Attentional capacity, a probe ERP study: Differences between children with attention-deficit hyperactivity disorder and normal control children and effects of methylphenidate


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Abstract

In the present study it was investigated whether the smaller P3s in attention-deficit hyperactivity disorder (ADHD) children are caused by a shortage of capacity underlying P3 processes or whether they are due to a capacity allocation problem. Also, effects of methylphenidate on these processes were investigated. Performance and event-related potentials (ERPs) of 14 ADHD and 14 control children were measured using an irrelevant-probe technique. Three types of task irrelevant visual probes—standards, deviants, and novels—were presented against the background of two visual tasks that varied in task difficulty. The parietal P3 wave was measured in response to task stimuli and probes. ADHD subjects made significantly fewer correct detections than normal controls in both the easy and the hard tasks. Controls showed an enhanced P3 to task-relevant stimuli in the hard task, whereas ADHD children did not. Probe P3 amplitudes decreased from the easy to the hard task to the same extent in both groups. Methylphenidate enhanced the percentage of correct responses and task P3 amplitudes in both the easy and the hard task but probe P3 amplitudes were not influenced by methylphenidate. It was concluded that ADHD children do not suffer from a shortage in attentional capacity; rather, the evidence is in favor of a problem with capacity allocation. Furthermore, methylphenidate had enhancing effects on performance and ERPs, but did not improve the capacity-allocation deficit.

Descriptors: Attention-deficit hyperactivity disorder, Attentional capacity, Probe task, Event-related potentials, P3, Methylphenidate

Extensive research has shown that children with attention-deficit hyperactivity disorder (ADHD) perform worse than normal control children on a wide range of attentional tasks (see Douglas, 1983, for a review). In addition, the attentional deficits of ADHD children are displayed in the amplitudes of event-related brain potentials (ERPs). The most frequently reported finding in ERP research with ADHD children is that, compared with normal control children, they show smaller P3 amplitudes to target stimuli. In most of these ERP studies so-called oddball tasks were used. In these tasks a series of stimuli is presented and subjects have to detect infrequently presented stimuli that deviate in some aspect from the more frequently presented standard stimuli. In oddball tasks, the P3 is thought to reflect activity related to the evaluation of target stimuli; for a recent review of the processing manifested in the P3 see Polich and Kok (1995). Additionally, research on human adults has led to the assumption that in so-called dual tasks, the amplitude of the P3 also reflects the amount of mental capacity that is invested in a task (for a review see Kok, 1997). In light of this capacity hypothesis regarding the P3 amplitude, an important question that arises is whether the often-reported smaller P3s in ADHD children are related to a deficit in attentional capacity. The present investigation was aimed at providing an answer to this question. For an understanding of the rationale behind the P3 as a measure of processing capacity, first an overview of the literature on this subject is provided below.

The concept of the P3 amplitude as a measure of processing capacity is supported by the results of dual-task studies. In these studies, a primary and a secondary task have to be performed at the same time, with the subject instructed to keep performance of the primary task at an optimal level. It is assumed that when the primary task is made more difficult, more processing capacity has to be invested in this task, and therefore less capacity will be available for processing of the secondary task. It has indeed been found that the reduction of processing resources available for the processing of secondary task stimuli is usually reflected by smaller P3 amplitudes to these stimuli (Isreal, Wickens, & Chesney, 1980; Wickens, Kramer, Vanasse, & Donchin, 1983). In contrast, the
investment of more capacity in the primary task when it gets more difficult is reflected by a P3 increment (Wickens et al., 1983). Although the finding of an increase in P3 amplitude with increasing task demands (and thus higher capacity investment) is not debated in dual-task paradigms, in some other studies (for reviews, see Kok & Looren de Jong, 1980; Polich, 1987) P3 amplitudes have been found to decrease with increasing task load. An explanation for these different findings was given by Okita, Wijers, Mulder, and Mulder (1985) and Wijers, Otten, Feenstra, Mulder, and Mulder (1989), who concluded that the decrease in P3 was found only in visual search tasks and was caused by an overlapping negativity induced by memory search processes that were specifically required in these tasks.

Another technique that can be used for studying capacity trade-off between easy and hard tasks is the so-called irrelevant-probe technique. In such studies, task-irrelevant stimuli (probes) are presented against the background of an ongoing task that can be varied in task difficulty. Task-P3 and probe-P3 amplitudes can be compared across easy and hard tasks to study capacity trade-off effects in the same way as in dual tasks. An important advantage of the probe method is that, in contrast to in dual tasks, the task-irrelevant probe stimuli do not interfere with the processing of the task stimuli because they are attended only passively. This scheme is especially advantageous for capacity research with children, in particular in clinical populations such as those with ADHD.

Several authors have used the irrelevant-probe method to study task load effects in normal adult volunteers on different early and late ERP waves in response to probes (Kenemans, Verbaten, Melis, & Slangen, 1992; Kramer, Trejo, & Humphrey, 1995; Verbaten, Huyben, & Kemner, 1997). As will be discussed below, the results from these studies are inconsistent. Kenemans et al. (1992) presented task-irrelevant visual probes to subjects during hard and easy versions of an active decision task, and measured probe amplitudes as a function of task load. Besides standard probes, infrequently occurring deviants were presented to induce an automatic deviant/standard discrimination (visible in the P3) that is indicative of stimulus evaluation processes. Such passive discrimination processes have been demonstrated in several other ERP studies using a passive “oddball” paradigm (McCullum, Barrett, & Po-cock, 1989; Polich, 1987, 1989; Roth, 1983). The results from the Kenemans et al. (1992) study showed that although N2-P2 amplitudes were higher to deviant than to standard probes, there was no interaction with the load manipulation. Probe-P3 amplitudes were higher to deviants than standards in the easy task but not in the hard task. Kenemans et al. concluded that stimulus evaluation/classification processes (as reflected by P3 amplitude) to irrelevant probe stimuli occurred in the easy task but could not be executed in the hard task because of a shortage of processing capacity. Accordingly, Kenemans et al. concluded that the P2-N2 and P3 reflect different stages of the attention orienting reaction to deviant irrelevant probes in which only the latter (P3) process is capacity limited. In contrast with these findings, in an auditory probe paradigm, Kramer et al. (1995) did not find task load effects on the probe-P3 but did report effects on the probe-N1 and N2. The differences in P3 findings between the two probe studies might be explained by modality-specific effects. In the Kenemans et al. (1992) study, visual probes were used, whereas Kramer et al. (1995) used auditory probes. There is some evidence from a study by Holdstock and Rugg (1995) that in the auditory modality the difference in pitch between passively attended standard and deviant stimuli plays an important role in eliciting a P3. In their task, standard, deviant, and highly deviant novel sounds were presented to subjects in a condition in which the stimuli were passively attended or in a condition in which the stimuli were ignored while the subjects were engaged in another task. The novel P3 had a higher amplitude when the stimuli were passively attended than when they were ignored, but a P3 to deviants was not found in either condition. Holdstock and Rugg concluded that, whereas the more salient novel sound always elicits a P3, the difference in pitch between standards and deviants might be critical for the elicitation of a passive P3 to deviants. In light of these findings, the absence of P3 amplitude differences between auditory standard and deviant probes in the Kramer et al. (1995) study might be explained by a too small pitch difference between standard and deviant tones. When there is no P3 to deviant probes no load effects can be found.

Although in the Kenemans et al. (1992) study a P3 response to deviant irrelevant visual probes was found, results from a study by Verbaten et al. (1997) showed that the “saliency” of probes may also play a role in the elicitation of a P3 in the visual modality. In this study, the same visual probe task as in the Kenemans et al. (1992) study was used but besides the standard and deviant probes, highly deviant (novel) probes were also presented; the strongest P3 load effects were found for the highly deviant probes (amplitudes decreased with increasing task load). The irrelevant-probe method seems suitable to measure capacity trade-off between easy and hard tasks when the probes are salient enough to attract attention and allow for P3 processing. Thus, in the present study capacity processes were investigated in ADHD and normal control children using the irrelevant-probe technique.

Until now no ERP studies have been performed to study capacity processes in ADHD children using a dual-task or the irrelevant-probe technique. There are, however, two behavioral studies that are important to mention in this respect. Schachar and Logan (1990) performed a dual-task study to investigate attentional capacity in three groups of 6- to 12-year-old boys: attention deficit disorder plus hyperactivity (ADDH), ADDH plus conduct disorder, and normal children. Their study used a primary-choice reaction-time task under two conditions: one with and one without a secondary task that also required a response. In the condition that included a secondary task, there were several time delays between primary and secondary task stimuli that influenced their overlap. The assumption was that more capacity would be required with increasing overlap of the tasks. The hypothesis was that if ADDH subjects are deficient in attentional capacity, when compared with normal controls, their reaction times (RTs) to the secondary task stimuli would increase more with increasing temporal overlap of the primary and secondary task stimuli. This hypothesis was not supported by the data. However, the RTs of ADDH subjects on the primary task increased more after introduction of the secondary task than did those of normal controls. Schachar and Logan concluded that the latter finding was an indication of a different allocation policy in ADDH children but that there was no capacity shortage because the increase in primary task RT after introduction of the secondary task was not influenced by the overlap of primary and secondary task stimuli.

Sergeant and Scholten (1985) investigated hyperactive children in a Sternberg memory comparison paradigm under three different instruction sets (speed, accuracy, and normal). Whereas the normal control subjects traded accuracy for speed in the speed set (more errors, shorter RTs), hyperactive subjects did not (more errors but also longer RTs). According to Sergeant and Scholten, this finding suggested a deficit in resource allocation strategy in hyperactive subjects. Thus, the only two studies that investigated capacity processes in ADHD children, albeit not with ERPs, concluded that the
performance problems that are encountered by ADHD children are due to inefficient allocation of capacity.

In the present study, for the purpose of studying attentional capacity in ADHD children, during a primary task differing in load, irrelevant novel, deviant, and standard probes were presented to ADHD and normal control children. In normal children larger task-P3 and smaller probe-P3 amplitudes were expected in the hard condition. Load effects on the probe-P3 were expected to be stronger to the more salient novel probes. For ADHD children, specific predictions about performance and ERP effects can be made in case of a capacity shortage deficit. If ADHD children have less attentional capacity than normal subjects, they should (1) show a larger performance decrement (more errors and/or longer RTs) than control subjects with increasing task difficulty, and (2) show a smaller (novel) probe P3 than control subjects (at least in the hard task). If these predictions are not confirmed by the results, the problems of ADHD children (if present in this experiment) might be attributed to allocation problems, for which no specific results can be predicted because there are multiple possibilities.

Furthermore, earlier ERP waves (P1, N1, P2, and NC) were also measured, because there is a possibility either that deficits at the P3 level are already (also) manifest in earlier waves (Kramer et al., 1995), or that there is no capacity deficit at the P3 level in ADHD children but deficits in processes represented by earlier ERP waves. In a study by Jonkman et al. (1997a), in which a selective attention task was presented to ADHD and control subjects, some support was found for the latter possibility because no deficits in stimulus processing were found at the P3 level, but deficits were apparent in earlier ERP peaks.

The first experiment in the present study was followed by a second experiment in which, in the same ADHD children, the effects of the stimulant drug methylphenidate on possible capacity problems that are revealed in the present experiment were investigated.

**EXPERIMENT 1**

**Method**

**Subjects**

The total sample consisted of 28 children, 7–13 years old. The control and clinical groups consisted of 14 subjects each (mean age 10.1 and 9.6 years, respectively). The 14 clinical subjects were all recruited from the Department of Child Psychiatry, Academic Hospital Utrecht, where they received treatment by child psychiatrists for behavioral and/or academic problems. Controls were recruited from two different local elementary schools and were screened on the basis of ratings on the Conners Teachers Rating Scale (Conners, 1985) and parents ratings on the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983).

All subjects were administered the Wechsler Intelligence Scale for Children Revised (WISC-RN), Dutch version. All children had full-scale IQ scores higher than 80. Furthermore, to check whether group means were different on hyperactivity scales, all parents filled out a CBCL (see above), and all teachers filled out the Conners Teachers Rating Scale.

The main inclusion criterion for the clinical group was a clinical rating of ADHD made by psychiatrists using DSM-III-R guidelines. Controls were required to have: (a) Conners teacher scores of < 15 on the abbreviated hyperactivity scale (scores ranged from 5 to 10, mean = 6.8 [SD 1.7]) and (b) parent CBCL ratings below the clinical range (T-score < 70) on the “attention problems” scale (scores ranged from 50 to 67, mean T-score = 53 [SD 5.1]). Of the 14 ADHD subjects, 6 did not have a Conners score > 15 and 5 scored below the clinical range on the CBCL. In this last group, scores on the Conners scale ranged from 8 to 20 (mean = 14.5 [SD 3.5]), on the CBCL scores ranged from 67 to 81 (mean T-score = 72 [SD 4.0]). Mean Conners, F(1,26) = 53.9, p < .0001, and mean CBCL scores, F(1,26) = 118, p < .0001, were significantly different between the groups. All subjects had normal or corrected-to-normal vision and no color blindness. All the control children were drug free, that is, they did not use any medication. Clinical subjects who were normally on medication stopped using it at least 3 days before each test day. Mean data and SDs of each group with respect to age, sex, and full-scale IQ are reported in Table 1. The groups did not differ in age but the full-scale IQ score was significantly different between the groups, t = 2.2, p < .05.

**Electroencephalographic (EEG) and Electrooculographic (EOG) Recordings**

EEG activity was recorded from tin electrodes by means of an electrocap. Activity was measured from the midline scalp electrodes, Fz, Cz, Pz, and Oz. Linked ear lobe electrodes, each connected with a 15-kΩ resistor, were used as reference. Horizontal EOG was recorded using tin electrodes attached to the outer canthus of each eye by means of adhesive rings. Similarly, vertical EOG was recorded from infraorbital and supraorbital electrodes placed in line with the pupil of the left eye. A ground electrode was attached to the middle of the forehead. For both EEG and EOG, electrode paste was used. Electrode impedance was kept below 10 kΩ. All EEG signals were amplified with a time constant of 10 s. EOG signals were amplified with a time constant of 36 s. All signals were filtered (40 Hz, 24 dB/oct low-pass) online and digitized by a computer at a rate of 256 Hz. Sampling was continuous. Signals were epoched offline with a window from 0.1 s before to 1.2 s after stimulus onset.

**Tasks**

During each test session a total of five experimental blocks was presented. Three of these tasks involved the Eriksen “flanker” paradigm and were used to study response-related processes in ADHD children; these data will be reported in another paper. The other two tasks were an easy and a hard decision task. In these tasks the level of difficulty was manipulated while probes (which were not different between the two task conditions) were presented. The order of presentation was balanced across subjects.

The duration of the decision tasks was about 10 min, stimulus duration was 0.3 s, and interstimulus intervals (ISIs) between primary task stimuli were randomized between 3.7 and 4.9 s. The tasks consisted of 140 stimuli, which were purple, red, green, and...
blue rectangles subtending a height of 5.3° of arc and a width of 4.5° of arc. Stimuli displayed on a monitor positioned approximately 70 cm from the subject’s eyes were presented in the center of the subject’s visual field. In the easy task, the instruction was to press the right-hand button whenever a blue rectangle was detected and to press the left-hand button to all rectangles of another color. In this condition there were 71 blue stimuli and 69 non-blue stimuli (23 purple, 23 red, 23 green) to ensure that as many left- as right-hand button presses were required. In the hard task, there were 35 stimuli of each color. In this condition, the subject’s task was to compare each rectangle that appeared on the screen with the preceding rectangle. When a rectangle was the same color as the preceding rectangle, subjects were instructed to press the right button. When two successive rectangles were of different colors, subjects had to press the left button. To ensure that as many right-as left-hand button presses were given, the probability that two successive stimuli had the same color (or different colors) was 50%. It is important to emphasize that in both the easy and the hard task each task stimulus was task relevant, for each stimulus required either a left or a right button press. The hard task could be properly executed only if the subject kept a running memory of the presented stimuli.

The probes consisted of 140 stimuli, which can be divided into three stimulus types. Standards (60%) consisted of a white abstract figure (resembling the shape of a left bracket, [ ] subtending a height of 3.3° of arc and a width of 1.6° of arc. Deviants (20%) were exactly the same figures as standards, only rotated to 180°. Novels (20%) were composed of 22 different abstract (8 of the 22 novels were presented twice to come to a total of 30 novels), multicolored patterns, subtending a height of approximately 15.5° of arc and a width of approximately 17.5° of arc (the same series of novels was used in both hard and easy tasks). A probe stimulus, with a duration of 0.3 s, was always presented after a task stimulus, with the ISI varying randomly between 1.7 and 2.3 s. The subjects were told that in between the task stimuli, other figures would be presented regularly but that they should not respond to them.

**Procedure**

On arrival, the child (accompanied by one of the parents) was familiarized with the procedure. During electrode attachment subjects watched a video movie. To motivate the children, they were told that they could choose a toy after the test if they performed well enough. After attachment of the electrode cap and EEG electrodes, the child sat down in a dentist’s chair in an acoustically and electrically shielded room. The chair was adjustable so that the child’s head could be positioned roughly parallel to a TV monitor, which was positioned above and in front of the child. A vacuum cushion attached to the top of the chair allowed fixing the child’s head in such a way that the child looked at the center of the screen and head movements were prevented. Next, the test session was started with the child receiving instructions for the first task. Instructions for the tasks were given by showing stimuli on the screen and pointing out which button to press until it was clear that the child understood the task. After the instruction, subjects had to perform a practice task (a short version of the real task containing 24 practice trials) of about 2 min, until a criterion of 70% correct responses was reached. Due to time contrictions the maximum amount of practice sessions was two. Four ADHD and two control subjects did not meet the 70% correct criterion in the hard task after two practice sessions; these subjects were not excluded from the study as will be discussed below. After the practice session, the child was instructed to move as little as possible during the task, and was also made aware of the presence of an intercom. Then, the experimenter and the parent left the room, the door was closed, and the task was started. Throughout the task the EEG was monitored on the screen by the experimenter. After each task, the experimenter entered the room and gave instructions for the next task.

**Signal Analysis**

All of the sampled EEG and EOG epochs were baseline corrected and filtered offline with a 30-Hz, 24-dB/oct digital low-pass filter. Then the EEG was corrected for vertical EOG artifacts by subtracting vertical EOG from EEG epochs using a regression method in the time domain (Kenemans, Molenaar, Verbaten, & Slangen, 1991). Finally, all EEG epochs containing artifacts (saturation of the A/D converter or an amplitude greater than −100 or 100 μV) were removed from the database.

ERPs were computed separately for task and probe stimuli. Task ERPs were computed by averaging only trials in which a correct response to a task stimulus was given (hit P3). Probe ERPs were computed by averaging only trials in which, in agreement with task instructions, no response was given. In this procedure, trials with differently colored task stimuli were averaged, because no effects of color on the ERPs were expected. With regard to the probe stimuli (to which no response had to be given), ERPs were computed for standards, deviants, and novels measured at each lead. Subsequently, P1, N1, P2, and NC peaks were scored in the averaged ERPs by measuring the highest amplitude in a predetermined time window relative to a 100-ms prestimulus (baseline) level, based on inspection of the grand-average waveforms. The NC is a broad, late frontal negativity that occurs in children (see Courchesne, Elmasian, & Yeung-Courchesne, 1987). Peaks were scored in the following windows: P1: 50–160 ms, N1: 50–170 ms, P2: 150–280 ms, NC: 250–500 ms. For the P3, area measurements under the curve were computed in a 400–800-ms window. Peak scoring was not used for the P3 because for children there is a large variability in the time of occurrence of the P3 and this wave is often composed of more components that will not be covered by scoring one peak.

**Statistical Analysis**

Although clinical and control groups differed in full-scale IQ, covariance analyses did not reveal any effects of IQ and therefore IQ was not included in further analyses.

To test the expectations discussed above, repeated-measures multivariate analyses of variance (MANOVAs) were performed for the performance measures (hit percentage and RT). In these tests there was one between-subjects factor of group, including ADHD and control children, and one within-subjects factor of load, including hard and easy tasks. The MANOVA directed at the Pz P3 induced by task and probe stimuli included the same factors with the addition, in the case of probes, of the within-subjects factor stimulus type, including standards, deviants, and novels. To test for unpredicted effects on ERP waves preceding the P3 (P1, N1, P2, and NC), separate MANOVAs were performed for task stimuli and probes consisting of the above factors, including an additional within-subjects factor leads (Fz, Cz, Pz, and Oz). Significance levels in all MANOVAs were set at 5%, two-tailed.

**Results**

**Performance**

Measures of performance were percentage of hits and mean RT to hits. A response was considered a hit when it was given within a
window of 150–1,500 ms after stimulus presentation. Means and SDs for each group are presented in Table 2. MANOVA showed that for hits, a significant group effect, $F(1,25) = 8.67, p < .01$, indicated that ADHD subjects had fewer hits than control subjects in both the hard and the easy tasks. RTs were not significantly different between the two groups. Furthermore, a significant load effect was found for both hits, $F(1,26) = 47.07, p < .0001$, and RT, $F(1,26) = 61.90, p < .0001$; for both groups there were more errors and slower RTs in the hard versus the easy task.

Finally, for percentage of hits, only a trend toward a Group × Task interaction was found, $F(1,26) = 2.43, p < .10$. Although ADHD children had lower hit percentages than controls in both easy and hard tasks, the interaction indicates that the performance of ADHD children deteriorated slightly more with increasing task difficulty than that of normal controls.

### P3 Analyses at the Pz Lead

**Task stimuli.** Mean P3 area amplitudes for task stimuli at Pz are presented in Table 3. In the multivariate P3 analysis for task stimuli at Pz, significant load, $F(1,26) = 9.69, p < .005$, and Group × Load, $F(1,26) = 4.21, p < .05$, effects were found. As shown in Figure 1, in the control group there was an increase in P3 amplitude from the easy to the hard condition (10.8 and 14.7 μV, respectively), whereas in the ADHD group, amplitudes did not differ between the two conditions (10.0 μV in the easy task vs. 10.9 μV in the hard task).

**Probe stimuli.** The multivariate analysis for probe stimuli at Pz revealed only a near-significant Load × Stimulus type effect, $F(2,25) = 3.21, p = .06$. Further univariate testing indicated that, as expected, novel amplitudes decreased from the easy to the hard task (5.6 vs. 3.0 μV), whereas this effect was not found for deviants and standards (see Figure 2). Although it seems that there also was a load effect regarding deviants and standards, indicating an

### Table 2. Performance Data for the Easy and Hard Task in the Control and ADHD Group and in Placebo MPH Conditions

<table>
<thead>
<tr>
<th>Task</th>
<th>Hits %</th>
<th>Reaction time (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Easy</td>
<td>Controls</td>
<td>89.7 (9.4)</td>
</tr>
<tr>
<td>ADHD</td>
<td>82.1 (8.1)</td>
<td>775 (169)</td>
</tr>
<tr>
<td>Placebo</td>
<td>76.72 (12.3)</td>
<td>841 (179)</td>
</tr>
<tr>
<td>MPH</td>
<td>86.46 (6.1)</td>
<td>753 (167)</td>
</tr>
<tr>
<td>Hard</td>
<td>Controls</td>
<td>74.0 (17.2)</td>
</tr>
<tr>
<td>ADHD</td>
<td>58.0 (18.6)</td>
<td>898 (193)</td>
</tr>
<tr>
<td>Placebo</td>
<td>63.21 (16.1)</td>
<td>895 (209)</td>
</tr>
<tr>
<td>MPH</td>
<td>76.09 (11.2)</td>
<td>811 (269)</td>
</tr>
</tbody>
</table>

*Note: ADHD = attention-deficit hyperactivity disorder, MPH = methylphenidate.*

### Table 3. Mean P3 Area Amplitudes in μV (400–800 ms) to Task Stimuli (Hits) in ADHD and Control Groups and in Placebo and Methylphenidate Conditions at the Four Midline Electrodes in Easy and Hard Tasks

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Easy condition</th>
<th>Hard condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fz</td>
<td>Cz</td>
</tr>
<tr>
<td>Controls</td>
<td>−3.69</td>
<td>4.17</td>
</tr>
<tr>
<td>ADHD</td>
<td>−4.41</td>
<td>3.89</td>
</tr>
<tr>
<td>Placebo</td>
<td>−2.32</td>
<td>2.92</td>
</tr>
<tr>
<td>Methylphenidate</td>
<td>−1.96</td>
<td>6.07</td>
</tr>
</tbody>
</table>

*Note: SD in parentheses. ADHD = attention-deficit hyperactivity disorder.*
increase from the easy to the hard task, this effect was not significant, $F(1,27) = 1.62, p = .21$. No main or interaction effects involving the group factor were found.

**Overall ERP Analyses**

Mean P3 area amplitudes at the four midline leads for task stimuli and probe stimuli (novels, deviants, and standards) are presented in Tables 3 and 4, respectively. Mean P3 amplitudes in both groups at the four midline leads for task and novel probe stimuli are visualized in Figure 3. Grand-average ERPs of ADHD and control subjects for task stimuli and novel probes (in both task conditions) are depicted in Figure 4.

**Task stimuli.** No significant load or group effects nor interactions involving these factors were found for the P1 and P2 waves.

![Figure 2. Mean parietal probe amplitudes in easy and hard tasks for control and attention-deficit hyperactivity disorder (ADHD) subjects.](image)

**Table 4. Mean P3 Amplitudes (400–800 ms) to Probe Stimuli in ADHD and Control Groups and in ADHD Children in Placebo and Methylphenidate Conditions at the Midline Leads in Easy and Hard Tasks**

<table>
<thead>
<tr>
<th></th>
<th>Novels</th>
<th>Deviants</th>
<th>Standards</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fz</td>
<td>Cz</td>
<td>Pz</td>
</tr>
<tr>
<td>Easy Controls</td>
<td>0.24</td>
<td>1.89</td>
<td>3.46</td>
</tr>
<tr>
<td>ADHD</td>
<td>-1.50</td>
<td>2.33</td>
<td>2.57</td>
</tr>
<tr>
<td>Placebo</td>
<td>2.39</td>
<td>-1.04</td>
<td>0.21</td>
</tr>
<tr>
<td>Methylphenidate</td>
<td>-2.52</td>
<td>-1.04</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>(0.27)</td>
<td>(5.47)</td>
<td>(4.56)</td>
</tr>
</tbody>
</table>

*Note: SD in parentheses. ADHD = attention-deficit hyperactivity disorder.*
With regard to the \( N1 \), the multivariate analysis revealed a group effect, \( F(1,25) = 9.88, p < .01 \), showing that controls had larger \( N1 \)s than ADHD subjects. Further univariate testing of Lead \( \times \) Group interaction, \( F(3,24) = 5.39, p < .05 \), showed that this effect was significant at Fz and Cz leads.

With regard to the \( NC \), multivariate analysis revealed a near-significant Group \( \times \) Load interaction, \( F(1,13) = 4.06, p < .06 \). Univariate testing indicated that control subjects did display a load effect, \( F(1,13) = 8.58, p < .05 \), and displayed more negativity in the easy than in the hard task, whereas ADHD subjects did not.

Probes. Multivariate analysis including all probe stimuli did not reveal effects of the factors group, load, or interactions between these factors on the \( N1 \), \( P2 \), or \( NC \) in the overall analysis, except for the \( P1 \), for which a significant Load \( \times \) Stimulus type \( \times \) Group interaction, \( F(2,25) = 4.29, p < .05 \), was noted. When further tested per Stimulus type, a significant Group \( \times \) Load interaction was apparent for deviants, revealing that in the easy task controls displayed higher \( P1 \) amplitudes in response to probe deviants than did ADHD subjects, \( F(1,26) = 4.14, p < .05 \).

Summary and Discussion

The main findings of Experiment 1 were that, compared with control children, ADHD subjects showed (1) worse performance in both easy and hard tasks; (2) no load effect on the task \( P3 \); in contrast with the control group, no change in amplitude was found across easy and hard tasks; and (3) an equally large decrement in novel probe \( P3 \) amplitude from the easy to the hard task. Thus, both ADHD and control subjects appeared to have the same amount of extra capacity (= \( P3 \) amplitude in the easy task) at their disposal, but the ADHD children did not, or were not able to invest it in the task when task demands increased (no increase in \( P3 \) amplitude was found in the hard task in ADHD subjects). On the basis of these data, it is concluded that the \( P3 \) results were indicative of a deficiency in capacity allocation rather than of a capacity shortage in ADHD children.

Assuming that capacity-allocation deficits play an important role in the attention problems of ADHD, it is important to find out whether such deficits can be counteracted by pharmacological treatment. Methylphenidate is the most effective and most frequently prescribed drug in treatment of ADHD children. Although the stimulant drug has been found to ameliorate the performance and \( P3 \) deficits of ADHD children in oddball tasks (Coons, Klorman, & Borgstedt, 1987; Fitzpatrick, Klorman, Brumaghim, & Borgstedt, 1992; Jonkman et al., 1997b; Verbatan et al., 1994), the effects of the drug on processing capacity in ADHD are not clear. Therefore, a second experiment was executed in which the effect of methylphenidate on the allocation deficit displayed by ADHD children in Experiment 1 was studied.

**EXPERIMENT 2**

The stimulant drug methylphenidate has an ameliorating influence on the performance- and \( P3 \)-deficits shown by ADHD children.
Not much is known about the effects of methylphenidate on processing capacity in ADHD. Carlson, Pelham, Swanson, and Wagner (1991) used a dual task to study the effects of methylphenidate (0.3 mg/kg) on capacity allocation in ADHD children. Subjects had to solve arithmetic problems (primary task) while simultaneously making foot presses to tone stimuli (secondary task) presented at different intervals before or after processing the arithmetic problems. Carlson et al. found an increase in accuracy in the primary task after ingestion of the drug, but the drug had no effect on performance in the secondary task (RT to auditory stimuli). Carlson et al. concluded that methylphenidate administration resulted in reallocation of capacity from the secondary to the primary task.
Assuming that methylphenidate does indeed lead to an improvement in resource allocation in ADHD children, as suggested by Carlson et al. (1991), we expected to find an increase in task P3 amplitude (and performance), particularly in the hard task. In other words, an improvement in reallocation strategy in ADHD under methylphenidate should result in a P3 pattern much like that of normal children in the first study, that is, larger task P3 amplitudes in the hard than in the easy task and smaller P3 amplitudes to novel probes in the hard than in the easy task. If these effects of methylphenidate on P3 amplitudes are found, they should concur with an increase in performance in the hard task. P3 effects were studied only at the parietal lead because at this lead the largest amplitudes and effects are usually found.

Method

Subjects

The total ADHD sample consisted of 14 children, 7–13 years old, who also participated in Experiment 1. Mean ages and diagnostics of the subjects are described in the Method section of Experiment 1.

Procedure and Signal Analysis

The same children that participated in the ADHD-control study also participated in the following drug study. The drug study started 1 week after the initial session of Experiment 1 and consisted of two laboratory sessions 1 week apart that were procedurally identical, except for the pharmacological substance (placebo or 15 mg methylphenidate) contained in a capsule and consumed at the beginning of each session. Testing started 50 min after intake of the capsule. The experimental procedure was identical to that in Experiment 1, as was the signal analysis procedure. Drug assignment was counterbalanced between subjects. Subjects that were normally taking medication had a washout period of 3 days before each test day. Body weights were measured and the received doses varied between 0.36 and 0.78 mg/kg (the group mean was 0.49 mg/kg, SD 0.16 mg/kg). In the present study there was 1 ADHD subject who (only in the placebo condition) did not meet the criterion of 70% correct responses in the practice session in the hard task. This subject was included in the analysis.

Statistical Analysis

To test the predictions formulated in the introduction, separate repeated-measures MANOVAs were performed for performance results (percentage of hits and RT), the parietal P3 amplitude to task and probe stimuli, and for earlier ERP waves preceding the P3 (P1, N1, P2, and NC). In the case of the performance and task P3 analyses, the MANOVA consisted of two within-subjects factors: drug (including placebo and methylphenidate) and load (including hard and easy tasks). In the probe analyses one extra within-factor, stimulus type with three levels (novels, deviants, and standards), was included. In the overall analyses for the earlier ERP waves, performed separately for task and probe stimuli, the additional within-factor leads was included, consisting of Fz, Cz, Pz, and Oz leads. All significance levels were set at 5%, two-tailed.

Results

Performance

Two types of performance measures were calculated, percentage of hits and mean RT to hits. A response was considered a hit when it was given within a window of 150–1,500 ms after stimulus presentation. Means and SDs for each drug and task condition are presented in Table 2. The MANOVA showed a significant drug effect for percentage of hits, F(1,13) = 8.34, p < .05; in both easy and hard tasks, methylphenidate enhanced the percentage of correct responses. RTs were not significantly different between the placebo and methylphenidate conditions, although RT was shorter under methylphenidate. Furthermore, a significant load effect was found for both percentage of hits, F(1,13) = 39.06, p < .0001, and RT, F(1,13) = 19.88, p < .001; there were more errors and slower RTs in the hard condition compared with the easy condition. No Drug × Load interactions were found.

P3 Analysis at the Pz Lead

Task stimuli. Mean midline P3 amplitudes in response to task stimuli and probes are presented in Tables 3 and 4, respectively. Mean P3 amplitudes in placebo and methylphenidate conditions at the midline leads and for task stimuli and novel probes are shown in Figure 5.

In the multivariate P3 analysis regarding task-relevant stimuli, a significant drug effect, F(1,13) = 4.96, p < .05, was found. This effect indicated that P3 amplitudes were higher in the methylphenidate (10.1 μV) than in the placebo condition (6.7 μV). The expected Drug × Load interaction was not found.

Probe stimuli. In the multivariate probe P3 analysis, no significant drug, load, or stimulus type effects were found. Because in Experiment 1 the expected load effects were found only for novel probes, an additional multivariate analysis was performed on this variable by unidirectional comparisons. This analysis revealed a significant load effect, F(1,13) = 3.67, p < .04, indicating that novel P3 amplitudes decreased from the easy (2.9 μV) to the hard task (0.14 μV). No drug effects were found.

Overall ERP Analyses

For placebo and methylphenidate conditions, grand-average ERP waveforms in response to task stimuli and novel probes are shown in Figure 6.

Task stimuli. No effects of drug or load were found for the N1, P1, P2, and the NC.

Probes. No effects were found for the NC.

N1. Further testing of a Drug × Load × Stimulus type × Leads interaction, F(6,8) = 35.26, p < .000, led to the finding that in the placebo condition novel amplitudes at Oz were higher in the easy than in the hard task.

P1. A multivariate load effect, F(1,13) = 7.37, p < .05, showed that, averaged over all three probe types and independent of drug, amplitudes were higher in the hard than in the easy task.

P2. Multivariate analysis revealed a load effect, F(1,13) = 13.48, p < .01, showing higher P2 amplitudes during the easy than the hard task, for all three probe types and independent of drug.

GENERAL DISCUSSION

The purpose of the present study was to investigate the possible presence of deficits in processing capacity or in allocation of resources in ADHD subjects versus normal control children. To this end, a so-called irrelevant probe technique was used. Task-irrelevant probe stimuli were presented against the background of two tasks that varied in difficulty. Earlier research (Kenemans et al., 1992; Verbaten et al., 1997) has shown that in such a paradigm the probe P3 can be used as an indirect measure of attentional capacity.
because the processes by which the probe stimuli are evaluated appear to be dependent on the available amount of capacity and will not be executed in case of a shortage of capacity. For the control group in the present study, as task load increased, the parietal P3 to novels decreased (less available spare capacity) and the task P3 increased (which means that more capacity is invested in the task). This effect suggests that for processing of the novel probes, capacity is drawn from the same resource pool as for processing the task stimuli, and that there was less spare capacity available for probe processing when the task was more difficult.

The main predictions were that if ADHD subjects have less capacity at their disposal than normal control children, they would show a larger performance decrement from the easy to the hard task and that this decrement would be accompanied by smaller novel probe P3s in the hard task as compared to those of control subjects. But that prediction was not found. In both the easy and the hard task, ADHD subjects made more errors than control subjects and showed only a trend toward a larger performance decrement with increasing task load. It has to be noted that even in the easy task, hit percentages were lower in the ADHD group while there were no differences between the groups in task P3 amplitude. This finding can be explained by the fact that although performance and the P3 amplitude are related, these variables are known to show some dissociation (see review by Koelega & Verbaten, 1991). Probe P3 analysis did not lead to the conclusion that ADHD subjects invest spare capacity in the more difficult task. Both ADHD and control subjects showed smaller P3s to novel probes in the hard than in the easy task (an indication that less spare capacity was available for P3 processes), but in the ADHD group this did not concur with an increase in P3 task amplitudes in the hard task. Apparently, ADHD subjects do not, like normal controls, invest spare capacity in the task to prevent greater deterioration. These results suggest the presence of a resource allocation problem in ADHD children: they do not invest all available capacity when it is needed. Instead, although the ERP evidence is not conclusive on this point, it seems that at least some of the remaining capacity is used for processing of the novel probes, because even in the hard task some processing of the probes still takes place (amplitudes were not zero). It has to be mentioned that, in the hard task condition, there were three ADHD subjects who performed at chance level. It might be argued that these chance performers were not performing the task seriously and were therefore not suitable for testing the capacity hypotheses. However, there is some evidence that ADHD subjects were performing the task. When in the hard task chance performers would have been pressing buttons arbitrarily (independent of the task demands), these subjects would have been expected to show more variable response times than the other subjects which would lead to higher SDs in the hard than in the easy task. However, mean RT SDs were only 24 ms larger in the hard than in the easy task. Thus, a more likely explanation is that the chance performers were executing the task seriously but the task was too difficult for some of the subjects to score above chance level. More proof for the latter explanation can be found in the fact that two of the three chance performers still showed considerably high P3 amplitudes in the hard task (10 and 31 μV), while they performed at chance level. When these subjects would have been pressing...
from the analysis because their problems seem to originate from their disorder and not from a lack of motivation.

The purpose of the second experiment was to investigate whether methylphenidate was capable of improving the resource allocation deficit that was found in ADHD children in the first experiment. It was expected that, if methylphenidate was capable of improving this capacity-allocation deficit in ADHD children, this improve-
Methylphenidate effects on attentional capacity deficits in ADHD

ment would become evident as an increase in the percentage of correct responses and higher P3 amplitudes, especially in the hard task. It appeared that, although methylphenidate had an enhancing effect on the amount of correct responses and task P3 amplitude of the ADHD children, these effects were the same in easy and hard tasks. Furthermore, methylphenidate, compared with placebo, did not affect the novel probe P3; in both placebo and drug conditions, P3 amplitudes were smaller in the hard than in the easy task, indicating that there was a certain amount of “rest capacity.” But the medication did not help ADHD children to invest this extra capacity in the hard task to prevent a large performance decrement. Summarizing, methylphenidate had general enhancing effects on the amount of invested capacity in all tasks but did not influence the allocation strategies of ADHD children; after methylphenidate ingestion they still did not invest all their capacity in the hard task to improve performance.

So, in contrast to the findings reported by Carlson et al. (1991), we cannot conclude that methylphenidate administration to ADHD subjects leads to a better allocation strategy in case of higher task demands; in that case a Drug × Load interaction should have emerged, which was not the case. There are, however, a few differences between the Carlson et al. study and the present study that might account for the different findings. First, Carlson et al. did not measure ERPs, which might have given additional information about capacity trade-off between the tasks. Second, the conclusion by Carlson et al. that ADHD children had an allocation deficit was not based on a comparison with controls, as it was in the present study. Third, Carlson et al. used two tasks that both required processing (divided attention) whereas in the present study the probes were task irrelevant.

Although hard–easy differences in P3 amplitude in ADHD subjects were the same in both studies, compared with the first experiment, absolute P3 amplitudes in the second experiment were higher to task stimuli and smaller to novel stimuli. These differences may be attributed to repetition effects: the higher task P3 amplitudes may in this sense reflect a learning effect (more correct responses were made in the second and third sessions), and the smaller novel amplitudes may be caused by a reduced novelty effect because the same novels were presented repeatedly.

Interestingly, as opposed to the P3, in the first experiment neither ADHD nor controls showed a load effect on the “probe” N1 (or other early peaks), with the exception for normal controls of a smaller P1 in response to deviant probes in the hard task. These findings are in accordance with findings by Kenemans et al. (1992) and Verbaten et al. (1997), who concluded that no load effects were found on early waves because these waves, as opposed to the P3, represent processes that are running more automatically and are thus less dependent on capacity resources. However, Kramer et al. (1995) did report load effects on early waves (N1, P2, and mismatch negativity) but not on the P3. There are a number of differences between the paradigm used in the present study and that used by Kramer et al. that might account for the differences regarding early waves. First, probes were presented in different modalities (auditory probes whereas in the present study visual probes were used). Second, there was a difference in the manipulation of task difficulty: perceptual stimulus discriminations in the Kramer et al. study versus memory comparison in the present study. Different processes might be manipulated by these different task requirements and these processes might be dependent on different capacity resources. In this sense the perceptual task manipulation might cause a depletion of the capacity resources that are used specifically for processes that are represented by the early waves, consequently leading to a shortage of capacity for the same processes to evaluate the probes. In the same way, the memory comparison manipulation might require more P3 processing, thereby enhancing the demand on P3 resources that can no longer be used for evaluation of the probes, leading to reduced probe P3 amplitudes. However, these explanations are speculative and further research is needed to clarify these issues. Also, the above explanation cannot explain why in the second experiment some load effects on early waves (N1, P1, P2) in response to probes did occur in the same group of ADHD children.

Some group differences that are unrelated to the capacity allocation deficit were found: compared with controls, at Fz and Cz, the ADHD subjects had smaller N1 amplitudes and larger P2 amplitudes at all four leads in response to hits. The meaning of these group effects is not clear; smaller N1s in ADHD children have been reported in selective attention studies and in these paradigms are indicative of deficient filtering of irrelevant information (Loiselle, Stamm, Maitinsky, & Whipple, 1980; Zambelli, Stamm, Maitinsky, & Loiselle, 1977). The meaning of the smaller N1 to hits in the present paradigm remains, however, unclear. Robaey, Breton, Dugas, and Renault (1992) also reported larger P2 amplitudes (to targets and nontargets) in ADHD children in a categorization task. They concluded that orienting to differences between successive stimuli was enhanced in hyperactive subjects, regardless of the relevance of these differences for the categorization process.

However, in the present study, methylphenidate had no effects on these early peaks to task stimuli; N1 and P2 amplitudes remained unchanged. The absence of methylphenidate effects on the N1 is in agreement with findings from an earlier study (Jonkman et al., 1997b).

Conclusions

In line with earlier findings (Schachar & Logan, 1990; Sergeant & Scholten, 1985) there is evidence in favor of a capacity-allocation deficit rather than a capacity-availability deficit in ADHD children. Thus, the fact that they often make more errors cannot be explained easily by less available capacity to perform a task; ADHD subjects invested the same amount of capacity in the easy task (reflected by task P3 amplitudes) as did normal subjects, but did not show the P3 increase of normal subjects in the hard task. This finding might be related to the trend toward a larger performance decrement from the easy to the hard condition in ADHD subjects. However, an explanation in terms of a shortage of capacity in ADHD children was not supported by P3 amplitudes to probe stimuli; ADHD and control subjects showed the same reduction in novel P3 amplitude with increasing task load. In other words, in the normal group the spare capacity, which was manifested in the easy task as a large P3 reaction to the novels, was used in the hard task to meet the higher task demands, as evidenced by an increase in task P3 and a decrease in “novel” P3. Although ADHD subjects had the same amount of spare capacity at their disposal, they were unable to invest it in the hard task, as indicated by the absence of an increase in P3. Finally, in contrast to the study by Schachar and Logan (1990), we found a difference in errors in both the easy and hard task: ADHD children made more errors than normal children.

Methylphenidate did not improve the capacity-allocation deficit of ADHD children found in the first study. Methylphenidate improved correct detections and increased P3 amplitudes in response to task-relevant stimuli, but there was no interaction of methylphenidate effects with task load, as was expected if in the hard task the drug would have ameliorated the allocation deficit.
REFERENCES


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