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# Inhibition in Children with Attention-Deficit/Hyperactivity Disorder: A Psychophysiological Study of the Stop Task

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**Background:** *The purpose of the study was to investigate and identify abnormal brain activity, as revealed by event-related potentials (ERPs) concurring with deficient inhibitory control in children with attention-deficit/hyperactivity disorder (ADHD).*

**Methods:** *Performance and ERPs from 16 children with ADHD and 16 control subjects were compared in the stop-signal paradigm.*

**Results:** *The ADHD children showed a lower inhibition percentage and their (estimated) response time to the stop signal was disproportionately longer compared to the slowing of reaction times to primary-task stimuli. In normal control subjects, fronto-central positivity (100–400 msec) after the onset of the stop-signal was larger in case of successful inhibition, relative to failed inhibition; this was less so in ADHD children. A late positive wave (500–700 msec), maximal at Oz on failed inhibition trials, and possibly related to error-detection, was smaller in ADHD children.*

**Conclusions:** *These results point to abnormalities in brain processes involved in motor inhibition and error-detection in ADHD children.* Biol Psychiatry 2002;51: 668–676 © 2002 Society of Biological Psychiatry

**Key Words:** Inhibition, attention-deficit/hyperactivity disorder, event-related potentials, stop task, error detection

## Introduction

Impulsivity is assumed to be a major characteristic of Attention-deficit/hyperactivity disorder (ADHD) (DSM-IV; American Psychiatric Association 1994). According to

the DSM-IV, impulsivity manifests itself as “1) difficulty in delaying responses, blurting out answers before questions have been completed, 2) difficulty awaiting one’s turn, 3) frequently interrupting or intruding on others to the point of causing difficulties in social, academic or occupational settings.” Many factors are assumed to be involved in impulsive behavior, such as deficiency in processes involved in preparing, initiating, delaying and executing ongoing responses, processes involved in interrupting and altering ongoing responses and effects of reward and punishments. In the current study we will focus on the process of inhibitory control involved in stopping of an ongoing response.

Individuals with deficient inhibitory control react impulsively in situations in which controlled behavior is required. Logan and Cowan (1984) developed a paradigm in which inhibitory control can be measured: the stop-signal paradigm. In this paradigm, participants perform a primary task, a visual choice reaction time task. The instruction is to respond as quickly and accurately as possible. Occasionally a stop signal is given, usually a tone, that tells them to withhold their response on that trial. The issue is whether the participants succeed in withholding their response on the stop signal or not.

The basic assumption in this paradigm is a horse-race between these two processes. If the response process related to the visual stimulus finishes earlier than the stop process, initiated by the auditory stop signal, the response is executed; however, if the stop process finishes before the response process, the response is inhibited. In this horse-race model, it is contended that the two processes are independent (De Jong et al 1990; Jennings et al 1992; Logan and Cowan 1984). The outcome of this race depends on the interval between the onset of the visual stimulus and the presentation of the stop signal (called stop-signal delay). If the stop signal occurs early enough, the response will always be inhibited, if the stop signal occurs too late, the response will always be executed.

The time course of inhibitory control can be measured

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by estimating the speed of the stopping process, called the stop-signal reaction time (SSRT), and it is assumed that the SSRT is constant. Because the SSRT cannot be observed directly, this measure is estimated from the distribution of reaction times to the visual trials without a stop signal, the probability of inhibition on the stop-signal trials, and stop-signal delay (Logan 1994; Logan and Cowan 1984).

Several studies using the stop-signal task have shown a worse inhibition performance and longer SSRTs in children with ADHD compared with a normal control group (Jennings et al 1997; Pliszka et al 1997, 2000; Schachar and Logan 1990; Schachar et al 1995, 2000).

A group of ADHD- and aggressive children (Oosterlaan and Sergeant 1996) and ADHD children with a comorbidity of oppositional defiant disorder (ODD) or conduct disorder (CD) (Jennings et al 1997) also showed a poor inhibitory control compared to a control group. A group of children with a combination of the diagnoses ADHD and aggression did *not* differ in inhibitory control compared to the ADHD children (Oosterlaan and Sergeant 1996). See for an overview Oosterlaan et al (1998) and Tannock (1998).

Therefore, we also expected in the present study that ADHD children would show a deficit in inhibitory control, in terms of a smaller percentage of inhibition on stop-signal trials and longer latencies of the stop process (SSRT), compared to a normal control group (Oosterlaan et al 1998; Tannock 1998).

Event-related potentials (ERPs) may provide direct information about the inhibition process in the brain in the order of milliseconds, which cannot be obtained by performance alone. De Jong et al (1990) conducted the first ERP study in the stop task, they reported larger fronto-central P3s for trials on which inhibition succeeded compared to trials in which inhibition failed.

Two ERP studies employed the stop task in children with ADHD. In the study of Brandeis et al (1998), no differences in inhibition performance nor any differences in brain activity between the ADD and control group were found that could be related to the processing of the stop signal. This might be attributed to the fact that the ADD children did not meet the DSM-III-R criteria for ADHD. [Pliszka et al (2000) found besides a deficient inhibition performance in the ADHD group a smaller right frontally N2 to the stop stimulus in ADHD children compared to a normal control group, but this was similar for successful and failed inhibition trials.]

Activation of the right inferior frontal lobe and the caudate nucleus in normal subjects during the stop task has been reported by Rubia et al (1999) in a functional magnetic resonance imaging (fMRI) study; activation of these areas were lower in adolescents with ADHD. Other fMRI studies on response inhibition also showed that the

frontostriatal circuitry is implicated in ADHD (Casey et al 1997; Vaidya et al 1998). Konishi et al (1999) found dominant no-go activity in the posterior part of the right inferior frontal sulcus in an event-related fMRI study in normal adults. A recent fMRI study in normal adult subjects investigated different types of the stop- and go/no-go tasks. Activation in the go/no-go tasks were predominantly left hemispheric mesial, medial and inferior frontal, and parietal cortices. For the stop tasks, activation was predominantly found in right hemispheric anterior cingulate, supplementary motor area, inferior prefrontal, and parietal cortices. Activation in inferior frontal lobes has been found to be most consistent across studies (Rubia et al 2001).

If impaired inhibition performance in ADHD children is due to a deficient processing of the stop signal, ERPs might give us insight in the electrical activity of the brain during situations of successful and unsuccessful inhibition, and given their high resolution in time, also indicate at which period in time such differences occur. This might become manifest in a smaller difference in fronto-central positivity between successful and failed inhibited trials. To accumulate enough trials for the ERPs while not making the task too long for the children, we presented stop signals on 40% of the trials with two fixed delays (or stimulus onset asynchronies [SOAs]), one of 125 msec and one of 200 msec. Jennings et al (1997) also used two stop-signal delays with results that were comparable with other stop-task studies using delays dependent on the subjects mean reaction time (Oosterlaan et al 1998; Tannock 1998).

## Methods and Materials

### Subjects

Participants were 16 ADHD children (all boys, aged [mean  $\pm$  SD] 10.4  $\pm$  1.4 years [range: 7–12 years], full-scale intelligence quotient [IQ] on Wechsler Intelligence Scale for Children-Revised [WISC-R] 95.4  $\pm$  14.6) with a diagnosis of ADHD according to DSM-III-R criteria (American Psychiatric Association 1987). They were referred to the outpatient clinic of the Utrecht Department of Child and Adolescent Psychiatry. All referred children and their parents participated in a series of extensive diagnostic evaluations, which followed a structured protocol. In the Diagnostic Interview Schedule for Children (DISC)-2.3 interview with the parents (Schwab Stone et al 1996; Shaffer et al 1996) the symptoms and diagnostic criteria of all DSM-III-R axis I disorders occurring in childhood were systematically and completely reviewed. Further, for all subjects, scores on the Child Behavior Checklist (CBCL) (Achenbach and Edelbrock 1983) completed by parents and on the 39-item Conners Teacher Rating Scale (CTRS) (Conners 1985) were available. Subjects were only included in the study if the following criteria were met: 1) diagnosis of ADHD according to

DSM-III-R criteria; and 2) scores in the clinical range on both the CBCL inattention factor ( $T$ -scores higher than 70, i.e., 98th percentile) and CTRS Hyperactivity factor (mean factor score  $> 2.2$  [items rated as 1 = not at all, to 4 = very much]). Children with a diagnosis of tic disorder or pervasive developmental disorder were excluded from the study. Further, the ADHD children had the following scores on the CBCL and CTRS (mean  $\pm$  SD): CBCL Inattention  $T$ -score  $74.4 \pm 8.0$ , Externalizing  $T$ -score  $59.5 \pm 8.2$ ; CTRS Hyperactivity  $2.7 \pm .5$ , Conduct  $1.9 \pm .7$ .

Six ADHD children had comorbid ODD, one child had a comorbid anxiety disorder, and three children had specific developmental disorders (developmental reading disorder, expressive language disorder, developmental coordination disorder). Approval of the Medical Ethics Committee and informed consent from the parents of the children were obtained. All children had normal or corrected-to-normal vision. All ADHD children used methylphenidate and stopped taking the drugs 3 days before they participated in the experiment.

The children of the normal control group were recruited from various elementary schools and were included as control subjects only if 1) all factors scores on CBCL and CTRS were in the normal range; 2) they had never been referred to a psychiatric or mental health unit; and 3) they satisfied two or fewer criteria of ADHD according to DSM-III-R in a parent interview and did not qualify for DSM-III-R ODD or CD. Sixteen control children participated in the study (14 boys and 2 girls; aged [mean  $\pm$  SD]  $10.3 \pm 1.5$  years [range: 7–12 years], full-scale IQ on WISC-R  $105.4 \pm 13.8$ ; CBCL Inattention  $T$ -score  $52.4 \pm 4.6$ , Externalizing  $T$ -score  $56.5 \pm 4.5$ ; CTRS Hyperactivity  $1.2 \pm .5$ , Conduct  $.8 \pm .03$ ).

The selection of the subjects and collection of data were completed before publication of the DSM-IV (American Psychiatric Association 1994). Afterwards we diagnosed the children according to DSM-IV categories. The ADHD diagnosis in DSM-III-R was found to be fully concordant with the DSM-IV diagnosis of ADHD combined type in all 16 children of the experimental group. The difference in IQ between both groups was near-significant [ $F(1,30) = 3.95, p = .056$ ]. Therefore, we determined correlations between IQ and each of the dependent variables in case significant differences between groups were found. In none of the cases, however, were these correlations significant, so there was no reason to conduct analyses of covariance.

### Stop Task

The primary (go) task was a choice reaction-time task, in which a colored clown's face was presented in the middle of a television screen. The clown had a feather on his head; if the feather pointed to the right a right-hand button press had to be made, and when it pointed to the left a left-hand button had to be made. The clown's face was 6.5 cm wide ( $6.2^\circ$ ) and 4.8 cm high ( $4.6^\circ$ ). The stop signal was a 1-kHz tone, 80 dB, with a duration of 400 msec, generated by the computer and administered binaurally through headphones.

Each trial began with a white dot as a warning signal for 500 msec in the center of the screen, followed by a blank screen for

500 msec. Then the clown's face appeared for 750 msec, followed by a variable inter-stimulus-interval of 1.0–1.25 sec in which the screen remained blank.

There were eight blocks of 120 trials (total of 960 trials). Each block started with three warming-up trials, which were excluded from the analysis. Stop signals were presented on 40% of the trials (384 trials) at 125 msec or 200 msec after the onset of the go-task stimulus. In each block the two primary task stimuli occurred equally often and the stop signals occurred equally often at each stop-signal delay, the sequence of trials was pseudorandomized, and each block lasted about 6 min.

### Procedure

On arrival the child was familiarized with the procedure. During electrode attachment the children watched cartoons. They were told that they could choose a toy after the test if they performed well enough (eventually each child received a toy). After the attachment of the electrocap and electro-oculographic (EOG) electrodes, the child sat down in an adjustable dentist's chair, in an acoustically shielded room. The experiment started with the AX-version of the Continuous Performance Test (CPT), which lasted 11 min (the data of this task are reported in Overtoom et al 1998). Because the stop task lasted a relatively long time it was always the second task. A button switch was located on the forefinger of each hand. Initially, the children were instructed only with respect to the choice-reaction time task. They received two practice blocks of 2.5 min, during which they had to respond as quickly and accurately as possible. Then a practice block for the stop task (2 min) followed. The instruction was the same as for the former practice blocks, but in addition the children were told that whenever they heard a tone they should *not* respond to the primary- (go) task stimulus. It was also emphasized that they should not wait for the tone. Finally, they were notified that after two experimental blocks they would get a break (about 10 min), and about the presence of an intercom and a camera. After each block the computer provided the mean reaction time (RT) and number of errors to the primary task, as well as their inhibition percentage. If the mean RT was more than 50 msec longer than the mean practice RT and the inhibition percentage was more than 10% higher than in the preceding block, this was taken as an indication that the child might have adopted a strategy to wait for the tone to increase the probability of inhibiting. In such cases we encouraged the child to respond as quickly to the go-task stimuli as he or she had done before in the practice blocks without the tones, and to reckon with the possibility of a stop signal to a lesser extent. As it turned out both groups showed somewhat longer RTs during the go task than during the practice task (ADHD: practice task 553 msec, go task 598 msec; normal control subjects: practice task 487 msec, go task 508 msec); but statistical testing showed that there was no significant difference between the groups in the difference between RT during the practice task and go task [ $F(1,30) = .31, ns$ ].

### Psychophysiological Recording

Electroencephalographic (EEG) as well as horizontal and vertical EOG activity was recorded from tin electrodes held in place by,

Table 1. Summary of Stop-Task Performance<sup>a</sup>

	RT (in msec)	Choice errors (%)	Omissions (%)	Inhibition (%)		Stop-signal RT (msec)	
				SOA125	SOA200	SOA125	SOA200
ADHD	598 (161)	9.97 (3.7)	8.1 (7.3)	55.5 (21.2)	46.8 (16.8)	472 (283)	435 (276)
Normal control subjects	508 (104)	4.89 (1.8)	2.9 (1.5)	75.8 (13.7)	62.4 (13.3)	279 (98)	245 (84)

ADHD, attention-deficit hyperactivity disorder; NC, normal control group; SOA125, stimulus onset asynchrony at 125 msec; SOA200, stimulus onset asynchrony at 200 msec; RT, reaction time.

<sup>a</sup>Mean values per group and SOA (for inhibition percentage and stop-signal RT), SD in parentheses.

respectively, an electrocap and adhesive rings. Scalp locations were at Oz, Pz, Cz, Fz (according to the 10–20 system; Jasper 1958), referenced to linked mastoid electrodes. A ground electrode was placed on the forehead. Resistance was less than 5 k $\Omega$ . Signals from EEG and EOG were amplified with, respectively, a time constant of 10 sec and 36 sec, in conjunction with, respectively, 200- and 300-Hz low-pass filters, and were thereafter filtered with a 40-Hz (24dB/oct) low-pass filter. All signals were sampled continuously, at a rate of 100 Hz.

### Signal Analysis (ERPs to the Stop Signal)

The EEG data were epoched for 1100 msec starting 100 msec before visual stimulus onset, filtered off-line by a 30 Hz (24 dB/oct) low-pass filter and checked for artifacts. Vertical and horizontal EOGs were subtracted from the ERPs (Kenemans et al 1991). Auditory ERPs were selectively averaged for successful and failed inhibition trials. The percentage of go trials (ERP to the visual stimulus) was 71% for the ADHD children and 86% for the control group. Percentage of failed inhibition trials was 39%–47% (SOA125 and SOA200) for the ADHD group and 24%–35% (SOA125 and SOA200) for the control subjects. The percentage of successful inhibition trials was 49%–42% (SOA125 and SOA200) for the ADHD group and 70%–58% (SOA125 and SOA200) for the control subjects. To remove the overlap from the preceding visual stimulus on the auditory ERPs we used the same procedure as De Jong et al (1990; see page 175). The resulting difference waves, the successful inhibition and failed inhibition ERPs, were baseline corrected (–100–0 msec) and analyzed in time segments of 50 msec from 100–400 msec after the onset of the auditory stimulus.

### Statistical Analysis

**PERFORMANCE.** Planned comparisons to test for Group differences were performed on the mean reaction time, choice-error rate, and omission rate separately in a univariate analysis of variance with Group as a between-subjects factor (ADHD and control subjects). Inhibition percentage and stop-signal reaction time were also tested separately for Group differences with one within-subjects factor SOA (125 and 200 msec) and the between-subjects factor Group. In all these cases significance levels were set at 5%, one-tailed, because the direction of these effects could be predicted: longer reaction times, more errors, and worse inhibition performance of the ADHD group compared to control subjects (Oosterlaan et al 1998). In all other cases two-tailed tests will be reported.

**ERPS.** A multivariate analysis of variance was conducted for each time window of 50 msec, from 100–400 msec after the onset of the auditory stimulus of the difference wave, using one between-subjects factor Group (ADHD and control subjects) and three within-subjects factors, SOA (125 and 200 msec), Inhibition (successful and failed inhibition trials), and Leads (Fz, Cz, Pz, and Oz). Significance levels were set at 5%, two-tailed. In case of a significant effect extending over multiple successive windows, only maximum and minimum *F*-values are reported.

## Results

### Performance

**GO TASK.** Planned comparisons showed that the ADHD children had longer reaction times [ $F(1,30) = 3.58, p < .05$ , one-tailed] made more choice errors [ $F(1,30) = 23.87, p < .001$ , one-tailed] and more omissions [ $F(1,30) = 8.10, p < .01$ , one-tailed] than the control children (see Table 1).

**STOP TASK.** Stopping performance is summarized in Table 1. The ADHD group showed a lower percentage of inhibition [ $F(1,30) = 10.23, p < .01$ ] compared to the control group. The inhibition rate decreased with increasing SOA [ $F(1,30) = 41.60, p < .001$ ], but there was no significant Group  $\times$  SOA interaction. To exclude the possibility that the inhibition performance of the ADHD children was contaminated by omitted responses to the visual go task, we used the corrected inhibition method as proposed by Tannock et al (1989)<sup>1</sup>. Applying this method yielded essentially the same results: Inhibition percentages

<sup>1</sup>Procedure for correcting inhibition proportion by Tannock et al (1989):

$$P_y = \frac{x - o}{N - o} = \frac{\frac{x}{N} - \frac{o}{N}}{\frac{N}{N} - \frac{o}{N}} = \frac{P_x - P_o P_x}{1 - P_o P_x}$$

$P_y$  = corrected inhibition proportion.

$x$  = number of stop trials with successful inhibition.

$N$  = total number of stop trials.

$o$  = number of omissions in stop-signal trials (unknown), estimated as  $P_o P_x N$ .

$P_o$  = number of omissions in go trials divided by the number of go trials (general omission probability).

$P_x$  = number of successfully inhibited stop trials divided by the number of stop trials (raw inhibition rate).

were smaller in the ADHD group [ $F(1,30) = 11.22, p < .001$ ]. Uncorrected inhibition rates are reported nevertheless, for reasons of comparability with average ERPs for which individual omission trials cannot be taken into account.

Stop-signal reaction times were longer for the ADHD children than for the control subjects [ $F(1,30) = 6.88, p < .01$ ] and longer for the shorter SOA than for the long one [ $F(1,30) = 15.98, p < .001$ ]. The Group  $\times$  SOA interaction was not significant.

**POST HOC ANALYSIS.** We also tested whether the deficient inhibition in ADHD children was due to a larger delay in stop-signal RT or to a larger delay in go RT to the primary task stimulus. A factor RT category was constructed with two levels, stop-signal RT and go RT. Further, the factor Group included two levels, ADHD and control subjects. The interaction Group  $\times$  RT category was significant [ $F(1,30) = 6.40, p < .05$ ]. This reflected that, as already mentioned above, ADHD children had slower reactions to the primary stimuli than did control subjects (598 vs. 508 msec), but they showed a disproportionately longer delay in SSRT (454 vs. 262 msec) [ $F(1,30) = 6.88, p < .05$ ].

### ERPs

**INHIBITION EFFECTS (DIFFERENCES BETWEEN SUCCESSFUL AND FAILED INHIBITION).** As can be seen in Figures 1 (SOA125) and 2 (SOA200), there was more positivity between 100 and 400 msec for the successful than for the failed inhibition trials, especially at Fz, Cz, and Pz. Significant main effects of Inhibition were found for all epochs between 100 and 350 msec (see Table 2 for the  $F$ - and  $p$ -values of the significant overall effects). An SOA  $\times$  Inhibition interaction was found from 150 to 200 msec; tests for the effect of inhibition for the separate SOAs revealed significance only for SOA125 [ $F(1,31) = 30.80, p < .001$ ]. An Inhibition  $\times$  Leads interaction was present from 150 to 400 msec. Tests for separate leads revealed significant effects of Inhibition between 150 and 400 msec at Fz [ $F(1,31) = 6.5\text{--}16.6, p < .05$ ], and Cz [ $F(1,31) = 7.1\text{--}36.2, p < .05$ ], and from 150 to 350 msec at Pz [ $F(1,31) = 7.1\text{--}15.1, p < .05$ ].

**GROUP EFFECTS.** Figures 1 and 2 also suggest that the difference in positivity between successful and failed inhibition trials was larger for the control subjects than for the ADHD group. This was confirmed by significant Group  $\times$  SOA  $\times$  Inhibition (100–200 msec, 350–400 msec) and Group  $\times$  Inhibition  $\times$  Leads (250–400 msec) effects.

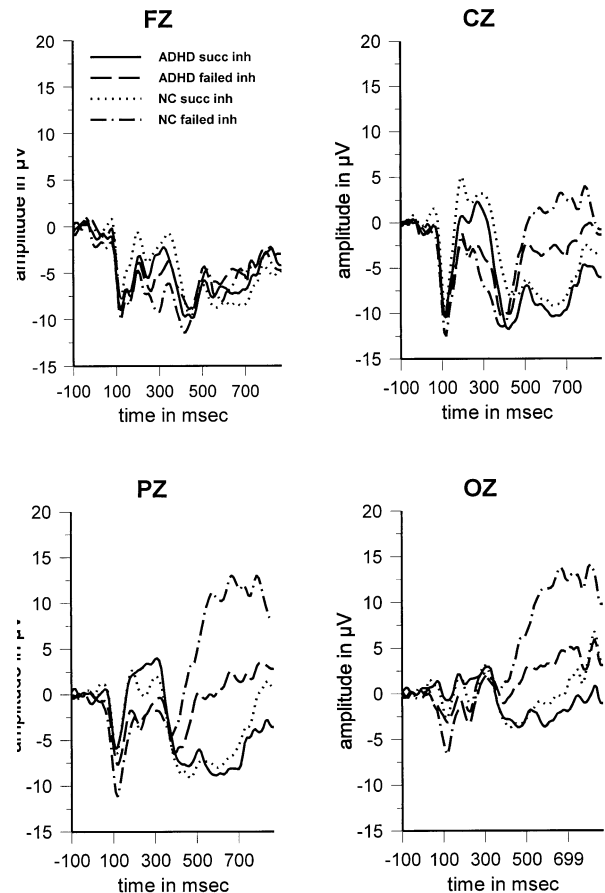


Figure 1. Auditory event-related potentials corrected for the overlap of the preceding visual go stimulus for SOA125 (stop signal presented 125 msec after visual stimulus) for attention-deficit/hyperactivity disorder (ADHD) and control (NC) group for successful (succ inh) and failed inhibition (failed inh) trials. Time point zero is the onset of the auditory stimulus.

Further analysis of the former interaction showed that between 100 and 200 msec, the Group  $\times$  Inhibition effect was only significant for SOA125 [ $F(1,30) = 4.5\text{--}5.1, p < .05$ ]. To further delineate this interaction for SOA125, Inhibition effects were tested per Group for this SOA only. Between 100 and 150 msec, the difference between successful and failed inhibition trials was significant only for the control group [ $F(1,15) = 12.41, p < .01$ ]. Between 150 and 200 msec the difference between conditions was significant for the control [ $F(1,15) = 28.49, p < .001$ ] and for the ADHD group [ $F(1,15) = 8.02, p < .05$ ], but smaller for the latter. Between 350 and 400 msec, for neither SOA was the Group  $\times$  Inhibition significant.

To further analyze the Group  $\times$  Inhibition  $\times$  Leads interaction (250–400 msec), we tested the Group  $\times$  Inhibition effect for separate leads. This yielded significance at Fz and Cz between 250 and 300 msec (approximately,  $p < .06$ ), and at Fz between 300 and 350 msec ( $p < .05$ ). For both these time windows, the Inhibition

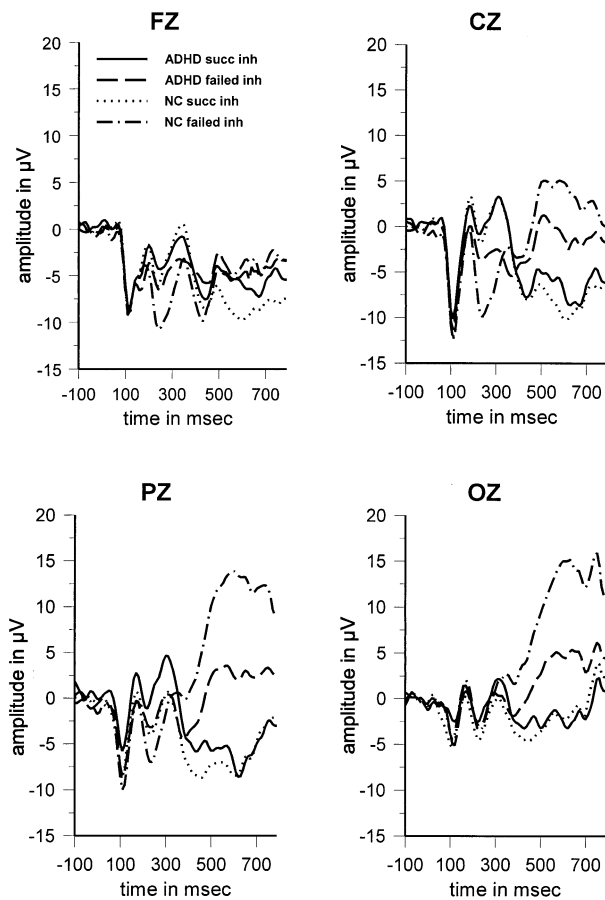


Figure 2. Auditory event-related potentials corrected for the overlap of the preceding visual go stimulus for SOA200 (stop-signal presented 200 msec after visual stimulus) for attention-deficit/hyperactivity disorder (ADHD) and control (NC) group for successful (succ inh) and failed inhibition (failed inh) trials. Time point zero is the onset of the auditory stimulus.

effect at Fz was significant for control subjects ( $p < .001$  for both windows), but not for ADHD children. The Inhibition effect at Cz was significant for both the ADHD ( $p < .05$ ) and control group ( $p < .005$ ). To corroborate the conclusion of a significant, lead-dependent difference between groups in inhibition-related positivity, we also

tested the Inhibition  $\times$  Leads interaction for separate groups. For control subjects this yielded significance in all three time windows ( $p < .005$ ,  $.005$ , and  $.01$ , respectively), but for the ADHD group between 250 and 300 msec ( $p < .05$ ) only.

An additional analysis was performed because, as evident in Figures 1 and 2, a huge positive deflection later in time was seen. For the time window 500–700 msec an Inhibition main effect [ $F(1,30) = 64.12, p < .001$ ], a Group  $\times$  Inhibition [ $F(1,30) = 7.41, p < .05$ ] and an Inhibition  $\times$  Leads interaction [ $F(3,28) = 11.92, p < .001$ ] were found. This latter interaction reflected significant inhibition effects at Pz [ $F(1,31) = 57.87, p < .001$ ] and Oz [ $F(1,31) = 39.99, p < .001$ ] with more positivity for the failed than for the successful inhibited trials. More importantly, the Group  $\times$  Inhibition effect reflected far larger amplitudes for the failed inhibitions for the control group than for the ADHD children [ $F(1,30) = 8.02, p < .01$ ].

## Discussion

### Performance

Inhibition performance in ADHD children was worse compared to that in a normal control group. Also, the latency of the stop process (SSRT) was longer for the ADHD than for the control group. These findings are in agreement with other studies on the stop-signal paradigm (Jennings et al 1997; Oosterlaan and Sergeant 1996; Schachar and Logan 1990; Schachar et al 1995). Furthermore, the ADHD group made more errors (choice errors and omissions) and showed longer mean reaction times to the visual go stimulus without a stop signal than did their normal peers.

Another finding was that the delay in SSRT was much longer than the delay in the go RT for the ADHD children compared to the control group. This could indicate that the impaired inhibition in the ADHD children was at least partly due to a specific deficit in the processing of the stop stimulus, a conclusion that was further substantiated by the ERPs findings.

Table 2. Significant Inhibition and Group Effects in the Interval 100–400 msec, per 50 msec

	100–150 msec	150–200 msec	200–250 msec	250–300 msec	300–350 msec	350–400 msec
Inhibition	9.62 <sup>a</sup>	23.87 <sup>b</sup>	43.91 <sup>b</sup>	32.21 <sup>b</sup>	23.68 <sup>b</sup>	
Inhibition $\times$ SOA		8.95 <sup>a</sup>				
Inhibition $\times$ Leads		4.09 <sup>c</sup>	5.99 <sup>a</sup>	10.19 <sup>b</sup>	10.35 <sup>b</sup>	6.32 <sup>a</sup>
Group $\times$ SOA $\times$ Inhibition	4.72 <sup>c</sup>	5.55 <sup>c</sup>				6.89 <sup>c</sup>
Group $\times$ Inhibition $\times$ Leads				3.31 <sup>c</sup>	3.01 <sup>c</sup>	3.11 <sup>c</sup>

Effects without the factor leads have  $F(1,30)$  values, effects with the factor leads have  $F(3,28)$  values. SOA, stimulus onset asynchrony.

<sup>a</sup> $F$ -value with  $p < .01$ .

<sup>b</sup> $F$ -value with  $p < .001$ .

<sup>c</sup> $F$ -value with  $p < .05$ .

### ERPs

Fronto-central ERPs to the auditory stop signals were marked by more negative (less positive) amplitudes for failed than for successful inhibition trials, from 100 to 400 msec poststimulus. This means that successful inhibition of a response concurs with more positive amplitudes. De Jong et al (1990) also found in adults more positive amplitudes for successful inhibited than for failed inhibited trials at Cz, from about 150 msec on. In largely the same interval we found that the inhibition-related positivity was smaller in the ADHD group, especially at Fz. Thus, the ERP recordings seem to provide converging evidence of a specific ADHD deficit in the processing of the stop signal.

There are several possibilities with respect to the interpretation of this inhibition-related positivity. The obvious interpretation is that the inhibition-related positivity is a direct reflection of the motor inhibitory process. In normal control children the significant inhibition-related positivity concurs in time with their SSRT (279–245 msec [mean SSRTs for the two SOAs]), thus the positivity could be a direct reflection of inhibitory processing.

The inhibition-related positivity of the ADHD children was much smaller than that of the control subjects, and the ADHD SSRTs were much longer (472–435 for the short and longer SOA) than 400 msec, exceeding the frontal inhibition-related positivity window. In this case the interpretation could be that smaller positivity reflects weaker inhibition, leading to SSRTs that are much longer or too long (failed inhibitions).

In the failed inhibition trials, we found a large late positivity at Pz and Oz, which was much larger than when inhibition was successful. Furthermore, this differential positivity was much larger for the control subjects than for the ADHD children. It could well be related to what Falkenstein et al (1991) have termed “error positivity” (“Pe”). The Pe is thought to reflect a more thorough evaluation of the incorrect response (Falkenstein et al 1991, 1995). Also according to others (Rösler 1983) errors elicit late ERP positivity, which may be related to processes like the adjustment or updating of the subject’s strategy following the recognition of an error (Donchin et al 1988). Thus, the Pe might reflect a kind of evaluation of the subject’s own performance. In turn, the smaller Pe in the ADHD group suggests that these children have a deficient evaluation of their incorrect responses.

Tasks in which a response has to be issued after a go stimulus but inhibited after an alternative no-go stimulus (Go/Nogo tasks) have revealed an increase in negativity to the no-go stimulus, relative to the go stimulus. In a variety of this task, the Ax-version of the Continuous Performance Task

(CPT-AX) (Overtom et al 1998), we found that ADHD children and control subjects did not differ with respect to this negativity (only a subgroup of ADHD with ODD did). The fact that they did differ with respect to the inhibition-related positivity in the stop task lends further credibility to the idea that the FzN2 negativity in the CPT-AX and the stop positivity reflect two distinct inhibitory processes. This may be related to the difference in demands imposed by the two tasks. In the CPT-AX, the response tendency that is built up before presentation of either the X or not an X, after the A, rarely results in a response before the presentation of that stimulus. In contrast, the stop task demands and elicits frequent responses to the go-task stimulus, which have to be inhibited only in a limited portion of the trials. It is possible therefore, that the inhibitory processes in the two tasks address different levels of the motor system, for example premotor-cortical processes in the CPT-AX and primary-motor activation in the stop task. A recent fMRI study (Rubia et al 2001) revealed a different involvement of brain structures during performance of the Go/Nogo task and during the stop-task, with stronger left fronto-parietal activation in the Go/Nogo task and involvement of supplementary motor cortex and the anterior cingulate gyrus in the stop task, supplying partial support for the contention of different structures involved in Go/Nogo tasks and stop tasks.

A contrasting result was reported by Pliszka et al (2000), who noted a frontal negativity (N2) to the stop trials that was smaller in ADHD children compared to control subjects in the stop task. The authors suggested that their N2 might be similar to the inhibition N2s reported by others in the Go/Nogo task. Furthermore, Pliszka et al (2000) reported a smaller frontal N2 in ADHD children, whereas Overtom et al (1998) did not find a difference in N2s, although in the CPT-AX, between the two groups. It has to be stressed that in the present study and in Overtom et al (1998) the same groups were investigated. This finding suggests that, at least in our lab, in the two tasks different neural systems are activated.

The reason we did not find an N2 in our stop task might be attributed to the fact that we used an auditory stop signal and Pliszka et al (2000) used a visual stop signal. Falkenstein et al (1999) found no N2 to auditory Nogo trials, whereas they did report an N2 in visual Nogo trials in the Go/Nogo task using the same subjects.

### Conclusion

In the present stop task, ADHD children exhibited reduced inhibition performance, as well as a slowing of stop-signal reaction time that was disproportionately larger than the slowing of the go RT. Event-related potential data also suggested a specific impairment in reacting to the stop signal. This impairment concerned an early processing

stage (100–400 msec after the stop signal) that might be related to motor-inhibition, and a later one (500–700 msec) that might reflect error processing. It might be interesting for future research to conduct an event-related fMRI study in children or adults with ADHD performing a stop task. In this way it would be possible to find out if different brain areas are involved in successful and failed inhibition trials and if these regions differ in ADHD.

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