School of Civil & Mechanical Engineering

Study of Human Postural Control based on Electroencephalography Signals

Kwang Leng Alex Goh

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Declaration

To the best of my knowledge and belief, this thesis contains no material previously published by any other person except where due acknowledgement has been made.

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

Human Ethics The research presented and reported in this thesis was conducted in accordance with the National Health and Medical Research Council National Statement on Ethical Conduct in Human Research (2007) – updated March 2015. The proposed research study received human research ethics approval from the Curtin University Human Research Ethics Committee (EC00262), Approval Number ENG-71-14

Signature: AlexGoh.....

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Abstract

Human movement requires adequate postural control. Optimal movement performance is benefited by the integration of sensory information from a multitude of systems including visual, vestibular and somatosensory systems. Stimulation of these individual sensory systems induces alterations in body sway. However, the role of cortical activity in maintaining balance remains unclear. Supporting evidence demonstrates attention and cognitive function affect postural stability. Thus, there is significant value to investigate the underlying cortical activity in control of balance response.

Cortical activity can be recorded using electroencephalography (EEG) for its non-invasive, portability, high temporal resolution and low-cost compared to other neuroimaging techniques. There are two main approaches to analysis EEG signals in postural control studies – evoked potentials (EP) and EEG Rhythm. Cortical representation of balance processing in EP can be identified by perturbations-evoked responses (PERs). However, studies so far have focused primarily on perturbations of the somatosensory system (tactile/proprioceptive). The visual processing systems have been shown to influence postural sway in both an immersive virtual reality environment and on a moving platform. However, not much research has been carried out in the study of the cortical representation of the visual contribution leading to postural control. On the other hand, EEG Rhythms such as theta, alpha, beta and gamma bands have been showned to be related to balance maintenance. Responsive integration and weighting of sensory information for postural control are critical for effective equilibrium. Thus the frequency bands could lead to better understanding of higher level postural control (i.e. sensory reweighting).

The purpose of this thesis was to extend the understanding of cortical involvement in human postural control. Specifically, the goals of this thesis were to examine the cortical role of vision in balance response and different postural demand tasks. Also,

this thesis extended its validity towards individuals with autism spectrum disorder (ASD) who have significant symptoms of sensory processing. The first study investigated the PERs to visual perturbation of postural stability in young typically developed (TD) adults. The second study investigated the effect of postural demand tasks and sensory input on cortical activity in young TD adults. The third study examined the PERs to visual perturbation under varied postural stability conditions in adults with and without ASD. Lastly, the fourth study was designed to enable classification of adults with ASD and TD adults based on machine learning with features derived from force plate data (i.e. centre of pressure) and EEG Rhythm.

The findings of these studies provide direct and indirect evidence for the cortical contribution to the visual system and postural demand in human balance control. The results also provide insights into the cortical responses which contributed to sensory integration in individuals with ASD. The future direction may focus on exploring the neurophysiological mechanism in balance control under altered sensory conditions and other neurological disorders. Ultimately, this thesis provides critical insight into the mechanisms of adaptive and maladaptive postural control.

Contents

Publications

- Goh, K. L., Morris, S., Rosalie, S., Foster, C., Falkmer, T., & Tan, T. (2016, March). Typically developed adults and adults with autism spectrum disorder classification using centre of pressure measurements. In *Acoustics, Speech and Signal Processing (ICASSP), 2016 IEEE International Conference on (pp. 844-848)*. IEEE.
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- Goh, K. L., Morris, S., Ring, A., & Tan, T. (2017). Typically developed adults and adults with Autism Spectrum Disorder classification using centre of pressure and electroencephalography signals.

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Abbreviations

ACC	Anterior cingulate cortex
ADI-R	Autism Diagnostic Interview-Revised
ADOS	Autism Diagnostic Observation Schedule
AP	Anterior posterior
AREA	Sway area
ASD	Autism Spectrum Disorder
AUC	Area under receiver operator characteristic
BOS	Base of support
COM	Centre of body mass
COP	Centre of pressure
CPz	Central-parietal site
Cz	Central site
DSM	Diagnostic and Statistical Manual of Mental Disorders
DT	Decision tree
EEG	Electroencephalography
EOG	Electrooculogram
EP	Evoked potential
ERD	Event related desynchronization
ERP	Event related potential
FCz	Frontal-central site
Fz	Frontal site
GND	Forehead ground
HF	Horizontal flow
ICA	Independent component analysis
ICA-R	Independent component analysis with reference
KNN	k-nearest neighbour
LSM	Least squares means
M1	Primary motor cortex
ML	Medial lateral
MMN	Mismatch negativity
MV	Mean velocity
NB	Naïve Bayes
NN	Neural network

OF	Optic flow
Oz	Occipital site
PER	Perturbation evoked response
PO	Parietal-occipital site
Pz	Parietal site
RANGE	Total range
RD	Resultant distance
RF	Random forest
RMS	Root mean square
ROI	Region of interest
SEP	Somatosensory evoked potential
SIT	Sitting task
SMA	Supplementary motor area
STD	Standing task
SVM	Support vector machine
TD	Typically developed
TOTEX	Total excursions
TP	Temporal-parietal
VO	Visual occlusion
VT	Visual transparent

Chapter 1

Introduction

1.1 Motivation

Humans need to adapt their postures to the changing environment to perform the activities of daily living. Inability to adapt will not only affect the psychosocial functions of the individuals but also increase the risk of fall-related injuries (de Mettelinge et al., 2013; Young & Williams, 2015; Zijlstra, Mancini, Chiari, & Zijlstra, 2010). Postural control is a complex skill that integrates multimodal sensory systems such as visual, vestibular and proprioceptive systems to respond to complex sensory environments. Thus, the ability to reweight sensory information effectively in response to alterations in environmental conditions, otherwise known as adaptive postural control, is crucial for maintaining postural stability.

The involvement of subcortical structures such as spinal cord, brain stem, basal ganglia and cerebellum in maintaining postural control has been demonstrated in human clinical studies for the last two decades. However, it is only recently that evidence of changes in cognitive function and attention have been associated with changes in postural stability, even for typical adults without cortical damage (Jehu, Desponts, Paquet, & Lajoie, 2015; Remaud, Boyas, Lajoie, & Bilodeau, 2013; Slobounov, Hallett, Stanhope, & Shibasaki, 2005). Moreover, it is evident that the cortex plays a role in motor adjustments to recover balance (Ackermann, Diener, & Dichgans, 1986; Dietz, Quintern, & Berger, 1984, 1985; Dietz, Quintern, Berger, & Schenck, 1985; Dimitrov, Gavrilenko, & Gatev, 1996; Duckrow, Abu-Hasaballah,

Whipple, & Wolfson, 1999). Thus, there is a growing interest in investigating the underlying cortical mechanisms used in adaptive postural control.

The role of the cerebral cortex in postural control is the subject of the recent investigation. Emerging studies using the dual-task paradigm, which requires individuals to perform cognitive and balance tasks simultaneously, have indicated attentional resources are required in balance control (Bogost, Burgos, Little, Woollacott, & Dalton, 2016; Fujita, Kasubuchi, Wakata, Hiyamizu, & Morioka, 2016; Little & Woollacott, 2015; Remaud et al., 2013). Furthermore, studies of visual attention, perturbation evoked responses and the use of transcranial magnetic stimulation to modulate specific cortical locations have provided evidence that coupled cognitive demands and cortical control (See review (Maki & McIlroy, 2007)). Recent findings offer support for the contribution of cortex towards the control of early compensatory arm reactions following whole-body perturbation (Bolton, Williams, Staines, & McIlroy, 2012). Additionally, it seems that balance is also managed with the role of cerebral cortex within the distributed network (Bolton, 2015).

Cortical activity related to postural control can be obtained using electroencephalography (EEG). EEG is a technique that monitors the electrical activities of the brain through the placement of electrodes on the scalp (Berger, 1929). Despite the introduction of other neuroimaging devices such as computerized tomography, functional near-infrared spectroscopy, functional magnetic resonance imaging, magnetoencephalography and positron emission tomography, EEG remains popular in both research and clinical settings for the reason that it is a safe, inexpensive, quiet and non-invasive way of monitoring the brain (Evans & Abarbanel, 1999; Luck, 2014). Also, EEG has a very high temporal resolution which can record on the order of milliseconds rather than seconds. Thus, the cortical activity obtained using electroencephalography (EEG) during postural control can provide valuable insights into the cortical contribution to balance control.

Nevertheless, the understanding of cortical involvement in postural control is still limited. Understanding the contribution of the cortex to postural control can provide insights into the mechanisms of adaptive and maladaptive postural control.

Thus, the purpose of this thesis was to extend the understanding of cortical involvement in human postural control using EEG. Specifically, the goals of this thesis were to examine the cortical role of vision in balance response and different postural demand tasks. Finally, the findings of the thesis work were extended to explore the postural control mechanisms in individuals with Autism Spectrum Disorder, for whom postural control is unusual and is highly associated with sensory impairments (Lim et al., 2017). Lastly, the thesis focussed on enabling classification of adults with ASD and TD adults based on postural control features from the force plate measurements of the centre of pressure and EEG.

1.2 Scope and Objectives

This thesis aims to provide a better understanding of cortical involvement in human postural control, specifically the cortical role of vision, postural demand tasks and sensory impairments in Autism Spectrum Disorder (ASD). In addition, this thesis explored ways to classify adults with and without ASD from postural control features.The detailed objectives of this study are as follows:

- To investigate the contribution of the visual system to postural control at the cortical level by comparing perturbation evoked responses to the visual occlusion condition under varying conditions of postural demand (i.e. standing and sitting) in typically developed adults.
- To investigate the effect of postural demand and visual input on cortical activity (i.e. alpha and theta activity) in typically developed adults by assessing them in conditions of sitting and standing during the availability and removal of visual input (i.e. visual transparent and visual occlusion)
- To investigate the perturbation evoked responses which contribute to the processing of visual occlusion in standing and sitting in young adults with and without Autism Spectrum Disorder.
- To classify typically developed adults and young adults diagnosed with Autism Spectrum Disorder based on the centre of pressure and EEG rhythm measurements during quiet standing.

1.3 Contributions of the thesis

In this thesis, the research focused on examining the cortical role of vision in balance response, postural demand tasks and sensory impairments in Autism Spectrum Disorder (ASD). The original contributions are as follows:

1. **Postural and cortical responses following visual occlusion in standing and sitting tasks in young typically developed adults**
   Perturbation-evoked responses (PERs) to a physical perturbation of postural stability have been detected using electroencephalography. However, PERs to a visual perturbation of posture have yet to be reported. Ten healthy young adults were exposed to unpredictable visual occlusion mediated through liquid crystal glasses. The responses to postural perturbation by sudden visual occlusion are similar in nature to that seen in relation to a physical perturbation. The study informs the relative importance of vision to postural stability, postural set and provides a protocol to objectively assess sensory-based postural disorders.

2. **Cortical activity in postural tasks with visual conditions**
   Earlier studies have used the spectral analysis of electroencephalography theta (4-7 Hz) and alpha (8-12 Hz) oscillations to evaluate cortical involvement in postural control. However, the information transmission when down-weights less reliable information and up-weights reliable information during balance maintenance could also contribute to the EEG rhythms. Thus, the purpose of this study was to investigate the effect of postural demands and visual input on cortical activity in healthy young adults. The study revealed that elevated theta and alpha activities during changes in postural demand suggesting that more brain resources are allocated to postural tasks that are more demanding. The results of this study establish a link between postural task and cortical activity.

3. **Postural and cortical responses following visual occlusion in adults with and without Autism Spectrum Disorder**

   Sensory impairment is highly disruptive and limits the performance of activities of daily living, especially for individuals with ASD. It is evident that individuals with ASD use visual and proprioceptive information differently than TD individuals. The aim of the study was to investigate the PERs to visual perturbation under varied postural demand conditions (standing and sitting) in adults with and without ASD. The finding supports evidence that the processing of proprioceptive information is intact in adults with ASD. The study also provides the first reported evidence indicating that postural response utilises more cortical resources to maintain balance upon visual perturbation in individuals with ASD.

4. **Typically developed adults and adults with Autism Spectrum Disorder classification using centre of pressure and electroencephalography signals**

   Due to the rise in the prevalence of ASD, there is an increased demand for ASD diagnostic assessments. However, most of these tools were developed for the use in children with ASD and may therefore not be adequate to identify ASD in adult populations. Moreover, current ASD diagnostic tools are expensive, time-consuming and require extensive training to use. The aim of this study was to use machine learning to classify TD adults and adults diagnosed with ASD based on COP and EEG measurements during quiet standing. The AUC result of this work is greater than the accuracy of the currently used Autism Diagnostic Observation Schedule Assessment (ADOS-2 Module 4) and the accuracy of Autism Diagnostic Interview-Revised assessment (ADI-R).

1.4 Thesis Outline

This thesis consists of eight chapters, and each chapter provides a stepping stone for the subsequent chapter or complements a previous chapter. The rest of this thesis is structured as follows:

- **Chapter 2** provides a brief introduction to the postural control system and neurophysiological aspects of postural control. An overview of the role of cortex in postural control and EEG postural control related studies are presented. Lastly, Autism Spectrum Disorder and sensory integration are briefly outlined in this chapter.
- **Chapter 3** provides a brief background on the techniques that were used to quantify and assess the physiological mechanisms of postural control.
- **Chapter 4** examines the perturbation-evoked responses to visual occlusion under varying conditions of postural demand in typically developed adults.
- **Chapter 5** investigates the effect of a postural task on cortical theta and alpha activities under differing visual conditions (i.e. visual transparent and visual occlusion) in typically developed adults.
- **Chapter 6** investigates the perturbation-evoked responses to visual perturbation under varied postural demand conditions (i.e. standing and sitting) in adults with and without Autism Spectrum Disorder.
- **Chapter 7** presents the machine learning models and quantifiable features from EEG and force plate measurements of the centre of pressure that best classify typically developed adults and adults with Autism Spectrum Disorder in quiet standing.
- **Chapter 8** provides a summary and findings of this thesis. This chapter also provides information about the future work.

Chapter 2

Literature Review

2.1 Overview

This chapter begins with a brief introduction to the postural control system and neurophysiological aspects of postural control. Following this, an overview of the role of cortex in postural control is presented, and the postural control studies related to electroencephalography (EEG) is thoroughly discussed in Section 2.5. In Section 2.6, a brief outline of autism spectrum disorder (ASD) and sensory integration is provided.

2.2 Postural Control System

The postural control mechanism in human is commonly described and visualised as a continuous process of stabilising an inverted pendulum (Loram & Lakie, 2002; Mergner, Maurer, & Peterka, 2003; Winter, 1995; Yoshikawa, Suzuki, Kiyono, & Nomura, 2016). In fact, postural control is a complex skill based on the interaction of multimodal sensory systems which are integrated to interpret and respond to complex sensory environments. Typically, postural orientation and postural equilibrium are two functional goals of postural control (Horak, 2006; Horak & Macpherson, 2011; Jacobs & Horak, 2007). Postural orientation is defined as the alignment of the trunk and head concerning gravity, support surface, visual surround and an internal reference. On the other hand, postural equilibrium is defined as the coordination of movement strategies to stabilise the centre of body mass (COM)

during both self-initiated and externally triggered postural perturbations (Horak, 2006; Maki & McIlroy, 1997; Massion, 1994; Winter, 2009). Postural equilibrium (or commonly known as balance) is particularly interesting as it maintains the relationship between the COM and the base of support (BOS) (Horak, 2006; Maki & McIlroy, 1997; Massion, 1994; Parokaran Varghese, 2016; Winter, 2009). It is also the state of an object when the resultant force acting upon it is zero (Bell, 1998). Above all, postural control is the act of maintaining, achieving or restoring a state of balance during any posture or activity (Horak, 1987; Massion, 1994; Pollock, Durward, Rowe, & Paul, 2000) whereas adaptive postural control involves adjusting sensory and motor response to postural and environment demand (Shumway-Cook & Woollacott, 2007). In this thesis, the cortical responses in adaptive postural control are emphasised. The literature concerning postural equilibrium (or balance) will be outlined in the following sections.

There are three major sensory systems that contribute to postural control: the visual, vestibular and proprioceptive systems. The importance of these sensory systems in balance maintenance are well documented; stimulation of individual senses such as vision (Bonnet & Baudry, 2016; Day, Steiger, Thompson, & Marsden, 1993; Dichgans, Mauritz, Allum, & Brandt, 1975; Dornan, Fernie, & Holliday, 1978), vestibular (Fitzpatrick & McCloskey, 1994; Matsugi et al., 2017; Pavlik, Inglis, Lauk, Oddsson, & Collins, 1999; Peterka & Benolken, 1995) and proprioception (Bergin, Bronstein, Murray, Sancovic, & Zeppenfeld, 1995; Eysel-Gosepath, McCrum, Epro, Brüggemann, & Karamanidis, 2016; Teasdale, Stelmach, & Breunig, 1991) induce alterations in body sway. Depending on the environment and the postural disturbance, the central nervous system down-weights less reliable information and up-weights reliable information to achieve balance control (Pasma, Boonstra, Campfens, Schouten, & Van der Kooij, 2012). While healthy people predominantly rely on their proprioceptive system to maintain stance (Ben-Itzhak et al. 2010; Peterka 2002), closing the eyes does increase postural sway (Kuo et al. 1998). The process of regulating the contribution of each sensory system to maintain balance is referred to as sensory reweighting (Assländer & Peterka, 2014, 2016; Kuo, Speers, Peterka, & Horak, 1998; Oie, Kiemel, & Jeka, 2002). In a well-lit

environment with a strong base of support; healthy subjects typically rely on vision (10%), vestibular (20%) and proprioceptive (70%) information (Ben-Itzhak, Herman, Giladi, & Hausdorff, 2010; Peterka, 2002). However, if subjects were to stand on an unstable surface, weighting on vestibular and visual is increased as the dependence on proprioceptive information decreases for postural equilibrium (Liston, 2013; Peterka, 2002). The ability to reweight sensory information effectively due to the alteration of environments requires an adaptive and sensitive postural control system and is crucial for maintaining postural stability.

2.3 The Neurophysiology of Postural Control

The involvement of subcortical structures such as spinal cord, brain stem, basal ganglia and cerebellum in maintaining postural control has been demonstrated in animal models (Deliagina, Orlovsky, Zelenin, & Beloozerova, 2006; Magnus, 1926; Sherrington, 1910). However, the results from the animal models have only been generalised to human clinical studies in the last two decades (Horak & Macpherson, 2011; Jacobs & Horak, 2007; Lewko, 1996; Maki & McIlroy, 2007; Mierau et al., 2017; Parokaran Varghese, 2016; Varghese, Merino, Beyer, & McIlroy, 2016). The details of each region for postural control are briefly discussed in the rest of the sections.

The spinal cord and the brain stem have been implicated in the maintenance of human upright stance since last century (Magnus, 1926; Sherrington, 1910). The inability to stand and walk following spinal cord injury results in frustration and often irreversible change in the quality of life (Behrman & Harkema, 2000; Kralj & Bajd, 1989). Furthermore, anticipatory postural reactions to unexpected perturbations were observed absent or delayed in this population (Diener, Ackermann, Dichgans, & Guschlbauer, 1985; Parokaran Varghese, 2016). Likewise, the brainstem plays a similar role as the spinal cord. Evidence from decerebrated cats maintaining balance in response to postural disturbance suggests the involvement of brainstem in postural balance (Honeycutt, Gottschall, & Nichols, 2009; Honeycutt & Nichols, 2006). Thus, the spinal cord and brain stem are crucial

in ongoing postural control that organises movement via the upper motor neuron pathways (Hall & White, 2004).

The basal ganglia appear to be involved in adaptive postural control. The dopamine depletion in the striatum was suggested to give rise to the motor disabilities of Parkinson's disease, demonstrating the dominant role of basal ganglia and its pathways (Halliday, Winter, Frank, Patla, & Prince, 1998; Nelson & Kreitzer, 2014). The basal ganglia seem to be primarily involved in the motor preparatory process by coordinating postural and focal commands during voluntary movements (Rogers, Kukulka, & Soderberg, 1987; Winter, Patla, & Frank, 1990). Similarly, the contribution of the basal ganglia towards adaptive postural control and gaining control of balance correcting responses have been observed in both patients and animals with basal ganglia lesions (Visser & Bloem, 2005). Altogether, the basal ganglia role in postural control is likely to operate at the level of planning, initiation, execution and termination of motor learning and motor programs (MacKay-Lyons, 2002; Wichmann & DeLong, 1996).

Impairment in motor adaptation has been associated with damage to the cerebellum (Bastian, 2008; Izawa, Criscimagna-Hemminger, & Shadmehr, 2012; Thach & Bastian, 2004). Patients with cerebellar lesions demonstrate large postural reactions when experiencing predictable perturbations suggesting that the cerebellum may function to approximate the scaling of the magnitudes of postural response for anticipatory postural adjustment (W.-H. Chang et al., 2010; Horak & Diener, 1994; Jacobs & Horak, 2007). However, patients with cerebellar damage can still improve their motor responses to a physical perturbation suggesting that the brain may engage another neural mechanism to compensate (Criscimagna-Hemminger, Bastian, & Shadmehr, 2010). Overall, the cerebellum plays an important role in the coordination of voluntary movement for postural adjustments (Bastian, 2006; Ioffe, 2013; Yanagihara, 2014).

Although the subcortical structures are important in balance maintenance, there is a role for cortical activity in adaptive postural control (Jacobs & Horak, 2007; Maki & McIlroy, 2007; Slobounov et al., 2005). While it is known that postural control is altered after damage to the cortical area of the brain (Hauer et al., 2003;

Rapport et al., 1993), recent evidence suggests changes in cognitive function and attention alter postural stability even for typical adults without cortical damage (Jehu et al., 2015; Remaud et al., 2013). Moreover, it is evident that the cortex plays a role in motor adjustment to recover balance (Ackermann et al., 1986; Dietz et al., 1984; Dietz, Quintern, & Berger, 1985; Dietz, Quintern, Berger, et al., 1985; Dimitrov et al., 1996; Duckrow et al., 1999). Thus, there is a growing interest in investigating the underlying cortical mechanisms used in the adaptive postural control. Understanding the contribution of these areas in postural control assists in providing insight into the mechanisms of adaptive and maladaptive postural control.

2.4 The role of the Cortex in Postural Control

The potential role of the cerebral cortex has been considered concerning postural control. Emerging studies using dual-task paradigm, which requires individuals to perform cognitive and balance tasks simultaneously, have indicated attentional resources were required in balance control (Bogost et al., 2016; Fujita et al., 2016; Little & Woollacott, 2015; Mierau et al., 2017; Remaud et al., 2013). Furthermore, studies from visual attention, perturbation evoked responses and the use of transcranial magnetic stimulation to modulate specific cortex were sources of evidence that coupled cognitive demands and cortical control (See review (Maki & McIlroy, 2007; Wittenberg, Thompson, Nam, & Franz, 2017)). Additionally, recent findings offer support for the contribution of cortex towards the control of early compensatory arm reactions following whole-body perturbation (Bolton et al., 2012). Therefore, it seems that balance is managed with the role of cerebral cortex within the distributed network (Bolton, 2015). Understanding the cortical process associated with balance control is essential, not only to provide insights on the cortical neurophysiology of impaired postural response but also provides accessibility for neuroplastic change with advanced intervention (Jacobs, 2014).

The cortical response has revealed maximum activation over frontal-central and central electrode sites following balance disturbance. Distinct movement-related potentials before the onset of anticipatory postural adjustment and also the

onset of foot-off during lateral stepping were observed in the mid-line frontal-central region, specifically the frontal-central and central sites (Varghese et al., 2016). The two electrodes were placed above the supplementary motor area (SMA) and primary motor cortex (M1), which were consistent with other literatures reporting the role of SMA and M1 in voluntary and external perturbations (Babiloni et al., 2003; Varghese et al., 2016; Weilke et al., 2001). Traditionally, the cortical response was initially hypothesized generated in anterior cingulate cortex (ACC) for the reason that the cortical response was strongly influenced by predictability and cognitive demand (Adkin, Quant, Maki, & McIlroy, 2006; Jacobs & Horak, 2007; Little & Woollacott, 2015; Mochizuki, Sibley, Esposito, Camilleri, & McIlroy, 2008; Quant, Adkin, Staines, Maki, & McIlroy, 2004). However, a recent study investigated the source of the cortical response in the postural task and cognitive task independently and revealed the source from postural task was generated from SMA and the cognitive task was generated from ACC (Marlin, Mochizuki, Staines, & McIlroy, 2014). Another recent study also supports the previous statement by estimating the source of the cortical response related to a whole-body surface translation with or without performing visual working memory task (dual and single-task paradigm) (Bogost et al., 2016). Moreover, they concluded that pre-motor, primary and SMA and somatosensory area were primary cortical sources related to reactive postural control (Bogost et al., 2016). Furthermore, supporting studies from Slobounov et al. (Slobounov et al., 2005) and Mihara et al. (Mihara, Miyai, Hatakenaka, Kubota, & Sakoda, 2008) point toward the involvement of SMA along with prefrontal cortex in generating motor plans for compensatory balance reactions. In summary, activations of the SMA from frontal-central and central sites have a strong correlation with postural responses.

2.5 EEG related to Postural Control

The cortical activity obtained using electroencephalography (EEG) during postural control provide valuable insights into the cortical contribution to balance control. The EEG rhythm and evoked potentials for static balance control are discussed in

this section.

2.5.1 EEG rhythm related to postural control

Spectral analysis of EEG theta (4 – 7 Hz) and alpha (8 – 12 Hz) oscillations has been used in the evaluation of cortical involvement in induced postural instability. Evidence that visual stimuli projected in a 3D environment induce greater postural instability and stronger EEG theta activity at the frontal-midline areas compared to a 2D environment suggests that EEG theta reflects the recruitment of brain resources to meet the spatial demands of the postural task (Slobounov, Ray, Johnson, Slobounov, & Newell, 2015; Slobounov, Teel, & Newell, 2013). Additionally, an increase in theta activity has been observed when the complexity of visuomotor task increases (Slobounov, Fukada, Simon, Rearick, & Ray, 2000) and in association with motor learning (Rozengurt, Barnea, Uchida, & Levy, 2016).

Aside from theta band, the alpha band is likely to be important in balance and postural control. Cortical alpha during bipedal stance referenced to unipedal stance was lower in athletes than non-athletes in the frontal, parietal, and central area, suggesting alpha activity is reduced in experts (Del Percio et al., 2009; Del Percio et al., 2007). It has been reported that alpha activity is correlated with sensory integration between the thalamus and the cortex (Hülsdünker, Mierau, & Strüder, 2015; Lőrincz, Kékesi, Juhász, Crunelli, & Hughes, 2009). Moreover, alpha plays a crucial role in the engagement and disengagement of the sensory regions (Haegens, Händel, & Jensen, 2011), an ability needed to optimise postural control. The alpha band is also closely related to cognitive function, with the oscillations of EEG alpha reflective of cognitive and memory performance (Klimesch, 1999). Also, alpha has been reported to modulate as a function of attentional demands (Başar, Schürmann, Başar-Eroglu, & Karakaş, 1997; Klimesch, Doppelmayr, Russegger, Pachinger, & Schwaiger, 1998).

An increase in theta and alpha activity has been associated with increasing postural demand and postural sway. Hülsdünker et al. reported that increasing the demands of balance tasks were accompanied by modulation of theta and alpha cortical activity at the frontal midline area in healthy young adults (Hülsdünker,

Mierau, Neeb, Kleinöder, & Strüder, 2015; Hülsdünker, Mierau, & Strüder, 2015). Additionally, Varghese et al. demonstrated a significant synchronisation of alpha activity in healthy young adults during a lean (stable) to release (sudden unstable) cable protocol (Varghese et al., 2014). Tse et al. also reported the difficulty of the balance training tasks was accompanied with an increase in alpha activity (Tse et al., 2013). Furthermore, modulation of EEG within theta and alpha band at the frontal, fronto-central and parietal was reported an indication of voluntary postural sway direction; medial-lateral (ML) sway requires more energy than anterior-posterior (AP) sway and was reflected in the modulation of EEG (Slobounov, Hallett, Cao, & Newell, 2008). Altogether, theta and alpha band at the frontal-central area had a strong correlation with postural sway.

Beta and gamma activity bands have also been associated with postural responses and sensorimotor integration. An increased in cortical beta event related desynchronization (ERD) in the central site suggests a decreased adaptability of postural responses as observed in Parkinson's disease studies when the postural effect was distinct in small perturbation but not to a great perturbation (Smith, Jacobs, & Horak, 2012, 2014). Likewise, task-related uncertainty was also accompanied with a larger beta ERD in the sensorimotor cortex during motor preparation (Tzagarakis, Ince, Leuthold, & Pellizzer, 2010). Thus, larger beta ERD could also relate to the cognitive aspect of the postural response. On the other hand, a burst of gamma activity at the frontal central was accompanied with a backward stepping postural reaction when the balance was in danger (Slobounov et al., 2005), or when subjects visually recognized non-stable postures (Slobounov, Tutwiler, Slobounova, Rearick, & Ray, 2000). At the same time, the increase of gamma band also demonstrated a strong vision effect particularly the processing of sensory feedback (C.-J. Chang, Yang, Yang, & Chern, 2016), such as the involvement of conscious attention when performed the tandem Romberg stance (Varghese, Beyer, Williams, Miyasike-daSilva, & McIlroy, 2015). Altogether, the increase of beta and gamma bands had strong relations with the complexity of the balance tasks (Petrofsky & Khowailed, 2014). Therefore, it is evident that gamma activity in the frontal-central area plays a major role in the integration of somatosensory and visual

information.

2.5.2 Evoked potentials related to postural control

Evoked potentials to a physical perturbation of postural instability are known as perturbation-evoked responses (PERs). PERs have been conducted using mechanical perturbations primarily to the proprioceptive system, to induce postural reactions (Bolton, 2015; Marlin et al., 2014). These responses which peak at the frontal-central site (FCz), demonstrate the detection component (P1) followed by the evaluation component (N1) after the onset of a postural perturbation (Bolton, 2015; Little & Woollacott, 2015; Mierau, Hülsdünker, & Strüder, 2015; Varghese et al., 2015).

The positive peak P1 is the earliest component which occurs before the onset of muscle activation (Dietz et al., 1984; Dietz, Quintern, & Berger, 1985; Duckrow et al., 1999) and is thought to indicate the initial primary sensory perturbation over the primary sensory cortex related to sensing instability (Jacobs & Horak, 2007). However, the P1 in response to a postural perturbation is not consistently reported as distinguishable (Maki & McIlroy, 2007; Quant, Adkin, Staines, & McIlroy, 2004; Quant, Adkin, Staines, Maki, et al., 2004).

On the other hand, a more consistently distinguishable component of PERs is the N1 component, which occurs 100 to 200 ms after the onset of a postural perturbation (Figure 2.1) (Huang, Zhao, & Hwang, 2014; Little & Woollacott, 2015; Marlin et al., 2014; Mierau et al., 2015; Varghese et al., 2014). The N1 component is suggested to reflect the sensory processing of the postural perturbation (Marlin et al., 2014). The N1 aspect of the PER is seen in response to; a lean and release of a cable system during quiet standing

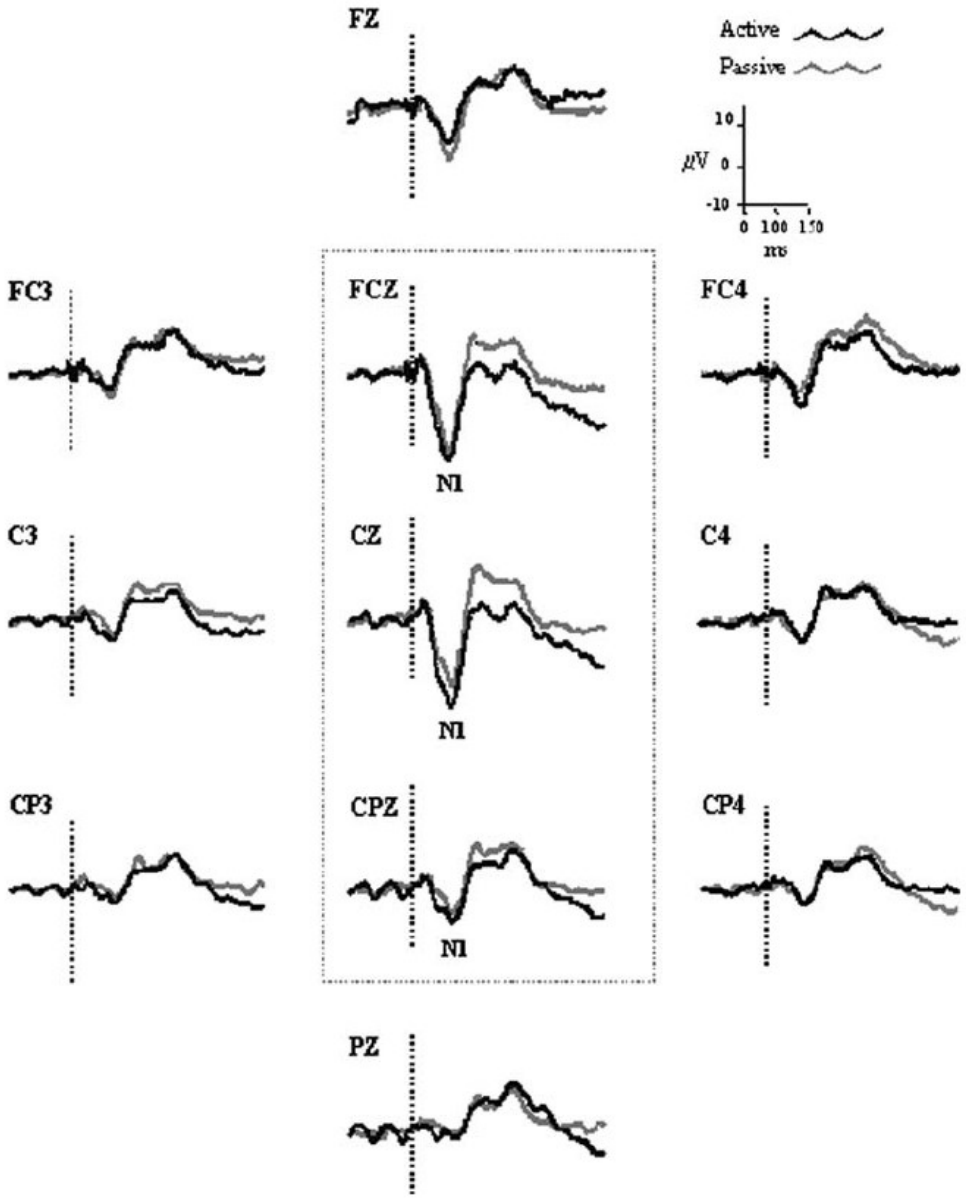

Figure 2.1 Grand average of perturbation-evoked responses recorded from the international 10-20 system (Quant, Adkin, Staines, & McIlroy, 2004).

(Mochizuki, Boe, Marlin, & McIlRoy, 2010; Mochizuki, Sibley, Cheung, & McIlroy, 2009), a single transient horizontal perturbation on the trunk (Adkin et al., 2006), a transient forward tilt of the inverted pendulum during sitting (Quant, Adkin, Staines, & McIlroy, 2004), a horizontal perturbation of the platform during standing (Mierau et al., 2015; Mochizuki et al., 2008), and a chair tilt backwards while sitting

(Mochizuki, Sibley, Cheung, Camilleri, & McIlroy, 2009). A recent study suggests the evoked N1 was responded in each time-locked medial-lateral centre of pressure displacement (Varghese et al., 2015). The N1 component is particularly interesting since the peak amplitude of the N1 component is known to be affected by age (Toledo, Manzano, Barela, & Kohn, 2016), predictability (Adkin et al., 2006; Jacobs & Horak, 2007; Mochizuki et al., 2008), the size of the stimulus (Staines, McIlroy, & Brooke, 2001), postural threat (Adkin, Campbell, Chua, & Carpenter, 2008), the challenge of postural task (Huang et al., 2014; Varghese et al., 2015), and cognitive tasks (Little & Woollacott, 2015; Quant, Adkin, Staines, Maki, et al., 2004). Furthermore, a larger peak amplitude of the N1 component has been suggested to indicate a modified "central postural set" (Little & Woollacott, 2015).

2.6 Autism Spectrum Disorder and Sensory Integration

Autism spectrum disorders (ASD) are a group of neurodevelopmental disorders that are characterised by impairments in social interaction and communication, and restricted or repetitive behaviour. According to the latest Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (American Psychiatric Association, 2013), sensory impairment is now included as part of definitions for ASD. Sensory impairment in ASD can be expressed as hyper or hypo-reactivity to any sensory aspects of the environment such as excessive smelling or touching of objects, adverse response to specific sounds or textures, apparent indifference to pain/temperature, visual fascination with lights or movement (American Psychiatric Association, 2013; Attwood, 2006). Numerous studies have reported at least 90% of children with ASD demonstrated evidence of unusual responses to sensory information (Crane, Goddard, & Pring, 2009; Tomchek & Dunn, 2007). Sensory impairments in ASD are known to be associated with social and behavioural challenges (Nebel et al., 2016). Aside from these challenges, evidence suggests that children with ASD often have impaired motor skills (Bhat, Landa, & Galloway, 2011; Gowen & Hamilton, 2013; Green et al., 2009). Furthermore, all of these difficulties (i.e. sensory, social and behavioural) often persist into adulthood (Bhat

et al., 2011; Schall & McDonough, 2010). As a result, sensory impairment is highly disruptive and limits the performance of activities of daily living, especially for individuals with ASD.

Postural instability can be the result of a sensory impairment and is often accompanied by an increase of postural sway as observed in an individual with ASD. In fact, a sensorimotor control process such as the control of upright stance had demonstrated severely disturbed in individuals with ASD (Doumas, McKenna, & Murphy, 2016; Fournier, Amano, Radonovich, Bleser, & Hass, 2014; Graham et al., 2015). A recent systematic review reported that postural sway in both anterior-posterior (AP) direction and medial-lateral (ML) direction is larger in adults with ASD than TD adults in the standing task (Lim, Partridge, Girdler, & Morris, 2017), especially the total excursion of the AP and ML sway directions (Graham et al., 2015; Stins, Emck, de Vries, Doop, & Beek, 2015). Moreover, larger postural sway was also evident under conditions of visual and somatosensory stimulation in this vulnerable population (Gowen & Hamilton, 2013; Lim et al., 2017). Postural control deficits are suggested to lie in problems of multimodality sensory integration of individuals with ASD (Minshew, Sung, Jones, & Furman, 2004).

Some studies have demonstrated sensorimotor impairments in ASD. The abnormal attentional focusing mechanism was observed in ASD using a simple target-detection task and could link to a dysfunctional top-down feedback from fronto-parietal network to early visual areas (Ronconi, Gori, Ruffino, Molteni, & Facoetti, 2013). Similarly, individuals with ASD had difficulties moving their focus from a central fixation to a peripheral target (Mosconi et al., 2013; Schmitt, Cook, Sweeney, & Mosconi, 2014) or following a moving light (Wilkes, Carson, Patel, Lewis, & White, 2015), demonstrating difficulties in decelerating saccades or delayed in initiating saccades. Also, individuals with ASD took more time to perform pointing task when no vision was available and relied more on proprioception compared to typical control group (Glazebrook, Gonzalez, Hansen, & Elliott, 2009). As a result, these abnormal visual perceptions in ASD could contribute to social impairments observed in ASD.

ERP studies on visual perceptions have been evaluated in individuals with and

without ASD. The high-level ventral and dorsal pathways in people with ASD were often reported distinct from those without ASD (Yamasaki et al., 2014). For example, a negative evoked occipito-temporal component with latency peaked around 170 ms that reflects the neural processing of faces (N170) (Rossion et al., 2000) revealed significantly delayed in response to face (McPartland, Dawson, Webb, Panagiotides, & Carver, 2004) or delayed and smaller amplitude in response to facial expression in adults with ASD (O'Connor, Hamm, & Kirk, 2005). Furthermore, delayed N170 in response to face but larger N170 in response to objects were observed in 3-4 years old children with ASD (Webb, Dawson, Bernier, & Panagiotides, 2006). As a result, these impairments in the visual recognition of faces revealed similar patterns to patients with brain lesions and were suggested damages in the ventral pathway (E. A. Prieto, Caharel, Henson, & Rossion, 2011). On the other hand, the dorsal pathway is concerned with global motion processing, and coherent motion stimulus such as radial optic flow (OF) and horizontal flow (HF) were commonly used to investigate the dorsal pathway (Yamasaki et al., 2014). Motion flow ERP studies suggested both OF and HF stimulus can evoke N170 component but only OF can evoked the parietal P200 component (Tobimatsu et al., 2008; Yamasaki et al., 2011; Yamasaki et al., 2012; Yamasaki et al., 2014). Adults with ASD demonstrated delayed N170 and P200 latencies in response to OF stimuli, but the peak latency of N170 was not affected by HF stimuli, suggesting the higher-level dorsal pathway was impaired in adults with ASD (Yamasaki et al., 2011; Yamasaki et al., 2014). Additionally, flicker contrast which explored the dorsal pathway at lower subcortical along with global motion was investigated in children with ASD (Pellicano & Gibson, 2008); the authors also concluded the higher-level dorsal pathway (global motion) was impaired in children with ASD, but the lower-level remained intact (Pellicano & Gibson, 2008). In summary, individuals with ASD seem to have difficulties with higher-order visual processing but not early stage visual processing.

Aside from visual perception, event related potentials (ERP) studies on auditory perception have also been evaluated on the individual with and without ASD. A simple pure-tone audiogram is often used as the "gold standard" to evaluate the

hearing sensitivity (i.e. lower-level auditory function), whereas the complex tones such as noise are useful to assess higher-level auditory function (Council, 2004; Yamasaki et al., 2014). A negative response over the fronto-central scalp with a peak latency around 100ms (N1) is the most prominent auditory sensory evoked potential that reflects activation of the auditory pathway from the cochlea to the cortex (Stapells, 2002; Woods, 1995). Several studies reported shorter or faster N1 latencies in response to simple pure tone stimuli in children with ASD, suggesting ASD involved in enhanced auditory processing (Ferri et al., 2003; Martineau, Garreau, Barthelemy, & Lelord, 1984; Oades, Stern, Walker, Clark, & Kapoor, 1990; Oades, Walker, Geffen, & Stern, 1988). On the other hand, the auditory mismatch negativity (MMN) has been studied intensively to investigate higher-level auditory processing in response to complex auditory stimuli; the MMN usually peaks negatively around 100 – 250 ms with a maximal response at the temporal and frontal area and occurs after a complex tone appears in a repetitive sequence of sound (i.e. oddball sequence) (Cheour, Leppänen, & Kraus, 2000; Dunn, Gomes, & Gravel, 2008; Javit, Steinschneider, Schroeder, Vaughan, & Arezzo, 1994; Mamashli et al., 2017; Näätänen, Paavilainen, Rinne, & Alho, 2007). However, the findings on MMN in ASD are not consistent (Dunn et al., 2008; Näätänen et al., 2007). For example, the MMN was reported smaller amplitude and delayed in latencies in individuals with ASD (Dunn et al., 2008; Jansson-Verkasalo et al., 2003; Kujala, Lepistö, Nieminen-von Wendt, Näätänen, & Näätänen, 2005; Roberts et al., 2011; Russo, Zecker, Trommer, Chen, & Kraus, 2009) whereas some studies reported larger MMN and shorter latencies in individuals with ASD (Ferri et al., 2003; Kujala et al., 2007). The inconsistencies of the findings could explain the differences in methodology (some experiments are dual tasks), and most importantly, the MMN is affected by attention (Campbell & Davalos, 2015; Müller, Achenbach, Oades, Bender, & Schall, 2002; Näätänen, Paavilainen, Titinen, Jiang, & Alho, 1993). Altogether, the findings suggest the basic auditory perception is intact or superior in individuals with ASD, but the impairments in the high-level auditory function in ASD might attribute to a higher order cortical area.

In general, the ERP studies on visual and auditory perceptions in ASD suggest

atypical neural activity in higher-level cortical processing but not lower-level processing. The findings from the ERP studies substantially support the sensory impairments as core deficits in ASD. Moreover, the temporal resolution of EEG can be used as a neuro marker to understand the mechanism and neural substrates underlying the impairments that define ASD.

Chapter 3

Methodology

3.1 Overview

In this section, the techniques that were used to quantify and assess the physiological mechanisms of postural equilibrium in the thesis are described.

3.2 Force Plates

Centre of pressure (COP) is the most common posturographic measurement in postural control studies. It is the point location of the vertical ground reaction force vector, and it represents an average of all the surface pressures points of an area in contact with the ground (Winter, 1995). In this thesis, the COP data was obtained using AMTI AccuGait portable force platform (Advanced Mechanical Technology Inc., Watertown MA, USA) and recorded using custom made program written in LabView (National Instruments Corporation, Austin TX, USA). The COP data was recorded with a sampling rate of 2000Hz.

The forces and moments recorded by the force platform were used to calculate the COP in the anterior-posterior (AP) and medial-lateral (ML) directions. The COP data was then down sampled to 1000 Hz to match the EEG sampling rate. The AP and ML time series were passed through a fourth-order zero phase Butterworth low-pass filter with a 5Hz frequency cut-off to eliminate high-frequency noise artefacts. Since the absolute positions of the participants standing on the force platform and sitting on the stool were not controlled, the linear trend of AP and ML time series were removed

before analysis (Duarte & Freitas, 2010; T. Prieto, Myklebust, Hoffmann, Lovett, & Myklebust, 1996). Subsequently, the resultant distance (RD) which was the composite measure of both the AP and ML in time series was computed. The AP, ML and RD time series were quantified into the following measures; RD root mean square distance (RMS), RD mean velocity (MV), sway area (AREA), total range (RANGE), total excursion in AP direction ($TOTEX_{AP}$) and ML direction ($TOTEX_{ML}$). All summations were computed from 1 to N, in which N is the number of data points; and T, the period is 5 seconds. The RMS is the root mean square distance value of the RD time series from the mean COP, where

$$RMS = \left(1/N \sum_{n=1}^{N} RD[n]^2\right)^{1/2}$$

The MV is the average velocity of the COP which also defined as the total excursion of the RD time series ($TOTEX_{RD}$) divided by the time elapsed, expressed by

$$MV = TEX/T$$

where

$$TEX = \sum_{n=1}^{N-1} [(AP[n+1] - AP[n])^2 + (ML[n+1] - ML[n])^2]^{1/2}$$

The $AREA$ is the sway area measured by approximately summing the area of the triangles formed by AP and ML time series and the mean COP, it is written as

$$AREA = 1/2T \sum_{n=1}^{N-1} |AP[n+1]ML[n] - AP[n]ML[n+1]|$$

The RANGE is the maximum distance between any two points on the COP path. Lastly, the total excursions are the total length of the COP path in the AP direction ($TOTEX_{AP}$) and ML direction ($TOTEX_{ML}$), and is estimated by the summation of the distances between the consecutive points in their respective time series such as

$$TOTEX_{AP} = \sum_{n=1}^{N-1} |AP[n+1] - AP[n]|$$

$$TOTEX_{ML} = \sum_{n=1}^{N-1} |ML[n+1] - ML[n]|$$

The analysis of the COP signal was also conducted using MATLAB 2015a (Mathworks Inc, Natick MA, USA). Further details of the measurement are described by Prieto et al. (T. Prieto et al., 1996).

3.3 Electroencephalography

Cortical activity related to postural control can be obtained using electroencephalography (EEG). EEG is a technique that monitors the electrical activities of the brain through the placement of electrodes on the scalp (Berger, 1929). Despite the introduction of other neuroimaging devices such as computerized tomography, functional near-infrared spectroscopy, functional magnetic resonance imaging, magnetoencephalography and positron emission tomography, EEG remains popular in both research and clinical settings for the reason that it is a safe, inexpensive, quiet and non-invasive way of monitoring the brain (Evans & Abarbanel, 1999; Luck, 2014). Also, EEG has a very high temporal resolution which can record on the order of milliseconds rather than seconds. The time contingent changes in the electrical field of the brain are captured by the electrodes on the scalp and are known as the EEG signals (Lee, 2014). The following section will discuss the creation of the electrochemical currents that are recorded by the EEG and its measurements.

3.3.1 The neurophysiology of human brains

The brain is the most complex organ in the human body and contains billions of neurons and hundreds of billions of interconnections that form the human neural network. In fact, an average human brain consists of 86 billion neurons with 16 billion them located in the cerebral cortex (Azevedo et al., 2009; Herculano-Houzel, 2009). The interconnections of the brain are maintained by these neurons via an electrochemical process allowing them to transmit signals. The neurons have three basic parts that perform the transmission function: the dendrites, the axon, and the cell body (also referred as the “soma”).

The contact points between two neurons, specifically the dendrites of one neuron

and the axon terminals of the other neurons are known as synapses. The function of the dendrites is to receive messages from the other neurons and transmit the impulses from the synapses to the cell body. The function of the cell body is to provide energy and introduce new proteins to the dendrites, axon and the axon terminals. Finally, the axons take the signals away from the cell body and transmit the information to the end of the axon terminals and onto other neurons.

Action potentials allow electrical signals to travel quickly through the axon to the dendrites via a series of depolarisations and repolarisations in part of the neural membrane along the axon. The release of neurotransmitters is triggered when an action potential arrives at the axon terminal. The potential changes in the membrane of the axon terminals are known as postsynaptic potentials. When the postsynaptic potentials accumulate and reach the threshold of the conduction level of the postsynaptic neuron, electric fields then spread to the scalp. These electric fields are then captured by a neuroimaging device such as electroencephalography (EEG).

3.3.2 EEG Measurements

In this thesis, the EEG signals were collected using a 40 channel Ag/AgCl electrode cap (Neuroscan, El Paso, TX, USA) based on the International 10-20 System (Figure 3.1) (Klem, Lüders, Jasper, & Elger, 1999). Additional electrodes were placed above and below of the left eye, and on the outer canthus of each eye to monitor electrooculogram (EOG) signals (See Appendix A for more details on the electrode locations with and without EOG). Both EEG and EOG signals were measured in a monopolar mode with a sampling rate of 1000 Hz. The recorded raw EEG signals are typically noisy and require processing for further analysis and application. Thus, a series of pre-processing steps such as referencing, bandpass filtering and artefact removal are required to increase the signal-to-noise ratio (Luck, 2014).

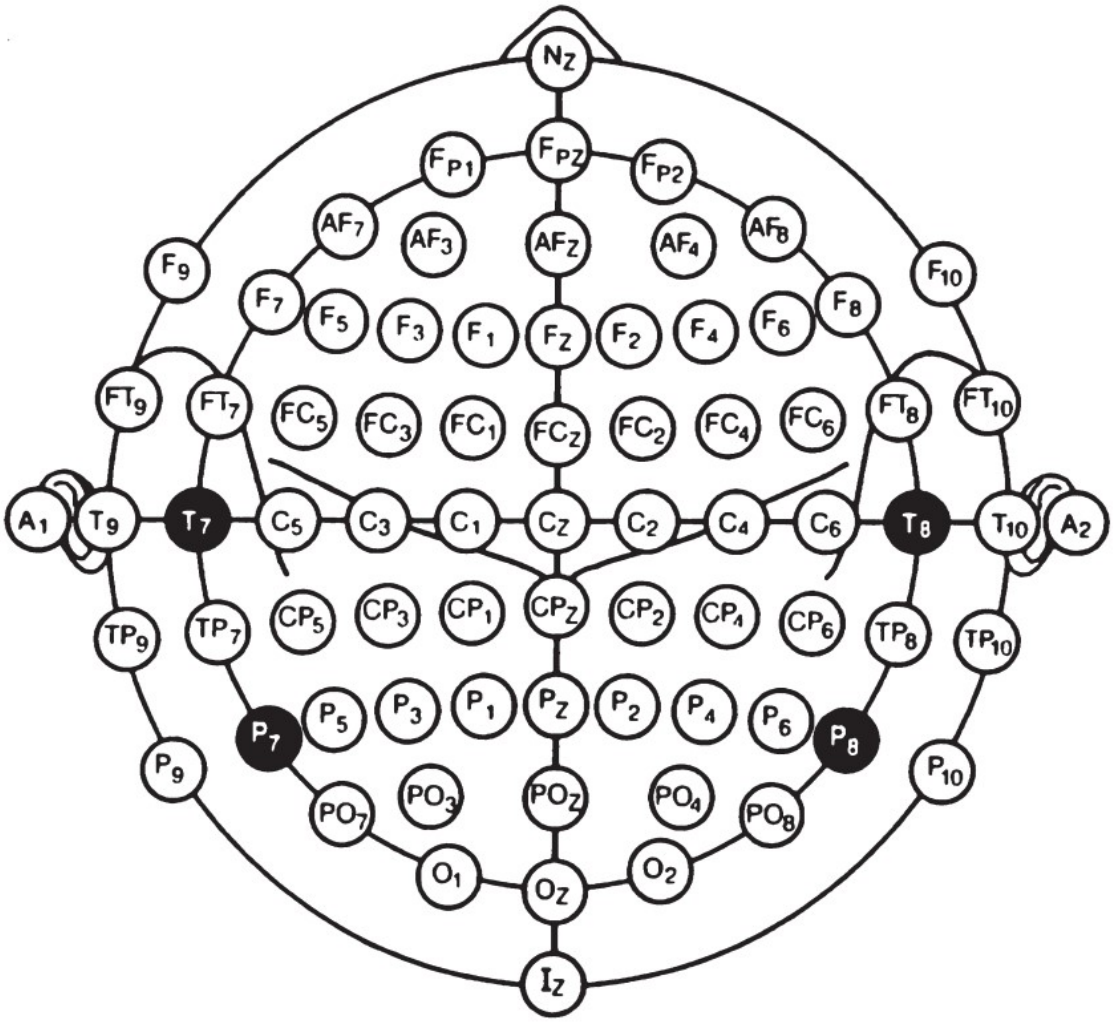

Figure 3.1 The International 10-20 electrode system (Klem et al., 1999).

Figure 3.2 Block diagram of EEG pre-processing techniques

The first stage of the EEG signal pre-processing begins with referencing to reduce the noise signals that appear in all active electrodes and the reference electrodes (Figure 3.2). In this thesis, all channels were referenced to the average of the electrode A1 and A2 located on the left and right mastoid with a forehead ground (GND). The impedance of all channels was maintained below 5kΩ throughout the experiment.

After referencing, noise such as baseline drift and power line interference may still

exist in the EEG signal. Thus, bandpass filtering is applied to suppress these artefacts (Figure 3.2). In this thesis, the EEG signals were bandpass filtered using second order zero phase 0.5 Hz elliptic high-pass filter and eighth order zero phase 40 Hz elliptic low-pass filter. In both low-pass and high-pass filters, the passband ripple of 0.1 dB and the stopband ripple of 70 dB were used.

Strong noise artefacts such as ocular artefacts are the primary source of contamination in EEG signals. Therefore, independent component analysis (ICA), a widely adopted EEG signal processing technique, was used to remove ocular artefacts and extract source signals (Makeig, Jung, Bell, Ghahremani, & Sejnowski, 1997; Rogasch et al., 2014). ICA is a generative learning technique that decomposes noisy mixed signals into a set of source signals (Hyvärinen, Karhunen, & Oja, 2004). However, the extractions in ICA require manual or blind selection. In this thesis, independent component analysis with reference (ICA-R) was performed on the EEG signals to remove ocular artefacts (Lee, Tan, Falkmer, & Leung, 2016). ICA-R is a technique that extracts the signal-of-interest directly through the guidance of a reference signal (i.e. EOG reference). ICA-R has demonstrated faster computation, more accurate and more consistent extraction performance compared to a traditional ICA (Lee et al., 2016).

After the EEG preprocessing steps are completed, the EEG signals are considered adequate for further analysis (Figure 3.2). There are two ways to analyse the ocular artefact free EEG signals: i) EEG rhythm and ii) evoked potential (EP).

The EEG rhythm is the time-frequency analysis of the EEG signals and it is characterized by unique frequency bands such as delta (0.5 – 4 Hz), theta (4 – 8 Hz), alpha (8 – 13 Hz), beta (13 – 30 Hz) and gamma (> 31 Hz) (Niedermeyer, 2005). It is also known as spontaneous EEG as it lacks an experimental stimulus or event of interest. In this thesis, the frequency bands of interest for each channel were computed based on the power spectrum estimated from the Welch method with no overlapping. These measurements reflect the total power of certain frequency range, and are commonly used in investigating sleep, Alzheimer's disease and epilepsy disorder

(Blinowska & Durka, 2006; Lizio et al., 2011; Sanei & Chambers, 2013).

In contrast, an evoked potential (EP) is a small voltage brain response time-locked to a specific repeated stimulus in milliseconds to hundreds of milliseconds (Zani, 2013). Unlike the event related potential (ERP) which reflects the higher order of brain processing related to mental activity such as memory, expectation or, attention, EP is simply evoked by an external physical stimulus (Zani, 2013). Nevertheless, there are additional preprocessing steps such as epoching, baseline correction and artefact detection before EP or ERP analysis (Figure 3.4). Firstly, the EEG signals are

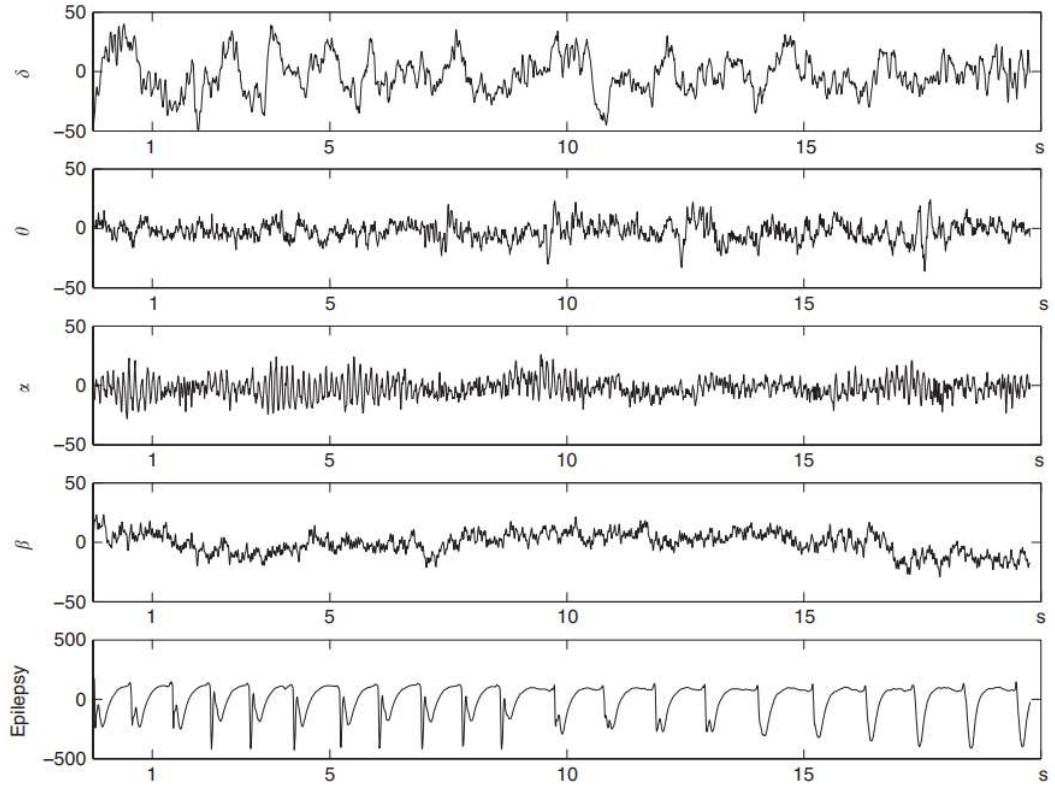

Figure 3.3 Characteristics of EEG rhythms, from the top: δ delta (0.5 – 4 Hz), θ theta (4 – 8 Hz), α alpha (8 – 13 Hz), β beta (13 – 30 Hz) and the lower trace-EEG during an epileptic seizure (Blinowska & Durka, 2006).

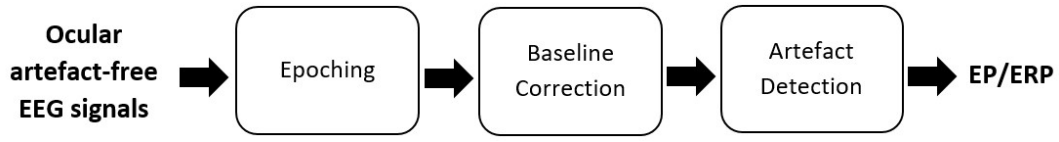

Figure 3.4 Block diagram of ERP additional pre-processing techniques

segmented into epochs (or trials) based on the onset of the stimulus (i.e. epoching). During or after the epoching step, a baseline correction is applied by subtracting the mean voltage of the epoch individually before the onset of the stimulus (e.g., 200ms to 0ms) for each epoch, so that the epoch reflects the voltage relative to the average pre-stimulus voltage. Lastly, even though ocular artefact removal was already performed in the pre-processing step of the EEG signals, the EEG could contain other artefacts such as muscle, mechanical or electrodes artefacts. Thus, artefact detection is performed on each epoch so that the epochs marked for rejection are not included in the grand average of EP/ERP. Artefact detection is a signal detection problem by which the artefact is compared with a threshold to make an inclusion or exclusion decision (Luck, 2014). The artefact detection was achieved by finding the difference between the largest and smallest values (also known as maximum peak-to-peak voltage) within a moving window across the epoch where the epoch would be rejected if the voltage exceeded a pre-set voltage threshold. The voltage threshold (100uV), moving window width (200 ms) and window step (50 ms) were consistent with all trials. Epochs were rejected if artefacts were found in this phase. Moreover, the epochs were visually inspected to remove any suspicious contaminated artefacts. The artefact free epochs for each participant were retained to minimise selection from which an average of EP/ERP per participant and a grand average of EP/ERP across all participants were constructed in order to increase the signal-to-noise ratio of the recorded EP/ERP (Duncan et al., 2009; Luck, 2014).

The analysis of the EEG signals was performed using MATLAB 2015a (Mathworks Inc, Natick MA, USA), specifically the EEGLAB toolbox (Delorme & Makeig, 2004) to pre-process EEG signals with referencing, bandpass filtering and artefact removal. Also, ERPLAB (Lopez-Calderon & Luck, 2014) was tightly integrated with EEGLAB for evoked potential (EP) processing, visualisation and analysis.

Chapter 4

Postural and cortical responses following visual occlusion in standing and sitting tasks in young typically developed adults

This chapter is covered by the following publication:

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Exp Brain Res
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RESEARCH ARTICLE

Postural and cortical responses following visual occlusion in standing and sitting tasks

Kwang Leng Goh[1] · Susan Morris[2] · Wee Lih Lee[2] · Alexander Ring[3] · Tele Tan[1]

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Abstract Perturbation-evoked responses (PERs) to a physical perturbation of postural stability have been detected using electroencephalography (EEG). Components of these responses are hypothesized to demonstrate the detection (P1) and evaluation (N1) of postural instability. Despite the important contribution of the visual system to postural control, PERs to a visual perturbation of posture have yet to be reported. Ten healthy young adults were exposed to unpredictable visual occlusion mediated through liquid crystal glasses under two conditions of postural demand: quiet standing and quiet sitting. The participants' PERs and postural responses were recorded and differences between conditions assessed using Wilcoxon signed-rank tests. In response to unpredictable visual occlusion, both P1 and N1 components of the PER were observed in both postural conditions. The amplitude of the P1 response remained consistent between postural conditions ($Z = -0.5606$, $p = 0.5751$), whereas N1 amplitude and postural responses were significantly smaller in the sitting condition ($Z = -2.2934$, $p = 0.0218$). This is the first study to demonstrate cortical responses to visual perturbation of posture. The responses to postural perturbation by sudden visual occlusion are similar in nature to that seen in relation to a physical perturbation. In addition, the amplitude of the N1 response is not only consistent with the relative magnitude of the perturbation, but also the underlying postural set, with a larger N1 seen in standing relative to sitting. The study informs the relative importance of vision to postural stability, postural set and provides a protocol to objectively assess sensory-based postural disorders.

Keywords EEG · Evoked potentials · Postural control · Proprioception · Vision · Sensory integration

✉ Kwang Leng Goh
kwangleng.goh@postgrad.curtin.edu.au

[1] Faculty of Science and Engineering, Curtin University, Bentley, WA, Australia

[2] Faculty of Health Sciences, Curtin University, Bentley, WA, Australia

[3] Otolaryngology, Head and Neck Surgery, University of Western Australia, Perth, WA, Australia

Introduction

Human postural control is a complex skill which is reliant on the integration of sensory information from the visual, vestibular, and somatosensory systems. The importance of these sensory systems in postural control is well documented, and stimulation of these individual systems induces alterations in body sway (Peterka 2002). While it is known that postural control is altered after damage to the cortical area of the brain (Rapport et al. 1993; Hauer et al. 2003), the underlying mechanism is unknown. A number of research studies have been conducted to study the involvement of cortical activity in postural control (Jacobs and Horak 2007; Maki and McIlroy 2007). Moreover, recent evidence suggests even for typical adults without cortical damage, and changes in cognitive function and attention alter postural stability (Remaud et al. 2013; Jehu et al. 2015).

Electroencephalography (EEG) has been widely used to measure perturbation-evoked responses (PER) in the cortex during postural perturbations, because EEG is non-invasive and has high temporal resolution (Dietz et al. 1984, 1985; Duckrow et al. 1999; Staines et al. 2001; Quant et al. 2004a, b; Adkin et al. 2006, 2008; Jacobs and Horak 2007; Maki and McIlroy 2007; Mochizuki et al. 2008, 2009, 2010;

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Marlin et al. 2014; Varghese et al. 2014; Bolton 2015; Little and Woollacott 2015). There are two main components of the PER that occur in relation to postural perturbations. The earliest component is the positive peak P1 component which occurs before the onset of muscle activation (Dietz et al. 1984, 1985; Duckrow et al. 1999) and is thought to indicate the initial sensory processing of the primary sensory cortex related to sensing instability (Jacobs and Horak 2007). P1 in response to a postural perturbation is not consistently reported as distinguishable (Quant et al. 2004a, b; Maki and McIlroy 2007). A more consistently distinguishable component of PER is the N1 component, which occurs 100–200 ms after the onset of a postural perturbation and peaks at the frontal–central site (FCz) (Adkin et al. 2008; Mochizuki et al. 2009, 2010; Marlin et al. 2014; Varghese et al. 2014). The N1 component is suggested to reflect the sensory evaluation of the postural perturbation (Marlin et al. 2014). The N1 aspect of the PER is seen in response to a lean and release of a cable system during quiet standing (Mochizuki et al. 2010), a single transient horizontal perturbation on the trunk (Adkin et al. 2006), a transient forward tilt of the inverted pendulum during sitting (Quant et al. 2004a), and a chair tilt backwards while sitting (Mochizuki et al. 2009). The evaluation role of the N1 is supported by the variation in N1 amplitude with stimulation of proprioceptors using vibration (Staines et al. 2001), behavioural and cognitive processes (Quant et al. 2004b; Little and Woollacott 2015), and with predictability of the stimulus (Adkin et al. 2006; Jacobs and Horak 2007; Mochizuki et al. 2008).

Predominately research on PERs has been conducted using mechanical perturbations involving primarily the proprioceptive system to induce postural reactions. Limited studies have been conducted to study PERs when perturbing vision. A few studies have attempted to evaluate the contributions of visual function to postural control by introducing eyes-open and eyes-closed conditions while perturbing proprioceptive systems during upright stance (Staines et al. 2001; Del Percio et al. 2007; Petrofsky et al. 2012; Tse et al. 2013). However, findings have been inconsistent and inconclusive. One study found no difference in cortical responses in the eyes-open versus eyes-closed condition (Staines et al. 2001). As the visual system contributes less (10%) than the proprioceptive system (70%) in maintaining balance during upright stance (Peterka 2002; Ben-Itzhak et al. 2011), it might be expected that cortical responses from the proprioceptive system to maintain balance overwhelm the cortical responses from the visual system. Changes in the EEG frequency of cortical activity following perturbation related to a change in the visual condition have been reported (Del Percio et al. 2007; Petrofsky et al. 2012), specifically an increase in the alpha frequency band at the centro-parietal site (Del Percio et al. 2007) or an increase in the beta and sigma frequency bands at the parietal area during the eyes-open condition (Petrofsky et al. 2012; Tse et al. 2013). Even though the EEG frequency is widely used to determine the brain areas engaged in a particular task, PERs are useful to evaluate brain function in response to specific sensory or motor events.

Vision is important to postural coordination. Alterations in vision, such as closing the eyes, increases postural sway (Kuo et al. 1998). Furthermore, moving visual environments can induce postural changes and motion sickness in healthy adults (Redfern et al. 2001). The aim of this study is to investigate the contribution of the visual system at the cortical level in postural control. It is hypothesized that the PER components P1 and N1 will be evident in response to visual occlusion. Furthermore, it is hypothesized that while the latencies of P1 and N1 will remain constant, the cortical response to visual occlusion will be attenuated in less demanding postural tasks; specifically, the N1 component will be smaller in sitting compared with standing. Finally, it is hypothesized that postural sway measures will be smaller in sitting condition compared to the standing condition.

Materials and methods

Participants

Ten healthy young adults (2 females and 8 males, 26.3 ± 7.0 years, 170.6 ± 7.4 cm, 66.2 ± 12.3 kg) participated in this study. Exclusion criteria included any self-reported neurological or musculoskeletal deficits that may have influenced postural control. This study was approved by Curtin University Human Research Ethics Committee (Approval No: ENG-71-14) with written informed consent from all subjects. All subjects gave written informed consent in accordance with the Declaration of Helsinki.

Data acquisition

Electroencephalography Electroencephalography (EEG) signals were recorded using a 40 channel Ag/AgCl electrode cap (Neuroscan, El Paso, TX, USA) based on the International 10-20 System. All channels were referenced to the average of the electrode A1 and A2 located over the left and right mastoid with a forehead ground (GND). The impedance of all channels was maintained below 5 kΩ throughout the experiment. Additional electrodes were placed above and below of the left eye, and on the outer canthus of each eye to monitor electrooculogram (EOG) signals. Both EEG and EOG signals were measured in a monopolar mode with a sampling rate of 1000 Hz.

Centre of pressure Centre of pressure (COP) data was obtained using AMTI AccuGait portable force platform

(Advanced Mechanical Technology Inc., Watertown MA, USA) and recorded using custom made program written in LabView (National Instruments Corporation, Austin TX, USA). The COP data was recorded with a sampling rate of 2000 Hz.

Procedure

All participants were barefoot and either stood on the force platform or sat on a customized stool with the force platform underneath (Fig. 1). The customized stool had no backrest, was 700 mm in height, 500 mm wide, and 400 mm in length and was attached firmly to the force platform. This stool also had an adjustable footrest to ensure knees and hips were flexed at 90° and the height of vision during sitting was approximately equivalent to the height of vision during standing. The force platform was 1.5 m away from the height-adjustable computer screen. In the standing task, participants were informed to stand with their feet slightly apart in their comfortable position with hands relaxed alongside their body. In the sitting task, participants were asked to sit centred on the stool with their hands at the side of the stool. The height of the computer screen was adjusted to the height of the participants eyes. Participants were positioned by the same investigator for all trials using standardized instructions.

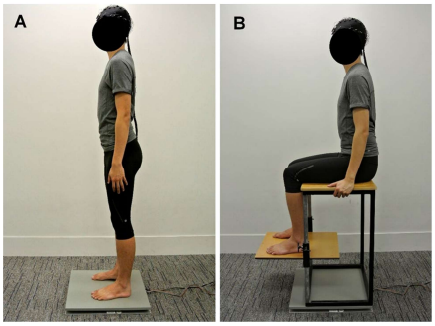

Fig. 1 Experimental setup for testing: **a** standing and **b** sitting

During the standing or sitting task, participants wore liquid crystal spectacles (PLATO, Translucent Technologies Inc, Toronto ON, Canada) and focused on the computer screen in front of them. The PLATO spectacles were able to shut and open rapidly and vice versa using liquid crystal cells. In the open state, participants were able to look through the lenses but in the closed state, the lenses were shut and participants were not able to perceive visual information (Milgram 1987).

The stimuli were presented using EPRIME (Psychology Software Tools Inc., Sharpsburg PA, USA) which demonstrates precise millisecond timing to ensure the accuracy of the data. The cycle of the task presentation for one trial is shown in Fig. 2. Participants were asked to look at the computer screen and mentally solve individual mathematics questions in their head, i.e., silent count. A random number of questions (n = 1–5) were displayed for 1–2 s followed by an "X" in the middle of the screen for 3 s, followed up by a 50% chance of PLATO spectacles being shut for 5 s (visual occlusion condition) or remaining the same

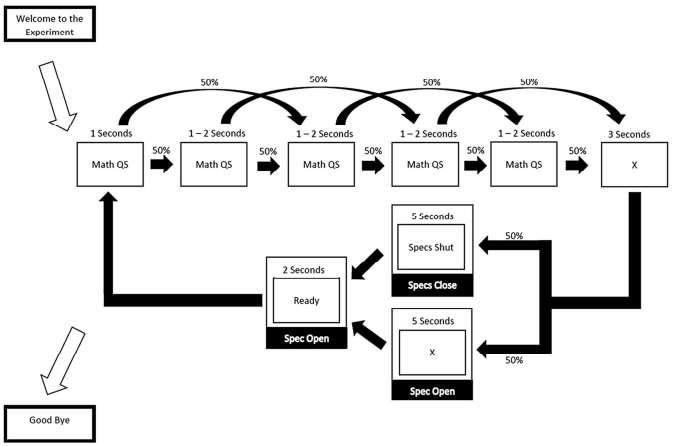

Fig. 2 Presentation of the experiment for a single trial

"X" for 5 s (visual transparent condition). At the end of this sequence, the PLATO spectacles would open if closed or remain open. The screen would then display "Ready?" for 2 s and followed up by the mathematics questions again. The 50% chance for visual occlusion and visual transparent conditions were introduced to avoid adaptation and habituation. The silent count for the mathematics questions was presented. Silent count is widely used in ERP studies as a distractor task Light et al. (2010) to engage participants' attention away from the main task (i.e., visual occlusion condition). The advantage of a silent count is to avoid contamination with motor-associated cortical activity (Kayser et al. 2010).

There were a total of eight blocks of trials four standing blocks and four sitting blocks. Blocks were presented in a consistent sequential order (i.e., stand, sit, stand, sit, etc) and the duration of each block ranged from 5 to 6 min. A resting period of 30–60 s between blocks was provided to minimise fatigue. Each block consisted of 20 trials. Additional blocks were conducted if there was a suspicion of noisy signals due to contamination of eye movements and blink artifacts or muscle movements. At least a total of 30 visual occlusion trials were collected from each participant.

Data processing

Analysis of the EEG signals was performed using MATLAB 2015a (Mathworks Inc, Natick MA, USA), specifically the EEGLAB toolbox (Delorme and Makeig 2004) to pre-process EEG signals with signal filtering and artifact removal. In addition, ERPLAB (Lopez-Calderon and Luck 2014) was tightly integrated with EEGLAB for event-related potential (ERP) processing, visualization, and analysis. The EEG signals were initially bandpass filtered using the second-order zero phase 0.5 Hz elliptic high-pass filter and eighth-order zero phase 40 Hz elliptic low-pass filter. Following this independent component analysis (ICA) was performed on the EEG signals to remove ocular, muscular, and line noise artifacts. The next step was to divide the ICA-pruned free continuous EEG signals into a set of fixed-length epochs using ERPLAB, and the epochs were time-locked to the visual occlusion condition in both the standing and sitting tasks. The range of the duration of epochs was between −200 and 1000 ms relative to visual occlusion stimulus with the pre-stimulus period of −200 to 0 ms as baseline correction. Artifact detection was performed on the epoched data again to identify artifacts that were not removed by ICA. This was achieved by finding the difference between the largest and smallest values (also known as maximum peak-to-peak voltage) within a moving window across the epoch, where the epoch would be rejected if the voltage exceeded a pre-set voltage threshold. The voltage threshold (100 μV), moving window width (200 ms), and window step (50 ms) were consistent for all trials. Epochs were rejected if artifacts were found in this phase. Moreover, the epochs were visually inspected to remove any suspicious contaminated artifacts. The first 30 artifact free epochs for each participant were retained to minimise selection from which an average of PERs per participant and a grand average of PERs across all participants was constructed to increase the signal-to-noise ratio of the recorded PER. The peak amplitude and latency of the cortical potential of P1 (50–100 ms) and N1 (100–200 ms) relative to the onset of the stimuli were measured for each participant to identify the differences in PER between the standing and sitting tasks. Analysis of the EEG measures was performed on FCz, because this was the site, where the negativity is maximal (Marlin et al. 2014; Varghese et al. 2014).

The forces and moments recorded by the force platform were used to calculate the centre of pressure (COP) in the anterior–posterior (AP) and medial–lateral (ML) directions. The COP data were down sampled to 1000 Hz to match the EEG sampling rate. The AP and ML time series were passed through a fourth-order zero phase Butterworth low-pass filter with a 5 Hz frequency cutoff to eliminate high-frequency noise artifacts. Since the absolute positions of the participants standing on the force platform and sitting on the stool were not controlled, the linear trend of AP and ML time series was removed before analysis. Subsequently, the resultant distance (RD) which was the composite measure of both the AP and ML in time series was computed. The AP, ML, and RD time series were quantified into the following measures: RD root mean square distance (RMS), RD mean velocity (MV), sway area (AREA), and total range (RANGE) as they have been reported to be significantly different between standing and sitting (Serra-AÑó et al. 2015).

All summations were computed from 1 to N, in which N, the number of data points is 5000; and T, the period of the time is 5 s. The RMS is the root mean square distance value of the RD time series from the mean COP, where

$$\mathrm{RMS} = \left(1/N \sum_{n=1}^{N} \mathrm{RD}[n]^2\right)^{1/2}. \tag{1}$$

The MV is the average velocity of the COP which also defined as the total excursion (TEX) divided by the time elapsed, expressed by

$$\mathrm{MV} = \mathrm{TEX}/T \tag{2}$$

where

$$\mathrm{TEX} = \sum_{n=1}^{N-1} [(\mathrm{AP}[n+1] - \mathrm{AP}[n])^2 + (\mathrm{ML}[n+1] - \mathrm{ML}[n])^2]^{1/2} \tag{3}$$

The AREA is the sway area measured by approximately summing the area of the triangles formed by AP and ML time series and the mean COP, it is written as

$$\text{AREA} = 1/2T \sum_{n=1}^{N-1} |\text{AP}[n+1]\text{ML}[n] - \text{AP}[n]\text{ML}[n+1]|. \quad (4)$$

The RANGE is the maximum distance between any two points on the COP path. Further details of the measurement are described by Prieto et al. (1996) and Duarte and Freitas (2010). The COP measurements were reported 5000 ms after the onset of the visual occlusion condition. Only COP trials matching the EEG trials were retained for data analysis to be consistent with the EEG data analysis. The analysis of COP signals was conducted using MATLAB 2015a (Mathworks Inc, Natick MA, USA). The measures from both EEG and COP were derived from averaging the raw time series data.

Statistical analysis

Statistical analysis was performed on data from the visual occlusion condition only. Data for each participant were represented by one averaged signal (EEG and COP) for each condition. Due to the skewed nature of the distributions, Wilcoxon signed-rank tests were performed to test the effect of postural condition (standing or sitting) on the dependent variables for EEG (the amplitude and latency of P1 and N1) and COP measures (RMS, MV, RANGE, and AREA). Post hoc analysis was performed using Bonferroni test. Median and percentiles (25th and 75th) were also reported. A difference of $p \leq 0.05$ (two-tailed comparison) was considered significant. These statistical analyses were performed using MATLAB 2015a Statistics and Machine Learning toolbox (Mathworks Inc, Natick MA, USA).

Results

P1 and N1 amplitudes were maximal at the frontal recording sites (Fig. 3). The peak amplitude of the P1 component occurred between 50 and 100 ms postvisual occlusion, while the peak amplitude of the N1 component occurred between 100 and 150 ms postvisual occlusion (Fig. 3).

The EEG responses to visual occlusion were consistent across participants and resulted in a positive potential (P1) followed immediately by a negative potential (N1) at FCz in both standing and sitting (Fig. 4). Data for the FCz site only are presented here. There was no significant difference in the P1 peak latency ($Z = 1.176$, $p = 0.2396$) and peak amplitude ($Z = -0.5606$, $p = 0.5751$) between the standing and sitting tasks (Table 1). There was no significant

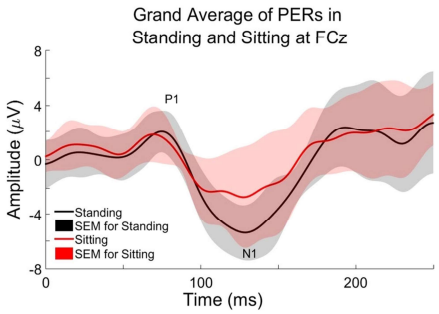

Fig. 3 Grand average of the EEG topographic maps of P1 and N1 based on the peak latencies for each subject. The power unit for the *scale bars* is μV and subjected to a $\log_{10}$ transform to normalise the distribution. **a** Standing P1. **b** Sitting P1. **c** Standing N1. **d** Sitting N1

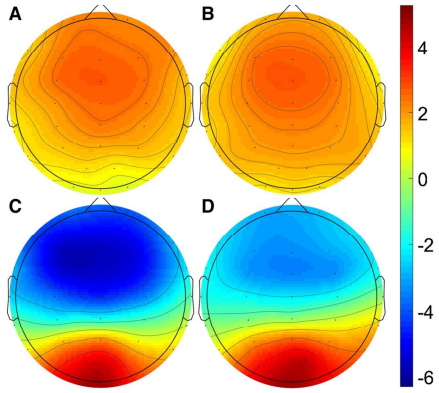

Fig. 4 Grand average of PERs in standing and sitting at frontal–central site. *SEM stands for the standard error of the mean

difference in the N1 peak latency ($Z = 1.0759$, $p = 0.2820$) between the standing and sitting tasks (Table 1). However, there was a significant difference in N1 peak amplitude with a greater amplitude in standing compared with sitting ($Z = -2.2934$, $p = 0.0218$) (Table 1).

Visual occlusion resulted in a backward movement of the COP in standing but not in sitting (Fig. 5). The median and percentiles (25th and 75th) of the COP measurements (RMS, MV, RANGE, and AREA) for standing and sitting are shown in Table 2. The effect of visual occlusion on the COP position was statistically larger in standing compared with sitting. Despite significant differences in RMS ($Z = 2.7011$, $p = 0.0069$

Table 1 Median and percentiles (25th and 75th) of peak latency and peak amplitude of P1 and N1 at FCz site in the standing and sitting tasks

		Median (percentiles)[a]		z^b	p^b
		Standing	Sitting		
Peak latency (ms)	P1	74 (67, 79)	68.5 (65.75, 74.75)	1.1760	0.2396
	N1	130.5 (121.75, 137.5)	128 (114.25, 132.5)	−0.5606	0.5751
Peak amplitude (μV)	P1	2.543 (1.3735, 3.9623)	2.4875 (1.5438, 3.5968)	−0.4590	0.6462
	N1	−6.4085 (−7.41, −4.8963)	−2.4315 (−6.7988, −1.9225)	−2.2934	0.0218

Wilcoxon signed-rank test z score and p value were measured between standing and sitting tasks

[a]Percentiles provided are median (25th and 75th)

[b]Z score and p value for Wilcoxon signed-rank test

Table 2 Median and percentiles (25th and 75th) of root means square (RMS), mean velocity (MV), total range (RANGE), and sway area (AREA) for standing and sitting tasks

	Median (percentiles)[a]		z^b	p^b
	Standing	Sitting		
RMS	3.6876 (1.8986, 4.7761)	0.8941 (0.8142, 1.1294)	2.7011	0.0069
MV	4.6193 (3.9767, 5.245)	4.2058 (3.0901, 5.163)	0.8644	0.3863
RANGE	11.4572 (5.5737, 15.9401)	4.0407 (3.5303, 4.9456)	2.7011	0.0069
AREA	11.1556 (5.5897, 17.57)	3.9516 (2.6903, 6.2211)	2.4973	0.0125

Wilcoxon signed-rank test z score and p value were measured between standing and sitting tasks

[a]Percentiles provided are median (25th and 75th)

[b]Z score and p value for Wilcoxon signed-rank test

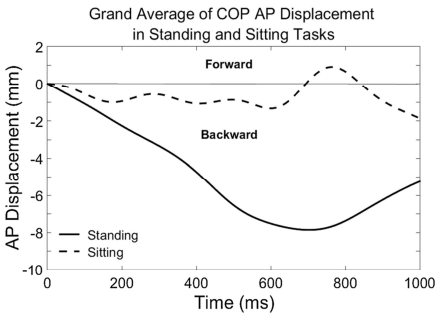

Fig. 5 Grand mean of anterior–posterior displacement of COP (mm) in standing and sitting plotted against time (ms) from the onset of visual occlusion. Zero displacement is the starting point of the normalized absolute position

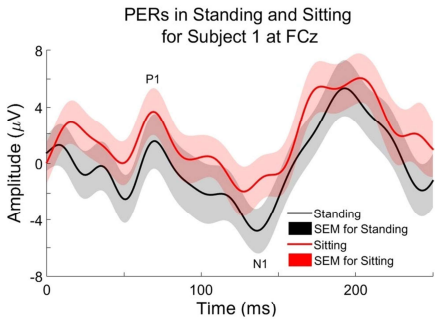

Fig. 6 PERs in standing and sitting conditions at the frontal–central region for Subject 1. *SEM stands for the standard error of the mean

), RANGE ($Z = 2.7011$, $p = 0.0069$), and AREA ($Z = 2.4973$, $p = 0.0125$), there was no significant difference between the two tasks for MV ($Z = 0.8644$, $p = 0.3863$). Examples of PERs and AP displacement of the COP from Subject 1 in standing and sitting condition are shown in Figs. 6 and 7.

Discussion

This study is the first to compare PERs to visual occlusion under varying conditions of postural demand. The EEG responses to sudden and unexpected visual occlusion in both standing and sitting resulted in a positive potential (P1) at around 60–80 ms followed by a negative potential (N1) at around 100–150 ms latency at the fronto-central (FCz) electrode site (Fig. 4). There were no significant differences in the latencies of each of these components between the two tasks; however, there was a significant

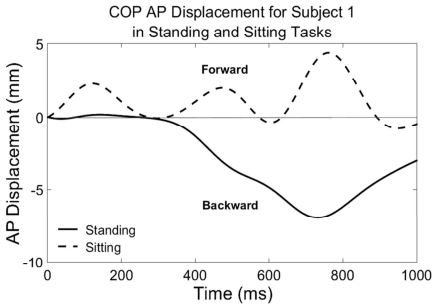

Fig. 7 Anterior–posterior displacement of COP (mm) in standing and sitting conditions plotted against time (ms) from the onset of visual occlusion for Subject 1. Zero displacement is the starting point of the normalized absolute position

increase in the amplitude of N1 (not P1) in standing compared with sitting following visual occlusion (Table 1). A backward movement in COP (8 mm over 700 ms) was observed in standing but not in sitting (Fig. 5), resulting in significant differences in RMS, RANGE, and AREA in response to visual occlusion. However, MV was similar in standing and sitting postvisual occlusion (Table 2).

The maximal P1 and N1 PER components at the FCz site observed in both standing and sitting were consistent with the previous research on proprioceptive perturbations to posture (Marlin et al. 2014; Bolton 2015). This P1 response is considered to be a somatosensory evoked potential (SEP) due to its occurrence before the onset of the muscle activation (Dietz et al. 1984; Quant et al. 2004a). Although P1 has been suggested to represent the primary sensory perturbation that senses postural instability, some perturbation studies reported that the P1 component was often absent (Quant et al. 2004a, b; Adkin et al. 2006; Maki and McIlroy 2007; Toledo et al. 2016) which is surprising. In our study, all participants demonstrated a consistent and distinguishable P1 response 60–80 ms after the onset of the visual occlusion. The latency of the P1 component was comparable with that seen when evoked by a perturbation of stance in a similar population to our study, i.e., young adults (Duckrow et al. 1999), where the latency of P1 was 60 ± 5 ms (mean $\pm$ standard deviation). Interestingly, older participants (≥70 years) were reported to have a delay in P1 response of up to 22 ms compared with young participants (Duckrow et al. 1999), suggesting that a delay in processing a sense of instability may occur with age. The topographic map for P1 in this study was also in line with other literature which suggested that P1 response was generated at the primary sensory cortex (Dietz et al. 1984; Duckrow et al. 1999). Our findings are consistent with the hypothesis that P1 reflects a sense of instability developed in the primary sensory cortex; however, the postural consequences (i.e., postural demand) of the instability are not considered at this level of processing.

The peak latency of N1 was consistent with the previous studies reporting that it occurred approximately 100–150 ms after the onset of visual occlusion (Adkin et al. 2008; Varghese et al. 2014). However, interestingly, the effect of a mechanical perturbation on the proprioceptive system has been reported to produce a larger N1 peak amplitude compared with that observed with visual perturbation in the current study (standing: $-6.1517 \pm 1.3853\,\mu V$); sitting: $-4.0393 \pm 2.8768\,\mu V$) (Quant et al. 2004a; Adkin et al. 2006; Mochizuki et al. 2009, 2010). In standing situations, Mochizuki et al. (2010) induced an average N1 peak amplitude (±SE) of $-30.1 \pm 17.7\,\mu V$ at the central (Cz) electrode site by discharging a load-release cable which required subject to step in response, and $-11.4 \pm 7.1\,\mu V$ by discharging a load-release cable, but there was an additional cable to hold the subject which does not require subject to step but to lean forwards slightly. Adkin and colleagues reported an average N1 peak amplitude of $-22.1 \pm 5.0\,\mu V$ in predictable and $-28.7 \pm 6.7\,\mu V$ in unpredictable conditions at the FCz site when single transient horizontal perturbations were performed to the trunk of the subject during upright stance with eyes closed (Adkin et al. 2006). In sitting, Quant and colleagues performed an experiment which required subjects seated with their eyes-closed and were instructed to balance an inverted pendulum using their ankle musculature (Quant et al. 2004a). They observed that the grand average of N1 peak responses at FCz was $-27.0 \pm 15.8\,\mu V$ when subjects were instructed to return the inverted pendulum to the original stable position using their ankle musculature and $-20.4 \pm 16.0\,\mu V$ when the subjects were instructed not to return the inverted pendulum to original stable position. Mochizuki and colleagues compared two conditions in which an electromagnetically released heavy load initiated perturbations in posterior direction in standing and a rapid chair tilted backward using the release of magnet (Mochizuki et al. 2009). They reported large N1 amplitude in standing ($39.08 \pm 4.51\,\mu V$) and sitting ($37.16 \pm 6.99\,\mu V$). In terms of potential consequences of a perturbation, disturbing posture by a physical push or pull is likely to be more destabilising than simply removing vision. This supports the study from Staines and colleagues who measured subjects' PERs to linear translations in backward direction with eyes open and eyes closed; they concluded no differences in cortical response regardless of visual stimulations (Staines et al. 2001).

Contrary to the previous findings of no differences in N1 amplitude for standing and sitting (Mochizuki et al. 2009), the peak amplitude of N1 in our study varied with postural demand. Perhaps, the difference in findings relates to the

type of perturbation as in our study, the proprioceptive and vestibular effects were minimised and only vision was perturbed. Since the amplitude of N1 is influenced by the size of a perturbation, therefore, it is understandable that postural demand also influences the amplitude of N1 response. Where postural demand is low, i.e., in sitting, the signal is low. Where postural demand is higher, i.e., in standing, the signal is higher. It is possible that the increase of N1 amplitude in standing over sitting in response to visual occlusion was associated with a change in attention (Quant et al. 2004b), since it is known that standing requires more attention than sitting (Vuillerme and Nafati 2007; Roerdink et al. 2011). Reducing the postural requirements of the body by lowering the centre of mass and increasing the base of support (standing to sitting) will also result in lesser consequences for posture. These findings support the conclusions drawn by Mochizuki and colleagues that the N1 is scaled to the expected consequence of postural instability (Mochizuki et al. 2010) and adds an effect of postural set (Horak et al. 1989; Beckley et al. 1991).

The average topographic representations for the N1 response revealed a maximal peak amplitude at the FCz site. It was initially hypothesized that the N1 response was generated in anterior cingulate cortex (ACC) in the frontocentral region (Adkin et al. 2006) for the reason that the amplitude of the N1 response was strongly determined by the predictability (Adkin et al. 2006; Jacobs and Horak 2007; Mochizuki et al. 2008) and the cognitive demand processes (Quant et al. 2004b; Little and Woollacott 2015). However, a recent evoked cortical source localization study has shed new light on the location of the specific neural generators at the FCz site for N1 response in balance reactions (Marlin et al. 2014). Marlin and colleagues used two task conditions; the first task (postural task) was a standing lean and release and the second task (cognitive task) was a flanker task which subjects were instructed to respond to the direction of the target arrow on the computer screen using left or right computer mouse button press (Marlin et al. 2014). It was expected that both tasks would elicit an ERN, but source localization analysis revealed that the sources of the N1 responses were different between tasks. Even though both N1 responses shared the same topographic map which evoked maximally at FCz site, the dipole source analysis inferred that the evoked N1 response was generated in supplementary motor area (SMA) in the postural task, whereas the ERN evoked N1 response in the cognitive task was generated in the ACC. Another recent study by Bogust and colleagues supports the aforementioned statement by which they estimate the location of the cortical sources related to a whole-body surface translation with or without performing visual working memory task (dual- and single-task paradigm) (Bogost et al. 2016). The authors incorporated brain dipole clustering algorithm to estimate distinct cortical sources and related each domain to the anatomical and Brodmann area probability values. They concluded that pre-motor, primary, supplementary motor areas (lesser extent in dual-task paradigm) and somatosensory areas were primary cortical sources related to reactive postural control. Supporting research from Slobounov et al. (2005) and Mihara et al. (2008) point toward the involvement of SMA along with the prefrontal cortex in generating motor plans for compensatory balance reactions.

Postural responses were also accompanied with the aforementioned cortical responses after the onset of visual occlusion. In particular, following visual occlusion in standing, the COP of the participants moved slightly backwards. The forward and backward movement of AP displacement was not observed in sitting task. It is possible that in standing while being attentive and concentrating on mathematics questions prior to visual occlusion, the participants leaned forwards, consistent with the findings reported by Maki and Mcllroy (1996). This may explain why, when vision was occluded and the focus of the participant changed, there was a resulting backwards shift of the COP. More analysis on quantitative COP measurements for 5 s after visual occlusion demonstrated significant differences in RMS, RANGE, and AREA. These findings were consistent with similar results observed in other studies on standing and sitting (Grangeon et al. 2015; Serra-AÑó et al. 2015). However, MV did not change between standing and sitting tasks ($Z = 0.8644$, $p = 0.3863$). It is likely that visual occlusion elicited a relatively small disturbance as compared to perturbation from proprioception, thus resulting the effects on MV may have been too small to measure. The findings here is inconsistent with the previous literature which reported mean velocity was significantly different between eyes-open and eyes-closed conditions (Prieto et al. 1996).

Previous studies reported that P1 and N1 components were evoked when proprioception was perturbed while maintaining balance (Marlin et al. 2014; Bolton 2015). The rhythmic activity of EEG has been reported in studies that include eyes-open and eyes-closed conditions while perturbing proprioceptive systems during upright stance (Staines et al. 2001; Del Percio et al. 2007; Petrofsky et al. 2012; Tse et al. 2013). In this study, evoked cortical responses, such as P1 and N1 components, were revealed in response to visual occlusion during standing and sitting while maintaining balance. Moreover, significant differences in the amplitude of N1 were found between the two tasks, suggesting that the N1 response was directly related to postural set. The topographic maps of both tasks further support the findings of this study as the locations that were maximal at the topographic map were similar to the topographic map resulting from proprioception. This suggests that the cortical processes,

such as the N1 amplitude, could potentially verify the weight of an individuals vision or proprioception towards postural control. However, a limitation of the current study was the small sample size ($n = 10$) reducing the potential generalisability of the findings. However the sample size is consistent with similar research in the area (Adkin et al. 2008; Mochizuki et al. 2010; Marlin et al. 2014) and reflects the complexity of EEG measurement and processing. While non-significant difference in the P1 between conditions in the study could be a type 2 error due to the small sample size, this is unlikely as the p values were relatively large.

Optimum balance is achieved through a distributed neural network (Bolton 2015), and the results from this study provide insights into the underlying mechanisms of cortical role in compensatory balance. Specifically, the study shows that a simple change in sensory information during postural control (in the absence of a physical perturbation) provides evidence that the N1 reflects cortical processing associated with instability. Understanding cortical responses is important, because the evoked components involved in postural control may be impaired leading to impaired postural responses (Jacobs 2014). If we can identify deficits in N1 in specific patient groups, we may be able to anticipate those at risk of falling. Ultimately, this work can provide critical insights to develop and support interventions and clinical outcomes for those with balance disorders (Jacobs 2014).

Conclusion

In summary, this study revealed that cortical activity was altered by visual occlusion and modulated by postural stability. The N1 components significantly varied in standing and sitting tasks, suggesting that postural demand altered the amplitude of N1. These cortical responses were then accompanied by postural responses and postural sway measures were smaller in sitting compared with standing. As such, these cortical and postural responses paralleled findings of the previous studies based on proprioceptive perturbation in balance maintenance. Further investigation is needed to explore the underlying neurophysiological mechanisms in balance control under altered sensory conditions and in neurological disorders. Ultimately to provide critical insights to develop and support interventions and clinical outcomes for those with disorders of postural control (Jacobs 2014).

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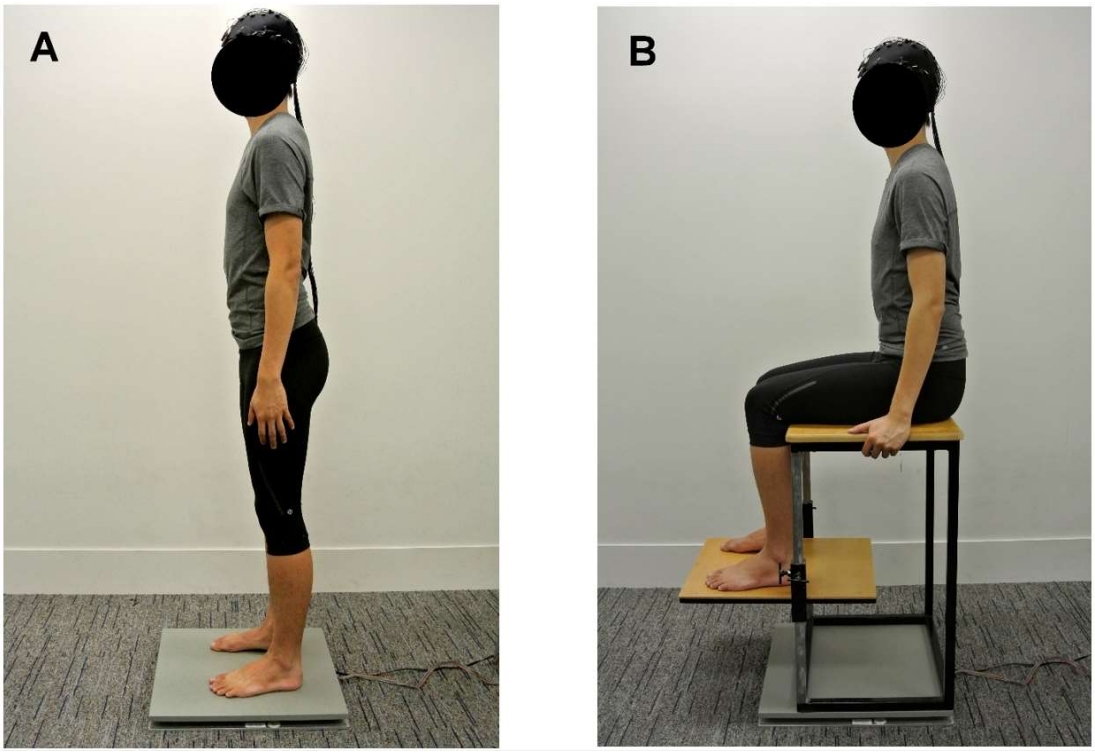
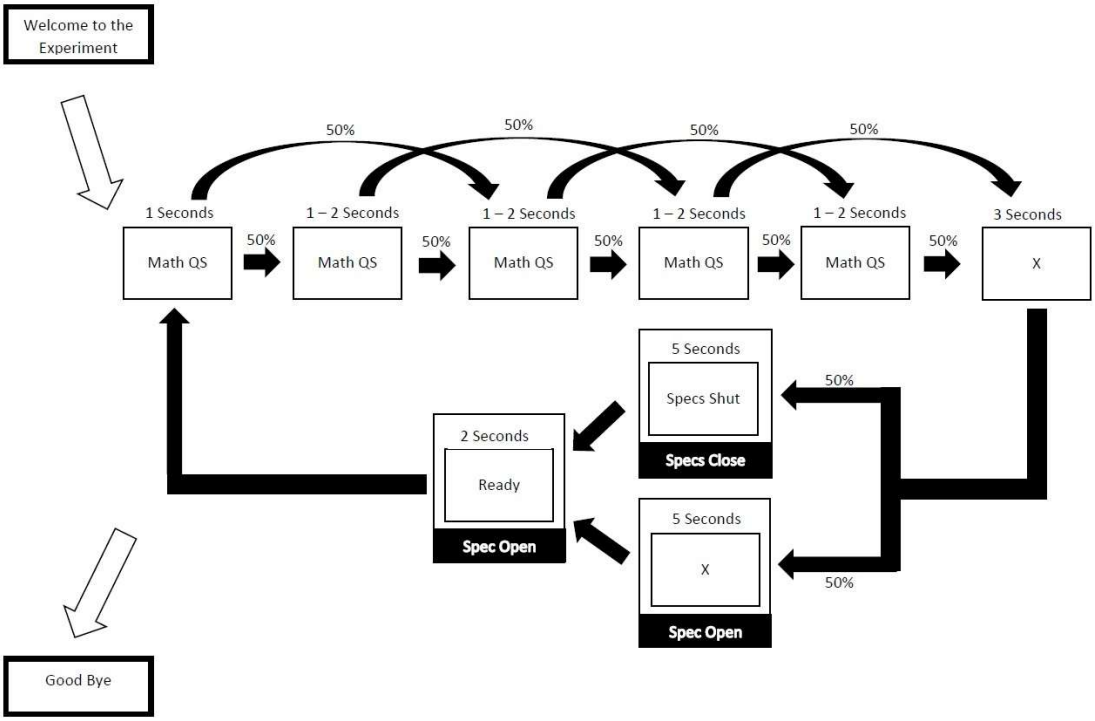
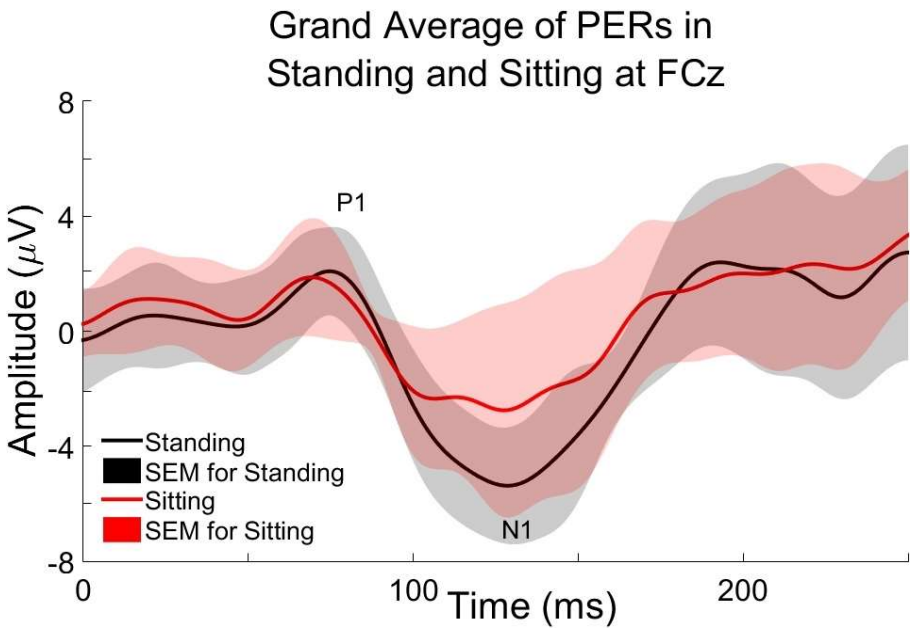


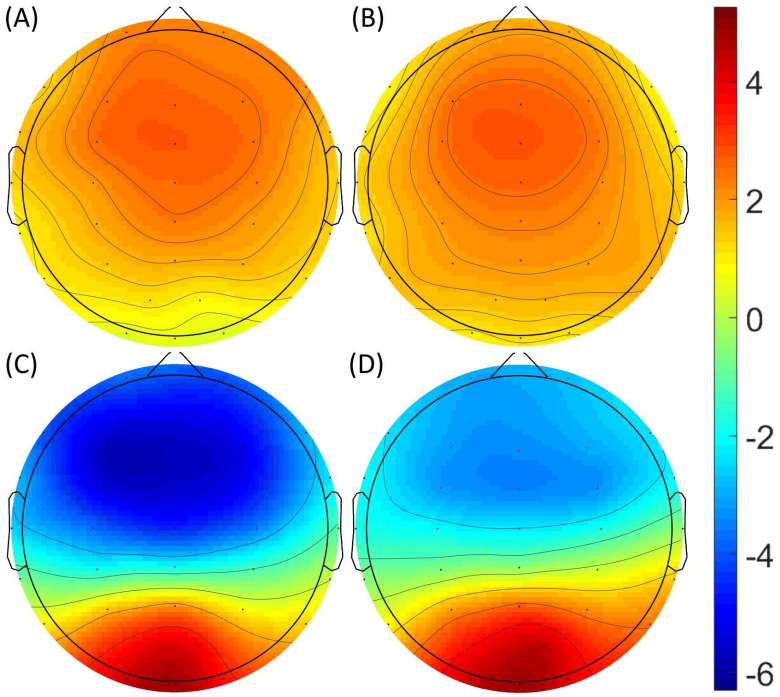
Supplementary Figure 4.1 Experimental setup for testing: (A) Standing (B) Sitting



Supplementary Figure 4.2 Presentation of the experiment for a single trial



Supplementary Figure 4.3 Grand average of PERs in standing and sitting at frontal-central site. *SEM stands for the standard error of the mean. See Appendix B for more details.



Supplementary Figure 4.4 Grand average of the EEG topographic maps of P1 and N1 based on the peak latencies for each subject. The power unit for the scale bars is μV and subjected to a log_{10} transform to normalise the distribution. (A) Standing P1 (B) Sitting P1 (C) Standing N1 (D) Sitting N1. See Appendix B for more details.

Supplementary Table 4.1 The median and percentiles (25th and 75th) of peak latency and peak amplitude of P1, N1 and P300 at FCz site in the standing and sitting tasks. Wilcoxon signed rank test z-score and p-value were measured between standing and sitting tasks.

		Median (percentiles)[a]		Z^b	p^b
		Standing	Sitting		
Peak latency (ms)	P1	74 (67, 79)	68.5 (65.75, 74.75)	1.1760	0.2396
	N1	130.5 (121.75, 137.5)	128 (114.25, 132.5)	-0.5606	0.5751
Peak amplitude (μV)	P1	2.543 (1.3735, 3.9623)	2.4875 (1.5438, 3.5968)	-0.4590	0.6462
	N1	-6.4085 (-7.41, -4.8963)	-2.4315 (-6.7988, -1.9225)	-2.2934	0.0218

Wilcoxon signed-rank test *z* score and *p* value were measured between standing and sitting tasks

[a] Percentiles provided are median (25th and 75th)

[b] Z-score and p-value for Wilcoxon signed-rank test.

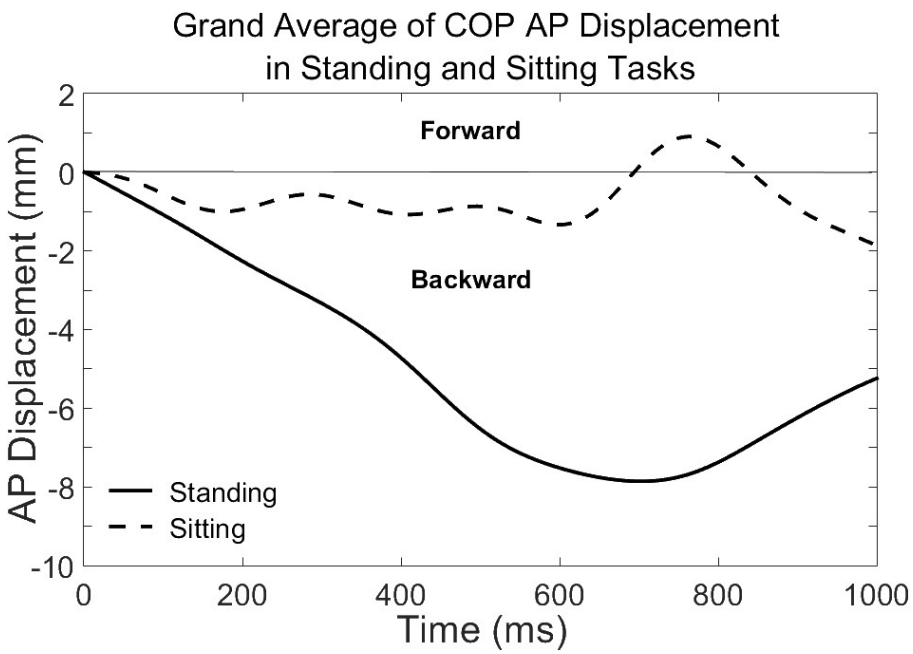
Supplementary Table 4.2 The median and percentiles (25th and 75th) of root means square (RMS), mean velocity (MV), total range (RANGE) and sway area (AREA) for standing and sitting tasks. Wilcoxon signed rank test z-score and p-value were measured between standing and sitting tasks.

	Median (percentiles)[a]		Z^b	p^b
	Standing	Sitting		
RMS	3.6876 (1.8986, 4.7761)	0.8941 (0.8142, 1.1294)	2.7011	0.0069
MV	4.6193 (3.9767, 5.245)	4.2058 (3.0901, 5.163)	0.8644	0.3863
RANGE	11.4572 (5.5737, 15.9401)	4.0407 (3.5303, 4.9456)	2.7011	0.0069
AREA	11.1556 (5.5897, 17.57)	3.9516 (2.6903, 6.2211)	2.4973	0.0125

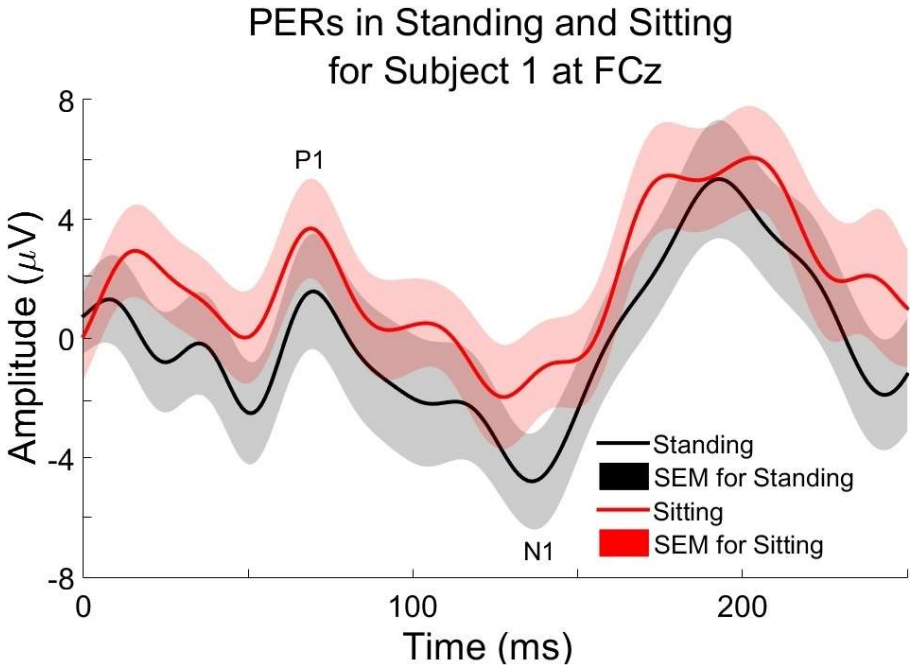
Wilcoxon signed-rank test *z* score and *p* value were measured between standing and sitting tasks

[a] Percentiles provided are median (25th and 75th)

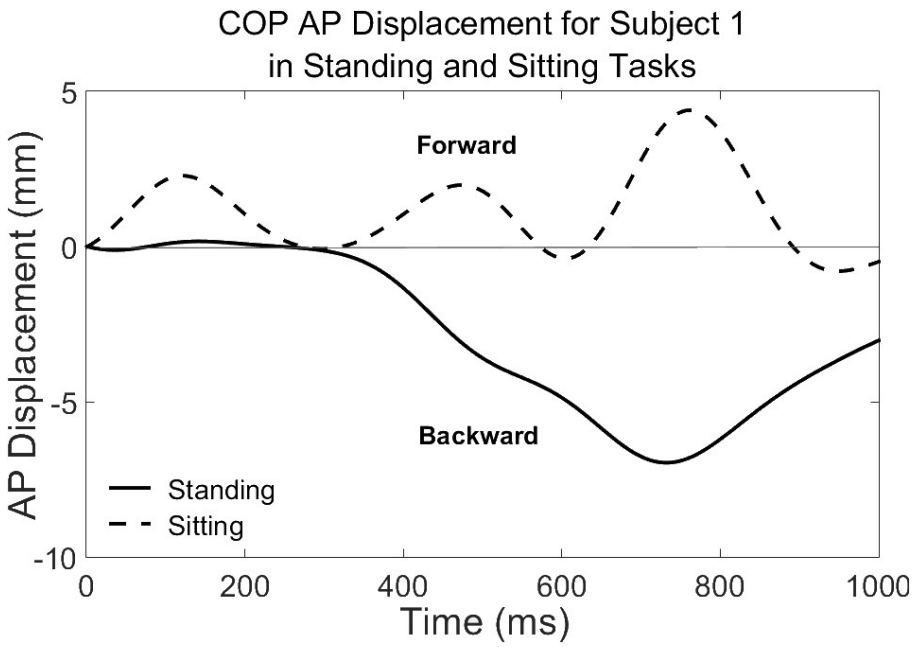
[b] Z-score and p-value for Wilcoxon signed-rank test.



Supplementary Figure 4.5 Grand average of anterior-posterior displacement of COP (mm) in standing and sitting plotted against time (ms) from the onset of visual occlusion. Zero displacement is the starting point of the normalized absolute position.



Supplementary Figure 4.6 PERs in standing and sitting at frontal-central site for Subject 1. *SEM stands for the standard error of the mean.



Supplementary Figure 4.7 Anterior-posterior displacement of COP (mm) in standing and sitting plotted against time (ms) from the onset of visual occlusion for Subject 1. Zero displacement is the starting point of the normalized absolute position.

Chapter 5

Cortical activity in postural tasks with visual conditions

5.1 Introduction

Earlier studies have used the spectral analysis of electroencephalography (EEG) theta (4-7 Hz) and alpha (8-12 Hz) oscillations to evaluate cortical involvement in postural control. Two studies found that observers showed greater postural instability and more EEG theta activity in a three-dimensional visual environment than in a two-dimensional visual environment (Slobounov et al., 2015; Slobounov et al., 2013). This increased EEG theta activity suggested a recruitment of brain resources to meet the spatial demands of the postural task (Slobounov et al., 2015; Slobounov et al., 2013). Other than in postural control, elevated EEG theta activities have been observed in studies investigating in visuomotor tasks, motor learning, and attention (Klimesch, 1999; Slobounov, Fukada, et al., 2000). Aside from EEG theta activity, the EEG alpha activity may also be used as an indicator of higher level balance control because of its crucial role in engagement and disengagement in the thalamus and motor cortex regions, which is essential for the processing of sensory information and necessary for posture control (Haegens et al., 2011; Hülsdünker, Mierau, & Strüder, 2015; Lőrincz et al., 2009). Moreover, the measurement of EEG alpha activity has been used to assess memory and attentional performances (Başar et al., 1997; Klimesch, 1999; Klimesch et al., 1998). Hence, EEG theta and alpha measurements may be useful as a physiological indicator of higher level balance control (i.e. balance control involving

cerebral cortex).

Studies that investigated the association between bipedal standing postural control and cortical activities in healthy young adults have yielded inconsistent results. Hülsdünker et al. found that participants produced elevated levels of EEG theta and alpha activities during experimental conditions that required a larger postural demand (Hülsdünker, Mierau, Neeb, et al., 2015; Hülsdünker, Mierau, & Strüder, 2015). In contrast, no significant associations were reported in other studies (Tse et al., 2013). Discrepancies in the results may be due to the inherently unstable nature of standing (Assländer & Peterka, 2014; Palmieri et al., 2003; Pasma et al., 2012; Peterka, 2002), inevitability generating cortical activities in the participants. Changing the experimental condition from a standing task to a sitting task may minimise discrepancies of the results because a sitting task requires lesser postural demand (Gatev, Thomas, Kepple, & Hallett, 1999).

The visual system has been known to play a major role in balance and postural control. While healthy people predominantly rely on their proprioceptive system to maintain stance (Ben-Itzhak et al. 2010; Peterka 2002), closing their eyes increases postural sway, indicating that vision does indeed play a role in postural stability (Kuo et al. 1998). The process of regulating the contribution of each sensory system to maintain balance is referred as sensory reweighting (Assländer & Peterka, 2014). Depending on the environment and the postural disturbance, the central nervous system down-weights less reliable information and up-weights reliable information to achieve balance control (Pasma et al., 2012). As a result, this information transmission could contribute to alpha and theta activity (Lőrincz et al., 2009). Thus, it is important to investigate the cortical contribution of the sensory systems in different postural demand tasks.

With sufficient sensory information, an optimal postural control system is achieved by the interplay of control mechanisms such as the afferent feedback from sensors and efferent feedforward of motor control. Afferent feedback has commonly been reported in evoked potential studies (Kasai, Kawai, Kawanishi, & Yahagi, 1997;

Shimazu et al., 1999) and the event-related potentials have been reported associated with an increase in theta rhythm (Hasselmo, 2006). Thus, afferent feedback could influence theta rhythm. Additionally, there have been reports suggesting theta activity in balance control is likely to reflect not only afferent feedback but as well as efferent feedforward responses (Gatev et al., 1999; Palmieri et al., 2003). While postural sway is the result of efferent processes, the theta oscillations may also be influenced by efferent feedforward motor control.

Therefore, the purpose of this study was to investigate the effect of postural demands and visual input on cortical activity (alpha and theta activity) in healthy young adults by assessing them in conditions of sitting and standing during the availability and removal of visual input (i.e. visual transparent (VT) and visual occlusion (VO)). It is hypothesised that EEG theta and alpha activities will increase as postural demands increases, reflecting greater recruitment of brain resources for the more challenging postural demand task (Lőrincz et al., 2009). Moreover, it was hypothesized that regulating the contribution of the sensory systems such as reduced visual input will be accompanied with an increase of theta reflecting afferent and efferent response signals (Gatev et al., 1999; Palmieri et al., 2003).

5.2 Methods

5.2.1 Participants

Twenty-four healthy young adults (5 females and 19 males, 26.3 ± 5.6 years, 171.9 ± 7.5 cm, 69.3 ± 14.4 kg) provided written informed consent to participate in this study. Sample size calculation, based on a post hoc power analysis of a pilot study (Goh, Morris, Lee, Ring, & Tan, 2016), accounting for an effect size of 0.695, indicated that 14 participants are required for this study to achieve a statistical power of 80%. Exclusion criteria for this study included any self-reported neurological or musculoskeletal atypicalities. The study was performed in accordance with the guidelines of the Helsinki Declaration and was approved by the Human Research

Ethics Committee of Curtin Universtiy (ENG-71-14-06).

5.2.2 Data Acquisition

Electroencephalography (EEG) signals were collected using a 40 channel Ag/AgCl electrode cap (Neuroscan, El Paso, TX, USA), set up according to the International 10-20 System. All channels were referenced to the average signal recorded in the A1 and A2 electrodes located on the left and right mastoid with a forehead ground, respectively. The impedance of all channels was maintained below 5kΩ throughout the experiment. Additional electrodes were placed above and below the left eye, and on the outer canthus of each eye to monitor electrooculogram (EOG) signals. Both EEG and EOG signals were measured in a monopolar mode with a sampling rate of 1000 Hz.

5.2.3 Experiment Protocol

Participants were allocated to either the sitting group or the standing group (Figure 5.1). All participants follow a standardised instruction in each of their respective groups. In the sitting group, participants sat on a stool placed on top of a force platform and placed their hands on the side of the stool. The customised stool measured 700 mm in height by 500 mm in width and 400 mm in length. This stool included an adjustable footrest for participants to maintain 90 degrees knees and hip flexion. Also, the stool could be adjusted for participants to achieve eye-level height with the computer screen. In the standing group, participants stood on a force platform with their feet shoulder-width apart and their hands relaxed beside the body. Participants from both groups were positioned 1.5 meters away from the height-adjustable computer screen. The first author conducted the experiments.

In both standing and sitting conditions, participants wore the liquid crystal spectacles (PLATO, Translucent Technologies Inc, Toronto ON, Canada) and looked at the computer screen in front of them. When the participants were relaxed and ready, the investigator began recording the EEG data. There were two experimental

conditions: (1) The liquid crystal spectacles being in the open state and participants were told beforehand to fixate on an “X” at the centre of the screen (VT); (2) The spectacles being in the closed state (VO). Participants were instructed to keep their eyes open throughout the experiment and to look directly forward. The duration of each condition recorded was 75 seconds. The tasks were presented in a sequential order of standing with the visual transparent condition, standing with the visual occlusion condition, sitting with the visual transparent condition and sitting with the visual occlusion condition. A one-minute rest period was included between conditions to minimise fatigue. Additional blocks were conducted if there were suspicions of unwanted signals from the eye movements, blink artefacts or muscle activity that may contaminate the data.

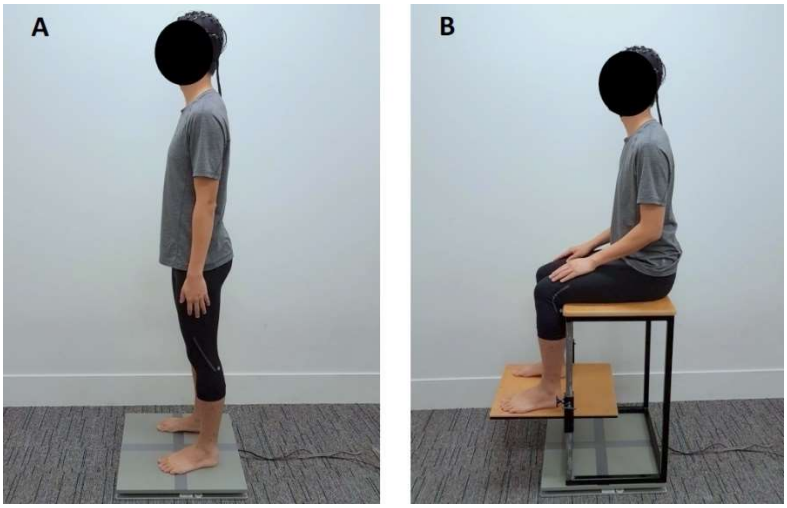


Figure 5.1 The experimental setup for testing: (A) Standing (B) Sitting.

5.2.4 Data Processing and Analysis

Analysis of the EEG signals was performed using MATLAB 2015a (Mathworks Inc, Natick MA, USA). Specifically, the EEGLAB toolbox (Delorme & Makeig, 2004) was used to pre-process EEG signals with signal filtering and artefact removal. The EEG signals were initially band-pass filtered using second-order zero phase 0.5 Hz elliptic high-pass filter and eighth order zero phase 40 Hz elliptic low-pass filter. Following this, one independent component concerning the visual artefact was removed using

independent component analysis with reference (Lee et al., 2016). The total length of the EEG was 60 seconds per participant and was segmented into 5 seconds for power spectrum analysis. The theta (4-7Hz) and alpha power (8-12Hz) for each channel was computed based on the power spectrum estimated from the Welch method with no overlapping. Three regions of interest (ROI) were highlighted; frontal-central (FC3, FCz, FC4), central (C3, Cz, C4) and central-parietal (CP3, CPz, CP4). These ROIs were selected as they are suggested to be associated with cognitive, premotor/motor processing and somatosensory processing (Hülsdünker, Mierau, & Strüder, 2015; Koessler et al., 2009). The average power values over the electrodes were computed for the ROIs. The EEG analyses were performed by omitting the first 15 seconds to avoid the immediate responses of participants due to visual occlusion or transparency. The Box-Cox transformation was applied to the absolute theta and alpha power to approximate normal distribution before statistical analysis.

5.2.5 Statistical Analysis

Statistical analyses were undertaken using SAS 9.4 (SAS Institute Inc., Cary, NC). Cortical activities in three regions were examined using mixed linear regression models with a random effect (participant) and two fixed effects (visual conditions and postural tasks). These three regions of interest included the frontal-central, central, and central-parietal area of the brain. A main effect is the effect of a single independent variable on a dependent variable whereas the interaction term is the effect of one independent variable may depend on the level of the other independent variable. Main effects and interaction terms were retained if p-value was considered significant. Post hoc comparison of group differences were performed by calculating the least squares means (LSM). An alpha level of $p < .05$ was used to indicate statistically significance in all tests. A term for time interval (in 5-second blocks) from the commencement of each experiment was also included in the model to investigate its influence.

5.3 Results

Time (i.e. the 60 seconds duration) was observed not contributing to the model and was removed from the analysis ($p > .20$ for all regions in theta power and $p > .13$ for all regions in alpha power). Table 5.1 shows the summary results of the model fitting for both theta and alpha power.

5.3.1 Theta Power

Table 0.1 The main effect of visual condition and the postural task of theta and alpha power and their interactions within each region. Results provided are the F-statistics (p-value). All degrees of freedom for the F-tests were (1,69).

			Frontal Central	*Central*	*Central Parietal*
Theta (μV^2)	Main Effect	Visual Condition	21.34 (<.0001)	17.98 (.0001)	9.53 (.0029)
		Postural Task	51.89 (<.0001)	36.08 (<.0001)	5.64 (.0203)
	Interaction		1.23 (.2716)	0.45 (.5069)	1.37 (.2465)
Alpha (μV^2)	Main Effect	Visual Condition	14.47 (.0003)	20.53 (.<.0001)	18.69 (<.0001)
		Postural Task	64.37 (<.0001)	83.46 (<.0001)	44.07 (<.0001)
	Interaction		0.02 (.9019)	2.43 (.1235)	0.28 (.6011)

There were no significant interactions in theta power between the visual condition and the postural task at the frontal-central region, central region or central parietal region (Table 5.1). The main effect of visual condition was statistically significant in all three regions, indicating that the theta power was significantly greater for VO condition than for VT condition (Figure 5.2). The main effect of postural task was also statistically significant in all three regions. The postural task (Stand > Sit) appeared to influence theta power at all of the regions in VT and VO condition, and theta power was significantly greater for STD task than SIT task (Figure 5.2).

5.3.2 Alpha Power

There was no visual condition by postural task interaction for alpha power at the frontal-central region, central region or central parietal region (Table 5.1). There were

also significant effects in alpha activity at the three regions between VO and VT tasks (Figure 5.3). The main effect analysis also showed that alpha power at the three regions was affected by postural task with increased alpha power in the standing condition (Figure 5.3).

The topographic maps of EEG theta and alpha activities during visual condition and postural task are presented in Figure 5.4 and 5.5.

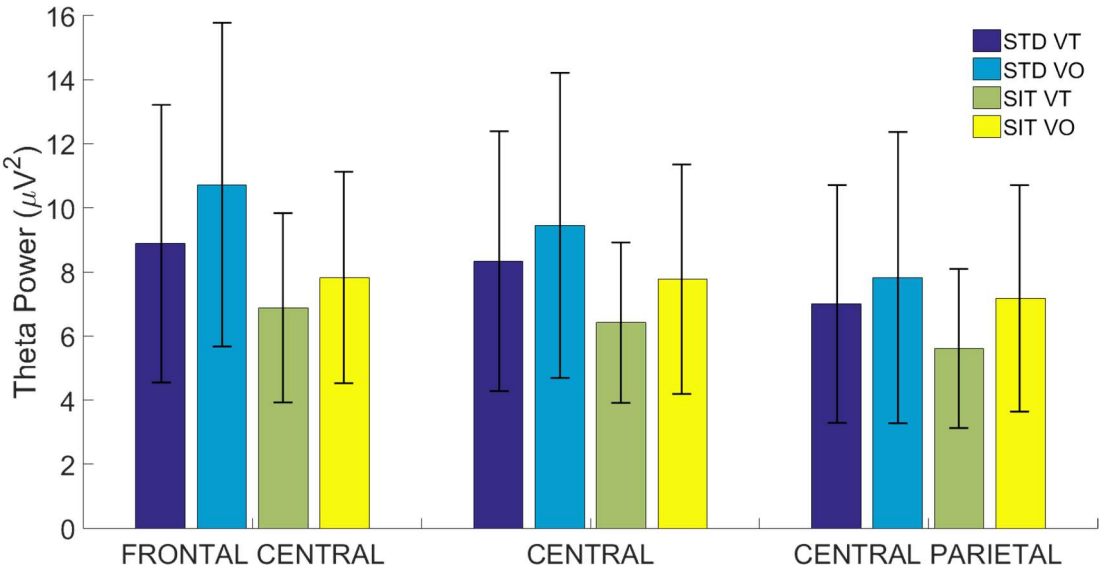


Figure 5.2 Bar chart of the channel-averaged theta power (raw data) at the frontal-central, central and central parietal regions of all participants in the various postural task and visual conditions. The error bar in each bar represents the standard deviation of the theta power.

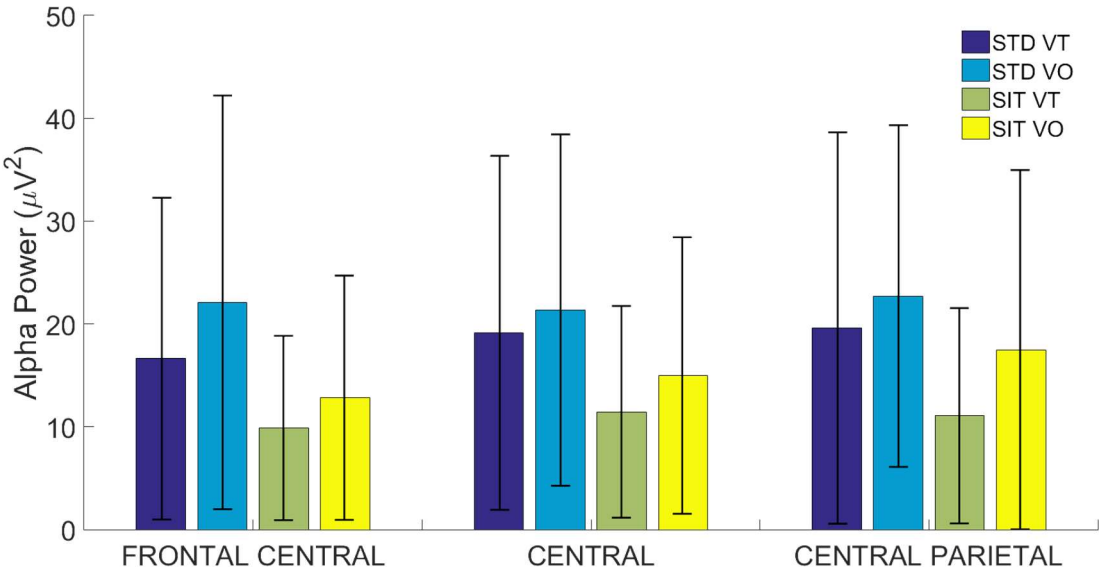


Figure 5.3 Bar chart of the channel-averaged alpha power (raw data) at the frontal-central, central and central parietal regions of all participants in the various postural task and visual conditions. The error bar in each bar represents the standard deviation of the alpha power.

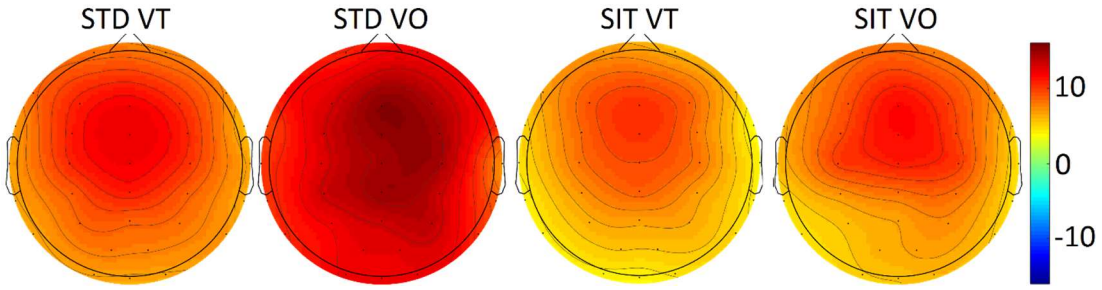


Figure 5.4 Topographic effects of the postural tasks. Each topographic map reveals the mean theta rhythmic activity across the single channel. The power unit for the scale bar is μV^2 and subjected to a log_{10} transform to normalise the distribution.

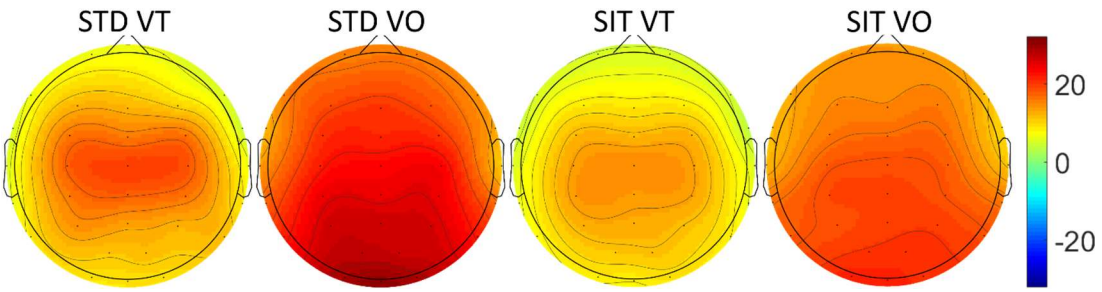


Figure 5.5 Topographic effects of the postural tasks. Each topographic map reveals the

mean alpha rhythmic activity across the single channel. The power unit for the scale bar is μV^2 and subjected to a log_{10} transform to normalise the distribution.

5.4 Discussion

This study examined the cortical activities of healthy young adults in conditions that influenced the postural demands and visual input of the individual. Consistent with our first hypothesis, EEG theta activities increased as postural demands increased. The results are in line with a previous study suggesting an increase in central-parietal theta power as a function of sensory integration and sensorimotor coordination (Hülsdünker, Mierau, & Strüder, 2015). Previous research has documented that the increase in theta power at the frontal-central region may be related to motor planning behaviour (Slobounov et al., 2015). Since the standing condition has smaller base of support and is accompanied by an increased postural sway (Carpenter, Frank, Winter, & Peysar, 2001; Tse et al., 2013), it appears that the increase in frontal-central theta power is related to a higher level of cortical processing of postural control as reported in other studies (Adkin et al., 2006; Hülsdünker, Mierau, Neeb, et al., 2015). However, the increase of EEG theta activities was not restricted to the frontal-central and central-parietal regions, with increased theta power also appearing in the central region, consistent with previous studies which have reported an increase in theta power at the central region during the transition to instability (Slobounov, Cao, Jaiswal, & Newell, 2009). This increase of the central theta could indicate the involvement of anterior cingulate cortex, which monitors postural stability (Slobounov et al., 2009). In summary, an increase in theta power over the frontal-central, central and central-parietal regions during a postural task are most likely related to the detection and processing of postural instability.

In this study, a reduction in theta was observed when visual input was available. It has been suggested that this reduction of theta reflects a global increase in cortical activation (Barry, Clarke, Johnstone, Magee, & Rushby, 2007). The role of theta in the processing of visual information is unclear. One possibility is that theta contributes to

the processing of afferent feedback when postural demand is present. The contribution of theta power oscillations has been associated with an increasing strength of afferent feedback (Hasselmo, 2006). Additionally, the afferent feedback mechanism is stimulated when posture is disturbed (Gatev et al., 1999). Thus, postural instability induced by higher postural demands in standing could have resulted in an increase in afferent feedback and hence higher theta power oscillations. Moreover, theta bursts using transcranial magnetic stimulation has after effects on the sensorimotor cortex in humans that alter evoked potentials (Ishikawa et al., 2007). Therefore, it is likely that the increase of theta power seen in our study reflects visual afferent feedback related to postural control.

Although an increase in theta power is reported to be related to increasing postural demands, it is not the only EEG activity related to postural control. The results for alpha power are consistent with our first hypothesis which shows a global increase in alpha power in the frontal-central, central and central-parietal region during higher postural task demands (Hülsdünker, Mierau, & Strüder, 2015); likely reflecting increased information transfer between the thalamus and the cortex (Lőrincz et al., 2009). These results also support the scalp distributions of alpha power with an increase in the frontal-central midline region suggested to reflect the role of concurrent sensorimotor and cognitive processes for postural control (Varghese et al., 2014). However, a significant reduction in alpha power has been reported at the central frontal region prior to medial-lateral sway (Slobounov et al., 2008) and at the occipital region when transitioning to instability (Slobounov et al., 2009). The reduction in alpha activities was thought to be closely related to cognitive function such as attention (Başar, 2012; Klimesch et al., 1998). If the attentional demands from vision modulated the alpha power, it might be expected that the alpha power in the standing and sitting tasks during the visual transparent condition may be the same, since the visual environment was the same. However, participants in the standing task showed a larger level of EEG alpha activity than in the sitting task, suggesting that an increased alpha power was related to the increase in postural demands. Thus, EEG alpha activity may

be a reliable indicator of postural control.

The modulation of alpha power was observed during the visual condition in all regions. These findings are consistent with studies reporting that alpha power was reduced from the eyes-closed to the eyes-open condition (Barry et al., 2007; Boytsova & Danko, 2010). The reduction of alpha activity from eyes-closed to eyes-open in standing could reflect an increase in arousal as a correlation between high skin conductance and global reduction of alpha during the eyes-open condition has been reported previously (Barry et al., 2007). However, this seems unlikely as a reduction of alpha activity from VO to VT condition from the results was also observed in the sitting task, and sitting in silence decreases the level of arousal (Nantais & Schellenberg, 1999; Thompson, Schellenberg, & Husain, 2001). The alpha power in visual condition is associated with the modulation of sensory input (Niedermeyer, 2005; Pfurtscheller, Stancak, & Neuper, 1996) and the engagement or disengagement of sensory regions (Haegens et al., 2011). As the sitting task had a lower postural demand compared with the standing task, the alpha power was modulated by postural task, rather than the visual input.

EEG theta and alpha activities were highest during the eyes closed condition, regardless of the postural demand. The results suggest that changes in cortical activity across the visual conditions and postural tasks are independent and therefore perhaps generated by different mechanisms in the brain. Postural demand manipulation primarily produced a higher cortical activity at the frontal region of the brain, which could relate to the detection and processing of the actual balance state (Slobounov et al., 2009). It is also evident that the frontal region is correlated with the activation of the motor and somatosensory evoked potentials (Ishikawa et al., 2007). A supporting study from functional near-infrared spectroscopy also suggested a significant increase in oxyhemoglobin concentration at the frontal lobe in the more challenging postural task (Fujita et al., 2016). In our study, the widespread activation of the entire cortex when there was visual input suggested that information processing was presented between the cortex and the thalamus (Başar, 2012). Thus, the results suggested that

different brain regions were involved in processing postural task and visual condition, respectively.

Bipedal upright stance is inherently unstable due to a large body mass kept at a high elevation and a relatively small base of support (Kouzaki & Masani, 2008; Peterka, 2002; Qu, Nussbaum, & Madigan, 2007). In this study, significant differences in theta and alpha activities across frontal central, central and central-parietal region were reported in the two postural tasks (STD, SIT) and two visual conditions (VT, VO). An increase in theta and alpha activities were consistent with the increase in postural demand. Moreover, when there was no vision, the results demonstrated that theta and alpha activity in the low postural demand condition was lower than in the high postural demand condition, supporting the suggestion of greater cortical resource allocation in a more demanding balance task (Hülsdünker, Mierau, & Strüder, 2015). The topographic maps of the postural tasks in the two visual conditions further support the findings of our study as global increases in theta activity were observed when postural demand alpha activity was elevated, and alpha activity was elevated posteriorly in VO compared to VT condition regardless of the postural task.

The limitation of this study was that the spatial resolution of the EEG scalp surface recordings is limited. However, the spatial resolution of EEG can be addressed using EEG source localisation techniques such as standardised low-resolution brain electromagnetic tomography (Pascual-Marqui, 2002) to represent local underlying brain sources more accurately. Further details on the source localisation techniques are described by Grech et al. (Grech et al., 2008).

5.5 Conclusion

Earlier studies have demonstrated the role of the cerebral cortex in balance (Bolton, 2015; Marlin et al., 2014; Mihara et al., 2008; Slobounov et al., 2005). This study revealed the alteration of EEG theta and alpha activities induced by the availability of visual input and by the postural task of varying postural demands. Elevated theta and alpha activities during changes in postural demand suggested that more brain resources

are allocated to postural tasks that are more demanding. These findings of this study were similar to findings of previous studies based on increasing postural demands and the eyes-open/eyes-closed condition. Furthermore, this study also revealed theta and alpha activities in low postural demand conditions were lower than high postural demand conditions, supporting our hypothesis on postural demand. The results of this study establish a link between postural task and cortical activity, specifically the modulation of theta and alpha activity.

Chapter 6

Postural and cortical responses following visual occlusion in adults with and without Autism Spectrum Disorder

This chapter is covered by the following publication:

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ORIGINAL PAPER

Postural and Cortical Responses Following Visual Occlusion in Adults With and Without ASD

Kwang Leng Goh[1] · Susan Morris[2] · Richard Parsons[2] · Alexander Ring[3] · Tele Tan[1]

Abstract
Autism is associated with differences in sensory processing and motor coordination. Evidence from electroencephalography suggests individual perturbation evoked response (PER) components represent specific aspects of postural disturbance processing; P1 reflects the detection and N1 reflects the evaluation of postural instability. Despite the importance of these cortical responses to postural control, PERs to a perturbation in adults with autism spectrum disorder (ASD) have yet to be reported. The aim was to compare PERs to visual perturbation under varied postural stability conditions in adults with and without ASD. This study is the first to report that while the assessment of postural set is intact, adults with ASD use more cortical resources to integrate and interpret visual perturbations for postural control.

Keywords ASD · Perturbation evoked response · Postural control · Postural disturbance · Vision · Sensory integration

Introduction

Sensory impairment is now accepted as a core symptom of ASD (Amerian Psychiatric Association 2013). Numerous studies have reported at least 90% of children with ASD demonstrate evidence of unusual responses to sensory information (Crane et al. 2009; Pfeiffer et al. 2011; Tomchek and Dunn 2007). Sensory impairments in ASD are associated with social and behavioural deficits (Baum et al. 2015; Nebel et al. 2016). In particular people with ASD demonstrate deficits in adaptive orientation to behaviourally relevant sensory information (Keehn et al. 2013). People with ASD also demonstrate poorer motor skills (Bhat et al. 2011), even when no sensory manipulations have been performed (Fournier et al. 2010). Poorer motor skill development in people with ASD may reflect reduced or altered motor learning secondary to these differences in adaptive orientation to relevant sensory information. Furthermore, sensory, social, motor and behavioural difficulties in ASD commonly persist into adulthood (Bhat et al. 2011; Minshew et al. 2004; Schall and McDonough 2010).

Sensory impairments can cause postural instability as evidenced by an increase of postural sway. A recent systematic review reported that anterior-posterior (AP) sway and medial-lateral (ML) sway is larger in people with ASD than typically developing (TD) people while standing quietly (Lim et al. 2017), especially the total excursion of the AP and ML sway directions (Graham et al. 2015; Stins et al. 2015b). The problem is reported to lie in problems of multimodality sensory integration of individuals with ASD (Minshew et al. 2004). Despite numerous studies investigating sensory impairments in individuals with ASD, there is a lack of research investigating the cortical responses which contribute to sensory integration in individuals with ASD.

Perturbation-evoked responses (PERs) to a physical or visual perturbation causing postural stability have been detected using electroencephalography (EEG). Predominantly research on PERs has been conducted using mechanical perturbations, involving primarily the proprioceptive system, to induce postural reactions (Bolton 2015; Marlin et al. 2014). However, a recent study revealed that the PERs were also evoked after the onset of sudden and unexpected visual occlusion (Goh et al. 2017). These responses which peak at the frontal-central site (FCz), demonstrate the detection component (P1) followed by the evaluation component

✉ Kwang Leng Goh
kwangleng.goh@postgrad.curtin.edu.au

[1] Faculty of Science and Engineering, Curtin University, Bentley, WA, Australia

[2] Faculty of Health Sciences, Curtin University, Bentley, WA, Australia

[3] School of Surgery, University of Western Australia, Perth, WA, Australia

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(N1) after the onset of a postural perturbation (Bolton 2015; Goh et al. 2017; Marlin et al. 2014; Mochizuki et al. 2010; Varghese et al. 2014). The N1 component is particularly interesting, since the peak amplitude of the N1 component is known to be affected by age (Toledo et al. 2016), predictability (Adkin et al. 2006; Jacobs and Horak 2007; Mochizuki et al. 2008), size of the stimulus (Staines et al. 2001), and cognitive tasks (Little and Woollacott 2015; Quant et al. 2004). A more recent study demonstrated an increase of N1 peak amplitude with an increase in postural demand i.e. in standing vs sitting (Goh et al. 2017). Thus, a larger peak amplitude of the N1 component likely indicates a modified "central postural set", which reflects influences of central drive on automatic postural responses to external perturbations as suggested by Little and Woollacott (2015). As individuals with ASD often demonstrate a larger postural sway, we questioned if the exaggerated postural responses seen in ASD are exhibited due to a different sense of instability (P1) or a difference in "central postural set" (N1)? Specifically, is the larger postural sway observed in ASD related to impaired postural disturbance detection (P1) or processing (N1)? Additionally if "central postural set" is affected, is this limited to an impaired assessment of postural demand or is it a more generalised issue? PERs are useful to evaluate brain function in response to specific sensory or motor events, and impairment in PERs during postural control could lead to impaired postural responses. To the extent of our knowledge, PERs have not been used to evaluate the brain responses to postural perturbations in individuals with ASD.

Where sensory information conflicts, vision plays an important role in postural control. It is evident that individuals with ASD use visual and proprioceptive information differently than TD individuals (Glazebrook et al. 2009; Morris et al. 2015). In particular, individuals with ASD appear to rely less on vision to control posture (Morris et al. 2015). The aim of the study was to investigate the PERs to visual perturbation under varied postural demand conditions (standing and sitting) in adults with and without ASD. It is hypothesized that the PERs will be evident in response to a visual occlusion in adults with ASD. Moreover, it is hypothesized that while the latencies of P1 and N1 will remain constant, the peak amplitude of the PERs to a visual occlusion in adults with ASD will be smaller than those seen in TD adults in the postural tasks, consistent with the previous evidence that vision is not used to control posture in ASD to the same degree as TD adults. Lastly, we hypothesize that reduced PERs will be accompanied by larger disturbance in postural sway in ASD due to reduced cortical processing and subsequent larger error in postural responses.

Methods

Participants

Twenty-six male adults (13 adults with ASD, age = 24.6 ± 2.78 years, height = 179.7 ± 7.2 cm, weight = 78.5 ± 14.0 kg; 13 TD adults, age = 25.5 ± 2.5 years, height = 175.1 ± 7.2 cm, weight = 79.9 ± 9.7 kg) were recruited via service providers, research institutions and social media in Perth, Western Australia. Adults with ASD were eligible for inclusion if they presented with a diagnosis of Autism Spectrum Disorder according to the Diagnostic and Statistical Manual for Mental Disorders 5th Edition (American Psychiatric Association 2013) or equivalent diagnosis of Autism Spectrum Disorder, Asperger Syndrome or Pervasive Developmental Disorder according to the DSM-4-TR (American Psychiatric Association 2000). Adults with ASD and TD adults were excluded if there were prescription medication for the past 3 months, history of severe brain injury or acute mental health illness, and intellectual disability. TD adults were excluded if there were any self-reported neurological or musculoskeletal deficits that may have influence postural control. The TD control group was matched to the ASD group on chronological age ($p = .3786$), height ($p = .1298$), weight ($p = .2815$) and gender. A post hoc power analysis from the previous pilot study (Goh et al. 2017) revealed that using the mean, standard deviation and between-groups comparison effect size (0.918), an N of approximate 12 would be required to achieve a statistical power of recommended 80% power (Cohen 1988). This study was approved by Curtin University Human Research Ethics Committee with written informed consent from all subjects. All subjects gave written informed consent in accordance with the Declaration of Helsinki.

Data Acquisition

Electroencephalography (EEG) signals were recorded using a 40 channel Ag/AgCl electrode cap (Neuroscan, El Paso, TX, USA) based on the International 10–20 System. The impedance of all channels was maintained below 5 kΩ, and all channels were referenced to the average of the electrode A1 and A2 located on the left and right mastoid with a forehead ground. Additional electrodes were placed above and below of the left eye, and on the outer canthus of each eye to monitor electrooculogram (EOG) signals. Both EEG and EOG signals were measured in a monopolar mode with a sampling rate of 1000 Hz.

Centre of Pressure (COP) data was obtained using AMTI AccuGait portable force platform (Advanced

Mechanical Technology Inc., Watertown MA, USA) and recorded using custom made program written in LabView (National Instruments Corporations, Austin TX, USA). The COP data was recorded with a sampling rate of 2000 Hz.

Procedure

The PERs to visual occlusion were evoked using the same experiment paradigm as in a previous study (Goh et al. 2017). Briefly, the visual occlusion condition was initiated by the liquid crystal spectacles (PLATO, Translucent Technologies Inc, Toronto ON, Canada) rapidly shutting so that participants were not able to perceive visual information (Milgram 1987). All participants were barefoot and either stood on the force platform or sat on the stool with the force platform underneath (Fig. 1). In the standing task, participants were informed to stand with their feet slightly apart in their comfortable position with hands relaxed alongside their body. In the sitting task, participants were asked to sit centered on the stool with their hands at the side of the stool. The participants were asked to fixate on the height-adjustable computer screen 1.5 m away in front and at eye level and were positioned by the same investigator for all trials using standardised instructions. The cycle of the task presentation using EPRIME (Psychology Software Tools Inc., Sharpsburg PA, USA) for one trial is shown in Fig. 2. Participants were asked to calculate in their heads a random number of mathematics questions (i.e. mental arithmetic task) followed by an "X" for 3 s before a 50% chance of visual occlusion (VO) condition or visual transparent (VT) condition. The mental arithmetic task was used to engage participants' attention away from the main task (i.e. visual occlusion condition) and the 50% chance of visual conditions was introduced to avoid adaptation and habituation. A total of eight blocks (20 trials each) were presented in a sequential order (i.e. stand, sit, stand, sit and so on) and the duration ranged from 5 to 6 min for each block. Participants were given a resting period to minimize fatigue. At least 30 VO trials were collected from each participant.

Data Processing

The data processing was similar to a previous study (Goh et al. 2017), where EEG signals were high-pass and low-pass filtered at 0.5 and 40 Hz, respectively. Independent component analysis with reference was performed on the EEG signals to remove ocular, muscular and line noise artefact (Lee et al. 2016). The process of extracting the ERPs for the VT condition was equivalent to that used in the Visual Occlusion (VO) condition. The epoch of the PERs ranging from −200 to 500 ms for VT conditions were time-locked to the onset of 5 s 'X' presentation and after the 3 s 'X' presentation. Epochs were rejected if the voltage exceeded 100 μV or suspicious contaminated artefacts were observed during the visual inspection. The first 30 artifact free epochs for each participant were retained to minimise selection from which an average of PERs per participant and a grand average of PERs across all participants was constructed to increase the signal-to-noise ratio of the recorded PER. The analysis of the EEG measures focused on FCz as it had the maximal negativity (Marlin et al. 2014; Varghese et al. 2014). The analysis of the EEG signals was performed using MATLAB 2015a (Mathworks Inc, Natick MA, USA) along with the EEGLAB (Delorme and Makeig 2004) and ERPLAB (Lopez-Calderon and Luck 2014) toolbox.

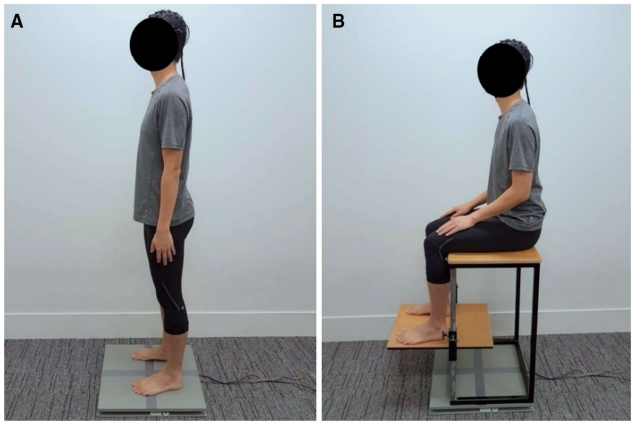

Fig. 1 Experimental setup for testing: **a** standing and **b** sitting

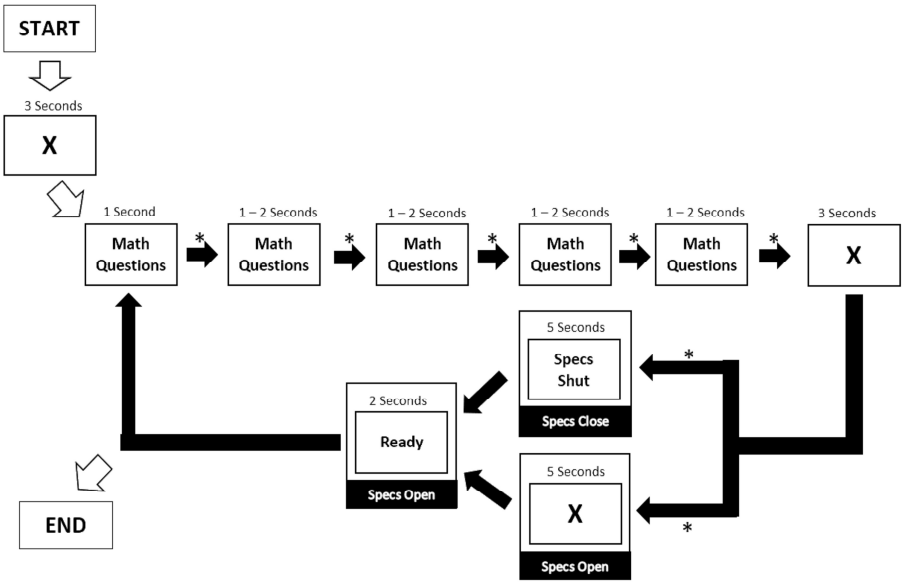

Fig. 2 Presentation of the experiment for a single trial. Asterisk denotes 50% of occurrence

The centre of pressure (COP) data in the anterior-posterior (AP) and medial-lateral (ML) directions was down-sampled to 1000 Hz to match the EEG sampling rate followed by the low-pass filtered at 5 Hz. The linear trends of the AP and ML time series were removed before quantified into the total excursions in the AP direction ($TOTEX_{AP}$) and ML direction ($TOTEX_{ML}$) as these have been reported to be significantly different between TD and ASD (Graham et al. 2015; Stins et al. 2015b). All summations were computed from 1 to N, which N, the number of data points is 5000. The total excursions are the total length of the COP path in the AP direction ($TOTEX_{AP}$) and ML direction ($TOTEX_{ML}$), and is estimated by the summation of the distances between consecutive points in their respective time series such as

$$TOTEX_{AP} = \sum_{n=1}^{N-1} |AP[n+1] - AP[n]|$$

$$TOTEX_{ML} = \sum_{n=1}^{N-1} |ML[n+1] - ML[n]|$$

The COP measurements were reported 5000 ms after the onset of the visual occlusion condition. Only COP trials matching the EEG trials were retained for data analysis to be consistent with the EEG data analysis. The analysis of COP signals was also conducted using MATLAB 2015a (Mathworks Inc, Natick MA, USA). In summary, both EEG and COP data were carefully filtered, processed, and averaged per participant before statistical analysis.

Statistical Analysis

Statistical analysis was performed on data from the visual occlusion condition only. Data for each participant was represented by one averaged signal for each of EEG and COP for each condition. A general linear model was used to investigate the effect of postural task (standing or sitting) and group (TD or ASD) on the dependent variables for EEG (the latency and amplitudes of P1 and N1) and COP measures (the total excursions in AP direction and ML direction). The interaction terms were retained if p value was considered significant. Post hoc comparisons of group differences were performed by calculating the least square means (LSM) and least square means differences of fixed effects. An alpha level of $p < .05$ was utilized to indicate statistically

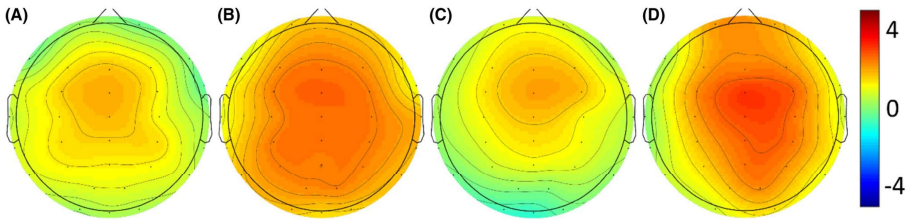

Fig. 3 Grand average of the EEG topographic maps of P1 based on peak latencies for each TD and ASD subject. The power unit for the colour bar is μV^2 and subjected to a $\log_{10}$ transform to normalise the distribution. **a** TD standing, **b** TD sitting, **c** ASD standing, **d** ASD sitting. (Color figure online)

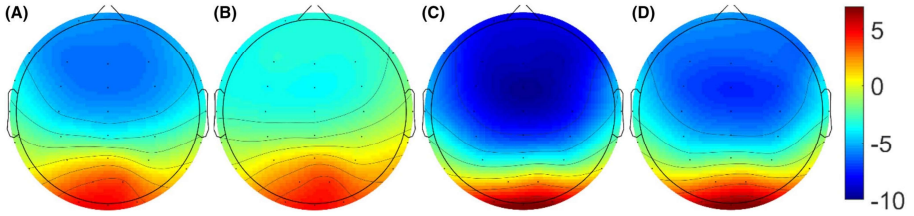

Fig. 4 Grand average of the EEG topographic maps of N1 based on peak latencies for each TD and ASD subject. The power unit for the colour bar is μV^2 and subjected to a $\log_{10}$ transform to normalise the distribution. **a** TD standing, **b** TD sitting, **c** ASD standing, **d** ASD sitting. (Color figure online)

significance in all tests. These statistical analyses were performed using SAS 9.4 (SAS Institute Inc., Cary, NC).

Results

Electroencephalography

The peak amplitudes of the PER components were maximal at the frontal-central recording sites (Figs. 3, 4). The peak amplitude of the P1 component occurred between 50 and 100 ms post visual occlusion (Fig. 5), while the peak amplitude of N1 component occurred between 100 and 150 ms post visual occlusion (Fig. 6). There were no cortical responses following the visual transparent condition during the standing and sitting tasks across TD (Fig. 5) and ASD (Fig. 6). Thus, the rest of the analysis will report only the visual occlusion condition.

The EEG responses to visual occlusion were consistent across both TD and ASD groups and resulted in a P1 component followed by an N1 component at FCz in both standing and sitting (Figs. 5, 6). Only the results for the FCz site are presented here because this was the site,

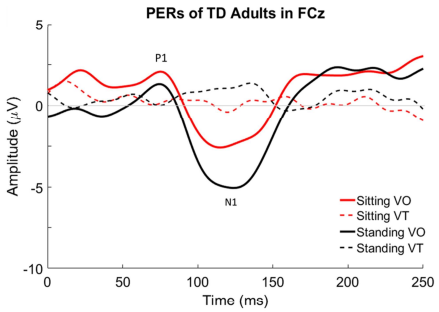

Fig. 5 Grand average of PERs in standing and sitting to visual occlusion (VO) and visual transparent (VT) conditions at the frontal-central site for TD adults

where negativity is maximal (Goh et al. 2017; Marlin et al. 2014; Varghese et al. 2014). There were no significant interactions in both P1 peak latency and the P1 peak amplitude between postural task and group (Table 1). No

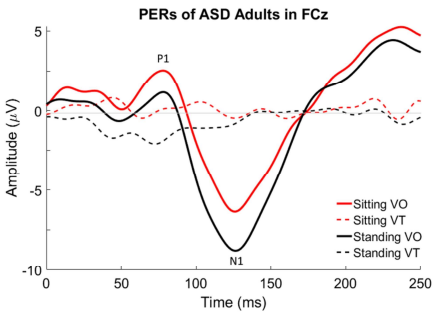

Fig. 6 Grand average of PERs in standing and sitting to visual occlusion (VO) and visual transparent (VT) conditions at the frontal-central site for adults with ASD

main effect of postural task, as well as the group, was observed in both peak latency and peak amplitude of P1 (Table 1).

The general linear model also yielded no postural task by group interaction for the N1 peak latency and the N1 peak amplitude. Main effect analysis demonstrated that the N1 peak latency was not affected by postural task and group (Table 1). However, the main effect of postural task on the N1 amplitude yielded a F ratio of$F(1,48) = 22.25$, $p < .0001$ indicating that the N1 amplitude was significantly greater in the standing task than in the sitting task for both groups (160% greater in TD and 132.7% greater in ASD) (Table 1). In addition, a significant main effect of group for the N1 amplitude was observed ($F(1,48) = 9.29$,$p = .0037$) with greater N1 amplitude in adults with ASD compared to TD adults (160.7% greater in standing and 193.8% greater in sitting) (Table 1).

Centre of Pressure

Visual occlusion resulted in a backwards movement then followed up by a forward movement in the standing task in both TD and ASD group but not in sitting task (Fig. 7). No significant postural task by group interactions was observed for the $TOTEX_{AP}$ and $TOTEX_{ML}$ (Table 1). The main effect of group (TD vs ASD) was significant ($F(1,48) = 7.30$, $p = .0095$) as well as the main effect of posture (standing vs sitting) for $TOTEX_{AP}$ ($F(1,48) = 41.88, p < .0001$), with significantly greater AP displacement in adults with ASD in the standing task (group: 134.0% greater in standing, 182.6% greater in sitting; posture: 315.0% in TD and 231.1% in ASD) (Table 1). The statistical test also demonstrated a significant effect of group ($F(1,48) = 4.60, p = .0371$) and a significant effect of posture ($F(1,48) = 5.08, p = .0288$) for $TOTEX_{ML}$; specifically, post hoc analyses reported greater ML displacement in adults with ASD in the standing task (Fig. 8) (group: 124.4% greater in standing, 130.3% greater in sitting; posture: 132.0% in TD and 126.0% in ASD) (Table 1).

Discussion

This current study investigated the cortical responses which contribute to the processing of visual occlusion during standing and sitting in individuals with ASD and TD adults. Our study found that there was no difference in the latency of the PERs between groups, suggesting no differences in early perturbation processing speed in adults with ASD. However, while the peak amplitudes of the P1 components (indicate the site is FCz rather than occipital) were similar between groups during the postural tasks, peak amplitudes of the N1 component were larger in the ASD group regardless of the postural task. This finding is in contrast to our initial hypothesis where we expected reduced cortical processing of the postural task in people with ASD and suggests that

Table 1 The means and standard deviations of peak latency and peak amplitude of P1 and N1, and the total excursions in the AP direction ($TOTEX_{AP}$) and ML direction ($TOTEX_{ML}$) at FCz site in the standing and sitting task

		Mean (± standard deviation)				Main effects[a]	
		TD Adults		Adults with ASD		Group	Posture
		Standing	Sitting	Standing	Sitting		
Peak latency (ms)	P1	71.69 (±10.91)	73.00 (±6.92)	76.31 (±7.75)	76.31 (±6.10)	0.08 (0.7730)	3.09 (0.0852)
	N1	123.92 (±11.92)	121.92 (±15.45)	125.46 (±12.07)	126.92 (±11.62)	0.01 (0.9401)	0.84 (0.3639)
Peak amplitude (μV)	P1	1.873 (±1.441)	2.560 (±1.462)	1.599 (±1.877)	2.926 (±1.589)	2.66 (0.1096)	0.01 (0.9412)
	N1	−6.176 (±2.052)	−3.859 (±2.346)	−9.927 (±3.671)	−7.480 (±2.924)	9.29 (0.0037)	22.25 (<0.0001)
$TOTEX_{AP}$ (mm)		5.066 (±1.027)	1.608 (±0.932)	6.790 (±3.668)	2.937 (±1.099)	7.30 (0.0095)	41.88 (<0.0001)
$TOTEX_{ML}$ (mm)		1.606 (±0.532)	1.217 (±0.719)	1.998 (±0.800)	1.586 (±0.444)	4.60 (0.0371)	5.08 (0.0288)

[a]The results of the main effects provided are t-score (p value)

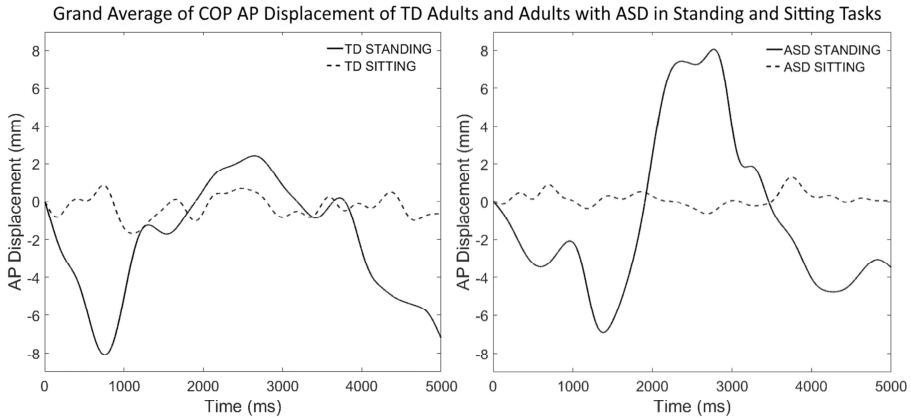

Fig. 7 Grand mean of COP anterior-posterior displacement (mm) of TD adults (left) and adults with ASD (right) in standing and sitting plotted against time (ms) from the onset of visual occlusion. Zero displacement is the starting point of the normalised absolute position

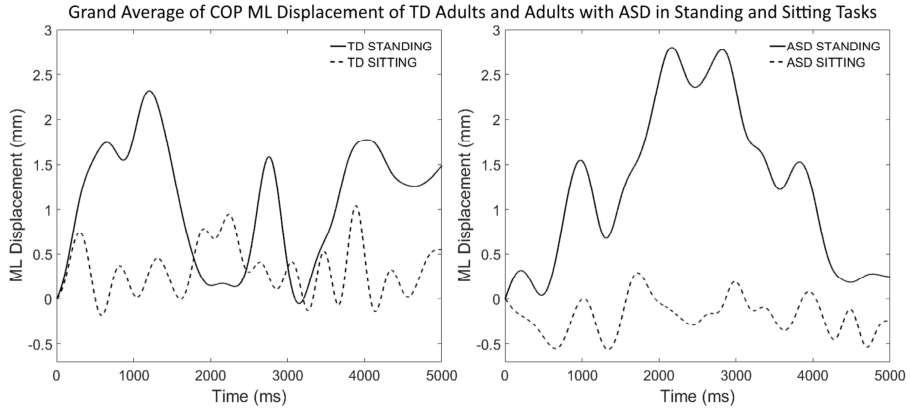

Fig. 8 Grand mean of COP medial-lateral displacement (mm) of TD adults (left) and adults with ASD (right) in standing and sitting plotted against time (ms) from the onset of visual occlusion. Zero displacement is the starting point of the normalised absolute position

while assessment of postural set related to postural demand is typical in ASD, planning of the postural response to visual occlusion utilises more cortical resources in ASD. To the extent of our knowledge, this study is the first to report PERs to visual occlusion in relation to postural stability in adults with ASD.

The PERs (P1 and N1) in response to visual occlusion during the postural tasks were observed in adults with ASD and TD adults. Although the P1 component (at FCz) is suggested to reflect the sensing of postural instability following a perturbation (Adkin et al. 2006; Jacobs and Horak 2007), some studies have reported that the P1 component is often absent (Adkin et al. 2006; Maki and McIlroy 2007). Additionally, the peak latency of the P1 component is reported up to be delayed by 22 ms in older adults compared with young adults reflecting a slowing of processing speed (Duckrow

et al. 1999). In our study, all adults (including TD adults) demonstrated a constant P1 component peaked at 60–80 ms and peak amplitude maximal at the FCz site (Fig. 3); suggesting that the P1 component was generated in the primary sensory cortex (Dietz et al. 1984; Duckrow et al. 1999) and unaffected by ASD. This finding is consistent with other studies which reported no significant differences in latency of P1 despite the variation in magnitude for the perturbations (Adkin et al. 2006; Duckrow et al. 1999). In our study, both P1 latency and magnitude were not different between groups and did not change with postural demand. However, this level of cortical processing does not involve the consequences of postural response.

The N1 component of the PER has been associated with processing the characteristics of a postural perturbation. The peak latency of the N1 component at FCz in this study was also consistent with previous studies as it occurred at 100–150 ms after the onset of visual occlusion (Goh et al. 2017) and postural perturbations (Bogost et al. 2016; Little and Woollacott 2015; Toledo et al. 2016; Varghese et al. 2014). A delay in N1 peak latency has previously been observed in older adults compared with young adults, which was associated with an increase in the central processing time (Toledo et al. 2016). Since the peak latencies of the N1 component were consistent across groups and postural tasks (Figs. 5, 6), this indicates that there were no cognitive processing speed differences in ASD for processing the characteristics of a postural perturbation. This supports the findings of a previous study which suggests processing speed remains intact among individuals with ASD (Wallace et al. 2009a, b). Apart from N1 peak latency, studies have demonstrated the peak amplitude of N1 associated with postural perturbations is influenced by age (Toledo et al. 2016), the predictability (Adkin et al. 2006; Jacobs and Horak 2007; Mochizuki et al. 2008) and size (Staines et al. 2001) of the stimulus, added cognitive tasks (Little and Woollacott 2015; Quant et al. 2004) as well as postural demand (Goh et al. 2017). N1 peak amplitudes in both TD adults and adults with ASD confirmed the peak amplitude of the N1 component varied with postural demand. However, the effect of visual occlusion in the current study produced a larger N1 peak amplitude in adults with ASD compared with TD adults in both postural tasks, inconsistent with our hypotheses. The larger rather than smaller N1 response to visual occlusion indicates that rather than ignoring visual information used for postural control, people with ASD appear to be hypersensitive to a change in visual information.

Sensory impairments have been identified as a key aspect of ASD clinical descriptions. Even though most ASD studies focus on children (Crane et al. 2009; Pfeiffer et al. 2011; Tomchek and Dunn 2007), evidence suggests that impairments in sensory processing persist into adulthood (Bhat et al. 2011; Lim et al. 2017; Minshew et al. 2004; Schall and McDonough 2010). While the N1 component is highly associated with sensory processing, impairments at any stage along the sensory processing pathway could have resulted in an elevated peak amplitude of N1 in adults with ASD. A delay in peak latency and attenuation in peak amplitude of auditory evoked components (i.e. P200 and P300) have been observed in individuals with ASD which suggested deficits in processing auditory information (Bomba and Pang 2004; Magliaro et al. 2010). Moreover, the N1 component of the visual evoked potential to a contrast-reversing checkerboard has been reported to be delayed in latency and of smaller amplitude in children with ASD (Siper et al. 2016). However, the N1 component in our current study reported a consistent peak latency and systematically greater peak amplitude across adults with ASD compared with TD adults.

Generally, there are two types of impairments in sensory responses observed in people with ASD—hypersensitivity (i.e. excessive sensitivity) and hyposensitivity (i.e. below normal sensitivity) (Crane et al. 2009). Hyposensitivity may be reflected in the delayed and attenuated evoked components observed by Siper and colleagues 2016 which included children with ASD of varying levels of cognitive functioning, although sensory sensitivity was not reported in this study. In contrast, our results may be explained by the sensory hypersensitivity experienced by individuals with ASD (Baranek et al. 2007; Baron-Cohen et al. 2009; Kern et al. 2006). The occipital region is primarily responsible for visual information processing and mapping the visual world. Post-hoc analysis was conducted on P1 in the occipital area (Oz) to determine if there was other evidence of hypersensitivity in the visual cortex responses to visual occlusion. The peak latency of occipital P1 was similar to the frontal central N1 reflecting parallel processing of the visual information (Figs. 9, 10). However, a significant main effect of group

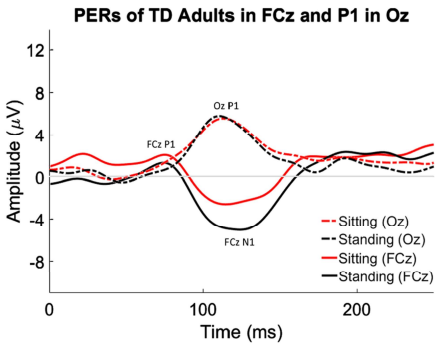

Fig. 9 Grand average of PERs at the frontal site and P1 at the occipital site in standing and sitting to visual occlusion (VO) in TD adults

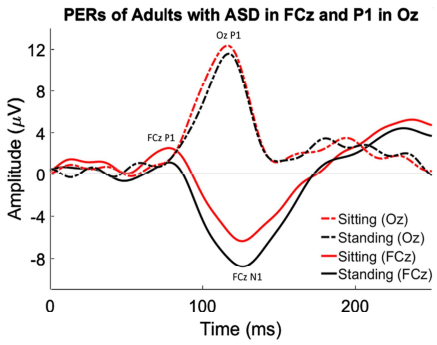

Fig. 10 Grand average of PERs at the frontal site and P1 at the occipital site in standing and sitting to visual occlusion (VO) in ASD adults

for occipital P1 component (occurred at 100–150 ms) was observed ($F(1,48) = 7.98, p = .0069$) with greater occipital P1 peak amplitude in adults with ASD than TD adults. In addition, the correlation analysis revealed a positive correlation between the amplitude of N1 at the frontal-central site and the amplitude of P1 at the occipital site across both TD adults and adults with ASD ($r = 0.5066$) (Figs. 9, 10). This finding suggests that there was a generalized over excitation across the cortex in response to sudden visual occlusion and may reflect excitation/inhibition imbalances in the neural system for individuals with ASD (Cline 2005; Orekhova et al. 2008). Additionally, this finding is also paralleled with a study suggesting the excitation at the posterior of the brain was accounted for the hyperactivation in individuals with ASD (Belmonte et al. 2004a). Therefore, the greater N1 peak amplitude in adults with ASD is likely affected by hypersensitivity to sensory stimuli. Hypersensitivity in this situation may reflect a problem of adaptive orientation to behaviourally relevant sensory information (Keehn et al. 2013). In this situation, whereas the TD adults may selectively attend to a smaller region of the visual field, information from the whole visual field may be processed in the ASD group resulting in an exaggerated response to occlusion. Further research is required to test this hypothesis.

Postural sway induced by the visual occlusion condition was greater in adults with ASD than TD adults as hypothesized. The total length of the postural sway path for both groups was assessed in two directions, AP (Fig. 7) and ML (Fig. 8). The findings in the current study were consistent with other studies reporting that individuals with ASD exhibited a greater amount of AP and ML sway compared to TD individuals with and without visual input (Doumas et al. 2016; Goh et al. 2016). Even though most studies reported distinct postural AP and ML sway in children with ASD (Fournier et al. 2010; Memari et al. 2013; Molloy et al. 2003; Stins et al. 2015a), it is suggested that age does not improve postural control in children with ASD (Fournier et al. 2010; Memari et al. 2013). Moreover, evidence from the empirical studies suggests that abnormal postural control resulted from poor sensory integration (Marco et al. 2011). Our findings on the postural way in ASD accompanied by the modulation of N1 peak amplitude support a hypothesis that abnormal postural sway in ASD could arise as a result of sensory hypersensitivity (Baron-Cohen et al. 2009). Despite the fact that most postural control studies in individuals with ASD were reported in the static standing condition (Lim et al. 2017), to the best our knowledge, there is no study that measures postural control of this population in the static sitting condition. Interestingly, postural AP and ML sway in the sitting condition was significantly different from standing in both groups, and adults with ASD presented greater AP and ML sway than TD adults in both conditions. Therefore, this suggests that greater postural AP and ML sway in individuals with ASD can be extended to the sitting condition and hence may be unrelated to postural demand. Increased postural sway in people with ASD may reflect exaggerated postural responses to sensory information perhaps related to problems in adaptive narrowing of visual attention to relevant cues for maintaining resilient postural stability. This hypothesis requires further research.

Since this is the first study that compares PERs to visual perturbation under varied postural stability conditions in adults with ASD and TD adults, there are limitations in our research to be considered. Firstly, the sample size ($n = 13$) for each group might not be enough to be considered representative of all those with ASD, particularly as it excluded those with intellectual disability. However, the sample size is consistent with similar research in the area (Bogost et al. 2016; Marlin et al. 2014; Mochizuki et al. 2010; Varghese et al. 2014). Additionally, the risk of type 2 error is higher where sample size is small. Secondly, despite the spatial resolution of the EEG scalp surface recording being limited, this issue can be addressed using advanced EEG source localization techniques to better understand the generation and propagation of the EEG source. A review on EEG source localization technique is described by Grech et al. (2008) and this will be explored in future work. It is also noteworthy to mention that the amplitude differences are affected by scalp thickness, scalp resistance and other conductance properties of the skull. These elements were not assessed in the current study. However, there is no evidence that support people with ASD have differences in scalp thickness and resistance. The best explanation for the difference in the EEG amplitude is the cortical activity differences. These cortical activity differences may occur in many cortical locations associated with many tasks, we are just reporting on one cortical location—FCz.

Sensory information is constantly processed in our brain to generate appropriate motor responses. Impairments in the sensory information processing can severely impact the social, emotional and behavioural responsiveness of the individual (Baker et al. 2008) as commonly observed in individuals with ASD (Crane et al. 2009; Pfeiffer et al. 2011; Tomchek and Dunn 2007). In this study, the PERs which were maximal at the frontal-central site were evident in both adults with ASD and TD adults during the two postural tasks and moderated by postural demand, indicating a relationship to postural control (Marlin et al. 2014). There were no significant differences in the P1 component at the same location, indicating that sensing instability (P1) is intact in adults with ASD. The peak latency of the N1 component was similar for both groups suggesting the cognitive speed of both groups was similar. Interestingly, the peak amplitude of the N1 component was larger in adults with ASD than TD adults. It is hypothesised that sensory hypersensitivity could play a role in the elevation of N1 as it is often experienced in adults with ASD (Baranek et al. 2007; Baron-Cohen et al. 2009; Kern et al. 2006). This hypothesis was supported by post hoc analysis on posterior of the brain that has been implicated in posterior hyperactivation in individuals with ASD (Belmonte et al. 2004b) and requires independent validation in future studies. Apart from PERs, the postural sway in AP and ML directions were consistent with previous findings (Doumas et al. 2016; Goh et al. 2016) which reported greater sway in adults with ASD compared with TD adults. In summary, current accumulated evidence suggests that the assessment of postural demand is intact in adults with ASD, but the postural responses in this population utilise more cortical resources to integrate and interpret visual perturbations, including those related to postural control. The results from this study provide insights into the cortical responses which contribute to sensory integration in individuals with ASD. Further investigations into the neural connections of individuals with ASD and how these may result in hyper and hyposensitivity would be relevant.

Conclusions

Adults with ASD detected the VO event (P1) similarly to the TD adults. The difference in N1 between sitting and standing indicates an equivalent ability to detect changes in the postural set. This finding supports evidence that the processing of proprioceptive information is intact in adults with ASD (Haswell et al. 2009; Morris et al. 2015). However, the postural and cortical responses to VO in adults with ASD were larger compared with TD adults regardless of posture. These findings support the role of the N1 as the cortical signal for the automatic postural response with appropriate latency, relationship to postural set and postural sway. A generalised increase in activation in the posterior brain supports the hypothesis that postural instability in ASD is due to hyper-responsiveness to visual/sensory information rather than inadequate motor responses. The study provides the first reported evidence indicating that postural response utilises more cortical resources to maintain balance upon visual perturbation in individuals with ASD. Further investigations are needed to explore the neurophysiological responses of individuals with ASD and provide insights to develop better clinical outcomes.

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Author Contributions KLG, SM and TT designed the experiment. KLG performed the experiments, analysed the data and wrote the Material and Methods sections. KLG, SM and RP performed the statistical analyses. AR contributed with expertise in sensory systems and balance disorder throughout the design and in writing the introduction and discussion/implementations sections together with KLG, SM and TT. The final editing was completed jointly by all five authors. The order of authors has been agreed upon among them and the corresponding author, KLG has taken due care and full responsibility of this matter.

Compliance with Ethical Standards

Conflict of interest All authors declare that they have no conflict of interest.

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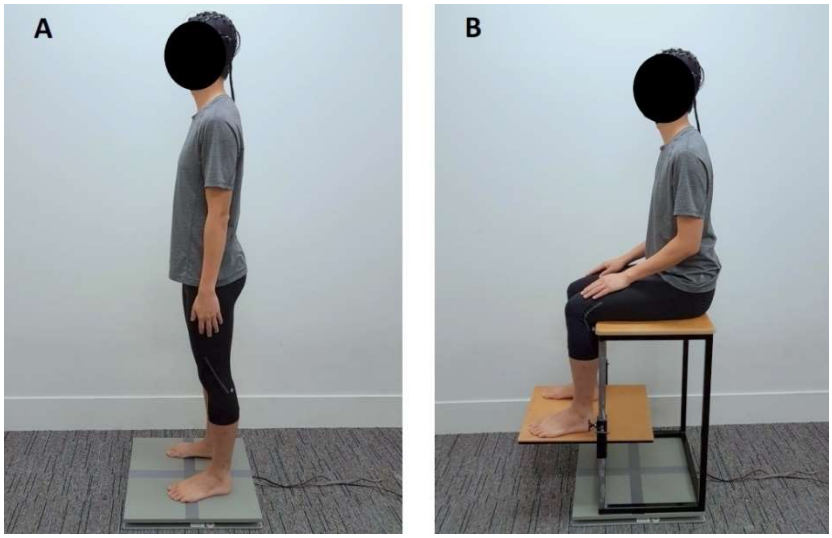
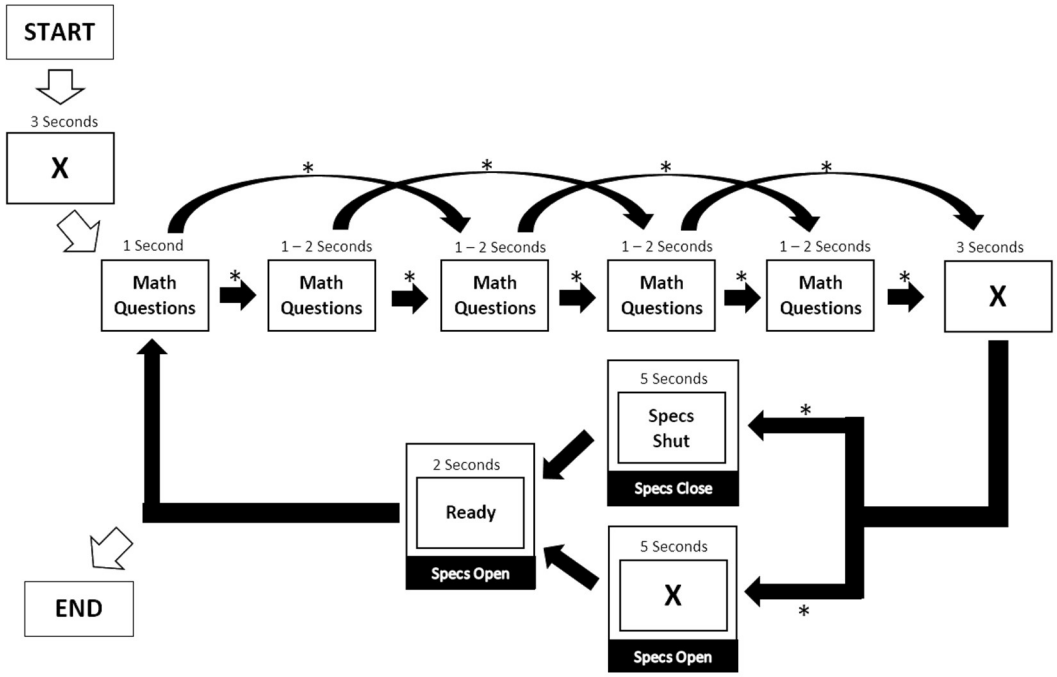
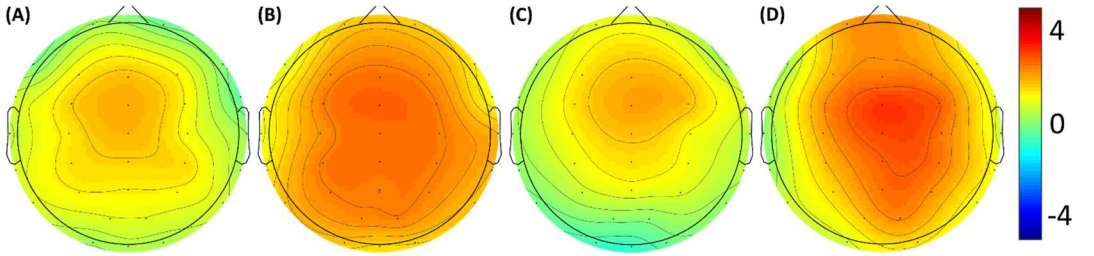


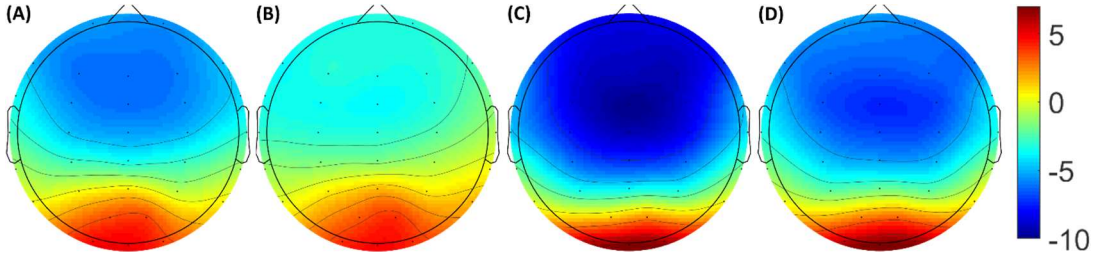
Supplementary Figure 6.1 Experimental Setup for testing: (A) Standing (B) Sitting



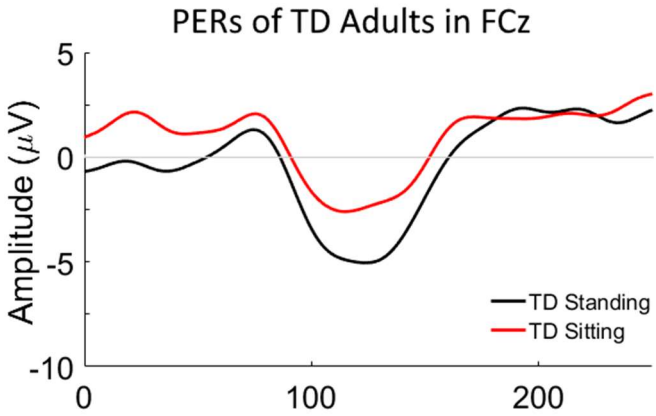
Supplementary Figure 6.2 Presentation of the experiment for a single trial. * denotes 50% of occurrence.



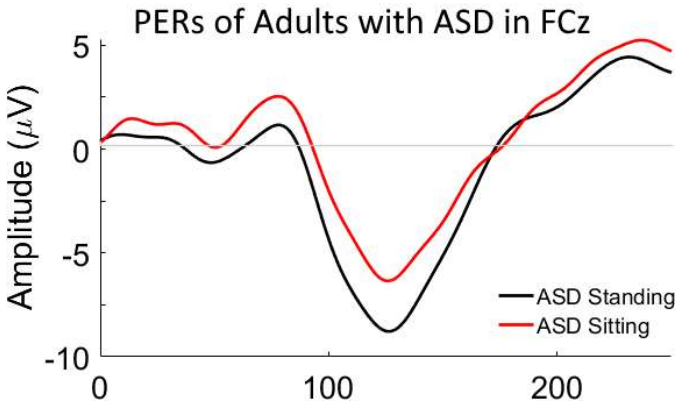
Supplementary Figure 6.3 Grand average of the EEG topographic maps of P1 based on peak latencies for each TD and ASD subject. The power unit for the *colour bar* is μV^2 and subjected to a log_{10} transform to normalise the distribution. (A) TD Standing (B) TD Sitting (C) ASD Standing (D) ASD Sitting



Supplementary Figure 6.4 Grand average of the EEG topographic maps of N1 based on peak latencies for each TD and ASD subject. The power unit for the *colour bar* is μV^2 and subjected to a log_{10} transform to normalise the distribution. (A) TD Standing (B) TD Sitting (C) ASD Standing (D) ASD Sitting



Supplementary Figure 6.5 Grand average of PERs in standing and sitting at the frontal-central site for TD adults.

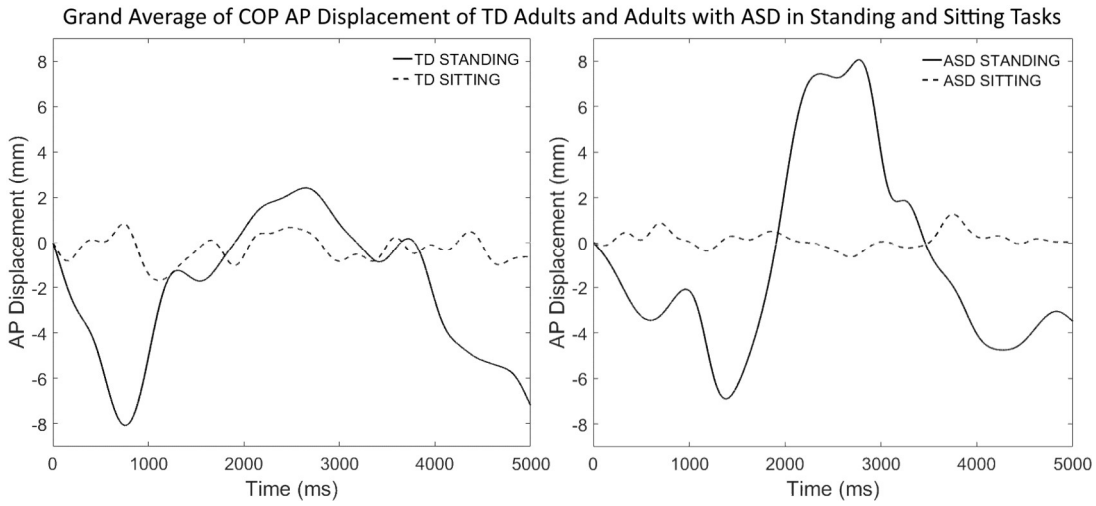


Supplementary Figure 6.6 Grand average of PERs in standing and sitting at the frontal-central site for adults with ASD.

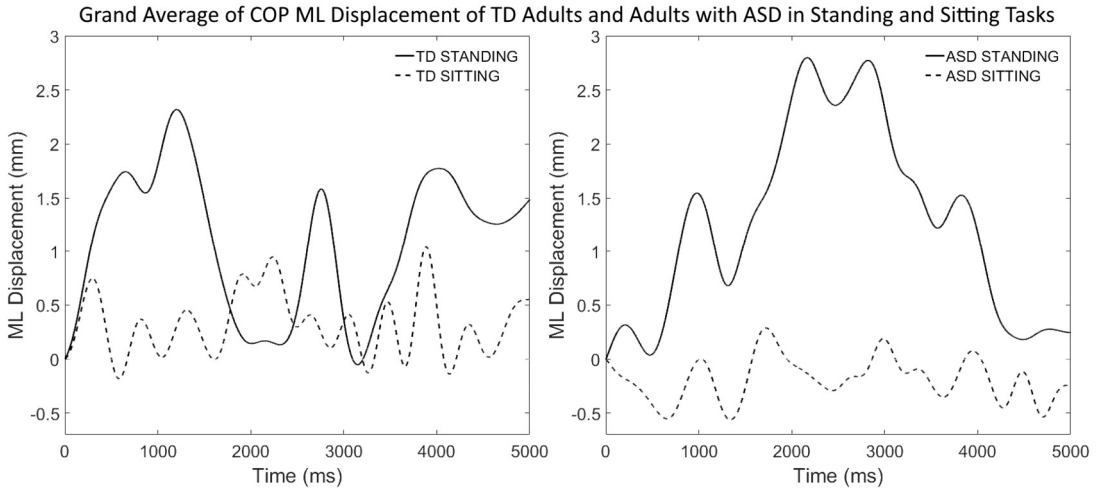
Supplementary Table 6.1 The estimated least square means of peak latency and peak amplitude of P1 and N1 at FCz site in the standing and sitting task.

		Estimated Least Square Means				Main Effects*	
		TD Adults		Adults with ASD		Group	Posture
		Standing	Sitting	Standing	Sitting		
Peak latency (ms)	P1	71.70	73.00	76.31	76.31	0.08 (.7730)	3.09 (.0852)
	N1	123.92	121.92	125.46	126.92	0.01 (.9401)	0.84 (.3639)
Peak Amplitude (μV)	P1	1.873	2.560	1.599	2.926	2.66 (.1096)	0.01 (.9412)
	N1	-6.176	-3.859	-9.927	-7.480	9.29 (.0037)	22.25 (<.0001)
$TOTEX_{AP}$ (mm)		5.066	1.608	6.790	2.937	7.30 (.0095)	41.88 (<.0001)
$TOTEX_{ML}$ (mm)		1.606	1.217	1.998	1.586	4.60 (.0371)	5.08 (.0288)

*The results of the main effects provided are t-score (p-value).



Supplementary Figure 6.7 Grand mean of COP anterior-posterior displacement (mm) of TD adults (Left) and adults with ASD (Right) in standing and sitting plotted against time (ms) from the onset of visual occlusion. Zero displacement is the starting point of the normalised absolute position.



Supplementary Figure 6.8 Grand mean of COP medial-lateral displacement (mm) of TD adults (Left) and adults with ASD (Right) in standing and sitting plotted against time (ms) from the onset of visual occlusion. Zero displacement is the starting point of the normalised absolute position.

Chapter 7

Typically developed adults and adults with Autism Spectrum Disorder classification using centre of pressure and electroencephalography signals

7.1 Introduction

Autism spectrum disorders (ASD) are a group of neurodevelopmental disorders characterised by impairments in social interaction and communication and restricted or repetitive behaviour (American Psychiatric Association, 2013). The population prevalence of ASD has been estimated to be 2.64% in South Korea (Kim et al., 2011) whereas, in the United States, the prevalence rate for ASD has been reported as 0.91% (Lord & Cook, 2013). The prevalence rate for ASD has increased over the years and the lifetime cost of raising a child with ASD is currently between USD 1.4 million to $2.4 million (Buescher, Cidav, Knapp, & Mandell, 2014). Thus, ASD results in significant financial and personal costs to both families and communities.

Due to the rise in the prevalence of ASD, there is an increased demand for ASD diagnostic assessments. In fact, to date, 131 ASD diagnostic assessments are available (Falkmer, Anderson, Falkmer, & Horlin, 2013). However, most of these tools were developed for the use in children with ASD (McConachie et al., 2015) and may therefore not be adequate to identify ASD in adult populations. Furthermore, many assessment tools lack an evidence base of high quality-independent studies (Falkmer et al., 2013). The combination of Autism Diagnostic Interview-Revised (ADI-R) and Autism Diagnostic Observation Schedule (ADOS) are currently considered the ‘gold standard’ of ASD diagnosis, with a large evidence base demonstrating these tools have the highest sensitivity and specificity (Falkmer et al., 2013). Even so, both ADOS and

ADI-R are still expensive, time-consuming and require extensive training to use (Skellern, McDowell, & Schluter, 2005). As a result, there is a strong call to enhance or develop more effective means to accurately diagnosis ASD.

While impairments in communication and social interaction are the core symptoms of ASD, emerging studies have demonstrated sensory impairments in this population (American Psychiatric Association, 2013; Fournier et al., 2010; Radonovich, Fournier, & Hass, 2013; Travers, Powell, Klinger, & Klinger, 2013). Postural instability can be the result of a sensory impairment where sensory impairment is often accompanied by an increase of postural sway. A recent systematic review reported that anterior-posterior (AP) sway and medial-lateral (ML) sway are larger in adults with ASD than typically developed (TD) adults in standing tasks (Lim et al., 2017), particularly the total excursion of the AP and ML sway directions (Graham et al., 2015; Stins et al., 2015). Studies using the centre of pressure (COP) range in the AP direction and ML direction, sway area and sway velocity had shown that children with ASD have a different pattern in postural control when compared with TD children (C.-H. Chang, Wade, Stoffregen, Hsu, & Pan, 2010; Fournier et al., 2014; Fournier et al., 2010; Funahashi, Karashima, & Hoshiyama, 2014; Memari et al., 2013). While a force plate is often used by researchers to measure postural steadiness, only a limited array of COP measurements derived from the force plate are commonly used in the analysis. Moreover, the COP data recorded from the force plate can be measured in the time domain, frequency domain or hybrid of both domains (Carpenter et al., 2001; T. Prieto et al., 1996; van der Kooij, Campbell, & Carpenter, 2011). Thus, it may be important to include various COP measurements from the different domains to provide a comprehensive assessments of postural control in ASD.

Extensive studies have been conducted to investigate the COP behavioural response of individuals with ASD, but very few studies have specifically examined sensorimotor integration in this disorder. A recent systematic review suggests ASD is associated with altered patterns of connectivity, particularly long-range underconnectivity (O'Reilly, Lewis, & Elsabbagh, 2017). As multisensory integration

relies on brain connectivity to encode information (Harris & Mrsic-Flogel, 2013), it may be a problem for individuals with ASD. Moreover, it has been suggested that the cerebellum is an important structure to understand ASD and sensorimotor control (Allen, Buxton, Wong, & Courchesne, 1997). At this point, electroencephalography (EEG) provides a unique opportunity to provide further insights to these hypotheses. EEG provides an inexpensive, safe and non-invasive way of investigating sensorimotor process in the brain. The time-frequency analysis of the EEG signals characterized by delta (0.5 – 4 Hz), theta (4 – 8 Hz), alpha (8 – 13 Hz), beta (13 – 30 Hz) and gamma (> 31 Hz) could provide vital information assisting to elucidate the nature of sensorimotor integration atypicality in this disorder. Thus, it may be valuable to investigate the rhythm features derived from EEG to assist in ASD assessment.

The aim of this study was to classify TD adults and adults diagnosed with ASD based on COP and EEG measurements during quiet standing. Data mining tools such as decision tree (Quinlan, 1993), naïve Bayes (Lewis, 1998), support vector machine (Platt, 1999), K-nearest neighbour (Altman, 1992) as referenced in (Wu et al., 2008) and random forest (Breiman, 2001), multilayer perceptron neural network (Haykin, 1998) are tools for the classification of data sets. In this study, the importance of the EEG and COP features were ranked from the forests of randomized trees (Louppe, Wehenkel, Sutera, & Geurts, 2013) and the classification results were presented in area under receiver operator characteristic (AUC).

7.2 Methods

7.2.1 Participants

Thirty-two male adults (16 adults with ASD, age=25.5 ± 3.97 years, height=179.56 ± 7.69 cm, weight=77.63 ± 12.86 kg; 16 TD adults, age=25.6 ± 5.69 years, height=176.09 ± 5.96 cm, weight=72.95 ± 10.28 kg) were recruited through service providers, research institutions and social media in Perth, Western Australia. Adults with ASD were eligible for inclusion if they presented with a diagnosis of Autism

Spectrum Disorder according to the Diagnostic and Statistical Manual for Mental Disorders 5th Edition (American Psychiatric Association, 2013) or equivalent diagnosis of Autism Spectrum Disorder, Asperger Syndrome or Pervasive Developmental Disorder according to the DSM-4-TR (American Psychiatric Association, 2000). Adults with ASD and TD adults were excluded if there were prescription medication for the past three months, history of severe brain injury or acute mental health illness, and intellectual disability. TD adults were excluded if there were any self-reported neurological or musculoskeletal deficits that may have influence postural control. The TD control group was matched to the ASD group on chronological age (p=.9430), height (p=.1264), weight (p=.3754) and gender. This study was approved by Curtin University Human Research Ethics Committee with written informed consent from all subjects. All subjects gave written informed consent in accordance with the Declaration of Helsinki.

7.2.2 Data Acquisition

Electroencephalography (EEG) signals were recorded using a 40 channel Ag/AgCl electrode cap (Neuroscan, El Paso, TX, USA) based on the International 10-20 System. The impedance of all channels was maintained below 5kΩ, and all channels were referenced to the average of the electrode A1 and A2 located on the left and right mastoid with a forehead ground. Additional electrodes were placed above and below of the left eye, and on the outer canthus of each eye to monitor electrooculogram (EOG) signals. Both EEG and EOG signals were measured in a monopolar mode with a sampling rate of 1000 Hz

Centre of Pressure (COP) data was obtained using AMTI AccuGait portable force platform (Advanced Mechanical Technology Inc., Watertown MA, USA) and recorded using custom made program written in LabView (National Instruments Corporations, Austin TX, USA). The COP data was recorded with a sampling rate of 2000 Hz.

7.2.3 Experimental Protocol

Participants attended a single session where they undertook a set of experimental trials in quiet standing. Potential candidates were first familiarized with the procedure and were provided with the opportunity to ask any questions before participation. Once the written informed consent was completed and it was verified that the participants met inclusion criteria, the height and weight of the participants were then measured. This paper reports on the baseline assessment of quiet standing which occurred at the start of the session.

The participants' task was to focus on the computer screen in front of them. Participants were informed to stand with their feet comfortably shoulder-width apart with hands relaxed alongside their body. Next, the participants were instructed to keep their eyes open throughout the experiment and to look directly forward. The duration of each condition recorded was 75 seconds. Additional sets were conducted if there were suspicions of unwanted signals from the eyes movements, blink artefacts or muscle activity that may contaminate the data.

7.2.4 Data Processing, Parameters Settings and Analysis

Subsequent processing was completed using MATLAB 2015a (Mathworks Inc, Natick MA, USA) and Scikit-learn (Pedregosa et al., 2011). The COP and EEG analyses were performed by omitting the first 15 seconds to avoid the immediate responses of the participants. The sample size was expanded by generating more samples with shorter duration. Thus, there were 5 seconds, 10 seconds and 15 seconds sampling duration datasets with no overlapping. Sampling durations that exceeded 15 seconds were excluded due to small sample size for classification. The details of the datasets are shown in Table 7.1.

EEG signals were initially band-pass filtered using second order zero phase 0.5 Hz elliptic high-pass filter and eighth order zero phase 40 Hz elliptic low-pass filter. After that, independent component analysis with reference (ICA-R) was performed on the EEG signals to remove ocular artefact (Lee et al., 2016). ICA-R is a technique that

extracts the signal-of-interest directly through the guidance of a reference signal (i.e. electrooculography reference). The total length of the EEG was 60 seconds per participant and was segmented into 5 seconds, 10 seconds and 15 seconds for power spectrum analysis. For each channel, the power of the EEG delta (0.5 – 4 Hz), theta (4 – 8 Hz), alpha (8 – 13 Hz), beta (13 – 30 Hz) and gamma (> 31 Hz) were computed with the frequency resolution of 1 Hz and no overlapping. Since the EEG has 34 individual channels, several individual channels were averaged to increase the signal to noise ratio. Thus, there were 10 regions of interest such as frontal (F3, FZ, F4), frontal-central (FC3, FCZ, FC4), central (C3, CZ, C4), central-parietal (CP3, CPZ, CP4), parietal (P3, PZ, P4), parietal-occipital (PO1, PO2), occipital (O1, OZ, O2), frontal-temporal (FT9, FT7, FT9, FT10), temporal (T3, T4, T5, T6) and temporal-parietal (TP7, TP8). The average of power values over the electrodes were computed for the ROIs.

Table 7.1 Datasets with various sample size and duration.

Sampling Period (seconds)	TD samples	ASD samples	Total samples
5	192	192	384
10	96	96	192
15	64	64	128

The centre of pressure (COP) data in the anterior-posterior (AP) and medial-lateral (ML) directions was down-sampled to 1000 Hz to match the EEG sampling rate followed by the low-pass filtered at 5 Hz. Since the absolute positions of the participants standing on the force platform were not controlled, the linear trend of AP and ML time series were removed before analysis. Similar to EEG signals, the total length of the COP was segmented into 5 seconds, 10 seconds and 15 seconds. Subsequently, the resultant distance (RD) which was the composite measure of both the AP and ML in time series was computed. The AP, ML, and RD time series were quantified into the following measures: mean distance (RD, AP, ML), sway path (AP, ML, RD), standard deviation (AP, ML, RD), amplitude of COP displacement (AP, ML,

RD), total excursion (RD, AP, ML), max distance (AP, ML, RD) , area (95% of COP data, 95% confidence circle area, 95% confidence ellipse area), sway area, mean frequency (AP, ML, RD), fractal dimension (confidence circle, confidence ellipse) and frequency domain measures such as total power (AP, ML, RD), mean power frequency (AP, ML, RD), peak frequency (AP, ML, RD), 50% power frequency (AP, ML, RD), 95% power frequency (AP, ML, RD), centroid frequency (AP, ML, RD) and frequency dispersion (AP, ML, RD). For the details of the time and frequency domain measures can refer to Prieto et al. (T. Prieto et al., 1996). The frequency domain measures were calculated from range 0.15 Hz to 5.0 Hz as in references (T. Prieto et al., 1996; van der Kooij et al., 2011). The power spectral density of AP, ML and RD directions was computed using Welch's periodogram technique by dividing the data into seven segments with 50% overlap. See Appendix C for more details of the EEG and COP features. The distribution of the feature vectors is shown in Table 7.2.

Table 7.2 The distribution of feature vectors.

Notation	Feature Set	No. of Features
A	EEG	50
B	COP	48
C	EEG + COP	98

Top data mining tools such as decision tree, naïve Bayes, support vector machine and k-nearest neighbour as recommended by Wu et al. (Wu et al., 2008) and random forest and multilayered perceptron neural network were used for comparison and classification in this chapter. A brief explanation and parameters settings of the algorithms were written below.

Decision tree decided the target class of a new sample based on selected features from available data using the concept of information entropy. The nodes of the tree were the attributes, each branch of the tree represented a possible decision, and the end nodes or leaves were the classes. Pruning was used to avoid overfitting the training data. Random forest worked by constructing multiple decision trees on various sub-samples of the datasets and output the class that appeared most often or mean

predictions of the decision trees. In the experiment, the random forest consisted of 100 trees.

The third classifier was the naïve Bayes classifier which is based on Bayes theorem with strong independent assumptions between features. The fourth classifier, multilayered perceptron neural network, is a non-linear feed-forward network model which mapped a set of inputs X onto a set of outputs y using multi weights connections. The network was trained by updating the weight and bias value using stochastic gradient-based optimizer (Kingma & Ba, 2014). One hidden layer was used in this chapter, and the number of hidden neurons was 100. The learning rate was set to 0.001, the momentum was set to 0.9 in each weight updating and the maximum number of epoch for each training time was set to 200.

Support vector machine was used to discriminate a set of high-dimension features using one or sets of hyperplanes that gave the largest minimum distance to separates all data points among classes. Radial basis function was used as a kernel for classification, and the kernel coefficient was set as 2.0 (default). Lastly, K-nearest neighbour was an instance-based learning algorithm that stored all available data points and classified the new data points based on similarity measure such as distance. There was no distance weighting for the setting, and Euclidean distance was used to search for the nearest neighbour.

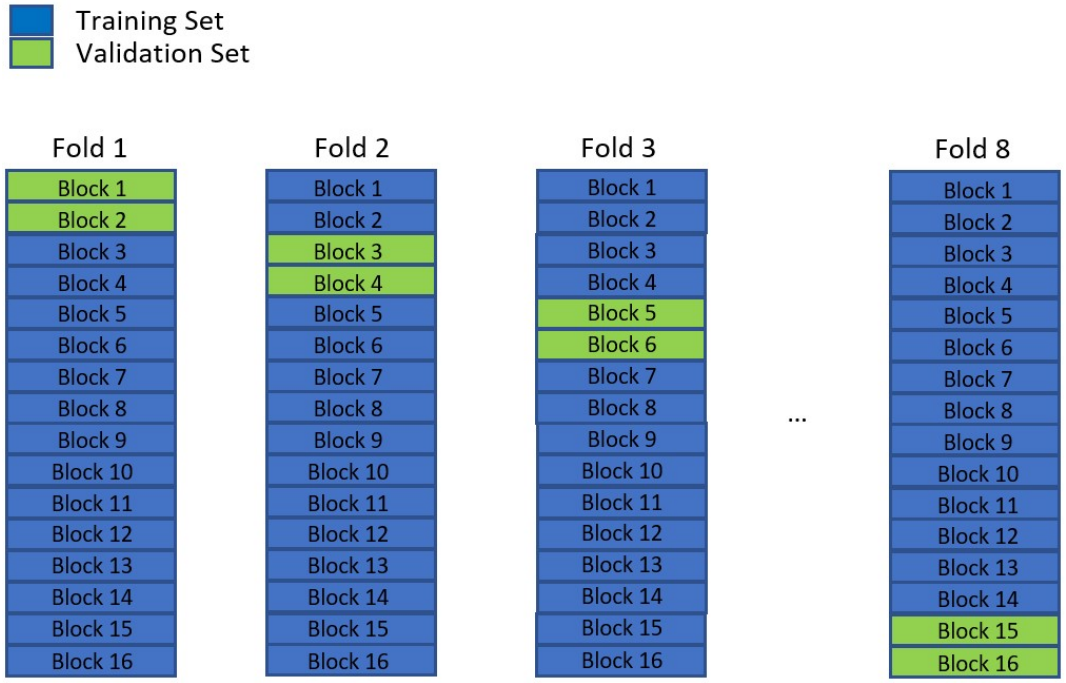


Figure 7.1 Diagram of k-fold cross validation with $k = 8$

Cross validation by participants was used to assess how accurately a model performed in practice and evaluated the performance of the algorithms. Since the 60 seconds COP and EEG durations of a participant was divided into 12 segments, 8-fold cross validation by participants was proposed to avoid the machine learning model training and predicting on segments from the same participant. Figure 7.1 denotes the diagram of the 8-fold cross validation, and each block represents all 12 segments of COP or EEG or both feature sets of the TD adult and adult with ASD. For example in Fold 1, the training set was completed on Block 3 to Block 16 (3rd TD adult to 16th TD adult and 3rd adult with ASD to 16th adult with ASD) and Block 1 and 2 (1st TD adult, 1st adult with ASD, 2nd TD adult and 2nd adult with ASD) were used for validation. Cross validation then averaged all the predictive measures to derive an accurate estimate of model prediction performance and evaluation.

Unlike precision and recall that depends on the particular threshold, the area under the receiver operating characteristic (AUC) was determined by plotting true positive rate vs. the false positive rate in various threshold value. Thus, AUC was emphasized to measure the performance because it does not depend on any threshold. The

randomized forests of trees were used to retrieve the importance of COP and EEG features (Louppe et al., 2013). The importances of the features indicate how useful or valuable each feature was in the construction of decision trees within the randomized forest. If a feature often appears across the decision trees within the random forest, the importance score of the feature will be relatively higher to those features that seldom appear.

7.3 Results

In this section, the AUC results on the datasets were summarized in Table 7.3. The results were reported based on the EEG features (*Feature Set A*), COP features (*Feature Set B*) and both EEG and COP features (*Feature Set C*). The highest AUC results were 0.858 in 5 seconds sampling period dataset, 0.869 in 10 seconds sampling period dataset and 0.873 in 15 seconds sampling period dataset. The best performances were all achieved by random forest (highlighted in bold).

Table 7.3 The AUC classification results of 5, 10 and 15 seconds sampling period datasets.

Sampling Period		5 seconds			10 seconds			15 seconds		
Feature Set		*A*	*B*	*C*	*A*	*B*	*C*	*A*	*B*	*C*
Model	*DT*	0.698	0.634	0.716	0.694	0.671	0.749	0.767	0.681	0.803
	RF	**0.789**	**0.692**	**0.858**	**0.823**	**0.720**	**0.869**	**0.825**	**0.696**	**0.873**
	NB	0.766	0.682	0.813	0.772	0.718	0.832	0.759	0.678	0.856
	NN	0.685	0.578	0.651	0.596	0.636	0.785	0.687	0.594	0.752
	SVM	0.704	0.624	0.661	0.675	0.613	0.692	0.727	0.614	0.720
	KNN	0.618	0.552	0.643	0.702	0.559	0.702	0.654	0.564	0.665

**DT – Decision Tree, RF – Random Forest, NB – Naïve Bayes, NN – Neural Network, SVM – Support Vector Machine, KNN – k-Nearest Neighbour*

Next, the randomized forests of trees were used to evaluate and rank the importance of the COP and EEG features (Louppe et al., 2013). The EEG and COP features of the 15 seconds sampling period dataset were ranked as the dataset had reported the highest AUC results among the three datasets (Figure 7.2 and Figure 7.3).

The delta rhythm in the frontal-central region (*FC-delta*) was the most significant feature to classify TD adults and adults with ASD during quiet stance, followed by the theta rhythm in the frontal-central region (*FC-theta*) whereas the total excursion in ML directions ($TOTEX_{ML}$) was the most significant feature to classify the two groups. In addition, the top 40 features from the combination of EEG and COP features were demonstrated in Figure 7.4. The abbreviations and acronyms were used in the figures (refer to Acronyms section for the full term).

Since random forest had achieved the highest AUC in Table 7.3, the model was used again for prediction by gradually adding in the ranked features from top 2 features to a total of 98 features from Figure 7.4. Figure 7.5 demonstrated the AUC results based on the ranked features. The highest AUC results were 0.9023 with 71 features in 5 seconds dataset, 0.9030 with 52 features in 10 seconds dataset and 0.9143 with 57

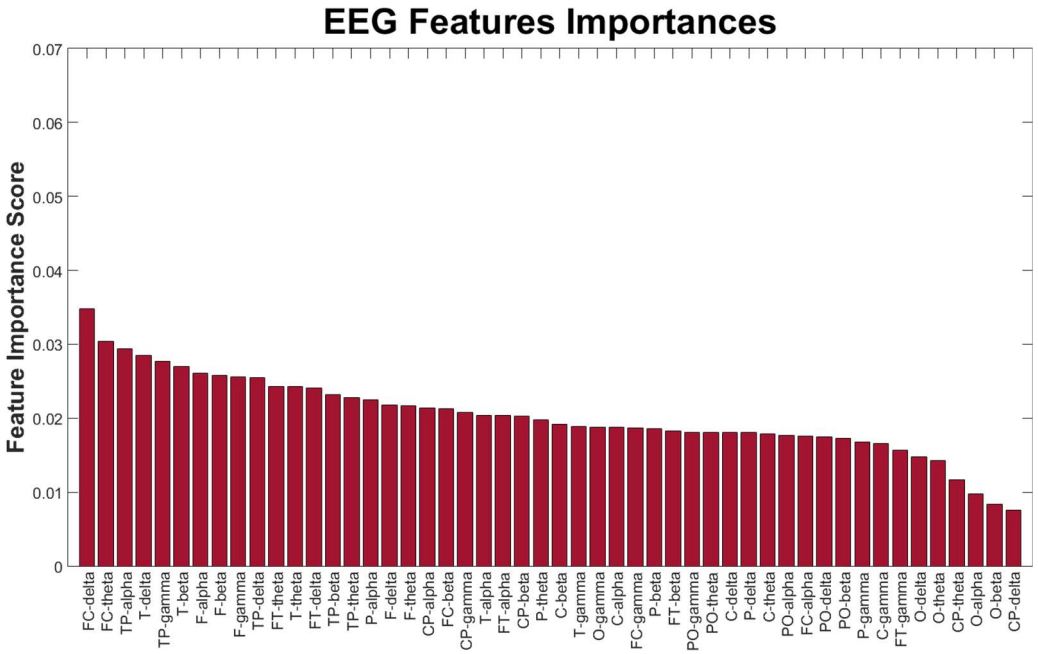


Figure 7.2 The feature importance score of all EEG features.

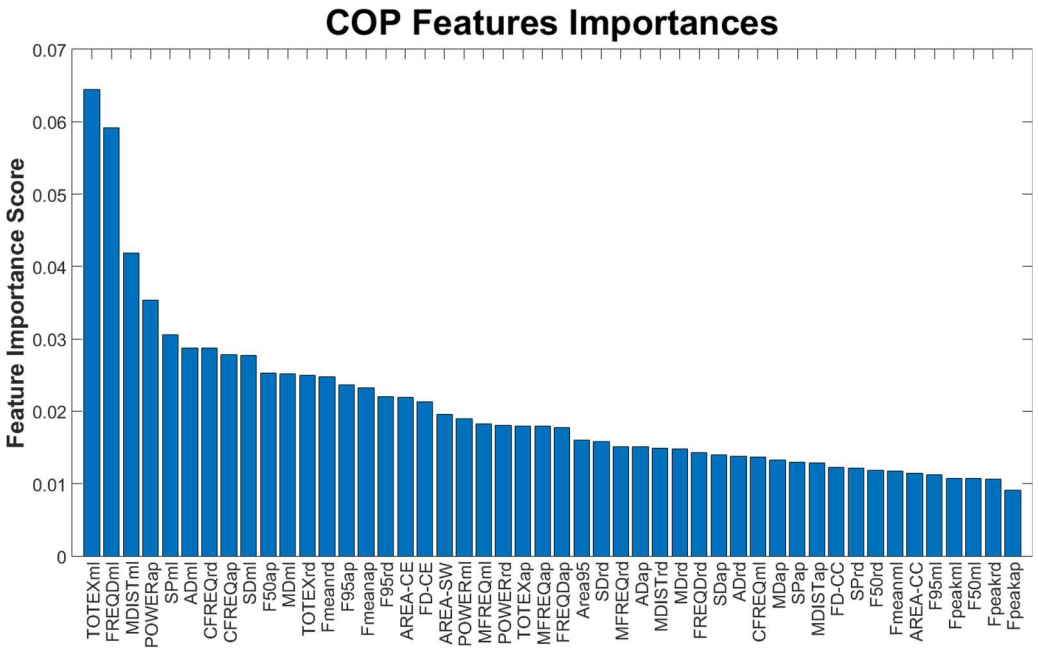


Figure 7.3 The feature importance score of all COP features.

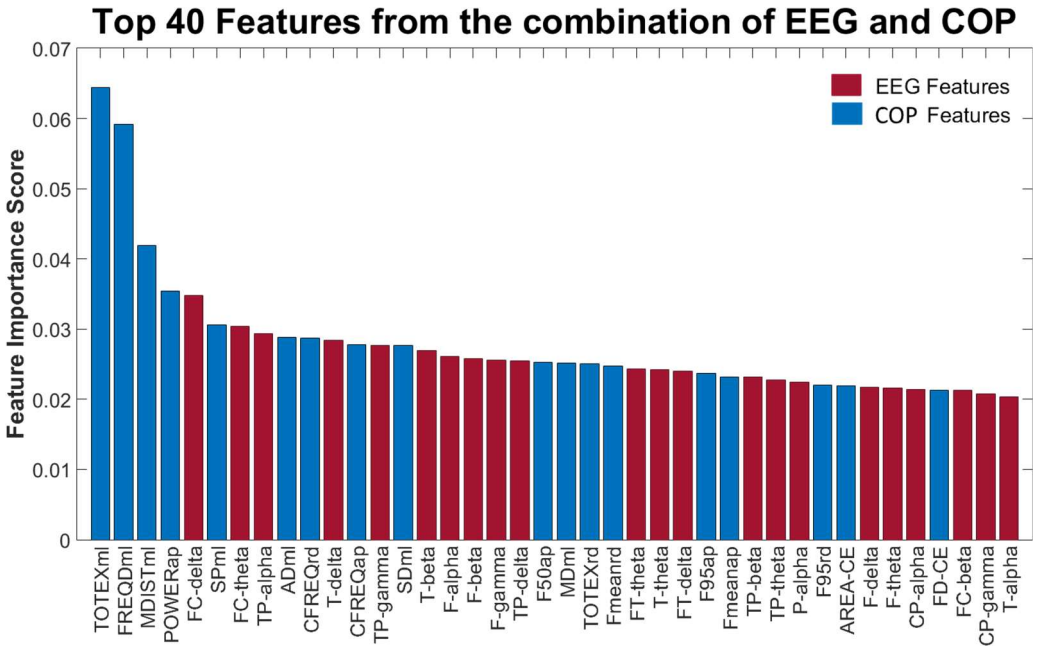


Figure 7.4 The feature importance score of the top 40 features.

features in 15 seconds dataset (Figure 7.5). Even so, at least 30 features were required to achieve more than 0.87 AUC in all three datasets, which was greater or equal to the highest AUC result using all features in different datasets (Table 7.3).

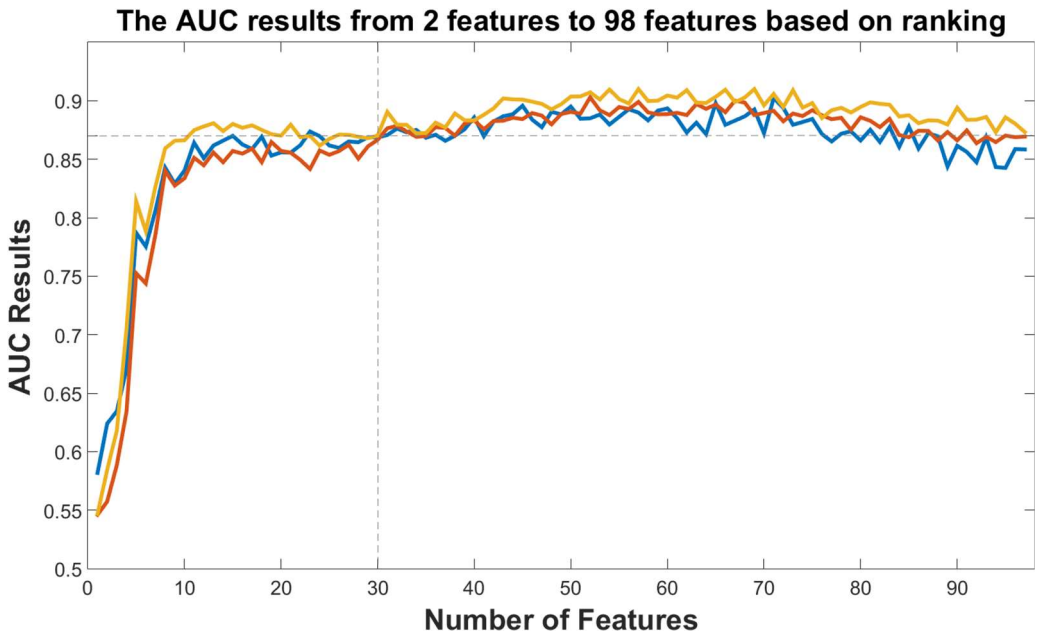


Figure 7.5 The AUC results of 2 features till 98 features using the random forest. The grey dash line across the *y*-axis denote as 0.87 AUC.

7.4 Discussion

From the observation of Table 7.3, the AUC results in feature set A demonstrated greater results compared to feature set B, suggesting the COP features were more significant than the EEG features in discriminating the groups. Subsequently, the AUC results from the combination of both EEG and COP features (feature set C) reported higher AUC results in all datasets than COP features alone. This suggests that some EEG features might work well with the COP features which increase the overall classification results. It is also noticeable that even though 5 seconds sampling period had the largest sample size, most AUC results from the 10 seconds and 15 second sampling period datasets were higher than the 5 second sampling period dataset, suggesting a minimum of 10 second sampling period is required to detect useful information in both EEG and COP feature sets. The random forest classifier showed greater AUC results compared to other algorithms in classifying TD adults and adults with ASD with up to 0.873 using both EEG and COP feature sets.

In Figure 7.2, the delta and theta rhythm in the frontal-central region were the most significant features to classify TD adults and adults with ASD during quiet stance. The

two rhythms in the frontal-central region have been associated with attention and concentration (Barry et al., 2007; Widagdo, Pierson, & Helme, 1998), this is consistent with previous studies which have suggested that individuals with ASD may have difficulties with sustained attention (Corbett & Constantine, 2006; Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Murphy et al., 2014). In Figure 7.3, the total excursion in ML directions ($TOTEX_{ML}$) was the most significant feature to classify both group consistent with previous studies which reported larger ML sway in ASD population (Graham et al., 2015; Stins et al., 2015).

A minimum of 11 features from EEG and COP was desired to achieve an AUC result greater than 0.850 in all different sampling duration datasets. However, a minimum of 30 features was required to achieve better results than the AUC results reported using all EEG and COP features – 0.870. The AUC result of this work is greater than the accuracy of the currently used Autism Diagnostic Observation Schedule Assessment (ADOS-2 Module 4) which reported 0.84 AUC and the accuracy of Autism Diagnostic Interview-Revised assessment (ADI-R) which reported 0.78 AUC (Fusar-Poli et al., 2017). Also, the proposed assessment took less time and cost to implement. Thus, the work in this chapter could provide valuable insights to assist in ASD assessment and may provide a potential avenue for the development of more accurate and effective ASD diagnostic tools. Meanwhile, the preliminary classification performance was performed based on the default settings of the machine learning models, fine-tuning the parameters of the individual classifiers (especially random forest) could achieve further improvement.

7.5 Summary

Current ASD diagnostic tools are expensive, time-consuming and require extensive training to use. As sensory impairments are now accepted as core symptoms of ASD, the features from EEG and COP during quiet stance were used to classify TD adults and adults with ASD in three different sampling period datasets. The results in all sampling period datasets were consistent. COP features were reported more significant

and valuable than EEG features but the combination of both COP and EEG features produce greater results than one feature set alone. Random forest reported the highest AUC result with up to 0.873 compared to other machine learning models. However, not all COP or EEG features were useful for the classification. The COP and EEG features were ranked accordingly based on variable importance using forests of trees, and it was reported that a minimum of 30 ranked features from EEG and COP was required to achieve greater or equal to 0.870 AUC. With the sampling period of 15 seconds, random forest managed to achieve 0.9143 AUC. Future research is required to extend the classification of the features to develop a potential screening tool for ASD in childhood.

Chapter 8

Conclusion and Future Works

8.1 Summary

The purpose of this thesis was to extend the understanding of cortical involvement in human postural control using EEG. Traditionally, the involvement of subcortical structures such as spinal cord, brain stem, basal ganglia and cerebellum in maintaining postural control has been demonstrated in humans (Horak & Macpherson, 2011; Jacobs & Horak, 2007; Lewko, 1996; Maki & McIlroy, 2007). The potential role of the cerebral cortex has recently considered in postural control with evidence from dual-task paradigms, visual attention and perturbation evoked responses (PERs) (See review (Maki & McIlroy, 2007; Wittenberg et al., 2017)). However, the understanding of cortical involvement in postural control is still limited. EEG has provided opportunities to investigate the cortical activities related to postural control, particularly, the PERs and EEG rhythms. Thus, this thesis investigates the cortical role of vision, the cortical role of postural demand tasks, and sensory impairments in Autism Spectrum Disorder (ASD). Lastly, this thesis focuses on enabling classifications on adults with and without ASD based on the centre of pressure and EEG measurements during quiet standing.

In Chapter 2, the postural control system was briefly introduced, and the neurophysiological aspects of postural control systems were thoroughly explained. Current literature suggests the cortical cortex plays a role in the human postural control

and EEG provides a unique opportunity to investigate the cortical activity related to postural control. However, the literature regarding cortical influence on postural responses is not well developed. Thus, the main contribution of this thesis is to examine the postural responses to visual perturbation and postural demand tasks. The findings from the studies above were then replicated to investigate individuals with ASD. Thus, a brief introduction to Autism Spectrum Disorder and sensory impairment was also presented in this chapter. Before diving into the studies, the pre-processing methods for EEG signals and centre of pressure (COP) are necessary to improve the quality of Signal to Noise ratio. Thus, the techniques that were used to quantify and assess the physiological signals were reviewed in Chapter 3.

Despite the significant contribution of the visual system to postural control, PERs to a visual perturbation of posture have yet to be reported. In Chapter 4, the cortical responses to visual perturbation of posture were demonstrated in ten typically developed (TD) adults. The responses to postural perturbation by sudden visual occlusion are similar to that seen about a physical perturbation, notably the detection (P1) and evaluation (N1) components. The amplitude of the N1 response is not only consistent with the relative magnitude of the perturbation, but also the underlying postural set, with a larger N1 seen in standing relative to sitting. This study informs the relative importance of vision to postural stability, postural set and provides a protocol to assess sensory-based postural disorders. Despite that, the neural oscillations of the EEG or the EEG rhythm potentially play a role in the synchronization of neural activity across the brain region.

As a result, the effect of postural task (i.e. standing and sitting) on cortical EEG rhythms such as theta and alpha activities was investigated about the differing visual condition (i.e. visual transparent and visual occlusion) in twenty-four TD adults at Chapter 5. This study revealed elevated cortical theta and alpha activities during changes in postural demand suggested that more brain resources are allocated to postural tasks that are more demanding. Additionally, the increase in these cortical theta and alpha activities during visual occlusion suggests a reduction of visual input.

The results of this chapter establish a link between postural task and cortical activity, specifically the modulation of theta and alpha activity.

Despite the importance of the cortical responses to postural control as mentioned in Chapter 4, PERs to a visual perturbation in adults with ASD have yet to be reported. In fact, it is evident that individuals with ASD use visual and proprioceptive information differently than TD adults. Therefore, the PERs to visual perturbation under varied postural stability conditions were investigated in adults with and without ASD in Chapter 6. This study found that there was no difference in the latencies of the PERs between groups, suggesting no differences in early perturbation processing speed in adults with ASD. However, while the peak amplitudes of the P1 components were similar between groups during the postural tasks, peak amplitudes of the N1 component were larger in the ASD group regardless of the postural task. The results suggest while the assessment of postural set is intact, adults with ASD use more cortical resources to integrate and interpret visual perturbations for postural control. The results from this study provide insights into the cortical responses which contribute to sensory integration in individuals with ASD.

Due to the rise in the prevalence of ASD, there is an increased demand for ASD diagnostic assessments. However, the ‘gold standard’ of ASD diagnosis is still expensive, time-consuming and require extensive training to use. Extensive studies have been conducted to investigate the COP behavioural response of individuals with ASD, but very few studies have specifically examined sensorimotor integration in this disorder. The cortical differences between adults with and without ASD highlighted in Chapter 6 had demonstrated the EEG as a potential neuro-marker for ASD diagnostic assessment. Also, the EEG rhythm could provide vital information assisting to elucidate the nature of sensorimotor integration atypicality in this disorder. Thus in Chapter 7, typically developed adults and adults with ASD were classified based on the centre of pressure (COP) measurements from force plate and EEG rhythm features. There were three datasets according to the sampling periods (5, 10 or 15 seconds) and e8-fold cross-validation was used to estimate of model prediction performance and

evaluation. The results suggest that COP features were reported more significant and valuable than EEG features. However, the combination of both COP and EEG features produce greater results than one feature set alone. Random forest reported the highest AUC result with up to 0.873 compared to other machine learning models. However, not all COP or EEG features were useful for the classification. The COP and EEG features were ranked accordingly based on variable importance using forests of trees, and it was reported that a minimum of 30 ranked features from EEG and COP was required to achieve greater or equal to 0.870 AUC. The AUC result of this work is greater than the accuracy of the currently used Autism Diagnostic Observation Schedule Assessment (ADOS-2 Module 4) which reported 0.84 AUC and the accuracy of Autism Diagnostic Interview-Revised assessment (ADI-R) which reported 0.78 AUC (Fusar-Poli et al., 2017).

8.2 Future Work

The studies from this thesis investigate the cortical responses to visual occlusion in typically developed adults and adults with Autism Spectrum Disorder (ASD). Moreover, the effect of postural task on cortical theta and alpha activities was also investigated and enable classification on typically developed adults and adults with ASD based on the centre of pressure and EEG rhythm features. However, further investigations are still needed to enhance the understanding of cortical involvement in human postural control. Below are the future works to close the research gap in this area:

1. In the first study (Chapter 4), the cortical responses to visual occlusion were investigated in young adults. Even though the effect of ageing had demonstrated in anticipatory postural control (Kanekar & Aruin, 2014), the cortical responses in balance response in this population are not well understood. Thus, an additional study has to be carried out to explore the cortical responses.
2. In the second study (Chapter 5), the limitation of this study was that the spatial

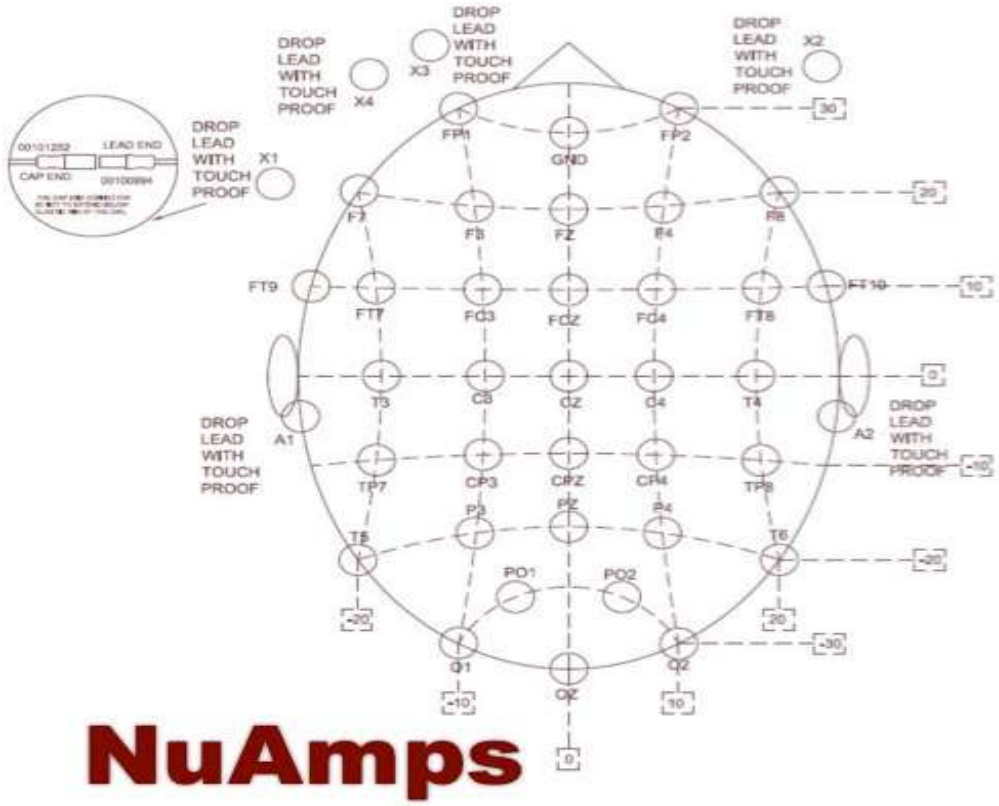
resolution of the EEG scalp surface recordings is limited. However, the spatial resolution of EEG can be addressed using EEG source localisation techniques such as standardised low-resolution brain electromagnetic tomography (Pascual-Marqui, 2002) to represent local underlying brain sources more accurately. Thus, future studies will include EEG source localisation to provide some insight into the potential role of the cortical activity.

3. In the third study (Chapter 6), the types of impairments in sensory responses observed in individuals with ASD, specifically hypersensitivity (i.e. excessive sensitivity) and hyposensitivity (i.e. below normal sensitivity) could potentially play a role in affecting the perturbation evoked responses differently. Thus, more studies needed to carry out in this direction.
4. In the fourth study (Chapter 7), the study on enabling classification on typically developed adults and adults with ASD is limited due to the sample size. In future, the research can be extended to collect more samples and potentially clinical trials.

Appendix A

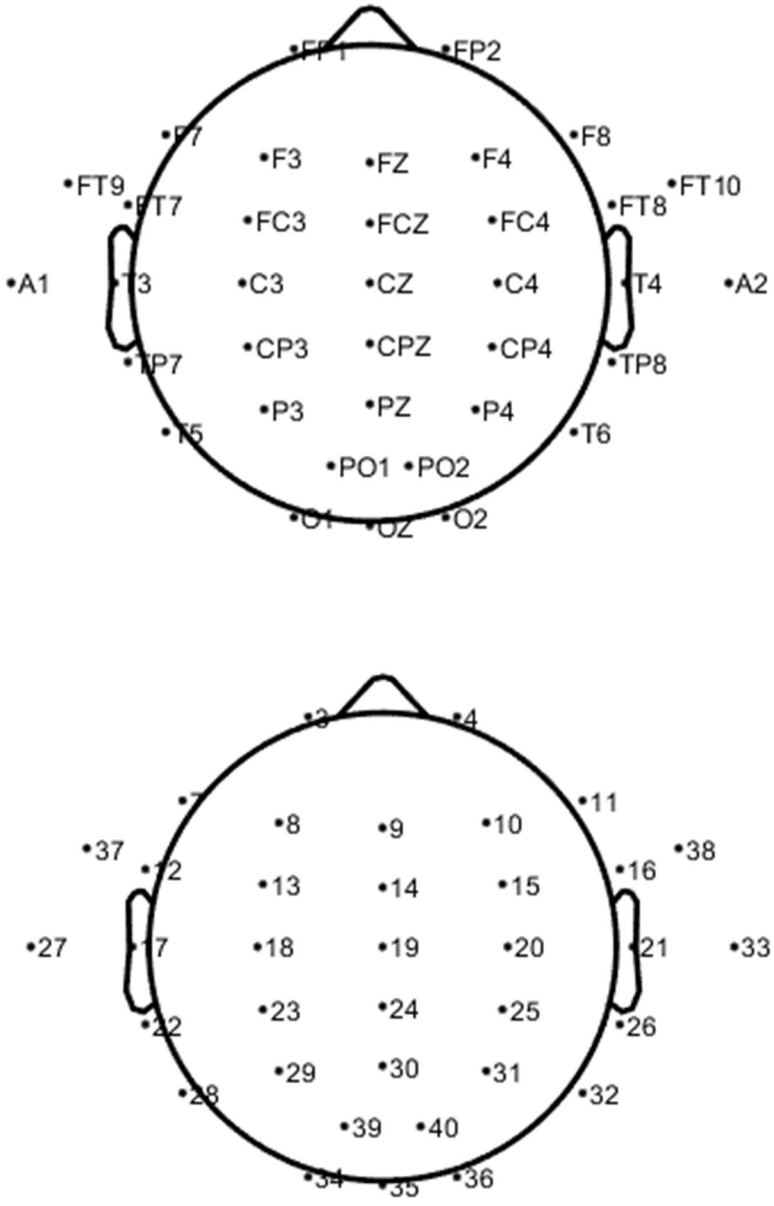
40 channels (including EOG)

1	'HEOL'	**11**	'F8'	**21**	'T4'	**31**	'P4'
2	'HEOR'	**12**	'FT7'	**22**	'TP7'	**32**	'T6'
3	'FP1'	**13**	'FC3'	**23**	'CP3'	**33**	'A2'
4	'FP2'	**14**	'FCZ'	**24**	'CPZ'	**34**	'O1'
5	'VEOU'	**15**	'FC4'	**25**	'CP4'	**35**	'OZ'
6	'VEOL'	**16**	'FT8'	**26**	'TP8'	**36**	'O2'
7	'F7'	**17**	'T3'	**27**	'A1'	**37**	'FT9'
8	'F3'	**18**	'C3'	**28**	'T5'	**38**	'FT10'
9	'FZ'	**19**	'CZ'	**29**	'P3'	**39**	'PO1'
10	'F4'	**20**	'C4'	**30**	'PZ'	**40**	'PO2'



34 channels (excluding EOG)

1	'FP1'
2	'FP2'
3	'F7'
4	'F3'
5	'FZ'
6	'F4'
7	'F8'
8	'FT7'
9	'FC3'
10	'FCZ'
11	'FC4'
12	'FT8'
13	'T3'
14	'C3'
15	'CZ'
16	'C4'
17	'T4'
18	'TP7'
19	'CP3'
20	'CPZ'
21	'CP4'
22	'TP8'
23	'T5'
24	'P3'
25	'PZ'
26	'P4'
27	'T6'
28	'O1'
29	'OZ'
30	'O2'
31	'FT9'
32	'FT10'
33	'PO1'
34	'PO2'



Appendix B

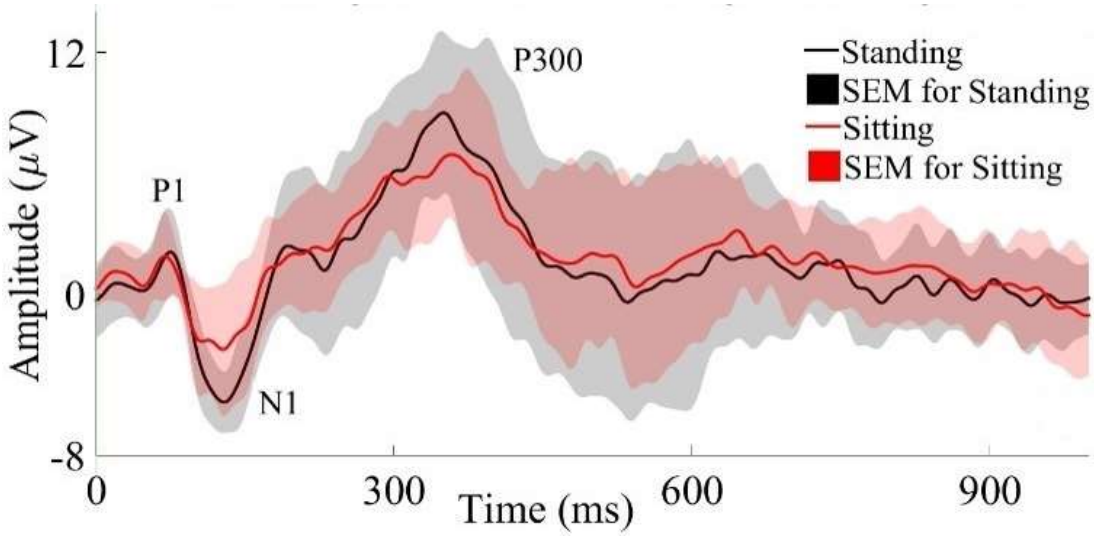


Figure B.1 Grand average of PERs in standing and sitting at frontal-central site from 0ms to 1000ms. *SEM stands for the standard error of the mean.

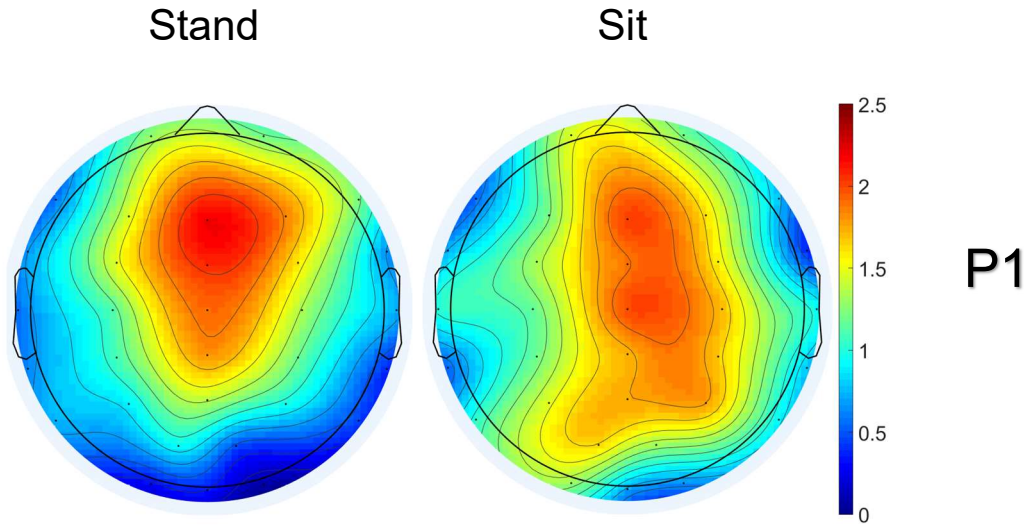


Figure B.2 Grand average of the EEG topographic maps of P1 based on the peak latencies for each subject. The power unit for the scale bars is μV and subjected to a log_{10} transform to normalise the distribution.

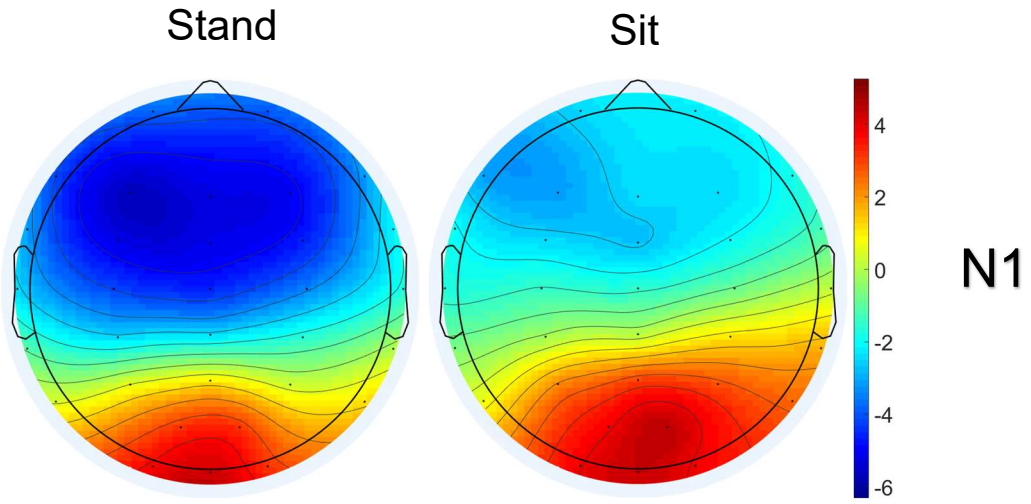


Figure B.3 Grand average of the EEG topographic maps of N1 based on the peak latencies for each subject. The power unit for the scale bars is μV and subjected to a log_{10} transform to normalise the distribution.

Appendix C

COP Features

ADap	Amplitude of CP displacement AP, Distance between the maximum and minimum CP displacement for each direction
ADml	Amplitude of CP displacement ML, Distance between the maximum and minimum CP displacement for each direction
ADrd	Amplitude of CP displacement RD, Distance between the maximum and minimum CP displacement for each direction
AREA_CC	The 95% confidence circle area (AREA_CC), the area of a circle with a radius equal to the one-sided 95% confidence limit of the RD time series
AREA_CE	The 95% confidence ellipse area (AREA_CE), the area of the bivariate confidence ellipse, which is expected to enclose approximate 95% of the points on the COP path.
AREA_SW	Swap area (AREA_SW) estimates the area enclosed by the COP path per unit of time
Area95	Area (95% of the COP data inside)
CFREQap	The frequency in thr AP direction at which spectral mass is concentrated
CFREQml	The frequency in thr ML direction at which spectral mass is concentrated
CFREQrd	The frequency in thr RD direction at which spectral mass is concentrated
F50ap	50% of the AP frequency spectrum
F50ml	50% of the ML frequency spectrum
F50rd	50% of the RD frequency spectrum
F95ap	95% of the AP frequency spectrum
F95ml	95% of the ML frequency spectrum
F95rd	95% of the RD frequency spectrum
FD_CC	Fractal Dimensions - Confidence Circle (FD_CC)
FD_CE	Fractal Dimensions - Confidence Ellipse (FD_CE)
Fmeanap	Median AP frequency of the spectrum
Fmeanml	Median ML frequency of the spectrum
Fmeanrd	Median RD frequency of the spectrum
Fpeakap	AP Peak frequency of the spectrum

Fpeakml	ML Peak frequency of the spectrum
Fpeakrd	RD Peak frequency of the spectrum
FREQDap	Unitless measure of the variability in the AP frequency content of the power spectral density
FREQDml	Unitless measure of the variability in the ML frequency content of the power spectral density
FREQDrd	Unitless measure of the variability in the RD frequency content of the power spectral density
MDISTap	Mean Distance-AP (MDISTap), Average AP distance from the mean COP
MDISTml	Average ML distance from the mean COP
MDISTrd	Mean Distance-RD (MDISTrd), Average RD distance from the mean COP
MDap	Max Distance-AP, the absolute value of the difference between the smallest and the largest value in AP time series.
MDml	Max Distance-ML, the absolute value of the difference between the smallest and the largest value in ML time series.
MDrd	Max Distance-RD, the absolute value of the difference between the smallest and the largest value in RD time series.
MFREQap	Mean Frequency-AP (MFREQap), Frequency of a sinusoidal oscillation in Hz with an average value of the mean distance-AP and a total path length of total excursions-AP.
MFREQml	Mean Frequency-ML (MFREQml), Frequency of a sinusoidal oscillation in Hz with an average value of the mean distance-ML and a total path length of total excursions-ML.
MFREQrd	Mean Frequency (MFREQrd), rotational frequency in revolutions per seconds or Hz of the COP if it had traveled the total excursions around a circle with a radius of the mean distance.
POWERap	Total Power in AP, integrated area of the power spectrum
POWERml	Total Power in ML, integrated area of the power spectrum
POWERrd	Total Power in RD, integrated area of the power spectrum
SDap	Standard Deviation AP, Dispersion of CP displacement from the mean position during a time interval
SDml	Standard Deviation ML, Dispersion of CP displacement from the mean position during a time interval
SDrd	Standard Deviation RD, Dispersion of CP displacement from the mean position during a time interval
SPap	Sway Path AP
SPml	Sway path ML
SPrd	Sway Path RD
TOTEXap	Total Excursion AP (TOTEXap), total length of the COP path is approximated by the sum of the distances between consecutive points in the AP series
TOTEXml	Total Excursion ML (TOTEXml), total length of the COP path is approximated by the sum of the distances between consecutive points in the ML time series

TOTEXrd	Total Excursion RD (TOTEXap), total length of the COP path is approximated by the sum of the distances between consecutive points in the RD series

EEG Features

C_alpha	Alpha rhythm at the central site
C_beta	Beta rhythm at the central site
C_delta	Delta rhythm at the central site
C_gamma	Gamma rhythm at the central site
C_theta	Theta rhythm at the central site
CP_alpha	Alpha rhythm at the central-parietal site
CP_beta	Beta rhythm at the central-parietal site
CP_delta	Delta rhythm at the central-parietal site
CP_gamma	Gamma rhythm at the central-parietal site
CP_theta	Theta rhythm at the central-parietal site
F_alpha	Alpha rhythm at the frontal site
F_beta	Beta rhythm at the frontal site
F_delta	Delta rhythm at the frontal site
F_gamma	Gamma rhythm at the frontal site
F_theta	Theta rhythm at the frontal site
FC_alpha	Alpha rhythm at the frontal-central site
FC_beta	Beta rhythm at the frontal-central site
FC_delta	Delta rhythm at the frontal-central site
FC_gamma	Gamma rhythm at the frontal-central site
FC_theta	Theta rhythm at the frontal-central site
FT_alpha	Alpha rhythm at the frontal-temporal site
FT_beta	Beta rhythm at the frontal-temporal site
FT_delta	Delta rhythm at the frontal-temporal site
FT_gamma	Gamma rhythm at the frontal-temporal site
FT_theta	Theta rhythm at the frontal-temporal site
O_alpha	Alpha rhythm at the occipital site
O_beta	Beta rhythm at the occipital site
O_delta	Delta rhythm at the occipital site
O_gamma	Gamma rhythm at the occipital site
O_theta	Theta rhythm at the occipital site
P_alpha	Alpha rhythm at the parietal site
P_beta	Beta rhythm at the parietal site
P_delta	Delta rhythm at the parietal site
P_gamma	Gamma rhythm at the parietal site
P_theta	Theta rhythm at the parietal site

PO_alpha	Alpha rhythm at the parietal-occipital site
PO_beta	Beta rhythm at the parietal-occipital site
PO_delta	Delta rhythm at the parietal-occipital site
PO_gamma	Gamma rhythm at the parietal-occipital site
PO_theta	Theta rhythm at the parietal-occipital site
T_alpha	Alpha rhythm at the temporal site
T_beta	Beta rhythm at the temporal site
T_delta	Delta rhythm at the temporal site
T_gamma	Gamma rhythm at the temporal site
T_theta	Theta rhythm at the temporal site
TP_alpha	Alpha rhythm at the temporal-parietal site
TP_beta	Beta rhythm at the temporal-parietal site
TP_delta	Delta rhythm at the temporal-parietal site
TP_gamma	Gamma rhythm at the temporal-parietal site
TP_theta	Theta rhythm at the temporal-parietal site

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