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EEG evidence for a new conceptualisation of attention deficit hyperactivity disorder

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Abstract

Objectives: This study investigated the presence of electroencephalographic (EEG) clusters within a sample of children with the inattentive type of attention-deficit/hyperactivity disorder (ADHD).

Methods: Subjects consisted of 100 boys with ADHD and 40 age-matched controls. EEG was recorded from 21 sites during an eyes-closed resting condition and Fourier transformed to provide estimates for total power, and relative power in the delta, theta, alpha and beta bands. Factor analysis was used to group sites into 3 regions; frontal, central and posterior. These data were subjected to cluster analysis.

Results: Two distinct EEG clusters of children with the inattentive type of ADHD were found. These were characterised by (a) increased high-amplitude theta with deficiencies of delta and beta activities, and (b) increased slow wave and deficiencies of fast wave activity.

Conclusions: These two subtypes are independent of current diagnostic categories, and consist of a cortically hypoaroused group and a group typified by a maturational lag in central nervous system (CNS) development. These results support a re-conceptualisation of ADHD based on the CNS abnormality underlying the disorder rather than the behavioural profile of the child. This has the potential to add a level of predictive validity, which is currently lacking in the present diagnostic systems.

Keywords: Attention deficit/hyperactivity disorder; Children; Electroencephalogram; Subtype; Diagnosis

1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) has become one of the most commonly treated disorders of childhood (Cantwell, 1996), with the last decade seeing an approximate 4-fold increase in the number of children diagnosed with this disorder (Brownell and Yogendran, 2001). ADHD has undergone considerable change in its conceptualisation, with debate still continuing over the exact nature of the disorder. The diagnostic and statistical manual of mental disorders (DSM) definition has changed from a single disorder with an emphasis on hyperactivity (American Psychiatric Association, 1968), to a two-dimensional disorder, allowing diagnosis of 3 types (American Psychiatric Association, 1994). In contrast, the tenth version of the International Classification of Diseases (ICD-10; WHO, 1993), widely used in Europe, lists criteria for a similar disability under the title of 'hyperkinetic disorder'. This differs from the DSM-IV in recognising only one type of the disorder, due to what was considered as a lack of empirical evidence for the hyperactive/impulsive and inattentive types listed in the DSM-IV. However, recent work from our laboratory has provided evidence which argues that the ICD classification needs to recognise the inattentive type as a valid diagnostic category (Clarke et al., 2002b).

The two-dimensional DSM-IV model of ADHD has been questioned on a number of issues. For example, children with the predominantly inattentive type of ADHD (ADHDin) have been found to differ behaviourally from children with the combined type of the disorder (ADHDcom) on more than just their level of hyperactivity (Lahey et al., 1987; Lahey and Carlson, 1991), leading some researchers to suggest that ADHDin may be better categorised as a subtype of a different disorder (Lahey et al., 1985). Another limitation of the present definitional criteria is that diagnosis is based on behaviour alone, without consideration of the cause of the behaviour. It is possible that there could be a number of different underlying causes that result in the behaviours seen in ADHD, and these

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causes may have different developmental paths and/or require different treatment regimes.

Electroencephalography (EEG) research over the past 30 years has found consistent differences between children with and without ADHD. ADHD children typically have an excess of slow wave activity, primarily in the delta and theta bands, and deficiencies of alpha and beta activities (Clarke et al., 1998, 2001b, 2001d, 2002a; Lazzaro et al., 1998; Chabot and Serfontein, 1996; Janzen et al., 1995; Mann et al., 1992; Dykman et al., 1982; Satterfield et al., 1972). These results have been interpreted as indicating that children with ADHD have a central nervous system (CNS) dysfunction, which has been characterised primarily as either a maturational lag (Mann et al., 1992) or cortical underarousal (Lubar, 1991). In a new approach to the investigation of CNS dysfunction in ADHD, we examined the existence of EEG-defined subtypes within a large sample of ADHDcom children (Clarke et al., 2001c). Despite these children having the same behaviour-based diagnosis, results indicated that there were 3 distinct EEG-defined clusters. These appeared to consist of a hypoaroused group characterised by increased high-amplitude theta activity and decreased delta and beta activities, a maturational-lag group with increased slow wave and deficiencies of fast wave activity, and an over-aroused group with excess beta activity.

The present study advanced that research by investigating the existence of EEG-defined subtypes within children with the inattentive type of ADHD, to determine the level of heterogeneity within this type of ADHD and its relationship to children with other types.

2. Study 1

2.1. Method

Subject inclusion criteria, testing procedures and statistical analysis in this study are the same as our previously published cluster study (Clarke et al., 2001c) except for the type of ADHD patient included.

2.2. Subjects

Subjects in this study consisted of 100 boys with a diagnosis of ADHDin and 40 age-matched male control subjects. All children were between the ages of 8 and 13 years. Subjects had a full-scale WISC-III IQ score of 85 or higher. The children with ADHD were drawn from consecutive new patients presenting at a Sydney-based paediatric practice for an assessment for ADHD from 1993 to 1995 until the target number was obtained. The clinical subjects had not been diagnosed as having ADHD previously, had no history of medication use for the disorder, and were tested before being prescribed any medication. The control group consisted of children from local schools and community groups. Ten children initially assessed as control subjects were rejected due to learning difficulties. The protocol was approved by the University of Wollongong, Human Research Ethics Committee, and informed consent was obtained from all subjects in this study.

Inclusion in the ADHD group was based on clinical assessments by a paediatrician and a psychologist; children were included only where both agreed on the diagnosis. DSM-IV criteria were used and children were included only if they met the full diagnostic criteria for ADHD, inattentive type. A clinical interview was used which incorporated information from as many sources as were available. The interview included a description of the presenting problem and a medical history given by a parent or guardian, a physical examination, assessment for neurological 'soft signs', review of school reports for the past 12 months seeking behavioural/learning problems, reports from any other health professionals, and behavioural observations during the assessment. Children were also assessed using the WISC-III, Neale analysis of reading and the Wide Range Achievement Test Revised (WRAT-R) spelling test. Children were excluded from the clinical groups if they had a history of a problematic prenatal, perinatal or neonatal period, a disorder of consciousness, a head injury with cerebral symptoms, a history of CNS diseases, convulsions or a history of convulsive disorders, paroxysmal headaches or tics.

Inclusion in the control group was based on: an uneventful prenatal, perinatal and neonatal period; no disorders of consciousness, head injury with cerebral symptoms, history of CNS diseases, obvious somatic diseases, convulsions, history of convulsive disorders, paroxysmal headache, enuresis or encopresis after the fourth birthday, tics, stuttering, pavor nocturnes or excessive nailbiting, obvious mental diseases, conduct disorders, and no deviation with regard to mental and physical development. Control subjects had to also score in the normal range on the measures of accuracy and comprehension on the Neale Analysis of Reading, and have a standard score of 90 or above on the WRAT-R spelling test. Assessment for inclusion as a control was based on a clinical interview with a parent or guardian similar to that of the ADHD subjects, utilising the same sources of information, and the same psychometric assessment as was used for the clinical subjects.

Any children who showed signs of depression, anxiety, oppositional behaviour or syndromal disorders were excluded from this study. Children were also excluded if spike wave activity was present in the EEG.

2.3. Procedure

Both the ADHD and control subjects were tested in a single session lasting approximately 2.5 h. Subjects were first assessed by a paediatrician, where a physical examination was performed and a clinical history was taken. Subjects then had a psychometric assessment consisting of a WISC-III, Neale Analysis of Reading and WRAT-R (spel-

ling). At the end of this assessment, subjects had an electrophysiological assessment consisting of a visual–auditory oddball evoked potential and an EEG. The EEG was recorded at the end of this session in an eyes-closed resting condition, while subjects were seated on a reclining chair. An eyes-closed condition was used due to greater demonstrated test–retest reliability than eyes-open data for relative power measures (John et al., 1980). Electrode placement was in accordance with the international 10-20 system, using an electrocap produced by Electrocap International. Activity in 21 derivations was recorded from Fp1, Fp2, Fpz, F3, F4, F7, F8, Fz, C3, C4, Cz, T3, T4, T5, T6, P3, P4, Pz, O1, O2 and Oz. A single electro-oculogram (EOG) electrode referenced to Fpz was placed beside the right eye and a ground lead was placed on the left cheek. A linked ear reference was used, and reference and ground leads were 9 mm tin disk electrodes. Impedance levels were set at less than 5 kOhm.

The EEG was recorded and Fourier transformed by a Cadwell Spectrum 32, software version 4.22, using test type EEG, montage Q-EEG. The sensitivity was set at 150 μV per centimeter, low frequency filter 0.53 Hz, high frequency filter 70 Hz and 50 Hz notch filter. The sampling rate of the EEG was 200 Hz and the Fourier transformation used 2.5 s epochs.

Thirty 2.5 s epochs were selected from the live trace and stored on a floppy disk. Epoch rejection was based on both visual and computer selections. Computer reject levels were set using a template recorded at the beginning of the session and all subsequent epochs were compared to this. The EOG rejection was set at 50 μV. The technician also visually appraised every epoch and decided to accept or reject it, based on the absence or presence of artefact. These were further reduced by a second technician to 24 epochs (1 min) for Fourier analysis. The EEG was analysed in 4 frequency bands: delta (1.5–3.5 Hz), theta (3.5–7.5 Hz), alpha (7.5–12.5 Hz) and beta (12.5–25 Hz), for relative power, as well as the total power of the EEG (1.5–25 Hz).

2.4. Statistical analysis

Initially, the data from the ADHDin group were converted to Z scores based on the data from the control group. This gave comparable estimates of excesses or deficiencies of power for each frequency band at each site, for each ADHD child, compared to normal children.

Principal component analysis with varimax rotation was then performed on the z-transformed power estimates at the 21 electrode sites in each frequency band, in order to explore ways of reducing the number of variables by grouping sites into regions. Each EEG measure was then averaged across sites in each region for further analysis. In the next stage, subjects were grouped with Ward's method of cluster analysis, using the squared Euclidian distance as the measure of dissimilarity. The variables used in the cluster analysis were regional averages for total power and power in each of the 4 frequency bands, as well as the child's age (included in the analysis to control for maturational effects). Discriminant function analysis was performed on the subject clusters identified in the cluster analysis, to determine the level of correct classification of subjects, based on the EEG data.

Analyses of variance (ANOVA) were performed examining the effects of region and group for each band in the total power and relative delta, theta, alpha and beta. The effects of region were examined in a 3-level repeated-measures factor. Planned contrasts compared the frontal region with the posterior region, and their mean with the central region. These orthogonal planned contrasts allow optimal clarification of site effects within the regions studied. In separate group analyses, control subjects were compared with the total ADHDin group, as well as with the two clusters. A Bonferroni procedure (Keppel, 1982) was employed to correct the multiple group comparisons to maintain the familywise error rate for each set of analyses at $\alpha = 0.05$. Only between-group effects and interactions are reported here for space reasons.

2.5. Results

Principal component analysis identified two similar factors within each relative power frequency band (see Table 1). The first factor primarily loaded on the frontal electrode sites and the second factor loaded on the posterior sites. However, there was a relatively high but inconsistent loading of the central sites and T3 and T4 on both factors. Hence T3, T4 and the central sites were clustered into a third regional grouping, as in our previous cluster study (Clarke et al., 2001c). The factor analysis thus suggested the grouping of scalp sites into 3 sagittal regions, frontal (Fp1, Fp2, Fpz, F3, F4, F7, F8, Fz), central (T3, T4, C3, C4, Cz), and posterior (T5, T6, P3, P4, Pz, O1, O2, Oz) for the relative power bands. In total power, two factors were also identified. However, the factors divided the electrode sites into an occipital region (O1, O2, Oz), and a second factor including the remaining electrode sites. As factor analysis was performed primarily for data reduction reasons, total power was also clustered into 3 regions, a frontal region consisting of the same sites as in relative power, a central region consisting of central, parietal and temporal electrodes, and a posterior region consisting of the occipital electrodes. EEG measures were averaged across electrodes within each of these regions for further analysis.

A summary of results are presented in Table 2. A comparison of the ADHDin group with the control group indicated that the ADHDin group had increased relative theta ($F(1, 138) = 34.05$, $P < 0.001$) and decreased relative alpha ($F(1, 138) = 12.21$, $P < 0.001$) and relative beta ($F(1, 138) = 7.00$, $P < 0.01$) across the entire scalp (see Fig. 1). In total power, the ADHDin group had more frontal and less posterior power than the control group ($F(1, 138) = 9.96$, $P < 0.01$), and less frontal and more

Table 1
Factor loadings are shown at each electrode site[a]

Site	Total Power		Relative Delta		Relative Theta		Relative Alpha		Relative Beta	
	Factor 1	Factor 2	Factor 1	Factor 2	Factor 1	Factor 2	Factor 1	Factor 2	Factor 1	Factor 2
Fp1	**0.890**	0.207	**0.802**	0.134	**0.909**	0.159	**0.860**	0.340	**0.721**	0.193
Fpz	**0.918**	0.276	**0.909**	0.276	**0.946**	0.179	**0.913**	0.327	**0.903**	0.321
Fp2	**0.909**	0.217	**0.781**	0.123	**0.902**	0.192	**0.861**	0.365	**0.844**	0.253
F7	**0.907**	0.263	**0.841**	0.240	**0.857**	0.323	**0.864**	0.365	**0.821**	0.328
F3	**0.930**	0.288	**0.921**	0.278	**0.926**	0.294	**0.921**	0.306	**0.885**	0.321
Fz	**0.901**	0.288	**0.902**	0.309	**0.921**	0.264	**0.917**	0.320	**0.885**	0.313
F4	**0.914**	0.287	**0.884**	0.346	**0.907**	0.321	**0.903**	0.359	**0.893**	0.328
F8	**0.903**	0.248	**0.861**	0.251	**0.863**	0.314	**0.862**	0.341	**0.824**	0.307
T3	*0.852*	0.326	*0.793*	0.261	*0.622*	0.496	*0.745*	0.393	0.330	*0.434*
C3	*0.854*	0.439	*0.792*	0.463	*0.641*	0.616	*0.713*	0.560	*0.779*	0.518
Cz	*0.771*	0.418	*0.741*	0.437	*0.651*	0.569	*0.732*	0.492	*0.786*	0.428
C4	*0.838*	0.434	*0.690*	0.548	0.622	*0.652*	*0.649*	0.598	*0.747*	0.525
T4	*0.566*	0.178	*0.710*	0.443	*0.602*	0.444	*0.817*	0.406	*0.612*	0.257
T5	*0.732*	0.450	0.500	**0.721**	0.372	**0.805**	0.514	**0.716**	0.444	**0.812**
P3	*0.801*	0.452	0.502	**0.784**	0.339	**0.874**	0.480	**0.797**	0.502	**0.802**
Pz	*0.695*	0.508	0.519	**0.759**	0.362	**0.840**	0.440	**0.775**	0.525	**0.751**
P4	*0.703*	0.517	0.407	**0.833**	0.298	**0.905**	0.400	**0.838**	0.435	**0.817**
T6	*0.596*	0.583	0.361	**0.791**	0.248	**0.850**	0.439	**0.740**	0.375	**0.831**
O1	0.307	**0.883**	0.141	**0.912**	0.165	**0.896**	0.297	**0.886**	0.258	**0.905**
Oz	0.215	**0.924**	0.143	**0.907**	0.168	**0.848**	0.234	**0.874**	0.194	**0.936**
O2	0.219	**0.930**	0.104	**0.925**	0.184	**0.874**	0.235	**0.888**	0.172	**0.889**

[a] Loadings given in bold are consistent across measures with the top and bottom sections, respectively, representing a frontal grouping (Factor 1) and a posterior grouping (Factor 2) in each measure. In the middle section, loadings in italics are the largest for that measure, indicating the variability in segregation of the central sites in the two factors.

posterior delta than controls ($F(1,138) = 6.26$, $P < 0.05$). However, cluster analysis indicated the presence of two distinct EEG-defined subtypes within the total ADHDin sample, with discriminant function analysis indicating 96% correct classification of these children using a two cluster model.

Cluster 1 accounted for 68% of the total sample. This cluster was characterised by increased relative theta compared with controls ($F(1,106) = 19.30$, $P < 0.05$), with a reciprocal decrease in relative beta ($F(1,106) = 6.10$, $P < 0.001$) across the scalp. Theta activity was greater in the frontal region than the posterior region in cluster 1 compared to control subjects ($F(1,106) = 10.47$, $P < 0.01$). Alpha activity was at normal levels. Cluster 2 contained 32% of the sample. This group had increased frontal and decreased posterior total power ($F(1,70) = 28.81$, $P < 0.001$), compared to control subjects. Delta activity was increased across the scalp ($F(1,70) = 12.34$,

Table 2
Mean *Z*-Scores for each cluster and the total ADHD sample

Region	Cluster 1 ($N = 68$)	Cluster 2 ($N = 32$)	Total ADHD Group ($N = 100$)
Total power frontal	0.529	0.707	0.586
Total power central	0.223	−0.068	0.129
Total power posterior	0.009	−0.670	−0.208
Delta frontal	−0.324	0.463	−0.072
Delta central	−0.226	0.673	0.061
Delta posterior	−0.081	1.155	0.314
Theta frontal	1.249	2.275	1.577
Theta central	0.687	2.074	1.131
Theta posterior	0.508	2.321	1.088
Alpha frontal	−0.196	−1.260	−0.536
Alpha central	−0.052	−1.257	−0.437
Alpha posterior	−0.150	−1.754	−0.663
Beta frontal	−0.445	−0.615	−0.499
Beta central	−0.359	−0.682	−0.462
Beta posterior	−0.357	−0.048	−0.258

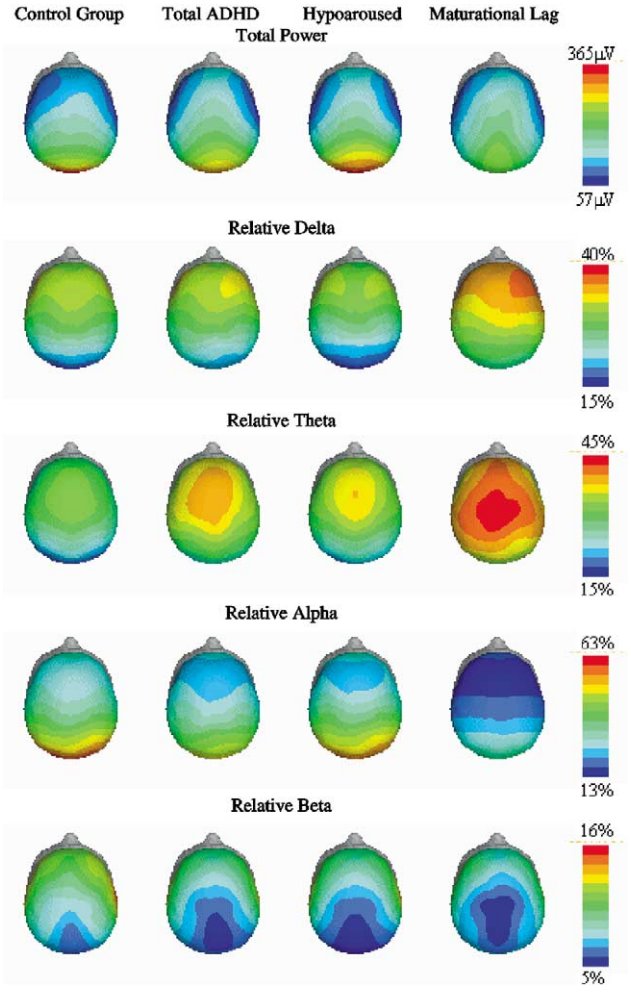

Fig. 1. Topographic group differences in total and relative power.

$P < 0.001$), with the increase being greater in the posterior region than the frontal region ($F(1, 70) = 15.93, P < 0.001$). Theta activity was increased ($F(1, 70) = 81.97, P < 0.001$) and alpha activity decreased ($F(1, 70) = 70.12, P < 0.001$) across the scalp. The deficiency in relative alpha was greater in the posterior region than the frontal region ($F(1, 70) = 11.48, P < 0.001$), and the central region had more power than the mean of the frontal and posterior regions ($F(1, 70) = 7.39, P < 0.01$). In the beta band, the difference between the frontal and posterior regions was greater in cluster 2 than the control group ($F(1, 70) = 8.00, P < 0.01$), and the maximum activity in the central region compared with frontal/posterior regions was reduced in cluster 2 compared with controls ($F(1, 70) = 9.00, P < 0.01$).

2.6. Discussion

EEG studies of the inattentive type of ADHD have found that these children have increased slow wave activity, primarily in the delta and theta bands, and deficiencies of alpha and beta activities (Clarke et al., 1998, 2001b, 2001d, 2002a; Lazzaro et al., 1998; Chabot and Serfontein, 1996; Janzen et al., 1995; Mann et al., 1992; Dykman et al., 1982; Satterfield et al., 1972). In the present study, the comparison of the total sample of ADHDin children and the control group found that the ADHDin group had increased theta activity, and deficiencies of alpha and beta, which is consistent with previous studies.

As with our previous cluster study in ADHDcom children (Clarke et al., 2001c), cluster analysis indicated the presence of distinct groups of ADHDin children which are not identified by mean group data based on the DSM-IV behavioural criteria. The first cluster consisted of children with increased relative theta, with a reciprocal decrease in relative beta across all regions. Beta activity increases during both physical and mental activities (Andreassi, 1995; Ackerman et al., 1994, 1995), and a number of studies have found that children with ADHD have lower levels of beta activity during cognitive tasks (Mann et al., 1992; Lubar, 1991). This decrease in beta activity has been interpreted as cortical hypoarousal (Lubar, 1991), which is supported by studies that have assessed ADHD children using skin conductance (Satterfield and Dawson, 1971), as well as regional cerebral blood flow and positron emission tomography (Lou et al., 1984, 1989; Zametkin et al., 1990). These results indicate that the primary deficit in this group is probably associated with cortical hypoarousal (Lubar, 1991; Clarke et al., 2001c; Satterfield et al., 1972).

The second cluster had increased frontal and decreased posterior total power, increased relative delta and relative theta, with decreased relative alpha across the scalp, and a decrease in fronto-central relative beta activity, with the maximal decrease being evident in the central region. With normal maturation, EEG frequencies increase as a function of age, with slow wave activity apparently being replaced by faster waveforms (Matousek and Petersen, 1973; Matthis et al., 1980, Clarke et al., 2001a). Benninger et al. (1984) found that theta activity decreased as alpha increased and that the speed of change in occipital areas was almost twice that of central areas. Topographic studies of maturation have shown that changes take place from posterior to anterior regions, in the delta, theta and alpha bands (Gasser et al., 1988). Beta waves developed earliest in the central region followed by parietal, occipital and then frontal regions. The increased delta and theta, with maximal differences in the posterior region (see Table 2), the reduced alpha, again maximal in the posterior region, and the central maximum for relative beta, all suggest a maturational lag in cluster 2.

In our previous study of EEG clusters in ADHDcom children (Clarke et al., 2001c), 3 distinct clusters were found. The first group was typified by cortical hypoarousal, and the second by a maturational lag. The third was a small group with excess beta activity, tentatively associated with cortical hyperarousal. We previously failed to find evidence for this third EEG profile in ADHDin children (Clarke et al., 2001e), and again failed in this study. In the context of our previous cluster study (Clarke et al., 2001c), the present

results indicate that the hypoaroused and maturationally lagged subtypes are found in both the combined and inattentive types of ADHD. This then raises the question, if similar CNS dysfunctions occur in both subtypes of ADHD, what causes the behavioural differences found in these two groups?

3. Study 2

The second phase of this investigation aimed to examine the presence of EEG differences between the hypoaroused and maturationally lagged clusters of children within the DSM-IV types of ADHD.

3.1. Method

3.1.1. Subjects

Subjects consisted of the 147 ADHDcom subjects from our previous cluster study (Clarke et al., 2001c), and the 100 ADHDin subjects from Study 1. Of the ADHDcom subjects, 78 were clustered as hypoaroused and 69 as maturationally lagged.

3.1.2. Statistical analysis

In separate ANOVAs, the hypoaroused ADHDcom group was compared with the hypoaroused ADHDin group, and the maturational lag ADHDcom group was compared to the maturational lag ADHDin group, to determine the relationship between the clusters of children with different DSM-IV diagnosis. The same planned contrasts for region were used as in Study 1.

3.2. Results

As shown in Fig. 2, the hypoaroused ADHDcom children had greater total power ($F(1,144) = 13.42$, $P < 0.001$), more relative theta ($F(1,144) = 7.34$, $P < 0.01$), less relative delta ($F(1,144) = 15.91$, $P < 0.001$) and beta ($F(1,144) = 6.82$, $P < 0.01$) across the entire scalp, than children in the hypoaroused ADHDin group. In total power ($F(1,144) = 5.92$, $P < .05$) and relative beta ($F(1,144) = 3.99$, $P < 0.05$), these group differences were greater in the posterior region than the frontal region.

In the comparison of the two maturationally lagged groups, no significant differences were found (see Fig. 3).

3.3. Discussion

In the comparison of the two hypoaroused groups, the ADHDcom children had EEG profiles that suggested that they were more hypoaroused than those with ADHDin. ADHDcom theta levels were increased, and beta activity decreased, with alpha activity being similar in both the groups. These results are consistent with the continuum model of ADHD (Levy et al., 1997), which suggests that the behaviours found in ADHD children represent the extreme end of a continuum that ranges from normal behaviour to behavioural disturbance, rather than there being a set point where normal ends and ADHD starts. The present results suggest that the more cortically hypoaroused a child is, the greater will be their level of behavioural disturbance.

In contrast to these results, no significant differences were found between the maturationally lagged subtypes in the ADHDcom and ADHDin groups, although differences in EEG topography are evident. Previously we have found subtle EEG differences between combined and inattentive types (Clarke et al., 2001b,d) which the present analysis would not be expected to identify. These differences need further investigation in order to explain the behavioural differences found between the two groups.

The present research indicates that, based on underlying CNS dysfunction, there are 3 distinct subtypes of children within the ADHD diagnosis, which are largely independent of the present behaviourally based diagnostic system. They consist of a cortical hypoarousal subtype and a maturational-lag subtype, both of which are found in groups of children with either the DSM-IV inattentive or combined type diagnoses. A third subtype, with excess beta activity, appears to occur in the combined type of ADHD, but not with inattention alone (Clarke et al., 2001e).

A number of researchers have proposed that ADHDin children have social and behavioural problems sufficiently distinct from those found in children with the hyperactive/impulsive type to warrant the removal of this diagnosis from the ADHD category (Lahey et al., 1987, 1985; Lahey and Carlson, 1991). This is not supported by the present results, as the inattentive type did not have CNS dysfunctions that were qualitatively distinct from those found in children with the combined type. This suggests that the inattentive type should be retained within the ADHD disorder. These results offer further support for the inclusion of an inattentive type of the hyperkinetic disorder within the ICD classification system, as the inattentive children were found to have abnormal EEG profiles, indicative of cortical dysfunction.

The present diagnostic systems, based on checklists of abnormal behaviours, have the limitation that a number of different factors may cause the behaviours seen in ADHD. While the present diagnostic types provide useful descriptions of behaviour, their predictive validity for treatment response, or the developmental time course of the disorder, is limited. With the addition of information relating to underlying CNS dysfunction, predictive validity can be increased. A child with cortical hypoarousal, irrespective of whether they have the combined or inattentive type of ADHD, may be more likely to respond to stimulant medications than a maturationally lagged child. In a previous study we found that stimulants increased beta and decreased theta activity in children with the combined type of ADHD (Clarke et al., unpublished data). This indicated that the stimulants were increasing arousal in children who were hypoaroused. However, complete EEG normalisation within the sample was not found. This may have resulted from a lack of change occurring in children who were

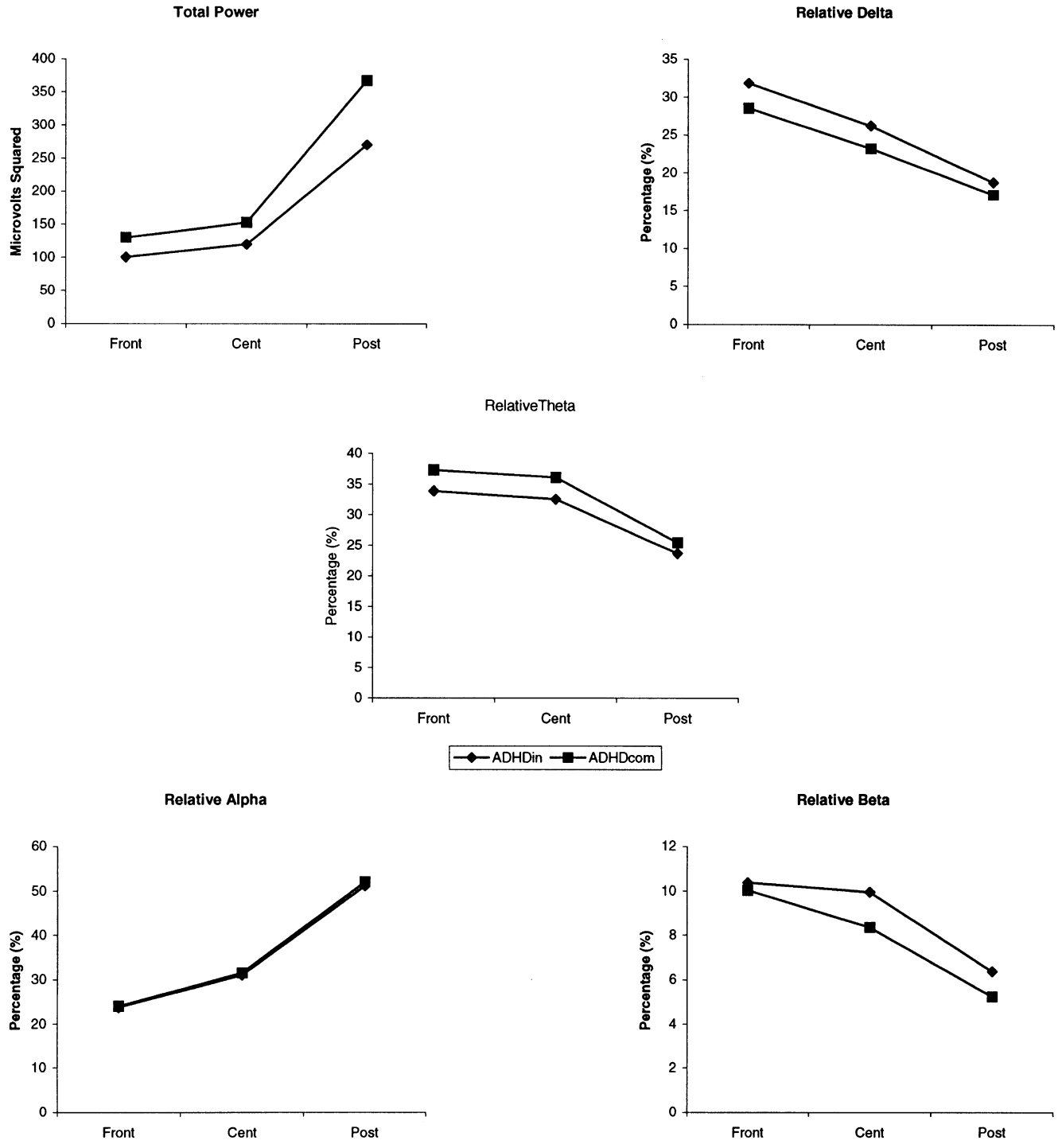

Fig. 2. Power distribution for the hypoaroused clusters, from frontal to posterior regions, for total power (top left), relative delta (top right), relative theta (centre), relative alpha (bottom left), relative beta (bottom right).

maturationally lagged, as these children would not be expected to respond well to stimulant medications, since their primary deficit is not reduced arousal requiring elevating to normal levels.

The behavioural profile of people with ADHD also undergoes substantial change with age. Hyperactivity is predominantly seen in younger children, with the degree of hyperactivity decreasing in early adolescence, and usually disappearing by late adolescence. Estimates indicate that between 30 and 70% of children with ADHD continue to suffer ADHD as adults, although this will almost exclusively be of the inattentive type (Bellak and Black, 1992). At present, there is no predictor of which children will continue to have ADHD as an adult. We consider that chil-

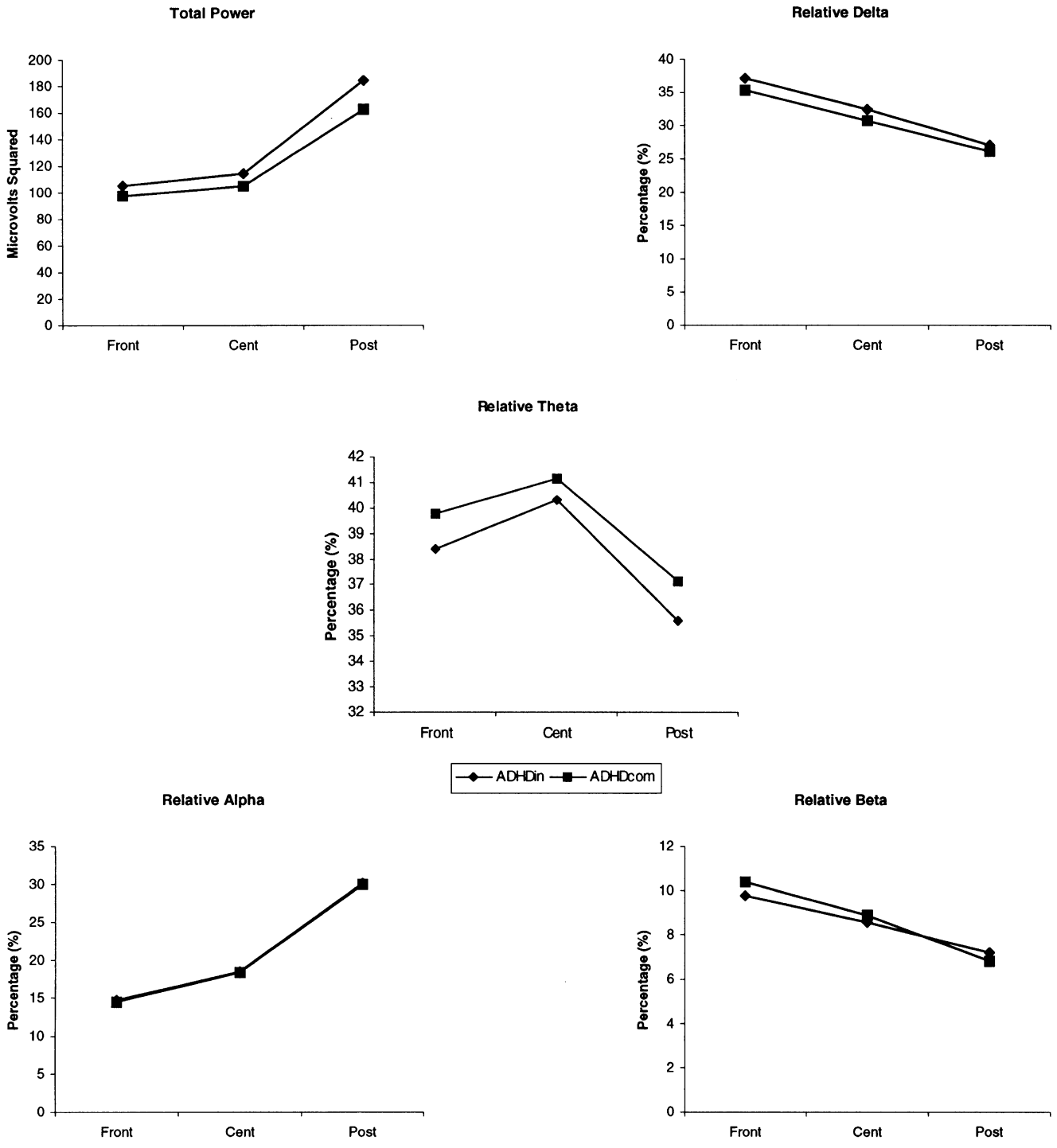

Fig. 3. Power distribution for the maturationally lagged clusters, from frontal to posterior regions, for total power (top left), relative delta (top right), relative theta (centre), relative alpha (bottom left), relative beta (bottom right).

dren with the maturational-lag profile are more likely to outgrow the disorder as their CNS attains normal adult levels, albeit at a later time than children without ADHD, than are children in the hypoaroused group. Complete normalisation is unlikely in the hypoaroused group, as the nature of their deficit indicates a more permanent CNS dysfunction. These hypotheses need testing.

From these results, we suggest that a new emphasis should be placed on the underlying CNS abnormality that results in ADHD, rather than typing based on behaviour alone. While behaviour is of paramount importance in the initial diagnosis of ADHD, it has little predictive value. The addition of causal factors to the diagnostic criteria has the potential to substantially improve treatment outcomes. If

these EEG-defined subtypes of ADHD children are able to predict treatment response and the developmental course of the disorder, their inclusion in revised diagnostic criteria would be indicated.

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