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Differentiation of High-Functioning Autism and Asperger's Disorder Based on Neuromotor Behaviour

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Abstract Autism and Asperger's disorder (AD) are characterised by impairments in social interaction, stereotypic behaviours or restricted interests. Although currently listed as distinct clinical disorders, the validity of their distinction remains controversial. This study examined gait in children with autism and AD. Eleven children with high-functioning autism and eleven children with AD completed a series of walking tasks. Results indicated distinct movement disturbance; these findings are discussed in light of seminal papers in this field by Vilensky et al. (Arch Neurol 38:646–649, 1981) and Hallett et al. (Arch Neurol 50:1304–1308, 1993) who interpret the gait of individuals with autism using parkinsonian and cerebellar-ataxia patient models, respectively. Distinctions in gait patterns implicating perhaps unique motor circuit disturbances support the hypothesis that autism and AD may have unique neurodevelopmental trajectories.

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Introduction

Autism is currently diagnosed on the basis of the 'clinical triad' of impaired social interaction, impaired communication, and repetitive behaviours or restricted interests. According to current DSM-IV-TR definitions (APA 2002), autism is differentiated from Asperger's disorder (AD) on the basis of at least two of the following: delayed development of spoken language, impaired ability to engage in reciprocal conversation, stereotyped use of language (including echolalia), or lack of make-believe play. In around 70–80% of cases, autism coexists with intellectual disability. By contrast, there is no significant delay in language or cognitive development in the clinical definition of AD, although original descriptions (Wing 1981) make reference to unusual aspects of language and pragmatics, such as pedantic speech and a tendency to talk at length about topics of interest.

Differential diagnosis of autism and AD on the basis of language development is complicated by the presence of high-functioning individuals with autism who have a history of language delay but no associated intellectual delay. These individuals do not exhibit the frank expressive language deficits originally described by Kanner (1943); instead, high-functioning individuals frequently display the unusual aspects of language and pragmatics typically associated with AD.

There have been numerous attempts to identify the core cognitive and neurological deficits that underlie the clinical features of autism and Asperger's disorder.

To date, the few imaging studies that directly compare autism and AD indicate differential patterns of anomalous cerebral structure and function in the two disorders (Lotspeich et al. 2004; McAlonan et al. 2008). A study of motor preparation found qualitative differences between autism and AD, with autism associated with a parkinsonian pattern of cortical activation that implicated fronto-striatal dysfunction (Rinehart et al. 2006a). Anomalous cortical inhibition in autism (Rinehart et al. 2008) may underlie some of the clinical features of this disorder such as repetitive and stereotyped movements.

Neuromotor Functioning in Autism and Asperger's Disorder

It has been suggested that neuromotor dysfunction may be a core diagnostic symptom of autism (Leary and Hill 1996; Minshew and Williams 2007) and may precede the emergence of social and communicative impairments (Teitelbaum et al. 2004; Teitelbaum et al. 1998). In one of the first studies linking behaviour and neurological dysfunction in autism, Damasio and Maurer (1978) proposed that dyskinetic and dystonic movements in autism were associated with abnormal function of mesolimbic cortical structures, with the motor loop between the striatum, thalamus, and medial frontal lobes (and anterior cingulate) as potential sites of dysfunction. There have since been a small number of gait studies addressing the neurobiological underpinnings of abnormal behaviour in autism. Gait analysis, which involves the measurement of several key variables such as speed and stride length, is a particularly useful method of examining brain-behaviour relationships, as it provides a means of examining the integrity of the central nervous system and allows for the detection of very subtle neuromotor impairments (Brasic and Gianutsos 2000), particularly in children, as a mature gait pattern is reached by 7 years of age (Sutherland et al. 1980). As there is no single measure that determines the presence of abnormal gait, multiple variables must be examined to assess gait patterning and equilibrium. According to Gabell and Nayak (1984), stride length and stride time (cadence) reflect gait patterning, while base of support and double support time measure equilibrium. High gait variability has also been implicated as an indicator of abnormal gait (Patla 1996; Winter and Eng 1995), and appears to be a reliable indicator of generalised neuromotor dysfunction, as it has been found in several movement disorders such as Parkinson's disease and cerebellar ataxia (Blin et al. 1990; Ebersbach et al. 1999; Palliyath et al. 1998).

In one of the first quantitative studies of gait in children with autism, Vilensky et al. (1981) found a classic parkinsonian gait characterised by shortened stride length, confirming their hypothesis of underlying fronto-striatal motor dysfunction in autism. However, subsequent studies have found irregular, highly variable gait in adults with autism (Hallett et al. 1993) and wide base of support in children with autism (Ambrosini et al. 1998) that implicate a cerebellar-ataxic gait pattern. Recent findings from structural imaging studies and post-mortem examinations have confirmed the presence of anatomical and physiological abnormalities in striatal and cerebellar brain regions, as well as generalised cerebral anomalies such as macroencephaly (Nayate et al. 2005).

With the introduction of AD as a separate diagnostic entity in the DSM-IV (APA 1994), there is emerging anecdotal clinical evidence that neuromotor function may be differentially affected in autism and AD. Recently, a dissociation between autism and AD was empirically validated by Rinehart et al. (2006b, c), who found parkinsonian postural features in both disorders implicating involvement of fronto-striatal motor circuits, with additional cerebellar gait patterning in autism. The current picture is of a complex and widespread movement disturbance that qualitatively differs between autism and Asperger's disorder, and which involves multiple brain regions, including the striatum, cerebellum, thalamus, and prefrontal motor areas.

Current findings in the gait literature are consistent with Minshew and Williams' (2007) complex information processing theory of autism. Autism is associated with increased local connectivity (i.e. increased short- and medium-range cortical connections) and a reduction in long-range, intra-hemispheric connections, which may explain intact or enhanced low-level abilities typically associated with autism (e.g. superior perceptual or constructional skills) and impaired high-level functions such as executive skills that require complex integration of information. In the context of gait, children with autism show intact performance at the level of basic ambulatory functions (i.e. no evidence of grossly abnormal gait such as in cerebellar ataxia or Parkinson's disease), but appear to show subtle features of abnormal gait (e.g. increased variability) that indicate a high-level difficulty in integrating information to regulate movement. Accordingly, it may follow that gait disturbances in neurodevelopmental disorders manifest only in novel, challenging, or cognitively demanding environments.

The overall goal of the current study was to further explore the neural mechanisms that underlie gait disturbances in children with autism and AD; specifically, whether the movement disturbances are qualitatively distinct or appear to be of similar pathogenesis, and the implications of this in light of the neurobiological distinction between the disorders. Neuromotor dysfunction was explored by systematically examining gait performance (i.e. as measured with kinematic gait variables such as stride length, base of support, variability, etc.) in a range

of different environments and demands, including those contexts that influence gait in movement disorders of known aetiology, such as Parkinson's disease and cerebellar ataxia. A significant advantage of the current project over Rinehart et al.'s (2006b) previous study was the use of a gait analysis system that additionally offers step-to-step analysis of footfalls.

The aims of this study were to (a) investigate gait control by manipulating walking speed, (b) investigate the effects of cueing strategies such as auditory pacing and visual cueing, which are used to improve gait, and (c) to investigate high-level information processing in the context of gait by utilising a dual-task gait paradigm. It was hypothesised that children with autism and AD would show differential responses to these imposed environmental demands, implicating differential involvement of cerebellar and basal ganglia motor regions. It is further hypothesised that gait abnormalities will be enhanced in conditions of high cognitive demand that requires complex information processing (i.e. dual task conditions).

Method

Participants

Children with autism and AD aged 7–18 were recruited through a specialised assessment service, in the same manner as in Rinehart et al. (2006b). A team of experienced clinicians was involved in their diagnosis. Diagnostic information was collected using the revised Autism Diagnostic Interview (ADI; Lord et al. 1994), structured parent interview, direct child observations, and information from other sources such as teachers and therapists. (Due to ethical guidelines, ADI scores were not available to researchers). Assessment of intellectual functioning was undertaken with the age-appropriate form of the Wechsler intelligence scales (WISC-III, WISC-IV, or WAIS-III). Nonverbal intellectual functioning was estimated for each child using the perceptual reasoning index (PRI) score from the WISC-IV, or the perceptual organisation index (POI) score from the

WISC-III and WAIS-III. Performances on the other Wechsler indices could not be obtained from all participants; therefore, only PRI/POI index scores are reported. Inter-rater reliability, calculated on a sample of 107 cases, generated a Cohen Kappa of 0.95 for autism and 0.94 for Asperger's disorder, indicating strong agreement. Normally developing children were recruited from the community as a control group. Children with genetic disorders (e.g. Fragile X), comorbid medical illness (e.g. tuberous sclerosis) or a history of acquired head or spinal injury were excluded from the study. A total of 33 children participated in the study: 11 children with autism (9 male, 2 female), 11 children with Asperger's disorder (10 male, 1 female) and 11 healthy controls (8 male, 3 female). Table 1 summarises group characteristics for age, height, weight, PRI/POI score, and medication status.

There were no significant between-group differences for age $[F(2, 30) = 0.478, p = .624]$, height $[F(2,30) = 0.442, p = .647]$, weight $[F(2,30) = .455, p = .645]$, or PRI/POI $[F(2,30) = 3.002, p = .065]$. Nine children were medicated at the time of the study—six from the autism group (55%) and three from the Asperger's disorder group (27%). Post hoc analyses revealed there were no significant between-group effects between the HFA children on medication and those without medication ($p > .05$), for any of the gait conditions. Similarly, there were no group differences between children with AD on medication and those without medication ($p > .05$).

Informed consent was obtained from parents/guardians of all participants, in accordance with the Declaration of Helsinki. Ethical approval was obtained from the relevant human research ethics committees.

Materials

Gait was measured using the GAITRite® system (CIR Systems Inc., Clifton, NJ), which consists of an electronic walkway, 830 cm × 89 cm, with pressure sensors spaced 1.27 cm apart, embedded in a horizontal grid. The recordable area of the mat is 732 cm × 61 cm. Sensors have a frequency of 80 Hz and a temporal resolution of 11 ms. The sensors are

Table 1 Demographic information for the autism, Asperger's disorder, and control groups

	Autism (n = 11)[a]	Asperger's disorder (n = 11)[b]	Controls (n = 11)
Age (months)	154.82 (36.98)	143.64 (44.21)	160.45 (42.62)
Height (cms)	159.91 (17.97)	152.82 (16.96)	156.15 (18.14)
Weight (kgs)	53.24 (19.79)	46.76 (17.31)	47.16 (16.86)
PRI/POI[c]	97.09 (9.90)	102 (16.12)	110.91 (13.46)

[a] Six children were medicated: *P1* methylphenidate, *P2–P4* fluoxetine, *P5* fluoxetine and sodium valproate, *P6* risperidone and sodium valproate

[b] Three children were medicated: *P1* imipramine, *P2* fluoxetine, haloperidol, and methylphenidate, *P3* methylphenidate and sodium valproate

[c] Perceptual reasoning index and perceptual organisation index from the WISC-IV and WISC-III, respectively

connected to a standard IBM-compatible computer, via a serial interface cable. A footfall activates the pressure sensors, which triggers the closure of a switch. The timing of switch closures is used to calculate spatial and temporal gait parameters, such as stride length, speed, cadence, base of support, double support time, and stride time.

Procedure

The general procedure for gait analysis was similar in each of the studies. Participants completed three trials of each walking condition. A 2-m non-recordable zone was added to each end of the gait mat to minimise effects due to acceleration and deceleration. Trials were repeated when participants stepped off the recordable area of the mat or when incomplete footfalls occurred.

Statistical Analysis

For each variable, GAITRite® produced step-to-step values for all measures of gait in each walk. The following measures were calculated in the analysis of mean gait values: *speed* (centimetres per second), *cadence* (steps per minute), *stride length* (length of left and right steps in each gait cycle), *heel to heel base of support* (distance from heel point of one footfall to that of the perpendicular line of progression of the opposite foot), and *y-axis range* (maximum distance traversed laterally in the horizontal plane over a single walk). Mean values for each walk were calculated and averaged over all trials in that condition, to produce a global mean of each gait variable in each condition.

Coefficient of variability (CoV) was calculated as a measure of gait variability, using the formula: SD/ Mean × 100. Coefficient of variability is a common measure of gait variability (e.g. Ebersbach et al. 1999; Rinehart et al. 2006b, c), and was calculated by using the mean values across all trials and the variability of the distribution of those means. Variability was calculated for speed, cadence, stride length, double support time, and heel to heel base of support.

The procedure, statistical analysis, results, and a brief discussion of the results of each study will be discussed separately, and are followed by a general discussion of the results at the end of the paper.

Study 1: Effect of Self-Determined Walking Speed on Gait

Aims and Procedures

The aim of the first study was to examine gait and the relationships between spatiotemporal measures under conditions of self-determined walking speed. This was achieved by instructing participants to walk at three speeds:

Preferred

Participants were asked to walk at their preferred speed.

Fast

Participants were asked to walk at faster-than-preferred speed, and were instructed to avoid running. Across all groups, average increase in speed ranged from 10 to 70% from preferred speed (average increase was 41%).

Slow

Participants were asked to walk at slower-than-preferred speed. The average decrease in speed from preferred speed was 9–70% (average decrease was 48%).

Statistical Analysis

Data were analysed using SPSS v 12.0.1. Group differences for each gait measure were examined in each condition using one-way analysis of variance. Relationships between stride length and cadence were evaluated by determining the average slope and intercept of the regression equation describing these two variables.

Results

Significant group differences were found for base of support in all three conditions; preferred [$F(2,30) = 5.576$, $p = .009$], fast [$F(2,30) = 4.980$, $p = .014$], and slow [$F(2,30) = 3.663$, $p = .038$] walking. Post hoc testing revealed a significantly wider base of support in autism than controls ($p = .039$) and AD ($p = .012$) during preferred walking. Similarly, during fast walking, the autism group had a wider base than controls ($p = .036$) and AD ($p = .018$). In the slow walking condition, significant differences were found between autism and control groups ($p = .039$) but not between autism and AD groups ($p = .077$) (Table 2).

Significant differences were found in base of support variability [$F(2,30) = 4.341$, $p = .022$] at preferred speed. Children with AD were significantly more variable than control ($p = .018$) and autism ($p = .034$) groups.

Stride Length and Cadence Relationship

The linear regression equation for the cadence and stride length relationship was calculated for each participant,

Table 2 Spatio-temporal gait measures and variability measures for autism, Asperger's disorder, and control groups during preferred-, fast-, and slow-speed walking

	Autism (n = 11)	Asperger's disorder (n = 11)	Controls (n = 11)
Preferred			
Speed (cm/s)	136.46 (18.56)	136.81 (18.20)	124.79 (16.54)
CoV	8.61 (8.46)	9.18 (6.82)	4.63 (2.05)
Cadence (steps/min)	111.56 (15.10)	116.98 (14.01)	114.29 (13.09)
CoV	6.02 (6.72)	5.35 (6.39)	2.62 (1.73)
Stride length (cm)	147.98 (21.00)	141.82 (21.34)	131.31 (11.97)
CoV	4.77 (3.02)	5.11 (3.08)	2.65 (1.21)
Base of support (cm)**	11.16 (3.71)	7.65 (2.01)	8.51 (1.41)
CoV*	14.03 (6.42)	21.53 (8.86)	12.42 (7.72)
Y-axis range (cm)	16.88 (4.12)	15.49 (3.67)	16.98 (2.21)
Fast			
Speed	186.64 (22.26)	186.99 (32.29)	175.30 (25.37)
CoV	8.00 (6.20)	6.54 (5.29)	4.71 (3.02)
Cadence	137.94 (18.03)	141.97 (17.62)	135.83 (16.82)
CoV	5.49 (3.76)	4.78 (3.68)	2.89 (1.71)
Stride length	163.82 (22.51)	156.56 (26.89)	155.83 (20.47)
CoV	3.54 (2.47)	3.33 (1.76)	2.81 (1.75)
Base of support*	11.91 (4.33)	7.99 (2.54)	8.70 (1.92)
CoV	12.64 (5.98)	21.69 (21.94)	18.00 (17.77)
Y-axis range	17.95 (5.42)	16.63 (2.52)	15.66 (3.74)
Slow			
Speed	94.78 (31.78)	100.61 (24.42)	90.69 (18.20)
CoV	10.30 (5.60)	12.02 (9.02)	7.34 (3.28)
Cadence	86.17 (21.84)	99.01 (15.08)	94.01 (11.86)
CoV	7.23 (4.16)	7.15 (5.01)	4.91 (3.65)
Stride length	129.46 (25.76)	121.75 (20.92)	115.39 (16.73)
CoV	6.65 (3.42)	6.34 (4.45)	4.17 (2.31)
Base of support*	11.61 (5.13)	8.42 (2.43)	8.01 (1.68)
CoV	12.11 (7.55)	19.27 (14.41)	13.75 (9.79)
Y-axis range	18.12 (4.95)	18.09 (4.49)	16.25 (3.30)

* $p < .05$

** $p < .01$

using all cadence and stride length values across the preferred, fast, and slow walking conditions. Slope and intercept for each regression equation was then compared across groups. Table 3 shows the mean values for the regression slope and intercept, for each group.

Significant between-group differences emerged for the intercept of the stride length-cadence relation, $F(2,30) = 5.025$, $p = .013$. Post hoc analyses revealed a smaller *intercept* in the control group, compared to the autism group ($p = .012$). There were no differences between controls and AD ($p = .106$), or between autism and AD ($p = .582$). The *slope* of the relation did not significantly differ between groups [$F(2,30) = 2.166$, $p = .132$] (Fig. 1).

Table 3 Mean slope and intercept values for the regression equations describing the relationship between cadence and stride length in the autism, Asperger's disorder, and control groups

	Autism	Asperger's disorder	Control
Slope	0.68 (0.40)	0.75 (0.31)	1.07 (0.63)
Intercept*	72.08 (45.10)	53.90 (29.28)	15.68 (50.51)

* $p < .05$

Discussion

Findings implicate involvement of striatal and cerebellar motor circuits in autism. Wide base of support was consistently found across the preferred-, fast-, and slow-speed
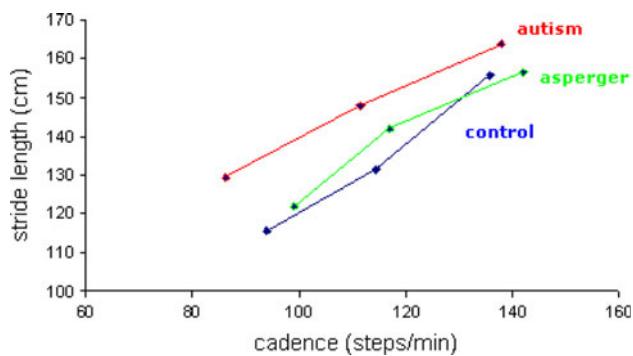
Fig. 1 Stride length and cadence relation for autism, Asperger's disorder, and control groups

walking conditions, which is clinically most closely linked with cerebellar disturbance (Hallett and Massaquoi 1993; Stolze et al. 2002; although see Palliyath et al. 1998) or frontal lobe lesions involving disruption of cerebellar tracts (Terry and Rosenberg 1995). In autism, this wide base of support implicating cerebellar involvement is consistent with previous gait studies in autism (e.g. Hallett et al. 1993; Rinehart et al. 2006c) and in line with documented cerebellar anomalies (e.g. Mostofsky et al. 2009).

Findings also revealed an unusual relationship between stride length and cadence in autism, with increased stride length at any given cadence compared to controls. Differences were most apparent during preferred- and slow-speed walking (around 14–16 centimetres greater than controls). This finding contrasts with the typical parkinsonian gait pattern where the stride length-cadence relation has a *smaller* intercept than controls (i.e. abnormally *shorter* stride length at any cadence; Morris et al. 1994). In Parkinson's disease, diminished activity of the basal ganglia reduces the state of "motor readiness" (sustained neural activity) necessary for the selection of stride length at any cadence and the maintenance of that stride length throughout the gait sequence. In autism, *increased* scaling of the intended movement, which produces a relatively abnormally *large* stride length, implicates a disturbance of motor set that results in a mismatch between intended and actual movements, and which may also be attributed to dysfunction of the fronto-striatal system. These findings are consistent with earlier electrophysiological evidence of abnormal pre-movement cortical activity in autism (Rinehart et al. 2006a), and may also account for the hyperkinetic, hyper-agile movements of Mari et al. (2003) in their reach-to-grasp task.

In contrast with autism, children with AD displayed variable base of support that was present only during preferred walking. Although the underlying mechanisms are not clear, variable gait may occur in any condition associated with neuromotor dysfunction, including Parkinson's disease, Huntington's disease, cerebellar pathology, and

subcortical arteriosclerotic encephalopathy (Blin et al. 1990; Ebersbach et al. 1999; Hausdorff et al. 1998; Palliyath et al. 1998). The amelioration of significant variability in the fast- and slow-speed conditions may reflect a relative normalisation of gait in AD when their attention is directed to a particular facet of walking (in this case, their walking speed), and may implicate cognitive/executive factors in the gait disturbance possibly implicating dorsolateral prefrontal compartments of the fronto-striatal system.

Study 2: Effect of Cueing

Aims and Procedure

The aim of this study was to examine the effects of visual cueing, a known technique for improving gait in Parkinson's disease, on gait in autism and AD. Participants completed three trials of each of the following two conditions:

Visually Cued

Participants were asked to walk on markers placed at equal intervals along the gait mat. White cardboard markers, each measuring 70 cm × 5 cm, were placed horizontally along the walking path at a distance 20% greater than individually determined preferred-speed stride length. Participants were instructed to walk at a comfortable speed while placing one foot on each marker. As the width of the markers exceeded the length of participants' feet, participants were asked to place their entire foot on the ground and ensure that part of their foot landed on each marker.

Non Cued

With the external visual cues removed, participants were asked to maintain the same step length as in the externally cued condition, by keeping the position of the markers 'in mind' while walking.

Statistical Analysis

Data were analysed using SPSS v 12.0.1. Two-way repeated-measures analysis of variance was used to determine group differences. Alpha was set at .05.

Results

Significant main effects of group were found for stride length variability [$F(2,30) = 4.143$, $p = .026$], and for base of support variability, [$F(2,30) = 5.673$, $p = .008$].

Table 4 Spatio-temporal gait measures and variability measures for autism, Asperger's disorder, and control groups during visually cued and un-cued walking

	Autism (n = 11)	Asperger's disorder (n= 11)	Controls (n = 11)
Visually cued stride length			
Speed	137.28 (24.88)	141.59 (33.09)	152.84 (26.65)
CoV	7.15 (4.71)	8.17 (4.26)**	6.36 (5.05)
Cadence	92.94 (19.00)	101.00 (18.80)	113.37 (16.77)
CoV	7.57 (5.00)	8.04 (4.31)	6.54 (5.35)
Stride length	180.38 (29.28)	168.92 (26.99)	162.14 (15.26)
CoV	1.20 (2.08)	0.76 (0.37)	0.66 (0.51)
Base of support*	10.33 (3.83)	7.18 (1.80)	8.04 (1.38)
CoV	17.46 (14.54)	30.03 (15.28)	19.19 (15.69)
Y-axis range	18.99 (3.46)	17.53 (3.65)	15.50 (3.32)
Non-cued stride length			
Speed	141.60 (34.87)	140.22 (37.70)	147.12 (17.95)
CoV	8.27 (7.96)	4.62 (3.05)**	5.06 (3.70)
Cadence	93.77 (21.83)	95.52 (18.76)	103.45 (11.84)
CoV	6.38 (6.86)	5.65 (4.79)	4.09 (3.61)
Stride length	183.68 (32.76)	175.73 (24.81)	171.64 (14.95)
CoV	6.24 (4.53)	3.90 (2.34)	2.56 (1.29)
Base of support	12.54 (5.68)	8.48 (2.79)	9.31 (2.92)
CoV	21.12 (12.24)	32.97 (17.67)	17.02 (8.65)
Y-axis range	16.37 (24.62)	14.95 (21.09)	13.07 (18.40)

* $p < .05$

** $p < .01$

Following a series of one-way ANOVAs, significant group differences were found during the non cued conditions for stride length variability [$F(2,30) = 4.128$, $p = .026$], and base of support variability [$F(2,30) = 5.673$, $p = .008$]. Post hoc tests revealed that the autism group had more variable stride length than controls ($p = .021$) and the AD group had more variable base of support than controls ($p = .024$), with no significant differences between autism and AD groups (Table 4).

Discussion

Increased stride length variability with visual cues provides further evidence of cerebellar involvement in autism (Ebersbach et al. 1999; Palliyath et al. 1998; Stolze et al. 2002), consistent with earlier gait research (Rinehart et al. 2006c). Increased variability contrasts with the typical parkinsonian response, where stride length cues produce a relative normalisation of stride length (Morris et al. 1994) and stride length variability (Stolze et al. 2001). In autism, increased variability with visual cues may reflect a difficulty of these individuals to efficiently integrate visual information with the basic gait program—a function subserved by the cerebellum (Kohen-Raz et al. 1992).

In AD, significantly higher base of support variability during the non cued condition may be attributable to the lack of external structure that was initially provided by the visual cue. Despite verbal instruction to maintain the increased stride length (i.e. implicit instruction to maintain attention to gait), the removal of the explicit cue may have unstructured the task to such an extent that this verbal instruction was ineffective. Of note, a previous study of cognitive-executive function utilising a random number generation task revealed an *improvement* in performance in children with AD with cueing, while there was no such improvement in autism. This may be attributable to the different cueing modality (i.e. visual in this study versus auditory in the random number generation study) or may support the notion of differential response of neurocognitive versus neuromotor performance in children with AD in response to visual cueing.

Study 3: Effect of Concurrent Task

Aims and Procedure

The purpose of this study was to examine gait under dual-task conditions. Each participant completed two dual-task

conditions. While walking at preferred speed, participants completed a counting task or a bimanual finger-thumb apposition task (see below). Participants completed three trials in each of the two conditions:

Preferred Walk and Counting

Participants walked at their preferred speed while completing a serial counting task (cognitive).

Preferred Walk and Tapping

Participants walked at their preferred speed while completing a finger-thumb apposition task (motor).

Due to the wide age range of participants, serial counting and finger-thumb apposition tasks were modified, with respect to difficulty, depending on the age and ability of the child. For the serial counting task, participants either counted backwards by 1s, backwards by 2s from an even number, or backwards by 2s from an odd number. For the finger-thumb apposition task, participants either completed a one-step movement (tapping the thumb and index finger repeatedly), or a four-step movement sequence (tapping the thumb to each finger in succession, starting with the index finger, and repeating). The most challenging counting/apposition task that the participant could successfully complete at baseline (i.e. fewer than two errors) was selected as the secondary task. All participants were able to complete at least one counting and one apposition task. Errors were uncorrected by the examiner throughout the testing session, and participants were encouraged to continue walking even if secondary task errors were made.

In each walking trial, the number of errors and the time taken to complete one counting/apposition cycle were taken as a measure of secondary task performance. Secondary task performance in each dual-task condition was evaluated in relation to single-task (i.e. baseline) performance. There were no confounding effects of secondary task performance, as performances of clinical participants did not significantly differ from that of controls.

Data were analysed using SPSS v 12.0.1. As in Study 2, two-way repeated-measures analysis of variance was used to determine group differences. Alpha was set at .05.

Results

Performances on the dual-task conditions were compared to walking at preferred speed (study 1) using two-way repeated-measures ANOVA. There were significant group × condition interactions for y-axis range for the cognitive task [$F(2,30) = 4.24$, $p = .024$] and for the motor task [$F(2,30) = 8.221$, $p = .001$]. Post hoc t tests revealed a significant increase in y-axis range in Asperger's

Table 5 Spatio-temporal gait measures and variability measures for autism, Asperger's disorder, and control groups under dual task walking conditions

	Autism (n = 11)	Asperger's disorder (n = 11)	Controls (n = 11)
Serial counting			
Speed	118.25 (40.21)	92.31 (23.60)	106.05 (40.21)
CoV	9.99 (7.07)	12.13 (17.59)	8.55 (4.10)
Cadence	94.96 (24.58)	90.41 (15.31)	99.61 (20.22)
CoV	6.10 (4.31)	11.62 (17.48)	5.13 (3.13)
Stride length*	147.73 (22.65)	122.55 (21.93)	127.80 (12.60)
CoV	5.92 (3.61)	4.89 (2.74)	3.68 (1.65)
Base of support	12.01 (5.54)	8.59 (2.39)	8.57 (2.05)
CoV	15.47 (10.04)	22.76 (13.91)	19.06 (9.18)
Y-axis range*	19.20 (5.65)	18.89 (4.21)	16.51 (2.91)
Finger-thumb apposition			
Speed	133.48 (31.71)	130.03 (36.65)	116.43 (21.00)
CoV	6.36 (3.62)	6.90 (4.10)	4.84 (2.02)
Cadence	109.39 (21.08)	115.26 (22.09)	108.90 (14.91)
CoV	3.88 (3.05)	4.11 (2.64)	2.71 (1.51)
Stride length	146.17 (18.16)	134.93 (20.95)	128.14 (13.80)
CoV	4.77 (3.26)	3.73 (1.65)	2.59 (1.23)
Base of support*	10.79 (4.95)	7.36 (1.84)	8.28 (1.65)
CoV	15.31 (7.92)	17.80 (14.34)	13.39 (13.73)
Y-axis range**	18.07 (5.97)	19.09 (3.32)	15.04 (2.43)

* $p < .05$

** $p < .01$

disorder with a secondary cognitive task ($p = .009$) and with a secondary motor task ($p = .009$). The autism group also showed an increase in y-axis range with a secondary cognitive task ($p = .049$); however, with adjusted alpha this difference was no longer significant (Table 5).

Discussion

Children with AD displayed increased y-axis range (i.e. greater number of footfalls occurring over greater width of the mat indicating variable direction of progression) under dual-task conditions. Increased y-axis range occurred irrespective of the nature of the secondary task (i.e. cognitive or motor), indicative of a decline in gait performance irrespective of the type of dual-task condition. This may result from dysfunction of high-level cognitive (executive) processes involved in attentional allocation to complete tasks simultaneously. Findings in AD are broadly consistent with a complex information processing difficulty (Minshew and Williams 2007) that leads to a difficulty with high-level integration of cognitive and/or motor

systems. A similar trend of increased y-axis range was observed in autism, although group differences did not reach significance.

General Discussion

The overall aim of this study was to further elucidate the neurobiological distinction between autism and AD through an analysis of gait under different imposed environmental demands. Consistent with our hypotheses and in-line with previous research, findings have shown a clear distinction in gait patterning associated with autism and AD. In autism, there was evidence of cerebellar (wide base of support, variable stride length with visual cues) and striatal (abnormally increased scaling of movement) involvement in the gait disturbance. However, the latter finding argues against previous studies (e.g. Damasio and Maurer 1978) that suggest a parkinsonian movement disorder in autism and implicate autism as an "early" form of Parkinson's disease. Rather, findings suggest that autism is associated with widespread neurobiological dysfunction that is qualitatively dissimilar from adult-onset disorders such as Parkinson's disease.

By contrast, children with AD generally responded differently to the range of gait conditions than the control group (and, in many conditions, the autism group). In some conditions, base of support variability in AD was only marginally higher than in controls; for example, walking at faster-than-preferred speed (difference of 3.69). In other conditions, however, AD had significantly higher base of support variability than controls; such as during the non-cued stride length walking task (difference of 15.95). The presence of such dramatic differences in base of support variability across conditions raises the possibility that cognitive/attentional factors associated with different conditions may be the primary influence on gait variability in AD, and may result from executive difficulties such as that associated with abnormal frontal lobe (dorsolateral prefrontal) or fronto-striatal function. Such marked differences in gait patterning were not apparent in autism, perhaps due to additional cerebellar involvement leading to a more static neuromotor disturbance that is consistently apparent across a range of conditions.

As hypothesised, a similar trend towards a decline in gait performance under conditions involving high level cognitive demands (i.e. dual task conditions) was found in both autism and AD, implicating complex information dysfunction as originally suggested by Minshew and Williams (2007) in autism. It appears that, while autism and AD are associated with qualitatively distinct patterns of cerebellar and striatal disturbance as evidenced under conditions of low cognitive demand, in more challenging conditions these low level disturbances manifest as a similar disorder of complex information processing. Clinically, these qualitative differences in low level neurobiology may account for the behavioural distinction between the two disorders (e.g. with regard to language function and the high co-occurrence of intellectual disability in autism), as well as the similarities in complex cognitive functions such as social interaction and repetitive/ritualised behaviours.

A strength of this project was the careful selection of three well-matched groups of children with high-functioning autism and Asperger's disorder, and normally developing children. This allowed for a direct examination of neuro-motor functioning without confounding effects of intellectual disability. However, the selection of children with 'high-functioning' autism limited the generalisability of findings, in light of the high coexistence of intellectual disability amongst individuals with autism. There is evidence that low-functioning children may display a qualitatively different motor impairment to high-functioning children (Mari et al. 2003). Functionally, low-functioning children with autism are likely to display greater impairment than the high-functioning children recruited in this study. A full understanding of neuromotor disturbance in autism, including the proposed interaction between motor and cognitive functions, would be enhanced by further research comparing low-functioning children with autism and an intellectually disabled, non-autistic comparison group.

Two limitations of these studies were the small sample and potential medication effects. Small sample size was the consequence of selecting a well-matched sample from a population of children with a rare disorder, to overcome confounding influences of age, intellectual functioning, height, and weight. The trend towards statistical significance that was observed in some studies may have resulted from insufficient power, due to the small sample. It will be important, therefore, to replicate the current preliminary findings using larger matched samples.

Medication effects were another possible consequence of examining groups of behaviourally disturbed children. Research in paediatric psychiatry is often unavoidably affected by group differences in medication status. In this study, medication effects could not be systematically investigated, due partly to the small sample but also to the wide range of medications prescribed to participants (stimulants, anti-depressants, anti-convulsant/mood stabilisers, and antipsychotics). Unwanted effects of medications include bradykinesia, dyskinesias, and tremor, which may affect gait performance and the ability of children to adapt their gait to external constraints. To off-set this limitation, the overall gait findings point away from a Parkinsonian-like gait in both clinical groups. Three children (2 autism, 1 AD) were medicated with sodium valproate, a medication

which may have a clinical effect on the cerebellum at high dose levels. As the 3 children in this study were medicated with low doses for mood stabilisation purposes, with close medical monitoring for side-effects including impact on motor functioning (author BT), it would be unlikely that the gait related cerebellar anomalies reported in this study were due to valproate.

Statistical comparison of participants on and off medication was complicated by the small sample; however, based on clinical observation and parent reports, there was no evidence of dyskinesia in any medicated child.

It is also important to point out that a general inspection of the gait data reveals that there are a number of instances where the clinical groups differed in a distinct manner and sometimes in an intermediary manner from the control group, for example, during fast walking, the autism group had a wider base than controls and AD, indicating a distinct pattern in this context, however, during the slow walking condition, the differences in base of support are less clearly distinguished between the clinical groups, and perhaps more accurately conceptualised as showing a milder anomaly in AD. Findings from our previous gait and EEG studies comparing children with autism and AD have shown similar intermediary differences, for example, in Rinehart et al. (2006b), the stride length variability recorded in the AD group falls in-between the autism and control groups (see also Rinehart et al. 2006a).

In conclusion, findings support the idea that some aspects of neuromotor functioning are qualitatively distinct in autism and AD, with areas where neuromotor anomalies are less pronounced in one or the other disorder. The different pattern of neuromotor findings may point to differential involvement of cerebral motor circuits. Subtle deficits in neuromotor function are clinically relevant as "surrogate markers" in not only improving early detection but also assisting in differential diagnosis, particularly in higher-functioning children with no significant developmental delay (Dowd et al. 2010). With regard to clinical definition, it may be more valuable to consider autism and AD as part of a 'fronto-striatal spectrum of disorders', rather than the current term 'autism spectrum disorders,' which is a clinically derived term that lacks international agreement as to its accurate use.

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