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Inhibition of return in children with attention deficit hyperactivity disorder

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Abstract Earlier studies have suggested an impairment in the attention and eye movement control of children with ADHD. An important phenomenon in the control of attentional shifts and eye movements is the inhibition of return (IOR), which states that our brain works in a way that prevents our attention from returning to a spatial location that has been attended to, either overtly or covertly. This current study addresses whether the IOR in oculomotor planning is compromised in children with ADHD. Eleven ADHD and 12 age- and gender-matched control subjects participated in a behavioral task, in which they made saccades to a peripheral target after a valid, invalid or neutral cue. The latency difference between cued and uncued saccades over a range of cue-target onset asynchrony as well as the positive component of this latency profile (i.e., IOR) was compared between groups. The results show that ADHD children demonstrate a biphasic latency profile that is grossly similar to that

observed in control subjects, although the magnitude of IOR appears to be slightly smaller in ADHD subjects. These preliminary results suggest that the inhibitory attention mechanism subserving IOR is at least not fully compromised in ADHD children.

Keywords ADHD · IOR · Attention · Oculomotor · Saccade

Introduction

Children with attention deficit hyperactivity disorders (ADHD) are characterized by their difficulty in giving attention to details and in carrying on sustained attention during certain activities. Many studies have tried to elucidate the underlying mechanism of the attention impairment in these children. However, the results obtained in the literature are far from convergent (Blondis et al. 1999); some have even challenged the existence of attention deficit in ADHD (Corkum and Siegel 1993; Van der Meere 1996). Among the psychological tests used to examine attention functions in ADHD, Continuous Performance Tests (CPT) appear to be most widely used and to have fared best in terms of detecting attentional dysfunction in subjects suffering from this disorder (Losier et al. 1996). On the other hand, some studies have suggested that the attention impairment demonstrated with the CPT may not be specific to ADHD (Swaab-Barneveld et al. 2000; Riccio and Reynolds 2001).

Other than the CPTs, variants of the Posner's spatial cuing paradigm have been used to explore attention function in children with ADHD (Carter et al. 1995; Epstein et al. 1997; McDonald et al. 1999; Nigg et al. 1997; Perchet et al. 2001; Swanson et al. 1991). For instance, evaluating endogenous and exogenous cuing effects separately, Carter and colleagues found that children with ADHD showed an asymmetrical performance deficit characterized by a loss of costs on endogenous attentional orienting to invalidly cued left visual field targets (Carter et al. 1995). They suggested

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that this deficit may be related to diminished right hemispheric frontal-striatal catecholamine activity. Employing similar covert attention tasks, McDonald and colleagues showed that ADHD children exhibited bilaterally greater benefits for validly cued trials and greater costs for invalidly cued trials than control subjects (McDonald et al. 1999). Moreover, based on a lack of field-size effect on reaction time in the left visual field, they also reasoned that the right hemisphere suffers greater dysfunction than the left hemisphere in ADHD. Other studies have examined overt attention shifting and described impairment in eye movement control for these children (Castellanos et al. 2000; Houghton et al. 1999; Klorman et al. 1999; Ross et al. 2000). The latter experiments largely identify poor inhibitory control as the core deficit in this disorder (Barkley 1997), with little discussion about the relevance of the oculomotor abnormality to aberrant attention functions.

Evidence has been accumulating that eye movement and attention control are intricately related (Kustov and Robinson 1996; Moore and Fallah 2001; Sheliga et al. 1994). The integration of these two processes furnishes an important mechanism for allocating our processing resources both in the spatial and in the temporal domains – the inhibition of return (IOR) of attention (Posner et al. 1985). A motor response to a visual target presented at a pre-cued spatial location is facilitated if the target is presented shortly after the cue. When the cue-target onset asynchrony approaches a few hundred milliseconds, the response becomes slower instead, compared to the one made to a target that appears at an uncued location. The latter, inhibitory effect has been termed IOR. It is suggested that when directing attention away from a spatial location that has just been attended to, IOR provides an important behavioral strategy for effective foraging in our complex visual environment (Klein 1988). Since children with ADHD appear to be impaired in attention and eye movement control, we think it is of interest to investigate whether IOR is also compromised in this disorder.

Materials and methods

Subjects, experimental setups, and procedures

Eleven children with ADHD, of the combined type (Diagnostic and Statistical Manual IV, American Psychiatric Association, 1994), and 12 control subjects matched as a group for age, sex and IQ were recruited for the study, with all of them having normal or corrected-to-normal vision. The patient subjects were recruited from the outpatient clinic of the Chang Gung Memorial Hospital. All control subjects were children of hospital employees. One of the patient subjects dropped out early in the study, for personal reasons. Prior to the experiments both control and ADHD children received a clinical interview by a child psychiatrist (second author) based on a Chinese version of K-SADS (Chambers and Puig-Antich 1985). They all also received a parental rating on a Chinese version of the Child Behavior Check List (CBCL, Achenbach 1991). The ADHD children had mixed symptoms of hyperactivity/impulsivity and attentional disturbance, and a higher average T score than the control subjects. Table 1 lists the demographic data of the subjects.

Table 1 Demographic data of the subjects

	Control	ADHD
Age (years) ^a	11.8 (0.7)	12.2 (0.8)
Sex ^b	Nine males, three females	Nine males, two females
IQ ^c	103.2 (5.2)	105.8 (9.2)
CBCL T-score ^d	44.9 (4.4)	68.5 (4.7)

Standard deviations in parentheses: ^a P=0.27 (two-tailed Student's *t*-test); ^b P=0.54 (Fisher's exact test); ^c P=0.41 (two-tailed Student's *t*-test); ^d P<0.001 (two-tailed Student's *t*-test)

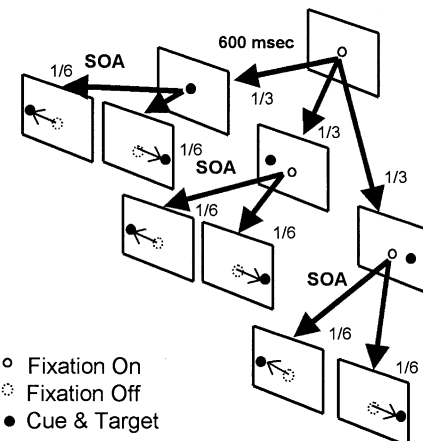


Fig. 1 The spatial cuing task. A center fixation point appeared along with two square boxes at the beginning of each trial. A peripheral cue showed up briefly (33 ms) in one of the boxes (each in one-third of the trials) or at the center, overlapping the fixation light (in another one-third of trials), after the subject acquired fixation for 600 ms. Following a pseudorandomized SOA, a target or imperative stimulus appeared at one of the two boxes. The subject then had to make a saccade to the target within 500 ms after its onset

None of the subjects had psychotic or mood disorders, or any history of neurological insult or of use of substance. ADHD children were medication free at the time of the experiment. After a complete description of the study was provided to the subjects and their parent(s), written informed consent was obtained. This study was carried out according to institute ethics guidelines.

The experiment was carried out in a dark room. Subjects were seated 50 cm in front of a ViewSonic P815 monitor, and their head was stabilized with a chin rest. Eye positions were recorded with a video-based eyetracker (Eyelink, SR Research, Toronto), using a sampling rate of 250 Hz and a spatial resolution of 0.1°, and were corrected for head movement. The visual stimuli were generated by a personal computer using a VGA graphic card and displayed in synchrony with a screen refresh rate of 60 Hz.

Figure 1 illustrates the behavioral task. A center fixation point appeared along with two square boxes (8° to the left and right of the fixation) at the beginning of each trial. A peripheral cue showed up briefly (33 ms) in one of the boxes (each in one-third of the trials) or at the center, overlapping the fixation light (in another one-third of trials), after the subject acquired fixation for 600 ms. Following a pseudorandomized SOA (50, 83, 100, 133, 167, 200, 400, 700, or 1,200 ms), a target or imperative stimulus appeared at one of the two boxes. The subjects had to maintain their eye within a window of 1.2°×1.2° throughout fixation and make a saccadic eye movement to the target within 500 ms after target onset.

The fixation point was a circle having a 0.7° visual angle in diameter and 4.6 cd/m² in luminance. Both the cue and the target

were a circle 0.7° in diameter and 18.75 cd/m^2 in luminance. The target appeared at the same location as the cue in a valid trial and at the opposite location in an invalid trial. In a neutral trial, in which the cue appeared at the center overlapping the fixation light, the target could appear at either side of the fixation. The locations of the cue and target were pseudorandomized in order that the cue did not predict where the target would appear.

The subjects were instructed that the cue did not necessarily predict the target's location and that they should make a saccade to the target as quickly as possible after its onset. Subjects initiated a trial by pressing the space bar on the keyboard and were allowed to move at their own pace in the experiment. There were a total of 9 (SOA) \times 2 (direction) \times 3 (cue validity) \times 10 (repetitions) = 540 trials in each experimental block. In case of an error, the trial is aborted and inserted at the end of the "stack." Each subject performed in three blocks, each on a separate day. A practice session of 30 trials was run before the experiment proper.

Data analysis

The trials that had a saccade latency of less than 100 ms were considered anticipatory and were excluded from further analyses. Saccade onset is defined as the time when the eye movement velocity exceeds $30^\circ/\text{s}$. We first compared the latency of the "neutral" or centrally cued trials between normal subjects and ADHD subjects. Second, for each individual subject, the latency difference was obtained between the valid and the invalid trials across all SOAs, separately for rightward and leftward saccades. The latency difference followed a biphasic profile, with the later, positive component being IOR. Likewise, we obtained the "benefit" of spatial cuing by subtracting the latency of neutrally cued trials from that of the validly cued trials and the "cost" of spatial cuing by subtracting the latency of neutrally cued trials from that of the invalidly cued trials. Repeated measures ANOVA was performed to compare in each case the latency difference between the two subject groups. The P values presented are results based on multivariate tests.

Results

General performance

In general, both ADHD and control children could perform the task well, although ADHD children often-times requested more breaks and took longer to complete a session. As an index of the general performance, we examined the frequency of errors (number of error trials divided by the total number of trials) made by the subjects in this behavioral task. The ADHD and control subjects had an average (\pm SD) error rate of $58.1 (\pm 11.2)\%$ and $43.7 (\pm 14.4)\%$, respectively. A repeated measures ANOVA with saccade direction and SOA as within-subject variables and group as between-subject variable showed

that the error rate differed between the two groups ($F_{(1,20)}=6.555$, $P=0.019$), but this difference did not appear to vary with saccade direction ($F_{(1,20)}=1.842$, $P=0.190$, group \times direction interaction) or SOA ($F_{(8,13)}=0.206$, $P=0.984$, group \times SOA interaction). There was also a main effect of SOA ($F_{(8,13)}=34.216$, $P=0.000$): the longer the SOA the higher the error rate. The direction main effect was not significant ($F_{(1,20)}=0.336$, $P=0.569$), nor was the SOA \times direction interaction ($F_{(8,13)}=1.987$, $P=0.130$).

We also examined the response variability of the subjects as another index of their general performance. To this end we computed for each individual the coefficient of variation of the latency of the neutrally cued saccades for each saccade direction and SOA. A repeated measures ANOVA with saccade direction and SOA as within-subject variables and the group as the between-subject variable showed that there is no main effect of group ($F_{(1,20)}=0.130$, $P=0.722$), direction ($F_{(1,20)}=0.632$, $P=0.346$) or SOA ($F_{(8,13)}=1.150$, $P=0.395$). None of the two-way interactions was significant ($F_{(1,20)}=1.934$, $P=0.180$, direction \times group; $F_{(8,13)}=0.401$, $P=0.900$, SOA \times group; $F_{(8,13)}=1.011$, $P=0.473$, direction \times SOA), but there was a significant group \times SOA \times direction interaction ($F_{(8,13)}=3.209$, $P=0.03$).

Latency difference profile and IOR

Table 2 lists the mean values for the latency of neutral, valid, and invalid saccades for both normal and ADHD subjects. First, we compared the latency of neutral saccades. A two-way repeated measures ANOVA with SOA as a within-subject variable showed that, while there was a significant effect of SOA ($F_{(8,13)}=9.234$, $P=0.000$), there was no overall difference in the latency of a neutral saccade between the two groups ($F_{(1,20)}=0.360$, $P=0.555$), nor a significant group \times SOA interaction ($F_{(8,13)}=0.964$, $P=0.502$). Second, to test for a difference in the latency difference profile between groups, we performed a repeated measures ANOVA with SOA and saccade direction as the within-subject variables and group as the between-subject variable. The results showed that there was no significant effect of saccade direction ($F_{(1,20)}=0.028$, $P=0.870$) or direction \times group interaction ($F_{(1,20)}=0.057$, $P=0.813$). We thus combined the data of rightward and leftward saccades and assessed for a group effect in another ANOVA with SOA as a within-subject

Table 2 Saccade latency; mean \pm SD (ms)

Group	Cue SOA	50 ms	83 ms	100 ms	133 ms	167 ms	200 ms	400 ms	700 ms	1,200 ms
Normal	Central	211 (± 30)	196 (± 28)	190 (± 25)	185 (± 24)	188 (± 25)	192 (± 23)	201 (± 26)	187 (± 24)	196 (± 27)
	Valid	200 (± 18)	204 (± 18)	209 (± 26)	222 (± 17)	235 (± 14)	238 (± 14)	242 (± 25)	227 (± 34)	214 (± 30)
	Invalid	251 (± 37)	227 (± 35)	228 (± 40)	211 (± 36)	204 (± 37)	200 (± 37)	210 (± 31)	197 (± 30)	198 (± 29)
ADHD	Central	216 (± 25)	206 (± 33)	198 (± 33)	190 (± 33)	190 (± 35)	193 (± 31)	212 (± 22)	203 (± 24)	198 (± 29)
	Valid	210 (± 24)	205 (± 23)	205 (± 28)	217 (± 24)	222 (± 19)	239 (± 30)	245 (± 33)	231 (± 30)	219 (± 37)
	Invalid	259 (± 28)	239 (± 23)	237 (± 27)	224 (± 27)	218 (± 35)	214 (± 30)	218 (± 35)	212 (± 35)	207 (± 42)

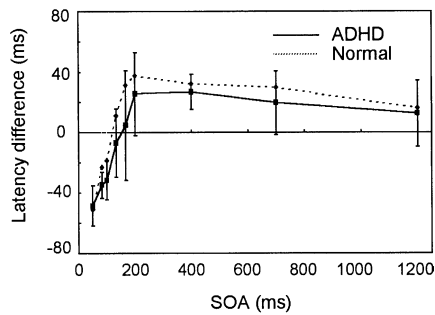


Fig. 2 IOR profile. Each data point represents the mean across subjects at each SOA of the latency difference between valid and invalid trials. Error bars are standard deviations. Neither the magnitude nor temporal profile differs between normal and ADHD subjects

and group as the between-subject variable. The results showed that, while there was a significant effect of SOA ($F_{(8,13)}=44.182$, $P=0.000$), there was no significant difference in the magnitude of latency difference between the two groups ($F_{(1,20)}=2.547$, $P=0.126$), nor a significant group \times SOA interaction ($F_{(8,13)}=0.911$, $P=0.536$). Figure 2 shows the latency difference profiles for both normal and ADHD children. For both groups, the latency difference is negative at an SOA under 100 ms and becomes positive afterwards, with the inhibition peaking around 200 ms.

To further investigate whether IOR differed between ADHD and healthy children, we focused on the inhibitory (positive) component of the latency difference. Although all subjects demonstrated a clear biphasic profile, the latency difference crossed over from negative to positive at different times for each individual subject. We computed the magnitude of IOR for each individual subject in three different ways (sum1) by summing up all the latency differences starting at the first SOA where the difference turned positive, (sum2) by summing up all the positive latency differences starting at the first SOA where the difference turned positive, and (peak) by taking the peak positive latency difference. Repeated measures ANOVA with the saccade direction as a within-subject and group as the between-subject variable showed that there was no effect of saccade direction ($F_{(1,20)}=0.048$, $P=0.828$, for “sum1”; $F_{(1,20)}=0.044$, $P=0.835$, for “sum2”; $F_{(1,20)}=0.176$, $P=0.679$, for “peak”), group ($F_{(1,20)}=2.248$, $P=0.149$, for “sum1”; $F_{(1,20)}=2.711$, $P=0.115$, for “sum2”; $F_{(1,20)}=1.218$, $P=0.283$, for “peak”) or direction \times group interaction ($F_{(1,20)}=0.001$, $P=0.970$, for “sum1”; $F_{(1,20)}=0.084$, $P=0.775$, for “sum2”; $F_{(1,20)}=1.994$, $P=0.173$, for “peak”). On the other hand, although the results did not reach statistical significance, there appeared to be a trend that the magnitude of IOR was slightly smaller in ADHD than in normal children (average of two directions: 169 ms vs. 120 ms, “sum1”; 171 ms vs. 120 ms, “sum2”; 55 ms vs. 47 ms, “peak”).

The results presented so far are concerned with latency difference between validly and invalidly cued trials. We also examined for a group difference in “cost” (latency

difference between invalidly cued and neutral trials) and in “benefit” (latency difference between validly cued and neutral trials). For each of the two measures, a three-way repeated measures ANOVA with saccade direction and SOA as within-subject variables and group as the between-subject variable was performed. The results showed that, for latency cost, only SOA came out significant ($F_{(8,13)}=20.08$, $P=0.000$); all other main effects and interactions were not significant (all P 's >0.21). Likewise, for latency benefit, the only significant effect was with SOA ($F_{(8,13)}=14.65$, $P=0.000$); all other main effects and interactions were not significant (all P 's >0.31). It thus seems that ADHD and control subjects did not differ from each other in terms of latency cost or benefit.

Discussion

Although ADHD children performed at a higher error rate than healthy controls, they were all able to complete this relatively lengthy behavioral task. Moreover, the response variability of both groups of children appeared to be similar. These results suggest that the spatial cuing paradigm can be a useful tool for exploring attention and oculomotor functions in ADHD. Visual inspection of the eye traces showed that the errors comprised failures to maintain fixation and premature saccades made to the cue. To better characterize these errors, we set up operational criteria to delineate different types of errors. For instance, fixation insufficiency is a blink or a non-goal-directed eye movement made during the entire fixation period before the target appears. A goal-directed eye movement is defined as a saccade made either to the cue or to the target (within a spatial window of 2.67° in radius). A distraction error is a goal-directed saccade made to the cue from 100 to 200 ms after its onset. An anticipation error is one that does not fit the definition of a distraction error and a goal-directed saccade made to the cue or the target between the time when the cue appears up to 100 ms after the target appears. Fixation insufficiency, distraction and anticipation each accounted for approximately 80%, 6% and 5% of the total errors in both groups of subjects, with the remaining errors being failures in initiating a saccade within the time window and in landing a saccade correctly on the target. ADHD and healthy children showed a nearly statistically significant difference in fixation insufficiency but not in the other types of errors (data not shown). Note that these results should be considered only as descriptive, as the use of short cue-target onset asynchrony in this behavioral task made it impossible to distinguish in every case whether a saccade was made to a cue or to a target. Moreover, some of the behavioral parameters used in the definitions of the errors were arbitrary. Nevertheless, the finding about fixation insufficiency is consistent with a recent study that reported more intrusive saccades during fixation in ADHD than in control subjects (Gould et al. 2001).

Children with ADHD did not differ from normal subjects in the latency of centrally cued saccades, which is consistent with an earlier report stating that the latency of “pro-saccades” is not altered in this disorder (Mostofsky et al. 2001). This finding suggests that the oculomotor circuitry involved in the generation of visual target-guided saccades is not compromised in these patients. Moreover, the profile of latency difference between validly and invalidly cued saccades in the ADHD subjects appears to follow that of control subjects fairly well. When the target follows the cue closely, the saccades that are directed to the target at the pre-cued spatial location are facilitated. As the onset asynchrony increases, the facilitatory effect turns into inhibitory; saccades directed to the pre-cued target location then become inhibited. This biphasic facilitation-inhibition profile is clearly visible in ADHD as well as in control children. On the other hand, although the overall latency profile does not appear to differ between the two groups of subjects, there perhaps is a trend that the overall magnitude of the positive latency difference (namely, IOR) is larger in control than in ADHD subjects. Indeed, isolating this component by three different measures, we show that this difference between the two subject groups is fairly consistent. It thus appears that, while the profile of the latency difference between validly and invalidly cued trials appears to be intact, the magnitude of IOR perhaps is slightly diminished in ADHD.

Studies investigating the neural correlates of IOR have mostly suggested a role for the cortico-collicular network in mediating this inhibitory effect. A recent functional magnetic resonance imaging study demonstrated that IOR involves activation of the supplementary and frontal eye fields and the supramarginal gyrus, structures that are important in oculomotor control and attentional orienting (Lepsien and Pollmann 2002). Examining a patient with a hemorrhagic infarct confined to the right superior colliculus (SC), Sapir and colleagues demonstrated a less efficient IOR when the visual stimulus was presented to the temporal visual hemifield of the left eye or the nasal visual hemifield of the right eye, compared to when the left, intact SC was engaged (Sapir et al. 1999). This finding complements earlier evidence and suggests a direct role of the SC in the generation of IOR. Therefore, the current result that IOR is grossly intact in ADHD children suggests that this network of brain areas is not severely impaired in coordinating eye movement and the shifting of attention that subserves this inhibitory function. This finding overall adds another dimension to our understanding of attention deficit in ADHD children.

Examining the latency cost and benefit, we found that there is no difference between the two groups, nor was there a directional effect (right-left asymmetry). These results appear to be at odds with several earlier studies that reported asymmetrical attention function in ADHD as revealed in spatial cuing tasks (Carter et al. 1995; Epstein et al. 1997; McDonald et al. 1999; Nigg et al. 1997). This discrepancy could reflect methodological differences between the current and earlier studies. While manual

reaction time was the dependent measure in the previous experiments, we monitored eye movement in the current study. As has been demonstrated recently, response modality modulates the magnitude and temporal profile of IOR (Briand et al. 2000; Li and Lin 2002) and presumably would alter the latency cost and benefit as well. Moreover, while the earlier studies employed a paradigm with more valid than invalid trials, the two trial types are equal in number in our experiment. In other words, the spatial cue in our behavioral task does not predict the location of the target, but it does (with varying predictabilities) in the previous experiments. Unpublished data in our laboratory have shown that this factor too could have an effect on the latency of validly cued trials. When the peripheral cue predicts the location of the target 100% of the time, saccades made to the cued location do not increase in latency, compared to the neutrally cued saccades (Li, unpublished data). Another task variable is whether attention is directed with an exogenous or endogenous cue. As has been demonstrated earlier, IOR does not obtain in an endogenous cuing task (Li and Lin 2002). These behavioral variables would thus have to be controlled for before the results obtained in the different studies can be compared. Finally, the present study only tested a small sample of subjects, so these results should be considered as preliminary. Further experiments with more subjects and with designs testing the same subjects with both oculomotor and manual responses are required to resolve this issue.

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