Can Electroencephalography (EEG) and Electromyography (EMG) be used to explain the adaptation of the feedforward postural response to repeated continuous postural perturbations

Caitlin Beard
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A thesis submitted in attainment of the requirements of the Manchester Metropolitan University for the degree of Master of Science (MSc by Research)

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Table of Contents

List of Abbreviations

ACC	Anterior Cingulate Cortex
AP	Anteroposterior
BOS	Base of Support
CNS	Central Nervous System
COG	Centre of Gravity
COM	Centre of Mass
EIP	External Initiated Perturbation
EEG	Electroencephalography
EMG	Electromyography
ERP	Event-Related Potential
ERN	Error-Related Negativity
GM	Gastrocnemius Medialis
PECR	Perturbation Evoked Cortical Response
PEP	Perturbation Evoked Potential
PC	Postural Control
R/L	Right/Left
TA	Tibialis Anterior

List of Figures

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Declaration

I declare that any of the work that is submitted throughout this thesis is my own and has not been submitted from any other degree or qualification. I have ensured any studies or references have been acknowledged appropriately.

Caitlin Beard

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1. Abstract

Introduction: A sudden balance perturbation is often accompanied by postural instability which can be causal to falls. However, repeated exposure to such balance perturbations is characterised by improved balance control, through adaptation of the reactive and/or feedforward postural responses. The oscillating platform paradigm provides an interesting and meaningful experimental approach that elicits both reactive and anticipatory responses when exposed to a few sinusoidal cycles within a trial-by trial basis. The N1 cortical response has been investigated as a function to different biomechanical characteristics of perturbations, however, research is limited regarding the N1 modulation and its contribution to the maintenance and adaptation of postural responses.

Aims: The aim of this thesis was to investigate how feedforward postural responses indicate adaptation to repeated continuous transient balance perturbations, through postural muscle organisation and changes in the N1 potentials relative to the onset of the perturbation and changes of frequency.

Hypothesis: It was hypothesised that postural adaptation would be manifested over numerous repeated trials, evidenced by reduced muscular activation relative to platform direction (anteroposterior), changes in centre of pressure metrics (reduced peak velocity), and a reduction in initial N1 potentials and amplitudes which reflects a decrease in cognitive workload over trials.

Methods: Twenty-one healthy young adults (12 males, 9 females; mean ± SD = 27 ± 4.83 years) participated in this study. Each participant was instrumented EMG electrodes (2000 Hz) on the GM and TA muscles on both legs. Participants were fitted with a 32-channel EEG cap (1000 Hz). Participants were stood on the oscillating platform barefoot and shoulder-width apart moving in an anteroposterior direction at 0.25 Hz for 10 cycles and 0.5 Hz for 15 cycles per trial (each trial lasting 95 seconds to 1-minute). Force plates embedded into the platform recorded ground reaction forces (1000 Hz) in which all relevant centre of pressure metrics were obtained. Postural adaptation was measured over the 10 repeating trials. Statistical analyses were performed using repeated measured (ANOVA) via SPSS statistics with within-subject factor of trial (T1-T10). Polynomial trend analyses found significant effects and post-hoc analyses were performed using pairwise comparisons.

Results: The N1 amplitude and EMG activity of both the TA and GM showed significant adaptation over the 10 trials with both of them dramatically reducing in terms of the onset of

the perturbation and change of frequency, this was also the case for COP dynamics which began with a large acceleration in both anteroposterior directions which reduced over time.

Discussion: This study demonstrated a significant interaction between the cortical N1 potential, muscular activation, and COP dynamics during postural equilibrium, providing further insight into neural control of balance. Both N1 amplitude and muscular activation reduced over time indicating the collaborative work of the neuro-muscular system in terms of balance control. COP adjustments in the backwards direction were significantly faster than in the forwards direction which indicates directional response properties of the balance control system. The results emphasise the efficiency of neuro-muscular coordination relative to repeated exposure to continuous perturbations, which is characteristic of adaptive processes occurring within the response strategies. Future research should be directed at investigating such responses in other populations (e.g. ageing and neurodegenerative disorders) and rehabilitative strategies that focus on adaptive postural control.

2. Introduction

2.1 Mechanisms in balance and Postural Control

Compensation for postural instability from predictable and unpredictable balance perturbations is vital when preventing the risks of accidents and falls and so it is highly essential for humans especially in daily life. Balance has been generally defined as "the control of the body's position in space for purposes of balance and orientation" (Sousa, 2012), and is traditionally biomechanically defined as the "ability to maintain the body's projected centre of mass (COM) within the limits of the base of support (BOS)" (Bugnariu, 2005). In regard to this, balance is usually sustained when COM is maintained within the BOS and can be identified as 'static' or 'dynamic' balance and is most commonly found in conditions of walking, standing or sitting (Forte et al, 2014). Static balance is referred to as maintaining a position with minimal support and the maintenance of an individual's centre of gravity (COG) within their BOS whilst keeping an upright position throughout quiet stance (Seyed and Asghar, 2010). On the contrary, dynamic balance manifests maintaining a stable BOS whilst completing a necessary movement, or the maintenance of upright posture whilst an individual's COG projects outside of the BOS (Winter et al, 1990; Seyed and Asghar, 2010), indicating that dynamic balance is fundamental when it comes to the execution of maintaining postural control (PC) after the loss of balance in unpredictable conditions, and is therefore essential in preventing the risk of falls and/or in extreme cases loss of mobility (Neptune and Vistamehr, 2019).

Previous research has demonstrated that when balance is disturbed by superficially initiated perturbations, centre of pressure (COP) displacement is modified, performance feedback is triggered and postural muscle activity is increased, growing attention to the fact initial muscle activity is ineffective and would require a substantial effort from lower limb extremities (Kennedy et al, 2013), particularly the Gastrocnemius Medialis (GM) Tibialis Anterior (TA) and the Soleus (Nardone et al, 1990: Gollhofer et al, 1989; Kennedy et al, 2013; Patel et al, 2009). The disequilibrium that the support surface creates leads to a continuous acceleration or sway (postural sway) to the body in a forward-to-backward (anteroposterior) direction, and as follows, the appropriate muscles and torques are applied to counteract these movements and create a familiar state of balance, therefore when an individual is suddenly displaced due to a perturbation, inertial reaction forces take control and reflex of the lower limb muscles

stretched by said perturbation contribute significantly to restoring equilibrium (Nardone et al, 1990; Nashner et al, 1976).

Human posture or 'bipedal stance' is often referred to as a single inverted pendulum as it continuously pivots around the axis of the ankle joints (Gunther and Wagner, 2016; Winter et al, 2001). The body's COM is generally maintained within a few centimetres of the anterior of the ankle joint during quiet stance, the body's COG acts within the stated pendulum and creates a forward torque in which the ankle plantarflexor muscles work simultaneously to create an elasticated element that pulls the pendulum backwards to counteract the gravitational torque and keep the COM within the necessary BOS and prevent toppling (Sasagawa et al, 2009). Research has identified that the constant development of dynamic balance is driven by a 'feedforward' response within an approximate period of time of 1 second however, beyond this time 'feedback' mechanisms are working considerably in order to control 'quiet' stance (Gunther et al, 2011; Gatev et al, 1999).

2.2 Systems Contributing to Balance

Postural control is regarded as a complex motor skill consisting of the interaction of multiple sensorimotor systems working simultaneously to regain and maintain balance. The Central Nervous System (CNS) plays an imperative role in executing the right responses and coordinating activity through different muscles and joints using sensory feedback which stems from the Somatosensory system, the Visual system and the Vestibular system (Horak, 2006; Gaerlan et al, 2012; Mills, 2018). Each of these responses must be unique and specific to interpret the appropriate control in different tasks and situations that may occur when disturbing equilibrium (Akram et al, 2008). The core components are the input that each system (somatosensory, visual, and vestibular) provides, i.e. feedback from the location and movement of objects in the external environment that has caused disturbance, orientation and location of the head and body in space, the relativity and locomotion of the body segments, higher order supraspinal centres that plan, initiate and perform appropriate movements based on desired objectives, final compensation and multisensory input, and lower order subcortical centers such as brainstem nuclei and spinal cord that integrate the correct motor commands with multisensory feedback to ensure the intended and automatic actions correspond to appropriate postural adjustments (MacKinnon, 2018).

As an environment changes for an individual, their sensory environment must change alongside it by reweighting their relative dependence on each system. When in an environment that is well lit and has a firm BOS, a healthy person relies on 70% somatosensory, 10% visual, and 20% vestibular information (Peterka, 2002; Horak, 2006). Contrarily, when stood on an unstable surface, surface somatosensory information is decreased and sensory weighting is passed onto visual and vestibular information for appropriate postural orientation (Horak, 2006). Therefore, the ability to counteract and re-weight sensory information depending on the context of sensory output is essential when maintaining stability. Individuals with peripheral (visual), vestibular, or somatosensory loss may struggle when re-weighting postural sensory information and are therefore at a higher risk of falls or accidents. However, it has been suggested that in conditions regarding peripheral neuropathies, balance may be arguably better within dynamic conditions such as standing on a perturbating balance platform that is continuously moving in an anteroposterior direction than under quiet bipedal stance. This is possibly due to other inputs and pathways that may step in and play a role to provide crucial information processing (Nardone et al, 2007). In addition, when a platform moves in an anterior-posterior direction it is usually an adaptable and predictable condition and so a feedforward control is activated due to the fact central fusion of somatosensory, visual, and vestibular information creates an effective production of necessary anticipatory controls to contradict disequilibrium (Nardone et al, 2007; Schieppati et al, 2002).

2.3 Reactive and Anticipatory control Mechanisms

Human movement and postural control can be measured in different environments whether external or internal. Both predictable and unpredictable perturbations are measured and usually compensated with reactive and anticipatory control mechanisms (Mills, 2018), both essential to prevent or reduce the risks of falls. Reactive balance mechanisms compensate for the unpredictability of postural perturbations and are elicited to regain balance, whereas anticipatory mechanisms reduce the effect of destabilisation, predictable perturbations, and unvoluntary movements (Varghese et al, 2016; Massion, 1992; Maki and McLlroy, 1997; Jacobs and Horak, 2007). It is suggested that reactive mechanisms are elicited based on the sensory signals which indicate disturbances related to postural control and balance (Massion, 1994). On the other hand, anticipatory adjustments do not acknowledge the onset of a disturbance like reactive balance does, instead it precedes it and reduces the effect of any future disturbance in a feedforward manner allowing the postural challenge to become more

manageable and maintainable in unpredictable disturbances (Maki and McLlroy, 1997; Massion, 1994). However, an individual's expectation of the disturbance is relative as they cannot anticipate external unpredictable threats during gait, therefore, reactive mechanisms begin to act after the unexpected perturbations (Ferber et al, 2002). On the other hand, if the individual is aware of the upcoming disturbance they can prepare and prevent postural instability. Regarding this, in perturbating platform experiments (moving in an anteroposterior direction), it Is conventional that after the first few cycles individuals start to predict the characteristics of the disturbance and the destabilising effects in which control mechanisms are set to minimise the effects (Akram et al, 2008).

It is recognised that anticipatory control is regulated in order to meet the demands of predictable perturbations, the magnitude of reactive responses can be diminished through precise anticipatory adjustments that are based on adapted internal portrayal of the system and predictable external responses (Bugnariu and Sveistrup, 2006; Wolpert et al, 1995; Pavol and Pai, 2002). Thus, prior knowledge of internal and external disturbances manifest results of habituation in which preparation occurs in order to counteract the anticipated perturbation, resulting in postural muscle activation in advance or concurrent with the perturbation (Mills and Sveistrup, 2018; Kennedy et al, 2013; Bugnariu and Sveistrup, 2006; Pavol and Pai, 2002). When faced with a series of perturbations, the magnitude of postural reactions and muscular activation decreases over repeated trials (Horak and Nashner, 1986; Chong et al., 2000), and the largest amplitude reduction across trials typically occurs promptly between the first and the second trial (Oude Nijhuis et al., 2009; Pai et al., 2010; Nanhoe-Mahabier et al., 2012). Teixeira et al (2019) highlight that 'online balance-related feedback' that is found to be active from the second perturbation and onwards may be associated with feedforward control that is based on sensorimotor outcomes resulting from previous exposure to continuous perturbations. This improvement of balance stability to repetitive unanticipated perturbations is characteristic of an adaptation of postural response.

The oscillating platform paradigm allows for a more experimental approach to elicit responses such as reactive and anticipatory. This measure focuses on perturbing support surfaces at specific frequencies and amplitudes to which the first perturbation(s) evokes a reactive response mechanism. As the platform continues to oscillate the individual will most likely switch to an anticipatory response due to an adaptation in anticipation with each perturbation (Mills and Sveistrup, 2018). These adaptations to the predicted perturbations have been

manifested to develop within a few sinusoidal cycles of the platform to rapidly meet the requirements of the environment and task at hand (Kennedy et al, 2013; Schmid et al, 2011). Research indicates that young healthy adults adapt from a reactive to an anticipatory response within just 3-5 sinusoidal platform oscillations (Kennedy et al, 2013).

2.4 Onset Muscle Latencies in Postural Control

The neuromuscular system is greatly responsible for the maintenance of trunk stability in terms of standing and sitting (Preuss and Fung, 2008; Milosevic et al, 2015). In lower limb muscles, it is proposed short latency electromyographic (EMG) responses to transient perturbations are adjusted based on muscle lengthening or joint motion and have been considered a monosynaptic reflex (based on amplitude and frequency of occurrence influenced by velocity of platform displacement), medium latency responses are often recognised in stretched muscle and have been calculated to be reflex responses relayed through polysynaptic spinal pathways feasibly mediated by group II afferents (Nardone et al, 1990; Nardone et al, 1996; Grey et al, 2001) which are likely to contribute to medium latency responses during standing. On the contrary, long latency responses are adjusted based on achieving and sustaining task-level goals i.e. upright stance or intended direction of limb movement (Safavynia and Ting, 2013).

Onset muscle activation ensuing the disturbance of quiet stance has been studied to understand accountability of neural mechanisms in the trunk muscles which sustain postural equilibrium. With respect to this, previous studies on reactive balance control, where perturbations are applied to the trunk i.e. perturbing a surface in which an individual is standing, have shown that leg and thigh muscles play significant roles in regaining equilibrium after a slip caused by a perturbed surface (Tang et al, 1998). The TA muscle is found to be particularly activated when the ankle joint is perturbed and released from its usual trajectory relative to plantarflexion, in contrast, the gastrocnemius is active when the ankle joint shows increased dorsiflexion (Tang et al, 1998). Sozzi et al (2016) highlights that once the body segments are displaced short and long muscle latencies are elicited by this which leads to a production of balance correcting effects (Nardone et al, 1990). Reactive balance has indicated that it differs when comparable to voluntary movements, it is constitutionally a feedback motor process in which relevant muscle activity is activated in direct response to task-related error (Welch and Ting, 2008; Welch and Ting, 2009).

Trunk onset muscle latencies are activated in respect to the onset of the perturbation and usually range between 25-200ms of the perturbation (Milosevic et al, 2015; Preuss and Fung, 2008; Cresswell et al, 1994), previous data suggests that both the level of muscle activation and onset latency varies in consideration of the direction of the perturbation in quiet stance (Tang et al, 1998; Henry, 1998). Welch and Ting (2014) also exhibit that, the influence of sensorimotor feedback response to muscle activity is initiated ~50ms on the account of spinally-regulated stretch responses in muscles that are extended by the perturbation, in addition, this primary feedback response is initially down to a larger and longer latency postural response, credible to reactive muscle activity after ~100ms from the perturbation onset. This response is often based on the displacement of the COM from its initial or desired upright stance and due to delays in stabilising effects of reactive muscle activity on COM displacement this may not be evident up to ~200ms after the muscle activity is elicited (Welch and Ting, 2014; Welch and Ting, 2008; Welch and Ting, 2009).

2.5 The Role of the Cerebral Cortex in Postural Control

It is clear the CNS and its complex network aid the control of postural stability and is exaggerated in circumstances where perturbations are anticipated. There is a broad network of high-level neural structures in the CNS such as the cerebral cortex, cerebellum, brainstem, spinal cord, basal ganglia, and sensorimotor cortex that interact to assist the prevention of disequilibrium (Dakin and Bolton, 2018). This network contributes in the development of an internal depiction of posture that is persistently updated based on multisensory feedback used to maintain postural stability, this is from feedforward commands (from the central and peripheral nervous systems) that assist in the control the positioning of the body in space, and detects deviations from a stable postural equilibrium by modulating/initiating corrective actions, either by directly contributing to postural responses or adapting the excitability of subcortical postural responses (Solis-Escalante et al, 2020; Bolton, 2015; Sousa, 2018). Relative to this, it has been observed in previous predictive postural control research, that increased excitability from the primary motor cortex to the spinal cord occurs prior to movement (Peterson et al, 2009). This insinuates that the cerebral cortex contributes to the spinal cord during anticipation to postural disequilibrium therefore, the spinal network works suitably to meet the demands of a postural disturbance (Dakin and Bolton, 2018).

In such cases where a perturbation is external, postural responses manifest a set of behaviours. Whilst postural adjustments are voluntarily modifiable, they cannot be supressed and the

onset of the response is much earlier than voluntary movements, yet later than a segmental spinal reflex (Ackermann et al, 1991; Jacobs et al, 2008). Consequently, the characteristics of postural adjustments (voluntary and reflexive) stipulate an integral role played by the cerebral cortex as it is known to contribute to generation and mapping of voluntary and reflexive movement within the brainstem and spinal cord (Jacobs et al, 2008) which both act when keeping upright stability.

Conditions where changes in postural equilibrium occur slowly over repeated trials (where the sensory condition remains consistent), amount to changes within the CNS often referred to as changes in 'central set' (Horak, 1996; Maki and McIlroy, 2007). Modifications in central set provide the basis for adaptive modification of compensatory postural responses, optimising postural control due to practice, and an increase of repeated exposure (Horak, 1996; Jacobs and Horak, 2007). Research has shown that the cerebral cortex is active responsive when adapting and learning a new motor task (Aizawa et al, 1991), through adaptation, the CNS composes and executes specific strategies to cope with previous conditions that it has been exposed to, overall leading to an improved motor memory developed from an acute change in balance or sensorimotor output to a long-lasting state caused by an adapted and repetitive strategy (Tjernstrom et al, 2001; Shadmehr and Brashers-Krug, 1997). Horak et al (1989) looked at the oscillating platform paradigm to systematically measure the comparable influence of central set and peripheral drive on automatic postural responses. Participants involved with this study had been exposed to different magnitudes of perturbations, however, were not told about any information regarding the amplitude of the forthcoming perturbation. Results established participants magnified the amplitude of their early muscle onset response to match the anticipated amplitude of the perturbation, particularly after repeated exposure to the perturbations. These results support the previous statements indicating that the central nervous system increases the volume of muscular activation based on anticipation and prediction over numerous trials and so diminishes or prevents the delay in which the body responds the perturbation (Horak et al, 1989; Dakin and Bolton, 2018)

The challenge of compensatory postural responses is heightened when individuals do not expect a perturbation. As this is a situation that requires a higher degree of movement, it increases the need for cortical involvement i.e. challenges of spatial navigation, change in support reactions, and acknowledging the surrounding environments, therefore, to maintain upright stance when disturbed by a perturbation, anticipatory reactions start to become

apparent and are active when disrupted by the onset of instability and so are followed with compensatory reactions (Bolton, 2015).

2.5.1 Adaptation and habituation

Cortical habituation and adaptation are the two predominant indicators when it comes to cortical response in balance control. Cortical habituation can be elaborated as the process of gradual improvement in postural control generally obtained through repetitive adaptation and improved motor control to a postural perturbation (Barollo et al 2020, Edmunds et al, 2019). Cortical adaptation can be described as transient changes to motor control strategy when exposed to acute changes in sensorimotor input (Welch and Ting, 2014). Keshner et al (1987) highlights, adaptation in balance is due to a generalised habituation in the neuromuscular system where initial EMG adaptation has been previously addressed with a series of distinct impulsive perturbations. The maintenance of upright stance can be popularised by the fusion of feedforward and feedback mechanisms founded by cortical and subcortical derivatives to create and maintain forces to keep balance, it is consistently sustained through adjustments to the correct body segments and extremities during adaptation or the influence of motor control (Edmunds et al, 2019). Postural control is seemingly an ability that is particularly acquired and modified through repetitive conditions and practice; therefore, the strategy of adaptation is fundamental within research.

2.6 Electroencephalography and the Measurement of Cortical Activity in Balance

As previously stated, there is a rapid growth in evidence suggesting cerebral cortex is responsible for assisting and regulating the excitability of subcortical postural responses to maintain balance and postural control. This is specifically evident in transient or externally initiated perturbations which indicate characteristic changes in electroencephalographic (EEG) activity also known as a perturbation evoked response (Mierau et al, 2015). EEG Scalp electrode recordings of cortical potentials evoked by postural perturbations have shown to provide critical temporal information related to the cortical processing of associated sensory and motor events (Maki and McIlroy, 2007). This type of brain imaging measures electrical cortical activity through analysis of frequencies and event-related potentials (ERP's) and are seen as an indication of cortical activity displayed through systematic changes in in electrical activity generated by cortical neurons (Read and Innis, 2017). It is understood that the cortical role in postural control may be parallel to ERPs that are evoked by alternate stimuli i.e. visual or auditory (Marlin et al, 2014; Varghese et al, 2014). ERP's are known to measure each stage

of information flow manifested through synchronisation and desynchronisation of EEG signals (Kropotov, 2010). To elicit such responses, ERPs need to be locked into an event which can be a stimulus (Pfurtscheller and Lopes da Silva, 1999) or movement and are known to assist an array of sensory, motor, and cognitive functions when maintaining balance (Read and Innis, 2017).

The involvement of cortical activity in balance control has been manifested through the quantification of evoked cortical potentials that have the highest amplitudes at the fronto-central region of the cortices and are apparent at the onset of a perturbation (Mochizuki et al, 2009). Fronto-central cortical regions such as the anterior cingulate cortex (ACC) is known to be substantially involved with action monitoring and the detection of error signals (Hulsdunker et al, 2015), in regard to balance control, error signals are established as a discrepancy between the expected and actual state of balance during transient balance perturbations (Adkin et al, 2006). Previous studies proclaimed that there is elevated fronto-central/ACC activity during the performance of a balance task that ascribed the detection of balance instability (Mochizuki et al, 2009). An error-related negativity (ERN) is a neural correlate of error-monitoring which explores individual disparities in developmental, and adaptive context. ERN is a negative deflection in the frontocentral midline and is thought to occur in neural circuits involving the ACC and supplementary motor areas (Payne et al, 2023).

Cortical responses to balance perturbations are visible with a large scalp distribution and a substantial spectral structure which reflects cognitive and sensorimotor processes that are relative to the integration of sensory information linked with sudden changes in posture (Peterson and Ferris 2018; Solis-Escalante et al, 2020). EEG signals are comprised of different frequency spectrums that oscillate simultaneously, these are; Delta (<4 Hz), Theta (4-7 Hz), Alpha (8-12 Hz), Beta (13-30 Hz) and Gamma (>35 Hz). It is accepted that the lower the frequency is, the greater the power, therefore, as power decreases the frequency increases (Mierau et al, 2017; Varghese et al, 2014; Read and Innis, 2017) e.g.. less alpha power means an increase in cognitive work. Theta, Alpha and Beta are the most common frequencies found in transient balance investigations. The activity of the ACC when balance is disturbed has been characterised as an increase in theta power which allows for error detecting and processing to take place in order to maintain balance (Hulsdunker et al, 2015). Spectral characteristics of perturbation-related cortical activity can allow for an in depth understanding of how much the cerebral cortex contributes to the control of balance. For example, time-frequency analysis of

EEG can reveal changes in specific cortical dynamics relative to environments that may be internal or external (Solis-Escalante et al, 2019). Different cortical rhythms have been seen to correlate against cognitive and sensorimotor functions, i.e. low-frequency cortical rhythms (< 13 Hz) are relative to perception and cognitive control, however, high-frequency rhythms (> 13 Hz) are relative to motor function (Cavanagh and Frank, 2014; Engel and Fries, 2010). In regard to this, beta oscillations are linked to the processing of sensorimotor information and motor control, however they are also found to decline when sudden changes in gait patterns are apparent (Ghosn et al, 2020; Sipp et al, 2013; Varghese et al, 2014).

2.7 N1 Response and its role in Postural Control

The control of human balance is orchestrated through numerous neuromuscular systems however, it specifically requires assistance from rapid and powerful neural ensembles that are distributed over several levels of the CNS (Solis-Escalante et al, 2020; Maki and McIlroy, 2007; Jacobs and Horak, 2007). Previous research has shown that unpredictable postural perturbations such as platform translations can elicit a multi-component response that consists of a small positive potential (P1) and a negative potential (N1) (Maki and McIlroy, 2007; Quant et al, 2004; Quant et al, 2005). It is argued that the P1 response represents a PECR which differs in amplitude due to the characteristics of the perturbation however, it is not consistent and is relatively small (1μV) (Quant et al, 2004; Quandt et al, 2005). The N1 peak is known to be a prominent and consistent feature of a perturbation-evoked cortical response (PECR) (Varghese et al, 2014) and carries a large amplitude in excess of 30μV (Maki and McIlroy, 2007). This response is broadly distributed and retains maximal response within the fronto-central cortical regions and occurs approximately at 100-200ms after the onset of the perturbation (Adkin et al, 2005).

It is found that the N1 response reflects the processing of the balance disturbance at the level of the cortex (Adkin et al, 2006). Such stated PECRs are modulated by the characteristics of the balance perturbation and can be down to any of the following: displacement, velocity, acceleration, and duration. It is manifested that the N1 response increases with perturbation intensity and the destabilising effects that follow with it (Mochizuki et al, 2010), suggesting that the N1 potential is relative to the involvement of processing of the multisensory input linked to sudden changes in task conditions and postural control (Maki and McIlroy, 2007). With respect to this, Varghese et al (2017) confirms that PECRs are elicited by multisensory

stimuli (somatosensory, visual, and vestibular) evoked by whole body perturbations and are characterised by a large N1 amplitude compared to an N1 generated by other stimuli.

It is improbable that the N1 response represents cortical activity that contributes to early onset reactive postural responses which has a latency of ~150ms and a weak correlation with rapid reactive muscle responses (Mierau et al, 2015; Payne et al, 2019), alternatively, it is more than likely that the N1 response indicates cognitive and sensorimotor processes that modify late onset postural responses (Solis-Escalante et al, 2020). Psychological and cognitive factors that are relevant and apparent when it comes down to activating an N1 response during postural instability can vary from; the predictability of a perturbation and its onset and magnitude (Mochizuki et al, 2010; Adkin et al, 2008), perceived postural threat (Mochizuki et al, 2010; Adkin et al 2008), attention to simultaneous tasks (Quandt et al, 2004), and habituation (Mierau et al, 2015). Considering these factors, attention to simultaneous task or repeated exposure to perturbations gradually decreases the N1 response due to habituation and anticipation (Miearau et al, 2015; Barollo et al, 2020), yet, imposed changes to postural stability of a uniformed magnitude elicit a stronger N1 response due to conditions that create increased postural threat and a lack of perturbation predictability (Adkin et al 2008; Solis-Escalante et al, 2020; Solis-Escalante et al, 2021). Reflecting on what was previously stated, it is undertsood that the N1 response is constantly regulated when challenged with postural instability. Furthermore, it is also reported that adaptation of the negative cortical potential (N1) and reduced muscle (co)contraction during a trial based balancing task under a standing balance condition is very much apparent (Mierau et al, 2015; Sozzi et al, 2016) highlighting an adaptation with the repetition of perturbating trials.

N1 potentials have been previously suggested by Quandt et al (2004) to display cortical processing of sensory information that has been elicited through perturbations, nonetheless, N1 is proven to increase with perturbation amplitude (Mochizuki et al, 2010; Staines et al, 2001), it is enhanced during an increase in postural threat and, reduced or absent when perturbation onset is predictable (Adkin et al, 2008; Adkin et al, 2006). Therefore, these results indicate the involvement of a higher cognitive process rather than simply just processing afferent sensory input (Mierau et al, 2015). Other pioneers in research have discussed that N1 responses occurring after postural instability indicate error-detection and is simply unrelated to sensory and motor events related to compensatory postural responses (Varghese et al, 2017; Adkin et al, 2008; Adkin et al, 2006; Mochizuki et al, 2010). Their observations found that

there were larger amplitudes of N1 when exposed to repeated trials of unpredictable postural perturbation, whereas predictable perturbations portrayed a much smaller N1 amplitude. Overall, they established that the amplitude of the response comes down to the interaction between 'actual postural state' and changes within expectations of the 'central state' and so it was concluded that the indisputably large N1 amplitude during unpredictable perturbations is due to a confusion between actual and expected postural responses, therefore N1 embodies an 'error signal' that generally detects the shift between the two states in lieu of sensory processing or balance control (Varghese et al, 2017; Adkin et al, 2008; Adkin et al, 2006).

Inverse mapping of the N1 potential stipulates that it is derived from the supplementary motor cortex with additional assistance of the ACC and posterior parietal cortex (Marlin et al, 2014; Mierau et al, 2015). Considering the spectral characteristics of PECR's, further insights into time-frequency analysis of EEG have been found to expose changes in particular cortical dynamics respective to internal and external events that affect postural stability (Pfurtscheller and Lopes da Silva, 1999). This analysis in terms of exposure to continuous mechanically induced perturbations has emphasised a transient spectral power increase of theta (4-7Hz), alpha (8-12Hz), and Beta (13-30Hz) rhythms that coincide with the latency and localisation of the N1 potential (Mierau et al, 2017; Varghese et al, 2014). With this said, it has been suggested that the N1 response and theta rhythm have been known to predict the response outcome to balance perturbations that acquire a stronger cortical dynamic in terms of a stepping response when compared to feet-in-place responses at similar perturbation magnitudes (Stokkermans et al, 2023; Solis-Escalante et al, 2020). Collectively, previous evidence has highlighted that the scalp-level N1 potential constitutes for various coinciding cognitive and sensorimotor institutes diffused throughout multiple areas of the cortices (Solis-Escalante, 2019).

2.8 Introduction Summary

The understanding of adaptation mechanisms to continuous transient balance perturbations remains limited, however, the oscillating platform paradigm allows for an interesting and meaningful experimental approach that elicits reactive and anticipatory responses when exposed to a few sinusoidal cycles within a trial. It is crucial for both sensory input and motor output to interact in order to create a postural stability that is essential for humans during daily life, and while both of these have been investigated separately, few studies have explored the extent of what they do together which creates an interesting and indispensable gap of knowledge within this field of research.

3. Aims and Hypothesis

There is an extensive amount of research that investigates reactive postural responses to superficial initiated perturbations and the affects it can have of postural control and balance, the same can also be said for cortical activity and the N1 response. Contrary to this there is an extremely limited amount of research that allows for these components to coincide together, therefore, this thesis will hopefully reduce this gap of knowledge and allow for some insight into feedforward mechanisms and how they assist in adaptation to continuous unpredictable postural perturbations.

The aim of this thesis is to explore and investigate how feedforward postural responses indicate adaptation to repeated continuous postural perturbations through postural organisation against the measurement of muscular activity, kinematics, and kinetics. Additionally, the aim is to investigate cortical activity and characterise changes in N1 potentials relative to postural state at the onset of the perturbation and the change of frequency of the perturbation. Furthermore, the changes found in the EEG activity data will reflect adaptations within postural control

It is hypothesised that postural adaptation would be apparent when exposed to transient perturbations. This is down to task-level balance control (Welch and Ting, 2014) during the responses to postural perturbations and changes in both reactive and anticipatory modes of postural stability. It is further hypothesised that the measure of EMG activity in the lower limb extremities (specifically the tibialis anterior and gastrocnemius medialis) will gradually decrease over trials with earlier muscular activation due to anticipatory and reactive responses. Furthermore, changes in cortical activity through reduced amplitudes of N1 responses during individual trials is expected which manifests a reduction in cognitive workload overtime.

4. Methodology

4.1 Participants

Twenty-two healthy young adults (12 males, 10 females) between the ages of 22-39 were recruited for, and participated in this research study however, one participant was removed due to data issues leaving twenty-one participants (12 males, 9 females; *M* age = 27 yrs, *SD* = 4.83; *M* height = 173.28 cm; *SD* = 9.58; *M* weight = 70.76kg, *SD* = 19.52). This sample size was chosen due to similar studies measuring EEG in balance referring to and also using this sample size (Castro et al 2022). Participants were recruited through word of mouth and given an information document followed by a written consent form prior to the onset of the study. This research study was reviewed and approved by the institutional ethics board at Manchester Metropolitan University.

Each subject that was recruited was ensured to fit into the inclusion criteria which insisted that each subject had no self-reported neurological or musculoskeletal disorders in order to explore specific individual responses to postural perturbations within a cohort of healthy individuals without any type of sensory dysfunctions.

4.2 Protocol

Participants attended the movement laboratory at Manchester Metropolitan University for a single 60-90-minute testing session. Once consent from each individual had been confirmed, each participant was instrumented with twenty-three retro-reflective markers on specific anatomical landmarks i.e. right and left (R/L) frontal bone, R/L occipital bone, R/L clavicle, sternum, R/L acetabulum, R/L Asis, R/L lateral mid-thigh, R/L lateral condyle, R/L tibia, R/L calcaneus, R/L lateral malleolus, R/L second metatarsal head. These markers were placed respectively to track the movement of the individual and estimate the positioning of the body's COM throughout each of the postural trials. Four more markers were placed on each corner of the oscillating platform in order to determine its placement within the area that was calibrated. A twenty-fourth marker was placed on the body at the sacrum at the end of each testing for a static measurement (i.e., to complete the pelvic model). This was due to the fact the backpack from the mobile EEG affected motion analysis for this marker throughout the trials. Motion analysis software recorded any postural movements at 100 Hz by using twelve Vicon cameras (Vicon, Oxford, UK).

Participants were instrumented with four EMG surface electrodes to measure any muscular activation that would occur through each trial (see section 4.3.1 for details), subsequently, once EMG electrodes had been placed, participants were fitted with the EEG cap to measure cortical activity within each trial (see section 4.3.2 for details). The preparation and overall set up would usually take around 30-45 minutes. Once fitted with all appropriate measurement devices, participants were asked to stand on the platform barefooted, shoulder-width apart, hands by their sides, and eyes open. Throughout each trial, participants were asked to keep eyes fixated on a cross marked on a wall at participants eye-level approximately 3-4 metres in front on them. Once the participant was asked if they were ready, a five second interval would take place before pressing ta synchronisation trigger which would deliver a square wave pulse from Qualisys to the EEG amplifier.

The oscillating platform paradigm was based on previously established methods (Kennedy et al, 2013; Mills and Sveistrup, 2018). Platform perturbations were delivered via a bespoke oscillating platform with embedded force plates (80 x 60 cm, Kistler, Winterthur, Switzerland) which translated in an anterior-posterior direction, 12cm peak-to-peak at two different frequencies ([1]). Participants experienced 1 block of 10 trials. Each trial consisted of 10 cycles at 25 Hz and 15 cycles at 50 Hz and lasted 1 minute per trial. Between trials, participants would be asked to step off the platform and take a 1-minute break whilst preparing for the next trial. The platform was accelerated using an electromagnetic actuator and customised software (Labview v19, SP1, National Instruments, Austin, Texas) via DAQ card (USB-6210, National Instruments) in which the two different frequencies were set to automatically change at set intervals unbeknownst to the participants.

[1] From participant 9, the force plates was switched and replaced from the Kistler plate to a Bertec force plate (Bertec Corporation, Ohio, USA) due to inaccurate measurement of mass and COP (this defect did not corrupt any of the previous data). Furthermore, participants stood on one force plate throughout data collection due to equipment failure/data only acquired from one plate.

4.3 Instruments and Equipment for Data Acquisition

4.3.1 Electroencephalography

Electroencephalographic (EEG) cortical signals were recorded at a rate of 1000 Hz from 32 active shielded AgCl electrodes fixed into a flexible fabric-based cap (eego sports, Ant Neuro, Hengelo, Netherlands), with a basis of the ActiveTwo recording system. The thirty-two channel EEG cap was positioned relative to the international 10-20 placement system (Jurcak et al., 2007). EEG electrodes in sites CPz and GND and Nasion and Inion anatomical landmarks were used and measured as a reference for accurate and approximate placement of the cap to correct sites of the cortices, Each of the thirty-two electrodes were inserted with conductive gel (Signa gel, Parker) once fitted to the head to increase impedance (kept below 20 kΩ) and allow for clear results of cortical activity and produce a better signal-to-noise ratio (Shad et al., 2020). The EEG cap was connected to an EEG mobile amplifier (eego sports, Ant Neuro, Hengelo, Netherlands) with supplemented auxiliary channels to allow for synchronous recording of bipolar surface EMG. Force and EEG data were synchronised through a Delsys surface electrode used as a trigger prior to the start of each trial.

4.3.2 Electromyography

Postural muscle activation was recorded through electromyographical (EMG) surface electrodes (Delsys Bagnoli EMG, Delsys Inc., Natick, USA) at 1000 Hz. Each surface electrode was placed accurately over the skin of the muscle belly of the right/left tibialis anterior (TA) and gastrocnemius medialis (GM) by following the SENIAM guidelines for accurate assessment of each muscle (Garcia et al., 2017). If necessary, any visible hair in appropriate area was shaved and skin was later cleaned with an alcohol wipe to allow for enhanced EMG signals, these were checked uniformly prior to every trial per participant.

4.4 Data Processing

4.4.1 EEG data processing

For EEG analyses, signals were band-pass filtered using the EEGLAB “basic FIR filter (new)” (1-45Hz, 3300 filter order, -6dB cutoff frequency, 1 Hz transition bandwidth). Data from each trial were then cut into two distinct epochs ranging from -2 to +2 s relative to the first perturbation onset, and from -2 to +2 s relative to the change of perturbation frequency at the onset of the

11th perturbation cycle. These epochs were visually inspected for large EEG contamination from muscular artifacts, and no bad EEG channels were identified. Independent component analysis (ICA) weights were obtained through the RunICA infomax algorithm running on EEG signals. ICA weights that presented obvious non neural activity upon visual inspection (e.g., eyeblinks, line noise, muscular artifact) were manually rejected. To assess cortical N1 responses, we extracted single trial N1 amplitudes that were baseline subtracted (-150 to -50 ms before perturbation onset) for each participant. The N1 was quantified as the largest negative peak 50-300 ms after perturbation onset and change of platform frequency. These processing steps were performed using EEGLAB (v2020.0) functions for MATLAB.

4.4.2 Postural Control analyses

Custom MATLAB scripts was used to determine the peak velocity of centre of pressure (COP) data in response to the initial backward portion of the first perturbation and the change of perturbation frequency. It explored both peak positive and peak negative COP velocity as the outcome variables. Firstly, for each event they were selected and low-pass filtered (5Hz, 2nd order bidirectional Butterworth filter) a 10-second AP-COP trace that spanned 5000ms pre-perturbation and 5000ms post-perturbation. Each of these traces were then corrected for offset using the estimated median AP sway during the 'baseline' period (based on the 2000-50ms pre-perturbation window). Peak forward velocity of the postural response to the perturbation was then identified as the first positive peak, whereas peak backward velocity was identified as the first negative peak, in the derivative of the AP-COP tract in the initial backward portion of the perturbation (spanning 0 to 2000ms post-perturbation). Movement tracking was measured and analysed through Qualisys (Qualisys, Gothenburg, Sweden).

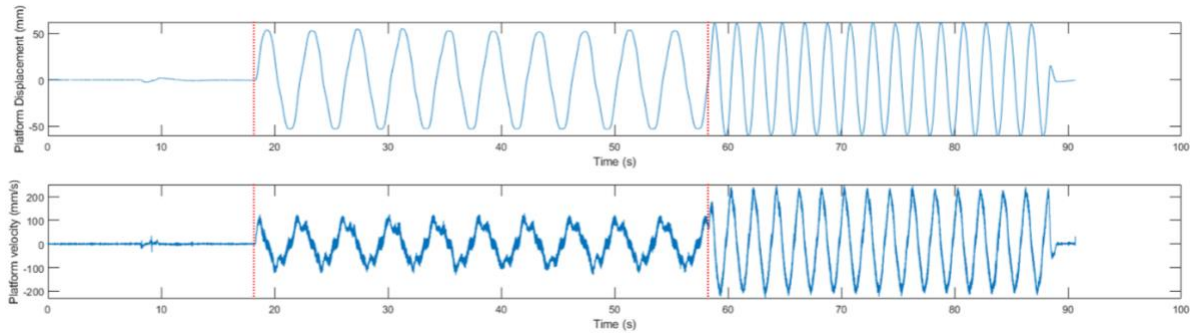

Figure 1 – Platform displacement (top) and velocity (bottom) for one trial. Red dotted line indicates onset of the platform oscillation frequency transition.

4.4.3 Muscular Activation

To calculate muscle activity, EMG signals were cut into epochs ranging from -2 to +2 seconds relative to the onset of the first perturbation and the change in perturbation frequency. EMG signals were then converted to the time-frequency domain via convolution with a family of complex Morlet Wavelets, defined as Gaussian-tapered sine waves. Three-hundred and ninety-four linearly spaced wavelets between 7 and 400 Hz were used. The number of cycles increased from 8 to 16 in logarithmic steps. Convolution was performed via frequency-domain multiplication, in which the Fourier derived spectrum of the EMG data was multiplied by the spectrum of the wavelet, and the inverse Fourier transform was taken. EMG power was defined as the squared amplitude of the complex result. Total muscle activity for the GM and the TA muscles were taken as the sum of the signal power contained between 7 and 400 Hz from 0 to +2 seconds relative to the onset of the first perturbation and the change in perturbation frequency. Muscular activation for the left and right lower limb muscles were averaged to provide a single value for TA and GM activity.

**Onset muscle latencies were not analysed in this thesis however they will be for publication of thesis*.

4.4.4 Statistical Analyses

Dependent variables were calculated separately for the onset of the first perturbation (onset of platform movement) and the onset of the change of frequency (instance when platform speed increased), and included; the cortical N1 amplitude, peak-forwards COP velocity, peak-backwards COP velocity, and EMG activity of the TA and GM. Statistical analyses were performed on all dependant variables using a repeated measures Analysis of Variance (ANOVA) with the within-subject factor of Trial (T1-T10). Significant effects were probed by polynomial trend analyses, and *post-hoc* analyses were performed using pairwise comparisons. Specifically, adaptation was assessed by comparing all trials against the first trial (T1) to determine how many trials were required for adaptation to occur, and by comparing trials 1 to 9 against trial 10 to determine the point at which any adaptive changes plateaued across the experimental trials. ANOVA effect sizes were reported using a partial eta squared (ηp^2), common indicative thresholds for which are small (0.01), medium (0.06), and large (0.14) (Field, 2013). All statical analyses were performed using IBM SPSS statistics (version 26) with an alpha level of ≤ 0.05.

5. Results

5.1 Onset of initial platform movement

5.1.1 Centre of Pressure

Peak forward COP velocity

A repeated measures ANOVA for peak forward COP velocity (PFCOPV) revealed a significant main effect of Trial, $F(9, 99) = 2.679$, $p = .008$, $\eta p^2 = .196$, that was best explained by a linear polynomial trend, $F(1, 11) = 12.103$, $p = .005$, $\eta p^2 = .524$. Pairwise comparisons revealed that the PFWCOPV was significantly greater for trial 1 compared to trial 7 ($p = .041$), Trial 9 ($p = .029$) and Trial 10 ($p = .024$) PFCOPV was also significantly smaller for trial 10 compared to trial 4 ($p = .044$). (Figure 2, graph B).

Peak backward COP velocity

A repeated measures ANOVA for peak backward COP velocity (PBCOPV) revealed a large significant main effect of Trial, $F(9, 99) = 11.118$, $p = <.001$, $\eta p^2 = .503$, that was best explained by a linear polynomial trend, $F(1, 11) = 57.453$, $p = <.001$, $\eta p^2 = .839$. Pairwise comparisons revealed that the PBCOPV was significantly greater for the first trial compared to all other trials ($ps < .045$). Additionally, PBCOPV significantly smaller for trial 10 when compared to all other trials ($ps < .05$) except from trial 7 ($p = .0321$) and trial 8 ($p = .347$). (Figure 2, graph C).

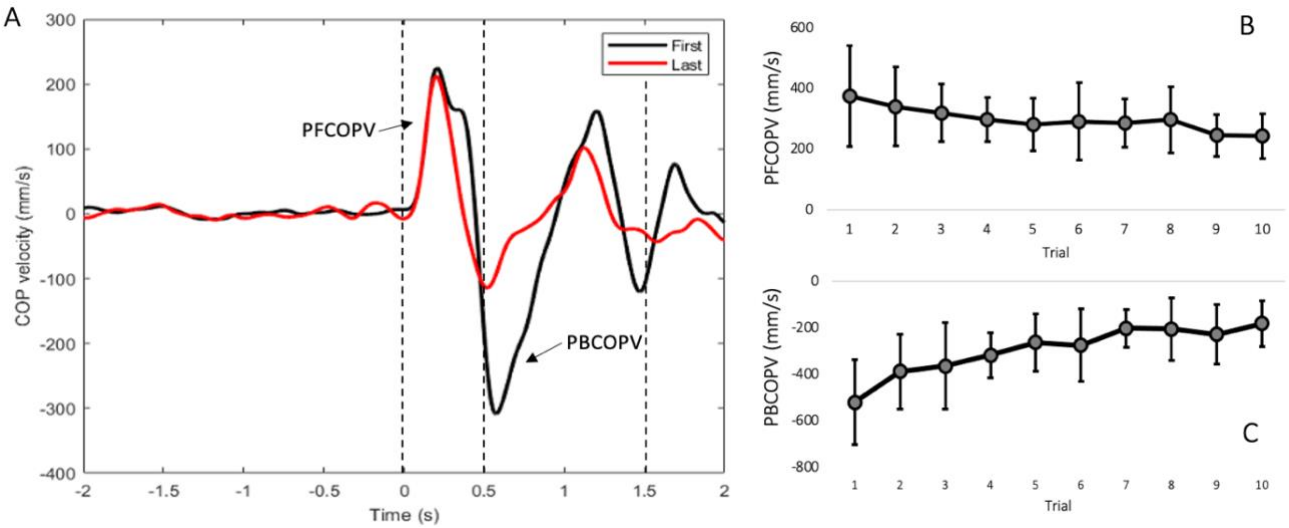

Figure 2 – Graph (A) shows comparisons of overall COP velocity (mm/s) from the first and last trial in response to the onset of initial platform movement. Graph (B) shows peak velocity (mm/s) for COP in response to the forward translation from the onset of the initial movement of the platform compared over trials 1-10. Graph (C) Graph (B) shows peak velocity (mm/s) for COP in response to the backwards translation from the onset of the initial movement of the platform compared over trials 1-10.

5.1.2 Electromyography

Peak Gastrocnemius Medialis activity

A repeated measures ANOVA for peak GM activity revealed a large significant main effect of Trial, F(9, 108) = 6.751, p = <.001, ηp^2 = .360, that was best explained by a linear polynomial trend, F(1, 12) = 15.710, p = .002, ηp^2 = .567. Pairwise comparisons revealed that peak GM activity was significantly greater during the first trial compared to all other trials (ps < .015), except for trial 3 (p = .051). Peak GM activity was lowest for trial 10 but was only significantly lower compared to trial 1 (p = .003), trial 3 (p = 0.15), and trial 7 (p = .038). (Figure 3, graph A).

Peak Tibialis Anterior Activity

A repeated measures ANOVA for peak TA activity revealed a large significant main effect of Trial, F(9, 126) = 14.378, p = <.001, ηp^2 = .507, that was best explained by a linear polynomial trend, F(1. 14) = 17.663, p = <.001, ηp^2 = .558. Pairwise comparisons revealed that peak TA activity was significantly largest for the first trial compared to all other trials (ps < .002). Pairwise comparisons also revealed that all trials were significantly greater (ps = .05) than Trial 1 (p = .110) when compared against trial 10. (Figure 3, graph B).

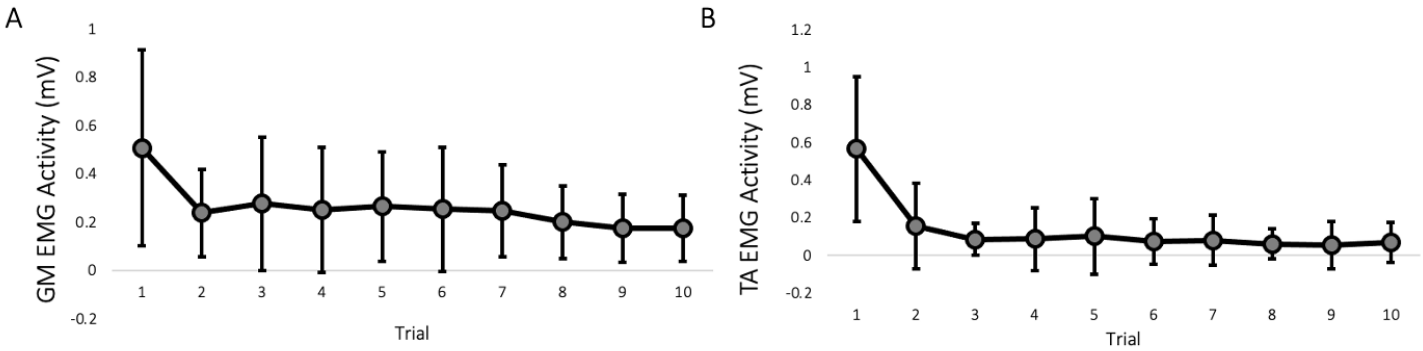

Figure 3 – Peak Electromyographic activity for the Gastrocnemius Medialis (GM) (Graph A) and Tibialis Anterior (TA) (Graph B) in response to the onset of the initial platform movement compared over trials 1-10.

5.1.3 N1 Response

A repeated measures ANOVA revealed a significant main effect of Trial, F(9, 108) = 2.887, p = .004, ηp^2 = .194, that was best explained by a linear polynomial trend, F(1, 12) = 10.331, p = .007, ηp^2 = .463. Pairwise comparisons revealed that compared to trial 1 (p = .002), the N1 was significantly smaller from trial 5 (p =.029) through to trial 10 (p = .002). The N1 was also significantly smaller for trial 10 compared to Trial 4 (p = .006), and trial 6 (p = .032).

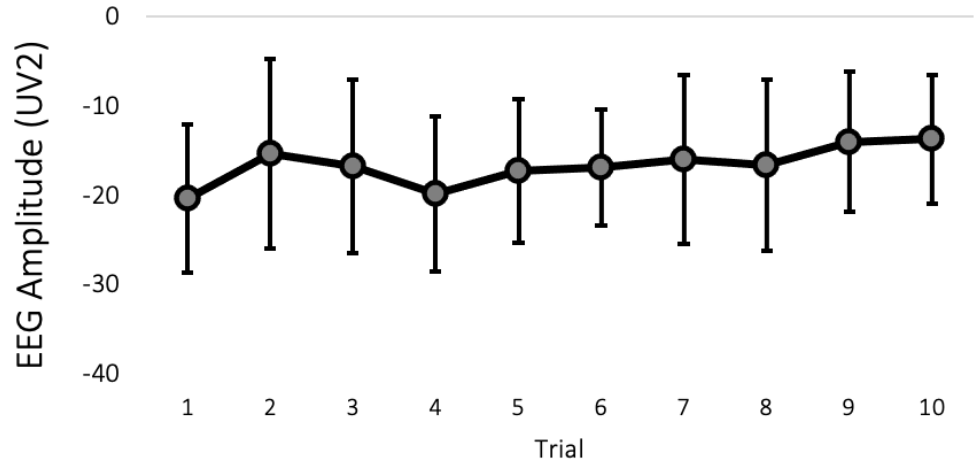

Figure 4 - Peak N1 Electroencephalographic (EEG) amplitude (UV2) in response to the onset of the initial platform movement compared over trials 1-10.

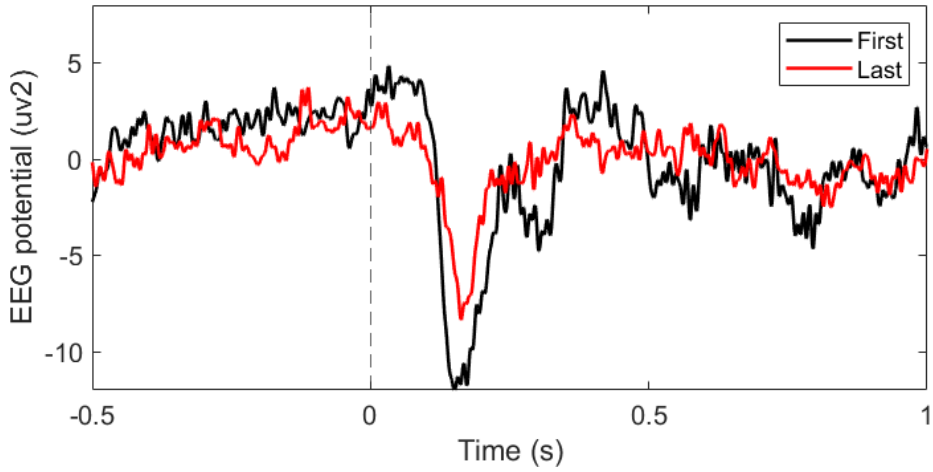

Figure 5 – N1 ERP time series of EEG channel Cz in response to the onset of the initial platform movement analysed from trials 1 and 10 (first and final trials).

5.2 Change of Frequency

5.2.1 Centre of Pressure

Peak forward COP velocity

A repeated measures ANOVA for peak forward COP velocity (PFCOPV) revealed there was no significant main effect of Trial, $F(9, 99) = 2943.066$, $p = .713$, $\eta p^2 = .061$. (Figure 6, graph B).

Peak backward COP velocity

A repeated measures ANOVA for peak backward COP velocity (PBCOPV) revealed a significant main effect of Trial, $F(9, 99) = 2.181$, $p = .03$, $\eta p^2 = .165$, that was best explained by a linear polynomial trend, $F(1, 11) = 11.420$, $p = .006$, $\eta p^2 = .509$. Pairwise comparisons revealed that

PBCOPV was significantly greater for trial 1 compared to trial 7 (p = .033) and trial 10 (p = .036). Pairwise comparisons also revealed that trial 6 (p = .151), trial 7 (p = .452) and trial 8 (p = .267) were significantly larger when compared against trial 10. (Figure 6, graph C).

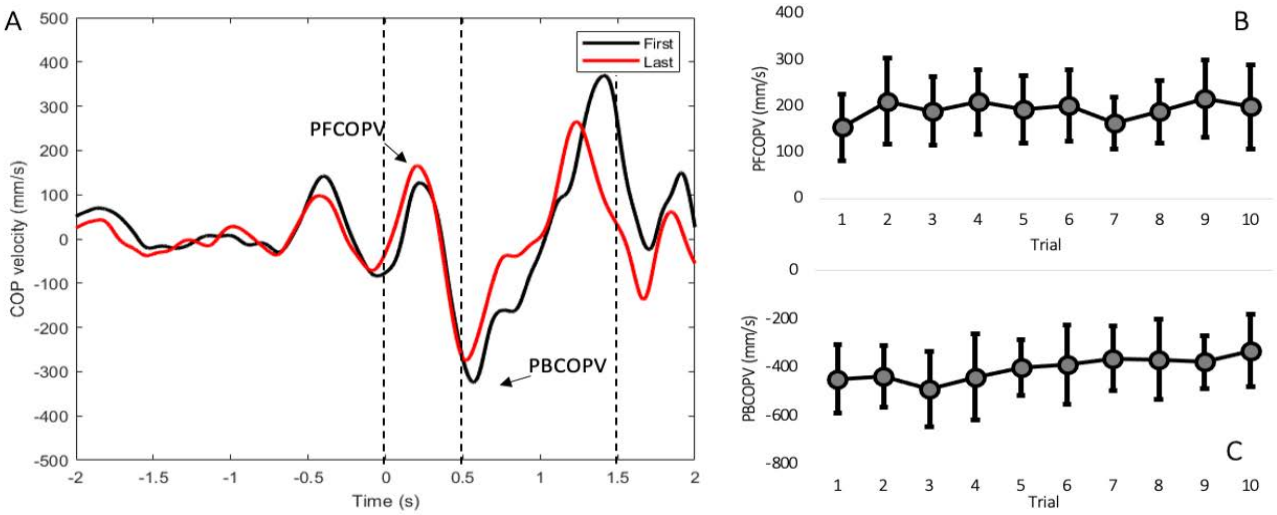

Figure 6- Graph (A) shows comparisons of overall COP velocity (mm/s) from the first and last trial in response to the change of frequency of the platform. Graph (B) shows peak velocity (mm/s) for COP in response to the forward translation to the change of speed of the platform compared over trials 1-10. Graph (C) Graph (B) shows peak velocity (mm/s) for COP in response to the backwards translation to the change speed of the platform compared over trials 1-10.

5.2.2 Electromyography

Peak Gastrocnemius Medialis activity

A repeated measures ANOVA for peak GM activity revealed a large significant main effect of Trial, F(9, 81) = 12.025, p = <.001, ηp^2 = .572, that was best explained by a linear polynomial trend, F(1, 9) = 37.986, p = <.001, ηp^2 = .808, Pairwise comparisons revealed that peak GM activity was significantly greatest for trial 1 compared to all other trials (ps < .002). Peak GM activity was also significantly lower for trial 10 compared to trial 2 (p = .013), trial 3 (p = .001), and trial 7 (p = .032). (Figure 7, graph A).

Peak Tibialis Anterior Activity

A repeated measures ANOVA for peak TA activity revealed a large significant main effect of Trial, F(9, 81) = 11.862, p = <.001, ηp^2 = .569, that was best explained by a linear polynomial trend, F(1, 9) = 28.833, p = <.001, ηp^2 = .766. Pairwise comparisons revealed that peak TA activity was significantly greatest for trial 1 (p = <.001) compared to all other trials (ps <.009). TA activity was also significantly lower for trial 10 compared to trial 2 (p = .031), and trial 3 (p = .012). (Figure 7, graph B).

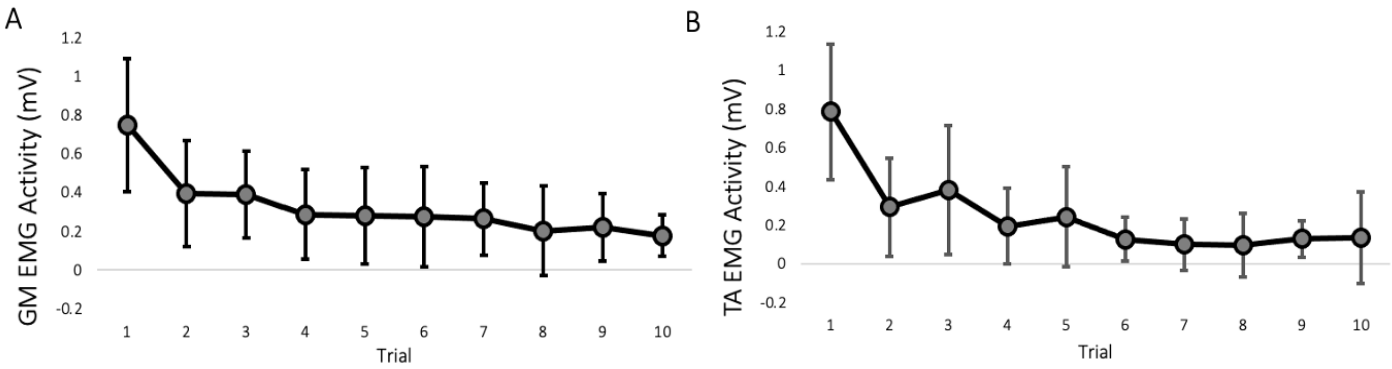

Figure 7 – Peak Electromyographic activity for the Gastrocnemius Medialis (GM) (Graph A) and Tibialis Anterior (TA) (Graph B) in response to the change of frequency compared over trials 1-10.

5.2.2 N1 Response

A repeated measures ANOVA revealed that there was no significant main effect of Trial upon the cortical N1 amplitude, $F(9, 117) = .718$, $p = .692$, $\eta p^2 = .052$.

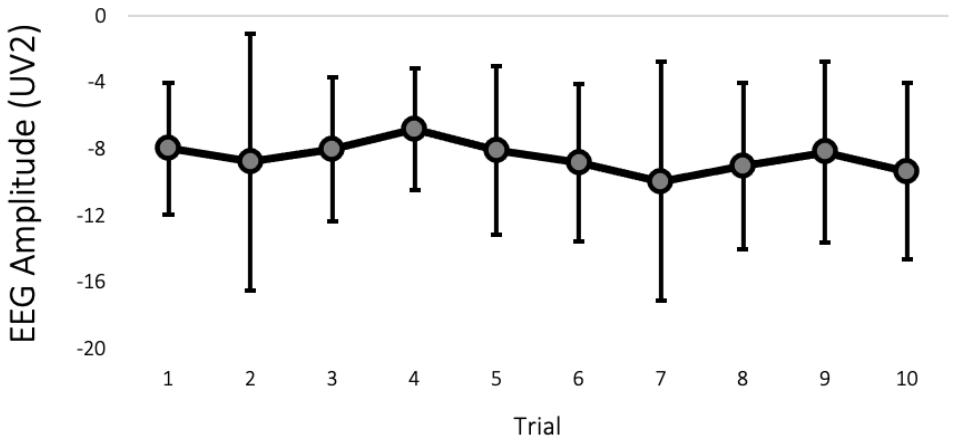

Figure 8 - Peak N1 Electroencephalographic (EEG) amplitude (UV2) in response to the change of frequency of the perturbation when compared over trials 1-10.

5.2.4 N1 Response to forward-to-backward change of direction following transition to increased oscillatory frequency

A repeated ANOVA revealed that there was a large significant main effect of Trial, $F(9, 117) = 4.883$, $p = <.001$, $np^2 = .273$, that was best explained by a linear polynomial trend, $F(1, 13) = 16.952$, $p = .001$, $\eta p^2 = .566$. Pairwise comparisons revealed that the N1 response was significantly greater for trial 1 compared to all trial ($ps < .043$) except trial 2 ($p = .095$) trial 4 ($p = .058$). Trial 2 ($p = .030$), trial 4 ($p = .012$), and trial 6 ($p = .042$) was smaller compared to trial 10.

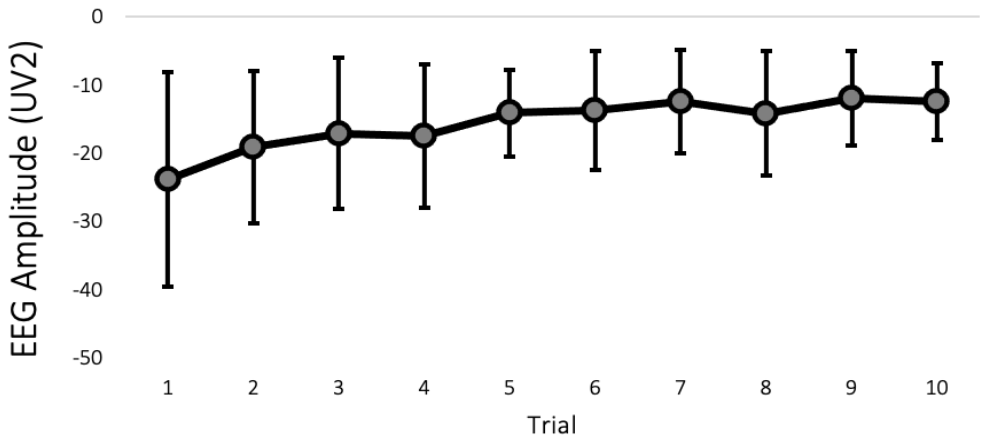

Figure 9 - N1 Electroencephalographic (EEG) amplitude (UV2) in response to the change of direction of the forward-to-backward translation following the transition to increased oscillatory frequency compared over trials 1-10.

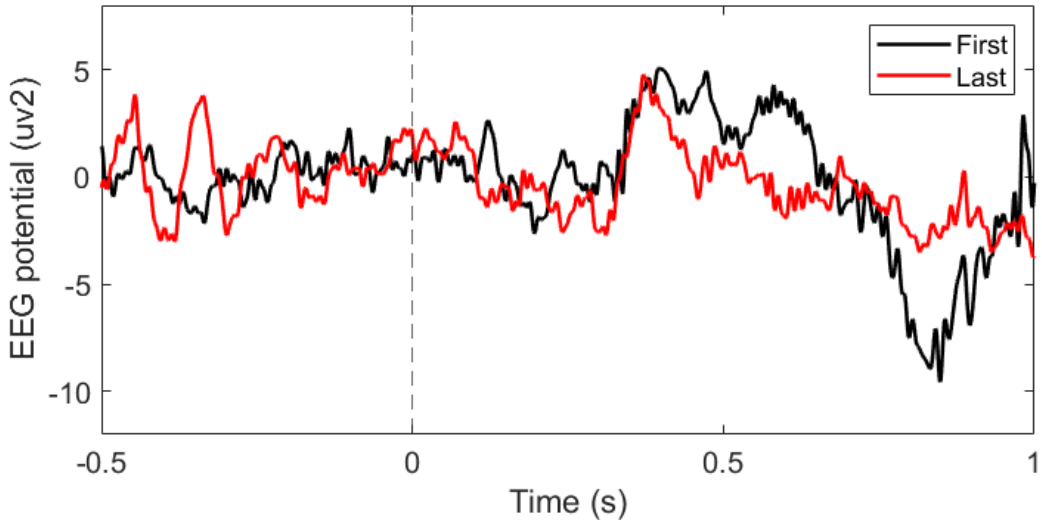

Figure 10 - N1 ERP time series of EEG channel Cz in response to the change of frequency of the perturbation analysed from trials 1 and 10 (first and final trials).

6. Discussion

This study explored how feedforward postural responses indicate adaptation to repeated continuous postural perturbations by measuring postural organisation through changes in muscular activation, centre of pressure positioning, and N1 cortical characteristics and activation relative to postural state at the onset of the perturbation, change of frequency and generally throughout the 10 individual trials with each perturbation moving in an AP direction. The findings support the hypothesis that repeated trials would result in significant postural and neural adaptations. The findings reveal significant adaptations across multiple measures that shed light on the dynamic interplay between sensory input, motor output and cortical output in maintaining balance and stability.

6.1 Centre of Pressure

In terms of COP positioning, it is essential to understand how balance is maintained and restored by participants throughout and after the perturbations. The adaptation to the onset of the first perturbation can be characterised as a rapid repositioning of COP, which is expressed by a slowing down of the magnitude of COP velocity (Park et al, 2023; Afschrift et al, 2022). Key findings in this study determined that COP dynamics were significantly reduced when exposed to the continuous unpredictable perturbations. Specifically, it was found that, in response to the initial perturbation, it took approximately seven trials to observe a significant reduction in the peak forwards velocity of the COP (Figure 2, graph B). Relative to this, Kennedy et al (2013) also found this information within his study with data highlighting that it took around seven trials to observe such a reduction in response to the perturbation. By contrast peak backwards COP velocity in response to the platforms first change of direction (backwards to forwards) significantly reduced from the first to second trial, and continued to reduce until trial 7, from which improvements appeared to stagnate (Figure 2, graph C). These postural adaptations may be due to relatively rapid motor-task learning or adjusting neuromuscular functioning during postural control. Studies have shown that this kind of rapid adjustment is likely due to the body's ability to quickly learn and implement effective postural strategies in response to repeated perturbations. Specifically, this phenomenon is linked to the nervous systems capacity to organise sensorimotor feedforward and feedback mechanisms which are crucial for maintaining stability (Woollacott and Shumway-Cook, 2002; Peterka, 2002)

The ten trials shows that the peak velocity of COP In both backwards and forwards directions, at first rapidly increased as a response to the initial onset of the unpredictable perturbation. Subsequently, there was a significant reduction in the COP velocity, which became steady after repeated exposure to the perturbation. Kennedy et al (2013) also found that this was the case, the first phase of COP displacement in their study was quite large, however, an improved postural response occurred rapidly and therefore a significant decrease in COP displacement was found between trials one and three in both forward and backwards directions. The results highlight that the adaptation to the disturbance was quicker in terms of the peak backwards velocity compared to the peak forwards velocity. This could indicate that natural responses to backwards falls counteracts the movement more aggressively than forward falls, possibly in this case as the risk of injury is increased. Previous research has highlighted that COP could adapt due to repeated exposure to perturbations, providing an improved postural stability over time (Horak et al, 1989). Maki and McIlroy (2007) demonstrated that adaptation in COP dynamics reflected anticipatory and reactive adjustments during postural control. The significant differences in the rate and magnitude of adaptation shown in this study compared with other work may be caused by differences in perturbation magnitude, frequency, direction, or specifics of the task used (Miall et al, 2004; Wolpert and Flanagan, 2016). These discrepancies could also be based on methodological differences, such as the accuracy of COP measurement or characteristics of the participants and their demographics (Palmieri and Ingersoll, 2002; Woollacott and Shumway-Cook, 2002). Neural mechanisms underlying these may depend on the individual's previous experience in controlling balance or intrinsic neuromuscular properties (Casadio et al, 2013).

For the change of frequency (moment at which platform oscillated at a faster speed), it was observed that no changes to the peak forward COP velocity across trials (Figure 6 graph B). By contrast, a linear reduction in peak backwards COP velocity was observed in response to the first change of platform direction following the increase in platform frequency (Figure 6, graph C). With that being said, peak backwards COP velocity observed during the first trial was only significantly different from trials 7 and 10, implying that there is some complexity within the interactions between the sensory inputs and motor outputs as the system adapted to faster oscillatory perturbation frequency with increased demands (Peterka, 2002). As the peak forwards COP occurs first, this is likely explained by the unpredictability and reactivity of this response (Maki and McIlroy, 1997). Longer times for COP adaptation to changes in frequency would, therefore, imply that both feedforward and feedback mechanisms in the control of

balance are of greater importance thank specifically one element alone. This may suggest that the CNS requires more time to process and integrate the sensory information of faster perturbations and allow for the correct motor outputs for the appropriate responses (Keyser et al, 2023). It was suggested by Keysner et al (2023), that under conditions of increased unpredictability and complexity, such as that applied in this current study, a change in the effectiveness of the anticipatory postural adjustments is observed, showing a slower adaptation rate to the changes in frequency. These dynamics point out the adaptability of the human postural control system but also its limit under rapidly changing and unpredictable conditions. However, regarding this, this human body can adapt extraordinary abilities to environmental changes and demands in which there is a bounded efficiency related to the frequency and variability of those changes (Lejarraga and Pindard-Lejarraga, 2020).

6.2 EMG Activity and Muscular Activation

Adaptation through EMG activity during the perturbation trials gives a clear insight into the strategies of muscular responses during balance disturbances. The findings indicate that EMG activity of the TA and GM muscles were significantly reduced across the 10 trials. As a primary dorsiflexor, the TA muscle was explicitly active at the onset of the perturbation, presumably down to the involvement in antagonising the forward shift of the body's COM. The GM was essentially involved in plantarflexion and highlighted significantly greater activation as a 'counter-regulatory' response to backward shifts. This muscular adaptation pattern is found to be relative to Maki and McIlroy (2007) and their work in which they observed strong adaptations in the muscular response of the lower limbs to balance perturbations. It was further observed that muscular activation decreased over time, which was explained by an increased input of the neural pathways and motor patterns with subjects developing familiarity with perturbations. This can also be referred to as habituation due to adaptation through the repetition of continuous perturbations (Keshner et al, 1987). On the contrary, differences in muscle activations levels reported may be due to different magnitudes of perturbations and/or characteristics of individuals themselves (Tang et al, 1998).

Stabilisation in the COP trajectories highlighted an increase in the overall efficiency of postural control while muscular activations decreased indicating adaptation. The results have also represented some inconsistencies in the rate at which EMG and COP adaptations took place. This is due to the fact equilibrium of COP often precedes any significant reduction in the

activation of the muscles, which would suggest that the initial muscular responses may be more reactive rather than anticipatory (Maki and McLlroy, 1997; Massion, 1994).

With the increase of perturbation frequency, the muscular responses demonstrated a pattern of adaptation. As the frequency of the platform increased it firstly increased much higher activation levels in both the TA and GM, representing an increased defence mechanism to the unexpected faster disturbance. Throughout the trials, the level of activation initially subsides due to the neuromuscular systems adaptations to the increase of frequency. This adaptation was proven to be slower than the responses to the onset on the perturbation, suggesting that the sensory and motor systems require more to equip and predict for increased frequency (Peterson and Moritz, 2015).

6.3 N1 Response and Cortical Adaptation

The results highlight significant decreases in the amplitude of the N1 potential throughout the study which indicates cortical adaptation to repeated balance perturbations. High N1 amplitudes were initially recorded, reflecting high cortical activity in response to the unpredictable balance disturbances. As participants continued through each trial, the N1 potential reduced overtime, suggesting that the cortical regions involved became significantly discreet in processing the sensory information relative to such perturbations. The amplitude of the N1 diminished subsequently, suggesting that the adaptation occurred at the neural level, a phenomenon known as the description that an individual's synapses become more efficient allowing for the movement of relevant sensory and motor data to migrate to the area where the information is needed (Dayan and Cohen, 2011). Neural adaptation reduces the need for activation of large numbers of cortical neurons and therefore decreased the N1 response (Dayan and Cohen, 2011). This process is essential for accelerating the rate whereby the brain can produce an accurate reaction to future stimuli such as the integration of neural and motor networks that assist in postural control (Wolpert and Flanagan, 2001).

The changes of the N1 amplitude provide crucial information on the functions of the cortex in long-term balance control. The decrease of the N1 response with repetitive perturbations indicates the decrease of cortical involvement, which is comprehensive in the initial state of balance disturbances, with anticipation and adaptation in instances after. This adaptation decreases the amount of effort and demand from the cortices so that the actions can be performed faster and automatically when it comes to balance disturbances (Sozzi et al, 2020).

These results underpin the features of the primary motor cortex of the CNS as the main cortices that controls posture and balance. Therefore, awareness of these mechanisms is essential in attempt to control such plasticity evident within the cortex to enhance postural stability within individuals, particularly the elderly or those affected by neural disorders (Pollock et al, 2000)

The adaptation of the N1 response to changes in frequency of perturbations is a critical aspect of how the cortex adjusts to increased demands on balance control. When the frequency of the perturbation increased, the initial N1 amplitudes were notably larger, reflecting a heightened cortical response to the unexpected velocity of the platform (Purohit and Bhatt, 2022). This suggests an increased attentional demand and sensory processing load caused by faster perturbations. Mochizuki et al (2010) highlight that N1 responses increase with the intensity of the perturbation and suggests that the N1 is relative to the processing of multisensory input linked to sudden changes in task conditions and postural control (Maki and McIlroy, 2007) which is evident in the results. Over the 10 trials, the amplitude of the N1 response decreased over time, indicating that the cortical areas became more efficient in processing the sensory inputs at increased frequencys. This cortical adaptation suggests a shift from an increased reliance on sensory input processing towards a more efficient, predictive neural mechanism that adapts to faster perturbations with reduced cortical activity (Mochizuki et al 2008).

6.4 N1 Response to Forward and Backward Directional Changes and Oscillatory Frequency

This study also observed the cortical responses to directional changes in perturbations after a change of frequency. As the perturbation direction switched from anterior to posterior, the N1 response initially exhibited increased amplitudes, similar to the response seen with the increase of frequency. This amplified N1 response to directional changes post-frequency velocity likely reflects the cortical systems need to re-establish a new anticipatory and reactive model that assists in faster and directionally variable perturbations (Hulsdunker et al, 2017). Over time, as the participants adapted to the changes in dynamics, the N1 amplitudes diminished, which can be suggestive of an integration of adaptive strategies that efficiently assists with changes in both direction and frequency, overall enhancing postural equilibrium. These observations are crucial as they demonstrate the cortices flexibility and capacity to adapt to multiple challenges and environments specifically those relative to balance (Solis-Escalante, 2019; Taubert et al, 2016). These findings provide deeper insights into the complex interplay between cortical activation, sensory processing, and motor control, emphasising the

sophisticated nature of human balance control systems. The ability to rapidly adapt to responses caused by changes in environmental demands is fundamental for research and rehabilitation aimed at improving balance and preventing falls, particularly in environments where sudden changes in balance are common (Horak, 2006; Shumway-Cook and Woollacott, 2000).

6.5 Interaction Between Cortical N1 Activity and Muscular Activation

These changes in the N1 amplitudes provided useful information on some crucial aspects of the cortex and its contribution to long-term postural control. The overall decrease in the N1 response amplitude shows that, although the cortical structures are revealing a high level of apprehension towards processing the balance-related disturbance during the initial stages of the reaction to the perturbation, the thalamus and other neural network possess the ability to anticipate the following challenges and probable scenarios attached with balance, and therefore reduces the cortical concern over repeated exposure to the perturbation (Dakin and Bolton, 2018). As a result of this, there is a decrease in cognitive load which is apparent with a decrease in the responsible neural resources, consequently increasing the frequency of the balance recovery process from the perturbation to make it more automatic and habitual (Shumway-Cook and Woollacott, 2000; Woollacott and Shumway-Cook, 2002; Seidler et al, 2004). It is apparent, that the cortical areas are working together for the concern of balance and posture, therefore, it is critical to understand the processes on which these adaptations are based, as well as the possibility of using interventions that have the potential to enhance such cortical plasticity in order to optimise the regulation of balance in the elderly and neurologically impaired (Hackney et al, 2017).

Overall, this study showed similar patterns and interactions between the cortical N1 potential and the muscular activation patterns recorded from the TA and GM muscles (in regard to the data collected and analysed). As the amplitude of N1 decreased over trials due to the cortical mechanisms adapting to the unpredictable perturbations, a reduction in both the amplitude and duration of the muscular activity was also apparent. This interaction indicates the balance between preparatory actions of the cerebral cortex and the precise muscular postural activities necessary for postural equilibrium. The changes in N1 amplitudes were shown to be consistent in line with changes in the reduced muscular activities, crediting that, the cortex gains more efficiency in predicting and processing feedforward and feedback information of balance postural disturbances, there should be minimal recruitment of the muscles to maintain

postural equilibrium over time due to adaption and habituation (Mochizuki et al, 2008; Horak and Macpherson, 1996). With the decrease in N1 cortical activity, reduced muscular activation allows for the transparency of integrative regulatory aspects of balance (Kuehn et al, 2018). This interaction suggests that there is an advanced neuromuscular organisation that optimises the response to external perturbations both through adaptation and anticipation (Horak, 2006; Woollacott and Tang, 1997).

Overall, these results support the hypothesis that the initial activity at the cortical and muscular levels is part of an adaptation process in which each system curates an appropriate response strategy. In terms of this, the systems are more efficient, and less cortical or muscular activity is necessary for maintain postural control and stability. The positive of such findings is that they provide insight into how neural control enables balance and could therefore, contribute to future rehabilitation strategies to improve balance in populations with different levels of impaired neuromuscular function. This study demonstrated the dynamic interaction of cortical and muscular adaptations in response to balance perturbations. The results emphasize the efficiency of neuro-muscular coordination across repeated exposure to perturbations, implying such adaptive conditions for enhanced rehabilitative strategies aimed at postural control and balance improvement in varied populations.

7. Limitations

Whilst this study was successful and carries many strengths, it was also limited by the small sample size that was recruited. Larger populations when relative to trial-by-trial measures in balance performance can quantify an interaction between cortical N1 activation and balance recovery characteristics, i.e. N1 response may represent cognitive events such as attentional demands or postural threat (Little and Woollacott, 2015), which may still impact motor control (Payne and Ting, 2020). Furthermore, the sample size within this study could have limited the opportunity to detect significant differences in muscular activation and patterns, therefore with a larger sample size, there may have been greater variability in the results which would have allowed for stronger conclusions for the cortico-muscular mechanisms related to postural control (Keenan et al, 2012; Farina and Merletti, 2001).

Previous researchers have found that there are numerous technological limitations that can restrict research into PEP's. Whilst measures of EEG have many positives such as, excellent temporal resolution (that captures transient neuronal activities and non-invasive techniques, it is often limited by the spatial resolution (1-10 cm) (Sakkalis, 2011), Consequently, there is a restriction to precisely predict and locate the underlying cortical source areas that generate the PEP's and other neuronal activity measured at the scalp (Michel and Brunet, 2019). For the purpose of divulging into the specific role of each cortical area, there is a requirement of additional applications/and or combinations of neural techniques i.e., functional near-infrared spectroscopy (fNIRS), transcranial magnetic stimulation (TMS), and functional magnetic resonance imaging (fMRI) (motor imagery of perturbation and balance reactions). Relatively, inhibiting or disrupting necessary and specific cortical locations using TMS or stimulating such cortical areas (using transcranial direct current stimulation tDCS), whilst measuring and analysing the amplitudes and timing patterns of muscular activation and PEPs, alternatively, lesion studies which examine PEPs and postural responses from cortical stroke patients could provide insight to the role of cortical activations in balance control (Varghese et al, 2011).
Another limitation to EEG is the excessive artifacts that may have corrupted the data, i.e. eye blinks/movements, head/facial movements, whole body movements and line noise from other measurement factors such as EMG, COP, etc.

The anticipation of unpredictable postural perturbations in a controlled setting such as laboratories may exaggerate the complexity of balance control when compared to real life situations (Horak and Macpherson, 1996). Individuals are exposed to a multitude of environmental factors including different sensory conditions which may significantly influence anticipatory postural adjustments (Peterka, 2002), consequently, the anticipatory responses observed in laboratory settings may apprehend the adaptive strategies employed by individuals in real life dynamic and unpredictable environments. In addition, the controlled nature of a laboratory may lead to task specific anticipatory responses that conclude in other scenarios (Horak and Macpherson, 1996), participants may adopt anticipatory strategies that are relevant to the specific characteristics of the experimental task, such as timing of perturbations, change of frequency, and direction of the perturbations rather than the natural adaptive mechanisms.

Finally, the inclusion criteria for this included only adults aged 22-39 without any neurological disorders, whilst this approach allowed us to control any potential discrepancies associated

with age-related changes in sensorimotor function and neurological impairments, it may have limited the validity of the findings regarding broader populations. Horak and Macpherson (1996) highlight that older individuals that carry neurological conditions such as Parkinson's disease, multiple sclerosis or stroke may exhibit different or less anticipatory strategies and more sensory-driven responses compared to younger, healthy adults (Tasseel-Poncho et al, 2015; Massion et al, 2004) due to altered sensorimotor function and impaired balance control which can influence their ability to anticipate and adapt to postural perturbations. Furthermore, focusing on this demographic, our data does not allow for age-related variations in postural adjustments. Older adults often experience age-related declines in sensorimotor function, proprioception, and muscle strength which can affect the ability to anticipate and reactively respond to postural perturbations (Maki and McIlroy, 2006). In studies of responses evoked by AP perturbations, previous researchers have found that older adults do not adapt and use anticipatory postural adjustments in response to unpredictable EIPs in the AP direction (Bugnariu and Sveistrup, 2006; Bugnariu and Sveistrup, 2001).

8. Conclusions

This thesis aimed to provide insight into the mechanisms involved in balance control and adaptation in response to unpredictable superficial perturbations from analysis of COP positioning, EMG muscular activity, and cortical N1 responses. The main research question addressed the adaptation of postural control mechanisms of the human body over repeated perturbations in terms of muscular and neural responses and the impact of on the efficiency of balance control in young healthy adults. The findings of COP positioning provide insightful information on how balance is maintained and adapted over time. Subsequently, the strategies are considered to have been acquired with the COP's adaptation, which lasted a shorter-term learning of the participants response behaviours to the disturbance of the perturbations. Furthermore, when the direction moved in a backwards translation, COP adjustments were significantly faster than in the forward direction, indicating the directional responsive properties of the balance control system. This adaptation was tested further by modulating the frequency of the perturbation and making it faster in the final 15 cycles of each individual trial. However, COP responses showed as destabilised in the beginning, and a new level of stability was gradually achieved over the 10 trials, underlying the dynamic capacity of the postural control system to adapt to increasing demands.

Concerning the muscular responses, significant adaptations occurred in the muscular activation patterns of the major lower limb muscles, particularly the TA and the GM. It was observed that the TA was significantly more active than the GM in the earlier stages of the trials and then reduced its activity over time. This suggests that the TA muscles are of prime importance in opposing the forward shifts of the body's COM. Conversely, the GM muscle was more active in the backwards translation of the perturbations and thus supported the critical role of this muscle helping restore upright stability by plantarflexion. The present findings emphasise the selective recruitment of muscles with the direction and nature of balance perturbations and, in doing so, show the specificity of the neuromuscular adaptations to maintain stability.

The N1 potential as a marker for cortical activity was analysed during the perturbation events following amplitude changes. A systematic reduction of the N1 response was found with increasing trials. Initially, large amplitudes reflected vigorous cortical processing activity while subjects were dealing with new sensory information relative to the postural disturbance. Over time, when the subjects had adapted to the perturbation, the decrease in amplitude of the N1 indicates that fewer cortical resources were required to deal with the same kind of balance disturbances and therefore cognitive demand was reduced. This reduction indicates better cortical efficiency due to the brain modifying its response to familiar stimuli. A key finding in the study was that it is evident that the modulation of the N1 response in the cortex is related to the modulation of the muscular reactions. As N1 amplitudes diminished, corresponding muscular activity progressively diminished the muscular responses, particularly in muscles that initially showed high response levels. The coordination of this modulation demonstrates a superior coordination of the anticipatory adjustments performed in the brain with the muscular actions necessary to preserve and fulfil postural equilibrium. Such results indicate that the overall postural control strategy was more efficient throughout each of the subjects and developed through improved cortical control and allowing moderation of reliance of muscular responses.

8.1 Synthesis of Findings

Integrating the findings from the dynamics of the COP, EMG activity, and cortical N1 adaptation would provide a more comprehensive view of these complex interactions and efficiencies of the balance control system. However, this study shows adaptive mechanisms within the muscular and neural domains are tightly integrated to provide an optimal response to balance

perturbations. One of the critical features of this system is the ability to learn after repeated exposures and consequently reduce the load on the neural and muscular systems. These features will go on a long way to realising more effective and efficient targeted treatment and rehabilitative strategies in terms of developing an enhanced state of balance and fall prevention, particularly in high-risk populations. These findings confirm investigations of the current theories of balance control and proceed by describing given adaptations at various level within the control system. As a result, the dynamic capacity of the human balance system is demonstrated and research in this area has relevance to neurophysiology, rehabilitation and biomechanics. This comprehensive analysis, alongside other pioneers in this field will lay the groundwork for future research to further dissect the individual components of balance control and their interactions under varied and more complex environmental demands.

8.2 Future Research

This research may be continued, proceeding with an investigation of the mechanisms of balance control in larger populations and demographics that include older adults, children, and various neurological patients such as those with Parkinson's disease or Multiple Sclerosis, in order to gain further insight into the differences in age and pathology regarding balance adaptation. Furthermore, the inclusion of newer technologies, such as applications of virtual reality systems for slightly more dynamic and realistic environments in balance control and adaptation testing. Finally, the combination of EEG with functional MRI, or near-infrared spectroscopy could be essential to localise brain activities more precisely and allow extraction of underlying neural mechanisms in more detail.

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