

# Attention-deficit hyperactivity disorder involves differential cortical processing in a visual spatial attention paradigm

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## Abstract

**Objective:** Inattention is undoubtedly one of the main characteristics of Attention-deficit hyperactivity disorder (ADHD). Nevertheless, a growing corpus of evidence shows that not all attentional processes are affected in this condition. This study aimed to explore the distribution of attentional resources in children with ADHD via a spatially shifted double-oddball visual task.

**Methods:** We recorded event-related potentials (ERPs) for all visual stimuli. Subjects were instructed to allocate attention in a specific area of visual space while ignoring all stimuli presented outside. Ten male children (age: 9–14; mean = 11.6 ± 2.1) who met DSM-IV criteria for the ADHD combined subtype participated in the study, along with ten age- and sex-matched healthy controls (9–14; mean = 11.2 ± 2.3).

**Results:** ADHD subjects showed late differential cortical responses to initially suppressed irrelevant stimuli. The amplitude of early N1–P1 components were mainly modulated by stimulus location and showed no significant differences between groups, but a late P300-like positivity was clearly evoked in the ADHD group by peripheral stimuli.

**Conclusions:** These results suggest that ADHD may not compromise the early attentional spatial filter but rather entails a different distribution of attentional resources at later stages of cortical processing. Perhaps these differences may be attributable to individual differences in attentional mechanisms.

**Significance:** ADHD may not affect initial focusing of visual attention but rather the allocation of processing resources in later stages. © 2006 International Federation of Clinical Neurophysiology. Published by Elsevier Ireland Ltd. All rights reserved.

**Keywords:** ADHD; Attention deficit; Visual spatial attention; ERP; P300

## 1. Introduction

Attention-deficit hyperactivity disorder (ADHD) is a very common neuropsychiatric disorder clinically characterized by inattention, impulsivity and hyperactivity (Steinhausen et al., 2003). Great research efforts have been

devoted to understanding the physiopathology of this condition. Some etiological factors, both genetic and environmental, have been identified and still appear as major topics of research in ADHD (Castellanos and Tannock, 2002). At the level of neuropsychological evaluation, impairment in some attentional processes was expected to be the main cognitive deficit. In fact, the well-known evaluation of ‘sustained attention’ by means of continuous performance tasks (CPT) has repeatedly shown an increased number of errors in ADHD subjects (Corkum and Siegel,

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1993). Nevertheless, recent reports of detailed neuropsychological explorations of attentional functions in ADHD suggest that other attentional subcomponents might be spared in this condition (Koschack et al., 2003; Huang-Pollock and Nigg, 2003; Sergeant et al., 2002; Barkley, 1997). ADHD children can even perform better than controls in some attentional tasks that involve divided attention (Koschack et al., 2003). New evidence points to poor inhibitory control as a central factor in explaining ADHD symptoms (Huang-Pollock and Nigg, 2003; Durston, 2003; Sergeant et al., 2002; Barkley, 1997). This deficiency could explain both the deficits in cognitive functioning and impulsive behaviors associated with the disorder (Barkley, 1997). These apparent disagreements highlight the need to revisit how attentional resources are used, distributed and controlled in a condition that is clinically characterized by inattention. The present study explores the amplitude of early and late ERP components which have been described to reflect resource allocation in visual spatial tasks (Luck et al., 1996; Mangun and Hillyard, 1990, 1991).

Due to their high temporal resolution, ERPs have been used frequently to study both normal attention and ADHD. Most visual ERP reports on ADHD focus on the neural correlates of the poor performance in extensive CPT and on its improvement after medication. Behavioral reports on the performance of ADHD children in visuo-spatial attention tasks suggest a differential pattern of reaction times compared to controls (McDonald et al., 1999), but only few ERP studies focus into spatial attention in this condition (Barry et al., 2003). It has been demonstrated that the amplitude of P1 and N1 components of the ERPs is modulated by the attention allocated to a specific visual stimulus (Barry et al., 2003; Kastner and Ungerleider, 2000; Clark and Hillyard, 1996). This evidence has supported the view of an early spatial filter for attentional selection in the visual system (Clark and Hillyard, 1996). In ADHD, early ERP components P1 and N1 are often described as delayed in latency and reduced in amplitude during visual-spatial attention tasks (Perchet et al., 2001; Steger et al., 2000). This might well be interpreted as a deficiency in the early spatial filter in ADHD. Nevertheless, increased amplitude of early positivities has also been described in children with this condition in other experimental designs, such as categorization and seriation tasks (Robaey et al., 1992). Previous studies have also reported differential amplitude for early ERP components in ADHD and related conditions. Buchsbaum and Wender (1973) reported larger amplitude for N140-P2 in children with the diagnosis of Minimal Brain Damage in a passive presentation paradigm. This finding was also supported by data from hyperactive children's responses to standard stimuli in an oddball paradigm (Callaway et al., 1983). Interestingly, reduced P1 amplitudes have been recorded in response to standard and deviant, but not to novel stimuli, in ADHD subjects (Kemner et al., 1996). This data indicates that the interpretation of the amplitude of early ERP components in ADHD is not always simple because

it can be modified by multiple factors, like the type of task or the cognitive strategy used to solve it. This emphasizes the need for more comprehensive designs to study attentional processes in ADHD.

Another ERP component frequently used to study attention and ADHD is a late positive deflection referred to as the P300. A delayed latency and decreased amplitude of the P300 is the most usual finding in ADHD studies (Barry et al., 2003). Jonkman et al. (2000) used a double-task paradigm to compare attentional capacity between ADHD and control children. Subjects had to solve two versions of a primary task (easy–hard) while they were passively viewing an oddball task. They found that control subjects had an increase of P300 amplitude to deviants from the easy to the hard version of the primary task. This increase was not found in ADHD subjects. This difference was not evident in the responses to 'novel targets' in which they found that P3 amplitudes decreased from the easy to the hard task to the same extent in both groups. They interpreted these results as indicative of a deficiency in capacity allocation rather than of a capacity shortage in ADHD children. They suggested that both ADHD and control subjects might 'have the same amount of extra capacity at their disposal, but the ADHD children did not, or were not able to invest it in the task when task demands increased'.

These results could also be indicative of a differential pattern of distribution of attentional resources in ADHD. It seems apparent that this differential pattern would become more evident under conditions of high attentional demands and task complexity. But ADHD children also have poorer results in simpler everyday tasks. The DSM-IV criteria describe that ADHD children "often do(es) not follow through on instructions..." and "fail to understand instructions". Exploring the amplitude modulation of ERP components sensitive to attention in a task that does not necessarily pose high attentional demands could give us more information about whether this differential pattern is only present when attentional capacity is challenged or if it is a manifestation of a usual style of resource allocation in this disorder. Investigations in this direction may lead to a better understanding of this highly prevalent condition.

In the present study, we designed a non-simultaneous visual double-oddball task intermixed in time and shifted in space to assess the children's ability to concentrate attention in a specific area of the visual field where they would have to distinguish between two stimuli (relevant-infrequent and irrelevant-frequent). They were asked to selectively ignore any stimulus presented outside this area. A permanently visible frame served as a spatial cue for the intended focus of attention. In accordance with this instruction, we anticipated a stimulus selection strategy based first on spatial location (spatially valid/spatially invalid) and second on stimulus relevance (relevant-infrequent and irrelevant-frequent). This was also anticipated to be reflected in a specific pattern of amplitudes of the

ERP components: (1) early components as P1/N1 would be larger for stimuli presented in the valid location than for those presented outside the frame; (2) the P300 effect elicited by infrequent target detection would be affected by the strategy of actively ignoring stimuli outside the spatially cued area and therefore it would appear only after relevant-infrequent stimuli presented inside the attended area. Comparing ADHD and Control subjects in these two stages of visual processing could give us new evidence regarding the temporal profile of resource allocation in ADHD.

## 2. Methods

### 2.1. Participants

Two groups of subjects were selected to participate in the case-control study:

**ADHD group:** Ten male children (age: 9–14;  $11.6 \pm 2.1$ ) who met DSM-IV criteria for the ADHD combined subtype and had no major comorbidity (American Psychiatric Association, 1994). These subjects were randomly selected out of a larger sample participating in a genetic association study on ADHD and reevaluated by a pediatric neurologist and a child psychologist. They were originally recruited from general (secondary care) psychiatric and neurological outpatient services. All of them had a clinically proven history of good response to stimulant medication, which they had been using for at least 3 months when recruited. Seven of the subjects were using methylphenidate and the other three were using D-amphetamine. They were asked to interrupt stimulant treatment the day before and the day that the exam was taken, consequently they were evaluated after more than 48 h without medication. Both they and their parents agreed to participate in the study and signed written consent forms.

**Control group:** Ten age- and sex-matched healthy controls (age: 9–14; mean =  $11.3 \pm 2.2$ ) who were selected out of a large group who volunteered for the study from city public schools. No monetary compensation was given. They underwent a complete physical and psychological examination and were classified using the same instruments as the ADHD group: Conners' Abbreviated Parent-Teacher Questionnaire (Rowe and Rowe, 1997) and DSM-IV. They and their parents agreed to participate in the study and signed written consent forms.

All participants were Chilean, native Spanish speakers, right handed according to the Edinburgh Inventory (Oldfield, 1971) and had no parental antecedents of left handedness. They had an average or higher IQ assessed by WISC-R (Wechsler, 1974), and agreed to be examined to rule out any morbidity.

Participants from ADHD and controls groups were compared regarding age, IQ (WISC-R) and the total scores from the 10-items Conners' Abbreviated Parents-Teachers Questionnaire. Conners' items are rated from 0 (not at all) to 3 (very much) and Total Score represents the sum of the points in all items. The Wilcoxon Matched Pairs test

showed no significant difference regarding age. Group Means and Standard Deviations (SD) were Controls: 11.6 (2.1), ADHD: 11.2 (2.3)  $p = 0.68$ . The IQ (WISC-R) was not significantly different between the groups: Controls: 110.5 (14.99), ADHD: 112.3 (13.0),  $p = 0.76$ . The total scores from the 10-items Conners' Abbreviated Parents-Teachers Questionnaire were compared between the groups using a non-parametric Mann-Whitney  $U$  Test. The scores of the ADHD group ranged from 18 to 23 (mean: 19.3, SD: 1.49) and from 1 to 4 in the Control group (mean: 2.3, SD: 0.67). Mann-Whitney  $U$  Test showed significant differences between the groups  $Z = -3.78$ ;  $p < 0.01$ . All individual items reported to be related with overactive – inattentive behaviors (items 1, 2, 4, 5 and 6) had significantly different scores between the groups ( $p < 0.01$ ). These results are in agreement with previous reports from studies that used larger samples (Rowe and Rowe, 1997).

### 2.2. Stimuli and procedure

The experimental procedures were approved by the Ethical Committee of the Faculty of Medicine, Universidad de Chile.

Visual stimulation was accomplished using E-Prime™ software. A fixed frame of  $3 \times 3$  cm, subtending  $3.5^\circ$  of visual angle, was permanently shown in the center of a computer screen. Subjects were asked to pay attention only to stimuli presented within this central frame. Stimuli consisted of  $2.5 \times 2.5$  cm facial photos of a male and a female. They were arranged in the following oddball sequence: the male face was the frequent stimulus (standard) and the female face the infrequent one (target). Subjects were asked to mentally count how many times the target stimulus appeared inside the central frame (Central Task) and to ignore any stimulus presented outside.

These same two faces were also randomly presented outside the central frame in one of 24 positions arranged in three concentric circles (Peripheral Task). The outer circumference represented  $16^\circ$  of eccentricity. Fig. 1 illustrates stimulus types or conditions: T1, Central Target; S1, Central Standard; T2, Peripheral Target; S2, Peripheral Standard.

A trial sequence started with a central cross that appears for 150 ms marking the fixation point. This was followed by a waiting period with a random duration between 50 and 150 ms before stimulus presentation (Stimulus Onset Asynchrony, SOA: 200–300 ms). Stimulus (T1, S1, T2 or S2) were presented for 300 ms and followed by another random duration period of 600–950 ms. The Standard : Target ratio was 9:1 for both the central and the peripheral oddball. The overall number of stimuli was 540 (270 S1, 30 T1, 216 S2 and 24 T2). The peripheral oddball was specially counterbalanced in order to avoid consecutive targets presentations in the center and periphery.

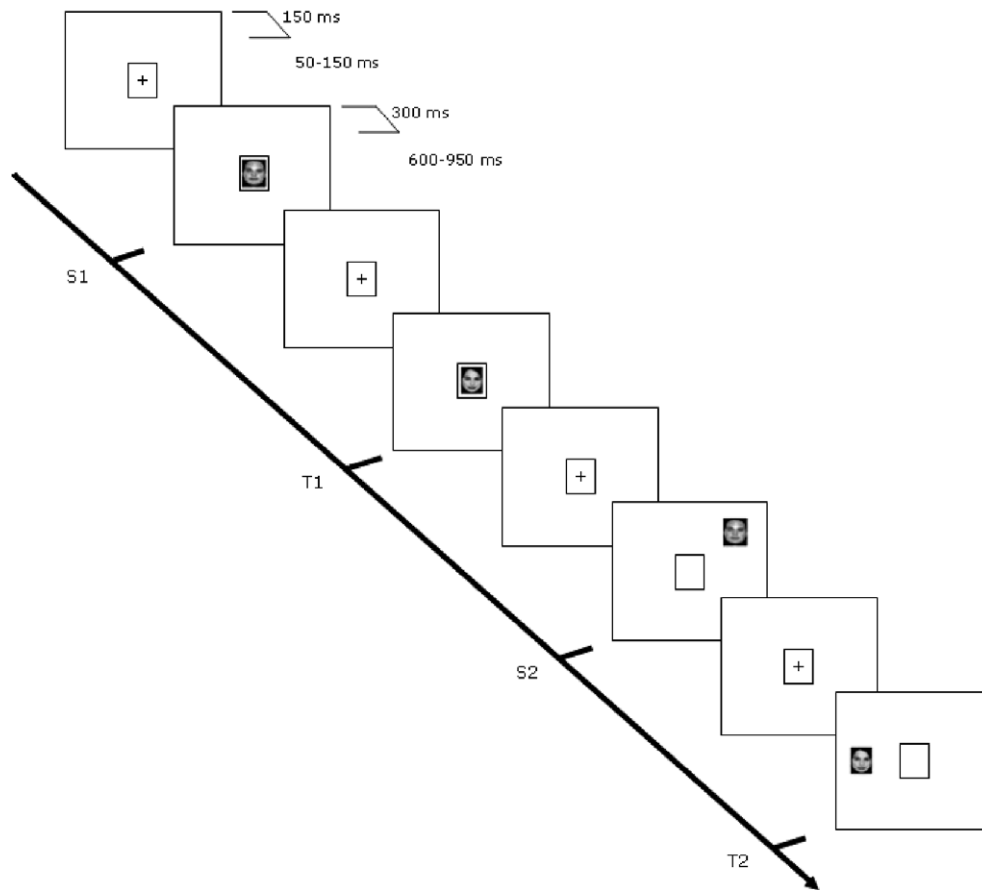


Fig. 1. Illustration of stimulus types or conditions. T1, Central Target; S1, Central Standard; T2, Peripheral Target; S2, Peripheral Standard. The intended area of attention was delimited by a frame permanently showed at the center of the screen. A trial sequence started with a central cross that appeared for 150 ms marking the fixation point. This was followed by a waiting period with a random duration between 50 and 150 ms before stimulus presentation (Stimulus Onset Asynchrony, SOA: 200–300 ms). Stimulus (T1, S1, T2 or S2) were presented for 300 ms and followed by another random duration blank (600–950 ms). Subjects were instructed to ignore any face outside the central frame.

### 2.3. Electrophysiological recordings

Electrophysiological signals were recorded using a 64-channel system from Electrical Geodesic Inc. with a 16-bit A/D converter and the NetStation™ software. The recording sampling rate was 1000 Hz. Analog filters were set at 0.1 and 100 Hz. A band pass digital filter between 0.5 and 30 Hz was later applied to remove unwanted frequency components. Signals were re-sampled at 250 Hz to reduce file size. During recordings the reference was set (by default) to vertex, but later average reference was calculated and applied. Two bipolar derivations were designed to monitor vertical and horizontal ocular movements (EOG).

Continuous EEG data were segmented from 200 ms prior to stimulus to 800 ms after it. All segments with eye movement contamination ( $>65 \mu\text{V}$ ) were removed from further analysis. Artifact free segments were averaged to obtain the ERPs. The number of remaining trials per category after artifact rejection in the control group were [mean (SD)]: S1: 249.8 (9.3); T1: 26.4 (2.6); S2: 199.8 (7.5); T2: 21.5 (1.4). In the ADHD group they were: S1: 237.9 (14.8); T1:

23.9 (4.0); S2: 190.0 (9.3) and T2: 20.5 (1.2). These did not differ significantly between the groups. The EEGLAB Matlab toolbox was used for EEG off-line processing and analysis (Delorme and Makeig, 2004).

ERP waveforms were averaged separately for the four experimental conditions: frequent male face at the attended central frame (S1), infrequent female face at the central frame (T1), frequent male face in the periphery (S2) and infrequent female face in the periphery (T2). Nine regions of interest (ROI) were defined to represent the scalp topography of the ERP components. Groups of electrodes were collapsed into these regions in order to avoid loss of statistical power (Oken and Chiappa, 1986). These regions were labeled combining the terms: Anterior, Central, Posterior, and Right, Central, Left (AL, AC, AR, CL, CC, CR, PL, PC and PR). Corresponding electrodes were AL (9, 13–16, 19, 20), AC (2, 3, 6–8, 11, 12), AR (1, 56–58, 60–62), CL (17, 21–25, 27), CC (4, 5, 18, 30, 43, 55), CR (47, 49, 50, 52–54, 59), PL (28, 29, 31–33, 35, 36), PC (34, 37–40) and PR (41, 42, 44–46, 48).

The waveform obtained from each ROI and condition was subdivided, after stimulus onset, into non-overlapping

50 ms intervals. Within each of these intervals, the average of all data points was computed, yielding an average amplitude measure that was later used in an exploratory statistical comparison between conditions. A very similar approach has been used before by other authors (Alho et al., 1989; Loewy et al., 1996). This allowed the identification of the time windows that should be considered in the definitive statistical analysis. Although there were various intervals with statistically significant differences, only a 50 ms interval centered on the peak latency of the ERP component of interest (P1, N1, P3) in each condition and from the ROI where the component has its larger scalp amplitude was chosen to perform the statistical analysis reported. These intervals were: P1 90–140 ms, posterior left ROI, (Peak latency: 114 ms); N1 140–190 ms, middle frontal ROI, (Peak latency: 165 ms). In the case of the P3-like positivity a wider 100 ms interval was used due to its temporal course. This interval spanned from 400 to 500 ms, middle central ROI.

#### 2.4. Statistical analysis

Although the figures show the ERPs grand averages from each group, all statistical calculations were done using individual waveforms. A mixed ANOVA design (Group  $\times$  Stimulus Location  $\times$  Stimulus Type) with repeated measures over the latter two factors was used to independently compare N1 and P3 amplitude across conditions and groups. The factor Stimulus Location had two levels: central and peripheral. The factor Stimulus Type had 2 levels Standard (S: Male face) and Target (T: Female face).

Topographic effects for the ERP components used in the present study have been described well elsewhere (Kutas and Van Petten, 1994; Hillyard and Anllo-Vento, 1998), therefore the statistical analysis was performed with amplitude data from the ROIs where the ERP component has its maximum amplitude. Univariate comparisons were done when necessary. Results were corrected with the Greenhouse-Geisser and Bonferroni's methods to adjust the univariate output of repeated measures ANOVA for violations of the compound symmetry assumption.

### 3. Results

#### 3.1. Behavioral responses

In the present experiment, the percent of correct responses on mental counting of primary target (T1) was over 98% in both groups. This result diverged from the findings of an increased number of errors of these same ADHD subjects in several other cognitive tasks they underwent in our laboratory.

#### 3.2. ERP analysis

ERP waveforms are shown in Fig. 2. Stimulus presentation elicited, in all conditions, an early posterior positive

deflection and a more frontal negative deflection, hereafter named as P1 and N1, respectively. The voltage scalp maps for these components in each condition are shown in Fig. 3.

The N1 component showed maximal amplitude over the anterior midline and its amplitude varied according to the eliciting condition. In both the ADHD and Control groups, the early N1 showed higher amplitude for those stimuli presented inside the central frame (S1 and T1) while it was reduced for those presented outside the central frame (S2 and T2). A mixed ANOVA (Group  $\times$  Stimulus Location  $\times$  Stimulus type) with repeated measures over the last two factors was conducted to contrast these amplitude modulations. The levels for stimulus location were central (S1, T1) and peripheral (S2, T2). The levels for stimulus type were standard (S1, S2) and target (T1, T2). Significant differences were found only for the main effect Stimulus Location ( $F_{(1,18)} = 53.7, p < 0.01$ ). No significant differences were found between groups ( $F_{(1,18)} = 2.01, p = 0.61$ ) nor between Stimulus Types ( $F_{(1,18)} = 0.74, p = 0.39$ ). There were no significant interactions found between Stimulus Location and Group ( $F_{(1,18)} = 1.78, p = 0.19$ ) nor between Stimulus Location and Stimulus Type ( $F_{(1,18)} = 1.53, p = 0.23$ ).

Due to the large differences in the number of trials per category after artifact rejection, two additional ANOVAs were conducted to corroborate the results described above. The second ANOVA (Group  $\times$  Stimulus Location) consisted of using only the N1 amplitude values from the standard stimuli (S1 and S2). Significant differences were found only for the main effect Stimulus Location ( $F_{(1,18)} = 31.6, p < 0.01$ ). No significant differences were found for the factor group ( $F_{(1,18)} = 2.53, p = 0.12$ ) and there were no significant interactions found between Group and Stimulus Location ( $F_{(1,18)} = 0.31, p = 0.58$ ).

A third ANOVA (Group  $\times$  Stimulus Location) was conducted using only the N1 amplitude values from the Target Stimuli (T1 and T2). These values are comparable since the number of remaining trials after artifact rejection is very similar between these categories. Again, significant differences were found only for the main effect Stimulus Location ( $F_{(1,18)} = 44.3, p < 0.01$ ). No significant differences were found for the factor group ( $F_{(1,18)} = 2.573, p = 0.07$ ) and there were no significant interactions found between Group and Stimulus Location ( $F_{(1,18)} = 2.04, p = 0.16$ ).

These results were corroborated by Bonferroni-corrected Pairwise Mean comparisons of N1 amplitude between the conditions: S1–S2 (Mean difference:  $-1.42$ , Standard Error:  $0.25, p < 0.01$ ) and T1–T2 (Mean difference:  $-1.8$ , Standard Error:  $0.27, p < 0.01$ ). Mean differences between S1–T1 and S2–T2 did not reach the statistical significance level. Thus, N1 amplitude was modulated more by stimulus localization (Central–Peripheral) rather than by stimulus type (Target–Standard) or Group.

The amplitude modulations of the P1 component (from the left posterior ROI) exhibited very similar results. A mixed ANOVA (Group  $\times$  Stimulus Location  $\times$  Stimulus type) with repeated measures over the last two factors

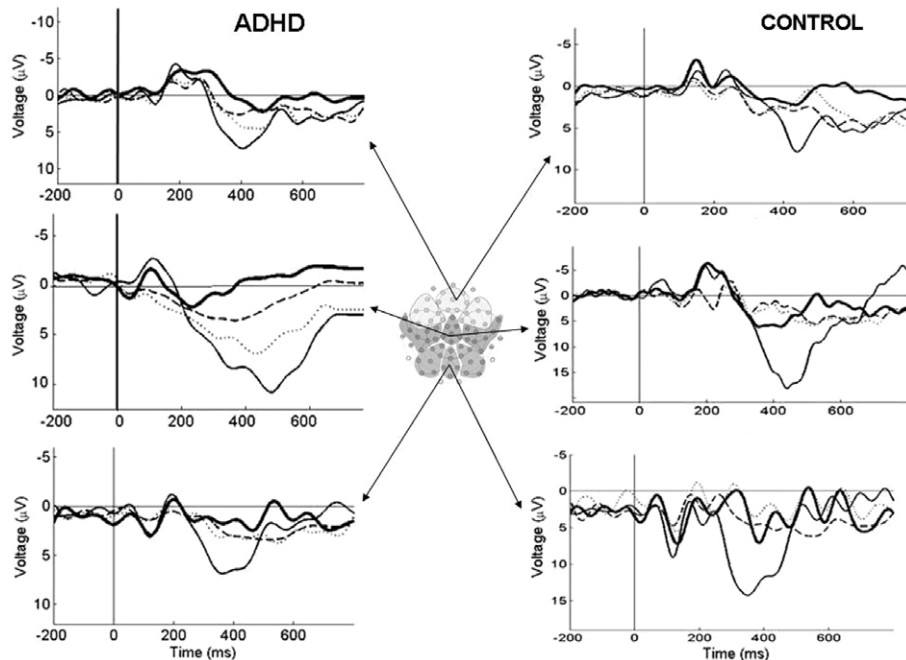


Fig. 2. ERP waveforms for every condition and group. Solid thick line T1, solid thin line S1, dotted line T2 and dashed line S2. According to the amplitude distribution in the scalp of N1/P1 and P3, midline ROIs were selected for illustration.

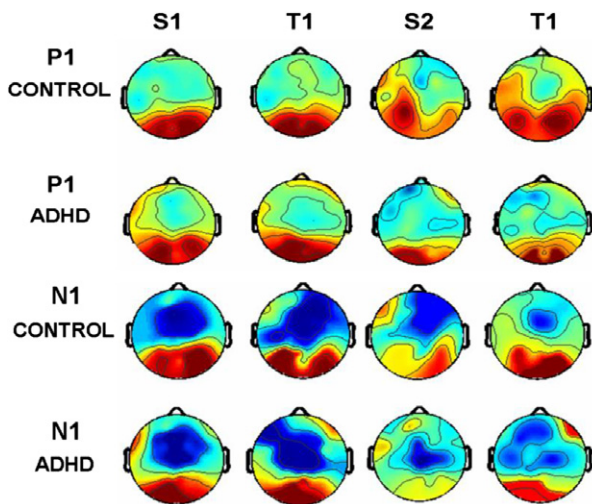


Fig. 3. Topographic maps showing the scalp distribution of the components P1 and N1 in the studied groups and conditions.

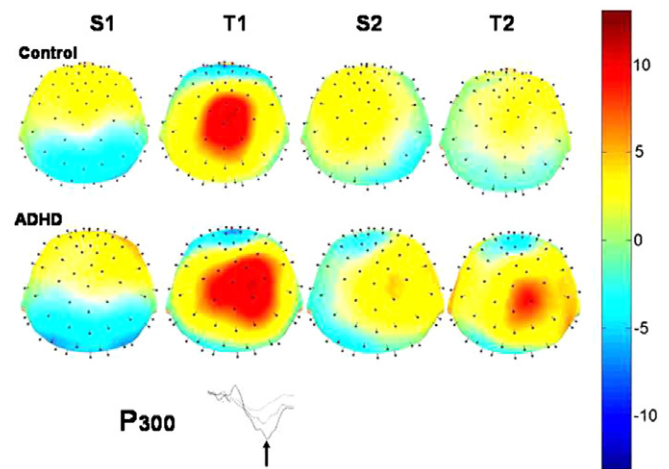


Fig. 4. Topographic maps showing the scalp distribution of the P3 component in the studied groups and conditions. T1 elicited significant P3 component both in ADHD and Control groups. T2 also elicited a significant P3 in ADHD.

showed significant differences only for the main effect Stimulus Location ( $F_{(1,18)} = 22.6, p < 0.01$ ). No significant differences were found between groups ( $F_{(1,18)} = 1.09, p = 0.71$ ) nor between Stimulus Types ( $F_{(1,18)} = 0.92, p = 0.23$ ), nor were any significant interactions found.

The late ERP components exhibited striking intra- and inter-group differences. As expected, the ERP elicited by T1 shows a large P3-like positive deflection peaking after 400 ms post-stimulus and with the topography usually described for this component (Barry et al., 2003) (see Fig. 4). The statistical analyses reported here were performed over the amplitude values of P3 from the Central

Midline ROI. A mixed ANOVA (Group  $\times$  Stimulus Location  $\times$  Stimulus Type) with repeated measures over the latter two factors was conducted to compare P3 amplitude across conditions and groups. The factor Stimulus Location had two levels: central and peripheral and Stimulus Type had also two levels, Standard (S: Male face) and Target (T: Female face).

There were significant differences for the main effects Group ( $F_{(1,18)} = 15.27, p < 0.01$ ); Stimulus Type ( $F_{(1,18)} = 51.03, p < 0.01$ ); and for Stimulus Location ( $F_{(1,18)} = 6.06, p = 0.024$ ). Significant interactions were also

found between Group and Stimulus Location ( $F_{(1,18)} = 11.20$ ,  $p < 0.01$ ), and between Stimulus Location and Stimulus Type ( $F_{(1,18)} = 18.7$ ,  $p < 0.01$ ). A three way interaction Group  $\times$  Stimulus Location  $\times$  Stimulus Type was also significant ( $F_{(1,18)} = 14.8$ ,  $p < 0.01$ ).

This pattern of results can be explained mainly by the fact that the expected amplitude reduction (or even disappearance) of the P300 component for peripherally located stimuli did not occur in the ADHD group. This explains the Group  $\times$  Stimulus location significant interaction and, as this effect is more evident for the targets, it may also account for the Stimulus Location  $\times$  Stimulus Type interaction and the Group  $\times$  Stimulus Location  $\times$  Stimulus Type interaction. Bonferroni-corrected univariate post hoc comparisons ( $MS = 3.1452$ ,  $df = 35.012$ ) confirmed these hypotheses. Follow-up univariate comparisons of the Group  $\times$  Stimulus Location interaction showed that the amplitude values corresponding to peripherally located stimuli in the control group were significantly smaller ( $MS = 2.76$ ,  $df = 35.9$ ) than those elicited in the ADHD group by stimuli located in the central cued area ( $p < 0.01$ ) and in the periphery ( $p < 0.01$ ). In the Stimulus Location  $\times$  Stimulus Type interaction ( $MS = 3.67$ ,  $df = 18$ ), the amplitude of P3 to target stimulus in the central location (T1) was, as expected, significantly larger than in any other condition ( $p < 0.01$ ). However, the main finding comes from the Group  $\times$  Stimulus Location  $\times$  Stimulus Type interaction. This analysis showed that a large P300 component elicited by target stimuli presented in a peripheral location in the ADHD group was significantly different from all other conditions ( $MS = 3.1452$ ,  $df = 35.012$ ,  $p < 0.01$ ). The amplitude value from the Standard stimuli presented in a peripheral location (S2) in the ADHD group was different from the S1 condition in both groups ( $p < 0.05$ ) and from S2 in the control group ( $p < 0.05$ ).

Univariate pairwise mean comparisons (Bonferroni corrected) showed that in both groups the P3 amplitude after the presentation of T1 was significantly larger than in all other conditions. Mean and Standard Errors (in  $\mu V$ ) were: Control Group (S1:  $-1.0$  (0.4), T1:  $4.0$  (0.6), S2:  $-0.8$  (0.5), T2:  $-0.6$  (0.5)). ADHD Group (S1:  $-0.8$  (0.4), T1:  $4.1$  (0.6), S2:  $0.8$  (0.5), T2:  $3.1$  (0.6)). In the Control group the waveform elicited by S1, T2 and S2 showed no significant differences beyond 300 ms. In contrast, in the ADHD group a graded P3 amplitude increase was noticed following the order  $S1 < S2 < T2 < T1$  (see Fig. 2). Pairwise comparisons showed significant differences within this group between conditions T2 and S2 ( $p < 0.01$ ) and also between T2–S1 ( $p < 0.01$ ) and T1–T2 ( $p < 0.01$ ).

#### 4. Discussion

The present investigation had three important results: (1) the absence of differences between ADHD and Controls regarding the amplitude modulation of the early ERP components according to stimulus localization, (2) the elicitation of a late P300 by target stimuli presented outside the

intended area of attention in ADHD but not in Controls, and finally, (3) the amplitude modulation of this ‘peripheral’ P300 in ADHD according to stimulus relevance in the assigned cognitive task.

N1 amplitude has been previously associated with the amount of attention allocated to a specific area of the visual field (Hillyard, 2000). As a matter of fact, the modulation by attention of these cortical electrical responses constitutes the main evidence of early spatial attentional selection in the visual system (Hillyard and Anllo-Vento, 1998). Our results suggest that this so-called ‘early spatial attentional filter’, as reflected by N1 amplitude modulation, could be intact in ADHD. Nevertheless, this might change in tasks demanding fixation of attention at a point for longer periods or in a more monotonous visual scenario. The behavioral responses in the present experiment evidenced it was an easy task for both ADHD and Controls. Dynamic changes in spatial visual filtering may explain the differences between our results and those reported in previous studies about N1 amplitude in ADHD (Barry et al., 2003).

It is widely accepted that, besides stimulus infrequency and novelty, attention is an important modulating factor for P300 amplitude (Kutas and Van Petten, 1994). The elicitation in ADHD subjects of a late P300-like positivity in the T2 and S2 conditions of the present experiment could be considered, therefore, as evidence of late attention allocation to these stimuli. In the light of the present result we suggest that, in ADHD subjects, after an initial attentional suppression of ‘irrelevant’ stimuli presented outside the attended area of the visual field, there is a redistribution of attentional resources according to stimulus relevance, generating a gradient of P300 amplitude. This is, perhaps, related to a higher processing level. In other words, ADHD implies a late differential distribution of attentional resources but not incapacity to initially focus attention over an area of the visual space. It is important to notice that attention allocation is not the same for all ‘irrelevant’ stimuli in the periphery: T2 elicited larger P3 than S2, although the targets in the attended central frame (T1) exhibited the largest P3 amplitudes. That rules out the possibility of considering the allocation of attention in ADHD as a senseless capture of attentional resources by any disruptive stimuli. On the contrary, the pattern of attentional resource distribution exhibited by our ADHD subjects is more likely a different style of allocating attention. This attentional ‘flexibility’ might allow ADHD individuals to occasionally have a more rapid response style, as has been evidenced by some neuropsychological studies (Koschack et al., 2003).

Mangun and Hillyard (1990) demonstrated that in visual attention tasks, higher processing stages reflected in P3 amplitude do not necessarily depend on stages reflected in early ERP components. Reductions in earlier selection processes (reflected in P1 and N1 amplitudes) need not be paralleled by proportional reductions in detection performance and the associated P300 component. They suggested

that in tasks requiring selective focusing of attention, sensory and central-cognitive processes do not always have to follow each other in a strictly serial or hierarchical order, and can operate in a more or less independent fashion. The early and late ERP components were considered as indices of ‘separate but interacting levels of attentional selection having different operating principles’. In this line we interpreted the elicitation of P300 by targets presented outside the cued area as evidence of a late reassignment of resources in ADHD that did not occur in controls. As a matter of fact, in ADHD subjects, the standard stimuli presented outside the cued area also elicited a significant P3. This pattern of modulation of P3 amplitude resembles previous reports of studies that used the focused attention paradigm with low presentation rates, where the attended non-target stimuli also elicited P3, although of a lower amplitude than attended target stimuli (van der Stelt et al., 1998; Looren de Jong et al., 1988).

Reporting electrophysiological changes without overt behavioral differences is still controversial. Nevertheless, this is not a new issue in the ERP literature (Kotchoubey, 2006; Gray et al., 2004) or in the ADHD (Karayanidis et al., 2000; Harter et al., 1988). Given the simplicity of the task, minimal or no differences in overall behavioral performance were expected between the two groups. One of the main reasons for using ERPs is that this technique can overcome some of the known limitations of behavioral measurements. The high temporal resolution of ERPs allows, at least from a theoretical point of view, a real time monitoring of the cortical processes underlying cognitive activity. In a recent review Kotchoubey (2006) addresses this topic and states that overt responses are, in the end, a product of multiple covert processes, but because different processes can lead to the same result, measuring only the end result would never allow the kind of inferences that can be made from ERP data. He also points at the necessity of identifying the processing of irrelevant signals that need no overt response as one of the conditions in which ERPs could be very helpful. This is the case in the present design. Evidence has accrued about the modulation of different ERP components by the allocation of attention (Hillyard and Anllo-Vento, 1998). This information can be used to presume attention allocation without the need of any overt response. A behavioral measurement in this condition would require the participant recognition of a psychological event and the translation of that recognition into an overt behavioral response (Gray et al., 2004). This would transform the whole design and certainly impede the “irrelevant condition”. According to Gray et al. (2004) the use of ERPs could allow the identification of cognitive resources allocation inaccessible to conscious awareness, as well as the time course of the cognitive processing that could not be assessed using reaction times. Nevertheless, it is worthy to consider that our poor knowledge about the neural correlates and functional significance of ERP components poses a serious limitation to this approach and therefore caution is recommended.

In the case of ADHD, Karayanidis et al. (2000) is a clear reference to ERP differences between ADHD and controls that did not impact behavioral responses. These authors review the need to consider electrophysiological findings regardless of whether they are or are not accompanied by behavioral differences. They argue that dissociation can exist between ERP and behavioral results and emphasize the need to explore if ADHD affects discrimination and selection processes in simple visual tasks. In a similar situation, Harter et al. (1988) suggested that the presence of ERP differences in the absence of a performance deficit may represent a processing deficit that behavioral measures are not sensitive enough to detect or, on the contrary, that ERPs could be reflecting compensatory processes, rather than the deficit itself. It is our opinion that the present results reflect a deficit or a deviation in ADHD that behavioral measures are not sensitive enough to detect, at least in simple tasks.

A recent report on the effect of action video games on visual skills showed that habitual playing enhances the capacity of visual attention and its spatial distribution (Green and Bavelier, 2003). Moreover, this capacity can also be trained in occasional players. These differences in the way the same visual scene is attended by different persons suggest a greater flexibility and individuality of attentional processes than previously supposed. The characteristics of attention processes in ADHD could also represent a different approach or implicit strategy to analyze the environment more than a real deficit of attention. The possibility of modifying these characteristics by means of training should not be overlooked. ADHD children seem to use their attentional tuning ‘differently’, but this may not be enough to explain the cause of all their real-life problems. Even though much research is still necessary to comprehend the physiopathology of ADHD, a better understanding of its cognitive profile could allow us to design new approaches to this condition, both in the medical and the educational systems.

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