Hyperfocus in adult ADHD: An EEG study of the differences in cortical activity in resting and arousal states

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Abstract

Attention deficit/hyperactivity disorder (ADHD), characterised by problems relating to attention, impulsive behaviour and hyperactivity, has become widely accepted to be a neurodevelopmental disorder that persists into adulthood for a large proportion of the ADHD population. ADHD symptoms are reported to undergo developmental transformation from childhood to adulthood, resulting in a significantly different clinical picture of adult ADHD. This signals a need to move away from reliance on childhood models of the disorder. Current conceptions of both childhood and adult ADHD do not discuss the occurrence of *hyperfocus* (or *flow*), a symptom which seems to connote an extreme form of sustained attention and which has been noted in clinical work. The possibility that hyperfocus could occur in ADHD is unexpected and poses a challenge to current formulations of inattention in ADHD. Media settings have been identified as providing the conditions needed for one to enter the hyperfocus state. This study set out to explore the possible symptom of hyperfocus in ADHD and to attempt to identify the neural correlates thereof, using electroencephalograph (EEG). A sample of 10 participants (5 ADHD, 5 non-ADHD) was recruited using the Adult ADHD Self Report Scale V-1.1 (ASRS V-1.1) Screener and a biographical questionnaire. A quasi-experimental research design was employed, whereby EEG recordings of frontal, frontal midline and parietal regions were taken for each participant during resting states (Eyes Closed and Eyes Open) and whilst playing a first person shooter game. Post-test survey questionnaires were also administered to examine the participants' time perception during game play. Between-group and within-group differences in absolute and relative power scores were examined, using non-parametric statistical methods (Mann-Whitney-U test and Wilcoxon Signed Rank Test) to analyse the data. Possible markers for hyperfocus were identified, namely significantly lower alpha and beta levels in the ADHD group, as well as a decrease in slow-wave activity over time, as well as post-test survey data that indicated a greater degree of distorted time perception in the ADHD group during game play. Significant between- and within-group differences found in the parietal region highlight the need for further research into the role of the parietal lobe in attention functions and in ADHD. Further, significant changes in cortical activity in the progression from Eyes Closed to Eyes Open in both groups warrant further investigation.

CONTENTS

Acknowledgments **ii**

Abstract **iii**

Chapter 1: Introduction **1**

1.1 Introduction 1

1.2 Historical roots of ADHD 3

1.3 Diagnostic and psycho-diagnostic controversies 9

1.3.1 Wender's diagnostic approach 10

1.3.2 Diagnosing adult ADHD using the DSM-IV-TR 11

1.4 Comorbidity 13

1.5 Adult ADHD as a separate disorder 14

1.6 Conclusion 15

Chapter 2: Attention, Inattention and Hyperfocus **17**

2.1 Introduction 17

2.2 Attention and inattention 18

2.3 Inattention and hyperfocus 21

2.3.1 Csikszentmihalyi's theory of flow 22

2.3.2 Proposed model of hyperfocus in ADHD 25

2.4 Computer games and flow 27

2.5 Neuroanatomy of ADHD 28

2.6 Neurophysiology of ADHD 30

2.7 Neurophysiology of hyperfocus 31

2.8 EEG models in ADHD research 32

2.9 Conclusion 33

Chapter 3: Methodology **35**

3.1 Introduction 35
3.2 Sampling and participants 36
3.3 Measures 37
3.3.1 Adult ADHD Self-Report Scale-Version 1.1 (ASRS-V1.1) 37
3.3.2 Biographical questionnaire 39
3.3.3 BIOPAC MP150 Data Acquisition System 39
3.3.4 Cognitive activation procedure 40
3.4 Procedure 40
3.4.1 Phase 1: Screening 41
3.4.2 Phase 2: Data collection 42
3.5 Data analysis 44
3.5.1 EEG data 44
3.5.2 Survey questionnaire 46
3.6 Ethical considerations 46
3.7 Conclusion 46
Chapter 4: Results **48**
4.1 Introduction 48
4.2 Frontal lobe 49
4.2.1 Descriptive statistics 49
4.2.1.1 Mean power scores in the frontal lobe 49
4.2.1.2 Mean relative wave activity in the frontal lobe 53
4.2.2 Between-group differences in the frontal lobe 54
4.2.2.1 Absolute values 54
4.2.2.2. Relative values for frontal lobe 55
4.2.3 Within-group differences 56
4.3. Frontal midline 58
4.3.1 Descriptive Statistics 58
4.3.1.1 Mean power scores in the frontal midline 58

4.3.1.2 Mean relative wave activity in the frontal midline 62
4.3.2 Between-group differences in the frontal midline 63
4.3.2.1 Absolute values in the frontal midline 63
4.3.2.2 Relative values in the frontal midline 64
4.3.3 Within-group differences in the frontal midline 65
4.4 Parietal lobe 66
4.4.1 Descriptive statistics for parietal lobe 66
4.4.1.1 Mean power scores in the parietal lobe 66
4.4.1.2 Mean relative wave activity in the parietal lobe 70
4.4.2 Between-group differences in the parietal lobe 71
4.4.2.1 Mean absolute power in the parietal lobe 71
4.4.2.2 Mean relative power in the parietal lobe 72
4.4.3 Within-group differences in the parietal lobe 73
4.5 Theta: beta ratios 74
4.6 Post-test survey data 76
4.7 Summary and conclusion 77
CHAPTER 5: DISCUSSION **80**
5.1 Introduction 80
5.2 Between-group differences across resting states 81
5.3 Was hyperfocus achieved? 82
5.4 Between-group differences 84
5.5 Within-group differences 85
5.6 Theta: Beta Ratios 87
5.7 Limitations & recommendations 87
5.7.1 Limitations 87
5.7.1.1 Sampling 87
5.7.1.2 Design 88
5.7.2 Recommendations 89

5.8 Conclusion 90

References **92**

Appendices **104**

Appendix A Biographical questionnaire 104

Appendix B Participant information sheet 108

Appendix C Informed consent form 112

Appendix D Instructions for participants prior to appointment 113

Appendix E Pre-EEG questionnaire 114

Appendix F Post-EEG questionnaire 115

LIST OF TABLES AND FIGURES

Tables

1	DSM-IV-TR criteria for ADHD	7
2	Changes in the DSM Definition of ADHD	8
3	Data collection procedure	44
4	Mean power scores per wave and condition for the frontal lobe	49
5	Relative wave activity across conditions in the frontal lobe	54
6	Between-group differences for the absolute power scores of all waves for the frontal cortical site	55
7	Between-group differences in relative values across waves and conditions	56
8	Within-group differences in the progressions between conditions for frontal lobe	57
9	Mean power scores per wave and condition for frontal midline	58
10	Relative wave activity across conditions in the frontal midline	63
11	Between-group differences for the absolute power scores of all waves for the frontal midline cortical site	64
12	Between-group differences in relative values across waves and conditions in frontal midline	65
13	Within-group differences in progressions between conditions for frontal midline	66
14	Mean power scores per wave and condition for parietal lobe	67
15	Relative wave activity across conditions in the parietal lobe	71
16	Between-group differences for the absolute power scores of all waves for the parietal lobe	72
17	Between-group differences in mean relative power in the parietal lobe	73
18	Within-group differences in progressions between conditions for parietal lobe	74
19	Between-group differences in theta: beta ratios	75
20	Between-group differences in theta: beta ratios per experimental condition and cortical site	75
21	Time perception and awareness of surroundings during game play	76
22	Summary of results	79

Figures

1	Proposed model of flow in context of self-efficacy	27
2	10-20 montage for electrode placement in electroencephalography	40
3	Delta wave mean power score in frontal lobe across conditions	50
4	Theta wave mean power score in frontal lobe across conditions	51
5	Alpha wave mean power score frontal lobe across conditions	52
6	Beta wave mean power score in frontal lobe across conditions	52
7	Delta wave mean power score in frontal midline across conditions	59
8	Theta wave mean power score in frontal midline across conditions	60
9	Alpha wave mean power score in frontal midline across conditions	61
10	Beta wave mean power score in frontal midline across conditions	62
11	Delta wave mean power score in parietal lobe across conditions	68
12	Theta wave mean power score in parietal lobe across conditions	68
13	Alpha wave mean power score in parietal lobe across conditions	69
14	Beta wave mean power score in parietal lobe across conditions	70
15	Time perception versus actual time spent during game play	77

CHAPTER 1: INTRODUCTION

1.1 Introduction

The diagnostic label 'attention-deficit/hyperactivity disorder' (ADHD) is used to describe a population that typically exhibits problems relating to attention, impulsive behaviour and excessive levels of activity (American Psychiatric Association (APA), 2000). Up until relatively recently, it was believed that ADHD symptoms did not persist into adulthood (Barkley, Murphy & Fischer, 2007). However, research suggests that symptoms will carry over into adolescence and adulthood for a large proportion of those diagnosed with ADHD in childhood (Barkley 1998; Wender, 2000). Furthermore, it has been suggested that people who are diagnosed with adult ADHD (aADHD) constitute an entirely separate subpopulation from children with the same disorder; possessing “not only a stable pathophysiology, but a different neurobiological or environmental background” (Schneider, Retz, Coogan, Thome & Rösler, 2006, p.I/32). This implies that research on ADHD in child populations cannot fully account for adult populations, indicating a need for more research into aADHD.

The DSM-IV-TR lists criteria for the different subtypes of ADHD, including symptoms such as inattention, hyperactivity, and impulsivity. A symptom that has (arguably) been omitted from the DSM-IV criteria is a phenomenon termed *hyperfocus*. According to Schecklmann et al. (2009) hyperfocus can be understood as “intensive concentration on interesting and non-routine activities accompanied by temporarily diminished perception of the environment” (p.104). This particular symptom, although known from clinical work, seems to have received very little attention in scientific research thereof (Schecklmann et al., 2009) and thus requires further research into the nature and causes of hyperfocus, specifically in ADHD. The possibility that hyperfocus could occur in ADHD is particularly crucial, as it seems counterintuitive to definitions of inattention that have been proposed for ADHD, and could pose a challenge to current formulations of the disorder. Given the way in which hyperfocus is described, one could argue that it is a state of

heightened sustained attention, and therefore, if it does occur in ADHD, the concept of inattention in ADHD is questioned.

This study aimed to research hyperfocus in ADHD, with a specific focus on adults with this disorder. Given that ADHD is currently understood to be a neurodevelopmental disorder with a strong neurobiological basis (Tye et al., 2012), electroencephalography (EEG) was utilised to explore possible neurological substrates of hyperfocus in adults with ADHD. The theory of *flow* developed by Csikszentmihalyi (1992) was used as a basis for conceptualising the construct of hyperfocus (the terms *hyperfocus* and *flow* refer to the same construct and thus are used interchangeably in this study). Based on this theory of flow, Sherry (2004) identified media settings as providing the conditions necessary for flow to occur. Subsequently, in a study by Weber, Tamborini, Westcott-Baker and Kantor (2009), playing a computer game was identified as an appropriate task to facilitate the flow experience. This study thus sought to establish differences in EEG readings between adults with ADHD and adults without ADHD across cortical resting states (Eyes Closed and Eyes Open) and cortical activation states (playing a computer game as a state of hyperfocus).

In order to understand current formulations of the disorder, it is important to trace back through the various ways that ADHD was conceptualised by the clinical and scientific community. Current definitions of ADHD reflect the culmination of clinical and scientific research spanning more than a century (Barkley, 2006). The earliest descriptions of ADHD are usually traced back to the works of paediatrician George Frederick Still, in which a lack of morality or a deficit in moral control was pinpointed as the primary cause of the disorder (Still, 1902, as cited in Timimi & Leo, 2009). Subsequently, as research on the topic has accumulated, aetiological theories have evolved.

Despite this long history, the diagnostic criteria for ADHD are still considered highly controversial, particularly in their applicability to aADHD. The clinical picture is further clouded by the high rate of comorbidity of ADHD with other disorders. The section that follows will thus trace through the origins of ADHD as a disorder. Thereafter, the diagnostic and psycho-diagnostic controversies relating to aADHD

will be explored. This will include problems identified in the DSM criteria, and shortcomings in the way that aADHD is diagnosed. The final section will address the diagnostic challenges posed by the high rate of comorbidity of aADHD with other disorders.

1.2 Historical roots of ADHD

In the first half of the 20th century, an epidemic of encephalitis led researchers and clinicians to the conclusion that brain damage can cause cognitive and behavioural disturbances. This conclusion was reached after symptoms of restlessness, personality changes and learning difficulties were noted in many of the children who had been affected in the epidemic (Timimi & Leo, 2009). Thereafter, interest grew in studying the causes and effects of brain damage. In various studies, a range of cognitive and behavioural impairments, including symptoms which are now associated with ADHD, were linked to brain injury (Barkley, 2006). It was in light of these findings that the diagnosis of minimal brain damage (MBD) was created.

MBD was the proposed diagnosis in children who exhibited symptoms of hyperactivity, even in the absence of evidence of brain injuries (Barkley, 2006). The approach taken here emphasises the biological and organic aspects of the aetiology, an approach which is echoed in current descriptions of ADHD. Descriptions of the clinical picture of children with ‘brain injury’ or ‘minimal brain injury’, found in early 20th century literature, match descriptions of what is now labelled as ADHD. For example, Strauss and Lehtinen (1947, p. 129) describe the behaviour of a child with MBD as follows:

> The brain-damaged organism, as we know, is abnormally responsive to the stimuli of his environment, reacting unselectively, passively, and without conscious intent. When such a hypervigilant organism - one whose reactibility is beyond his own control - is placed in a situation of constant and widespread stimulation, he can only meet the situation with persistent undirected response…He pivots in his seat to watch the activity of any children sitting near him. His attention is caught and held by any child who leaves his place. Any noise may cause him to attend; any motion seemingly

> insignificant may attract him. The teacher will repeatedly find him gazing at pictures and decorations while the lesson is neglected on his desk.

The above description fits the clinical picture of ADHD perfectly. Clinicians linked these symptoms to brain injury, even in the absence of physical evidence to corroborate the existence of brain injuries (Barkley, 2006; Timimi & Leo, 2009). Inevitably, when researchers were compelled to acknowledge that there was insufficient evidence of such brain injury in many children who exhibited these symptoms, the diagnosis of 'brain injury' evolved into 'minimal brain damage', and later still into 'minimal brain dysfunction' (MBD) (Goodman, 2009). Other terms used to refer to these symptoms included 'organic learning and behaviour disorders', 'organic deviation' and 'CNS deviation' (Goodman, 2009). This diagnosis was eventually abandoned when the *Diagnostic Statistical Manual, Second Edition* (DSM-II) was released in 1968 (APA, 1968).

In the years leading up to the publication of the DSM-II, there was a move away from attributing the aforementioned symptoms to a purely biological cause. The release of the DSM-II saw the introduction of the label 'hyperkinetic reaction of childhood' for what was previously referred to as MBD (APA, 1968; McGough & McCracken, 2006). It was generally believed that the symptoms would gradually and spontaneously improve with age, and that the hyperkinetic reaction did not persist into adulthood (e.g. Laufer & Denhoff, 1957). Whereas MBD was attributed purely to organic causes, the hyperkinetic reaction of childhood swung to the opposite extreme, viewing the symptoms as "a reaction to family environment" (McGough & McCracken, 2006, p. 1673). The focus thus turned to the effect of the environment on behaviour as the primary cause.

In 1980, the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III) (APA, 1980) was released, introducing a completely new way of conceptualising the hyperkinetic reaction to childhood. The three core symptoms of inattention, hyperactivity and distractibility would now form the foundation of the diagnostic criteria, an approach which has continued to inform the selection of criteria for ADHD in all subsequent editions of the DSM (Goodman, 2009). This change is quite significant; previous notions of ADHD emphasised the hyperkinesis

or over activity associated with the disorder, with little reference to the aspects of attention associated with the disorder. The DSM-III removed hyperactivity as the central symptom, and moved towards recognising the key role of attention and concentration in the symptomatology. This is evident in the name change in the DSM-III to attention-deficit disorder (ADD) (APA, 1980). As demonstrated in the name, ADD was understood to stem primarily from a deficit in attention, and was believed to exist either with or without the additional symptom of hyperactivity. Accordingly, ADD subtypes were created: ADD with hyperactivity (ADD +H) and ADD without hyperactivity (ADD -H). DSM-III criteria were met if the individual exhibited two or three symptoms **from each of the three** behavioural categories (inattention, impulsivity, and hyperactivity) (Searight & McLaren, 1998).

Further changes were made in subsequent editions of the DSM, to include hyperactivity in the name, hence returning this symptom to the spotlight. The DSM-III-R renamed the disorder attention-deficit hyperactivity disorder (ADHD), and collapsed the three behavioural categories into one list of 14 symptoms, 8 of which needed to be exhibited for an individual to be diagnosed with ADHD (APA, 1987). The single list of symptoms implies that ADHD was now understood to be a unitary disorder, rather than a disorder with subtypes. The ADD-H subtype was thus removed. Further, the majority of the symptoms referred to behaviours associated with hyperactivity and impulsivity, signalling a return to a greater emphasis on hyperactive and impulsive behaviour rather than inattention (Searight & McLaren, 1998). According to Barkley (2006), a major reason for doing away with the ADD -H subtype was the lack of empirical evidence to support its placement as a subtype of ADD; it was unknown whether the attention deficit associated with this subtype was qualitatively similar or different to ADD +H.

The current name, attention-deficit/hyperactivity disorder, was coined in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) (APA, 1994), and was kept unchanged in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (DSM-IV-TR) (APA, 2000). The slight change alludes to the acknowledgement that inattention does not necessarily imply hyperactivity. The DSM-IV criteria for ADHD have been based on a much larger field trial than previous editions, making the criteria the most empirically-

based in the history of the disorder (Barkley, 2006). The criteria for ADHD remained unchanged from the DSM-IV to the DSM-IV-TR. Three subtypes were included, namely ‘Predominantly Inattentive Type’ (similar to DSM-III’s ADD -H), ‘Predominantly Hyperactive-Impulsive Type’ and ‘Combined Type’. Whereas the DSM-III had listed criteria in three separate behavioural categories (inattention, hyperactivity and impulsivity), the DSM-IV reduced this to two categories by combining hyperactivity and impulsivity into one category. For a diagnosis to be made, a minimum of six symptoms of either inattention or hyperactivity/impulsivity would have to be present in two or more settings for at least six months before the age of seven years, and it must be clear that these symptoms negatively affect one’s academic, social or occupational functioning. Further, these symptoms are not exclusively observable during the course of a Pervasive Developmental Disorder, Schizophrenia or another Psychotic Disorder, and would not be better accounted for by another mental disorder (APA, 2000). The criteria in the DSM-IV and DSM-IV-TR also mark the first time that the criteria having been worded in a manner that attempts to include adults. The criteria of the DSM-IV-TR are shown in Table 1. The adjustment of the symptoms to include adults is a topic of debate and will be discussed later on.

The definition of ADHD has undergone numerous significant changes since its symptoms were first noted (see Table 2). Opinions regarding age of onset and duration of ADHD have also varied considerably through the many changes in definition of ADHD. Until relatively recently, it was believed that ADHD symptoms did not persist into adulthood (Barkley et al., 2007). However, research suggests that symptoms will carry over into adolescence and adulthood for many of those diagnosed with ADHD in childhood, and that the disorder is "associated with significant psychopathology and dysfunction in later life" (Wilens, Biederman & Spencer, 2002, p.114).

Table 1

DSM-IV-TR criteria for ADHD (APA, 2000)

Criterion	Symptoms
A	Six or more symptoms of either (1) inattention or (2) hyperactivity/impulsivity for a minimum of 6 months to a degree that is maladaptive and incongruent with one's developmental level:
	(1) Inattention
	(a) Often fails to pay attention to details or makes careless mistakes in schoolwork, work, or other tasks.
	(b) Often struggles to sustain attention while performing tasks or in play activities.
	(c) When spoken to directly, often doesn't seem to be paying attention.
	(d) Often fails to follow through on instructions or on tasks, such as schoolwork, household duties, or workplace tasks (not due to lack of understanding or defiant behaviour).
	(e) Often struggles to organise tasks and activities.
	(f) Avoids, or doesn't enjoy partaking in tasks that demand sustained mental effort (e.g. Homework or class work). (g) Often misplaces or loses things which are needed for certain activities (e.g. School books, stationery, toys etc.).
	(h) Often gets distracted by irrelevant stimuli.
	(i) Forgetful in day to day tasks.
	(2) Hyperactivity/impulsivity
	Hyperactivity
	(a) Tends to fidget with hands or move around in one's seat.
	(b) Often gets up from chair in class or other situations where sitting is required/expected.
	(c) Frequently runs around or climbs excessively in inappropriate environments (adolescents/adults may just experience a subjective feeling of restlessness).
	(d) Struggles to engage in leisure activities quietly.
	(e) Frequently "on the go" or seems as if "driven by a motor".
	(f) Often excessively talkative.
	Impulsivity
	(g) Often blurts out answers before the full question has been asked.
	(h) Struggles to wait his/her turn
	(i) Frequently interrupts others or intrudes on people's activities (e.g. Butting into a conversation or game)
B	Some symptoms of hyperactivity/impulsivity or inattention which caused impairment were evident before 7 years old.
C	Symptoms cause impairment in two or more settings (e.g. Work, home).
D	There is clear evidence of impairment that is clinically significant in one's social, academic or occupational functioning.
E	Symptoms are not limited to the course of a Pervasive Developmental Disorder, Schizophrenia or another Psychotic Disorder, and symptoms cannot be better accounted for by another mental disorder.

Table 2

Changes in the DSM Definition of ADHD

	DSM-III	DSM-III-R	DSM-IV	DSM-IV-TR
Name	Attention-deficit disorder (ADD)	Attention-deficit hyperactivity disorder	Attention-deficit/hyperactivity disorder (AD/HD)	Attention-deficit/hyperactivity disorder (AD/HD)
Subtypes	•ADD with Hyperactivity (ADD+H) •ADD without Hyperactivity (ADD-H)	No subtypes	•Inattentive Type •Hyperactive Type •Combined Type	Inattentive Type Hyperactive Type Combined Type
Diagnostic criteria	•Three behavioural categories (inattention, impulsivity, hyperactivity) •Minimum 2 or 3 symptoms from each behavioural category required for diagnosis	-Single list of 14 symptoms Minimum 8 symptoms required for diagnosis Majority of criteria refer to behaviours associated with hyperactivity and impulsivity	Two behavioural categories of Inattention and Hyperactivity/Impulsivity. Minimum 6 symptoms of either inattention or hyperactivity/ impulsivity	Two behavioural categories of Inattention and Hyperactivity/Impulsivity. Minimum 6 symptoms of either inattention or hyperactivity /impulsivity

Barkley et al. (2007) pinpoint the 1960s and 1970s as the period in which research first addressed the possibility of an adult version of ADHD (then referred to as minimal brain damage or dysfunction (MBD)). Studies conducted during this period began to explore the possibility of MBD symptoms persisting into adulthood. For example, Menkes, Rowe and Menkes (1967) undertook a 25 year follow-up study of individuals diagnosed with MBD, concluding that symptoms had persisted to some degree in most subjects who were examined. Later research provided biological evidence for ADHD, as well as further support for an adult version of the disorder. For example, in a study of adoptive and biological parents of children with and without hyperactivity, Alberts-Corush, Firestone and Goodman (1986) found that biological parents of children with hyperactivity had more attentional difficulties than all other parents in the study. Further, it was found that parents of hyperactive children had often themselves been hyperactive as children and often suffered from other forms of psychopathology in adulthood (Cantwell, 1975; Morrison & Stewart, 1971). Additionally, research by Wood, Reimherr, Wender and Johnson (1976) demonstrated that adults with MBD-like symptoms responded well to drug therapy typically administered to children with MBD. Although these and other studies took promising steps in paving the way for ADHD to be recognised as a disorder affecting adults, professionals and the general public only began to accept the

legitimacy of aADHD in the 1990s (Barkley, 2006). The DSM-III-R was the first to include adults in the definition of the disorder, in recognition of research results which reported persistence of symptoms into adulthood (Goodman, 2009).

With a steadily growing body of research addressing the nature, causes and prevalence of adult ADHD, findings from many studies provide evidence of high rates of persistence of ADHD symptoms from childhood into adulthood (Wilens et al., 2002). There is considerable variance in reports as to the exact percentage of cases of ADHD that persist into adulthood. Wender (2000) reports that 33% to 66% of childhood cases of ADHD persist into adulthood, which translates into prevalence rate of between 1% and 5%. Barkley (1998) reports that ADHD symptoms and associated behavioural problems persist into adulthood in 50% to 65% of cases of ADHD identified in childhood. However, differences in prevalence rates seem to be a function of methodological differences in the studies (Wilens et al., 2002).

1.3 Diagnostic and psycho-diagnostic controversies

Many professionals have criticised the way in which both childhood and adult ADHD have been conceptualised, and have called to question the validity of the DSM-IV-TR's diagnostic criteria, despite the fact that the diagnostic criteria for ADHD in the DSM-IV-TR are the most empirically based to date (Barkley, 2006). Some have even gone so far as to question the existence of ADHD as a real disorder. For example, Moncrief and Timimi (2010) believe that pharmaceutical companies have been promoting aADHD as a valid disorder because it promises to provide a lucrative market for stimulants and other related drugs. They argue that there is insufficient evidence for the effectiveness of specific treatments, and that the diagnostic criteria are far too vague to be relied on for diagnosing adults with ADHD (Moncrief & Timimi, 2010). Although many of the objections and critiques are applicable to both childhood and adult ADHD, the primary focus of the discussion will be on aADHD. The full argument for the validity of the diagnosis of childhood and aADHD falls outside of the scope of this specific paper. The stance taken in this study is that ADHD is a valid disorder which still requires the establishment of appropriate diagnostic criteria.

Given the recency of aADHD having been acknowledged as a valid diagnostic category, the lack of consensus as to the exact clinical picture of aADHD is not surprising. There is still debate around which symptoms of aADHD are necessary for diagnosis, as opposed to which are common but not necessary for diagnosis (Wender, 2000). The lack of agreement among professionals can be attributed to a number of factors, such as the divergent approaches which have been adopted for the diagnosis of aADHD (McGough & Barkley, 2004), the developmental transformation which the key symptoms are said to undergo (Pary, Lewis, Matuschka & Lippmann, 2002), as well as the high rate of comorbidity with other psychiatric disorders (Torgersen, Gjervan & Rasmussen, 2006).

The method of diagnosing aADHD has not been standardised. McGough and Barkley (2004) identify two dominant diagnostic approaches which have been adopted in the diagnosis of aADHD, but which do not seem to correlate in the sets of patients identified. These two divergent approaches are: (1) the approach led by Wender and colleagues (Wender, 1996), and (2) the approach based on an adapted version of DSM criteria (McGough & Barkley, 2004). The problems associated with each of these approaches are discussed below.

1.3.1 Wender's diagnostic approach

Wender's approach for diagnosing aADHD primarily entails the retrospective diagnosis of childhood ADHD based on interviews of the patient and another informant, preferably a parent (Wender, 1996). Originally, Wender suggested that aADHD symptoms could be categorised into seven clusters: inattention; hyperactivity; mood lability; irritability and hot temper; impaired stress tolerance; disorganisation; and impulsivity (Wender, 1996). Individuals and their informants are interviewed based on these seven symptom clusters. A diagnosis of aADHD is made if a retrospective diagnosis of childhood ADHD can be made, both hyperactivity and inattentiveness are identified as on-going symptoms, and at least two more of the remaining symptoms are present (McGough & Barkley, 2004). The retrospective diagnosis is made with the help of the Wender Utah Rating Scale (WURS), which requires the patient and informant to respond to 25 items descriptive of typical ADHD behaviour in childhood (Ward, Wender & Reimherr, 1993).

The approach of Wender and his colleagues (1996; 2000) have highlighted some important points to keep in mind when diagnosing aADHD. There is growing support for the premise that aADHD is preceded by childhood ADHD (e.g. Barkley, 2006; Wilens et al., 2002), thus providing justification for the need to establish a history of childhood ADHD symptoms, as required by Wender and colleagues (2000). Although the symptom clusters identified by Wender and colleagues (1996) have not withstood the changes in the conceptualisation of ADHD which have occurred with emerging research, the approach that they pioneered has established that there is a need for "retrospective determination of childhood ADHD, use of third-party informants, and recognition of developmental changes in adult presentation" for the diagnosis of aADHD (McGough & Barkley, 2004, p.1953).

The use of retrospective reports of ADHD symptoms, however, presents an additional problem for accurate diagnosis: Pary, Lewis, Matuschka and Lippmann (2002) point out that retrospective data collected from the individual and from other sources regarding the individual's past behaviour are not necessarily reliable, as recollection of past events is often inaccurate. This problem cannot be resolved easily in the cases of adults who have not previously been diagnosed with ADHD and thus have no documented history of ADHD symptoms (Pary et al., 2002). Wender and his colleagues address this problem partially by using three different approaches simultaneously to collect data. First, the patient's parents are spoken to; second, the patient's parent is asked to complete the Parent Rating Scale (Wender and others, 1981; as cited in Wender, 1996), which provides a report on the patient's childhood ADHD-related behaviour; finally, the WURS is administered, which has been standardised on normal adults, adults with ADHD, and adults with a major depressive disorder (Wender, 1996).

1.3.2 Diagnosing adult ADHD using the DSM-IV-TR

The second dominant approach used for diagnosing aADHD, which bases the diagnosis on the DSM-IV-TR criteria, is quite widely used. In order to meet criteria for ADHD, the individual must: exhibit six or more symptoms listed under either the category of 'inattention' or 'hyperactivity/impulsivity' for at least six months to a degree which causes significant impairment and is inconsistent with one's

developmental level; have had some symptoms which caused impairment before the age of seven years; and have impairment from symptoms in at least two settings (APA, 2000). There is much controversy surrounding the use of the DSM-IV criteria for diagnosing aADHD, and it has been argued that the DSM-IV criteria are not appropriate for diagnosis of ADHD in adults (McGough & Barkley, 2004; Wiita & Parish, 2008). Firstly, the threshold of six symptoms for diagnosis seems to be unfounded in adult populations, and fails to identify all adults who experience clinically significant impairment (McGough & Barkley, 2004). Wilens et al. (2002) report that 90% of adults who were diagnosed in childhood with ADHD experience impaired overall functioning despite not meeting the full diagnostic criteria for ADHD. This implies that the diagnostic criteria as delineated by the DSM-IV-TR may be inherently flawed in that an individual's overall functioning may continue to be significantly affected in adulthood, even if the person does not exhibit the minimum required symptoms for a 'valid' diagnosis.

Apart from the unfounded threshold of six symptoms necessary for a diagnosis of aADHD, the wording used in the DSM-IV criteria excludes adults due to the over-specificity of the context and behaviour needed in making a diagnosis. Symptoms include references to difficulties associated with schoolwork and homework, classroom behaviour, and also cite behaviours such as excessive running or climbing (APA, 2000). As pointed out by McGough and Barkley (2004), these symptoms lack face validity for adults, as they are developmentally inappropriate. Furthermore, research indicates that symptoms of ADHD undergo changes with normal development, and thus the presentation of the essential symptoms of hyperactivity, inattentiveness and impulsivity may present differently at different ages (Pary et al., 2002; Wasserstein, 2005). Pary et al. (2002) outline the differences in the symptoms which present at various developmental trajectories. School-aged children may exhibit impulsiveness and hyperactivity through their inability to wait their turn and blurting out answers before being called upon, and their mood and performance are inconsistent and unpredictable. Symptoms of hyperactivity tend to decrease with age, so that adolescents display more age appropriate levels of activity, and rather experience problems with setting priorities and with being organised (Greenhill, 1998, as cited in Pary et al., 2002). Adults with ADHD typically exhibit impairments which are predominantly attributable to

attention deficits and disorganisation, with a decrease in hyperactive and impulsive symptoms (Schneider et al., 2006). Symptoms of hyperactivity may evolve into a sense of restlessness and consequently often manifest as an unstable lifestyle of changing jobs and moving house, as well as short-lived or poor relationships (Pary et al., 2002; Wiita & Parish, 2008). These individuals also characteristically perform poorly in tasks which require vigilance, motor inhibition, planning, organisation, verbal learning, and memory (Wadsworth & Harper, 2007).

The changes in symptom expression from childhood to adulthood and the inappropriate wording of the DSM-IV criteria for adult populations indicate that it is unsuitable to attempt to apply a single set of diagnostic criteria to both childhood and adult ADHD. Furthermore, the diagnostic criteria listed in the DSM-IV-TR for ADHD have not been validated for adult populations (McGough & Barkley, 2004). Thus, it is vital that criteria are developed and validated for aADHD (Fedele, 2008).

1.4 Comorbidity

The high rate of comorbidity found in individuals with ADHD is perhaps a major factor which contributes to the difficulties associated with creating a valid and reliable set of diagnostic criteria for aADHD. Approximately 65-89% of adults with ADHD reportedly suffer from at least one comorbid psychiatric disorder, most often mood and anxiety disorders, substance use disorders or personality disorders (Sobanski, 2006), which may share similar features to ADHD (Torgersen et al., 2006). Pary et al. (2002) report that up to 75% of adults who have attention deficits are diagnosed with a comorbid psychiatric condition. This makes it difficult to discern which symptoms are attributable to other comorbid conditions and which are specific to ADHD. Moreover, the diagnosis of ADHD may be missed in adults because the comorbid disorders are usually the focus of attention and treatment of mental health professionals (Wasserstein, 2005). Comorbid psychiatric disorders have also been reported to complicate the clinical picture of ADHD regarding diagnostics, treatment and outcome (Sobanski, 2006). Compounding the issue of comorbidity, there is no objective assessment available for making an accurate diagnosis of ADHD in adults (Goodman, 2009; Torgersen et al., 2006).

1.5 Adult ADHD as a separate disorder

Neurocognitive and biological findings observed in children with ADHD are similar to findings in adults with ADHD (Biederman, 2005; Schneider et al., 2006). Thus the research findings on childhood ADHD serve as a good starting point for conducting research into aADHD. However, despite the seemingly strong connection between childhood and adult ADHD, it has been suggested that due to the differences between the core clinical features of ADHD in adults and children, adults with ADHD constitute an entirely separate subpopulation from children with the same disorder; possessing “not only a stable pathophysiology, but a different neurobiological or environmental background” (Schneider et al., 2006, p.I/32). This implies that research conducted on ADHD in child populations cannot be assumed to be fully applicable to adult populations without further investigation. This underscores the need for more research to establish diagnostic criteria specifically for aADHD, as opposed to making minor adjustments to existing criteria.

In light of the problems identified with the DSM-IV-TR criteria, a few suggestions have been made to improve the method of diagnosing adults with ADHD until the next edition of the DSM is released. McGough and Barkley (2004) suggest utilising the DSM-IV criteria as a guideline, rather than a strict set of criteria, and make the following recommendations:

(1) Due to the lack of evidence to support criterion B requiring that there be symptoms before the age of seven years, any symptoms before puberty should be considered significant.
(2) Clinicians should establish if there have been “pervasive impairments over the lifespan, even if current complaints are limited to a single domain”, and that “clinicians can be comfortable treating adults with childhood histories of ADHD, evidence of current ADHD-related impairment, and a minimum of four current hyperactive-impulsive or inattentive symptoms” (p.1953).
(3) Clinicians should try to obtain information from third parties when possible and document all evidence which points to the presence of ADHD.

The DSM-V has been in development for the past few years, with its final release date set for May 2013 (APA, 2012). Surprisingly, the proposed revisions for ADHD

are relatively minor in proportion to the criticisms made about the current diagnostic criteria. In response to the criticism that the criteria are inappropriate for adults, the APA has changed the threshold for adolescents and adults (older than 17 years) from six symptoms to four symptoms to be diagnosed with ADHD. Regarding the lack of empirical basis for the requirement of criterion B that symptoms were present before 7 years, the cut-off age has been changed to 12 years (APA, 2013). New symptoms of hyperactivity/impulsivity and inattention have been added to criterion A, and are more suitable for identification of adults with ADHD. Although the proposed changes make the criteria more appropriate for adults than the DSM-IV-TR criteria, attempting to arrive at a unitary set of criteria for both adults and children ignores the research findings that suggest that aADHD may constitute an entirely separate disorder from childhood ADHD (Schneider et al., 2006). Furthermore, changing the cut-off age in criterion B from 7 years to 12 years repeats the same error of defining an arbitrary cut-off age in the absence of empirical basis. Finally, reduction of the threshold for diagnosis may result in increased cases of misdiagnosis (Frances, 2010).

1.6 Conclusion

Through the numerous developments in the definition of ADHD, it has become widely accepted that ADHD is a neurodevelopmental disorder that persists into adulthood for a large proportion of the ADHD population (Tye et al., 2012). The developmental transformation which ADHD symptoms are reported to undergo from childhood to adulthood result in a significantly different clinical picture of adult ADHD, signalling a need to move away from reliance on childhood models of the disorder. The differences in symptom presentation have been recognised by the editors of the DSM-IV-TR who have made some changes to the diagnostic criteria in an attempt to include adults into the diagnostic criteria, although the changes are very subtle, limited to small changes in wording (Wiita & Parish, 2008). These minor changes are clearly not sufficient for making an accurate diagnosis of adult ADHD. The proposed changes for DSM 5 also fail to address the problems with the current criteria, and again, do not draw enough of a distinction between childhood and adult ADHD. The overlap of symptoms between ADHD and comorbid disorders makes the task of defining a clear clinical picture for ADHD more difficult (Sobanski, 2006), and hence a challenge to create an undisputed set of DSM criteria for ADHD.

These issues imply a need for further research to establish a more solid model of adult ADHD which does not model itself exclusively on childhood models of the disorder, and consequently a separate set of criteria to be used in the diagnosis of adult ADHD.

Current conceptions of both childhood and adult ADHD do not make much mention of the occurrence of hyperfocus, despite this symptom having been noted in clinical work (Schecklmann et al., 2009). Chapter 2 will explore the symptom of hyperfocus in the context of definitions of attention and inattention. Hyperfocus will be conceptualised through the works of Mihaly Csikszentmihalyi, who has researched hyperfocus (or “flow”, as he refers to it) extensively. The close link between hyperfocus and sustained attention creates a contradiction in the terminology used for inattention in ADHD. The way in which inattention is conceptualised in the literature is not compatible with the possibility that an individual with ADHD can exhibit hyperfocus. This implies that, should hyperfocus be a phenomenon which can occur in individuals with ADHD, then there is a need to reformulate the way that inattention is understood. More specifically, if inattention is defined as the “inability to sustain attention” (Weyandt et al., 2003, p. 283), this dictates that the individual with ADHD cannot exhibit hyperfocus. This does not seem to be accurate, given reports of hyperfocus in individuals with ADHD.

CHAPTER 2: ATTENTION, INATTENTION AND HYPERFOCUS

2.1 Introduction

Throughout the many changes in definition of ADHD, deficits in attention have always been recognised. In the literature describing the 'brain injured' child, these deficits are described as 'abnormally responsive to the stimuli of his environment' and 'hypervigilant…one whose reactibility is beyond his own control' (Strauss & Lehtinen, 1947, p.129). The DSM-II identified distractibility and short attention span as two of the symptoms characteristic of the 'hyperkinetic reaction to childhood' (APA, 1968). From DSM-III to DSM-IV-TR, the centrality of the problems associated with inattention was fully acknowledged, with inattention being recognised as a cardinal symptom of ADHD and thus included in the name of the disorder (Goodman, 2009). Interestingly, inattention is the only symptom which is always recognised as part of the clinical picture of ADHD, across all subtypes; although hyperactivity and impulsivity are easily observable in many cases of ADHD and are considered cardinal symptoms of ADHD, the presence of these two symptoms is not required for a diagnosis of ADHD to be made (APA, 2000). Moreover, there is increasing evidence that symptoms associated with inattention are far more persistent than those of hyperactivity/impulsivity and are associated with more significant impairment (Kordon, Kahl & Wahl, 2006).

Typically, attention is conceptualised by dividing the functions of attention into separate components or processes, thereby demonstrating a general consensus that attention is a multifaceted construct in which more than one process is at play. This is where the consensus ends in the literature, as there is considerable variability in the theories and models of attention. For example, Knudsen (2007) identifies four components of attention, namely, (i) working memory, (ii) competitive selection, (c) top-down sensitivity control, and (d) salience filters. In contrast, Petersen and Posner (2012) describe attention in relation to three primary anatomical networks (alerting, orienting and executive). Theories of executive function often include components of attention such as sustained and selective attention (e.g. Cubillo, Halari, Smith, Taylor & Rubia, 2012; Lovejoy et al., 1999)

and are thus often closely related to models of attention. However, the manner in which executive functions are described and understood is by no means uniform, largely due to “the inconsistent and interchangeable use of both psychological and anatomical definitions of executive and frontal functions” (Stuss & Alexander, 2000, p. 289). This further obscures the way in which attention is understood.

The sections that follow will explore the construct of attention/inattention through the various ways in which it has been conceptualised in the literature. In the first section, attention/inattention and hyperfocus are explored and defined through extant literature. These definitions are used as a base for critical examination of the way in which inattention has been defined in ADHD, demonstrating that this conception of inattention is incompatible with the possibility that hyperfocus can occur in ADHD. This implies that, should hyperfocus be a phenomenon which can occur in individuals with ADHD, there is a need to reformulate the way that inattention is understood. A model of flow/hyperfocus, based on Csikszentmihalyi’s theory of flow (Csikszentmihalyi, 1992) will be proposed as it applies to ADHD. This model will be linked to current research on the neuropsychological correlates of ADHD and sustained attention, as well as current EEG research on ADHD.

2.2 Attention and inattention

Currently, the diagnostic criteria and clinical picture of ADHD are based upon the three cardinal symptoms of the disorder, namely: hyperactivity, impulsivity and inattention (APA, 2000; Goodman, 2009). Historically, the concept of attention has been confusing due to the variety of definitions, as well as the many suggested subdivisions of attentional processes (Zillmer, Spiers & Culbertson, 2001). This confusion is described clearly by Zillmer, et al. (2001):

> … The term attention can refer to general level of alertness or vigilance; a general state of arousal; orientation versus habituation to stimuli; the ability to focus, divide, or sustain mental effort; the ability to target processing within a specific sensory arena (such as visual attention or auditory attention); or a measure of capacity. Researchers have also asked whether attention implies

> a general state of cortical tone or energy, or functions as a network or set of specific structures or networks within the brain … (p.170)

As is evident in the above excerpt, the term attention has been used to describe a variety of phenomena and processes. The general consensus of researchers is that, rather than attention being a unified concept, it is “a multifaceted concept that implies multiple behavio[u]ral states and cortical processes that various subsets of cerebral structures control” (Zillmer et al., 2001, p. 170). This is supported by research findings from neuroimaging, neuropsychological, clinical and neuroanatomical studies, which reveal that attentional functions cannot be localised to one single area (Tucha et al., 2008).

Given the strong evidence indicating that attention is a complex construct governed by a variety of interlinked processes, descriptions of inattention in the literature on ADHD seem to be oversimplified; descriptions of inattention often paint a picture of a consistent weakness or inability in the individual to sustain attention. For example, Weyandt et al. (2003, p.382) define inattention as the “inability to sustain attention”, while Biederman (2005, p.1215) provides examples of inattention, namely, “daydreaming, distractibility, and difficulty focusing on a single task for a prolonged period”. This understanding of inattention does not account for the occurrence of hyperfocus. The possibility that individuals with ADHD could exhibit the ability to hyperfocus is noted in clinical work, (Schecklmann et al., 2009), and many popular media articles addressing ADHD take it for granted that hyperfocus is a regular symptom of ADHD (e.g. Danoff, 2011; Flippin, 2005; Low, 2009). Definitions of hyperfocus appear to be closely linked to those of sustained attention. According to Catania (1998, as cited in Sagvolden, Aase, Johansen & Russel, 2005), attention can be described by how a stimulus or stimulus property affects one's behaviour, taking into account the sensory and motivational processes at play. When a stimulus or stimulus property changes one's behaviour, this implies that the individual is attending to the stimulus or stimulus property. Sagvolden et al. (2005) extend this general description of attention to explain that sustained attention refers to a situation in which one's behaviour is controlled over time by a stimulus or stimulus property. Similarly, Zillmer et al. (2001) define sustained attention as “the ability to maintain an effortful response over time”, and state that it is linked to one’s

ability to “persist and sustain a level of vigilance” (p.173). Both hyperfocus and sustained attention connote a situation in which the individual's behaviour is controlled by a stimulus or stimulus property over time. However, hyperfocus appears to refer to a more specific (and perhaps extreme) type of sustained attention in which the individual's behaviour is controlled for a long period of time by a task which is 'non-routine' or of interest to him/her, to the point that his/her awareness of the environment is considerably diminished (Sagvolden et al., 2005; Schecklmann et al., 2009). If hyperfocus connotes a mode of sustained attention, it seems paradoxical that it could occur in individuals with ADHD, a disorder characterised by inattention.

Kaufmann, Kalbfleisch and Castellanos (2000) provide an alternative description of the attentional difficulties associated with ADHD that seems to be more compatible with the occurrence of hyperfocus in ADHD individuals. They state that “ADHD is not characteri[s]ed by an inability to sustain attention, but rather by the inability to appropriately regulate the application of attention to tasks that are not intrinsically rewarding and/or that require effort” (p.2). Leimkuhler (1994) echoes this view of ADHD, stating that the attentional difficulties do not necessarily manifest as a consistent weakness, but rather occur in an inconsistent fashion, resulting in a “‘maldistribution’ of attention”. Accordingly, the inattention “may quite literally disappear when the individual is occupied with something of great interest to him or her” (p.180). Similarly, Thompson and Thompson (2005) suggest that a person with adult ADHD struggles to sustain attention “when material is slow paced, repetitive, boring or deemed irrelevant” (termed *routine*) (p.123). Conversely, the individual is able to **hyperfocus** on tasks which are of particular interest to the person, which can be considered *non-routine* (Thompson & Thompson, 2005).

According to the above descriptions, the occurrence of hyperfocus appears to be task-dependant, and tends to occur when the task is interesting to the individual. In this context, it appears that the following constructs are linked to hyperfocus: (i) interest, (ii) motivation and (iii) intrinsic motivation. If hyperfocus tends to occur in tasks which are interesting to the individual, this implies that interest plays an important role in providing motivation to attend to a task, a position which is supported strongly in the literature (Ainley, 2006; Edelson & Joseph, 2001; Reeve,

1989; Schiefele, 1991). Edelson and Joseph (2001) assert that interest in a task acts as an intrinsic motivation for the individual to attend to and engage in the task, without the need for external rewards. Research indicates that if an activity arouses interest in the individual, this leads to intrinsic motivation (Reeve, 1989). This is corroborated by a study conducted by Schiefele (1991), in which a significant positive correlation was found between interest and intrinsic motivation. Similarly, Ainley (2006) conceptualises interest as an affective state which facilitates motivation through eliciting alertness, arousal, attention and concentration when an individual is presented with a novel task. The supposition that motivation underlies hyperfocus is important for providing a link to ADHD, as research supports the notion that motivation plays an important role in mediating the behaviour in an individual with ADHD. Neurobiological research identifies deficits in executive functions in ADHD which play a role in the regulation of affect and motivation (Cubillo et al., 2012).

Thus far, hyperfocus has been explored through the very scant descriptions provided in ADHD literature. The section that follows will elaborate on the construct hyperfocus through the work of Mihaly Csikszentmihalyi, whose study of hyperfocus (or flow, as he calls it) over the past few decades forms the most significant body of research on the topic.

2.3 Inattention and hyperfocus

Csikszentmihalyi's studies and publications have approached the study of flow through the paradigm of positive psychology, and have focused on trying to understand what components are at play which leads to flow. Csikszentmihalyi has subsequently used these findings to formulate a theory of flow (Csikszentmihalyi, 1992). Although Csikszentmihalyi's work on flow is quite extensive, he has not really addressed the presence of hyperfocus or flow in ADHD. Thus, it seems that very little research has addressed this construct as it relates to ADHD (Schecklmann et al., 2009). This model of flow will be integrated with current research pertaining to ADHD.

2.3.1 Csikszentmihalyi's theory of flow

Csikszentmihalyi (1992) describes flow as 'the state in which people are so involved in an activity that nothing else seems to matter; the experience itself is so enjoyable that people will do it even at great cost, for the sheer sake of doing it' (p.4). He also refers to this state as optimal experience. The following major components which constitute the phenomenology of the flow experience have been identified through data collected over many years: (i) The experience usually takes place during a challenging task which requires skill; (ii) The individual must be able to concentrate on the task; (iii) the task usually has clear goals and provides immediate feedback; (iv) due to the deep but effortless investment into the task at hand, one's awareness is removed from all other thoughts; (v) a sense of control over one's actions is felt; (vi) one's awareness of self becomes diminished; and (vii) one's sense of time becomes warped.

(i) Challenging tasks that require skill

Csikszentmihalyi (1992) reports that in all activities that participants in his study were engaged in, their sense of enjoyment came at a specific point: "whenever the opportunities for action perceived by the individual are equal to his or her capabilities" (p.52). In other words, an activity becomes enjoyable when a person feels challenged to the degree that he/she possesses the suitable skills to succeed, but still feels the need to invest his/her skills into the activity. Csikszentmihalyi describes this point where enjoyment begins as "the golden ratio between challenges and skills", and states that "enjoyment appears at the boundary between boredom and anxiety, when the challenges are just balanced with the person's capacity to act" (p. 52). This seems to be linked to Bandura's self-efficacy theory in which Bandura posits that the individual's perceived abilities in a given task will affect the individual's motivation to attempt and persist in performing the task (Bandura, 1977). The link between Bandura's theory of self-efficacy and flow will be discussed later.

(ii) Full concentration on the task

Descriptions of the flow experience highlight the full concentration on the activity at hand. This seems to occur as a function of the previous component: the challenges of the activity demand that the individual calls on all of his/her relevant skills,

resulting in the individual becoming completely absorbed in the task. This does not leave any room for any other stimuli to be processed (Csikszentmihalyi, 1992). Csikszentmihalyi (1992) describes this element as the most distinct feature of the flow experience: “people become so involved in what they are doing that the activity becomes spontaneous, almost automatic; they stop being aware of themselves as separate from the actions they are performing” (p.53).

(iii) Clear goals and immediate feedback

Flow is most likely to occur in situations where the individual has clear goals and will receive immediate feedback as the activity progresses. For example, a tennis player knows that he/she needs to return the tennis ball to his/her opponent’s side of the court, and will know immediately if this goal has been achieved. Csikszentmihalyi (1992) notes that in creative activities, although the goals may not be as clear cut, the individual generally has developed an internal sense of what he/she intends to achieve; as the creative activity progresses, the individual will assess the progress according to the internal criteria of what is “good” or “bad”. Regardless of the form that the feedback takes, it provides confirmation to the individual that he/she has succeeded in what he/she set out to achieve. Interestingly, this sort of immediate feedback can also be noted in video or computer games. In a New York Times article, Dr Christopher Lucas notes that children with ADHD are able to sustain attention when playing video games because there are “frequent intermittent rewards” (Klass, 2011). Video games have also been identified by Sherry (2004) and Weber et al. (2009) as providing the ideal conditions for flow to occur.

(iv) Removal of awareness from other thoughts

This element of flow is linked to the ability to concentrate on the task at hand. Because the task requires the individual’s full attention, irrelevant information and thoughts are not allowed to enter the individual’s mind. Consequently, the worries and anxieties that normally enter one’s mind during an activity, which may distract one from the task, become null and void, and have no influence on the individual’s ability to focus on the flow activity (Csikszentmihalyi, 1992).

(v) Experiencing a sense of control

Individuals typically relate their sense of control over the activity in which they experience flow. This sense of control does not necessarily reflect definite success in the task at hand, but rather the possibility of success or control in that particular task. In other words, the individual is aware that there is a chance of failure, but this possibility does not worry the individual - it is enough for the individual to know that there is potential for his/her world to be controlled (Csikszentmihalyi, 1992).

Csikszentmihalyi (1992) addresses this element of control specifically in activities which are considered dangerous yet are enjoyed by some people, such as extreme sports. It would seem paradoxical that one would experience a sense of control in an activity which is intrinsically dangerous, lacking safety nets. However, Csikszentmihalyi (1992) reports that individuals who take part in these extreme activities typically describe an augmented sense of control in these situations. Reference to the experience of flow in these sorts of activities is particularly interesting in the context of ADHD. Csikszentmihalyi (1992) says the following:

> It is usual to explain the motivation of those who enjoy dangerous activities as some sort of pathological need: they are trying to exercise a deep-seated fear, they are compensating, they are compulsively reenacting an Oedipal fixation, and they are “sensation seekers.” While such motives may be occasionally involved, what is most striking, when one actually speaks to specialists in risk, is how their enjoyment derives not from the danger itself, but from their ability to minimize it. So rather than a pathological thrill that comes from courting disaster, the positive emotion they enjoy is the perfectly healthy feeling of being able to control potentially dangerous forces (p.60).

In the context of the above excerpt which describes sensation seeking in flow, it seems that the so-called sensation seeking behaviours which are reported in individuals with ADHD may actually be activities in which the individuals engage so as to experience a sense of control over the given activity. Research indicates that there is a high incidence of sensation seeking behaviours in adults with ADHD (Zimak, 2008).

(vi) Loss of self-consciousness

As previously mentioned, the individual's awareness is completely removed from all stimuli which are not directly related to the flow activity. This extends to awareness of self. During the flow experience, one becomes so involved in the task that one no longer feels separate from the task. This also translates to a reduction in self-consciousness, in that the individual does not feel insecure or threatened in any way (Csikszentmihalyi, 1992).

(vii) A changed sense of time

During a flow experience, people typically describe their changed sense of time. The conventional flow of time is experienced differently - the subjective experience of time changes, in that hours seem to pass like minutes. This may be due to the complete involvement in the task, which results in one losing track of time (Csikszentmihalyi, 1992).

2.3.2 Proposed model of hyperfocus in ADHD

As noted by Csikszentmihalyi (1992), one is most likely to experience flow when one is involved in a task that is challenging, requiring skills that are possessed by the individual, and over which the individual will be able to feel a sense of control. The conditions of hyperfocus which are noted in literature on adult ADHD (Freeman, 2004; Schecklmann et al., 2009; Thompson & Thompson, 2005) seem to correspond quite closely with the conditions of flow described by Csikszentmihalyi. It seems reasonable to then use Csikszentmihalyi's model as the basis upon which hyperfocus in ADHD can be understood. Csikszentmihalyi (1992) further notes that the flow experience is facilitated by the individual receiving immediate feedback whilst engaged in the flow activity, which then spurs the individual on to continue with the task undisturbed. This can be linked to research which found that children with ADHD did not show any deficit in sustained attention as compared to controls when they were provided with frequent reinforcers during an activity (Aase & Sagvolden, 2006). Further, Luman, Oosterlaan and Sergeant (2005) found in a review of the literature that children with ADHD preferred immediate over delayed rewards.

Figure 1 depicts a proposed model of flow in which the components of the flow experience have been reorganised according to how they relate to self-efficacy and sustained attention. Two of the conditions of flow proposed by Csikszentmihalyi (1992) appear to be closely related to self-efficacy, namely: a challenging task that requires skill; and experiencing a sense of control. The degree of an individual's belief in his/her efficacy to exercise control over his/her actions or environment has been shown to affect outcomes of success (Bandura, 1993). Furthermore, research has shown that the degree of perceived self-efficacy influences the difficulty or level of goals which individuals set for themselves, as well as the degree to which individuals will persevere to achieve those goals (Bandura, 1993). Thus, in the context of the flow experience, if one's perceived self-efficacy in a certain task is high, this high sense of self-efficacy creates the platform for the individual to commit fully to participating in the task. This commitment to the task can be understood as a high level of motivation to apply oneself to the situation. Thus, through a perceived sense of self-efficacy in the task at hand, one is motivated to engage in the task fully, and sustain attention. This state of sustained attention is descriptive of the other three conditions of flow, namely: the ability to concentrate on the task; removal of one's awareness from unrelated thoughts; and a diminished awareness of self. With these conditions satisfied, the flow activity can occur, and the individual's sense of time becomes warped. The flow activity provides clear goals and immediate feedback to the individual, which then reinforces the individual's sense of self-efficacy, and hence the continued motivation to sustain attention on the task.

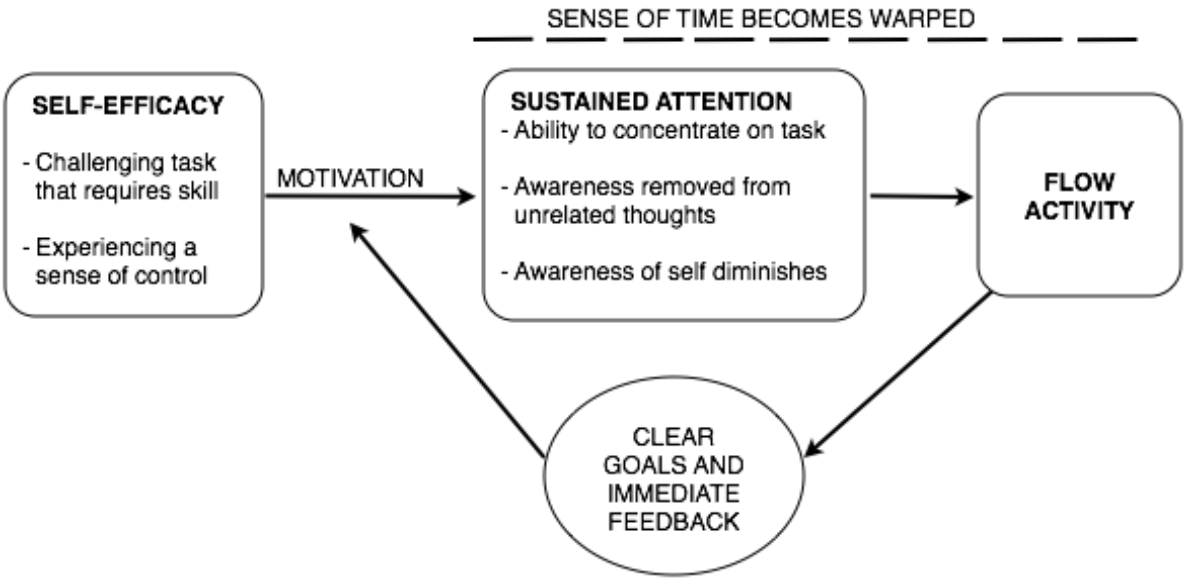

Figure 1: Proposed model of flow in context of self-efficacy

Differences in motivational behaviours have been noted between individuals with ADHD and without ADHD (Ernst et al., 2003 as cited in Schneider at al., 2006). It is further noted by Taylor (1998) that problems in sustaining attention may be a function of motivation, as this 'deficit' only tends to become apparent when one is faced with a task that is unappealing. Taylor (1998) notes that motivation and attention are likely to interact with one another in the context of whether the activity is favourable in the eyes of the individual. The motivational factors which are hypothesised to affect sustained attention in ADHD fit in very nicely with the model of flow outlined above; in the context of ADHD, an individual is hypothesised to be able to sustain attention when a task is deemed by the individual to be relevant, interesting or novel (Thompson & Thompson, 2005). Furthermore, this model supports the notion that immediate feedback and reinforcement enable individuals with ADHD to sustain attention at an equal level as controls (Aase & Sagvolden, 2006).

2.4 Computer games and flow

Based on descriptions of flow provided by Csikszentmihalyi (1992), media settings have been identified as providing the necessary conditions (the balance between challenge and skill) for flow to occur (Sherry, 2004; Weber et al., 2009). Firstly, as required by the theory of flow, media use can be enjoyable. Second, media is often

used “to escape and to forget”, which is linked to the intense concentration and “loss of self-consciousness” associated with the flow experience (Sherry, 2004, p.339). Third, media use has the potential to distort time perception; as Sherry (2004) points out, “video games are notorious time wasters, with players staying up very late at night to conquer the next level” (p. 339). Finally, media use has been found to be at least partially intrinsically motivating (Sherry, 2004). Weber et al. (2009) argue that video games, in particular, provide a unique opportunity for challenge and skill to be balanced, thus making gaming a particularly suitable form of media for producing the flow state. Video games often contain the following elements which are conducive to producing flow; video games typically: (a) have defined goals and achievable rules; (b) contain settings that can be manually or automatically tailored to our proficiency level; (c) provide players with immediate feedback through provision of scores, progress to higher levels, completion of tasks, and the likes; and (d) “have abundant visual and aural information that helps screen out distraction and facilitate concentration” (Sherry, 2004, p.339).

2.5 Neuroanatomy of ADHD

Current thinking describes ADHD as a neurobehavioural disorder with a complex aetiology involving genetic, environmental and biological factors (Biederman, 2005). Although cognitive findings in adult ADHD populations have reflected similarities to the findings in children with ADHD, the body of neurological research is substantially smaller for adult ADHD than for child and adolescent ADHD populations (Biederman et al., 1993, 2004 as cited in Biederman, 2005; Schneider et al., 2006). The differing clinical presentation in adult ADHD as compared to childhood ADHD further highlights the need to expand on the current neurological research on adult ADHD, rather than relying on child ADHD models (Schneider et al., 2006).

Neurological research into ADHD has focused on the brain regions which are related to executive functions and attention networks (Schneider et al., 2006). As such, research has focused on fronto-striatal and fronto-subcortical networks. The cortico-striatal-thalamo-cortical (CSTC) circuits, which are believed to underlie almost all neuropsychiatric disorders, have been implicated in ADHD (Swanson et

al., 1998, as cited in Castellanos, 2001). These multiple parallel circuits link (1) the prefrontal cortex to the input nuclei of the basal ganglia, namely the striatum, which comprises of the caudate nucleus, putamen and nucleus accumbens; then from (2) the output nuclei of the basal ganglia, namely the globus pallidus internal segment (GP) or substantia nigra pars reticulata (Snr), to the thalamus; and then finally (3) from the thalamus nuclei back to the cortex (Alexander et al., 1986, as cited in Castellanos, 2001). These neural circuits facilitate the regulation of behaviours relating to executive functions, behavioural inhibition, motor control and reward systems (Biederman, 2005). The dorsal part of the anterior cingulate cortex (dACC) has also been pinpointed in research on ADHD due to its extensive connections to the forebrain and its key role executive functioning, interference control and reward based decision making (Schneider et al., 2006). Research which has linked these subcortical networks to ADHD supports the hypothesis that functional networks are affected, rather than specific cortical regions (Biederman, 2005; Schneider et al., 2006).

The frontal lobe seems to play a major role in the neurobiology of ADHD. Deficits in the frontal lobe and connected subcortical projections are consistently implicated in neuroimaging, neuropsychological and neurobiological studies of ADHD (Biederman, 2005, Lovejoy et al., 1999). Although the exact nature of the role of frontal lobe function in ADHD is not well understood, frontal lobe function has consistently been linked to executive functions (Biederman, 2005; Schneider et al., 2006). Research of the frontal lobe in ADHD has focused on the ventrolateral and dorsolateral prefrontal cortex (VLPFC and DLPFC) because these regions have been linked to attentional functions (vigilance, selective and divided attention, attention shifting) (Petersen & Posner, 2012).

The dACC is associated with a number of complex cognitive functions, including behavioural inhibition, motivation, performance monitoring, target and error detection, and the modulation of reward-based decision-making (Bush, Valera & Seidman, 2005; Schneider et al., 2006) and is strongly connected to the DLPFC, parietal lobe and striatum. Numerous imaging studies have found abnormalities in the dACC in ADHD populations, making it a key area of interest in ADHD (Bush et al., 2005). Bush et al. (2005) suggest that the dACC's role in motivation and

reward-based decision making may explain “the seeming paradoxical ability of ADHD subjects to perform normally on some tasks (when motivated) but to show deficient performance when the task is not deemed salient” (p. 1275). This observation may point to a possible neurological correlate of hyperfocus.

The parietal cortex has been somewhat neglected in ADHD research, despite its involvement in certain attentional functions (Bush et al., 2005; Petersen & Posner, 2012; Schneider et al., 2006). Findings related to the parietal cortex signal a need for further research into the role of the parietal lobe in ADHD. For example, Cubillo et al. (2012) found in a follow up of medication-naive adults diagnosed with ADHD in childhood exhibited dysfunctions in lateral fronto-striatal-parietal regions during sustained attention.

2.6 Neurophysiology of ADHD

Neuroimaging literature often refers to cortical activity, cortical activation and cortical arousal. These terms are sometimes used interchangeably. In this study, cortical activity/activation is used to refer to any observed activity of the different wave types, such as an increase or decrease in delta waves (Koehler et al., 2009), and cortical arousal is used to refer to the different patterns of neuronal activity, which are correlated with different psychological and behavioural states (“Experiment 32: Electroencephalogram (EEG) wave patterns and cortical arousal”, 2004). Although findings have been mixed in ADHD research, a fair amount of studies support the hypothesis of cortical underarousal in the frontal regions characterised by increased level of slow-wave (theta) activity (Lovejoy et al., 1999; White, Hutchens & Lubar, 2005). This finding has been supported in EEG studies which have demonstrated increased theta activity in individuals with ADHD compared to normal samples throughout development (Bresnahan, Anderson & Barry, 1999; Chabot & Serfontein, 1996; Koehler et al., 2009; Loo & Barkley, 2005). Although theta levels have been found to decrease with age in both normal and ADHD samples, increased theta power has still been observed in adult ADHD samples across resting states (Eyes Closed and Eyes Open) (Chabot & Serfontein, 1996; Bresnahan et al., 1999). Chabot and Serfontein (1996) found that the increased absolute and relative theta power was accompanied by decreased fast wave (alpha and beta waves) mean frequencies in 29.8% of the children in their

study. It must be noted, however, that in a study of resting state EEGs in ADHD across the lifespan, Liechti et al. (2012) found no consistent theta or theta/beta increases. Slow-wave activity (theta and low-alpha) is generally understood to have an inverse relationship with the degree of cortical involvement in a task (i.e. The more slow wave activity, the less cortical involvement in a task); fast wave activity (high-alpha and beta) is positively related to levels of cortical activation (i.e. the more fast wave activity, the higher the cortical activation) (White et al., 2005).

2.7 Neurophysiology of hyperfocus

Dietrich (2004) provides a theory of flow based on theories of information processing in cognitive neuroscience. It has been suggested that there are two means of processing information: 'Explicit' information processing is a conscious process whereby rules govern the acquisition of knowledge, and processing relies on a conscious effort on the part of the individual. The content of the explicit system can be communicated verbally. Research seems to indicate that the prefrontal regions control explicit information processing (Ashby & Casale, 2002; Dehaene & Naccache, 2001; Dietrich, 2003, all cited in Dietrich, 2004). Conversely, the implicit information processing system is not governed by rules, but rather by one's actual experience and performance of a given task. The content of implicit processing is not readily accessible to one's conscious awareness (Ashby & Casale, 2002; Dienes & Perner, 1999; Schacter & Bruckner, 1998, all cited in Dietrich, 2004). Dietrich (2004) posits that flow occurs through a top-down process, in which the skills required for a specific task are acquired through the explicit system, and become implicit through practice, and suggests that the automatic process associated with the experience of flow is made possible by the temporary suppression of frontal function, "a state of transient hypofrontality" in which the explicit system is inhibited and the task is governed by the implicit system (p.757). Weber et al. (2009) argue against Dietrich's theory of hypofrontality underlying the flow experience, and posit that there is significant evidence from brain imaging studies which demonstrates increased prefrontal activity when focused attention is in use (Raz & Buhle, 2006, as cited in Weber et al., 2009).

In a study by Schecklmann et al. (2009), higher performance was found to be correlated with decreased cortical activation - ADHD participants exhibited higher verbal fluency in comparison with matched controls despite diminished cortical activation. This led Schecklmann et al. (2009) to suggest that decreased cortical activation does not necessarily signify a deficit, but may also be indicative of enhanced cognitive function, implying that as individuals master a task or find the task less challenging, less cognitive effort is required. Schecklmann et al. (2009) suggest that this may point to an explanation for hyperfocus, but recommend further research. The proposal that decreased cortical activity is linked to enhanced performance is supported by other research. For example, according to White et al. (2005), elevated alpha wave levels imply increased cortical activation in the region of interest, and may be indicative of individuals needing to invest more effort to maintain performance during a cognitive activation task.

2.8 EEG models in ADHD research

As has been mentioned previously, this research is based on electrophysiology (EEG) recordings. The literature indicates that there are various ways in which data can be recorded and reported in EEG research. The majority of EEG studies in children with ADHD have been based on recordings taken during an eyes-closed resting state, and have yielded relatively consistent findings. In contrast, there are very few studies that have examined resting states in older individuals with ADHD, albeit with inconsistent results. Additionally, there are few studies that have investigated the relationships between resting state EEG findings and performance on attention and cognitive tasks; the few studies that have been conducted in this vein are limited to children and adolescents, and are inconsistent in their findings (van Dongen-Boomsma et al., 2010). In addition to the disparate findings in resting state EEG studies of aADHD, it is questionable whether EEG recordings in resting states in ADHD can be generalised to describe the cortical activity involved in cognitive activities requiring attention. The symptoms of ADHD manifest behaviourally in individuals in a variety of settings whilst they are engaging in daily tasks which require attention, and not specifically during resting states. It thus seems unlikely that an EEG reading taken solely during a resting condition is adequately related to the cortical activity which may occur during an activity requiring attention.

2.9 Conclusion

There is a consensus in the literature that attention is a multifaceted construct governed by many interlinked processes. However, this complexity has not come through adequately in the ADHD literature, where inattention is often oversimplified to connote the inability to sustain attention. Reports of hyperfocus occurring in individuals with ADHD seem to imply that it is possible for individuals with ADHD to sustain attention in specific settings, when the individual is engaged in a task that is enjoyable. This possibility requires that the definition of inattention be revised and expanded. Flow theory provides a suitable framework for understanding the conditions which may predispose an individual to hyperfocus in certain situations. This framework seems to complement existing literature on ADHD that addresses the role that motivation plays in the ability of individuals with ADHD to sustain attention.

The previous chapter explored the many ways in which ADHD has been conceptualised in the past. Whether ADHD symptoms were attributed to brain damage, familial conflict or an inherent dysfunction in the individual, the uniting factor in all approaches was that ADHD was viewed as a liability, a ‘disorder’. Some authors view ADHD in a manner that reframes it in a positive framework. For example, Hartmann (1997) views ADHD from an evolutionary perspective, describing the ADHD personality as having descended from hunters. In this context, hyperfocus is conceptualised as the ability for the hunter to focus in his prey and block out all extraneous stimuli. In line with this, hyperfocus considered a positive symptom of ADHD, as it affords one a pronounced ability to sustain attention in certain settings. However, if the occurrence of hyperfocus does have drawbacks that impact negatively on the individual’s functioning (such as disengaging from one’s surroundings and loss of time perception), then this needs to be looked at more closely in terms of the symptoms experienced by people with ADHD. Before deciding on the implications of hyperfocus in ADHD, however, we need to determine if it is indeed a symptom that occurs frequently in ADHD.

The dACC, an area implicated in ADHD, has been pinpointed as an area which may be implicated in the occurrence of hyperfocus. Because the dACC is a subcortical region, its activity cannot be examined directly using electroencephalography, and would require more sophisticated brain-imaging equipment such as functional magnetic resonance imaging. However, the exploratory nature of this study does not justify the use of such costly equipment. As a starting point for exploring possible neurobiological correlates of hyperfocus, the frontal cortex and parietal cortex were identified as areas of interest, due to their strong connections with the dACC.

CHAPTER 3: METHODOLOGY

3.1 Introduction

This chapter describes the research design and methodology employed in the study, and provides justification for the selection thereof. The first section outlines the sampling method used and participant information. The next section describes the instruments and equipment used. Thereafter the procedure followed for screening and data collection is explained. Methods of data analysis are then explained. The chapter concludes with a discussion of the ethical considerations that were relevant to the study.

In order to meet the aims of the study (as discussed previously), a quantitative method was utilised, or more particularly, a quasi-experimental research strategy. An experimental research design aims to establish whether there is a causal relationship between variables. For a research design to be considered experimental, certain conditions need to be met: one variable (the independent variable) must be manipulated to create two or more treatment conditions; a second variable (the dependent variable) must be measured in each treatment condition; the scores in each treatment condition are compared for consistent differences; and all extraneous variables are controlled for (Gravetter & Forzano, 2009). A research design is classified as quasi-experimental when there are factors inherent in the study which prevent the researcher from adhering to the full level of control of a 'true experiment'. This prevents the researcher from drawing an unambiguous conclusion about the causal relationship between variables. Typically, a quasi-experimental study "uses a nonmanipulated variable or a noncontrolled variable in place of the manipulated variable used in a true experiment" (Gravetter & Forzano, 2009, p.133). In this study, two treatment conditions were required: (1) adults high in ADHD symptoms (ADHD group/'ADHDs'), and (2) adults low in ADHD symptoms (non-ADHD group/'non-ADHDs'). Because the assignment of participants to each group was predefined by the variable 'ADHD symptoms', the researcher could not control to which group the participants would be assigned. This study is therefore classified as quasi-experimental. The researcher utilised a non-equivalent groups

(in terms of the independent variable ‘ADHD’) repeated measures pre-test post-design.

The following hypotheses were addressed in this study:
Between-groups:
1) There will be significant differences in cortical activation in the frontal lobe, parietal lobe and frontal midline between the ADHD group and the non-ADHD group during resting state.
2) There will be significant differences in cortical activation between the ADHD group and the non-ADHD group in the frontal lobe, parietal lobe and frontal midline in the hyperfocus state.

Within-groups:
3) Adults with ADHD exhibit significant differences in cortical activation patterns in the frontal lobe, parietal lobe and frontal midline in resting state as opposed to in an activity in which hyperfocus has been induced.
4) Adults without ADHD will not exhibit significant differences in cortical activation patterns in the frontal lobe, parietal lobe and frontal midline in resting state as opposed to in an activity in which hyperfocus has been induced.

3.2 Sampling and participants

Nonprobability sampling methods were used to recruit participants for the study. Sampling methods employed included initial convenience and snowball recruiting, but purposive sampling was used for the final two groups. An important aspect of sampling is the degree to which the sample selected is representative of the population from which it is drawn. A sample can be considered representative if the characteristics of the sample reflect the characteristics of the population being studied (Kerlinger, 1986, as cited in Strydom & Venter, 2002). For the purposes of this study, the sample needed to comprise of adults who were rated as high in ADHD symptoms and adults who were low in ADHD. The use of probability sampling methods is generally preferred in quantitative studies, as this improves the extent to which results can be generalised to the target population (Heppner & Heppner, 2004). However, due to the high degree of specificity of the sample needed for the study, as well as the exclusion criteria which are suggested for EEG

studies, it was decided that nonprobability sampling methods would be more effective.

Participants were recruited from two primary sources, namely the undergraduate student population at the University of Johannesburg (UJ) and via social media channels (i.e. Twitter). The final sample was selected purposively from a participant pool of 577 undergraduate students at UJ and nine respondents from Twitter (N=586), according to specific selection and exclusion criteria (to be discussed in the procedure section). The sample consisted of five adults in the ADHD group (the 'ADHDs'), and five adults in the non-ADHD group (the 'non-ADHDs'). Participants were classified as 'high' or 'low' in ADHD symptoms using the Adult ADHD Self-Report Scale V1.1 Screener (Adler, Kessler & Spencer, 2003a) according to the four-stratum classification system recommended by Kessler et al. (2007) (to be discussed in section 3.3.1). Four participants in the ADHD group fitted into the lower positive stratum (14-17) and one fitted into the higher positive stratum (18-24). In the non-ADHD group, four participants fitted into the lower negative stratum (0 − 9), and one participant was in the higher negative stratum (10 − 13). Six (60%) out of 10 participants were UJ students and four (40%) social media respondents. Participants' ages ranged from 20-31 years ($\overline{x}$ = 24.25 years; SD = 3.59 years). Each group consisted of three (60%) male and two (40%) female participants.

3.3 Measures

There were two categories of measurement utilised in this study. The first category included instruments used for the selection and screening of participants, and the second category the instruments used for data collection. These measures are described below.

3.3.1 Adult ADHD Self-Report Scale-Version 1.1 (ASRS-V1.1)

The Adult ADHD Self-Report Scale (ASRS) (Adler, Kessler & Spencer, 2003b) is a World Health Organization instrument which has been based on the DSM-IV-TR ADHD criteria and was adapted for use with adult populations (Murphy & Adler, 2004). The instrument consists of 18 questions, in which the first six questions act as a screener for symptoms of inattention, hyperactivity and impulsiveness (Part A) (Murphy & Adler, 2004) and the remaining 12 questions measure the frequency of

symptoms (Part B). The items in Part A have been used in an abridged version of the ASRS, the Adult ADHD Self-Report Scale-V1.1 (ASRS-V1.1) Screener (Adler et al., 2003a), which is a more efficient screener for adult ADHD due to its brevity. Because the ASRS was used in this study as a screening instrument, it was only necessary to use the scores in Part A for selecting participants. Items are rated on a four-point Likert-type scale (Never/rarely = 0, Sometimes = 1, Often = 2, Very often = 3). Two approaches are used for scoring responses on the ASRS-V1.1. In the first approach, a participant is given a score from 0 to 6 based on the pattern of responses in part A. In the second approach, a participant is given a score of 0-24 based on the responses on the Likert scale. As recommended by Kessler et al. (2007), the 0 - 24 scoring approach was used in this study, with a four-stratum classification of the scores (0 − 9; 10 − 13; 14 − 17; and 18 − 24). The two lower strata are effective in identifying individuals without ADHD, and the two higher strata are used for identifying individuals with ADHD. The higher the score, the higher the probability is that the individual has ADHD.

The ASRS-V1.1 has been shown to outperform the full ASRS in identifying individuals with ADHD (Kessler et al., 2005). Kessler et al. (2007) found a test retest reliability of 0.58-0.77 and internal consistency reliability in the range of 0.63-0.72. Kessler et al. (2007) attribute the lower estimates of reliability to the method of item selection used for the ASRS Screener (i.e. stepwise logistic regression) and recommend that evaluating the concordance of the ASRS screener with clinical diagnoses is a more effective method of evaluating the instrument's utility. To measure concordance, the area under the receiver operating characteristic curve (AUC) was used, a calculation that "can be interpreted as the probability that a randomly selected clinical case would score higher on the ASRS Screener than a randomly selected non-case" (Kessler et al., 2007, p. 56). The AUC was found to be 0.90 (Kessler et al., 2007). Murphy and Adler (2004) report acceptable internal consistency alpha values. Burke, Austin and Waldeck (2011) found an overall internal consistency value of 0.886 for the scale in the South African context. Based on the psychometric evidence, it seems that the ASRS-V1.1 Screener can be an effective tool in discriminating between ADHD and non-ADHD cases.

3.3.2 Biographical questionnaire

A biographical questionnaire was designed by the researcher based on the exclusion criteria formulated for the study (see Appendix A). These exclusion criteria were informed by the studies of White et al. (2005) and Karch et al. (2010) to screen participants for characteristics which may confound the results of an EEG (see discussion in 3.4.1). Potential participants were required to complete this questionnaire as part of the screening process to ensure their suitability for the study. Questions covered demographic details of participants, education and employment, medical history and video/computer game habits.

3.3.3 BIOPAC MP150 Data Acquisition System

The BIOPAC MP150 Data Acquisition System (BIOPAC Systems, Inc., Goleta, CA) is a computer-based system which can be used to acquire physiological data for a broad range of applications such as electrocardiograms (ECG), electrodermal activity (EDA) and electroencephalograms (EEG). Incoming signals are converted by the MP System into digital signals and stored on the computer to be processed by the researcher (BIOPAC Systems, Inc., n.d.). The MP150 System consists of 16 analog and 16 digital channels that can record individual signals simultaneously. For this study, the BIOPAC MP150 System was used to collect EEG data in participants. A CAP100C electrode cap was connected to the BIOPAC MP150 system and fitted to participants. The electrode cap consists of 19 tin electrodes which have been positioned according to the international 10/20 montage. This system minimises errors in positioning the electrodes correctly for measurement of electrical activity for specific cortical areas (BIOPAC Systems, Inc., n.d.).

The figure below illustrates the placement of the electrodes for the measurement of three cortical areas (frontal, frontal midline, parietal):

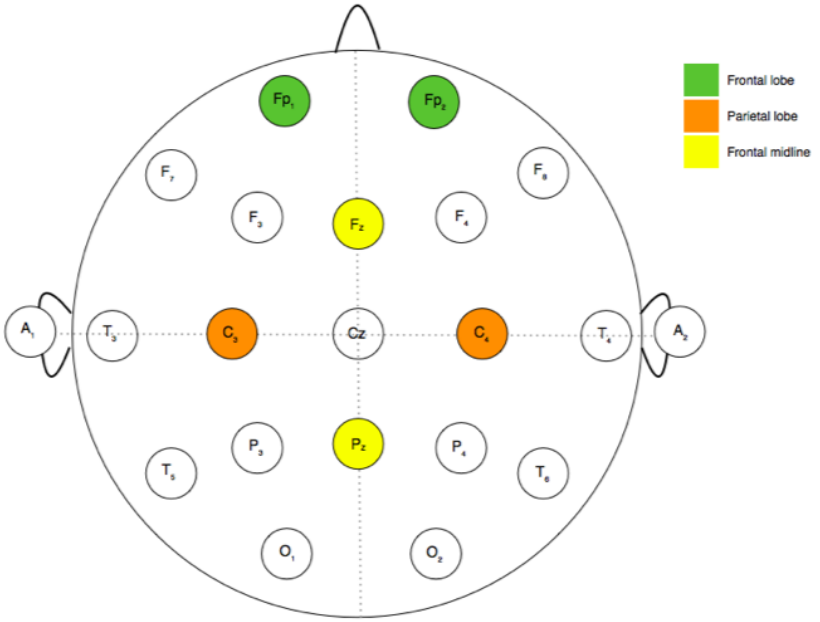

Figure 2: 10-20 montage for electrode placement in electroencephalography (adapted from diagram in MP system hardware guide (BIOPAC Systems, Inc., n.d.)

3.3.4 Cognitive activation procedure

A cognitive activation procedure that was likely to 'induce' a state of hyperfocus was selected. As recommended by Weber et al. (2009), a tactical shooter PC game was selected for its ability to provide the conditions described by Csikszentmihalyi (1990) as conducive for the occurrence of flow or hyperfocus. The game used is called Conflict: Desert Storm (Gotham Games, 2002). The game is a tactical shooter game in which the player must enter a combat zone to rescue a fellow soldier and destroy a bridge. The player must concentrate on operating the controls to navigate through the combat zone, while defending him/herself from enemy fire. Further, the player needs to try to find the captured soldier, as well as complete the mission of destroying the bridge.

3.4 Procedure

The procedure can be divided into two phases. The first phase includes the screening procedure that was used to select suitable participants. The second

phase consists of the procedure followed in the collection of the EEG data, which in itself consists of three stages.

3.4.1 Phase 1: Screening

As discussed in section 3.2, participants were recruited from within the UJ population, as well as through social media channels and snowball sampling. Differences in communication channels necessitated a slightly different recruitment process for each group.

As part of a broader research project that was being conducted at UJ, an online questionnaire was made available to UJ students to complete through the student portal by a certain date. The questionnaire covered demographic details (e.g. age, ethnicity, level of education), interest in computer games, any history of an ADHD diagnosis, as well as the full version of the ASRS. Students were also asked to indicate whether they would be willing to participate in further research. The researcher contacted students who had indicated an interest in computer games as well as a willingness to participate in further research to invite them to participate in her study. In addition to the UJ students, the researcher recruited volunteers through social media channels (Twitter), asking for individuals who enjoyed playing computer games who were interested in participating in the study to contact her. Interested respondents from both Twitter and UJ students were sent the following documentation by email: (i) participant information sheet (document describing the study and what would be required of participants if they chose to consent to take part in the study - see Appendix B); (ii) informed consent (see Appendix C); the ASRS (with the exception of UJ students who had already completed this previously); and (iv) the biographical questionnaire (described in section 3.3; see Appendix A). Once all forms were completed and returned, the researcher scored the responses of Part A of the ASRS according to the 0 − 24 four-strata scoring method (Kessler et al., 2007). As an extra measure, the mean and standard deviation (SD) of all respondents' Part A scores were calculated ($\overline{x}$ = 10.21; SD = 3.56). Participants were shortlisted for the study if they obtained scores at least one standard deviation above or below the mean (ADHDs: 13.77 or higher; non-ADHDs: 6.65 or lower). In relation to the four strata identified by Kessler et al. (2007), the

ADHD group's scores corresponded with the two higher strata (14-17; 18-24) and the non-ADHDs' scores corresponded with the lowest stratum (0 – 9).

Further screening was carried out using the following exclusion criteria which were informed by other EEG studies (Barry, Clarke & Johnstone, 2003; White et al., 2005; Bekker et al., 2005):

i) Age: Individuals below the age of 19 years were excluded.
ii) Medication: Individuals who were on chronic psychiatric medication, with the exception of methylphenidate (Ritalin), were excluded. In the case of individuals on Ritalin, they were asked to refrain from taking the medication for a minimum of 12 hours prior to the assessment.
iii) History of a neurological disorder or head injury.
iv) History of substance abuse in the previous two months.
v) Evidence of another Axis I or Axis II disorder

Participants were then assigned either to the ADHD or non-ADHD group and contacted to arrange an appointment.

3.4.2 Phase 2: Data collection

Participants from both groups underwent the same procedure. Once participants had confirmed an appointment for an EEG, they were emailed information regarding what activities to avoid prior to attending their appointment (e.g. consuming caffeine, smoking, etc. - See Appendix D). Upon arrival for his/her appointment, the participant was required to complete a brief questionnaire confirming whether he/she had refrained from the activities which may affect EEG readings (see Appendix E). The researcher then explained to the participant the process that would be followed. The electrode cap was fitted onto the participant's head, and electro-gel used to create contact between the electrodes and the participant's scalp. The participant was asked to confirm that he/she could feel the electro-gel on his/her scalp for each electrode when the researcher pressed the electrode down. If the participant reported that he/she could not feel the gel for any of the electrodes, more gel was applied, and each electrode was checked a second time.

The participant was asked to sit comfortably and to refrain from moving around too much during the procedure. Two baseline EEG readings were taken, first with the participant's eyes closed (EC), and then with the participant's eyes open (EO). The participant was then shown the computer game and was given a printout of the game controls to refer to whilst playing. The participant was instructed to complete the 'basic training' session in the game as a means of familiarising him/herself with the game controls. No recording was taken during the training session. Once the participant had completed the basic training, he/she was instructed to play the game until told to stop, and to restart if he/she was 'killed'. Each participant played the game for approximately 35-40 minutes, during which two separate readings were taken. After being asked to stop playing, the participant was asked to complete a brief questionnaire. This questionnaire aimed to examine the distorted time perception in flow activities described by Csikszentmihalyi (1992) and thus included items focused on the participant's perception of time and awareness of his/her surroundings whilst playing the game (see Appendix F). Because the researcher wanted to examine whether there was a distorted perception of time, participants were left to continue playing the game for approximately 10 minutes after the second reading was completed, and participants were not aware of how long they would play the game.

Table 3 provides an outline of the procedure followed with each participant. The researcher sat behind the participant and took observational notes to help with removing contaminated data from the EEG readings - any sudden movements or events such as the character dying were recorded. The total time spent by each participant playing the game was also recorded.

Table 3

Data collection procedure

Activity	**EEG Reading**	**Length of time**
Eyes closed (EC)	Yes	3 minutes
Eyes open (EO)	Yes	3 minutes
Basic Training	No	10-20 minutes
Start game (Game 1)	Yes	3 minutes
Continue game	No	3 minutes
Continue game (Game 2)	Yes	3 minutes
Continue game	No	10 minutes
Post-game questionnaire	No	2 minutes
Total time		**+/-35 − 40 minutes**

3.5 Data analysis

3.5.1 EEG data

EEG data were recorded and transformed by means of the Acqknowledge 3.9.1 software (BIOPAC Systems, Inc, n.d.). The data files for all the participants were coded by the supervisor to ensure that data extraction and analysis performed by the researcher was conducted blindly. The EEG data analysis process comprised of two stages: (i) Data extraction and (ii) Statistical analysis.

(i) Data extraction

Approximately 12 minutes of data were recorded for each participant (3 minutes per condition). Data was transformed using the Acqknowledge software with the digital filters set at 0.001Hz to 25Hz. For each condition, nine epochs of data, 20 seconds apart, were extracted (i.e. at 20s, 40s, 60s, 80s, 100s, 120s, 140s, 160s, and 180s) for the frontal, frontal midline and parietal regions. A frequency graph was produced for each epoch by computing a logarithm function, followed by a Fourier transformation/power spectral density. A power score was then determined for each frequency band. The frequency bands used were: delta (0 − 4 Hz), theta (4 - 8 Hz), alpha (8 - 13 Hz) and beta (13 - 30 Hz) (Alaraj, Fukami & Ishikawa, 2012).

The power scores can either be reported as is (i.e. absolute power) or can be transformed to be reported as follows:

- Relative power: The percentage of power in any band compared with the total power in the patient's EEG (e.g. "relative theta" is the percentage of theta of the combined sum of delta, theta, alpha, and beta).
- Theta/beta ratios: The percentage of power in theta divided by the percentage of beta. This is a slow-to-fast wave relationship measurement.

(ii) Statistical analysis

Non-probability sampling was used and added to the fact that the sample size was small, it was assumed that the results cannot be generalised to the larger population of ADHDs or the larger population of non-ADHDs. Furthermore, due to the sampling, it was assumed that the dependent variables under investigation were not normally distributed in the sample, therefore, despite the fact that the measurement of the independent variables was on an interval scale, non-parametric statistical techniques were used to analyse the data. The following statistical analyses were performed on the data, more specifically the absolute power scores as well as the relative power scores for the different waves and the theta: beta ratios:

- Descriptive statistics: Mean rank scores, means and standard deviations were calculated and reported
- Within-group differences: In order to determine whether there were significant differences in the independent variables across the different conditions, the Wilcoxon Signed Rank test was calculated.
- Between-group differences: In addition to the within-group analyses, it was also necessary to determine to what extent the ADHD group differed from the non-ADHD group in terms of the independent variable scores for the different conditions. The Mann-Whitney-U test was performed to determine whether the scores differed significantly.
- Effect size: In addition, the effect size was calculated, in order to establish the size of a statistically significant difference. Cohen's *d* was calculated (see Tables 6, 7, 11, 12, 16 & 17) by means of the following formula (Cohen, 1988; 1992):

$$d = \frac{\bar{x}_t - \bar{x}_c}{\sqrt{\frac{(n_t - 1)s_t^{\,2} + (n_c - 1)s_c^{\,2}}{n_t + n_c}}}$$

> Cohen (1992) states that *d* = 0.2 is indicative of a small effect size, *d* = 0.5 a medium effect size and *d* = 0.8 a large effect size.

Although the data was on an interval scale, the sample size was small, suggesting that the variables were not normally distributed in the sample. This necessitated the use of non-parametric statistics. Between-group differences were evaluated using the Mann-Whitney U test.

An EEG reading was taken for each of the different conditions in order to analyse cortical activity changes across different experimental conditions for each group. The Wilcoxon Signed-Rank test was used to analyse within-group analysis of differences in brainwave activity between the different stages.

3.5.2 Survey questionnaire

The mean time spent playing the game was calculated for each group, as well as the mean perceived time spent playing. The mean discrepancy between the two calculations was calculated and compared.

3.6 Ethical considerations

This project forms part of a greater ADHD research project for which ethical clearance was given by the faculty ethics committee. All the participants in this study were of a consenting age and could therefore agree to participate in the study. Participants were also told that they could withdraw from the study at any stage without prejudice. The participants all received detailed information about the procedures, but not the aim of the study as this may have influenced their performance. They were however informed of the aim of the study on completion of the procedures. All participants agreed to participate in the study by signing a consent form (see Appendix C). All participants were given the option of receiving feedback of their results after completion of the study.

3.7 Conclusion

In order to answer the research question, a quasi-experimental design, as described, was designed, using the BIOPAC Data Acquisition System to assess

cortical wave activity in selected areas for an ADHD group and a non-ADHD group. In order to compare these two groups, EEG data was extracted and transformed and statistical analysis was performed. The results of the statistical analysis will be reported in chapter 4.

CHAPTER 4: RESULTS

4.1 Introduction

In this chapter, the results of the EEGs are presented and described. The results are divided according to cortical regions, namely the frontal lobe, followed by the frontal midline and finally the parietal region. For each cortical region, the following results are presented for ADHD and non-ADHD: (1) the mean power scores and standard deviation calculated for each experimental condition (Eyes Closed (EC), Eyes Open (EO), Game 1, Game 2) per wave; (2) the between-group differences in absolute and relative wave activity across conditions; and (3) the within-group differences in cortical activity during the progression from one condition to the next. The significant results for each region are highlighted (where $p < 0.05$). There are instances where results were not statistically significant, but where interesting patterns are noted. The instances where results were significant at $p < 0.1$ are also noted as areas for further investigation. Thereafter, the survey questionnaire results, which provide context to some of the EEG results, are presented.

The following definitions are applicable in understanding the results presented in this chapter:

- Absolute power: The actual power (power score) in the patient's EEG database (Power is microvolts squared).
- Relative power: The percentage of power in any band compared with the total power in the patient's EEG (e.g. 'relative theta' is the percentage of theta of the combined sum of delta, theta, alpha and beta).
- Theta/beta ratios: The percentage of power in theta divided by the percentage of beta. This is a slow-to-fast wave relationship measurement.

4.2 Frontal lobe

4.2.1 Descriptive statistics

4.2.1.1 Mean power scores in the frontal lobe

Table 4 summarises the mean power scores found for each group across conditions in the frontal region. These values are displayed in the figures that follow according to wave type, to demonstrate the different patterns of cortical activation between the two groups.

Table 4

Mean power scores per wave and condition for the frontal lobe

		ADHD		Non-ADHD	
Wave	**Condition**	**Mean**	**SD**	**Mean**	**SD**
Delta	EC	.0129	.0113	.0046	.0012
	EO	.0187	.0072	.0143	.0054
	Game 1	.0213	.0177	.0108	.0035
	Game 2	.0190	.0150	.0146	.0025
Theta	EC	.0217	.0199	.0071	.0015
	EO	.0315	.0173	.0170	.0045
	Game 1	.0317	.0225	.0186	.0056
	Game 2	.0267	.0153	.0228	.0051
Alpha	EC	.0142	.0134	.0047	.0009
	EO	.0200	.0119	.0091	.0013
	Game 1	.0203	.0148	.0130	.0046
	Game 2	.0169	.0087	.0147	.0039
Beta	EC	.0078	.0072	.0026	.0006
	EO	.0123	.0091	.0078	.0030
	Game 1	.0127	.0104	.0071	.0030
	Game 2	.0120	.0086	.0085	.0022

The delta wave activity for the two groups is demonstrated in Figure 3. During the EC stage, non-ADHDs had lower delta waves than ADHD ($\bar{x}_{ADHD}$ = .0129; $\bar{x}_{nADHD}$ = .0046), with both groups showing increased delta wave activity in EO, but a more visible difference in non-ADHDs ($\bar{x}_{ADHD}$ = .0187; $\bar{x}_{nADHD}$ = .0143). From EO to Game

1, non-ADHDs showed a decrease in delta wave activity ($\overline{x}_{nADHD}$ = .0108), whereas ADHDs showed a slight increase in delta wave activity ($\overline{x}_{ADHD}$ = .0213). From Game 1 to Game 2, ADHDs showed a decline in delta wave activity for the first time ($\overline{x}_{ADHD}$ = .0190), whereas non-ADHD showed an increase in delta wave activity ($\overline{x}_{nADHD}$ = .0146).

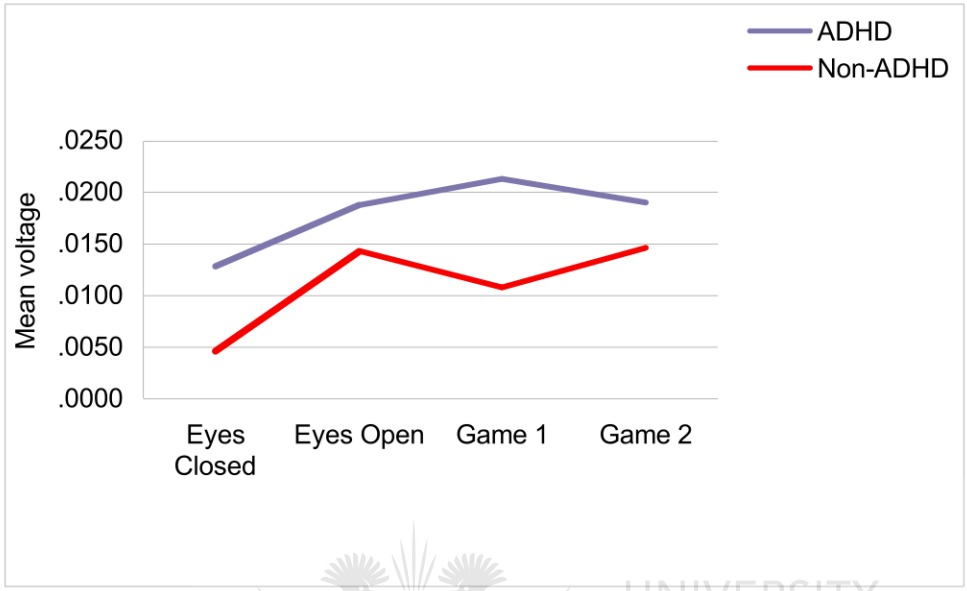

Figure 3: Delta wave mean power score in frontal lobe across conditions

The mean power scores for theta waves in the frontal lobe across conditions are displayed in figure 4. ADHDs displayed consistently higher theta wave activity than non-ADHDs in the frontal lobe. At EC, the mean power score for ADHDs was .0217, compared to .0071 in non-ADHDs. Both groups had increased theta waves from EC to EO ($\overline{x}_{ADHD}$ = .0315; $\overline{x}_{nADHD}$ = .0170). From EO to Game 1, both groups showed only a slight increase in theta waves, with ADHDs only increasing to .0317 and non-ADHDs increasing to .0185. From Game 1 to Game 2, ADHDs showed a decrease in theta waves for the first time ($\overline{x}_{ADHD}$ = .0267), and non-ADHDs showed a slightly larger increase than before ($\overline{x}_{nADHD}$ = .0228).

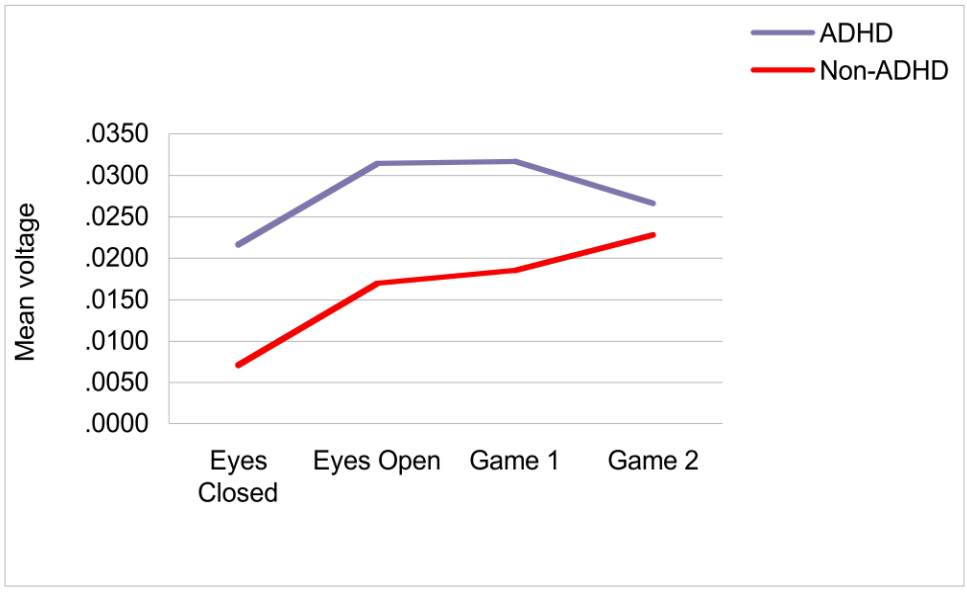

Figure 4: Theta wave mean power score in frontal lobe across conditions

Figure 5 shows the mean alpha wave activity per condition in the frontal lobe. ADHDs exhibited higher alpha wave activity than non-ADHDs in all four conditions. At EC, the mean power score for ADHDs was .0142, whereas the mean power score for non-ADHDs was .0047. From EC to EO, both groups showed increased alpha wave activity, with ADHDs' mean power score calculated as .0200, and non-ADHDs' mean as .0091. From EO to Game 1, ADHDs' alpha waves only increased marginally ($\overline{x}_{ADHD}$ = .0203), whereas non-ADHDs exhibited a steady increase in alpha waves ($\overline{x}_{nADHD}$ = .0130). From Game 1 to Game 2, ADHDs' alpha wave activity decreased for the first time ($\overline{x}_{ADHD}$ = .0169), and non-ADHDs' alpha waves continued to increase, although to a slightly lesser extent ($\overline{x}_{nADHD}$ = .0147).

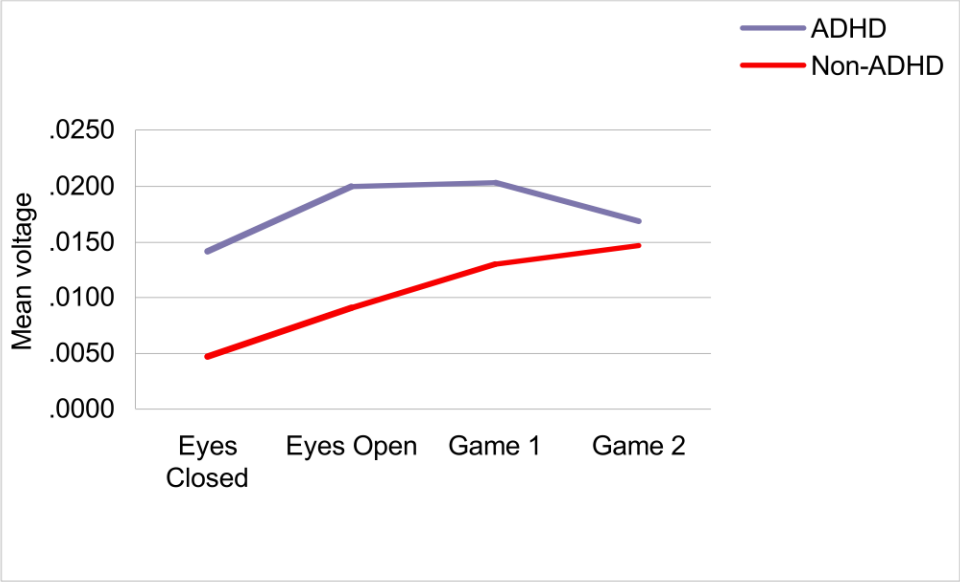

Figure 5: Alpha wave mean power score frontal lobe across conditions

Mean beta wave values in the frontal lobe are displayed in Figure 6. ADHDs exhibited higher beta waves than non-ADHDs through all four conditions. At EC, ADHDs had a mean power score of .0078, whereas non-ADHDs' mean beta power score was .0026. From EC to EO, both groups' beta waves increased, with the ADHDs having a mean power score of .0123 and non-ADHDs a mean power score of .0078. From EO to Game 1, ADHDs' beta waves only increased slightly ($\bar{x}_{ADHD}$ = .0127), whilst non-ADHDs' beta waves decreased ($\bar{x}_{nADHD}$ = .0071). From Game 1 to Game 2, non-ADHDs' beta waves increased again ($\bar{x}_{nADHD}$ = .0085), whereas ADHDs' beta waves decreased for the first time ($\bar{x}_{ADHD}$ = .0120).

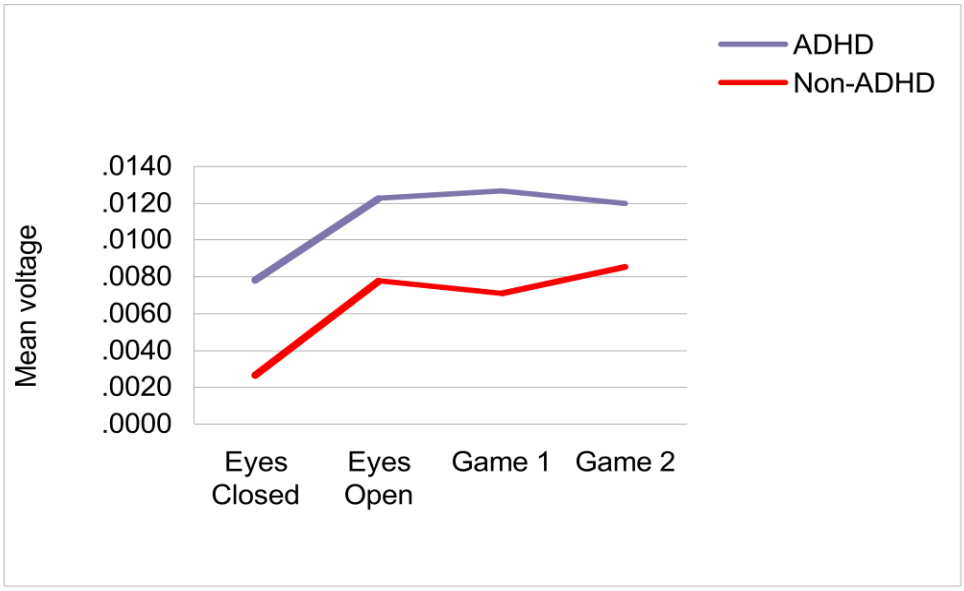

Figure 6: Beta wave mean power score in frontal lobe across conditions

4.2.1.2 Mean relative wave activity in the frontal lobe

Table 5 displays the mean relative wave activity in the frontal lobe for the two groups across the four conditions.

For ADHDs, mean relative delta was 13.90% at EC, 16.83% at EO, 16.06% at Game 1 and 16.67% at Game 2. For non-ADHDs, mean relative delta was 14.12% at EC, 16.23% at EO, 13.82% at Game 1 and 13.85% at Game 2. One can note that the mean relative delta activity for the ADHDs was initially less than that of non-ADHDs at EC, but that mean relative delta activity was greater in the ADHDs than non-ADHDs at EO, Game 1 and Game 2.

Theta waves were more prominent than the other wave types for both groups across all conditions. Relative theta values were quite similar between the two groups for each condition. For the ADHDs, relative theta was 41.32% at EC, 40.99% at EO, 41.65% at Game 1, and 42.04% at Game 2. For non-ADHDs, relative theta was 41.27% at EC, 41.71% at EO, 41.85% at Game 1 and 40.91% at Game 2.

Relative alpha values differed slightly between the two groups. For ADHDs, relative alpha was 32.42% at EC, 30.31% at EO, 30.08% at Game 1 and 29.77% at Game 2. For non-ADHDs, mean relative alpha was 32.53% at EC, 29.99% at EO, 32.04% at Game 1 and 32.49% at Game 2.

Relative to the other wave types, beta waves were consistently the least active across all conditions for both groups. For ADHDs, mean relative beta was calculated to be 12.37% at EC, 11.88% at EO, 12.20% at Game 1 and 11.52% at Game 2. For non-ADHDs, the mean relative beta was 12.09% at EC, 12.06% at EO, 12.29% at Game 1 and 12.74% at Game 2.

Table 5

Relative wave activity across conditions in the frontal lobe

		ADHD		Non-ADHD	
Wave	**Condition**	**Mean**	**Sd**	**Mean**	**Sd**
Delta	EC	.1390	.0132	.1412	.0108
	EO	.1683	.0605	.1623	.0299
	Game 1	.1606	.0277	.1382	.0140
	Game 2	.1667	.0505	.1385	.0092
Theta	EC	.4132	.0153	.4127	.0181
	EO	.4099	.0203	.4171	.0257
	Game 1	.4165	.0108	.4185	.0104
	Game 2	.4204	.0042	.4091	.0144
Alpha	EC	.3242	.0165	.3253	.0134
	EO	.3031	.0437	.2999	.0392
	Game 1	.3008	.0167	.3204	.0065
	Game 2	.2977	.0388	.3249	.0139
Beta	EC	.1237	.0044	.1209	.0062
	EO	.1188	.0040	.1206	.0092
	Game 1	.1220	.0148	.1229	.0042
	Game 2	.1152	.0125	.1274	.0057

4.2.2 Between-group differences in the frontal lobe

The mean scores for each group are summarised below in terms of the different waves during each condition. In each group, the mean relative wave activity in the frontal lobe was calculated for each wave type. This was done by dividing the mean wave activity of each wave type by the total wave activity in each condition (e.g. mean delta for EC divided by total mean wave activity at EC).

4.2.2.1 Absolute values

Table 6 summarises the mean rank scores for the two groups per wave and condition in the frontal lobe. No significant differences were found between the two groups for any wave type or condition.

Table 6

Between group differences for the absolute power scores of all waves for the frontal cortical site

Wave	**Condition**	**Mean Rank**		***z***	***p***	**Cohen's d**		
		ADHD	Non-ADHD			d	Effect size	Category
Delta	EC	6.20	4.80	-.731	.465	1.32	0.25	Small
	EO	6.60	4.40	-1.149	.251	0.70	0.15	Small
	Game 1	6.40	4.60	-.940	.347	0.99	0.20	Small
	Game 2	5.00	6.00	-.522	.602	0.51	0.11	Small
Theta	EC	6.20	4.80	-.731	.465	1.36	0.25	Small
	EO	7.00	4.00	-1.567	.117	1.33	0.25	Small
	Game 1	6.20	4.80	-.731	.465	0.94	0.19	Small
	Game 2	6.20	4.80	-.731	.465	0.38	0.09	Small
Alpha	EC	6.20	4.80	-.731	.465	1.32	0.25	Small
	EO	7.00	4.00	-1.567	.117	1.64	0.29	Small
	Game 1	6.00	5.00	-.522	.602	0.75	0.16	Small
	Game 2	6.20	4.80	-.731	.465	0.34	0.08	Small
Beta	EC	6.20	4.80	-.731	.465	1.33	0.25	Small
	EO	6.60	4.40	-1.149	.251	0.74	0.16	Small
	Game 1	6.20	4.80	-.731	.465	0.83	0.17	Small
	Game 2	5.80	5.20	-.313	.754	0.65	0.14	Small

4.2.2.2 Relative values for frontal lobe

When comparing the relative alpha activity between the two groups in the frontal lobe (see Table 7), it seems as if the non-ADHDs had significantly higher relative alpha wave activity ($p < .05$) during the Game 1 stage than the ADHDs ($MR_{nADHD} = 7.6$; $MR_{ADHD} = 3.4$). Furthermore, there was a medium to large effect size of this condition on the alpha wave activity. The non-ADHDs showed significantly higher relative Beta wave activity than the ADHDs in the frontal lobe during the Game 2 stage ($MR_{nADHD} = 7.6$; $MR_{ADHD} = 3.4$). Despite the fact that the differences were significant, the effect size was only medium.

Table 7

Between-group differences in relative values across waves and conditions

Wave	Condition	Mean Rank		*z*	*p*	Cohen's d		
		ADHD	Non-ADHD			d	Effect size	Category
Delta	EC	5.20	5.80	-.313	.754	-0.18	-0.05	Small
	EO	5.00	6.00	-.522	.602	0.13	0.03	Small
	Game 1	7.00	4.00	-1.567	.117	1.07	0.21	Small
	Game 2	6.80	4.20	-1.358	.175	0.94	0.19	Small
Theta	EC	5.20	5.80	-.313	.754	0.03	0.01	Small
	EO	5.40	5.60	-.104	.917	-0.31	-0.08	Small
	Game 1	5.60	5.40	-.104	.917	-0.19	-0.05	Small
	Game 2	7.00	4.00	-1.567	.117	1.22	0.23	Small
Alpha	EC	5.20	5.80	-.313	.754	-0.07	-0.02	Small
	EO	5.80	5.20	-.313	.754	0.08	0.02	Small
	Game 1	3.40	7.60	-2.193	.028*	-1.69	-0.73	Medium/ Large
	Game 2	4.00	7.00	-1.567	.117	-1.03	-0.35	Small
Beta	EC	6.40	4.60	-.940	.347	0.53	0.12	Small
	EO	4.80	6.20	-.731	.465	-0.27	-0.07	Small
	Game 1	5.20	5.80	-.313	.754	-0.09	-0.02	Small
	Game 2	3.40	7.60	-2.193	.028*	-1.34	-0.50	Medium

Note: values marked with * are significant at $p < .05$; values marked with ** are significant at $p < .10$

4.2.3 Within-group differences

Regarding the changes in the levels of wave activity during the progressions between the four conditions, significant results in the frontal lobe were only found for non-ADHDs, and exclusively during the transition from EC to EO ($p < .05$). This was found for all four wave types. It can be noted that for alpha in the progression from EO to Game 1, the difference was significant at $p < .10$.

Table 8

Within-group differences in the progressions between conditions for frontal lobe

Wave	Condition	ADHD				Non-ADHD			
		Mean Ranks		*z*	*p*	Mean Ranks		*z*	*p*
		Negative	Positive			Negative	Positive		
Delta	EC – EO	2.00	3.67	-.944	.345	.00	3.00	-2.023	.043*
	EO – Game 1	3.50	2.67	-.135	.893	2.75	4.00	-.944	.345
	Game1–Game 2	3.00	3.00	-.405	.686	2.00	3.25	-1.483	.138
Theta	EC – EO	3.00	3.00	-1.214	.225	.00	3.00	-2.023	.043*
	EO – Game 1	4.00	2.33	-.135	.893	2.33	4.00	-.135	.893
	Game1–Game 2	2.67	3.50	-.135	.893	.00	3.00	-1.214	.225
Alpha	EC – EO	3.00	3.00	-1.214	.225	.00	3.00	-2.023	.043*
	EO – Game 1	3.00	3.00	-.405	.686	1.00	3.50	-1.753	.080**
	Game1–Game 2	2.67	3.50	-.135	.893	5.00	2.50	-.674	.500
Beta	EC – EO	2.00	3.25	-1.483	.138	.00	3.00	-2.023	.043*
	EO – Game 1	4.50	2.00	-.405	.686	2.67	3.50	-.135	.893
	Game1–Game 2	3.00	3.00	-.405	.686	5.00	2.50	-.674	.500

Note: values marked with * are significant at $p < .05$; values marked with ** are significant at $p < .10$

4.3 Frontal midline

4.3.1 Descriptive Statistics

4.3.1.1 Mean power scores in the frontal midline

The mean power scores found for each group across conditions in the frontal midline are presented in Table 9. Thereafter, the different patterns of cortical activation for the frontal midline are demonstrated graphically per wave type.

Table 9

Mean power scores per wave and condition for frontal midline

		ADHD		Non-ADHD	
Wave	**Condition**	**Mean**	**SD**	**Mean**	**SD**
Delta	EC	.0079	.0116	.0022	.0017
	EO	.0149	.0173	.0058	.0017
	Game 1	.0056	.0066	.0078	.0025
	Game 2	.0101	.0121	.0099	.0037
Theta	EC	.0133	.0197	.0030	.0022
	EO	.0220	.0260	.0078	.0018
	Game 1	.0091	.0104	.0136	.0067
	Game 2	.0126	.0146	.0133	.0067
Alpha	EC	.0085	.0127	.0019	.0014
	EO	.0140	.0167	.0046	.0015
	Game 1	.0053	.0060	.0096	.0049
	Game 2	.0074	.0087	.0084	.0045
Beta	EC	.0046	.0069	.0012	.0009
	EO	.0130	.0192	.0031	.0006
	Game 1	.0030	.0033	.0050	.0021
	Game 2	.0055	.0067	.0051	.0021

The mean delta wave activity is displayed in Figure 7. During EC, the ADHD group exhibited higher delta wave activity in the frontal midline ($\overline{x}_{ADHD}$ = .0079) than the non-ADHD group ($\overline{x}_{nADHD}$ = .0022). From EC to EO, both groups showed an increase in delta wave activity, with the ADHD group displaying a more marked increase ($\overline{x}_{ADHD}$ = .0149; $\overline{x}_{nADHD}$= .0058). From EO to Game 1, the ADHD group showed a significant drop in delta wave activity ($\overline{x}_{ADHD}$ = .0056), whereas the non-ADHD group continued to exhibit increased delta waves ($\overline{x}_{nADHD}$ = .0078). From

Game 1 to Game 2, the non-ADHD group's delta wave activity continued to increase steadily ($\bar{x}_{nADHD}$ = .0099). After the ADHD group's initial drop in delta waves at Game 1, this group's delta waves increased again from Game 1 to Game 2 ($\bar{x}_{ADHD}$ = .0101).

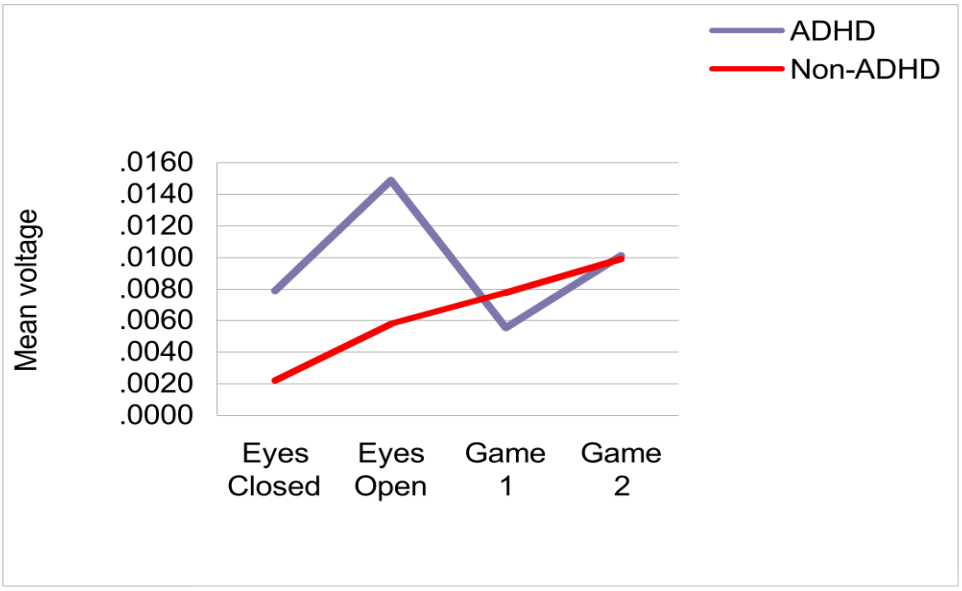

Figure 7: Delta wave mean power score in frontal midline across conditions

In the frontal midline region, the ADHD group exhibited higher theta wave activity than non-ADHD group at EC ($\bar{x}_{ADHD}$ = .0133; $\bar{x}_{nADHD}$ = .0030) (see Figure 8). Both groups had increased theta waves from EC to EO, with the ADHD group having a mean of .0220 and the non-ADHD having a mean of .0078. The ADHD group's theta waves decreased significantly from EO to Game 1 ($\bar{x}_{ADHD}$ = .0091), whereas the non-ADHD group's theta waves increased steadily ($\bar{x}_{nADHD}$ = .0136). From Game 1 to Game 2, the theta waves decreased for the first time in the non-ADHD group ($\bar{x}_{nADHD}$ = .0133), whereas the ADHD group's theta waves started to increase again ($\bar{x}_{ADHD}$ = .0126).

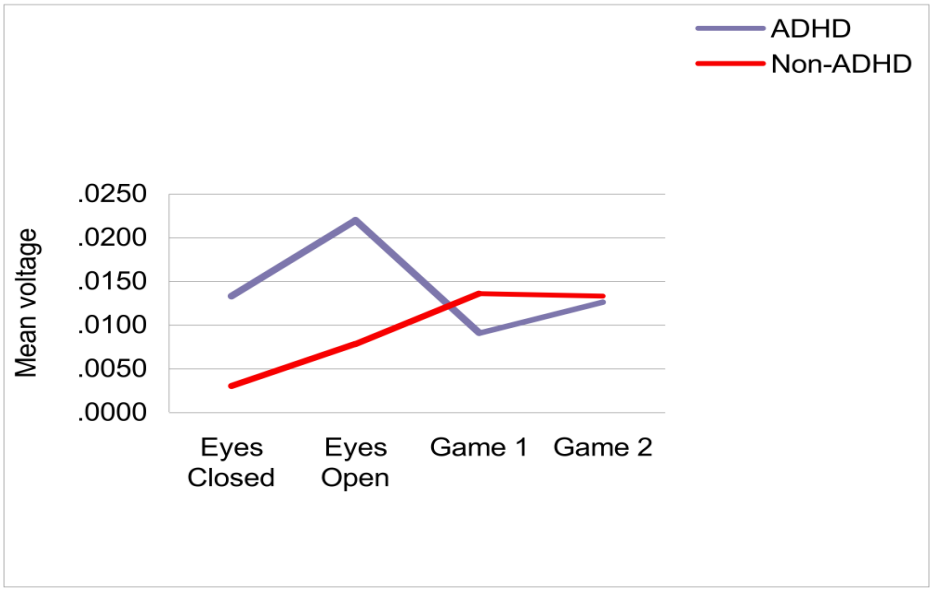

Figure 8: Theta wave mean power score in frontal midline across conditions

As displayed in Figure 9, the ADHD group initially exhibited higher alpha wave activity than the non-ADHD group at EC ($\bar{x}_{ADHD}$ = .0085; $\bar{x}_{nADHD}$ = .0019). From EC to EO, both groups' alpha waves increased, with the ADHDs showing a more notable increase than the non-ADHDs ($\bar{x}_{ADHD}$ = .0140; $\bar{x}_{nADHD}$ = .0046). However, from EO to Game 1, the ADHD group's alpha waves dropped significantly ($\bar{x}_{ADHD}$ = .0053), whereas the non-ADHD group's alpha waves continued to increase, albeit a bit more markedly than before ($\bar{x}_{nADHD}$ = .0096). From Game 1 to Game 2, the ADHD group's alpha waves increased again ($\bar{x}_{ADHD}$ = .0074) whereas the non-ADHD group's alpha waves decreased for the first time ($\bar{x}_{nADHD}$ = .0084).

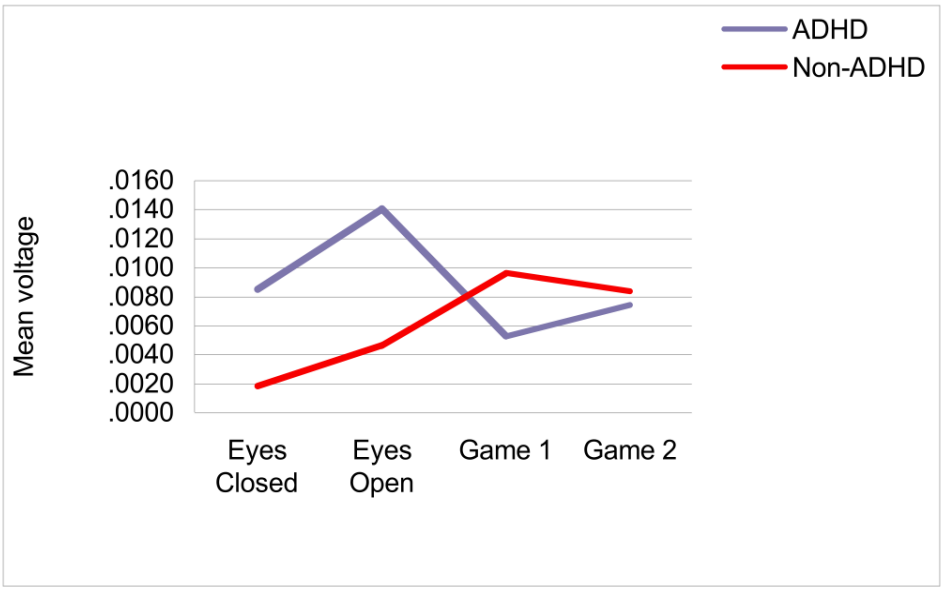

Figure 9: Alpha wave mean power score in frontal midline across conditions

In terms of beta wave activity, the ADHD group initially exhibited higher beta wave activity than the non-ADHD group, with a mean power score at EC of .0046 (as opposed to .0012 in the non-ADHD group). The ADHD group then displayed a marked increase beta wave activity in the switch from EC to EO ($\bar{x}_{ADHD}$ = .0130), whereas the non-ADHD group increased less dramatically ($\bar{x}_{nADHD}$ = .0031). In the switch from EO to Game 1, the ADHD group's beta waves dropped quite significantly ($\bar{x}_{ADHD}$ = .0030), whereas the non-ADHD group's beta waves continued to increase steadily ($\bar{x}_{nADHD}$ = .0050). From Game 1 to Game 2, the ADHD group's beta waves started to increase again ($\bar{x}_{ADHD}$ = .0055), whereas the non-ADHD group's beta waves continued to increase but to a lesser degree than before ($\bar{x}_{nADHD}$ = .0051).

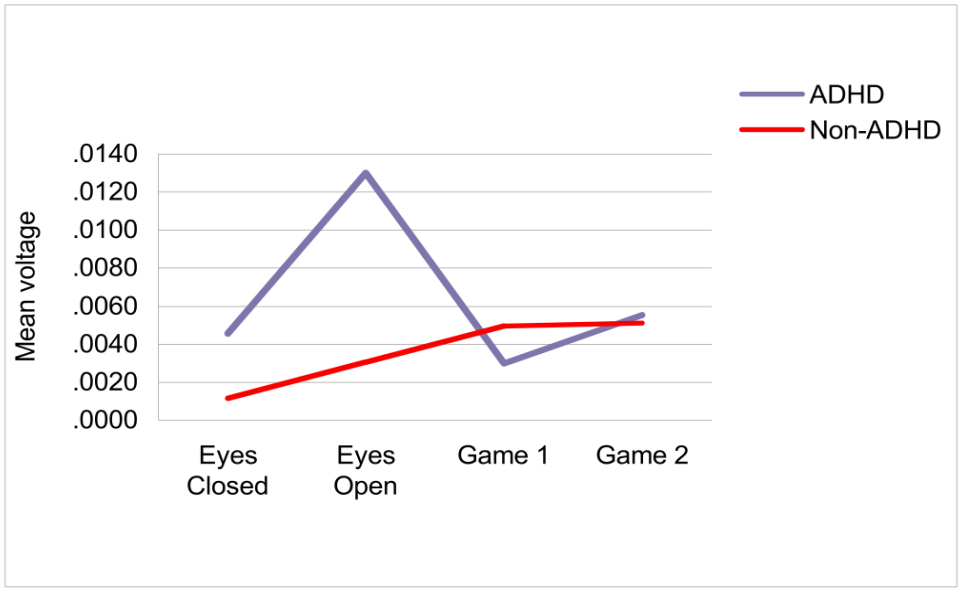

Figure 10: Beta wave mean power score in frontal midline across conditions

4.3.1.2 Mean relative wave activity in the frontal midline

Table 10 presents the mean relative wave activity for the two groups across all conditions.

Mean relative delta was consistently higher in the ADHDs across all conditions. For ADHDs, mean relative delta was 18.02% at EC, 21.94% at EO, 15.30% at Game 1 and 15.53% at Game 2. For non-ADHDs, mean relative delta was 16.38% at EC, 17.97% at EO, 14.62% at Game 1 and 15.53% at Game 2.

Mean relative theta activity was consistently the most active wave type in both groups across conditions. In the ADHD group, relative theta was 40.12% at EC, 37.49% at EO, 42.42% at Game 1 and 41.75% at Game 2. For the non-ADHD group, relative theta was 41.10% at EC, 41.43% at EO, 41.26% at Game 1 and 42.49% at Game 2.

In the ADHD group, the mean relative alpha was 29.16% at EC, 26.42% at EO, 29.59% at Game 1 and 30.37% at Game 2. In the non-ADHD group, mean relative alpha was 31.16% at EC, 28.24% at EO, 32.10% at Game 1 and 29.98% at Game 2.

Beta waves were the least active relative to other wave types in both groups across conditions. In ADHDs, mean relative beta activity was 12.71% at EC, 14.15% at EO, 12.60% at Game 1 and 12.35% at Game 2. Mean relative beta was slightly lower in non-ADHDs than in ADHDs, with the exception of Game 2. Mean relative beta was 11.36% at EC, 12.36% at EO, 12.01% at Game 1 and 12.69% at Game 2.

Table 10

Relative wave activity across conditions in the frontal midline

		ADHD		non-ADHD	
Wave	**Condition**	**Mean**	**SD**	**Mean**	**SD**
Delta	EC	.1802	.0781	.1638	.0338
	EO	.2194	.0553	.1797	.0389
	Game 1	.1539	.0369	.1462	.0244
	Game 2	.1553	.0343	.1485	.0214
Theta	EC	.4012	.0245	.4110	.0193
	EO	.3749	.0376	.4143	.0181
	Game 1	.4242	.0263	.4126	.0139
	Game 2	.4175	.0134	.4249	.0262
Alpha	EC	.2916	.0561	.3116	.0167
	EO	.2642	.0378	.2824	.0333
	Game 1	.2959	.0333	.3210	.0176
	Game 2	.3037	.0243	.2998	.0324
Beta	EC	.1271	.0419	.1136	.0114
	EO	.1415	.0213	.1236	.0150
	Game 1	.1260	.0313	.1201	.0063
	Game 2	.1235	.0118	.1269	.0080

4.3.2 Between-group differences in the frontal midline

In this section, the between-group differences in the frontal midline are presented in terms of the absolute and relative wave activity.

4.3.2.1 Absolute values in the frontal midline

As demonstrated in Table 11, no significant differences in absolute power were found between the two groups for any of the wave types in the frontal midline. At EO, however, the difference in mean beta activity was significant at $p < .10$.

Table 11

Between-group differences for the absolute power scores of all waves for the frontal midline cortical site

Wave	Condition	Mean Rank		*z*	*p*	Cohen's d		
		ADHD	Non-ADHD			d	Effect size	Category
Delta	EC	5.80	5.20	-.313	.754	0.86	0.18	Small
	EO	6.40	4.60	-.940	.347	0.95	0.19	Small
	Game 1	5.00	6.00	-.522	.602	-0.49	-0.14	Small
	Game 2	5.00	6.00	-.522	.602	0.03	0.01	Small
Theta	EC	5.60	5.40	-.104	.917	0.94	0.19	Small
	EO	6.20	4.80	-.731	.465	1.02	0.20	Small
	Game 1	4.60	6.40	-.940	.347	-0.53	-0.15	Small
	Game 2	5.00	6.00	-.522	.602	-0.07	-0.02	Small
Alpha	EC	5.60	5.40	-.104	.917	0.94	0.19	Small
	EO	6.20	4.80	-.731	.465	1.03	0.20	Small
	Game 1	4.20	6.80	-1.358	.175	-0.80	-0.25	Small
	Game 2	5.00	6.00	-.522	.602	-0.14	-0.04	Small
Beta	EC	5.80	5.20	-.313	.754	0.88	0.18	Small
	EO	7.20	3.80	-1.776	.076**	1.00	0.20	Small
	Game 1	4.60	6.40	-.940	.347	-0.72	-0.22	Small
	Game 2	5.00	6.00	-.522	.602	0.10	0.02	Small

Note: values marked with * are significant at $p < .05$; values marked with ** are significant at $p < .10$

4.3.2.2 Relative values in the frontal midline

In terms of the mean relative values, no significant differences were found between the two groups for any of the wave types (see Table 12).

Table 12

Between-group differences in relative values across waves and conditions in frontal midline

Wave	Condition	Mean Rank				Cohen's d		
		ADHD	Non-ADHD	*z*	*p*	d	Effect Size	Category
Delta	EC	5.00	6.00	-.522	.602	0.29	0.07	Small
	EO	6.60	4.40	-1.149	.251	0.84	0.17	Small
	Game 1	5.60	5.40	-.104	.917	0.25	0.06	Small
	Game 2	6.00	5.00	-.522	.602	0.24	0.06	Small
Theta	EC	4.80	6.20	-.731	.465	-0.45	-0.13	Small
	EO	4.20	6.80	-1.358	.175	-1.42	-0.55	Medium
	Game 1	6.60	4.40	-1.149	.251	0.58	0.13	Small
	Game 2	5.80	5.20	-.313	.754	-0.37	-0.10	Small
Alpha	EC	4.80	6.20	-.731	.465	-0.55	-0.16	Small
	EO	4.80	6.20	-.731	.465	-0.51	-0.15	Small
	Game 1	4.00	7.00	-1.567	.117	-0.99	-0.33	Small
	Game 2	5.20	5.80	-.313	.754	0.14	0.03	Small
Beta	EC	5.40	5.60	-.104	.917	0.51	0.11	Small
	EO	7.00	4.00	-1.567	.117	0.99	0.20	Small
	Game 1	6.20	4.80	-.731	.465	0.31	0.07	Small
	Game 2	5.00	6.00	-.522	.602	-0.34	-0.09	Small

Note: values marked with * are significant at $p < .05$; values marked with ** are significant at $p < .10$

4.3.3 Within-group differences in the frontal midline

The within-group differences in the transitions between conditions in the frontal midline are summarised in Table 13. Both the ADHD and non-ADHD groups showed significant changes in cortical activity in the progressions between conditions in the frontal midline area. This is in contrast with the frontal lobe, where significant differences were only found in non-ADHDs. Significant differences in the frontal midline were found for both groups in all wave types in the progression from the EC to EO conditions. The ADHDs also showed a significant change in delta wave activity in the progression from the Game 1 to the Game 2 conditions. In the progression from Game 1 to Game 2, the change in theta wave activity was only significant at $p < .10$ for the ADHD group. In the progression from EO to Game 1 in

the non-ADHD group, the change in alpha wave activity was only significant at $p < .10$.

Table 13

Within-group differences in progressions between conditions for frontal midline

Wave	Condition	ADHD				Non-ADHD			
		Mean Ranks		*z*	*p*	Mean Ranks		*z*	*p*
		Negative	Positive			Negative	Positive		
Delta	EC – EO	.00	3.00	-2.023	.043*	.00	3.00	-2.023	.043*
	EO – Game 1	3.33	2.50	-.674	.500	1.50	4.00	-1.214	.225
	Game1 – Game 2	.00	3.00	-2.023	.043*	3.00	3.00	-1.214	.225
Theta	EC – EO	.00	3.00	-2.023	.043*	3.00	3.00	-2.023	.043*
	EO – Game 1	.00	3.00	-.944	.345	2.00	3.25	-1.483	.138
	Game1 – Game 2	2.75	4.00	-1.753	.080**	3.50	2.67	-.135	.893
Alpha	EC – EO	.00	3.00	-2.023	.043*	.00	3.00	-2.023	.043*
	EO – Game 1	2.75	4.00	-.944	.345	1.00	3.50	-1.753	.080**
	Game1 – Game 2	2.00	3.25	1.483	.138	3.33	2.50	-.674	.500
Beta	EC – EO	.00	3.00	-2.023	.043*	.00	3.00	-2.023	.043*
	EO – Game 1	3.00	3.00	-1.214	.225	2.00	3.25	1.483	.138
	Game1 – Game 2	1.50	4.00	-1.214	.225	3.00	3.00	-.405	.686

Note: values marked with * are significant at $p < .05$; values marked with ** are significant at $p < .10$

4.4 Parietal lobe

4.4.1 Descriptive statistics for parietal lobe

The mean power scores found for each group across conditions in the parietal region are presented in Table 14. These mean power scores are presented per wave type in Figures 11, 12, 13 and 14.

4.4.1.1 Mean power scores in the parietal lobe

Both groups exhibited similar patterns of delta wave activity in the parietal region (see Table 14 and Figure 11). However, the ADHD group delta wave means were consistently higher than the non-ADHD group at each condition. At EC, the mean power score for the ADHDs was .0085, whereas the non-ADHD mean power score was .0019. From EC to EO, both groups displayed increased delta waves ($\bar{x}_{ADHD}$ =

.0148; $\bar{x}_{nADHD}$ = .0066). Both groups displayed decreased delta waves from EO to Game 1 ($\bar{x}_{ADHD}$ = .0110; $\bar{x}_{nADHD}$ = .0022), and then increased delta waves from Game 1 to Game 2 ($\bar{x}_{ADHD}$ = .0154; $\bar{x}_{nADHD}$ = .0076).

Table 14

Mean power scores per wave and condition for parietal lobe

Wave	**Condition**	**ADHD**		**Non-ADHD**	
		Mean	**SD**	**Mean**	**SD**
Delta	EC	.0085	.0080	.0019	.0017
	EO	.0148	.0102	.0066	.0077
	Game 1	.0110	.0074	.0022	.0015
	Game 2	.0154	.0089	.0076	.0053
Theta	EC	.0122	.0126	.0020	.0016
	EO	.0178	.0130	.0095	.0132
	Game 1	.0162	.0074	.0044	.0040
	Game 2	.0188	.0110	.0096	.0058
Alpha	EC	.0122	.0126	.0012	.0012
	EO	.0103	.0079	.0059	.0097
	Game 1	.0100	.0043	.0033	.0032
	Game 2	.0111	.0067	.0050	.0021
Beta	EC	.0049	.0047	.0013	.0013
	EO	.0081	.0059	.0036	.0051
	Game 1	.0062	.0023	.0019	.0016
	Game 2	.0084	.0052	.0033	.0018

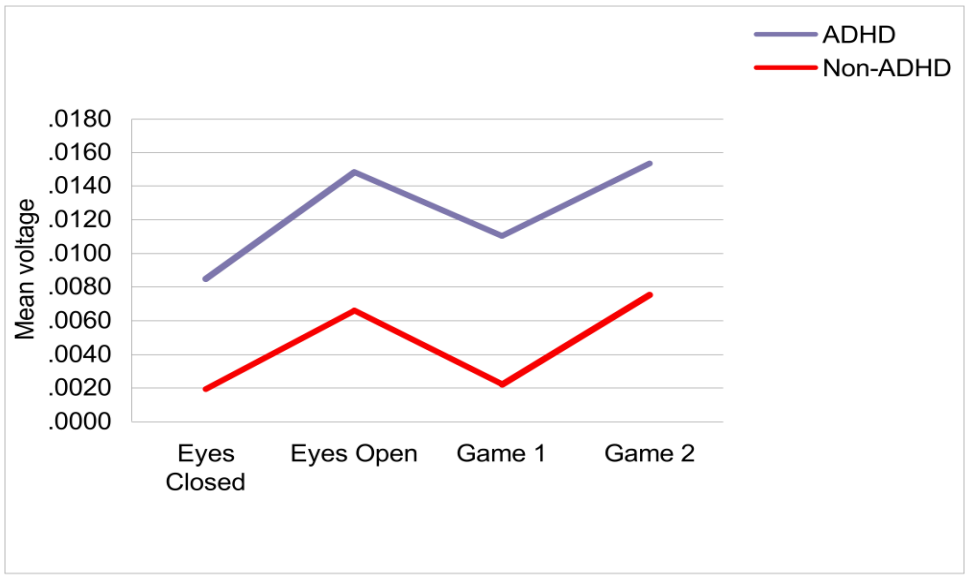

Figure 11: Delta wave mean power score in parietal lobe across conditions

The ADHD group exhibited consistently higher theta wave activity than the non-ADHDs in the parietal lobe (see Table 14 and Figure 12). At EC, the ADHD group's mean power score for theta waves was .0122, whereas the non-ADHD group had a mean power score of .0020. Both groups had an increase in theta waves from EC to EO ($\bar{x}_{ADHD}$ = .0178; $\bar{x}_{nADHD}$ = .0095). From EO to Game 1, both groups had decreased theta waves, with the non-ADHD group showing a more significant drop ($\bar{x}_{ADHD}$ = .0162; $\bar{x}_{nADHD}$ = .0044). Both groups' theta waves then increased again from Game 1 to Game 2, with the non-ADHD group displaying a more marked increase ($\bar{x}_{ADHD}$ = .0188; $\bar{x}_{nADHD}$ = .0096).

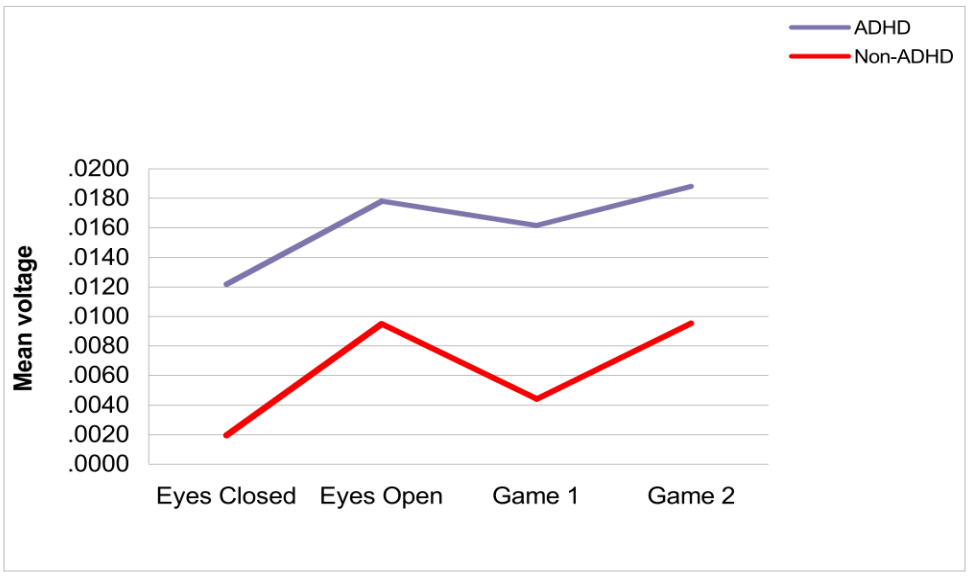

Figure 12: Theta wave mean power score in parietal lobe across conditions

In the parietal lobe, the ADHD group's alpha wave activity was consistently higher than the non-ADHD group's (see Table 14 and Figure 13). The mean power score recorded at EC was .0122 for the ADHD group, and .0012 for the non-ADHD group. From EO to EC, both groups' alpha wave activity increased ($\bar{x}_{ADHD}$ = .0103; $\bar{x}_{nADHD}$ = .0059). Both groups exhibited a decrease in alpha wave activity from EO to Game 1, with the non-ADHD group displaying a more notable decrease ($\bar{x}_{ADHD}$ = .0010; $\bar{x}_{nADHD}$ = .0033). Both groups then exhibited increased alpha waves from Game 1 to Game 2 ($\bar{x}_{ADHD}$ = .0111; $\bar{x}_{nADHD}$ = .0050).

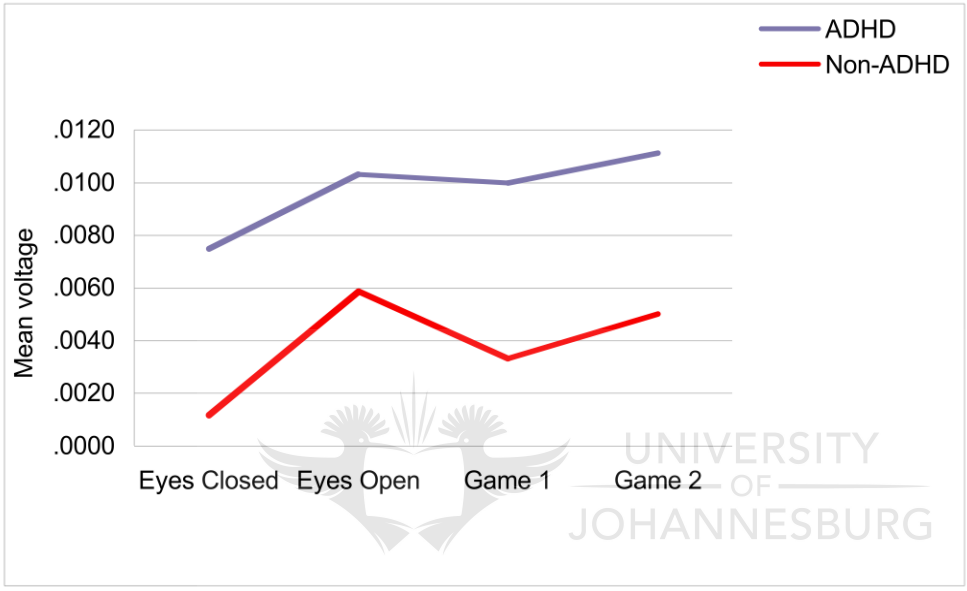

Figure 13: Alpha wave mean power score in parietal lobe across conditions

Both groups exhibited similar patterns in beta wave activity in the parietal region. The ADHD group, however, displayed higher beta waves through all four conditions. At EC, the mean power score for the ADHD group was .0049, whereas the non-ADHD group's mean power score was .0013. From EC to EO, both group exhibited an increase in beta waves ($\bar{x}_{ADHD}$ = .0081; $\bar{x}_{nADHD}$ = .0004). The beta wave activity dropped for both groups from EO to Game 1, with the ADHD group having a mean power score of .0062 and the non-ADHD group .0019. From Game 1 to Game 2, there was an increase again in beta wave activity ($\bar{x}_{ADHD}$ = .0084; $\bar{x}_{nADHD}$ = .0033).

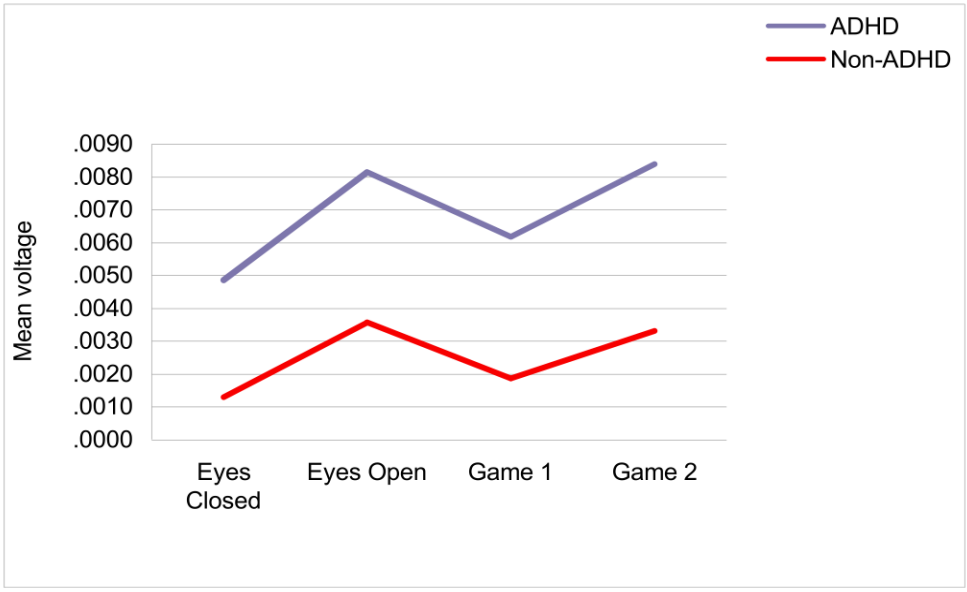

Figure 14: Beta wave mean power score in parietal lobe across conditions

4.4.1.2 Mean relative wave activity in the parietal lobe

The mean relative wave activity in the parietal region across conditions is displayed in Table 15.

Mean relative delta activity was generally higher in ADHDs, with the exception of the EC condition. Mean relative delta values for ADHDs were 15.50% at EC, 21.23% at EO, 18.85% at Game 1 and 16.90% at Game 2. For the non-ADHD group, mean relative delta was 24.63% at EC, 20.45% at EO, 15.18% at Game 1 and 15.75% at Game 2.

Mean relative theta wave activity was dominant compared to other wave types for both groups, with relatively similar values. In ADHDs, mean relative theta was 41.76% at EC, 40.57% at EO, 41.18% at Game 1 and 40.54% at Game 2. In non-ADHDs, mean relative theta was 42.65% at EC, 41.64% at EO, 41.40% at Game 1 and 41.85% at Game 2.

In terms of mean relative alpha wave activity, the ADHD group's mean relative alpha was 31.21% at EC, 25.95% at EO, 27.88% at Game 1 and 29.65% at Game 2. The non-ADHD group had higher mean relative alpha values for all conditions

except for the EC condition. At EC, the non-ADHDs' mean relative alpha was 24.04%, then 26.65% at EO, 30.93% at Game 1 and finally, 30.35% at Game 2. Beta waves were least active relative to other wave types across conditions in the parietal lobe. In the ADHD group, mean relative beta was 11.54% at EC, 12.24% at EO, 12.08% at Game 1 and 12.90% at Game 2. In non-ADHDs, mean relative beta was very low at EC (8.68%), 11.26% at EO, 12.49% at Game 1 and 12.09% at Game 2.

Table 15

Relative wave activity across conditions in the parietal lobe

		ADHD		non-ADHD	
Wave	**Condition**	**Mean**	**SD**	**Mean**	**SD**
Delta	EC	0.1550	0.0171	0.2463	0.1119
	EO	0.2123	0.0306	0.2045	0.0689
	Game 1	0.1885	0.0569	0.1518	0.0215
	Game 2	0.1690	0.0505	0.1575	0.0379
Theta	EC	0.4176	0.0088	0.4265	0.0491
	EO	0.4057	0.0181	0.4164	0.0506
	Game 1	0.4118	0.0224	0.4140	0.0111
	Game 2	0.4054	0.0271	0.4185	0.0156
Alpha	EC	0.3121	0.0163	0.2404	0.0853
	EO	0.2595	0.0272	0.2665	0.0534
	Game 1	0.2788	0.0445	0.3093	0.0170
	Game 2	0.2965	0.0362	0.3035	0.0274
Beta	EC	0.1154	0.0049	0.0868	0.0480
	EO	0.1224	0.0175	0.1126	0.0407
	Game 1	0.1208	0.0071	0.1249	0.0073
	Game 2	0.1290	0.0166	0.1206	0.0058

4.4.2 Between-group differences in the parietal lobe

4.4.2.1 Mean absolute power in the parietal lobe

The between-group differences calculated in the parietal lobe in terms of absolute power can be seen in Table 16. An interesting finding emerged which was unique to the parietal region: ADHDs exhibited significantly higher mean power scores than

non-ADHDs in the parietal region across all four wave types during Game 1. However, despite these differences being significant, the effect sizes were only small. Additionally, ADHDs' mean beta power score was considered significantly higher at EO when $p < .10$.

Table 16

Between-group differences for the absolute power scores of all waves for the parietal lobe

Area	Condition	Mean Rank		*z*	*p*	Cohen's d		
		ADHD	Non-ADHD			d	Effect size	Category
Delta	EC	6.40	4.60	-.940	.347	1.35	0.25	Small
	EO	7.00	4.00	-1.567	.117	0.92	0.19	Small
	Game 1	8.00	3.00	-2.611	.009*	1.97	0.33	Small
	Game 2	6.60	4.40	-1.149	.251	1.10	0.21	Small
Theta	EC	6.40	4.60	-.940	.347	1.44	0.26	Small
	EO	6.40	4.60	-.940	.347	0.64	0.14	Small
	Game 1	7.80	3.20	-2.402	.016*	2.07	0.34	Small
	Game 2	6.80	4.20	-1.358	.175	1.10	0.22	Small
Alpha	EC	6.60	4.40	-1.149	.251	1.60	0.29	Small
	EO	6.40	4.60	-.940	.347	0.50	0.11	Small
	Game 1	7.80	3.20	-2.402	.016*	1.77	0.31	Small
	Game 2	7.00	4.00	-1.567	.117	1.38	0.26	Small
Beta	EC	6.40	4.60	-.940	.347	1.19	0.23	Small
	EO	7.20	3.80	-1.776	.076**	0.82	0.17	Small
	Game 1	7.80	3.20	-2.402	.016*	2.22	0.36	Small
	Game 2	7.00	4.00	-1.567	.117	1.45	0.27	Small

Note: values marked with * are significant at $p < .05$; values marked with ** are significant at $p < .10$

4.4.2.2 Mean relative power in the parietal lobe

Between-group differences in mean relative power in the parietal region were investigated. The results appear in Table 17. No significant differences were found between the two groups.

Table 17

Between-group differences in mean relative power in the parietal lobe

Area	Condition	Mean Rank				Cohen's d		
		ADHD	Non-ADHD	*z*	*p*	d	Effect size	Category
Delta	EC	4.80	6.20	-.731	.465	-1.42	-0.55	Medium
	EO	6.00	5.00	-.522	.602	0.16	0.04	Small
	Game 1	6.40	4.60	-.940	.347	0.94	0.19	Small
	Game 2	6.40	4.60	-.940	.347	0.26	0.06	Small
Theta	EC	5.80	5.20	-.313	.754	-0.31	-0.08	Small
	EO	5.20	5.80	-.313	.754	-0.31	-0.08	Small
	Game 1	6.00	5.00	-.522	.602	-0.13	-0.03	Small
	Game 2	4.40	6.60	-1.149	.251	-0.61	-0.18	Small
Alpha	EC	6.20	4.80	-.731	.465	1.41	0.26	Small
	EO	5.00	6.00	-.522	.602	-0.17	-0.05	Small
	Game 1	4.60	6.40	-.940	.347	-0.99	-0.33	Small
	Game 2	4.60	6.40	-.940	.347	-0.22	-0.06	Small
Beta	EC	6.40	4.60	-.940	.347	1.08	0.21	Small
	EO	5.40	5.60	-.104	.917	0.34	0.08	Small
	Game 1	4.40	6.60	-1.149	.251	-0.57	-0.17	Small
	Game 2	6.60	4.40	-1.149	.251	0.75	0.16	Small

4.4.3 Within-group differences in the parietal lobe

The results for within-group differences in the progressions between conditions in the parietal lobe appear in Table 18. An interesting pattern emerged in the parietal lobe where the ADHD group showed significant increases in delta and beta wave activity in the progression from EC to EO. Although the ADHD group's increase in theta wave activity was from EC to EO was not significant at $p < .05$, it was significant at $p < .10$.

In contrast to the ADHD group, the non-ADHD group's delta, theta and beta wave activity increased significantly in the progression from Game 1 to Game 2. Although the non-ADHDs' increase in alpha wave activity from Game 1 to Game 2 was not significant at $p < .05$, it was significant at $p < .10$.

Table 18

Within-group differences in progressions between conditions for parietal lobe

Wave	Condition	ADHD				Non-ADHD			
		Mean Ranks		*z*	*p*	Mean Ranks		*z*	*p*
		Negative	Positive			Negative	Positive		
Delta	EC – EO	.00	3.00	-2.023	.043*	3.00	3.00	-1.214	.225
	EO – Game 1	4.50	2.00	-.405	.686	3.67	2.00	-.944	.345
	Game1 – Game 2	2.50	3.33	-.674	.500	.00	3.00	-2.023	.043*
Theta	EC – EO	1.00	3.50	-1.753	.080**	4.00	2.33	-1.214	.225
	EO – Game 1	3.00	3.00	-.405	.686	3.50	2.67	-.135	893
	Game1 – Game 2	3.00	3.00	-.405	.686	.00	3.00	-2.023	.043*
Alpha	EC – EO	1.50	4.00	-1.214	.225	4.00	2.75	-.944	.345
	EO – Game 1	2.67	3.50	-.135	.893	4.00	2.33	-.135	.893
	Game1 – Game 2	2.33	4.00	-.135	.893	1.00	3.50	-1.753	.080**
Beta	EC – EO	.00	3.00	-2.023	.043*	4.00	2.75	-.944	.345
	EO – Game 1	3.33	2.50	-.674	.500	4.00	2.33	-.135	.893
	Game1 – Game 2	4.00	2.75	-.944	.345	.00	3.00	-2.023	.043*

4.5. Theta: beta ratios

The theta: beta ratios were calculated to investigate the between-group differences in the slow- to fast-wave relationship in each cortical region for each of the conditions. No significant differences in theta: beta ratios were found between the two groups (see Tables 19 and 20).

Table 19

Between-group differences in theta: beta ratios

		Theta		Beta	
Cortical site	**Condition**	**Mean**	**SD**	**Mean**	**SD**
Frontal	Eyes Closed	2.8144	.1653	2.6875	.1497
	Eyes Open	2.6516	.5228	2.2728	.3625
	Game 1	2.7537	.4778	2.7287	.4203
	Game 2	2.4764	.5465	2.6961	.2942
Frontal Midline	Eyes Closed	2.8177	.6480	2.7570	.3985
	Eyes Open	2.0934	.6357	2.5552	.2940
	Game 1	2.8793	.5396	2.7117	.2324
	Game 2	2.5918	.6049	2.5315	.2725
Parietal	Eyes Closed	2.6606	.4409	2.0907	.8491
	Eyes Open	2.0993	.4450	2.6725	.5311
	Game 1	2.5758	.3325	2.3966	.2775
	Game 2	2.3318	.2558	2.8893	.5381

Table 20

Between-group differences in theta: beta ratios per experimental condition and cortical site

Area	**Condition**	**Mean Rank**		***z***	***p***
		ADHD	**Non-ADHD**		
Frontal	EC	6.60	4.40	-1.149	.251
	EO	7.00	4.00	-1.567	.117
	Game 1	5.60	5.40	-.104	.917
	Game 2	5.00	6.00	-.522	.602
Frontal Midline	EC	5.40	5.60	-.104	.917
	EO	4.00	7.00	-1.567	.117
	Game 1	6.60	4.40	-1.149	.251
	Game 2	5.20	5.80	-.313	.754
Parietal	EC	6.60	4.40	-1.149	.251
	EO	4.00	7.00	-1.567	.117
	Game 1	6.00	5.00	-.522	.602
	Game 2	4.20	6.80	-1.358	.175

4.6 Post-test survey data

Table 21 summarises the findings based on the survey data. Three out of five participants (60%) in the ADHD group were unaware of background noise while playing the game. In the non-ADHD group, two out of five participants were unaware of background noise. Participants provided an estimate of how long they thought that they had been playing the game. When compared to the actual time spent, the mean discrepancy was 9:09 minutes in the ADHD group, and 6:41 minutes in the non-ADHD group (see Figure 15).

Table 21

Time perception and awareness of surroundings during game play

Group	Participant	Gender	Aware of background noise	Estimated time spent playing (minutes)	Actual time spent	Discrepancy
ADHD	A1	M	No	30	21m 0s	9 m 0s
	A2	M	No	20	30m 30s	-10 m30s
	A3	M	Yes	40	29 m 47s	10m 13s
	A4	F	Yes	30	40 m 0s	-10m 0s
	A5	F	No	35	28m 57s	6 m 3s
	Mean scores			28 m 45 s	31m 5 s	5 m 46 s
Non ADHD	N1	M	No	35	37 m 26s	-2 m 26s
	N2	M	Yes	22	28 m 30s	-6 m 30s
	N3	M	Yes	20	28 m 00s	-8 m 00s
	N4	F	No	45	31m 17s	13 m 43s
	N5	F	Yes	30	32 m 46s	-2 m 46s
	Mean scores			30 m 15 s	31 m 6s	6 m 36 s

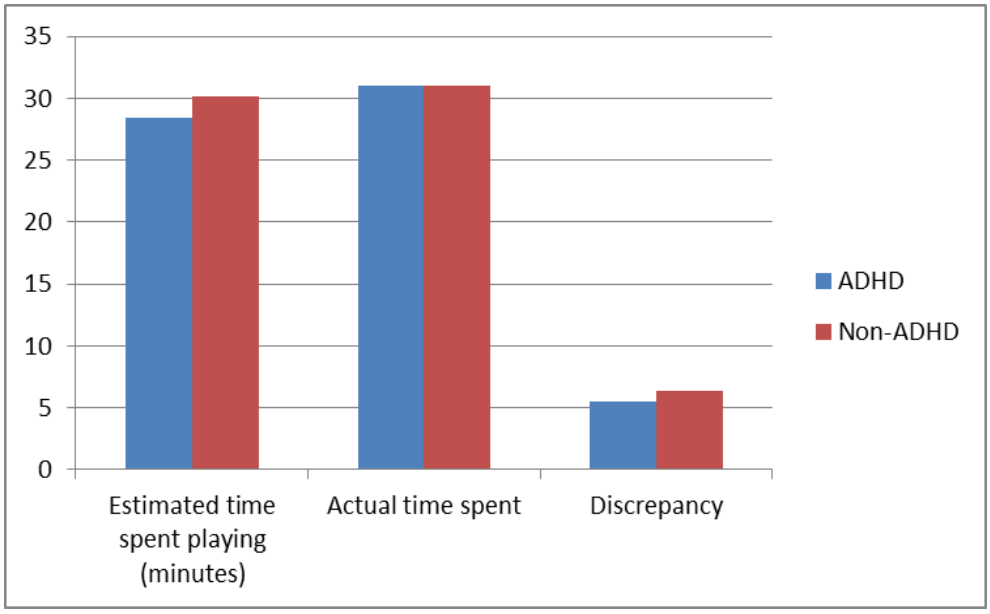

Figure 15: Time perception versus actual time spent during game play

4.7 Summary and conclusion

As mentioned previously, an EEG recording of brainwave activity across 4 experimental conditions. This was done in order to determine changes in brainwave activity across conditions, as well as to determine to what extent there were differences between the ADHD and non-ADHD groups during the different conditions. In order to draw conclusions, these differences are summarised in Table 22.

In the frontal lobe, three significant findings were made: (1) mean relative alpha was significantly higher in non-ADHDs than in ADHDs during Game 1; (2) mean relative beta activity was significantly higher in non-ADHDs than in ADHDs during Game 2; and (3) there were significant changes in the activity of all wave types from EC to EO for the non-ADHD group, whereas the ADHD group did not show the same significant changes. In the non-ADHD group, the change in alpha during EO to Game 1 was noted as significant at $p < .10$.

In the frontal midline, no significant between-group differences were found. In terms of within-group differences, the following significant findings can be noted: (1) In the progression from EC to EO, both groups exhibited significant increases in cortical activity for all wave types; and (2) the ADHD group showed a significant increase in

delta wave activity from Game 1 to Game 2. Three findings were noted for their significance at $p < .10$: (1) Mean absolute beta was higher in the ADHD group at EO; (2) In the non-ADHD group, the increase in alpha from EO to Game 1; and (3) In the ADHD group, the increase in theta from Game 1 to Game 2.

There were three significant findings in the parietal lobe: (1) During Game 1, mean absolute power was significantly higher for all wave types in the ADHD group; (2) The ADHD group showed significant increases in delta and beta from EC to EO; and (3) There were significant increases in delta, theta and beta in the non-ADHD group during the progression from Game 1 to Game 2. Three findings were noted as significant when $p < .10$: (1) Mean absolute beta was higher in ADHDs than non-ADHDs during EO; (2) The increase in theta during EC to EO in the ADHD group; and (3) the increase in alpha activity from Game 1 to Game 2 in the non-ADHD group.

Table 22

Summary of results

	Between group differences			Within group differences		
		Absolute	Relative		ADHD	non-ADHD
Frontal lobe	**EC**	.	.	**EC-EO**	.	Significant for all wave types
	EO	.	.	**EO-Game 1**	.	Alpha significant at $p < .10$
	Game 1	.	nADHD: Significantly higher alpha	**Game 1-Game 2**	.	.
	Game 2	.	nADHD: significantly higher beta			
Frontal midline	**EC**	.	.	**EC-EO**	Significant increase for all wave types	Significant increase for all wave types
	EO	ADHD: higher beta significant at $p < .10$	.	**EO-Game 1**	.	Alpha significant increase at $p < .10$
	Game 1	.	.	**Game 1-Game 2**	- Significant for delta - Theta increase significant at $p < .10$	.
	Game 2	.	.		.	.
Parietal lobe	**EC**	.	.	**EC - EO**	- Significant changes in delta and beta - Theta significant at $p < .10$	
	EO	- ADHD beta significant at .10	.	**EO - Game 1**	.	.
	Game 1	- ADHD significantly higher for all wave types	.	**Game 1 – Game 2**	.	- Significant Delta, theta and beta - Alpha significant at 0.1
	Game 2	.	.		.	.

CHAPTER 5: DISCUSSION

5.1. Introduction

This study set out to explore the possible symptom of hyperfocus in aADHD and to attempt to identify the neural correlates thereof. In this chapter, the findings for each cortical region are discussed in relation to the hypotheses of the study. The between-groups hypotheses addressed in this study were: (1) “There will be significant differences in cortical activation between the ADHD group and the non-ADHD group during resting state”; and (2) “There will be significant differences in cortical activation between the ADHD group and the non-ADHD group in the hyperfocus state”. The within-groups hypotheses were: (3) “Adults with ADHD exhibit significant differences in cortical activation patterns in resting state as opposed to in an activity in which hyperfocus has been induced”; and (4) “Adults without ADHD will not exhibit significant differences in cortical activation patterns in resting state as opposed to in an activity in which hyperfocus has been induced”.

As discussed in chapter 4 an EEG recording was taken for each participant through the different conditions. Other studies have usually been limited to only one or two conditions of cognitive activation tasks or to EC/EO resting states. If one wants to study hyperfocus, which has been conceptualised as a state of sustained attention in which the individual’s behaviour is controlled over time by a stimulus (Sagvolden et al., 2005), then one needs to look at the way in which shifts occur from the resting state to the cognitive activation state, as well as changes in the cognitive activation state over time.

There are different possible approaches to discussing the results, for the purposes of this study, it was decided to first look at expected significant results, followed by expected non-significant results and then unexpected significant results. In this study, there were cases where results were not significant at the 5% level, but that were significant at the 10% level. These results have been recognised as trends that are potential areas for further research. The first section explores the results in relation to hypothesis 1, which deals with between-group differences across resting

states. Before addressing hypotheses 2, 3 and 4, the success of inducing the hyperfocus state in the study will be discussed. Thereafter, the results will be discussed in relation to the last three hypotheses.

5.2 Between-group differences across resting states

The first hypothesis set out to investigate whether there would be significant differences between the two groups during resting states (EC and EO). Of particular interest was the theta power, which was expected to be significantly higher in the ADHD group (Chabot & Serfontein, 1996; Koehler et al., 2009; Loo, & Barkley, 2005; Lovejoy et al. 1999; White et al., 2005). No significant differences were found between the two groups at the .05 level for any of the wave types during resting states. The failure to replicate previous findings in this study could possibly be due to the decrease in theta levels which has been noted across developmental trajectories in both normal and ADHD samples (Chabot & Serfontein, 1996). It must be noted that although increased theta has been pinpointed as a consistent marker of ADHD, Liechti et al. (2012) did not find significant increases in theta or theta: beta values in their study, and suggest that this could provide support for the hypothesis that "ADHD is a heterogeneous disorder, where the resting state is not consistently characterised by maturational lag" (p. 135). The small sample size or the method employed in screening and assigning participants to the two groups may have also resulted in non-significant differences in theta. In addition to using non-parametric statistical procedures, to compensate for the small sample, effect size, using Cohen's d was also calculated. The effect sizes also only ranged from small to medium.

Of note, at EO, ADHDs' absolute beta power was considered significantly higher in both the frontal midline and the parietal region at the .10 level. It is uncertain whether, with a larger sample size, the increased absolute beta would have proven significant at the 0.05 level. If this were indeed found to be the case, it would stand in contrast to previous research which has identified a decrease in beta power in ADHD samples (White et al., 2005). This highlights an area for further investigation.

5.3 Was hyperfocus achieved?

At the outset of the study, it was challenging to the select a task that would induce a state of hyperfocus, and to find an appropriate method of measurement. Firstly, the activity needed to meet the criteria set out by Csikszentmihalyi (1992) for flow to occur. Secondly, an appropriate means of measuring the flow experience needed to be identified. Finally, steps needed to be taken to provide more assurance that hyperfocus/flow had indeed occurred. A computer game was selected as a task which was likely to facilitate the occurrence of hyperfocus (Sherry, 2004; Weber et al., 2009). The accurate measurement of flow has long proven difficult, with lack of agreement among researchers on how to operationally define flow, as well as finding a method of collecting data which accounts for the context-specific and experiential aspects of flow (Hoffman & Novak, 2009; Novak & Hoffman, 1997; Weber et al., 2009). In the context of this study, the researcher wanted to collect data in real time without interrupting the flow activity; the automatic experience of flow does not allow for feedback from the individual in real time, as this would interrupt the flow activity. Therefore, the EEG was selected as a suitable means of measuring flow, as it could obtain real time data from the participants without interrupting the actual activity. Although video games have been identified as providing the appropriate conditions for creating flow (Sherry, 2004; Weber et al., 2009), this was not guaranteed. Survey questions were thus formulated to provide more evidence of whether flow was achieved.

It is important to note that Csikszentmihalyi (1990) views flow as a state which can be achieved and cultivated by almost anyone through the correct practice and discipline, although some people are more receptive to entering the flow state. To date, there is no research which specifically addresses the occurrence of flow in ADHD. Csikszentmihalyi (1990) actually asserts that “attentional disorders not only interfere with learning, but effectively rule out the possibility of experiencing flow as well” because the individual with an attentional disorder is unable to control attention (p.84). This assertion conflicts with reports of hyperfocus in ADHD (Schecklmann et al., 2009). In this study, the hypothesis was made that adults with ADHD are more predisposed to experience hyperfocus than adults without ADHD.

The results of the survey data seem to provide some evidence that hyperfocus did indeed occur in the ADHDs according to the conditions required by Csikszentmihalyi (1992) for the occurrence of flow. Firstly, three out of five (60%) of the ADHD participants indicated that they were not aware of background noise whilst playing, indicating full concentration on the task and inability to process any other stimuli. Secondly, the mean discrepancy between the actual time spent and the perceived time spent playing the game was greater for the ADHDs (9:09 min) than the non-ADHDs (6:41 min), indicating a possible changed sense of time. It seems likely that hyperfocus was experienced by at least some of the non-ADHD participants. Two out of five non-ADHDs reported that they were not aware of background noise, and in one case, the participant's estimate of playing time was 13m43s longer than the actual time spent.

As discussed in Chapter 2, a decrease in cortical activation may be linked to enhanced cognitive function, and perhaps may even point to the occurrence of hyperfocus (Schecklmann et al., 2009). In relation to this, a number of interesting points can be drawn from the results for the frontal lobe. When the relative wave activity was compared across the two groups, significant differences only existed in two instances in the frontal lobe: firstly, the non-ADHDs exhibited significantly higher alpha activity than the ADHD group during Game 1 (MR_{nADHD}= 7.6; MR_{ADHD}=3.4; p = .028); secondly, the non-ADHDs also showed significantly higher relative beta levels than the ADHD group during Game 2 (MR_{nADHD}= 7.6; MR_{ADHD}=3.4; p = .028). The significantly higher relative alpha and beta levels suggest that increased effort was exerted by the non-ADHD group as time went on in order to maintain performance in the cognitive activation task (White et al., 2005). These findings may point to the occurrence of hyperfocus. Finally, the ADHD group exhibited an increase in slow-wave activity in the frontal midline region (where delta's increase was significant at $p < .05$ and theta was significant at $p < 0.1$). It would be interesting to investigate whether the increase in theta would be found to be significant at $p < .05$ in a larger sample; Aftanas and Golocheikine (2001, p.57) found that increased theta in the frontal midline was associated with an "emotionally positive 'blissful' experience", a state which may be associated with hyperfocus. This suggestion, however, is speculatory, and requires further study.

Two trends found in the within-groups results could point to additional potential markers for hyperfocus. Although within-group differences in alpha and beta levels in the progression from Game 1 to Game 2 were not found to be significant for either group, two trends warrant being mentioned in light of the above significant findings: in the ADHD group, alpha and beta levels decreased from Game 1 to Game 2, whereas alpha and beta levels continued to increase for the non-ADHD group from Game 1 to Game 2. It is interesting that the fast-wave activity increased in the non-ADHD group and decreased in the ADHD group in the shift from Game 1 to Game 2, rather than the opposite. If it is true that ADHD implies the inability to sustain attention over time (Biederman, 2005; Weyandt et al., 2003), one would expect that the ADHD group would need to invest increased cortical effort (i.e. increased relative alpha and relative beta activity) as the cognitive activation task as compared to the non-ADHD group, as suggested by White et al. (2005). However, the opposite occurred. In line with the suggestion by Schecklmann et al. (2009), this decrease in activation in the ADHD group may signify the occurrence of hyperfocus or flow as the participants became more involved in the game.

5.4 Between-group differences

An interesting result emerged which was unique to the parietal lobe: the mean rank score of the ADHD group was found to be significantly higher than that of the non-ADHD group across all wave types, specifically in the Game 1 experimental condition. The presence of significant between-group differences in the parietal cortex in this study provide further justification for the need to expand on the current research on the role of the parietal lobe in attention deficits (Schneider et al., 2006). This significantly higher parietal activation in the ADHD group was surprising and stands in contrast to other studies that found significantly higher parietal activation in the control groups of other studies (Schneider et al., 2010; Tamm, Menon & Reiss, 2006). Tamm et al. (2006) suggest that the significantly lower activation in the parietal lobe noted in their study may point to impairments in directed attention, sustained attention/vigilance and set shifting. If one assumes that increased parietal activation signifies the activation of the above-mentioned attentional networks, then this provides evidence for the hypothesis that the impairments in attention associated with ADHD are context-specific (Leimkuhler, 1994; Thompson & Thompson, 2005). However, this assumption requires further investigation.

One can note that there was more slow wave activity (delta and theta waves) in the ADHD group across all conditions. This is consistent with the literature which identifies hypoarousal and excessive cortical slowing in the frontal cortex as a common expression of ADHD (Pary et al., 2002; Monastra et al, 1999, as cited in White et al., 2005). Although the slow wave activity was consistently higher for the ADHD group, it is interesting to note that, whereas the delta and theta levels continued to increase for the non-ADHD group across all four conditions, there was a decrease in slow wave activity for the ADHD group in the transition from Game 1 to Game 2. This is unexpected: If one assumes that ADHDs have a problem with inattention and cortical hypoarousal, one would expect the ADHD group to become less engaged with the task as the game continued, indicated by an increase in slow wave activity. However, given that there was a decrease in slow-wave activity, it seems that there was continuously more engagement with the task, as opposed to the non-ADHD group, which indicates a possible marker for hyperfocus.

5.5 Within-group differences

The actual changes in cortical activity across different experimental conditions were examined for each group, to determine whether these changes were significant. It is notable that significant results were found in all three cortical regions during the progression from EC to EO. In the frontal lobe, significant results were only found for the non-ADHD group across all wave types. In the frontal midline, significant increases were found for both groups in the progression from EC to EO for all wave types. In the parietal lobe, the ADHDs showed significant increases in delta and beta. The shifts observed across these states for both groups highlights a relatively untapped area of research.

Although functional studies have provided evidence that certain attentional functions are linked to the parietal region (Posner et al., 1990, as cited in Schneider et al., 2006), there is a surprisingly small amount of research which explores the parietal lobe's structure and function in ADHD (Schneider et al., 2006). The fact that there were significant findings for both groups highlights the need for more research specifically in the role that the parietal lobe plays in attentional functions. Further, the presence of significant differences between the two groups in terms of the

progressions between the four experimental conditions provides more reason to explore the exact role that the parietal lobe plays specifically in ADHD.

When the relative values in the parietal lobe were examined for each group to ascertain any significant changes during progressions between experimental conditions, it was interesting to note that significant changes for the ADHD group were only found in the progression from EC to EO, and only for Delta and Beta waves. In contrast, the non-ADHD group exhibited significant changes in the progression from Game 1 to Game 2, for Delta, Theta and Beta waves.

Previous qualitative electroencephalography (QEEG) research has indicated that adults with ADHD exhibit a significant delay in the maturation of the prefrontal cortex, resulting in lower cortical arousal than adults without ADHD (Monastra et al, 1999, as cited in White et al., 2005). Thus the absence of significant changes in cortical arousal in the ADHD group could possibly be attributed to lower cortical arousal generally noted in adults with ADHD. Interestingly, this 'hypofrontality' which is noted as underlying the symptomatology in ADHD is also pinpointed by Dietrich (2004) as the condition which he proposes to underlie flow.

The within-group differences support the evidence for between-group differences for hyperfocus. As previously discussed, previous research indicates that theta levels are expected to increase "as demands of a task become less engaging" (Lubar, 1995, as cited in White et al., 2005, p. 119). Specifically in this study, one would expect that theta levels would increase in both groups from Game 1 to Game 2. Although theta levels decrease for both groups for the frontal midline and the parietal lobe in the switch from Game 1 to Game 2, there was a decrease in theta levels for the ADHD group from Game 1 to Game 2. This seems to indicate that the ADHD group continued to be engaged in the task, as opposed to the non-ADHD group that did not show this trend. This is contrary to previous findings which suggest that theta levels will increase as a task becomes less engaging (Drummond et al., 2005 as cited in Helps, James, Debener, Karl & Sonuga-Barke, 2008). Again, if one assumes that people with ADHD have impaired sustained attention, one would have expected the ADHD group to become less engaged in

the game, which would have been indicated by a gradual increase in theta wave activity.

5.6 Theta: Beta Ratios

One cannot look at different waves only in isolation. To get the full picture, one must also look into the way that the waves work together. In light of this, as the task progressed over time one would expect that there would be an increase in the theta wave activity, as mentioned above and an increase in beta wave activity, therefore the less engaged a person is, the greater the ratio, and the more engaged, the greater the ratio. A ratio of 1:1 (theta: beta) would arguably indicate full engagement.

Theta: Beta if ratio is equal (1), this signifies full engagement.

In the present study, no significant differences were in the theta: beta ratios for either group. This is contrary to research which demonstrates higher theta-beta power ratios in ADHD samples when compared to non-ADHD groups (Thompson & Thompson, 2005). This would only be true if ADHDs indeed have impaired sustained attention. The fact that there were no significant differences between the two groups is in itself a significant finding. However, given the small sample size, this is an aspect that warrants further research.

5.7 Limitations & recommendations

5.7.1 Limitations

Given that this study addressed an element of adult ADHD that has not been addressed in the literature, there was little to no other research upon which to base the method of data collection. The method employed was thus quite exploratory, and a lot of lessons were learnt in the process. In retrospect, the following limitations were identified:

5.7.1.1 Sampling

A larger sample size generally allows for the results of a study to be generalised more easily to the population being studied (Gravetter & Forzano, 2009). In this study, the sample size was kept small due to practical reasons - the process of extracting data from the EEG recording is particularly lengthy, with the frequencies

for each wave type needing to be recorded manually per epoch. However, although interesting findings were made, the sample size was too small to draw any final conclusions. For generalisability, one must also ensure that the sample is representative of the population in terms of gender. Although gender was matched across groups, it is uncertain whether the groups were reflective of the proportions of males to females with adult ADHD. However, because there are very few prevalence studies in South Africa, it is very difficult to define the population parameters. Regarding the screening process, the ASRS was used for screening because it is a widely used screening instrument for adult ADHD. Although the ASRS has proven useful in successfully identifying samples of adults with and without ADHD, the screening of the participants could have been more rigorous to ensure that participants met all criteria and exclusion criteria.

Furthermore, in selecting the ADHD sample, no provision was made for the different sub-types of ADHD, and it may be that the different sub-types of ADHD may have produced different results. It must be noted however, that there is scant literature on the different sub-types of ADHD in adulthood and that the most of the studies done on adults samples the inattentive or combined types, or treat ADHD in adulthood as a homogenous disorder. Burke and Edge (in press) argue that ADHD cannot be seen as a homogeneous disorder in adulthood, and that further research is indicated to distinguish the sub-groups. In this research, the default position of ADHD as a homogenous disorder was assumed.

5.7.1.2 Design

The quasi-experimental design is inherently limited in that there are factors that do not allow for all confounding variables to be controlled for. Although these factors (such as non-random sampling), were controlled for to the greatest extent possible, any conclusions drawn from the study remain tentative (Gravetter & Forzano, 2009). Furthermore, only one type of task, i.e. a first person shooter game, was used in this study. It may be useful, in future research to include more than one group of ADHD, not only based on the sub-type of ADHD, but also the type of activity that the groups are exposed to. It must be noted, however, that the EEG technology that is currently available, places constraints on the type of activities that participants can be exposed to.

In this study, one specific type of computer game was used. It is possible that other types of computer games or other activities would have facilitated the occurrence of hyperfocus more effectively. Further, after conducting the experiment, it became clear that the post-test survey questions did not fully capture the element of time perception as initially desired. Regarding the EEG recording, the equipment available at the time only allowed for three cortical areas to be recorded simultaneously. Finally, although every effort was made reduce the effects of environmental factors on results, the test environment was not fully controlled (i.e. air-conditioning, light, noise, etc.). Additionally, although we tried to test all participants at the same time of day, to eliminate the effects of circadian rhythms, it was not always possible due to practicalities of arranging a suitable time.

5.7.2 Recommendations

In light of the limitations mentioned above, a number of recommendations can be made for future studies. Firstly, it is recommended that future studies utilise a larger sample so that results can be generalised further. Generalisability could also be improved by conducting similar studies in which comparative groups are included of people who have reported hyperfocus in a particular experience (e.g. in playing an instrument or writing, etc.). Further, experimental conditions can be manipulated. Secondly, screening methods can be improved on by including additional means of obtaining information, whilst keeping in mind the diagnostic controversies identified in chapter one. One must make assumptions about adult ADHD which will inform the choice of screening methods. Hence, if ADHD is conceptualised as a neurodevelopmental disorder, then evidence from childhood must be included in the screening. It is recommended that an inventory which screens for childhood symptoms is included (e.g. Wender Utah Rating Scale) along with a clinical interview. The data collection can be improved upon by looking at more cortical sites to provide a more holistic picture. Further, additional measures can be put in place to assess whether hyperfocus occurred: (1) introduce a distractor during the hyperfocus condition, to test the level of ‘absorption’ in the task; (2) Refine the qualitative questions; and (3) Increase the duration of the experiment and the recording time, to see if the pattern continued.

5.8 Conclusion

Based on the preceding discussion of the results, a few conclusions can be drawn from this study regarding hyperfocus in adult ADHD. Keeping in mind the limitations discussed in the previous sections, as well as the exploratory nature of this study, these conclusions remain tentative and require further investigation. Firstly, it seems that this study made progress in establishing the use of a computer game and EEG recording, as previously done by Weber et al. (2009), as a method of measuring and recording hyperfocus that does not interfere with the actual experience of hyperfocus. The experience of hyperfocus is, by definition, context-dependant, which implies that the cognitive activation task must be suited to the interests of the participant. Further, the recording of the progression from resting states to cognitive activation states appears to have been an appropriate choice specifically for the measurement of hyperfocus. Traditionally, EEG research in ADHD has used Eyes Closed and Eyes Open as experimental states in which brain wave activity is recorded. These types of studies have not only yielded contradictory findings, but one could also question the extent to which they provide information in terms of different contexts (van Dongen-Boomsma et al., 2010). This holds true especially for the study of hyperfocus, which is not only contextually bound, but also for the temporal qualities of hyperfocus. It is for this reason that an EEG recording was taken during each condition, as it was postulated that it would address some of the aforementioned problems.

The findings in this study also highlighted a need for further research into certain aspects of ADHD. Firstly, the significant changes in cortical activity found in the progression from EC to EO warrants further investigation. Furthermore, the unexpected finding that there were significant between-group differences and within-group differences in the parietal lobe highlights the need for further research into the role that the parietal lobe plays in the attentional functions as well as in ADHD, given the scant research into this particular region.

In addressing the original hypotheses from Chapter 3 for between-group and within-group differences, a few possible markers for hyperfocus were identified, that seemed to fit well with the suggestion that decreased cortical activation may point to enhanced cognitive function, and possibly hyperfocus (Schecklmann et al.,

2009). Possible markers of hyperfocus included the significantly lower alpha and beta levels noted in the ADHDs during game play, as well as the trend noted in the ADHD group that slow-wave activity (delta and theta) decreased from Game 1 to Game 2; these results suggest that the ADHDs seemed to become more engaged with the game as time went on. This is further corroborated by the qualitative results which seemed to indicate that ADHD participants experienced a distorted perception of time during game play. In line with the second hypothesis, these findings provide empirical evidence that there are significant differences in cortical activation between the two groups during the hyperfocus state, suggesting that adults with ADHD may be more predisposed to hyperfocus. This challenges the stance of Csikszentmihalyi (1992) that individuals with attention disorders are not able to enter the flow state. Perhaps more importantly though, these findings seem to indicate that the ADHDs were able to sustain attention on the task, in that their behaviour was controlled over time by the task (Sagvolden et al., 2005), thus challenging the notion that ADHDs are unable to sustain attention. This poses a challenge to current formulations of inattention in ADHD, and hence the current diagnostic criteria of ADHD.

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APPENDICES

Appendix A: Biographical questionnaire

Dear Participant

Please take the time to complete this biographical questionnaire. The information from this questionnaire will assist the researcher to select suitable participants for the study. All information will be kept confidential - after the study, the researcher will only use the information provided to report on statistics for the study.

Please be as honest as possible when answering the questions.

Demographic Details	
Name:	
Surname:	
Date of Birth:	DD / MM / YYYY
Tel No:	( )

1) Gender:

1	Male
2	Female

2) Ethnicity (Please tick one):

1	Black
2	White
3	Indian
4	Coloured

Education and Employment

3) What is your Highest level of education (completed)?

1	Matric

2	Undergraduate degree
3	Diploma
4	Honour's Degree
5	Master's Degree
6	Doctorate

4) Are you currently a student?

1	Yes
2	No

5) If you are a student, please indicate your year of studies in 2012:

1	1st year
2	2nd year
3	3rd year
4	4th year
5	Honour's
6	Master's
7	Doctorate

6) Are you currently employed?

1	Yes
2	No

7) If you are employed, please indicate whether you are employed full-time or part-time:

1	Full-Time
2	Part-time

8) Occupation: _____

Medical History

9. Are you taking any chronic medication (medication on an ongoing basis)?

1	Yes
2	No

10. If you answered yes to question 9, please list what medication you are taking:

11. Have you ever had a head injury where you lost consciousness for longer than 10 minutes?

1	Yes
2	No

12. If you answered yes to question 11, please indicate when this head injury occurred (please provide the month and the year):

13a. Are you receiving treatment for any psychiatric conditions?

1	Yes
2	No

13b. If you don't mind disclosing your diagnosis, please write what you are receiving treatment for:

14. Have you used any recreational drugs in the past 60 days?

1	Yes
2	No

Video Games

15. Do you play video games/Playstation/Xbox or anything similar?

1	Yes
2	No

16. If you answered yes to question 15, how much time on average do you spend playing these games?

1	Every day
2	Several times a week
3	A few hours per week
4	Every few weeks
5	Once a month
6	Less than once a month

Thank you for taking the time to complete this questionnaire.

Appendix B: Participant information sheet

Dear Participant,

Thank you for volunteering to participate in this study! As a research team we are committed to improve people's lives by informing clinicians, diagnosticians and therapists by means of scientific research. Through personal experience, as well as extensive review of current scientific documents, we have come to the conclusion that there is much confusion surrounding adult ADHD (aADHD) as a diagnosis. Some practitioners strongly believe in this diagnosis, and others are strongly opposed to this diagnosis. This confusion may lead to misdiagnosis, which may lead to inappropriate treatments. We are striving to clarify this confusion.

Please read the details of the study below. The following information is included: a copy of the agreement of "informed consent" that you will be required to sign should you agree to participate in the study; a description of the study; and a description of what will be required of you should you choose to participate in this study.

If you have any questions, please do not hesitate to contact me.

Many thanks and kind regards

Ms. Rony Sklar

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Tel (work): 011 559 4760

Email: Rony.Sklar@gmail.com

Description of the Study

Aim of this Study

This study aims to explore brain functions associated with the symptom of “hyperfocus” or “flow” in adult ADHD. Hyperfocus/flow is defined as “intensive concentration on interesting and non-routine activities accompanied by temporarily diminished perception of the environment” (Schecklmann et al., 2009, p.104). This symptom has been noted in many articles about ADHD, but has not been researched sufficiently.

Participants in the study will be connected to an Electroencephalograph (EEG) whilst playing a video game.

Screening Procedure

The purpose of the study as well as the method of collecting data necessitate that strict criteria are adhered to as to who can be included as participants. All volunteers are thus asked to undergo the following screening process in order to assist in the selection of suitable participants.

1) ASRS (Adult ADHD Self Report Scale)

Because this study is exploring a symptom in adult ADHD, individuals who volunteer are requested to complete the Adult ADHD Self Report Scale (ASRS), a short questionnaire which rates respondents from low to high in adult ADHD symptoms. This allows the researcher to draw conclusions about the results as they relate to Adult ADHD.

2) Biographical Questionnaire

Certain criteria need to be met by participants to be considered suitable for participating in a study that uses an EEG to record brainwave activity. This is because there are various factors that will impact on the results of an EEG reading. Therefore, all volunteers will be required to complete a Biographical Questionnaire to rule out the factors that could impact on the results. Participants will notice that some of the questions are highly personal - all questions are included specifically for screening purposes, and to ensure that research findings are as reliable as possible. Please be reminded that all responses are kept completely confidential - please be as honest as possible in answering the questions.

The Main Study

Once participants have been selected through the screening procedure outlined above, the researcher will contact participants to arrange a time for the participant to come in for the EEG session. Participants will be requested to refrain from certain substances for a certain amount of time before being tested (e.g. nicotine, caffeine, alcohol, Ritalin). Participants will be asked to fill in a brief questionnaire disclosing this information (i.e. that they did or did not refrain from the substances) just before undergoing the EEG test.

Instruments

- Electroencephalograph (EEG)

The EEG is an instrument that can be used to measure brainwave activity. There are contact points inside the cap that are positioned to measure the activity in different parts of the brain. This cap is connected with wires to the main EEG device as well as a computer, which then allows for the researcher to record brainwave activity.

When fitting the cap, the researcher applies small spots of gel to the inside of the cap to ensure that the electrodes establish contact with the participant's scalp. This gel is harmless and any excess gel remaining after the session can be wiped away quite easily.

- Computer Game

Participants will be required to play a computer game whilst connected to the EEG. The video game is a "first-person shooter game".

Outline of the Procedure

- Participant will complete a consent form, expressing consent to take part in the study and return it to the researcher.
- The participant will complete the Adult ADHD Self Report Questionnaire and the Biographical Questionnaire and return them to the researcher.
- After ensuring that the participant is suitable for the EEG study, the researcher will contact the participant to arrange a time to undergo the EEG test.
- The participant will be asked to refrain from using certain substances for a certain time frame before undergoing the EEG test (to ensure that the test results are reliable). The participant will be asked to disclose this information in a brief questionnaire immediately before the EEG test. The substances and time frames that the participants are asked to adhere to are as follows:

- •Please do not drink caffeinated drinks (Coffee, Tea, Coke, etc.) in the few hours prior to the test
- •If you are a smoker, please do not smoke for 4 hours prior to the EEG test
- •If you take Ritalin, please refrain from taking your dose of Ritalin in the 12 hours prior to the EEG test
- •Please do not consume any alcohol in the 24 hours before the test
- •Please do not take any recreational drugs in the days before coming in for the EEG test

- •The researcher will explain procedure in detail to the participant.
- •Participant will be fitted with the EEG cap.
- •Participant's brainwave activity will be measured for 3 minutes while eyes are closed, then 3 minutes with eyes open (this provides a 'baseline' for the researcher)
- •Participant will be given a few minutes to familiarise him/herself with the computer game.
- •Participant's brainwave activity will be measured while playing the game.
- •Following the EEG, participants may be interviewed briefly regarding certain aspects of the experiment.

The entire EEG test process should not take longer than an hour.

Appendix C: Informed consent form

I _____ (full name Participant) agree to participate in the study based on the following:

Confidentiality

My confidentiality will be ensured at all stages before, during and after the study.

All information that I provide will be kept completely confidential, and will only be used for screening purposes, and for reporting on statistics. When statistics are reported, my identity will not be revealed in any way.

All information that I provide will be kept in a secure place.

Voluntary Participation

My participation in this study is completely voluntary.

I may withdraw from the study at any point without suffering any negative consequences.

Ethics

The procedures and method of this study have been approved by the UJ Faculty Higher Degrees Committee.

I will not be harmed in any way during this study.

The results of the study will be provided to me (on request) after the study.

Date: _____

Signature (Participant):_____

Signature (Researcher):_____

Appendix D: Instructions for participants prior to appointment

Dear

Once again, thank you for your willingness to participate.

Please diarise the following details for your EEG assessment:

Date:
Time:
Venue: Prof Burke's Office - Psychology Department (C-Ring 4), Office 435

Please arrive a few minutes early to ensure that we can start the assessment on time.

To ensure that the EEG results can be used, please remember to adhere to the following before coming for your assessment:

1) Please refrain from consuming any alcohol in the 24 hours before your appointment.
2) Please refrain from using any recreational drugs at all before your appointment.
3) If you have been prescribed Ritalin, please do not take your dosage for 12 hours before the assessment (you may bring this with you to take straight after the assessment).
4) Please do not drink coffee or any other caffeinated drinks for 4 hours before your appointment.
5) If you are a smoker, please do not smoke a cigarette in the 4 hours before your appointment.

If for any reason, you need to reschedule your appointment, please contact me as soon as possible to notify me. This is very important, as other researchers need to book the equipment, so will need to know about any changes in the booking schedule.

Thank you again and I look forward to meeting you.

Kind regards

Ms. Rony Sklar

Rony.Sklar@gmail.com
072-216-7044

Appendix E: Pre-EEG questionnaire

Brief Questionnaire before EEG

The recordings obtained from an EEG can be affected by various factors. The following information is important for ensuring that the results are as reliable as possible. Therefore we ask that you answer honestly.

1. Have you consumed any alcohol in the past 24 hours?

☐ Yes
☐ No

2. Have you used any drugs (besides for prescription medication) in the past 60 days?

☐ Yes
☐ No

3. If you have been prescribed Ritalin to be used daily, have you taken Ritalin today?

☐ Yes
☐ No

4. Have you drunk coffee or any other drink with caffeine in the past few hours?

☐ Yes
☐ No

5. Have you smoked a cigarette in the past 4 hours?

☐ Yes
☐ No

6. If you have smoked a cigarette in the past 4 hours, please indicate approximately how long ago your last cigarette was:

_____

Appendix F: Post-EEG questionnaire

Quick Questionnaire

Name: _____

Surname: _____

1. Without looking at your watch, how much time do you think you spent playing the game (including the 'basic training' (give an estimate in minutes)?

_____

2. Were you aware of anything happening around you while you were playing (e.g. noises, movements)?

☐ Yes
☐ No

Please specify:

_____

_____

Thank you for taking the time to be a part of this study.