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Effects of stimulant medications on the EEG of children with Attention-Deficit/Hyperactivity Disorder Predominantly Inattentive type

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Abstract

Stimulant medications are the most commonly-used treatments for Attention-Deficit/Hyperactivity Disorder (ADHD) in North America and Australia, although it is still not entirely known how these medications work. This study investigated the effects of stimulant medications on the EEG of children with the Inattentive type of ADHD. An initial EEG was recorded during an eyes-closed resting condition and Fourier transformed to provide absolute and relative power estimates for the delta, theta, alpha and beta bands. Theta/alpha and theta/beta ratios were also calculated. Subjects were placed on a 6-month trial of a stimulant and a second EEG was recorded at the end of the trial. Subjects were included in this study only if they showed a good clinical response during the trial. The unmedicated ADHD group had significantly greater absolute and relative theta, less relative alpha, and higher theta/alpha and theta/beta ratios than the control group. The stimulant medications resulted in a normalisation of the EEG, with changes in the theta, alpha and beta bands being most evident. These results suggest that stimulants act to increase cortical arousal in children with ADHD, normalising their EEG.
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Keywords: Attention-Deficit/Hyperactivity Disorder; Children; EEG; Stimulants; Medication

1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common psychiatric conditions of childhood, effecting between 4 and 6% of school-age children (Pelham et al., 1992; Lindgren et al., 1990; APA, 1994). ADHD can be a pervasive problem with symptoms persisting into adulthood in between 40 and 70% of individuals (Bellak and Black, 1992). It interferes with many areas of normal development and functioning in a child's life, and if untreated, it predisposes the child to psychiatric and social pathology in later life.

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For over half a century ADHD has been treated with stimulant medications such as Methylphenidate and Dexamphetamine. In North America clinical guidelines recommend an initial trial of medication for the treatment of ADHD before other interventions are explored (Swanson et al., 1998). In numerous controlled trials (Wilens and Biederman, 1992), and clinical reports (Swanson et al., 1993), nearly 80% of children with ADHD have demonstrated clinically significant benefits from medication, with improvements in the core symptoms of the disorder, namely increasing attention, and decreasing impulsivity and gross motor activity.

While stimulants have a long-standing history for the treatment of ADHD, their precise effects on brain functioning are still not fully understood. One model of the disorder proposes that ADHD results from hypofunctionality of catecholaminergic pathways projecting to prefrontal cortical areas (Todd and Botteron, 2001). This is supported by neuroimaging studies that have found structural changes in both basal ganglia structures and the prefrontal lobes (Castellanos et al., 1996; Filipek et al., 1997). However, it is not clear whether the problems seen in ADHD result from decreased activity in these pathways or less responsivity of the targets of these paths. Stimulant medications appear to produce their therapeutic affect by increasing catecholamines in the synaptic cleft (Spencer et al., 1996), although again, the exact regions of the brain that these medications act on are not entirely clear.

EEG studies of children with and without ADHD have typically found that ADHD children have increased theta activity, which is often maximal in the frontal regions (Satterfield et al., 1972; Janzen et al., 1995; Chabot and Serfontein, 1996; Lazzaro et al., 1998; Clarke et al., 1998, 2001b,c; Barry et al., in press), increased posterior delta (Matousek et al., 1984; Clarke et al., 1998, 2001b,c) and decreased alpha and beta activity (Dykman et al., 1982; Callaway et al., 1983; Barry et al., in press), also most apparent in the posterior regions (Clarke et al., 1998, 2001b,c; Lazzaro et al., 1998; Barry et al., in press). Calculations of ratios of EEG activity between frequency bands have also been used to assess differences between clinical groups, with ADHD children having an increase in the theta/alpha (Matousek et al., 1984; Ucles and Lorente, 1996; Clarke et al., 1998, 2001b,c) and theta/beta ratios (Lubar, 1991; Janzen et al., 1995; Clarke et al., 1998, 2001b,c) compared to normal children. These results have been interpreted as indicating that ADHD children have a maturational lag in CNS development (Clarke et al., 1998) or are cortically hypoaroused (Lubar, 1991).

The majority of these studies have investigated children suffering the Combined type of ADHD, which has hyperactivity as a major component of the disorder, with few EEG investigations of the Inattentive type of ADHD. Studies that have investigated the Inattentive type of ADHD have found that these children have EEG abnormalities substantially similar to those of children with the Combined type, particularly increased theta activity, and deficiencies of alpha and beta activity (Mann et al., 1992; Clarke et al., 1998, 2001b,c; Chabot and Serfontein, 1996), but the degree of abnormality in their EEGs is not as extreme as that found in hyperactive children (Clarke et al., 1998, 2001b,c; Chabot and Serfontein, 1996).

In comparison to the number of EEG studies of children with ADHD, only a few studies have investigated changes in the EEG due to stimulant medications, and little consensus has been achieved within these. Chabot et al. (1999) found that 56.9% of a sample of children with ADHD showed a normalisation of the EEG after the administration of a stimulant, while 33.8% remained unchanged and 9.3% showed an increase in EEG abnormality. Swartwood et al. (1998), Lubar et al. (1999) investigated the effects of Methylphenidate in 23 boys with ADHD. Results from these studies failed to identify any global changes in the EEG due to medication. From this it was concluded that Methylphenidate may affect the brainstem and other subcortical areas rather than cortical functioning. In a preliminary report Loo et al. (1999) found that, after administration of Methylphenidate, good responders had decreased theta and alpha, and increased beta activity in the frontal regions, while poor responders showed the opposite EEG changes. The limitation of that study was that only 10 ADHD

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