Electrophysiological evidence of enhanced distractibility in ADHD children


Cognitive Brain Research Unit, Department of Psychology, University of Helsinki, Helsinki, Finland
Department of Psychiatry, Yale University and VA-Connecticut Healthcare System, West Haven, CT, USA
Cognitive Neuroscience Research Group, Department of Psychiatry and Clinical Psychology, Faculty of Psychology, University of Barcelona, Catalonia, Spain
Department of Child Neurology, Hospital for Children and Adolescents, Helsinki University Central Hospital, Helsinki, Finland
Helsinki Brain Research Centre, Helsinki, Finland
Collegium of Advanced Studies, University of Helsinki, Finland
Department of Psychology, University of Helsinki, Helsinki, Finland

Received 26 June 2004; received in revised form 14 October 2004; accepted 21 October 2004

Abstract

Abnormal involuntary attention leading to enhanced distractibility may account for different behavioral and cognitive problems in children with attention deficit hyperactivity disorder (ADHD). This was investigated in the present experiment by recording event-related brain potentials (ERPs) to distracting novel sounds during performance of a visual discrimination task. The overall performance in the visual task was less accurate in the ADHD children than in the control children, and the ADHD children had a higher number of omitted responses following novel sounds. In both groups, the distracting novel sounds elicited a biphasic P3a ERP component and a subsequent frontal Late Negativity (LN). The early phase of P3a (180–240 ms) had significantly smaller amplitudes over the fronto-central left-hemisphere recording sites in the ADHD children than in the control group presumably due to an overlapping enhanced left-hemisphere dominant negative ERP component elicited in the ADHD group. Moreover, the late phase of P3a (300–350 ms) was significantly larger over the left parietal scalp areas in the ADHD children than in the controls. The LN had a smaller amplitude and shorter latency over the frontal scalp in the ADHD group than in the controls. In conclusion, the ERP and behavioral effects caused by the novel sounds reveal deficient control of involuntary attention in ADHD children that may underlie their abnormal distractibility.

Keywords: Attention deficit hyperactivity disorder; Distraction; Children; Orienting; P3a; Reorienting negativity (LN/RON)

Attention deficit hyperactivity disorder (ADHD) is a multi-dimensional disorder that has its onset in childhood and that is characterized by persistent problems of inattention, impulsivity, and hyperactivity [2,15]. In ADHD patients, difficulties with sustained attention tasks [11] can be caused by different alternatives, one being abnormal distractibility. Therefore, the present study compared involuntary attention and distractibility in ADHD and healthy children, as indicated by distraction of visual task performance by task-irrelevant novel sounds and by event-related brain potentials (ERPs) to these sounds.

Highly deviant, task-irrelevant auditory and visual stimuli elicit a P3 component of ERP called the P3a or novelty P3 [5,21]. The P3a is maximal over the fronto-central left-hemisphere recording sites in the ADHD children than in the control group presumably due to an overlapping enhanced left-hemisphere dominant negative ERP component elicited in the ADHD group. Moreover, the late phase of P3a (300–350 ms) was significantly larger over the left parietal scalp areas in the ADHD children than in the controls. The LN had a smaller amplitude and shorter latency over the frontal scalp in the ADHD group than in the controls. In conclusion, the ERP and behavioral effects caused by the novel sounds reveal deficient control of involuntary attention in ADHD children that may underlie their abnormal distractibility.

© 2004 Elsevier Ireland Ltd. All rights reserved.

Keywords: Attention deficit hyperactivity disorder; Distraction; Children; Orienting; P3a; Reorienting negativity (LN/RON)
switching is evidenced by the fact that P3a-eliciting novel sounds also distract visual task performance in adults [6] and children [9].

The P3a to novel sounds has two phases: an early phase (eP3a) peaking at 200–250 ms and a late phase (IP3a) peaking at 300–350 ms from sound onset [6]. In adults, the eP3a has its maximum amplitude over the central scalp, whereas the IP3a is distributed widely and has its maximum over the frontal scalp. According to scalp current density analysis [25], magnetoencephalography [1], and studies in patients with local brain lesions [14], the eP3a is generated in the auditory and temporoparietal cortices, whereas the IP3a gets a major contribution from the prefrontal cortex [13,25]. Thus, multiple brain areas are involved in involuntary attention and auditory novelty detection. This appears to be true also in children. The scalp distributions of the eP3a and IP3a to novel sounds in children aged over 11 years are similar to those in adults [9]. However, in 7–10-year-olds, the IP3a has its maximum over the centro-parietal scalp suggesting that the auditory attention networks are not yet fully developed at this age [9].

The P3a to distracting sounds is followed by a reorienting negativity (RON) component in adult ERPs [19] and the Late Negativity (LN) in children [9]. RON and LN have maximum amplitudes over fronto-central scalp areas at 400–700 ms after onset of a distracting sound and may be associated with the prefrontal brain mechanisms involved in reorienting attention back to task performance after a distracting event [9,19].

Previous studies found no differences between ADHD and control children in P3a responses to novel visual stimuli [10,11]. However, Kemner et al.’s [11] data suggest smaller P3a responses over the parietal scalp to task-irrelevant novel sounds in ADHD children than in healthy controls, although this effect did not reach statistical significance. In contrast, Kilpeläinen et al. [12] found enhanced frontal P3 deflections (presumably the P3a) in distractible children in comparison with non-distractible children in response to infrequent target tones occurring among frequent tones. In the present study, we investigated these issues further by recording ERPs to repeating tones and novel sounds from ADHD children and control children in P3a responses to novel visual stimuli. The children were instructed to ignore the sounds, to focus their gaze on a white fixation cross (1 cm × 1 cm) continuously presented at the center of the screen and another button with their left thumb to a non-animal image. The children’s motivation to perform the task was indicated by their genuine interest to their hit rate reported to them after each block.

The electroencephalogram (EEG; 0.1–100 Hz, sampling rate 250 Hz) was recorded from frontal (Fp1, Fp2, F7, F3, Fz, F4, and F8), central (C3, Cz, C4), temporal (T3, T4, T5, T6), parietal (P3, Pz, P4), and occipital (O1, O2) scalp sites and from the left and right mastoids (LM and RM, respectively). Voltage changes caused by eye movements and blinks were monitored with recordings from the forehead sites (Fp1, Fp2) and from additional electrodes placed at the left and right canthi. The common reference electrode was placed at the tip of the nose. ERPs were obtained separately for the tones and novel sounds by averaging EEG epochs over a 900 ms pe-
period starting 100 ms before each sound onset. These EEG epochs were digitally band-pass filtered at 1–30 Hz. Epochs with extracerebral artifacts exceeding ±100 μV at any electrode were excluded from averaging. Also, the epochs for the first four stimuli of each block and the epochs for any tone occurring right after a novel sound were excluded. For each child, averaged ERPs to tones and novel sounds consisted of at least 380 and 90 acceptable EEG epochs, respectively.

Because the children were instructed to perform the visual discrimination task with an “accuracy instruction” (stress on performance accuracy, not on speed), cf. [17]), changes in the rates of correct responses (“hits”) and incorrect responses (wrong button presses and response omissions) caused by the task-irrelevant novel sounds were used as major behavioral indexes distracting.

A correct button press given 200–1500 ms after visual stimulus onset was classified as a hit. Within-group difference of means rates of hits, wrong and missed responses, and hit re-action times (RTs) were analyzed with t-tests, while between-group difference of those were analyzed with Mann–Whitney U-test (dependent variables: tone versus novel).

The P3a and LN amplitudes were measured from ERP difference waves obtained by subtracting ERPs to tones from those to novel sounds [6,9]. Their amplitudes were measured as mean voltages over fixed latency windows determined after visual inspection of the grand-average ERP difference waves at Fz and Cz electrodes. The mean amplitudes of the eP3a and IP3a were measured over 180–250 and 300–350 ms from sound onset, respectively, in relation to the mean difference-wave amplitude during the 100 ms prestimulus baseline. The LN amplitudes, in turn, were measured over two latency windows: 420–490 and 490–560 ms. Between-group differences in the P3a and LN amplitudes were examined with ANOVAs and Newman–Keuls post hoc tests. Greenhouse–Geisser corrections were used for the F-values when appropriate. ANOVAs for the eP3a and IP3a, included electrode factors Frontality (frontal F3, Fz, F4 versus central C3, Cz, C4 versus parietal P3, Pz, P4 electrodes) and Laterality (left-hemisphere F3, C3, P3 versus midline Fz, Cz, Pz versus right-hemisphere F4, C4, P4 electrodes).

Table 1 shows mean RTs, and the mean rates of hits, wrong responses, and response omissions in the visual task for the ADHD and control groups. In both groups, the distraction effect in the visual task caused by novel sounds was significant: An occurrence of a novel sound decreased the number of correct responses to the following visual stimulus, in comparison with visual stimuli preceded by a tone (e.g., the F3 electrode), this effect might be caused by an extracerebral artifact exceeding ±100 μV at any electrode. A Mann–Whitney U-test for the rates of missed responses indicated that an occurrence of a novel sound (in comparison with an occurrence of a tone) enhanced significantly more the number of response omissions in the ADHD children than in the controls (U(17) = 2.49; P < 0.01), this effect of the preceding sound on the number of omissions being 4% in the ADHD group but only 1% in the control group (see Table 1). No significant group difference or effect of preceding stimulus was found for the number of wrong responses.

Fig. 1 displays grand-average ERPs elicited by the auditory-visual stimulus pairs. In the ADHD and control groups, tones elicited only a small auditory N1 component (peak around 100 ms from sound onset at the Cz electrode), presumably due to a relative immaturity of the auditory cortex [18]. A P3a response was elicited by novel sounds in each age group (Fig. 1).

Table 1

<table>
<thead>
<tr>
<th>Performance</th>
<th>Stimulus</th>
<th>ADHD mean (S.D.)</th>
<th>Controls mean (S.D.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RT (ms)</td>
<td>Tone</td>
<td>476 (78)</td>
<td>498 (122)</td>
</tr>
<tr>
<td></td>
<td>Novel</td>
<td>488 (88)</td>
<td>533 (142)</td>
</tr>
<tr>
<td>Hit rate (%)</td>
<td>Tone</td>
<td>65 (13)</td>
<td>81 (8)</td>
</tr>
<tr>
<td></td>
<td>Novel</td>
<td>62 (12)</td>
<td>78 (10)</td>
</tr>
<tr>
<td>Wrong-response (%)</td>
<td>Tone</td>
<td>13 (5)</td>
<td>6 (3)</td>
</tr>
<tr>
<td></td>
<td>Novel</td>
<td>12 (6)</td>
<td>9 (3)</td>
</tr>
<tr>
<td>Miss-response (%)</td>
<td>Tone</td>
<td>22 (9)</td>
<td>12 (10)</td>
</tr>
<tr>
<td></td>
<td>Novel</td>
<td>26 (9)</td>
<td>13 (10)</td>
</tr>
</tbody>
</table>

A correct button press given 200–1500 ms after visual stimulus onset was classified as a hit. Within-group difference of means rates of hits, wrong and missed responses, and hit re-action times (RTs) were analyzed with t-tests, while between-group difference of those were analyzed with Mann–Whitney U-test (dependent variables: tone versus novel).

A correct button press given 200–1500 ms after visual stimulus onset was classified as a hit. Within-group difference of means rates of hits, wrong and missed responses, and hit re-action times (RTs) were analyzed with t-tests, while between-group difference of those were analyzed with Mann–Whitney U-test (dependent variables: tone versus novel).
Fig. 1. Grand-average ERPs elicited by tones (solid line) and novel sounds (dashed line) at selected electrodes in the ADHD and control children. Note that the auditory ERPs are followed and partially overlapped by the ERPs to the subsequent visual stimulus (visual-stimulus onset indicated by vertical dashed line).

Fig. 2. Grand-average ERP difference waves obtained by subtracting the ERPs to tones from those to novel sounds. The P3a elicited by novel sounds consists of two phases: the eP3a and lP3a, which are followed by the LN.

An ANOVA for the lP3a revealed a significant Group × Frontality × Laterality interaction (F(4,72) = 2.56; P < 0.04). According to the subsequent Newman–Keuls tests, the ADHD children had significantly larger lP3a amplitudes at the left and midline parietal scalp sites (4.8 versus 3 μV at P3; 6.7 versus 4.6 μV at Pz; P < 0.01). This effect is seen in Figs. 2 and 3a.

Following the P3a, a frontal LN was observed in both groups (Figs. 2 and 3a). The LN appeared to be smaller in amplitude and to peak earlier in the ADHD group than in the controls. Therefore, the LN amplitudes were measured at frontal electrode sites (F8, F4, Fz, F3, F7) as mean voltages over two time windows, i.e., over 420–490 and 490–560 ms from sound onset. The earlier of these windows covers the peak latency of the LN in the grand-average ERP difference waves of the ADHD group and the later window covers the peak latency of the LN of the control group. An ANOVA for these LN amplitudes including factors Group, Laterality (F8 versus F4 versus Fz versus F3 versus F7) and Time Window (420–490 ms versus 490–560 ms) revealed a significant Group × Laterality × Time Window interaction (F(4,72) = 6.18; P < 0.002) caused by the fact that over the frontal midline, the LN was larger in amplitude in the ADHD group than in the controls during the earlier time window (mean amplitudes −2.8 μV versus −1.2 μV, respectively; Newman–Keuls test: P < 0.001) but smaller in the ADHD group than in the controls during the later time window (mean amplitude 0.01 μV versus −1.7 μV, respectively; P < 0.001).
In conclusion, the present performance data are in accordance with our previous studies [9] by showing that novel sounds distract children’s performance in a visual discrimination task as indicated by decreased hit rates and increased RTs after an occurrence of a novel sound in comparison with occurrence of a repeating tone. Moreover, larger number of response omissions after a novel sound in the ADHD group than in the control group revealed higher distractibility in the ADHD group.

Abnormal involuntary attention in the ADHD children was also supported by the ERPs to the distracting novel sounds. These sounds elicited a biphasic P3a response with an early phase (eP3a) that had significantly smaller amplitude over the left-hemisphere in the ADHD children than in the control group. Thus, reduced eP3a amplitudes in the ADHD were presumably caused by a left-hemisphere dominant negative ERP component evoked by the novel sounds in the ADHD group but not in the control group or evoked with much smaller amplitudes in the latter group. Alternatively, the abnormal left-hemisphere activity in the ADHD children around 240 ms from novel-sound onset might be caused by a genuine reduction in the activity of one of the left-hemisphere generator sources contributing to the P3a response at the latencies between the eP3a and IP3a peaks (cf. [25]). For example, this reduction might be associated with a deficit in controlling verbalized encoding of these distracting environmental sounds that may be based on automatic stimulus identification and classification [6]. Moreover, the IP3a showed enhanced amplitudes in the ADHD group in comparison with the controls in parietal electrode sites especially over the left-hemisphere. These findings are in agreement with previous findings showing differences between ADHD and controls at left-hemisphere sites to auditory stimulus changes in ADHD patients compared with control subjects [16].

Interestingly, Gomot et al. [8] reported in 5–9-year-old autistic children concentrating on watching a silent movie atypical activity elicited by frequency changes in a repeating sound. This activity occurred at the latency of the mismatch negativity (MMN) and was localized with scalp current density analysis to the left frontal cortex. Their study together with the present results suggests left-hemisphere abnormality that may underlie various attentional problems in children.

Finally, in the present study, the IP3a was followed by the LN component that had a smaller amplitude and shorter latency in the ADHD children than in the controls. In healthy children, the LN has been interpreted to correspond the reorienting negativity (RON) observed in adults and proposed to be generated by prefrontal mechanisms involved in reorienting of attention back to the distracted task performance [3,9,19,24]. Therefore, the reduced LN in the ADHD chil-
children might be associated with a prefrontal cortical dysfunction [4] leading to problems in orienting attention back to the distracted task performance. This problem in reorienting attention is indicated in the present study by enhanced number of omitted responses in the visual task after a novel sound. The decrement in the LN peak latency observed in the ADHD children in relation to the controls might, in turn, be associated with a higher degree of impulsivity, which is a symptom of ADHD [2,15].

In summary, the present study suggests that ERPs recorded in the task-irrelevant novel stimuli distracting the children’s performance provide us with evidence supporting the observations of attentional and behavioral deficits in ADHD. Although, the present study should be considered as preliminary, given the relatively small size of the two groups, the present results suggest that a multimethodological approach combining neuropsychological, behavioral and electrophysiological measures of distractibility and involuntary orienting of attention improve our understanding of the brain-behavior relationship in ADHD.

Acknowledgements

The authors thank Ms. Kirsi Jarvinen for her help in recruiting the control children, as well as Ms. Leena Wallendahr and Mr. Timo Saarinen for assistance with the electrophysiological recording. V. Gumenyuk was supported by the Academy of Finland grant no. 55606. R. Näätänen and V. Gumenyuk were supported by the Academy of Finland grants nos. 77322 and 102516. C. Escera was supported by the grant no. P599-0167 of the Spanish Ministry of Science and Technology.

References