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An Examination of Movement Kinematics in Young People with High-functioning Autism and Asperger's Disorder: Further Evidence for a Motor Planning Deficit

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Abstract

This paper examines upper-body movement kinematics in individuals with high-functioning autism (HFA) and Asperger's disorder (AD). In general, the results indicate that HFA is more consistently associated with impaired motoric preparation/initiation than AD. The data further suggest that this quantitative difference in motor impairment is not necessarily underpinned by greater executive dysfunction vulnerability in autism relative to AD. Quantitative motoric dissociation between autism and AD may have down-stream effects on later stages of movement resulting in qualitative differences between these disorder groups, e.g. “motor clumsiness” in AD versus “abnormal posturing” in autism. It will be important for future research to map the developmental trajectory of motor abnormalities in these disorder groups.

Keywords

High-functioning autism; Asperger's disorder; Movement kinematics; Motor preparation kinematics; Motor preparation

Autism is a pervasive developmental condition defined by impairments in communication, social reciprocity, and repetitive—stereotyped behavioral patterns (American Psychiatric Association, 1994). Although motor functioning deficits are widely reported in the literature (Berkeley, Zittel, Pitney, & Nichols, 2001; Beversdorf et al., 2001; Brasic & Barnett, 1997; Ghaziuddin, Butler, Tsai, & Ghaziuddin, 1994; Manjiviona & Prior, 1995; Miyahara et al., 1997; Rinehart et al., 2001a), there is debate about how to describe and define motor abnormalities in this population (e.g. clumsy movement versus poorly coordinated movement versus poorly planned movement), and debate about whether the nature of motor

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impairment is different in autism and Asperger's disorder (AD). However, characterizing the nature and extent of motor abnormalities may have important implications for differential diagnosis and early detection of autism spectrum disorders (Rinehart et al., 2001a; Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998).

Motor functioning has been studied in these disorders using behavioral neurological assessment (Damasio & Maurer, 1978; Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998), gait analysis (Hallet et al., 1993; Maurer & Damasio, 1982; Vilensky, Damasio, & Maurer, 1981), analysis of postural control (Kohen-Raz, Volkmar, & Cohen, 1992), hand-writing analysis (Beversdorf et al., 2001), standardized motor batteries (e.g. Griffith's gross motor sub-scale, Bruininks–Osertsky test of fine and gross motor skills, Test of Motor Impairment-Henderson Revision, the Movement Assessment Battery for Children and the Pegboard test of motor coordination) (Ghaziuddin & Butler, 1998; Ghaziuddin et al., 1994; Gillberg, 1989; Manjiviona & Prior, 1995; Miyahara et al., 1997; Szatmari, Tuff, Finlayson, & Bartolucci, 1990), finger-tapping tasks (Muller, Pierce, Ambrose, Allen, & Courchesne, 2001; Rinehart et al., 2001a), and more recently, analysis of movement kinematics during reach- and - grasp experimental tasks (Mari et al., 2003; also see Hughes, 1996).

Several studies using standardized tests of motor functioning have directly compared children with high-functioning autism (HFA) and AD. Although it was originally thought that children with AD may have more significantly impaired motor functioning, than children with autism, as manifested by motoric clumsiness (Ghaziuddin, Tsai, & Ghaziuddin, 1992; Gillberg, 1989), more recent studies have reported similarly impaired motor functioning in both groups (Ghaziuddin et al., 1994; Manjiviona & Prior, 1995; Szatmari et al., 1990). DSM-IV-TR (APA, 2000) cites “motor clumsiness” (p. 81) as a feature of AD, whereas autistic disorder is associated with “abnormalities of posture” (p. 71). Motoric clumsiness is referred to in the ICD-10 (World Health Organization, 1992) as a symptom often found in AD; however no mention of motor clumsiness is made in the ICD-10 clinical description of autism (World Health Organization, 1992).

Some of the controversy as to whether motor functioning is indeed differentially impaired in these clinical groups may stem from a failure to consider ‘neuropsychological overshadowing’ of executive impairments. For example, comparative motor studies do not traditionally control for ‘executive’ dysfunction, a neurobehavioral component that may be more impaired in children with HFA than those with AD (Szatmari et al., 1990; Rinehart, Bradshaw, Moss, Brereton, & Tonge, 2001b; Rinehart, Bradshaw, Tonge, Brereton, & Bellgrove, 2002b). We reported subtle motor differences between individuals with HFA and AD using a simple motor reprogramming task (Rinehart et al., 2001a), which was thought to be more likely to tap the motor end of the attention—motor continuum than more cognitively demanding standardized tests of motor function. This experiment used our serial-choice button-pressing task, which yielded separate measures of motor preparation and execution time. Four target buttons (two central and two flanking) could be illuminated by an LED set into the based of each button. Upon illumination of the central buttons, participants made leftward and rightward movements in a repeating sequence as quickly as possible. Reprogramming of direction was manipulated by the introduction of an oddball to the basic reciprocating sequence. The oddball occurred at one of the two flanking buttons, either to the left or right of the central buttons. After detecting the oddball, participants were required to rejoin the reciprocating sequence. Movement preparation time was taken as the time between the illumination of one LED and the release of the previous button. Movement execution time was taken as the time between releasing one button and pressing the next. This study indicated that individuals with autism and AD have atypical movement *preparation* with an intact ability to *execute* movement. This finding is comparable to that of Hughes (1996) who used a reach, grasp, and place task. The autism and AD groups

displayed different patterns of motor preparation deficits. The participants with AD were slower at the point in the reprogramming sequence following the oddball, in contrast to the normal controls who were able to quickly re-engage in the back and forth reciprocating sequence. The motor preparation anomaly displayed by the autism participants was characterized by a failure to adjust their motor preparation time in response to an 'expected' versus 'unexpected' movement, in contrast to the normal controls who were, as one would predict, faster at preparing movement for an 'expected' versus 'unexpected' movement (Rinehart et al., 2001a). The subtle motor preparation deficits observed in these disorder groups were discussed in the context of a possible dissociable involvement of the basal ganglia thalamocortical circuitry, specifically the supplementary motor area and anterior cingulate, the latter area for its involvement in 'attention for action' and motivational aspects of behavior (see Pantelis & Brewer, 1996; Rinehart et al., 2001a). The basal ganglia thalamocortical circuitry has also been implicated by Vilensky et al.'s (1981) and Damasio and Maurer's (1978) analyses of gait, and Muller et al.'s (2001) recent study using functional magnetic resonance imaging during a finger-tapping task. In contrast, on the basis of their gait analysis of participants with autism, Hallet et al. (1993) argued that the nature of motor impairment in children with autism is more consistent with dysfunction of the cerebellum than the basal ganglia. Given the neurological and psychiatric complexities of autism/AD, it is more likely that both of these regions are involved to some extent (Bradshaw, 2001). The subtler the motor anomaly, the more difficult it is to ascribe, with any great certainty, to circumscribed neural circuitry. Indeed, Beversdorf et al. (2001) reported that the macrographic hand-writing observed in adults with HFA and AD is seen in both patients with cerebellar and basal ganglia pathology (Phillips, Bradshaw, Chiu, & Bradshaw, 1994a). (Note: Beversdorf et al., 2001, did not provide separate data for the autism and AD participants.)

Mari et al. (2003) have recently applied kinematic analysis techniques to measure *movement execution* characteristics of children with autism during a reach and grasp task. This study showed that specific deficits in movement kinematics only emerged when children were sub-grouped according to intellectual ability: 'low' (full scale IQ below 80) versus 'average /high' (full scale IQ between 80 and 109). Mari et al. reported that the low ability group displayed a parkinsonian-like bradykinesia with longer movement duration and deceleration, lower peak velocity, and later time of maximum grip aperture. In contrast, the average/high ability group showed 'intact' movement kinematics that were, however, more rapid than both the controls and low ability groups. Mari et al. suggested that the apparent 'hyperagility and hyperdexterity' (p. 402) may have a negative impact on every-day goal directed movement for children with HFA/AD because it may result in an inability to use additional external—environmental cues to modulate movement once a program is set in action. Although Mari et al. included children with autism and AD in their sample, separate data were not reported for these groups.

In summary, past research studies and contemporary clinical diagnostic criteria (i.e. DSM-IV-TR & ICD-10) provide conflicting reports about the nature of motor impairment in autism and AD. Moreover, the contribution of past research to the question of whether motor impairment is similar or different in autism and AD is limited by the use of standardized motor assessment batteries which do not separate out executive from motor dysfunction. While some studies have used more careful experimental approaches, for example, Mari et al.'s kinematic analysis of motor impairment, they have failed to consider potential motor differences between autism and AD in their analyses. Our previous examination of motor functioning in autism and AD using a reprogramming task pointed to dissociation in motor planning ability between the disorder groups (in the context of an intact ability to skillfully execute movements) (Rinehart, 2002b). However, this finding is yet to be replicated. Thus a study which provides separate data for autism and AD groups,

controls for potentially confounding executive functioning deficits, and separates motor preparation from motor execution, is clearly warranted.

The aim of the present study was to investigate movement kinematics (i.e. movement preparation, movement execution, and the shape of the movement trajectory (time spent in accelerative versus decelerative phases) in children with HFA and AD using a kinematic paradigm similar to that used in previous motor investigations of patients with neurodegenerative disorders (see Phillips, Martin, Bradshaw, & Iansek, 1994b; Bellgrove et al., 1997). The task was designed to also examine the impact of an executive load on movement kinematics by including expectancy and inhibitory components. The basic task involved the movement of a special stylus from a center start position toward either a left or right target according to the presentation of a visual cue (the illumination of an LED directly above the left or right target). There were three different task 'Levels'. In Level 1, participants were simply required to move to the left or right targets that were presented in a pseudo-random order. Fifty percent of the targets appeared on the left side, and the rest occurred on the right side. Participants were told that half of the targets would appear to the left and the other to the right. This task is therefore analogous to a choice reaction time (RT) task[1] with the addition of a more prolonged motor execution component, i.e. moving a stylus towards the target, rather than merely pressing a button.

In Level 2, 75% of targets appeared to one side and 25% to the other side. Thus, in Level 2 participants were instructed that they should move in the direction indicated by the LED as quickly as possible. Additionally, they were instructed to use the expectancy manipulation to facilitate their movement preparation and execution.

Level 3, was similar to Level 2 except that an additional *inhibitory* component required participants to always move to the un-cued target location, instead of the cued target location. For example, if the visual cue appeared on the left side, the participant was required to move to the right target, and vice versa. This motoric task is therefore somewhat analogous to antisaccade tasks within the cognitive domain. The expectancy manipulation remained in that the participant was told to expect most cues on one side as opposed to the other.

On the basis of previous studies we predicted that children with HFA and AD would show qualitatively different motor planning deficits (Hughes, 1996; Rinehart et al., 2001a). However, based on the recent findings of Mari et al. (2003) and our previous study (Rinehart et al., 2001a) we predict both groups will show generally intact movement execution. Mari et al.'s kinematic study suggested that individuals with autism and AD (IQ > 80) may be unable to efficiently modulate movement once a motor program is set in action, however, separate data was not provided for each group, so we can not be certain whether this finding relates more to autism or AD. Thus we predict that either or both clinical groups might spend a prolonged time in the more effortful terminal guidance/decelerative phase of movement in which final adjustments are made for target acquisition (see Bellgrove et al., 1997 for a discussion of the components of voluntary movement). In our review of past motor studies we suggested that individuals with autism may perform more poorly on motor tasks than individuals with AD due to their reportedly greater impairment in executive functioning (Szatmari et al., 1990; Rinehart, Bradshaw, Moss, Brereton, & Tonge, 2001b; Rinehart, Bradshaw, Tonge, Brereton, & Bellgrove, 2002b). If motor functioning deficits in children with autism are primarily underpinned by executive functioning deficits, then we

[1]We acknowledge that Reaction Time is not a pure measure of 'motor preparation' as the involvement of attentional deployment and other cognitive factors can not easily be extracted from this measure. Notwithstanding, the separation of the early and late stage components of this kinematics task is a very close approximate to the preparation and execution aspects of movement.

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would expect deficits in movement preparation time to manifest in the more complex and cognitively demanding Levels 2 and 3 where expectancy and inhibitory components were introduced, but be relatively intact in Level 1.

Methods

Participants

Twelve individuals with HFA (10 males and 2 females) as well as 12 control participants matched on age, sex, and full scale IQ, participated in the study. In addition, 12 individuals with AD (10 males and 2 females) and another 12 controls were recruited and matched according to age, sex, and full scale IQ. It is important to note that, while it would have been optimal to directly compare the HFA and AD groups, this was not deemed appropriate because of the developmentally critical age difference between the two groups, and the impact that this would have on the development of executive functioning ability and motor development in the two groups. One-way ANOVAs uncovered no significant age difference between the HFA and control group, $F(1,22) = .01$, $p = .93$ (HFA, mean age 8.1 years: controls, mean age 8.1 years) or between the AD and matched control groups, $F(1,22) = .01$, $p = .94$, (AD, mean age 12.0 years: controls, mean age 11.9 years) (see Tables 1 and 2 for participant details).

The participants for this study were recruited as part of ongoing research (Rinehart, Bradshaw, Moss, Brereton, & Tonge, 2000; Rinehart et al., 2001a, b). The participants with HFA fulfilled DSM-IV (APA, 1994) criteria for autistic disorder. The participants in the AD group satisfied DSM-IV criteria for AD. Four experienced clinicians were involved, at various times, in diagnosis. Diagnostic information was gathered using the revised Autism Diagnostic Interview (Lord, Rutter, & Le Couteur, 1994), structured parent interview, direct child observations, and information from other sources such as teachers and therapists. Inter-rater reliability, calculated on a sample of 107 cases of autism and AD, generated a Cohen κ of .95 for autism and .94 for AD, thereby indicating strong agreement.

Participants were included only if their performance and verbal IQ exceeded 70. In addition, participants were excluded if they had previously experienced the following conditions: comorbid medical (e.g. tuberous sclerosis), hearing or visual, neurological (e.g. epilepsy), psychiatric (e.g. Tourette's, attention deficit hyperactivity disorder) or genetic disorders (e.g. Fragile X disorder), other than the primary diagnosis of HFA or AD. The cognitive functioning of the HFA and AD groups was established from previous cognitive assessment undertaken at the time of the original diagnosis. If three or more years had lapsed since diagnosis, cognitive functioning of participants was reassessed using an age-appropriate Wechsler Intelligence test. It was only possible to report full scale IQ *range* because some of the participating assessment services did not provide specific IQ *scores*. For the purposes of statistical comparison, the mid-point of each IQ range was utilized; for example, a full scale IQ in the Average range (90–109) was recorded as 99.5.

Intellectual functioning in control participants was established using a short form of the Wechsler Intelligence Scales (either WPPSI-R, WISC-III-R, or WAIS-R), consisting of two Verbal (information and vocabulary) and two Performance (picture completion and block design) subtests. This particular short form loads highly on verbal comprehension and visual-perceptual organization skills, and is a reliable estimate of full scale IQ scores (Sattler, 1992). Control participants were matched to clinical participants on the basis of full scale IQ. There was no significant difference in IQ between the HFA and matched control groups, $F(1,22) = .31$, $p = .58$, or between the AD and matched control groups, $F(1,22) = .06$, $p = .80$ (see Tables 1 and 2).

Normal behavioral functioning was screened in both control groups using the Parent form of the Child Behavior Checklist (CBC-L) (Achenbach, 1991). None of the control participants was reported to have levels of clinically significant behavioral problems.

Apparatus

Participants sat directly in front of a WACOM SD420 digitizing tablet. The surface of the digitizing tablet lay at an angle of 7° from horizontal. The digitizing tablet, measuring 420 × 420 mm with an active surface of 305 × 305 mm, was connected to a Toshiba Notebook computer (440 CDT Satellite Pro; active screen) (see Fig. 1). The digitizing tablet recorded the movements of a non-inking electronic pen, and pen-tip position was sampled at a rate of 200 Hz. Data were recorded in the X and Y axes, and were accurate to ±2 mm.

Targets (i.e. LEDs) were positioned in the top left- and right-hand corners of the digitizing tablet, and the start position was positioned at the bottom, center of the tablet (see Fig. 1). The distance from the center of the start position and the center of each target was 170 mm, with each target at an angle of 45° to the start position. All three positions were depicted on the tablet as unfilled black-rimmed circles with a diameter of 25 mm. Directly, above each target circle was a small box housing the LEDs (see Fig. 1).

Procedure

The kinematic task was comprised of three different levels. The basic task involved the movement of a non-inking stylus from the center start position toward either a left or right target in response to the illumination of the left or right LED (see Fig. 1).

Level 1: No manipulation of target-side expectancy (Instructions: Move towards the target)—In Level 1 participants were instructed to move the stylus towards the target as quickly as possible in response to the illumination of the LED (i.e. draw an imaginary line from the start position to the target). Performance for target side was pseudo-randomly ordered, with 50% left-sided and 50% right-sided targets. Target side was not analyzed.

Level 2: Manipulation of target-side expectancy (Instructions: Move towards the target)—Level 2 involved two conditions for each participant. In one condition 75% of the targets were left-sided, and 25% were right-sided, while in the second condition the exact opposite occurred (i.e. 75% right targets, 25% left targets). Participants were instructed on which side the majority of targets would appear. When targets were presented at an unexpected location, the participant had to *inhibit* moving to the expected location. There were 32 trials in each condition (24 expected, 8 unexpected). Again, performance relative to target side was not analyzed.

Level 3: Manipulation of target-side expectancy (Instructions: Move to the opposite side of the target)—Level 3 used the same expectancy manipulation (i.e. 75:25) as Level 2; however, participants were now required to move to the *opposite* location to that indicated by the target. For example, if the LED appeared on the right side, participants were instructed to move to the left-sided target. Again, there were two conditions. In the first condition there were 32 trials where the *right side* was the ‘expected’ location (i.e. 24 trials with left LEDs, that required movements to right targets, and 8 trials with right LEDs, that required movement to left targets). Condition 2 was analogous to Condition 1, except that the left side was the ‘expected’ direction of movement. Again, each participant completed both conditions and order of presentation was counterbalanced across subjects.

Any trials in which participants initiated movements towards the incorrect target location were recorded as errors and not included in the motor kinematic analyses. The extent of movement errors was estimated in three categories: Mild errors (0–5 cm in the incorrect direction), moderate errors (5.1–12 cm), and severe errors (12.1–17 cm). These error categories were able to accommodate the three common error patterns which were observed across participants; for example, moving a third of the way, two-thirds of the way, or completely, to the wrong target.

Kinematic Measures

Horizontal and vertical vector components were obtained for each movement, and the data were filtered (low-pass, 10 Hz cut-off) using a recursive, dual pass, second order Butterworth filter (Mattingley, Phillips, & Bradshaw, 1995). Displacement data were differentiated using a 9-point central-finite-differences algorithm. Automatic algorithms were used to determine movement preparation time, i.e. the time spent within the start position before commencing movement towards the target (see Fig. 1).

The kinematic dependent measures included: (a) Preparation Time: analogous to reaction time, indexing preparatory responses, (b) Movement Time: the total time (ms) spent in motion, providing a measure of the time spent in the execution phase of movement, and (c) Asymmetry Ratio: The Asymmetry Ratio reflects the shape of the movement trajectory, measured by the time to peak velocity divided by the Movement Time. An efficient movement should have a symmetrical shape with equal time spent in accelerative and decelerative phases and a ratio of .5. A ratio greater than .5 indicates greater time spent in the accelerative phase, whereas a ratio of less than .5 indicates prolonged periods of deceleration and guidance (see Bellgrove et al., 1997 for a discussion of the components of voluntary movement).

Statistical Analyses

For Task 1, each of the movement indices were compared using a one-way ANOVA. For Tasks 2 and 3, each of the movement indices were analyzed using mixed-model ANOVAs, with between subjects factors of Group (HFA versus controls; AD versus controls) and within-subjects factors of Expectancy (expected versus unexpected).

There were insufficient errors in each of the three predefined categories (i.e. Mild, Moderate, Severe) for analysis: thus, the errors were collapsed across type and submitted to mixed-model ANOVAs, with between subject factors Group (HFA versus controls; AD versus controls) and within-subjects factors of Errors (Level 1, 2, 3).

Results

In view of the large number of statistical analyses conducted, only significant results ($p < .05$) and trends ($p < .08$) will be reported. All clinical and control groups committed comparable Errors across Levels, with no significant Group differences in Error Type ($p < .05$): HFA (mean % errors = Level 1 = .1%, Level 2 = 1.3%, Level 3 = 3.5%), HFA-matched controls (mean % errors = Level 1 = .2%, Level 2 = .4%, Level 3 = 1.6%); AD (mean % errors = Level 1 = .05%, Level 2 = 1.0%, Level 3 = 1.0%), AD-matched controls (mean % errors = Level 1 = .14%, Level 2 = .75%, Level 3 = 1.9%). Errors were therefore not analyzed further.

High-functioning Autism

Preparation Time

Level 1: No manipulation of target-side expectancy (Instructions: Move towards the target): Children with HFA were slower to prepare their movements (M = 724 ms, SD = 251) than control children (M = 538 ms, SD = 165), [$F(1,22) = 4.606$, $p = .04$], indicating a generalized impairment in motor planning.

Level 2: Manipulation of Target-side Expectancy (Instructions: Move towards the Target): A two-way ANOVA with factors of Group (HFA, controls) and Expectancy (expected, unexpected) revealed a main effect of Group [$F(1,22) = 4.39$, $p = .05$] indicating that the HFA group (M = 597.90 ms, SD = 194.55) were slower at preparing movement in comparison to controls (M = 466.58 ms, SD = 123.00).

Level 3: Manipulation of target-side expectancy (Instructions: Move to the opposite side of the target): An analogous two-way ANOVA again revealed a main effect of Group [$F(1,22) = 4.59$, $p = .04$] indicating that the HFA group were significantly slower (M = 547. 90 ms, SD = 186.4) at preparing movements in comparison to controls (M = 471.10, SD = 109.10).

Movement Time

Level 1: No manipulation of target-side expectancy (Instructions: Move towards the target): There was no main effect of Group, suggesting that HFA is not associated with a simple motor slowness.

Level 2: Manipulation of target-side expectancy (Instruction: Move towards the target): There were no Group differences, both Groups showed similar movement times for both expected and unexpected movements.

Level 3: Manipulation of target-side expectancy (Instructions: Move to the opposite side of the target): A two-way ANOVA revealed a Group by Expectancy interaction [$F(1,22) = 5.56$, $p = .03$]. Whereas the HFA group was relatively unaffected by the expectancy manipulation (M Exp. = 676.60 ms, SD = 220.75; M Unexpect. = 643.96 ms, SD = 171.16) the controls were significantly faster for the expected condition in comparison to unexpected condition (M Exp. = 588.03 ms, SD = 117.93; M Unexpect. = 638.79 ms, SD = 116.68). Subsequent analysis confirmed significant expectancy—unexpectancy effects for Controls, $t(11) = 2.79$, $p = .02$, but not for the HFA group $t(11) = -1.08$, $p = .31$.

Asymmetry Indices

Level 1: No manipulation of target-side expectancy (Instructions: Move towards the target): Both HFA and controls displayed approximately symmetrical asymmetry indices.

Level 2: Manipulation of target-side expectancy (Instructions: Move towards the target): A two-way ANOVA of Group (HFA, controls) and Expectancy (expected, unexpected) revealed a trend towards a main effect of Expectancy [$F(1,22) = 3.63$, $p = .07$]. There was a trend towards a Group by Expectancy interaction [$F(1,22) = 3.58$, $p = .07$]. Inspection of the means revealed that although the Asymmetry Indices of the control group were identical, as a function of Expectancy, the HFA tended to spend more time in the decelerative phase for expected (M = .45, SD = .08) than unexpected movements (M = .50, SD = .08).

Level 3: Manipulation of target-side expectancy (Instructions: Move to the opposite side of the target): An analogous two-way ANOVA revealed a main effect of Expectancy [$F(1,22) = 4.64$, $p = .042$], such that participants in both Groups spent more time in the accelerative phase for expected ($M = .53$, $SD = .08$) than unexpected movements ($M = .49$, $SD = .04$).

Asperger's Disorder

Preparation Time

Level 1: No manipulation of target-side expectancy (Instructions: Move towards the target): There was a trend towards a main effect of Group [$F(1,22) = 4.055$, $p = .056$], such that children with AD tended to prepare their movements more slowly ($M = 516$ ms, $SD = 169$) than controls ($M = 407$ ms, $SD = 82$).

Level 2: Manipulation of target-side expectancy (Instructions: Move towards the target): A two-way ANOVA (Group, Expectancy), revealed a main effect of Expectancy [$F(1,22) = 6.16$, $p = .02$], indicating that both groups took longer to prepare movements in response to unexpected ($M = 405$ ms) targets in contrast to expected ($M = 381$ ms) targets. Thus, the AD group and control group displayed optimal movement preparation in response to expected versus unexpected movements.

Level 3: Manipulation of target-side expectancy (Instructions: Move to the opposite side of the target): Both groups prepared movement similarly, and target expectancy did not impact on movement preparation times for either group. Thus, in contrast to the HFA/control comparison where the HFA group showed slower motor preparation for Levels 2 and 3, the AD group showed comparable motor preparation to their matched controls across task levels.

Movement Time

Level 1: No manipulation of target-side expectancy (Instructions: Move towards the target): There was no significant effect of Group.

Level 2: Manipulation of target-side expectancy (Instructions: Move towards the target): Similar to the HFA/control comparison, both the AD and control groups showed similar movement times for both expected and unexpected movements (and to each other).

Level 3: Manipulation of target-side expectancy (Instructions: Move to the opposite side of the target): Both groups exhibited similar movement times, and target expectancy did not significantly impact on movement times for either group (AD M Exp. = 675.31 ms, $SD = 192.73$; M Unexpect. = 739.04 ms, $SD = 204.46$: AD-controls M Exp. = 546.82 ms, $SD = 157.48$; M Unexpect. = 558.05 ms, $SD = 157.36$). This contrasts with the HFA analysis in that while the HFA group were relatively unaffected by the expectancy manipulation, their control group were significantly faster for the expected than unexpected targets.

Asymmetry Indices

Level 1: No manipulation of target-side expectancy (Instructions: Move towards the target): Both AD and controls displayed approximately symmetrical asymmetry indices.

Level 2: Manipulation of target-side expectancy (Instructions: Move towards the target): There were no significant effects involving Group or Expectancy.

Level 3: Manipulation of target-side expectancy (Instructions: Move to the opposite side of the target): There were no significant effects involving Group or Expectancy.

Discussion

Autism and AD are largely characterized by global and multifaceted social and communicative impairment and repetitive and restricted behavioral patterns; these symptoms are highly salient diagnostically and unequivocally understood as unique in character to these disorders. Motor impairment, although less well defined and understood in terms of diagnostic relevance, is an attractive target of neuropsychological investigations that seek to understand brain–behavior relationships, and the potential diagnostic separability of these disorders. Although several studies have focused on understanding gait abnormalities using kinematic analysis, this is only the second study to examine upper-body movement using kinematic methods (see also Mari et al., 2003).

Consistent with our hypothesis, deficits at the motor planning, rather than execution stage, were more predominate in both clinical groups. These results suggest that the movement deficits observed clinically for these individuals do not reflect a simple motor slowness. Given the high-functioning nature of our sample, this finding is comparable to Mari et al.'s (2003) study which showed movement execution problems were a feature of children with a full scale IQ below 80, and not apparent in more cognitively intact children with HFA/AD.

While the HFA group displayed a clear *motor preparation* deficit in comparison to controls, across all three Task Levels, there was only a trend towards impaired motor preparation for the AD group, and this occurred only at the least cognitively demanding task level (Level 1). The manifestation of a motor preparation deficit for a simple task, but not for a complex task, might be described as a type of *kinesia paradoxa*, where “the individual who typically experiences severe difficulties with the most simple of movements may suddenly perform complex, skilled movements…’ (Leary & Hill, 1996, p. 41). Leary and Hill note that this phenomenon is an important manifestation of movement disorder in children with autism spectrum disorders. This description of motor disturbance fits well with the finding that these children may have extreme difficulty with one aspect of motor functioning, for example, mastering pencil grip and writing (e.g. Beversdorf et al., 2001), but show exceptional ability on other motor tasks such as performing a rapid, complex sequence of movements on a computer joy-stick.

While on the surface, this study suggests that motor preparation may be more consistently impaired in children with HFA compared to those with AD, given the four-year age difference between the groups, caution is needed for this interpretation (despite the use of matched-control groups). Indeed, it may be that motor preparation anomalies are more pronounced at a younger age for children with autism, but that the deficit becomes less apparent or manifests as a *kinesia paradoxa* with age as fronto-striatal-basal ganglia thalamocortical pathways develop. Mapping the manifestation of cognitive-motor impairments in these groups over time could test the existence of such an abnormal motor developmental trajectory.

Analysis of Movement Time for Level 3 indicated the HFA participants were relatively unaffected by the expectancy manipulation. The HFA-control group, on the other hand, was significantly faster for expected than unexpected movements. This finding *somewhat parallels* our previous study using a motor tapping reprogramming task where movement for the HFA group, relative to matched controls, was characterized by a ‘lack of anticipation’, with motor preparation times not differing regardless of whether the movement was predictable or unpredictable (Rinehart et al., 2001a). However, as AD and AD-control

participants also did not show advantages for ‘expected’ movements in Level 3 (a finding which may be developmentally linked), these data need to be interpreted with caution. Nevertheless, the failure of the HFA group to benefit from the expectancy manipulation in order to execute expected movements more quickly than unexpected ones, could reflect a down-stream effect of a motor planning deficit. Indeed, analysis of the Asymmetry Indices suggested that the HFA group tended to spend greater periods in the decelerative/terminal guidance phase for expected movements- a pattern that is indicative of a poorly planned movement.

Some of the controversy amongst studies examining motor features in children with HFA and AD may be a result of the failure to adequately control for executive functioning problems, an area that appears more impaired in HFA than AD (Szatmari et al., 1990; Rinehart et al., 2001b, 2002b). It may be, for example, that contradictory reports of motor dysfunction in HFA and AD have not taken into consideration the possibility that HFA children may have poorer executive ability, which interferes with motor performance. The present study was able, to some extent, to parse out executive and motor dysfunction. For example, if motor functioning deficits in children with autism were primarily underpinned by executive functioning deficits, then we would have expected motor deficits in the more complex and cognitively demanding task levels where attention-set shifting and inhibitory components were introduced, with relatively intact motor functioning on Level 1 that involved a choice decision. The finding that motor preparation deficits were evident across all Levels regardless of cognitive complexity (i.e. the addition of inhibitory and expectancy components) would suggest that executive dysfunction does not confer significant and additional vulnerability for motor planning in individuals with HFA. Nevertheless, as argued above, there was evidence for task complexity effects within the execution phase of movement which might reflect a down-stream manifestation of impaired motor planning, since a poorly planned movement will need to be adjusted and guided on-line. To this extent our results resemble those seen in traditional movement disorders such as Parkinson's disease. For example, patients with Parkinson's disease (a classical hypokinesia disorder) show motor planning and execution deficits when required to perform sequential movements under conditions of reduced ‘advanced’ information; a task manipulation somewhat analogous to the ‘expectancy’ manipulations used in the current motor task (Georgiou et al., 1993, 1994).

The difficulty preparing for/initiating movements observed in the HFA group, and to a lesser extent in the AD group who displayed what might be described as *kinesia paradoxa*, can potentially be linked to a ‘parkinsonian like’ abnormality in dopaminergic activity resulting in inadequate output to the pre-motor areas of the brain, including the supplementary motor area (SMA). There is some neuroimaging evidence implicating frontostriatal regions such as the striatum, caudate, and putamen in autism (Fernell et al., 1997; Happe et al., 1996; Siegel et al., 1992). Functional neuroimaging studies have also found decreased metabolic and neurotransmitter activity in the thalamus, basal ganglia and cortical regions of individuals with autism (Chugani et al., 1997; Horwitz, Rumsey, Grady, & Rapoport, 1988). Future work will need to determine the neural circuitry that might underpin motor planning deficits in these disorder groups.

As discussed above, the HFA group tended to spend a prolonged period in the decelerative phase of movement when responding to an ‘expected’ but not an ‘unexpected’ target in Level 2. Normal time in the accelerative phase of movement with prolonged time spent in the more effortful terminal guidance phase in which final adjustments are made for target acquisition, is consistent with Mari et al.'s (2003) suggestion that these individuals may be unable to efficiently modulate movement once a program is set in action. Such difficulty with visually guided hand movements is somewhat consistent with the movement of

cerebellar patients which is characterized by a failure to accurately modulate later stages of movement in order to efficiently 'home-in' on targets (Rosenbaum, 1991). Anecdotally, young adults with HFA report that they have difficulty 'catching' a ball because 'at the last minute' they do not know what to do as the ball approaches. This might relate to a cerebellar target acquisition deficit. Conversely, patients with Parkinson's disease typically have no difficulty catching a ball (more like a reflex), but have difficulty throwing a ball (a more purely voluntary action) (Georgiou et al., 1994). The cerebellum has been extensively implicated in autism using behavioral, neuroimaging, and postmortem analyses (see Courchesne, 1999; Courchesne, Hesselink, Jernigan, & Yeung-courchesne, 1987; Courchesne et al., 2001; Courchesne, Kilman, Galambos, & Lincoln, 1984; Courchesne, Muller, & Saitoh, 1999; Courchesne et al., 1994a, b; Courchesne, Yeung-courchesne, Press, Hesse-Link, & Jernigan, 1988).

There are two clear limitations of this study; first, while it would have been desirable to directly compare HFA & AD groups, in view of the developmentally critical age-gap between groups it was not deemed appropriate to incorporate age as a covariate in data analyses. It is important to note, however, that the four-year age difference between our HFA and AD sample is entirely consistent with the different ages at which children who meet these diagnostic criteria present to assessment services: HFA (mean age at the time of diagnosis = 5.5 years) and AD (mean age at the time of diagnosis = 11 years) (Howlin & Asharian, 1999). The second limitation of note is that the small sample size is likely to be responsible for the inconsistent between-task findings, for example, the HFA group showing a tendency towards prolonged decelerative phase of movement in Level 2, but not Levels 1 and 3. Again, it is difficult to avoid this type of limitation given the relative difficulty associated with recruiting young people with HFA and AD who are matched intellectually and without comorbid medical conditions.

Notwithstanding these limitations, this research utilizes an innovative methodological approach to address the controversial issue of whether motor functioning is similarly impaired in HFA and AD. The overall finding that motor abnormalities (in particular motor preparatory function) appear more impaired in individuals with HFA than AD, contrasts with findings from standardized test batteries which show, for example, that these groups perform similarly when assessed on tests of ball skill, balance, and manual dexterity (Manjiviona & Prior, 1995; Miyahara et al., 1997). Although one study does not necessarily negate the findings of the other, the inconsistency does suggest that while on some tasks individuals with HFA and AD may share functional motoric impairment, (e.g. difficulty catching a ball), the fundamental underlying nature of motor impairment may be distinct (e.g. differential involvement of the cerebellum and fronto-striatal-basal ganglia motor circuitry).

In summary, this is the second study to examine upper-body movement using kinematic methods, and the first to consider whether kinematic motor functioning may be differentially impaired in HFA and AD. In general, the results indicate that individuals with HFA have more consistently impaired motoric preparation/initiation than individuals with AD. A possible quantitative dissociation in motor function between these groups may have differential down-stream effects on gross motor function and result in qualitative differences, such as those described in DSM-IV-TR, e.g. AD—motor clumsiness versus HFA-abnormal posturing (APA, 2000, p. 71, 81). It will be important for future research to use experimental movement paradigms sensitive to the clumsy motor patterns frequently reported in this literature. We are currently undertaking such a study to examine bimanual motor coordination.

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References

Achenbach, TM. Manual for the child behavior checklist, 4–18 and 1991 profile. Burlington: University of Vermont, Department of Psychiatry; 1991.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed.. Washington, DC: American Psychiatric Association Press; 1994.

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed.. Washington, DC: American Psychiatric Association Press; 2000. TR

Bellgrove MA, Phillips JG, Bradshaw JL, Hall KA, Presnell I, Hecht H. Response programming in demential of the Alzheimer type: A kinematic analysis. Neuropsychologica. 1997; 35:229–240. [PubMed: 9051672]

Berkeley SL, Zittel LL, Pitney LV, Nichols SE. Locomotor and object control skills of children diagnosed with autism. Adapted Physical Activity Quarterly. 2001; 18:405–416.

Beversdorf DQ, Anderson JM, Manning SE, Anderson SL, Nordgren RE, Felopulos GJ. Brief report: macrographia in high-functioning adults with autism spectrum disorder. Journal of Autism and Developmental Disorders. 2001; 31:97–101. [PubMed: 11439759]

Bradshaw, JL. Developmental disorders of the fronto-striatal system: Neuropsychological, Neuropsychiatric and Evolutionary perspectives. Hove, East Sussex: Psychology Press Ltd; 2001.

Brasic JR, Barnett JY. Hyperkinesias in a prepubertal boy with autistic disorder treated with haloperidol and valproic acid. Psychological Reports. 1997; 80:163–170. [PubMed: 9122323]

Chugani DC, Muzik O, Rothermel RD, Behen ME, Chakroaborty PK, Manger TJ. Altered serotonin synthesis in the dentato-thalamo-cortical pathway in autistic boys. Annals of neurology. 1997; 41:666–669. [PubMed: 9382481]

Courchesne E. An MRI study of autism: The cerebellum revisited. Neurology. 1999; 52:1106–1107. [PubMed: 10102448]

Courchesne E, Hesselink JR, Jernigan TL, Yeung-courchesne R. Abnormal neuroanatomy in a non-retarded person with autism. Archives of Neurology. 1987; 44:335–341. [PubMed: 3827686]

Courchesne E, Karns CM, Davis HR, Ziccardi R, Carper RA, Tigue ZD. Unusual brain growth patterns in early life in patients with autistic disorder: An MRI study. Neurology. 2001; 57:245–254. [PubMed: 11468308]

Courchesne E, Kilman BA, Galambos R, Lincoln AJ. Autism: Processing of novel auditory information assessed by event-related brain potentials. Electroencephalography and Clinical Neurophysiology: Evoked Potentials. 1984; 59:238–248. [PubMed: 6203714]

Courchesne E, Muller RA, Saitoh O. Brain weight in autism: Normal in the majority of cases, megalencephalic in rare cases. Neurology. 1999; 52:1057–1059. [PubMed: 10102428]

Courchesne E, Townsend J, Akshoomoff NA, Saitoh O, Yeung-Courchesne R, Lincoln AJ. Impairment in shifting attention in autistic and cerebellar patients. Behavioral Neuroscience. 1994a; 108:848–865. [PubMed: 7826509]

Courchesne, E.; Townsend, JP.; Akshoomoff, NA.; Yeung-Courchesne, R.; Press, GA.; Murakami, JW. A new finding: Impairment in shifting attention in autistic and cerebellar patients. In: Broman, SH.; Grafman, J., editors. Atypical cognitive deficits in developmental disorders: Implications for brain development. Hillsdale, NJ: Erlbaum; 1994b. p. 101-137.

Courchesne E, Yeung-courchesne R, Press GA, Hesse-Link JR, Jernigan TL. Hypoplasia of cerebellar vermal lobules VI and VII in autism. New England Journal of Medicine. 1988; 318:1349–1354. [PubMed: 3367935]

Damasio AR, Maurer RG. A neurological model for childhood autism. Archives of Neurology. 1978; 35:777–786. [PubMed: 718482]

Fernell E, Watanabe Y, Adolfsson I, Tani Y, Bergstrom M, Hartvig P. Possible effects of tetrahydrobiopterin treatment in six children with autism—clinical and positron emission tomography data: A pilot study. Developmental Medicine and Child Neurology. 1997; 39:313–318. [PubMed: 9236697]

Georgiou N, Bradshaw JL, Iansek R, Phillips JG, Mattingley JB, Bradshaw JA. Reduction in external cues and movement sequencing in Parkinson's disease. Journal of Neurology, Neurosurgery, and Psychiatry. 1994; 57:368–370.

Georgiou N, Iansek R, Bradshaw JL, Phillips JG, Mattingley JB, Bradshaw JA. An evaluation of the role of internal cues in the pathogenesis of Parkinsonian hypokinesia. Brain. 1993; 116:1575–1587. [PubMed: 8293289]

Ghaziuddin M, Butler E. Clumsiness in autism and Asperger syndrome: A further report. Journal of Intellectual Disability Research. 1998; 42:43–48. [PubMed: 9534114]

Ghaziuddin M, Butler E, Tsai L, Ghaziuddin N. Is clumsiness a marker for Asperger syndrome? Journal of Intellectual Disability Research. 1994; 38:519–527. [PubMed: 7841689]

Ghaziuddin M, Tsai LY, Ghaziuddin N. Brief report: A reappraisal of clumsiness as a diagnostic feature of Asperger syndrome. Journal of Autism and Developmental Disorders. 1992; 22:651–656. [PubMed: 1483982]

Gillberg C. Asperger syndrome in 23 Swedish children. Developmental Medicine and Child Neurology. 1989; 31:520–531. [PubMed: 2680690]

Hallet M, Lebiedowska MK, Thomas SL, Stanhope SJ, Denckla MB, Rumsey J. Locomotion of autistic adults. Archives of Neurology. 1993; 50:1304–1308. [PubMed: 8257307]

Happe F, Ehlers S, Fletcher P, Frith U, Johansson M, Gillberg C. 'Theory of mind' in the brain. Evidence from a PET scan study of Asperger syndrome. Neuroreport. 1996; 8:197–201. [PubMed: 9051780]

Horwitz B, Rumsey JM, Grady CL, Rapoport SI. The cerebral metabolic landscape in autism. Intercorrelations of regional glucose utilization. Archives of Neurology. 1988; 45:749–755. [PubMed: 3260481]

Howlin P, Asharian A. The diagnosis of autism and Asperger's syndrome: Findings from a survey of 770 families. Developmental Medicine & Child Neurology. 1999; 41:834–839. [PubMed: 10619282]

Hughes C. Brief Report: Planning problems in autism at the level of motor control. Journal of Autism and Developmental Disorders. 1996; 26:99–108. [PubMed: 8819773]

Kohen-Raz R, Volkmar FR, Cohen DJ. Postural control in children with autism. Journal of Autism and Developmental Disorders. 1992; 22:419–432. [PubMed: 1383190]

Leary MR, Hill DA. Moving on: Autism and movement disturbance. Mental Retardation. 1996; 34:39–53. [PubMed: 8822025]

Lord C, Rutter M, Le Couteur A. Autism diagnostic interview-revised: A revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. Journal of Autism and Developmental Disorders. 1994; 24:659–685. [PubMed: 7814313]

Manjiviona J, Prior M. Comparison of Asperger syndrome and high-functioning autistic children on a test of motor impairment. Journal of Autism and Developmental Disorders. 1995; 25:23–29. [PubMed: 7608032]

Mari M, Castiell U, Marks D, Marraffa C, Prior M. The reach-to-grasp movement in children with autism spectrum disorder. Philosophical Transactions of the Royal Society of London. 2003; 358:393–403. [PubMed: 12639336]

Mattingley JB, Phillips JG, Bradshaw JL. Impairments of movement execution in unilateral neglect: A kinematic analysis of directional bradykinesia. Neuropsychologia. 1995; 32:1111–1134. [PubMed: 7991078]

Maurer RG, Damasio AG. Childhood autism from the point of view of behavioral neurology. Journal of Autism and Developmental Disorders. 1982; 12:195–205. [PubMed: 7174608]

Miyahara M, Tsujii M, Hori M, Nakanishi K, Kageyama H, Sugiyama T. Brief report: Motor incoordination in children with Asperger's syndrome and learning disabilities. Journal of Autism and Developmental Disorders. 1997; 27:595–603. [PubMed: 9403374]

Muller RA, Pierce K, Ambrose JB, Allen G, Courchesne E. Atypical patterns of cerebral motor activation in autism: A functional magnetic resonance study. Biological Psychiatry. 2001; 49:665–676. [PubMed: 11313034]

Pantelis, C.; Brewer, W. Neurocogntive and neurobehavioural patterns and the syndromes of schizophrenia: Role of frontal-subcortical networks. In: Pantelis, C.; Nelson, HE.; Barnes, TRE., editors. Schizophrenia: A neuropsychological perspective. Chichester: John Wiley & Sons; 1996. p. 317-343.

Phillips JG, Bradshaw JL, Chiu E, Bradhsaw JA. Characteristics of hand-writing of patients with Huntington's disease. Movement Disorders. 1994a; 9:521–530. [PubMed: 7990847]

Phillips JG, Martin KE, Bradshaw JL, Iansek R. Could bradykinesia in Parkinson's disease simply be compensation? Journal of Neurology. 1994b; 241:439–447. [PubMed: 7931446]

Rinehart NJ, Bradshaw JL, Moss SA, Brereton AV, Tonge BJ. Atypical interference of local detail on global processing in high functioning autism and Asperger's disorder. The Journal of Child Psychology and Psychiatry. 2000; 41:796–778. [PubMed: 11039689]

Rinehart NJ, Bradshaw JL, Brereton AV, Tonge BJ. Movement preparation in high-functioning autism and Asperger's disorder: A serial choice-reaction time task involving motor reprogramming. Journal of Autism and Developmental Disorders. 2001a; 31:79–88. [PubMed: 11439757]

Rinehart NJ, Bradshaw JL, Moss SA, Brereton AV, Tonge BJ. A deficit in shifting attention present in high functioning autism but not Asperger's disorder. Autism: An International Journal of Research and Practice. 2001b; 5:67–80. [PubMed: 11708391]

Rinehart NJ, Bradshaw JL, Tonge BJ, Brereton AV, Bellgrove MA. A neurobehavioural examination of individuals with high-functioning autism and Asperger's Disorder using a fronto-striatal model of dysfunction. Behavioral and Cognitive Neuroscience Reviews. 2002b; 1:164–177. [PubMed: 17715591]

Rosenbaum, DA. Human motor control. London: Academic Press Limited; 1991.

Sattler, JM. Assessment of children—Revised and updated third edition. 3rd ed.. San Diego: Jerome M. Sattler, Publisher, Inc.; 1992.

Siegel BV, Asarnow R, Tanguay P, Call JD, Abel L, Ho A. Regional cerebral glucose metabolism and attention in adults with a history of childhood autism. The Journal of Neuropsychiatry and Clinical Neurosciences. 1992; 4:406–414. [PubMed: 1422167]

Szatmari P, Tuff L, Finlayson AJ, Bartolucci G. Asperger's syndrome and autism: Neurocognitive aspects. Journal of the American Academy of Child and Adolescent Psychiatry. 1990; 29:130–136. [PubMed: 2295566]

Teitelbaum P, Teitelbaum O, Nye J, Fryman J, Maurer RG. Movement analysis in infancy may be useful for early diagnosis of autism. Proceedings of the National Academy of Sciences: Psychology. 1998; 95:13982–13987. [PubMed: 9811912]

Vilensky JA, Damasio AR, Maurer RG. Gait disturbances in patients with autistic behavior. Archives of Neurology. 1981; 38:646–649. [PubMed: 7295109]

World Health Organization. The ICD-10 classification of mental and behavioral disorders: Clinical descriptions and diagnostic guidelines. Geneva: Author; 1992.

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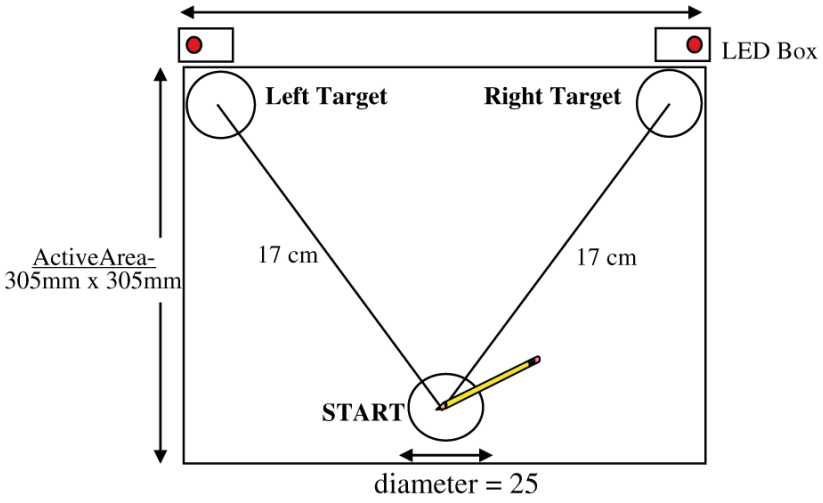

Fig. 1.
Configuration of targets and start position on the digitizing tablet. Small solid red circles represent the target LEDs

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Table 1

Sex, age, and full scale IQ range for the HFA group

Participants	Sex	Age	FSIQ range (IQ mid-point)
1	M	6.9	AV (99.5)
2	M	10.3	LAV (84.5)
3	M	9.1	LAV (84.5)
4	F	5.8	AV (99.5)
5	M	8.5	HAV (114.5)
6	M	5.5	AV (99.5)
7	M	7.3	LAV (84.5)
8	M	7.3	HAV (114.5)
9	M	7.9	AV (99.5)
10	F	11.8	AV (99.5)
11	M	9.6	LAV (84.5)
12	M	6.9	AV (99.5)
Mean		8.1	97.0
SD		1.9	10.8

Note. FSIQ range: LAV = Low average, AV = Average, HAV = High average

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Table 2

Sex, age, and full scale IQ range for the AD group

Participants	Sex	Age	FSIQ range (IQ mid-point)
1	M	15.7	LAV (84.5)
2	M	10.9	HAV (114.5)
3	M	7.0	SUP (124.5)
4	M	9.5	AV (99.5)
5	M	9.1	SUP (124.5)
6	M	19.8	HAV (114.5)
7	M	9.8	LAV (84.5)
8	M	16.1	BORD (74.5)
9	M	12.3	SUP (124.5)
10	F	6.2	LAV (84.5)
11	M	15.1	BORD (74.5)
12	F	12.2	HAV (114.5)
Mean		12.0	101.6
SD		4.1	20.1

Note. FSIQ range: BORD = Borderline, LAV = Low average, AV = Average, HAV = High average, SUP = Superior