, subjects' perceived instability and anxiety across the stimuli amplitudes was modulated by verbal priming despite there being no change in sway path ($F_{(2,50)}$=4.049, p<0.05 for instability; $F_{(2,50)}$=6.758, p<0.01 for anxiety, Figure 13B). Hence, misleading information about the task influenced the subjective, but not objective sway parameters.

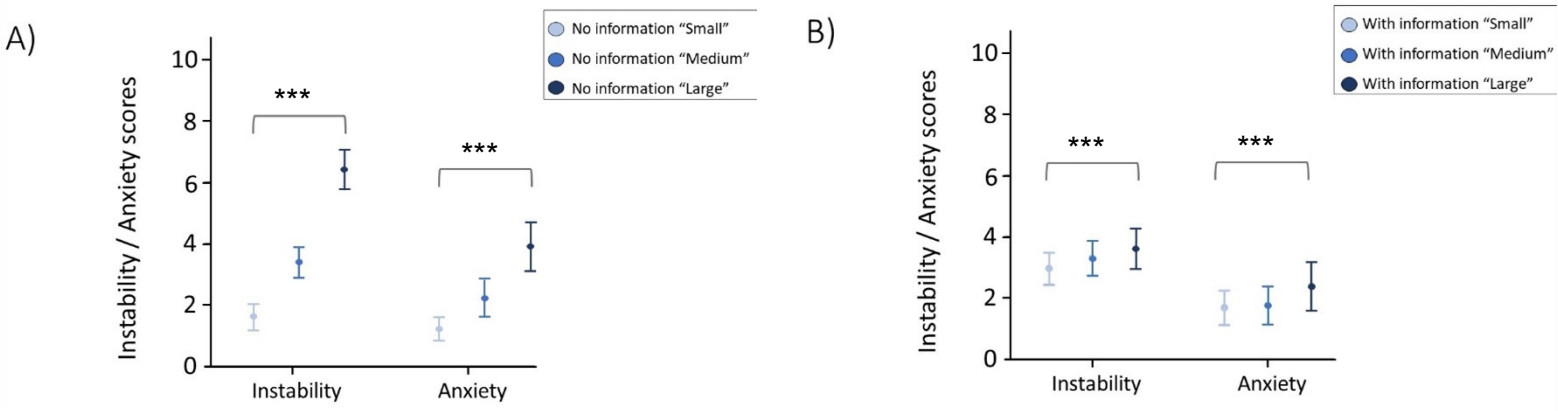

Figure13

A) Subjective instability and Anxiety scores provided by subjects during the three amplitudes without previous information but with a real sled velocity change. There was a significant increase in both Subjective instability and Anxiety across the three amplitudes. B) Subjective instability and Anxiety scores given by subjects during the three amplitudes with previous information provided but with no objective sled velocity change. There was a significant increase in both Subjective instability and Anxiety across the three amplitudes. Bars represent 95% CI. ***p <0.001.

Regarding the objective-subjective instability function, a regression with curve estimation showed a logarithmic function during the 'no information' condition (r^2:0.574, p<0.05), but an absence of any relationship when erroneous prior information was provided (r^2: 0.04, p=0.58, Figure 14). Finally, there was a tight relationship between instability and anxiety for both conditions (r^2:0.707 for 'no information' and r^2:0.569 for trials where verbal information was provided).

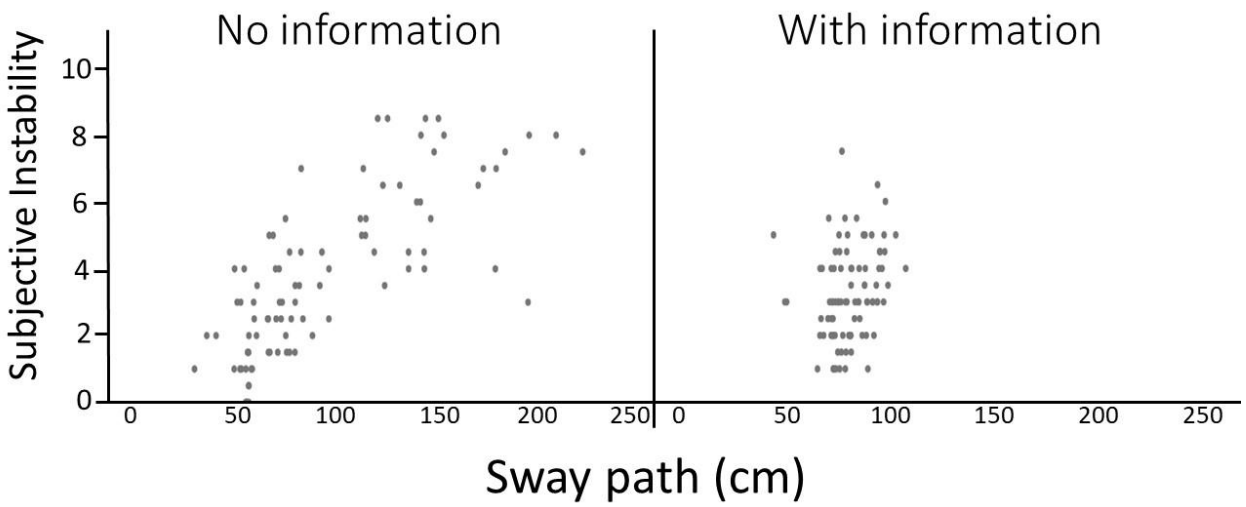

Figure 14

Relationship between Objective (Sway path) and Subjective instability for both conditions. A significant logarithmic function was observed in the trials where no information was given, but a sled velocity change occurred. This relationship was not significant in the trials with previous information but no platform velocity modification.

6. Discussion

The current investigation sought to identify whether modulating prior belief systems surrounding the difficulty of an upcoming postural task via the induction of an expectational mismatch may distort the relationship between objective and subjective instability. In line with the hypothesis, it was observed that erroneous visual and verbal priming disrupted the known relationship between the perception of stability and actual bodily displacement.

Several examples of dissociation between actual and subjective instability were observed. In the visual priming paradigm, body sway increased during the latter part of the task for subjects who had viewed the unsteady actor. Despite this increase in objective sway, their perception of their instability was not significantly different from the group that had viewed the steady video. Accordingly, the question that arises is why would an individual sway more but not report greater instability?

A possible explanation may be that the mismatch generated by the video primed the individual to expect a task that challenged their postural control. Considering that responses to upcoming events are mediated by prior beliefs or assumptions (Dryden, 1994), when participants came to actually perform the task, they found the task easier to perform in comparison to the priming-driven prediction regarding the difficulty of the upcoming task. Accordingly, such a mismatch could have generated overconfidence and the adoption of a more relaxed strategy, leading to increased sway but “no worries” about instability.

Such a proposition is supported by previous work indicating that the motor cortex generates motor plans for expected experiences. These plans need to be modified accordingly when a mismatch between the expected experience and the physical experience occurs (Grisoni, Dreyer & Pulvermüller, 2016). Applying this to the observations in this present study, it is postulated that during the initial part of the postural task subjects were bracing themselves for (i.e. expecting) a challenge that they predicted after viewing the actor in the unsteady video (Calvo-Merino et al., 2006). When this challenge never arose, subjects adopted a more relaxed postural strategy that would be compatible with the results of experiments exploring the motor cortex. This is consistent with the notion that the motor cortex can be activated with semantic incongruences related to movement, and

that such activation can change the plan that the motor cortex has selected for the given task (Grisoni, Dreyer & Pulvermüller, 2016).

An alternative, albeit conceptually close and non-mutually exclusive explanation is provided from the action-observation literature, which suggests that cortical resources in such areas are shared with those involved in task execution (Calvo-Merino et al., 2006). Accordingly, then, it was expected that the visual priming would generate in the subjects an equivalent change in the motor postural strategy in expectation of the forthcoming postural instability. However, in the present paradigm, when the real experience clashed with the expectation, this mismatch generated an opposite effect, i.e. subjects who expected a more destabilising task experienced less instability compared to their actual performance, and vice-versa. Given the absence of any modulation in the subjective ratings accompanying this objective change in body sway, it is possible to argue that either perceptual ratings are formulated early on during task performance or that such a change in postural strategy is not a fully conscious process.

The decision to adopt the early/late epoch analysis is borne out of the idea that if you have primed an individual to receive an expectational mismatch regarding the difficulty of an upcoming task, that will be at its greatest during the start of the trial (Early epoch). As the trial goes on (late epoch), the subject will adapt to the expectational mismatch. Accordingly, adopting the early/late epoch analysis approach is important to understand how postural strategies change in accordance with online evaluation of an expectational mismatch. Additionally, as seen with the anxiety analysis, the video did not generate significant differences in anxiety between the groups in the pre-task measurement, but the mismatch did generate a significant change in anxiety in one group, supporting the idea of a possible change during the task, which was probed further.

In the verbal priming experiment, it was observed that erroneous information about the amplitude of the forthcoming trials did not modulate the objective measures of body sway. However, it did distort the objective-subjective instability relationship, mediated by the increased ratings of subjective perception of instability and task-related anxiety in the absence of any physical change in platform motion or associated body sway.

How may priming distort the normally coupled objective-subjective instability measures (Castro et al., 2019b; Schieppati et al., 1999)? Critically, this uncoupling between objective measures and perceptual ratings of stability was observed only in participants that received erroneous visual or verbal priming by watching the unsteady video or receiving erroneous verbal cues regarding platform velocity. These dissociations indicate that when priming modifies subjects' expectation it can alter not only the objective motor performance, but also the subjective postural rating. Furthermore, given that both the erroneous visual and verbal priming distorted the normal relationship between objective measures of body displacement, perceived instability and task-related anxiety during the postural task, one could argue that such modulation is driven by emotional factors.

A possible mechanistic account for such influences can be derived from observations of a cortico-basal loop between areas of the limbic system and motor areas, implying that the limbic system provides an on-line threat assessment regarding body motion in space (Aoki et al., 2019). Surprisingly, limbic areas without any motor input were evidenced to generate motor output, hence showing how motor responses and their associated perceptual outputs can be modulated by: i) emotion and motivation (Aoki et al., 2019), and ii) threat and risk assessment (Adkin et al., 2002; Bunday & Bronstein, 2009; Carpenter et al., 2004; Cleworth & Carpenter, 2016; Green et al., 2010). Finally, the connections revealed between the limbic and motor areas postulate that novel internal and external threats exert an influence over motor output (Aoki et al., 2019). Considering that one of the main inputs used in the construction of perceived self-stability is actual body movement, it is plausible that such a loop connecting the emotion and motor control brain areas could modulate the normal relationship between objective and subjective measures of instability.

Regarding the directionality of the reported effect, the question remains as to whether increased anxiety increases self-reporting heightened levels of instability, or, vice versa, whether experiencing increased instability leads to higher anxiety levels. The data illustrated in Figure 7 shows that having watched the steady video induced anxiety during the task. This was presumably mediated by a combination of a fear of falling and/or startle, as the task was harder than predicted. On finding the task harder than predicted, fear of failure and falling set in, despite the fact that the subjects' objective sway levels remained unchanged. This suggests that anxiety is not primarily driven by objective instability levels

but by a central “risk assessment” process linked to arousal and ultimately involved in generating motor predictions (Green et al., 2010).

Complementary findings came from the subjects who developed ‘late’ objective instability after viewing the Unsteady video. In addition, the unsteady video disrupted the objective-subjective instability relationship by creating a ceiling effect on Subjective Instability (Fig 5), likely via the ‘over-confidence’ mechanism described before. This hypothesis is supported by the fact that watching the Unsteady video did not make people more anxious during/after the task. On the contrary, watching the Unsteady video reduced the coupling between Subjective instability and Anxiety (Fig 6), essentially because there were no longer high levels of Subjective instability during the task. In summary, on experiencing a task that was less challenging than predicted/expected, subjects seemed to ‘let go’ and not worry about their instability, which in turn lowered their anxiety levels. Critically, the paradigm used in this investigation was able to dissociate objective from subjective instability during a postural task, and in turn identified that subjective instability is the reason for developing higher levels of anxiety.

7. Conclusions

Taken together, the present results illustrate that modulating prior belief systems by inducing an expectational mismatch via either visual or verbal priming can distort the objective-subjective instability relationship. The findings presented in this chapter highlight the importance of prior expectation and anxiety in the perception of self-stability. These outcomes may have clinical implications for balance disorder patients, particularly those with functional neurological disorders (i.e. Persistent Postural Perceptual Dizziness (Staab et al., 2017) as well as those with functional gait disorders (Lin et al., 2020; Sokol & Espay, 2016). It can be suggested that balance therapists consider introducing priming to re-regulate patients' perceptual sensitivity by recalibrating the dynamics between prior expectation and perceived performance. As a practical example, therapists should be wary of presenting the retraining exercises to their patients using terms such as 'very easy' or 'very difficult'. In some subjects, the former may induce anxiety on performing the task whereas the latter might induce over-confidence.

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Chapter 3

Vestibular cortex dominance and postural control

1. Overview

The content of this chapter has been published in Brain Stimulation (Castro, P., Kaski, D., Al-Fazly, H., Ak, D., Oktay, L., Bronstein, A. & Arshad, Q. (2019) Body sway during postural perturbations is mediated by the degree of vestibulo-cortical dominance. Brain Stimulation: Basic, Translational, and Clinical Research in Neuromodulation. 12 (4), 1098–1100. doi:10.1016/J.BRS.2019.05.008).

I declare that I completed most of the work presented in this chapter, including the design, data collection and data analysis, the generation of the manuscript for submission, and the revision of the final version for publication. The contribution of the co-authors to this article is as follows: Fazly, H., Ak, D., and Oktay helped with the data collection. Drs. Kaski, D., Bronstein, A. & Arshad, Q. are the supervisors of this PhD and in this project provided support in the conceptualisation, design, and revision of the manuscript for publication.

In the previous chapter, it was possible to deepen the knowledge of the influence of expectations and anxiety on postural control, clarifying the contribution of emotional components to objective and subjective instability in healthy young subjects.

To continue my quest to understand posture, the effect of higher-order mechanisms involved in balance control will be explored. Accordingly, in this chapter, the possible influence of vestibular cortical areas and the right hemispheric dominance on postural control is investigated. Brain stimulation has been shown to modulate vestibular responses. The degree to which a vestibular nystagmus response is reduced following non-invasive brain stimulation has previously been used as a measure of right vestibular cortex dominance. Tactically, I will assess the effect of inhibiting the left vestibular cortex (to enhance the right vestibular dominance) on postural control in healthy young adults.

The normal control of posture necessitates vestibular and spinal reflexes; however, the influence of the cerebral cortex for postural control is less clear. Here cathodal tDCS is applied to inhibit cortical excitability in the left parietal cortex (in turn enhancing the individual right hemispheric dominance) to study its effect on posture. Balance function was measured following balance perturbations presented while subjects stood on a mobile sled. By examining postural measures before and after brain stimulation it is possible to

determine whether enhancement of a right hemispheric dominance modulates postural control in healthy individuals. If so, one can predict enhanced stability of upright posture following an unpredictable balance perturbation.

This work provides information on the role of the cerebral cortex and vestibular lateralisation in postural control, which is of particular relevance to patients with brain damage.

2. Abstract

Maintenance of posture requires the integration of multi-sensory cues. Recent studies have shown how during increasingly demanding postural tasks, EEG alpha power decreases proportionately in the central and parietal regions. Furthermore, this reduction in alpha power reveals an asymmetric distribution, whereby it is greater in the right hemisphere. However, to date the exact contribution that the lateralisation of the vestibular cortex plays upon maintaining postural control remains to be elucidated. Accordingly, the relationship between vestibulo-cortical dominance and body sway is investigated here using postural perturbations. A previously used biomarker of vestibulo-cortical hemispheric dominance was employed, namely the reduction of caloric vestibular nystagmus following transcranial direct current stimulation (tDCS) over the left Posterior Parietal Cortex (PPC).

Twenty-four right-handed participants (16 males, mean age: 21.6 years) took part in the study. tDCS was applied on two separate stimulation conditions: cathodal (test condition) or anodal (control) over the left PPC. A postural task with random left-right oscillations was used to assess postural control in the medio-lateral direction. Objective measures of body sway and subjective instability ratings were recorded.

No differences between the pre/post tDCS sway path or velocity measures were seen in either condition. The degree of right vestibulo-cortical dominance however showed a significant negative correlation with the sway path and sway velocity change after tDCS only in the cathodal group. This finding implies that individuals with a greater right hemispheric cortical representation swayed less and with a reduced velocity after stimulation. No significant correlation was observed in the anodal stimulation group.

The current findings illustrate how the individual degree of vestibulo-cortical hemispheric dominance modulates postural control, potentially influencing balance performance. These findings have potential clinical implications for neurological patients with balance and gait disorders where the degree of vestibular cortical dominance and the localisation of cortical lesions could affect their performance and treatment efficacy.

3. Introduction

Maintenance of posture requires the integration of multi-sensory (e.g., vestibular, visual and proprioceptive) cues (Chiba et al., 2016; Gill-Body, Beninato & Krebs, 2000). Contributions to postural control were historically noted to primarily implicate subcortical, spinal and cerebellar structures. More recent work, predominantly using electroencephalography (EEG), has shown that the cerebral cortex changes during postural tasks (Mierau et al., 2017; Hülsdünker, Mierau & Strüder, 2016). An alpha peak frequency increase has been observed with higher demands during a postural task, suggesting an increase in information transmission (Hülsdünker, Mierau & Strüder, 2016), and has been suggested to help the visual information processing needed for balance (Mierau et al., 2017). Additionally, it has been proposed that theta activity is related to error detection when postural demands increase (Hülsdünker, Mierau & Strüder, 2016) and to information processing and integration (Mierau et al., 2017).

Although postural control has been studied for years, the cortical areas and networks involved in postural maintenance and its perception are incompletely understood.

Imaging studies have identified that the parietal lobe is intimately involved in integrating vestibular, visual and proprioceptive information, three vital sensory inputs used in postural control (Chiba et al., 2016; Mierau et al., 2017; Gill-Body, Beninato & Krebs, 2000).

Additionally, it has recently been demonstrated that during increasingly demanding postural tasks, EEG alpha power decreases proportionately to the subjective rating of the task difficulty in the central and parietal regions. Furthermore, the reduction in alpha power is asymmetrically distributed, such that it is greater in the right hemisphere, suggesting an asymmetric cortical representation of vestibular information related to posture (Edwards et al., 2018).

Such asymmetry has been hypothesised to be dominant when processing vestibular information. Dietrich et al. (2003) showed how caloric stimulation activates cortical and subcortical structurespreviously related to the vestibular system predominantly in the non-dominant hemisphere, which is dependent on handedness. In the same line, Arshad et al. (Arshad et al., 2014) provided further support for the existence of lateralised responses

when the vestibular system is stimulated. In particular, Arshad et al. based their work on the reduction of the velocity of the caloric nystagmus after tDCS inhibiting the left parietal lobe (Arshad et al., 2014; Bednarczuk et al., 2017; Arshad, 2017a). Interestingly, the reduction in response does not occur when the right parietal lobe is the one being inhibited or when the left posterior parietal cortex is excited, hence suggesting an asymmetric cortical representation and control of vestibular responses (Arshad et al., 2014; Nigmatullina et al., 2016; Arshad et al., 2015b). This asymmetric distribution implies involvement of the vestibular cortical processing areas in the non-dominant hemisphere during active postural control (Edwards et al., 2018). It has also been proposed that the asymmetric cortical representation of vestibular responses implies a right hemispheric dominance in right handers, which in turn could mean that the vestibular hemispheric dominance could define handedness (Dieterich et al., 2003). This asymmetric response and the implications for different vestibular and non-vestibular functions has also been studied, with similar results (Arshad et al., 2015b; Arshad, Nigmatullina & Bronstein, 2013; Arshad et al., 2019a, 2016).

TDCS has been shown to modulate cortical excitability (Cohen Kadosh et al., 2010; Sparing et al., 2009; Orban de Xivry & Shadmehr, 2014) and, when conducting cathodal stimulation over the left Posterior Parietal Cortex (PPC), has been observed specific modulation of subcortical vestibular responses (Bednarczuk et al., 2017; Arshad, 2017b).. The inhibition generated by the cathodal stimulation (Orban de Xivry & Shadmehr, 2014) will unveil the degree of right hemispheric dominance (Arshad, 2017a; Arshad et al., 2014), and the extent to which this dominance will affect postural performance. If the information regarding posture maintenance and its perception is processed in this cortical area, it is necessary to identify whether the lateralisation of the vestibular function could also affect posture maintenance.

The experimental paradigm described by Arshad and colleagues (Arshad et al., 2014) can be used to enhance the vestibular right hemisphere dominance using transcranial direct current stimulation (tDCS) and provides a quantification of the degree of lateralisation of the vestibular cortex in a given individual (Arshad, 2017a; Bednarczuk et al., 2017). The procedure encompasses the reduction of a nystagmic response after tDCS, which will be different for each subject depending on their degree of right hemispheric dominance (Bednarczuk et al., 2017; Arshad, 2017a).

The tDCS applied to this parietal area (P3 on a 20-20 electrode montage) can be excitatory (anodal) or inhibitory (cathodal); the latter enhances the vestibular cortex asymmetry by inhibiting the left hemisphere, hence boosting the right hemispheric dominance (Bednarczuk et al., 2017; Arshad, 2017a). The nystagmic response will change after this stimulation and the percentage change in the nystagmic response after tDCS has been called the Nystagmus Suppression Index (NSI) and varies depending on each subject's degree of right hemispheric dominance. A high negative NSI implies a larger right hemispheric dominance, as the inhibition of the left parietal lobe will generate a larger suppression in the response, which is expected to be mediated by the dominant right hemisphere. That is how the degree of right vestibular dominance, measured with the NSI, can be related to different functions of the system, and even cortical functions such as numeric magnitude (Arshad et al., 2016) and self-motion perception (Arshad et al., 2019b).

The experiment described in this chapter aimed to identify whether the vestibular cortex dominance, enhanced by tCDS on the left PPC, can modulate objective and subjective measures of postural instability in a postural task that requires vestibular information and its integration with additional inputs in order to be executed effectively.

Due to the modulation exerted by the right hemispheric dominance on vestibular responses (Bednarczuk et al., 2017; Arshad et al., 2015b; Nigmatullina et al., 2016; Arshad et al., 2014), an effect on postural control was expected. If the degree of right vestibular cortex dominance influences postural control, a reduced amount of body sway after tDCS would be anticipated in subjects with more right vestibular hemispheric dominance, quantified by the NSI. An inhibitory stimulation (cathodal) on the left Posterior Parietal Cortex would reveal the right hemispheric dominance, which would contribute to a steadier performance. The anodal stimulation has not been observed to be related to vestibular hemispheric dominance (Arshad, 2017a; Arshad et al., 2014); hence no correlation between the degree of right hemispheric dominance (quantified with the NSI) and body sway would be expected in subjects receiving this stimulation condition.

4. Material and methods

To address the aim of this investigation, tDCS was used over the left parietal lobe, PPC specifically, to enhance the vestibular cortical dominance. The NSI was calculated as a measure of this dominance and was later correlated with an individual subjects' postural performance.

4.1 Participants

Twenty-four right-handed participants (16 males, mean age: 21.6 years, S.D.=1.35), that had no history or active neurological, ophthalmological, otological and psychiatric disorder were recruited. No subject had any brain-stimulation contraindications. All procedures were performed in accordance with the guidelines of the Helsinki declaration, and all subjects provided written informed consent.

4.2 Postural task

Subjects performed a postural task as described in previous chapters. A moving sled powered by two linear motors was used to deliver dynamic balance stimuli to participants. All subjects were blindfolded and wore earmuffs to avoid visual or auditory cues about the sled oscillation. Participants were asked to stand on the platform, to try not to hold on to the side safety bars, and to take corrective steps if required. During this task, objective measures of body sway were recorded: upper body displacement was obtained using a sensor placed over the level of the C7 vertebra, and the velocity of the centre of mass was assessed with an accelerometer over the right iliac crest (Figure 1). Additionally, after each trial, instability and anxiety were measured using two cartoon-aided scales, ranging from 0 ("very stable"/"not at all anxious") to 10 ("so unsteady I would fall"/"extremely anxious"). Differently to the investigations shown in previous chapters, subjects stood perpendicular to the sled displacement, so the body displacement was sideways (Figure 1). The stimulus consisted of a combination of 4 harmonically unrelated sine waves of different frequencies (0.18, 0.37, 0.69 and 0.9 Hz) delivering leftward and rightward oscillations for 30s in a fixed pseudo-random sequence (Figure 2). For this task, two velocities were used: "slow"

(maximum velocity of 0.05 m/s) and “fast” (maximum velocity was 0.2 m/s). These velocities were selected to include an oscillation that would cause a small sway path and then would be easy to complete and a larger sled oscillation that implied a postural challenge, based on previous studies using this paradigm (Castro et al., 2019b). Subjects completed this task twice, before and after tDCS and the calculation of NSI.

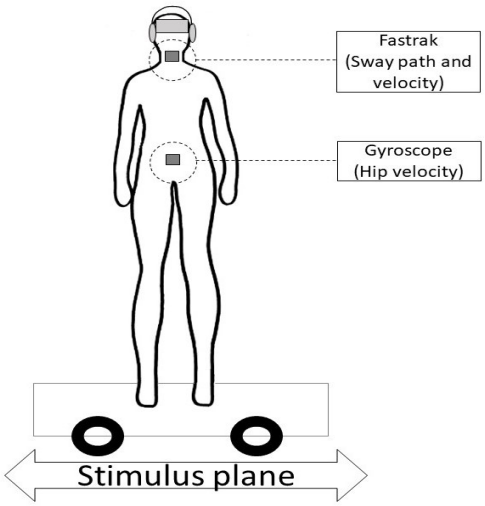

Figure 1

Platform task. Subjects stood sideways to the sled displacement and two movement sensors were put in place in order to record their body movement. One sensor was placed on the upper trunk and the other at the hip. Subjects were blindfolded and wore earmuffs to avoid visual or auditory cues.

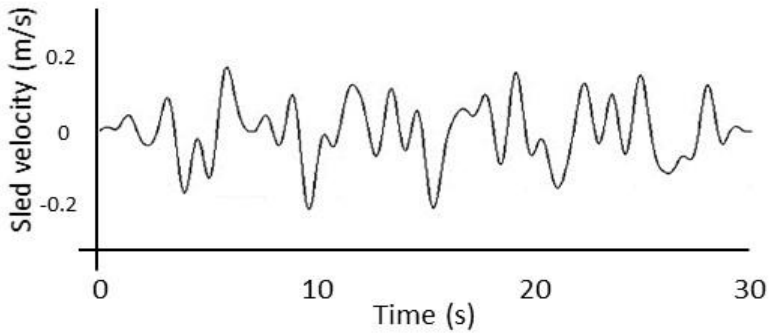

Figure 2

Stimulus profile used to drive the sled movement depicted as a velocity profile. The stimulus consisted of a combination of 4 harmonically unrelated sine waves of different frequencies (0.18, 0.37, 0.69 and 0.9 Hz). The first and last seconds of the stimulus were tapered to zero to avoid sudden accelerations and decelerations. This stimulus-induced left and right oscillations for 30s in a fixed pseudo-random sequence. Finally, the waveform was amplified using a linear potentiometer at 3 times the original waveform amplitude to generate trials of differing velocities (range 0–0.2 m/s in each direction).

4.3 Caloric test

Subjects underwent a caloric stimulation in order to obtain their nystagmus Slow Phase Velocity (SPV) at two time points, pre- and post-tDCS. To elicit the caloric nystagmus, subjects lay on their back on a medical couch with their head lifted 30 degrees to place the horizontal canal in an optimal position (vertical orientation) for testing. Subjects then had a cold caloric irrigation with water at 30^{o}C, for 40 seconds with a flow rate of 500 ml/min, on one ear at a time. The nystagmus was recorded using infrared Video NystagmoGraphy (VNG) goggles (ICS Chartr 200 VNG/ENG system, Otometrics) and the slow phase velocity (SPV) component was extracted using the clinical software (ICS Chartr 200 VNG/ENG software), providing the peak eye velocity, which was collected for analysis.

4.4 NSI calculation

The Nystagmus Suppression Index (NSI) was used as a quantification of the vestibular right hemisphere dominance [9]–[11]. The NSI corresponds to the percentage change in the nystagmus SPV after tDCS, using data from the pre and post caloric tests. The calculation is performed using the following formula: subtracting the peak post-tDCS SPV from the peak pre-tDCS SPV divided by the peak pre-tDCS SPV × 100.

4.5 tDCS

Electrical stimulation, tDCS (neuroConn GMBH, Ilmenau, Germany) was applied using a current of 1.5mA for 15min. Subjects were pseudo-randomly allocated to receive either cathodal (test condition, 12 subjects) or anodal (control condition, 12 subjects) stimulation. The anodal stimulation was used as a control condition due to the lack of vestibular modulation evidenced in previous studies (Arshad et al., 2014). The head electrode was the (+) for subjects in the Anodal condition and (-) for those in the Cathodal group, and was placed over the left PPC (P3; 10–20 international EEG classification; electrode placement area 25cm ^{2}). The reference electrode was placed on the ipsilateral shoulder (see Figure 3). The current had a ramp-up time of 10s at which point a constant current (1.5mA) was applied for 15 minutes. The current was ramped down in a 10s fade-out period [6 7 8].

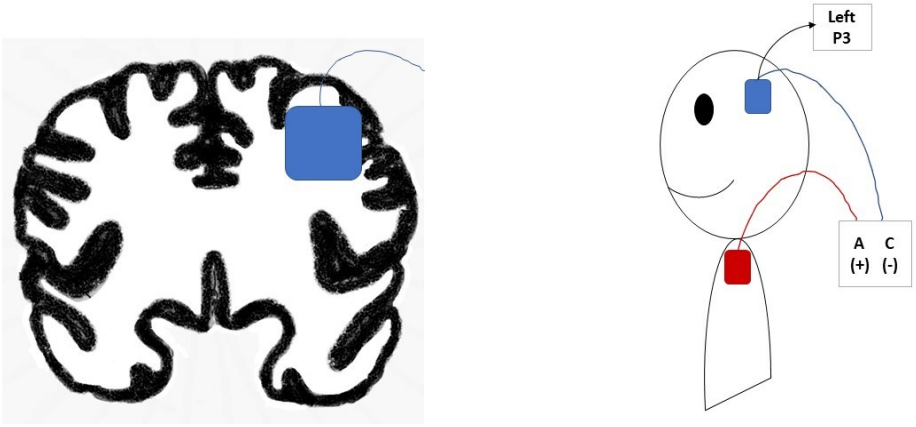

Figure 3

Electrode placement for the cathodal stimulation condition. The blue electrode represents the cathodal polarity (-), which was placed on the head (Left PPC). The anode (+) was placed on the ipsilateral shoulder. For anodal stimulation the electrode placement was inverted.

4.6 Experimental protocol

Subjects performed a baseline postural task, which provided the body sway information for the pre-stimulation time point. Immediately after this, they underwent a caloric test to obtain the baseline SPV measure (pre-tDCS) to be used later in the NSI calculation. Later, subjects were randomly allocated to receive tDCS on “Anodal” or “Cathodal” polarity. After the stimulation was finished, the caloric test was repeated on both ears (post tDCS) and the percentage change in SPV from the baseline was defined as the NSI. Finally, the postural task was repeated with the same settings. Subjects and examiners were blind to the group allocation. A schema of the protocol can be observed below (Figure 4).

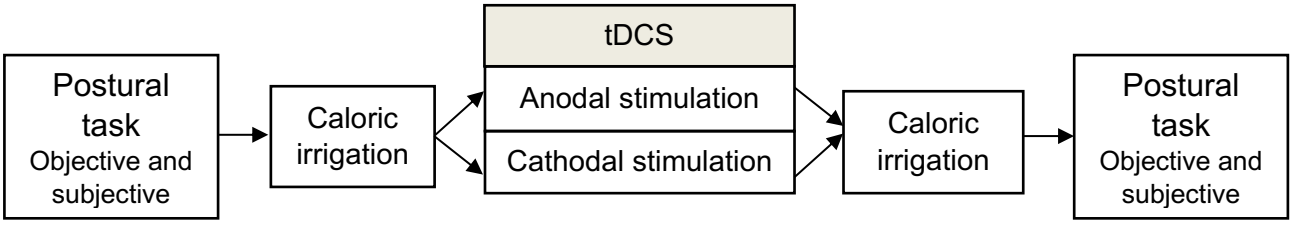

Figure 4

Schematic representation of the study protocol. All subjects followed this protocol, with random allocation to tDCS anodal or cathodal stimulation.

4.7 Statistical analysis

The statistical analysis was performed using SPSS version 27. Independent t-tests were used to compare the variables between the two condition groups. A repeated measures ANOVA was used to identify differences in the paired data, such as pre and post tDCS postural measures. A Pearson correlation was used to correlate the variables. The significance level was considered significant when $p<0.05$.

5. Results

A significant suppression of the caloric slow phase eye velocity after both the anodal and cathodal stimulation was observed (mean 51.71%, s.d. 18.91 reduction on cathodal and 11.03%, s.d. 16.82 on anodal; F:47.630, $p < 0.01$; repeated measures ANOVA; Figure 5). This reduction, determined as the NSI, was significantly larger for the cathodal stimulation group (t:-7.872, $p<0.001$; Figure 6).

Regarding the sway analysis during the postural perturbations, we observed no differences when comparing the pre and post tDCS sway path or sway velocity measures between the two stimulation conditions (F:0.55, p:0.46 for sway path and F:1.34, p:0.253 for sway velocity, repeated measures ANOVA).

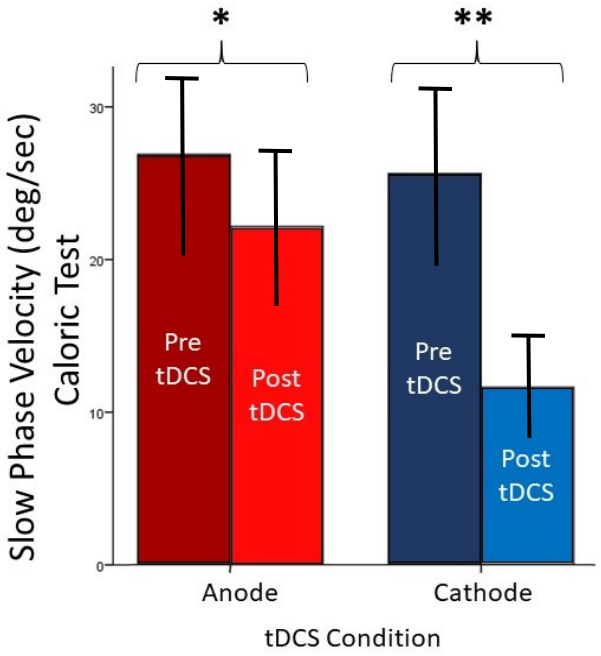

Figure 5

Nystagmus Slow Phase Velocity (SPV) during caloric stimulation before and after tDCS for both stimulation groups, anode and cathode. The black lines represent 95% CI. * p<0.05, ** p<0.01.

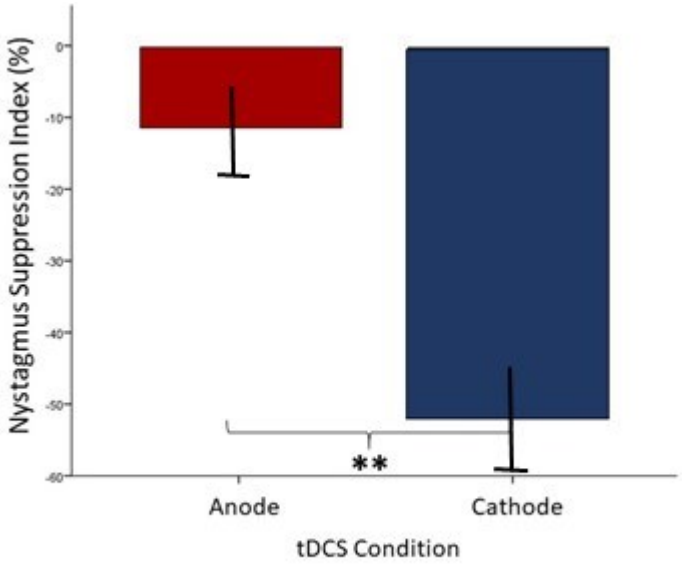

Figure 6

Average Nystagmus Suppression Index (NSI) for both stimulation groups, anodal and cathodal. The black lines represent 95% CI. ** p<0.01.

To identify any change in sway path occurring by the repetition of the task, the sway path and sway velocity from the trial pre tDCS was compared to those from the performance after stimulation. No significant change was observed between pre and post

tDCS sway path (t:0.802, p:0.427) or sway velocity (t:1.709, p:0.094). To specifically assess whether an individual's body sway was related to the degree of vestibulo-cortical dominance, the NSI was correlated with the measures of sway path and sway velocity change after tDCS. The percentage change in sway path and sway velocity from pre to post tDCS was calculated for each subject and later correlated with their individual NSI. There was a significant negative relationship between the degree of hemispheric dominance represented by the NSI and the sway path (r: -0.721, $p < 0.01$, Figure 7) and sway velocity (r: -0.65, $p < 0.05$, Figure 8) change. This correlation was only present and significant in the cathodal group and not in the subjects who received the anodal stimulation (r: -0.40, p:0.193 for sway path and r: -0.46, p:0.13 for sway velocity). To identify whether the hemispheric dominance would affect each direction of body perturbation differently, the sway traces were separated into right and left translations by taking the initial position of the subject for each trace as the "centre". Later, the traces were divided into either rightward or leftward movements from the centre. Again, the change in sway path and sway velocity was correlated with the NSI. The same negative correlation was present for both the rightward (r: -0.79, $p < 0.01$ for sway path and r: -0.66, $p < 0.05$ for sway velocity) and leftward (r: -0.59, $p < 0.05$ for sway path and r: -0.62, $p < 0.05$ for sway velocity) perturbations . Again, no significant correlations were observed in the anodal stimulation group.

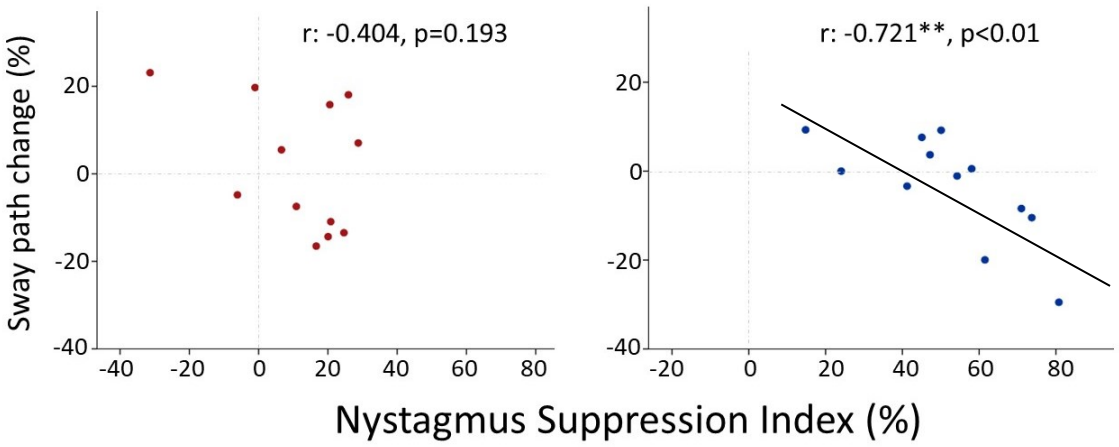

Figure 7

Correlation between the NSI and the overall (left and right) sway path percentage change in both the anodal (red dots) and cathodal groups (blue dots). A significantly negative correlation was observed only in the cathodal group, illustrating that individuals with a greater nystagmus suppression index swayed less after tDCS.

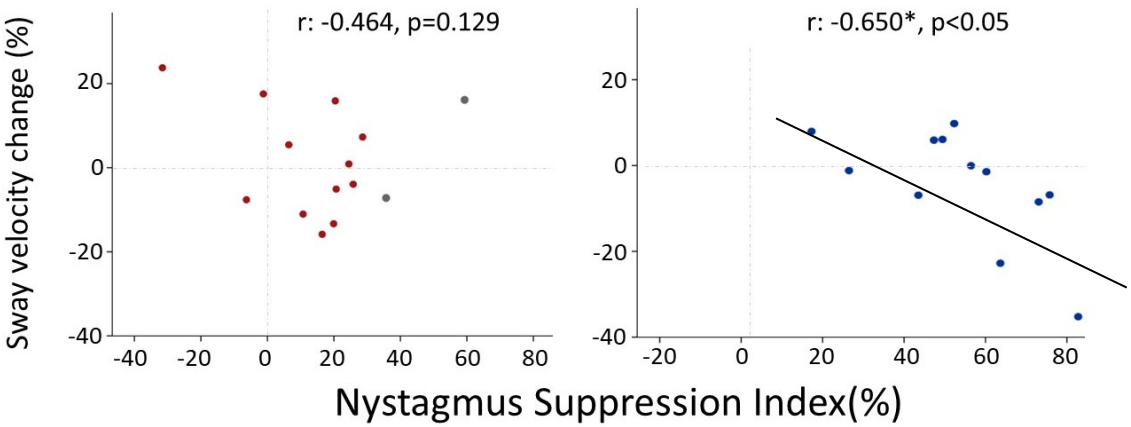

Figure 8

Correlation between the NSI and the overall (left and right) sway velocity percentage change in both the anodal (red dots) and cathodal groups (blue dots). A significantly negative correlation was observed only in the cathodal group, illustrating how individuals with a greater nystagmus suppression index swayed with a slower velocity after tDCS.

The correlations were found only in the "fast" perturbation stimuli, with no significant finding observed in the "slow" oscillations (all correlations $r<0.2$, $p>0.3$).

The subjective measures used during the task did not differ between the two stimulation groups ($p=0.57$ for subjective instability and $p=0.59$ for anxiety) or between the pre and post stimulation times ($p=0.58$ for subjective instability and $p=0.84$ for anxiety) and no correlation was observed with NSI; hence the change and correlations observed were only present for the objective measures of body movement.

6. Discussion

The main finding was a reduction in sway path and velocity following cathodal stimulation over the left PPC. This reduction was correlated with an individual's degree of right vestibulo-cortical hemispheric dominance as measured using the NSI. Accordingly, those individuals with a greater right hemispheric cortical representation for gravito-inertial processing swayed less and with a reduced velocity after stimulation.

As presented in the introduction, the right hemispheric cortical representation is believed to play a relevant role in the processing of vestibular information (Nigmatullina et al., 2016; Bednarczuk et al., 2017; Arshad, 2017a). To date, mostly subcortical vestibular functions, such as the vestibulo ocular reflex, have been linked to right hemispheric dominance (Bednarczuk et al., 2017; Arshad et al., 2014). This investigation provides evidence of the contribution of the cerebral cortex, and specifically the right hemispheric representation of vestibular information, to postural control. Rightward body perturbations result in a leftward sway, perhaps shifting spatial attention leftwards mediated by the right hemisphere. Since left hemisphere cathodal stimulation inhibits the left PPC, individuals who are more right-hemisphere dominant will be better able to cope with this shift, hence compensating more effectively for the left hemisphere inhibition. On the other hand, during leftward platform perturbations, the body sways rightward and shifts spatial attention rightwards, mediated by the left hemisphere. Thus, subjects with a more dominant right hemisphere will be less affected by the left hemisphere inhibition, and therefore will not be expected to experience significant instability. Conversely, those with a less dominant right hemisphere will be more affected by the inhibition of the left hemisphere, which will therefore have a greater destabilising effect upon their postural control. An alternative explanation is that participants with a higher right hemispheric vestibulo-cortical dominance favour the use of gravito-inertial cues, and hence sway less, a notion supported by previous lesion data (Pérennou et al., 2008).

The fact that the slow platform oscillations were not different between the conditions and did not correlate with the subject’s right hemispheric dominance suggests that this dominance might not be evident when the tasks are not challenging. During fast oscillations, the body movement was larger; hence, more postural adaptations were needed

to keep balance. These complex balance situations are where the hemispheric dominance might be playing a role in postural maintenance in healthy individuals. However, this could be different in patients with cortical lesions, where subjects exhibit postural disturbances (Tasseel-Ponche, Yelnik & Bonan, 2015; Dougherty, Carney & Emmady, 2022; Baggio et al., 2016; Mihara et al., 2012), especially if the lesions are in the right hemisphere (Manor et al., 2010); in this case, simple postural tasks could become extremely challenging for those individuals who are more right hemisphere dominant.

Similarly, subjective instability did not show differences between the conditions or a correlation with objective instability (body sway) change or the degree of right hemispheric dominance. This finding points to a less conscious role of the hemispheric dominance, modulating postural control, but not necessarily influencing how subjects perceive their balance performance. Right hemispheric dominance has been shown to affect cortical and subcortical functions (Bednarczuk et al., 2017; Arshad et al., 2016, 2014); hence, its influence in postural control could come from a cortical modulation of peripheral motor activity related to posture, as seen with other motor tasks (Orban de Xivry & Shadmehr, 2014). Surprisingly, the subjective instability scores were not influenced by right hemispheric dominance, despite hemispheric dominance previously being linked to cortical functions such as trait anxiety (Bednarczuk et al., 2018) and visual excitability (Arshad et al., 2019b). The construction of stability perception, however, might involve more complex and conscious processes (Ellmers, Kal & Young, 2021) that may be less influenced by hemispheric dominance.

An influence of vestibular hemispheric dominance on posture would bring clinical implications for the assessment and management of balance disorders in patients with brain damage, as their compromise could vary depending on the specific hemispheric representation.

7. Conclusions

The findings of this chapter illustrate how vestibular-cortical dominance impacts upon postural control. Additionally, these results contribute to the lateralisation hypothesis and highlight the importance of identifying the impact it has on other functions related to the vestibular system. There are potential clinical implications, particularly for neurological patients with balance and gait disorders (e.g. patients with SVD), in whom their diagnoses and possible impairment could be influenced by the degree of vestibular cortical dominance and the localisation of any cortical lesion. It is important to note that these findings correspond to healthy young subjects and, therefore, how the cerebral cortex modulates the objective and subjective aspects of postural control in patients with balance disorders still needs to be examined. Finally, there could be implications for treatment definitions and implementation based on the individual hemispheric dominance, which could be used to increase the efficacy of balance training and could also improve the prognosis of patients with impaired balance. In the next chapter, the characteristics of brain activity in patients without vestibular function will be studied to understand the reorganisation that the cortex undergoes when no vestibular inputs are present. Understanding the changes in brain activity and investigating how they might be linked to right hemispheric dominance, enhanced by brain stimulation, is of clinical relevance. Indeed neuro-modulatory treatment options for balance disorders could be guided by these and future findings.

8. References

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Chapter 4

Vestibular loss disrupts visual reactivity in the alpha electroencephalography rhythm

1. Overview

The content of this chapter is currently being prepared to be submitted for publication in a scientific journal.

In the previous chapter, I observed how the vestibular cortex modulates postural control in healthy young subjects when completing a dynamic postural task. Although these findings provide an insight into a general cortical involvement in the control of posture, the exact areas and processes involved in the maintenance of balance in patients with postural impairment still need further clarification. Accordingly, this chapter studies the alterations in brain activity when patients with reduced peripheral vestibular inputs complete a postural task. Due to the importance of multisensory integration for the correct functioning of the vestibular system, patients with no vestibular function are likely to have a reweighting of the sensory afferents and a reorganisation of their processing at the cortical level to preserve postural control. Changes in brain activity in these patients could imply possible cortical restructuring that occurs after a vestibular insult in order to preserve balance function. Although the vestibular system can compensate after a peripheral organ insult, the occurrence of specific changes in brain activity related to vision has not been fully investigated. Accordingly, here I explore the influence of vision on brain activity in patients with peripheral vestibular loss.

The primary aim of this study was to identify changes in brain activity with and without vision (eyes open vs. closed) in patients with bilateral vestibular hypofunction in sitting and standing positions.

Electroencephalography (EEG) was used to record brain activity in patients and age matched controls in order to identify differences in alpha band reactivity (i.e. increased alpha frequency activity on eye closure) in different postural conditions. Additionally, the possible relationship between alpha band reactivity and visual dependency (the degree to which an individual is reliant on vision for balance) was also assessed. Understanding the nature of the cortical changes that follow a vestibular insult could help us better predict the generation of symptoms related to vestibular loss and thus design preventative and management strategies for these individuals.

This chapter contributes to the main aim of this PhD by helping to identify the compensatory changes occurring at cortical level to maintain postural control in the context of vestibular loss. Finally, these findings encourage further research to inspect the role of vision and visual dependence on balance difficulties in other disorders, and to develop diagnostic and therapeutic approaches relevant to clinical practice.

2. Abstract

Postural control relies on effective multisensory integration. Multisensory integration is frequently studied using known interactions between vestibular and visual systems: Visual motion stimuli deactivate vestibular cortices and vice versa. Relatedly, vestibular loss leads to increased visual dependence - a bias in multisensory perceptual judgements (e.g. 'which way is up?') towards vision. These findings imply that normal vestibular function is essential for the tuning of visual cortical activity. We test this hypothesis using alpha EEG power as a measure of visual cortical activity measured in patients with bilateral vestibulopathy and controls.

EEG recordings were undertaken in 15 bilateral vestibulopathy patients (mean age 62 +/- 18 years) and 15 age-matched controls (62 +/- 20 years) in upright standing and seated postures with eyes open and closed. Measures of visual dependence were also collected.

With eyes open, the average alpha EEG power in both the seated and standing positions was comparable across the patients with bilateral vestibulopathy and the controls. An Alpha EEG power enhancement on eye closure (also known as visual reactivity) was observed in both the patients and controls ($p<0.05$); however, the participant group showed significantly less alpha enhancement with eyes closed ($p<0.05$). More residual vestibular function, quantified by the vestibulo-ocular reflex (VOR) gain, correlated with increased alpha reactivity in patients ($p<0.05$). Similarly, greater visual dependence correlated with less alpha enhancement ($p<0.05$), whereas no significant correlation was present in the controls. More visually dependent patients thus had less alpha visual reactivity.

Our results show that vestibular loss disrupts visual reactivity in alpha EEG oscillations. The correlations between greater visual dependence and vHIT gait with alpha enhancement in the patients suggests that both, the degree of vestibular loss and central visuo-vestibular interaction, drive the reduction in alpha reactivity. Our finding of a relationship between the alpha rhythm and vestibular function may have broad implications for cortical functioning following vestibular disease.

3. Introduction

3.1 Multisensory integration in balance control

Balance maintenance is a basic but complex function, requiring the integration of multiple pieces of information coming from different sensory systems in the body (Peterka, 2002; Mergner, Maurer & Peterka, 2003). The peripheral vestibular organ, including the otolith organs and semicircular canals, is just one of the sensory contributors to postural control. Plantar sensors and ankle proprioceptors also provide relevant information to maintain balance (Mergner, Maurer & Peterka, 2003). Finally, vision plays a very important role in providing the environmental information to place the body in space (Bronstein, 2016). When the integration of this multisensory information is disrupted, postural control will be impaired, highlighting the importance of this process in balance performance (MacKinnon, 2018).

The importance of a healthy peripheral vestibular organ in posture is widely accepted. Patients who present with a peripheral vestibular impairment will often have posture and gait problems (Gill-Body, Beninato & Krebs, 2000; Lucieer et al., 2016), and these will be even more marked if the dysfunction affects both labyrinths (Bilateral Vestibular Hypofunction, BVH). Consequently, after a BVH, the vestibular system of these patients will require the reorganisation of sensory information to happen, and other inputs to compensate for the lack of vestibular inputs in order to maintain balance (Curthoys, 2000). Interestingly, the participation of vision is believed to be fundamental for balance maintenance in these patients (Herdman, 2014). However, an abnormal integration of visual information could lead to the development of “visual vertigo” (Bronstein, 2016), where patients experience dizziness when exposed to certain visual stimuli, such as optokinetic stimulus or visually charged environment (busy streets or supermarket aisles) (Bronstein, 2016). Additionally, patients can become extremely reliant on visual information for balance after a vestibular insult, and may become “visually dependent” (Bronstein, 2016). Conversely, vision is used in vestibular rehabilitation to help the substitution of other strategies to replace the peripheral organ lost function (Curthoys, 2000; Herdman, 2014), as this is a reliable sensory input. However, the use of visual substitution in rehabilitation must

be within certain limits, as an over-reliance on visual information or increased visual dependency can become a chronic problem for patients (Bronstein, 2016). Because of its nature, it is not surprising that visual dependency is more marked in patients with BVH. Despite visual dependency being common in these patients, the rationale behind how the integration of visual information is affected at the cortical level when no peripheral vestibular function is present is not fully understood.

3.2 Brain activity

Electroencephalography (EEG) provides useful insights into brain function (Bazanova & Vernon, 2014) and hence has been widely used to understand brain activity. EEG response is analysed in different frequency bands: delta, theta, alpha, beta, and gamma, which have been linked to different cortical activities such as attention, memory, motor planning and cognitive processing (Herrmann et al., 2016). An 8-13 Hz posterior-predominant "alpha" rhythm first described by Hans Berger (La Vaque, 2008) was observed to be present with eyes closed and suppressed on eye opening. The alpha rhythm then is reactive, being enhanced by eye closure, and suppressed by eye opening, as well as following somatosensory, auditory and vestibular stimuli (Gale et al., 2016).

3.3 Alpha band visual reactivity

Visual reactivity in alpha oscillations – the so-called 'Berger effect' (Barry et al., 2007) - is a marker of neural efficiency relevant to cognitive health. Alpha rhythm enhancement on eye closure diminishes with brain ageing (Duffy et al., 1984), and is associated with cognitive underperformance in patients and healthy subjects (Van Der Hiele et al., 2008; Babiloni et al., 2010). In healthy individuals, the visual reactivity of the alpha band suggests a link between alpha brain activity and visual processing. Other determinants of visual alpha EEG reactivity are not well understood but known functional interactions between the visual and the vestibular systems suggests that vestibular function may be important. If we were to hypothesise that visual integration could be impaired in patients with vestibular disorders who develop visual dependency, it would not be unreasonable to think that those patients would have an abnormal integration of visual information, which could be evidenced in an

abnormal alpha band reactivity. However, this proposition has not been formally tested.

3.4 Visual alpha reactivity and postural control after vestibular failure

Functional interactions between the visual and vestibular cortices are important in health and disease. In health, the visual and vestibular cortices show reciprocal inhibition, such that visual motion stimuli deactivate the parieto-insular vestibular cortex, and vestibular stimuli deactivate the visual cortices (Brandt et al., 1998). The networks interconnecting the visual and vestibular areas support perceptual judgements on verticality and self-motion (Ibitoye et al., 2022). In patients with vestibular loss, the usual reciprocal visuo-vestibular cortical interactions are diminished (Bense et al., 2004) and the visual cortical stimulus responses differ (Dieterich et al., 2007). This occurs in the context of widespread structural changes in multisensory areas and altered functional connectivity (Göttlich et al., 2014). Dysfunctional functional connectivity in visuo-vestibular networks likely accounts for vestibular symptoms in patients (Göttlich et al., 2014), as well as greater visual dependence (Roberts et al., 2018; Ibitoye et al., 2022). Due to the reorganisation process that occurrs after a bilateral vestibular lesion, the visual information could suffer a restructuring in its processing, which could be evidenced with an unexpected alpha reactivity. Relatedly, as vestibular loss leads to increased visual dependence - a bias in multisensory perceptual judgements towards vision- a normal vestibular function would be essential for the tuning of visual cortical activity. This leads to the prediction that the usual deactivation of the visual cortex on eye closure is impaired following vestibular loss, in association with more visual dependence.

In this work, the effect of vestibular loss on visual alpha EEG reactivity is investigated. It is proposed that intact vestibular functioning facilitates deactivation of the visual cortices in health - a corollary being the enhancement of alpha oscillations on eye closure. Resting state seated EEG was measured in controls and patients with BVH to identify whether chronic bilateral vestibulopathy was associated with less alpha EEG enhancement on eye closure in the patients compared to the healthy controls and whether this reduced visual reactivity was related to vestibular and visual dependence.

4. Material and methods

4.1 Participants

Fifteen patients with chronic (> 6 months) BVH (9 female, median age 70 years, interquartile range 51 to 77 years) were recruited from a vestibular neurology clinic (diagnosis established by a neurotology specialist based on Bárány Society criteria on the basis of caloric, video head impulse test (vHIT, Otometrics) or rotational chair testing (Strupp et al., 2017)). The patients did not have any other vestibular, neurological or musculoskeletal disorder. Fifteen age-matched control participants were recruited (9 female, median age 70 years, interquartile range 50 to 74 years). They had no history of vestibular or balance disorders and had a normal neuro-otological examination. The vHIT was used to quantify the VOR function in both the patients and controls.

4.2 Behavioural measures

All participants completed 2 questionnaires prior to their participation: the Dizziness Handicap Inventory (DHI) to provide information about the handicap derived from their dizziness (Jacobson & Newman, 1990), and the Vertigo Symptom Scale (VSS) to identify their vertigo symptoms (Yardley et al., 1992).

After completion of the questionnaires, subjects completed a Visual Dependence task using the rod and disc task, as used in previous studies (Cousins et al., 2014; Roberts et al., 2016).

The patients sat in front of a laptop where they viewed the screen through a 30 cm deep viewing cone. On the screen, a vertical white line (rod) was surrounded by 220 white dots. The rod was off-set and the task consisted of aligning the rod to a vertical position using keyboard arrows. This task was completed in three conditions: (1) with the surrounding dots not moving or “Static” (S), (2) with the surrounding dots moving at 30deg/sec in a “Clockwise” (CW) direction, and (3) with the dots going around the rod at 30deg/sec in a “Counter-clockwise” (CCW) direction ((Cousins et al., 2014), Figure 1). Once the person had agreed with the vertical alignment of the rod, this was offset to be realigned

to the vertical position. This procedure was repeated such that a total of 6 trials were completed per condition. The stating angles were 20, 40, 70, -20, -40 and -70, and were presented randomly between the conditions. The errors in degrees to the right or left of the vertical position were recorded and then averaged per condition. The error in the S condition was also known as the Subjective Visual Vertical (SVV). To obtain a Visual Dependence (VD) score, the average error in the CW and CCW conditions was subtracted from the S condition and subsequently averaged for a single value (Figure 2).

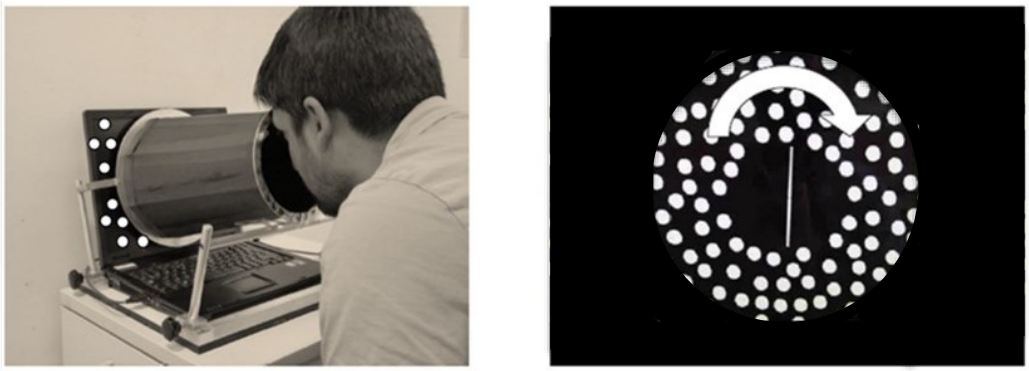


Figure 1

Image taken from (Cousins et al., 2014). Visual dependency protocol. The left image shows the setting of the laptop and the cone where the subjects had to align the rod. The right side shows the image the subjects saw and a white arrow representing the direction that the dots moved around the rod in the CW condition.

$$Visual\ Dependence\ = \frac{(|\underline{CW - S}| + |\underline{CCW - S}|)}{2}$$

Figure 2

Equation to calculate the VD. CW = Error in angles from vertical position for clockwise rotation of the visual background. CCW = Error in angles from vertical position for counter-clockwise rotation of the visual background. S = the error from vertical position for a static visual background (SVV).

4.3 EEG acquisition and conditions

All subjects then underwent a 32 channel EEG recording (Waveguard^{TM} cap, ANT Neuro, Enschede, The Netherlands) with a sampling rate of 1250Hz in three positions: (1) Seated (Sit), (2) Standing with feet 20cm apart (FA), and (3) Standing with feet together, separated by 2cm (FT). This last condition, however, was not included in the analysis due to the data being noisy, and therefore unreliable. The foot positioning was marked to ensure consistency across conditions, and between participants. Additionally, each of these three positions was completed with eyes open (EO) and eyes closed (EC) for a total of 6 conditions (Sit-EO, Sit-EC, FA-EO, FA-EC, FT-EO, FT-EC). Each condition had two recordings of 2 minutes, for a total recording period of 24 mins). The Seated position was always completed first, but the eyes open or closed order was randomised within subjects. The Standing conditions order was randomised among subjects to limit a possible order effect. All subjects had a rest of 1-2 minutes in between each trial. During the recording, an evaluator remained in the Faraday room with each patient for safety reasons.

4.4 EEG processing

The EEG data were pre-processed using methods as described in previous work (Ibitoye *et al.*, 2021) within the EEGLAB toolbox (Delorme & Makeig, 2004). The first and last 10 seconds of each recording were discarded. A 100 Hz low-pass filter was applied to eliminate high frequency noise. Additionally, the 50 Hz noise line was removed using an adaptive filter. The individual subject data was concatenated across conditions, and then independent component analysis (ICA) was applied for further denoising. The resultant components were automatically classified using ICLabel - a pre-trained classifier (Pion-Tonachini *et al.*, 2019). Non-brain components such as muscle contraction, eye movements or blinking, were identified using ICA to be later rejected on the basis of thresholds as defined from the ICLabel training dataset (Pion-Tonachini *et al.*, 2019).

The alpha frequency band was defined as 8 - 13 Hz. For each channel, the mean power spectrum density (PSD, microvolts^2/Hz, hereon referred to as power) across each recording was quantified (Delorme & Makeig, 2004). Individual peak alpha EEG frequencies were also determined for each recording at channel Oz by undertaking a Fourier transform

of the EEG data and then determining the maximum amplitude peak in the frequency range of 7 - 14 Hz. We hereon refer to this measure as the *peak alpha frequency*.

4.5 Statistical analysis

The statistics were evaluated for significance across the whole scalp (defined as all channels excluding M1 and M2 [mastoids]).

All of the statistical analysis was conducted in SPSS® Statistics 27 (IBM® Corp.). Independent t-tests were used when comparing a single variable between groups. A 2-way repeated measures analysis of variance (ANOVA) was performed on the alpha band (8-13Hz) power data of the standing and sitting positions separately for the eyes open and eyes closed conditions as the first factor and group as the second factor. A Pearson correlation was used to identify possible relationships between the variables, and stepwise regression analysis was used to identify the elements contributing to the alpha reactivity.

5. Results

5.1 Clinical features

The peripheral vestibular function, measured by the vHIT, showed a significantly reduced function in the patients, as expected (mean 0.54, standard deviation 0.26, t: -58.453, $p<0.001$, Table 1). The questionnaires showed how the patients reported more handicap related to their dizziness symptoms compared to the controls (t:6.561, $p<0.001$ for DHI, and t:3.190, $p<0.01$ for VSS, Figure 3).

5.2 Alpha Power, group and vision

5.2.1 Seated position

When comparing the average alpha power in the seated position, there were no significant differences between the patients and controls for EO (t:-1.732, $p=0.083$, Figure 4), but a significant variation was found for EC (t:-3.174, $p<0.01$, Figure 4), where the patients showed reduced alpha power compared to the controls.

ID	Age	Sex	Aetiology	VHIT	SVV / °	VD / °
1	67	F	Idiopathic	0.28	1.5	10.0
2	77	M	Idiopathic	0.59	1.0	25.5
3	54	M	Meniere's disease	0.98	0.5	6.4
4	70	M	Idiopathic	0.73	-2.7	7.2
5	36	F	Idiopathic	0.66	0.7	8.6
6	68	F	Idiopathic	0.79	1	11.5
7	74	M	Idiopathic	0.69	-2.9	15.6
8	48	M	Idiopathic	0.69	1.0	3.0
9	48	F	Idiopathic	0.2	6.1	11.7
10	70	F	Idiopathic	0.74	1.9	15.4
11	72	F	Gentamicin	0.05	1.1	19.1
12	17	F	Idiopathic	0.72	0.4	1.0
13	84	F	Idiopathic	0.41	-0.5	24.3
14	74	F	Idiopathic	0.39	0.0	12.2
15	71	M	Idiopathic	0.85	-0.2	4.8

Table 1

Patient characteristics: Age, Sex, Aetiology of the Bilateral Vestibular Hypofunction (BVH), video Head Impulse Test (vHIT) gain, Subjective Visual Vertical (SVV) and Visual Dependence (VD).

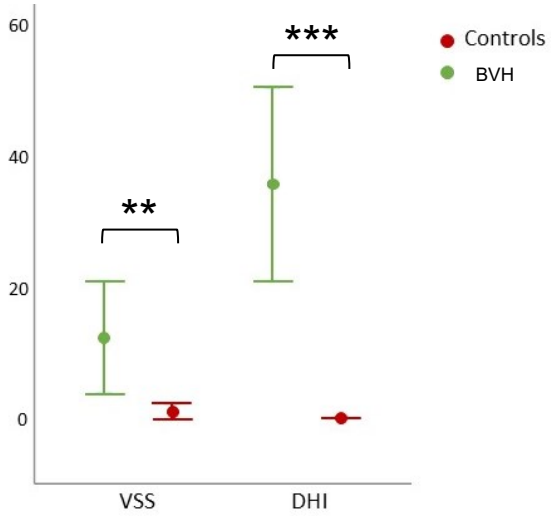


Figure 3

Questionnaire scores for patients with BVH and controls. Bars represent 95% confidence intervals. ** p<0.01, *** p<0.001.

There was a significant main effect of enhancement of alpha power with eye closure in all regions; it was most marked in the occipital areas, in both the patients and controls ($F(1,2046)=995.402$, $p<0.001$, Figure 4). The interaction between groups was significant, meaning the alpha enhancement with eye closure was smaller in the patients compared to the controls ($F(1,2046)=11.582$, $p < .001$, Figure 4).

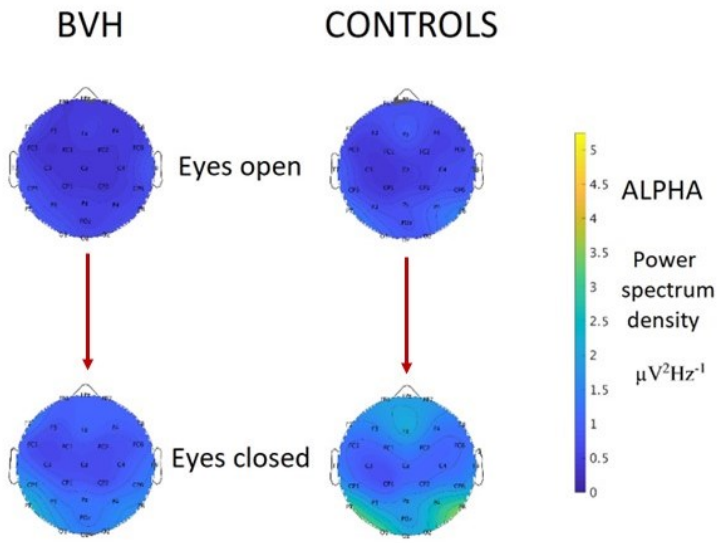


Figure 4

Alpha EEG power in patients with BVH and controls in sitting position. Head plots of average alpha EEG power spectrum density are illustrated with eyes open (top row) and eyes closed (bottom row), for patients with BVH (left column) and healthy controls (right column). The effect of eye closure on EEG power is shown by red arrows.

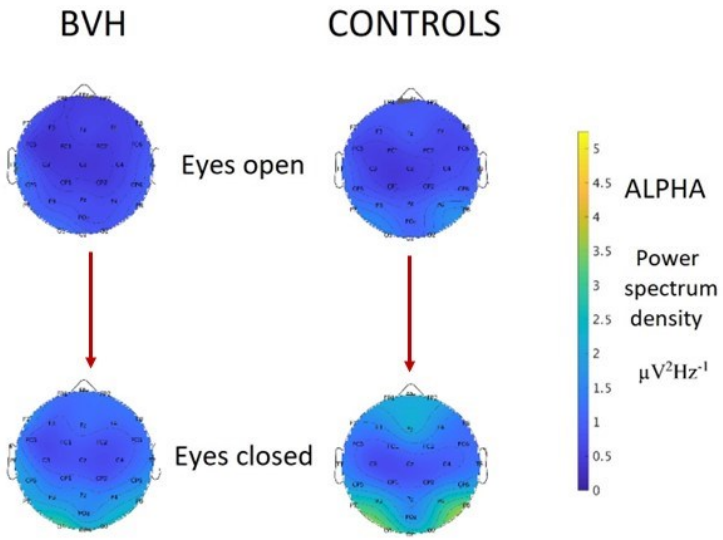
5.2.2 Standing position

As mentioned in the methods section, the Feet Together condition was unfortunately too noisy due to increased sway in the subjects and has not been included in the analysis. The standing with feet apart data condition was the one used as the only standing position.

The total average alpha power in the standing position was not significantly different between the patients and controls for EO (t:0.03, p=0.976) or EC (t:-0.397, p=0.346, Figure 5).

Similarly to the seated position, there was a significant main effect of eye closure on increase in alpha activity ($F(1, 2046)=689.200$, $p<0.001$, Figure 5). However, the interaction between groups was not significant ($F(1, 2046)=0.077$, $p=0.782$, Figure 5).

Figure 5



Alpha EEG power in patients with BVH and controls in standing position. Head plots of average alpha EEG power spectrum density are illustrated with eyes open (top row) and eyes closed (bottom row), for patients with BVH (left column) and healthy controls (right column). The effect of eye closure on EEG power is shown by red arrows.

5.3 Alpha reactivity and VOR gain

Further analysis of the factors influencing alpha enhancement on eye closure was performed in the group of patients with BVH by correlating the alpha power reactivity with the VOR gain measured with the vHIT. The sitting and standing positions were then analysed separately to identify whether this correlation was present on both conditions. Borderline significant correlations were observed between the alpha reactivity and vHIT gain in patients in the seated position ($r:0.590$, $p=0.07$) and the standing position ($r:0.514$, $p=0.08$).

5.4 Alpha reactivity and questionnaires.

To identify whether there was a relationship between the visual alpha EEG enhancement and chronic dizziness symptoms and handicap in the patients, a correlation was performed with the questionnaires scores. Again, both the sitting and standing positions were combined to have a general idea of this relationship. In the combined data, neither the DHI nor VSS significantly correlated with the alpha EEG enhancement (r:-0.127, p=0.497 for DHI and r:-0.139, p=0.457 for VSS). Subsequently, the two positions were separated to identify whether a correlation was present on one or both. In the seated position, the DHI and VSS scores did not significantly correlate with the EEG enhancement (r:-0.234, p=0.401 for DHI, r:-0.188, p=0.502 for VSS). Similarly, no relationship with DHI (r:-0.048, p<0.860) or VSS (r:-0.101, p=0.709) was observed when standing.

5.5 Alpha reactivity and visual dependency

The patients with BVH evidenced a higher visual dependence compared to the controls (mean: 11.3°, s.d.: 7.2° for patients and mean: 6.5°, s.d.: 5.3° for controls, t: 2.844, p<0.01; Figure 6).

The relationship between visual dependence and alpha EEG enhancement on eye closure was investigated in the BVH patient group. In both positions combined, visual dependence correlated negatively with alpha EEG enhancement on eye closure (r:-0.430, p<0.05), meaning patients who showed greater visual dependence had less alpha visual reactivity. The controls instead showed a positive nearly significant correlation between visual dependence and alpha EEG enhancement (r:0.320, p=0.074). In summary, greater visual dependence correlated with less alpha EEG enhancement in the patients, whereas this relationship was reversed in the controls (greater visual dependence correlated with more alpha EEG enhancement).

When separating the conditions, these results were maintained in the standing position (r:-0.516, p<0.05), meaning that in patients with BVH, greater visual dependence is linked to less alpha EEG enhancement on eye closure.

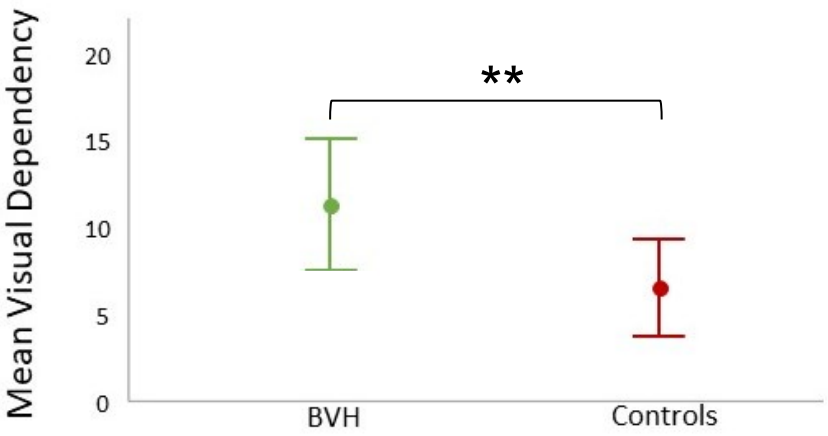


Figure 6

Visual dependence in degrees for patients with BVH and controls. Bars represent 95% CI. ** $p<0.01$.

5.6 Multivariable analysis

A regression stepwise was performed to identify the variables contributing to the EEG alpha reactivity between visual dependency, VOR gain, questionnaires scores and age. Visual dependence, vHIT and DHI were identified as contributors to the EEG alpha reactivity in all subjects and all conditions (R^2: 0.108, $p<0.001$). This result confirms that visual dependency is linked to the differences in alpha frequency reactivity, but also that VOR gain and the perceived handicap modulate the increase or decrease in brain activation with eyes closed.

6. Discussion

In the present study we investigated the relationship between visual alpha EEG reactivity and loss of peripheral vestibular function. The patients had significantly lower alpha enhancement when closing their eyes (i.e. reduced alpha reactivity), compared to the controls. Interestingly, this was more prominently observed when seated than standing, suggesting a generalised change in brain reactivity when peripheral vestibular inputs are lost. vHIT gain was shown to be correlated with visual reactivity, meaning that residual VOR function is linked to alpha reactivity. Finally, the patients showed more visual dependence - a measure dependent on cortical visuo-vestibular interactions –compared to the controls, and this visual dependence was inversely linked to alpha reactivity. The current results suggest that vestibular dysfunction impairs alpha visual reactivity and highlight a link between visual dependence, as a measure of multisensory integration, and visual alpha reactivity.

Alpha frequency oscillations are involved in the control of cortical activity and excitability. Alpha oscillations propagate from higher to lower order sensory cortices (Halgren et al., 2019) in keeping with their role in the top-down control of sensory processing (Haegens, Händel & Jensen, 2011). Local increases in alpha activity lead to reductions in cortical excitability (Sauseng et al., 2009), metabolic activity (Cook et al., 1998), and blood flow (Goldman et al., 2002), likely mediating the suppression of task-irrelevant processing (Foxe & Snyder, 2011), in turn meaning better cognitive performance (Haegens et al., 2010; Meeuwissen et al., 2011). Changes in alpha oscillatory activity thus reflect neurophysiologic processes relevant to performance and cognition.

No significant differences were observed in the average alpha EEG power between the patients with bilateral vestibulopathy and the controls except in the sitting position with EC. However, a previous study by (Gale et al., 2016) tested patients with BVH and, similarly, found no significant differences in alpha power between the groups when they were seated with eyes open in the dark (Gale et al., 2016). The increase in alpha power in the healthy controls was higher than in the patients, which could account for the difference observed in the current results. Perhaps the effects of keeping the eyes open in the dark were not comparable to eye closure. Previous literature has shown how the pattern of brain activity

recorded using fMRI with eyes open in darkness differs from that one observed with eyes closed (Hüfner et al., 2008; Marx et al., 2003), the latter being associated with the activation of vestibular areas (Marx et al., 2003). Accordingly, the differences observed between the current results and previous work could be due to methodological variations.

The present investigation found less visual enhancement of alpha EEG power in the patients with BVH compared to the controls. Additionally, the correlations and the regression analysis showed a link between VOR gain and visual alpha EEG enhancement in the BVH patient group. These results support a general effect of vestibular loss on visual reactivity in the alpha rhythm, and also suggest that this effect could depend on the degree of vestibular loss. The fact that the differences between the patients and controls were observed in frontoparietal areas suggests that the neurophysiologic effects of vestibulopathy on alpha activity are not specific to the visual cortices, but likely involve more generalised attentional networks (Misselhorn, Friese & Engel, 2019). Given the established link between visual alpha reactivity and visuospatial attention (Van Diepen, Foxe & Mazaheri, 2019), less visual reactivity in the alpha rhythm in patients with BVH would imply or reflect disruption in their visuospatial processing (see paragraph below).

Abnormal visuospatial processing has been observed in patients with BVH through measures of biases in the subjective vertical (Bisdorff et al., 1996), such as 'visual dependence' (Bronstein et al., 1996). The present results confirmed that the patients with BVH were more visually dependent than the controls. Other evidence of impaired visuospatial processing in BVH patients has been seen in visuospatial tasks such as navigation, where patients with BVH were tested using the Morris water maze task (Hamilton, Driscoll & Sutherland, 2002) and exhibited significantly poorer spatial memory and navigation skills (Brandt et al., 2005).

The present findings regarding the relationship between visual dependency and EEG alpha reactivity suggest that visual dependence influences (or is influenced by) visual reactivity in alpha oscillations. Although it is well known that visual dependence increases following vestibulopathy (Bronstein et al., 1996), the neural basis and neurophysiological associations of this measure have not yet been fully explained (Roberts *et al.*, 2018). A recent study identified a bi-hemispheric network connected to parieto-insular vestibular

areas, as a substrate for the integration of visual and vestibular information (Ibitoye et al., 2022). The current findings suggest that visual reactivity in the alpha rhythm is a neurophysiologic correlate of visual dependence in bilateral vestibulopathy.

The relationship between visual dependence and alpha reactivity differed between the patients and controls. A negative correlation between the two variables was found in the patients, but a positive correlation in was found in the controls. This opposite relationship suggests that different mechanisms underpin the relationship between visual dependence and visual alpha reactivity in the patients compared to the controls. In health, the trend observed where individuals with greater visual reactivity in the alpha frequency seemed more visually dependent would suggest that visual alpha reactivity is a measure of how the visual system's excitability corresponds with an overweighting of visual motion information. However, this correlation was borderline significant, more data is needed to confirm this theory.

On the other hand, for patents with BVH, apparently the loss of ascending vestibular signals - necessary for normal inhibition of visual cortices (Brandt et al., 1998) – generates a reverse process to the one happening 'in health'. Accordingly, the loss of vestibular inputs would imply less visual alpha EEG enhancement on eye closure (i.e. less inhibition of visual cortical activity (Goldman et al., 2002), and a parallel re-weighting of multisensory inputs, different from the vestibular, and more focussed on visual information (i.e. more visual dependence). This enhanced reliance on vision, which is linked to the observed changes in cortical activity, could be contributing to the increased balance difficulties experienced by patients with vestibular disorders, when vision is denied.

7. Conclusions

The current investigation showed how impairment of the peripheral vestibular function reduces visual reactivity in the alpha EEG frequency. Furthermore, this reduced reactivity was linked to the magnitude of such loss and visual dependence levels in patients. The current findings open up new research possibilities to fully unveil the influence of vision in the central compensatory mechanisms following a peripheral vestibular insult. Finally, the changes in brain activity observed in this investigation support the idea of a restructuring process occurring in the cortex to help postural control after a lesion, which could hopefully, in the future, be used as a diagnostic or prognosis marker and a guide for rehabilitation.

8. References

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General discussion

1. Final comments

The experimental work conducted over the course of this PhD and presented in this thesis sought to better understand the underlying mechanisms involved in postural control in health and disease.

Posture was initially thought to be a purely subcortical function, mainly driven by reflexes. Still, in the last few decades, there has been an appreciation of the various cortical networks required for this critical function. Despite the evident cortical role for postural control, the specific areas and processes involved have yet to be fully elucidated. The amount of research on the brainstem mechanisms controlling vestibulo-ocular and vestibulo-spinal function is an order of magnitude higher than that on cortical vestibular processes.

The findings detailed in this thesis illustrate how postural control – lower and higher levels – constructs a perception of stability that is fed by afferent (objective) body signals but is also very dependent on other factors such as anxiety, brain activity during eye closure, hemispheric dominance, cerebral white matter hyperintensity load and expectations/beliefs about one's postural control. This fact gives postural control a significant cortical lead, which was possible to observe in the effect that vestibular cortex asymmetry has on balance performance, in how the visual cortex modifies its activity when the vestibular peripheral organ input is impaired and how cortical disconnection syndromes can cause permanent dizziness and imbalance.

2. Subjective instability perception

In Chapter 1, I concluded that ID patients exhibit a somewhat distorted perception of instability. The question is whether this finding is a disorder of perception, a sensory integration deficit or a functional symptom. Imaging and EEG studies performed in older individuals with idiopathic dizziness have revealed a role for cerebral white matter

abnormalities in the generation of persistent dizziness and instability symptoms and, indeed, albeit clinically, their balance performance (Ibitoye et al., 2022; Ahmad et al., 2015). Such findings hint at a dysfunction of the central multimodal integration necessary to maintain body posture. Although postural abnormalities are not always evident in patients compared to healthy controls, in the current project, a subclinical postural abnormality was observed in their stepping response, showing what may be labelled as a more sensitive stepping threshold could also be feeding their heightened perception of instability.

However, the postural abnormality I found in these patients does not tell the whole story of how subjective instability perception is generated. The fact that the perceived subjective instability can be modulated in healthy young individuals (Chapter 2) suggests additional factors that are not fully accounted for by simple objective postural impairments. Indeed, in Chapter 2, it was possible to observe how watching a stable or unstable performance before performing a postural task actually modified anxiety levels towards the task and the degree to which objective and subjective instability measures correlate. It is thus inevitable to speculate that if healthy young subjects, without postural difficulties and with no task-related anxiety, show changes in their perceived and actual performance, it is quite feasible to expect modulation of these responses in older patients with balance difficulties and increased anxiety when facing challenging postural tasks. Expectations, then, are a further element to factor in when considering how the perception of instability is generated. Interestingly, in the group of young individuals, it was shown that the expectation of a very challenging task that was, in fact, not challenging (i.e. mismatch) generated a more “relaxed” stance and a lower sense of anxiety and instability, breaking the tight relationship between objective and subjective instability (Chapter 2). However, it is unclear whether this is also the case for patients. That is an interesting question and the starting point to investigate whether the different reactions to priming could be a distinctive measure in various balance disorders. Further work is required to explore whether modulating expectation through priming can enhance the effectiveness of the rehabilitation of balance disorders across neurological or older populations, including those with permanent instability perception and those with high levels of anxiety and fear of falling.

3. Expectations' role in objective and subjective postural control

Prior knowledge of an upcoming postural challenge has previously been shown to modify performance. Even in purely motor tasks, subjects change their execution based on the information given to them as priming (Grossi, Maitra & Rice, 2007) or feedback (Lamarche, Gammage & Adkin, 2011). In this project, it was observed how prior information given to subjects by either visual or verbal modality generated expectations, which in turn modified their objective performance and subjectively perceived instability (Chapter 2). Accordingly, these findings are not completely surprising, as emotional components have been seen to influence posture (Young et al., 2016; Carpenter et al., 2004; Castro et al., 2019b; Ellmers, Kal & Young, 2021; Cleworth & Carpenter, 2016; Wada, Sunaga & Nagai, 2001). Interestingly, however, the visual prime generated an effect that was the opposite of what was expected. The fact that subjects watching an unstable task performance swayed more suggests that it is possible to modify a postural motor plan to account for the revised difficulty of a task faced. It is important to note that this was observed in healthy young adults, suggesting that expectations are essential in the generation of a motor plan to face postural challenges, but that these are not fixed and can be adapted to accommodate any new need presented. This flexibility might be an essential characteristic, and then it would be relevant to know whether patients with high anxiety levels, or a functional neurological or vestibular disorder (e.g. PPPD) are able to permanently modify their postural adjustments based on the peripheral input feedback and the constant revision of their expectations with the actual experience. Accordingly, the next step would be to investigate how priming affects the objective performance of patients with balance disorders and different emotional involvement.

An interesting finding was that verbal priming, as presented in this current thesis, modulated healthy young adults' perception of instability and task-related anxiety in the absence of an objective body sway change. That is, it dissociated or distorted the usually fairly tight coupling between objective and subjective instability levels. Knowing that verbal information related to the difficulty of an upcoming postural task is able to modify an individual's perception of their objective performance opens up a new line of investigation. Clarifying the effect of verbal information on patients with different degrees of vestibular

emotional impairment could open up new rehabilitation avenues for these conditions, where physical activity can be combined with more cognitive approaches.

Although both priming modalities (verbal and visual) generated a degree of modulation of either objective or subjective instability (or both), watching an 'unstable' actor generated an expectation of instability that was 'mismatched' to the actual experience, which reduced the perception of instability, despite the greater objective sway. Conversely, experiencing the task after receiving verbal information, in fact, increased their perceived instability. Suppose that different priming modalities have other effects on objective and subjective postural control. In that case, future research should also focus on identifying those differences that make the best use of these tools for the rehabilitative treatment of balance disorders.

4. Right hemispheric dominance

Another interesting result described in this thesis (Chapter 3) is the effect of hemispheric vestibular dominance upon postural control. Cathodal tDCS applied to the left posterior parietal cortex inhibited the vestibular cortical activity, as evidenced in the reduced response to caloric stimulation post tDCS and then the unveiling of the underlying right hemispheric dominance. Conversely, anodal stimulation did not seem to affect cortical activity, as the nystagmic response to caloric stimulation was not significantly suppressed, as observed in previous investigations (Arshad et al., 2015a; Arshad, 2017a; Bednarczuk et al., 2017). In young, healthy individuals, a higher degree of right hemispheric dominance - measured using the Nystagmus Suppression Index - was associated with a reduced body sway following tDCS. This supports the notion that the right hemispheric vestibular cortex plays a relevant role in postural control. This is the starting point to understanding how right hemispheric dominance could modulate posture in patients with balance disorders. It has previously been shown that anxiety is also related to the degree of hemispheric dominance (Bednarczuk et al., 2018). Accordingly, it is not unreasonable to think that postural impairment and higher levels of anxiety could somehow be linked to hemispheric dominance in patients. If this were the case, a new avenue for treatment based on brain

stimulation would open up, as it has been successfully used in patients with neurological disorders (Nitsche et al., 2008).

I did not observe a correlation between right hemispheric dominance and instability perception in young, healthy individuals (Chapter 3). As such, it needs to be clarified whether hemispheric dominance also contributes to the generation of perceived imbalance in a healthy population. However, as mentioned previously, given that anxiety appears to be linked to this hemispheric dominance, individuals who have high levels of postural anxiety, or "fear of falling" may be a more relevant group in which to explore the relationship between right vestibular hemisphere dominance and balance perception. Investigating how tDCS affects postural control in patients with balance disturbance and task-related anxiety seems like the next step to better understanding its role, contribution, and, hopefully, its potential therapeutic use.

5. Visual cortex excitability

It is already known that head motion input (provided mainly by the peripheral vestibular organ), vision and proprioception are the primary sensory inputs needed to maintain balance. When the peripheral vestibular organ is injured, the vestibular and balance systems experience a reorganisation of the information provided by the remaining sensory inputs of the vestibular system, i.e. proprioception and vision (Herdman, 1998). Accordingly, the role of vision for balance and spatial orientation is upregulated and becomes even more relevant (Bronstein, 2004). By examining subjects with no peripheral vestibular function with electroencephalographic recordings (Chapter 4), it was possible to observe how cerebral cortex excitability is modified when faced with postural challenges. The results in this thesis show potential changes that are made in the cortex to cope with vestibular organ input failure. This diminished enhancement of the alpha rhythm in different parts of the cortex in patients with BVH compared to controls, shows how the loss of peripheral vestibular function has cortical consequences for how the information is processed and weighted. The fact that the individual vestibulo-ocular reflex gain and visual dependence levels contribute to the degree of visual alpha reactivity confirms that these three factors are intimately related. Future studies of vestibular compensation or

treatments could use quantitative EEG as a potential marker of brain reorganisation after a vestibular lesion.

Other cortical areas could also reorganise in response to vestibular input. The changes in other areas might be more subtle, and therefore harder to record or more artefact prone if they involve higher frequency EEG bands. Further research with a larger sample size could provide evidence of whether the reorganisation of the cortex in response to peripheral vestibular organ hypofunction is a generalised process or whether it is more localised. Unilateral lesions, which are far more frequent than bilateral ones, would provide larger patient groups but this initial study, which involved bilateral patients, was more effective as a proof of principle. Whether these changes in the visual cortex could be related to the degree of visual dependence that subjects experience or maybe even to conditions that carry a bad clinical prognosis, such as visual vertigo, is yet to be explored.

As compensation mechanisms are expected to be different depending on disease onset (acute versus insidious), evolution (static, episodic or progressive) and location (peripheral versus central) looking at the changes in brain excitability in vestibular conditions with different characteristics plus patients' subjective report might provide an interesting insight into the compensation mechanisms for each condition and the consequent relevance for rehabilitation.

6. Clinical implications

As previously mentioned, a better understanding of the areas and processes involved in postural control would allow clinicians to complete a better diagnosis and generate and implement more effective treatment plans for people with objective and subjective imbalance.

In the case of older patients with a permanent perception of instability, the current results suggest that it might be helpful to obtain objective/quantitative imaging studies, such as MRI, to identify their white matter hyperintensity volume, which could contribute to their symptomatology. Additionally, including relatively neglected aspects of the balance examination in dizzy patients, like stepping responses, would help identify whether the

patient fits the “idiopathic dizziness” (likely small vessel-related) group, which, in turn, would guide further management.

This thesis has also contributed to our knowledge of perceived instability. As evidenced throughout the investigations, such a process is a complex multifactorial mechanism. Therefore, the assessment should include factors such as anxiety and expectations, as these could modify the way that subjects face different postural tasks and their perceived performance. A clear example of these findings’ applicability is the common use of encouragement with patients to make them think the task they need to face is easy (“this is very simple, you can do it”). Unfortunately, instead of giving them more confidence, it could increase their anxiety and frustration if they cannot complete the task successfully. Accordingly, the information in this thesis could become valuable when generating treatment plans, as it could guide the future use of the information given to subjects to promote proper recovery.

Finally, in terms of cortical areas, this thesis showed how hemispheric dominance influences postural control. In particular, it highlights the neglected issue of considering vestibular hemispheric dominance, especially in patients with brain lesions. Identifying clinical correlates of the right vestibular cortex dominance in patients with vestibular disorders and healthy controls could open novel avenues into tailored rehabilitation plans to promote better balance rehabilitation which could even include tDCS if proven useful. Additionally, understanding the changes generated in the alpha cortical excitability after a bilateral vestibular lesion would allow us to know how the cortical sensory reorganisation works after a peripheral insult and then use VOR and visual dependence as markers of this reorganisation and future recovery. If rehabilitation can address the contribution of vision to postural control after a lesion, maybe alpha reactivity can give us insights into how the cortex compensates for the loss of peripheral function.

7. Future research

The current thesis focused on understanding the contribution of different cortical areas and cognitive elements to postural control. Some of the investigations tested only young, healthy controls as a first approach to identify a possible effect of factors such as

priming and hemispheric dominance on posture. The following steps will be to identify a possible age effect of these elements on postural control and to determine whether these factors contribute differently in older healthy subjects. Subsequently, to translate these findings into clinical practice, it will be essential to study how these elements correlate and contribute to balance in patients with various vestibular disorders. This thesis marks a starting point to investigate possible markers to improve the diagnosis of postural disturbances and potential treatment techniques to restore balance function and subjective instability in those patients.

Furthermore, findings from this body of work may facilitate the development of diagnostic tools for balance impairment across vestibular disorders. Accordingly, the following steps will include replicating the investigations completed in healthy controls in patients with vestibular disorders and hopefully identifying distinctive patterns for different vestibular conditions. For example, the effect of varying priming modalities in patients with postural difficulties and different anxiety levels might be specific to a specific group of patients (say, vestibular neuritis). It could then aid clinical diagnosis of that condition in the future. Similarly, there needs to be more clarity on the contribution of vestibular cortical dominance to the development and presentation of postural disturbances in patients with vestibular conditions. Consequently, clarifying how this dominance influences balance impairment in patients would add another marker to benefit the diagnostic process. Along the same lines, describing the contribution of brain activity and imaging abnormalities in patients with postural impairment would also allow us to complement the clinical assessment and diagnosis.

8. Conclusion

In summary, I hope that the investigations completed during this PhD have contributed to understanding the role of cerebral mechanisms in objective and subjective postural control. Hemispheric dominance, white matter hyperintensity volume, alpha visual reactivity, anxiety and previous balance-related experiences are additional factors to consider when diagnosing and treating objective and subjective balance impairments. Further research into the specific contribution of these elements to posture in different vestibular conditions is the next step.

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Publications

1. Publications related to this thesis

1.1 Article related to Chapter 1

In preparation for submission to a scientific journal. Article currently under review on Nature Aging.

1.2 Article embedded in Chapter 2:

Castro, P., Papoutselou, E., Mahmoud, S., Hussain, S., Bassaletti, C.F., Kaski, D., Bronstein, A. & Arshad, Q. (2022) Priming overconfidence in belief systems reveals negative return on postural control mechanisms. Gait and Posture. 94, 1–8. doi:10.1016/J.GAITPOST.2022.02.015.

1.3 Article embedded in Chapter 3:

Castro, P., Kaski, D., Al-Fazly, H., Ak, D., Oktay, L., Bronstein, A. & Arshad, Q. (2019) Body sway during postural perturbations is mediated by the degree of vestibulo-cortical dominance. Brain Stimulation: Basic, Translational, and Clinical Research in Neuromodulation. 12 (4), 1098–1100. doi:10.1016/J.BRS.2019.05.008.

1.4 Article related to Chapter 4:

Ibitoye RT, **Castro P**, Ellmers TJ, Kaski DN, Bronstein AM. (2023) Vestibular loss disrupts visual reactivity in the alpha EEG rhythm. Neuroimage Clinical. 11 (39):103469. doi: 10.1016/j.nicl.2023.103469. Epub ahead of print. PMID: 37459699.

2. Other publications

Barbosa, P., Kaski, D., **Castro, P.**, Lees, A.J., Warner, T.T. & Djamshidian, A. (2019) Saccadic Direction Errors are Associated with Impulsive Compulsive Behaviours in Parkinson's Disease Patients. Journal of Parkinson's Disease. 9 (3), 625–630. doi:10.3233/JPD-181460.

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San Pedro Murillo, E., Bancroft, M.J., Koohi, N., **Castro, P**. & Kaski, D. (2022) Postural misperception: a biomarker for persistent postural perceptual dizziness. Journal of neurology, neurosurgery, and psychiatry. jnnp-2022-329321. doi:10.1136/JNNP-2022-329321.

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