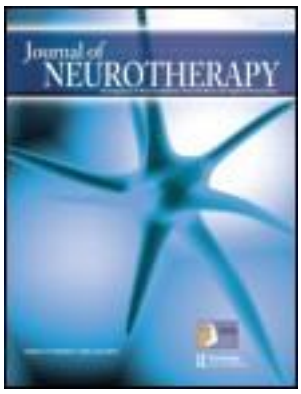

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EEG Activity in Subtypes of Attention-Deficit/ Hyperactivity Disorder

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EEG Activity in Subtypes of Attention-Deficit/Hyperactivity Disorder

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ABSTRACT. This article is a review of electroencephalography (EEG) studies of different types and subtypes of Attention-Deficit/Hyperactivity Disorder (AD/HD). The review outlines the definitional history of

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AD/HD, and changes that have been made to the conceptualization of the disorder as these different definitions have impacted on the EEG literature. EEG studies are examined using various models of AD/HD based on either behaviour or underlying central nervous system (CNS) abnormalities. From these studies, it appears that AD/HD children generally have increased absolute and relative power in the theta band, either at the frontal electrode sites or over the entire scalp. Reductions in absolute and relative power in the alpha and beta bands have also been found in several studies, although relative power measures appear to be more reliable than absolute power. Increased delta activity in both absolute and relative power has also been noted in several studies. These results are discussed in terms of existing CNS-based models of AD/HD, which attribute the disorder to hypoarousal or a maturational lag in CNS development. Implications of these data for clinical use and future research and development in AD/HD are considered.

KEYWORDS. Attention-deficit/hyperactivity disorder, electrophysiology, electroencephalography, EEG, review

INTRODUCTION

Attention Deficit/Hyperactivity Disorder (AD/HD) is one of the disorders most commonly treated by child and adolescent psychiatrists in North America, with these children comprising as much as 50% of child psychiatry clinic populations (Cantwell, 1996). The disorder, as defined in the fourth edition of the Diagnostic and Statistical Manual (DSM-IV), is characterized by varying levels of hyperactivity, impulsivity and inattention which may change with development from preschool through adulthood (APA,1994). AD/HD interferes with many areas of normal development and functioning in a child's life, and if untreated, it predisposes a child to social and psychiatric pathology in later life.

Prevalence rates vary according to the population that is sampled, the diagnostic criteria, and diagnostic instruments that are used. However, the DSM-IV (APA, 1994) estimates the prevalence of AD/HD in the general population at approximately three to five percent of school-age children. In both clinical and epidemiological samples, AD/HD is more

common in males than females (James & Taylor, 1990), with relative rates of up to nine to one in clinical samples, and four to one in epidemiological studies (APA, 1994), although community-based studies have found ratios of males to females as low as two to one (Szatmari, 1992; Taylor, Heptinstall, Sonuga-Barke, & Sandberg, 1998).

One of the major problems in determining who has AD/HD is that there is no international consensus on what cluster(s) of behaviours actually warrant classification as a valid disorder. Substantial changes have occurred in consecutive editions of the DSM, and the criteria in the DSM-IV are not in agreement with the criteria from other diagnostic systems. This lack of consensus has been used as one of the major criticisms of the use of quantitative electroencephalographic (qEEG) markers in diagnosing AD/HD (Levy & Ward, 1995).

While there are good descriptions of AD/HD which date back to early last century (Still, 1902), it was not until 1968 that the first formal diagnostic criteria were published in the DSM-II (APA, 1968). In this edition, the disorder was listed under the title "hyperkinetic reaction of childhood." This was a disorder with only one type which was characterised by over activity, restlessness, distractibility and short attention span, with the major emphasis being placed on the overt behavioural aspects. The DSM-II was criticized as having major limitations in childhood psychiatric disorders generally (Cantwell, Russell, Mattison, & Will, 1979), including categories which were too inclusive and vague, or too limited. In relation to AD/HD, subsequent research (Douglas, 1972; Douglas & Peters, 1979) found that problems of poor sustained attention and impulse control were as important, if not more so, than the hyperactivity.

Following on from this research, the DSM-III (APA, 1980) renamed the disorder "Attention Deficit Disorder," with a greater emphasis being placed on its attentional aspects (Edelbrock, Costello, & Kessler, 1984). The DSM-III distinguished between two types, Attention Deficit Disorder with hyperactivity (ADD/H) and Attention Deficit Disorder without hyperactivity (ADD/WO). The main difference between these two types was that children with ADD/WO exhibited the inattention and impulsive features of the disorder, but not hyperactivity.

In the next manual version, DSM-III-R (APA, 1987), the disorder was renamed "Attention Deficit Hyperactivity Disorder" (ADHD) and was considered unidimensional in nature, with a single behavioural checklist being given for the diagnosis. For a diagnosis of ADHD, a child had to exhibit any eight out of fourteen behaviours. A second category, "Undifferentiated Attention Deficit Disorder" (UADD), was also

included, with the predominant feature being inattention. Under the category of UADD the DSM-III-R did not give specific diagnostic criteria, stating that "further research is necessary to determine if this is a valid diagnostic category and, if so, how it should be defined" (APA, 1987, p. 95).

One reason the DSM-III-R reconceptualized ADHD and dropped the distinct types was that at publication there was not enough empirical support for the DSM-III conceptualization of two distinct types (Barkley, 1990; Frick & Lahey, 1991; Lahey et al., 1988; Lahey & Carlson, 1991; Schaughency & Rothlind, 1991). By conceptualizing ADHD as a unidimensional construct, DSM-III-R also avoided the problem of trying to categorize each symptom under a distinct domain (Newcorn et al., 1989). This approach was also consistent with the criteria for other DSM-III-R disorders, and with other empirical approaches to classification (Barkley, 1990).

At the time, several researchers argued that DSM-III-R was published prematurely and that the APA should have waited for more empirical evidence about the validity of the DSM-III criteria (Cantwell & Baker, 1988; Werry, Reeves, & Elkind, 1987). Suggestions were also made that the revisions were more substantial than were warranted (Cantwell & Baker, 1988; Werry, 1988). The most significant effect in relation to AD/HD was that the group of children assigned the ADHD diagnosis was more heterogeneous than that assigned an ADD/H diagnosis (Lahey et al., 1990; Newcorn et al., 1989).

A number of researchers (Goodman & Poillion, 1992; Shaywitz & Shaywitz, 1991; Weinberg & Emslie, 1991) have identified problems with the DSM-III and DSM-III-R diagnostic systems and the ambiguities associated with the ADD or ADHD diagnosis. However, research published shortly before and after the introduction of DSM-III-R helped to clarify the nature of the behavioural syndrome, and guided the development of DSM-IV. Factor-analytic studies suggested that AD/HD had two dimensions, the first dimension being inattention, and the second being a hyperactivity/impulsivity dimension (Bauermeister, 1992; Bauermeister, Alegria, Bird, Rubio-Stipec, & Canino, 1992; Lahey et al., 1988; Lahey et al., 1994, Pelham, Gnagy, Greenslade, & Milich, 1992; Morgan, Hynd, Ricco, & Hall, 1996). Thus, in the DSM-IV (APA, 1994), the diagnostic criteria changed again, to a two axis disorder which allowed the diagnosis of three types, a predominantly hyperactive/impulsive type, a predominantly inattentive type (AD/HDin) or a combination of the two called the combined type (AD/HDcom). However, there are still problems and disagreement with the DSM-IV crite-

ria, with members of the expert group which developed the criteria (e.g., Lahey, Schaughency, Frame, & Strauss, 1985) suggesting that the inattentive type may be better categorized as a different form of disorder (i.e., depressive) and removed from the AD/HD category.

A second, almost parallel set of criteria for an AD/HD-like syndrome is presented in the World Health Organisation's International Statistical Classification of Diseases and Related Health Problems (ICD). The latest revision of the ICD, the ICD-10 (WHO, 1993) lists criteria for a single disorder entitled Hyperkinetic Disorder. For a child to meet the criteria for this disorder they must show symptoms of inattention, hyperactivity and impulsivity. One of the main differences between the DSM-IV and ICD-10 criteria is that the ICD-10 diagnosis requires symptoms to be present in all three categories of inattention, hyperactivity and impulsivity (Swanson et al., 1998). This means that a diagnosis of Hyperkinetic Disorder under the ICD-10 is similar to a DSM-IV diagnosis of AD/HD Combined type, although different threshold levels are used in the two systems. This allows a child who is diagnosed as having AD/HD predominantly inattentive type (DSM-IV) to possibly meet criteria for the ICD-10 diagnosis.

The ICD-10 also notes that a child may be "sub-threshold" for hyperkinetic disorder. "Children who meet criteria in other ways but do not show abnormalities of hyperactivity/impulsiveness may be recognised as showing *attention deficit*; conversely, children who fall short of criteria for attention problems but meet criteria in other respects may be recognised as showing *activity disorder* . . . These conditions are not yet included in the main classification because of insufficient empirical predictive validation" (WHO, 1993, p. 157).

EEG research into AD/HD has been conducted under all of these diagnostic systems, so it is important to understand how well results from previous systems relate to the present criteria. Morgan et al. (1996) found that the DSM-III diagnoses of ADD/WO and ADD/H corresponded fairly closely with the DSM-IV diagnoses of AD/HD predominantly inattentive type and combined type, respectively. No significant relationship was found between the DSM-III-R diagnosis of ADHD and the DSM-IV diagnosis of AD/HD combined type.

The AD/HD predominantly hyperactive/impulsive type is a new diagnostic category which has received little previous theoretical or investigative attention, and has no relation to previous types in earlier versions of the DSM (Newcorn et al., 1989). At this point, there are no qEEG studies of this type.

Although diagnostic criteria for multiple types of AD/HD have existed since 1980, it was not until 1996 that the first study was published which actually investigated EEG differences between types. However, before discussing these studies of type differences, we will briefly review previous work which investigated a single type of AD/HD, as this work resulted in models of CNS functioning which are still in use today.

The EEG of Children with AD/HD

The majority of early studies were of hyperactive children (Satterfield, Cantwell, Lesser, & Podosin, 1972; Satterfield, Cantwell, Saul, Lesser, & Podsin, 1973; Satterfield, Cantwell, Saul, & Yusin, 1974; Satterfield, Lesser, Saul, & Cantwell, 1973; Satterfield & Cantwell, 1974; Dykman, Holcomb, Oglesby, & Ackerman, 1982; Callaway, Halliday, & Naylor, 1983; Matousek, Rasmussen, & Gilberg, 1984; Matsuura et al., 1993) although two were of children who were primarily inattentive (Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Janzen, Graap, Stephanson, Marshall, & Fitzsimmons, 1995). While these studies used a number of different diagnostic categories and different EEG measures, several results were consistently found. The most robust of these is an increase in theta activity (measured as absolute power, relative power or mean amplitude), either at the frontal electrode sites or over the entire scalp. Reductions in alpha and beta activity have also been found in several studies, although reductions in relative power measures appear to be more reliable than absolute power. Increased delta activity in both absolute and relative power, and amplitude, has also been noted in several studies.

This pattern of results has been interpreted in a number of ways. One interpretation is that these children have a maturational lag in central nervous system (CNS) development. EEG studies of normal children have consistently found that with increasing age, delta and theta activity decrease and alpha and beta increase (Matousek & Petersen, 1973; Matthis & Scheffner, 1980; John et al., 1980; Benninger, Matthis, & Scheffner, 1984; Gasser, Jennen-Steinmetz, Sroka, Verleger, & Mocks, 1988; Clarke, Barry, McCarthy, & Selikowitz, 2001a). Topographical differences in maturation have also been noted (Gasser, Verleger, Bacher, & Sroka, 1988), with delta, theta and alpha activity developing earliest in occipital regions followed by parietal, central and frontal regions. Beta waves develop earliest in central regions, followed by parietal, occipital and then frontal regions. Using the results from these studies and the changes in topography, the typical AD/HD profile of increased delta

and theta activity, with deficiencies of alpha and beta, has been seen as indicative of a maturational lag in CNS development in AD/HD children (Satterfield et al., 1973; Matsuura et al., 1993; Clarke, Barry, McCarthy, & Selikowitz, 1998; Lazzaro et al., 1998; Mann et al., 1992). This model has been seen as being able to explain the fact that hyperactivity appears to diminish with increasing age, indicating that the CNS is reaching maturity albeit at a slower rate than in the normal population. However, hyperactivity is not regarded today as the only symptom of AD/HD, and although hyperactivity does decrease with age, inattention and impulsivity appear to remain into adulthood in between 40% and 60% of sufferers (Bellak & Black, 1992). A second limitation of all of these studies is that they have hypothesised the existence of a maturational lag from the obtained EEG profile without actually comparing their results to those of younger normal children to see if they would appear normal at a younger age. Without this procedure being conducted, it is not possible to say with certainty that this qEEG profile in AD/HD children is actually indicative of a maturational lag.

The second electrophysiology-based model which has been influential within the literature has been the hypoarousal model of AD/HD. This was first proposed by Satterfield and Dawson (1971) who conducted a study of skin conductance levels (SCLs) in hyperactive children. This study initially aimed to test an *overaroused* model of hyperactivity. However, 50% of the hyperactive children had abnormally low SCLs, indicating underarousal in CNS functioning. From the EEG literature in AD/HD, a few studies have determined that their results could not be indicative of a maturational lag, and hence have interpreted them in hypoarousal terms (Satterfield et al., 1972, 1974; Satterfield et al., 1973; Grunewald-Zuberbier, Grunewald, & Rasche, 1975; Bresnahan, Anderson, & Barry, 1999).

QEEG Differences in DSM Types of ADHD

Several studies have investigated qEEG differences between types of AD/HD listed in the DSM-III, DSM-III-R, and DSM-IV. All of these studies have compared the hyperactive variants of the disorder with an inattentive type.

The first published study was by Kuperman, Johnson, Arndt, Lindgren, and Wolraich (1996) who used DSM-III-R criteria to investigate qEEG differences between children with ADHD, UADD and a control group. The control group was found to have more relative delta than the UADD subjects and less relative beta than both groups of children with

ADHD, during an eyes-open resting condition. The UADD group had hemispheric differences, with decreased relative delta and increased relative beta in the left hemisphere. In relative alpha and beta, the UADD group had more extreme EEG results, in comparison to the control group and the ADHD group.

Chabot and Serfontein (1996) studied qEEG differences in 407 children diagnosed using DSM-III criteria for ADD/W and ADD/WO, with results being compared to a normative database (John, Prichep, & Easton, 1987). Children with ADD had an increase in absolute and relative theta, primarily in the frontal regions and at the frontal midline. A slight elevation in relative alpha, and a diffuse decrease in mean frequencies in the alpha and beta bands were also found in some children with ADD. These results were determined to represent a deviation from normal development, probably hypoarousal. This study also noted qEEG differences between the two ADD groups which were mainly in the degree of abnormality of the EEG rather than the two groups showing distinct qEEG abnormalities.

These results are also consistent with a continuum model of AD/HD in which AD/HD is part of a continuum of behaviour that ranges from normal to abnormal. At some point on the continuum, the child's behaviours cease being deemed as within normal limits, and an AD/HD diagnosis is made. The problem with this model is that there is no clear definition of where the threshold between normal and abnormal is, or whether there is a distinct line that divides normal from abnormal. In terms of the EEG, more abnormal behaviour is associated with more abnormal qEEGs.

Clarke, Barry, McCarthy, and Selikowitz (1998, 2001d) investigated differences between children with AD/HDcom and AD/HDin using DSM-IV criteria. The two AD/HD groups had increased levels of absolute and relative theta and decreased levels of relative alpha and beta. In posterior regions, relative delta was elevated compared to the control group. In both of these studies, the degree of qEEG abnormality was the major difference between the combined and inattentive groups, with the AD/HDin children having qEEG profiles which were not as deviant as those found in children with AD/HDcom. However, Clarke et al. (2001d) indicated the presence of a frontal lobe dysfunction in the AD/HDcom group which was not evident in the AD/HDin group. This was typified by an increase in frontal theta activity. While these results are largely consistent with a continuum model of AD/HD, they also suggest that AD/HDcom children have a frontal lobe deficit which is not found in AD/HDin children.

In both of these studies, the nature of the underlying CNS abnormality was discussed. Clarke et al. (1998) determined that their data were consistent with a maturational lag model of AD/HD, but this explanation was not supported by the second study (Clarke et al., 2001d). Clarke et al. (2001d) calculated ratio coefficients (in terms of lower/higher frequencies) between permutations of the four frequency bands, as well as the mean frequency for each frequency band. It was hypothesised that if AD/HD results from a maturational lag, all ratio coefficients should have been higher and mean frequency lower in the AD/HD groups compared to a control group. In the delta band, the AD/HD group had a higher mean frequency than the control group, but this was reversed in the alpha and beta frequency bands. Similarly, the delta/theta ratio was lower and the theta/beta ratio was higher in the AD/HD group than the control group. These results suggested that there was a truncation of both ends of the normal EEG spectrum in the AD/HD group, which could not have resulted from a maturational lag.

The relationship between the two types was further investigated in a study that examined age-related changes in 160 children with AD/HDcom or AD/HDin types, and 80 controls (Clarke, Barry, McCarthy, & Selikowitz, 2001b). The two AD/HD groups had greater total power and absolute delta and theta, more relative theta, higher theta/alpha and theta/beta ratios, and less relative alpha and beta than controls. Again the qEEGs of the AD/HDin group were similar to, but not as extreme as, those of children with AD/HDcom. With increasing age, the EEG profile of the AD/HDin group was found to change at a similar rate to the changes found in the control group, with the differences in power levels remaining constant. In the AD/HDcom group, power was found to change at a greater rate than in the AD/HDin group, with power levels of the two AD/HD groups becoming similar with age. These results suggested that the hyperactive/impulsive component of the disorder was maturing with age but the inattentive component was stable, which is consistent with behavioural data for this disorder.

Only one study has investigated type differences in the EEG using ICD-10 criteria (Clarke, Barry, McCarthy, & Selikowitz, 2003b). That study investigated EEG differences between children with Hyperkinetic Disorder (HKD), HKD sub-threshold attention deficit (HKDsub) and control children. Results indicated that the HKD group had greater total power and absolute delta and theta, more relative theta, and less relative alpha and beta than the control group. The HKDsub group had EEG profiles which were different from both control children and HKD chil-

dren, with the HKDsub group generally placed between the other two groups.

Summary of qEEG Findings

Most studies have reported that AD/HD groups show elevated levels of slow wave activity in comparison to normal children (see Table 1 and Figure 1). The most reliable measure of this has been relative theta power. Reduced relative alpha and beta have also been found in most power studies, while absolute alpha and beta are less reliable discriminators. Increased delta activity in both absolute and relative measures has also been found in AD/HD, but with far less consistency. In general, qEEG abnormalities appear to be greater in children with AD/HDcom than AD/HDin, which is supportive of a continuum model of AD/HD, whereby more behaviourally-disordered children have more abnormal qEEGs. These studies also support keeping AD/HDin as an AD/HD diagnosis, since their EEGs are qualitatively similar to those from children with the hyperactive variant of the disorder.

EEG-Defined Subtypes of AD/HD

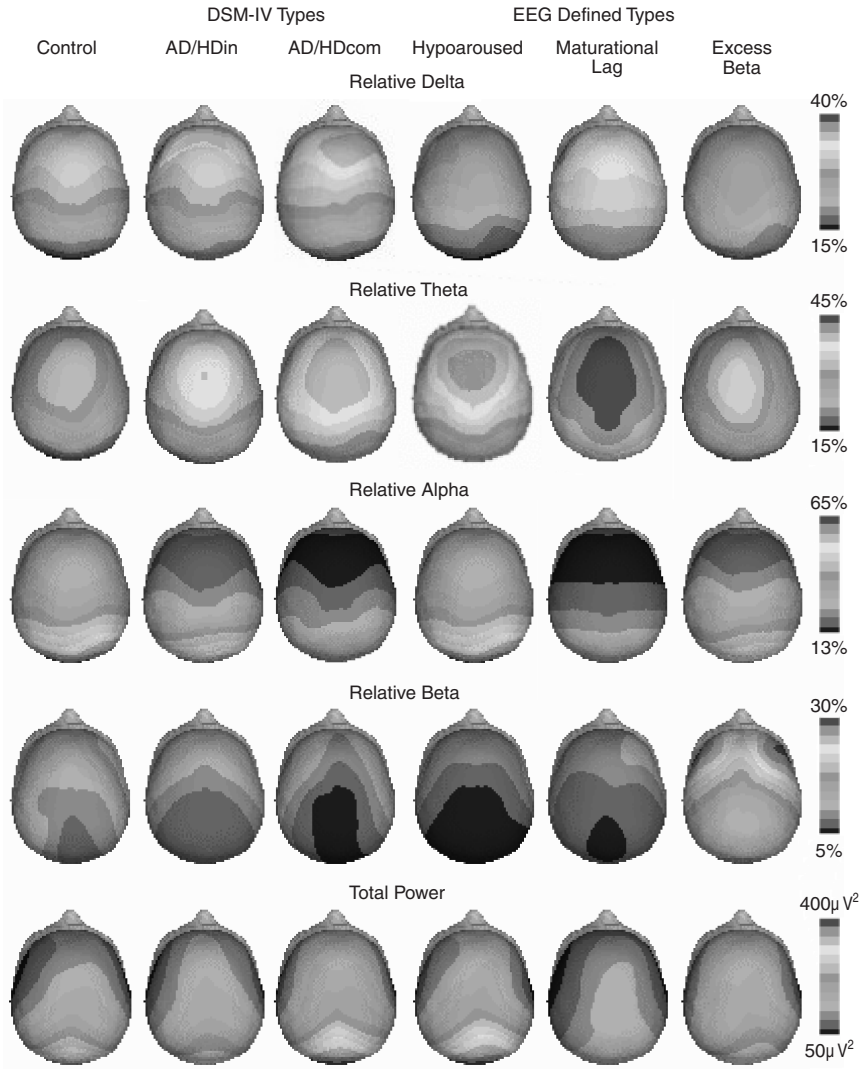
One limitation of most AD/HD studies is that the clinical populations are considered to be homogenous, although there is considerable evi-

TABLE 1. Summary of Typical qEEG Results Within the Literature for DSM and EEG-Defined Types of AD/HD.

Region	AD/HDin	AD/HDcom	Hypoaroused	Maturational Lag	Excess Beta
Delta Frontal	↑	↑	↓↓	↑↑	↓↓
Delta Posterior	-	-	↓↓	↑↑	-
Theta Frontal	↑↑	↑↑	↑↑	↑↑	↓↓
Theta Posterior	↑	↑	↑↑	↑↑	↓↓
Alpha Frontal	↓	↓	-	↓↓	↓↓
Alpha Posterior	↓	↓	-	↓↓	↓↓
Beta Frontal	↓	↓	↓↓	↓↓	↑↑
Beta Posterior	↓	↓	↓↓	↓↓	↑↑
Total Power Frontal	↑	↑	↑↑	↓↓	↑↑
Total Power Posterior	-	-	↑↑	↓↓	-

↑ = Increased power compared to normal, ↓ = Decreased power compared to normal, - = No difference compared to normal, ↑↑ or ↓↓ Consistently found difference, ↑ or ↓ Sometimes found.

FIGURE 1. Typical qEEG topographic head maps for children with DSM-IV combined and inattentive types, as well as EEG-defined subtypes of AD/HD.



dence to the contrary. If this lack of homogeneity is the case, the reported group differences may not accurately reflect the true nature of EEG deviances in these children. A number of studies have reported subgroups of children with distinct EEG profiles. Clarke et al. (1998, 2001d) found between 15 and 20% of children with AD/HDcom had significantly *elevated* levels of beta activity in their EEG. In those studies such cases were removed as statistical outliers, however, the validity of these EEG profiles as indicating a distinct subgroup of AD/HD was tested in a follow-up study (Clarke, Barry, McCarthy, & Selikowitz, 2001e). In a sample of 298 children with AD/HD, children with excess beta activity constituted approximately 18.8% of males with the combined type but only 2.2% of females. This profile was less common in children with AD/HDin, with 3.9% of males having the profile and no females having excess beta activity. These excess-beta children were also found to have a behavioural profile slightly different from other children with AD/HDcom, with an increased rate of temper tantrums and moody behaviours.

Developing from this investigation of children with excess beta activity, Clarke, Barry, McCarthy, and Selikowitz (2001c) explored the possibility that there may be other distinct sub-populations within their group-averaged data. That study examined the EEG of 184 boys with AD/HDcom. Comparison of the total AD/HD sample with the control group found results similar to other AD/HD studies–children with AD/HD had increased theta and deficiencies of alpha and beta activity. However, cluster analysis identified three distinct EEG-defined subtypes in the group (see Table 1 and Figure 1 for an overview of typical results). The first cluster had increased total power, relative theta and theta/beta ratio, with decreased relative delta and beta across all regions. This profile was determined to be indicative of cortical hypoarousal. The second cluster was characterised by increased delta and theta, and deficiencies of alpha and beta activity. Based on the qEEG profile, which utilised all frequencies and their topography, it was determined that this cluster probably indicated a maturational lag in CNS development, although their theta levels were slightly higher than expected for a simple maturational lag, suggesting an additional dysfunction. The third cluster had excess beta activity, and was labeled an over-aroused group because their qEEG profile appeared to be the same as that for the hypoaroused group except for a replacement of the dominant theta activity by beta activity. This study was followed by a replication in children with AD/HDin (Clarke, Barry, McCarthy, Selikowitz, & Brown, 2002). As with the previous study, the entire sample had a typical AD/

HD qEEG profile, although cluster analysis again identified the presence of different EEG profiles. The first of the two clusters obtained was characterised by reduced frontal relative delta and an increase in relative theta, with a reciprocal decrease in relative beta across the scalp, suggesting cortical hypoarousal. The second cluster had increased frontal and decreased posterior total power, increased centro-posterior relative delta, increased relative theta and decreased relative alpha across the scalp, and a decrease in fronto-central relative beta activity, indicative of a maturational lag.

A comparison of the maturational lag and hypoarousal groups from the two studies was next conducted. Results suggested that the hypoarousal cluster in the AD/HDcom group was more hypoaroused than the corresponding children in the AD/HDin group. In the comparison of the two maturational lag groups, no significant differences were found. The former results further supported a continuum model for the hypoaroused groups, but no explanation for the lack of differences between the maturational-lag groups can be given at this time.

In a third study, a cluster analysis was performed on the qEEGs of girls with both the inattentive and combined types of AD/HD (Clarke et al., 2003a). This study identified two EEG clusters in these children. The first cluster comprised 96% of the total sample and was characterised by increased total power, more relative theta, and less relative delta and beta than control subjects. This group appeared to be hypoaroused. The second cluster had substantially-increased total power and relative theta activity, with deficiencies in all other bands. On examination of the second cluster, the subjects appeared to be those with the most deviant qEEGs from the main group rather than qualitatively different from the other group. This indicated that, unlike boys with AD/HD, girls taken from a clinical population represent a far more homogenous population. This finding raises important questions about AD/HD in girls. There is no theoretical reason why a maturational lag group and a group with excess beta should not exist in girls, but children with such profiles do not appear to be referred for treatment, at least in Australia. This leads to two possible hypotheses–either these patterns of brain activity do not result in behavioural problems in girls, or we are simply not identifying all the girls with AD/HD. If the first hypothesis is correct, it may follow that boys with these qEEG profiles are not as substantially disordered (perhaps these are the ones who will outgrow the disorder without substantial ill effects, and may not need a high degree of intervention). On the other hand, if these profiles do represent serious dysfunction and there are girls in the community with this disorder, then we may have

identified a serious health issue in that such girls are not being identified. Knowing that childhood AD/HD predisposes an individual to greater levels of psychopathology in later life, and early intervention appears to reduce later problems, it becomes vital to question whether these girls actually exist in the community and whether they do have unrecognised symptoms of AD/HD.

From this research a new model of AD/HD was proposed (Clarke et al., 2002) which focused on the underlying dysfunction rather than the behavioural profile. The model proposes the existence of three distinct subtypes within AD/HD, and these are relatively independent of the DSM-IV diagnostic category. They consist of a cortical-hypoarousal subtype and a maturational lag subtype, both of which are found in groups of children with either AD/HDcom or AD/HDin. A third EEG subtype, with excess beta activity, appears to occur in AD/HDcom, but not in AD/HDin. From this model, novel hypotheses can be derived regarding the different medication responses and developmental pathways found within the AD/HD population. It was hypothesised that the hypoaroused group contains those children who are most likely to respond to stimulant medications, while the maturational lag group contains those who will outgrow the disorder as adults. However, these hypotheses have not yet been tested.

Different qEEG profiles have also been independently identified by Chabot and Serfontein (1996), who found three main EEG-determined subtypes of ADD, with 38% having excess theta activity, 28% excess alpha activity, and 13% excess beta. Subtypes of children with ADD characterised by excess relative alpha and beta were also found in Chabot, Orgill, Crawford, Harris, and Serfontein (1999). These studies further support our assertion that children diagnosed with AD/HD are heterogeneous, with different underlying electrophysiological abnormalities.

Clinical Implications and Future Directions

From this body of research a number of points emerge that need consideration in clinical practice. The first is that there appears to be a number of different underlying brain dysfunctions that result in the typical behavioural profile of AD/HD. This means that it is important to conduct an initial EEG assessment prior to implementing any treatment, including neurofeedback training. To assume that increasing beta in an AD/HD child will have a therapeutic effect is fundamentally flawed, as some AD/HD children have increased beta activity to begin with. Fur-

ther increases in beta are likely to be counter-productive and possibly, or even probably, detrimental in such children. Similarly, reducing theta activity may not be appropriate, as some of these children do not have elevated theta in the first place. For optimal patient outcomes to be achieved, neurofeedback training must be differentially based on a pre-treatment qEEG.

A second question that needs further investigation is the nature and occurrence of AD/HD in girls. Electrophysiological studies have determined that there is far more variance within the male AD/HD population than the female population, with results suggesting that certain CNS abnormalities in girls do not result in behaviours that warrant referral for an AD/HD assessment. This means that there may be girls in the community with AD/HD who are never recognised. Since untreated AD/HD is a precursor for so many other problems in later life, it is of major importance that research be conducted to determine if these children actually exist, and whether they have problems that warrant treatment.

Unfortunately, the routine use of qEEG as part of an initial assessment is not widely accepted–rather, it has received considerable criticism from statutory bodies in the western world. Despite this criticism, there still remains a major problem of misdiagnosis of AD/HD, which usually translates into the over-use of medications. Part of the problem is that there are no independent tests for AD/HD. QEEGs remain one of only a few assessment procedures which have the potential to provide an independent marker of this disorder. Consistent qEEG findings have been reported for nearly 30 years, supporting the use of this procedure as a diagnostic tool *when used as part of a comprehensive clinical assessment for AD/HD*. These data need to be communicated clearly to those statutory bodies which provide ill-informed criticism of the field. In addition, while there have been some qEEG studies of AD/HD addressing issues of discriminability (Chabot & Serfontein, 1996) and specificity (e.g., Bresnahan & Barry, 2002), these are relatively rare in the literature, and there is an imperative need for workers in the field to seek to supply such data. Further, in the light of the data reviewed here, greater acceptance of the value of qEEG in AD/HD requires a wider investigation into EEG-defined subtypes of the disorder, and the development of treatment protocols which can accommodate a number of such subtypes. By undertaking this research, and further quantifying the exact nature of underlying dysfunctions in these children, it will be possible to develop accurate diagnostic tests and better treatment regimes.

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