

Simultaneous EEG and EDA measures in adolescent attention deficit hyperactivity disorder

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Abstract

Adolescent unmedicated ADHD males and age- and sex-matched normal control subjects were examined simultaneously using EEG and EDA measures in a resting eyes-open condition. ADHD adolescents showed increased absolute and relative Theta and Alpha1 activity, reduced relative Beta activity, reduced skin conductance level (SCL) and a reduced number of non-specific skin conductance responses (NS.SCRs) compared with the control subjects. Our findings indicate the continuation of increased slow wave activity in ADHD adolescents and the presence of a state of autonomic hypoarousal in this clinical group. © 1999 Elsevier Science B.V. All rights reserved.

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Attention Deficit Hyperactivity Disorder (ADHD) is a behavioural syndrome of unknown aetiology, one of the most disruptive psychiatric disorders of childhood, with a prevalence that has been estimated to affect as much as 10% of school-age boys (Dulcan, 1997). The principal features of this disorder are lack of sustained attention, hyperactivity and impulsivity [American Psychiatric Association (APA, 1994)] associated with low self-esteem and poor academic performance. There are a number of features of this disorder that are age specific. Younger children tend to exhibit signs of gross hyperactivity and motor activity whereas in older children and adolescents, the hyperactivity has in most cases abated but may be experienced as inner feelings of restlessness (APA, 1994). The inattention and impulsivity, however, tend to endure.

Non-invasive measures of brain activity, such as electroencephalography (EEG) have been utilised as a means of elucidating underlying neural substrates associated with this disorder. Previous EEG studies in ADHD have examined mostly pre-adolescent children and have found increased slow wave activity (mostly Theta) (Satterfield et al., 1972; Lubar, 1991; Mann et al., 1992; Matsuura et al., 1993; Janzen et al., 1995; Chabot and Serfontein, 1996; Defrance et al., 1996; Bresnahan et al., 1997; Clarke et al., 1998) in ADHD children compared with normal control subjects. Mann et al. (1992) reported that ADHD pre-adolescents without hyperactivity or specific learning disabilities showed significantly increased Theta activity in the anterior-central regions compared with normal control subjects. Other studies employed a ratio of Theta to Beta that takes into account individual variability to examine regional activity in ADHD. Lubar (1991) found that this ratio was increased in the anterior region of their ADHD sample while Janzen et al. (1995) found an increase in the posterior region. Furthermore, Defrance et al. (1996) reported that children with ADHD showed significantly increased Theta activity over the entire scalp compared with control subjects under both resting and attend conditions.

Other studies have also reported a consistently reduced Beta activity in ADHD children (Callaway et al., 1983; Satterfield et al., 1984; Mann et

al., 1992; Bresnahan et al., 1997; Clarke et al., 1998; Lazzaro et al., 1998). Satterfield et al. (1984) found an interaction between age and Beta activity. Younger ADHD children were found to show reduced Beta activity compared with control subjects.

Electrodermal activity (EDA) in ADHD has been investigated by a number of studies (for reviews see Rosenthal and Allen, 1978; Zahn, 1986). Most of these studies examined pre-adolescent ADHD children using electrodermal indices of arousal such as the skin conductance level (SCL), the skin conductance response (SCR) and the number of non-specific skin conductance responses (NS.SCRs) during resting, non-attend and attend conditions (Satterfield and Dawson, 1971; Satterfield et al., 1972, 1984; Cohen and Douglas, 1972; Spring et al., 1974; Montagu and Swarbrick, 1975; Montagu, 1975; Zahn et al., 1975, 1978, 1980; Rapoport et al., 1980; Pliszka et al., 1993; Shibagaki et al., 1993; Zahn and Kruesi, 1993). Some of these studies have reported reduced SCL (Satterfield and Dawson, 1971; Satterfield et al., 1972) and reduced task-related SCR amplitudes (Satterfield and Dawson, 1971; Cohen and Douglas, 1972; Spring et al., 1974; Zahn et al., 1975; Shibagaki et al., 1993) in unmedicated ADHD children compared with control subjects. The number of NS.SCRs was also found to be significantly lowered in ADHD children compared with control subjects (Satterfield and Dawson, 1971).

The behavioural disturbances characteristic of pre-adolescent ADHD children such as impulsivity and attentional deficits are known to continue into adolescence (Barkley, 1990). Only a paucity of studies have examined EEG and EDA measures in adolescent ADHD. Suffin and Hamlin Emory (1995) in an EEG study, employed an eyes-closed condition and neurometric EEG analysis to separate a subgroup of ADHD adolescents who showed both excessive frontal Theta activity and which were also responsive to stimulant medication, from a larger cohort of ADHD patients. Bresnahan et al. (1997) examined as part of a larger study, adolescents diagnosed with ADHD and found increased Theta activity in this group compared with control subjects. Our group (Laz-

zaro et al., 1998) examined EEG activity in 26 ADHD adolescents (11–17 years of age) during a 2-min resting eyes-open condition. Theta activity was significantly larger in the ADHD group compared with the control subjects in the anterior region and in the left and right hemispheres. With respect to EDA, Zahn and Kruesi (1993) examined a clinical sample of boys ranging in age from 6 to 17 years from the disruptive behaviour disorder spectrum consisting of ADHD, Conduct and Oppositional Defiant Disorders. There were no significant differences in SCL between the disruptive disorder sample and the normal control subjects. SCR frequency, however, was found to be significantly larger in the control subjects than in the disruptive behaviour group.

EEG and EDA measures have generally been explored separately in ADHD. No study has examined interactions between these central and peripheral measures in this disorder, although it is known that there are central networks exerting inhibitory and excitatory effects on electrodermal activity (Roy et al., 1993; Sequeira and Roy, 1993). Furthermore, our group in a previous study involving adult normal control subjects has shown that during a non-attend auditory task, Alpha, Beta and Theta power were each negatively correlated with SCL (Lim et al., 1996).

This current study served to explore simultaneously acquired EEG activity and autonomic electrodermal measures of arousal in a group of adolescent males diagnosed with ADHD and in normal control subjects during an eyes-open resting condition. The main hypotheses of this study are that Theta activity in the ADHD adolescents would be increased anteriorly and that simultaneously acquired SCL and the number of NS.SCRs would be significantly reduced in this group compared with control subjects. Increased Theta activity would also be associated with decreased arousal in the ADHD group.

Fifty-four adolescent males diagnosed with ADHD (mean age = 13.7 years; S.D. = 1.4; age range = 11–17 years) and 54 age- and sex-matched normal control subjects (mean age = 13.4 years; S.D. = 1.5; age range = 11–17 years) were examined in this study. ADHD patients were re-

ferred by paediatricians, clinical psychologists and psychiatrists who considered them to have a diagnosis of ADHD. All patients (accompanied by a parent) were subsequently interviewed by our team using a semi-structured interview based on DSM-IV criteria for ADHD (APA, 1994). For older children, symptom features present before the age of 7 years were established retrospectively with the aid of their parents. For children with a prior diagnosis of ADHD, records were available. Forty-seven patients fulfilled the criteria of ADHD of the Combined Type diagnosis while seven patients fulfilled the criteria for ADHD of the Predominantly Hyperactive–Impulsive Type. Thirty-four ADHD patients were drug naive at the time of electrophysiological testing while the remaining 20 were withdrawn from stimulant treatment for a period of 2 weeks or longer prior to testing. Each patient was then rated using the Conners' Parent (48-item) and the Conners' Teacher (28-item) Rating Scales (Conners, 1989) and the Achenbach Child Behaviour Check List for parents (Achenbach, 1991a) and Teacher's Report Form (Achenbach, 1991b). Patients were accepted into this study if they showed raised scores on the Hyperactivity Index on both the Conners' Parent and Teacher Rating Scales. For the Conners' Parent Rating, entry cut-off was set at a *T*-score of 1.0 S.D. above the norm while for the Conners' Teacher Rating, entry criteria was set at 1.5 S.D. above the norm. All patients were to have no history of neurological disorder or substance abuse.

Age-matched normal control boys were recruited from local high schools. Each control participant was interviewed prior to acceptance to ensure no history of ADHD, neurological disorder or substance abuse. Only control subjects with a *T*-score of < 1.0 S.D. above the norm on the Conners' Parent and Teacher Rating Scales were accepted into the study. Boys in both groups were further assessed for intellectual ability and learning achievement using the Kaufman Brief Intelligence Test (K-BIT: Kaufman and Kaufman, 1991) and the Wechsler Individual Achievement Test (WIAT: Psychological Corporation, 1992). Criterion for sample entry was a K-BIT compos-

ite IQ estimate of 75 or greater.

EEGs were acquired continuously for 2 min during a resting eyes-open condition. This formed the baseline of a larger study consisting of non-attend and attend conditions to be presented at a later stage. The EEGs were recorded using an electrocap (Blom and Anneveldt, 1982) from 19 electrode sites (Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, T3, T4, T5, T6, Pz, P3, P4, O1, O2) of the International 10–20 system. Linked earlobes served as reference. EOG activity was monitored via two bipolar electrodes placed 1 cm at the outer canthus of each eye to measure horizontal EOG and a separate bipolar montage was placed above and below the centre of the left eye to record vertical eye movement. All electrode impedances were $< 5 \text{ k}\Omega$. All subjects were seated in a reclining comfortable dental chair. During the recording, subjects were instructed to look at a small circular dot (to limit eye movement) placed 60 cm on a screen in front of them. All potentials were acquired on a Syn Amps (NEURO SCAN Inc.) 32-channel DC system with a gain of 200 and digitisation rate of 250 Hz. All signals were band-limited to 50 Hz. EOG correction was carried out post acquisition using the Gratton et al. (1983) procedure in which linear regressions were calculated between each of the EOG and the EEG channels. The regression coefficients were then determined from which correction factors were derived and applied to correct the EEG data.

Skin conductance was recorded simultaneously and continuously with the EEG. A pair of silver–silver chloride electrodes, approximately 0.8 cm^2 in contact area, filled with electrode paste (0.05 M NaCl in an inert ointment base) were placed on the volar surface of the distal phalanges of digits II and III of the non-dominant hand of each subject. The electrode pairs forming part of the input circuit were excited by a constant voltage of 0.5 V (Lykken and Venables, 1971; Fowles et al., 1981) and the current change representing conductance was recorded using the Syn Amps DC amplifier and digitised at 250 Hz.

A fast Fourier transform (FFT) analysis was applied to 60 1-s epochs of the EEG. An average power spectrum was then computed for each sub-

ject. This was then used to determine absolute and relative EEG activity (power) in the Delta (1.0–3.0 Hz), Theta (4.0–7.0 Hz), Alpha1 (8.0–9.0 Hz), Alpha2 (10.0–13.0 Hz), Alpha (8.0–13.0 Hz) and Beta (14.0–30.0 Hz) bands. Each frequency band was submitted separately to a repeated two-way analysis of variance, in which group (control subjects vs. ADHD) was a between subject factor and site was a repeated within subject factor. Between group regional analyses were then undertaken that examined the midline (Fz, Cz, Pz), anterior (Fp1, Fp2, Fz, F3, F4, F7, F8), posterior (Pz, P3, P4, T5, T6, O1, O2), left hemispheric (Fp1, F3, F7, C3, T3, P3, O1) and right hemispheric (Fp2, F4, F8, C4, T4, P4, O2) activity. Data was initially screened for normality of distribution and homogeneity of variance. To obtain normality of distribution, absolute power scores were log transformed. Relative power scores ($x\%$) were transformed using $\ln[x/(100 - x)]$ (Gasser et al., 1982). With repeated measures design, Greenhouse–Geisser correction was used for adjusting univariate results for violations of compound symmetry assumptions. Bonferroni-type adjustments were applied to control for type 1 error where appropriate.

For each 1-s epoch in which an FFT was calculated, the tonic SCL (μS) for that epoch was determined by calculating the average within that epoch. This resulted in 60 SCL values across the 2-min period. These 60 values were then averaged to form a mean SCL for each subject. SCL was then submitted to a two-sample *t*-test that compared the control subjects with the ADHD group. NS.SCRs were obtained for each subject using a curve fitting procedure (SCORES) based upon the sigmoid-exponential SCR model (Lim et al., 1997). This method allowed the decomposition of skin conductance into tonic and phasic components from which the NS.SCRs were determined. The number of NS.SCRs across the 2-min period for each subject was then obtained by counting the number of responses which occurred during this period. The number of NS.SCRs were then submitted to a non-parametric Mann–Whitney test for group comparison. Associations between absolute and relative Theta power with the EDA measures in every region were explored using

Table 1

Mean and standard deviations (S.D.) for the *T*-scores of the Conners' Teacher and Parent Rating Scales for the ADHD and normal control groups^a

Rating subscale	Control subjects <i>T</i> -scores (S.D.)	ADHD <i>T</i> -scores (S.D.)	Group differences (<i>P</i> -values)
Conners' Parent Hyperactivity Index	41.3 (4.8)	78.8 (10.0)	<i>P</i> < 0.0005
Conners' Teacher Hyperactivity Index	43.9 (5.1)	78.0 (11.9)	<i>P</i> < 0.0005
Conners' Parent Impulsive-Hyperactivity	41.1 (6.6)	71.9 (10.8)	<i>P</i> < 0.0005
Conners' Teacher Hyperactivity	45.0 (4.5)	74.4 (15.4)	<i>P</i> < 0.0005

^aSignificant group differences are shown. All group analysis based upon the non-parametric Mann-Whitney test.

correlational analyses. All statistical analyses were carried out using the Statistical Package for the Social Sciences (SPSS 6.1.3).

There were no significant differences in mean age ($t = 1.14$; $P > 0.1$) between ADHD patients and control subjects. Mean K-Bit composite IQ scores for the control subjects were (mean IQ = 103; S.D. = 9.7) and for the ADHD patients (mean

IQ = 96.3; S.D. = 10.5). ADHD patients had significantly larger mean rating scores on the Hyperactivity Index, Impulsive-Hyperactive and Hyperactivity subscales for the Conners' Teacher and Parent Rating Scales (Table 1).

Fig. 1 shows absolute anterior EEG activity for the control and ADHD groups in all the frequency bands. There were no significant differ-

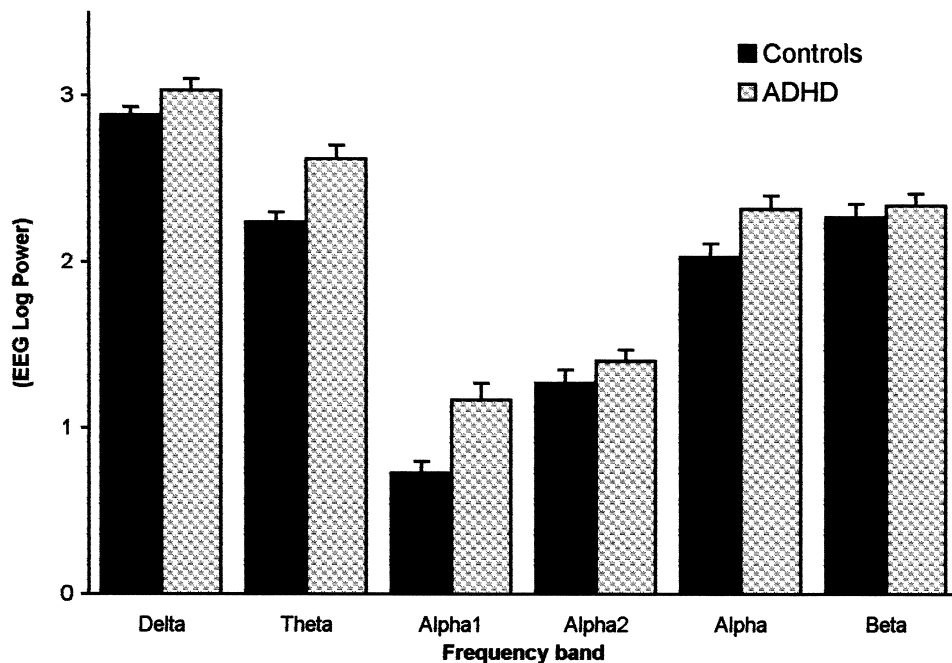


Fig. 1. Mean absolute anterior EEG power (log transformed) for the (Delta, Theta, Alpha1, Alpha2, Alpha and Beta) frequency bands with S.E. bars during the resting eyes-open condition in the Control and ADHD groups.

ences between control subjects and ADHD patients with respect to absolute Delta activity in any region. ADHD patients showed significantly increased absolute Theta activity in the midline ($F_{1,106} = 12.6$, $P = 0.001$), anterior ($F_{1,106} = 15.3$, $P < 0.0005$), posterior ($F_{1,106} = 8.99$, $P = 0.003$), left hemispheric ($F_{1,106} = 13.2$, $P < 0.0005$) and right hemispheric ($F_{1,106} = 12.96$, $P < 0.0005$) regions compared with control subjects. There were no significant group \times site interactions. ADHD patients also showed significantly larger midline ($F_{1,106} = 11.4$, $P = 0.001$), anterior ($F_{1,106} = 12.1$, $P = 0.001$), posterior ($F_{1,106} = 7.31$, $P = 0.008$), left hemispheric ($F_{1,106} = 10.8$, $P = 0.001$) and right hemispheric ($F_{1,106} = 9.51$, $P = 0.003$) absolute Alpha1 activity compared with control subjects. Alpha2 activity did not differentiate the groups. ADHD patients also showed significantly larger absolute Alpha activity compared with control subjects across the midline sites ($F_{1,106} = 7.79$, $P = 0.006$). However, with respect to anterior Alpha activity, there was a significant group \times site interaction in which ADHD patients showed significantly larger Alpha activity compared with control subjects at Fz, F3 and F7. There were no significant between group regional differences or interactions in absolute Beta activity.

With respect to relative EEG, relative Delta activity did not show any significant group differences. ADHD patients, however, showed significantly increased relative Theta activity across the midline ($F_{1,106} = 8.54$, $P = 0.004$), anterior ($F_{1,106} = 7.50$, $P = 0.007$), posterior ($F_{1,106} = 8.22$, $P = 0.005$), left hemispheric ($F_{1,106} = 7.24$, $P = 0.001$) and right hemispheric ($F_{1,106} = 9.06$, $P = 0.003$) regions compared with control subjects. There were no significant group \times site interactions. ADHD patients also had significantly larger relative Alpha1 activity across the midline ($F_{1,106} = 8.09$, $P = 0.005$), anterior ($F_{1,106} = 8.01$, $P = 0.006$), posterior ($F_{1,106} = 6.15$, $P = 0.015$), left hemispheric ($F_{1,106} = 8.92$, $P = 0.004$) and right hemispheric ($F_{1,106} = 9.39$, $P = 0.003$) regions compared with control subjects. There were no significant group differences with respect to either relative Alpha2 or Alpha activity. Relative Beta activity, however, was significantly reduced in ADHD patients compared with control sub-

jects at the midline ($F_{1,106} = 9.98$, $P = 0.002$), posterior ($F_{1,106} = 13.2$, $P < 0.0005$), left hemispheric ($F_{1,106} = 12.97$, $P < 0.0005$) and right hemispheric ($F_{1,106} = 8.3$, $P = 0.005$) regions. There were no significant group \times site interactions with respect to relative Beta activity.

The average tonic SCL across the 2-min period for the control and the ADHD groups is shown in Fig. 2 (upper). Control subjects (mean SCL = 6.59 μ S; S.D. = 2.6) showed significantly larger mean SCL across the 2-min period than the ADHD group (mean SCL = 5.49 μ S; S.D. = 2.5) ($t = 2.22$, $P < 0.03$). Also from Fig. 2 (lower), control subjects had a significantly larger number of NS.SCRs than the ADHD patients ($U = 1079$, $P < 0.02$).

Furthermore, the number of NS.SCRs across the 2-min period for each subject in the control and ADHD groups is shown in ascending order in Fig. 3. NS.SCRs were absent in 12 control subjects and in 19 ADHD patients.

Correlations were then explored between regional absolute and relative Theta power with the EDA measures for the control and the ADHD groups separately. Pearson's correlation between Theta activity and SCL did not reveal any significant correlations. However, a non-parametric Spearman correlation between Theta activity and the number of NS.SCRs resulted in a number of significant correlations. Absolute Theta power was found to be negatively correlated with the number of NS.SCRs for all regions in the ADHD group (Table 2). There were no significant correlations either in the control group or between relative Theta power with the EDA measures.

The aim of the present study was to examine whether increased anterior Theta activity (the most commonly reported EEG abnormality in pre-adolescent ADHD), lower SCL and a reduction in the number of NS.SCRs would be present in unmedicated ADHD adolescents.

The results of this study indicate that Theta activity is increased in ADHD adolescents compared with normal control subjects not just anteriorly but in all regions. These results as well as our previous findings of increased Theta activity (Lazzaro et al., 1998) suggests the continuation of slow wave activity into adolescent ADHD. Whether this continues into adulthood needs to

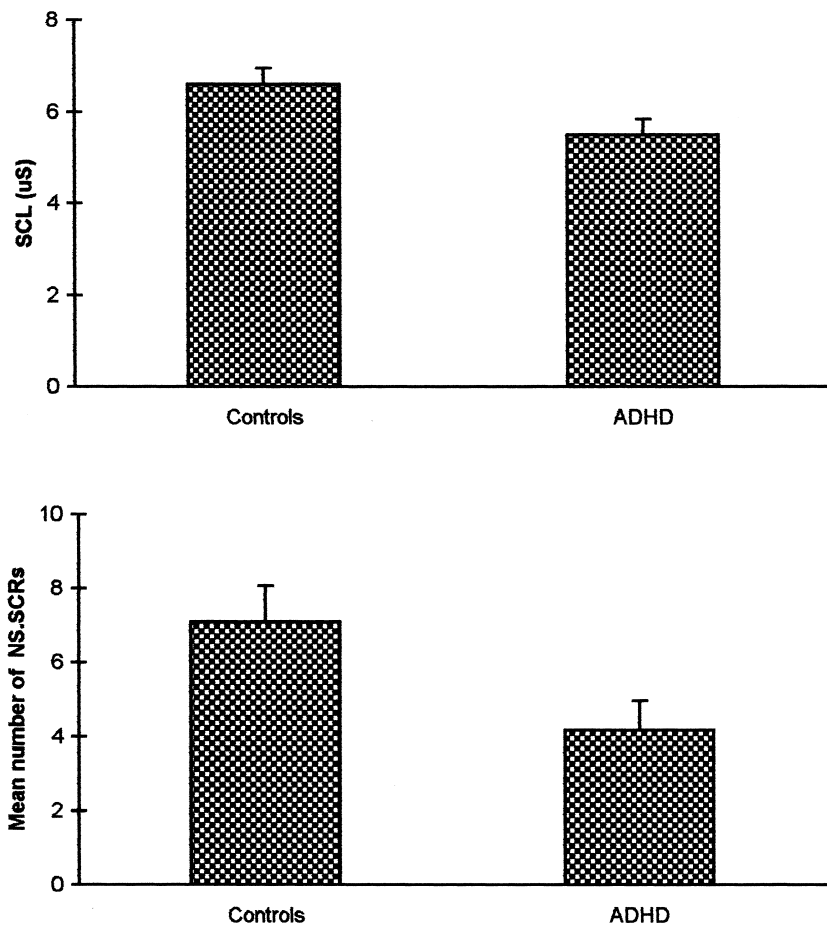


Fig. 2. Upper figure: Mean SCL with S.E. bars for the Control and the ADHD groups. Lower figure: Mean number of NS-SCRs with S.E. bars for the Control and the ADHD groups.

be further explored through a longitudinal study, although a study by Bresnahan et al. (1997) which examined preadolescent, adolescent and adult ADHD patients, indicated that although Theta activity decreased with advancing age, it remained elevated in all three clinical groups compared with their respective normal control subjects.

Increased Theta activity in our study is consistent with previous studies that have examined preadolescent ADHD children. However, the underlying neural substrates giving rise to this enhanced slow wave activity remains unclear. Some studies (Satterfield et al., 1972, 1974; Defrance et al., 1996) have interpreted the presence of increased slow wave activity during resting condi-

tions as evidence for a state of low CNS arousal in ADHD children. Mann et al. (1992) concluded that excessive Theta activity during activation tasks reflected reduced cortical arousal due to dysfunctional sub-cortical centres. From animal studies, it is known that Theta is associated with hippocampal-septal networks (Steriade et al., 1990).

The presence of excessive Theta activity may also reflect a neurodevelopmental delay in ADHD (Satterfield et al., 1974, 1984; Lubar, 1991; Mann et al., 1992; Matsuura et al., 1993; Clarke et al., 1998; Lazzaro et al., 1998). This may be evident from a number of developmental studies undertaken in normal children that have demonstrated

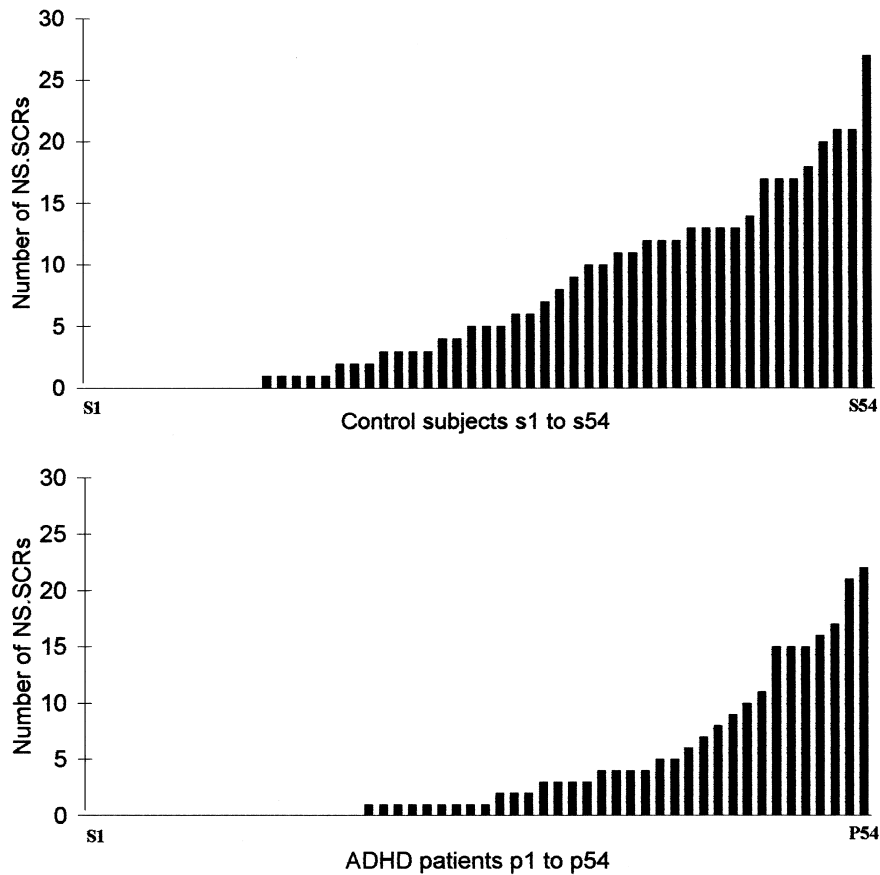


Fig. 3. The number of NS.SCRs for each Control subject (s1–s54) and for each ADHD patient (p1–p54) arranged in ascending order.

the decline of Theta activity with advancing age and also that the rate of this decline occurs more slowly over the anterior-central regions compared with the posterior (Benninger et al., 1984; Matsuura et al., 1985; Gasser et al., 1988a,b).

Elevated Theta activity in ADHD may also be associated with reduced cortical metabolic activity. Ingvar et al. (1976) found a positive correlation between cerebral blood flow and mean EEG frequency in cortical grey matter in a sample of adult chronic patients. The presence of increased anterior Theta activity in ADHD may be consistent with a regional cerebral blood flow (rCBF) study by Lou et al. (1984) that reported frontal hypometabolism in ADHD patients compared with normal control subjects and with a positron emission tomography (PET) study (Zametkin et

al., 1993) which found reduced glucose metabolism in the left anterior frontal lobe of ADHD adolescents compared with control subjects. Furthermore, the presence of increased Theta activity in the left hemisphere and in the posterior regions of our ADHD patients may also be consistent with Sieg et al. (1995) that reported reduced cerebral metabolism in the left anterior and left parietal regions in ADHD children using single photon emitted computed tomography (SPECT).

Increased Alpha1 activity (slow Alpha) in our adolescent ADHD patients seems to parallel our findings of increased Theta. Whether there is a coupling between these bands is uncertain. Gasser et al. (1988a) has shown that Alpha1 activity follows the same developmental course as Theta.

Table 2

Correlations between absolute and relative Theta power in each region with the number of NS.SCRs for the control and the ADHD groups^a

	Control	ADHD
Absolute Theta power		
Midline	NS	($r = -0.38, P = 0.005$)
Anterior	NS	($r = -0.31, P = 0.021$)
Posterior	NS	($r = -0.33, P = 0.016$)
Left hemisphere	NS	($r = -0.33, P = 0.017$)
Right hemisphere	NS	($r = -0.31, P = 0.022$)
Relative Theta power		
Midline	NS	($r = -0.25, P = 0.066, NS$)
Anterior	NS	NS
Posterior	NS	NS
Left hemisphere	NS	NS
Right hemisphere	NS	NS

^aNS = not significant.

Steriade et al. (1990) has indicated that Theta may be a slowing down of Alpha activity during certain pathological states. Raine et al. (1990) found both increased Theta and Alpha1 activity in 15-year-old males with a predisposition to criminality and suggested that this represented a state of low cortical arousal in criminal behaviour. Omori et al. (1995) reported that the concurrent increases in fast Theta and Alpha1 activity in never-treated schizophrenic patients reflected dysfunctional reticulothalamic pathways. Voeller (1991) and Castellanos (1997) have posited that the behaviours characteristic of ADHD may result from dysfunctions along cortical-striatal-thalamic networks. Chabot and Serfontein (1996) have suggested that the presence of increased Theta and Alpha activity in ADHD children may reflect such disturbances.

The reduction in relative Beta activity in our ADHD sample compared with the control subjects is consistent with previous studies. Some of these studies (Mann et al., 1992; Lazzaro et al., 1998) have suggested that this reflects a state of cortical hypoarousal in ADHD since increased Beta activity has been shown to be an index of cortical activation (Morihsa et al., 1983; Miyauchi et al., 1990; Kuperman et al., 1996). Bresnahan et al. (1997) reported that the difference in relative Beta activity between their normal control and

ADHD groups decreased with increasing age at the anterior-central regions and became normalised by adulthood. They also suggested that Beta activity was related to the hyperactivity dimension in ADHD which is known to abate with increasing age.

With respect to our concomitant EDA measures, ADHD patients showed significantly reduced mean SCL across the 2-min period. This is in agreement with Satterfield and Dawson (1971) and Satterfield et al. (1972) but is inconsistent with the majority of studies that have not found significant group differences with respect to SCL (Cohen and Douglas, 1972; Spring et al., 1974; Montagu, 1975; Zahn et al., 1975; Rapoport et al., 1980; Satterfield et al., 1984; Pliszka et al., 1993). The number of NS.SCRs, which is an index of arousal (Raine et al., 1990; Boucsein, 1992) was also found to be significantly lowered in our unmedicated ADHD group compared with the control subjects. This is consistent with Satterfield and Dawson (1971) but is in contrast to previous studies that did not find significant group differences in the number of NS.SCRs (Spring et al., 1974; Zahn et al., 1975, 1978; Pliszka et al., 1993). Also the significant number of negative correlations between absolute Theta power and the number of NS.SCRs in the ADHD group not only further supports the notion of a state of low arousal in this group but also that there is an interaction between cortical and electrodermal brain stem measures. Satterfield et al. (1972) although not employing NS.SCRs as their index of arousal did report that ADHD patients who showed both increased slow wave activity and low SCL were the best responders to stimulants on pre-treatment testing while the converse was true for the worst responders to stimulant medication. Reduced EDA in ADHD has been postulated to result from the attenuated activity of the noradrenergic pathways of the locus coeruleus (Satterfield and Dawson, 1971; Satterfield et al., 1974, 1990). The locus coeruleus is a brainstem nucleus that is associated with the level of arousal and from animal studies has been found to modulate cortical EEG activity (Berridge and Foote, 1991; Berridge et al., 1993a,b). Bilateral inhibition of the locus coeruleus by α 2-noradrenergic agonists

gave rise to increased cortical slow wave activity in the forebrain of a rat (Berridge et al., 1993a,b). This seems to suggest that enhanced cortical Theta activity (particularly in the anterior) in ADHD may result from inadequate activation of subcortical structures. The underlying mechanisms of this interaction, however, needs to be further elucidated, perhaps using orienting responses in cognitively engaging paradigms where attention deficits in the ADHD group may be more pronounced.

To summarise, our study has indicated that increased slow wave EEG activity (Theta) continues into adolescent ADHD. ADHD adolescents are also in a state of autonomic hypoarousal as indexed by their lower SCL and their reduced number of NS.SCRs. Theta activity was also found to be related to EDA in the ADHD group.

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