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RESEARCH ARTICLE

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Inhibitory control of eye movements during oculomotor countermanding in adults with attention-deficit hyperactivity disorder

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Abstract Children with attention-deficit hyperactivity disorder (ADHD) are impulsive, and that impulsiveness can be measured using a countermanding task. Although the overt behaviors of ADHD attenuate with age, it is not clear how well impulsiveness is controlled in adults with ADHD. We tested ADHD adults with an oculomotor countermanding task. The task included two conditions: on 75% of the trials, participants viewed a central fixation marker and then looked to an eccentric target that appeared simultaneous with the disappearance of the fixation marker; on 25% of the trials, a signal was presented at variable delays after target appearance. The signal instructed subjects to stop, or countermand, an eye movement to the target. A correct movement in this case would be to hold gaze at the central fixation location. We expected ADHD participants to be impulsive in their countermanding performance. Additionally, we expected that a visual stop signal at the central fixation location would assist oculomotor countermanding because the signal is presented in the "stop" location, at fixation. To test whether a central stop signal positively biased countermanding, we used a three types of stop signal to instruct the stop: a central visual marker, a peripheral visual signal, and a non-localized sound. All subjects performed best with the central visual stop signal. Subjects with ADHD were less able to countermand eye movements and were influenced more negatively by the non-central signals. Oculomotor countermanding may be useful for quantifying impulsive dysfunction in adults with ADHD especially if a non-central stop signal is applied.

Keywords Attention-deficit hyperactivity disorder · Saccades · Response inhibition

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Introduction

Impulsiveness, or deficient control of inhibition (Cairney et al. 2001; Crosbie and Schachar 2001; Logan et al. 1997; Nigg 1999; Schachar and Logan 1990; Schachar et al. 2000; Solanto et al. 2001; Tannock 1998; Williams et al. 1999) is perhaps the core deficit (Quay 1997) in children with attention-deficit hyperactivity disorder (ADHD) [Diagnostic and Statistical Manual IV (DSM-IV), American Psychiatric Association 1994]. One paradigm used to study inhibitory control in children with ADHD is the countermanding task (e.g., Schachar and Logan 1990). In countermanding, most trials require a movement to a target. On a small percentage of trials, however, a "stop" signal occurs after varying delays indicating that the target-directed movement should be countermanded or stopped. When the delay between target appearance and stop-signal appearance is brief, participants usually inhibit a movement to target, but as the delay increases, subjects do not always stop (Cabel et al. 2000; Hanes and Carpenter 1999). The ability to countermand is a function of the various stop-signal delays, and differences in inhibitory control may be seen in the slope of that function.

Inhibitory control in countermanding changes with age in the non-clinical population (Kramer et al. 1994; Williams et al. 1999), and task performance varies with impulsiveness (Logan et al. 1997), making it a useful task for evaluating children with ADHD. ADHD children are impaired at this task compared to children with no psychopathology or with conduct disorder (Schachar et al. 2000). Impulsive, overt ADHD behaviors attenuate with age (Barkley 1998; Biederman et al. 2000; Sachdev 1999) and, therefore, performance differences in children with ADHD may not be replicated in adults. The countermanding task may be a tool that provides the sensitivity to assess impulsiveness in adults with ADHD. We tested ADHD adults using an oculomotor countermanding task and compared their performance with that of age and sex-matched control participants.

Oculomotor countermanding often uses a central visual stimulus (e.g., the re-appearance of the central fixation marker) to signal a stop (e.g., Cabel et al. 2000; Hanes and Carpenter 1999). The appearance of a central visual stimulus on the fovea halts a saccade in several ways (Cabel et al. 2000). For example, the abrupt onset attracts attention reflexively; hence, participants will make an eye movement toward the newest stimulus that coincidentally appears at the stop location, the place where the fixation marker had appeared. Alternatively, an eye movement can be stopped by a voluntary restriction of eye movement in order to follow the instruction to stop moving. By contrast, a stop signal that appears away from the central foveal location will still draw attention, but to the stop signal location and not to the location required for a stopped eye movement; hence, for non-central stop signals, control of impulsive behaviors plays a more important role in effective stopping. Cabel and colleagues demonstrated that, in an oculomotor countermanding task, a central visual stop signal positively biased stop success when compared to a peripheral auditory stop signal (Cabel et al. 2000). We extend their approach by also including a visual stop signal that was not presented at the fovea.

We hypothesize that responses to the peripheral visual stop signal will match responses to an auditory stop signal because neither attracts the participant to the correct stopping location, at fixation. Without the abrupt onset at the fovea, responses should be less successful generally, but we expect that ADHD participants will be more disadvantaged by the absence of the central visual stop stimulus because they will be forced to rely on their limited impulse control in order to succeed.

Materials and methods

Participants

The experimental procedures were reviewed and approved by the Queen's University Human Research Ethics Board. All participants provided informed consent. Subjects completed two 1-h sessions and were paid $10 for each session. All participants had normal or corrected-to-normal visual acuity.

Fourteen ADHD adults (eight men, mean age 34.6 years; six women, mean age 29.5 years) were recruited from the subject pool at the eye-movement laboratory at Queen's University (Munoz et al. 1998) or from the local community via notices placed in doctors' offices. Each adult in the group had an existing diagnosis of ADHD that was confirmed by a clinical psychologist and based on DSM-IV interview criteria (American Psychiatric Association 1994). Three of the ADHD group (all women) were also under treatment for comorbid conditions: one for depression, one for anxiety, and one for severe back problems. The 14 non-clinical control subjects were matched to the ADHD subjects in age and sex (men, mean age 34.4 years; women, mean age 30.3 years). ADHD and control participants were assessed using Brown's Attention Deficit Disorder Scale (BADDS), administered by the same clinical psychologist. The ADHD group produced reliably higher BADDS scores (mean ±SE of 77.4±2.4; men 79.3, women 73.7), than did non-clinical controls (23.8±2.2; men 24.1, women 23.5), $t_{(24)}$=43.5, P<0.001. BADDS scores greater than 50 indicate a strong likelihood of ADHD; no participant with ADHD scored below 64, and no control participant scored above 34.

Materials

Visual stimuli were presented on a 17-inch monitor using video resolution of 640×480 pixels and a frame rate of 60 Hz. Eye-movement data were collected using a video-based eyetracker (Eyelink; SR Research Ltd., Mississauga, ON, Canada) that was mounted on a subject's head with an adjustable headband. The eyetracker uses infrared cameras to track the movement of the left pupil, measuring vertical and horizontal eye position and pupil size at a sampling rate of 250 Hz. It also provided spatial information about head position for head motion compensation. Acceleration and velocity thresholds were set to detect saccades greater than 0.15°.

Procedure

Participants sat approximately 60 cm from the display monitor. Each trial started with the presentation of a 1-cm white octagonal fixation spot subtending a visual angle of 0.95° (1.40 cd/m^2) centered on a dark background. Figure 1 illustrates the paradigm. When ready, subjects initiated the trial with a button press. After a random delay that varied on each trial between 200 and 1000 ms, the fixation marker disappeared and a green target (0.72 cd/m^2), equal in size and shape to the fixation marker, appeared randomly 5° to the left or right of center. Participants were instructed to look to the eccentric target when it appeared unless a stop signal was presented. In the presence of a stop signal (25% of trials), participants were instructed to suppress a saccade to the eccentric target. Within a block of trials, stop signals consisted of a visual signal (12.5%) or an auditory signal (12.5%) presented with equal probability. The red visual stop signal changed across blocks of trials: in the foveal stop-signal condition, it was a 1-cm octagonal marker (1.07 cd/m^2) centered on the monitor; in the peripheral stop-signal condition, it was a 0.1-cm wide continuous band (1.21 cd/m^2) approximately 0.6 cm from all four edges of the monitor presented around the edge of the computer screen. The auditory stop signal used in both visual stop stimulus conditions was a broadband noise burst (80 dB) emitted for 100 ms[1] from a speaker located approximately 2 m overhead. When it occurred, the stop signal followed target appearance after delays of 0, 50, 150, and 350 ms. Delay duration matched the range of delays used by Cabel et al. (2000) with one exception: they used ten equal-sized increments of delay; we doubled the difference between delays at each increment, and used only four values. The visual stop signal and the eccentric target then remained illuminated for the duration of the trial.

Design

The experiment included two blocks, one for each type of *visual* stop signal. Each session had a total of 256 trials reflecting four replicates of the factorial combination of target direction (left versus right), stop-signal delay (0, 50, 150, 350 ms), stop-signal type (visual versus auditory) and trial type (GO:STOP ratio 3:1). Trials were presented in a random order that was determined separately for each subject and each block. Each block lasted approximately 45 min and the order of the blocks was randomized.

Analysis

We report ZRFT, the standardized relative finishing time, a function that is normalized, and therefore centered on zero, which was calculated for each participant across each delay interval. This function is based on a race model (Logan and Cowan 1984) between the independent responses to GO to the target and to STOP that movement (see *inset* of Fig. 1). In the model, GO responses

[1] Anecdotal evidence from pilot work revealed that longer noise bursts for the auditory signal were offensive to participants.

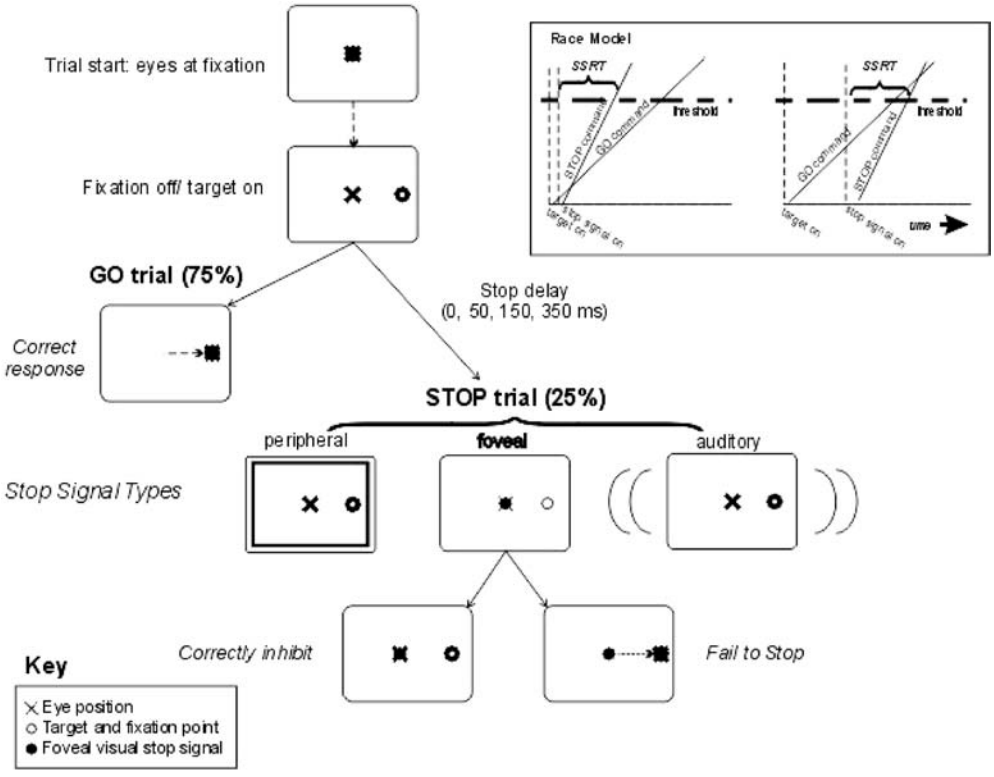

Fig. 1 The countermanding paradigm. Participants fixate a central marker and, on *GO trials*, look to an eccentric target when it appears. However, on a small percentage of trials (*STOP trials*), a signal to stop the eye movement appears at varying delays following the appearance of the target. At short delays, participants correctly inhibit an eye movement but at longer delays they fail to stop the eye movement. Performance at the task is explained in terms of a race model (*inset*) (*SSRT* stop-signal reaction time)

start when the target appears; STOP responses start when the stop is signaled. At short stop-signal delays, the STOP response has time to reach a critical threshold, or finish the race, before the GO response reaches criterion. As a result, participants are able to countermand or inhibit an eye movement. As the stop-signal delay increases, the time before the STOP response can begin the race is increased, and the GO response reaches threshold (i.e., finishes the race) first; hence, participants are less likely to stop at the longer delays. In the Logan and Cowan (1984) race model, each participant has a unique latency from stop signal to STOP response. That latency can be estimated by integrating the GO-trial distribution (the measure of a participant's typical GO latency) until the integral matches the probability of stopping at each stop-signal delay. This latency estimate is the stop-signal reaction time (SSRT; Logan and Cowan 1984), the length of the typical STOP response in the race model. To compare performance across participants, we normalize the relative finishing times (ZRFT) of the GO/STOP race by considering average reaction time divided by the variability in those times. ZRFT also takes into account individual SSRT at each stop-signal delay. ZRFT was normalized using Logan and Cowan's (1984) formula:

$$ZRFT = \frac{MSRT - SSD - SSRT}{SD}, \quad (1)$$

where MSRT is the mean GO-trial saccadic reaction time (SRT), SSD is the stop-signal delay, and SD is the standard deviation of the GO-trial SRT distribution.

The slope of the best-fit linear regression through the ZRFT function indicates the relationship between the two response processes in the race model. Because the function has accounted for speed and accuracy differences, the slope should be approximately the same for all conditions. If the slope is not the same, it is thought to reflect processing that is driven either by the participants, due to some pathology, or by some unique stimuli. For example, a change in slope might occur if an observer takes longer to respond on GO trials but does not benefit from this strategy with improved accuracy on STOP trials. Logan and his colleagues (De Jong et al. 1990; Logan and Cowan 1984; Osman et al. 1986; Schachar and Logan 1990) attribute slope changes to the inhibitory control of a movement. For example, Schachar and Logan (1990) found that the slope of the ZRFT function for ADHD children with pervasive hyperactivity was less steep than slopes found for groups of children with various other psychopathologies or with no psychopathology. As a result, they claimed that the slope difference for ADHD children with pervasive hyperactivity indicated distinct control processes, and thus, that the group should be considered separately for diagnostic purposes. Cabel et al. (2000) also found slope differences related to the modality of the stop signal; a steeper slope occurred when the stop signal was a central visual stimulus compared to a non-localized auditory stimulus.

A GO trial was scored as correct when a saccade with an amplitude greater than 3° was made in the direction of the target, i.e., towards the same half of the computer monitor as the target. The reaction time analysis program, RTSYS Version 1.0 (Heathcote 1996) provided summary data for correct GO trials on each participant's SRT distribution. Analyses of variance were performed on the mean. STOP-trial errors occurred when participants moved their eyes under instructions to maintain their gaze at fixation.

Results

The primary measure of interest in this study is ZRFT, a standardized measure of the relative finishing time for each individual (see Analysis section). The slope of the ZRFT function can differentiate impulsive behaviors. ZRFT is calculated using the cumulative GO-trial SRT distribution, countermanding accuracy on STOP trials, and an estimate of internal stop time, SSRT. We report these measures before discussing ZRFT.

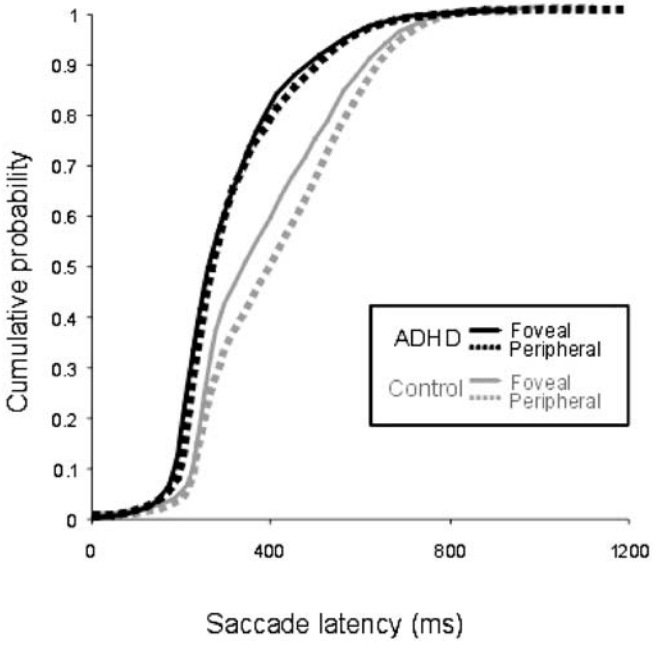

Fig. 2 Cumulative saccadic reaction time GO-trial distributions for the attention-deficit hyperactivity disorder (ADHD) and control groups in blocks defined by visual stimulus type (foveal or peripheral)

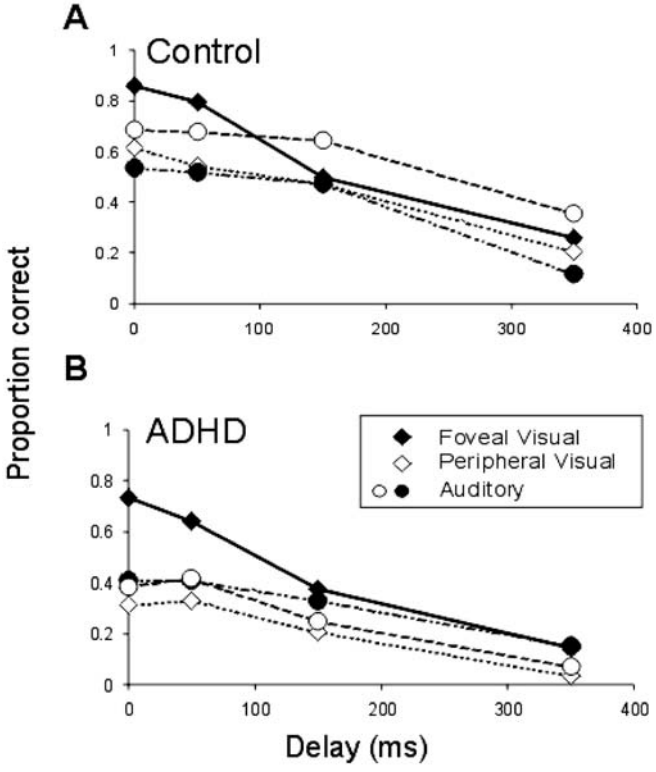

Fig. 3A–B STOP-trial accuracy as a function of stop-signal delay for control (**A**) and ADHD (**B**) adults under foveal (*filled diamonds*) and peripheral (*open diamonds*) and auditory (*open* and *filled circles*) stop-stimulus trials

GO-trial saccadic reaction time

Figure 2 shows cumulative GO-trial SRT distributions for the ADHD and control groups under the two experimental blocks defined by the type of visual stop stimulus. Note that participants do not see a stop signal on GO trials; however, by discriminating across blocks we can assess whether willingness to GO was affected by the type of stop signal that *might* appear on any trial. ADHD response times were shorter (mean ±SE 324±18 ms) than control SRT (400±29 ms), $F_{(1,13)}$=4.63, P=0.05. Controls took less time to respond in the foveal-stimulus block (382±27 ms) than in the peripheral-stimulus block (417±32 ms), $F_{(1,13)}$=5.23, P<0.05, whereas the ADHD group did not show altered response times across blocks (foveal 325±18 ms versus peripheral 323±19 ms). Target direction did not affect SRT for either group, $F_{(1,13)}$<1, P>0.50.

STOP-trial accuracy (the inhibition function)

Figure 3 illustrates the probability of withholding a saccade given a stop signal for each delay for the control (Fig. 3A) and ADHD (Fig. 3B) groups. As expected, accuracy decreased across stop-signal delay. When scores were averaged across delay, control subjects were more accurate (mean proportion correct ±SE 0.53±0.09) than ADHD participants (0.33±0.09), $F_{(1,52)}$=10.2, P<0.01. But in other ways, the two groups showed similar patterns of performance. Accuracy was highest when the stop signal was the central, foveal visual stimulus, but only at the short delays between 0 and 50 ms; at longer delays, the foveal stimulus did not yield accuracy different from the non-foveal stop signals. The short/long delay differences resulted in an interaction among the factors of stop-signal type (visual versus auditory), delay (0, 50, 150, and 350 ms) and visual stimulus type (foveal versus peripheral), $F_{(3,156)}$=5.9, P<0.001. Thus, the central visual stop signal facilitated countermanding for both groups, but only at short delays. At long delays, stop accuracy was poor for all stop-signals types.

Stop-signal reaction time, SSRT

SSRT, an estimate of the average time needed to inhibit a response to the target (see Analysis section), was compared across groups and used to calculate the ZRFT function (see below). Figure 4 shows mean SSRT ±SE for each visual stop-signal block (foveal or peripheral) and stop-signal mode (visual or auditory). The shortest SSRTs were obtained for the foveal visual stop signal, and the longest for the peripheral visual stop signal with the auditory stop-signal SSRTs being intermediate. This was confirmed in the statistical analysis: the visual SSRT was shorter in the foveal block (mean ±SE 226.1±40.4 ms) than the auditory signal (foveal 315.2±45.4 ms), and the

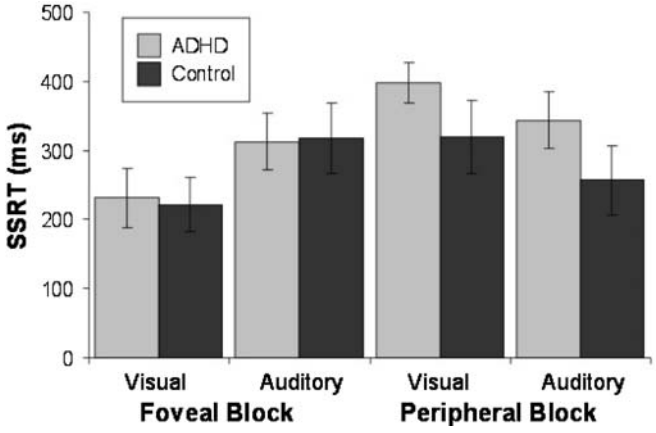

Fig. 4 Mean stop-signal reaction time (SSRT) and its standard error as a function of stop-signal mode (visual or auditory), visual stop-stimulus conditions (foveal or peripheral) and group (ADHD adults or controls)

peripheral visual SSRT was longer (359.1±43.7 ms) than the auditory signal (peripheral 300.3±47.0 ms), $F_{(1,13)}$=94.21, P<0.0001. When SSRT was collapsed over the visual and auditory modes, control participants showed no reliable difference between visual blocks (foveal 269.5±46.5 ms, peripheral 288.3±52.1 ms). The ADHD group matched control performance in the foveal block (foveal 271.9±42.6 ms), but in the peripheral block, ADHD SSRT (peripheral 371.0±35.8 ms) was greater than for controls or for ADHD in the foveal block. The single greater SSRT for the ADHD group in the peripheral blocks resulted in an interaction between the factors of group and visual stop-stimulus block, $F_{(1,13)}$=4.68, P<0.05. The SSRT estimate was shortest under the foveal visual condition and mode, and ADHD participants were most disadvantaged by the peripheral stop signals.

The normalized inhibition function, ZRFT

Figure 5 illustrates ZRFT functions for the control (Fig. 5A) and ADHD groups (Fig. 5B) under conditions defined by visual stimulus type and stop-signal mode. Figure 6 shows the same data collapsed across delay to clarify contrasts between groups; Fig. 6A shows mean ZRFT as a function of stop-stimulus type and Fig. 6B illustrates the slope of the best-fit linear regression on the ZRFT function. Like the standard normal, Z, the ZRFT function is likely to be centered at zero. All four of the control functions in Fig. 5 have means around zero; however, for the ADHD group only the foveal stop signal ZRFT function is close to zero, implying non-normal performance for the ADHD group on trials involving non-foveal stop signals. This is clearer in Fig. 6A; there are large negative mean ZRFT values for the ADHD group under the peripheral visual and auditory stop signals. Further, the slopes of the foveal functions are steeper than the slopes for the auditory and peripheral functions, and

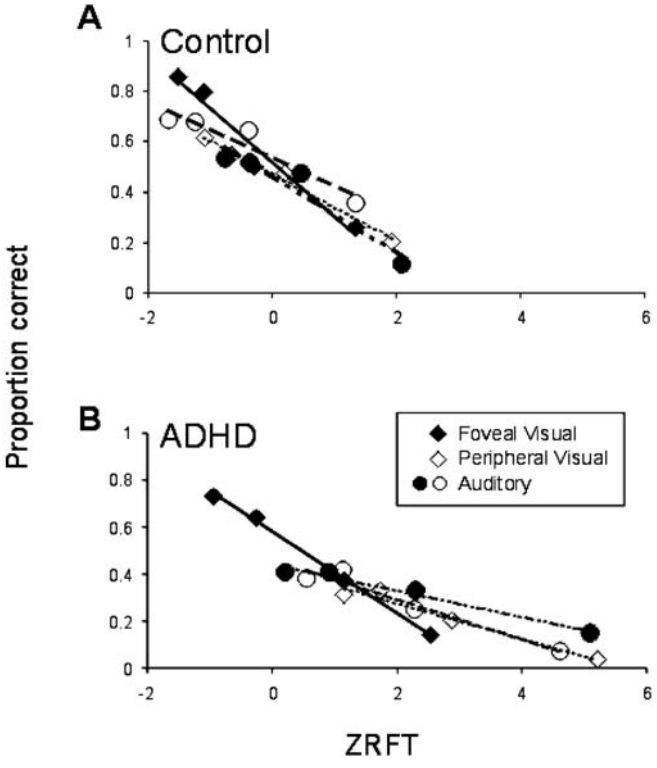

Fig. 5A–B Standardized relative finishing time (ZRFT) functions for controls (**A**) and ADHD (**B**) adults under foveal (*filled diamonds*) and peripheral (*open diamonds*) and auditory (*open* and *filled circles*) stop-stimulus trials

these functions appear to have similar slopes (Fig. 6B). A within-subjects analysis of variance on the ZRFT values across delays using the factors stop-signal mode (visual versus auditory), visual stop-stimulus block (foveal versus peripheral) and group (ADHD versus control) confirmed these observations. The control participants' data normalized closer to zero than the ADHD group, $F_{(3,39)}$=3.94, P<0.05. In the foveal visual stop-signal block, ZRFT was larger for visual signals (Fig. 5, *filled diamonds*) than for auditory signals (*filled circles*), a difference not seen under the peripheral visual stop-stimulus block (*open symbols*), resulting in an interaction between stop-signal mode and visual stop-stimulus block, $F_{(1,13)}$=31.17, P=0.0001. Control subjects showed steeper (larger) ZRFT functions than ADHD participants, implying a stronger association between successful stopping and GO-trial performance, $F_{(1,13)}$=11.24, P<0.01. Both groups had a larger ZRFT slope in the foveal visual condition than in the remaining non-foveal conditions, resulting in an interaction between mode and group, $F_{(1,13)}$=4.26, P=0.05. The normalized scores showed distinct performance in both groups when the foveal visual stimulus was presented, which was the only condition that provided an image to hold gaze. Thus, the slope of the normalized measure of Logan and Cowan (1984) indicated a deficit in inhibitory control for ADHD participants, except under the foveal visual stop-stimulus condition.

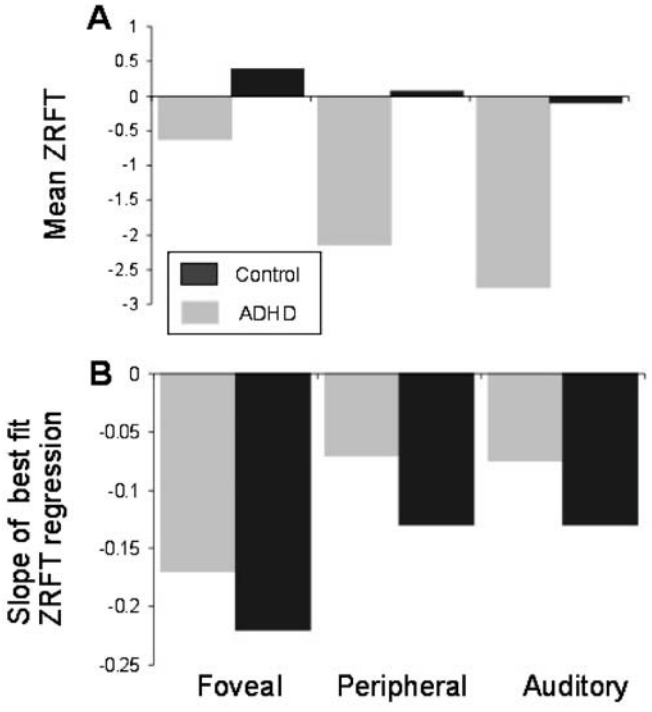

Fig. 6A–B Mean standardized relative finishing time (ZRFT) (**A**) and slopes of the best-fit ZRFT regression (**B**) as a function of stop stimulus for control and ADHD adults

Discussion

The oculomotor countermanding task demonstrated that (1) ADHD participants made more impulsive eye movements than age-matched controls, and (2) a central visual stop stimulus directed towards the fovea positively biased performance. ADHD participants were faster to respond to a target (Fig. 2) but their speed was costly: they were more likely to fail to countermand an eye movement on STOP trials (Fig. 3). We used Logan and Cowan's (1984) measure, ZRFT, to account for the trade-off between speed and accuracy; also, we used its slope to assess inhibitory control. For both groups, the slope of the ZRFT function was most negative under the foveal stop-signal condition implying stronger impulse control, i.e., there is a stronger link between STOP accuracy and the foveal visual signal. Hence, like controls, ADHD participants were more able to suppress impulsive eye movements only when an abrupt visual stimulus onset appeared at the fovea. Without the foveal stimulus, both groups showed a change in ZRFT slope; the ADHD group showed the least negative slopes and the most negative mean ZRFT under non-foveal conditions, whereas the control group's mean ZRFT was consistently near zero. We conclude that ADHD participants were less able to override impulsive eye movement commands without the help of an abrupt foveal stimulus.

This finding is consistent with results from studies of children with ADHD. Schachar and Logan (1990) measured button-press response time in a countermanding task and found that the slope of the normalized inhibition function, ZRFT, was less steep for children with ADHD in combination with pervasive hyperactivity than in children with other psychopathologies and those with no psychopathology. In contrast, in their meta-analysis of eight studies using the stop-signal paradigm, Oosterlaan and colleagues found that ADHD children were less able to inhibit a response on a Stop trial compared with controls, had slower mean SSRT, but they had no slope differences in the ZRFT function (Oosterlaan et al. 1998).

Our findings differ from earlier work using oculomotor countermanding in two ways. Firstly, mean SSRT values were much longer than those described previously (Arress and Carpenter 2001; Cabel et al. 2000; Hanes and Carpenter 1999). Cabel et al. (2000) used the same apparatus that was used in the current study. In their experiment, SSRT values were between 94 and 126 ms whereas our SSRT values ranged from 220 to 400 ms. The difference may result first from the number and range of stop-signal delays used in the two studies, and second from age differences between the participant groups. Cabel et al. (2000) used ten delays, nine of them occurring before or at 250 ms. As a consequence, subjects' exposure to long delays occurred on only 10% of STOP trials. In contrast, we used only four delays and the longest delay, 350 ms, occurred on 25% of the STOP trials. A greater proportion of long delays may have encouraged a "wait-and-see" strategy in our participants. Such a strategy would increase GO-trial reaction time, and, as a consequence, increase SSRT. Moreover, participants in the Cabel et al. (2000) study ranged in age from 22 to 25 years, whereas our subjects were on average almost a decade older (range 22–54), and there is some evidence that inhibitory control is age-dependent (Kramer et al 1994; Williams et al. 1999).

Secondly, Arress and Carpenter (2001) found no difference in SSRT between central and peripheral stop signals in an oculomotor countermanding task. Participants in the Arress and Carpenter (2001) study were aged 20 to 26 years, but included one participant aged 54. Therefore, like the Cabel et al. (2000) study, their subjects were younger, and their one older subject was an author of the study. Also, both studies used more than twice the number of trials than were used in the current study. Thus, practice may have also speeded their stop-signal reaction times. Arress and Carpenter (2001) used a potential target as their peripheral stop signal; thus, participants were already monitoring target and stop-signal locations for the potential target. Their punctate target was easily localized, unlike our peripheral visual stop signal, which was generalized around the entire computer monitor. In the current study, participants monitored the same two target locations as in the Arress and Carpenter (2001) study (because the target could appear either left or right of the fixation point) but, in addition, our participants had to monitor the very periphery of the computer monitor and listen for the auditory stop signal. These important differences in experimental design regarding the peripheral stop signals could account for the different findings.

Finally, Arress and Carpenter (2001) used a GO:STOP ratio of 6:1, whereas we had more frequent stop trials (GO:STOP ratio 3:1). The frequency of STOP trials in the current study could have induced participants to slow down on GO trials as a precaution against errors.

Finally, in earlier studies (e.g., Schachar and Logan 1990), the experimenters adjusted the timing of the stop-signal delay for each subject to guarantee STOP performance. Thus, performance variability was restricted by the experimenters. We chose not to restrict ADHD performance arbitrarily, and this, too, may have affected SSRT.

Countermanding and brain function

By definition, symptoms of ADHD include inattentiveness, impulsiveness, and sometimes hyperactivity (DSM-IV). The condition has been characterized as a disorder of executive function (Castellanos 1997), and executive function is well measured with the countermanding task (Solanto et al. 2001). Two obvious reasons why ADHD persons might have more trouble with the countermanding task are: first, they are not vigilant—a common indicator of ADHD is poor performance on the continuous performance task, a task of vigilance (Lezak 1995)—and, second, even when vigilant, they may be less able to switch attention rapidly, and hence they cannot "recover" from identifying the target in time to correctly detect the stop signal (Hollingsworth et al. 2001). A third explanation is that people with ADHD have less stable gaze control (Munoz et al. 1999, 2003).

Rubia et al. (1999) used functional magnetic resonance imaging (fMRI) to reveal reduced activity in the prefrontal cortex of boys with ADHD during a countermanding task. Mehta et al. (2000) showed that methylphenidate, a drug known to enhance cognitive performance and commonly prescribed for people diagnosed with ADHD, reduced blood flow to the dorsalateral prefrontal cortex and to the posterior parietal cortex, areas of the brain known to influence inhibition that is necessary for the suppression of reflexive saccades. Using fMRI, Vaidya et al. (1998) found ADHD children had atypical frontal-striatal function. Brandeis et al. (1998) found increased neuroelectric activity in the frontal lobes of ADHD children performing a countermanding task. Areas of the dorsolateral prefrontal cortex are instrumental in the control of impulses, or inhibitory control (Fuster 1997; Stuss and Levine 2002), and in the control of eye movements (Carpenter 1988; Leigh and Zee 1999). These areas are smaller in children with ADHD (Swanson et al. 1998).

To countermand or halt initiation of a saccade requires a signal to overcome pre-saccadic processing. Central and peripheral stop signals likely elicit differing activity patterns in the brain. The central stop signal can enhance activity in brain areas controlling saccade suppression either via direct inputs from visual areas with a foveal representation or via executive processing following interpretation of the suppression signal. In other words, the foveal stop signal can map directly onto fixation neurons in the superior colliculus and onto omnipause neurons in the paramedian pontine reticular formation (Everling et al. 1998; Munoz and Wurtz 1993a). The peripheral and auditory stop signals cannot access the fixation system as directly, and instead, these signal must be processed for interpretation before being remapped onto the fixation system. Such processing presumably requires prefrontal cortex (Fuster 1997; Guitton et al. 1985; Pierrot-Deseilligny et al. 1991).

Most models of saccadic reaction time attribute variations in SRT to either changes in the rate of accumulation toward a threshold and/or variations in baseline activity at the time of target appearance (Carpenter and Williams 1995; Kopecz 1995; Trappenburg et al. 2001). Using an oculomotor countermanding task, Hanes and Schall (1996) found that neurons in the primate frontal eye field (FEF) have a fixed threshold of discharge rate that is achieved 10–20 ms before saccade initiation. Variability in SRT was related to a variable rate of rise in discharge rate toward this threshold. More recently, Hanes and Paré (1998) have made similar observations for saccade-related neurons in the intermediate layers of the primate superior colliculus (SC). The sudden appearance of a visual stimulus on the fovea will elicit a robust phasic visual response among fixation neurons in the SC and FEF (Krauzlis et al. 1997; Munoz and Wurtz 1993a, 1993b) and this response will interact locally with the developing saccade signal via lateral inhibitory connections to delay or prevent saccade initiation (Meredith and Ramoa 1998; Munoz and Istvan 1998). Such a mechanism does not require input from prefrontal cortex, and is therefore presumably intact in ADHD. Therefore, it is not surprising that ADHD participants had no difficulty in oculomotor countermanding responses with a foveal stop signal.

Appearance of the peripheral or auditory stop signal is not likely to lead to direct activation of fixation neurons in the FEF and SC in the same manner as a foveal stop signal. Additional processing is therefore required involving prefrontal cortex and executive function to interpret these stimuli as stop commands before being relayed in a top-down manner to pre-oculomotor areas such as the FEF and SC to prevent saccade initiation. These higher centers may include structures in the frontal cortex and basal ganglia that are believed to be abnormal in ADHD (Swanson et al. 1998). Therefore, ADHD subjects should be expected to have abnormal countermanding abilities with non-foveal stop signals that require higher brain areas for interpretation, and these are dysfunctional in ADHD. Consequently, the ADHD subjects' abnormal performance with the peripheral visual and auditory stop signals may be the result of frontostriatal pathophysiology.

An alternative viewpoint is that both visual stop signals and the auditory stop signal are endogenous cues, i.e., they provide indirect information to stop an eye movement. The foveal stop signal is also an exogenous

cue, directing the eye to the location required when an eye movement is stopped. Hence, the foveal signal provides participants with more attentionally accessible information, making it easier to stop.

Conclusion

The oculomotor countermanding task confirmed a deficit in inhibitory control in adults with ADHD that has been shown previously in children. The deficit was especially severe when the signal to stop an eye movement was not at the fovea, and therefore when a saccade could not inhibited reflexively. Oculomotor countermanding with various stop signals may provide an important means to assess inhibitory control in ADHD.

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