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Gait Analysis in Autism: A Potential Diagnostic Tool

Forster, Katharine

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Temporal Variability of Gait in High Functioning Autism: A Potential Diagnostic Tool

Katharine Ruth Forster

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Declaration

This work has not previously been accepted in substance for any degree and is not being concurrently submitted in candidature for any degree. This project is entirely the work of the writer's own investigation.

Signed...

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Gait analysis research has suggested development of a diagnostic tool to distinguish between diagnoses on the autistic spectrum. Research into anomalous temporal synchrony, coordination, Purkinje cells, 'clock genes' and the cerebellum combines to create a theory of temporal variability in autism. The gait of 16 participants with High Functioning Autism (HFA) and 16 age-matched typically-developing (TD) controls was assessed using VICON MX and 12 MXF40 cameras, sampling at a temporal resolution of 250Hz. Each individual participant had a mean and coefficient of variation (CoV) value calculated for the time taken to execute 'Step', 'Stance' and 'Swing.' No average timing difference was found. A significant difference in temporal variability ($p<0.001$) was identified by CoV values between HFA and TD groups for 'Step', 'Stance' and 'Swing.' No relationship existed between temporal variability of gait and Autism Diagnostic Observation Schedule scores. There was a trend for greater temporal variability in Left than Right Step within the HFA group, especially for younger participants, and those suspected to have Asperger's not HFA. No correlation existed for gait variability and age, suggesting suitability of gait analysis as a diagnostic tool. Results are consistent with previous theories implicating cerebellar dysfunction in the gait of individuals with autism.

Autism is a developmental disorder, characterised by problems with social interactions and communication, and spanning a wide spectrum of intellectual ability. Understanding how temporal synchrony and the cerebellum influence the heterogeneous nature of Autistic Spectrum Disorders could contribute toward a cohesive theory of the autistic aetiology and symptomatic presentation, specifically regarding the autistic gait. This research conducted gait analysis on children with autism to substantiate previous findings of anomalous gait in autism, by focusing on temporal variability, potentially providing support for gait analysis as a diagnostic tool.

Gait in Autism Spectrum Disorders

Clumsiness in Asperger's is reported consistently across cultures; motor delay and inadequate coordination with objects (i.e. balls) was reported in children with Asperger's (Miyahara, Tsujii, Hori, Nakanishi, Kageyama & Sugiyama, 1997). Movement disturbance is found in children with Autistic Spectrum Disorder, with anomalies identified in both the planning and execution of movement in this patient group (Mari, Castiell, Marks, Marraffa & Prior, 2003). Motor impairment in children with Asperger's was examined in contrast with children with a Specific Developmental Disorder of Motor Function (SDD-MF), both possessing an IQ over 80 (Green, Baird, Barnett, Henderson, Huber & Henderson, 2002). The Asperger's group were equally, if not more severely, motor impaired than the SDD-MF group.

The clinical picture presented in a study on autistic adults' locomotion suggested a disturbance of the cerebellum (Hallet, Lebiedowska, Thomas, Stanhope, Denckla & Rumsey, 1993). Research has shown variable stride length in the gait of children with autism, neurologically implicating the cerebellum and basal ganglia (Rinehart, Tonge, Iansek, McGinley, Brereton, Enticott & Bradshaw, 2006b). The authors hypothesized that basal ganglia and cerebellar dysfunction in autism would demonstrate developmental trajectory

effects upon motor symptoms. A GAITRite Walkway was used to gather spatiotemporal gait data (relevant for cerebellum and basal ganglia influences); Variable stride length measures reached a significant result compared to controls, consistent with the gait of cerebellar ataxia. The participants were blindly qualitatively rated, using Visual Analogue Scales, as being less coordinated and more variable within their own gait pattern, plus postural abnormalities were noted. Variability and coordination is indicative of cerebellar and basal ganglia involvement and posture is related to fronto-striatal basal ganglia dysfunction. The cerebellum is involved in controlling end-stage movement and thus dysfunction involves variable and irregular movements, whereas initiation of movement is more closely linked with basal ganglia. It was found that these features were stable throughout core developmental ages, which inspires possible use of gait analysis in diagnosis of autism in children.

Shetreat-Klein, Shinnar and Rapin (2012) found that all of the joints of autistic children, apart from the elbow, demonstrated greater suppleness than the control group. Using parental report measures, children with autism started walking over a month later on average than typically developing children, suggesting development delays. In conclusion, a total of 68% of the autistic children had a dysfunctional gait whereas only 13% of the typically developing children did. Wide-based gait was a commonly observed trait of the autistic gait, suggesting a lack of balance and postural control. Stride and step length and width were measured in children with autism using an ELITE system (Nobile, Perego, Piccinini, Mani, Rossi, Bellina & Molteni, 2012).   The authors found a reduced range of motion in children with autism, and that to increase stability they reduce speed, widen steps and shorten stride.

Balance and Posture

A range of stereotypies were compared between children with autism and

developmentally impaired children, covering purposeless movements of a repetitive or

rhythmic nature.   The autistic children demonstrated greater prevalence of stereotypies and

specifically displayed the most gait and hand or finger stereotypies, but not trunk stereotypies

(Goldman, Wang, Salgado, Greene, Kim & Rapin, 2008). However, the trunk is a relevant

component as a feature of postural sway, as found in other studies. Postural control in

children with ASD was investigated within both static and dynamic tasks (Fournier, Kimberg,

Radonovich, Tillman, Chow, Lewis, Bodfish & Hass, 2010). Children with ASD

demonstrated normalized mediolateral postural sway with 438% greater frequency. The

Centre of Pressure (COP) shift mechanism is a measure of competence for initiating walking

from a static posture. This was shown to be normal in autism for posterior COP, but there

were reduced lateral COP shifts; suggesting a lack of stability, or substituting different

strategies to achieve mediolateral momentum. Although these participants were capable of

forward momentum, dynamic balance was impaired.

Higher probability of flat footedness in children with autism is reported in a study

investigating foot pressure variables and temporal spatial measurements (Yang, Choi & Lee,

2012). This is thought to be due to a lack of control of plantar flexion. Double support time

and stance time were found to be longer, plus step width was wider than for controls,

suggesting balance difficulties.

Anticipatory postural adjustments (APA) occur for example during prediction of

unloading an object from one hand into another hand. Short-term adaptation of APA is

obsolete in cerebellar patients, with additional problems in this patient group in learning a

new APA in a novel situation. The cerebellum was also shown to be necessary for accurately

timing behaviours and movements that had previously been learned (Diedrichsen, Verstynen,

Lehman & Ivry, 2004).

Movement, posture, gait and balance disturbances in autism are thought to occur due to

neurological impairments. Kohen-Raz, Volkmar and Cohen (1992) examined posture in children with autism using a modern computerized posturographic method. Autistic children demonstrated greater variability and lateral sway, alongside decreased stability and tended to place disproportionate weight on a singular foot, toe or heel. In a later study, autistic individuals were subject to dynamic posturography (Minshew, Sung, Jones & Furman, 2004). The authors demonstrated reduced postural stability and delayed postural development in individuals with autism. Instability of posture was particularly evident with somatosensory input disruption, implicating deficits in sensory integration of multiple modalities at a neural level. Research on postural anticipation in children with autism (Vernazza-Martin, Martin, Lepellec-Muller, Ruto, Massion & Assaianate, 2005) investigated modulation of gait parameters, equilibrium control problems and whether locomotion was modified in accordance with verbal directions communicated by the researcher. A kinematic analysis of gait (ELITE system) was used, finding that the core deficits in autistic children regarding gait are associated with the 'goal of action'. The goal of action involved walking 5 metres to a playhouse, which resulted in greater gait anomalies than mere self-directed walking, implying movement-planning deficits. Internal control of multiple-joint coordination was appropriate, due to reliance on automatic level of walking, but when an external request was made, the cognitive demands interfered with efficient motoric action.

FMRI Investigations of Motor Impairments

Using fMRI, Muller, Pierce, Ambrose, Allen and Courchesne (2001) observed motor impairments in autism. Comparisons were made between visual stimulus instructing finger movements, and plain visual stimulation minus this motor command requirement.

Activations in the basal ganglia, supplementary motor area, cerebellum and thalamus were less pronounced in the group with autism in comparison to the control group. Interestingly, control subjects showed strongest activations in the contralateral central sulcus; yet

individuals in the autism group had an inconsistent array of locations showing the strongest

activations. This is possibly due to a wide range of compensatory mechanisms in autism that

differ between individuals. MRI research in patients with ASD has shown localised

structural basal ganglia anomalies (Qiu, Adler, Crocetti, Miller & Mostofsky, 2010). The

right hemispheric basal ganglia structure is deformed at the posterior putamen, responsible

for motor skill and the bilateral anterior and posterior putamen, responsible for praxis.

There is fMRI evidence to suggest a different neurobiology with regard to the Primary

Motor Cortex (responsible for motor control and learning) in children with autism (Nebel,

Joel, Muschelli, Barber, Caffo, Pekar & Mostofsky, 2012). Amongst all age groups and both

TD and ASD groups, the common pattern was a symmetrical dorsomedial to ventrolateral

organization, with the legs and trunk aspect of the motor homunculous corresponding to the

dorsomedial region and the oro-facial aspects of movement corresponding to the

ventromedial region. However, in children with ASD the dorsomedial cluster was

significantly greater, implying that the distinctions between upper limb and lower limb are

not as precisely defined, providing a possible explanation for fine motor control impairments

in autistic children. A developmental delay in specialisation of function within the Primary

Motor Cortex could explain the data of the ASD participants up to 12 years of age resembling

that of the TD 8-9 year old group.

Using a finger-tapping task, it was found that functional connectivity within motor

control regions is significantly reduced in autism (Mostofsky, Powell, Simmonds, Goldberg,

Caffo & Pekar, 2009). The ipsilateral anterior cerebellum demonstrated less activity in the

High Functioning Autism group, who instead displayed a higher degree of activation in the

Supplementary Motor Area. The lack of structural connectivity here can explain the Weak

Central Coherence symptoms of autism. In autism short connective fibres were evident in the

frontal and temporal areas, and even a smaller corpus callosum (structure connecting the two

hemispheres). Autistic children showed an excess of localised cortical connections and a greater proportion of radiate white matter volume within the Primary Motor Cortex, which positively correlated with motor impairment (Mostofsky, Burgess & Larson, 2007). This local over-connectivity is contrasted by long-range under-connectivity due to low prevalence of long-range fibres.

Diagnostic Function of Gait Analysis

The Eshkol-Wachman Movement Analysis System was used in conjunction with still-frame videodisc analysis to investigate retrospective videos of autistic children, from when they were just 4-6 months old (Teitelbaum, Teitelbaum, Nye, Fryman & Maurer, 1998). The analysis of autistic infants detected varying types of movement disturbance inherent in facial movement or developmental milestones including crawling, sitting, lying. Indeed, motor skills in participants diagnosed with an ASD at the age of two were reliably predictive of subsequent outcome (Sutera, Pandey, Esser, Rosenthal, Wilson, Burton, Green, Hodgson, Robins, Dumont-Mathieu & Fein, 2007). Those who subsequently display less autistic symptoms on a socialisation measure had lower original scores on tests examining fine motor control.

Research on children with autism used a GAITrite analysis system to investigate spatiotemporal patterns, finding variable stride length and time, a reduced ability to walk in a straight line and more mis-placed steps during a walking task along a white line (Rinehart, et al., 2006b). These features remained stable throughout development, suggesting that gait analysis is an appropriate diagnostic tool. In adults with autism gait remains anomalous; motion of the knee shows decreased flexibility. No correlation found between chronological age and gait anomaly (Vilensky, Damasio & Maurer, 1981) justifies the use of gait analysis, as this suggests gait anomalies in young children with autism are not simply a manifestation of developmental delay in physical maturation.

Early Diagnostic Method; Distinguishing Autism from Learning Disability

It is important to isolate features of autistic gait from a gait characteristic of a child with a learning disability, as co-morbidity is frequently noted. Eposito and Venuti (2008) researched the gait of toddlers with autism to assess whether gait could be distinguished from those with mental retardation or of typical development. Home videos were used in conjunction with the Walking Observation Scale and varying patterns of walking were reliably identified between the groups. In terms of diagnosing variation within the autistic spectrum, participants with the most anomalous gait scores also demonstrated the most profound autistic symptomatic presentation. According to Eposito, Venuti, Apicella and Muratori (2011), gait analysis is a valuable bio-marker for establishing gait differences between both control groups and children with Autistic Disorder. Significant differences were found between groups for the Positional Pattern for Symmetry during Walking and the Walking Observation Scale, with cerebellar influences deemed to be the most appropriate neurobiological explanation. A lack of correlation between IQ and gait anomaly suggests that the gait differences in children with autism are not simply a manifestation of mental retardation (Vilensky et al., 1981).

VICON 3-D Motion Capture System

A VICON system is excellent at capturing specific timing and was recommended as an improvement by Rinehart, Tonge, Bradshaw, Iansek, Enticott and McGinley (2006a) for gait research focusing on temporal variation. A study comparing 12 children with autism and 22 age-matched normative controls was conducted examining patterns of kinematic and kinetic gait (Calhoun, Longworth & Chester, 2011). The data was gathered utilising four force plates, 20 markers and an eight-camera motion capture system, obtaining twenty trials per child prior to analysis. Cadence, peak hip and ankle kinematics and kinetics were found to be significantly different between the two groups. Increased dorsiflexion angles in early stance

through to pre-swing were found in coincidence with reduced plantarflexor moments. Children with autism also had decreased hip extensor moments. No spatial-temporal differences were found but variability wasn't assessed. Subsequent research using VICON Motion Capture methodology and force plates focused on symmetry, and found no differences in symmetry between the autistic and control groups (Calhoun & Chester, 2012). Symmetry can be a biological marker of neurological abnormality but the current research proposes to establish neurological abnormalities by investigating timing anomalies specifically.

Further research has explored the effect of intention on locomotion, with the theoretical link of motivation in movement intent. Deficient abilities in movement planning are thought to induce this difficulty in goal orienting and accurately distinguishing the required trajectory. Planned motor response is evidently a significant problem in autistic locomotion, requiring preparation and timing governed by fronto-striatal systems (D'Cruz, Mosconi, Steele, Rubin, Luna, Minshew & Sweeney, 2009). One study required participants to walk in a straight line to pick up an object possessing either negative or positive motivation. Using a VICON Motion Capture System to analyse the gait, it was shown that the aversive emotional valence object reduced the efficacy of executive functions for movement planning in children with autism (Longuet, Ferrel-Chapus, Oreve, Chamot, Vernazza-Martin, 2012) resulting in motoric delays. Motivation is modulated by dopaminergic limbic reward systems, cohesive with theories regarding similarities between Parkinsonian and Autistic gait styles. An explanation is offered at a neurobiological level; movement planning is affected due to a deficient cerebral frontal and parietal cortex in autism. Emotional valence is modulated areas of the prefrontal cortex and only a positive emotional object will preserve the movement planning sufficiently to execute the motor goal in autism.

Differentiating Between Diagnoses on the Autistic Spectrum

The criteria for diagnoses of Autistic Spectrum Disorder (ASD) include a dichotomy

between Autistic Disorder and Asperger's Syndrome [Appendix X]. High Functioning

Autism (HFA) lies within Autistic Disorder, possessing average or above average intellectual

functioning. Asperger's criteria states average intelligence, with no history of language

impairment or delay.

Developmental dyspraxia was investigated in children with autism in terms of gesturing

ability in response to imitation and command and tool use, finding impairments in these tasks

for this clinical group (Steinman, Mostofsky & Denckla, 2010). The group with High

Functioning Autism (HFA) showed more tool-for-body-part errors than the Asperger's

Syndrome (AS) group, a deficit linked to the right supramarginal gyrus. This area is

implicated in language dysfunction, which can be a distinguishing symptomatic factor of

HFA from Asperger's.

Motor signs could distinguish children with autism from controls by using the PANESS

assessment tool PANESS (Physical and Neurological Exam for Subtle Signs), but did not

differ in motor signs between HFA and Asperger's (Jansiewicz, Goldberg, Newschaffer,

Denckla, Landa, & Mostofsky, 2006). Brasic and Gianutsos (2000) speculate gait to be

suitable for distinguishing subcategories of the ASD diagnosis because the cerebellar and

basal ganglia neurological variations between people on the spectrum are manifested in the

heterogeneous gait movements. Manjiviona and Prior (1995) compared children with AS and

HFA on motor impairment tasks, using the standardized 'Test of Motor Impairment –

Henderson Revision'. About 50% of children with Asperger's were found to have motor

impairment at a clinically significant level, compared to 67% of the HFA group.

Furthermore, assessment using the Bruininks-Oseretsky test of Motor Proficiency was

conducted amongst HFA and Asperger's participants (Ghaziuddin, Butler, Tsai &

Ghaziuddin, 1994); although motor clumsiness and coordination may not be a reliable

method of distinguishing the two conditions, there may be differences between their

movements qualitatively.

A comparison of gait between children with Asperger's Syndrome and those with

autism was undertaken (Nayate, Tonge, Bradshaw, McGinley, Iansek & Rinehart, 2012)

using a GAITRite System.   One component of this research focused on visual cues, with

markers on the floor to guide the positioning of foot placement. In autism, increased

variability in stride length was induced by this task, suggesting cerebellar dysfunction

because of the difficulty to integrate the visual cues with the demanding motor

component. Another feature of Nayate et al's (2012) study investigated dual tasks i.e.

counting or tapping while walking at a preferred speed. The Asperger's group demonstrated

a haphazard direction of progress across the gait analysis floor area; more foot placements in

a wider space. This difficulty to execute high-level cognitive tasks in conjunction with gait is

further support for a theory of dysfunctional complex information processing in

autism. There was no variation between Asperger's Disorder and Autism in this task.

In conclusion, the research conducted by Nayate et al. (2012), demonstrates a greater

neurobiological dysfunction in Autism as evidenced by more severe gait anomalies, than the

Asperger's group who showed some profoundly significant differences in some tasks, but in

other instances only a slightly higher degree of variability than controls. This is attributable to

cognitive faculties – in Asperger's only some of the tasks required more of the planning than

they could muster, whereas the group with Autism found that the higher processing required

to execute the tasks was usually in excess of their competencies. Thus cognitive faculties,

particularly in the dorsolateral prefrontal or frontostriatal regions of the frontal lobe, are

thought to contribute to the varying gait between these two clinical groups. Greater

cerebellar disturbance in Autism is thought to be the explanation behind a more consistent

neuromotor dysfunction according to task.

A study investigated whether Asperger's, Low Functioning Autism (LFA: IQ < 70) and High Functioning Autism (HFA: IQ > 70) – established by a combination of ADOS, ADI-R and DSM-IV scoring - differed in their neuroanatomy (Lotspeich, Kwon, Schumann, Fryer, Goodlin-Jones & Buonocore, 2004). Asperger's was found to be on the mildest end of the Autistic spectrum in terms of cerebral gray tissue. For the HFA and Asperger's groups, cerebral white matter and age bore a significant positive correlation; conversely, cerebral gray tissue reduced as age increased in children with autism, suggesting a complicated relationship of developmental trajectories.

Multiple research projects by Rinehart and colleagues have investigated differences in gait between patients diagnosed with Asperger's Syndrome or autism. Differences in movement abnormalities were outlined using a serial choice reaction time task; with deficits in motor control in autism seen as a result of a lack of anticipation and in Asperger's were perceived as a problem in motor preparation (Rinehart, Bradshaw, Brereton & Tonge, 2001). The fronto-striatal region is thought to be the influencing faculty, with the anterior cingulate and the supplementary motor area showing most involvement. The anterior cingulate is implicated in attention to action and is part of the basal ganglia thalamocortical circuitry. Along with the Supplementary Motor Area, this is thought to induce the executive dysfunction symptoms of autism, a presentation more prevalent in High Functioning Autism than Asperger's Syndrome. Participants with Asperger's displayed a lack of preparation, whereas those with HFA showed a lack of anticipation within the preparation of movements. Varying frontostriatal involvement is again thought to explain these motoric differences. A button-pressing task was used, with HFA failing to correctly anticipate the next button in the sequence, whereas control participants become faster in sequences. The Asperger's group actually became slower. Movement execution is intact in these clinical groups but atypical preparation is the hindering factor.

Another study (Rinehart, Bellgrove, Tonge, Brereton, Howells-Rankin & Bradshaw, 2006c) used a kinematic paradigm, allowing separate analysis of the expectancy and inhibitory components of executive functioning in movement execution. Autistic participants demonstrated greater impairment in initiation and preparation of movement. Elements of their gait can often suggest cerebellar difficulties and not the basal ganglia related explanation of executive dysfunction. It was found that the motor planning deficits differed between the two groups. Planning, and not the execution of movement, was impaired. HFA showed a more pronounced degree of this planning impairment. When a demand was expected, the typically developing group were faster than for an unexpected demand, whereas HFA were unaffected.

Research specifically focusing on the gait elements of motor disorder also revealed significant differences between the degree of autistic diagnosis. In a comparison between the gait of people with autism and those with Asperger's disorder Rinehart et al. (2006a) used matched controls (age, IQ, weight, performance, height) and a Clinical Stride Analyzer. Those with autism had increased variability of stride-length compared to the other groups. The participants with Asperger's were the only ones who demonstrated anomalous head and trunk posture. It was suggested that the variation between autism and Asperger's in gait was due to differing cerebellum effects and the similarities were thought to be due to the congruent influence of the basal-ganglia frontostriatal region. The cerebellum is thought to be more heavily implicated in this autism because the gait is more variable, whereas the Asperger's Disorder group showed no specific gait abnormalities, only upper body and postural anomalies, which are more associated with the basal ganglia (Rinehart et al., 2006b.

Focus on A-rhythmic Gait in Autism

Variable stride length and duration is consistently found across other autistic gait studies too, thus substantiating the cerebellum and basal ganglia influences in timing of motor

performance in autism. Vilensky et al. (1981) conducted kinesiologic analysis of autistic

gait, showing that they presented reduced stride lengths and increased stance time. They also

found decreased knee extension and ankle dorsiflexion as the foot contacts the floor, plus

greater hip flexion at 'toe off'. Interestingly the ankle joint angle became closer to the

normal range as a function of higher IQ in autism. It was acknowledged that there were

similarities between the gait of participants with Parkinson's disease and the participants with

Autism. This was thought to be result of anomalies in the striatum and frontal cortex, as these

areas project to the basal ganglia, especially the Supplementary Motor Area and the anterior

cingulate gyrus. The findings of Vilensky et al. (1981), in which the reported mean cycle

durations showed no significant difference between the typically developing and the ASD

group, are of particular interest. The current study hypothesises high temporal variability in

gait cycle timing but no overall difference in cadence.

Aetiology of the Autistic Gait: 'Clock Genes'

A seminal study investigating the possible hereditary nature of autism showed a 60%

concordance rate in monozygotic twins, yet no concordance rate found in dizygotic twins

(Bailey, Le Couter, Gottesman, Bolton, Simonoff & Yuzda, 1995). Subsequent work on

genetic correlates of autism contributed evidence that the dysfunctional circadian rhythms of

autistic individuals and their consequential social and motor deficits are influenced by

anomalous 'clock genes'. Single-nucleotide polymorphisms were found at a significant level

($P< 0.05$), two in both *per1* and *npas2* (Wimpory, Nicholas & Nash, 2002; Nicholas,

Rudrasingham, Nash, Kirov, Owen, & Wimpory, 2007). These epistatic clock genes hold an

aetiological function in High-Functioning Autism, potentially influencing temporal

synchrony; the capacity for rhythmic turn-taking communication between adults and pre-

verbal infants with integrated gestures (Feldman, 2007).

The connection between 'clock genes' and social communication is evident even in

the fruit fly, Drosphila (Kyriacou & Hall, 1980) with their courtship rituals of wing-flapping

conforming to a rhythm. This rhythm is governed by 'per' regulation of the short-period

oscillator entwined with this communicative wing-flapping (Konopka & Benzer, 1971;

Konopka, Kyriacou & Hall, 1996; Ritchie, Halsey, & Gleason, 1999). Considering the

anomalies of 'per' in autism, this is an intriguing theory for social timing deficits.

Several other genes have been identified and postulated to also have an

influence. Those shown to influence timing distortions in the brain are likely to be of

considerable importance. A pathway, involving synaptic cell adhesion molecules (NRXN1,

NLGN 3-4) and SHANK3, a scaffolding protein operating at post-synaptic level, has been

linked to a genotypical propensity to develop autism (Bourgeron, 2007). SHANK3 is needed

for synapse function and in maintaining appropriate ratios of inhibition and excitation within

neurons.

Genetic Correlates in Relation to Purkinje Cells

Purkinje cells are neurons responsible for inhibitory projection and are located in the

cerebellum, a structure vital for motor control. Further genetic links have been found

implicating the genes RORA (Messer & Kang, 2000; Nguyen, Rauch, Pfeifer & Hu,

2010). These epistatic genes affect circadian rhythms; RORA in particular is involved in the

cerebellum. Mutation of RORa-*staggerer* retards cerebellar Purkinje cell development in

neonates. The cells that survive the apoptotic death of being blocked by the RORA-*staggerer*

mutation are smaller and have insufficient dendritic formation, thus severely reducing

postsynaptic site connections.   Post-mortem research confirms that Purkinje cells in people

with autism showed a 24% size reduction; indeed some of the brains demonstrated Purkinje

cells that were 50% smaller (Fatemi, Halt, Realmuto, Earle, Kist, Thuras & Merz, 2002).

Furthermore, neuropathological studies in autism have demonstrated a shortage of

cerebellar Purkinje cells (Palmen, Engeland, Hof & Schmitz, 2004). Brain-derived

neurotrophic factor (BDNF) is a neurotrophin necessary for Purkinje cells and has been found

to be present in a higher concentration in the neonatal blood of children with autistic

spectrum disorder; overexpression of the neurotrophin is likely to contribute to Purkinje cell

abnormalities in autism (Nelson, Grether, Croen, Dambrosia, Dickens & Jelliffe, 2001).

The Olivocerebellar System

The inferior olive is embedded within the medulla oblongata (located in the brain

stem) and regulates autonomic functions like respiration and cardiac rhythms. Much of the

brain's electrical synaptic processes occur within the inferior olive (Llina's, Baker, & Sotelo,

1974; Sotelo, Llina's & Baker, 1974). The olivocerebellar system originates in the inferior

olive and includes the inferior olive neurons within the ventral medulla, deep cerebellar

nuclear neurons and Purkinje cells of the cerebellar cortex. It is important to note that

Purkinje cells receive projections of inferior olive axons and project to the deep cerebellar

nuclear neurons.

In autism the delicate arrangement of anatomy within the olivocerebellar structures is

disrupted by ectopic groups of inferior olive neurons, thinning inferior olive neuropil and

duplicated inferior olive subnuclei (Bailey et al.,1995). The structural arrangement of

inferior olive neurons is problematic in the autistic brain, with a clumping of neurons

occurring around the periphery and not distributed evenly as in the typically developing brain

(Bauman & Kemper, 1985; Kemper & Bauman, 1993).

Neural Correlates

Approximately 89% of autistic individuals demonstrate cerebellar pathology in the

form of hypoplasia of the cerebellar hemispheres and vermis. An 18% reduction in size of

the midsagittal region of the vermian lobules (Courchesne, Townsend, Akshoomoff, Saitoh,

Yeung-Courchesne, Lincoln, James, Haas, Schreibman & Lau, 1994) specifically a shortage

of cerebellar neurons, with a severe Purkinje neuron loss of around 50% (Arin et al., 1991), a

hypoplasia that appears to decline within the first year (Hashimoto, Tayama, Murakawa,

Yoshimoto, Miyazaki, Harada, & Kuroda, 1995).

In a later study examining neuropathological findings in autism, Palmen et al. (2004)

repeat these conclusions. Cell packing density is considerably greater while the general size

of the neurons is on average smaller in autism. Purkinje cells were found to be reduced in

quantity and morphological changes were found in relation to age in the cerebellar

nuclei. The cerebral cortex was also found to display signs of cortical dysgenesis in most

cases. These findings are important when considering the implications of a neural aetiology

for timing dysfunctions in gait execution.

Differences in cortical folding abnormalities have been found between Low and High

Functioning Autism (LFA/HFA) and Asperger's Disorder (Nordahl, Dierker, Mostafavi,

Schumann, Rivera, Amaral & Van Essen, 2007). Magnetic Resonance Imaging research

revealed that LFA showed cortical folding abnormalities at the pars opercularis of the inferior

frontal gyrus, an area partially covering the insula, forming Broca's area in conjunction with

pars triangularis. Subjects with HFA demonstrated a different cortical folding abnormality in

parietal operculum and ventral post-central gyrus. The former region is part of the parietal

lobe and adjacent to the Secondary Somatosensory Cortex, responsible for sensory and motor

integration. It is connected with the parietal ventral cortex, which projects to the premotor

cortex. The ventral post central gyrus contains the primary somatosensory cortex and sensory

homunculous. Asperger's Disorder showed an alternative abnormality in the folding of the

intraparietal sulcus, responsible for perceptual-motor coordination and perception of depth

from stereopsis. Weak central coherence theories are linked to abnormal parietal cortex

connectivity. As the brain develops regions are pulled together if continuously reinforced by

links, forming gyri. Conversely, unconnected areas drift apart to form sulci. These findings

are important to consider when noting differences between specific ASD diagnoses and their corresponding distinctive gait; the disorders do not suggest a simple continuum of one neurobiological anomaly.

Deficient Visuocerebellar Pathway

Motor imitation is found to be a deficit in autistic children, posited to contribute to limited use of gesture (Jones & Prior, 1985). Mirror neuron system abnormalities are implicated in autism, with evidence to suggest that postural knowledge and imitation of skilled movements are deficient as a result (Steinman et al., 2010). Autistic participants experienced difficulties when transforming a spatiotemporal version of movements into action, a task reliant on left parietal pre-motor networks to guide goal-directed motion. There is excessive reliance on proprioceptive feedback as opposed to visual feedback when modulating motoric action. Interestingly this advantages children with autism in a moving room scenario task as they are more stable in this context than participants with Asperger's and controls (Gepner & Mestre, 2002). A reduced reliance on visual input to modify posture means they are hyporeactive due to visuopostural detuning, and thus can maintain stability under a moving room illusion more efficiently than other groups in this study. A deficient visuocerebellar pathway is a possible explanation.

It is interesting to note that without timing competence, there appears to be no corresponding acuity in the observation of biological motion. Usually infants as young as one day old will prefer to look at the motion of people, with areas of neural responsibility implicated in the same brain regions that process facial expressions and social cues. Infants with autism however, preferentially attend to non-biological aspects of the motion displays that are disregarded by typically developing children (Klin, Lin, Gorrindo, Ramsay & Jones, 2009).

Basal Ganglia and Cerebellum Comparisons

Movement disorders in autism and the neurobiological influence of the basal ganglia compared to the cerebellum were investigated (Nayate, Bradshaw & Rinehart, 2005). The authors stated that the cerebellum allows locomotive control to be executed in an integrated manner by coordinating information from higher and lower brain centres. The Cerebellar Locomotor Region is specified as having a critical role, lying adjacent to the fastigial nucleus. The fastigial nucleus enables coordination and balance by integrating sensory information. The cerebellum receives input from the premotor cortex regarding intended motion and actual motion input from ascending pathways. Conversely, the basal ganglia are implicated in successful initiation and maintenance of motion via minute nuancical postural alterations. Their connections to the Supplementary Motor Area are postulated to be influential in executing complex sequences of locomotion.

MRI research into the basal ganglia in autism demonstrated anomalies (Sears, Vest, Mohamed, Bailey, Ranson & Piven, 1999). The caudate, putamen and globus pallidus were subject to volumetric measurements, finding increased caudatal volume in the autistic group; this was associated with complex motor mannerisms and compulsions.

Basal Ganglia and Gait

The basal ganglia are heavily implicated in gait and the execution of movement sequence and are especially important for alterations in posture and initiation of movement. The abnormalities of this neural structure in Parkinson's Disease suggest that the gait of affected individuals will demonstrate a greater stride-to-stride variability with difficulties experienced in maintaining a steady rhythm in the gait cycle. Quantitative measures of Parkinsonian gait substantiate this basal ganglia theory, with positive correlations between severity of gait variability and disease (Hausdorff, Cudkowicz, Firtion, Wei & Goldberger, 1998). In Autism there is evidence for a larger caudate volume and a decreased ability to metabolise glucose in the left posterior putamen (Nayate et al., 2005), a

component of the basal ganglia. It is thought that this originates from prenatal abnormalities, with caudate volumes showing unusual neuronal migration. However, Asperger's disorder shows different basal ganglia abnormalities, with less grey matter. The Supplementary Motor Area, implicated in sequences of locomotion, is connected to the basal ganglia; the basal ganglia prepares information by regulating the coordination of subcomponents of a motor sequence hence anomalies in this area contribute to gait abnormalities.

Parkinson's Disease and the autistic gait. Research into lesion location and Parkinsonian gait disturbance found that pallidotomy led to variable improvement in motor symptoms according to the lesion position in posteroventral globus pallidus internus (Gross, Lombardi, Lang, Duff, Hutchison, Saint-Cyr, Tasker & Lozano, 1999). The globus pallidus is a significant component of the basal ganglia thus influencing motor control. The subthalamic nucleus (STN) is also situated within the basal ganglia system and has been found to be influential in gait in Parkinson's Disease (Yokoyama, Sugiyama, Nishizawa, Yokota, Ohta & Uemura, 1999).

Similarities have been observed between the Parkinsonian gait anomalies and the gait patterns of individuals with autism (Damasio and Maurer 1978; Eposito & Venuti, 2008; Mari et al. 2003; Vernazza-Martin et al. 2005; Vilensky et al. 1981). Hollander, Wang, Braun and Marsh (2009) investigated shared pathology and brain circuitry involving the basal ganglia, with attention to shared dysfunctional neural mechanisms, particularly the dopaminergic system. Blin, Ferrandez and Serratrice (1990) conducted a quantitative analysis of Parkinsonian gait, resulting in specific interest in the variability of stride length. There were differences between PD and controls for spatio-temporal and kinematic parameters. As the disease progressed, the stride length variability increased further. Stride-to-stride variations in Parkinson's have also linked to basal ganglia dysfunction by other authors (Hausdorff et al., 1998).

Cerebellar Ataxia and Parkinson's Disease. Quantitative gait analysis has been conducted comparing patients with cerebellar ataxia and PD, showing that both groups had slower velocity and diminutive step length compared to normative populations, but the cerebellar ataxia participants also had increased variability in step timing and disproportionate stride length as velocity requirements changed (Ebersbach, Sojer, Valldeoriola, Wissel, Muller & Tolosa, 1999). Cerebellar ataxic gait was investigated by Ilg, Golla, Thier and Giese (2007), by conducting quantitative analysis of gait and spatial/temporal variability of intra-joint coordination, showing significant correlation between temporal variation and intra-limb coordination for goal-oriented locomotion.

Earhart and Bastian (2001) investigated whether cerebellar patients with gait ataxia could appropriately adjust peak joint ankle amplitudes in correspondence with varying levels of inclining surfaces. Peak amplitudes of individual joint angles were appropriately adjusted, apart from the timing of ankle movement, namely the peak ankle plantarflexion. Coordination deficits were noted when participants were required to integrate multiple joints simultaneously. The authors postulate that the demanding locomotive task is subject to decomposition in cerebellar subjects; placing ankle joint in dorsiflexion simplifies the otherwise complex movement. Ivry (1996) has suggested that the internal clock and timing, including movement acuity, is influenced by subcortical structures; the cerebellum and basal ganglia.

Cerebellum and Modulation of Movement

The concept of timing continues to hold relevance as the neurology of such a mechanism is explored. Morton and Bastian (2007) highlight the influence of the cerebellum in control of movement and locomotion, for eliciting the correct limb movement patterns and allowing dynamic modulation of appropriate posture. The cerebellum modulates actual movements if they do not correspond to the information from the premotor cortex regarding

intended movements (Nayate et al., 2005). Cerebellar ataxia is a condition caused by lesion in

this area, resulting in a poorly regulated gait pattern that lacks coordination and rhythm.

Clinical features of cerebellar ataxia include highly variable gait measures, increased

instability during locomotion and inappropriate foot placement timing (Stolze, Klebe,

Peterson, Raethjen, Wenzelburger & Deuschl, 2002). Cerebellar patients with mild/moderate

gait ataxia differed from controls in the timing of peak ankle plantarflexion and demonstrated

higher intra-variability of peak hip and knee joint angles. Their coordination of joints

showed severe deficits between hip, knee and ankles, suggesting that the cerebellum is

necessary for modulating the relative motion of these joints (Earhart & Bastian,

2001). Cerebellar patients were found to have deficits in maintaining simple rhythm and

differentiating between interval durations (Ivry & Keele, 1989). It was postulated that the

influence of the cerebellum as a timing mechanism is a specific function of the motor control

system. The aforementioned abnormalities in the cerebellum in autism provide the

neurological justification for anomalous gait in this group.

This Research

This research study used a VICON Motion Capture System (recommended as an

improvement by Rinehart et al., 2006a) to assess temporal parameters of gait in people with

High Functioning Autism. The sophisticated technology can detect very specific timing of

events within the walking cycle.   It is expected that a significant effect will be found for

autistic gait in comparison to the Typically Developing controls, due to the multitude of

studies implicating the cerebellum for motor control clock gene and temporal synchrony

hypothesis (Wimpory et al., 2002) and. It is hypothesised that gait abnormality will correlate

with the degree of autism as measured by ADOS (Autism Diagnostic Observation Schedule).

The majority of measures on timing parameters in other studies have looked at cadence

or irregularities in step length or stride length patterns, which is an effective way to examine

anomalies in walking cycles. However, it was not intended to replicate findings of inconsistencies in length of step or to determine whether on average the cadence of a person with autism differs from the norm. The present research focused on timing variability, recording the length of time it takes participants to execute four components of the gait cycle: From toe off to foot strike ('Swing time'), from foot strike to toe off ('Stance time'), one foot strike to the next foot strike ('Step time') and strike of one foot to the next strike of the same foot ('Stride time'). The technology used allows the time it takes for each element of the step process to be recorded to a resolution of 250Hz, or every 4 milliseconds. This allows comparisons to be made between autistic and control participants regarding the consistency of the time taken for elements of the walk and brings an alternative mode of timing data to research on the autistic gait. If a significant result is obtained in the aforementioned domains, it will provide support for a link between the dysfunctional circadian rhythms as demonstrated by clock gene research (Wimpory et al., 2002; Nicholas et al. 2007), neurological implications of timing function in the cerebellum, Purkinje cells and basal ganglia and their corresponding influence upon gait variability often displayed in individuals with autism.

Hypothesis. Children on the autistic spectrum are expected to have a higher variability in their timing within all elements of the gait cycle compared to age-matched controls. A positive relationship between variability of timing in the gait cycle and participant's ADOS scores is expected. It is expected that average speed is no different between groups, only the variability is hypothesised to present differently. To be able to substantiate diagnostic methods as a distinguishing tool for varying levels of autism, or to diagnose a child earlier, could benefit clinicians involved in autism and Asperger's.

Method

Participants

37 participants were contacted, adhering to defined ethical protocol. They were existing clients of Dr Wimpory (Consultant Clinical Psychologist for Autism, Betswi Cadwaldar University Health Board, Lecturer/Practitioner), or Clinical Psychologist colleagues. Participants with High Functioning Autism were defined in the request, to avoid co-morbid Learning Disabilities. 16 male participants with autism were recruited between the ages of 7 and 35. There was no exclusion on the grounds of ethnicity. Inclusion criteria required that the participants are ambulant due to the nature of the research. Parents of participants under 18 were requested to attend the participation sessions, and for those over 18 the parents were invited. Data tables include participant information with an anonymous number of identification, according to the order they were contacted (1-37).

TD participants were recruited from local schools by specifying to the schools the Date of Birth required; the same age within 5 months of the participant they are to be matched with. As this recruitment process slowed, psychologists at Bangor University were contacted about possible TD databases, plus friends of Dr Dawn Wimpory.

Materials

Bilingual versions of all documents were provided, courtesy of the NHS translating system. Dr Wimpory in clinical psychologist role sent out an invitation letter [Appendix A] to the home address of current clients. If the potential participants were not on her current clinical case-load, then they were approached initially by their current treating clinician / GP. This Invitation Letter was written from the clinician on the first page [Appendix B], with space to insert the clinician's details, and attached overleaf was our generic invitation letter.

A different version of invitation letter was sent to schools to distribute to appropriately aged TD children [Appendix C]. Stamped self-addressed envelopes were included for consent forms to be returned prior to direct contact, in agreement with ethical protocol. [Appendix D]. All

participants or their parents signed a consent form. Versions were available for adults without

capacity for consent; parents would sign a 'Consultee Declaration Form' [Appendix E].

An information sheet included brief information on the VICON motion capture system with

VICON Nexus software. The purpose and processes of the research were explained, as well as the

potential benefits of another diagnostic tool. No risks were identified for the participant, and full

right to withdraw was made explicit. Contact details were provided. Different versions of the

information sheets [Appendix F] were written according to the age of the participant, their capacity

to consent status and whether they were in the Typically Developing group or the group with High

Functioning Autism. An easy-read informative leaflet was compiled [Appendix G] by Ms Oonagh

Eason, describing the VICON Motion Capture System. This was distributed to all potential HFA

and TD participants. After participation, debrief forms were distributed [Appendix H], with a

version for parents, adult participants, and parents of TD participants. Participants were sent a

follow up letter explaining the findings of the study [Appendix I] and a letter was sent to the

participants' GPs to inform them of their patient's participation [Appendix J].

Apparatus

A 3D VICON Motion Capture System with 12 (MXF40) cameras sampling at a rate of

250Hz and 20 reflective markers with specialist double sided adhesive tape were used in the Gait

Analysis Laboratory at Bangor University's Sports Science department, Normal Site, Holyhead

Road, Bangor, Gwynedd. VICON Nexus software (Version 1.4.115) was used to process the data

detected by cameras for a range of 4 metres by 4 metres within the room. The software plots the

data derived from the reflective markers and shows an unidentifiable computerised image of a

walking stick figure, mapping biological kinematics.

The Autism Diagnostic Observation Schedule (ADOS) was used to provide a quantifiable

measure of autism severity. Most participants had an existing ADOS score, but for those who didn't

we used Module 1,2, 3 and 4 [Appendix K] to assess these clients, tailored to the age and language

capacity of the individual. The assessment measure involves a series of structured and semi-

structured tasks that require a social interaction between the client and the administrator. The

observations are assigned to predetermined categories, which produce a quantitative score. Module

1 is for a child without much spoken language, and Module 4 is for fully verbal adolescents or

adults, with Module 2 and 3 lying somewhere between on the continuum. The cut-off points are

determined by research to identify the degree of autism spectrum disorder via a standardized

assessment of autistic behaviour. Bangor University purchased ADOS boxes.

Design

      This research is based on observational assessment and biomechanical measurement.

      Main Effect: Gait variability. The primary interest was in whether people with autism

presented a gait that was significantly different from the age-matched controls, focusing on timing

parameters. Gait analysis was conducted to ascertain anomalies and inter-subject variability of

timing parameters; Step time, Stride time, Swing time, Stance time and Cadence. These functioned

as the dependent variables of a between-subjects design comparing quasi-independent variables;

HFA subjects with age-matched typically developing controls. Kolomogorov-Smirnov tests and

Levene's tests were conducted for all tests to assess for normality and homogeneity of variance

respectively [Table 19, Appendix W]. If these parametric criteria were satisfied, then independent

measures ANOVA were run, using age-matched subjects as a between-subjects design. In the

instance of non-parametric tests being required, Mann-Whitney U tests were run with effect size.

For the purpose of simplicity, the Coefficient of Variability shall be abbreviated to 'CoV'

throughout. Each individual participant has a CoV value for each measure (i.e. Left Step), and it is

these that are pooled together for analysis purposes [Table 18, Appendix W].

      Each participant had 12 Coefficients of Variation (CoV) scores to represent the variability in

the timing of their Step, Stride, Swing and Stance (Left, Right, plus Left and Right combined).

Mann-Whitney U and Analysis of Variance (ANOVA) tests were run using SPSS (Statistics 20) to

identify any significant differences between the CoV of these walking parameters in HFA

participants and age-matched TD controls. Each participant also had 12 'Average' timing scores for

Step, Stride, Swing and Stance timing (Left, Right, plus Left and Right combined). In addition, Cadence (average time taken for 6 steps) 'Average' and 'CoV' values were analysed.

Secondary and tertiary factors. Secondary and tertiary factors involve severity of autistic symptoms and developmental age. To ensure no distorting relationship between secondary and tertiary factors, a Spearman's rho correlation analysis will be conducted using raw ADOS score and age in months. An additional analysis constituting of Mann-Whitney U comparisons using cohort age data ("12 and below" and "13 and above") will be run with ADOS score as the dependent variable.

Degree of autism. Any relationship between the degree of autism, quantified by the Communication (C) and Reciprocal Social Interaction (RSI) components of ADOS scores [Table 14, Appendix V], and the severity of the individual's 'Autistic gait' as specified by timing anomalies was investigated as a secondary factor. Spearman's rho were planned to seek a relationship between the variability of gait timing and degree of ADOS scores attained. Separate analyses were run to establish any relationships between Communication and CoV values for Step, Stance, Swing and Stride, repeated for RSI and the four CoV values, and a final analysis run for a combined C and RSI score and the four CoV values.

Developmental age. Links between the gait variability scores and the participant's age were also considered as a tertiary factor. A 2 (HFA and TD) X 2 (12 and under/13+) ANOVA was run with the gait variability CoV values functioning as dependent variables. An additional Pearson's correlational test between Stride CoV and age in months was run.

Procedure

Recruitment. Ethics approval was granted by R&D and REC committees (Ysbyty Gwynedd, Betsi-cadawaladr University Health Board). Independent ethics approval was also obtained from Bangor University. The agreed protocol was adhered to throughout the research. Potential participants were selected by Dr Wimpory or clinical psychologists for whom she has an Autism Spectrum Disorder-lead role. Ms Oonagh Eason, Research Nurse Learning Disability,

BCUHB was employed to get in touch with Clinical Psychologist colleagues of Dr Dawn Wimpory, who could identify cases fitting the inclusion criteria. Current treating clinicians (Clinical Psychologist, or General Practitioner if a case was closed to Psychology) of the potential participants sent out an invitation letter and information pack. Emphasis was placed on this not affecting their right to receive treatments and benefits available to them. If clinicians obtained assent, potential participants could be contacted. Stamped envelopes addressed to the clinician for reply were provided. Verbal assent on phone/in person was also acceptable if that was easier for the clinicians. Alternatively the participants could simply send a consent form directly to the Research Team. Ms Oonagh Eason or Bethan Griffiths (PA to Dr Dawn Wimpory) telephoned anyone who returned forms and an appointment could be arranged.

To recruit typically developing children, we contacted head teachers at local schools (Ysgol Tryfan and Ysgol Garnedd of Bangor, Gwynedd) with a letter introducing the research to them. The typically developing children took an invitation letter and information sheet home from school to their parents. If they were willing to participate, the parents could return a consent form in Stamped Addressed Envelope to then be contacted and given an appointment time.

The gait laboratory. The research took place in the Sports Science Research Laboratory at Bangor University, Normal Site, Holyhead Road, Bangor, Gwynedd. The families were greeted by Katharine Forster and Oonagh Eason and encouraged to settle into the laboratory with refreshments provided. Oonagh Eason dealt with informed consent. The participants were shown where to change into shorts to allow the markers to be placed accurately. Their height and weight were put into the VICON Nexus software to establish their personal profile. Markers were placed on the left and right side for each of the following anatomical locations: Anterior Superior Iliac Spine, Posterior Superior Iliac Spine, Thigh, Knee (Medial and Lateral Epicondyle of the Femur), Tibia, Ankle (Fibula Apex of Lateral Malleolus and Tibia Apex of Medial Malleolus), Heel (Calcaneus) and Toe (Head of Second Metatarsus) [Appendix L].

The participants were required to walk diagonally across the room to allow the cameras to

pick up the maximum amount of data from the 4 metre X 4 metre recording zone. The session in the

Gait Laboratory took approximately an hour and a half including obtaining consent, fitting markers,

ensuring enough examples of their 'typical' walk had been recorded and debriefing. Many families

opted to stay longer to have a chat with Ms Oonagh Eason and to allow the participant to engage in

some recreational activities (led by the participant's enthusiasm) e.g. playing golf with a VICON-

compatible reflective golf ball.

The number of walking trials varied for participants, as some were excluded afterwards if

hesitant or curved [Table 25, Appendix Z]. Approximately seven steps, captured in the centre of the

room, were used for data analysis. Participants were offered to watch videos of themselves as a

robotic stick figure on the VICON screen [Appendix M]. Participant payment and debrief forms

were provided.

Autism Diagnostic Observation Schedule. Katharine Forster received 88% Autism

Diagnostic Observation Schedule (ADOS) reliability approval, awarded November 2012 by

external ADOS assessor Dr Fiona Scott, Chartered Psychologist and former Research Associate at

the Autism Research Centre. Participants who hadn't already undergone an ADOS were assessed at

the gait laboratory by Katharine Forster after the gait data had been collected. This took

approximately one hour and was video-recorded by Kate Shakespeare (Assistant Psychologist,

BCUHB) or Marie Remouit (Research Project Support Officer, BCUHB). Katharine Forster blindly

recoded the video recordings of participants who had previous ADOSes. Afterwards, a Cohen's

Kappa (Landis & Koch, 1977) was conducted to ascertain the inter-raterreliability.

Miscellaneous. GP letters were sent out if the tick box in the consent form was checked to

give permission to inform GPs that their patients had participated in research. The research findings

were sent out to the participants and parents in a letter, thanking them for their contribution

[Appendix I]. All data was anonymised and coded numerically according to agreed ethical

protocol. The NHS code of confidentiality was followed at all times.

Processing VICON data. After gait data had been recorded, each video recording was

labelled; the computerised stick leg videos were marked at points corresponding to the markers

attached to the participant [Appendix M]. Some data was excluded [Table 25, Appendix Z] if

participants started to walk in a curve (this would affect their time in swing phase and step length

times) and if the participant had hesitated or broken into a jog. Following the labelling process, an

'event' was entered for every visible Left foot strike, Right foot strike, Left toe off and Right toe

off.

The VICON system was set to 250Hz i.e. recording frames at a rate of 250 frames per

second or every 4 milliseconds. The resultant biomechanical video of the participant can be moved

forward frame by frame until the event occurs (strike or toe-off). The frame number is displayed at

the base of the screen, thus a precise temporal measurement can be recorded [Appendix N]. Data

was subsequently multiplied by 4 to give an estimate of the milliseconds for each event and the

specific times of each 'Foot strike' and 'Toe off' were obtained [Appendix O].

A spreadsheet [Appendix P] was constructed to calculate the mean, standard deviation and

Coefficient of Variation (CoV) in milliseconds for each stage of the gait cycle for each participant

[Appendix Q, R, S, & T].

1) Step time = One foot striking the floor to the next foot striking the floor.

2) Swing time = One toe off the floor to the same foot striking the floor again

3) Stance time = One foot striking the floor to toe off of same foot.

4) Stride time = One foot striking the floor to the same foot striking the floor again.

Results

Gait Variability

Each participant had 12 Coefficient of Variability (CoV) timing values, one for every

phase of the gait cycle: Step, Stance, Swing and Stride (Left, Right and Left & Right

combined), calculated from all examples within their multiple trials, average $n = 37.15$ [Table

23, Appendix Z]. Normality and homogeneity of variance assumptions were not satisfied, so

Mann-Whitney U tests were employed [Table 19, Appendix W]. A one-tailed hypothesis

predicted more variability in the timing of gait in High Functioning Autism (HFA) group than

the Typically Developing (TD) age-matched controls group.
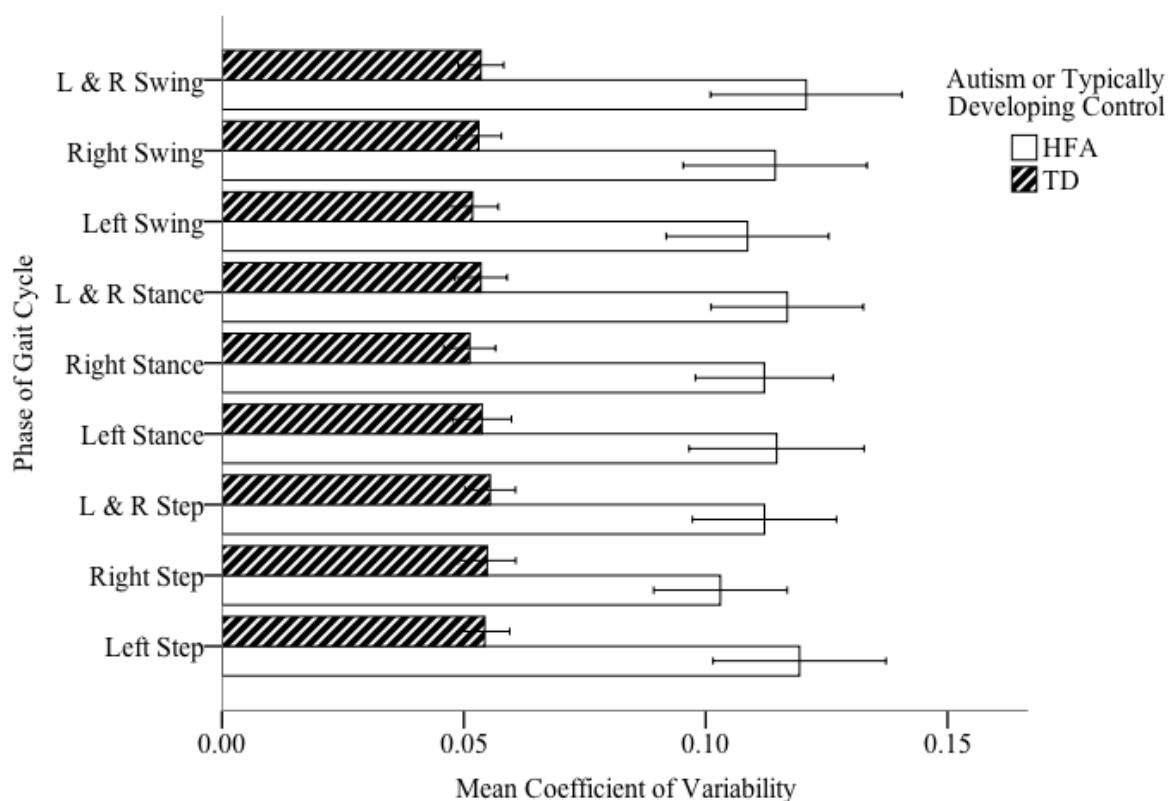
Figure 1. Variation in timing of Step, Stance, Swing and Stride, comparing $n = 16$ HFA

participants to $n = 16$ TD controls. Error bars +/- 1SEM.

Variability of step timing. The CoV of timing for one foot strike to next foot strike

[Appendix Q], was assessed using a Mann-Whitney U test, comparing the $n = 16$ HFA

participants with the $n = 16$ TD controls. The results indicate a significant difference for Left

step between HFA (*Mdn* = 0.0837) and TD (*Mdn* = 0.0522) participant groups, *U* =37, *z* =

-3.43, *p* < .001, *r* = -.61.   Right Step timing also shows a significant difference, HFA (*Mdn* =

0.0811) and TD (*Mdn* = 0.0528) *U* = 35, *z* = -3.51, *p* < .001, *r* = -.62. Left and Right Step

combined shows an overall significant difference between HFA (*Mdn* = 0.0879) and TD

(*Mdn* = 0.0527) participants, *U* = 34, *z* = -3.54, *p* < .001, *r* = -.63. The participants with

autism executed a more variable Step time than their TD controls [Table 18, Appendix W].

     Variability of swing timing. The data for the CoV values for Swing timing (toe-off

to foot strike [Appendix R]) revealed significant results using a Mann-Whitney U test to

compare the ranks for the *n* = 16 HFA participants with the *n* = 16 TD controls, for Left

Swing, HFA (*Mdn* = 0.0895) and TD (*Mdn* = 0.0441), *U* = 35, *z* = -3.51, *p* < .001, *r* = -. 62;

Right Swing, HFA (*Mdn* = 0.0896) and TD (*Mdn* = 0.0483) *U* = 36, *z* = -3.47, *p* < .001, *r* = -.

61; and for the combination of Left and Right Swing data together, HFA (*Mdn* = 0.0909) and

TD (*Mdn* = 0.0495), *U* = 35.5, *z* = -3.49, *p* < .001, *r* = -. 62. Swing timing is more variable

in participants with autism than the control participants [Table 18, Appendix W].

     Variability of stance timing. Stance timing variability was assessed using CoV

values of Foot Strike to Toe Off [Appendix S]; a Mann-Whitney U test compared the ranks

for the *n* = 16 HFA participants to the *n* = 16 TD controls. The results indicate a significant

difference between the two groups for Left Stance, HFA (*Mdn* = 0.0820) and TD (*Mdn* =

0.0451), *U* = 37, *z* = -3.43, *p* < .001, *r* = -. 61; Right Stance, HFA (*Mdn* = 0.0966) and TD

(*Mdn* = 0.0474), *U* = 20, *z* = -4.07, *p* < .001, *r* = -. 72; and combined Left and Right Stance,

HFA (*Mdn* = 0.0941) and TD (*Mdn* = 0.0479), *U* = 21, *z* = -4.03, *p* < .001, *r* = -. 72.

Individuals with autism demonstrated a more variable Stance phase than age-matched control

subjects [Table 18, Appendix W].

     Variability of stride timing. Stride timing was assessed using CoVs of one Foot

Strike to same Foot Strike [Appendix T] and analysed using a Mann-Whitney U test to

compare the ranks for the *n* = 16 HFA participants compared to the *n* = 16 TD controls, indicating a significant difference for Left Stride, HFA (*Mdn* = 0.0661) and TD (*Mdn* = 0.0232), $U = 29$, $z = -3.73$, $p < .001$, $r = -. 66$; Right Stride, HFA (*Mdn* = 0.0826) and TD (*Mdn* = 0.0366), $U = 20$, $z = -4.07$, $p < .001$, $r = -. 72$; and Left and Right Stride combined, HFA (*Mdn* = 0.0669) and TD (*Mdn* = 0.0373), $U = 21$, $z = -4.03$, $p < .001$, $r = -. 71$. The overall gait timing as defined by Stride was more variable in the participants with autism than the control participants [Table 18, Appendix W].

Average Timing

Each participant also had 12 Mean timing values, one for every phase of the gait cycle: Step, Stance, Swing and Stride (Left, Right and Left & Right combined), calculated from all examples within their multiple trials, average *n* = 37.15 [Table 23, Appendix Z]. In contrast to variability, it was predicted that there would be no difference between the HFA and TD group in their average timing for the four elements of the gait cycle. Normality and homogeneity of variance assumptions were satisfied in all four tests of average timing, so ANOVA tests were employed [Table 19, Appendix W].
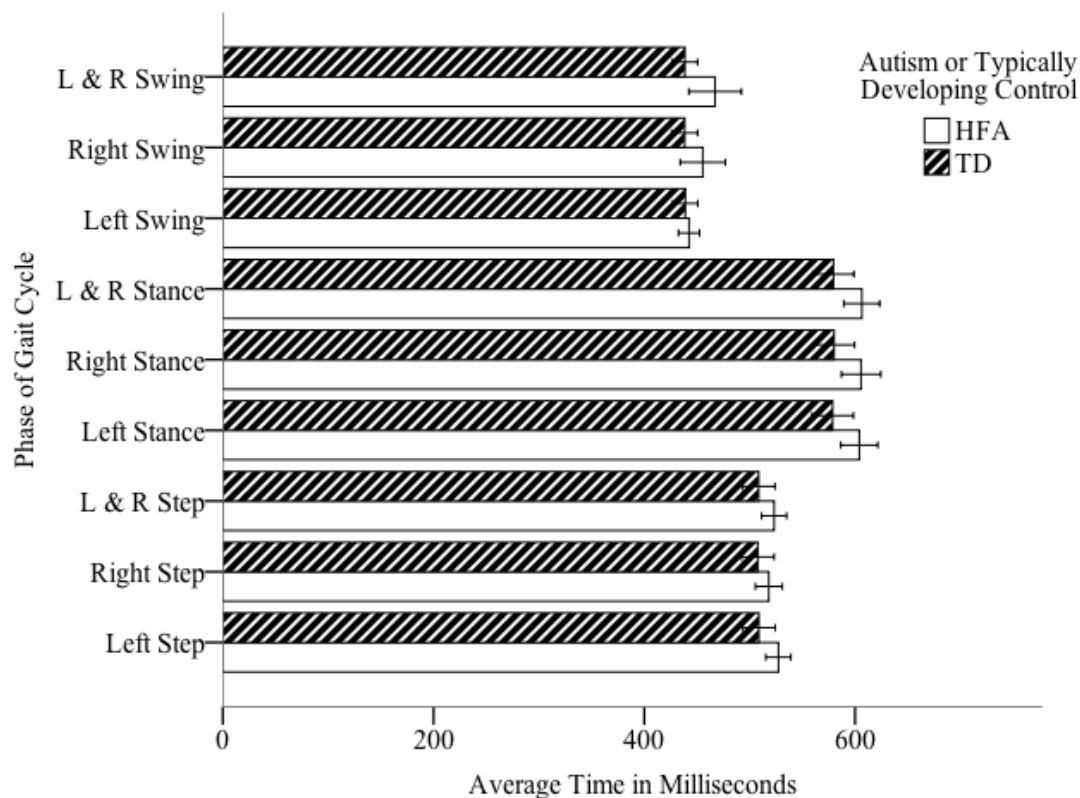
Figure 2. Average time taken in milliseconds for each stage of the gait cycle, comparing all

HFA participants (*n* = 16) with TD participants (*n* = 16). Error bars set to +/- 1SEM.

Average step timing. Data for average timing of one foot strike to next foot strike

[Appendix Q] indicated no significant difference in *n* = 16 HFA participants compared to *n* =

16 TD controls for Left Step, $F(1, 30) = 0.88$, $p < .36$, $\eta^2 = 0.03$, Right Step, $F(1, 30) = 0.27$,

$p < .61$, $\eta^2 = 0.009$ and both Left and Right Steps combined, $F(1, 30) = 0.57$, $p < .46$, $\eta^2 =$

0.02 [Table 18, Appendix W].

Average swing timing. Data for average time of toe-off to foot strike [Appendix R]

revealed no significant difference in average timing between *n* = 16 HFA participants

compared to *n* = 16 TD controls for Left Swing, $F(1, 30) = 0.05$, $p = .82$, $\eta^2 = 0.002$, Right

Swing, $F(1, 30) = 0.48$, $p = .49$, $\eta^2 = 0.016$ and both Left and Right Swing combined, $F(1,$

$30) = 1.06$, $p = .31$, $\eta^2 = 0.034$ [Table 18, Appendix W].

Average stance timing. The data for average time of foot strike to toe off [Appendix

S] demonstrated no significant difference between *n* = 16 HFA participants compared to *n* =

16 TD controls, for Left Stance $F(1, 30) = 0.91$, $p = .35$, $\eta^2 = 0.029$, Right Stance, $F(1, 30) =$

0.90, $p = .35$, $\eta^2 = 0.029$ and both Left and Right Stance combined, $F(1, 30) = 1.08$, $p = .31$,

$\eta^2 = 0.035$ [Table 18, Appendix W].

Average stride timing. The data for average time of one foot strike to same foot

strike [Appendix T] revealed no significant difference in average timing between the $n = 16$

HFA participants and the $n = 16$ TD controls for Left Stride $F(1, 30) = 0.86$, $p = .36$, $\eta^2 =$

0.028, Right Stride, $F(1, 30) = 0.91$, $p = .35$, $\eta^2 = 0.029$ and both Left and Right Stride

combined, $F(1, 30) = 0.88$, $p = .36$, $\eta^2 = 0.029$ [Table 18, Appendix W].

Cadence

To confirm that cadence, defined in this instance as the time taken to execute 6

consecutive Foot Strikes, was not affected by autism, tests were run to assess for differences

between groups for Mean Cadence values and CoV Cadence values [Appendix U]. A one-

way analysis of variance confirmed no significant difference between the average Cadence of

the $n = 16$ HFA participants and the $n = 16$ TD controls $F(1, 30) = 0.93$, $p = .34$, $\eta^2 = 0.03$. A

between-subjects Mann-Whitney U test was performed using the CoV value for each

participant to walk 6 steps, comparing $n = 16$ HFA participants and $n = 16$ TD control

participants. There was a significant difference between groups, HFA (*Mdn* = 0.0672) and

TD (*Mdn* = 0.0319), $U = 47$, $z = -3.05$, $p = .002$, $r = -.54$. The Cadence CoV for the HFA

participants was higher on average than for the TD control participants; the time taken to

execute 6 steps was more variable in HFA than TD [Table 18, Appendix W].
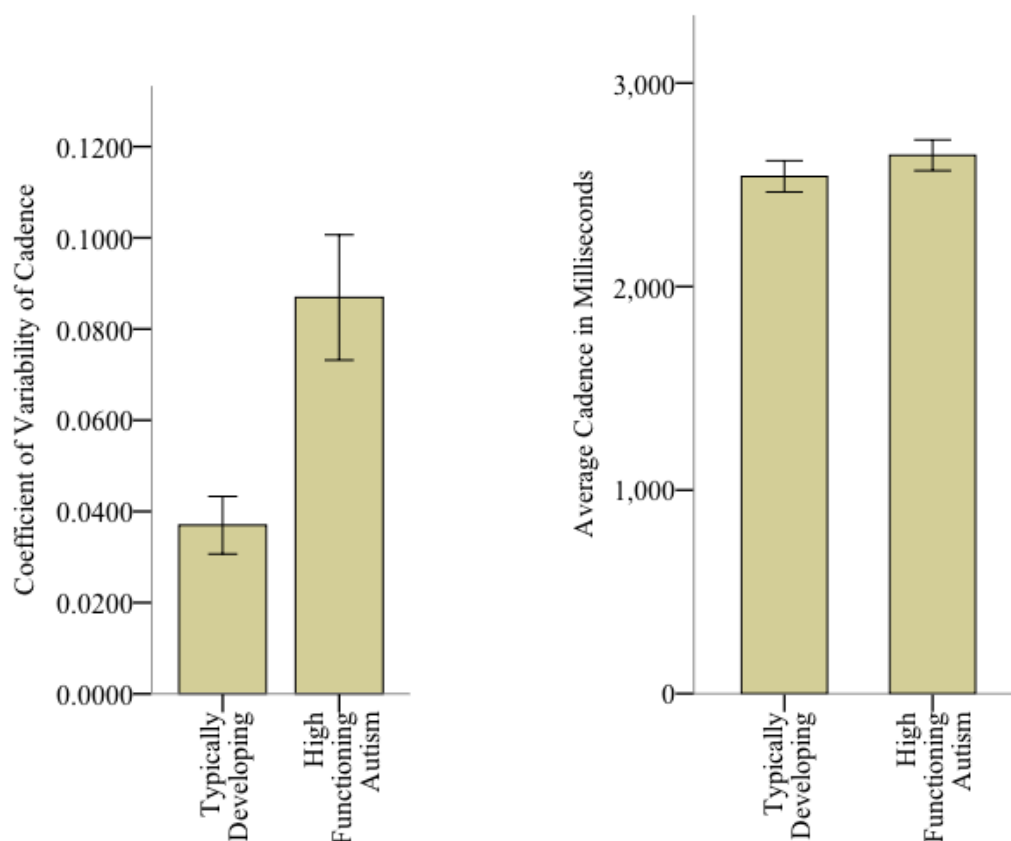
Figure 3. Average and Coefficient of Variation for Cadence: time taken in milliseconds for 6 Foot Strikes. Error bars set to +/- 1SEM.

Separating Secondary and Tertiary Factors

Tests were run to ensure no correlation between raw Autism Diagnostic Observation Schedule (ADOS) scores and chronological age in months. The data sets satisfied homogeneity of variance and normality assumptions [Table 19, Appendix W]. A Pearson's test revealed no relationship between Age and Communication score, $r = -.30$, $n = 14$, $p = .31$, Age and Reciprocal Social Interaction score $r = -.36$, $n = 14$, $p = .21$ or Age and the combined overall ADOS score, $r = -.06$, $n = 14$, $p = .84$. Distortion of gait factors could have arisen if the distribution of ADOS scorers was not equal throughout the age cohorts. One way analysis of variance was run with ordinal categories of Age (12 and below/13+), using the combined Communication and Reciprocal Social Interaction ADOS score as the dependent

variable, $n = 14$, revealing no significant difference in ADOS scores between the HFA

participants aged 12 and below, $n = 9$ and those aged 13 and above, $n = 5$, $F(1, 12) = 0.13$, p

$= .73$, $\eta^2 = 0.01$ [Table 17, Appendix V; Table 19, Appendix W; Table 21, Appendix Y]. Any

subsequent significant effect found between age and gait variability or ADOS scores and gait

variability reflect age or ADOS scores in isolation.
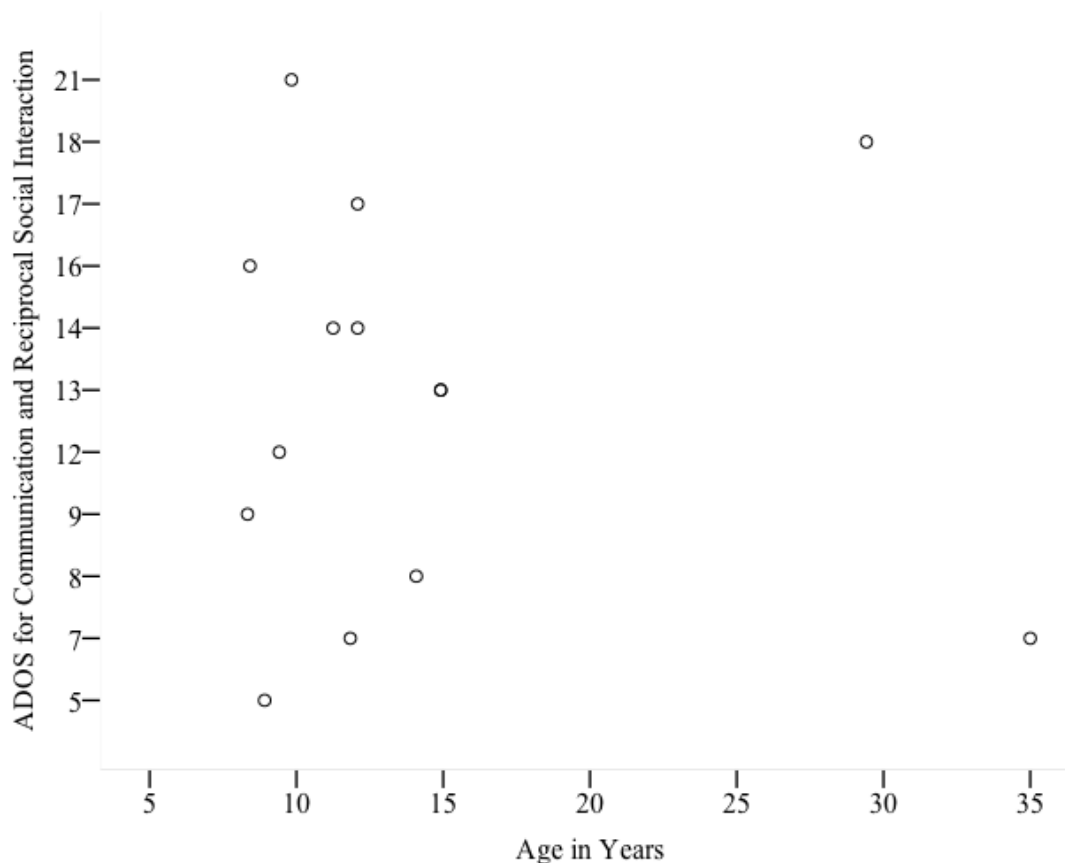
Figure 4. No correlation found between severity of autistic symptoms and age in HFA

participants, $n = 14$.

Autism Diagnostic Observation Schedule

Analyses included testing for inter-rater reliability, plus any relationship between autistic

symptoms as measured by the Autism Diagnostic Observation Schedule (ADOS) and

variability of gait scores, measured by Coefficient of Variation.

      Inter-rater reliability. Tests were run to ensure that various Clinicians' ADOS

scores did not differ significantly from Katharine Forster's scores (reliability approved

externally). In the majority of cases (8 out of 10) previously conducted ADOS assessment

videotapes were obtainable and functioning ($n = 8$), so K Forster blindly scored these and

subsequently compared it to the Clinicians' scores. It is important to ascertain for the two

tapes that weren't recoded how likely it is that the Clinicians' ratings are similar to the

reliable standard. The inter-rater reliability for K Forster and the varying Clinicians for the

Communication component of the ADOS score was assessed using Cohen's Kappa = .51 ($p =$

.002), 95% CI (0.203, 0.817). For the Reciprocal Social Interaction (RSI) component of the

ADOS score, Kappa = .439, ($p = .00$), 95% CI (0.102, 0.776). However, the inter-rater

reliability for both of these subcategories of ADOS scores is classified as only 'Moderate

Agreement' (Landis & Koch, 1977).

It was thought that due to the wide range of possible scores (up to 12 for RSI)

Cohen's Kappa might not have been a suitable test, because the similarity of the other rater's

score wasn't taken into account. For example, one person could rate 11 and the other 12, and

this would be good similarity, but wouldn't count as a congruent score for Cohen's Kappa. A

further test, Spearman's rho, was run to ensure a high correlation between K Forster and

Clinicians' scoring ($n = 9$), with a positive significant result for Communication score, $r =$

.86, $p = .007$; Reciprocal Social Interaction score, $r = .83$, $p = .010$; and combined ADOS

score, $r = .96$, $p < .001$. The value of the coefficient of determination, $r^2 = 0.92$ for the

combined ADOS score, implying the two ADOS tapes not attainable for re-coding were

appropriately coded by the Clinician at a likelihood of 92% [Tables 14 & 15, Appendix V].
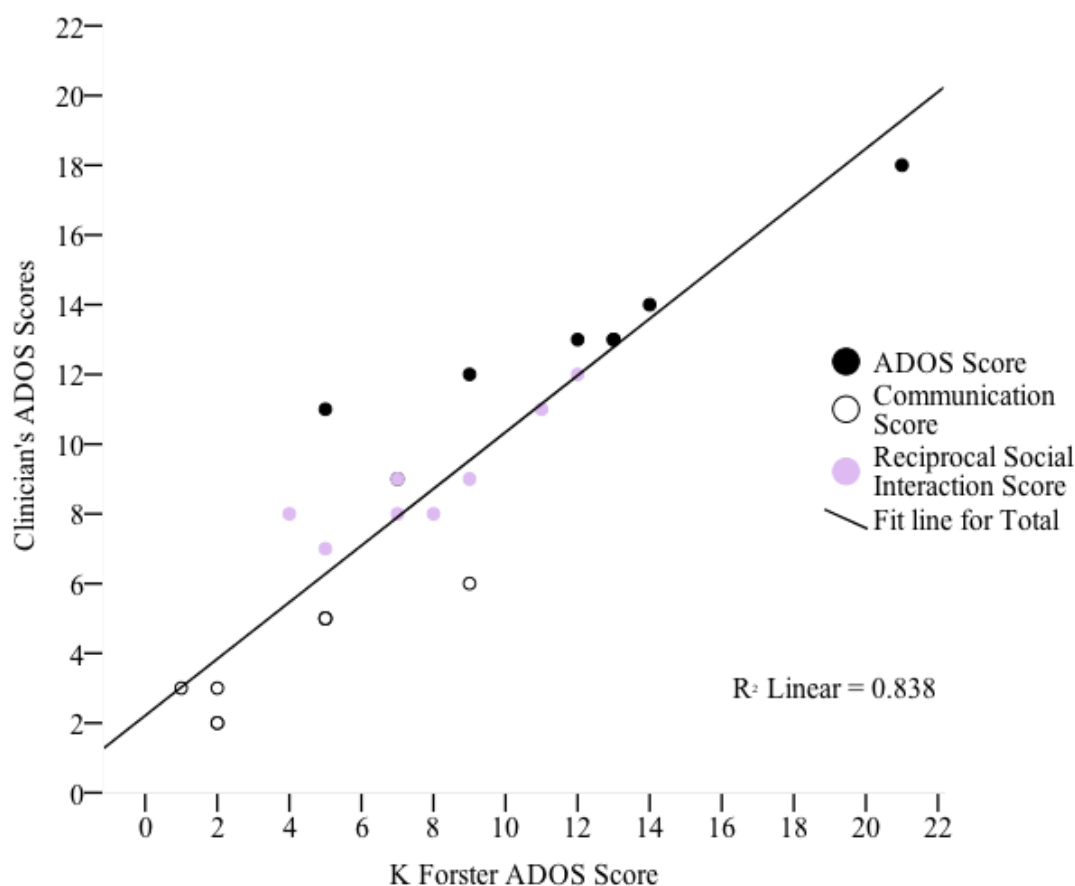
Figure 5. ADOS scores by Katharine Forster and various Clinicians for *n* = 8 HFA

participants. Some participants' scores duplicated, hence not 8 markers per ADOS score

category.

   ADOS and gait variability. A correlative analysis was run using Spearman's rho to

establish any relationship between CoV of Step, Swing, Stance and Stride [Q, R, S, & T], and

the HFA participant's ADOS scores [Table 14, Appendix V; Table 18, Appendix W].   For

the Communication component of ADOS, a Spearman's rho correlation for the data revealed

that Communication ADOS score and gait variability were not significantly related for Step

time, *r* = .11, *n* = 14, *p* = .72; Stance time, *r* = .05, *n* = 14, *p* = .87; Swing time, *r* = .21, *n* =

14, *p* = .47; or Stride time, *r* = .06, *n* = 14, *p* = .84. For the Reciprocal Social Interaction

component of ADOS a Spearman's rho correlation for the data revealed that gait variability

was not significantly related for Step time, *r* = .15, *n* = 14, *p* = .61; Stance time, *r* = .39, *n* =

14, $p = .16$; Swing time, $r = .34$, $n = 14$, $p = .23$; or Stride time, $r = .30$, $n = 14$, $p = .30$. No correlation was found when combining the Communication and Reciprocal Social Interaction scores for Step time, $r = .24$, $n = 14$, $p = .41$; Stance time, $r = .38$, $n = 14$, $p = .18$; Swing time, $r = .43$, $n = 14$, $p = .12$; or Stride time, $r = .31$, $n = 14$, $p = .28$.
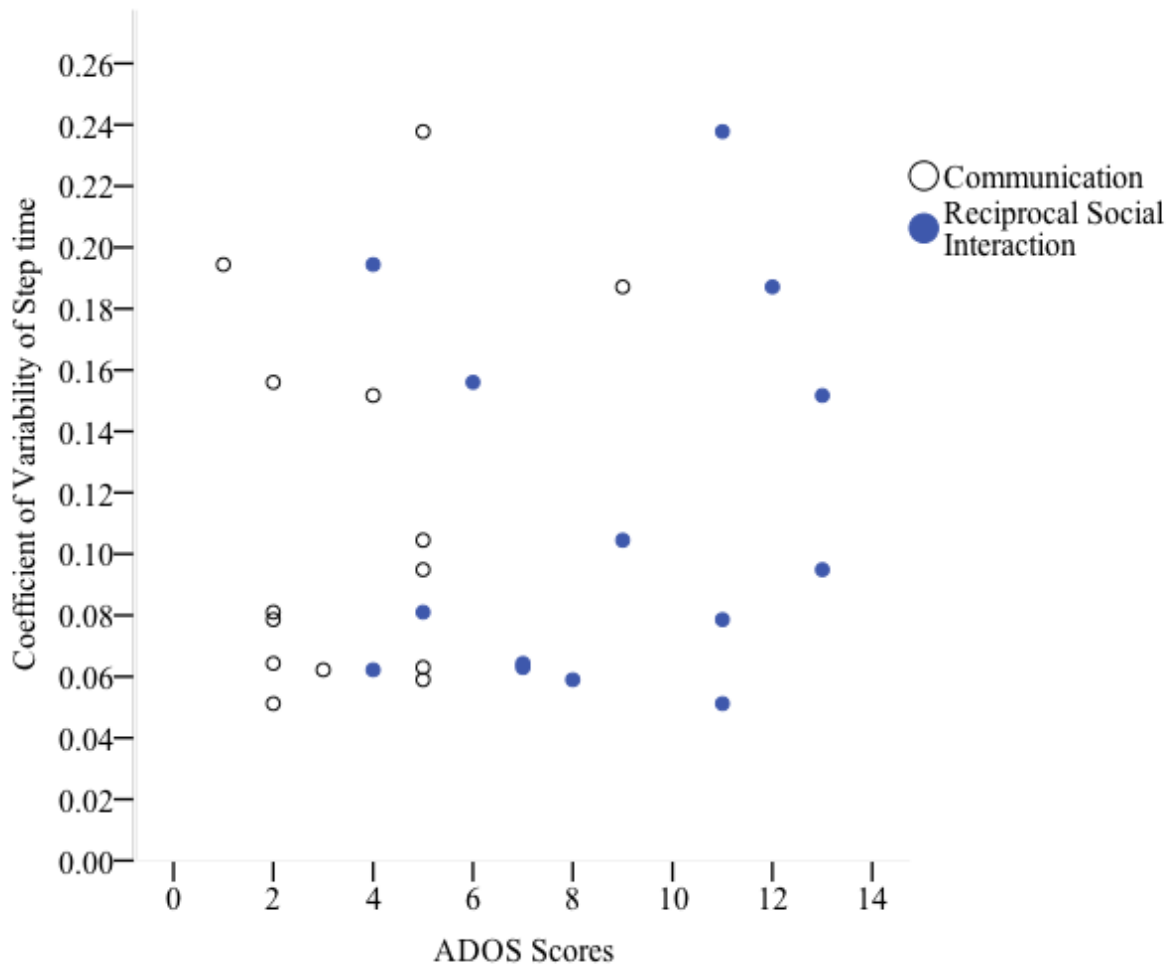
Figure 6. No correlation found between ADOS scores and CoV Step timing in HFA participants, $n = 14$.

For the combined score of Communication plus Reciprocal Social Interaction, a Mann-Whitney U test analysing the CoV of Stride (Stride is representative of gait cycle in general) timing in all participants with HFA, $n= 14$ and their ordinal category of ADOS score was run, with no significant difference found between ADOS score cohorts, 'ADOS Score <12' ($n = 6$, $Mdn = 0.0614$) and 'ADOS Score >13' ($n = 8$, $Mdn = 0.0730$), $U = 17$, $z = -.90$,

p = .41, *r* = -. 24. There was no discernible relationship to be found between the severity of autistic symptoms as scored by ADOS and the variability of gait [Table 17, Appendix V].

Degree of autism and gait variability – another approach. Despite no significant relationship between ADOS scores and gait variability, it was noted that the level of variability in the HFA group lay on a wide spectrum. The criteria were set so that those classified as 'Low variability' fit into the same range as TD participants. A consensus could not be reached on the reasons for the gait timing disparity between the participants, following consultation by Dr Dawn Wimpory with the various clinical professionals involved in the individuals' diagnosis. An additional categorisation was set for Step using the same principles, to allow for analysis of any lateralisation effects between Left and Right step.

Table 1

Participants with High Functioning Autism and Their Allocated Stride and Step Timing Variability Cohorts

Cohort	Range of CoV Values for Stride, Left and Right Leg Combined		Participant ID for Stride cohort	Range of CoV Values for Step, Left and Right Leg Combined.		Participant ID for Step cohort
	Lowest	Highest		Lowest	Highest	
TD Control	.0210	.0498	All control subjects (apart from one*)	.0323	.0648	All control subjects (apart from one*)
HFA low variability	.0367	.0470	35, 36 and 29	.0512	.0643	18, 35, 36, 29, 15 and 37

HFA mid-variability	.0523	.0796	17, 3, 37, 20, 18, 28 and 15	.0786	.0949	17, 20 and 3
HFA high variability	.1179	.2229	4, 2, 14, 13, 11, 12 and one anomalous TD Control	.1045	.2378	11, 12, 13, 28, 14, 2 and 4 and one anomalous TD control

Note. *One typically developing (TD) participant had CoV values fitting the cohort 'HFA with high variability'.

An artificial dichotomy was created using these cohorts, with all HFA participants in the 'Low Variability' or 'Moderate Variability' cohort grouped together, and the HFA participants with 'High Variability' grouped separately. Normality assumptions were not satisfied for all tests [Table 19, Appendix W]. Mann Whitney U tests for differences between 'Low/Moderate Variability', $n = 10$ HFA participants and $n = 10$ TD controls, revealed a significant difference in Left Stride, HFA (*Mdn* =0.0542) and TD (*Mdn* = 0.0379), $U = 10$, $z = -3.02$, $p = .002$, $r = -.68$; Right Stride, HFA (*Mdn* =0.0534) and TD (*Mdn* = 0.0366), $U = 6$, $z = -3.33$, $p = .000$, $r = -.74$; Left and Right Stride combined, HFA (*Mdn* =0.0543) and TD (*Mdn* = 0.0356), $U = 5$, $z = -3.40$, $p = .000$, $r = -.76$. The $n = 6$ HFA participants in the unusually 'High Variability' cohort haven't distorted the overall results unrepresentatively with regard to differences between the timing of HFA and TD, because there is still a highly significant difference between groups for the 'Low/Moderate Variability' cohort [Table 11, Appendix T; Table 18, Appendix W].

Similarly, the 'Low/Moderate Variability' cohort for Step timing demonstrated significant differences between $n = 9$ HFA and $n = 9$ TD for Left Step, HFA (*Mdn* =0.0605)

and TD (*Mdn* = 0.0524), *U* = 12, *z* = -2.52, *p* = .01, *r* = -. 59; Right Step, HFA (*Mdn*

=0.0648) and TD (*Mdn* = 0.0522), *U* = 16, *z* = -2.16, *p* = .03, *r* = -. 51; and for Left and Right

Step combined, HFA (*Mdn* =0.0630) and TD (*Mdn* = 0.0523), *U* = 10, *z* = -2.70, *p* = .006, *r* =

-. 63. The participants with lower variability still demonstrate significantly higher CoV

values than their age-matched control participant, confirming that the few 'High Variability'

HFA participants haven't unduly distorted the overall conclusions [Appendix Q; Table 18,

Appendix W].
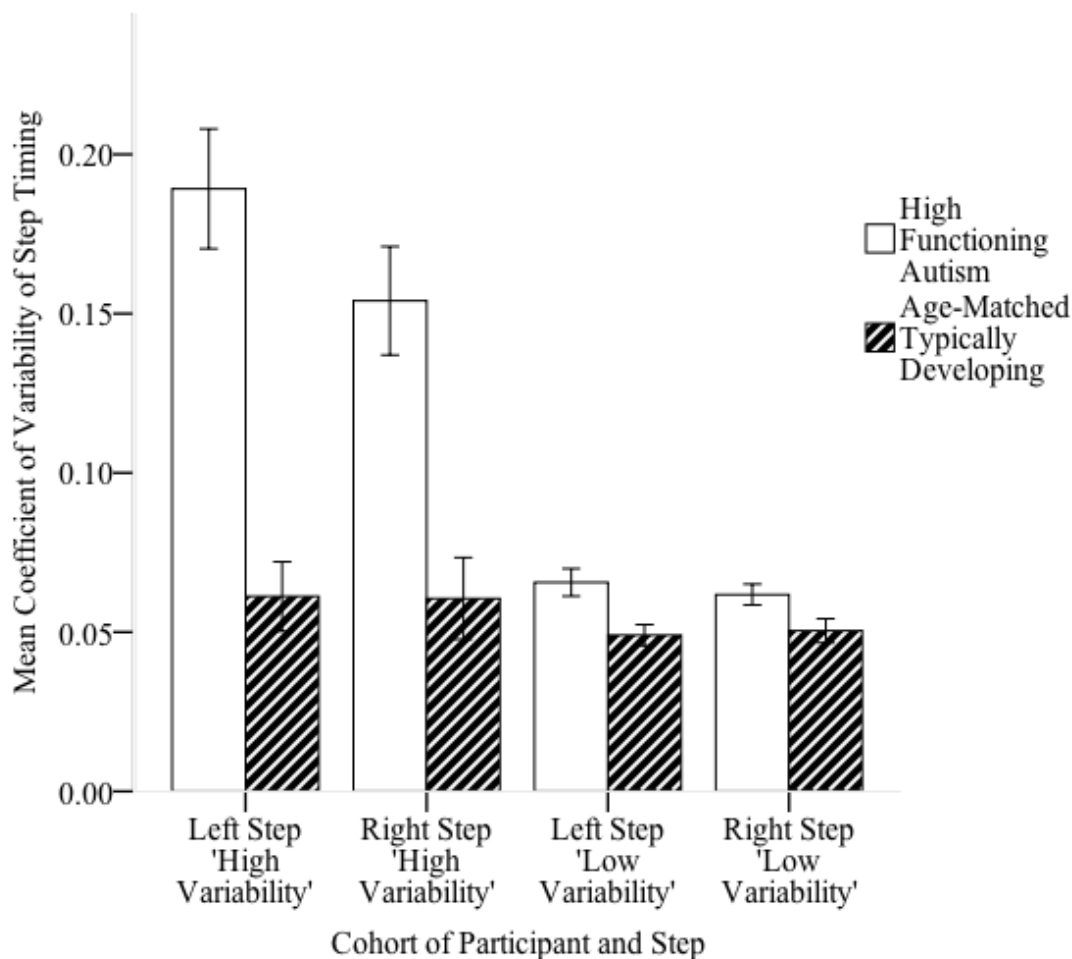
Figure 7. Significant differences on variability of Step timing between HFA and TD controls

remains for both the Low/Moderate Variability and the High Variability cohorts.   'Low

Variability' encompasses 'Low/Moderate Variability.' Error bars set to +/- 1SEM.

Extraneous Diagnostic Factors – Asperger's Or Autism.

The intention was to examine any extraneous factors that could influence gait variability for reasons other than High Functioning Autism. When initially approached, clinicians were requested to identify High Functioning clients, but subsequent diagnostic information was sought from clinical files and Clinicians were consulted for their opinion to ensure any participants with suspected Learning Disabilities (LD) were removed from a version of the data.

Following data collection of Clinician's diagnosis from clinical files [Table 16, Appendix V] the dichotomy of Asperger's ($n = 6$) or Autism ($n = 10$) was used to assess any differences in gait according to diagnosis [Table 6 & 17, Appendix V]. The data satisfied homogeneity of variance and normality assumptions [Table 19, Appendix W]. One-way ANOVA revealed no difference between diagnosis for Stance $F(1,14) = 0.13$, $p = .73$, $\eta^2 = 0.00$, Swing $F(1,14) = 0.07$, $p = .79$, $\eta^2 = 0.009$, Stride $F(1,14) = 0.04$, $p = .84$, $\eta^2 = 0.00$, Cadence $F(1,14) = 0.04$, $p = .85$, $\eta^2 = 0.00$.

Age

An age dichotomy was constructed consisting of one group aged '12 years and below', $n = 18$, and the other '13 years and above', $n = 14$, for the pooled data of both HFA and TD participants. Stride was the selected measure as it encompasses the other phases of the gait cycle (Stance, Step and Swing). The two sets of data divided by age satisfied normality assumptions and homogeneity of variance criteria [Table 19, Appendix W]. Analyses were run comparing these two age groups to see if one is more variable in Stride timing than the other.

A two-way ANOVA with factors of age ('12 and under,' or '13 and above') and diagnosis (HFA or TD) showed no interaction of Stride CoV timing, $F(1,32) = .377$, $p = .54$, $\eta^2 = 0.02$ [Table 21, Appendix Y]. Selecting HFA participants only, a one-way ANOVA

showed no difference between age cohorts for the Stride variability scores (CoV), $F(1,15) =$ 2.50, $p = .14$, $\eta^2 = 0.15$; Step variability scores $F(1,15) = 2.74$, $p = .12$, $\eta^2 = 0.17$, Stance variability scores, $F(1,15) = 0.35$, $p = .56$, $\eta^2 = 0.02$ or Swing variability scores $F(1,15) =$ 0.03, $p = .87$, $\eta^2 = 0.00$. To ensure the cohort approach wasn't obscuring age effects, a correlational method using Pearson's was employed, revealing no correlation between Stride CoV and age in months $r = - .38$, $n = 16$, $p = .15$. Gait variability is not attributable to developmental age, nor does age affect the gait of people with autism to a greater degree than the typically developing controls.
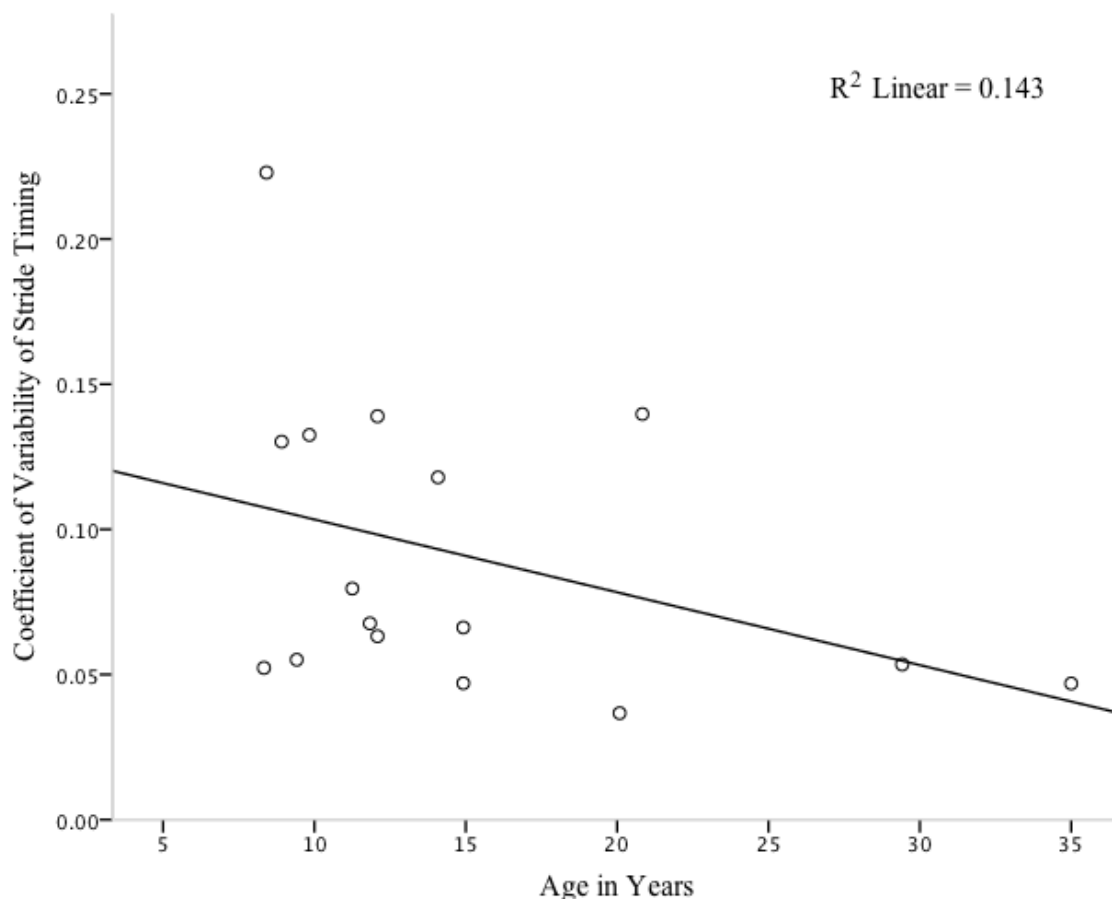
Figure 8. No significant correlation found between variability of gait timing, as measured by Coefficient of Variation of Stride timing, and developmental age in years, in participants with High Functioning Autism, $n = 16$.

Lateralisation of Gait Variability

Comparisons of the timing of the left steps versus the right steps were conducted to assess any lateralisation of gait variability [Appendix Q] in High Functioning Autism. Left versus right timing data was compared for CoV scores for Step, Stance and Swing using Wilcoxon's Signed Ranks [Table 19, Appendix W]. There were no lateralisation effects for CoV of Stance, Left (*Mdn* = 0.0820) versus Right (*Mdn* = 0.0966), *Z* = 0.00, *p* = 1.00, *r* = .00; or Swing, Left (*Mdn* = 0.0895) versus Right (*Mdn* = 0.0896), *Z* = -0.72, *p* = .469, *r* = -.181. However, there was a lateralisation effect for Step timing, with a higher CoV in the Left Step (*Mdn* = 0.0837) than the Right Step (*Mdn* = 0.0811) for participants with High Functioning Autism only, *Z* = -2.17, *p* = .030, *r* = -.543. Typically Developing control participants' data for CoV of Step timing was analysed separately, finding no difference between Left (*Mdn* = 0.0529) and Right (*Mdn* = 0.05187) Step, *Z* = -0.36, *p* = .717, *r* = -.095. The timing of the Left Step is more variable than the Right Step in the participants with autism only [Table 18, Appendix W].

Asperger's and lateralisation. A two (Asperger's or Autism) X two (Left or Right Step CoV) ANOVA revealed no differences between Asperger's and autism diagnosis groups for coefficient of variability of Step $F(1,31) = 0.048$, $p = .83$, $\eta^2 = 0.00$, between Left and Right $F(1,31) = 0.504$, $p = .48$, $\eta^2 = 0.00$, or the interaction between the two, $F(1,31) = 0.015$, $p = .90$, $\eta^2 = 0.003$.

However, using Wilcoxon Signed Ranks to test for differences in rank order between Left Step CoV and Right Step CoV for diagnostic cohorts, *n* = 6 Asperger's and *n* = 10 Autism; only the Asperger's group pertained to the aforementioned lateralisation effect. There was no left/right leg timing disparity for the Autism only cohort, Left (*Mdn* = 0.0732) versus Right Step (*Mdn* = 0.0811), *Z* = -.56, *p* = .575, *r* = -.18. The Asperger's Disorder cohort showed a lateralisation effect, Left (*Mdn* = 0.1000) versus Right Step (*Mdn* = 0.0803), *Z* = -2.20, *p* = .028, *r* = -.90. In all *n* = 6 participants with Asperger's, the Left Step timing

was higher than the Right Step timing variability (CoV) [Table 17, Appendix V].
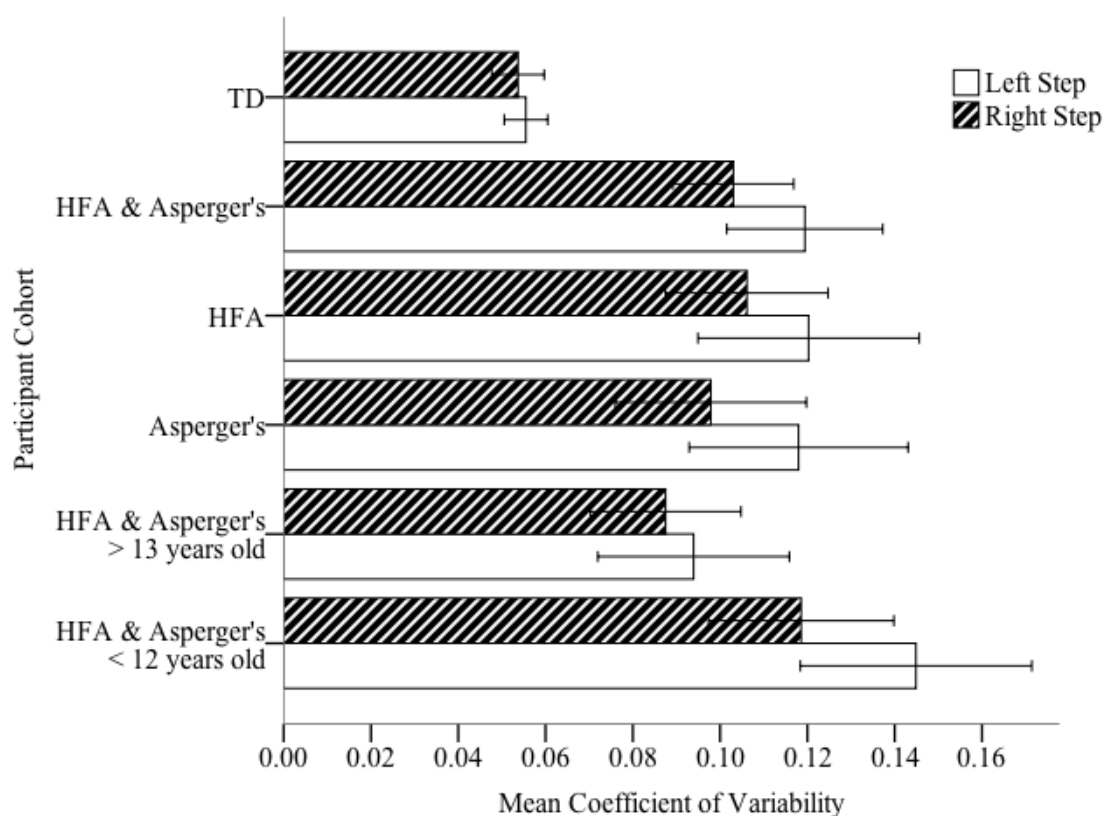
Figure 9. Variation in timing of Left and Right Step in different participant cohorts.

Lateralisation and age. No difference was found between age cohorts ('12 and

under', or '13 and above') for Step time, Stance time or Swing time, as stated previously.

However, in HFA participants there was a trend towards a correlation between age in months

for Left Step CoV, $r = -.48$, $n = 16$, $p = .06$, but not Right Step CoV, $r = -.28$, $n = 16$, $p =$

.30.

When looking at the youngest HFA cohort (age 12 and below, $n = 8$) in isolation

using Wilcoxon's Signed Ranks, there was a significant difference between Left and Right

Step, $Z = -2.24$, $p = .03$, $r = -.79$, with Left Step variability (*Mdn* = 0.1338) being higher than

Right Step variability (*Mdn* = 0.1051).   However the older cohort, $n = 8$, did not show a Left

(*Mdn* = 0.0646) versus Right (*Mdn* = 0.0711) disparity, $Z = -2.24$, $p = .03$, $r = -.79$ [Table 17,

Appendix V]. This age and lateralisation effect is only present in the HFA group; the young

TD cohort in isolation demonstrate no lateral disparity, $Z = -0.42$, $p = .67$, $r = -.79$, between

Left (0.0542) and Right Step (*Mdn* = 0.0592).

Given the finding that only the lowest age cohort of all participants with autism

(Asperger's and HFA combined) showed a lateralisation effect, it is important to establish

that the group with Asperger's don't merely consist of a disproportionate number of the

young cohort. A one-way ANOVA revealed no difference in chronological age in months

between the participants with Asperger's and the participants with High Functioning Autism,

$F(1,15) = 0.37$, $p = .55$, $\eta^2 = 0.03$. Despite only some cohorts showing significant

lateralisation, there appears to be an overall trend for the Left Step to be more variable than

the Right Step (*Figure 9*).

ADOS: Left versus right step. A correlative analysis was conducted to determine

any relationship between ADOS scores and Left Step CoV or Right Step CoV separately.

Using Pearson's correlation coefficient, no significant relationship was found for Left Step in

Communication score, $r = .02$, $n = 14$, $p = .95$; Reciprocal Social Interaction, $r = -.02$, $n =$

14, $p = .94$; or combined ADOS score, $r = .03$, $n = 14$, $p = .92$. No significant correlation

was found for Right Step either, in Communication score, $r = .31$, $n = 14$, $p = .29$; Reciprocal

Social Interaction, $r = .22$, $n = 14$, $p = .46$; or combined ADOS score, $r = .31$, $n = 14$, $p = .29$.

Using the ADOS score dichotomy, a one-way analysis of variance revealed no

significant difference between those who scored 12 and below ($n = 6$) or 13 and above ($n = 8$)

for Left Step, $F(1, 12) = 0.001$, $p = .97$, $\eta^2 = 0.00$; or Right Step, $F(1, 12) = 0.33$, $p = .58$, $\eta^2 =$

0.23. The Lateralisation effects detailed earlier were not also a function of ADOS scores

[Appendix Q; Table 17, Appendix V; Table 18 & 19, Appendix W].

Anomalous HFA participant. One participant, 'Number 20', presented an unusual

habit. During gait trials his gait appeared symmetric, though quite slow and deliberate.

When the mother said, "Do your proper walk now, like when we go for a walk together" an

entirely different gait was instigated. The second was the version used throughout the

analysis as it appeared more genuinely representative of this individual's gait. Mann-

Whitney U tests assessed differences between average values of the two walking styles,

finding a significant difference between first (*Mdn* = 518.40 milliseconds) and second walk

(*Mdn* = 472.68 milliseconds) for Left average Swing time, $U = 1$, $z = -2.46$, $p = .012$, $r = -.$

74. A significant difference was also apparent between first (*Mdn* = 509.60 milliseconds)

and second walk (*Mdn* = 433.32 milliseconds) for Right average Swing time, $U = 1$, $z =$

-2.46, $p = .012$, $r = -.$ 74. The first walk implemented longer Swing times on average.

Lateralisation was investigated, finding that for the first walking style, there was no

difference between the average time for Left Swing (*Mdn* = 518.40 milliseconds) and Right

Swing (*Mdn* = 509.60 milliseconds), $U = 14$, $z = -1.34$, $p = .21$, $r = -.$ 36. However, for the

second more 'genuine' walking style, there was a significant difference between Left (*Mdn* =

472.68 milliseconds) and Right (*Mdn* = 433.32 milliseconds) average swing time, $U = 0$, $z = -$

2.31, $p = .029$, $r = -.$ 82, with the Left Swing time average exceeding the Right Swing time

average [Table 23 & 24, Appendix Y]. This enhances the lateralisation effects detailed

earlier, with some substantiating Left leg anomalies relating to timing in one individual.

Body Mass Index

An analysis was run excluding data from the participants who scored anything but a

'healthy weight' using the NHS BMI generator,

http://www.nhs.uk/Tools/Pages/Healthyweightcalculator.aspx. Using the HFA data only,

CoV values of gait timing in 'healthy weight' and 'overweight' were analysed to see if

weight significantly impacted the variability of the results [Table 22, Appendix Y]. Data

satisfied normality and homogeneity of variance assumptions in most instances [Table 19,

Appendix W]; one-way analysis of variance was employed to seek differences between the

CoV of timing in all participants with HFA and their weight cohort; Healthy, $n = 12$, and

Other (Overweight or Obese), $n = 4$, with no significant difference between weight cohort found for Left Step $F(1,15) = 0.26$, $p = .62$, $\eta^2 = 0.00$, Right Step $F(1,15) = 0.01$, $p = .92$, $\eta^2 = 0.01$, Right Stance $F(1,15) = 0.15$, $p = .71$, $\eta^2 = 0.02$, Left Swing $F(1,15) = 0.00$, $p = 1.00$, $\eta^2 = 0.00$ or Right Swing $F(1,15) = 0.09$, $p = .77$, $\eta^2 = 0.01$.   A Mann Whitney U test was run for Left Stance showing no effect between Healthy (*Mdn* = 0.0752) and Overweight/Obese cohort (*Mdn* = 0.0897), $U = 22$, $z = -0.24$, $p = .86$, $r = -.06$. There is minimal risk that the overweight participants distorted the overall results.
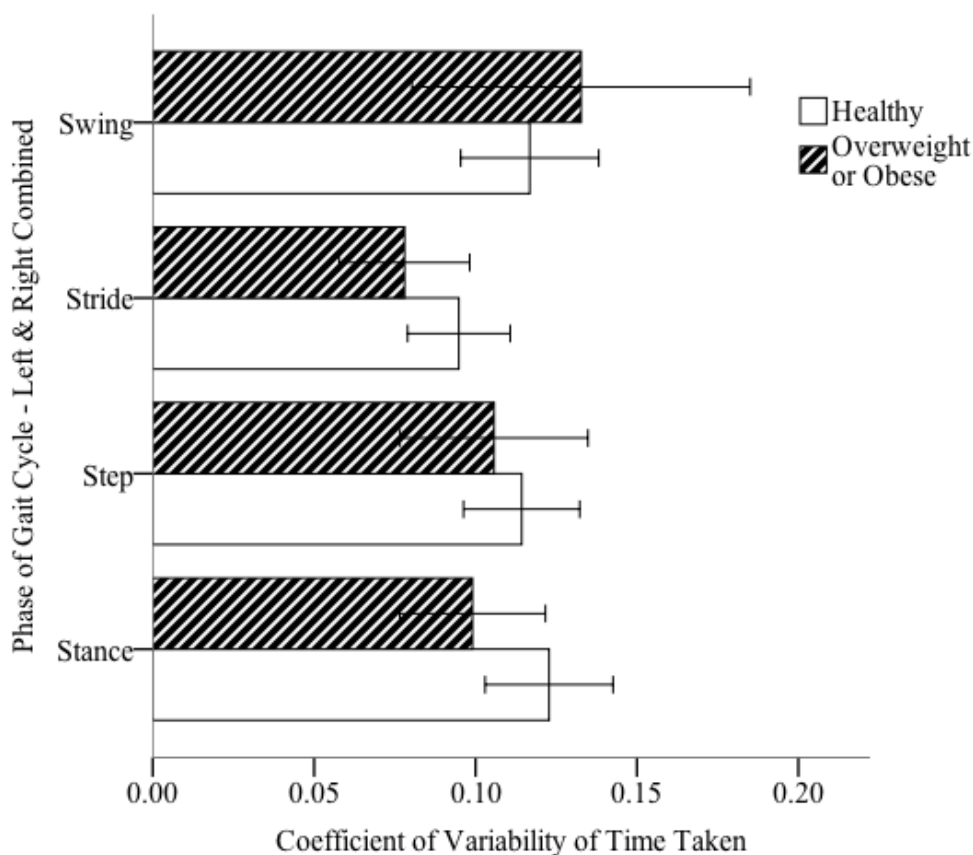
Figure 10. No significant difference found between gait variability (Step, Stance, Swing or Stride) according to the Body Mass Index of HFA participants. Healthy, $n = 12$; Overweight and Obese, $n = 4$.

Extraneous Diagnostic and Medical Factors

Any genetic or comorbid diagnoses would be potentially confounding, and medication can affect locomotion. Comorbid Learning Disabilities (LD) is a potential confound because

of anomalous gait in individuals with LD. Medical records were sought from Child Health

Database and no concerning factors were identified for genetic abnormalities, comorbid

diagnoses or medication [Table 16, Appendix V]. There were no participants with a Low

Birth Weight, threshold set at < 2.5kg (Figure 18, Appendix Z).

 Anthropometric anomalies. A participant in the High Functioning Autism group

was found to have one leg unusually longer than the other when placing the markers on the

required anthropometric locations of the knee. The physical discrepancy: Left leg length =

101cm, Right leg length = 99cm. A Paired samples t test [Table 19, Appendix W] was run to

assess if the variability in his gait cycle differed significantly between his Left and Right leg,

treating the Left and Right leg as a repeated measures variable. There was no significant

difference between legs, $t(3) = 2.66$, $p = .076$, $r = .74$. Therefore, a subsequent analysis

excluding the data from this participant was not considered necessary, as it is unlikely to

distort research conclusions [Table 20, Appendix V].

Suspected Learning Disabilities

 Following diagnostic information from clinical files and consultation with clinicians,

participants number 2 and 13 were considered to have a comorbid LD. Mann-Whitney U

Tests for Coefficient of Variability of gait timing were re-run to establish if increased gait

variability in autism compared to controls was still apparent despite removing these two

confounding individuals. For Step CoV there was still a significant difference between $n =$

14 HFA ($Mdn = 0.0807$) and $n = 14$ TD ($Mdn = 0.0522$) for Left Step, $U = 25$, $z = -3.35$, $p <$

.001, $r = -.63$; Right Step, HFA ($Mdn = 0.0680$) and TD ($Mdn = 0.0518$), $U = 21$, $z = -3.54$, p

$< .001$, $r = -.67$. There was also a significant difference between groups for combined Left

and Right Stance, HFA ($Mdn = 0.0898$) and TD ($Mdn = 0.0473$), $U = 9$, $z = -4.09$, $p < .001$, r

$= -.77$ and for combined Left and Right Swing, HFA ($Mdn = 0.0825$) and TD ($Mdn =$

0.0495), $U = 24.5$, $z = -3.38$, $p < .001$, $r = -.64$. Removing participants Number 2 and

Number 13 from a version of the data analysis for Step, Swing and Stance induced no

alterations to the statistical conclusions of increased temporal variability in participants with

High Functioning Autism [Table 18, Appendix W].

Discussion

This research focused on temporal variability in gait, comparing participants with High Functioning Autism with Typically Developing controls. There is a multitude of existing research stating variability in step and stride length, proposing theories implicating fronto-striatal systems, the basal ganglia and the cerebellum, with an inconsistent influence of these systems contributing to the heterogeneous nature of the autistic disorder. The intention was not to focus on the distance covered with each step, but the variability in timing taken to execute four components of the gait cycle. As expected, no differences in average timing but significant differences in temporal variability were found between autism and controls, for Step, Stance, Swing and Stride.

The work of Rinehart et al. (2006b) reports similar results using a GAITRite walkway, with no difference in mean values but a significant variability regarding stride length duration in 11 children with autism. Calhoun et al. (2011) investigated kinetic and kinematic gait patterns in 12 children with autism, finding a higher cadence in children with autism using a VICON Motion Capture System, conflicting with the current finding of congruent average time taken to execute six steps between HFA and TD groups. It was concluded (Calhoun et al., 2010), converse to the present results, that there were no differences in temporal-spatial parameters. It was acknowledged that variability of temporal parameters were not assessed, which were considered the imperative research measure in the present research.

A contradictory finding was noted (Mari et al., 2003), with participants with Asperger's showing slower movements, whereas no average timing difference was found in our participants. However, it was only the low IQ group that showed this, whereas our research recruited only participants with a normal IQ.

Current Results in the Context of Neurobiological Theories of Gait

Previous literature suggests a combination of clock gene and temporal synchrony theories for autism, and neurobiological explanations focus on differential involvement of anomalous basal ganglia and cerebellum according to diagnosis, plus the influence of Purkinje cells, which link to both genetic and cerebellum theories.

Basal ganglia. The basal ganglia structure is associated with a Parkinsonian style walk, which has been compared to the autistic gait (Damasio & Maurer 1978; Eposito & Venuti, 2008; Hausdorff et al., 1998; Mari et al. 2003; Vernazza-Martin et al. 2005; Vilensky et al. 1981). The Parkinsonian gait is characterised by smaller step length, slower speed and increased stance time. Stride length variability increases as a function of disease progression (Blin et al., 1990), suggestive of cumulative dysmorphology of the basal ganglia (Hausdorff et al., 1998).

Smaller step length was reported in many autistic gait studies (Nobile et al., 2012; Vernazza-martin et al., 2005), but no spatial parameters were recorded in the current research. This is not a deficit of the study because the intention was to investigate temporal effects in isolation, and not replicate the multitude of studies existing regarding step lengths. No average timing differences for any gait cycle component or overall 'Cadence' were identified in the present research. The intention was to verify that it is predominantly the variability of gait timing within a typical gait cycle (not the ability to walk at a regular speed), which is affected by autism.

Fronto-striatal system theories of gait posit that neural pathways involving the basal ganglia are responsible for motoric mediation and can explain both the Parkinsonian and the autistic symptomatic presentation (D'Cruz et al. 2009), especially in relation to preparation and timing of locomotion. The current research didn't involve any measures distinguishing planning faculties in gait so the anomalies found are unlikely to be a function of fronto-striatal circuitry dysfunction. Furthermore, the current research didn't find a slower speed or

an increased Stance time on average, suggesting more of a temporal variability anomaly and not a Parkinsonian similarity. The variable timing concept links with gait due to mutual connections with cerebellar function. Ivry and Spencer (2004) found that the cerebellum is largely responsible for motor and perceptual tasks requiring very specific timing, and lesions here commonly induced increased variation in temporality. The basal ganglia are implicated for slightly larger time frames and for elements of temporal processing involving decision processes; dopaminergic input modulates this mechanism. This is relevant when considering the differences between autism and ASD and their complex basal ganglia differences (Nayate et al., 2005; Rinehart et al., 2006) and Parkinson's Disease for dopamine correlates (Eposito & Venuti, 2008). Ebersbach et al. (1999) found that cerebellar ataxic patients, but not patients with Parkinson's Disease, demonstrated greater temporal variability during execution of a step. Thus a cerebellar anomaly, inducing motoric temporal variability, seems more appropriate than an explanation implicating the basal ganglia structures.

Cerebellum. The cerebellum is responsible for adaptive timing and this function is diminished in individuals with autism (Grossberg & Seidman, 2006). The cerebellar pathology found in the majority of autistic patients (Courchesne et al., 1994) provides physical evidence of the deficient function of the cerebellum in this clinical group. Coordination is modulated partly by the Cerebellar Locomotor Region (Nayate et al., 2005). Inconsistent coordination could contribute to the variability in timing of steps, because postural sway and ill-coordinated limbs could induce an arrhythmic gait. The wide array of studies reporting decreased stability in ASD (Kohen-Raz et al., 1992; Minshew et al., 2004; Vernazza-Martin et al., 2005; Yang et al., 2012) could suggest that this influences the rhythm of the gait, thus affecting temporal measures of the Step, Stance, Swing and Stride variability with participants in the current research. Postural sway is commonly reported in autism (Minshew et al., 2004), however there are some conflicting conclusions. Gepner and

Mestre (2002) only report postural differences in individuals with Asperger's, but not

autism.   The latter would suggest that as the current participants have autism and

demonstrate temporal variability, then posture and balance is not the explanatory factor for

temporal variability.

The Cerebellar Locomotor Region integrates sensory input and premotor cortex

information to coordinate movements (Nayate et al., 2005), so variability in step timing could

be explained in this context instead of balance or posture. The cerebellum is responsible for

modulating patterns of limb movement (Morton & Bastian, 2007). Patients with cerebellar

ataxia struggle to maintain a rhythm and have variable gait timing (Ivry & Keele, 1989;

Stolze et al., 2002) and lesions in the cerebellum impact the capacity to execute movements

requiring very specific timing (Ivry & Spencer, 2004). The ability to adaptively time each

step according to forward trajectory is necessary to maintain a rhythm, thus could explain the

temporal variability found in the gait of the participants with autism in the current research.

Purkinje cells and clock genes. It is pertinent to consider the neural correlates of

autism that contribute to the timing deficits and other anomalous features of the autistic

gait. Grossberg and Seidman (2006) conducted research on neural correlates of timing,

motor processes, emotion and cognition in autism. The prefrontal and temporal cortex,

hippocampus, cerebellum and amygdala are thought to interact to produce autistic symptoms.

The adaptive timing function of the cerebellum is diminished in people with autism,

contributing to motor symptoms, because the cerebellum is inherent in motor function. A

model is postulated for the cerebellum's adaptive timing of motor commands, involving the

aforementioned deep cerebellar nuclei; Purkinje cells. People with autism have been found to

have a reduced quantity and structural arrangement of Purkinje cells (Arin et al., 1991;

Fatemi et al., 2002; Hashimoto et al., 1995; Kemper & Bauman, 1993; Palmen et al., 2004).

The mutation of the epistatic RORA-staggerer gene in autism induces retarded

Purkinje cell development in neonates (Messer & Kang, 2000), suggesting a tentative linking

of Purkinje cell and cerebellar theories to clock gene research in autism (Nicholas et al.,

2007; Wimpory et al., 2002). If Purkinje cells are responsible in some part in the adaptive

timing function of the cerebellum, then this is an appropriate explanation for variable

temporality in the Step of participants with autism in the present research. The genetic

implications suggest that modulation of circadian rhythms could be facilitative in subsequent

temporal synchrony abilities and motor control involving temporal variability in individuals

with autism.   Temporal synchrony in the brain is enabled in some capacity by inferior olive

neurons.

Inferior olive. Inferior olive nuclei are located in the medula oblongata, part of the

brain stem, and connect to the phylogenically most recent region of the cerebellum, the

neocerebellum or posterior lobe, via the olivocerebellar pathways (Biller, Gruener & Brazis,

2011). The fully developed human brain exhibits its highest density of electrical synapses

within the inferior olive (Llina's et al., 1974; Sotelo et al., 1974). The interaction of

electrically coupled inferior olive neurons and inhibitory GABAergic deep cerebellar nuclear

nuclei create local inhibition of the neuronal coupling, which subsequently prevents neural

oscillations from continuing. This inhibitory chemical synaptic action is vital for second-by-

second spatial structure within the coupling of inferior olive dendrites (Lang, Sugihara &

Llina's, 1996). Other deep cerebellar nuclear nuclei are, conversely, excitatory to other

structures, including the cerebellum.   Inferior olive neurons fire action potentials in

synchrony with others in a 'cluster' (Llina's & Yarom, 1981; Sasaki, Bower & Llina's, 1989)

and modulate and reinforce oscillations occurring at a subthreshold level (Llina's & Yarom

1986; Long, Deans, Paul, & Connors, 2002), thus enabling groups of inferior olive neurons to

behave as one amalgamative structure (Welsh, Ahn, & Placantonakis, 2005).

Buzsaki and Draguhn (2004) explain how mammalian cortical neurons are organised

to create oscillating neuronal networks of different sizes for specific behaviour requirements, are selective to the sensory input, link neurons into temporally-relevant groups and promote plasticity of synaptic actions.

The defective inferior olive physiology in people with autism (Bailey et al., 1998; Kemper & Bauman, 1993) substantiates evidence that they operate within a different temporality when processing stimuli. In human subjects, removal of the inferior olive disallowed regular responses to conditioned stimulus and unconditioned stimulus when displayed at 250 – 500 millisecond intervals (Mintz, Lavond, Zhang, Yun & Thompson, 1994). Subthreshold oscillations of inferior olive neurons function as a metaphorical clock, governing a tempo of excitation for the neuronal ensembles, which enable processing of rapid stimuli, including language. This has obvious implications in the brain of a person with autism and helps to explain the reasons behind their timing, language, executive function, multi-modal sensory integration and social interaction difficulties.

A quantitative measure designed to assess these autistic symptoms is the Autism Diagnostic Observation Schedule (Lord, Rutter, DiLavore, & Risi, 2001), used in the present research. It picks up social interaction and communicative difficulties in a subtle way by taking into account variation in language ability and intelligence. The tasks involve conversation for some participants, but at a pre-verbal level there are tasks designed to see how requests are made (i.e. any eye contact), if turn-taking between the administrator and the participant is observed during interactive play, and whether anything the participant finds enjoyable is shown to the administrator with reciprocal social intent. This assessment has been extensively developed by the Autism Research Centre since 1989 and is used widely by clinicians as a diagnostic tool. The quantitative scoring format and high reliability attained by the administrator (K Forster) made it a sensible choice to utilise when obtaining a measure of autism severity.

No Correlation Between ADOS and Gait

It was expected that a correlation would be found between gait variability and the severity of symptomatic presentation in the participants with High Functioning Autism, as measured by a composite Communication and Reciprocal Social Interaction score from the Autism Diagnostic Observation Schedule. However, no relationship could be found between any element of the gait cycle and either component of the ADOS assessment. It was initially suspected that perhaps the sample of high functioning subjects with average or above average intelligence didn't allow much space for variation within the group's symptomatic presentation, because previous studies found patterns between motor impairment and intellectual functioning (Green, Charman, Pickles, Loucas, Chandler, Simonoff & Baird, 2009).

However, the ADOS scores did span a large range. Despite the ADOS assessment possessing high credibility as a diagnostic test alternative methods were sought to establish any relationship between autism severity and gait variability. Clinician's opinions were sought by Dr Dawn Wimpory, Consultant Clinical Psychologist in Autism, providing a list of those with 'Low Variability', 'Moderate Variability' and 'High Variability' scores to assess what symptomatic differences could be contributing to the vastly different gait scores. No clinical opinions shed light on the reasons for the variability categories in the participants.

Distinctions Between Diagnoses

In one pivotal study (Rinehart et al., 2006) the group with autism was shown to have greater variability in stride length than both the control participants and the Asperger's disorder group. There were stride length differences in terms of spatial variability but not average scores, complimenting the current temporal findings.

They assessed the differences between the clinical groups during walking governed by white

floor markers and normal preferred walking, finding cued differences between autism and Asperger's. The authors concluded that participant gait differences arose from the differences in the cerebellum, and the common motoric characteristics suggestive of more comparable basal ganglia fronto-striatal abnormalities. No overall timing variability differences were found between categorical diagnoses in the current research participants; the average times and CoV times were no different between Asperger's and High Functioning Autism.

The discrepancy between the results of Rinehart et al. (2006) and the present research could be explained by the method of grouping the two clinical diagnoses of autism or Asperger's disorder. Four experienced clinicians, with inter-rater reliability of over .94, diagnosed the participants for the former research and rated their behaviour according to DSM-IV (APA, 1994) criterion. So it is possible that the reason for the differences between Asperger's and autism in Rinehart et al (2006) and minimal differences between HFA/Asperger's dichotomy in the current research could be due to misdiagnosis in the latter. Although this seems unlikely due to excellent communication between Dr Dawn Wimpory (Consultant Clinical Psychologist) and the highly experienced clinicians involved, it is arguable that the former research had a more thorough system for dividing into the two diagnoses, with inter-rater reliability criterion. The Diagnostic and Statistical Manual of Mental Disorders-IV [Appendix X] was under critical scrutiny regarding these diagnoses and there is much controversy about where to place a child on the autistic spectrum (Fitzgerald & Corvin, 2001). Alternatively, it could be that the distinguishing elements of the gait between autism and Asperger's (Rinehart et al., 2006) are picked up by the Clinical Stride Analyzer (CSA; B&L Engineering, CA, USA) but not the VICON system used in the current research. Congruent to the current research, no average values were found to differ between the three groups (Rinehart et al., 2006).

Nayate et al. (2012) also investigated gait differences between autism and Asperger's.

Differences in gait characteristics between diagnostic categories suggest distinct motor circuit anomalies for each diagnosis, leading to a specific cascade of neurodevelopment. Their research studied the extent of control the children had over their walking patterns by requesting different walking speeds (preferred, fast and slow) and the consequences of auditory pacing and visual cueing on gait patterns. Dual-task conditions were also imposed to investigate whether additional complex demands would require information processing that detracted from the child's ability to perform a normal gait. These hypotheses were all investigated by a GAITRite System, recruiting participants aged 7-18 diagnosed with either Asperger's Disorder or Autism. Significant differences between the Autism and Asperger's Disorder groups were found in terms of stride length-cadence relation. All walking speeds showed a wide base of support in autism, implicating striatal and cerebellar motor circuits to control balance. In autism, ~16 cm greater stride lengths were observed regardless of cadence, especially during slow or normal cadence.

Despite similarities between Parkinsonian gait disturbances and Autistic gait anomalies, the typical Parkinsonian gait trend demonstrates a shorter stride length regardless of speed, due to reduced function of the basal ganglia restricting readiness for motor activity. In contrast, the large stride length seen in autism is thought to be the result of a disturbance to the fronto-striatal system, inducing an inability to execute the intended movement, so a larger scale stride is performed. Conversely, the Asperger's Disorder group demonstrated a more varied base of support in preferred walking but not in requested fast/slow cadence. This suggests greater intent and cognitive factors provoking a more consistent gait and the finding provides further support for fronto-striatal system theories, specifically the dorsolateral prefrontal regions. Further trials included research on visual cues and simultaneous tapping, to encompass cognitive demand. This research didn't investigate spatial measures or cognitive tasks so are not directly comparable, but the observation by

Nayate et al. (2012) that the Asperger's participants were more variable during their preferred

walking pace is of interest. Perhaps temporal gait differences between these two diagnoses

can only be observed when simultaneous tasks are required of the participants.

Lateralisation Effects

Reduced neural connectivity in autism is attributable in some portion to corpus

callosum anomalies, as it is one of the largest structures of white matter in the brain (Piven,

Bailey, Ranson, & Arndt, 1997). People with autism demonstrate decreased long-range white

matter connectivity in favour of excessive short-range connections (Belmonte, Allen, Beckel-

Mitchener, Boulanger, Carper, & Webb, 2004) and a higher proportion of white matter in the

Primary Motor Cortex, correlating with motor impairment (Mostofsky et al., 2007). A

reduction in size of the corpus callosum affects inter-hemispheric connectivity (Hardan,

Pabalan, Gupta, Bansal, Melhem, Federov, Keshavan & Minshew, 2009) and reduced

connectivity is thought to be one of the neurobiological explanations for the autistic

aetiology. Variable time taken to execute gait tasks such as 'Step' could be due to

dysfunctional neural communication via decreased connectivity. Perhaps reduced

hemispheric connectivity due to a dysfunctional corpus callosum could explain the

lateralisation effects found in the present research participants with autism.

Dichotomy of Asperger's and autism. No gait differences were found between sub-

categories of the HFA group as dictated by ADOS scores. Diagnostic information was

derived from Clinical files, specifying a diagnosis of Asperger's or Autism. High Functioning

Autism had been requested initially. Some of the participants recruited were incidentally

more closely fitted to a diagnosis of Asperger's than High Functioning Autism; clinicians are

aware of how DSM-IV distinctions can be ambiguous, especially in a client presenting

enigmatic symptoms (Fitzgerald & Corvin, 2001). The recruitment criteria specifying high

functioning subjects could have presented a bias in the clinicians to select clients with intact

language, and thus the diagnostic cohorts were an unforeseen confound. A dichotomy was created on these grounds to assess if any difference in gait could be established between the two clinical groups. There were no significant differences between the gait of the two diagnostic cohorts, with no difference in Average or CoV timing scores for any component of the gait cycle.

It was not expected to find any lateralisation effects, as to our knowledge no previous gait studies have referred to such findings. There was an increased Left Step variability compared to Right Step in the Asperger's/HFA group, and no disparity in TD controls. When seeking lateralisation trends between diagnoses, it was found that only participants diagnosed with Asperger's demonstrated a significantly increased Left Step over Right Step timing variability. There was no significant Left/Right Step disparity in participants with a diagnosis of High Functioning Autism, although there is a trend for greater Left Step across the all non-TD cohorts [*Figure 9*]. Other studies have found differences between autism and Asperger's (Nayate et al, 2012; Rinehart et al., 2006a; Rinehart et al., 2001).

Previous research in relation to motor asymmetry in autism. Chester and Calhoun (2010) found no asymmetries in the gait of older children using a VICON Motion Capture System and collecting temporal-spatial measurements and symmetry indices. However, they selected the most representative (nearest to the mean) example of each participant to subsequently analyse, so variability within each participants' trials was lost.

Some asymmetries were found in infant studies regarding the positions that babies lie, with movement disturbances noted generally to occur on the right hand side of the body (Teitelbaum et al., 1998), which conflicts with our left hand side anomalous findings. In toddlers, asymmetries were found in those with autism in both a static and dynamic modality (Eposito et al., 2011). The Positional Pattern for Symmetry during Walking was used with significant findings between participants with autism and controls, but no specificity of

directional asymmetry was reported.

Handedness and footedness. Research is varied regarding whether individuals with autism have a higher prevalence of left-handedness, and connectivity theories have largely replaced handedness theories. Some claim that there is a disproportionate degree of preferential left handedness in autism (Hauck & Dewey, 2001) but elsewhere conclusions are mixed; Cornish and McManus (1996) suggests that the prevalence lies in the youngest children with autism, whose typical lateralisation switch from right hemisphere to left hemisphere (Chiron, Leboyer, Leon, Jambaque, Nuttin & Syrota, 1995) is subject to developmental delays, and that left handedness decreases with age as it does in typically developing children.

Footedness is potentially a better measure of neural hemispheric dominance than handedness, because of fewer neural resources for lower than upper limbs (Gabbard, 1993). There was no discrepancy between left and right handedness or footedness in children with Asperger's and High Functioning Autism and typically developing controls (Markoualakis, Scharoun, Bryden & Fletcher, 2012), assessed by a variety of tasks like kicking a ball, or squashing a fake bug on the floor, but proportionally, eight out of 12 of the children with autism showed a right foot preference. No effect was expected between left and right leg in the current research. The incidental finding is a benefit of the research despite not being able to conclusively rule out handedness as an extraneous variable.

Issues of contralateral and ipsilateral effects. Cerebral hemispheres are renowned to affect the body contralaterally (a right hemisphere lesion would induce left side motor deficits). Conversely, cerebellar hemispheric lesions on the contrary induce ipsilateral signs. So anomalies in the right cerebellar hemispheres would potentially cause anomalies on the right hand side of the body (Biller et al., 2011). Thus the ideal scenario for providing an aetiological explanation for a Left Step anomaly would be to find previous research noting

cerebellar differences in the Left cerebellar hemisphere in participants on the autistic spectrum, specifically in Asperger's.

Previous research on lateralisation in Asperger's. In individuals with Asperger's, the right hemisphere is frequently noted as being anomalous, not the left (Ellis, Ellis, Fraser, & Deb, 1994; Fein, Humes, Kaplan, Lucci & Waterhouse, 1984). The right hemispheric differences in the participant with Asperger's could explain the contralateral Left Step variability as required, especially considering there is no lateralisation finding when participants with High Functioning Autism were separated from those with Asperger's.

Gesturing ability was examined in children with High Functioning Autism and Asperger's Syndrome (Steinman et al., 2010), concluding that the HFA group presented more tool for body-part errors, thus implicating right supramarginal gyrus deficits. The right hemispheric location is a suitable finding to coordinate with the current Left leg anomalies, but gesturing is not similar enough to gait to draw any direct comparisons or suggest neurobiological implications. Perhaps more apt is the discovery that the right hemispheric basal ganglia structure is deformed in participants with ASD at the posterior putamen (Qiu et al., 2010) and was predictive of deficient motor abilities.

Asymmetrical motor findings in 8 – 12 years olds with autism involved finger-tapping tasks and simultaneous Functional Magnetic Resonance Imaging (Mostofsky et al., 2009). There was a Left Hand finger sequencing speed difference found with a corresponding right posterior cerebellum anomaly. There was significantly less activation in this region of the cerebellum in children with autism, and decreased connectivity across the network responsible for motoric action. The posterior cerebellum is involved in fine motor control. Interestingly, the posterior cerebellum is activated during rhythmic tapping tasks, requiring temporal control (Thaut, 2003).

Research by Markoualakis et al. (2012) using foot tapping tasks garnered an interesting result; only the group with Asperger's or HFA showed a preference to use the right foot for foot tapping tasks involving rhythm, and longer mean interval durations when using the left foot to tap. Of importance is the finding that the standard deviation of left foot tap duration was higher than the right foot for the group with autism, mirroring the Left Step higher variability in timing in the current research.

Temporal Variability of 'Left Step' in Asperger's

The aetiological reasons behind a higher temporal variability in Left Step in Asperger's but not High Functioning Autism is beyond the scope of the present research. The accidental inclusion of some participants with Asperger's Disorder and not High Functioning Autism has lead to a myriad of tentative theories regarding the aetiology of the greater lateralised gait timing variability in Asperger's

Right hemisphere dysfunction is examined in participants with Asperger's syndrome using single photon emission computer tomographic (SPECT) imaging (McKelvey, Lambert, Mottron, & Shevell, 1995). Right hemisphere anomalies were found including an enlarged right lateral ventricle, decreased right hemisphere uptake and a smaller right cerebellar hemisphere. Lower volume of grey matter is reported in the right cerebellar hemisphere in Asperger's Disorder, alongside a lower volume of right putamen in Asperger's Disorder (Yu, Cheung, Chua, & McAlonan, 2011).

The right hand side anomalies found in the putamen (Yu et al., 2011) and lateral ventricles, plus lesser right hemisphere uptake (McKelvey et al., 1995) are subject to the typical contralateral limb affects, so are appropriate for postulating causes of left leg anomalies in Asperger's. However, white matter deficits identified in the left cerebellum in participants with Asperger's syndrome (McAlonan et al., 2002) are the appropriate cerebellar hemispheric location for ipsilateral motoric effects on the Left leg and the cerebellum is the

area of most interest.

Alternatives; autism, Asperger's and hemispheric development. Escalante-Mead, Minshew & Sweney (2003) compared people with autism, some with language developmental disorders and some with typically developing language. Those with early language delays demonstrated greater atypical cerebral dominance, suggesting disturbance of brain maturational processes. Language and the cerebellum were subsequently investigated in autism (Hodge, Makris, Kennedy, Caviness, Howard, McGrath, Steele, Frazier, Tager-Flusberg & Harris, 2010) in a Magnetic Resonance Imaging method. The cerebellum of participants with Specific Language Impairment, and participants with autism, some of whom had impaired language, were compared. There was a reversed asymmetry in the language-impaired groups regarding the posterior-lateral cerebellar lobule, with a larger left hand side in the groups with impaired language, with or without autism. The autistic participants without language impairment showed a larger right hand side posterior-lateral cerebellar lobule. This is relevant when considering laterality in Asperger's participants only; the diagnosis involves no language impairment.

Rumsey and Hamburger (1988) found that basic language abilities controlled by the left hemisphere were normal in their participants with autism, but that elements of language mediated by the right hemisphere were anomalous i.e. pragmatics and prosody. Reports of left hemisphere dysfunction had suggested a disparity between autism and Asperger's disorder, because language faculties are predominantly in the left hemisphere and language is relatively intact in Asperger's disorder (Yu et al., 2011). Participants with autism were shown to have anomalous left hemisphere performance during tasks requiring executive function. The participants with Asperger's showed laterality effects similar to the control participants for both tasks. A fronto-corticocerebellar circuit abnormality was posited as an explanation (Hodge et al., 2010), consistent with the proposal of frontostriatal models for autism

(Bradshaw, 2001; Rinehart, Bradshaw, Brereton, & Tonge, 2002) as an alternative to the

previously pertained to notion of Left Hemisphere Dysfunction (LHD). However, it has been

suggested that frontostriatal circuitry disturbance of laterality may only be present in autism

not Asperger's (Rinehart et al., 2002).

Age and Gait

It is complicated to disentangle the rationale behind the lateralisation findings. The

whole group of participants with autism demonstrated a significantly higher Left than Right

Step. Removing participants with High Functioning Autism and only examining participants

with a diagnosis of Asperger's showed this effect, but examining participants with High

Functioning Autism, and not Asperger's, did not show this effect. Furthermore, only the

youngest cohort of all the participants (Asperger's and High Functioning Autism combined)

showed this Left Step disparity.

There was a lateralisation effect for age, with only the youngest (12 and under)

HFA/Asperger's participants demonstrating a larger Left Step CoV than Right Step CoV.

The HFA/Asperger's participants aged 13 years and above didn't show this lateralisation.

Following the significant result of finding Left Step disparities according to specific clinical

diagnosis, Asperger's but not High Functioning Autism, it was confirmed statistically that

there wasn't simply a greater number of younger participants in the group diagnosed with

Asperger's.   It's impossible to conclude for certain whether the specificity of diagnosis or the

age cohort is the explaining factor. The two significant results are independent, so no

conclusion takes precedence over another.

Chiron et al., (1995) used Single Photon Emission Computed Tomography (SPECT)

to measure regional cerebral blood flow (rCBF) in children aged 4 – 17 years with autism. In

controls the rCBF values were higher in the left than the right hemisphere, but in autism this

was reversed, independent of age and handedness.   The lateralisation effect in the youngest

cohort suggests that a developmental delay in switching from Right to Left hemispheric dominance could be an area of interest. This suggests that a dysfunctional right hemisphere may exist in both autism and Asperger's, with additional LHD developing in autism.   The differences between Asperger's and autism could be explained by variations in the period of emergence of lateralisation in the brains of these individuals (Rinehart et al., 2002). In typically developing children, SPECT demonstrated CBF dominance in the right hemisphere between the ages of 1 – 3 years old (Chiron, Jambaque, Nabbout, Lounes, Syrota, & Dulac, 1997). At the age of 3, hemispheric dominance shifts to the left hemisphere, likely to be due to the emergence of language. In autism language is delayed, so this hemispheric shift could be delayed. A developmental dysfunction of the left hemisphere could explain some of the lateralisation effects found in the present study as a function of age cohort. Typically developing controls demonstrated a more consistent hand preference from the age of 3 to 12 years old, whereas participants with autism were less consistent at the age of 3 and this consistency didn't increase to the same level as the controls by the age of 12 (Cornish & McManus, 1996). Furthermore, Left handedness decreases to 15% from 33% between the ages of 3 to 12. A hemispheric dominance switch to the left hand side at a developmentally later stage of around twelve, is a suggested link for the occurrence of Left Step greater variability than Right Step in the youngest cohort (cut-off point of 12 years) of participants with HFA/Asperger's.   The conflicting aforementioned research conclusions regarding hemispheric dominance and diagnosis of Asperger's and High Functioning Autism make this an interesting feature for future research, but it is not possible to postulate neurobiological reasons behind this finding in the present research.

Further tests confirmed no difference between age cohorts ('12 and under' or '13 and above') for combined Left and Right Step, Stance or Swing time, implying that gait analysis is still suitable as a diagnostic tool. No correlation was found between overall gait variability

and participant's chronological age. This is a benefit of the research conclusions, because it means that the significant findings are not merely picking up on motoric developmental delays, which have been reported in the autistic population (Provost, Lopez & Heimerl, 2007).

A lack of age and gait correlation substantiates previous research (Vilensky et al., 1981) and subsequently the opinion that gait analysis is a suitable diagnostic method to implement amongst a range of other tools at the disposal of a psychologist during assessment for autism. Sutera et al. (2007) found motor skills at 2 years of age were predictive of subsequent autistic symptoms. Anomalous gait in autism is not merely a delayed developmental phenomenon and is a consistent symptom across all age groups with this diagnosis. Gait analysis a suitable diagnostic method providing potential for asymmetries in younger participants is noted.

Anomalous participant with High Functioning Autism

One participant (number 20) with High Functioning Autism presented a very odd gait. His first eight walking trials looked a bit clumsy, deliberative, and similar to the remaining HFA participants to the eye. At this point his mother intervened and told him to "Just walk how you normally do when we go out for a walk." This instruction induced a strikingly different gait pattern, with an unusual lopsided hop, rapid and asymmetrical but rhythmic. The Coefficient of Variation was not extreme; he was so rhythmic with this unusual walking pattern, fitting into the 'Moderate Variability' cohort. Yet subjectively, he presented as the most unusual of all the participants. A significantly different average Swing timing was found for his second gait style, with the Left Swing taking longer on average than the Right Swing. There was no difference between his Left and Right Step or Swing time in terms of variability for the unusual gait, suggesting that the method of analysis CoV of Step timing was not sufficient to detect his particular gait quality. The method of gait analysis is

important to consider, and will be discussed in the context of previous research.

Criticique of Gait Analysis Systems

   ELITE. The ELITE Gait Analysis System was used by Nobile et al. (2012) and

Vernazza-Martin et al. (2005) for their research into the autistic gait. The ELITE system is

automatic and analyses kinematic and kinetic measurements of gait, using pattern recognition

of anatomically placed markers (Giannini, Catani, Benedetti & Leardini, 1994). Force

platform information is analysed in synchrony with electromyographic data and foot switch

(for temporal parameters) data. The temporal resolution is 50Hz – 100Hz, which isn't as

high as the VICON system used currently, but the synchronous measurements are undeniably

a benefit not present in the current research.   Temporal parameters were of central focus in

the current research, so having a higher temporal resolution was of greater precedence.

Variability in timing of Steps detected by the 250Hz VICON may not have been picked up by

a less sensitive GAITRite system.

   The Clinical Stride Analyzer. The Clinical Stride Analyzer focuses on temporal

parameters and is used by Rinehart et al. (2006a), collecting measures of cadence, velocity,

gait cycle duration, duration of single and double support by assessing contact at heel, big toe

and 1st and 5th metatarsal.   However, the foot switches worn inside participant's shoes as

insoles are arguably not allowing a natural walk. Barefoot walking permits a natural walk,

not mediated by the range of different participant's shoe styles. There is the option of taping

the insoles to bare feet but this would likely induce an unusual gait due to it feeling novel or

uncomfortable. This could be especially inappropriate for testing the gait of people on the

autistic spectrum, who frequently experience unusually sensitive reactions to sensory

input. Rinehart et al. (2006a) recommended the use of a VICON Motion Capture system for

future research, following their use of a Clinical Stride Analyzer.

The GAITRite System. The GAITrite system used for autism research by Rinehart et al. (2006b), Nayate et al. (2012) and Yang et al. (2012) has been assessed in terms of concurrent validity and test-retest reliability against a Clinical Stride Analyzer (CSA) (Bilney, Morris & Webster, 2003). It collects temporal and spatial data with a temporal resolution of 80Hz, or every 11 milliseconds – the VICON system in this study captures at 250Hz, which is every 4 milliseconds. The GAITRite was found to be reliable in comparison with the CSA in terms of stride length and cadence, weak in terms of proportion of gait cycle in double support. Test-retest reliability with the GAITRite shows variable data when participants are at slow speed, but good when walking normally. The GAITRite system has been compared with a VICON512 and found to be reliable (Youdas, Hollman, Aalbers, Ahrenholz, Aten & Cremers, 2006). Yang et al (2012) found a longer stance time in participants with autism using a GAITRite system, but also a lower cadence. The longer stance time conflicts with the current research findings whereby no average temporal differences were found for any phase, including Stance. The aforementioned lower reliability of the GAITRite system at slow speed could be the reason for this discrepancy. Furthermore, it is stated that the hands of the participants were sometimes held to encourage the walking task, suggesting that the gait may not be very natural (Yang et al., 2012).

The GAITRite has an impressive selection of spatial and temporal parameters. The present research did not intend to replicate the large quantity of existing research on Step Length and Stride Length variability, so temporal parameters were of greater priority. The temporal parameters offered by the GAITRite system include 'First Contact', 'Heel Contact', 'Last Contact'. This is superior to the current research, whereby an 'Event' was manually entered named 'Step' (signifying foot contact with ground) due to the lack of kinetic measures through forceplates. The GAITRite also calculates Step Time, Stride Time, Ambulation Time, Stance and Swing Time, Single Support and Double Support as well as

various ratios of phases within the gait cycle. For future research into temporal variability in autism, a GAITRite system could be the most appropriate substitute to VICON; however, the VICON system had a much higher temporal resolution at 250Hz and was readily available at the University. The GAITRite walkway is 2' X 12', which could induce a less representative walk from participants who feel like they're performing. The VICON laboratory used in the current research was in a large room and the participants just had to walk in a straight line across the room.

VICON – advantages and disadvantages. Wearing the reflective markers could be a potential discomfort for the participants. In previous gait research it is noted that there were some non-compliance issues where child participants wouldn't wear the markers (Longuet et al., 2012). In the current research there was mostly full compliance, apart from a few cases the children would be self-conscious and wear markers on top of long shorts, and be reluctant to have their t-shirt rolled up slightly to reveal hip bones or put shorter shorts on. Any slight reluctance was respected due to ethics standards, so markers were fitted on top of clothing in these cases. Participant comfort was considered priority. It was also more beneficial to the research to have a higher number of participants, and many would have opted to decline to participate if insistence on less clothing had been instigated. This does not affect validity of data collected, because for analysis purposes only foot strike and toe off were ultimately examined for timing. The markers create an excellently proportioned robotic figure based on the participant's body if placed accurately, but accuracy for this research was only truly important on the feet. It was always ensured that ankle, toe and heel markers were visible. It seems most important that the participant feels comfortable, hence obtaining a more representative and genuine gait, than to induce a stiff or self-conscious gait by insisting all markers were applied on bare legs and exposed hipbones.

Similarly, participant enjoyment and a calm emotional state were considered of

paramount importance when they attended the gait laboratory session. It seemed helpful to

make the participant relax and feel less self-conscious, so golf and football activities were

encouraged prior to serious data collection as a tool to soothe the participant and prevent the

feeling of the session being a formal clinical assessment. All were given the option to do this,

and 9 participants chose to do humorous walking, 11 participants chose to play with

reflective golf/footballs. In the information packs designed for children, information was

provided about the technology used and how computer games and animated films have used

the same system. This was to encourage the potential participants to anticipate the

experience in a positive way and not feel intimidated by expecting a formal assessment.

Training for Katharine Forster was received for specific anatomical placement of the

reflective markers for VICON courtesy of Mr William Bromwich (Research Physiotherapist

employed at Orthotic Research and Locomotor Assessment Unit at the Robert Jones and

Agnes Hunt Orthopaedic Hospital, Oswestry). VICON Nexus software training was received

from Dr Martin Warner, Experimental Officer at the Centre for Innovation and Leadership in

Health Sciences, Southampton University.

A 3D VICON MX Motion Capture System was used for this research, with 12

cameras and a 250Hz sampling-rate. In terms of test-retest reliability, VICON has been used

for many purposes including clinical gait analysis.

VICON reliability. GaitMat II was compared with VICON, finding excellent

consistency regarding temporal measures (Barker, Craik, Freedman, Herrmann & Hillstrom,

2006). An assessment of inter-tester reliability and test-retest reliability (Tsushima, Morris &

McGinley, 2003) was run using a VICON system, sessions of 5 trials on two days two weeks

apart, and two individual testers. Test-retest reliability was assessed in two trials separated

by two hours using a VICON 512, finding a very high correlation for time-distance

parameters (Westhoff, Hirsch, Hefter, Wild & Krauspe, 2004). Due to the prerogative of this

research to focus on temporal parameters, it is of benefit to note that these measures have

been assessed as reliable by several independent pieces of research. The VICON MX system

is a high precision system with excellent cameras, software and hardware. An assessment of

various motional analysis systems was held at Nippon Engineer College in Tokyo (27-

29/07/2002); the findings show that VICON has the lowest variability as measured by

standard deviation for measuring the accuracy of distance between two points, angle accuracy

and accuracy of virtual points.

Using the VICON system, Chester and Calhoun (2012) found no differences in

symmetry, despite having a more prestigious system in terms of force-plates for kinetics,

which conflicts with the current Left and Right Step difference in timing in HFA. This could

be due to a lower number of trials than the current research, and only a 60Hz sampling-rate

used. Our greater number of trials and focus on variability not averages, plus a 250Hz

sampling-rate VICON system could account for finding some temporal variability in the

symmetrical presentation of the autistic gait. It is worth noting that a truly objective

quantitative method was not achieved. Due to a lack of a force-plate at the Gait laboratory,

and no heel clicker inserts, the method of establishing when a 'Foot Strike' and 'Toe Off' had

taken place was not ideal. VICON Nexus training was received from Dr Martin Warner

(Experimental Officer, Biomechanics) of Southampton University who suggested the best

method in the absence of a force-plate was to manually insert these 'events'. It is possible,

due to the extremely high temporal resolution of the system used, to play the video in slow

motion by dragging it along the time scale frame-by-frame, until the foot contacts the

ground. At this point an event is marked, I.e. 'Foot Strike', and then the number displayed at

the side of the scale is recorded. This is the time at 250 Hz sampling rate (1/250ths of a

second = to a 4 millisecond accuracy level). Therefore calculating between alternating foot

strikes gives the time it takes to execute a Step, for example. The same event marking is

done for 'Toe Off', to enable calculation of Stance and Swing when combined with 'Foot Strike' times. The number display [Appendix N] reduces the potential subjectivity of this data recording method and it is not considered to have been a risk for experimenter bias. Also, one of the typically developing controls displayed variability at the degree of a highly variable HFA participant, further suggesting that this method does not enable any bias subtly pertaining to experimental hypotheses.

Anomalous control subject

It was initially hoped that gait analysis could be a useful diagnostic tool when distinguishing between enigmatic cases on the edge of the spectrum, using ADOS scores as the measure of autism. Intriguingly, despite no ADOS correlation with gait, the example of the anomalous Typically Developing control could suggest that this theory need not be discarded. Following a manually constructed sub-categorisation of the HFA participants according to whether their gait variability scores were 'High', 'Moderate' or 'Low' (latter = within the range of the control subjects), it was noted that one of the control subjects had gait variability scores falling into the 'Highly Variability' HFA cohort (Table 1). The mother of this control participant spoke of her interest in the research because she used to work with children with autism. She claimed to have always thought her son was on the spectrum and that he had some autistic symptoms, referring to his distress when routines or planned events don't occur as expected and his demand to know when everything will happen in advance. During the gait session her comments were responded to with reassuring tones, but during subsequent analysis of the TD participant's data, this anomalous finding became apparent.

No autism assessment was done on this child (or any of the TD participants) and he didn't present as noticeably autistic, although was withdrawn and shy and didn't interact much. This was not taken much notice of at the time, and only biased conclusions can be drawn from assessing his behaviour retrospectively from memory.   The mother was

subsequently contacted and she stated that she had had conversations with psychiatric nurses following her curiosity about her son's habits, who agreed that he is very sensitive and shows some symptoms. In addition to the necessity for routines, he is uncoordinated, sensitive to noises and light and she has felt it necessary to reward him for giving eye contact. This finding is of considerable interest due to the original intention to be able to assess borderline cases using gait analysis.

Thorough Analysis of Potential Confounds

Statistically, this research employed rigorous use of Levene's test for homogeneity and the Kolomogorov-Smirnof test for normality [Table 19, Appendix W]. If either of these assumptions were violated then non-parametric statistical methods were implemented in place of Analysis of Variance (Dytham, 2010; Gravetter & Wallnau, 2007). Median values were reported for non-parametric tests (Field, 2009). Participants performed a varied number of trials, as more were requested if the participant had curved or hesitated or been distracted in some way. Each trial consisted of 6 – 8 steps in the centre of the room; they represented the most natural steps of the walk; after the rhythm of the walk had been established, and before slowing down to stop for the end of the walk. Total *n* for each stage of the gait cycle was the number of trials multiplied by the examples of that stage of the gait cycle within each trial. Overall an average of 34 – 40 examples of the gait cycle were collected for (Left and Right) Step, Stance, Swing and Stride for each participant [Table 25, Appendix Z]. Hence, the CoV values for each individual were derived from a large and representative sample of their gait. Coefficient of Variation was calculated, which is the standard method of assessing variability in gait (Nayate et al. 2005) because it establishes the variation within the context of the mean, thus accounting for an expected variation in average speed according to age and leg length.

Multiple extraneous statistical analyses were run to ensure experimental validity and

reduce chance of confounding variables distorting the research conclusions.

Controlling for overweight participants. Following the data collection session, the

participants' Body Mass Index (BMI) was calculated. If a participant's BMI was stated as

overweight, they were separated in the data analysis stage. This followed advice from the

Orthotic Research & Locomotor Assessment Unit (ORLAU) at the Robert Jones & Agnes

Hunt Orthopaedic & District Hospital NHS Trust in Oswestry. A senior bioengineer (Dr

Caroline Stewart, Senior Bioengineer, ORLAU) suggested that overweight participants might

present anomalies in the data, as locating specific points in the skeletal and muscular

structure may not be reliable. NHS BMI resources were utilised

(http://www.nhs.uk/Tools/Pages/Healthyweightcalculator.aspx). Results still remained

significant between HFA and TD participant groups; thus it is unlikely that overweight

participants were disproportionately influencing the gait variability.

ADOS and age; separating factors. Despite age-matched controls, it is important

to note that extraneous age-related variables may cause distortion when analysing any

relationship between ADOS and gait variability. Hypothetically, a prevalence of higher

ADOS scores in the younger age cohort would imply that any relationship found between

ADOS and CoV could just be a developmental effect of younger children demonstrating a

more variable gait, and not a positive correlation between high ADOS scores and high

variability. It was established that no relationship existed between ADOS and age, thus

ensuring that any potential correlation found between ADOS and gait variability or Age and

gait variability was not distorted, permitting experimental validity.

Ensuring reliable ADOS coding. Two of the original ADOS tapes ($n = 10$)

could not be viewed. Inter-rater analysis between K Forster's re-coded scores ($n = 8$) and

scores of the attainable clinical tapes suggest that the unattainable tapes ($n = 2$) were likely to

be scored in a manner congruent with K Forster's scoring. K Forster was granted an external,

research-standard, approval of ADOS administration and coding reliability at 88% by Dr

Fiona Scott of Autism Research Centre (November 2012), making the scoring system valid

according to the 'Gold Standard'. Additionally worth noting; one of the two un-checked

tapes was coded by Dr Fiona Scott and her accuracy is not under question.

Gender. To achieve a mixed-gender balanced design would have been problematic,

due to a higher prevalence of males with autism, so only male participants were recruited for

this research to ensure no gender-related confounds were introduced. Other research has

seemingly recruited a minimal number of female participants but not separated in analysis

and not of equal numbers as male participants (Rinehart et al, 2006b), which is an ostensible

methodological confound.

Age-matching. Advice regarding recruiting a control group of Typically Developing

children for the 3D Motion Capture data was sought from a senior bioengineer (Dr Caroline

Stewart, Senior Bioengineer, ORLAU, RJAH Orthopaedic Hospital, Oswestry). It was

established that matching for age is of paramount importance due to gait varying at different

developmental ages.

A reasonable sample size with a stringent recruitment criteria was obtained, with $n =$

16 participants with High Functioning Autism and $n = 16$ age matched controls. The control

subjects were age-matched within a five-month range if they were below 18; it was not

considered as important to match adults as strictly as they are physically fully

developed. Adults were matched within a couple of years and this seemed adequate. No IQ

tests were run on either group, which is a deficit in comparison to some research where

intelligence scores had been matched for as well as age (Rinehart et al., 2006a).

It is also worth being aware that height, weight and IQ were not matched for; our

method of recruiting control subjects did not allow prior knowledge of anthropometric details

of the Typically Developing children, so the research team were unaware of their physical

characteristics prior to their arrival at the gait laboratory. Given no correlation between age

and gait variability, and the method of using Coefficient of Variation, which accounts for

mean timing, it is unlikely that varying heights within the age-matched pairs will have

instigated false positive results.

Only children over the age of 7 were recruited, following advice from Dr Caroline

Stewart that this is a period of developmental transition in terms of gait, thus avoiding any

developmental complication confounds. It was advised that a methodological design would

have to have a balanced number below and above this key age, and there were not sufficient

quantities of participants to recruit a balanced number below the age of 7 years old. Thus, the

results of this timing variability study cannot extrapolate to children under the age of

7. However, previous research (Teitelbaum et al., 1998) has found gait or crawling

anomalies in babies as young as 4 months old, using rudimentary retrospective home video

analysis. Similar conclusions were reached utilising more advanced methods of gait analysis

in toddlers with autism (Eposito et al., 2011), and autism could even be distinguished from

mental retardation in toddlers (Eposito & Venuti, 2008).   So it is possible that extending

research using VICON and gait variability would garner similar results for younger children

and even toddlers.

Medical history. A participant with one leg unusually longer than the other was

excluded from a version of the analysis with no impact on the significantly different

variability between HFA and control subjects.

The main motivation for seeking medical history is to reduce the possibility for

additional diagnoses i.e. co-morbid Learning Disability that could confound the conclusions

of autistic gait characteristics. Participant 4, 20 and 36 are adult participants for whom

medical history could not be obtained, but did not present as having a Learning Disability;

two have attended courses of Higher Education, and one has some accounting work experience.

Exclusion of Co-morbid Learning Disabilities

Initial recruitment criteria excluded participants with a co-morbid Learning Disability, due to the prevalence of anomalous gait within LD populations regardless of autism and intent to reduce potential confounds. Green et al. (2009) found a greater incidence of motor impairments in children with ASD and an IQ less than 70. The recruitment criteria in our introductory letters to other Clinicians requested that they select only High Functioning clients; so although every effort was made to avoid recruiting any participants with a sub-normal IQ, it was suspected that two had a Learning Disability.   Following diagnostic specificity from clinical files, participant Number 2 was suspected to not fit the criteria of HFA due to possible LD. Following the ADOS assessment, participant Number 13 was suspected to not be high functioning; he had very little language but seemed to have good mechanical and analytical skills when playing with toys, and could sing a tune (but not words) in synchrony with the administrator (K Forster). It was required to switch to a version of the ADOS tailored for individuals with no language (Module 1), and thus without speech it is not appropriate to put him in a category of High Functioning Autism. Following consultation with clinicians, numbers 2 and 13 were confirmed to have a co-morbid LD. Number 20 has a specific learning disability, related to language but is not typically LD (accounting work experience) so he was not excluded from subsequent version of data analysis.

Once their data was removed, the differences in variability between the clinical group and the control group still remained significant. Although these two participants constituted a third of the 'High Variability' cohort, this is not considered a problem because this cohort also contained a participant who was at University. It wasn't merely a cohort containing all

the participants with the lowest intellectual ability. The neurological or diagnostic

explanation behind these variability cohorts within the HFA group remains an enigma. It is

possible that varying degrees of cerebellar dysfunction could explain the dichotomies

regarding temporal variability, as the cerebellum is thought to be responsible for motor

variability (Rinehart et al., 2006b). All other participants ($n = 14$) were deemed to have

average or above average intellectual functioning and fitted the criteria of High Functioning

Autism/Asperger's, as diagnosed by their clinicians.

HFA participants' medical history was sought via the Child Health Database,

confirming that there were no suspected concerns for anomalous genetic or medical

influences distorting results. Of particular interest were genetic comorbidities – given the

interplay of genetic influences in autism (Muhle, Trentacoste & Rapin, 2004), especially

Phenylketonuria (PKU), and birth weight. Birth weight is important because babies that are

born underweight have a higher propensity for developmental delays (Vohr, Wright, Dusick,

Mele, Verter, Steichen, Simon, Wilson, Broyles, Bauer, Delaney-Black, Yolton, Fleisher,

Papile & Kaplan, 2000). PKU can cause autistic symptoms (Baieli, Pavone, Meli, Fiumara &

Coleman, 2003; Miladi, Larnaout, Kaabachi, Helayem & Hamida, 1992); it was observed that

all participants whose records we obtained had had routine screening for PKU in infancy,

eliminating this potential confound. The normal range for birth weight is 2.5-4kg. Some of

the participants were up to 535g over the 4kg range but being overweight is not concerning;

the intention was to ensure no participants had a birth weight below the threshold of 2.5kg,

thus eliminating any potential confounds of being underweight at birth contributing to

developmental abnormalities including Learning Disabilities.

The nature of gait analysis suggests it could be a suitable diagnostic tool because the

participants largely enjoyed the experience, something that perhaps formal clinical

assessments lack if they are conversationally demanding. However, any requests to cease

proceedings were adhered to; for example, in the case of one young participant with autism

who was exceptionally shy, and didn't wish to participate upon arrival. Overall though, the

Gait Laboratory experience was a positive one for the participants; many opted to stay much

longer than was required, so they could play golf or football and watch videos of their

robotically mapped selves performing these sports afterwards.

       Close professional relationships. The close professional relationships amongst the

Psychologists was an advantage to this research, enabling information about potentially

confounding variables like medical history to be sought, thus excluding the risk of

confounding genetic disorders or medication effects. Additionally, conversations with

colleagues allowed rejection of certain theories i.e. the lack of distinguishing characteristics

between 'Low Variability' and 'High Variability' gait participants. Clinical file information

and liaising with clinicians enabled participants suspected to have a Learning Disability to be

excluded from a version of the data analysis, enhancing validity of the research conclusions.

Specific diagnostic criteria could be examined, leading to the discovery that the participants

with Asperger's, not High Functioning Autism, demonstrated a more variable Left than Right

Step timing pattern.

       Advantageously for the participants, their anonymous data was made accessible

within ethical protocol to the Superintendent and Clinical Specialist Paediatric

Physiotherapist (Elaine Owen, Head of the Community Paediatric Physiotherapy Department

for North West Wales, with postgraduate qualifications in Lower Limb Orthotic

Biomechanics and Clinical Gait Analysis). One referral was made to the physiotherapist team

at the Child Development Centre, Bangor, following a mother's concern about her son's gait

and the research team's observations that he had a flat-footed gait.

Future Research

       The ultimate aim of gait analysis in autism is not only to establish ways in which

neurological dysfunction logically match the motor symptoms in order to understand the disorder better, but to provide a potential diagnostic capacity. Gait analysis could allow earlier diagnosis to enable provision of intervention therapies sooner for the child. It may also function as an additional tool for children presenting ambiguous symptoms, with reference here to the control participant with some autistic symptoms and a gait score of 'High Variability'.

Investigations into the reasons behind the differential temporal variability within the High Functioning Autism group would be useful to establish different neurobiological developmental trajectories. Muller et al. (2001) found using fMRI that the typically developing group showed activations in the contralateral central sulcus when instructed to execute finger movements, whereas the participants with autism showed no consistent location of strongest activation. It was concluded that a wide array of compensatory mechanisms are likely to show in the neurobiology of individuals with autism. There appeared to be no diagnostic, symptomatic, intellectual, language-related or clinical characteristics (as assessed by ADOS and clinical information) fitting the reasons for the wide range of gait variability, but the difference within the group was pronounced.

Further research is required to assess the neural functions implicated in greater temporal variability in the Left Step observed in the present research, both in those below the age of 12 and in participants with Asperger's. Exploring these could establish distinguishing methods of gait analysis, according to neurological differences between the diagnoses of Asperger's or High Functioning Autism. Cautious extrapolation of previous research conclusions suggest the left cerebellar hemisphere (McAlonan et al., 2002) and right putamen (Yu et al., 2011) could be starting points for future research into greater Left Step temporal variability in Asperger's. The Left Step disparity according to age cohort is another factor to consider, with developmental delays in hemispheric dominance posited as an explanation.

Temporal synchrony in autism does not follow a typical developmental trajectory due to fundamental neurobiological timing differences in infants with autism in neonates (Feldman, 2007), potentially linked to clock gene anomalies (Wimpory et al., 2002). Without these early biological rhythms, pre-verbal interactions with caregivers are out of synchrony and their ability to engage in a social turn-taking rhythm is reduced. This has an influence upon the neurological cascade of development, particularly in the cerebellum – vital for motor control. Gait temporal variability links with theories pertaining to the trajectory of Clock genes (Nguyen et al., 2010; Messer & Kang, 2000) leading to anomalous Purkinje cell development (Palmen et al., 2004), impacting the neurobiology of the cerebellum, thus exacerbating the autistic phenotype and motor coordination. The temporal variability in Step, Stance and Swing in all participants (High Functioning Autism and Asperger's) is attributable to cerebellar abnormality.

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Appendices

Appendix A: Invitation Letters for Participants with High Functioning Autism

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Invitation to participate in Gait Analysis study – Parent Version</u>

Dear _____

We are conducting a study into whether the technique of Gait Analysis (the way a person walks) can be used to assist diagnosis of autism and we would like to invite
_____ to participate.

The purpose of this study is to investigate Gait Analysis as an effective diagnostic technique by using it to analyse the different walking styles of people of different ages and varying degrees of autism in the hope of identifying an 'Autistic Gait' – i.e. a specific way that a person with autism walks.

This research study will be conducted by a mixed team of clinicians and researchers from both Bangor University and the BCUHB (Betsi Cadwaladr University Health Board). This project will be overseen by Consultant Clinical Psychologist for Autism Dr Dawn Wimpory. This research study is split into two parts, the first part lasting about an hour and a half and the second part just an hour. The first part of this study takes place in the Bangor University Sports Science Department where biological motion analysis with specialist criteria and equipment will be used to measure the way people walk around the room. During this part of the study your son will have small reflective balls fastened to their skin. These are not painful and take around 5 minutes on average to put on in the correct places. Secondly, on a separate occasion, we may conduct further tests by asking your son to simply walk up and down on a series of floor plates that measures the force of their steps in the Bangor Child Development Centre, although it is looking unlikely at the moment that we will need to ask you to return to the second venue.

Additional information on this Sports Science equipment is included in the other information sheet, with pictures of the laboratory used. It is very interesting technology and may be of interest to your son; the system with the reflective balls is similar to equipment used for creating the cartoon-like CGI animations using human actors in films i.e. Avatar!

As _____ is under the age of 18, we request that a parent/guardian attends the sessions.

Any information collected during this study will be kept confidentially and stored securely by the Consultant Clinical Psychologist overseeing this study.

Enclosed are two information sheets (the one with pictures may be most suitable for a younger participant to look at), however if you still have unanswered questions please feel free to contact a member of the research team at <u>kitty_forster@hotmail.com</u> or leave a message on 01248 382514. A member of the NHS team will contact you to discuss the

project in more detail and is willing to come out to your home if you want.

You are not obliged to take part, and declining participation will not affect your right to treatment or specialist services.

Yours faithfully,

Research Team:

01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com

GIG CYMRU NHS WALES
Bwrdd Iechyd Prifysgol
Betsi Cadwaladr
University Health Board

PRIFYSGOL BANGOR UNIVERSITY
1884

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Invitation to participate in Gait Analysis study – Adult Participant version</u>

Dear _____

We are conducting a study into whether the technique of Gait Analysis (the way a person walks) can be used to assist diagnosis of autism and we would like to invite you to participate.

The purpose of this study is to investigate Gait Analysis as an effective diagnostic technique by using it to analyse the different walking styles of people of different ages and varying degrees of autism in the hope of identifying an 'Autistic Gait' – i.e. a specific way that a person with autism walks.

This research study will be conducted by a mixed team of clinicians and researchers from both Bangor University and the BCUHB (Betsi Cadwaladr University Health Board). This project will be overseen by Consultant Clinical Psychologist for Autism Dr Dawn Wimpory.

This research study takes place in the Bangor University Sports Science Department where biological motion analysis with specialist criteria and equipment will be used to measure the way people walk around the room. During this part of the study you will have small reflective balls fastened to your skin. These are not painful and take around 5 minutes on average to put on in the correct places.

Additional information on this equipment is included in the other information sheet, with pictures of the laboratory used. It is very interesting technology and may be of interest to you; the system with the reflective balls is similar to equipment used for creating the cartoon-like CGI animations using human actors in films i.e. Avatar!

Any information collected during this study will be kept confidentially and stored securely by the Consultant Clinical Psychologist overseeing this study.

Enclosed are two information sheets (one contains pictures for an easy way to get an idea of what to expect), however if you still have unanswered questions please feel free to contact a member of the research team at kitty_forster@hotmail.com or leave a message on 01248 382514. A member of the NHS team will contact you to discuss the project in more detail and will offer to come out to your home if you want.

You are not obliged to take part, and declining participation will not affect your right to treatment or specialist services.

Yours faithfully,
Research Team:
01248 382514
Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com

Appendix B: Initial Page of Invitation Letter – 'Other Clinician'.

Insert header of Clinician's department logo}

{Insert address of Clinician}
<u>   Invitation to participate in Gait Analysis study</u>
Dear _____{Insert name of parent}_____,
I am writing to inform you about a research study coming up led by Dr Dawn Wimpory
(Consultant Clinical Psychologist for Autism, NHS BCUHB). The study is about the way
people with autism walk and would involve going to a room in Bangor University to
participate in gait analysis (more details enclosed).   Would you be happy for me to share
your contact details with the research team?
 Please fill in the below slip of paper and return it to me using the stamped self addressed
envelope I have included. Then when a member of the research team contacts me I will pass
on your contact details if you have given assent for this. This will allow a member of the
research team to contact you to ask about your decision to participate. If you decide from
reading the other information provided that you would like to contact them directly, then you
can email or telephone members of the research team to get in touch faster without having to
send this form back to me. Contact details of the research team are at the end of the enclosed
invitation letter and information sheet. Please be very aware that your son's right to
treatment or support from us is **not** affected by your decision to participate or not participate
in any way.

Yours faithfully

{Insert name of clinician here}

Your
Name:_____

Please tick this box if you are not interested in being contacted by any research teams:

Please tick this box if you are happy to be contacted:

Signature _____
Date_____

Appendix C: Invitation Letter for Typically Developing

Children

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514   Invitation Letter

We are conducting a study into whether the technique of Gait Analysis (the way a person walks) can be used to assist diagnosis of autism and we would like to invite your child to participate as a typically developing child, so we have data to compare typical walking patterns with how children with autism walk.

The purpose of this study is to investigate Gait Analysis as an effective diagnostic technique by using it to analyse people of different ages and varying degrees of autism in the hope of identifying an 'Autistic Gait' – i.e. a specific way that a person with autism walks.

This research study will be conducted by a mixed team of clinicians and researchers from both Bangor University and the BCUHB (Betsi Cadwaladr University Health Board). This project will be overseen by Consultant Clinical Psychologist for Autism Dr Dawn Wimpory.

This research study is split into two parts, the first part lasting about an hour and a half and the second part just an hour. The first part of this study takes place in the Bangor University Sports Science Department where biological motion analysis with specialist criteria and equipment will be used to measure the way children walk around the room. During this part of the study your son will have small reflective balls fastened to their skin. These are not painful and take around 5 minutes on average to put on in the correct places. Secondly we may conduct further tests by asking your son to simply walk up and down on a series of floor plates that measures the force of their steps in the Child Development Centre in Bangor, although it is looking unlikely at the moment that we will need to ask you to return to the second venue.

Additional information on this Sports Science equipment is included in the other information sheet, with pictures of the laboratory used. It is very interesting technology and may be of interest to your son; the system with the reflective balls is similar to equipment used for creating the cartoon-like CGI animations using human actors in films i.e. Avatar!

We request that a parent/guardian attends the sessions to lend support and sign the consent form. In the room there will be two or three researchers plus your son and his parent/guardian.

Any data collected during this study will be kept confidentially and stored securely by the Consultant Clinical Psychologist overseeing this study.

Enclosed is a participant information sheet, however if you still have unanswered questions please feel free to contact a member of the research team at kitty_forster@hotmail.com or leave a message on 01248 382514. A member of the NHS team will contact you to discuss the project in more detail if you provide assent for your contact details to be passed on to our research team by the school.

You are not obliged to take part, and declining participation will not affect your right to treatment or specialist services. Participants will be paid £5 and travel costs reimbursed.

Yours faithfully,

Research Team:

01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student kitty_forster@hotmail.com

Appendix D: Consent Forms

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

CONSENT FORM – Adult Participants
Title of Project: Gait Analysis in Autism: A Potential Diagnostic Tool
Name of Researcher: Miss Kitty Forster

Please initial box

1. I confirm that I have read and understood the information sheet
    dated 20/12/2011 (version 3) for the above study and have
    had the opportunity to ask questions.

2. I understand that my participation is voluntary and
    that I am free to withdraw at any time, without giving any
    reason, without any medical care or legal rights being affected.

3. I understand that sections of any of my medical notes may
    be looked at by responsible individuals employed by BCUHB,
    where it is relevant to their taking part in research i.e. any autism-related
    or other relevant assessments. I give permission for these individuals
    to have access to my records.

4. I agree to a letter being sent out to my GP, to inform them
    of my participation in this research. No confidential information will
    be sought.

5. Please would you supply a phone number for us to contact you to arrange an
    appointment. Thank you. Number:

    _____

If you do not wish to be contacted for future research opportunities, please tick this box
This will not affect your access and right to treatment.

_____   _____   _____
Name of Participant   Date   Signature.

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

CONSENT FORM – Parent of Participant
Title of Project: Gait Analysis in Autism: A Potential Diagnostic Tool
Name of Researcher: Miss Kitty Forster

Please initial box

1. I confirm that I have read and understood the information sheet
   dated 20/12/2011 (version 3) for the above study and have
   had the opportunity to ask questions.

2. I understand that my son's participation is voluntary and
   that he is free to withdraw at any time, without giving any
   reason, without any medical care or legal rights being affected.

3. I understand that sections of any of my son's medical notes may
   be looked at by responsible individuals employed by BCUHB,
   where it is relevant to their taking part in research i.e. any autism-related
   or other relevant assessments. I give permission for these individuals
    to have access to my son's records.

4. I agree to a letter being sent out to my son's GP, to inform them
    of my participation in this research. No confidential information will
    be sought.

5. Please would you supply a phone number for us to contact you to arrange an
    appointment. Thank you. Number:
    _____

If you do not wish to be contacted for future research opportunities, please tick this box.
This will not affect your access and right to treatment.

_____   _____   _____
Name of Participant   Date   Signature.

Appendix E: Consultee Declaration Form

Title of Project: Gait Analysis in Autism: A Potential Diagnostic Tool

Name of Researcher: Miss Kitty Forster, Dr Dawn Wimpory and Professor Robert Ward

Please initial box

I, …………………………….have been consulted about ………………………………..
  (name of consultee)   *(name of potential participant)*
participating in this research project. I have had the opportunity to ask questions
about the study and I understand what is involved.

In my opinion he would have no objection to taking part in the above study

I understand that I can request he is withdrawn from the study at any time,
without giving any reason and without his care or legal rights being affected.

I agree to him being filmed during their ADOS assessment (if they do

not already have one) to assist in coding their assessment at a later date

I agree that their data will be kept confidentially unless information is

disclosed regarding the safety of a child or vulnerable adult in which case

researchers will report this information to the relevant authority.

I understand that their GP will be informed of their participation

Name of Consultee _____

Relationship to participant: _____

Date: _____   **Signature** _____

Researcher _____

Date _____   **Signature** _____

Appendix F: Information Sheets (Parent of HFA, Adult HFA,

Consultee, TD versions)

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Parent Information Sheet for Participants under 18</u>
GAIT ANALYSIS IN AUTISM: A POTENTIAL DIAGNOSTIC TOOL?
<u>Invitation</u>
Your son is being invited to take part in a research project. Before you decide we would like you to understand why the research is being done and what it will involve. Please take time to read this information carefully and discuss it with others if you wish. Ask us if there is anything you're not clear on or if you would like more information. Take time to decide whether or not you would like to take part. Thank you for reading this.
<u>What is the purpose of the study?</u>
The purpose of this research study is to find out whether 116ehaviour the individual way someone walks ("gait") can be a good way to help diagnose autism at an early age. We will also investigate whether there is a relationship between the degree of autism diagnosis and the type of gait.
<u>Why have I been chosen?</u>
Your family has been contacted as you have a son with autism (who may now be an adult) who fits the correct inclusion criteria (aged 7-30, male, diagnosis of High Functioning Autism) necessary for this study. We aim to recruit around sixteen willing participants.
<u>Do I have to take part?</u>
It is entirely your decision as to whether or not to allow your son to take part. Refusal to take part will not result in any penalty or loss of benefits or service to which you are entitled. Your son may withdraw from the experiment at any time, without any need to provide a reason. If you do decide to consent for your son to take part in this research you will be asked to keep these information sheets and to sign a consent form. Please find a consent form attached, which you can bring with you to the session or post back to us. We have spare consent forms on site should you forget to bring it. A legal custodial guardian is the appropriate person to sign this form on behalf of a child. There is no need to be concerned about the costs of transport to the Bangor facilities, as you will be reimbursed for public transport/petrol costs. Your son will be paid £10 for participating.
<u>The design of the study: What will happen to my son if taking part?</u>
The first part of the experiment will take place in the Bangor University Sports Science Department in a new state of the art Gait Analysis room and can take up to 2 hours. In this stage your son will have tiny light reflecting balls attached to the skin with double sided sticky tape. In order for the markers to be visible, your child will have to wear some shorts rolled down enough to expose the lower back, hip bones (just below naval) and short enough

to show the thighs. A t-shirt may be worn as long as the hem is short enough to expose a bit of the hipbone area (not a really long t-shirt). It would be good if you could bring these with you please, but we can provide some if you forget. There is an area to change clothing in privacy. Your son will then be asked to walk around in the large room wearing these balls and the light that reflects from the balls is picked up by a start of the art computer programme via 12 cameras on the walls. It is called a 3D Vicon Motion Capture System with accompanying Vicon Nexus Computer Software. It is used for gait analysis, sports science and also computer graphics i.e. computer games and CGI animations. This will give us information about the particular way they move and the rhythm of their steps. This stage of the study will also measure whether your son has even or uneven strides when walking as people with autism are often found to have irregular stride lengths. The entire procedure is totally harmless but if you or your son feels distress at any point the experiment will be stopped immediately.

After this, we will look at the data and decide which people have the sort of walk we are looking for. If they do, then they may be asked to come back and participate in the second part of the research.

If the preliminary glance at the data suggests we need a different type of measure as well, a second part of this study will take place in the NHS (BCUHB) Child Development Centre on Holyhead Road, Bangor and will take about an hour. In this part of the research study your son would be asked to walk on a 'gait pathway'. This is a series of mats that contain a 'force plate' which measures the pressure caused by different parts of a foot when walking. From this we can work out if the foot is placed down in a particular way. A common example of this is that people with autism often put a lot of pressure on their toes in a gait known as 'toe walking'.

After the practical elements of the research are completed the stick figure computerised videos of your child's gait will be analysed using advanced computer software to attempt to identify any walking patterns.

We will try to accommodate your time schedules when arranging a suitable session to attend the gait laboratory. Weekends and evenings are negotiable if week days are not convenient for you.

It is likely that your son will have an existing ADOS (Autism Diagnostic Observation Schedule) assessment conducted by their treating clinician. A staff member employed by Betsi Cadwaladr University Health Board will look at your child's medical records to establish the existence of this and other relevant assessments. However, if no ADOS assessment has been done, you will be asked to attend a subsequent session which will last approximately an hour at the Child Development Centre.   Again, travel costs will be reimbursed. Alternatively, the ADOS administrator could be requested to visit your home if you think your son would be more comfortable there. The ADOS assessment involves a series of interactive tasks and games with an ADOS administrator, for example reading a book together, playing with figures etc.

What are the possible risks and benefits of taking part?

Although there are no identifiable risks in this study everyone's reaction to new surroundings and people will be different therefore it is possible that your son could show signs of distress. If this happens we will simply stop the experiment immediately and you are under no obligation to have any further involvement with the research.

The benefit of this study is that by helping us improve our diagnostic methods you may know that you are improving the accuracy of Autistic Spectrum Disorder diagnosis and in the long run making life significantly better for families of children with ASD by making future diagnosis more conclusive.

What will happen if I withdraw from the study?

If you withdraw from the study there will be no penalty and you are under no obligation to continue with the research.

Whilst no factors have been identified that could go wrong, if anything happens that you feel affects you/your child in a negative way then the experiment will be stopped immediately. If you question the conduct of experimenters in this research or want to make a complaint you may contact the School Manager, Hefin Francis (01248 388339 or h.francis@bangor.ac.uk). The Head of the School of Psychology at Bangor University is Dr Charles Leek (01248382948).

Will my taking part in this study be kept confidential?

Only the research team will have access to any personal data; it will be kept in a locked cabinet, which Dr Wimpory has the key to.

What will happen to the results of this study?

Once the research study is complete these results will very likely be published in a research journal. Again all data will be totally confidential.

Who is organising and funding this research?

Bangor University and Betsi Cadwaladr University Health Board. Financial funding from KESS (*Knowledge Economy Skills Scholarships).*

Who has reviewed this study?

Scientists from the Psychology Department at Bangor University

Contact for further information

Kitty Forster at kitty_forster@hotmail.com or leave a message on 01248 382514. A member of the NHS team, Ms Oonagh Eason (NHS Registered Nurse Learning Disability) will contact you to discuss the project in more detail and is willing to come out to your home if you have any other queries.

Research Team:

01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com

Thank you

**Bwrdd Iechyd Prifysgol
Betsi Cadwaladr
University Health Board**

**PRIFYSGOL
BANGOR
UNIVERSITY**

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Participant Information Sheet for Participants over 18</u>
GAIT ANALYSIS IN AUTISM: A POTENTIAL DIAGNOSTIC TOOL?
<u>Invitation</u>
You are being invited to take part in a research project. Before you decide we would like you
to understand why the research is being done and what it will involve. Please take time to
read this information carefully and discuss it with others if you wish. Ask us if there is
anything you're not clear on or if you would like more information. Take time to decide
whether or not you would like to take part. Thank you for reading this.
<u>What is the purpose of the study?</u>
The purpose of this research study is to find out whether 119ehaviour the individual way
someone walks ("gait") can be a good way to help diagnose autism at an early age. We will
also investigate whether there is a relationship between the degree of autism diagnosis and
the type of gait.
<u>Why have I been chosen?</u>
You have been contacted because you fit the inclusion criteria (aged 7-30, male, diagnosis of
High Functioning Autism) necessary for this study. We aim to recruit around sixteen willing
participants.
<u>Do I have to take part?</u>
It is entirely your decision as to whether or not to take part. Refusal to take part will not result
in any penalty or loss of benefits or service to which you are entitled. You may withdraw
from the experiment at any time, without any need to provide a reason. If you do decide to
take part in this research you will be asked to keep these information sheets and to sign a
consent form. Please find a consent form attached, which you can bring with you to the
session or post back to us. We have spare consent forms on site should you forget to bring it.
There is no need to be concerned about the costs of transport to the Bangor facilities, as you
will be reimbursed for public transport/petrol costs. You will be paid £10 for participating.
<u>The design of the study: What will happen to me/my child if taking part?</u>
The first part of the experiment will take place in the Bangor University Sports Science
Department in a new state of the art Gait Analysis room and can take up to 2 hours. In this
stage you will have tiny light reflecting balls attached to the skin with double sided sticky
tape. In order for the markers to be visible, you will have to wear some shorts rolled down
enough to expose the lower back, hip bones (just below naval) and short enough to show the
thighs. A t-shirt may be worn as long as the hem is short enough to expose a bit of the
hipbone area (not a really long t-shirt). It would be good if you could bring these with you
please, but we can provide some if you forget. There is an area to change clothing in privacy.
You will then be asked to walk around in the large room wearing these balls and the light that
reflects from the balls is picked up by a start of the art computer programme via 12 cameras
on the walls. It is called a 3D Vicon Motion Capture System with accompanying Vicon

Nexus Computer Software. It is used for gait analysis, sports science and also computer graphics i.e. computer games and CGI animations. This will give us information about the particular way you move and the rhythm of your steps. This stage of the study will also measure whether you have even or uneven strides when walking as people with autism are often found to have irregular stride lengths. The entire procedure is totally harmless but if you feel distress at any point the experiment will be stopped immediately.

After the practical elements of the research are completed the stick figure computerized videos of your gait will be analysed using advanced computer software to attempt to identify any walking patterns.   We will try to accommodate your time schedules when arranging a suitable session to attend the gait laboratory. Weekends and evenings may be negotiable if week days are not convenient for you.

It is likely that you will have an existing ADOS (Autism Diagnostic Observation Schedule) assessment conducted by your treating clinician. A staff member employed by Betsi Cadwaladr University Health Board will look at your medical records to establish the existence of this and other relevant assessments. However, if no ADOS assessment has been done, you will be asked to attend a subsequent session which will last approximately an hour, travel costs will be reimbursed. Alternatively, the ADOS administrator could be requested to visit your home if you think you would be more comfortable there. The ADOS assessment involves a series of interactive tasks and games and topics of conversation with an ADOS administrator.

What are the possible risks and benefits of taking part?

Although there are no identifiable risks in this study everyone's reaction to new surroundings and people will be different therefore it is possible that you may feel discomfort. If this happens we will simply stop the experiment immediately at your request and you are under no obligation to have any further involvement with the research.

The benefit of this study is that by helping us improve our diagnostic methods you may know that you are improving the accuracy of Autistic Spectrum Disorder diagnosis and in the long run making life significantly better for families of children with ASD by making future diagnosis more conclusive.

What will happen if I withdraw from the study?

If you withdraw from the study there will be no penalty and you are under no obligation to continue with the research.

What if something goes wrong?

Whilst no factors have been identified that could go wrong, if anything happens that you feel affects you in a negative way then the experiment will be stopped immediately. If you question the conduct of experimenters in this research or want to make a complaint you may contact the School Manager, Hefin Francis (01248 388339 or h.francis@bangor.ac.uk). The Head of the School of Psychology at Bangor University is Dr Charles Leek (01248382948).

Will my taking part in this study be kept confidential?

Only the research team will have access to any personal data; it will be kept in a locked cabinet, which Dr Wimpory has the key to.

What will happen to the results of this study?

Once the research study is complete these results will very likely be published in a research journal. Again all data will be totally confidential.

Who is organising and funding this research?

Bangor University and Betsi Cadwaladr University Health Board. Financial funding from KESS (***Knowledge Economy Skills Scholarships).***

Who has reviewed this study?

Scientists from the Psychology Department at Bangor University

Contact for further information

Kitty Forster at kitty_forster@hotmail.com or leave a message on 01248 382514. A member of the NHS team, Ms Oonagh Eason (NHS Registered Nurse Learning Disability) will contact you to discuss the project in more detail and will offer to come out to your home if you have any other queries.

Research Team:

01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com

Thank you

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Parent Information Sheet for typically developing children</u>
GAIT ANALYSIS IN AUTISM: A POTENTIAL DIAGNOSTIC TOOL?
<u>Invitation</u>
Your son is being invited to take part in a research project. Before you decide we would like you to understand why the research is being done and what it will involve. Please take time to read this information carefully and discuss it with others if you wish. Ask us if there is anything you're not clear on or if you would like more information. Take time to decide whether or not you would like to take part. Thank you for reading this.
<u>What is the purpose of the study?</u>
The purpose of this research study is to find out whether 122ehaviour the individual way someone walks ("gait") can be a good way to help diagnose autism at an early age. We will also investigate whether there is a relationship between the degree of autism diagnosis and the type of gait.
<u>Why have I been chosen?</u>
Your family has been contacted because you have a typically developing son between the ages of 7-18. Their walking style will be used as a normal walk to compare with the walk of a child with autism. This will help us decide whether 122ehaviour the way people walk could help diagnose people with autism earlier on and thus use this method to help provide interventions and treatments earlier on.
<u>Do I have to take part?</u>
It is entirely your decision as to whether or not to take part. Refusal to take part will not result in any penalty or loss of benefits or service to which you are entitled. You may withdraw from the experiment at any time, without any need to provide a reason. If you do decide to take part in this research you will be asked to keep these information sheets and to sign a consent form. Please find a consent form attached, which you can bring with you to the session or post back to us. We have spare consent forms on site should you forget to bring it. A legal custodial guardian is the appropriate person to sign this form on behalf of a person under the age of 18. There is no need to be concerned about the costs of transport to the Bangor facilities, as you will be reimbursed for public transport/petrol costs. Your child will be paid £5 for participating.
<u>The design of the study: What will happen my child if taking part?</u>
The first part of the experiment will take place in the Bangor University Sports Science Department in a new state of the art Gait Analysis room and can take up to 2 hours. In this stage your child will have tiny light reflecting balls attached to their skin with double sided sticky tape. In order for the markers to be visible, your child will have to wear some shorts rolled down enough to expose the lower back, hip bones (just below naval) and short enough to show the thighs. A t-shirt may be worn as long as the hem is short enough to expose a bit of the hipbone area (not a really long t-shirt). It would be good if you could bring these with

you please, but we can provide some if you forget. There is an area to change clothing in privacy. Your child will then be asked to walk around in the large room wearing these balls and the light that reflects from the balls is picked up by a start of the art computer programme via 12 cameras on the walls. It is called a 3D Vicon Motion Capture System with accompanying Vicon Nexus Computer Software. It is used for gait analysis, sports science and also computer graphics i.e. computer games and CGI animations. This will give us information about the particular way your child moves and the rhythm of their steps. This stage of the study will also measure whether your child has even or uneven strides when walking as people with autism are often found to have irregular stride lengths. The entire procedure is totally harmless but if you or your child feels distress at any point the experiment will be stopped immediately.

After the practical elements of the research are completed the stick figure computerised videos of your son's gait will be analysed using advanced computer software to attempt to identify any gait markers.

After this, we will look at the data and decide if more is required; if so, your child may be asked to come back and participate in the second part of the research.

If the preliminary glance at the data suggests we need a different type of measure as well, a second part of this study will take place in the NHS (BCUHB) Child Development Centre on Holyhead Road, Bangor and will take about an hour. In this part of the research study your child would be asked to walk on a 'gait pathway'. This is a series of mats that contain a 'force plate' which measures the pressure caused by different parts of a foot when walking. From this we can work out if the foot is placed down in a particular way. A common example of this is that people with autism often put a lot of pressure on their toes in a gait known as 'toe walking'. However, it is very unlikely that anyone will need to come to this session.

We will try to accommodate your time schedules when arranging a suitable session to attend the gait laboratory. Weekends and evenings could be negotiated if week days are not convenient for you.

What are the possible risks and benefits of taking part?

Although there are no identifiable risks in this study everyone's reaction to new surroundings and people will be different therefore it is possible that your child could show signs of distress. If this happens we will simply stop the experiment immediately and you are under no obligation to have any further involvement with the research.

The benefit of this study is that by helping us improve our diagnostic methods you may know that you are improving the accuracy of Autistic Spectrum Disorder diagnosis and in the long run making life significantly better for families of children with ASD by making future diagnosis more conclusive.

What will happen if I withdraw from the study?

If you withdraw from the study there will be no penalty and you are under no obligation to continue with the research.

What if something goes wrong?

Whilst no factors have been identified that could go wrong, if anything happens that you feel affects you/your child in a negative way then the experiment will be stopped immediately. If you question the conduct of experimenters in this research or want to make a complaint you may contact the School Manager, Hefin Francis (01248 388339 or h.francis@bangor.ac.uk). The Head of the School of Psychology at Bangor University is Dr Charles Leek (01248382948).

Will my taking part in this study be kept confidential?

Only the research team will have access to any personal data and will be kept in a locked cabinet, which Dr Wimpory has the key to.

What will happen to the results of this study?

Once the research study is complete these results will very likely be published in a research journal. Again all data will be totally confidential.

Who is organising and funding this research?
Bangor University and Betsi Cadwaladr University Health Board. Financial funding from KESS (*Knowledge Economy Skills Scholarships*).

Who has reviewed this study?
Scientists from the Psychology Department at Bangor University.

Contact for further information
Kitty Forster at pspc12@bangor.ac.uk or leave a message on 01248 382514. A member of the NHS team will contact you to discuss the project in more detail.

Research Team:

01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com
Thank you

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Consultee Information Sheet –</u>
<u>For parent of participant over 18 without capacity for consent</u>
GAIT ANALYSIS IN AUTISM: A POTENTIAL DIAGNOSTIC TOOL?

<u>Invitation</u>
Your son is being invited to take part in a research project. Before you decide we would like you to understand why the research is being done and what it will involve. Please take time to read this information carefully and discuss it with others if you wish. Ask us if there is anything you're not clear on or if you would like more information. Take time to decide whether or not you would like to take part. Thank you for reading this.

<u>What is the purpose of the study?</u>
The purpose of this research study is to find out whether 125ehaviour the individual way someone walks ("gait") can be a good way to help diagnose autism at an early age. We will also investigate whether there is a relationship between the degree of autism diagnosis and the type of gait.

<u>Why have I been chosen?</u>
Your family has been contacted as you have an adult son with autism who fits the correct inclusion criteria (aged 18-30, male, diagnosis of High Functioning Autism) necessary for this study. We aim to recruit around sixteen willing participants. You have been given this sheet because, after assessment by Ms Oonagh Eason, it was thought best that you provide consent on behalf of your son and decide whether or not he understands what the procedure entails.

<u>Do I have to take part?</u>
It is entirely your decision as to whether or not to take part. Refusal to take part will not result in any penalty or loss of benefits or service to which you are entitled. You may withdraw from the experiment at any time, without any need to provide a reason. If you do decide to take part in this research you will be asked to keep these information sheets and to sign a consent form. Please find a consent form attached, which you can bring with you to the session or post back to us. We have spare consent forms on site should you forget to bring it. A legal custodial guardian is the appropriate person to sign this form on behalf of your son. There is no need to be concerned about the costs of transport to the Bangor facilities, as you will be reimbursed for public transport/petrol costs. Your son will be paid £10 for participating.

<u>The design of the study: What will happen to my son if taking part?</u>
The first part of the experiment will take place in the Bangor University Sports Science Department in a new state-of-the-art Gait Analysis room and can take up to 2 hours. In this stage your son will have tiny light reflecting balls attached to his skin with double sided sticky tape. In order for the markers to be visible, he will have to wear some shorts rolled down enough to expose the lower back, hip bones (just below naval) and short enough to

show the thighs. A t-shirt may be worn as long as the hem is short enough to expose a bit of the hipbone area (not a really long t-shirt). It would be good if you could bring these with you please, but we can provide some if you forget. There is an area to change clothing in privacy. Your son will then be asked to walk around in the large room wearing these balls and the light that reflects from the balls is picked up by a start of the art computer programme via 12 cameras on the walls. It is called a 3D Vicon Motion Capture System with accompanying Vicon Nexus Computer Software. It is used for gait analysis, sports science and also computer graphics i.e. computer games and CGI animations. This will give us information about the particular way your son moves and the rhythm of their steps. This stage of the study will also measure whether your son has even or uneven strides when walking as people with autism are often found to have irregular stride lengths. The entire procedure is totally harmless but if you or your son feels distress at any point the experiment will be stopped immediately.

After the practical elements of the research are completed the videos of your son's gait will be analysed using advanced computer software to attempt to identify any gait markers.

We will try to accommodate your time schedules when arranging a suitable session to attend the gait laboratory. Weekends and evenings are negotiable if week days are not convenient for you.

It is likely that your son will have an existing ADOS (Autism Diagnostic Observation Schedule) assessment conducted by their treating clinician. A staff member employed by Betsi Cadwaladr University Health Board will look at your son's medical records to establish the existence of this and other relevant assessments. However, if no ADOS assessment has been done, you will be asked to attend a subsequent session.   Again, travel costs will be reimbursed. Alternatively, the ADOS administrator could be requested to visit your home if you think your son would be more comfortable there. The ADOS assessment involves a series of interactive tasks and games and conversational topics with an ADOS administrator.

<u>What are the possible risks and benefits of taking part?</u>

Although there are no identifiable risks in this study everyone's reaction to new surroundings and people will be different therefore it is possible that your son could show signs of distress. If this happens we will simply stop the experiment immediately and you are under no obligation to have any further involvement with the research.

The benefit of this study is that by helping us improve our diagnostic methods you may know that you are improving the accuracy of Autistic Spectrum Disorder diagnosis and in the long run making life significantly better for families of children with ASD by making future diagnosis more conclusive.

<u>What will happen if I withdraw from the study?</u>

If you withdraw from the study there will be no penalty and you are under no obligation to continue with the research.

<u>What if something goes wrong?</u>

Whilst no factors have been identified that could go wrong, if anything happens that you feel affects your son in a negative way then the experiment will be stopped immediately. If you question the conduct of experimenters in this research or want to make a complaint you may contact the School Manager, Hefin Francis (01248 388339 or h.francis@bangor.ac.uk). The Head of the School of Psychology at Bangor University is Dr Charles Leek (01248382948).

<u>Will my taking part in this study be kept confidential?</u>

Only the research team will have access to any personal data; it will be kept in a locked cabinet, which Dr Wimpory has the key to.

<u>What will happen to the results of this study?</u>

Once the research study is complete these results will very likely be published in a research journal. Again all data will be totally confidential.

Contact for further information
Kitty Forster at kitty_forster@hotmail.com or Ms Oonagh Eason (NHS Registered Nurse
Learning Disability). You can leave a message on 01248 382514.

Research Team:
01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com
Thank you

Appendix G: Easy-Read Information Sheet

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Easy-read Information Sheet</u>

<u>Finding out about how people walk, called Gait Analysis Research</u>

*   These pages will to tell you about Gait Analysis or Finding Out About Walking.

*   We hope what is written here will help you to decide if you want to come and walk
    for us or not.

*   They will also tell you about what will happen when you come if you say yes.

*   There is a telephone number and a list of our names at the bottom of these pages,
    please call us if you have questions or would like us to tell you more.

We could come to meet you at home to talk about it if you like

*   Everybody has their own special way of walking.

We are asking you to help us so that we can learn more about it.

We want to find out if people who have autism all walk in the same kind of way. And if
knowing about this can help us to help them when they are very young.
To help us, you would need to come to Bangor and do some walking in a big room with
cameras all around. Here are some pictures of the room.

Sports Science Gait Laboratory.

If you say yes, we will send you a letter. This will tell you the day and the time we would like you to come.

We want you to wear or bring a pair of shorts with you please. They need to be quite short on your legs so we can put markers on the right places.
Your height and weight will be measured.
You will walk in bare feet around the room, so it doesn't matter what shoes you arrive in.
If you agree to come, we will send you personalized directions from Google maps along with your appointment time.
There are a few carparks around Normal Site. You can park here free of charge.
If you are coming by bus, let us know and we will send you bus route information from where you are travelling from.

This is a close up of the building that is circled above.

Go to the second ramp

Kitty Forster and Robin Kramer outside of the building.

The building itself looks like this.   Once inside you will meet –
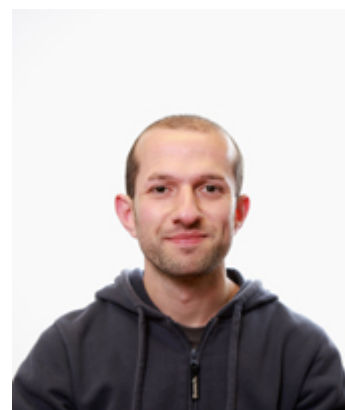
Kitty Forster **Dr Dawn Wimpory** **Robin Kramer**
Oonagh Eason
Mres Student **Clinical Psychologist** **PhD Student** **NHS**
nurse

We will go into this room
We will show you where to find things you might need like toilets and where to change into your shorts.

We will talk with you about what things we would like you to do.
Then we will ask you to go and put your shorts on.

We will talk to you and the person you have with you about you being happy to take part. If you say yes, we will ask you / your parent or guardian to sign forms saying you agree

Then Kitty will stick special sticky shiny balls
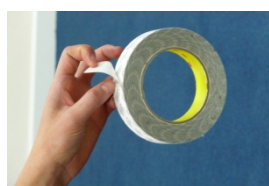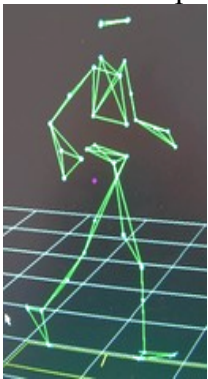
on your legs and hips and toes.

When they are all stuck on you will look a little bit like this

When we show you your picture on the special computer we're using

you will look a bit like this

Next we will ask you to walk around the big room.
The green mat will not be there on the day you come.

We would like you to do this how you always walk. The same way you would when you walk at home, in school, or to the shops and other places.
It is important that we know the way you <u>always</u> walk when you're not thinking about it.
While you are walking the special cameras and computer machines you saw in the pictures already will be videoing you.
The pictures will end up looking like a film show and not really like you – picture examples……..
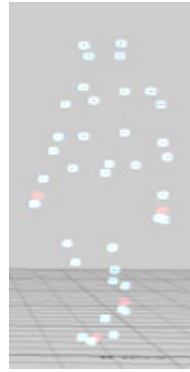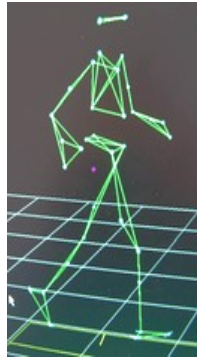
      After about one hour we will have finished.

It will be time to look at the videos the computer made of you walking.

 Then change out of your shorts and into your outdoor clothes to go home.

The pictures take a very long time to sort out. We need to get special people who have studied the way people walk to look at them and help us sort them out. When we have learned what we can from your walking, we will write and tell you all about it.
The people studying the pictures of your walking might want to see you walk again. If this

happens, we will write you a letter again and tell you all about it

   In that letter we might ask if you would come and do it all again in the same place. Or, we might ask you to come to a different place in Bangor. Here we would ask you to walk up and
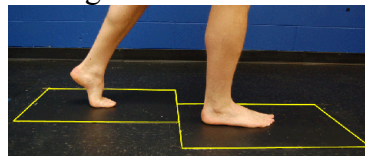
down a special walkway like in the picture   instead of all around the room.
We will tell you more about it if we ask you to come there.

All the pictures of you walking will be locked away   and only the people with special permission will be allowed to look.
When you come to see us we think you will have a lovely time and lots of fun.

If at any time, or for any reason you don't like it, please let us know, and we will stop. You can choose to go home, and that's it, finished. Or you could choose to come another day instead if you like

We hope we have told you all about what Gait Analysis or what Finding Out about Walking is all about, and what will happen if you choose to come and walk for us.
If there is anything else you would like to know about please contact us.

oonagh.eason@wales.nhs.uk

kitty_forster@hotmail.com

The telephone number to call if you would like to talk to somebody about this before you make up your mind is (01248) 382 514

Appendix H: Debrief Form

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Debrief Form: Gait Analysis in Autism: A potential diagnostic tool?</u>

Dear Participant,

Thank you for your participation into this study on Gait Analysis as a potential diagnostic tool for autism. We hope that from this research we can begin to investigate the possibility of using Gait Analysis as a supplementary part of autism diagnosis. If a classifiable 'Autistic Gait' is identified then it is possible that in the future clinicians may be trained on how to identify autism through certain markers in the way children walk. This could potentially make diagnosis of ASD (Autistic Spectrum Disorder) quicker and more accurate in the future. Gait Analysis is an area currently receiving increased research attention and we are positive that our results will make a major contribution to this field of research that couldn't have been achieved without your participation.

If you would like to receive a letter detailing our findings in this study please contact Kitty Forster on kitty_forster@hotmail.com or leave a 'phone message for her on 01248 382514.

If you feel that you/your child has experienced psychological distress in this study or have any comments or questions regarding the conduct of this research or want to make a complaint you may contact the School Manager, Hefin Francis (01248 388339 or h.francis@bangor.ac.uk). The Head of the School of Psychology at Bangor University is Dr Charles Leek (01248382948).

Many thanks for your participation in this study; your contribution has allowed us to analyse the gait of children with autism and ascertain if there are any differences between their walk and the walk of a typically developing child.

Research Team:
01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, NHS BCUHB
Ms Oonagh Eason – NHS Nurse   oonagh.eason@wales.nhs.uk
Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com

_____

If you do not wish to be contacted for future research opportunities, please tick this box.
This will not affect your access and right to treatment.   ☐

Appendix I: Follow-up Letter

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

Thank you for your participation in
'Gait Analysis in Autism: A Potential Diagnostic Tool'

Dear _____

Thank you for coming to the gait analysis session at Bangor University and contributing towards the research 'Gait Analysis in Autism: A Potential Diagnostic Tool.'
The research is now complete and the results were very interesting. We recruited males with High Functioning Autism between the ages of 7 and 35 and were looking into the timing of their walking rhythm. These temporal measures were examining very specific units of time taken to execute Step (time from one foot strike to next foot strike), Swing (time from one toe off to same foot strike) and Stance (time from foot strike to same foot toe off). The VICON Motion Capture system you saw at the gait laboratory (all the cameras around the room) can analyse movements correct to a degree of 250 Hz or 4 milliseconds (or 1/250ths of a second!). This is a much higher temporal resolution than many gait analysis systems used in previous research into the autistic gait. Previous research has often found variability in length of step, but we didn't want to replicate these findings, choosing time instead as the main factor to investigate. Time is interesting in the context of autism for many reasons. Dr Wimpory (supervisor to this research) has researched 'temporal synchrony' in autism, which is a way of describing the rhythm of interaction between two people. The gestures and turn-taking in conversation can be difficult for people with autism. 'Clock genes' are genes that every animal has, which govern their circadian rhythms (sleep/wake cycles). Even in a fruit fly, if these genes are mutated then there is an unusual timing of wing flapping patterns during courtship. In individuals with autism, it has been found that 'clock genes' can be anomalous. This means that when a very young infant with these genes is engaging with a parent/caregiver, the pre-verbal interaction may not possess 'temporal synchrony' because the infant cannot perceive the interaction in the same way as a typically developing infant. Without these pre-verbal reciprocal interactions as a foundation, the neurobiological development in the brain goes through an unusual trajectory. One part of the brain thought to be affected is the cerebellum. This is an 'old' part of the brain, which developed in our ancestors before the higher cognitive parts evolved. The cerebellum is very much involved in motor coordination. The Cerebellar Locomotor Region integrates information to enable coordination of limbs and posture. The cerebellum is important for very specific timing of movement. In autism, structural differences have been found in the cerebellum (post-mortem examination, MRI research etc). In patients with cerebellar ataxia (lesions in the cerebellum), their walking style becomes arrhythmic and uncoordinated. It is thought that the walk of people with autism is not noticeably like this, but at a subtle level shows some signs of not having a perfect rhythm.

This is exactly what we found. Each participant had about 30 examples of 'Step' time (and 30 for Stance and 30 for Swing). On average the time taken didn't differ between people with autism or control participants. However, calculating a value of variability means that a value suggesting the range of time taken is generated.

For example, on a test, someone could score 5, 5, 5, 5 and 5. Their average value would be: $5 + 5 + 5 + 5 + 5 = 25/5 = 5$.

But if someone else scored 3, 4, 6, 2, 10, then their average value would also be 5: $3 + 4 + 6 + 2 + 10 = 25/5 = 5$.

Clearly the two people's scores are very different, because the first is a very consistent score, and the second is on average the same but shows a lot of variability within the range of scores. With the current research it was found that participants with autism demonstrated a significantly more variable time to execute a Step, Stance and Swing but were no slower or faster on average than the control participants. This lack of rhythm is not visible to the eye, but the 250Hz technology can pick up on extremely specific intervals.

We hope that, with further research, this sort of diagnostic tool could be useful to help diagnose young children so interventions could be suggested at a stage in their cognitive and behavioural development when it is most effective. Dr Wimpory has done research into Music Interaction Therapy, and has found that this can help facilitate the sort of 'temporal synchrony' between child and parent that generally occurs as an infant, during preverbal interaction, to provide this important stage into communicative development.

It was also hoped that gait analysis could help diagnose clients on 'the edge' of the autistic spectrum, when their symptoms are not very obvious or clear.

Additionally, a 'Left Step' difference was found in the participants with autism. The variability score was significantly higher in the Left than the Right leg. This has implications for hemispheric specificity. The right hemisphere generally controls the left leg, but with the cerebellum it's more complicated and the effect is generated in the same side of the body. So we haven't understood a possible explanation for this yet, but it would be an area for potential future research.

Please remain assured that all data is anonymised and confidential, in keeping with data protection protocol within the BCUHB (Betsi Cadwaladr University Health Board). This project was overseen by Consultant Clinical Psychologist for Autism Dr Dawn Wimpory.

I hope that you enjoyed your session at the gait laboratory. Thank you once again for contributing your time to help us conduct this research. If any more information is required then do not hesitate to get in touch.

Yours faithfully,

Research Team:

01248 382514

Dr Dawn Wimpory- Consultant Clinical Psychologist for Autism, Lecturer/Practitioner, NHS BCUHB

Miss Kitty Forster- Mres Psychology Student   kitty_forster@hotmail.com

Appendix J: GP Letter

Bangor University
Brigantia Building
Penrallt Road
Bangor
Gwynedd
LL57 2AS
01248 382514

<u>Information for GP of participant</u>

Dear Dr…………………………,
I am writing to inform you that your patient
…………………………………………………………………... has agreed to participate in
research investigating the relationship between gait anomaly and degree of autism. This
research will be lead by Consultant Clinical Psychologist Dr Dawn Wimpory who is also in
charge of selecting participants; either her current/previous clients or those of her clinical
colleagues to whom she offers supervision. Medical records may be consulted by Betsi
Cadwaladr University Health Board employed staff in terms of autism diagnosis and any
other relevant assessments.

There is a very small chance that this experiment may cause frustration and distress, but this
is highly unlikely. All members of the research team have experience working with people
with autism and we ask parent/guardian to stay in the research laboratory with your patient at
all times (if under 18). All participation in research is voluntary and all participants have the
right to refuse to participate or withdraw their participation at any time during the research
project, without penalty.

The first part of this study takes place in the Bangor University Sports Science Department
where biological motion analysis will be used to measure the way in which the person walks.
During this part of the study the participant will have reflective balls fastened to their skin
whilst they walk around a large room (cameras record 4X4 metres of their walking range).
During the second part of the study, the participant simply walks up and down on a series of
floor plates that measures the force of their steps in the Bangor Child Development Centre
though it is unlikely they would have to return to this venue.

We hope that from this research we can begin to investigate the possibility of using Gait
Analysis as a supplementary part of autism diagnosis. If a classifiable 'Autistic Gait' is
identified then it is possible that in the future clinicians may be trained on how to identify
autism through certain markers in the way people walk. This could potentially make
diagnosis of ASD (Autistic Spectrum Disorder) quicker and more accurate in the future.
Please let us know if you have any further queries about the objectives or processes of this
research.

Yours faithfully,

Research Team:

01248 382514

Appendix K: Autism Diagnostic Observation Schedule: Modules 2 and 3

Student Name: _____   Date: _____   Observer: _____

ADOS-Module 2

- In general, the order of tasks and events should be flexible and determined by flow of the interaction.
- Begin by putting student at ease. This should include some indication that the session will involve a variety of different activities or tasks. Need to present student with a list of tasks that we will be completing. Present the list orally. If the student seems anxious and not appearing to be engaged in task, then present the student with a visual chart of task, that then can be checked off as it is completed.
- It is essential to provide an interactive model, commenting on the student's activities or statements and introducing brief observations about the Psych's own interests or activities.
- Overall – note if student directs attention to a different place in the room, specific item, etc.
- Eye contact – if a student doesn't make eye contact, try to do something/make a noise that will catch his attention "Whoa! Look at this!"

1. Construction Task (puzzle and pattern board)

• Sit far apart and place some blocks out of reach • create opportunity for child to ask for help. **Instructions: *"Show me how you would put these blocks together to look like this picture. Let me know if you need more blocks."*** • The remainder of the blocks are placed on the other side of the examiner's arm. • Indicate where they are. • Turn slightly away from the child when initial blocks have been used. • If child does not ask or gesture towards/for the remaining blocks, ask **Query: *"Are you doing alright?" or "How are you doing"*** • Still no gesture or communication, say: **Query: *"Do you need more blocks?"*** • When finished place container in front of child, say: • **Instructions: *"Time to clean up!"***	o Observe interactive 141ehaviour o Does the student make eye contact? When? o Does student ask for pieces? How? o Note details of conversation (reciprocal)

Table 34.   Make-Believe Play (contents of bag 3-2, action figures & props)

Instructions: *"Here are three characters to use to make up a story. Could you play with these for a while?"* • Lay all the materials out with some description of each item • If the child does not pick up any objects, say: **Query***: "I'll play with these."* • Examiner should begin play with the objects (limited) • If the child still does not play with the objects, say: **Query:** *"What are you doing with yours?"* • If child appears to be reluctant, frame the task as making a MTV video or TV show. • Examiner should be interactive and show interest in the play. • Try to elicit info from child by asking, **Query:** *"That looks interesting. What are you doing?"* • Crucial to see child's creative/imaginative play (not a recital of the examiner's introduction)	• The focus is to see if the child can engage in imaginative play • Do the action figures interact with one another? • Are there developing of play themes? • Does student participate in sequence (i.e., one hits another, the 2nd one falls, then something else happens)? • Does student engage in role playing? Does student pretend to be characters of telling what characters are doing ("The knight is going to…")

Table 34.   Joint Interactive Play (same materials as #2)

• After make-believe play has been evaluated, the examiner joins in. **Instructions:** "*Can I play too?"* • Manipulate object and press for interactive play • Try this up to 4 times • Examiner must join in and attempt to elicit flexibility in play. • Make comments about what is happening. Add a third figure if only two are being used. **Query:** "*Can you be they boy?', or, "Can I do that?"*	• Focus is on reciprocity through interactive play • Participant must develop the interaction • Not just a reaction to the examiner's overtures • Flexibility in changing themes when examiner joins in? • When Psych puts out bag, does student start/help clean up toys?

4. Demonstration Task (Hand towel and soap)

Instructions: *"Now I want you to play a pretend game with me. Let's pretend this is a sink in the bathroom."* • Pretend to draw a sink with the faucets. Then say: "*This is a pretend toothbrush and this is the pretend toothpaste."* • Make gestures to indicate where those object are. Say: *"Now I want you to teach me how to brush my teeth. Can you show me and tell me? Start at the beginning. You just came into the bathroom. What do you do now?"* • Repeat all the instructions if the student does not understand. • If student still does not begin, demonstrate for the student. • If they demonstrate only an isolated action, say: *"That's good, now tell me and show me again."* • Now repeat the sequence with the hand towel and soap. • Can attempt 2nd task, if student not successful with 1st.	• Focus is the child's ability to represent familiar actions with gestures. • Are there verbal explanations of the gestures? • How much detail is provided? • Can the child report on a familiar event.? • Overall – looking for student's ability to describe a story AND provide gestures. Does student do one (describe) but not provide gestures or vice versa? • How does student respond to having to sequence a task? Can student initiate task? • If student is not able to do through gestures, have student demonstrate using real objects. Does this help student sequence the process better? How specific were the sequencing details when real objects were used (increase or decrease)?

Table 34.   Description of a Picture (American montage scene or resort scene)

• Use only one scene unless the child does not respond to one • Can shift down to level 2 scene if not appropriate for developmental level. **Instructions: *Let's look at this picture now. Can you tell me about it? What is happening in the picture?*** • The aim is to generate language • Encourage student and respond positively • Show interest and enthusiasm in what the child says • Make statements or ask questions to encourage more communication. • Want the child to relate what they see to personal experience • If the child does not offer any conversation other than stating factual info, say: • **Query: *"What is this? Who is this? What are they doing?"***	• Looking for student's language level, ability to describe details. • Does student look at the picture as a whole or does student just describe details in the picture? • Does student point to any objects in the picture? • Try to get a good sample of the child's spontaneous language and communication • Consider if the examiner had to ask questions to elicit verbal exchanges. • Look for the child's interests • Any reciprocity? • Did the child only provide basic facts or detailed info and explanations?

Table 34.   Telling a Story from a Book (either of the picture books) Younger-Frog Book, Older-*Free Fall*

• Show the child a book and say: **Instructions: *"Have a look at this book. It tells a story in pictures. See it starts out with …..(describe the first picture). Can you tell me the story as we go along? You go first, then I will take a turn."*** 　• Hand the book to the child and encourage them to begin 　• Only give two specific prompts to begin 　• If the child focuses on specific details say: *"You're right. Can you tell the story? Or What are the pictures all about?"* 　• Once the child has described the book for a few minutes, say: **"That was great. Now I'll take a turn."** • Quickly complete the story. If the child is determined to complete the story. Make a note and let them do so. • 1st – See what student does without Psych asking student questions. After, Psych can ask probing questions about the book: "What is the man thinking?" • Psych can have student slow down and tell sequence. • Ok to make comments, "Gosh, look at that!"	• Try to get a good sample of the child's spontaneous language and communication • Consider if the examiner had to ask questions to elicit verbal exchanges. • Look for the child's interests • Any reciprocity? • Did the child only provide basic facts or detailed info and explanations in sequences? • Also look for the child's response to humor. • Demonstrate social understanding (related to characters in the book)? • Notice affect, intonation, emotional range on face and reflection. • Does student recognize humor in the book (i.e., recognize details on the people's faces. • Does student have ability to retell story (real or imaginative)? • Why is the story imaginative?

7.   Cartoons (Card set A [fisherman,cat] B[two monkeys])

• Child is told to look at the cartoons and then retell the story • Examiner presents each set of cartoons in order while giving basic info about the cartoon • Initially, do not give too much info • If the child is confused about the story provide clarification • After presentation of each set, ask the student to step back from the table and cartoons and say : *"Can you tell me the story?" "Tell me and show me with your hands what happened in the cartoon?"* 　• The student needs room to gesture, standing up	• Does student recognize emotions on the page? • Observe the child's use of gestures as coordinated with speech • Any sense of humor apparent? • Note the amount of language provided • Obtain an idea of insight (ability to inference from concrete visuals) • Ability to adapt narrative to the audience (make it understandable). Concerned about audience understanding. • Comments about affect or relationships of characters. • Can the child describe character motivations?

• Have no access to the cartoons (nothing in hands) • If limited gesturing on the first cartoon set, then administer the second.	• Retell in sequence?

8. Description of a Picture (Feast scene or resort scene)

• Use only one scene unless the child does not respond to one • Can shift down to level 2 scene if not appropriate for developmental level. **Instructions: *"Let's look at this picture now. Can you tell me about it? What is happening in the picture?"*** • The aim is to generate language • Encourage student and respond positively • Show interest and enthusiasm in what the child says but do not ask questions. • Make statements or ask questions to encourage more communication. • Want the child to relate what they see to personal experience • If the child does not offer any conversation other than stating factual info, say: • **Query: *"What is this? Who is this? What are they doing?"***	• Looking for student's language level, ability to describe details. • Does student look at the picture as a whole or does student just describe details in the picture? • Does student point to any objects in the picture? • Try to get a good sample of the child's spontaneous language and communication • Consider if the examiner had to ask questions to elicit verbal exchanges. • Look for the child's interests • Any reciprocity? • Did the child only provide basic facts or detailed info and explanations?

Table 34.   Telling a Story from a Book (either of the picture books)

• Show the child a book and say: **Instructions: *"Have a look at this book. It tells a story in pictures. See it starts out with …..(describe the first picture). Can you tell me the story as we go along? You go first, then I will take a turn."*** • Hand the book to the child and encourage them to begin • Only give two specific prompts to begin • If the child focuses on specific details say: ***"You're right. Can you tell the story? Or What are the pictures all about?"*** • Once the child has described the book for a few minutes, say: **"That was great. Now I'll take a turn."** • Quickly complete the story. If the child is	• Try to get a good sample of the child's spontaneous language and communication • Consider if the examiner had to ask questions to elicit verbal exchanges. • Look for the child's interests • Any reciprocity? • Did the child only provide basic facts or detailed info and explanations in sequences? • Does student have ability to retell story (real or imaginative)? • Why is the story imaginative?

determined to complete the story. Make a note and let them do so. • 1st – See what student does without Psych asking student questions. After, Psych can ask probing questions about the book: "What is the man thinking?" • Psych can have student slow down and tell sequence. • Ok to make comments, "Gosh, look at that!"	

Table 34. Free Play (TOYS ON TABLE-pop-up toy, board book, toy telephone, pieces of yarn, textured block)
(TOYS ON FLOOR- music box, jack-in-the-box, dump truck, baby doll, letter blocks, medium   size ball, two identical cars, two pairs of small balls, two pairs of utensils, and four small plates)

• The goal is to allow the child to play with the items alone. • Allow the child to play with the items for several minutes.	Purpose: • Give student a break from the social demands of the assessment • Provide an opportunity to observe his/her 146ehaviour in less structured circumstances. Focus of Observation: • Does the child spontaneously seek engagement with the examiner? • The extent to which the child explores the materials symbolically or functionally. • How long does the student stay with an activity? • Extent to which the child engages in repetitive activity.

11. Birthday Party (Baby doll, plate, fork, cup, knife, napkin, play-dough, four candles, and blanket.)

Instructions: • Complete this task at a slow pace if possible • Put the doll on the table or in a second chair • Say, **"Look, here's the baby!"** • Provide an opportunity for the child to touch, hug, or speak to the doll. • In an animated voice say, **"It's the baby's birthday! Let's have a birthday party for the baby!"** • Make a cake out of the play dough on the plate • Put one candle in the cake and say,	**"Okay, the party's over. Now what will the baby do?"** • Lay the doll and the blanket on the table. If the child does not attempt to cover the baby or put it to bed, then say, **"The baby is tired. Time for the baby to sleep."** • Pause and then give the blanket to the child. No response, cover the doll with the blanket and say, **"Night, Night baby"** • Give the doll to the child and see if they give it a kiss or put it to bed. Purpose:

"Here are the candles." • Give the second candle to the child and leave the other two within reach. • If not done independently, help the child put the other candles on the cake. • Pretend to light the candles, and blow out the match, say, **"Hot! What should we do now?"** • If the child does not respond, say, **"Let's sing Happy Birthday!"** • At the end of the song, clap and cheer. • If the child does not blow out the candles or have the doll do so, say, **"Let's blow out the candles!"** • Then follow these 4 steps. Say, **"What's next?"** • Open your mouth and make a blowing expression, then blow out the candles. Then clap and cheer. • Give the fork to the child and say, "**The baby's hungry"** • If the child does not begin to feed the doll, say **"The baby wants some birthday cake."** • If the child begins to feed the baby make, **"Yum, Yum"** sounds. • If the child does not demonstrate feeding the baby then say, **"Let's feed the baby"** • Give the child the fork and point to the cake. • Pretend to pour some juice in the cup. • Put napkins on the table and pretend to spill the juice saying, **"Oh No! I spilled the juice! What a mess. What should we do?"** • If the child does not respond, say **"Can you help clean up?"** If still no response, then hand the child a napkin. Then say,	• To create and opportunity for the child to engage in symbolic and functional play. Focus of Observation: • The child's level of interest or ability to join in with a familiar script. • Note whether the child treats the baby as an animate being • Spontaneously contributes to the to the enactment • Can imitate the examiner's actions or join in when directed.

12. Snack (Small cup, water or juice in a clear container, paper plate, and two kinds of small cookies or crackers in plastic containers)

Instructions:	Purpose:
• Child should be seated at the table and say, "It's time for a snack." • Place the plate in easy reach of the child and put one type of cracker on the plate. Say,	• To give the child the opportunity to make requests in a familiar context. Focus of the observation: • To determine if and how the child makes a preference and requests food.

"We have cookies and crackers." • Hold each container in a hand 18-24 inches from the child and say, **"What do you want?"** • Wait for and note response. If no response, hold up each container and say, • **"Crackers or cookies?"** Hold up both containers and say, **"What do you want?"** • Give the child what they have requested by any means (pointing, reaching, verbal). • Repeat holding up the containers and asking the student what they want until they have had enough. Offer a drink if necessary.	• How does the child use gaze, gesture and facial expressions, and vocalization to communicate requests?

13.   Anticipation of Routine with Objects (a balloon or cause and effect toy)

Instructions:
• Blow up the balloon, pinch the neck to prevent air from leaking and hold the balloon directly in front of the child.
• Let the child touch the balloon. Say,
"Ready, set, go!"
• Let go of the balloon, then blow it up and release it again.
• After the balloon lands, wait for the child to bring it to you. Wait to see if they request that it be blown up again.
• Do not let the child try to blow up the balloon.
• Again, show the child the balloon, and repeat the steps, pausing after each step to see the child's reaction.
   o Hold the balloon in front of your mouth.
   o Say, **"Ready, set, go!"**
   o Put the balloon to your mouth.
   o Blow up the balloon
   o Hold the inflated balloon over your head
   o Release
• Repeat the procedure two more times, waiting to see if the child initiates the sequence.
	Purpose:
	• Purpose: To assess the child's anticipation and initiation of the repetition of an action routine.
	
	Focus of Observation:
	• Another opportunity to observe the child's affect, initiation of joint attention, shared enjoyment, requesting and motor 148ehaviour.

- 14. Bubble Play (bubble gun and liquid)

Instructions:	Purpose:
• Have the child engage with a book or a toy on the floor. • Move about 5 ft from the child with the bubble gun and liquid. • Begin blowing bubbles with the bubble gun holding it away from your body • Continue blowing bubbles until about 5 seconds after the child sees them • Note whether the child gestures or vocalizes • For full credit the child must act while the bubbles are present. Note reaction within 5 seconds of bubbles appearing. • Partial credit if the child gestures or looks towards parent/or other examiner • After making notations on joint attention, wait for the child to request more bubbles. • If the child does not, put the bubble gun in a location so they can hand the gun to examiner. • Examiner keeps the bubble liquid.	• To elicit eye contact and vocalization from the child in coordination with pointing or reaching for the purpose of directing attention to a distant object. Unusual sensory behaviors or movements may be noted. Focus of Observation: • To observe the child's affect, initiation of joint attention, shared enjoyment, requesting and motor 149ehaviour. • Initiation of joint attention requires a shift in gaze from the object to person to object with no other purpose than to share interest or pleasure.

SCORING:
- When stuck between 2 scores, choose the lower score. EXCEPT for SECTION D; If we see it at ALL, CODE!

Module 3

Student Name: _____   Date: _____   Observer: _____

ADOS-Module 3
- In general, the order of tasks and events should be flexible and determined by flow of the interaction.
- Begin by putting student at ease. This should include some indication that the session will involve a variety of different activities or tasks. Need to present student with a list of tasks that we will be completing. Present the list orally. If the student seems anxious and not appearing to be engaged in task, then present the student with a visual chart of task, that then can be checked off as it is completed.
- It is essential to provide an interactive model, commenting on the student's activities or statements and introducing brief observations about the Psych's own interests or activities.
- Overall – note if student directs attention to a different place in the room, specific item, etc.
- Eye contact – if a student doesn't make eye contact, try to do something/make a noise that will catch his attention "Whoa! Look at this!"

2. Construction Task (puzzle and pattern board)

• Put at least a few pieces to the side so you can tell if student asks for pieces of puzzle (asks for help). Make sure the Psych gestures to the blocks so the student can see these blocks. • Sit far apart and place some blocks out of reach • Create opportunity for child to ask for help. **Instructions: *"Show me how you would put these blocks together to look like this picture. Let me know if you need more blocks."*** • The remainder of the blocks are placed on the other side of the examiner's arm. • Indicate where they are. • Turn slightly away from the child when initial blocks have been used. • If child does not ask or gesture towards/for the remaining blocks, ask **Query: *"Are you doing alright?" or "How are you doing"*** • Still no gesture or communication, say: **Query: *"Do you need more blocks?"***	○ Observe interactive 150ehaviour ○ Does the student make eye contact? When? ○ Does student ask for pieces? How? ○ Note details of conversation (reciprocal)

| • When finished place container in front of child, say:
• **Instructions: *"Time to clean up!"*** | |

Table 34.   Make-Believe Play (contents of bag 3-2, action figures & props)

| **Instructions: *"Here are three characters to use to make up a story. Could you play with these for a while?"***
 • Lay all the materials out with some description of each item
 • If the child does not pick up any objects, say:
Query: *"I'll play with these."*
 • Examiner should begin play with the objects (limited)
 • If the child still does not play with the objects, say:
Query: *"What are you doing with yours?"*
 • If child appears to be reluctant, frame the task as making a MTV video or TV show.
 • Examiner should be interactive and show interest in the play.
 • Try to elicit info from child by asking,
Query: *"That looks interesting. What are you doing?"*
 • Crucial to see child's creative/imaginative play (not a recital of the examiner's introduction) | • The focus is to see if the child can engage in imaginative play
• Do the action figures interact with one another?
• Are there developing of play themes?
• Does student participate in sequence (i.e., one hits another, the 2nd one falls, then something else happens)?
• Does student engage in role playing? Does student pretend to be characters of telling what characters are doing ("The knight is going to…") |

Table 34.   Joint Interactive Play (same materials as #2)

| • After make-believe play has been evaluated, the examiner joins in.
Instructions: "***Can I play too?"***
 • Manipulate object and press for interactive play
 • Try this up to 4 times
 • Examiner must join in and attempt to elicit flexibility in play.
 • Make comments about what is happening. Add a third figure if only two are being used.
Query: "***Can you be they boy?', or, "Can I do that?"*** | • Focus is on reciprocity through interactive play
• Participant must develop the interaction
• Not just a reaction to the examiner's overtures
• Flexibility in changing themes when examiner joins in?
• When Psych puts out bag, does student start/help clean up toys? |

Table 34.   Demonstration Task (Hand towel and soap)

| **Instructions: *"Now I want you to play a*** | • Focus is the child's ability to represent |

pretend game with me. Let's pretend this is a sink in the bathroom."
   • Pretend to draw a sink with the faucets. Then say:
"*This is a pretend toothbrush and this is the pretend toothpaste."*
   • Make gestures to indicate where those object are. Say:
"Now I want you to teach me how to brush my teeth. Can you show me and tell me? Start at the beginning. You just came into the bathroom. What do you do now?"
   • Repeat all the instructions if the student does not understand.
   • If student still does not begin, demonstrate for the student.
   • If they demonstrate only an isolated action, say:
"That's good, now tell me and show me again."
• Now repeat the sequence with the hand towel and soap.
• Can attempt 2nd task, if student not successful with 1st.
	familiar actions with gestures.
	• Are there verbal explanations of the gestures?
	• How much detail is provided?
	• Can the child report on a familiar event.?
	• Overall – looking for student's ability to describe a story AND provide gestures. Does student do one (describe) but not provide gestures or vice versa?
	• How does student respond to having to sequence a task? Can student initiate task?
	• If student is not able to do through gestures, have student demonstrate using real objects. Does this help student sequence the process better? How specific were the sequencing details when real objects were used (increase or decrease)?

5. Description of a Picture (American montage scene or resort scene)

• Use only one scene unless the child does not respond to one
   • Can shift down to level 2 scene if not appropriate for developmental level.
Instructions: *Let's look at this picture now. Can you tell me about it? What is happening in the picture?*
   • The aim is to generate language
   • Encourage student and respond positively
   • Show interest and enthusiasm in what the child says
   • Make statements or ask questions to encourage more communication.
   • Want the child to relate what they see to personal experience
   • If the child does not offer any conversation other than stating factual info, say:
• **Query: "*What is this? Who is this? What are they doing?"***
	• Looking for student's language level, ability to describe details.
	• Does student look at the picture as a whole or does student just describe details in the picture?
	• Does student point to any objects in the picture?
	• Try to get a good sample of the child's spontaneous language and communication
	• Consider if the examiner had to ask questions to elicit verbal exchanges.
	• Look for the child's interests
	• Any reciprocity?
	• Did the child only provide basic facts or detailed info and explanations?

Table 34.   Telling a Story from a Book (either of the picture books) Younger-Frog Book, Older-*Free Fall*

• Show the child a book and say:
Instructions: ***"Have a look at this book. It tells a story in pictures. See it starts out with …..(describe the first picture). Can you tell me the story as we go along? You go first, then I will take a turn."***
  • "Tell me and show me with your hands what happened in the cartoon?"
  • Hand the book to the child and encourage them to begin
  • Only give two specific prompts to begin
  • If the child focuses on specific details say:
"You're right. Can you tell the story? Or What are the pictures all about?"
  • Once the child has described the book for a few minutes, say:
"That was great. Now I'll take a turn."
• Quickly complete the story. If the child is determined to complete the story. Make a note and let them do so.
• 1st – See what student does without Psych asking student questions. After, Psych can ask probing questions about the book: "What is the man thinking?"
• Psych can have student slow down and tell sequence.
• Ok to make comments, "Gosh, look at that!"
	• Try to get a good sample of the child's spontaneous language and communication
	• Consider if the examiner had to ask questions to elicit verbal exchanges.
	• Look for the child's interests
	• Any reciprocity?
	• Did the child only provide basic facts or detailed info and explanations in sequences?
	• Also look for the child's response to humor.
	• Demonstrate social understanding (related to characters in the book)?
	• Notice affect, intonation, emotional range on face and reflection.
	• Does the student demonstrate the story using gestures?
	• Does student recognize humor in the book (i.e., recognize details on the people's faces.
	• Does student have ability to retell story (real or imaginative)?
	• Why is the story imaginative?

9.   Cartoons (Card set A [fisherman,cat] B[two monkeys])

• Child is told to look at the cartoons and then retell the story • Examiner presents each set of cartoons in order while giving basic info about the cartoon • Initially, do not give too much info • If the child is confused about the story provide clarification • After presentation of each set, ask the student to step back from the table and cartoons and say : ***"Can you tell me the story?" "Tell me and show me with your hands what happened in the cartoon?"***	• Does student recognize emotions on the page? • Observe the child's use of gestures as coordinated with speech • Any sense of humor apparent? • Note the amount of language provided • Obtain an idea of insight (ability to inference from concrete visuals) • Ability to adapt narrative to the audience (make it understandable). Concerned about audience understanding. • Comments about affect or relationships of characters.

• The student needs room to gesture, standing up • Have no access to the cartoons (nothing in hands) • If limited gesturing on the first cartoon set, then administer the second.	• Can the child describe character motivations? • Retell in sequence?

8. Conversation and Reporting

Materials: None	Purpose:
Instructions: • Psych must provide sufficient leads, guides & prompts on a topic. • Psych needs to make appropriate use of student's own interests by incorporating his/her earlier statements, comments, or questions whenever possible. • Topic cannot be centered exclusively around student's strongest interest; but should include some discussion of age-appropriate topics of interest • Avoid question-and-answer style. • In order to emphasize the reciprocal nature of task, psych should make a point of including brief statements about own interests, activities, or feelings and then see if student can build upon or follow up on such comments. • Student must be given the opportunity to describe a nonroutine event (i.e., vacation or family celebration). The event should be something that actually occurred, as opposed to an account of a film or story. • As time goes on, the psych should stop maintaining the conversation and remain silent for a few seconds while looking interested, to see if student can take the initiative without a specific prompt.	• Assess ability to engage in a conversation with to- and fro interchange. • Describe an event/situation for which there are no current visual cues. • Provides an opportunity to generate a language sample in less structured settings (versus with picture tasks). • Student's ability to recount a nonroutine event is evaluated. Focus of Observation: • Extent to which the student builds on the psych's statements and takes a full role in back and forth conversation. • How does student report routine and nonroutine events? • How does student describe relationships and emotions? • Observe student's features of communication: use of gaze, facial expression, intonation and gesture.

Table 34.   Emotions

Materials: None	Purpose:

Instructions:
• Often appropriate to begin or end with "happy" or other positive emotions, but any order may be used.
• These questions can be integrated throughout the ADOS presentation where they fit most casually. If Psych doesn't get all information needed for subtest, can put on visual schedule as "Talk with Karly".
Interview Questions: (need description of 2 emotions)
• ***"What do you like doing that makes you feel happy and cheerful?"***
• ***"What kinds of things make you feel this way? How do you feel when***
	• Psych should probe until the student has given detailed descriptions of:
	   o 2 emotions
	   o the context in which they arise
	   o & what the student's individual experience of these emotions is like.
	Focus of Observation:
	• Identify what events or objects elicit different emotions in the student, especially whether the events/objects are social in nature or not
	• To observe how student describes his/her emotions.

you're happy? Can you describe it?" • *"What about things that you're afraid of?"* • *"What makes you feel frightened or anxious? How does it feel? What do you do?"* • *"What about feeling angry?"* • *"What kinds of things make you feel that way? How do you feel "inside" when you're angry?"* • *"Most people have times when they feel sad. What kinds of things make you feel that way?"* • *"How do you feel when you're sad? What is it like when you're sad? Can you describe that?"* • Can give leading questions: *"You know the other day made me so happy. This is what I did… What makes you happy?"* • Can support conversation with visuals. However, may want to start conversation off without using visuals. Does the introduction of visual picture of friend/name of friend generate more language?	

Table 34.   Social Difficulties and Annoyance

Materials: None Instructions: **Interview Questions:** • *"Have you ever had problems getting along with people at school?"* • *"Are there things that other people do that irritate or annoy you? What are they?"* • *"Were you ever teased or bullied? Why, do you think?"* • *"What about things you do that annoy others?"* • *"Did you ever try to change these things? Did you ever do anything so that others wouldn't tease you? Did it work?"*	Purpose: • To assess student's insight into personal social difficulties and sense of responsibility for his/her own actions. Focus of Observation: • On student's perception of social difficulties • His/her insight into the nature of these problems • And whether he/she has made any attempt to change his/her own 156ehaviour in order to fit in with others more smoothly. • Psych should pay attention to the student's understanding of the appropriateness and implications of his/her feelings.

Table 34.   Break (shape puzzle, paper and markers, pin art, spin pen, small radio, newspaper & magazine, bags 2 & 3)

• Break may be given at any time during the	Purpose:

assessment and/or several breaks may be taken.

Instructions:

- At an appropriate time, the Psych says *"Let's take a break"*.
- Psych indicates that she needs some time to make notes in order to remember what she and the student have done.
- Psych should point to the specified "break" materials and express hope that the student can find something of interest among them.
  - o If the student is not sure about the materials, the Psych should demonstrate how they work (i.e., take the cap off the spin-pen; stick a toy in the pin art, etc.)
- The Psych should either:
  - o Move her chair back from the table OR
  - o Move to another chair
    - ▪ Psych wants to be sitting within view, but away from table where student is sitting.
- If student isn't interested in any of the materials, Psych should ask if there is something else he/she would like to do, or else offer a snack.
- Once student is settled and everything possible has been offered (even if nothing is of interest), the Psych should work on notes for at least 2 minutes (longer is fine).
- If the student initiates an interaction, the Psych should respond briefly and positively, but indicate that she has to finish more paperwork before talking.
- Later, a few seconds, the Psych should look up, catch the student's eye and smile briefly in encouragement.
- GOAL – To create an occasion for the student to initiate an interaction. If this doesn't occur, Psych can return to her notes or say, *"I'll just be a few more minutes"*.
- After several minutes, the Psych should return to the table. If helpful, food and/or drink should be offered.
  - o Psych should give herself a plate and a cup, but not take any food unless the student offers.
  - o Psych should say, *"May I join you*

- Give student a break from the social demands of the assessment
- Provide an opportunity to observe his/her 157ehaviour in less structured circumstances.

Focus of Observation:

- How does student occupy self during free time?
- How does student respond to examiner's withdrawal from the interaction?
- If and how student initiates and participates in an unstructured conversation or interaction with the Psych.
- How does student interact with snack? Does he touch it/smell it?

| *before we get back to work? What would you like to talk about?”* | |

12. Friends and Marriage

| Materials: None
Instructions:
Interview Questions:
• *“Do you have some friends? Can you tell me about them?”* (Note the friends’ ages. It can be helpful to ask for names if the student is very general about who they are.)
• *“What do you like doing together? How did you get to know them? How often do you get together?”*
• *“What does being a friend mean to you?”*
• *“What is different about a friend than someone whom you just work with or go to school with?”*
• *“Do you have a girlfriend/boyfriend? What is her/his name? How old is she/he?”*
• *“When did you see her/him last?’*
• *“What is she/he like? What do you like to do together?”*
• *“How do you know she/he is your girlfriend/boyfriend?”*
• *“Do you ever think about having a long-term relationship or getting married (when you are older)?”*
• *“Why, do you think, some people get married when they grow up?’*
• *“What would be nice about it? What might be difficult about being married?”* | Purpose:
• Obtain a detailed description of one ore more relationships that the student would describe as friendships
• Obtain a general description of his/her understanding of the concept of friendship and the idea of establishing a family or building a long-term relationships as a couple
Focus of Observation:
• Not on whether the student has friends, but on how student understands:
  o the concept of friendship and/or marriage
  o the nature of these relationships
  o on how student perceives his/her own role in these relationships
• The questions pertaining to marriage and long-term relationships focus on:
  o Why a person might want to be part of a long-term relationship
  o Student’s understanding of his/her own possible role in such a relationship |

13. Loneliness

| Materials: None
Instructions:
Interview Questions:
• *“Do you ever feel lonely?”*
• *“Do you think other (young) people in your circumstances ever feel lonely?”*
• *“Are there thing that you do to help yourself feel better?”* | Purpose:
• Provide another opportunity to assess the student’s insight into his/her social situation
• Ability to describe his/her emotional reaction to it
Focus of Observation:
• Questions address whether the student understands the concept of loneliness and how he/she feels it pertains to him/her |

	• Does student understand meaning of word "lonely" or does student describe the word "alone"

- 14. Creating a Story (6 items with a definite purpose and 6 items with no clear purpose)

Instructions:	Purpose:
• Psych tells student, ***"Now you and I are going to make up stories using of these objects"*** • Student is to use 5 items to make up a story, newscast, or commercial • Psych may choose which items will be used or allow the student to choose them • Psych should model choosing 5 items and making up a simple narrative discussing the items in ways for which they are not intended (i.e., using a toy parasol as a basket). • Psych story should be simple so student doesn't seem like he/she can't create a story that competes with Psych story. • Psych should create a story that is geared to developmental age of student. • One object should be used as an "actor" in the story (i.e, "Mr. Flame woke up one morning," using a candle stick). • Psych replaces the 5 items used and gestures to student to choose a new group of 5 items.	• Observe creativity in a play-like situation that is appropriate for older children, adolescents, and adults Focus of Observation: • On student's creative use of objects in telling a novel story or creating a newscast or commercial. • Does the student's story have a beginning, middle, and end?

SCORING:
- When stuck between 2 scores, choose the lower score. EXCEPT for SECTION D; If we see it at ALL, CODE!

Appendix L: Anthropometric Marker Placement for VICON 'Plug-in-Gait'
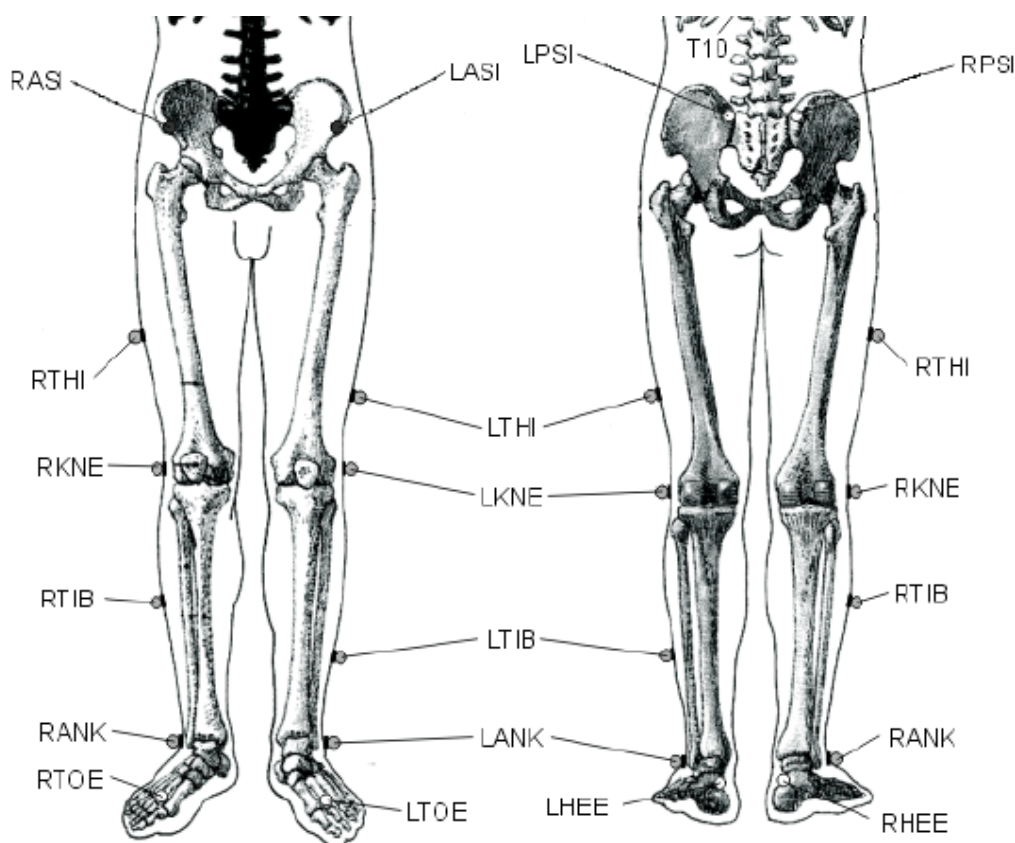
Figure 16. Front and back view of anthropometric marker placement for VICON gait analysis purposes. Diagram from http://www.idmil.org/mocap/Plug-in-Gait+Marker+Placement.pdf

Lower Body

Pelvis

LASI	Left ASIS	Placed directly over the left anterior superior iliac spine
RASI	Right ASIS	Placed directly over the right anterior superior iliac spine

The above markers may need to be placed medially to the ASIS to get the marker to the correct position due to the curvature of the abdomen. In some patients, especially those who are obese, the markers either can't be placed exactly anterior to the ASIS, or are invisible in this position to cameras. In these cases, move each marker laterally by an equal amount, along the ASIS-ASIS axis. The true inter-ASIS Distance must then be recorded and entered on the subject parameters form. These markers, together with the sacral marker or LPSI and RPSI markers, define the pelvic axes.

LPSI	Left PSIS	Placed directly over the left posterior superior iliac spine
RPSI	Right PSIS	Placed directly over the right posterior superior iliac spine

LPSI and RPSI markers are placed on the slight bony prominences that can be felt immediately below the dimples (sacro-iliac joints), at the point where the spine joins the pelvis.

Leg Markers

LKNE	Left knee	Placed on the lateral epicondyle of the left knee

To locate the "precise" point for the knee marker placement, passively flex and extend the knee a little while watching the skin surface on the lateral aspect of the knee joint. Identify where knee joint axis passes through the lateral side of the knee by finding the lateral skin surface that comes closest to remaining fixed in the thigh. This landmark should also be the point about which the lower leg appears to rotate. Mark this point with a pen. With an adult patient standing, this pen mark should be about 1.5 cm above the joint line, mid-way between the front and back of the joint. Attach the marker at this point.

LTHI	Left thigh	Place the marker over the lower lateral 1/3 surface of the thigh, just below the swing of the hand, although the height is not critical.

The thigh markers are used to calculate the knee flexion axis location and orientation. Place the marker over the lower lateral 1/3 surface of the thigh, just below the swing of the hand, although the height is not critical. The antero-posterior placement of the marker is critical for correct alignment of the knee flexion axis. Try to keep the thigh marker off the belly of the muscle, but place the thigh marker at least two marker diameters proximal of the knee marker. Adjust the position of the marker so that it is aligned in the plane that contains the hip and knee joint centers and the knee flexion/extension axis. There is also another method that uses a mirror to align this marker, allowing the operator to better judge the positioning.

LANK	Left ankle	Placed on the lateral malleolus along an imaginary line that passes through the transmalleolar axis
LTIB	Left tibial wand marker	Similar to the thigh markers, these are placed over the lower 1/3 of the shank to determine the alignment of the ankle flexion axis

The tibial marker should lie in the plane that contains the knee and ankle joint centers and the ankle flexion/extension axis. In a normal subject the ankle joint axis, between the medial and lateral malleoli, is externally rotated by between 5 and 15 degrees with respect to the knee flexion axis. The placements of the shank markers should reflect this.

Foot Markers

LTOE	Left toe	Placed over the second metatarsal head, on the mid-foot side of the equinus break between fore-foot and mid-foot
LHEE	Left heel	Placed on the calcaneous at the same height above the plantar surface of the foot as the toe marker

*Note. These anthropometric marker instructions were provided and taught to K Forster by physiotherapists at the Royal Jones and Agnes Hunt Orthopaedic Hospital, Oswestry. A soft copy was found for Appendix purposes at http://www.idmil.org/mocap/Plug-in-Gait+Marker+Placement.pdf

Appendix M: Anonymised Stick-Figure With Markers
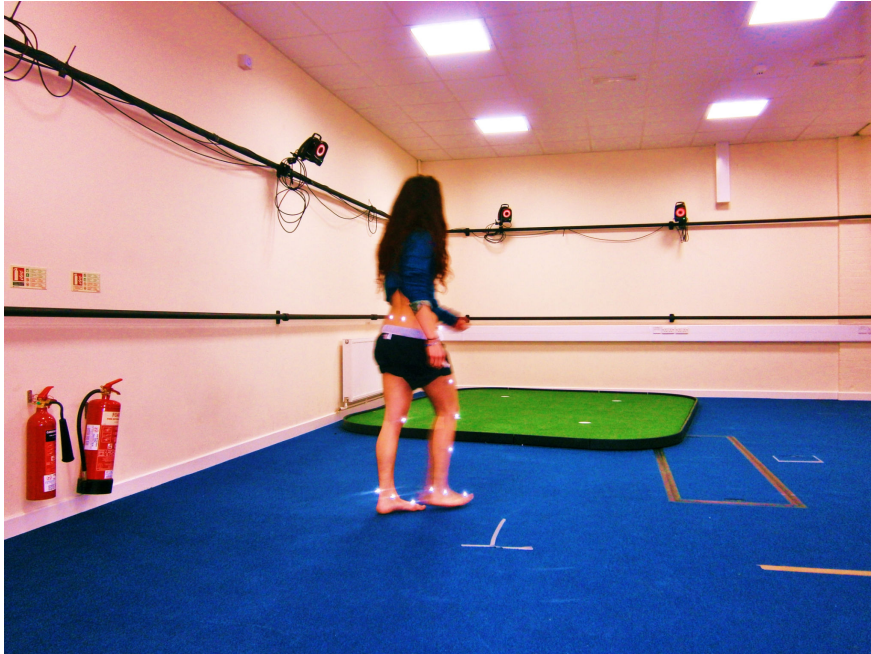
Figure 17. Katharine Forster demonstrating the procedure of walking across the VICON Gait

Laboratory, School of Sport, Health and Exercise Science, Bangor University, Normal Site,

Holyhead Road, Bangor, Gwynedd. LL57 2AS.

Figure 18. Reflective marker used during Gait Analysis. 20 Reflective markers are used on

each participant.
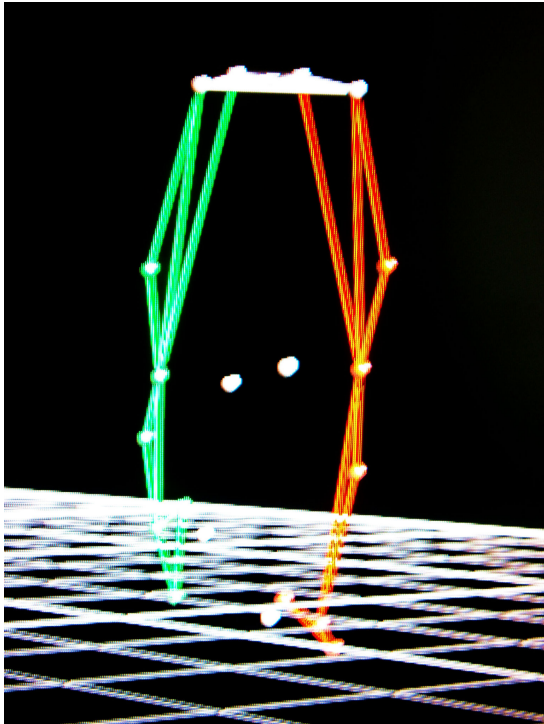
Figure 19. Computerised version of Katharine Forster's walk on VICON Nexus software.

Appendix N: Image of Foot Strike **and On-Screen Time Frame**
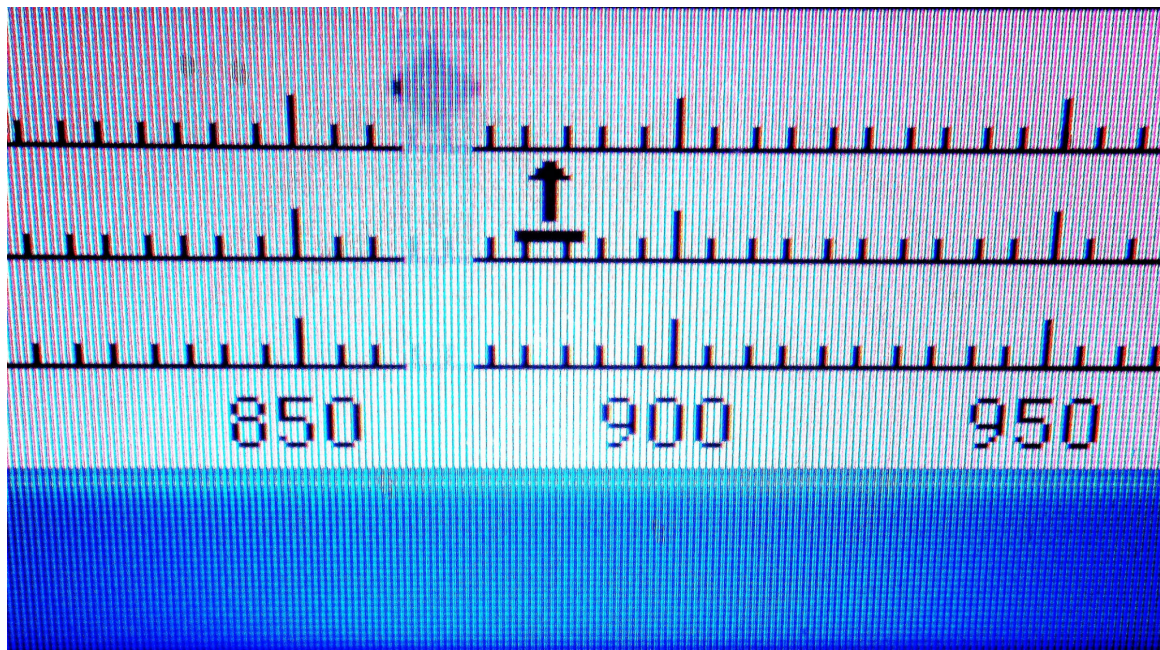
Figure 20. Temporal recordings from a VICON trial; on-screen VICON Nexus screen shot.

'Foot Strike' event is depicted by black diamond shape, occluded by the vertical blue

translucent line used for temporally navigating the participant trial. The blue line can be

moved across the scale to display the exact timing to an accuracy of 250Hz.
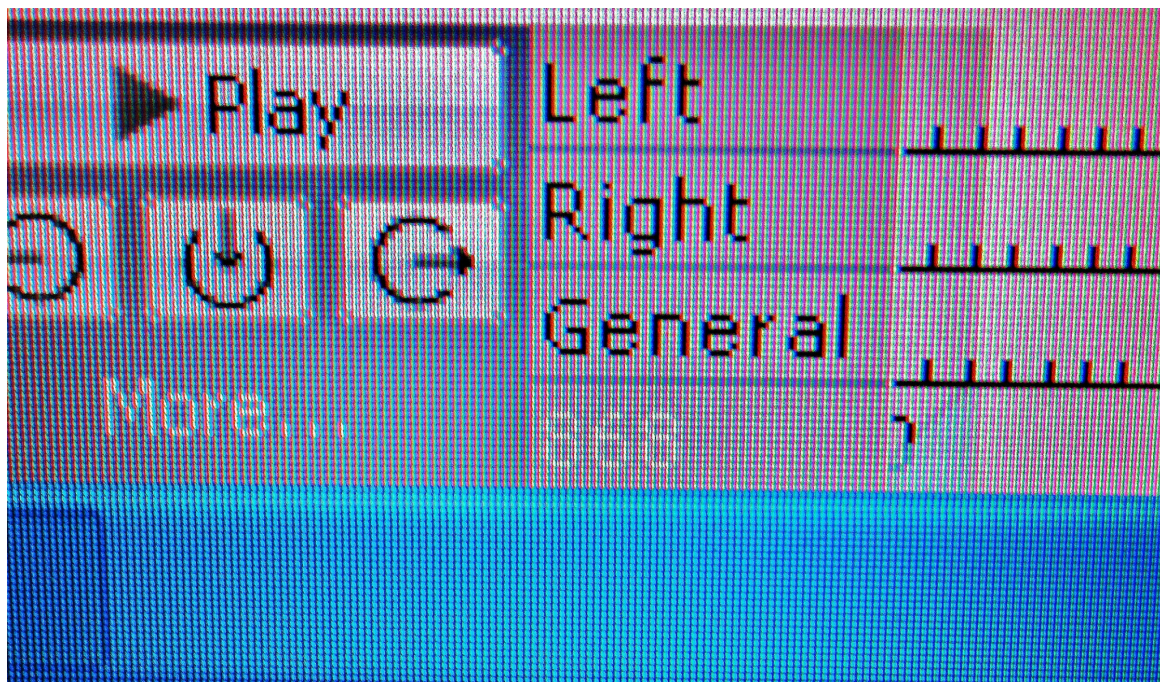
Figure 21. The exact temporal resolution to an accuracy level of 250 Hz. Displayed to the

left of the time scale of a recorded participant trial. This 868 corresponds to the blue line

placement of '*Figure 16'* and is accurate to 4 milliseconds; a Foot Strike 3472 milliseconds

into the trial.

Appendix O: Converting 250Hz Sampling Frequency Data to Milliseconds
Table 2.

Stance Example of Data Converted From Nexus Figure at 250Hz Sampling Frequency Rate

To Milliseconds by Multiplying by 4.

	Stance at 250Hz		SD/M	Stance in ms		SD/M
Participant	SD	M	CoV	SD	M	CoV
4	109.304	534.44	0.2045	437.216	2137.76	0.2045206
17	56.32	587.52	0.0959	225.28	2350.08	0.0958606
3	44	637.08	0.0691	176	2548.32	0.0690651
18	30.808	700.28	0.044	123.232	2801.12	0.0439938
Moi	29.796	539.68	0.0552	119.184	2158.72	0.0552105
12	170.04	635.76	0.2675	680.16	2543.04	0.2674594
14	77.4	534.68	0.1448	309.6	2138.72	0.1447595
Dion	40.6	623.12	0.0652	162.4	2492.48	0.065156
Jazzy	24.86	646.96	0.0384	99.44	2587.84	0.0384259
Aled	30.08	466.24	0.0645	120.32	1864.96	0.0645161
28	39.48	473.32	0.0834	157.92	1893.28	0.0834108
Aron	21.32	523.36	0.0407	85.28	2093.44	0.0407368
2	97.4	692.32	0.1407	389.6	2769.28	0.1406864
13	173.4	687.56	0.2522	693.6	2750.24	0.2521962
29	31.16	594.12	0.0524	124.64	2376.48	0.0524473
35	36.32	701.56	0.0518	145.28	2806.24	0.0517703
36	37.28	623.4	0.0598	149.12	2493.6	0.0598011
Iain	29.76	734.52	0.0405	119.04	2938.08	0.0405163
Timmy	25.04	618.24	0.0405	100.16	2472.96	0.0405021

Note. Participants with autism provided in anonymous numbers. For example purposes only;

mid-way through participant recruitment and data collection, not complete data for Stance

data.

Appendix P: Example of Original Raw Data

Table 3

Example Raw Data From Gait Trials From VICON Nexus Frame Numbers

Trial	LS*	RS**	LO***	RO****
11	2116	1970	2272	2139
11	2373	2243	2541	2395
11	2649	2523	2805	2672
11	2908	2777		2929
11				
25	195	329	349	223
25	446	560	587	466
25	690	815	849	709
25	950			
31	390	491	512	609
31	598	706	726	829
31	815	931	941	1050
31	1039	1132	1140	
32	509	403	414	516
32	702	599	616	718
32	910	801	821	928
32	1120	1020	1035	1132
32		1220	1246	
33	385	491	506	399
33	604	712	734	618
33	836	940	1055	848
33	1180	1169	1280	1070
33	1420	1404	1525	1300
34	249	338	360	260
34	445	559	577	461
34	673	782	802	688
34	894	999	1025	911
34				1139
35	261	372	379	490
35	471	580	590	686
35	672	779	785	890
35	880	970	995	1100
35	1081	1211	1211	

	LS	RS	LO	RO
36	503	404	425	519
36	710	610	630	729
36	921	821	840	936
36	1140	1022	1050	1156
36		1250	1269	
38	500	629	650	780
38	755	880	895	1010
38	1000	1111	1135	1239
38	1220	1355	1368	1476
38	1461	1576	1598	
39	549	651	670	775
39	753	870	885	996
39	979	1088	1105	1212
39	1191	1303	1321	1449
39	1432	1550	1574	
40	270	381	410	528
40	502	625	640	754
40	741	860	875	993
40	972	1099	1115	1228
40	1210			
41	560	430	700	822
41	800	687	941	1069
41	1041	912	1189	1317
41	1299	1174	1440	1565
41	1540	1421	1695	1832
41		1673		

Note. Frame numbers sampled at a frequency rate of 250 Hz (1/250seconds). All raw trial

data from High Functioning Autism participant Number 14, for Foot Strike and Toe Off.

*LS = Left Strike

**RS = Right Strike

***LO = Left Off

****RO = Right Off

Calculating Step, Stance, Swing and Stride times

Table 4

Spreadsheet of Raw Data Calculations for Step, Stance, Swing and Stride Times

Step Time*		Stance Time**		Swing Time***		Step to Step****	
RS-LS	LS-RS	LO-LS	RO-RS	LS-LO	RS-RO	LS-LS	RS-RS
127	146	156	169	101	104	257	273
150	130	168	152	108	128	276	280
128	126	156	149	103	105	259	254
			152				
		154	137			251	231
134	117	141	149	97	106	244	255
114	130	159		103	94	260	
125	135			101	106		
						208	215
101	107	122	118	86	97	217	225
108	109	128	123	89	102	224	201
116	108	126	119	98	82		
93		101					
						193	196
90	106		113	95	83	208	202
99	103	107	119	86	83	210	219
110	109	119	127	89	92		200
100	100	125	112	85	88		
		126					
						219	221
106	113	121	127	98	92	232	343
108	124	130	136	102	94	344	114
104	240	219	130	125	92	240	235
	251	100	131	140	99		
		105					
						196	221
89	107	111	123	85	78	228	223
114	114	132	129	96	98	221	217
109	112	129	129	92	94		
105		131	140		88		
						210	208
111	99	118	118	92	90	201	199
109	92	119	106	82	93	208	191

107	101	113	111	95	80	201	241
90	111	115	130	86	111		
130		130					
					207	206
107	99	127	115	78	91	211	211
111	100	130	119	80	92	219	201
101	100	129	115	81	86		228
110	118	129	134	90	94		
129	126	150	151	105	100	255	251
125	120	140	130	105	101	245	231
111	109	135	128	85	116	220	244
135	106	148	121	93	100	241	221
115		137					
					204	219
102	102	121	124	83	95	226	218
117	109	132	126	94	92	212	215
109	103	126	124	86	91	241	247
112	129	130	146	111	101		
118		142					
					232	244
111	121	140	147	92	97	239	235
123	116	138	129	101	106	231	239
119	112	134	133	97	106	238	
127	111	143	129	95			
					240	257
127	130	140	135	100	90	241	225
112	113	141	157	100	105	258	262
133	129	148	143	110	104	241	247
122	125	141	144	100	108		252
133	119	155	159				

Note: *RS – LS = Right Strike minus Left Strike = time taken from Left Strike to Right Strike = 'Right Step'

LS – RS = Left Strike minus Right Strike, time taken from Right Strike to Left Strike = 'Left Step'

**LO – LS = Left toe Off minus Left Strike, time taken from Left Strike to Left toe Off = 'Left Stance'

RO – RS = Right toe Off minus Right Strike, time taken from Right Strike to Right toe Off =

'Right Stance'

***LS – LO = Left Strike minus Left toe Off, time taken from Left toe Off to Left Strike =

'Left Swing'

RS – RO = Right Strike minus Right toe Off, time taken from Right toe Off to Right Strike =

'Right Swing'

****LS – LS = Left Strike minus Left Strike = 'Left Stride.' RS – RS = Right Strike minus

Right Strike = 'Right Stride.'

Appendix Q: Step Timing Data

Table 5

Comparing Coefficients of Variation of Step Timing in Milliseconds: Participants with High Functioning Autism and Typically Developing Controls

Participant	CoV Left Step		CoV Right Step		CoV Left & Right Step	
	HFA	TD	HFA	TD	HFA	TD
4	0.1729	0.0325	0.1349	0.0370	0.1554	0.0347
17	0.0763	0.0624	0.0770	0.0522	0.0786	0.0498
3	0.0911	0.0493	0.0648	0.0389	0.0810	0.0457
18	0.0564	0.0564	0.0456	0.0632	0.0512	0.0600
35	0.0492	0.0372	0.0527	0.0385	0.0527	0.0383
2	0.1792	0.0510	0.1766	0.0588	0.1871	0.0501
36	0.0561	0.0546	0.0652	0.0514	0.0627	0.0523
29	0.0592	0.0566	0.0537	0.0562	0.0592	0.0600
28	.1088	.0346	.0957	.0533	.1045	.0531
13	.2446	.1169	.2265	.1344	.2378	.1250
14	.2486	.0688	.1145	.0603	.1944	.0648
15	.0605	.0407	.0660	.0615	.0630	.0542
11	.1588	.0725	.1440	.0429	.1517	.0647
12	.2112	.0519	.1859	.0371	.1560	.0446
20	.0851	.0331	.0700	.0322	.0949	.0323
37	.0675	.0551	.0603	0596	.0643	.0589

Note: HFA – High Functioning Autism, TD – Typically Developing Control, CoV – Coefficient of Variation

Table 6

Comparing Average Step Timing in Milliseconds: Participants with High Functioning Autism and Typically Developing Controls

	Mean Left Step		Mean Right Step		Mean Left & Right Step	
Participant	HFA	TD	HFA	TD	HFA	TD
4	555.24	533.88	550.52	536.96	553.04	535.40
17	509.04	472.00	530.00	475.04	519.92	473.32
3	543.52	471.36	560.65	459.68	552.45	465.60
18	590.62	555.24	586.40	550.52	588.40	553.04
35	587.56	608.48	571.56	617.40	580.04	612.92
2	548.00	420.64	615.92	425.80	580.88	423.24
36	568.08	564.52	553.40	574.04	560.88	574.00
29	520.52	555.24	539.44	550.52	529.68	553.04
28	428.24	458.67	411.12	485.04	420.04	470.53
11	498.30	517.23	487.81	492.31	493.35	504.77
12	515.54	556.91	528.97	564.89	529.80	560.97
13	510.38	476.32	554.32	463.00	531.25	469.80
14	478.84	433.14	456.00	440.73	466.92	437.02
15	464.74	556.41	464.64	540.82	464.69	548.00
20	534.46	539.29	478.29	539.38	507.29	539.33
37	496.83	403.60	487.56	391.48	492.36	397.44

Note: HFA – High Functioning Autism, TD – Typically Developing Control

Appendix R: Swing Timing Data

Table 7

Comparing Coefficients of Variation of Swing Timing in Milliseconds: Participants with

High Functioning Autism and Typically Developing Controls

	CoV Left Swing		CoV Right Swing		CoV Left & Right Swing	
Participant	HFA	TD	HFA	TD	HFA	TD
4	.2358	.0329	.1998	.0393	.2196	.0362
17	.0986	.0793	.0925	.0481	.0960	.0661
3	.0881	.0471	.0694	.0484	.0792	.0488
18	.0564	.0536	.0526	.0468	.0546	.0515
35	.0483	.0345	.0519	.0417	.0500	.0380
2	.1969	.0419	.1691	.0446	.2863	.0429
36	.0641	.0662	.0651	.0576	.0661	.0613
29	.0581	.0536	.0492	.0468	.0625	.0515
14	.1222	.0674	.1018	.0670	.1118	.0688
28	.0908	.0421	.1040	.0529	.0985	.0517
13	.2573	.1137	.2317	.1157	.2516	.1136
15	.0476	.0431	.0510	.0546	.0491	.0492
12	.1234	.0382	.3076	.0341	.2250	.0385
11	.1352	.0411	.1365	.0407	.1359	.0453
20	.0561	.0317	.0867	.0454	.0859	.0389
37	.0593	.0452	.0608	.0525	.0606	.0498

Note: HFA – High Functioning Autism, TD – Typically Developing Control, CoV –

Coefficient of Variation

Table 8

Comparing Average Swing Timing in Milliseconds: Participants with High Functioning Autism and Typically Developing Controls

Participant	Mean Left Swing		Mean Right Swing		Mean Left & Right Swing	
	HFA	TD	HFA	TD	HFA	TD
4	472.00	452.24	493.72	449.08	776.92	450.64
17	451.08	406.76	437.52	407.68	444.32	407.18
3	467.43	409.96	461.40	419.44	464.46	414.76
18	480.12	480.16	486.68	493.72	483.16	487.12
35	459.64	492.28	460.32	492.68	460.00	492.48
2	471.56	380.12	738.96	380.68	605.28	380.40
36	499.68	505.96	515.12	506.60	507.24	506.12
29	468.96	480.16	440.32	493.72	455.20	487.12
14	383.64	384.70	386.72	397.49	385.20	391.32
28	367.24	416.80	353.92	399.85	360.72	408.16
13	379.71	394.80	431.41	387.40	403.15	391.10
15	406.15	466.46	404.10	462.48	405.10	464.36
12	444.75	478.32	417.10	492.38	431.61	485.63
11	427.27	445.57	414.60	428.00	421.24	437.18
20	474.67	446.77	430.67	450.56	452.67	448.63
37	424.36	355.00	414.81	347.14	419.10	351.07

Note: HFA – High Functioning Autism, TD – Typically Developing Control

Appendix S: Stance Timing Data

Table 9

Comparing Coefficients of Variation of Stance Timing in Milliseconds: Participants with

High Functioning Autism and Typically Developing Controls

Participant	CoV Left Stance		CoV Right Stance		CoV Left & Right Stance	
	HFA	TD	HFA	TD	HFA	TD
4	.2045	.0405	.1342	.0389	.1698	.0394
17	.0959	.0552	.0867	.0449	.0909	.0498
3	.0691	.0407	.0862	.0510	.0778	.0467
18	.0440	.0652	.1353	.0455	.1020	.0569
35	.0518	.0405	.0472	.0376	.0494	.0390
2	.1407	.0645	.1691	.0493	.1588	.0574
36	.0598	.0384	.0516	.0469	.0575	.0479
29	.0524	.0652	.0653	.0455	.0642	.0569
14	.1448	.0583	.1085	.0620	.1287	.0607
28	.0834	.0454	.1065	.0380	.0972	.0464
13	.2522	.1340	.2546	.1262	.2551	.1287
15	.0699	.0522	.0685	.0517	.0688	.0515
11	.1547	.0447	.1677	0.0399	.1615	0.0458
12	.2675	.0389	.1740	.0478	.2360	.0436
20	.0806	.0283	.0759	.0324	.0887	.0303
37	.0634	.0710	.0624	.0565	.0630	.0644

Note: HFA – High Functioning Autism, TD – Typically Developing Control, CoV –

Coefficient of Variation

Table 10

Comparing Average Stance Timing in Milliseconds: Participants with High Functioning

Autism and Typically Developing Controls

Participant	Mean Left Stance		Mean Right Stance		Mean Left & Right Stance	
	HFA	TD	HFA	TD	HFA	TD
4	623.12	618.24	614.08	620.84	618.96	619.52
17	587.52	539.68	603.10	541.44	594.64	540.56
3	637.08	523.36	642.36	513.44	639.76	518.48
18	700.28	623.12	716.28	614.08	708.48	618.96
35	701.56	734.52	703.24	732.20	702.36	733.40
2	692.32	466.24	738.96	470.48	714.84	468.24
36	623.40	646.96	606.40	642.92	615.16	644.88
29	594.12	623.12	625.08	614.08	608.76	618.96
14	534.68	486.60	526.80	476.00	530.92	481.56
28	473.32	526.80	448.08	548.77	481.00	537.00
13	687.56	551.80	633.04	543.62	661.44	547.61
15	522.80	637.20	526.56	638.00	524.72	637.59
11	555.16	566.00	572.89	586.83	564.23	576.00
12	635.76	642.13	580.97	630.30	610.54	636.12
20	536.62	626.24	584.00	628.71	561.85	627.55
37	559.04	440.69	570.07	450.00	564.77	445.26

Note: HFA – High Functioning Autism, TD – Typically Developing Control, CoV –

Coefficient of Variation

Appendix T: Stride Timing Data

Table 11

Comparing Coefficients of Variation of Stride Timing in Milliseconds: Participants with High Functioning Autism and Typically Developing Controls

Participant	CoV Left Stride		CoV Right Stride		CoV Left & Right Stride	
	HFA	TD	HFA	TD	HFA	TD
4	.1414	.0300	.1388	.0286	.1397	.0289
17	.0638	.0387	.0624	.0291	.0632	.0336
3	.0684	.0371	.0680	.0387	.0676	.0676
18	.0413	.0494	.0823	.0363	.0662	.0424
35	.0407	.0331	.0326	.0320	.0367	.0324
2	.1270	.0442	.1387	.0331	.1325	.0393
36	.0458	.0444	.0483	.0401	.0469	.0420
29	.0472	.0494	.0473	.0363	.0470	.0424
14	.1163	.0525	.1439	.0476	.1302	.0498
28	.0767	.0333	.0829	.0266	.0796	.0305
13	.2232	.1236	.2259	.1174	.2229	.1179
15	.0571	.0401	.0536	.0432	.0551	.0414
12	.1173	.0332	.1203	.0298	.1179	.0309
11	.1269	.0346	.1507	.0401	.1390	.0369
20	.0561	.0184	.0867	.0236	.0535	.0210
37	.0523	.0484	.0532	.0459	.0523	.0469

Note: HFA – High Functioning Autism, TD – Typically Developing Control, CoV – Coefficient of Variation

Table 12

Comparing Average Stride Timing in Milliseconds: Participants with High Functioning

Autism and Typically Developing Controls

Participant	Mean Left Stride		Mean Right Stride		Mean Left & Right Stride	
	HFA	TD	HFA	TD	HFA	TD
4	1301.40	1072.80	1264.72	1071.88	1284.00	1072.28
17	1038.76	949.76	1043.08	947.52	1040.88	948.64
3	1103.44	933.36	1104.12	931.04	1103.80	932.12
18	1176.28	1109.60	1149.88	1107.48	1161.38	1108.68
35	1156.56	1225.68	1162.08	1222.88	1159.40	1224.32
2	1161.88	845.00	1168.08	851.84	1164.96	848.16
36	1121.32	1149.80	1128.64	1133.44	1124.92	1143.68
29	1060.72	1109.60	1068.20	1107.48	1064.28	1108.68
14	924.56	870.53	913.20	870.38	918.84	870.45
28	837.76	939.56	844.48	951.38	841.20	945.36
13	1070.70	941.04	1071.52	933.00	1071.09	933.24
15	930.80	1093.57	931.33	1100.00	931.05	1096.73
12	1044.61	1119.33	1056.00	1123.63	1049.94	1121.94
11	974.00	1009.74	989.64	1011.82	981.92	1010.76
20	1009.85	1077.39	1026.91	1079.30	1017.67	1078.35
37	984.36	793.29	987.23	798.00	985.92	795.56

Note: HFA – High Functioning Autism, TD – Typically Developing Control, CoV –

Coefficient of Variation, M – Mean

Appendix U: Cadence Data for HFA and TD

Table 13

Coefficient of Variability and Mean Values for Cadence; Time in Milliseconds Taken To

Execute Six Steps

Participant	CoV Cadence		Mean Cadence	
	HFA	TD	HFA	TD
4	.1753	.0315	3319.56	2677.45
17	.0554	.0287	2580.00	2365.50
3	.0626	.0301	2758.00	2337.09
18	.0212	.0294	2925.82	2761.50
35	.0225	.0323	2899.43	3059.69
2	.1024	.0331	3006.00	2124.80
36	.0614	.0361	2786.22	2875.00
29	.1575	.0197	2514.93	713.22
14	.0882	.0454	2304.00	2164.00
28	.0718	.0141	2114.00	2371.50
13	.1933	.1232	2729.85	2352.80
15	.0511	.0366	2327.27	2743.50
12	.1265	.0514	2608.40	2754.40
11	.1298	.0270	2431.00	2532.00
20	.0295	.0108	2559.00	2694.00
37	.0421	.0420	2452.00	1987.43

Note. CoV = Coefficient of Variability

Appendix V: ADOS Scores, Age, Diagnoses and Medical History of Participants

Table 14

Existing Autism Diagnostic Observation Schedule Scores Assessed by Clinicians Compared to Research Standard Score

Participant	Communication Score		Reciprocal Social Interaction Score	
	Clinician	K Forster	Clinician	K Forster
17	3	n/a*	11	n/a*
18	2	2	11	11
3	2	2	7	5
14	3	1	8	4
2	6	9	12	12
28	5	5	9	9
29	5	5	8	8
15	5	5	8	7
12	2	Dr Fiona Scott	6	Dr Fiona Scott
37	3	2	9	7
36	No existing ADOS	3	No existing ADOS	4
13	No existing ADOS	5	No existing ADOS	11
11	No existing ADOS	4	No existing ADOS	13
20	No existing ADOS	5	No existing ADOS	13

Note: Dr Fiona Scott = ADOS trainer, assessed reliability of K Forster

*Tape not functioning for recoding purposes.

Table 15

Cohen's Kappa Inter-Rater Reliability of K Forster and Clinicians' Scores for The

Communication Component Autism Diagnostic Observation Schedule

	Clinician				
K Forster	1	3	5	6	Total
1	2	1	0	0	3
2	0	1	0	0	1
5	0	0	3	0	3
9	0	0	0	1	1
Total	2	2	3	1	8

Table 16

Age, ADOS Score, Birth Weight, Clinical Diagnostic Information and Medical History of

Participants.

P	Age in months	ADOS score	Clinician's Diagnosis	Birth Weight (kg)	Genetic	Comorbid Diagnoses	Medication at time of Gait Lab.
4	250	n/a***	Asperger's and Developmental Delays – Education/ literacy	3.22	No record of routine screening but is definitely High Functioning (at University) so not deemed a problem.		
17	145	14	Autism	3.08	x	x	x
3	142	7	Asperger's – Able	2.67	x	x	x
12	169	8	Asperger's, Average IQ, inconsistent between	4.00	x	x	x

				verbal/non-verbal IQ			
14	107	5	Autism, Average IQ, slow visual processing speed	4.14	x	x	x
13	101	16*	Autism. Language delay*	2.77	x	x	x
35	241	n/a***	Clinician Mike Jackson found this client after requesting HFA participants	2.91	Only records of test for Phenylketonuriapku and Congenital Hypothyroidism		x
36	420	7	Clinician Karen Keemish found this client after requesting HFA participants	Not on Child Health Database (older). Not deemed a problem – enjoys engineering, excellent language, High Functioning.			
28	135	14	Asperger's Disorder	4.24	x	x	x
29	179	13	High Functioning Autism	3.49	x	x	x
15	124	12	Autism, Average IQ, some support required	3.77	No routine test for Muscular Dystrophy – but zero suspicion of suffering from MD.		X
2	118	21	Autism, Inconclusive cognitive assessment; disengaged**	3.41	x	x	x
18	179	13	Asperger's Disorder with average IQ	4.54	x	x	x

11	145	17	Autism, specific speech and language	2.91	x	x	x
37	100	9	Asperger's	4.27	x	x	x
20	353	18	Autism. Literacy problems, developmental speech and language disorders, specific LD with speech.	2.52	No tests recorded.		

Note: P = Anonymised Participant number, x = no problems noted in records, CAMHS =

Child and Adult Mental Health Services. Age provided in months not D.O.B. for

confidentiality purposes. ADOS Score by K Forster.

*Suspected not High Functioning Autism: ADOS assessment required Module 1 due to

minimal speech; unclear if just a language delay or Learning Disability.

**Clinician suspected Learning Disabilities.

***No ADOS score due to individual declining to participate.

Table 17

Descriptive Statistics for Analyses of Participants According to ADOS Score Cohort, Age

Cohort and Clinical Diagnoses of Autism or Asperger's Disorder.

Participant Cohort and Component of Gait Cycle	Mean Coefficient of Variability*	Standard Deviation**
Stride ADOS $\leq$ 12	.0783	.04
Stride ADOS $\geq$ 13	.1005	.06
Stance ADOS $\leq$ 12	.1053	.07

Stance ADOS ≥ 13	.1273	.06
L Step ADOS ≤ 12	.1225	.08
L Step ADOS ≥ 13	.1211	.07
R Step ADOS ≤ 12	.0928	.05
R Step ADOS ≥ 13	.1111	.06
Step Autism	.1182	.07
Stance Autism	.1124	.06
Swing Autism	.1351	.09
Stride Autism	.0927	.06
Cadence Autism	.0891	.06
Left Step Autism	.1203	.08
Right Step Autism	.1061	.06
Step Asperger's	.1021	.05
Stance Asperger's	.1243	.07
Swing Asperger's	.1229	.08
Stride Asperger's	.0872	.03
Cadence Asperger's	.0833	.06
Left Step Asperger's	.1180	.06
Right Step Asperger's	.0979	.05
Asperger's age in months***	162.50	51.10
HFA age in months***	192.20	111.61
L Step 12y&under	.1449	.08
R Step 12y&under	.1186	.06
L Step 13y&over	.0939	.06
R Step 13y&over	.0875	.05

TD L Step 12y&under	.0634	.02
TD R Step 13y&over	.0614	.03

Note. Communication and Reciprocal Social Interaction scores combine for ADOS score

cohorts. Diagnosis of Autism of Asperger's is derived from clinical files, not from ADOS

assessments.

Table 34.   Values to 4 decimal places

** Values to 2 decimal places

*** Age in months, not Coefficient of Variability of gait

Appendix W: Descriptive Statistics and Appropriate Statistical Methods

Table 18

Averages for Participants with High Functioning Autism and Typically Developing Controls;

Gait Timing Coefficient of Variability and Mean Values

Gait Cycle	Coefficient of Variability of Gait Timing				Average Gait Timing in Milliseconds**			
	HFA		TD		HFA		TD	
	M*	SD**	M*	SD**	M	SD	M	SD
L Step	.1194	.07	.0544	.02	527.20	47.69	508.83	61.98
R Step	.1030	.06	.0548	.02	518.21	50.74	507.75	62.12
Step L&R	.1121	.06	.0555	.02	523.19	47.23	508.50	62.06
L Stance	.1147	.07	.0538	.02	604.02	70.37	578.80	78.86
R Stance	.1121	.06	.0512	.02	605.74	74.30	580.20	77.77
Stance L&R	.1168	.06	.0535	.02	606.40	68.08	579.46	78.13
L Swing	.1086	.07	.0518	.02	442.39	40.07	438.90	47.58
R Swing	.1144	.08	.0531	.02	455.46	85.88	438.27	49.46
Swing L&R	.1208	.08	.0535	.02	467.21	99.83	438.62	48.27
L Stride	.0878	.05	.0430	.02	1056.06	114.82	1016.88	124.45
R Stride	.0960	.05	.0406	.02	1056.82	108.01	1017.74	123.12
Stride L&R	.0906	.05	.0415	.02	1056.33	111.26	1017.19	124.11
L Step – LD	.1073	.06	.0501	.01	522.07	44.37	517.08	61.21
R Step – LD	.0879	.04	.0489	.01	513.50	52.71	517.17	60.16
L&R Step – LD	.0978	.05	.0509	.01	518.49	47.85	517.35	60.70
L Stride –	.0751	.03	.0372	.01	1047.46	119.39	1034.58	121.73

LD								
R Stride – LD	.0836	.04	.0356	.01	1047.82	111.37	1035.64	120.31
L&R Stride – LD	.0782	.04	.0362	.01	1047.51	115.22	1035.27	121.17
L Step L/M	.0656	.01	.0490	.01	543.74	39.62	526.25	66.63
R Step L/M.V.	.0618	.01	.0504	.01	540.57	45.90	524.84	67.52
L&R Step L/M.V.	.0652	.01	.0500	.01	652.29	42.19	526.03	67.26
L Step H.V.	.2026	.04	.0642	.03	528.25	48.99	485.83	59.45
R Step H.V.	.1637	.04	.0655	.04	521.72	40.98	484.63	61.96
L&R Step H.V.	.1804	.03	.0639	.04	525.87	40.84	485.29	60.54
L Stride L/M.V.	.0551	.01	.0370	.01	1053.21	116.29	1041.17	131.67
R Stride L/M.V.	.0597	.02	.0352	.01	1053.98	112.27	1042.13	129.45
L&R Stride L/M.V.	.0578	.01	.0360	.01	1053.36	113.91	1041.83	130.72
L Stride H.V.	.1420	.04	.0530	.04	1079.52	135.98	976.41	110.05
R Stride H.V.	.1530	.04	.0494	.03	1077.19	125.27	977.09	110.17
L&R H.V.	.1470	.04	.0506	.03	1078.46	130.69	976.14	110.53
L Stride	.0911	.06	.0449	.03	1058.50	108.06	1025.85	115.89

minus O

| R Stride | .1016 | .06 | .0440 | .02 | 1057.62 | 98.28 | 1025.85 | 115.85 |

minus O

| L&R Stride – | .0948 | .06 | .0440 | .02 | 1057.90 | 103.00 | 1025.72 | 116.33 |

O

Note. L = Left. R = Right. HFA = High Functioning Autism participants. AS = Asperger's

participants. TD = Typically Developing age-match control participants. M = Mean. SD =

Standard Deviation. LD = Learning Disabilities; participants 2 and 13 (suspected to have

additional LD) and their age-matched controls removed from a version of the analysis.

L/M.V. = Low/Moderate Variability Cohort. H.V. = High Variability Cohort. O =

Overweight/Obese; A version of the analysis was conducted minus these participants. Y =

years.

* Values to 4 decimal places

** Values to 2 decimal places

Table 19

Assessing Data Suitability for Parametric or Non-parametric Statistical tests; Normality Assumptions (Kolmogorov-Smirnov) and Homogeneity of Variance Assumptions (Levene's)

CoV values between HFA and TD participants	Levene's F	p	Kolmogorov-Smirnov, Z	P	Statistical Test
Left Step	30.94	.000	1.77	.004	Mann-Whitney U
Right Step	14.36	.001	1.95	.001	Mann-Whitney U
Left & Right Step	23.39	.000	1.77	.004	Mann-Whitney U
Left Stance	17.22	.000	1.59	.013	Mann-Whitney U
Right Stance	13.34	.001	2.30	.000	Mann-Whitney U
Left & Right Stance	15.87	.000	2.12	.000	Mann-Whitney U
Left Swing	12.31	.001	1.77	.004	Mann-Whitney U
Right Swing	16.20	.000	1.77	.004	Mann-Whitney U
Left & Right Swing	21.12	.000	1.591	.013	Mann-Whitney U
Left Stride	11.07	.002	1.77	.004	Mann-Whitney U
Right Stride	14.12	.001	2.30	.000	Mann-Whitney U
Left & Right Stride	14.79	.001	2.30	.000	Mann-Whitney U
Cadence	12.8	.001	1.77	.004	Mann-Whitney U
Average values between HFA and TD participants	**Levene's F**	**p**	**Kolmogorov-Smirnov, Z**	**p**	**Statistical Test**
Left Step	2.79	.11	0.88	.420	One way ANOVA
Right Step	1.36	.25	0.53	.940	One way ANOVA
Left & Right Step	2.62	.12	0.71	.699	One way ANOVA

Left Stance	0.32	.58	0.53	.94	One way ANOVA
Right Stance	0.45	.51	0.71	.70	One way ANOVA
Left & Right Stance	0.75	.40	0.71	.70	One way ANOVA
Left Swing	0.90	.35	0.53	.94	One way ANOVA
Right Swing	0.37	.55	0.50	.94	One way ANOVA
Left & Right Swing	1.23	.28	0.53	.94	One way ANOVA
Left Stride	0.67	.42	0.71	.70	One way ANOVA
Right Stride	1.12	.30	0.71	.70	One way ANOVA
Left & Right Stride	0.92	.35	0.71	.70	One way ANOVA
Cadence	0.154	.70	0.71	.70	One way ANOVA

Other Tests	Levene's F	p	Kolmogorov-Smirnov, Z	p	Statistical Test
Assessing correlation between ADOS scores and Age in months	0.16	.69	0.64	.81	Pearson's
Comparing Stride CoV between Age dichotomy (pooled HFA and TD)	2.73	.11	0.91	.38	One way ANOVA
Age Dichotomy (HFA)	1.81	.20	1.00	.27	One way ANOVA & Pearson's
Number 4 L vs R	0.391	.555	1.061	.21	Wilcoxon Signed Ranks (small sample)
Age cohort & Step	3.29	.09	1.00	.27	One way ANOVA
Age cohort & Stance	0.01	.92	0.50	.96	One way ANOVA

Age cohort & Swing	0.31	.59	0.50	.96	One way ANOVA
Just HFA Healthy vs Overweight/Obese	Levene's F	p	Kolmogorov-Smirnov, Z	p	Statistical Test
Left Step CoV	2.757	.119	.433	.992	One way ANOVA
Right Step CoV	0.313	.584	.433	.992	One way ANOVA
Left Stance CoV	4.915	.044	.772	.675	Mann-Whitney U
Right Stance CoV	0.440	.518	.433	.992	One way ANOVA
Left Swing CoV	0.216	.649	.577	.893	One way ANOVA
Right Swing CoV	1.889	.191	.577	.893	One way ANOVA
HFA vs TD Participants CoV minus overweight participants	Levene's F	p	Kolmogorov-Smirnov, Z	p	Statistical Test
Left Stride	8.002	.010	1.429	.034	Mann-Whitney U
Right Stride	10.243	.004	2.041	.000	Mann-Whitney U
Left & Right Stride	10.902	.003	2.041	.000	Mann-Whitney U
L/M V. HFA participants compared to TD	Levene's F	p	Kolmogorov-Smirnov, Z	p	Statistical Test
Left Stride CoV	0.693	.416	1.57	.015	Mann-Whitney U
Right Stride CoV	4.85	.04	2.01	.001	Mann-Whitney U
Left and Right Stride combined CoV	2.309	.146	2.01	.001	Mann-Whitney U
Left Step CoV	0.650	.432	1.414	0.37	Mann-Whitney U
Right Step CoV	0.349	.563	1.179	.124	Mann-Whitney U
Left and Right Step CoV	0.136	.717	1.414	0.37	Mann-Whitney U
H.V. HFA participants	Levene's	p	Kolmogorov-	p	Statistical Test

compared to TD	F		Smirnov, Z		
Left Stride CoV	0.054	.820	1.443	.031	Mann-Whitney U
Right Stride CoV	0.013	.911	1.732	.005	Mann-Whitney U
Left & Right Stride CoV	0.040	.845	1.443	.031	Mann-Whitney U
Left Step CoV	2.237	.161	1.604	.012	Mann-Whitney U
Right Step CoV	1.423	.256	1.604	.012	Mann-Whitney U
Left & Right Step CoV	1.044	.327	1.604	.012	Mann-Whitney U
Autism compared to Asperger's Disorder	Levene's F	p	Kolmogorov-Smirnov, Z	p	Statistical Test
Step CoV	3.240	.093	.581	.888	One-way ANOVA
Stance CoV	0.007	.936	.516	.952	One-way ANOVA
Swing CoV	0.266	.614	.581	.888	One-way ANOVA
Stride CoV	3.216	.095	.829	.482	One-way ANOVA
Cadence CoV	.000	.991	.323	1.00	One-way ANOVA
Left Step CoV	1.722	.211	.516	.952	One-way ANOVA
Right Step CoV	0.136	.718	.581	.888	One-way ANOVA
Mean Cadence	1.274	.211	.452	.987	One-way ANOVA
Mean Step	5.429	.035	.645	.799	One-way ANOVA
Asperger's compared to Autism for Right Step CoV	0.136	.718	.581	.888	2(Diagnosis) X
Asperger's compared to Autism for Left Step CoV	1.722	.211	.516	.952	2(Left and Right Step CoV) ANOVA
No. 20 First gait version,	0.211	.654	0.802	.541	Mann Whitney U

average Left & Right

Swing time

| No. 20 Second gait version, average Left & Right Swing time | 2.945 | .137 | 1.414 | | .037 | Mann Whitney U |

| No. 20 Left variability for Step, Stance, Swing between First and Second gait version. | 0.646 | .467 | 1.368 | | .047 | Mann Whitney U |

| No. 20 Right variability for Step, Stance, Swing between First and Second gait version. | 3.612 | .130 | 1.368 | | .047 | Mann Whitney U |

| Asperger's & HFA, age in months | 3.325 | .09 | 0.45 | | .99 | One way ANOVA |

Note. Levene's *F* statistic for homogeneity of variance between samples. Kolmogorov-

Smirnov *Z* statistic for assessing normality of the sample distribution.   L/M V. HFA

Participants = Low/Moderate Variability Cohort participants with High Functioning Autism.

H.V. HFA participants = High Variability Cohort participants with High Functioning Autism.

Any reference to ADOS scores pertains to participants with High Functioning Autism only,

not TD controls.

Appendix X: Autism Diagnostic Criteria, Diagnostic Statistical Manual Version IV

Tools: DSM-IV Criteria for ASDs

299.00 Autistic Disorder
A total of six (or more) items from (1), (2), and (3), with at least two from (1), and one each from (2) and (3):
qualitative impairment in social interaction, as manifested by at least two of the following:
- marked impairment in the use of multiple nonverbal behaviors, such as eye-to- eye gaze, facial expression, body postures, and gestures to regulate social interaction
- failure to develop peer relationships appropriate to developmental level
- a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest)
- lack of social or emotional reciprocity

qualitative impairments in communication, as manifested by at least one of the following:
- delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
- in individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
- stereotyped and repetitive use of language or idiosyncratic language

lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level
restricted, repetitive, and stereotyped patterns of behaviour, interests, and activities as manifested by at least one of the following:
- encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
- apparently inflexible adherence to specific, behaviour, personal routines or rituals
- stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting or complex whole-body movements)
- persistent precoccupation with parts of objects

Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years: (1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play.
The disturbance is not better accounted for by Rett's disorder or childhood disintegrative disorder.

299.80 Pervasive Developmental Disorder, Not Otherwise Specified
This category should be used when there is a severe and pervasive impairment in the development of reciprocal social interaction or verbal and nonverbal communication skills, or when stereotyped behaviour, interests, and activities are present, but the criteria are not met for a specific pervasive developmental disorder, schizophrenia, schizotypal personality disorder, or avoidant personality disorder. For example, this category includes "atypical autism" –presentations that do not meet the criteria for autistic disorder because of late age of onset, atypical symptomatology, or subthreshold symptomatology, or all of these.

299.80 Asperger's Disorder
Qualitative impairment in social interaction, as manifested by at least two of the following:
- marked impairment in the use of multiple nonverbal behaviors, such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction

- failure to develop peer relationships appropriate to developmental level
- a lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest to other people)
- lack of social or emotional reciprocity

Restricted, repetitive, and stereotyped patterns of behaviour, interests, and activities, as manifested by at least one of the following:

- encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
- apparently inflexible adherence to specific, behaviour, personal routines or rituals
- stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements)
- persistent preoccupation with parts of objects
- The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning.
- There is no clinically significant general delay in language (e.g., single words used by age 2 years, communicative phrases used by age 3 years).
- There is no clinically significant delay in cognitive development or in the development of age-appropriate self-help skills, adaptive behaviour (other than in social interaction), and curiosity about the environment in childhood.

Criteria are not met for another specific pervasive developmental disorder or schizophrenia.

299.80 Rett's Disorder

All of the following:

- apparently normal prenatal and perinatal development
- apparently normal psychomotor development through the first 5 months after birth
- normal head circumference at birth
- Onset of all of the following after the period of normal development:
- deceleration of head growth between ages 5 and 48 months
- loss of previously acquired purposeful hand skills between ages 5 and 30 months with the subsequent development of stereotyped hand movements (i.e., hand-wringing or hand washing)
- loss of social engagement early in the course (although often social interaction develops later)
- appearance of poorly coordinated gait or trunk movements
- severely impaired expressive and receptive language development with severe psychomotor retardation

299.10 Childhood Disintegrative Disorder

Apparently normal development for at least the first 2 years after birth as manifested by the presence of age-appropriate verbal and nonverbal communication, social relationships, play, and adaptive behaviour.

Clinically significant loss of previously acquired skills (before age 10 years) in at least two of the following areas:

- expressive or receptive language
- social skills or adaptive behaviour
- bowel or bladder control
- play
- motor skills

Abnormalities of functioning in at least two of the following areas:

- qualitative impairment in social interaction (e.g., impairment in nonverbal behaviours,

       failure to develop peer relationships, lack of social or emotional reciprocity)
- qualitative impairments in communication (e.g., delay or lack of spoken language, inability to initiate or sustain a conversation, stereotyped and repetitive use of language, lack of varied make-believe play)
- restricted, repetitive, and stereotyped patterns of behaviour, interests, and activities, including motor stereotypies and mannerisms

The disturbance is not better accounted for by another specific pervasive developmental disorder or by schizophrenia.

Diagnostic and Statistical Manual, 4th Edition, ï¿½1994, American Psychiatric Association

Appendix Y: Age and ADOS, BMI, Anthropometric Anomalies.

Table 20

Gait Cycle Coefficient of Variations of Participant Number 4* Left and Right Leg Comparisons

Gait Cycle Phase	Left Leg	Right Leg
Step	0.1729	0.1349
Stance	0.2045	0.1342
Swing	0.2358	0.1998
Stride	0.1414	0.1388

Note. *Participant with High Functioning Autism

Table 21

Coefficient of Variability (CoV) of Stride Timing and ADOS Scores as a Function of Participants' Age

	12 years and below		13 years and above	
Participant Cohort	M	SD	M	SD
High Functioning Autism CoV	.1047	.06	.0765	.05
Age-Matched Typically Developing CoV	.0482	.03	.0328	.01
Communication ADOS Score*	4.33	2.29	2.80	1.30
Reciprocal Social Action ADOS Score*	9.44	3.40	7.2	2.59
Combined ADOS Score*	12.78	5.09	11.8	4.44

Note. *Cohort containing participants with High Functioning Autism only. M = Mean. SD = Standard

Deviation. ADOS Scores and SDs to 2 decimal places. CoV values to 4 decimal places.

Table 22

Height (cm) and Weight (kg) Attributes of Participants with High Functioning Autism and

Age-Matched Typically Developing Controls.

HFA	HFA Height	HFA Weight	Age-specific NHS Verdict	TD Height	TD Weight	Age-specific NHS Verdict
17	152	50	93rd % = Overweight	155	47	80th % = Healthy
3	146	42	84th % = Healthy	147	40	67th % = Healthy
4	181	76	23.2 BMI = Healthy	175	64	20.9BMI= Healthy
18	175	68	85th % = Healthy	170	67	90th % = Healthy
12	173	54	37th % = Healthy	169	54	50th % = Healthy
14	139	31	50th % = Healthy	137	30	47th % = Healthy
2	126	32	94th % = Overweight	140	32	49th % = Healthy
28	150	50	96th % = Overweight	140	55	99th % = Obese
13	122	20	2nd % = Healthy	132	26	23rd % = Healthy
15	137	28	20th % = Healthy	138	33	77nd % = Healthy
29	170	59	68th % = Healthy	174	61	61st % = Healthy
35	180	108	33.3 BMI = Obese	178	78	24.6BMI =Healthy
36	192	80	21.7 BMI = Healthy	171	70	23.9BMI =Healthy
11	147	35	22nd % = Healthy	159	58	95th%=OverW
20	183	63	18.8 BMI = Healthy	181	64	19.5BMI =Healthy
37	140	35	86th % = Healthy	127	30	91st% = OverW

Note. Under-18 year olds receive a percentile range (%) for age-specific norms. Adults are

assigned a Body Mass Index (BMI).

Table 23

The Coefficient of Variability of Timing of Participant Number 20 According to First or Second Gait Style, for Left and Right Step, Stance and Swing

Gait Phase	Left Coefficient of Variability		Right Coefficient of Variability	
	First Gait Style	Second Gait Style	First Gait Style	Second Gait Style
Step	0.0740	0.0700	0.0708	0.0851
Stance	0.0535	0.0806	0.0511	0.0759
Swing	0.0973	0.0561	0.1028	0.0867

Table 24
Average Swing Timing for Participant Number 20 According to First or Second Gait Style

Left Swing average timing at 250Hz		Right Swing average timing at 250 Hz		Left Swing average timing converted to milliseconds		Right Swing average timing converted to milliseconds	
First	Second	First	Second	First	Second	First	Second
128.50	117.00	129.00	99.33	514	468	516	397.32
140.00	116.67	127.40	106.33	560	466.68	509.6	425.32
120.50	121.67	134.25	110.33	482	486.68	537	441.32
128.00	119.33	121.20	114.67	512	477.32	484.8	458.68
141.50		113.25		566		453	
129.60		137.75		518.4		551	
136.50		118.00		546		472	

Note. For the First version of the gait style, $n = 7$ trials, for the Second version, $n = 4$ trials.

*Data converted from the sampling frequency of the VICON, 250Hz, to milliseconds by multiplying the values by four.

Appendix Z: World Health Organization Birth Weight

Weight-for-age BOYS
Birth to 13 weeks (percentiles)

World Health Organization

Week	L	M	S	1st	3rd	5th	15th	25th	50th	75th	85th	95th	97th	99th
								Percentiles (weight in kg)						
0	0.3487	3.3464	0.14602	2.3	2.5	2.6	2.9	3.0	3.3	3.7	3.9	4.2	4.3	4.6
1	0.2776	3.4879	0.14483	2.4	2.6	2.7	3.0	3.2	3.5	3.8	4.0	4.4	4.5	4.8
2	0.2581	3.7529	0.14142	2.7	2.8	3.0	3.2	3.4	3.8	4.1	4.3	4.7	4.9	5.1
3	0.2442	4.0603	0.13807	2.9	3.1	3.2	3.5	3.7	4.1	4.5	4.7	5.1	5.2	5.5
4	0.2331	4.3671	0.13497	3.2	3.4	3.5	3.8	4.0	4.4	4.8	5.0	5.4	5.6	5.9
5	0.2237	4.6590	0.13215	3.4	3.6	3.7	4.1	4.3	4.7	5.1	5.3	5.8	5.9	6.3
6	0.2155	4.9303	0.12960	3.6	3.8	4.0	4.3	4.5	4.9	5.4	5.6	6.1	6.3	6.6
7	0.2081	5.1817	0.12729	3.8	4.1	4.2	4.5	4.8	5.2	5.6	5.9	6.4	6.5	6.9
8	0.2014	5.4149	0.12520	4.0	4.3	4.4	4.7	5.0	5.4	5.9	6.2	6.6	6.8	7.2
9	0.1952	5.6319	0.12330	4.2	4.4	4.6	4.9	5.2	5.6	6.1	6.4	6.9	7.1	7.4
10	0.1894	5.8346	0.12157	4.4	4.6	4.8	5.1	5.4	5.8	6.3	6.6	7.1	7.3	7.7
11	0.1840	6.0242	0.12001	4.5	4.8	4.9	5.3	5.6	6.0	6.5	6.8	7.3	7.5	7.9
12	0.1789	6.2019	0.11860	4.7	4.9	5.1	5.5	5.7	6.2	6.7	7.0	7.5	7.7	8.1
13	0.1740	6.3690	0.11732	4.8	5.1	5.2	5.6	5.9	6.4	6.9	7.2	7.7	7.9	8.3
						WHO Child Growth Standards								

Figure 18. World Health Organization Birth Weight Norms, from http://www.who.int/en/

Table 25

Number of trials for each stage of the gait cycle for participants with High Functioning

Autism

Participant	Left Step	Right Step	Left Stance	Right Stance	Left Swing	Right Swing	Left Stride	Right Stride
2	60	64	61	57	57	57	57	57
3	37	34	33	34	35	34	29	32
4	34	37	33	36	34	30	31	28
11	34	35	30	31	33	32	33	33
12	34	37	34	29	33	30	33	29
13	42	38	38	35	41	34	37	33
14	45	49	51	46	44	44	42	43
15	43	44	40	42	39	41	40	36
17	28	26	24	26	26	26	23	22
18	34	32	28	29	30	26	27	30

20a*	31	33	31	29	30	32	30	27
20b**	14	14	13	13	12	12	13	11
28	50	46	46	50	48	46	41	43
29	44	47	49	44	41	38	40	36
35	47	53	51	47	45	50	42	44
36	52	54	53	50	52	50	45	43
37	37	36	34	33	32	32	35	36
Mean	39.18	39.94	38.18	37.12	37.18	36.12	35.18	34.29

Overall Trial $N = 37.15$

Note. * The first walk of participant number 20. ** The second walk of participant number 20.