EEG brain wave activity at rest and during evoked attention in children with attention-deficit/hyperactivity disorder and effects of methylphenidate

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Short title: EEG brain wave activity in children with ADHD

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Abstract

Objective: The aim of this study was to assess baseline EEG brain wave activity in children with ADHD and to examine the effects of evoked attention and methylphenidate on this activity.

Method: Children with ADHD (n=19) were tested while they were stimulant-free and during a period in which they were on stimulant (methylphenidate) medication. Control subjects (n = 18) were tested once. EEG brain wave activity was tested both at baseline and during focussed attention. Attention was evoked and EEG brain wave activity was determined by means of the BioGraph Infiniti biofeedback apparatus.

Results: The main finding of this study was that control subjects and stimulant-free children with ADHD exhibited the expected reactivity in high alpha wave (11-12 Hz) activity from baseline to focussed attention; however, methylphenidate appeared to abolish this reactivity.

Conclusion: Methylphenidate attenuates the normal cortical response to a cognitive challenge.

Clinical applications

These results indicate that methylphenidate may have a suppressive effect on the normal cognitive stress response. The implications of this suppression on the cognitive and psychological well-being of these children warrants the need for further investigation.

Limitations of the study

- Only 19 children with ADHD and 18 control subjects were tested.
- Further studies should include prior testing in order to exclude children with possible co-existing learning disabilities.

Keywords

ADHD, methylphenidate, Ritalin, EEG, high alpha activity, focussed attention, cortical reactivity, cognitive stressor.

Introduction and background information

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with normal functioning or development and directly impacts on social and academic or occupational activities [1]. The disorder occurs in most cultures with a prevalence rate of about 5% of children, according to the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) [1]; making it the most prevalent psychiatric disorder of childhood. In fact, it has been reported that ADHD cases may well represent as much as 50% of child psychiatric clinic populations in America [2]. This disorder is not limited to childhood with studies suggesting that approximately 50-70% of children with ADHD will continue to suffer from this disorder into adulthood [2].

Resting electroencephalogram (EEG)

The normal paediatric resting EEG is characterised by a small amount of 4-8 Hz theta activity, significant 8-13 Hz alpha activity and scattered 14 Hz and above beta activity [3]. Significant deviations from this pattern have however emerged in children suffering from ADHD. Indeed, studies have shown that children with ADHD display slow-wave EEG elevations, manifested by elevated levels of delta [4–6] and theta power [2,4–8], and fast-wave EEG deficits, as indicated by decreased levels of alpha [2,4–6], beta [2,4–8] and even gamma [4] activity, when compared to control subjects. Recent research has, however, suggested that there exists an atypical subgroup of children with ADHD that display excess beta activity as opposed to the excess theta power typical of this disorder [9].

Research has advocated the use of an attentional index in the diagnosis of patients with ADHD [10]. This attentional index involves the calculation of a theta/beta power ratio and is generally calculated as an average across baseline, reading, listening and drawing tasks [10]. This index is defined as a "biological" measure of attention [11], and its determination is said to allow for the assessment of the presence and severity of ADHD [10]. Studies have shown that children with ADHD display elevated resting levels of both theta/beta [2,5,7,8] and theta/alpha ratios [2,6]. Theta/beta ratios have been shown to discriminate subjects with ADHD from control subjects with 86% sensitivity and 98% specificity [2].

EEG during focussed attention

During cognitive stress, changes that are characteristically seen in the EEG include a reduction in alpha power and the replacement of these waves by higher frequency beta waves [12]. Again, deviations from this characteristic pattern have been shown to occur in children suffering from ADHD. Studies have shown that children with ADHD show a greater increase in slow cortical theta activity [2,13], and a greater decrease in fast cortical alpha [13] and beta activity [2,13] during tasks that require sustained concentration or attention such as continuous performance tasks. Results are however sometimes contradictory, as it has also been shown that a cognitive challenge such as a continuous performance task can actually induce an alpha power increase in children with ADHD while evoking an alpha power decrease in control subjects [14]. These findings are suggested to be due to the inability or decreased ability of children with ADHD to suppress or deactivate activity in the default-mode network during tasks that require

cognitive focus [15]. The default-mode network consists of a widely distributed network of related cortical regions that are active during resting states, and whose activity is believed to be incompatible with goal-directed activity such as focussed attention [16]. The activity of this default-mode network is believed to be synchronized through very low frequency (0.06-0.2 Hz) oscillations in the resting brain [17]. The disruption of this network in individuals with ADHD is elegantly addressed by the "default-mode interference hypothesis" of Sonuga-Barke and Castellanos [16] which states that fluctuations in the quality of focussed attention occur in these individuals due to failure to effectively transition from default-mode activation to an active processing mode during a cognitive task. In other words, the low frequency EEG activity that is normally attenuated during goal-directed tasks such as focussed attention persists into or re-emerges during active processing to the extent that it competes with task-specific neural processing and results in attentional lapses [16]. Indeed, this hypothesis was tested by Helps *et al* [17,18] who found that children with ADHD showed less very low frequency attenuation during rest-to-task transition. Furthermore, this reduced attenuation appeared to be associated with a number of measures of active task (two choice reaction time task) performance [17] and inversely associated with ADHD symptoms [18]. In other words, participants who were less able to attenuate the default-mode network made more errors, were slower and more variable that those who could effectively attenuate this network during the cognitive task [17]. Interestingly, studies using functional magnetic resonance imaging (fMRI) have shown that stimulant medication appears to have the ability to significantly improve the suppression of default-mode activity in the ventral anterior cingulate cortex in children with ADHD [19].

Effect of stimulant medication on the EEG

Studies that have investigated the effect of stimulant medication commonly prescribed for the treatment of ADHD on the EEG of children with ADHD are limited. However, an interesting study by Clarke *et al* [6] has shown that the use of stimulant medication in children with ADHD appears to have the ability to normalise the EEG by specifically decreasing theta and increasing beta activity. Studies in children suffering from ADHD that exhibit excessive beta activity, on the other hand, have shown that in this subgroup specifically stimulant usage appears to have the ability to decrease this excessive high frequency activity [20]. However, it should be noted here that these changes were suggested to be due to the reduction in total power shown by these participants and not a normalization of beta activity [20]. Moreover, a study by Ozlem *et al* [21], has shown that 3 month treatment with stimulant medication was unable to induce any change in EEG activity in 57.1% of children with ADHD, while 23.8% showed improvement and 19.0% showed worsening of EEG activity.

The aim of this study, therefore, was to investigate baseline EEG brain wave profiles of children with ADHD, as compared to control subjects; as well as to examine the effects of both evoked attention and methylphenidate on these profiles.

Methods

The study protocol was submitted to and approved by the Faculty of Health Sciences Research Ethics Committee, University of Pretoria, clearance number S30/2007, and conducted in accordance with the Declaration of Helsinki and CGP/ICH guidelines. The

protocol was, furthermore, submitted to and approved by the South African Department of Health, DOH trial number DOH-27-0808-1816. Patient recruitment and supervision was conducted by a registered psychiatrist involved in the study. Only children from whom and from whose guardians/parents voluntary informed assent/consent could be obtained were included in the study.

Subjects

The experimental group consisted of 19 children diagnosed with ADHD by a registered psychiatrist, according to the text revised Fourth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (13 boys and 6 girls, ages 6 to 15 years, mean 9.53 years). Although high male: female ratios of ADHD are sometimes seen in clinic settings, it is believed that females are generally underrepresented in such settings due to, amongst other factors, referral bias [22]. Results from population studies, on the other hand, have found male:female ratios closer to 2:1, suggesting that females are underdiagnosed in the community [22]. Interestingly, a recent study in Nigeria has even found that no association between gender and ADHD exists [23]. Exclusion criteria included children with co-morbidities such as conduct disorder, children on medication other than methylphenidate (including other stimulant mediation, as well as medication known to affect the autonomic nervous system), overtly malnourished children, children suffering from mental retardation and children with the inability to understand and give informed assent. Children were also excluded if informed consent was not obtained from their parent or guardian. The children with ADHD were regarded as being on stimulant medication if they had been taking methylphenidate (Ritalin) consistently for at least 10

days at the dosage prescribed specifically for them by their psychiatrist. Eighteen of the children with ADHD tested in our study were taking short-acting methylphenidate at a dosage of 10 mg, while one child was on long-acting methylphenidate at a dosage of 20 mg. These same children were tested after they had refrained from taking methylphenidate for a period of approximately three weeks during their school holidays and were then considered to be stimulant-free. The practise of taking children with ADHD off their stimulant medication during their school holidays is normal and was not introduced into the treatment regime as part of the study. The control group consisted of 18 age- and gender-matched control subjects (12 boys and 6 girls, 7 to 13 years, mean 9.17 years) who did not have any psychiatric illnesses, were not on any medication, were not overtly malnourished, did not suffer from mental retardation and could understand and give informed assent. The control subjects were recruited from a local primary school by the registered psychiatrist involved in the study using the aforementioned criteria. Voluntary informed consent was also obtained from the parents/guardians of the control subjects.

Electroencephalography values

EEG values were measured by means of a BioGraph Infiniti biofeedback program designed specifically to increase attentive abilities in individuals with ADHD. This program extracts EEG frequency components and feeds them back to the participant in the form of a game, using an audio-visual loop. The specific biofeedback program used in this study was a bowling EEG speed game. The EEG signal was collected through silver-silver chloride electrodes attached to the skull. Electrode positioning was

monopolar with the active electrode positioned at C4 according to the neurofeedback protocol used for this game. Mastoid reference electrodes were placed on both of the subject's earlobes. Electrode skin contact was checked and all impedances were kept below 10kΩ. Data was sampled while the children were sitting in a quiet environment at a constant room temperature. Baseline recordings were made over a period of 5 minutes. The 5 minute baseline recordings were directly followed by 10 minute recordings during focussed attention. The biofeedback program used contains within it an assessment program which uses an analog-to-digital converter to sample the unfiltered analog EEG signal in order to alter it into a digitised signal. This signal is then filtered with a Fast Fourier Transformation in order to produce a digitally filtered signal. This signal is then amplified by a digital-to-analog converter in order to produce the final filtered analog signal. This software also contains within it a function that allows for the auto-rejection of artifact signals, which was set at ±50 μV. The assessment program was used to determine the power within each of the following brain waves: theta (4-8 Hz), thalpha (6-10 Hz), low alpha (8-10 Hz), high alpha (11-12 Hz), SMR (13-15 Hz), beta 1 (16-20 Hz) and beta 2 (13-21 Hz), as well as theta/beta and theta/SMR ratios.

Statistics

All data was statistically analysed using Stata™ Data Analysis Software. The Mann-Whitney test was used when comparing values obtained for the children with ADHD to those obtained for the control subjects. When comparing values obtained while the children with ADHD were on stimulant medication to those obtained while they were stimulant-free, the Wilcoxon signed-rank test was used. The Wilcoxon signed-rank test

was also used when comparing participants' values at baseline to values during focussed attention.

Results

No statistically significant effects were found with regards to any of the brain waves other than alpha waves. Therefore the results will focus on alpha wave activity. Means and standard deviations for alpha power of all three groups are presented in Table 1.

Baseline alpha power

No statistically significant differences in baseline alpha power were found when comparing children with ADHD (whether on stimulant medication or stimulant-free) to control subjects. However, when baseline alpha power values obtained while the children with ADHD were on stimulant medication were compared to values obtained while they were stimulant-free, a statistically significant difference in high alpha power ($p=0.0298$) was found, with high alpha power found to be significantly greater in stimulant-free children with ADHD. This suggests that methylphenidate usage appears to attenuate baseline high alpha 11-12 Hz wave activity in children with ADHD.

Alpha power during evoked attention

When comparing alpha power during focussed attention of stimulant-free children with ADHD to control subjects, no statistically significant differences were found. Interestingly though, when these same children were compared to control subjects while they were on stimulant medication, statistically significant differences in low alpha

(p=0.0418) and high alpha (p=0.0164) power were found, with low alpha power found to be significantly lower and high alpha power found to be significantly higher in children with ADHD on stimulant medication. This suggests that methylphenidate has the ability to influence alpha power activity during focussed attention in these individuals. In the final analysis, alpha power during focussed attention was compared between children with ADHD while they were on stimulant medication and while they were stimulant-free. In this comparison a statistically significant difference in high alpha power (p=0.0486) was found, with high alpha power found to be significantly greater in children with ADHD while on stimulant medication. This finding therefore suggests that methylphenidate has the ability to disrupt the attenuation of high alpha power that occurs during focussed attention in children with ADHD.

Difference in alpha power between baseline and focussed attention

When examining the shift in alpha power from baseline to focussed attention in control subjects, the expected statistically significant decrease in high alpha power was found (p=0.0016). This represents the characteristic alpha block that occurs during the cognitive shift from resting state to focussed attention. When examining the shift in alpha power from baseline to focussed attention in stimulant-free children with ADHD the same statistically significant decrease in high alpha power was seen (p=0.0022). Interestingly, when these same children with ADHD were tested while they were on stimulant medication, no statistically significant difference in high alpha power between baseline and focussed attention was found. This finding suggests that methylphenidate has the potential to disrupt the shift in high alpha power from baseline to focussed

attention such that the alpha block characteristic of states associated with focussed attention is not seen in individuals taking this stimulant.

Delta values of alpha power

Delta values of alpha power were calculated by determining the difference between baseline values and values during focussed attention (Δ = focussed attention – baseline). Means and standard deviations for delta values of all three groups are represented in table 2.

No statistically significant differences in delta values of alpha power were found when comparing stimulant-free children with ADHD and control subjects, suggesting once again that both of these groups displayed the normal alpha block characteristic of the cognitive shift from baseline to focussed attention. When comparing the children with ADHD while they were on stimulant medication to control subjects, however, a statistically significant difference in the delta values of high alpha ($p=0.0127$) was found, such that delta values for children with ADHD on stimulant medication were found to be attenuated when compared to the delta values of control subjects. This once again suggests that methylphenidate usage may block the characteristic alpha reactivity normally seen during the shift from baseline to focussed attention. To confirm these findings, we compared delta values of alpha power between children with ADHD while they were on stimulant medication and while they were stimulant-free and, once again, a statistically significant difference in delta values of high alpha power ($p=0.0048$) was found, with delta values of high alpha power found to be larger in stimulant-free children

with ADHD. This confirms the finding that methylphenidate has the potential to block the reactivity of high alpha power from baseline to focussed attention in children with ADHD.

Discussion

Although previous studies have suggested that individuals suffering from ADHD may be distinguished from control subjects on the basis of specific EEG findings both at baseline and during a cognitive stressor, our findings did not support this. This could be due to the small sample sizes of our study, as well as the sensitivity of the EEG equipment used; however, the fact that differences in alpha wave activity were still found despite these limitations suggests that the alpha wave may be the most sensitive EEG wave to differences in cortical activity between subjects with ADHD and controls. This suggests that alpha wave activity, and more specifically high alpha wave activity, may possess the most potential in the development of an EEG diagnostic tool for ADHD.

Alpha wave activity of the cortex is believed to inversely represent cortical activity, such that high levels of cortical activity are associated with low levels of alpha wave activity and vice versa [12]. The alpha wave can, however, be divided into low and high frequency components, with low alpha wave activity believed to inversely represent general cortical arousal while high alpha wave activity inversely represents task-specific attentional processes [24]. Low alpha power is believed to be associated with more internalized attention by providing a relaxed, defocused witness perspective to multimodal information processing, while high alpha power is more associated with the

encoding and processing of semantic information [25]. In other words, low alpha power is associated with the switching off of external attention [26] while high alpha power represents external semantic processing [27]. Therefore a decrease in high alpha activity is believed to represent heightened task-relevant attentional processing since it is believed to reflect oscillations of selective neural systems involved in the elaboration of task-related information and efficient information transfer within thalamocortical pathways [28]. In support of this, a decrease in high alpha power has been shown to occur during both a psychological and a physical stressor [29]. Furthermore, a greater attenuation of high alpha wave activity over the motor cortex has been shown to occur in golf experts, when compared to novices, during putting preparation [30]. Moreover, greater attenuation of high alpha activity preceded putts that were holed by these golfers [30]. This confirms the suggestion that an attenuation of high alpha wave activity represents a focusing of attention and the mobilising and devotion of neural resources to task-specific response programming [30]. This suggestion has recently been supported in a study by Cooke *et al* [31] which found that less high alpha power was found in golfers following a putting error – a state that is known to be associated with the allocation of more resources to response programming. Moreover, the ability to attenuate high alpha wave activity during a task is believed to underlie successful performance in that task [30]. Similarly, decreases in high alpha wave activity have been shown to occur during working memory tasks and are therefore believed to represent the enhancement of task-related short-term memory processes [28].

The most concerning result of the present study therefore is the finding that methylphenidate usage has the ability to switch off this task-specific attentional processing since children with ADHD on stimulant medication were unable to display the characteristic decrease in high alpha wave activity associated with focussed attention. On closer inspection it becomes apparent that this decreased reactivity of the high alpha wave is due to the ability of methylphenidate to decrease baseline high alpha activity and furthermore increase high alpha activity during focussed attention, decreasing the reactivity of this wave between baseline and evoked attention. The decreased baseline high alpha activity can be explained by the fact that stimulant medications like methylphenidate are known to activate the cortex; however it seems that this high baseline cortical activation has the detrimental effect of abolishing the ability of the cortex to then react to a cognitive stressor which requires focussed attention. One can only assume that by increasing baseline cortical activity a ceiling effect may be created whereby the high baseline cortical activity exhausts reserve cortical activation in response to a cognitive challenge. Interestingly, the same effect on the autonomic functioning of these individuals has previously been found [32].

Conclusions

Methylphenidate appears to interfere with the normal reactivity of the cerebral cortex to a cognitive challenge in children with ADHD. This may very well have negative implications for the psychological and cognitive well-being of these children.

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Conflicts of interest

Both authors declare that they have no conflicts of interest.

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