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Impaired visuomotor adaptation in adults with ADHD

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Abstract

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent psychiatric disorder in children that often continues into adulthood. It has been suggested that motor impairments in ADHD are associated with underlying cerebellar pathology. If such is the case, individuals with ADHD should be impaired on motor tasks requiring healthy cerebellar function. To test this, we compared performance of individuals with ADHD and ADHD-like symptoms with non-ADHD controls on a visuomotor adaptation task known to be impaired following cerebellar lesions. Participants adapted reaching movements to a visual representation that was rotated by 30°. Individuals with ADHD and those with ADHD-like symptoms took longer to correct the angle of movement once the rotation was applied relative to controls. However, post-adaptation residual effect did not differ for individuals with ADHD and ADHD-like symptoms compared to the control group. These results are consistent with the hypothesis that mild cerebellar deficits are evident in the motor performance of adults with ADHD.

Keywords

ADHD; adaptation; cerebellum; reaching

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is one of the most prevalent psychiatric disorders. While first established in the 1970's as a disorder in children, the diagnostic criterion for ADHD was expanded to include adolescents and adults (Conrad and Potter 2000). Approximately 65% of ADHD cases persist into adulthood (Biederman et al. 2012) and about 8 million adults in the United States alone have a diagnosis of ADHD (Kessler et al. 2006).

ADHD has well-established and recognizable behavioral symptoms such as excessive motor activity, distractibility, restlessness, and impulsivity (American Psychiatric Association

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Conflict of Interest

The authors have no conflicts to declare.

2000). Less well known is that a large proportion of individuals with ADHD have motor difficulties. For example, when children with ADHD and matched controls were assessed with the Movement Assessment Battery for Children (MABC), which examines manual dexterity and balance, individuals with ADHD performed worse than controls. Moreover, motor difficulties were more common in individuals with ADHD and many were borderline for need of intervention (Piek, Pitcher and Hay 1999). Children with ADHD exhibit increased variability in movement timing and fail to correct for timing errors in the open-loop manner that control participants employ (Zelaznik et al. 2012). Additionally, children with ADHD are more likely than controls to be rated as clumsy by both medical professionals and teachers on a questionnaire of motor-dysfunction (Kadesjö and Gillberg 1998), have worse handwriting (Racine et al. 2008), and have more frequent developmental motor delays (Yochman, Ornoy and Parush 2006a; Yochman, Ornoy and Parush 2006b). While little is known of motor deficits in adults with ADHD, a similar deficit as that seen in children has been suggested (Carr, Nigg and Henderson 2006; Malloy-Diniz et al. 2007).

Motor impairments in the ADHD population may be related to abnormalities in cerebellar structure and function. Children and adolescents with ADHD have significantly reduced grey matter volume within the cerebellum (Castellanos et al. 2002; Carmona et al. 2005; Mackie et al. 2007; Valera et al. 2005) and this reduction has been associated with poorer performance on a response inhibition task (McAlonan et al. 2009). In addition to structural differences in the brain, diffusion tensor imaging has shown abnormalities in white matter microstructure within the cerebellum in children with ADHD, possibly reflecting reduced axonal integrity (Nagel, et al. 2011). Additionally, hypoactivation of corticocerebellar circuits in children and adolescents with ADHD is associated with impaired motor timing (see review: Hart et al. 2012) and motor response inhibition (Schulz et al. 2004; Suskauer et al. 2008).

Similar to children, adults with ADHD also have reduced cerebellar volume. When adults who were diagnosed with ADHD as children were re-examined after 33 years, ADHD probands had significant cerebellar grey matter volume deficits compared with controls (Proal et al. 2011). Additionally, adults with ADHD have been shown to have reduced functional activity within the cerebellum during performance of both motor and non-motor tasks (Valera et al. 2005; Valera et al. 2010).

The cerebellum is necessary for adaptive motor learning - a critical ability that allows for adjustment of movements to the dynamic surrounding environment. Lesions to the cerebellum prohibit visuomotor adaptation from taking place (Martin et al. 1996; Maschke et al. 2004; Rabe et al. 2009; Tseng et al. 2007). For example, Martin and colleagues (1996) investigated accuracy of throwing in individuals with cerebellar lesions and matched controls. To examine visuomotor adaptation, throws were made with and without prism glasses. Unlike healthy control participants, individuals with cerebellar lesions were unable to adapt throws to compensate for the modified visual information.

Individual differences in healthy cerebellar structure and function have also been shown to relate to performance on visuomotor adaptation tasks. Greater cerebellar activation is associated with visuomotor learning and automaticity (Della-Maggiore and McIntosh 2005).

Similarly, greater density of the white matter tracts connecting the cerebellum with the motor and premotor cortical regions is associated with a faster rate of adaptation (Della-Maggiore et al. 2009). Therefore, even within healthy individuals, cerebellar connectivity can predict the rate of adaptive motor learning.

In the present study, we examined whether adults with ADHD exhibit movement impairments compared to controls. In addition to testing individuals with ADHD, individuals with ADHD-like symptoms (score ≥4 on part A of the ADHD Self-Report Scale) were also included. Adult ADHD is under-diagnosed with respect to its prevalence (Clarke, Heussler and Kohn 2005; Hines, King and Curry 2012); in a large-scale survey, only approximately 11% of adults who met the diagnostic criteria for ADHD had received treatment in the previous year (Kessler et al. 2006). By including individuals with symptoms consistent with ADHD diagnosis, regardless of actual diagnosis, we sought to examine a more representative adult sample.

To probe motor performance, we used a visuomotor adaptation task known to rely on cerebellar function (e.g., Martin et al. 1996; Maschke et al. 2004; Rabe et al. 2009; Tseng et al. 2007). Given that the cerebellar deficits associated with ADHD are of reduced volume and white matter connectivity, but not complete lesions, we hypothesized that individuals with ADHD would be able to adapt to a visuomotor rotation task but to a lesser extent than the non-ADHD control group.

Materials and Methods

Participants

Participants were 91 (49 female) young adults ranging from 18 to 30 yrs (M = 21.4 ± 1.95 yrs; Table 1) who received course credit for participation. Exclusion criteria included a history of neurological disorder or premature birth (< 38 weeks gestational age) as premature birth has been shown to affect the cerebellum (Parker et al. 2008).

Diagnostic measure

The ADHD Self-Report Scale (ASRS) was used to classify subjects into “ADHD” or control groups. The ASRS has high internal consistency (Cronbach’s α=0.88) and high intra-class correlation coefficients for subset symptom scores (intra-class correlation coefficients = 0.83; Adler et al. 2006). Given that the first 6 questions (part A) are most predictive of symptoms consistent with ADHD (Kessler et al. 2005), only part A of the ASRS was used. All participants eligible to receive credit for research participation (students enrolled in Psychology courses) completed the 6 questions along with a larger pool of prescreening questions. A total of 1414 individuals completed the prescreening questions. Of those, 20 declined to answer the 6 questions, and 411 (29.5%) of those who responded were identified as having symptoms consistent with ADHD. Individuals were invited to participate based on their responses to part A of the ASRS (scored ≥ 4 were invited to the ADHD group; scores < 4 were invited to the control group). When individuals came in to participate, they were given the ASRS again and scores on the day of participation were used to classify individuals into groups. Forty-nine individuals scored ≥ 4 and composed the ADHD group.

Of these, 20 had a present self-reported diagnosis of ADHD and 15 were currently taking medication to treat their disorder. These individuals were asked to refrain from taking their medication in the morning before their testing session. The remaining 29 individuals in this group were classified as ADHD-like in sub-analyses reported below.

Apparatus

A Polhemus Liberty electromagnetic tracker was used to record movements. A small kinematic sensor (0.375 in. x 0.375 in. x 0.375 in.) taped to the index finger of the participant's dominant hand sampled the 3-D position at a sampling rate of 256 Hz. The Polhemus was connected to a computer with a horizontally mounted LCD monitor. The 22-in LCD monitor was elevated 10 in. above the table (~70 cm viewing distance) so the participant could comfortably move their hand in the space between the monitor and the table to perform the task (Fig 1). Their hand was occluded from their view during the task by the monitor. A yellow cursor on the LCD screen corresponded to the 2-dimensional (forward-backward/left-right) position of the participant's index finger as they moved below the screen.

Procedures

All procedures were approved by the University of Massachusetts Institutional Review Board and informed consent was obtained before the study commenced. The visuomotor rotation task was adapted from Kagerer, Contreras-Vidal, and Stelmach (1997). The display on the screen consisted of five white rings. Rings were 2 cm in diameter, and those in the four corners of the screen were targets (Fig 1). A center circle was the starting and ending location of each movement. While the finger rested at the center ring, one of the four outer targets illuminated (black center turned white). The participant was asked to make a reaching movement with their arm (shoulder and elbow joints) in order to move the cursor to the cued target. The distance from the center circle to the target (center-to-center) was 15 cm. Participants were instructed to reach as quickly and accurately as possible to the target then return to the center. Targets were cued in a random order with each appearing an equal number of times within each phase. Participants were allowed to freely move their eyes during the task as there was no fixation point.

The task was divided into three phases. The *pre-adaptation phase* consisted of 32 trials of baseline reaching performance. The *adaptation phase* consisted of 120 trials. In this phase, the cursor position, the visual indication on the monitor of the finger position, was rotated 30 counterclockwise in relation to the actual finger position. This perturbation occurred in the first trial of this phase, and was thus sudden, rather than gradual. A 30° rotation has been shown to be an effective perturbation to observe visuomotor adaptation in control populations (Krakauer, Pine, Ghilardi and Ghez, 2000; Galea, Vazquez, Pasricha, Orban de Xivry and Celnik, 2011; Krakauer, Ghez and Ghilardi, 2005), and is not considered to be excessively difficult (Rentsch and Rand, 2014) which may be important for use with the ADHD population. To successfully complete a reach in the adaptation phase, participants had to correct for this rotation by moving their finger 30 in the clockwise direction from the actual target location. The *post-adaptation phase* consisted of 12 trials in which the rotation was removed, meaning that the movement of the hand corresponded accurately with the

placement of the cursor on the screen. The post-adaptation phase gauged the existence and intensity of a residual effect, or the continued compensation for the rotation immediately following its removal.

Data Analysis

Each trial was visually inspected to ensure that the participant performed the task as instructed. Trials in which the participant exhibited a highly irregular movement pattern were withheld from analysis; for example, reaches were removed if the individual first reached toward an incorrect target before adjusting to the correct target. In addition, the first two trials of the pre-adaptation phase were withheld from analysis as they were considered "practice".

Reaction time (RT), movement time (MT) and angle at maximum velocity (AMV) were determined for each reach. RT is the time it took the participant to move the cursor outside of the starting circle following the illumination of the target. MT was the time it took the participant to direct the cursor from the starting circle to the target. Consistent with prior studies of visuomotor adaptation (Paz et al. 2003; Karakauer, Ghez and Ghilardi 2005; Wang and Sainburg 2005; Wang and Sainburg 2009), AMV was measured as the angle of deviation from the direct line between the center of the home circle and the center of the target circle at the point of maximum velocity during a trial. The point of maximum velocity represents a point in each trial when the participant corrected for their error.

Baseline trials were grouped into a single average for all 30 trials given the low variability in these trials. Adaptation trials were averaged into epochs of 6 trials per epoch. Performance for post-adaptation trials was also averaged across 6 trials, creating 2 epochs. Averaging removes differences in movement parameters due to direction of movement or noise in performance. However, due to a program error, the fifth adaptation epoch had five trials in a row towards the same target. Given that this epoch was not representative of the rest of the phase, it was removed from the analysis. Independent samples t-tests were used to analyze pre-adaptation differences in performance (RT, MT, and AMV) between the ADHD and control groups. For the adaptation and post-adaptation phases respectively, 2 x 19 and 2 x 2 mixed design ANOVAs were used to analyze the group differences (ADHD and Control) in RT, MT and AMV across epochs. Given the violation of the assumption of sphericity in the adaptation phase for both AMV and MT measures, the Huynh-Feldt correction was used. These analyses were also run examining differences in diagnosed and undiagnosed individuals to ensure that our categorization of the "ADHD" was appropriate.

Post-hoc independent samples t-tests were used to identify differences in group performance across epochs when significant interactions were observed. To control for multiple comparisons, a more stringent alpha cutoff of $p \leq 0.01$ was used for these analyses. In addition, to better quantify the differences in learning rates, significant interactions were further examined with simple linear regressions to determine the slope of the lines. A caveat of this method is that learning rates are likely to be more quadratic than linear in trajectories.

Results

The ADHD and control groups did not differ by age ($t(89) = -0.861$, $p = 0.391$) or gender (ADHD = 53.1% female, Control = 54.8% female; Table 1). Expectedly, there were significant differences between ASRS scores of the ADHD group and the control group ($t(89) = 13.35$, $p < 0.001$; Table 1). Interestingly, within the ADHD group, there were no significant differences in ASRS scores between those who had been diagnosed with ADHD and undiagnosed individuals ($t(47) = -1.415$, $p = 0.164$; Table 1).

An average of 5.9% (SD = 5.6%) of trials were withheld from analysis due to substantial irregularities in the reach trajectory. Significantly more trials were removed from the ADHD group than the control group ($t(89) = 2.107$, $p = 0.038$). Moreover, the percentage of trials removed per subject was significantly correlated with severity of ADHD symptoms as determined by the ASRS ($r = 0.286$, $p = 0.006$). It is possible that, given the nature of this disorder, individuals in the ADHD group made greater numbers of irregular reaches due to being more distracted or inattentive, or more impulsive on individual trials. Notably, it is necessary to eliminate these trials from subsequent analyses as measures of MT, RT, and AMV in these trials may reflect a different process than those with similar trajectories for the two groups.

Pre-adaptation phase (baseline)

There were no significant baseline differences in RT ($t(89) = -1.21$, $p = 0.230$; ADHD 0.14 ± 0.04 sec, Control = 0.15 ± 0.03 sec) or MT ($t(89) = 1.41$, $p = 0.163$; ADHD = 1.60 ± 0.20 sec, Control = 1.54 ± 0.22 sec; Fig 2). Baseline AMV assessed in the pre-adaptation phase also did not differ between the ADHD (11.24 ± 5.06°) and control groups (10.34 ± 3.90°; $t(89) = 0.94$, $p = 0.349$; Fig 3). Therefore, regardless of group, all individuals performed equivalently at baseline.

Adaptation phase

Across the adaptation phase, there was a trend level difference in RT for the ADHD compared to the control group ($F(1, 89) = 3.06$, $p = 0.083$). However, the effect size of this measure was small (partial $eta^2 = 0.033$). There was also no significant main effect of epoch on RT ($F(18, 72) = 1.43$, $p = 0.142$) nor a group by epoch interaction ($F(18, 72) = 1.04$, $p = 0.433$).

However, with respect to MT, there was a significant main effect of epoch ($F(9.77, 996.41) = 91.05$, $p < 0.001$) indicating a gradual reduction in MT as the phase progressed. While there was no main effect of group on MT ($F(1, 89) = 0.28$, $p = 0.600$), the epoch by group interaction was significant ($F(9.77, 996.41) = 1.77$, $p = 0.05$). These data indicate that while there are no overall differences in MT, the rate of MT correction was slower for individuals with ADHD symptoms (Fig 2). Post-hoc analyses further support no overall group differences in MT across adaptation as MT was not significantly different between groups at any individual epoch.

We further examined whether MT performance differed between those individuals within the ADHD group, comparing those with and without an ADHD diagnosis. Within the

ADHD group alone (diagnosed and undiagnosed), the significant main effect of epoch on MT remained ($F(7.89,370.90) = 44.50$, $p < 0.001$); however, there was no significant main effect of diagnosis subgroup ($F(1, 47) = 1.53$, $p = 0.223$), nor a subgroup by epoch interaction ($F(7.89,370.90) = 1.05$, $p = 0.402$). Therefore, the use of the ASRS for grouping into ADHD and control groups appears sufficient for this measure. Additionally, when comparing only those with a current ADHD diagnosis to the control group, the significant interaction between epoch and group remained ($F(14, 280.82) = 1.86$, $p = 0.030$). When the undiagnosed individuals with ADHD symptoms are compared to controls, this effect is no longer observed ($F(10.42, 698.181) = 1.51$, $p = 0.128$).

When AMV was compared between the ADHD and control groups in the adaptation phase, there was a significant main effect of epoch ($F(15.69, 1396.28) = 20.84$, $p < 0.001$), indicating that participants were adapting to the rotation as expected as illustrated in Figure 4. There was also a significant main effect of group ($F(1, 89) = 8.77$ $p = 0.004$), such that AMV was greater for the ADHD ($19.70 \pm 0.66°$) compared to the control group ($16.82 \pm 0.72°$). Importantly, there was a significant epoch by group interaction ($F(15.69, 1396.28) = 1.73$, $p = 0.038$), supporting that the rate of adaptation differed between the groups (Fig 3). The adaptation to the rotation was significantly slower in ADHD group as indicated by a reduced slope ($m = -0.451$) compared to the controls ($m = -0.517$). Additionally, post-hoc t-tests were used to compare group performance across individual epochs. Significant differences in performance were observed across the adaptation phase indicating that the ADHD group was consistently more affected by the rotation throughout this phase (Fig 3).

We assessed whether AMV performance on the adaptation phase differed between those individuals within the ADHD group based on diagnosis status. Consistent with the above analysis, there was a significant main effect of epoch ($F(14.87,698.65) = 9.60$ $p < 0.001$); however, there was no significant main effect of diagnosis subgroup ($F(1, 47) = 1.00$, $p = 0.322$), nor a subgroup by epoch interaction ($F(14.87,698.65) = 0.58$, $p = 0.888$). This suggests that individuals within the ADHD group did not differ on their performance during the adaptation phase based on whether or not they had been diagnosed with ADHD. Further, when we compared AMV across the adaptation phase between those with an ADHD diagnosis to controls, we still found a significant main effect of group ($F(1,60) = 11.06$, $p = 0.002$), although the group by epoch interaction was no longer significant ($F(14.89, 893.08) = 1.00$, $p = 0.447$). When the undiagnosed individuals in the ADHD group were compared to the control group, again the main effect of group remained significant ($F(1, 67) = 4.43$, $p = 0.039$), as did the group by epoch interaction ($F(14.50, 971.74) = 1.97$, $p = 0.016$).

Lastly, we considered whether the group differences in MT were related to the differences in AMV across the adaptation phase; the two measures are not necessarily independent of one another. A reach with a larger AMV is likely to take longer to complete than one with a more direct trajectory. In these data, AMV was significantly correlated with MT for both the ADHD group ($r = 0.88$, $p < 0.001$) and for the controls ($r = 0.94$, $p < 0.001$). This suggests that these two measures are not mutually exclusive and both represent the level of adaptation to the applied rotation.

Post-adaptation phase

In the post-adaptation phase, RT did not significantly differ across epochs ($F(1, 89) = 1.27$, $p = 0.262$), or by group ($F(1, 89) = 0.69$, 0.408). Additionally, there was no significant group by epoch interaction for this variable ($F(1, 89) = 0.31$, $p = 0.580$).

However, MT did significantly differ across epochs ($F(1, 89) = 60.66$, $p < 0.001$) such that the duration of the reach was greater for the first epoch ($M = 1.62 \pm 0.02$) compared with the second ($M = 1.50 \pm 0.019$); however, there was no main effect of group ($F(1, 89) = 0.09$, $p = 0.769$), and there was no significant epoch by group interaction ($F(1, 89) = 0.07$, $p = 0.794$; Fig 2). Therefore, the ADHD and control groups did not differ in their MT performance in the post-adaptation phase.

There was a significant effect of epoch on AMV ($F(1, 89) = 20.681$, $p < 0.001$) in the post-adaptation phase; however, again, there was no significant main effect of group ($F(1, 89) = 0.97$, $p = 0.328$), nor a significant group by epoch interaction ($F(1, 89) = 0.01$, $p = 0.915$; Fig 3). Like with MT, the ADHD and control groups did not differ in their level of adaptation to the removal of the rotation in the post-adaptation phase.

Discussion

In this study, we assessed whether adult individuals with diagnosed ADHD and ADHD symptoms show diminished visuomotor adaptation abilities. Given that ADHD is associated with cerebellar pathologies, and a known function of the cerebellum is visuomotor adaptation, it was predicted that the ADHD group, regardless of diagnosis status, would demonstrate reduced visuomotor adaptation compared to the control group. In support of our prediction, individuals with ADHD symptoms showed evidence of impaired visuomotor adaptation. This impairment is evidenced by the greater AMV and slower reduction of MT across the adaptation phase. In contrast, the control group made faster and more direct reaches throughout the adaptation phase.

These results are consistent with the hypothesized role of the cerebellum in movement impairments in individuals with ADHD. On a similar task, Della-Maggiore and colleagues implicate decreased cerebellar activation (Della-Maggiore and McIntosh 2005) and cerebellar connectivity (Della-Maggiore et al. 2009) in a reduced rate of motor adaptation in healthy young adults. Therefore, it seems likely that a reduction in cerebellar connectivity and function plays a predominant role in movement impairments in adults with ADHD.

An alternative explanation for the differences in the ADHD and control groups could be the manifestation of ADHD symptoms of impulsivity or inattentiveness. Inattentive symptoms during the task due to symptoms of ADHD should correspond with significant increases in RT or greater irregularities in movements. Impulsivity should correlate with decreased RT times. However, there were no significant group differences in RT across any measures of the task; therefore impulsivity is unlikely to explain the adaptation results. While there were significantly more irregular reaches in the ADHD group, possibly indicating greater inattentiveness, these trials were removed from the analysis preventing them from biasing the adaptation results.

Having firmly adapted to the rotation, participants make slower movements (MT) and more extensive errors in reach direction (AMV) at the beginning of the post-adaptation phase once this rotation is removed. However, there were no differences between groups for MT or AMV for the post-adaptation phase, nor significant group by epoch interactions. This suggests that, although the rate of adaptation is slower in the ADHD group, they nonetheless successfully adapt to the rotation by the end of the phase. Therefore the impairment is specific to the rate of learning this task, as opposed to whether or not adaptation is possible. Again, this mimics the effects seen in healthy individuals with reduced cerebellar activation and white matter connectivity; these individuals successfully adapted to the rotation despite doing so at a slower rate than those with stronger cerebellar connections (Della-Maggiore and McIntosh 2005; Della-Maggiore et al. 2009).

While we assume the observed deficits in adaptation in the ADHD group reflect a cerebellar deficit, it is possible that they instead reflect a role of the posterior parietal cortex (PPC). The PPC has been implicated in ADHD symptomology as part of the attentional network (see review: Katsuki and Constantinidis, 2012). Animal models of ADHD show reduced metabolic capacity in the PPC (Gallo, Gonzalez-Lima and Sadile, 2002) and one of the most common genentic polymorphisms associated with ADHD, the DRD4 7-repeat allele, has also been associated with thinner cortical structure in the PPC (Shaw et al., 2007). With respect to visuomotor adaptation, the PPC has been implicated in error detection and in the acute strategic control processes of prism adaptation (Clower, Hoffman, Votaw, Faber, Woods and Alexander, 1996; Pisella, Michel, Grea, Tilikete, Vighetto and Rossetti, 2004; Newport and Jackson, 2006; Luauté et al., 2009). Comparatively, the cerebellum is thought to be important for the adaptive component, or the more gradual realignment of the visual and proprioceptive representations (Pisella, Michel, Grea, Tilikete, Vighetto and Rossetti, 2004; Newport and Jackson, 2006; Luauté et al., 2009). Therefore, while deficits in the PPC in the ADHD group might explain early differences in adaptation (within the first 5–15 trials; Newport and Jackson, 2006), the prolonged group differences in adaptation are more in line with cerebellar deficits. Additionally, the measure of AMV has been shown to reflect the more gradual sensorimotor realignment attributed to the cerebellum compared to other measures of adaptation, such as end-point error (O'Shea et al., 2014).

It is important to note that the ADHD-related deficits in the rate of adaptation were observed using a sudden, rather than a gradual, perturbation of the visual information. While the striatum and other brain regions have been implicated in adaptation to large, sudden perturbations (Venkatakrishnan, Banquet, Burnod and Contreras-vidal, 2011), there is also strong evidence for the cerebellum's role in such adaptation. Reversible inactivation of the dentate nucleus in nonhuman primates impaired adaptation to both gradual and sudden shifts although deficits were greater with a gradual perturbation (Robertson and Miall, 1999). Conversely, individuals with severe cerebellar degeneration were profoundly impaired at adapting their movements when a visual shift was applied suddenly and yet were still capable of demonstrating visuomotor adaptation when the perturbation was applied gradually (Criscimagna-Hemminger, Bastian and Shadmehr, 2010). This study suggested that motor learning of smaller errors may be possible using other neural strategies, whereas adaptation to large perturbations were very reliant on cerebellar involvement. Recently, the cerebellum has been implicated in adaptation to both gradual and sudden perturbations

through observations in patients with cerebellar ataxia (Schlerf, Xu, Klemfuss, Griffiths and Ivry, 2013). In sum, these data are consistent with a cerebellar role in motor adaptation even under sudden perturbations such as the one used here.

While most of the current literature focuses on motor deficits in children with ADHD, this study contributes to the significant gap in the literature regarding motor deficits in ADHD adults. Future research probing the changes in ADHD motor symptoms across the lifespan is warranted. The results of this behavioral study further demonstrate the need to examine cerebellar dysfunction in individuals with ADHD. The cerebellum is clearly implicated in movement coordination (see review: Thach, Goodkin and Keating 1992), motor sequencing (Braitenberg, Heck and Sultan 1997; Spencer and Ivry 2009), and movement timing (Ivry and Spencer 2004). Some of the earliest motor behavioral research on ADHD showed delays in timed repetitive and sequential movements (Denckla and Rudel 1978). Working to expand our understanding of how the cerebellum is affected in individuals with ADHD across all age groups will facilitate the formulation of strategies for improving adaptive motor learning ability, and alleviating the motor symptoms of ADHD.

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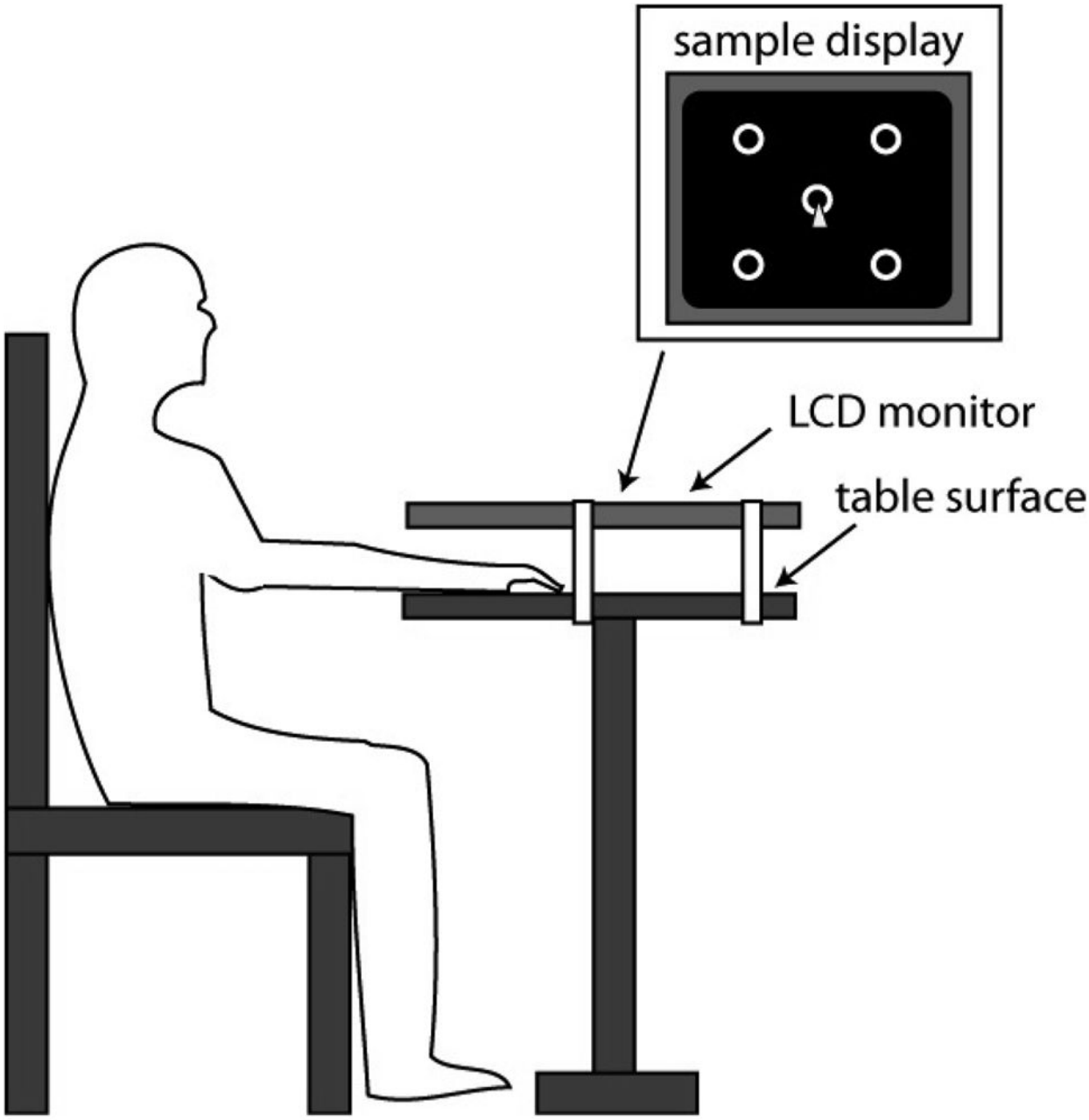

Fig 1.

Visuomotor rotation task setup. The four outer white rings are targets, whereas the center ring is the “home”. During the task, one of the targets will illuminate to cue the reach. The triangle represents the cursor that corresponds with the participant’s finger beneath the monitor.

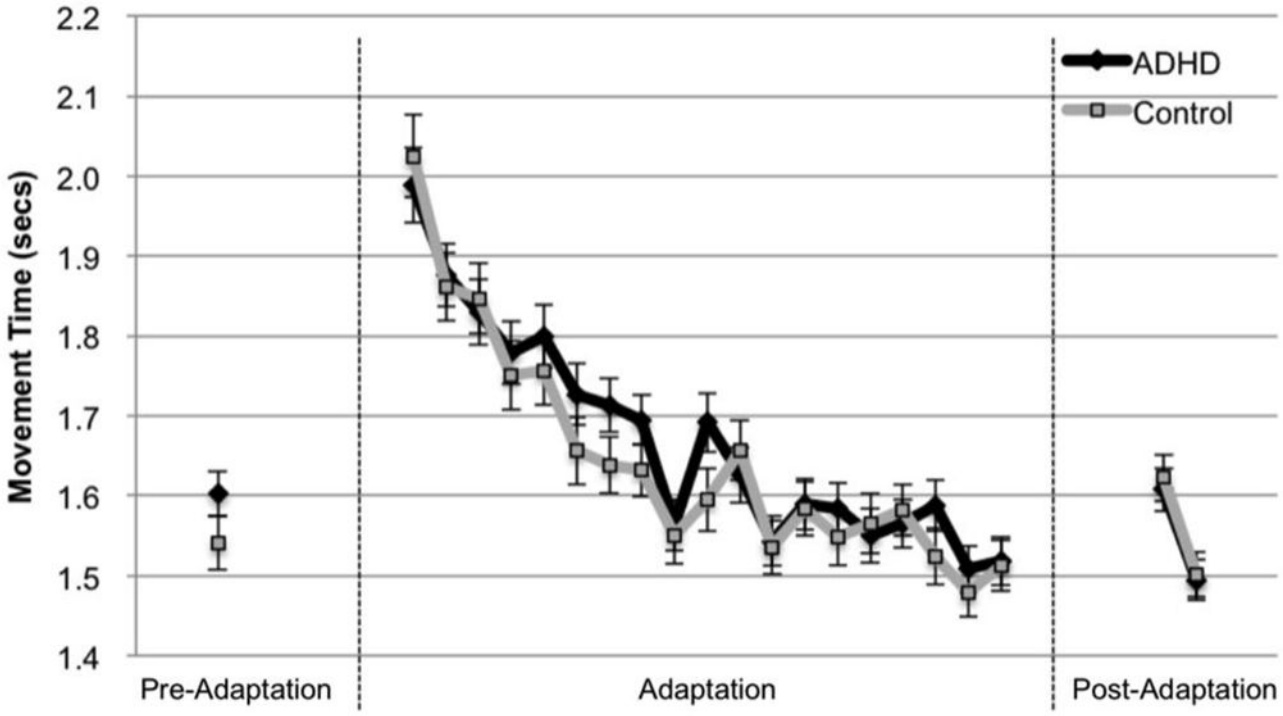

Fig 2.

The movement time for both the ADHD and control groups across the three phases of the visuomotor adaptation task. Pre-adaptation is represented by the average across all trials in this phase, whereas the Adaptation and Post-Adaptation is represented by epochs (groups of 6 trials each). Error bars reflect the standard error.

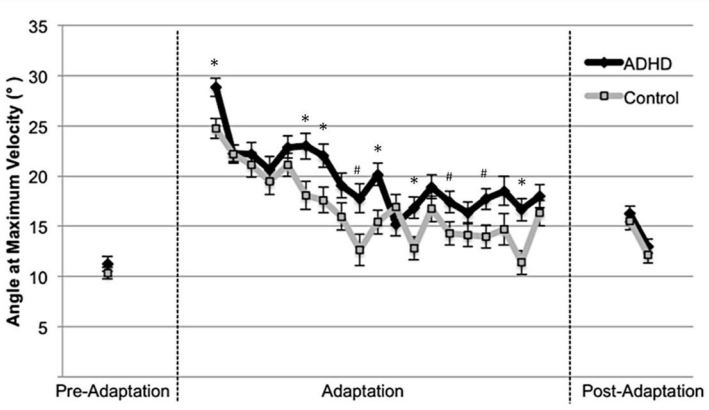

Fig 3.

The angle at maximum velocity for both the ADHD and control groups across the three phases of the visuomotor adaptation task. Error bars reflect the standard error. * = $p<0.01$, # = $p < 0.05$.

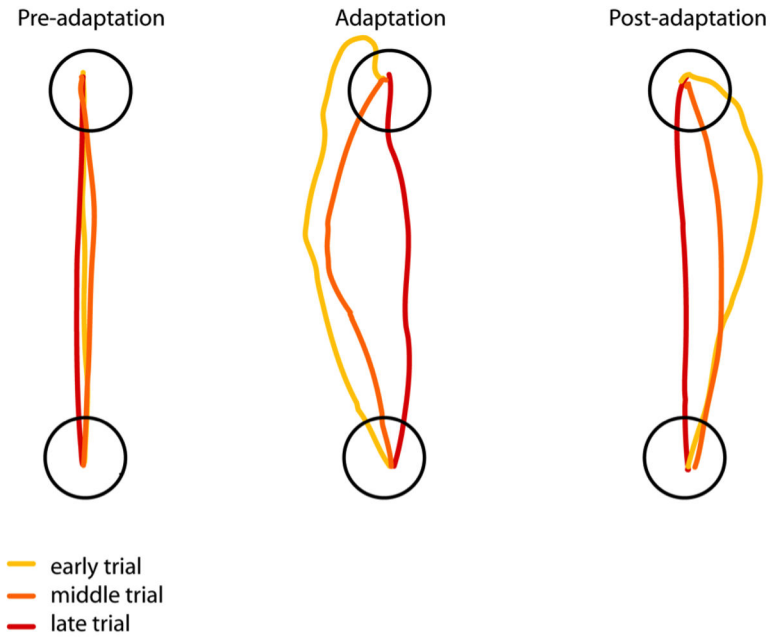

Fig 4.

Example reach movements in each of the phases of the experiment. The black rings are the start and end point of the reaches. Yellow lines represent reaches early in the phase, the orange lines are trials in the middle of the phase, and the red lines are trials at the end of the phase.

Table 1

Participant demographics

	N	Age Mean (SD)	Gender Female, Male	ASRS Score Mean (SD)
ADHD				
ADHD-diagnosed	20	21.50 (2.59)	13, 7	4.70 (1.26)
ADHD-like (undiagnosed)	29	21.44 (2.31)	13, 16	4.28 (0.84)
Control	42	21.26 (1.29)	23, 19	1.52 (1.04)