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Excess beta activity in children with attention-deficit/hyperactivity disorder: an atypical electrophysiological group

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Abstract

Studies of children with attention deficit/hyperactivity disorder (ADHD) have typically found elevated levels of slow wave activity in their EEGs, but in two of our previous studies, a small subset of ADHD children with excess beta activity in the EEG was identified. The aim of this study was to determine whether children with excess beta activity represent a distinct electrophysiological subtype of ADHD, to quantify the differences in their EEGs, and to determine if this group of children with ADHD have behavioural profiles different from other children with ADHD. Results indicated that children with excess beta represent a small independent subset of children diagnosed with ADHD, which primarily consists of children with a diagnosis of ADHD combined type. Behaviourally, this group was similar to other children with ADHD, although the excess-beta group were more prone to temper tantrums and to be moody. The excess in beta activity was found primarily in the frontal regions and may be associated with frontal lobe self-regulation and inhibition control.

Keywords: Attention deficit/hyperactivity disorder; Children; EEG; Maturation

1. Introduction

Children with attention deficit/hyperactivity disorder (ADHD) typically have increased electroencephalographic (EEG) slow wave activity, primarily in the theta band, compared to normal children (Capute et al., 1968; Wikler et al., 1970; Satterfield et al., 1973b). Increased relative delta activity in posterior regions is also common in children with ADHD (Matousek et al., 1984). Mann et al. (1992) found children with ADHD had an increase in absolute amplitude in the theta band, which primarily occurred in frontal regions during a resting condition. During cognitive tasks, ADHD children showed a greater increase in theta activity in frontal and central regions, and a decrease in beta activity in posterior and temporal regions. Based on studies of

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developmental changes in the EEGs of normal children, these results have been interpreted as ADHD representing either a maturational lag (Mann et al., 1992) or a developmental deviation (Chabot and Serfontein, 1996) of the central nervous system.

As a child becomes older, developmental changes occur in the EEG (Wada et al., 1996; Clarke et al., 2001a), with a reduction in slow wave activity and an increase in faster waveforms (Matthis and Scheffner, 1980). Delta, theta and slow alpha decrease with age and faster waveforms increase, with a strong complementary decrease in theta and increase in alpha being identified (Gasser et al., 1988). These changes in the frequency of the EEG appear to be linear in nature and can be predicted with a high degree of accuracy for relative power, based on age (John et al., 1980). However, the differences within an age group appear to be greater than the differences between age bands (Benninger et al., 1984). It is from these results that a maturational lag model of ADHD has been proposed.

In two of our previous ADHD studies (Clarke et al., 1998, 2001b) a subset of ADHD children with excessive levels of beta activity was identified. In both of the studies, the subset constituted approximately 20% of children with ADHD Combined Type (ADHDcom), using DSM-IV criteria (American Psychiatric Association, 1994). One subject with a diagnosis of ADHD Predominantly Inattentive Type (ADHDin) was also identified in our second study (Clarke et al., 2001b). Chabot and Serfontein (1996) are the only other investigators to have documented the existence of this subset of children with ADHD. In their study, approximately 13% of ADHD children were found to have excess beta activity.

Beta activity rarely occurs in children and adolescents and should not exceed 25 μV in amplitude (Fisch, 1994). In normal awake children, Kellway (1990) found beta activity was 20 μV or less in 98% of subjects and less than 10 μV in 70%. Increased levels of beta activity have typically been associated with drug usage (Kozelka and Pedley, 1990) and beta can be increased by the use of benzodiazepines (Pichlmayr and Lips, 1980; Glaze, 1990). Studies of beta activity have found excessive levels in patients with psychiatric illnesses (Gibbs and Gibbs, 1950). Increased beta activity has been found in schizophrenia (Morihisa et al., 1983) and major depressive disorders (Pollock and Schneider, 1990) and is also associated with anxiety (Kiloh et al., 1981).

The aim of this study was to ascertain the percentage of children diagnosed as having ADHD who have excess levels of beta activity in their EEG and to determine how their EEGs differ from those of other children with ADHD. A second aim was to determine if ADHD children with excess beta activity have behavioural profiles different from those of other children with ADHD.

2. Method

2.1. Subjects

Two hundred and ninety-eight children diagnosed with ADHD and 80 control subjects participated in this study. Two hundred and eight subjects were diagnosed as ADHDcom and 90 were diagnosed as having ADHDin. All children were between the ages of 8 and 12 years and right-handed and -footed. Subjects had a full-scale WISC-III IQ score of 85 or higher. The clinical group of children was drawn from new patients referred to a Sydney-based pediatric practice for an assessment for ADHD. The ADHD 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.

Inclusion in the ADHD groups was based on a clinical assessment by a pediatrician and a psychologist; children were included only where both agreed on the diagnosis. DSM-IV criteria were used and children were included in either the ADHDcom or ADHDin groups if they met the full diagnostic criteria for the subtype. A structured clinical interview was used in the assessment of all subjects. This 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 information on behavioural/learning problems, reports from any other health professionals, and behavioural observations during the assessment. Handedness was assessed by ascertainment of the hand used for writing, catching and throwing a ball and holding a bat, and the foot used to kick a ball. Children were excluded from the ADHD 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 central nervous system diseases, convulsions or a history of convulsive disorders, paroxysmal headaches or tics.

Inclusion in the control group required: an uneventful prenatal, perinatal and neonatal period; no disorders of consciousness, head injury with cerebral symptoms, history of central nervous system 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; and no deviation with regard to mental and physical development. The CPRS-48 Connors rating scale was used, with subjects who scored above a T-score of 65 on any of the six measures being excluded. Assessment for inclusion as a control was based on a structured clinical interview, with a parent or guardian, similar to that of the ADHD subjects and utilising the same sources of information.

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

2.2. Procedure

All subjects were tested in a single session lasting approximately 2.5 h. Subjects were first assessed by a pediatrician; a physical examination was performed and a clinical history taken. Subjects then had a psychometric assessment consisting of a WISC-III, Neale Analysis of Reading and Wide Range Achievement Test-R, spelling. At the end of this assessment, subjects had an electrophysiological assessment consisting of evoked potentials and an EEG. The EEG was recorded at the end of this session with an eyes-closed resting condition in which subjects were seated on a reclining chair. Electrode placement was in accordance with the international 10–20 system, using an electrocap produced by Electrocap International. The activity in 21 derivations was divided into nine regions by averaging in each region. These regions were the left frontal (Fp1, F3, F7), midline frontal (Fpz, Fz), right frontal (Fp2, F4, F8), left central (T3, C3), midline central (Cz), right central (T4, C4), left posterior (T5, P3, O1), midline posterior (Pz, Oz) and right posterior (T6, P4, O2). 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. Linked ear references were used with all EEG, and reference and ground leads were 9-mm tin disk electrodes. Impedance levels were set at less than 5 kΩ.

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/cm, 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 to floppy disk. Epoch rejection was based on both visual and computer selection. 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. Epochs were further reduced to 24 epochs (1 min) for Fourier analysis by a second technician. The EEG was analyzed in four 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 both absolute and relative

power, as well as the total power of the EEG (1.5–25 Hz).

2.3. Statistical analysis

The analysis of this data set was conducted in three stages. In stage 1, the control subjects were divided into two groups based on sex. Means and standard deviations in the relative beta band were calculated across sites for both groups of control subjects. The EEGs of the ADHD subjects were then compared to the means of the same-sex control group. In ADHD subject was deemed to have an excess of relative beta if the percentage of beta power was greater than two standard deviations above the control mean.

In stage 2, ADHD subjects with excess beta were matched on age, sex and diagnosis with a comparison group of subjects with ADHD, without excess beta, and an age- and sex-matched control group. An analysis of variance was performed examining the effects of region and group for each band in absolute and relative power. The effects of region were examined in two orthogonal three-level repeated-measures factors. The first of these was a sagittal factor, within which planned contrasts compared the frontal regions with the posterior regions, and their mean with the central regions. The second factor was laterality, within which planned contrasts compared activity in the left hemisphere with that in the right hemisphere, and their mean with the midline regions. These planned contrasts and their interactions allow optimal clarification of site effects within the regions studied. Within the group factor, planned contrasts compared the ADHD subgroup without excess beta activity with the control group (to establish ADHD differences from normal and to provide a 'typical' ADHD comparison group), and the excess beta group with both the typical ADHD and control groups. These single degree of freedom F tests obviate problems arising from asymmetry of the variance-covariance matrix often found with repeated-measures analyses of physiological data, and hence do not require Greenhouse-Geiser type adjustments. As all these contrasts are planned, and there are no more of them than the degrees of freedom for effect, no Bonferroni-type adjustment to α is required (Tabachnick and Fidell, 1989). Only between-group effects and interactions are reported here for space reasons.

In stage 3, only subjects diagnosed with the ADHD combined subtype were included due to numbers being too small to allow accurate analysis of the ADHD inattentive subtype. The behavioural profile obtained in the diagnostic interview was compared between the typical ADHD group and the excess beta group. This included behaviours listed in DSM-IV for the diagnosis of the ADHD combined type as well as any other problematic behaviours that were reported in the initial assessment.

3. Results

3.1. Age and IQ

No significant age differences were found between groups (see Table 1). However, the control group had a significantly higher mean IQ than the typical ADHD group ($t_{1,254} = 17.3,\ P < 0.001$).

3.2. Prevalence of ADHD with excess beta

The percentages of ADHD subjects with excess beta are presented in Table 2. From the total sample of 298 subjects, 34 had excess beta. Excess beta was found in approximately 15% of ADHD-com subjects and 2% of subjects with a diagnosis of ADHDin, with this difference in prevalence being significant ($\chi^2_{(1)} = 10.76,\ P < 0.001$). Excess

Table 1
Mean (S.D.) ages and IQ scores for the excess beta, typical ADHD and control groups

	Control group	Typical ADHD group	Excess beta group
Age (months)	125.3 (17.4)	123.5 (19.8)	123.6 (17.9)
Full scale IQ (WISC-III)	107.5 (12.3)	98.7 (11.2)	102.9 (14.7)

Table 2
Percentage of excess beta subjects by sex and diagnostic category

Group	Total sample		Male subjects		Female subjects	
	ADHDcom ($N = 208$)	ADHDin ($N = 90$)	ADHDcom ($N = 159$)	ADHDin ($N = 51$)	ADHDcom ($N = 49$)	ADHDin ($N = 39$)
ADHD	84.6%	97.8%	81.2%	96.1%	96.0%	100%
Excess beta	15.4%	2.2%	18.8%	3.9%	4.0%	0.0%

beta was more common among male ADHDcom subjects than female ADHDcom subjects ($\chi^2_{(1)} = 6.29$, $P < 0.05$); no female ADHDin subjects were found with excess beta.

A review of the EEGs found that the excess beta activity was primarily located in frontal and central electrode sites. Beta activity within the group varied from excesses of normal amplitude beta (Fig. 1, panel A), to high amplitude beta (Fig. 1, panel B). Beta spindles were apparent in 10% of the beta group (Fig. 1, panel C). The mean frequency ranged from 16.7 to 19.3 Hz in frontal sites and 15.8 to 17.7 Hz in the posterior sites. The beta group had a mean amplitude of 5.8 μV in the frontal regions and 4.7 μV in the posterior regions. This compared with 2.9 μV (frontal) and 3.5 μV (posterior), in the typical ADHD group, and 3.1 μV (frontal) and 4.0 μV (posterior) in the control group.

3.3. Typical ADHD vs. control group

A summary of significant group differences is shown in Table 3. In absolute delta (Fig. 2, top right), the typical ADHD group had more power than the control group ($F_{1,99} = 7.35$, $P < 0.01$). Laterally, the midline had greater power than the two hemispheres, and this difference was greater in the typical ADHD group than the control group ($F_{1,99} = 5.55$, $P < 0.05$), with maximal power occurring at the midline (Fig. 3, top right). In relative delta (Fig. 4, top left), the difference between the typical ADHD group and the control group was greater in the frontal regions than the posterior regions ($F_{1,99} = 6.31$, $P < 0.05$).

In absolute theta (Fig. 2, centre), the typical ADHD group had more power than the control group ($F_{1,99} = 15.88$, $P < 0.001$) and this difference was greater in the posterior regions than the frontal regions ($F_{1,99} = 5.45$, $P < 0.05$). Laterally (Fig. 3, centre), the difference between the midline and the two hemispheres was greater in the typical ADHD group than the control group ($F_{1,99} = 16.7$, $P < 0.001$). The typical ADHD group had more relative theta (Figs. 4 and 5, top right) than the control group ($F_{1,99} = 86.11$, $P < 0.001$). The

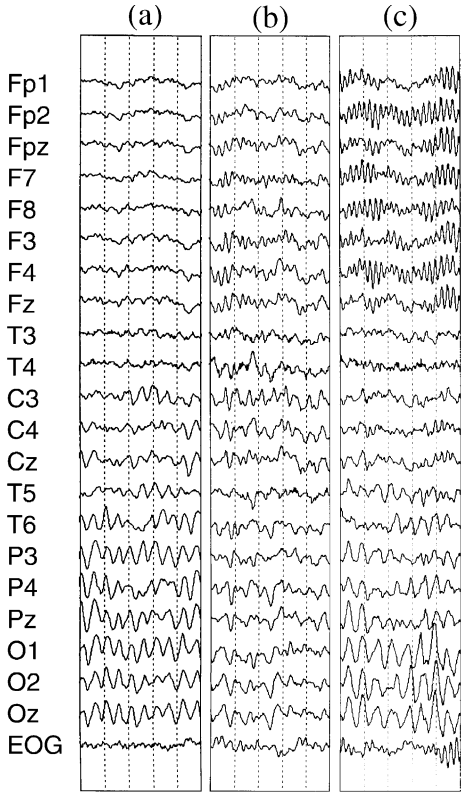

Fig. 1. Representative 1-s epochs from: (a) normal amplitude excess beta subjects; (b) high amplitude excess beta subjects; (c) excess beta subjects with frontal beta spindles.

difference between the midline and the two hemispheres was greater in frontal regions than posterior regions in the typical ADHD group compared with the control group ($F_{1,99} = 8.38$, $P < 0.01$).

The typical ADHD group had less relative alpha (Fig. 4, bottom left) than the control group ($F_{1,99} = 31.25$, $P < 0.001$). This difference was greater in the posterior regions, compared with the frontal regions ($F_{1,99} = 12.88$, $P < 0.001$) and less at the central regions than the mean of the frontal/posterior regions ($F_{1,99} = 5.46$, $P < 0.05$). In relative beta (Fig. 4, bottom right), the typical ADHD group had less power than the control group ($F_{1,99} = 12.49$, $P < 0.001$). No significant differences were found in total power, absolute alpha or absolute beta (Fig. 2 and Fig. 3, top left, bottom left and bottom right).

3.4. Excess beta vs. typical ADHD group

The excess beta group had less relative delta (Figs. 4 and 5, top left) than the typical ADHD group ($F_{1,99} = 8.42$, $P < 0.01$), and this difference was greater in the frontal regions in comparison with the posterior regions ($F_{1,99} = 8.87$, $P < 0.05$). Laterally, the difference between the midline and the two hemispheres (Fig. 5, top left) was less in the beta group than the typical ADHD group ($F_{1,99} = 5.12$, $P < 0.05$), and this difference was greater in the frontal regions than the posterior regions ($F_{1,99} = 8.15$, $P < 0.01$).

Table 3
Summary of significant comparisons between groups

	Absolute power					Relative power			
Comparison	Total	Delta	Theta	Alpha	Beta	Delta	Theta	Alpha	Beta
Typical ADHD vs. control									
Main effect		**	**				***	***	***
Main effect × F vs. P			*			*		***	
Main effect × F/P vs. C								*	
Main effect × L/R vs. M		*	**						
Main effect × F vs. P × L/R vs. M							**		
Excess beta vs. typical ADHD									
Main effect			***		***	**	**	**	***
Main effect × F vs. P			*		***	*		***	***
Main effect × F/P vs. C					***				
Main effect × L vs. R					***		*		*
Main Effect × L/R vs. M			**			*			***
Main effect × F vs. P × L vs. R					**				
Main effect × F vs. P × L/R vs. M						**			**
Excess beta vs. control									
Main effect					***	*		**	***
Main effect × F vs. P					***	***	*		***
Main effect × F/P vs. C					***				**
Main effect × L vs. R				*	***		*		
Main effect × L/R vs. M						*			**
Main effect × F vs. P × L vs. R				**	*				

Note. $^{*}P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$; F, frontal; P, posterior; C, central; L, left hemisphere; R, right hemisphere; M, midline; F/P, mean of the combined frontal and posterior regions; L/R, mean of the combined left and right hemisphere regions.

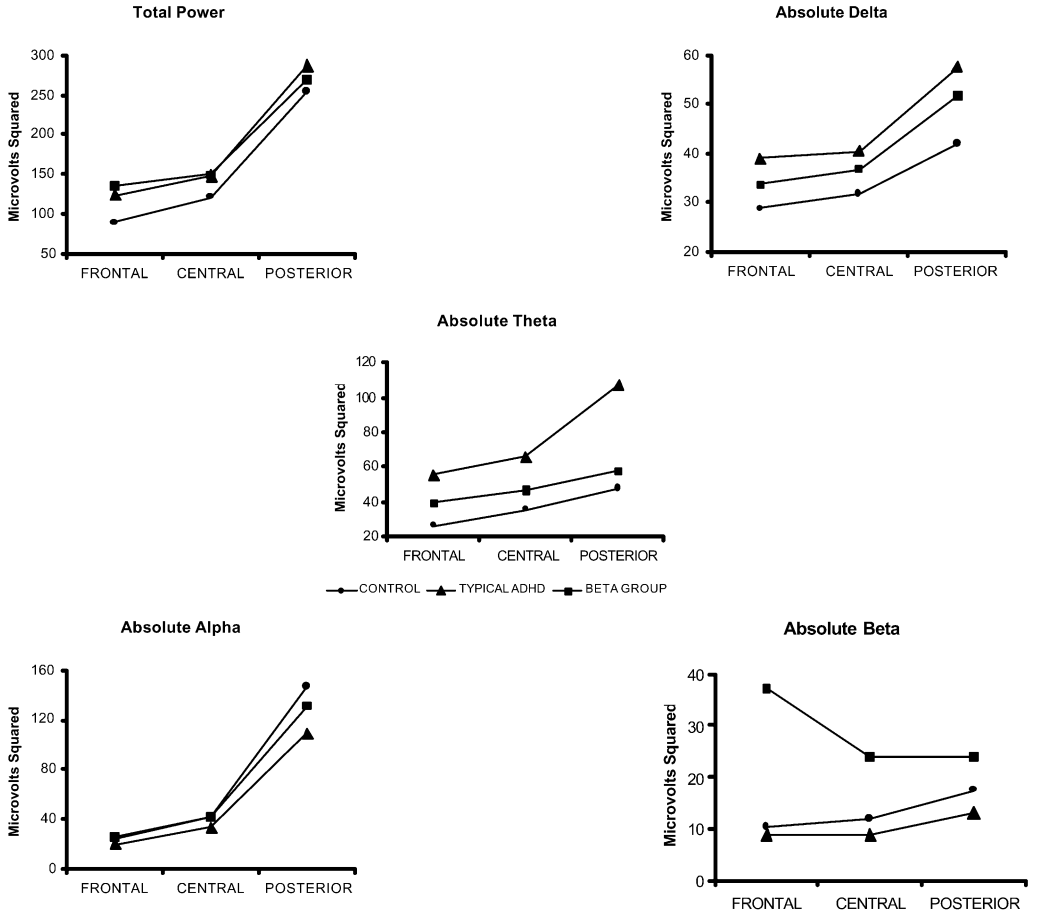

Fig. 2. Absolute power from frontal to posterior regions: Total Power (top left), Absolute Delta (top right), Absolute Theta (centre), Absolute Alpha (bottom left), Absolute Beta (bottom right).

For absolute theta (Fig. 2, centre), the excess beta group had less power than the typical ADHD group ($F_{1,99} = 86.11$, $P < 0.001$), and this difference was greater in the posterior regions than the frontal regions ($F_{1,99} = 6.69$, $P < 0.05$). The difference between the midline and the two hemispheres (Fig. 3, centre) was less in the beta group ($F_{1,99} = 8.88$, $P < 0.01$). In relative theta (Figs. 4 and 5, top right), the excess beta group had less theta than the typical ADHD group ($F_{1,99} = 7.98$, $P < 0.01$). Laterally, greater power occurred in the left hemisphere (Fig. 5, top right), and the difference between the left and right hemispheres was greater in the excess beta group ($F_{1,99} = 5.26$, $P < 0.05$).

The excess beta group had more relative alpha (Figs. 4 and 5, bottom left) than the typical ADHD group ($F_{1,99} = 7.54$, $P < 0.01$), and this effect was greater in the posterior regions, compared with the frontal regions ($F_{1,99} = 11.14$, $P < 0.001$).

As expected, the excess beta group had greater absolute beta (Fig. 2, bottom right) than the typical ADHD group ($F_{1,99} = 60.15$, $P < 0.001$). This difference was greater in the frontal regions than the posterior regions ($F_{1,99} = 23.4$, $P < 0.001$) and less at the central regions than the mean of the frontal/posterior regions ($F_{1,99} = 14.32$, $P < 0.001$). Laterally (Fig. 3, bottom right), the difference between groups was greater in the right hemisphere than the left hemisphere ($F_{1,99} =$

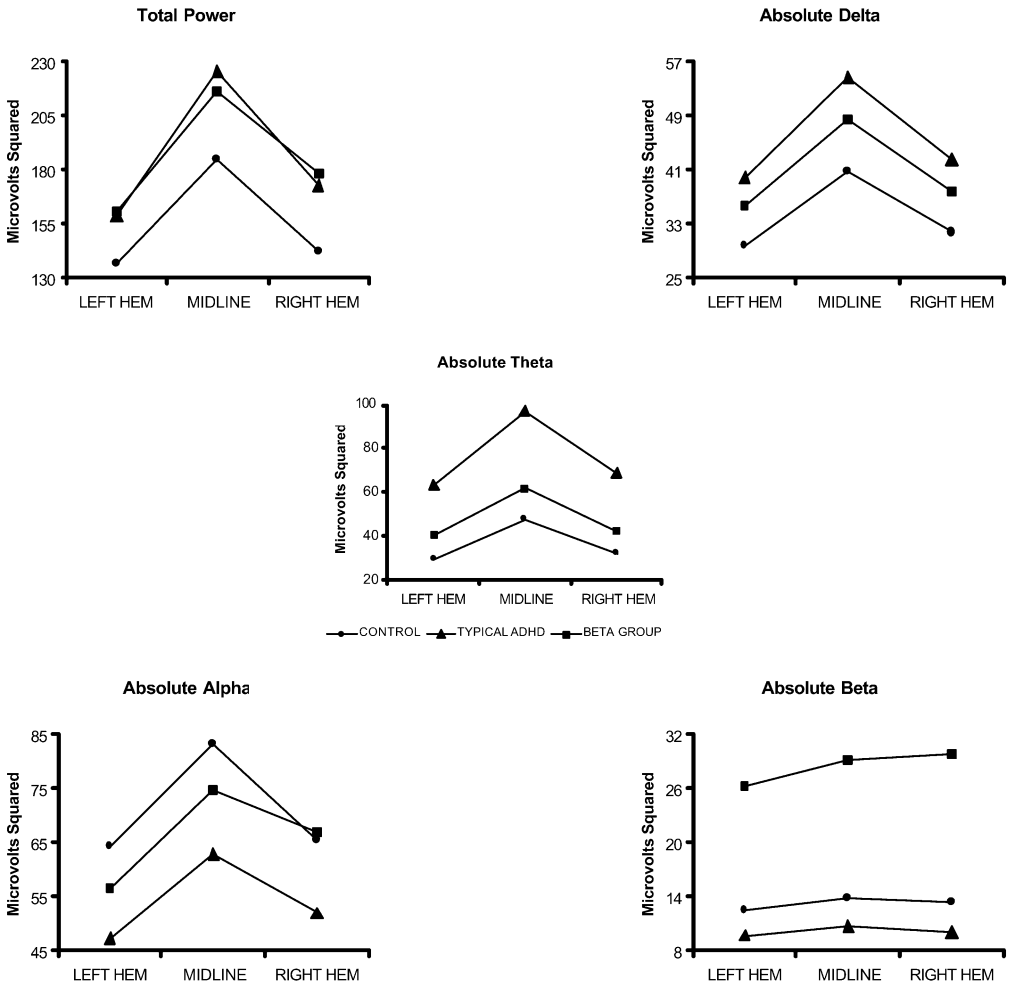

Fig. 3. Sagittal section for: Total Power (top left), Absolute Delta (top right), Absolute Theta (centre), Absolute Alpha (bottom left), Absolute Beta (bottom right).

19.28, $P < 0.001$), and this difference was greater in the frontal regions than the posterior regions ($F_{1,99} = 9.5$, $P < 0.01$). That is, the excess absolute beta was most apparent in the right frontal area. The excess beta group also had more relative beta (Fig. 4 and Fig. 5, bottom right) than the typical ADHD group ($F_{1,99} = 164.66$, $P < 0.001$) and this was greater in the frontal regions, compared with the posterior regions ($F_{1,99} = 109.13$, $P < 0.001$). Laterally (Fig. 5, bottom right), the difference between groups was greater in the right hemisphere than the left hemisphere ($F_{1,99} = 4.62$, $P < 0.05$). The difference between the midline and the two hemispheres was greater in the excess beta group ($F_{1,99} = 21.27$, $P < 0.001$), with the midline having less power than the two hemispheres. This group difference was greater at the frontal regions than the posterior regions ($F_{1,99} = 8.68$, $P < 0.01$). No significant group differences or interactions were found in total power, absolute delta or absolute alpha (Fig. 2 and Fig. 3, top left, top right and bottom left).

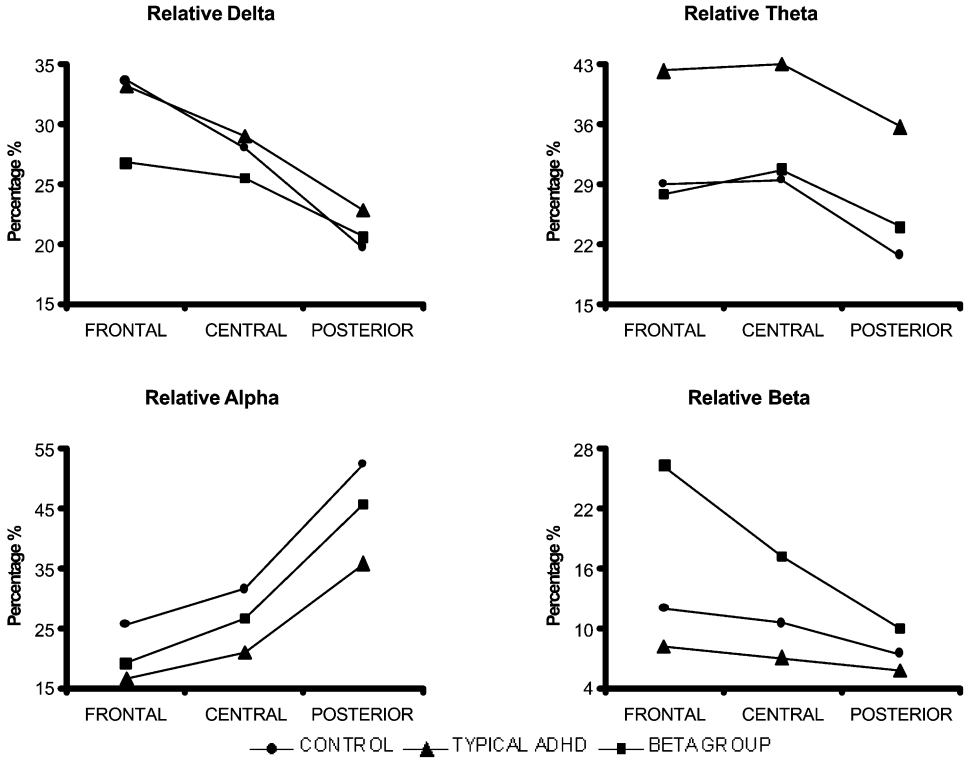

Fig. 4. Relative power from frontal to posterior regions: Relative Delta (top left), Relative Theta (top right), Relative Alpha (bottom left), Relative Beta (bottom right).

3.5. Excess beta vs. control group

The excess beta group had significantly less relative delta (Fig. 4 and Fig. 5, top left) than the control group ($F_{1,99} = 4.02$, $P < 0.05$), with the difference being greater in the frontal regions than the posterior regions ($F_{1,99} = 30.16$, $P < 0.001$). The midline had less power than the two hemispheres (Fig. 5, top left), with the difference less in the excess beta group than in the control group ($F_{1,99} = 5.16$, $P < 0.05$).

In relative theta (Fig. 4, top right), the excess beta group had a more posterior distribution than the control group ($F_{1,99} = 6.19$, $P < 0.05$). Laterally (Fig. 5, top right), the excess beta group had more theta in the left hemisphere compared with the right hemisphere than the control group ($F_{1,99} = 5.76$, $P < 0.05$).

In absolute alpha (Fig. 3, bottom left) the difference between the excess beta group and the control group was greater in the left hemisphere than the right hemisphere ($F_{1,99} = 6.38$, $P < 0.05$), with the difference being maximal in the left posterior region ($F_{1,99} = 6.83$, $P < 0.01$). Across sites, the excess beta group had less relative alpha (Fig. 4 and Fig. 5, bottom left) than the control group ($F_{1,99} = 8.09$, $P < 0.01$).

The excess beta group had greater absolute beta (Fig. 2, bottom right) than the control group ($F_{1,99} = 41.71$, $P < 0.001$). This difference was greater in the frontal regions than the posterior regions ($F_{1,99} = 31.58$, $P < 0.001$) and less at the central regions than the mean of the frontal/posterior regions ($F_{1,99} = 13.9$, $P < 0.001$). Laterally (Fig. 3, bottom right), the difference between groups was greater in the right hemisphere than the left hemisphere ($F_{1,99} = 14.23$, $P < 0.001$), with a maximal difference occurring at

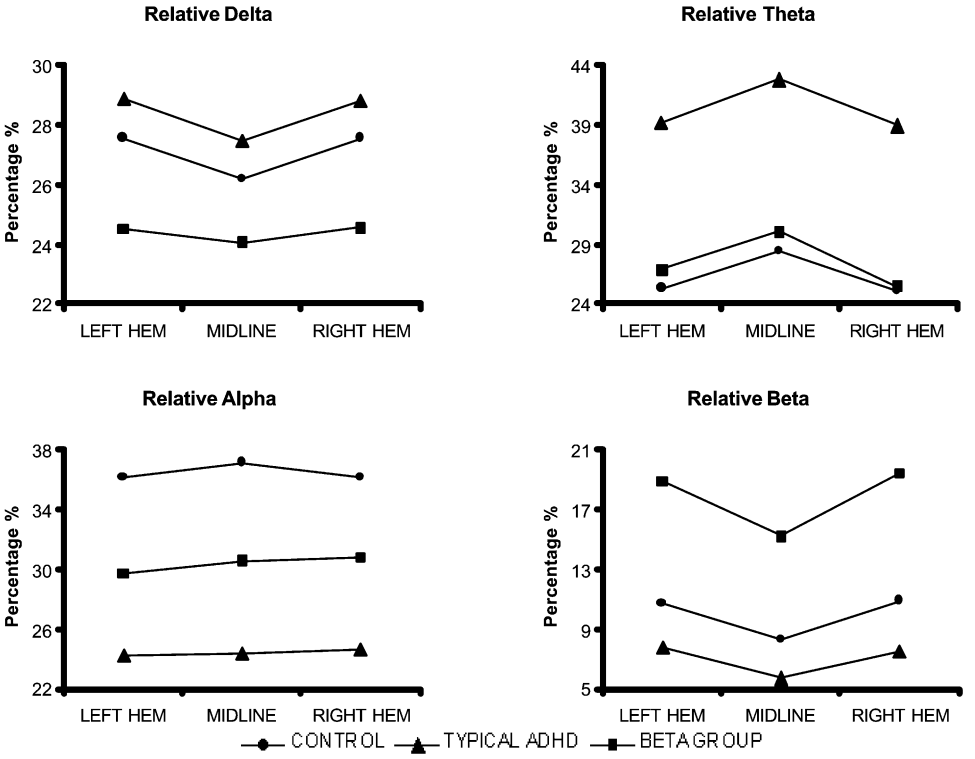

Fig. 5. Sagittal section for: Relative Delta (top left), Relative Theta (top right), Relative Alpha (bottom left), Relative Beta (bottom right).

the right frontal region ($F_{1,99} = 5.41$, $P < 0.05$). The excess beta group had more relative beta (Fig. 4 and Fig. 5, bottom right) than the control group ($F_{1,99} = 84.72$, $P < 0.001$). This difference was greater in the frontal regions than the posterior regions ($F_{1,99} = 76.2$, $P < 0.001$) and less at the central regions than the mean of the frontal/posterior regions ($F_{1,99} = 8.07$, $P < 0.01$). In the frontal regions, the difference between the midline and the two hemispheres was greater in the excess beta group than the control group ($F_{1,99} = 7.89$, $P < 0.01$).

No significant group differences or interactions were found in total power, absolute delta and theta (Fig. 2 and Fig. 3, top left, top right and centre).

3.6. Symptom profile differences

No significant differences were found between the two ADHD groups' behavioural profiles in regard to the DSM-IV Hyperactive/Impulsive or Inattentive checklists. Five behaviours, other than those in the DSM-IV checklists, were noted in the records of the diagnostic assessments. These were *poor self-esteem*, *moody/prone to temper tantrums*, *situationally aggressive at home* (not sufficient to be diagnosed as having a conduct or oppositional defiant disorder), *easily frustrated* and *socialisation problems*. The only behaviour for which a significant difference was found between the typical ADHD and excess beta groups was *moody/prone to temper tantrums*. Typical ADHD children's files noted this in 23.5% of cases, compared with 76.5% of cases for the children with excess beta ($\chi^2_{(1)} = 6.48$, $P < 0.05$).

4. Discussion

A significant difference was found between the IQ scores of the control group and the two ADHD

groups. Previous studies of ADHD have reported similar differences, but this has not been considered likely to have affected the EEG results (Satterfield et al., 1972; Clarke et al., 2001b). All groups in this study had a mean IQ in the average range, and as there were no significant IQ differences between the two ADHD groups (which are the focus of this article), IQ is not likely to have affected the present investigation.

Children with ADHD have generally been found to have elevated slow wave activity when compared with normal children (Capute et al., 1968; Satterfield et al., 1973a; Satterfield and Cantwell, 1974). Mann et al. (1992) found that ADHD subjects had an increase in absolute amplitude in the theta band in frontal regions and a decrease in beta in posterior regions. This was confirmed by Lazzaro et al. (1998), who found that the ADHD group had increased absolute theta and alpha 1 activity in frontal regions and reduced relative beta in posterior regions. Matsuura et al. (1993) found that children with ADHD had a higher average amplitude of delta, higher percentage time of delta and slow theta, and lower percentage time of alpha than normal control subjects. Janzen et al. (1995) found that ADHD children had a significantly higher theta amplitude than control subjects. In the ADHD groups, Chabot and Serfontein (1996) found an increase in absolute and relative theta, with the greatest increase being noted in frontal regions and at the midline, in comparison with normal control subjects.

Clarke et al. (1998, 2001b) found EEG differences between children with ADHDcom, ADHDin and control subjects. These differences occurred across all sites, with the ADHD groups having greater absolute and relative theta and less relative alpha and beta than control subjects. In posterior regions the ADHD groups had more relative delta than the control group. In each of these measures, the ADHDin group was positioned between the ADHDcom group and the control group.

Chabot and Serfontein (1996) reported that approximately 13% of children with ADHD had excess beta activity. In two of our previous ADHD studies (Clarke et al., 1998, 2001b), a subset of the ADHD children was identified with excessive levels of beta activity, constituting approximately 20% of children with ADHDcom. These children were excluded from the group analyses in those two studies. The present larger study found that approximately 15% of children with ADHDcom and 2% of children with ADHDin have excess beta activity, which is consistent with these other studies. The beta activity was of a higher amplitude than in the typical ADHD and control groups, but was still within the normal range reported in other studies (Kellway, 1990). In the frontal regions, beta activity varied from 16 to 19 Hz. These results are within the normal range reported in our previous study (Clarke et al., 2001b), which found beta frequencies in the control group to vary from 16 to 20 Hz. Beta in the 18–25 Hz range is often viewed as a medication effect (Fisch, 1994), but in this study, no subjects had a history of medication for ADHD and subjects were not prescribed any other form of medication at the time of assessment. For this reason, the beta levels cannot be seen as the result of medication. In children, beta levels of up to 60 μV have also been found in stage 1 and 2 sleep (Kellway and Fox, 1952). All our subjects were constantly checked for drowsiness during the recording of the EEG, and for this reason, the results are not believed to be associated with the subjects' state of alertness.

Sex differences were also noted in this group, with excess beta activity occurring more often in male subjects. Within clinical settings, referral biases have been found between males and females presenting for treatment (Arcia and Conners, 1998). In comparison to boys with ADHD, ADHD girls display greater intellectual impairment and lower rates of hyperactivity and other externalizing behaviours (Gaub and Carlson, 1997). It is possible that a referral bias in the sample resulted in the identification of fewer females with excess beta activity rather than that excess beta activity is a sex-linked characteristic.

To quantify exactly how the excess beta group differed from typical ADHD subjects, a sex- and age-matched ADHD group was used to compare both EEG and behavioural differences. The 'typical' ADHD comparison group was first compared

with normal control subjects, to determine whether this group had EEGs which were consistent with other ADHD studies. The typical ADHD group had more absolute delta and frontal relative delta, greater absolute and relative theta, and less relative alpha and beta than control subjects. The typical ADHD group also had an increase in frontal midline theta. These results are consistent with our two previous studies (Clarke et al., 1998, 2001b).

The excess beta group had EEG profiles which were different from both normal subjects and typical ADHD subjects. In absolute power, the excess beta group had less theta and more beta than the typical ADHD group. The excess beta group also had more fast wave activity in the relative alpha and beta bands. In comparison to this, the typical ADHD group was found to have more slow wave activity, with significant group differences being found in relative delta and theta. No group differences were found in total power, which would suggest that there was a replacement of theta activity by beta in the EEG of the excess beta group.

In both absolute and relative beta, maximal group differences were found in the frontal regions. Studies of children with ADHD have found neuropsychological deficits which suggest that the frontal cortex or regions projecting to the frontal cortex are dysfunctional in at least some ADHD children (Faraone and Biederman, 1998). In hyperactive children, these appear to be frontal-lobe mediated self-regulation deficits, such as in inhibition control (Rubia et al., 1998). Neuropsychological studies of subtypes of ADHD in adults have found deficits in the area of executive control functioning, which could be linked to dysregulation of frontal lobe systems (Gansler et al., 1998). Most of the subjects in the excess beta group had a hyperactive component to their diagnosis, and as the excess beta activity predominated in the frontal regions, it would appear that frontal lobe functioning is also involved in the behavioural disorders found in this group of ADHDcom subjects.

The only diagnostic behavioural difference between the two ADHD groups was that the excess beta group were more prone to temper tantrums and were more moody. All other behavioural symptoms were the same in both groups. Both mood and temper problems could be associated with dysfunction in the frontal-lobe systems associated with self-regulation and inhibition control, although more research is needed to determine why these systems are functioning in such a different way in two groups of children who are behaviourally very similar.

Although EEG studies of children with ADHD have primarily found increased levels of slow wave activity, the interpretation of what these results represent has been contentious. Some researchers have proposed that ADHD is the result of a maturational lag in the CNS (Kinsbourne, 1973; Mann et al., 1992), while others have viewed these results as representing a developmental deviation of the CNS (Chabot and Serfontein, 1996). Bresnahan et al. (1999) investigated changes in the EEG of ADHD subjects who where divided into three age groups: children, adolescents and adults. The results indicated that theta activity remained elevated in adults, but there was a decrease in beta activity with age. From these results, and a consideration of symptom profiles, it was suggested that beta activity may be linked to hyperactivity and increased theta activity to impulsivity, though this needed further investigation.

Clarke et al. (2001c) found that with increasing age, power changed at a greater rate in the ADHDcom group than in the ADHDin group, with power levels between the two groups becoming similar with age. These results suggest that ADHD has two components, with the hyperactive/impulsive component maturing with age and the inattentive component remaining more stable. The excess beta group in this study would have to be viewed as having an EEG profile that represents a developmental deviation of the CNS, rather than a maturational lag. This EEG profile could not be seen as normal for a person at any age. If our hypothesis is correct, and the beta is being generated by the same systems that are normally associated with excess theta in ADHDcom subjects, then this poses the question of whether these systems mature with age, as has been found in typical ADHDcom subjects. If maturation does

occur in this group, and the beta normalises with age, then these two groups of ADHDcom children are probably neuroanatomically similar. If maturation does not occur, then it is possible that this group of children represents a separate subgroup of the disorder. It is also not known whether the behavioural symptoms found in this group change with age, as is found in other groups with ADHD, or how well this group responds to medication. Further research is needed to investigate such questions within this group.

This study found that children with excess beta represent a small independent subset of children diagnosed with ADHD, primarily males with a diagnosis of ADHDcom. Behaviourally, this group is similar to other children with ADHD, although more prone to temper tantrums and moodiness. The excess beta activity is primarily found in the frontal regions and is probably associated with systems associated with self-regulation and inhibition control in the frontal lobes, although it is not known why these systems are functioning in such a different manner.

These results indicate that EEG measures have potential for use in the diagnosis of subtypes of ADHD. As the excess beta form of ADHD is behaviourally very similar to other forms of ADHD, EEG measures may provide an accurate method of differentiating between the two types. This would be especially pertinent if the excess beta group responds differently to medication and has a different developmental path. The reliability and specificity of these factors need to be further examined at an individual level for the clinical use to be properly evaluated.

References

American Psychiatric Association, 1994. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. American Psychiatric Press, Washington, DC.

Arcia, E., Conners, C., 1998. Gender differences in ADHD? Journal of Developmental and Behavioral Pediatrics 19, 77–83.

Benninger, C., Matthis, P., Scheffner, D., 1984. EEG development of healthy boys and girls. Results of a longitudinal study. Electroencephalography and Clinical Neurophysiology 57, 1–12.

Bresnahan, S., Anderson, J., Barry, R., 1999. Age-related changes in quantitative EEG in attention deficit disorder. Biological Psychiatry 46, 1690–1697.

Capute, A., Niedermeyer, E., Richardson, F., 1968. The electroencephalogram in children with minimal cerebral dysfunction. Pediatrics 41, 1104–1114.

Chabot, R., Serfontein, G., 1996. Quantitative electroencephalographic profiles of children with attention deficit disorder. Biological Psychiatry 40, 951–963.

Clarke, A., Barry, R., McCarthy, R., Selikowitz, M., 1998. EEG analysis in attention-deficit/ hyperactivity disorder: a comparative study of two subtypes. Psychiatry Research 81, 19–29.

Clarke., A., Barry., R., McCarthy., R., Selikowitz., M., 2001a. Age and sex effects in the EEG: development of the normal child. Clinical Neurophysiology 112, 806–814.

Clarke, A., Barry, R., McCarthy, R., Selikowitz, M., 2001b. EEG differences in two subtypes of attention-deficit/hyperactivity disorder. Psychophysiology 38, 212–221.

Clarke., A., Barry., R., McCarthy., R., Selikowitz., M., 2001c. Age and sex effects in the EEG: Differences in two subtypes of attention-deficit/hyperactivity disorder. Clinical Neurophysiology 112, 818–826.

Faraone, S., Biederman, J., 1998. Neurobiology of attention-deficit hyperactivity disorder. Biological Psychiatry 44, 951–958.

Fisch, B., 1994. Spehlmann's EEG Primer. Elsevier Science Publishers B.V, Amsterdam.

Gansler, D., Fucetola, R., Krengel, M., Stetson, S., Zimering, R., Makary, C., 1998. Are there cognitive subtypes in adult attention deficit/hyperactivity disorder? Journal of Nervous and Mental Disease 186, 776–781.

Gasser, T., Verleger, R., Bacher, P., Sroka, L., 1988. Development of the EEG of school age children and adolescents. I. Analysis of band power. Electroencephalography and Clinical Neurophysiology 69, 91–99.

Gaub, M., Carlson, C., 1997. Gender differences in ADHD: a meta-analysis and critical review. Journal of the American Academy of Child and Adolescent Psychiatry 36, 1036–1045.

Glaze, D., 1990. The effects of drugs on the electroencephalogram. In: Daly, D., Pedley, T. (Eds.), Current Practices of Clinical Electroencephalography, 2nd ed. Raven Press, New York.

Gibbs, F., Gibbs, E., 1950. Methodology and Controls. Addison-Wesley, Cambridge, MA.

Janzen, T., Graap, K., Stephanson, S., Marshall, W., Fitzsimmons, G., 1995. Differences in baseline EEG measures for ADD and normally achieving preadolescent males. Biofeedback and Self-Regulation 20, 65–82.

John, E., Ahn, H., Prichep, L., Trepetin, M., Brown, D., Kaye, H., 1980. Developmental equations of the electroencephalogram. Science 210, 1255–1258.

Kellway, P., 1990. An orderly approach to visual analysis: parameters of the normal EEG in adults and children. In: Daly, D., Pedley, T. (Eds.), Current Practices of Clinical Electroencephalography, 2nd ed. Raven Press, New York.

Kellway, P., Fox, B., 1952. Electroencephalographic diagnosis of cerebral pathology in infants during sleep. I. Rationale, technique and the characteristics of normal sleep in infants. Journal of Pediatrics 41, 262–287.

Kiloh, L., McComas, A., Osselton, J., Upton, A., 1981. Clinical Electroencephalography. Butterworths, London.

Kinsbourne, M., 1973. Minimal brain dysfunction as a neurodevelopmental lag. Annals of the New York Academy of Science 205, 268–273.

Kozelka, J., Pedley, T., 1990. Beta and mu rhythms. Journal of Clinical Neurophysiology 7, 191–207.

Lazzaro, I., Gordon, E., Whitmont, S., Plahn, M., Li, W., Clarke, S., Dosen, A., Meares, R., 1998. Quantified EEG activity in adolescent attention deficit hyperactivity disorder. Clinical Electroencephalography 29, 37–42.

Mann, C., Lubar, J., Zimmerman, A., Miller, C., Muenchen, R., 1992. Quantitative analysis of EEG in boys with attention deficit hyperactivity disorder: controlled study with clinical implications. Pediatric Neurology 8, 30–36.

Matousek, M., Rasmussen, P., Gilberg, C., 1984. EEG frequency analysis in children with so-called minimal brain dysfunction and related disorders. Advances in Biological Psychiatry 15, 102–108.

Matsuura, M., Okubo, Y., Toru, M., Kojima, T., He, Y., Hou, Y., Shen, Y., Lee, C., 1993. A cross-national EEG study of children with emotional and behavioural problems: a WHO collaborative study in the Western Pacific region. Biological Psychiatry 34, 52–58.

Matthis, D., Scheffner, D., 1980. Changes in the background activity of the electroencephalogram according to age. Electroencephalography and Clinical Neurophysiology 49, 626–635.

Morihisa, J., Duffy, F., Wyatt, R., 1983. Brain electrical activity mapping (BEAM) in schizophrenic patients. Archives of General Psychiatry 40, 719–728.

Pichlmayr, I., Lips, U., 1980. EEG effects of diazepam. Studies on 16 patients. Anaesthesist 29, 317–327.

Pollock, V., Schneider, L., 1990. Quantitative, waking EEG research on depression. Biological Psychiatry 27, 757–780.

Rubia, K., Oosterlaan, J., Sergeant, J., Brandeis, D., Leeuwen, T., 1998. Inhibitory dysfunction in hyperactive boys. Behavioural Brain Research 94, 25–32.

Satterfield, J., Cantwell, D., 1974. CNS function and response to methylphenidate in hyperactive children. Psychopharmacology Bulletin 10, 36–37.

Satterfield, J., Cantwell, D., Lesser, M., Podosin, R., 1972. Physiological studies of the hyperkinetic child: 1. American Journal of Psychiatry 128, 103–108.

Satterfield, J., Cantwell, D., Saul, R., Lesser, M., Podsin, R., 1973a. Response to stimulant drug treatment in hyperactive children: predictions from EEG and neurological findings. Journal of Autism and Childhood Schizophrenia 3, 36–48.

Satterfield, J., Lesser, M., Saul, R., Cantwell, D., 1973b. EEG aspects in the diagnosis and treatment of minimal brain dysfunction. Annals of the New York Academy of Sciences 205, 274–282.

Tabachnick, B., Fidell, L., 1989. Using Multivariate Statistics. Harper Collins, New York.

Wada, M., Ogawa, T., Sonoda, H., Sato, K., 1996. Development of relative power contribution ratio of the EEG in normal children: a multivariate autoregressive modeling approach. Electroencephalography and Clinical Neurophysiology 98, 69–75.

Wikler, A., Dixon, J., Parker, J., 1970. Brain function in children and controls: psychometric, neurological and electroencephalographic comparisons. American Journal of Psychiatry 127, 634–645.