Motor Dysfunction in Asperger's Disorder: An Analysis of Timing, Motor Planning and Visual Perception of Movement

by

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Abstract

Although motor deficits are presumed to be common in Asperger Syndrome (AS), the causes and characteristics of these deficits are unknown. This study addressed whether one or more of several variables discussed in the literature on Developmental Coordination Disorder, such as timing deficits, motor planning, and visual perception of movement, could account for clumsiness in AS. There were 14 AS participants (7 to 23 years old), and an age and gender matched group of 16 normal controls with no group IQ differences. Participants completed tests of timing perception and production, motor planning, visual perception of static forms, random moving dot kinematograms, biological motion, and postural stability in a moving virtual reality environment. Participants with AS were more erratic in their timing production, though there were no differences in the mean inter-response intervals of the two groups. There were also no differences in the perception of rhythms or in reaction time in responding to the perception task. Timing deficits therefore appear to implicate the motor system more than a central timing mechanism, and could be consistent with dysfunction within the extrapyramidal system. There were no group differences on a motor planning task, which may relate to the demands of the task. Participants with AS were less able to

perceive biological motion, appearing less sensitive to normal movements than the control group, and less stable in virtually moving rooms, especially at the initiation of movement. While social experience could account for deficits in human movement perception, it could not account for hypersensitivity to virtual movement, potentially implicating abnormalities in the dorsal visual stream. Each of these group differences (i.e., timing variability, perception of biological motion, and stability in a virtual environment) was correlated with severity of motor deficits, measured using a clinical examination. The study is the largest to directly examine the components of motor dysfunction in a group of children with AS and also extends the age range into older adolescence, suggesting that motor dysfunction does not improve significantly with maturation into adulthood. Although a single causal factor is unlikely to accurately characterize motor deficits in AS, it is possible that the visual deficits could be the result of the interaction of the visual and motor systems, and that they may be a symptom, rather than a cause of motor dysfunction.

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Motor Dysfunction in Asperger's Disorder: An Analysis of Timing, Motor Planning and Visual Perception of Movement

Under the heading of Pervasive Developmental Disorders (PDD), the Diagnostic and Statistical Manual of Mental Disorders – 4th edition (DSM-IV, American Psychiatric Association, 1994) lists five disorders that vary in their developmental course. These disorders include Autistic Disorder, Rett's Disorder, Childhood Disintegrative Disorder, Asperger's Disorder [also know as Asperger Syndrome (AS)], and Pervasive Developmental Disorder Not Otherwise Specified (NOS). While the distinction between disorders presupposes inherent differences in features, course, and etiology, the disorders are often much less distinct in individuals. The genetic overlap among these disorders has led to the adoption of the term, "Broad Autistic Phenotype" (BAP) or "Autism Spectrum Disorders" (ASD) to better conceptualize these disorders as falling along a continuum that progresses in terms of severity and number of symptoms, in addition to innumerable unknown variables. The features of Rett's Disorder are distinct from the others (i.e., it features deceleration of head growth, loss of previously acquired purposeful hand movements, and is seen only in females), as is the course of Childhood Disintegrative Disorder (i.e., normal development in the first two years of life followed by a loss of previously acquired skills). However, the features and course of autism, AS, and PDD NOS are much more difficult to distinguish, especially in individuals with average to high levels of cognitive ability. A common difficulty with nosological classification systems is that the diagnostic criteria used to separate individuals into groups are then central to determining the features associated with each disorder. Contrasting groups after separating them based on diagnostic criteria often provides

information only on the results of separating them. This circular logic may be less useful than assessing symptoms within the autism spectrum in order to understand the etiology, neuroanatomical underpinnings, and functional consequences of those symptoms. The present study then, is not designed to understand the nosological classification of AS versus any other syndromes within the autism spectrum (particularly differences between AS and autism), but rather, understanding one symptom that is found in many syndromes within the autism spectrum.

The present study is designed to advance the understanding of one symptom seen in the autism spectrum, most frequently noted in AS: motor dysfunction. Motor dysfunction has been reported more frequently in the AS population than autistic populations; motor skills have indeed been seen as a relative strength in autism. However, it may be that while motor skills are a relative strength (ipsitively), they are weak in comparison to other children (normatively). Motor skills dysfunction has been a central diagnostic criterion adopted by some AS researchers, and is included as an "associated feature" in the DSM-IV criteria (i.e., it is frequently associated with AS but not necessary for a diagnosis, and is seen in nearly 80% of cases [Bonnet & Gao, 1996]). In fact, although Developmental Coordination Disorder is a DSM-IV category which captures motor skills dysfunction, the presence of an autism spectrum disorder is an exclusionary criterion for the diagnosis, and DCD cannot be diagnosed as comorbid with a PDD. As such, motor skills dysfunction has largely been ignored in the neuropsychological literature addressing AS. However, characteristic motor impairment, in conjunction with the neurocognitive phenotype of PDDs might suggest an underlying etiological factor (Manjiviona & Prior, 1995; Smith, 2000; Voeller, 1998). Examination

of the motor system provides an alternate method of studying the localization of deficits seen in the autism spectrum. The present study attempts to address processes or components of motor skills that may underlie the clumsiness seen in AS, including hypothesized deficits in motor planning, timing, and difficulty with aspects of visual perception of movement.

Asperger Syndrome

While AS is a newer addition to the DSM than autism, the disorders were described almost simultaneously. Kanner (1943, cited in Wing, 1998) described autism in an American journal only a year before Asperger described his disorder (1944, cited in Wing, 1998), which he labelled autistic psychopathy, in a German journal (Gillberg & Coleman, 1992). Asperger was familiar with Kanner's work but considered the syndromes to be unique. Though Kanner's work attracted widespread recognition throughout psychiatric circles, Asperger's work was essentially known only in German speaking areas (Wing, 1998). It was not until 1981 when Lorna Wing reintroduced Asperger's work that scientific research into the differences between the disorders began. Asperger's Disorder was added to the DSM in 1994 with the publication of the fourth edition. Prior to 1994, AS was diagnosed using a number of slightly different sets of criteria; as such, research published on AS before 1994 is often comprised of AS samples that are dissimilar to the disorder described in the DSM-IV (Gillberg & Ehlers, 1998). Particularly important for the present study is that even when examining the presence of group differences in motor skills, it is not always clear whether motor skills were used as a diagnostic criterion to create the groups.

The current diagnostic criteria for AS, cited in the DSM-IV (1994) are presented in Table 1.

Table 1. Diagnostic Criteria for AS (DSM-IV, 1994, p. 77)

Diagnostic Criteria for 299.80 Asperger's Disorder

A. Qualitative impairment in social interaction, as manifested by at least two of the following:
    1) Marked impairment in the use of multiple nonverbal behaviours, such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
    2) Failure to develop peer relationships appropriate to developmental level
    3) 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)
    4) Lack of social or emotional reciprocity
B. Restricted repetitive and stereotyped patterns of behaviour, interests, and activities, as manifested by at least one of the following:
    1) Encompassing preoccupation with one or more stereotyped and restricted patterns of interests that is abnormal either in intensity or focus
    2) Apparently inflexible adherence to specific, non-functional routines or rituals
    3) Stereotyped and repetitive motor mannerisms (e.g., hand or finer flapping or twisting, or complex whole-body movements)
    4) Persistent preoccupations with parts of objects
C. The disturbance causes clinically significant impairments in social, occupational, or other important areas of functioning.
D. There is no clinically significant general delay in language (e.g., single words used by age 2 years, communicative phrases by age 3 years).
E. There is no clinically significant delay in cognitive development or in the development of age-appropriate self-help skills, adaptive behavior (other than in social interaction), and curiosity about the environment in childhood.
F. Criteria are not met for another specific Pervasive Developmental Disorder or Schizophrenia.

Epidemiological studies of autism and AS using a number of different diagnostic systems suggest that autism is found in 0.0007 to 0.00016% of the population though this number may be increasing; AS is approximately 10 times as common, found in .00036 to .0071% of the population (Gillberg & Ehlers, 1998; Howlin & Asgharian, 1999). Gender

ratios in autism suggest that it is between two and three times as common in males as females, while AS is 10 to 15 times more common in males.

Neuroimaging Results in AS

Although there have been a number of neuroimaging studies of people with autism, there have been very few with people with AS. The neurobiological findings of autism have long been used as a possible model for AS, given their phenomenological and neuropsychological similarities and genetic overlap (Schultz, Romanski, & Tsatsanis, 2000). Three major findings have been noted in the autism literature. The first is accelerated neural growth and cell migration leading to increased brain (and head) size (in males only), and cortical malformations. These include a lack of enlargement of the frontal lobes and a reduction in size of the posterior 40% of the corpus callosum (part of the body and the entire splenium), even when controlling for overall brain volume and gender (Schultz et al., 2000). The second major finding is neuronal abnormalities in temporal and limbic areas, including the amygdala, hippocampus, and entorhinal cortex. Studies using functional magnetic resonance imaging suggest that in both autism and AS, facial recognition is accomplished by inferior temporal gyrus rather than the fusiform gyrus; in normals, the inferior temporal gyrus is involved in object recognition (Schultz et al., 2000). Whether this is a cause or effect of a lack of social reciprocity, or whether it is unrelated, is unknown. Abnormalities of the amygdala may implicate a failure to attach an emotional valence to faces in autism (Afifi & Bergman, 1998). Less activity in the frontal lobes has been noted in functional imaging (Schultz et al., 2000), and

frontostriatal and cerebellar underpinnings have also been proposed (Damasio & Maurer, 1978).

McAlonan et al. (2002) completed magnetic resonance imaging (MRI) with 21 subjects with AS and 24 controls aged 18 to 49 years. In contrast to the controls, adults with AS did not show normal decreases in overall brain volume with age, perhaps indicating developmental differences in neurogenesis and apoptosis. Further, they found an average of 16% less grey matter bilaterally in three areas: (1) from the basal ganglia to the thalamus and ventral striatum, (2) within the medial frontal lobe and cingulate gyrus, and (3) within the cerebellum. They also found an average of 21% less white matter in the AS subjects, concentrated in the left hemisphere, apparently within long fibre tracts such as the inferior and superior longitudinal fasciculi and the occipitotemporal fasciculus (though, note that in humans, the occipitotemporal and inferior longitudinal fasciculi are the same thing [Afifi & Bergman, 1998]), and within the pons and left cerebellum. Finally, an average of 42% more white matter was found in subjects with AS bilaterally around the basal ganglia, incorporating the external capsule. They suggest that the concentration of abnormalities in the frontostriatal circuitry likely has significant functional consequences in terms of motor disturbances and social communication.

Murphy et al. (2002), noting that frontoparietal connections are disrupted in autism, suggested that frontal abnormalities might account for some of the symptoms of autism and AS (e.g., stereotyped obsessional repetitive and ritualistic behaviour). They compared the metabolic activity and membrane turnover of circumscribed right medial prefrontal and right medial parietal areas in 14 men with AS and 18 controls using proton

magnetic resonance spectroscopy. Their results suggested increased prefrontal metabolic activity and membrane turnover in subjects with AS, but no difference in parietal areas. Further, the prefrontal metabolic rate was correlated with the severity of obsessive and repetitive behaviour and membrane turnover was correlated with impaired social communication. Their results suggest that while postnatal neuronal membrane turnover normally decreases between birth and adulthood, this down-regulation is absent in AS, and may be related to some of the clinical symptoms in adulthood.

This literature implicates underdeveloped frontostriatal and cerebellar structures (Dewey & Bottos, 2004), smaller cortical association fibres (particularly in the left hemisphere), and a variety of abnormalities in programmed cell death (particularly in the frontal lobes) of adults with AS. The functional consequences have been linked to social communication, severity of obsessive and repetitive behaviours, and motor disturbances.

An fMRI study of movement-related activation patterns in autism suggests that the pattern of cortical activation and deactivation is less distinct in autism that in normal controls (Müller, Pierce, Ambrose, Allen, & Courchesne, 2001). While rapidly pressing a button, the control group showed activation in contralateral perirolandic regions, while the autistic group showed a less distinct pattern, though the autistic group actually pressed the button faster than the control group. The authors suggest that this lack of distinct activation may be due to an attentional failure of the autistic group to down-regulate posterior activity to focus on the requirements of the task.

Motor Skills Dysfunction in AS

Although children with autism tend to have delays in motor milestones and show less goal directed movement, motor symptoms are often downplayed because they are generally preserved relative to other areas of development (Smith & Bryson, 1994). In children with AS, motor symptoms have been more conspicuous.

Early discussions of AS included clumsiness as a descriptor, and in some cases, a diagnostic feature. Wing (1981, cited in Wing, 1998), in reintroducing the concept of AS to modern psychiatry, suggested that patients with AS had clumsy and poorly coordinated gross motor movements, and argued later (Burgoine & Wing, 1983, cited in Ghaziuddin, Butler, Tsai, & Ghaziuddin, 1994) that motor skills dysfunction, in addition to odd posture, was a major clinical feature.

Early studies of motor dysfunction in AS concentrated on the role of motor skills in diagnostic systems and the potential of this symptom to differentiate AS from autism. Ghaziuddin, Tsai, & Ghaziuddin (1992) provided the first review of clumsiness as a diagnostic feature, noting that although 55% of the 42 articles available on AS mentioned clumsiness or poor coordination as a feature of AS, only 10% assessed motor skills objectively. Of the four studies that assessed motor skills, they [somewhat errantly (Gillberg, 1993)] state that one used an arbitrary cut-off score on a standardized measure, and two others used non-standardized measures. They noted that all too often, the presence of clumsiness is established through clinical judgement rather than more objective means. Ghaziuddin et al. (1992) recommended that future research adopt an operational definition of clumsiness, and suggested the following: "an impairment of motor skills on standardized tests of motor impairment, below the expected level of

intelligence, in the absence of a known neurological disease". They noted the importance of taking intellectual abilities into account, as intelligence and motor skills are correlated, especially for individuals in the low and mentally retarded range. They also recommended that because motor skills dysfunction is not unique to AS or even to PDDs, contrasts should be made with other disorders with known motor deficits, such as Attention-Deficit/Hyperactivity Disorder, expressive and receptive language disorders (Ghaziuddin, Butler, Tsai, & Ghaziuddin, 1994), and Developmental Coordination Disorder.

Research on motor skills in AS has also been undermined by heterogeneity in the set of diagnostic criteria used to define the groups. For example, Klin, Volkmar, Sparrow, Cicchetti, and Rourke (1995), who noted motor dysfunction in their AS sample, acknowledged that they included motor clumsiness as a necessary diagnostic criterion, ameliorating its significance as an associated feature. Most other researchers, however, have not included motor deficits as a diagnostic criterion, especially those working after the publication of the fourth edition of the DSM (1994).

Evidence of Motor Skills Dysfunction in AS

One of the first studies to address motor skills dysfunction in AS (Szatmari, Tuff, Finlayson, & Bartolucci, 1990) used the Grooved Pegboard test and found large differences between adolescents (ages 8 to 18) with AS versus autistic patients and outpatient controls. While the AS group and autistic group were both significantly slower than controls when using their dominant hand, only the AS group differed from controls when using their non-dominant hand [a finding consistent with McManus,

Murray, Doyle, and Baron-Cohen (1992) who noted a dissociation between skill and preference in handedness in autism]. They interpreted their results, which also implicated flexibility and abstraction, as evidence of fronto-subcortical dysfunction and consistent with the theory proposed by Damasio and Maurer (1978) who suggested that the neuropsychological and neuromotor profile of AS was similar to that of older adults with Parkinson's disease and other forms of subcortical dementia. However, the assessment of motor skills in this study was extremely limited, restricting the significance of the findings.

Ghaziuddin, Butler, Tsai, & Ghaziuddin (1994) using a well standardized measure of motor skills, attempted to measure fine (response speed, visual-motor control, and upper limb speed and dexterity) and gross (running speed and agility, balance, bilateral coordination, upper limb coordination, and strength) motor skills in children with AS (n = 11) and HFA (n = 9). Overall, they found that both groups scored equally poorly, scoring well below published population norms. Their AS group was somewhat more intelligent than the HFA group, which may have negated any possible group differences. Ghaziuddin et al. concluded that future studies should focus on the determinants of clumsiness in order to establish whether motor skills dysfunction in AS manifests in a qualitatively different way than in autism. They recommended assessing information processing, visual perception of distance and spatial relationships, and kinaesthetic sensitivity.

Ghaziuddin and Butler (1998) replicated this study, this time including a PDD NOS group (N = 12 for each of the three groups), and found that all three groups showed problems with motor coordination. Specifically, the AS group was significantly less

impaired than the autistic group, with the PDD NOS group not significantly different than either group. Again, however, there was a significant difference between IQ across groups; IQ correlated with motor skills, and when IQ was covaried, group differences failed to reach a significant level. This study demonstrated again that children with AS were more impaired than the general population in terms of motor skills.

A similar study compared 12 children with AS and 9 children with HFA using the Test of Motor Impairment – Henderson Revision (TOMI-H), a standardized measure of manual dexterity, ball skills (catching and throwing), and balance (Manjiviona & Prior, 1995). They found that half of the AS and two thirds of the HFA sample had definite motor problems, generally affecting both gross and fine motor skills. Thus, like Ghaziuddin et al. (1994), there was clear evidence of motor skills dysfunction in children with a PDD diagnosis, though group differences were not significant. They noted delays in meeting developmental milestones, including walking at 16 to 24 months, undeveloped hand preference, trouble controlling the force and direction of the ball when throwing, and difficulty slowing down when told that they were rushing through a task. They concluded that these symptoms might reflect cerebellar ataxia, but did not elaborate.

Miyahara et al. (1997) compared Japanese children with AS (n = 26) to children with learning disabilities (LD; n = 16), citing similarities between the two groups (generally normal intelligence with an uneven pattern of cognitive development). Their dependent measure was the Movement Assessment Battery for Children (Movement-ABC), which measures manual dexterity (e.g., manipulation, drawing, cutting), ball skills (throwing and catching), and balance. The groups differed only on manual dexterity (in which children with AS did better than children with LD, though children with LD had

slightly lower IQs), but the prevalence of the ICD-10 diagnosis of Specific Developmental Disorder of Motor Function (SDD-MF, which requires a two standard deviation discrepancy between intellectual and motor skills development) was 42 to 44 times higher in these two groups than in the general population. Of children with AS, 85% met criteria for SDD-MF, as did 88% of children with LD. The results clearly implicate motor dysfunction in AS, but illustrate that motor dysfunction, as measured in this study, is not pathognomonic.

Miller and Ozonoff (2000) included motor skills in a study of the neuropsychological validity of AS. Like Miyahara et al. (1997), they used the Movement-ABC in a sample of 26 children with HFA and 14 with AS. Unfortunately, while Miyahara et al. reported raw scores, Miller and Ozonoff reported percentiles, so scores could not be compared directly between studies. Miller and Ozonoff (2000) found no difference between the PDD groups, but when they controlled for intelligence, a marginally significant group difference emerged, suggestive of poorer motor performance in the AS group. Among the Movement-ABC subscales, there was a significant difference on the manual dexterity subtest, with the HFA group scoring better (this is the same subtest that Miyahara et al. noted group differences, in favour of AS over LD). Both HFA and AS children were scoring in the borderline range at the 9th percentile on this measure; after covarying intellectual ability, the HFA group was at the 14th percentile while the AS group scored at the 5th percentile.

Iwanga, Kawasaki, & Tsuchida (2000) assessed preschoolers with AS (n = 10) and HFA (n = 15) using the Japanese version of the Miller Assessment for Preschoolers (JMAP) which assesses five developmental areas, including cognitive, verbal, and

sensory-motor functioning using 26 items. They found that two of these areas, Foundation (ten items including sense of position and movement, sense of touch, and development of the basic components of movement), and Coordination (seven items – three of which overlap with the Foundation area – including gross, fine, and oral motor abilities) were significantly lower than IQ for both the AS and HFA groups. Furthermore, the AS group scored significantly lower than the HFA group on the Foundation index, especially showing differences in balance and walking a straight line, which they felt may explain reports of stiff or awkward ways of walking and odd posture in children with AS. All children with AS scored below the 5th percentile on the Foundation score, which suggests a greater level of motor skills dysfunction than other studies, a finding that Iwanga et al. attributed to the accurate scoring system employed by the JMAP and the young age of their subjects, all of whom were between 58 (4 years, 10 months) and 74 (6 years, 2 months) months of age.

Green et al. (2002) attempted to determine whether the symptoms of motor skills dysfunction seen in AS were distinct from motor skills dysfunction seen in SDD-MF, a disorder described in the International Classification of Diseases (ICD-10; World Health Organization, 1992) that is similar to Developmental Coordination Disorder in the DSM-IV, and defined as a "marked" (ICD-10) or "serious" (DSM-IV) "impairment in the development of motor coordination that is not solely explicable in terms of general intellectual retardation or any specific congenital or neurological disease". The study is informative in particular in identifying the severity of functional consequences of motor skills dysfunction in AS. They used a measure of motor impairment, the Movement-ABC, and a measure of gesture production, the Gesture Test with 11 children with AS

and 9 with SDD-MF aged 6 to 10 years. Notably, three children identified as having SDD-MF through a school-based referral system had a sufficient number of symptoms of AS on a recognized diagnostic scale that they were moved into the AS group. On each of the components of the Movement-ABC (manual dexterity, ball skills, and balance), the AS group showed a trend toward being even more impaired than the SDD-MF group, and motor impairment was universal in the group with AS. The AS group was most impaired on the manual dexterity and ball skills components in this sample. Children with DCD have also been shown to have more symptoms of AS (and also of ADHD) than children without (Kadesjö & Gillberg, 1999). Interestingly, Green et al. (2002) have apparently begun a study that is designed to determine whether ball skills may be deficient in subjects with AS because they may be oblivious to the social pressures that drive other children to engage in ball-related sports, an interesting hypothesis that would provide valuable evidence in creating a theory of motor skills in AS.

Green et al. (2002) also assessed gesture production, and found that the group with AS performed significantly worse on this measure, which is similar to findings in the autism literature, in which gestures are viewed as an integral component of a general communication deficit. Scores on the Movement-ABC correlated .67 with gesture production in the AS group. It is significant that the gesture production is correlated with movement dysfunction, as it suggests that there may be a role for motor problems in the genesis of communication deficits. Green et al. (2002) hypothesized that difficulties in gesture production may result from a deficit in executive functioning; that is, it stems from an inability to plan a sequence of movements, and hold that plan in working memory until it is executed.

This body of literature clearly illustrates both the presence and severity of motor skills dysfunction in AS. These studies are, however, generally based on global measures which are useful in describing the functional limitation that children with AS have in adaptive living skills, but is less informative in describing the neuromotor components of movement that lead to these limitations. While there appears to be some diversity of results in comparing motor skills AS and autism, Smith (2004) noted that "this is not to say that patterns of motor performance…may not vary across the spectrum of autism. More studies are appearing that test specific hypotheses about motor and perceptual-motor impairments in ASD" (p. 157). These studies are reviewed below.

Conceptualising "Clumsiness"

Although clumsiness is an important feature of AS, it is not unique to AS. Academic interest in clumsiness in children was first noted in France in the 1910s and 1920s, and was termed "developmental dyspraxia" by Orton in 1937 (Tupper & Sondell, 2004). Gubbay provided the first definition of the clumsy child syndrome: "The clumsy child is to be regarded as one who is mentally normal, without bodily deformity, and whose physical strength, sensation, and co-ordination are virtually normal by the standards of routine conventional neurological assessment, but whose ability to perform skilled, purposive movement is impaired" (Gubbay, 1975, p. 246). Clumsiness itself is the core feature of a number of largely synonymous disorders across clinical disciplines, including Clumsy Child Syndrome (historically), Developmental Dyspraxia (neurology), Sensory Integrative Dysfunction (occupational therapy), and Developmental Coordination Disorder (psychiatry) (Missiuna & Polatajko, 1995). Motor dysfunction is

symptomatic of a number of neuropsychiatric conditions, including Attention-Deficit/Hyperactivity Disorder, childhood anxiety disorders especially obsessive-compulsive disorder, autism, and schizophrenia (Dewey, Crawford, Wilson, & Kaplan, 2004; Kadesjö & Gillberg, 1999; Kroes, et al., 2002; Voeller, 1998) and an even larger number of neurological and genetic conditions.

Despite this apparent lack of specificity, there are a variety of characteristic *patterns* of motor dysfunction that are indicative of involvement of particular brain structures. Analysing movements in terms of the contributions of known and hypothesized movement components permits the use of disordered movement in identifying corresponding dysfunctional contributions of the central nervous system (CNS). As such, the motor system can potentially provide analogous CNS information on the etiology of neuropsychological dysfunction in neuropsychiatric disorders.

Measuring clumsiness has proven difficult. Tupper and Sondell (2004) include a discussion of theories of motor control from a historical perspective, where they note a general progression from reactive sensory-motor theories (i.e., reflexes as the building blocks of complex motor behaviour) to perception-action interactions, to organism-environment (i.e., the child as an active explorer of the environment) theories.

Although a number of descriptive measures have been developed that rate the effectiveness of movements, measurement of movement has limitations. Principal among these is motor redundancy (Latash, 2000), also referred to as the degrees-of-freedom problem (Tupper & Sondell, 2004), the notion that there are almost an infinite number of ways of completing a movement, furthermore, a single individual may complete a movement in more than one way on different occasions, even if the movement is made

under similar conditions. As such, it is impossible to judge movements in terms of the "best" way of executing them. Latash provided an analogy with language, pointing out that there are almost an infinite number of ways of formulating a sentence, but that there are constraints applied by rules, or grammar. As such, the question of assessment of motor skills can be redefined in terms of determining what rules the CNS applies. Measuring motor skills cannot be done in a way that conceptualises one way of moving to be better than others.

Despite the difficulty in defining clumsiness, there are a number of characteristics of movements that can be considered clumsy, a number of which are described by Latash (2000). Delays in meeting milestones in motor development are apparent. Slowness in initiating and completing motor tasks are frequently observed. Variability in movement patterns (e.g., kinematics, dynamics, and patterns of electrical stimulation of muscles) is also noted; this variability is seen in almost all disorders of motor skills, both developmental and acquired. Variability leads to movements that are not as smooth as possible, and most importantly, that are not reproducible.

In addressing the causes of clumsiness, researchers have focused on a wide variety of potential causes, including visual-perceptual skills (e.g., Parush, Yochman, Cohen, & Gershon, 1998), kinaesthetic-perceptual skills (e.g., Coleman, Piek, & Livesay, 2001; Piek & Coleman, 1995), visual memory (e.g., Skorji & McKenzie, 1997), reaction time (Hatzitaki, Zisi, Kollias, & Kioumourtzoglou, 2002), timing (Keele, Ivry, & Pokorny, 1987), force control (e.g., Piek & Skinner, 1999), movement planning (e.g., Rinehart, Bradshaw, Brereton, & Tonge, 2001), spatial orientation (e.g., Whiting,

Savelsbergh, & Faber, 1988), depth perception (Hatzitaki et al., 2002) and visual perception of movement (e.g., Sigmundsson, Hansen, & Talcott, 2003).

Any number of these variables may lead to dysfunction within the motor system. In determining which variable or variables may account for motor deficits in AS, we might look for clues from the descriptive literature discussed above. Green et al. (2002) among others suggested that ball skills, including catching, are particularly difficult for children with AS. It is possible that some of the causes of motor dysfunction listed above may affect such skills. In catching, it is important to view and predict the trajectory of the ball (visual sensitivity to motion), attain an appropriate hand position (motor planning), and determine the precise time to grasp the ball (timing control). Other skills may certainly be involved, but this subset of motor components provides a useful means of generalizing motor dysfunction to brain structures. Evidence linking such skills to discrete areas of the brain is presented below.

Neuromotor Components of Movement

A limitation of the literature on motor skills dysfunction in AS is the fact that assessment methods, with a few exceptions, have been largely descriptive, without linking deficits to motor control theories. Like many other areas of neuroscience, there have been difficulties in parsimoniously ascribing specific functions to the various structures that are implicated in motor control. Included below is a brief and largely incomplete discussion of current theories of the contributions of a variety of structures to movement.

The motor system is organized in an opposite direction than sensory systems; the flow of information is generally from the frontal cortex to the thalamus, basal ganglia, and cerebellum (all of which then feed back to the cortex via the thalamus), then via the motor cortex to the spinal cord and the peripheral effectors. The precise role of each of the major components, however, has yet to be determined.

The frontal cortex plays a number of roles in movement through the pyramidal system, the executive system responsible for voluntary initiation of movement that involves the frontal systems, corticospinal tract, and spinal motor neurons (Tupper & Sondell, 2004). The lateral orbitofrontal cortex is involved in response inhibition, recognition of reinforcing stimuli, stimulus-reinforcer learning, coding for changes in reinforcement contingencies, and emotionality, personality, and autonomic functions, including obsessional and compulsive behaviour. Simply stated, this area appears to be important in determining *what* to do, perhaps by eliminating or inhibiting what *not* to do (Bradshaw, 2001). The dorsolateral prefrontal cortex is Luria's (1973) tertiary integration area for the unit for programming, regulation and verification of activity, combining diverse information from posterior sensory and memory structures, holding and manipulating the information in working memory, and leading to goal directed behaviour and movement. Again, simply stated, this area is involved in determining *how* to complete an action (Bradshaw, 2001). The mesial and anterior cingulate regions are involved in motivation, exploration, and attention, and appear to be involved in determining *when* to complete an action (Bradshaw, 2001). Other areas of the frontal cortex involved in movement include the premotor and supplementary motor cortices, involved in control of movement requiring external and internal guidance of movement,

respectively (Cunnington, Bradshaw, & Iansek, 1996). These areas appear to have a "vocabulary" of movements that specify the trajectory of a movement and the context in which the movement occurs (Gazzaniga, et al., 1998). The supplementary motor cortex also appears to be involved in bimanual coordination and sequencing. Finally, the motor cortex, and to some extent the somatosensory cortex, through the pyramidal system, appear to be involved in coding movements more generally. Patterns of cellular activation (population vectors) recorded in the motor cortex suggest that this area is responsible for planning movements, but not for actually leading to movement by activating muscles (Gazzaniga, Ivry, & Mangun, 1998).

Other cortical areas are involved in movement, including the somatosensory area in the postcentral gyrus, and more extensive areas in the parietal lobe. The parietal is implicated in apraxia, an area of clinical interest that has led to a varied literature that is difficult to disentangle (Gazzaniga et al., 1998). The diagnosis of apraxia is made when a patient has coordination problems that cannot be linked to difficulty in controlling the muscles themselves. However, comprehension difficulties and frank aphasia are often comorbid, especially in left sided lesions, making the diagnosis more difficult. Apraxia may also follow frontal lesions, which is likened to deficits in implementing an action plan, while posterior lesions are implicated in retrieving and storing plans (Gazzaniga et al., 1998).

The frontal cortex is connected to, but considered somewhat distinct from the "vaguely defined extrapyramidal system" (Tupper & Sondell, 2004, p. 17) which includes the basal ganglia, cerebellum, substantia nigra, and red nucleus. The basal ganglia, including the caudate and putamen (together referred to as the striatum), as well

as the globus pallidus, nucleus accumbens, and olfactory tubercle (Afifi & Bergman, 1998), connect to the frontal lobes via five reafferent corticostriatothalamocortial loops. Input to the basal ganglia is through the striatum, while efferent fibres leave through the internal segment of the globus pallidus (GPi; these projections return to the premotor and supplementary motor areas via the thalamus) and the pars reticulata of the substantia nigra (SNr; these projections are to the superior colliculus and are involved in eye movement). The first loop is a skeletomotor loop implicated in the basal ganglia's contributions to movement. The second is an oculomotor loop involved in visual fixation. A dorsolateral prefrontal loop appears to be involved in maintaining or shifting attentional set, organization, retrieving memories, and flexibility. The lateral orbitofrontal loop is likely involved in personality and social restraint, empathy, inhibition of interference, and self-monitoring. Finally, a limbic, or anterior cingulate loop is important in drive or motivation; damage to structures within this loop leads to apathy, reduced initiative, and akinetic mutism. As such, dysfunction of the basal ganglia can have disparate effects on movement, cognition, and emotion (Afifi & Bergman, 1998; Afifi & Uc, 1998; Bradshaw, 2001). All five of the loops appear to include both direct pathways involved in the initiation of a response, and indirect, inhibitory pathways. In the skeletomotor loop, the direct pathways select and amplify desired movements, while the indirect pathways inhibit and suppress unwanted movement and prevent shifts in limb position from interfering with maintained motor activities, including normal posture (Houk & Wise, 2002). In Parkinson's disease, the lack of nigrostriatal dopamine in the skeletomotor loop leads to hypokinesia. Chorea, seen in Huntington's disease, involves the degeneration of the indirect pathway of the skeletomotor loop, leading to a

lack of inhibition and hyperkinesia. Similar effects in the other four loops appear to lead to problems in the cognitive and affective realms, with, for example, the release of tics or obsessive-compulsive behaviours.

The overall functions of the basal ganglia are still contested. Traditionally, based on clinical studies of degeneration of the various components, the basal ganglia were thought to be involved in disinhibiting, or releasing, motor responses and scaling the size of their initial agonist bursts, while inhibiting inappropriate activity (Bradshaw, 2001; Houk & Wise, 2002). The focus was entirely motor, without incorporating cognitive aspects that were thought to be fed from higher cortical areas. Clearly though, there are cognitive and affective correlates of basal ganglia dysfunction.

Noting that individual differences in the variability of force control correlate across effectors (e.g., finger, arm, and foot), Keele, Ivry, and Pokorny (1987) suggest that force control is mediated centrally, as opposed to being associated with separate effectors. The same relationship is found in variability in timing across effectors, such that the upper limits of such skills as typing speed can be predicted by assessing tapping speed in fingers or even feet. These researchers sought to determine whether force and timing control were associated with separate neuroanatomical structures (Lundy-Eckman, Ivry, Keele, & Woollacott, 1991). They separated children scoring in the clumsy range on a motor skills test into one of two groups, based on the presence of soft signs of motor dysfunction. Neurological "soft signs" appear to be sensitive signs of biological involvement in a variety of developmental neuropsychiatric disorders (Taylor, 1987), but their specificity in correlating with neuropathology remains to be determined (Roy, Bottos, Pryde, & Dewey, 2004; Voeller, 1998). Lundy-Ekman et al. (1991), however,

argued quite cogently for the validity of the soft signs they selected. One group (n = 10) demonstrated at least one of the following soft signs associated with dysfunction of the basal ganglia: choreiform movements (similar to the chorea associated in Huntington's with degeneration of the caudate and putamen), athetotiform movements (similar to the slow, writhing movements of athetosis, associated with dysfunction of the putamen and globus pallidus), or synkinesia (overflow movements associated with dyskinesias of basal ganglionic origin). The second group (n = 10) demonstrated at least one of the following soft signs associated with dysfunction of the cerebellum: dysmetria (inability to produce correct distance for movements), dysdiadochokinesis (inability to perform rapid, alternating movements), and intention tremor (low frequency oscillations during intended movements). They then tested both groups and a normal control group (n = 10) on measures of variability in force and timing control, arguing that disruptions in components of motor skills would likely lead to increased variability. They found that the group with soft cerebellar signs were more variable on timing tasks, implicating the speed of their internal "clocks," while the group with soft basal ganglionic signs produced more variable forces. The findings suggest that timing and force control are separate components of the motor system, and provides weak evidence to suggest that timing is associated with cerebellar function, while force control is associated with basal ganglionic function. Piek and Skinner (1999), using a similar methodology, have argued that force control is dependent on timing, and that clumsiness is more likely to be attributable to motor planning or central timing deficits that lead to force control deficits.

The basal ganglia have also been implicated in time estimation (Meck & Benson, 2002; Wing, 2002). Time estimation can be experimentally manipulated using

pharmacological substances that act on dopamine (D_2) receptors in the striatum. Clinical studies implicate timing problems, that is, difficulty reproducing intervals and distinguishing between intervals, in Parkinson's disease, and in schizophrenia, Attention/Deficit-Hyperactivity Disorder, and even in patients with prefrontal lesions, especially on the right side (Meck & Benson, 2002).

Another hypothesis of basal ganglia function is that they are involved in filtering and selecting certain courses of action for release, while inhibiting others. In this way, the basal ganglia can be conceptualized in a model that includes "set" (scaling the amplitude of movements to fit the context) and "cue" (a pre-movement plan or potential generated internally) (Bradshaw, 2001). The basal ganglia can therefore be viewed as holding in readiness set-related activity in the motor, cognitive, and limbic domains, and providing internal cues to initiate movement plans in the correct order and with the right timing. The basal ganglia may therefore be involved in "binding" and synchronizing cortical activity (including movements) in order to sequence and group thoughts or actions (Bradshaw, 2001).

Houk and Wise (1995) suggest that the striatal spiny neurons' capacity for computations leading to contextual pattern recognition are employed to recognize contexts relevant to behaviour, including the state of the organism, the desirability of an action, the actions planned in the near future, the location of targets of action, and sensory inputs relevant for selecting or triggering motor programs. Instead of simply releasing movements, they propose that the basal ganglia are central to recognizing the context appropriate for the release of movements. Failures of this system lead to a failure in detecting a context appropriate for suppression of an action, or falsely signalling a

context for action; these failures would lead to behaviour out of context, that is, tics, ballismus, chorea, derangement of volitional movement (athetosis), and repetitive, uncontrollable, obsessive thoughts. Within this framework, difficulty shifting set, or recognizing that there has been a change of context implicates the basal ganglia in the executive dysfunction that is seen in Parkinson's.

The role of the cerebellum is also controversial. The cerebellum consists of a small vestibulocerebellum that is separate from the main part of the cerebellum, and is involved in controlling balance and coordinating eye movements with movements of the body. The main part of the cerebellum is divided into two functional regions: the spinocerebellum (consisting of the midline vermis and the intermediate zone), which receives extensive multimodal sensory information, and the lateral neocerebellum, which is connected to cortical motor and sensory areas. The cerebellum is a large structure, and different areas have been implicated in different neurological soft signs. Damage to the spinocerebellum leads to difficulty smoothly controlling movement, especially postural muscles following vermal damage, and limbs following damage to the intermediate zone. The anterior superior aspect of the vermis appears to be involved in stability of gait and balance, while the lateral aspect of the hemispheres is associated with dysdiadochokinesia. Damage to the neocerebellum leads to ataxia and hypermetria, especially noticeable when sequencing movements (Sullivan, Desmond, Lim, & Pfefferbaum, 2002).

While Keele et al. (1987) and Lundy-Eckman et al. (1991) view the cerebellum as a being responsible for timing, other theories suggest that it is much more complex and may have a sensory role. A sensory role is suggested by the existence of extensive

multimodal sensory inputs (Gazzaniga et al., 1998), and by the cerebellum's activity in an fMRI study during a haptic discrimination task (Miall & Reckess, 2002). Cerebellar patients also display intention tremors and dysmetria when guiding movements visually, but not when guiding movements without visual feedback. When visual feedback is delayed 200 to 300 ms in a visual tracking task, the performance of healthy subjects resembles that of cerebellar patients. Miall and Reckess (2002) suggest that the reason for this is that under visual guidance of movement, feedback is dependent on rather slow (100 to 200 ms) pathways; they suggest that visual guidance of movement cannot be based on feedback systems, but rather on feedforward systems that are not dependent on sensory feedback. They suggest that the cerebellum functions as a "sensory predictor" (p. 212) that generates predictions about the consequences of motor acts, and uses the predictions to control and adjust movements. It also processes sensory information to update the last known information about the movements of the body in space.

Miall and Reckess (2002) suggest that the cerebellum forms detailed models of the motor system and combines them in certain ways to generate output commands. Houk and Wise (1995) suggest that the cerebellum's neurons, Purkinje cells, are suited to recognize and classify patterns of movements, and simply combine information about limb and target positions, velocity, and force along with afferent sensory information that signals when the goal of an action is about to be reached, and then inhibit cortico-cerebellar activity to terminate the movement. Like Miall and Reckess they suggest that Purkinje cells learn (through inputs that transmit training information that adjusts their synaptic weights) to recognize input patterns at a significant phase advance so as to predict goal acquisition, rather than depending on delayed feedback. Both Houk and

Wise (1995) and Miall and Reckess (2002) view the cerebellum as a means for using delayed sensory information to predict the current location of a limb in movement, and send output that signals the appropriate time to terminate actions. Excessively restrictive or permissive movement pattern classifications lead to errors of starting and stopping, amplitude, direction, acceleration, and force.

Although elaborate theories of motor function may suggest otherwise, Wing (2002), in his discussion of timing states, "neuropsychological data do not, at present, distinguish unequivocally between basal ganglia and cerebellar contributions to timekeeping and motor implementation. Moreover, both neural structures may contribute to common timekeeping functions in time interval production and perception" (pp. 15-16).

Attempts to Examine the Components of Motor Skills Dysfunction in AS

Weimer, Schatz, Lincoln, Ballantyne, & Trauner (2001) estimated that prevalence rates of "motor dyspraxia" in AS were approximately 80%. Their study was designed to more carefully assess the components of motor skills to provide a basis for further investigation. They assessed motor speed (Finger Tapping, Grooved Pegboard), fine motor control (Grooved Pegboard), motor planning [Finger-Thumb Apposition (repetitive and serial)], balance [Fegly-Graybiel tests of ataxia (including one-leg balance with eyes open and closed, and tandem gait)], visuomotor function (Grooved Pegboard, Trail Making, Developmental Test of Visuomotor Integration), and praxis (ideomotor, buccofacial, and ideational praxis, and Finger-Thumb Apposition) in ten male children and adolescents aged 9 to 19 with AS, and a control group of 10 age, VIQ, and

socioeconomic status matched males. Their results indicate that children with AS had more difficulty balancing on one leg with their eyes closed and walking heel-to-toe, that is, they found exactly the same symptoms as Iwanga et al. (2000). This is consistent with dysfunction of the cerebellar vermis (Sullivan et al., 2002). In addition, Weimer et al. (2001) found that their AS group was slower on repetitive Finger-Thumb Apposition using the dominant hand (which Sullivan et al. would classify as dysdiadochokinesia, a cerebellar hemispheric soft sign), and had more difficulty with whole body ideomotor postures (i.e., gesturing a boxer's stance, a baseball player's stance, a soldier marching in place, use of a shovel, and use of a vacuum) than the control group. Note that their assessment of ideomotor postures required subjects to assume a "role" which may invoke Theory of Mind-type dysfunction. They suggested that while the AS group showed no classic signs of motor skills dysfunction, their difficulties appeared to be the result of deficits in sensory, rather than motor pathways. The fact that balance was poor when their eyes were closed, and given that they do not complain of vertigo or dizziness, led Wiemer et al. to conclude that their difficulties were due to dysfunction of the proprioceptive sensory system, and not the visual or vestibular systems. Wiemer et al. did not discuss the implication of cerebellar dysfunction.

The findings of Weimer et al. (2001) and Iwanga et al. (2000) that people with AS have more difficulty walking is consistent with findings in the autism literature that suggest that autistic children have an unusual gait. Vilensky, Damasio, & Maurer (1980) studied the gait of 21 autistic children and 15 normal controls, and 5 nonautistic psychiatric controls using kinematic techniques available at the time. They found that autistic children took relatively shorter strides, had an elongated stance ("defined as the

period between initial foot contact and toe-off", p.647), flexed their thigh more at the point when the toe lifts off the surface, reduced knee extension at initial toe-off, contacted the ground with a more plantarflexed ankle, had markedly flexed elbows, reduced or uncoordinated upper-limb movements, and dystonic postures of the extremities. The normative data that they were able to use to make comparisons suggested that the gait of children with autism was more similar to the locomotor disturbances seen in parkinsonian patients than the gait seen in normal adults. Vilensky et al. therefore argued that the motor dysfunction seen in autism was related to pathology of the basal ganglia (particularly the striatum) and the frontostriatal circuitry (particularly connections to the anterior cingulate and supplementary motor cortex). It was these results that Szatmari et al. (1990) used to build their theory of frontosubcortical dysfunction in AS.

Hallet et al. (1993) used a similar technique using the VICON motion system, which provides a more sophisticated kinematic analysis of gait in 5 autistic adults and five matched controls. They also found an overall difference across several variables, particularly the range of motion of the ankle. They also found a significant number of neurological soft signs indicative of cerebellar deficits in their autistic sample, but unfortunately, had no normative data of gait in people with cerebellar damage to which they could compare their results. They argued that the findings reported by Vilensky et al. (1980) are non-specific and cannot be attributed to basal ganglia dysfunction until cerebellar dysfunction can be ruled out. In particular, they pointed to the lack of bradykinesia in AS. Beversdorf et al. (2001) found macrographia in adults with autism, rather than micrographia which is normally seen in Parkinsonian patients, and were

unable to specify whether their findings pointed to basal ganglia (i.e., choreoform symptoms of Huntington disease) or cerebellar abnormalities.

Rinehart, Bradshaw, Brereton and Tonge (2001) argue that poor performance on tests of motor skills in AS is not necessarily due to dysfunction of the motor system. They argue that people with AS, as well as those with HFA, are impaired in flexibly shifting their attention, one of the executive functions supported by frontostriatal systems (Hughes & Russell, 1993; Ozonoff & Jensen, 1999; Ozonoff, Rogers, & Pennington, 1991; Pennington & Ozonoff, 1996; Rinehart, Bradshaw, Moss, Brereton & Tonge, 2001). They devised a study in which they tried to measure motor preparation and motor execution separately. They devised a button pressing apparatus in which 6- to 19-year-old subjects with HFA (n = 11), AS (n = 12), and matched controls (n = 24) were required to press one of four buttons in response to light signals that followed a particular pattern. The pattern was a somewhat predictable back and forth movement between two buttons, but on each trial of 15 button presses, there was an "oddball" where the pattern was disrupted and one of the other two lights was illuminated. Following the oddball, the back-and-forth pattern continued as it had started until the end of the 15 button-press trial. Thus, a strategy of cautious responding up to and including the oddball stimulus, followed by a shift into rapid responding thereafter, would be the most efficient strategy. To measure movement preparation time (i.e., frontally mediated motor planning and inhibition/disinhibition) they used the button "down time" (or how long the button is held down while the subject is supposedly deciding where to move next). To measure movement execution (i.e., efficiency of the motor execution system) they used the movement time (or the time the finger is "in flight" between buttons). Children with

HFA executed their movements as efficiently as controls, but their motor preparation was unusual in that they did not appear to employ a particular strategy to improve performance; they responded to the oddball and the following stimuli at the same rate as the previous stimuli, without speeding-up like the controls. Children with AS also executed their movements efficiently, but their motor preparation actually *slowed* following the oddball stimulus, implicating difficulty in flexibly shifting to a different cognitive set. The authors suggested that the lack of anticipation shown by the HFA group may reflect anterior cingulate (AC) involvement, as the AC is involved in attention and motivation for action. They suggest that the deficit in movement preparation shown in AS may reflect dysfunction of the AC/supplementary motor area (SMA) circuitry; the SMA dysfunction would lead to additional difficulties internally generating a motor program. Unfortunately, this study involves a limited assessment of motor skills; while it does implicate executive dysfunction in HFA and AS, it does not explain whether executive dysfunction can account for motor skills dysfunction addressed above. For example, balance and walking do not involve the same level of cognitive flexibility and strategy formation that are inherent in Rinehart et al.'s task. Their lack of findings in motor execution do not contribute to the literature that has documented much more generalized impairment than speeded hand-eye coordination. Smith (2004) noted too, that the complexity and attentional demands of the task may also have affected the outcome. However, the implication that children with AS have difficulties flexibly shifting set suggest problems with detecting the appropriate context to inhibit or disinhibit a plan of action, similar to what is seen in Parkinson's disease, and implicating the basal ganglia.

Hughes (1996) tested her theory (Hughes & Russell, 1993) of fronto-striatal mediated executive dysfunction in autism by hypothesizing that motor planning would be deficient in autistic patients. She tested 36 12- to 13-year-old autistic subjects, 24 IQ matched subjects, and 28 normal age-matched controls on a paradigm that required subjects to pick up painted wooden rods and then insert them into coloured discs in different orientations. Subjects could complete the task with either a properly planned grip that would result in a comfortable grip at the end of the movement, or a poorly planned grip that would result in an awkward grip. She found that the autistic subjects planned their motions poorly, ending up with awkward grips more often, suggesting that people with autism are developmentally delayed in acquiring the skills necessary to execute goal-directed motor acts. She suggested that people with autism may have difficulty with one or more of several different processes of executive control: sequencing ability, predicting the consequences of motor acts (though perhaps on a higher level than that discussed earlier in terms of cerebellar predictions of the sensory outcome of movements), or visual control of movement (i.e., using external feedback to guide behaviour).

Finally, though not actually attempting to study quality of movement, Müller et al. (2001) noted differences in the regularity of button-presses between an autistic and a control group; the autistic group was able to press the button *faster*, but less regularly. This may implicate poor timing of movements in PDDs. Interestingly, the activation of their motor cortex was monitored with fMRI, with the autistic group showing *less* activation.

Possible Confounds in the Assessment of Motor Skills in AS

In their review of deficits in imitation and movement in autism, Smith and Bryson (1994) point out that there are two, not mutually exclusive explanations of motoric deficits. First is an impairment of movement programming, a non-social information-processing deficit. Second is a conceptual, perhaps executive deficit in joint attention, a non-social precursor to the representation of the mental state of others, an important process in social relationships.

Baron-Cohen (1989; Camaioni, Perucchi, Muratori, & Milone, 1997; Goodhart & Baron-Cohen, 1993) studied both the comprehension and production of a particular movement in autistic children: pointing. They noted that there are at least three different types of pointing that range in level of abstraction from non-communicative pointing, to pointing to establish joint attention. They compared autistic children, children with Down Syndrome, and normally developing controls and found that though children with autism were unimpaired in terms of lower level pointing, they were severely impaired in comparison to the two control groups in terms of pointing to establish joint attention. They interpreted their findings as being indicative of deficits in the developmental precursors of "theory of mind". In terms of measurement of movement, it is therefore important to control for any sort of confounds that involve interpersonal communication or theory of mind (c.f., Weimer et al., 2001).

Visual Perception of Movement

While planning and execution of movement are important areas of investigation in addressing "clumsiness" in AS, deficits in visual perception of the objects and space

upon which the individual is acting are also plausible etiological factors (Laszlo, Bairstow, Bartrip, & Rolfe, 1988; Sigmundsson, Hansen, & Talcott, 2003). Smith and Bryson (1994) propose that a general representational deficit may exist in autism, what they term a developmental spatial neglect syndrome, which implicates lower level attentional and perceptual rather than social-cognitive processes in imitation deficits in autism. While a complete review of visual systems is clearly beyond the scope of this section, a description of major efferent extrastriate systems follows.

In 1982, Ungerleider and Mishkin identified two separate visual streams: one that projected dorsally from the striate cortex to the posterior parietal lobe, and one that projected ventrally to the inferotemporal cortex. They hypothesized that the dorsal stream computed spatial information such as the location of an object in space ("where"), while the ventral stream computed information about objects, such as their size, shape, orientation, and colour that was ultimately involved in object recognition ("what"). Livingstone and Hubel (1988) noted the existence of separate streams of visual information evident at the level of retinal ganglia cells: Pα or Y-cells which respond transiently to visual stimuli, have relatively large receptive fields, and project to the magnocellular area of the lateral geniculate nucleus of the thalamus (LGN), and Pβ or X-cells which have small receptive fields, respond in a sustained fashion to visual stimuli, and project to the parvocellular layers of the LGN. Livingstone and Hubel attempted to correlate these two systems with extrastriate projections to the dorsal and ventral streams. To a large extent, this has been shown to be the case, although there is much more "cross-talk" between the systems within the striate cortex than was hypothesized (Milner & Goodale, 1995).

Milner and Goodale (1995) re-evaluated the theory that the dorsal stream was involved in spatial information while the ventral stream was involved in object recognition. They invoked evolutionary theory to suggest that distinctions between the two systems are not a result of their input, but rather, their functional output. They suggest that the ventral stream is involved in representing the relatively enduring aspects of objects and their relations to one another, while the dorsal stream is involved in computing the information required for visually-guided movement. Rather than coding solely spatial information, the dorsal system codes for egocentric effector-specific spatial information. In addition, there appear to be cells sensitive to the visual appearance of objects to be acted upon; for example, so that effectors can assume the proper orientation to grasp an object. They suggest that the differences between the ventral and dorsal stream are essentially "perception versus action" (p. 65). In the present discussion of clumsiness in AS, it is dysfunction within the dorsal "action" system that is of interest.

Milner and Goodale support their view of the role of the dorsal stream with several lines of evidence. One such line is based on the observation that this area, in contrast to the rest of the visual system, is functionally silent to visual stimuli while an animal is under anaesthesia, suggesting that this system requires interaction between the organism and its environment.

The dorsal stream might best be conceptualized as providing those visual functions that are available to a person who demonstrates cortical blindness. Cortical blindness is the result of selective damage to the primary visual area, V1. Based on anatomical inputs to the ventral and dorsal streams, Milner and Goodale demonstrate that damage to V1 deafferents the ventral stream, resulting in the phenomenology of

blindness, but that retinal afferents to the pulvinar nucleus of the thalamus project directly to the dorsal stream without passing through V1, thus leaving the dorsal stream relatively intact. Individuals with cortical blindness have no conscious perception or recognition of objects, but can use visual stimuli or "blindsight" to move through and act upon the environment. When asked to localize visual stimuli they often profess ignorance, but can point to the stimuli at levels far greater than chance. Milner and Goodale describe this as "action without perception" (p.68).

Studies of visual perception in PDDs have generally focused on the role of the ventral visual stream. Here, an unusual distinction has been noted. While children with PDDs have demonstrated facial recognition deficits, adults with autism and AS actually respond faster than normals on the Embedded Figures Test, in which they need to attend to fine detail while ignoring the gestalt (Joliffe & Baron-Cohen, 1997, Kenworthy, et al., 2002). Clearly there are abnormalities within the ventral stream that have yet to be elucidated.

Several studies have addressed the integrity of the dorsal stream in individuals with PDDs. Gepner and Mestre (2002) assessed how children with autism and AS reacted to the perception of movement. While some studies have used the "moving room" phenomenon to test this, these researchers projected a graphically simulated tunnel, projected onto a large screen. Participants, including nine normal children, three with autism, and three with AS stood on a force platform in front of the screen. Without any visual manipulations, the three groups were equally stable. However, compared to the normal controls perceiving the movement, children with autism were hyporeactive to visual motion while children with AS were hyperreactive. With their eyes closed, AS

children were less stable that the other two groups. These differences were not statistically significant, likely due to the low power associated with the extremely small sample sizes. Assuming replication with larger sample sizes yielding statistically significant results, this appears to suggest that children with AS are more affected by visual motion and that their postural stability is dependent on visual inputs. Parallels to Weimer et al. (2001) are of interest, as the results implicate deficits in proprioception in AS, given that they were overwhelmed by the visual inputs and unable to rely on kinaesthetic input to steady themselves. Further, Masterton and Biederman (1980) demonstrated that when reaching for objects with and without visual input, children with autism were more reliant on proprioception than were normals.

Given the abnormalities in postural stability, Spencer et al. (2000) sought to determine whether there were differences in perceptual sensitivity to movement between children with autism (n = 23) and normal controls (n = 50) matched for verbal intelligence. They used an array of dots that oscillated back and forth; the child's task was to find a group of dots that oscillated in reverse phase. They found that their autism group showed normal age related improvements, but at each of the four age groups (ages 7 to adult), their performance was inferior to that of the normal controls. The authors suggested that this was indicative of dysfunction of the dorsal visual stream. Milne et al. (2002) replicated the study with autistic children (n = 25) and a different control group, matching by age and non-verbal intelligence (n = 22). They also found that children with autism had an unusually high motion coherence threshold, which they interpreted as indicative of magnocellular impairment, possibly within the parietal lobes. Finally, Pellicano, Gibson, Maybery, Durkin, and Badcock (2005) also found that autistic (n = 15)

and PDD NOS (n = 5) children performed worse on a global motion task, but not on a Flicker Contrast Sensitivity task. The Flicker Contrast Sensitivity task assesses low-level visual processing and does not necessarily involve movement, but would implicate magnocellular, and perhaps dorsal stream involvement.

While a global motion task has not been done with children with AS, a similar experimental paradigm was used with 'clumsy' children. Sigmundsson et al. (2003) assessed the 13 clumsiest children (i.e., the lowest scores on the Movement-ABC) in a school-based sample of 54 children, and compared their performance on the movement perception test to the 13 children who scored the best on the Movement-ABC. They found significant differences in their sensitivity to movement, and also to their sensitivity to forms, which would also implicate ventral streams. These authors did not control for IQ, but argue that their results are likely to be valid given that all children were in normal classrooms and free of learning disabilities. They also point to research (Braddick, O'Brien, Wattam-Bell, Atkinson, & Turner, 2000) suggesting that while cortical areas involved in the perception of form and motion with this paradigm have been shown not to overlap in fMRI research, they do not appear to map well to the dorsal and ventral streams.

The failure of such perceptual sensitivity to movement and flicker tasks to correlate with the dorsal and ventral streams in imaging research may relate to the task demands. Milner and Goodale suggest that the dorsal system is involved in vision *for action*. Visual sensitivity to movement is certainly implicated in the dorsal stream, but may also be involved in ventral stream processing.

The dorsal stream is activated when subjects view people carrying out actions. 'Mirror neurons' in the primary motor cortex are activated when subjects view human movements, though not to the extent that they are activated when the person carries out the action themselves (Avikainen, Kulomäki, & Hari, 1999; Stevens, Fonlupt, Shiffrar, & Decety, 2000; see also Smith, 2004). Avikainen et al. (1999) compared four children with AS and one with autism to eight controls using magnetoelectroencephalography (MEG). They found that the response of the mirror neurons in these children was similar to the response seen in the normal children. These results are, however, limited by the extremely small sample sizes and the possibility (depicted in Figure 2), that a single outlier may have affected their results.

A recent study (Blake, Turner, Smoski, Pozdol, & Stone, 2003) has addressed the ability of autistic children to differentiate between biological and non-biological motion, with the aim of understanding whether deficits in autism may be attributable to differences in perception of other people's movements. They had 12 children with autism and nine controls view point-of-light animations either of people making movements or random dot movements. Their task was to determine whether or not the animation depicted a person. Using signal-detection methods, they determined that there was a significant difference between groups, and that within the autism group, there was a significant correlation between this task and severity of autistic symptoms as measured by two clinical measures. Furthermore, on a measure of non-moving form detection there was no difference between groups. There was, however, a difference in mental age between their samples, which introduceed a possible confound. Nonetheless, these

authors suggest that there is convincing evidence of dorsal stream pathway impairment in autism.

Using such visual perceptual paradigms may prove useful in assessing the visual systems in AS. Milner and Goodale (1995) propose that the ventral stream would be active in recognition of the identity of a person, while the dorsal stream would be involved in the analysis of their actions and behaviour. It may be that while children with AS would be able to identify people well, they would have difficulty visually perceiving their movement. Therefore, the question of whether there are dorsal stream deficits in children with AS may not yet have been adequately posed.

Hypotheses

There appears to be general agreement in the literature that children with AS are clumsy. Questions remain, however, in regard to the reasons why they might be clumsy. It is the patterns of motor or perceptual-motor differences that may ultimately be associated with aspects of AS, and this is the direction in which research must now turn (Smith, 2004). At least three causes of clumsiness were identified as possibilities (discussed above in terms of the 'catching' analogy), and the present study was designed to address each of these three possible causes separately.

Movement timing, that is, the ability to independently produce and perceive rhythmic, accurately timed movements (i.e., Lundy-Eckman et al., 1991) was hypothesized to account for some degree of motor dysfunction. Certainly timing production deficits have been reported in the autism literature (Müller et al., 2001), but measures of the perception of timing have not. The present study included an analysis of

both the perception of variations in intervals, and the ability of the motor system to produce accurately timed intervals.

Movement planning (e.g., Hughes, 1996), an executive premotor or prefrontal function of planning out a movement before executing it was also hypothesized to be implicated in motor dysfunction. It was thought possible that the clumsiness seen in AS is a result of ineffective planning of movements. The present study addressed motor planning in a planned grasping task.

The third possible hypothesis related to processing of what Milner and Goodale (1995) term 'vision for action'. It was hypothesized that there are visual deficits that undermine the attempts of children with AS to accurately represent their environment when acting upon it. This was measured in four steps. First, visual perception of static patterns was measured to serve as a control measure. Second, a methodology similar to that used by Sigmundsson et al. (2003), Spencer et al. (2000), and Milne et al. (2002) that addressed the AS group's threshold in the perception of movement was completed. This methodology appears to address basic movement perception at the level of areas V5 and V3 (Braddick et al., 2001), but not necessarily the dorsal stream more generally. So in addition, a measure of the accuracy of the AS group's perception of human movement was administered; this required participants to view human movements, which relies on the activation of mirror neurons in the dorsal stream. Ostensibly, the perception of movements and the ability to produce them are related (Avikainen et al., 1999; Jacobs, Pinto, & Shiffrar, 2004). Finally, the ability of children with AS to maintain postural stability in the face of perceived movement was hypothesized to provide information regarding the integrity of their dorsal stream (e.g., Gepner & Mestre, 2002). This relies

not on movements to visually perceived targets, but rather, movements to counteract the visual perception of motion without overriding proprioceptive afferents that signal that no motion has occurred.

Analysis of these three variables included a comparison to a matched control group to determine whether there were differences between children and adolescents with AS and normal controls on any of these variables.

A final variable was included to capture clumsiness. A standardized test of motor skill was also administered. The purpose of this variable was to provide a criterion of clumsiness to be accounted for by the three variables that look at specific components of movement and motor control.

Method

Participants

Permission to involve human subjects in the present study was granted by the University of Victoria Human Research Ethics Committee (Protocol Number 069-04, Project Title: Motor Skills in Asperger's Disorder), and the Vancouver Island Health Authority's Child, Youth, and Maternal Health Research Advisory Committee (letter dated March 11, 2004). The permission of this committee was required because of their involvement in recruiting participants. Consent forms were approved by both committees, and are included in the Appendix. In addition, the Mental Health Operating Group at the Queen Alexandra Centre for Children's Health (QACCH) endorsed inviting their clients to participate in the study.

Most of the participants were recruited through a family and peer support group for children and adolescents with Asperger Disorder at the QACCH by (1) having an impartial third party address the parent group on three occasions to maximize the number of parents seen, and (2) through an electronic mail distribution list to all of the families potentially in this group. This electronic mail notice theoretically reached all families with a child or adolescent diagnosed through the QACCH on Vancouver Island. Two others participants were recruited through word of mouth, and one was recruited through the Autism Society of BC newsletter. Exclusionary criteria included the presence of a clinically significant language impairment (which would preclude a diagnosis of AS in favour of autism), significant neurological disorders or physical anomalies that would interfere with movement, and a Full-Scale IQ of at least 70. Control participants were recruited through advertisements throughout the city, though one was an unaffected

sibling of a child with Asperger Disorder. Exclusionary criteria included a history of psychiatric or neurological diagnoses or significant learning disabilities. However, no participants were excluded due to any of these criteria. Consent for all participants under 19 years of age was provided by their legal guardian.

There were two samples in this study, one group of 14 children and adolescents with a diagnosis of Asperger Disorder, and a control group of 16 individuals matched for age. All participants with AS were male, so no female controls were sought. This bias toward a male sample is in keeping with estimates of the gender ratio of AS in the population (i.e., 10 to 15 times more common in males). Children and adolescents of all ages were recruited to increase the sample size, as there is little literature to suggest that the motor symptoms of AS change over time [though Iwanga et al. (2000) felt that a strength of their study was the relative youth of their sample]. There was no difference between the average age of the Asperger group (mean = 14.14 years, SD = 4.80 years, range = 7.75 to 23.00 years) and the control group (mean = 14.08 years, SD = 4.61 years, range = 7.42 to 23.67 years), $t(28) = .034$, $p > 0.5$. The diagnosis of Asperger Disorder was, in almost all cases, made by a multidisciplinary team at the QACCH Mental Health Program, though several of the diagnoses were made by other psychologists and psychiatrists. All diagnoses were confirmed using symptom checklists to ensure that clinically significant levels of the symptoms outlined in Table 1 were present. Four symptom inventories were used, which constitute essentially all inventories measuring symptoms of AS currently developed (these symptom inventories are described in the "Materials" section below). These include the Gilliam Asperger Disorder Scale (GADS, Gilliam, 2001), the Asperger Syndrome (and high functioning autism) Diagnostic

Interview (ASDI, Gillberg, Gillberg, Råstam, & Wentz, 2001), and the high-functioning Autism Spectrum Screening Questionnaire (ASSQ, Ehlers, Gillberg, & Wing, 1999). These questionnaires were completed by the parent who accompanied the participant, though five of the older control participants were not accompanied and parents did not return the questionnaires, though an addressed stamped envelope was provided to their child to pass along to them. One of the parents of a child with Asperger's missed several items of the ASDI, so the score was not included. In addition to the above parent questionnaires, the Asperger's Questionnaire (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) was completed by the participant themselves. One control participant missed one of the three pages, so his score was not recorded. Scores on these questionnaires are presented in Table 2.

Table 2. Symptom severity across diagnostic inventories by group.

	Asperger Group (N = 14)			Control Group (N = 16)		
	N	Mean (SD)	Range	N	Mean (SD)	Range
GADS	14			11		
Social Interaction		10.64 (2.47)	6-13		1.18 (0.60)*	1-3†
Restricted Behavior		10.50 (2.47)	7-14		1.45 (1.04) *	1-4†
Cognitive Patterns		10.50 (2.77)	5-14		2.64 (2.01)*	1-6
Pragmatic Skills		10.64 (2.73)	5-15		1.36 (0.67)*	1-3†
Asperger's Quotient		104.0 (14.01)	77-127		44.36 (5.10)*	40-57†
ASDI	13			11		
Total Score		13.69 (3.42)	7-19		0.36 (0.67)*	0-2†
Criteria		5.08 (1.04)	3-6		0.09 (0.31)*	0-1†
ASSQ	14	30.21 (6.12)	18-42	11	2.72 (2.37)*	0-8†
AQ	14	26.86 (7.77)	18-45	15	17.13 (6.09)*	7-28

* $p < .001$

† Non-overlapping ranges

As is evident from Table 2, there was a clear separation of symptom frequency between the groups. All measures of symptomatology were significantly different

between the two groups at the $p < .001$ level. For seven of nine measures, the distribution of scores did not overlap at all. On the Cognitive Patterns scale of the GADS, there was mild overlap, but the overlap was more substantial on the self-report AQ. This measure was normed on adults and may require more self-awareness than is typical of children and adolescents, particularly those with Asperger Disorder. In terms of diagnostic cut-offs, the GADS showed clear separation with only one of the participants with Asperger Disorder in the "Borderline" range, and the rest in the "High/Probable" range, and all of the controls in the "Low/Not Probable" range. The authors of the ASSQ suggest a cut-off score of 19 for diagnosis with Asperger Disorder; again, only one participant had a score below 19 (his score was 18), and no controls had a score above 8. On the ASDI, a cut off of 5 critical items is recommended; four people with Asperger Disorder had scores less than 5 (three had a score of 4 and one had a score of 3). Interestingly, one of the criteria on the ASDI relates to motor clumsiness. All but 2 people in the AS sample were noted to have performed poorly on neurodevelopmental examinations of motor clumsiness in the past, while no one in the typically developing control group endorsed this item. The AQ (Baron-Cohen et al., 2001) has a suggested cut-off score of 32. Only three participants rated themselves that high on the AQ in the present study; all three had diagnoses of AS. Although there was clearly a significant difference between the groups on this measures, a number of factors may have led participants with AS diagnoses to rate themselves lower than the established cut-off for the measure. First is that for the most part, the normative data for the AQ was based upon university aged students, whereas most of the participants in the present study were considerably younger. Second, the AQ is self-rated, so some participants with AS may

have been embarrassed to acknowledge symptoms of AS. Finally, self-rating forms require some level of self-awareness which typically develops in adolescence. Interestingly, in the control group there was a trend toward a negative correlation such that in older participants, scores were lower. However, in the AS sample, there was a trend toward a positive correlation, suggesting that with age, participants with AS report more symptoms. This could suggest that older participants are more aware of their differences. Because of sample size issues, it was not possible to look at differences in correlations between older and younger participants in each group directly.

In terms of requirements to exclude participants, one participant's data was reviewed. The person with the borderline GADS score was the same person to have the ASSQ score of 18, and had an ASDI score of 4. This person was diagnosed by a reputable psychologist and psychiatrist; his mother noted that he was diagnosed with a "mild case" of Asperger Disorder. He was also one of the oldest people in the study at 23 years, 0 months. Despite his borderline parent ratings, his AQ score was 33, which is above the average for the group. As such, he was judged to meet the criteria for Asperger Disorder and was retained in the subject pool. All other participants demonstrated clear differentiation from controls. There are no empirically validated measures that differentiate Asperger Disorder from high-functioning autism, so it was not possible to definitively rule out the possibility that one or more of these participants may have high-functioning autism. Such a distinction is a clinical decision not aided by any standardized methods; it is assumed that the diagnosticians took a history of language delay into account in their differential diagnoses.

Participants' intellectual abilities were also measured to ensure that no group differences in IQ were present. IQ is correlated with motor ability such that if there were significant differences between the groups, IQ would need to be statistically controlled. The Wechsler Abbreviated Scale of Intelligence (WASI, Wechsler, 1999, described in more detail below in the "Materials" section) Vocabulary subtest was used to estimate verbal intellectual ability; there was no difference between the Asperger (T = 60.14, SD = 9.44) and control (T = 57.81, SD = 9.29) groups, $t(28) = 0.68$, $p > .50$. There was only a small effect size favouring the AS group (Cohen's d = .26). Likewise, there was no difference between the Asperger (T = 57.14, SD = 7.43) and control (T = 52.63, SD = 9.29) groups on the Matrices subtest, a measure of nonverbal intellectual ability, $t(28) = 1.46$, $p > 0.10$. There was a medium effect size (Cohen's d = .55) according to Cohen's (1965) criteria, again, favouring the AS group over the control group. And finally, as might be expected based on the above data, there was no difference between the Asperger (Standard Score = 115.43, SD = 12.76) and control (Standard Score = 108.94, SD = 10.68) groups on the two-subtest WASI Full-Scale IQ (FSIQ-2), $t(28) = 1.52$, $p > 0.10$. Again, this was a medium effect size (Cohen's d = .57) favouring the AS group. As such, the groups were essentially equivalent in terms of intellectual abilities and IQ was not considered to be a potential confound requiring statistical control.

Materials

Unfortunately, there are few adequate measures of AS symptomatology that differentiate between AS and high functioning autism (Howlin, 2000). Even measures that are typically used in diagnostic clinics, such as the Childhood Autism Rating Scale

(CARS; Schopler, Reichler, & Rochen Renner, 1988), the Autism Diagnostic Interview – Revised (ADI-R; Rutter, Le Couteur, & Lord, 2003) and the Autism Diagnostic Observation Schedule – Generic (ADOS-G; Lord, Rutter, DiLavore, & Risi, 1999) are sufficient only in separating children with Pervasive Developmental Disorders from their typically developing peers. These measures are insufficient in diagnosing differences between AS and autism, and given that these devices were not designed with AS in mind, they were not deemed appropriate for the present study. There are a number of instruments that were designed specifically to address AS and appear to be somewhat sensitive to its presence, including the screening questionnaire for Asperger Syndrome and other high functioning autism spectrum disorders in school age children (ASSQ, Ehlers et al., 1999), the Asperger Syndrome Diagnostic Interview (ASDI, Gillberg et al., 2001), and the Gilliam Asperger Disorder Scale (GADS, Gilliam, 2001). Parents of participants in the present study were asked to complete all three measures.

The ASSQ (Ehlers, et al., 1999) is a 27-item questionnaire that was developed by interviewing 110 children referred to a neuropsychiatric clinic, including 21 children with autism spectrum disorders, 58 with disruptive behaviour disorders, including Attention-Deficit/Hyperactivity Disorder, but not autism spectrum disorders, and 31 with Learning Disorders but not autism spectrum disorders. A separate sample of an additional 34 boys aged 6 to 16 with AS was used to determine the validity of the scale. The ASSQ is completed by parent or caregivers, and has good test-retest (r = .94) and inter-rater (r = .66) reliability. This measure can be used to differentiate between children with and without PDDs with 82% sensitivity when a cut-off score of 19 is used (Ehlers, et al., 1999). This was the suggested cut-off for making decisions about differential diagnoses

with other neuropsychiatric disorders, and was used in the present study. A copy of the items on the ASSQ is in the public domain and is reprinted in the Appendix.

The ASDI (Gillberg et al., 2001) was designed to address the diagnostic criteria advanced by Gillberg and Gillberg (1989), as opposed to the DSM-IV or ICD-10 criteria. The scale, which is in the public domain and is therefore included in the Appendix, is comprised of 20 items that address the Gillbergs' six criteria for diagnoses. These criteria include (1) severe impairments in reciprocal social interaction (extreme egocentricity), (2) all absorbing narrow interest pattern(s), (3) imposition of routines, rituals, and interests, (4) speech and language peculiarities, (5) non-verbal communication problems, and (6) motor clumsiness. The measure was validated using a sample of 24 participants aged 6 through 55, 17 of whom had a neuropsychiatric disorder. Thirteen were found, through clinical interview, to have AS, and all of these individuals met five or more of the six criteria for AS on the ASDI. Of the other 11 participants, only one met as many as five criteria, and this individual was diagnosed with dissociative personality disorder. The measure was noted to have good inter-rater (kappa = .91) and test-retest (kappa = .92) reliability.

The GADS is a 32-item scale parent-interview form normed on a population of 371 individuals (314 males) with AS from 46 American states, Canada, Great Britain, Mexico, Australia, and other countries (though only 31 were from outside of the USA). It can be used for people from the ages of 3 to 22 years of age. The coefficient alpha for the internal consistency reliability for the primary score, the Asperger's Disorder Quotient (ADQ) was .87 in a sample of 360 people with diagnoses of AS. The test-retest reliability of the ADQ was .93, though this estimate was based on a sample of only 10

people with AS. In a study of 371 people with AS and 78 without, the GADS scores were able to classify 83% of people correctly. Scores were, as hypothesized, unrelated to age. The subscales of the GADS correlate significantly with one another, and with the overall ADQ. There were significant group differences between the GADS scores of people with AS and people with other disorders, including autism (n = 54) or other disabilities (including mental retardation, ADHD, learning disabilities, etc; n = 35). The corresponding autism rating scale, the GARS, has been criticized for being insufficiently sensitive (South et al., 2002); however, no other research was available on the GADS. The ADQ correlated significantly (r = .58) with the Autism Quotient on the GARS.

In addition, because multiple informants improve the validity of the information gathered, the self-report Autism-Spectrum Quotient (AQ, Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) was also administered. This measure was developed on a heterogeneous sample of 58 adults with AS/HFA (mean age 31.6 years, gender ratio representative of AS at 3.5:1 male:female). This sample was compared with a sample of 174 randomly selected adults. A cut-off score was determined above which 79.3% of the AS/HFA group scored, compared to 2% of controls. Interesting group differences were also detected with this measures within 840 Cambridge undergraduates in a variety of disciplines and 16 winners of the UK Mathematics Olympiad, showing a trend toward higher scores for science, and particularly mathematics students, which was used to suggest that AS may lie on a spectrum of impairment. This measure is available on-line, and is included in the Appendix. The AQ is a self-report measure, and was completed by the participants themselves (not their parents) at the end of the testing session.

All participants completed the Full-2 scale of the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). This scale is comprised of two subtests, Vocabulary, which required participants to provide definitions of words, and Matrices, which required participants to analyze patterns in geometrical forms, and choose the missing piece using a five-item multiple choice format. The WASI was selected for use in the present study because it provides a single measure for assessing intellectual ability across the adolescent age range, and correlates significantly with other Wechsler scales, including the Wechsler Intelligence Scale for Children and the Wechsler Adult Intelligence Scale.

Procedure

Participants with AS were generally contacted first, so that demographics for matching the control group could be collected. Consent forms were reviewed with participants and their parents together when possible; some of the older participants had reached the age of majority and were not accompanied by a parent or guardian. When present, parents were provided with a copy of the consent form for their records, and a copy was stored in the laboratory, separate from the data collected.

Parent-completed symptom rating forms (including the ASSQ, the ASDI, and the GADS) were provided to parents so that they could complete them before the end of the testing session; in cases in which parents did not attend the session, participants were provided with the forms and an addressed, stamped envelope so that their parents could complete the forms and mail them in. As such, data from the rating forms were not used *a priori* to make decisions as to which group to assign participants, but were used later to

confirm group differences. A number of older participants, particularly controls, indicated that their parents did not live in the city and that they may be unable to have the forms completed.

Basic demographic data were recorded for matching purposes, including age, which was calculated to the nearest month. Date of testing, age, gender, and names of participants and their parents were recorded and participants were assigned a pre-determined random code number. The code number did not identify the diagnostic status of the participant so that later analyses could be completed blindly. All forms used in the study were stamped with a matching code number. All participants also had an anonymous (with the exception of their code number) Testing Checklist to ensure that data was completed, including parent measures, many of which were mailed in after the fact.

Participants began testing and completed the subtests of the Wechsler Abbreviated Scale of Intelligence (WASI, Wechsler, 1999) necessary to calculate the "Full-2" IQ score. Following completion of this test, participants completed the following measures in the following order: Timing Perception Test, then the Coloured Rod Test, the Motion Perception Test, the Form Perception Test, Biological Motion, Dean Woodcock, Tapping Beat, Postural Stability Test, and then the Asperger's Questionnaire. Each of these measures, many of which were constructed solely for the present study, is described in turn below. Once testing was complete, participants were paid a $15 to $20 honourarium in appreciation for their time. A final checklist was reviewed to establish that the data, electronic and paper, was complete. Data collection took 2.5 to 3 hours per participant.

Data addressing the study's hypotheses was derived from the following measures.

Hypothesis #1: Timing Production and Perception.

Detection of group differences in timing ability was measured in two ways. The first method was to use a perceptual discrimination task. The second method was to have participants produce a rhythm by tapping a computer key at a constant rate. Each of these measures is described below.

Timing Perception Test

In order to determine whether timing abilities are affected generally or specifically within the motor system, a motor-free timing perception task was used. Lundy-Ekman et al. (1991) presented two tones separated by 400 msec, and then one second later presented two more tones. Participants were required to report whether the interval between the second pair of tones was shorter or longer than that of the first pair of tones. A similar task was used in the present study using a computer program written for this study. For a random duration (varying between 5 and 10 seconds), a pacing beat was played with an interstimulus interval of 400 msec; that is, a pseudorandom number of these tones were sounded with this constant interstimulus interval. Then, a second beat played, again, for a random duration between 5 and 10 seconds with an interstimulus interval of $400 + X$ msec on four of eight trials, or an interstimulus interval of $400 - X$ msec on the remaining four trials. The order in which the eight tones were presented was randomized. The participant was required to judge whether the second rhythm was longer or shorter than the pacing beat. The pacing beat was presented each time for eight trials, and if the person could get 6 of the 8 trials correct, they moved to a more difficult

trial; if they could not answer correctly on 6 of the 8 trials, they moved to a less difficult trial until a perceptual threshold was established. The threshold of 6 out of 8 trials was selected to allow room for error, but to establish that the participant's success was above chance. In the first block of eight trials, the second beat had an interstimulus interval of either 410 or 390 msec (four of each in a random order); as such, each trial required that the participant perceive 10 msec differences, which was judged to be a noticeable difference by most people on whom the task was piloted. If the participant passed the first block, they moved to eight trials in which the interstimulus interval was either 408 or 392 msec (i.e., an 8 msec discrimination). Blocks then moved through 6 (406/394 msec), 4 (404/396 msec), and 2 (402/398 msec) msec discrepancies from the 400 msec pacing beat. If the participant could not answer correctly 6 out of 8 times on the first block, the interstimulus intervals increased by 2 msec to 412/388 msec, etc., until they were able to answer correctly 6 of 8 times. In this case, the perceptual threshold would continue to be the shortest interstimulus interval block in which they answered accurately.

Because the task was performed on the computer, it was possible to measure a reaction time, given that it was possible that the groups may have differed in the amount of time needed to make a decision (though note that their reaction time did not have an effect on the duration for which the beat played). Many participants found this task to be quite boring, and had to be reminded that it would last only five to ten minutes. Instructions for the task are listed in the Appendix under "Timing Perception Task (Time Comparison)".

Timing Production Task

A timing production task was also included to assess the temporal consistency with which participants could make movements. Lundy-Eckman et al. (1991) had subjects tap whenever they heard a paced beat (consisting of 13 tones with a 550 msec interstimulus interval), then continue with the same response rate for 30 more intervals without a pacing beat. This procedure was repeated so that there were 18 trials per subject. The variable of interest was the variability of the inter-response intervals produced without the pacing signal.

In the present study a similar program was created that set a pace of 400 msec for 12 beats, then subjects were to continue tapping a keypad at that same rate 31 more times (i.e., 30 more intervals). A standard pacing signal was important because the variability in inter-response intervals is proportional to the length of the inter-response intervals (Wing, 2002); had participants set their own paces, some would have been expected to have spuriously greater variability. This procedure was repeated for ten trials and the mean and the standard deviation of each trial were averaged across all 10 trials, producing an average mean and an average standard deviation. All participants completed all ten trials; there was no missing data.

Lundy-Eckman based their statistical analysis of tapping on the work of Wing and Kristofferson (1973a, 1973b). This method has been cited in over 109 studies over the past 30 years, including 18 times in 2004 attesting to its contemporary value. There are several levels to the analysis of this data. First, it is important to determine whether the groups differ in their mean tapping rate (i.e., whether one group "drifted" in their rate to elongate or shorten the interval overall). If this were the case, it would suggest difficulty

with time estimation; the timing mechanism of the central nervous system would be judged to be inaccurate. The second analysis is in regard to the variability in intervals (i.e., the average standard deviation of inter-response intervals). This would indicate the degree to which there is some variability in the response patterns. However, the source of the variability, be it an erratic central timer or an erratic motor system, would be in question. The Wing-Kristofferson (W-K) model statistically breaks this variability into these two separate components: a central timer and a motor implementation delay. The central timer refers to the person's own internal clock, or sense of time, while the motor implementation delay refers to the delay between the central clock indicating that it is an appropriate time to respond, and the implementation of the hand movement to press the response key.

The key concept of the Wing-Kristofferson model is that even with a central timer that is accurate in keeping a certain pace, a person's tapping response may be variable due to a delay in implementing the motor response. If the central timer is accurate, however, the person should correct themselves (e.g., if the last interval was judged to be too long, the next interval would intentionally be too short to re-establish the pace). The correlation between the duration of successive intervals (i.e., the lag-one autocorrelation) should be bounded by 0 and -0.5; an autocorrelation of 0 suggests that there is no adjustment by the central timer and the response pattern is random, while a correlation approaching -0.5 indicates that the central timer is accurate and that variability is due to motor implementation. Correlations over -0.5 would not be expected as successive intervals should alternate between being shorter and longer. In this way, one can

determine whether variability in the inter-response intervals is due to central timing dysfunction or motor dysfunction.

Data was collected by a program designed for the study that recorded the time, in milliseconds between taps of a button on an external keypad. These were then analyzed to determine the mean and standard deviation of each trial, and the autocorrelation. These three variable were averaged across the ten trials. In order to ensure that the W-K parameters were calculated accurately, the Collier-Ogden Drift program (Collier & Ogden, 2002) was used to reanalyze the raw data. This program calculated a range of variables, including the mean, standard deviation, autocorrelation, and the Wing-Kristofferson estimates of the clock and motor variance for each trial. Replication of each calculation was performed for at least one trial to ensure the accuracy of calculations. Directions used for the task are listed in the Appendix under "Timing Production Task".

Hypothesis #2: Motor Planning.

Coloured Rod Test

Motor deficits in AS have implicated frontally mediated planning and executive processes (Rinehart et al., 2001). Hughes (1996) developed a technique to address motor planning in children with autism that was used in the present study. Hughes presented participants with a 30 cm wooden rod painted half red and half black, and two disks (one blue and one red). The rod could be placed upright into either disk. Participants were asked, for example, to "pick up the black half of the rod and place it in the blue disk." By varying the position of the rod, the researchers could manipulate the likelihood that participants would use an underhand or overhand grip to grasp the rod. Participants

could place the rod into the disk using a well-planned, comfortable (thumb-up) grip, or a poorly planned, uncomfortable (thumb-down) grip. Previous research suggested that most people naturally use an overhand grip to grasp such objects, so the criterion for success was an appropriate (thumb-up) grasp on at least three of the four underhand trials. Only underhand trials were counted, as the use of the underhand grasp implied motor planning. This technique was repeated in the present study, but there were eight trials rather than four. This was done because the present study was using higher-functioning individuals than the original study, and the incorporation of more trials was hypothesized to allow for more variability and a more sensitive task. In addition, the rod and wells were all different colours in the present study, rather than having one half of the rod and one well the same colour, simply to make the directions more explicit, at Hughes' recommendation (personal communication). Directions for this task are included in the Appendix under "Coloured Rod Test".

Hypothesis #3: Dorsal Visual Stream

Motion Perception Test

A methodology similar to that conducted by Sigmundsson et al. (2003), Milne et al., (2002) and Spencer et al. (2000) was used to address the participants' threshold for movement perception. These authors used similar paradigms using random dot kinematograms. Sigmundsson et al.'s technique involved having the participants view a computer screen on which dozens of dots were moving; some were moving in random directions, while others were moving synchronously in one direction. Participants were asked to indicate whether the movement was to the left or to the right. The ratio of

synchronously moving to randomly moving dots was termed the coherence. Individual dots would disappear after 200 ms so that participants could not simply track a single dot. The measure was not timed – only accuracy was recorded, and the coherence was varied to the detection threshold starting from an initial 75% coherence that could be detected by all participants. Similarly, Spencer et al. had subjects identify an area on either the right or left side of a computer screen in which dots were oscillating in reverse phase to surrounding dots. The present study was conducted using a task similar to the measure used by Sigmundsson et al. (2003) and Milne et al. (2002). This psychophysical paradigm has been used in a number of published studies of autism, clumsiness, reading, and dyslexia (e.g., Cornelissen & Hansen, 1998; Cornelissen et al, 1998; and Talcott, Hansen, Assoku & Stein, 2000). Participants completed the task in a darkened room and viewed a similar kinematogram to that used by Sigmundsson et al. They were asked to press one of two buttons to signify which side of the screen had the coherent motion, and to guess if they were unsure. All images were displayed for the same amount of time, and participants were not able to respond until after the images disappeared. After each trial, the coherence value achieved was displayed; the meaning of this number was explained to participants so as to motivate them to perform better on the next trial. Most participants reported that they found the task quite engaging. There were four trials for each participant, and the average across all four trials, as well as the scores for each trial were recorded and analyzed. Directions used for the task were provided by Dr. Hansen, and are included in the Appendix under "Running the Subject: MOTDX Program".

Form Perception Test

The paradigm provided by Dr. Hansen also consisted of a measure of form coherence thresholds to control for differences in visual perception. The task was analogous to the Motion Perception Test, though this task involved simultaneously viewing two rectangular patches, one with lines arranged in random orientations, and one with a variable number of lines arranged so as to form a large circle somewhere in the patch. There was no motion involved in this paradigm. Again, participants were asked to identify which of the two patches contained a circular pattern, and to guess if they were unsure. Responses could not be made until the images disappeared after a standard amount of time had passed. This task was somewhat less engaging than its counterpart, but the provision of coherence scores after each trial was reported to be quite motivating. Directions used for the task were provided by Dr. Hansen, and are included in the Appendix under "Running the Form Program".

Biological Motion

Next, the groups viewed human movements, and the accuracy of their perception was measured for an indication of the integrity of their dorsal stream. Ostensibly, the perception of movements and the ability to produce them are related (Avikainen et al., 1999). As such, participants were presented with images of people walking; the images were point-of-light walkers generated by a 3D VICON motion capture system (Oxford Metrics, Oxford, UK) so as to obscure details of the person's body and require the participants to view the gestalt of the movement.

To create the point-of-light images, the archived gait-analysis data of volunteers participating in other aspects of research in the Centre for Human Movement Analysis (CHUMA) were sampled. Ten archived individuals were selected; five of these people had one of a variety of impairments in their gaits, such as limps or other asymmetries, while the remaining five had essentially intact gaits. No formal analyses of the gaits was completed, except to note that the five individuals with impaired gaits had been involved in studies due to documented and recognized gait anomalies, often with known medical etiologies. The video images selected from the archives featured the person walking relatively straight for approximately two to five seconds across a room. Two video images were selected from each individual's file; the images included similar paths, and always moved in the same direction. Two point-of-light walkers were created from these images, using the technology available with the VICON motion capture system. The VICON tracks movement through three dimensions by using six infrared cameras with sampling frequencies of 120 Hz. The cameras were mounted on tripods and detect the motion of reflective markers that were 14 mm in diameter and attached to people as they move around a room. Thirty-two markers had previously been labelled on each volunteer so that the archives included an inventory of the position of each marker. Each point-of-light image was composed of six points representing the person's ankles, knees, and hips. Images were replayed using the VICON Polygon Viewer program. All point-of-light walkers were viewed from the person's side, such that in some cases, the hip joints overlapped and one point was obscured so that only five points were visible.

A number of manipulations were made to these images. First, while one point-of-light walker was left intact for each person (resulting in 10 non-manipulated images), the

second point-of-light walker was created by sampling the points of one leg (e.g., the right leg) from the intact image, and the other leg (e.g., the left leg) from the second video image. As such, the resulting point-of-light walker was an overlap, or chimera, of two events (resulting in 10 chimeric images). This procedure is consistent with the procedure employed by Thornton, Vuong, & Bulthoff (2003) and Pinto and Shiffrar (1999). Often the image appeared to be a typical gait, and often it appeared to be asynchronous. For example, the points representing the hips might start out aligned, but one hip (and the rest of the leg) would move faster than the other, which would constitute a physically impossible movement. The ten chimeric images varied in the extent to which they appeared atypical or impossible.

A second manipulation was made to the 10 chimeric and 10 non-chimeric images. The playback speed of each of the 20 images was accelerated to 150% of their original speed to create twenty faster gaits. The images were also decelerated to 30% of their original speed to create twenty slower gaits. The purpose of manipulating the speed at which the images were replayed was to ensure that the difficulty level was varied, given that the images were created solely for this project, and that there was not a sufficient population of people with AS to merit piloting the data beforehand.

In total then, there were 60 images. Half (30) were based on volunteers with typical gaits, and half (30) were based on volunteers with atypical gaits or limps. Also, half (30) were chimeric images, and half were based on a single event. Finally, one-third (20) were played back at the speed at which the volunteer naturally walked, while one-third (20) were accelerated, and one-third (20) were decelerated. Due to software compatibility issues, five images were found to be unsuitable and were removed from the

experiment, yielding a total of 55 images in the final experiment. Those that were excluded included all three chimeric displays and one non-chimeric display of a volunteer with a normal gait. The chimeric, accelerated display of one of the volunteers with an atypical gait was also deleted due to software issues.

Given the complexity of the task and the need for the participants to understand the variables presented, the instructions for the task were extensive. Instructions for the task are included in the Appendix under "The Walkers Test". Briefly, each participant viewed a video clip of a volunteer walking in the CHUMA lab, and were then shown how that image was turned into a point-of-light walker. The participants were then shown a second video clip of the same volunteer walking somewhat differently. The participants were then shown the chimeric point-of-light walker, and told how it was created. Participants were then told how the program worked, and the questions they would be required to respond to. All participants were asked to verify their understanding of the task, and any misunderstandings were corrected to the extent that they were apparent.

Given the large number of images to be viewed, a computer program was designed to facilitate the display of the images to reduce the amount of time needed between trials, and to facilitate efficient data collection. Participants were posed two questions, serially. The first question appeared simultaneously with the appearance of each point-of-light walker. Point-of-light walkers were displayed on a repeating loop until both questions were answered. The first question queried whether the image was of one event, or a chimera of two events, by asking whether it was 1 or 2 walkers overlaid. Participants responded by clicking, with the mouse, their choice. Once their choice was made, the second question appeared, asking whether the gait was normal or atypical.

Again, participants responded by clicking with the mouse. Response times for both choices were recorded, and the next point-of-light walker launched after the second choice was made. Some participants required encouragement in order to get through the task, but most were able to complete it with some assurance that they were progressing efficiently (e.g., being told when they were half-way through).

Postural Stability Test

Next, the ability of children and adolescents with AS to maintain stability in the face of virtual movement of the environment and the availability of proprioceptive compensation (e.g., Gepner & Mestre, 2002) was addressed. Gepner and Mestre eschewed the usual technique, which is to construct a room in which the walls can be moved independently of the floor thereby separating visual and proprioceptive experiences, by simply projecting computer simulated movement (sinusoidal lines) on a large screen in front of participants, and measuring their steadiness by using a force plate. In the present study, a similar technique was used, but instead of projecting movement upon a screen, a virtual reality paradigm that simulated movement was displayed while participants stood on a force plate. The dependent variable used was, as per Gepner and Mestre, the shift of the centre of pressure on the force plate.

Two virtual images were programmed. The first image was of a corridor with a door at the end. The hall was lined with a variety of pictures that appeared to be paintings, and the overall appearance was that of a school hallway. The image ran for approximately 30 seconds and involved movement toward the end of the corridor, with a number of side-to-side rotations suggestive of glances with head turning toward the pictures on the wall. The image approached door at the end of the hall, and at the end of

the sequence, appeared to run right into the door such that all that was visible was the colour of the door. The second image was identical, but in front of the door was an image of an immobile human figure. The features of the human were not detailed, such that it looked similar to a mannequin. The end of the sequence involved a movement through the human figure and into the door.

Data regarding the shift of the centre of pressure was collected using an AMTI AccuSway force plate which was connected to the AMTI Balance Clinic Program (Advanced Mechanical Technology, Inc., Watertown, MA). The virtual reality equipment was not connected directly to the AMTI Balance Clinic Program; their coordination was achieved through standardized timing techniques described below.

Data collection commence by having participants remove their shoes and stand on a piece of paper upon which outlines of feet had been drawn. The outlines were aligned with the AMTI AccuSway force plate to facilitate instructions of where to stand. The AMTI AccuSway force plate required the input of a number of Base Of Support (BOS) points, so participants' actual feet were traced, then they stepped off of the plate so that the positions of the BOS points could be entered and the plate zeroed to tare and reset the platform. The platform was zeroed between each trial. Seven data trials were completed for each participant, and the instructions used for each can be found in the Appendix under "Balance Test". During the first trial, the participant simply stood on the platform for 45 seconds while trying to remain as still as possible. During the second trial they were asked to do this again, but with their eyes closed, removing accurate visual input to balance adjustments. The third trial was included to control for any additional movement associated with wearing a virtual reality headset; participants were instructed to wear the

headset, which showed an image of the immobile Windows® desktop. During the fourth trial the first virtual reality sequence, that of the empty hallway, was displayed. In order to standardize data collection, the AMTI data collection was started five seconds before the virtual image started to play. As such, participants spent the first 5 seconds of the 45 second data collection period looking at an immobile hallway, then suddenly, at 5 seconds, there was a largely unexpected sudden movement forward as the image began to play. The image ran for 30 seconds before the screen went black, and participants were reminded to remain still for an additional 10 seconds until the data collection was completed. The reason for collecting data longer than the virtual reality sequence was to ensure that, even if there was a delay in starting the image, all the responses to the virtual movement would be recorded. During the fifth trial, the hallway with the human figure was played with a procedure identical to that of the fourth trial. A sixth trial paralleled the third trial in which the participants wore the virtual reality headset, but saw only a blank desktop. However, during this sixth trial, participants were instructed to turn their head from the left to the right every five seconds for 45 seconds commencing at the 10 second mark (to allow time to start the virtual reality image at the 5 second mark in the seventh trial, described below). Their shoulders were lightly tapped to remind them when and where to turn their head. This head-turning procedure was repeated in the seventh and final trial in which the virtual reality headset displayed the moving hallway with a human figure again. The purpose of the latter two trials was to introduce proprioceptive movements that were discordant with the virtual input to the visual system.

Data was recorded from the AMTI system for each of the seven trials. Maylor, Allison, and Wing (2001) suggested that the most appropriate variables to record when measuring postural stability were the average velocity and the standard deviations in the anterior/posterior (Y) and lateral (X) axes. All three were recorded for the present study, in addition, the range of the anterior/posterior and lateral axes was also recorded. It was hypothesized *a priori* that a single sway may be more typical of the reactions to the initiation of the virtual movement than movement throughout the image, and that the range would more accurately reflect the degree of the sway than the standard deviation.

Clumsiness criterion

Dean-Woodcock Neuropsychological Battery

Given the possibility that a number of the hypotheses addressed above may suggest differences between the groups, it is necessary to determine whether these differences account for the clumsiness seen in AS. A criterion of clumsiness was included as a dependent variable against which the measures above would be compared to determine whether they accounted for a reasonable proportion of the variance.

Selecting an appropriate norm-referenced measure was difficult, as few tests of motor ability span the adolescent age range (Barnett & Peters, 2004). For example, the Movement Assessment Battery for Children or the Bruininks-Oseretsky have been used frequently in the literature with younger children and are tools that are sensitive to motor dysfunction seen in AS. However, the Movement ABC had norms extending only to 12 years, and the Bruininks-Oseretsky was only re-normed to capture the age range up to age 21 years, after the data collection phase of this project was completed. As such, a

relatively new measure was used, the Dean-Woodcock Neuropsychological Battery (Dean & Woodcock, 2003) Sensory-Motor Battery.

The Dean-Woodcock Neuropsychological Battery Sensory-Motor Battery is composed of 18 tests of sensory and motor abilities, but only the nine motor tests were administered. These nine tests are described in turn. Gait and Station provided guidelines for making clinically relevant observation of participants' gait while they walked naturally, walked heel-to-toe, hopped on one foot, and stood stationary. The test was designed to be sensitive to ataxia, muscular weakness, or spasticity. Romberg was a measure of unsteadiness associated with cerebellar or vestibular dysfunction and involved rating participants' balance while standing with their eyes closed and their feet together, oriented toe-to-heel, or on one foot. Construction measured visuocostructive ability and visuomotor praxis and provided guidelines for assessing drawing ability of an outline of a cross, and of a clock. Coordination measured coordination of skilled movements and required participants to alternate between touching their nose and the examiner's finger, and to alternate between touching the front and back of their hand to their thigh. Mime Movements, a typical test of ideomotor apraxia, required participants to pantomime a number of movements, including brushing their hair, opening a door, etc. Left-Right Movements was administered to all participants, but because of a ceiling effect and lack of variability, it was discarded from further analyses, but briefly assessed left-right orientation. Finger Tapping measured fine motor speed bilaterally; participants were required to tap the equals (=) button on a computer keypad, where the button was in the bottom right corner of the keypad, which tallied the number of times they could oscillate their finger in 10 seconds. The score was an average of five trials per hand. Expressive

Speech assessed fluency of speech production and the presence of dysarthria and required the repetition of complex words and phrases (e.g., "the ragged rascal ran"). Finally, Grip Strength (Hand Dynamometer) measured hand strength bilaterally.

The Dean-Woodcock was standardized on 1,011 healthy subjects without psychiatric or neurological conditions between the ages of 2 and 95 years, though normative data were available only between 4 and 80 years. The sample was stratified in accordance with the 2000 US census for sex, race, age, and handedness. The Dean-Woodcock scores are derived from Rasch modelling, a single-parameter logistic test model derived from item-response theory. The Dean-Woodcock is co-normed with a number of other Woodcock-Johnson tests.

Because of the use of Rasch modelling, standard scores, which are typically computed with norm-reference tests, are not included in the Dean-Woodcock. Instead, W scores are presented. Unlike standard scores, W scores demonstrate consistent change with development, so the scores are compared to the typical W score of a person of a particular age category, which is referred to as the Reference W. Deviations from the mean within an age category are expressed as a difference between the obtained W score and the Reference W. The Reference W can be centred arbitrarily, but is typically centred with a score of 500 being the average raw score of a 10 year 6 month old participant in the normative sample. Scores increase and decrease as a function of development, such that age-equivalents are always directly referenced by a W score on a particular measure. Deviations above the Reference W score signify advanced abilities, while differences of -6 and above signify abilities that are within normal limits, differences of -7 to -13 are labelled mildly impaired to within normal limits, differences

of -14 to -30 are labelled mildly impaired, differences of -31 to -50 are labelled moderately impaired, and differences of less than -50 are labelled severely impaired.

Inter-rater reliability for the Dean-Woodcock is reported to be good (>0.60) to excellent (> 0.80) in terms of agreement of impairment levels using the linear agreement weighting Kappa. Gait and Station and Romberg had the lowest inter-rater agreement because of the significant amount of clinical judgement required. As such, given that the present study was not completed blind to diagnostic status, the correlation between the Romberg test scores and an eyes-closed trial on the AMTI AccuSway force platform were analyzed to assess the likelihood that judgements of the Dean-Woodcock Romberg test were biased by prior knowledge of the participant's diagnostic status.

Developmental influences on the Dean-Woodcock, an indication of the test's validity, were evaluated by examining growth curves in the normative sample; these curves showed regular change and improvement with age into adulthood, and decline in later adulthood. A factor-analysis of the Dean-Woodcock revealed three partly overlapping factors: a sensory factor, a factor reflecting higher cortical motor skills, and a third factor, characterized by tests assessing subcortical motor skills. This third factor was composed primarily of Romberg (Toe-to-Heel) and Gait and Station, and to some extent, Coordination (Finger-to-Nose). Perhaps due to significant overlap of factors, no factor scores are computed for the test.

The Gait and Station subtest of the Dean-Woodcock required a sample of the participants' gait, which was completed in an infrequently used straight 20.5 meter long and 1.5 meter wide hallway outside the laboratory. Participants were asked to walk down the hallway while their gait was observed with the purpose of examining any anomalies

as part of the Gait and Station subtest. When they reached the end, they were asked to turn around and return to their start point blindfolded, a condition not required in the Dean-Woodcock. Participants were reminded that they could elect not to complete the blindfolded portion of the test, and were reassured as to steps taken to ensure that they would be safe in the hall. All participants agreed to continue. The purpose of blindfolding participants was twofold. The first purpose was to determine how far participants could successfully walk straight before bumping into a wall; that is, without visual input, was proprioceptive input sufficient to direct their movements in a consistent direction? The second purpose was to determine whether there was any tendency for groups to differ in terms of the direction in which they tended to veer. There is evidence in schizophrenia research of a tendency to veer to the left when walking blindfolded due to right-sided inattention, which in animals has been associated with asymmetrical dopamine systems, which may have lateralizing implications (Mohr, Bracha, & Brugger, 2003).

Results

Although a large amount of data was collected, the analysis of the data does not include an analysis of each possible variable. A number of tasks were included simply as control measures to understand the meaning of any differences on other tasks. The results that follow, then, are designed to test the primary hypotheses, rather than address all the data collected.

Global Motor Skills

As discussed above, clumsiness has consistently been noted to be a feature of Asperger Disorder. The purpose of gathering direct measures of global motor abilities in the present study was (a) to confirm that the AS group had weaker motor abilities and are therefore representative of the AS participants sampled in past research in this regard, (b) to determine the severity and pervasiveness of any impairments, and (c) to use this measure to address the veridical ecological validity of any of the hypotheses that are supported below. All participants completed the Motor Tests of the Dean-Woodcock Sensory Motor Battery. Although the eight tests produce 15 scores, only 12 scores were analyzed; this is because there was no variability (all perfect scores) in a left-right orientation task, and the scoring criteria for Finger-to-Nose: Right and Finger-to-Nose: Left were too vague to allow reliable judgement without further training. So instead of using a 15 subtest overall score, a 12 subtest overall score was substituted. Psychometrically, this was not problematic, because the total score was simply an average of norm referenced W difference scores which correspond directly to impairment levels; the overall score on the Dean-Woodcock is not normed separately. There was a significant difference and a large effect size between the Asperger (mean W diff = -6.58,

SD = 4.40) and control (mean W diff = 1.01, SD = 4.31) groups on the overall Dean-Woodcock Motor Tests, suggesting that the Asperger group was significantly less coordinated, $t(28) = -4.77$, $p < .001$, Cohen's d = 1.81. This suggests that the average person in the Asperger's group falls just below the "within normal limits" range (for which the cut-off is a W difference score of -6) in the "mild [-ly impaired] to within normal limits" range. Eight of the 14 participants with Asperger's had W difference scores below the normal range, with only one actually falling within the mildly impaired range (for which the cut-off is a W difference score of -14). In contrast, only four of the sixteen control participants fell below the normal range and none fell within the mildly impaired range (refer to Figure 1). However, this is illustrative of the fact that although there are highly significant differences on tests of motor ability, only about two-thirds of the Asperger's group scored below the average range, while a quarter of normal controls fell below the average range. As such, scores on global measures of motor ability alone are unlikely to be particularly useful in diagnostic decisions. A separate set of analyses were conducted within the AS group independently to determine whether the severity of impairment on the Dean-Woodcock correlated with the severity of scores on any of the measures of AS symptomatology; however, within this small sample (n = 14), no significant correlations were noted.

Because the assessment of motor skills was not done blind to diagnosis, there was a possibility that the Dean-Woodcock subtests that require more subjective judgements may have been somewhat biased. This was assessed in two ways. First, a second analysis of group differences on the Dean-Woodcock was calculated using only the eight subtests that did not require significant levels of clinical judgement. Even using only

these eight subtests, the difference between groups was present, $t(28) = -4.05$, $p < .001$, and was reduced by less than 1%, suggesting that experimenter bias was not a significant factor.

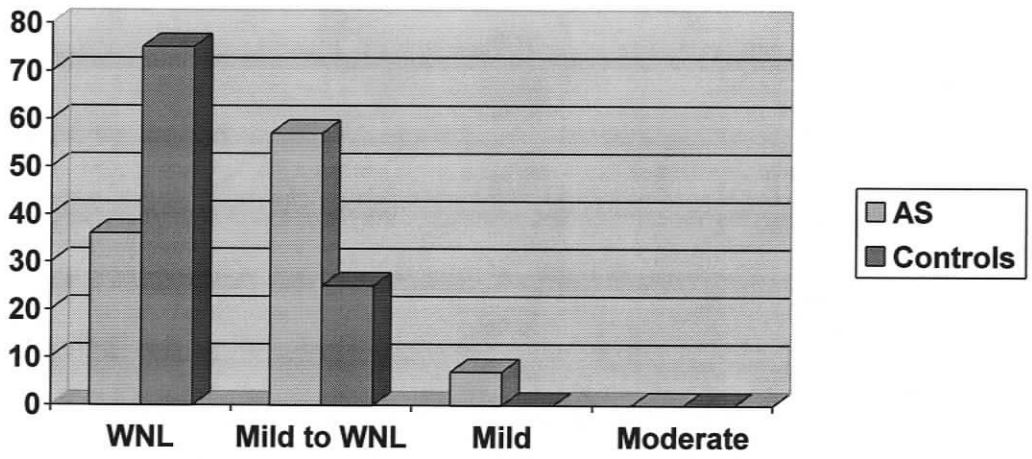

Figure 1. Percentage of each group falling within specified Dean-Woodcock Impairment Levels. WNL = within normal limits.

As a second measure of bias, the Dean-Woodcock Romberg Test, which involves standing stationary with eyes closed in three positions: feet together, toe-to-heel, and on one foot, was analysed. An eyes-closed stationary stance on a force platform was completed with all participants, and the data on this trial on the force platform were analyzed in relation to the Dean Woodcock Romberg Test. The Romberg W score correlated significantly and in the expected direction with all variables measured on this trial on the force platform, as shown in Table 3.

Table 3. Correlations between Romberg tests requiring clinical judgement and objective measurement.

	Dean-Woodcock Romberg Test
Balance Platform: Eyes Closed	
X SD (cm)	-.420(*)
Y SD (cm)	-.487(**)
X Range (cm)	-.420(*)
Y Range (cm)	-.425(*)
Velocity Average (cm/sec)	-.372(*)

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).

As a post-hoc analysis only, differences between the two groups on the Dean-Woodcock appeared to be due to significant differences on Gait and Station, Construction: Cross, Coordination: Hand to Thigh (right), Coordination: Hand to Thigh (left), Mime Movements, Finger Tapping: (dominant), and Finger Tapping (nondominant). There were no significant differences on the Romberg test, Construction: Clock, Expressive Speech, Grip Strength (dominant), or Grip Strength (nondominant).

Results of the hypotheses of the study are discussed below.

Hypothesis #1: Timing Production and Perception

Timing Production

The primary hypothesis of the timing production task was that there would be more variability in the inter-response intervals in the group with Asperger Disorder. The first analysis was to determine whether the two groups differed in the duration of their inter-response intervals, because longer inter-response intervals (1) could spuriously lead to greater variability (Wing, 2002), and (2) would indicate significant differences in the functioning of the central clock. Although the pacing beats had an interstimulus interval

of 400 ms, both groups had a tendency to elongate their inter-response intervals, which is consistent with past research (Lundy-Eckman et al., 1991). The Asperger group (mean = 550.26 ms, SD = 41.99) was actually more accurate than the control group (mean = 555.27, SD = 38.08), however the difference was not significant and the effect size (Cohen's d = 0.13) was negligible. This indicates that there should be no spurious group differences in variability due to the length of inter-response intervals, and that the groups' internal clocks were equally inaccurate.

The second analysis is the *a priori* analysis of variability in the inter-response intervals. A power analysis was performed beforehand, based on Lundy-Ekman et al.'s (1991) estimate of a large effect size (Cohen's d = 1.43), which had suggested that a sample of 24 participants (12 with AS) was required to produce a power of .96.

The analysis of variability in the inter-response intervals is based on the mean of the standard deviations of each of the ten trials. As hypothesized, the variability in inter-response intervals was significantly greater in the Asperger group (mean = 76.51 ms, SD = 49.58) than in the control group and the effect size was large (mean = 45.61, SD = 22.20), $t(28) = 2.25$, $p < .05$, Cohen's d = 0.85. The responses of the group with Asperger Disorder were more erratic.

Next, the planned post-hoc analysis was designed to determine the source of the added variability in the Asperger group. The Wing-Kristofferson model separates the variability into clock variance, and motor implementation variance. However, the model is based upon several assumptions. The most important assumption is that the lengths of successive intervals are correlated (i.e., an autocorrelation) between 0 and -0.5. An autocorrelation of 0 indicates that the central timer is not aware of, or able to implement,

adjustments to intervals to correct errors; an autocorrelation of -0.5 indicates that as one interval increases in length, the other corrects the error by decreasing in length, and that this is repeated over successive trials. The number of violations of this assumption were analysed by assessing the frequency with which trials had autocorrelations outside of the range of 0 to -0.5 across the 10 trials, and the magnitude of the violations (i.e, how far outside the 0 to -0.5 range the autocorrelation fell, summed across the 10 trials). The assumptions of the model were violated by both the Asperger group (mean = 5.93 out of 10 trials, SD = 2.46) and the control group (mean = 5.75, SD = 2.38) frequently, but there was no significant difference in the frequency of violations between groups. Essentially all violations of the model involved autocorrelations that were positive which might be expected with the equal elongations of the inter-response intervals; only one participant produced autocorrelations in the range of -1.0 to -0.5. There was also no significant difference in the magnitude of the violating autocorrelations by the Asperger group (mean = 1.61, SD = 1.19) and the control group (mean = 1.18, SD = 0.85). That is, one group was not making smaller violations than the other.

Because the assumptions of the Wing-Kristofferson model were violated on more than 50% of the trials in both the Asperger group and the control group, the model was considered invalid in the present study. An initial analysis of the data, calculated using the Collier-Ogden Drift Model program (Collier & Ogden, 2004) to calculate the Wing-Kristofferson statistics also revealed no potentially informative patterns.

In summary, the AS group was more erratic in their timing production, but the source of the inconsistency is unclear.

Timing Perception

As another test of timing ability, a motor free timing task was included. An *a priori* power analysis based on Lundy-Ekman et al.'s (2001) estimate of a large effect size (Cohen's d = 1.01) had suggested that a sample of 44 participants (22 with AS) was required to produce a power of .95. In the present study, there was no difference between the groups, though the Asperger group (mean = 8.86, SD = 5.42) mean was somewhat better than the controls (mean = 10.13, SD = 2.47), however this did not approach significance and the effect size (Cohen's d = 0.08) was negligible. Likewise, there was no difference in the groups' reaction times after the cessation of the beats. In fact, most people in both groups were prepared to respond before the cessation of the beats such that decision time was not measured well.

These results suggest that there are no significant differences in the internal clock of participants with and without Asperger Disorder. Combined with the results of the timing production task, a motor implementation delay may be implicated in the inconsistency of the beats produced by the AS group.

Hypothesis #2: Motor Planning

An *a priori* power analysis based on Hughes' (1996) estimate of a very large effect size (Cohen's d = 4.98) had suggested that a sample of only 4 participants (2 with AS) was required to produce a power of .91. However, in the present study, there were no significant differences between groups on this task. Hughes (1996) used two separate methods to evaluate her data. One was to use the number of correct (thumb-up) movements, for a data range of 0 to 4 across the four trials. The second method was to

use a dichotomous variable in which participants either did or did not get 3 or 4 out of four trials correct. Both of these methods were used in the present study, though eight trials were administered in the present study. This resulted in two different tests of this hypothesis, neither of which showed significant differences. There was a trend toward better motor planning in the control group (mean correct underhanded grips of 8 trials = 5.31, SD = 3.61) than the Asperger's group (mean = 3.43, SD = 3.59), which produced a medium effect size (Cohen's d = 0.55), substantially smaller than Hughes' estimate. Interestingly, in a direct comparison with Hughes' study, and looking only at the first four trials in the present study, as per her method, it is evident that the Asperger group performed similarly (mean correct underhand grips in 4 trials = 1.71, SD = 1.86), though somewhat better that Hughes' autism group (mean = 1.44, SD = .42). The difference is that in Hughes' study, her control group approached perfection (mean = 3.64, SD = .50), while this control group did not (mean = 2.63, SD = 1.78). One might hypothesize that this is due to the age of the participants; Hughes' control groups were preschoolers, who were most likely to use an underhand grip, and a middle childhood (mean age of 11 years) group, who were more likely to use an underhand grip than the autistic group, but less likely than the preschool group. It is possible that older participants are more "flexible" in their grips and that there is a developmental effect, such that the underhand grip was not required for the present, older (mean age of 14 years) sample. To test this hypothesis, an analysis of the effect of age in predicting success on this task was completed for both groups separately and pooled into one group, but none of these analyses approached significance.

It is also possible that a factor in the instructions for the task led people, especially controls to use an unnatural group. It is possible that the participants expected the task to be difficult, so used an unnatural grip thinking they were supposed to do so. Despite the lack of significance, it is interesting that the results for the Asperger group are so similar to the results of Hughes' autistic group, though there is much more variability in the present study.

Hypothesis #3: Dorsal Visual Stream

Motion Perception

It was hypothesized that visual perception of motion may be affected in participants with Asperger's, so two tasks, one of static visual perception and one of dynamic motion perception were completed. An *a priori* power analysis based on Milne et al.'s (2002) estimate of a large effect size (Cohen's d = .84) had suggested that a sample of 64 participants (32 with AS) was required to produce a power of .95.

As expected, there were no significant differences between the groups in terms of static visual perception, though the controls did somewhat better, with a small effect size (Cohen's d = 0.40). However, contrary to expectations, there was no significant difference between the groups in motion vision, either. Again, controls performed better, but the effect size here was even smaller (Cohen's d = 0.24). These results suggest that there is no difference between typically developing controls and participants with Asperger Disorder in terms of perception of simple motion.

Biological Motion Perception

Despite the lack of evidence of motion perception deficits, it was still possible that perception of human movement may have been affected. The biological motion task included two primary variables: whether the stimulus was whole or chimeric and whether the stimulus had a normal or abnormal gait. There were therefore four different types of correct responses, (1) correctly identifying whole walkers (a hit), (2) correctly identifying chimeric walkers (a correct rejection), (3) correctly identifying normal gaits (a hit), and (4) correctly identifying abnormal gaits (a correct rejection). There were 55 trials.

In terms of distinguishing between whole walkers and chimeric walkers, a signal detection approach was used to analyze the date. The d' (d prime) statistic was calculated for each participant based on the proportions of hits and false alarms produced; d' is person's sensitivity to the unified walker, the distance between the noise alone trials, and the signal+noise trials. The d' statistic is the difference between the *z*-transformations of the hit rate and the false alarm rate. There was a significant difference between the sensitivity (d') of the Asperger group (mean = 1.57, SD = .96) and the control group (mean = 2.28, SD = .73), producing a large effect size, $t(27) = -2.27$, $p < .05$, Cohen's d = 0.88.

In terms of distinguishing between normal and abnormal gaits, there was only a trend toward a significant difference between the sensitivity (d') of the Asperger group (mean = .26, SD = .58) and the control group (mean = .62, SD = .51), producing a medium effect size, $t(27) = -1.76$, $p < .10$, Cohen's d = 0.69.

Interestingly, when these four hit and correct rejection scores were summed to produce 110 possible correct responses, the control group (mean = 81.93, SD = 8.00)

responded correctly significantly more frequently than the Asperger group (mean = 71.29, SD = 10.23) producing a large effect size, $t(27) = -3.13$, $p < .005$, Cohen's d = 1.21. This illustrates that success on the two choices on the task were additive.

These results accord with the hypothesis that possible movements are more easily identifiable than impossible (chimeric) gaits. Discriminations between normal and abnormal gaits may have been more difficult for both groups, since the abnormal gaits are not actually impossible. There were no differences in response times, suggesting that certainty did not affect the rate at which people responded in any analyses.

Postural Stability in the Presence of Virtual Movement

It was hypothesized that in a virtual environment in which there is visual movement, a hyper- or hyposensitive dorsal visual stream may affect the amount of sway in a person's stance, even though they are consciously aware that there is no movement. Based on the small sample findings of Gepner and Mestre (2002), it was hypothesized that participants with Asperger's would be hyperreactive to the movement presented. An *a priori* power analysis based on Gepner and Mestre's (2002) estimate of a medium effect size (Cohen's d = 0.69) in their very small sample (n = 3) research had suggested that a large sample would be required, but it was not known what type of effect sizes might be expected.

Although Maylor, Allison, and Wing (2001) suggested that the most appropriate variables to use when measuring postural stability were the average velocity and the standard deviations in the anterior/posterior (Y) and lateral (X) axes, the standard deviations were recorded, but replaced in the primary analyses with the ranges in the present study. This was done *a priori*; in the present experimental design there was one

point during the 45 seconds of measurement at which time it was expected that the participants might make an abnormal movement. In the first hallway presentation, the participant was expected to make a movement along the anterior/posterior axis when initially presented with virtual motion. This movement was not expected in the second hallway, when this initial virtual motion would be anticipated by participants. However, in the second hallway, movement along the lateral axis was expected, especially in the control group, when approaching, and perhaps trying to avoid virtually bumping into, the humanoid figure at the end of the hall.

Consistent with results of the Romberg Test on the Dean-Woodcock, there were no significant differences in postural stability in the two groups when standing on the force plate with the virtual reality equipment on, with a motionless screen. However, when the hallway virtual motion was presented, there was a significant difference in the range of motion in the anterior/posterior (Y) axis between the participants with AS (mean = 4.26 cm, SD = 2.02 cm) and the control group (mean = 2.90 cm, SD = 1.18 cm) which produced a large effect size, $t(27) = 2.22$, $p < .05$, Cohen's d = 0.87. There was no significant difference in the range on the lateral axis. The standard deviations of these same variables reflected the same pattern; significant differences along the anterior/posterior axis, but not the lateral axis. There was no difference in the average velocity. This indicates that participants with Asperger Disorder were more reactive to the motion, and less stable. The hyperreactivity of the AS group was in keeping with the findings of Gepner & Mestre (2002).

In the second virtual presentation, which included the humanoid figure, there was no significant difference between groups along the anterior/posterior axis. However,

there was more movement along this axis in the second presentation than in the first in both groups, $F(1,27) = 5.38$, $p < .05$. However, in the second virtual presentation, the Asperger group (mean = 4.11 cm, SD = 1.67 cm) had a larger range of movement along the lateral axis than the control group (mean = 2.96 cm, SD = 1.14 cm) which produced a significant group difference and a large effect size, $t\ (27) = 2.19$, $p < .05$, Cohen's d = 0.84. There was no significant difference in the standard deviation or the average velocity. Because the presence of the humanoid figure was the only difference between this second virtual presentation and the first, it was possible that the movement in the Asperger group could be attributed to movement in reaction to the presence of the humanoid figure. However, there was no difference in the amount of movement along the lateral axis between the first and second virtual presentations; the AS group was simply less variable in the second presentation, which resulted in the significant group difference.

These results suggest a greater degree of movement in the centre of pressure in the AS group on tasks with a virtual reality presentation, but not on tasks that did not include virtual movement. However, it is difficult to attribute these differences to differences in the task parameters. Differences in the lateral or anterior/posterior axis may be the result of a similar mechanism.

Veridical Ecological Validity

A number of variables that are putatively related to motor skills are described above. Although several group differences have been identified, and although the Asperger group has been identified as being less coordinated than the control group, the

link between group differences on the variables of interest have not been linked to clumsiness directly. In order to do this, correlations with the clumsiness criterion were calculated for the variables for which there were significant group differences. These include: (1) variability in inter-response intervals in timing, (2) d' for the sensitivity to whole versus chimeric movements on the biological motion task (3) overall correct responses on the biological motion task, (4) anterior/posterior range of movement on the virtual motion task, and (5) lateral range of movement on the virtual motion task that included a humanoid figure. The 2-tailed Pearson correlations of each of these five variables with the Dean-Woodcock Total Motor score (based on 12 rather than 15 subtests, as described above) were significant, as shown in the column titled "Entire Sample" in Table 4.

Table 4. Correlation of variables with group differences with the Dean-Woodcock Total Motor score.

	Correlation with Dean-Woodcock Total Motor	
	Entire Sample	Group Partialed Out
Tapping Beat - Std Dev for 400 ms	-.68***	-.62***
Biological Motion – d' for whole/chimeric	.46*	.28
Biological Motion – correct responses	.63***	.44*
Balance Test: Hallway - Y Range	-.65***	-.57**
Balance Test: Hall with Human - X Range	-.60***	-.23

*** Correlation is significant at the 0.001 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

This suggests that not only were the groups significantly different in their success on these five tasks, but that performance on these tasks is correlated with performance on a standardized measure of motor ability, in the predicted direction. Improvements in Dean-Woodcock scores were associated with smaller amounts of variability on the

tapping task, a greater number of correct responses on the Biological Motion task, and smaller ranges of movement on the Balance Test.

However, it was possible that these correlations may be significant simply because there are group differences on all tasks, *and* the Dean-Woodcock. As such, these correlations were also calculated after controlling for the effect of group membership, and are presented in the columns titled "Group Partialled Out" in Table 4. It is apparent that for three out of the five variables, the correlations were still significant, and were still meaningful. In particular, the movement in the anterior/posterior direction in the virtual hallway, and the variability on the Tapping Beat task are still correlated with motor ability after controlling for the influence of group differences.

The correlation between the variability on the Tapping Beat task and the Dean-Woodcock was not simply due to the fact that the Dean-Woodcock includes a finger-tapping test. Although the Tapping task did correlate well, and in the anticipated direction, with Finger Tapping (Dominant) (r = -.59), it also correlated well with other motor tasks. These included dominant hand Grip Strength (r = -.57), non-dominant Grip Strength (r = -.64), Gait and Station (r = -.46), Romberg (r = -.57), Construction (Cross) (r = -.57), Construction (Clock) (r = -.59), Hand-to-Thigh (dominant) (r = -.63), and Hand-to-Thigh (non-dominant) (r = -.64).

Discussion

The present study builds upon an extensive literature that documents significant motor dysfunction in children with AS. The results described here are largely consistent with previous findings that suggested that 80% of cases of AS are associated with motor dysfunction (Bonnet & Gao, 1996), though the prevalence of motor problems on the Dean-Woodcock Sensory-Motor Battery Motor Tests was only 57% in this sample (8 of 14). However, this study is unusual in that older adolescents were sampled along with children; many other studies have incorporated younger samples. It demonstrates that although there may be a declining rate of motor dysfunction with age, that even in this older sample, motor dysfunction is extensive and measurable on traditional neurological and neuropsychological tests of motor ability.

This study also builds upon the literature by responding to the call for more analyses of specific hypotheses about motor and perceptual-motor impairments in ASD (Smith, 2004). The study makes a significant contribution to the literature for two reasons. One is that this is one of the first to apply the contemporary literature on clumsy children to address motor dysfunction in AS. The second is that, despite the somewhat small sample size, this was the largest study to date to address potential causes of clumsiness in children and adolescents with AS (i.e., the AS sample was larger than those of Weimer et al., 2001 and Rinehart et al., 2001, the only two studies published on the components of motor dysfunction in AS). This latter fact reflects the reality that even in major diagnostic health science centres it can be difficult to assemble large samples of youths with AS.

Interestingly, there is a significant amount of overlap between the Dean-Woodcock and the measures completed by Weimer et al. (2001). Specifically, they found no significant differences on tests of basic motor skills in comparison with typically developing controls, but did find differences on tests of apraxia (especially whole body posturing), tandem gait, and one-leg balance (especially with eyes closed), and repetitive finger-thumb apposition. They interpreted these results as being indicative of sensory, and especially proprioceptive dysfunction, rather than motor dysfunction, though this conclusion may have been somewhat premature. Similarly, the present study also documented differences on tests of apraxia (Mime Movements), gait (Gait and Station), and repetitive movements [Coordination: Hand to Thigh (right) and (left)]; however, balance (Romberg) was not an issue, whether it was measured by clinical judgement or using the very sensitive force platform. In addition, the present study documented significant differences in construction (a cross, but not a clock) and finger tapping, bilaterally. Neither of these findings were noted by Weimer et al., who also assessed finger-tapping and construction (using the Beery-Buktenika Test of Developmental Visuo-Motor Integration). The reasons for the differences between the studies are unclear, especially for finger-tapping differences. The methodology of the present study is more reliable and valid in terms of measurement of balance (Romberg) because of the use of the force-plate, though the measurement of construction in Weimer et al.'s study may be stronger due to their more extensive assessment of construction. However, due to the significant differences on Finger-Tapping, their assertion that clumsiness in AS is due to proprioceptive deficits would not be supported.

The results of the Dean-Woodcock Motor Battery provide few insights as to the locus of motor dysfunction in the present study, spanning the cortical and subcortical motor factors. This failure to document a single causal factor is in keeping with the other results of the study.

Three primary hypotheses were tested in the present study; the relevance of findings of each are discussed in turn below.

Hypothesis #1: Timing Production and Perception

The purpose of including measures of timing ability was to determine whether the clumsiness seen in AS might be a result of accurate movements completed at inaccurate times. This hypothesis also has ramifications for theories of the causes of social problems in AS, though this hypothesis was not tested directly at this time. Wing (2002) has suggested that there are two causes of timing errors (1) an inaccurate central timer which might also be implicated in time estimation deficits and (2) a motor implementation delay that is likely variable and unpredictable. Although Wing's methodology could not be applied to the data collected in the present study because of the failure of the model's assumptions to be met, these two causes can be addressed on the basis of the data that is available.

First, there were no differences in the timing perception task, a perceptual discrimination task. There are two possible causes of this finding, (a) there are no differences in the central timer of the two groups, or (b) that a property of the task itself made it insensitive to subtle group differences. The latter cause can not be addressed without further research, but further data are available to address the former. On the

timing production task, it was the variability in inter-response intervals that was the primary variable of interest; however, the overall mean duration of intervals was similar in both groups. Both groups had a tendency to elongate their inter-response intervals, but there was no difference in the extent to which the two groups did this (both elongated the 400 ms intervals to just over 550 ms, and group differences were negligible with similar standard deviations). Had the central timer of the AS group been inaccurate, their average inter-response intervals would have likely differed to a greater extent, or the variability in the average inter-response intervals would have been greater. As such, the evidence from the timing production task supports the findings of the timing perception task in suggesting that there were no differences in central timing ability.

However, the group with AS was more variable in the implementation of their repeated taps. Within the ten trials of 30-interval tapping tasks, there was more variability within each trial in the AS group than the control group. So although there is no evidence of central timing problems, the AS group did differ in their timing ability, but the cause of the timing difficulties is likely to lie within the motor system. Further, this variability in tapping is highly correlated with other aspects of motor ability, including such diverse skills as grip strength, gait and station, construction, and hand-to-thigh movements.

Lundy-Eckman et al. (1991) found that this type of variability was seen in their group of children with soft signs indicative of cerebellar deficits; however, in their study, these children also had difficulties with the perceptual task. It may be early to ascribe neuroanatomical underpinnings to group differences on one measure, but cerebellar deficits have been classically linked to dysmetria, a disorder of judgement of distances.

Miall and Reckess (2002) cite compelling literature that suggests that dysmetria (overshooting a motor target) may result from a lack of feed-forward input from the cerebellum (a "sensory predictor"), and may reflect dysfunction in motor timing. However, the basal ganglia also contribute to timing abilities, so at the present time, all that can be said is that it is likely that extrapyramidal systems are involved in the genesis of the timing dysfunction noted in the present study (Wing, 2002).

It was not apparent why there were so many violations of the Wing-Kristofferson model in the present study, not only by the AS group, but also by the controls. It is possible that this may be a developmental effect; most evaluations of the Wing-Kristofferson model have been completed in adult samples. It is possible that the model may not hold in pediatric samples.

Hypothesis #2: Motor Planning

Hughes (1996) found a sizeable difference between her autistic sample and normally developing, but younger sample that was matched by developmental level. As discussed above, differences in the present study and Hughes' data reveal that the AS and the autistic samples performed rather similarly, but that Hughes' control group performed much better than the control group in the present study. A developmental effect is the most likely explanation, though the developmental effect is somewhat unusual in that the older group performed "worse" than the younger group. Although success on the task, as Hughes defined it, was a "comfortable" thumb-up grip, this grip may not be "uncomfortable", more difficult, or less efficient than a thumb-down grip in older individuals as they try to put the rod in the well. With development, increased skill in a

variety of motor behaviours may make the comfort differences between the grip styles inconsequential. That is, while the younger children in Hughes' control group may have "planned" a comfortable grip so as to avoid an uncomfortable thumb-down grip and successfully place the rod in the well, the older control sample in this study may not have found that the thumb-down grip changed their level of comfort or level of success, and therefore, not needed to plan their movement in advance.

This task, then, may not have assessed motor planning in the way that was anticipated, given that it had not been attempted in older samples before. Further assessments of motor planning may be necessary in future studies.

The developmental effect on this task reflects Latash's (2000) work on motor redundancy, also referred to as the degrees of freedom problem (Tupper & Sondell, 2004), which suggests that there are an infinite number of ways of completing a movement, and that it can be difficult to label one type of movement "better" than another without completely understanding the movement.

Hypothesis #3: Dorsal Visual Stream

The results of this study suggest that while there are no differences in the ability of people with AS to visually detect movement, their perception of human movement may be inaccurate. Furthermore, they are hyperreactive to virtual motion.

The finding that people with AS were similar to controls in terms of their ability to perceive motion was unexpected, and inconsistent with the findings of Spencer et al. (2000) and Milne et al. (2002) who examined this finding in autism. Both studies provided statistics that suggest a large effect size (Cohen's d = .85), while the effect size

noted in the present study was small (Cohen's d = .24) suggesting that the difference was not due to the sample size. However, Spencer et al. noted a developmental trend in their data that suggested smaller effect sizes in their oldest (10 to 11 year old) group than in their youngest (7 year old) group. As such, the older sample of the present study may have eliminated differences. However, if motion perception differences improve with age, this might be a less compelling cause of motor dysfunction, as the motor dysfunction continues into adolescence, and other aspects of visual dysfunction are apparent in this older sample (see below). It is also possible that simple motion perception deficits are seen only in autism, though the findings of Sigmundsson et al. (2003) suggest that these findings extend even to 10 to 11 year old clumsy children without a PDD diagnosis. Replication of the results with a younger AS group would be very informative.

A lack of motion perception deficits, however, does not necessarily suggest an intact dorsal visual stream (Braddick et al., 2000). Blake et al. (2003) found deficits in the perception of point-of-light walkers in autism; the results of the present study suggest a similar finding in AS.

These findings are consistent with the theory that the perception of motion is separate from the perception of stimuli for the purpose of action (Milner & Goodale, 1995). Perception of motion that is not related to biological movement may be a ventral stream task or involve primary areas of the dorsal stream, including area MT/V5. In fact, performance on a global motion task was affected in patients with cerebellar lesions; these same patients had no impairment on tests of biological motion, suggesting that the perception of global motion and the perception of biological motion are at least somewhat independent (Jokisch, Troje, Koch, Schwarz, & Daum, 2005).

To assess the integrity of the dorsal stream, action-relevant tasks may be required; in the present study the biological motion and virtual reality tasks were used to assess this aspect of visual perception. There is evidence to suggest that the perception of movements is linked to the ability to produce them; for example, people's perception of human movement is much more sensitive than their perception of non-human movement (Jacobs, et al., 2004). There are two primary reasons for why this may be, the first relating to a functional linkage between the visual and motor systems (i.e., that the dorsal visual stream interacts with motor systems, perhaps through mirror neurons), and the second being that visual sensitivity to human movement is enhanced by the frequency with which human movements are viewed in social contexts. As such, the weakness that the AS group had on the biological motion task in this study could potentially have at least two causes: (1) deficits in biological motion perception are independent of experience and simply a reflection of a less functional motor system, or (2) that people with AS have social deficits that reduce their exposure to and experience with biological motion that are independent of their motor abilities. Indeed, in the present study, the biological motion task showed significant group differences, but when controlling for group differences, it was not significantly correlated with participants' motor skills. Research has suggested that in normally developing individuals, it is a combination of experience dependent and experience independent processing that underlie visual analysis of human movement (Jacobs et al., 2004).

Finally, the virtual reality task did demonstrated hyper-reactivity to virtual motion, which was in keeping with the findings of Gepner and Mestre (2002), though the children with AS in the present study did not show less stability with their eyes closed as

they did in previous research. This finding is consistent with previous findings of a linkage between the visual and motor systems, given that all participants were aware of the fact that any perceived motion was simply virtual, but that the AS participants were more likely to errantly use that virtual visual information to adjust their balance. This finding does not support the experience dependent theory of visual motion processing (because the groups should not differ in the amount of room-movement they have perceived), but rather a coupling of the visual and motor systems.

Limitations of the Present Study

The present study has a number of limitations, the most significant of which is the small sample size (14 cases with AS). This is a common problem in research on AS, due to the low prevalence of AS, and although this is the largest study of its kind completed, there is a need in the literature to correct this problem. In addition, while the present study was designed to survey a number of areas of possible dysfunction (e.g., are there problems with movement timing in AS?), future studies may need larger samples in order to address more refined hypotheses (e.g., what is the source of the timing deficits in AS?).

A second limitation of the study was the use of a clinically referred sample. Although considerable effort was made to use measures of AS symptomatology, these cannot compare with a full clinical assessment, which might also include detailed histories (e.g., the ADI-R and/or Childhood Autism Rating Scale), measures of autism-spectrum symptoms (e.g., the ADOS-G), and neuropsychological assessments of cognitive ability and associated symptoms (e.g., a nonverbal learning disability). The

source of the referrals was a children's health centre that was equipped to complete all of the above, so diagnoses may be presumed to reflect this level of investigation.

A third limitation of this study was that there were actually few direct analyses of movement (save the analysis of resistance to movement in the virtual reality environment). Eventually, in order to understand clumsiness, there will need to be a greater focus on the dynamics of movements that lead to the clumsiness. However, because of the degrees-of-freedom or motor redundancy issues it is currently difficult to make judgements as to what constitutes a "good" movement. As such, instead of looking at the outcome of a movement, an information-processing approach was used to understand at what level of information processing a movement might be ineffective or clumsy. Future research that ties the information processing approach with an analysis of movement outcome would be important. Such research might, for example, use documented movement timing problems to predict where a movement might break down and assess the relationship of the timing problem to the movement outcome. The reach-to-grasp movement, for example, which has been extensively studied in normally developing individuals, might highlight movement timing problems.

The second reason for not addressing movement outcome directly was that although the equipment was available (the VICON motion analysis system), without a theoretical basis upon which to predict motor dysfunction, it would be difficult to postulate an *a priori* hypothesis of the movements that would be expected to be clumsy. There was very little extant literature describing the *type* of movement dysfunction upon which to base any specific hypotheses. Many articles described classes of motor dysfunction in functional terms (e.g., gross motor skills, ball throwing skills), but did not

elaborate or qualitatively describe their observations. In the context of the complexity of the modelling of human movement (i.e., dozens of possible angles and trajectories of movements are measured with the VICON), it would be difficult to complete valid research with small (i.e., low statistical power) samples.

Although the biological motion perception task and the postural stability task are referred to as being associated with dorsal visual stream functions, it is important to note that this has yet to be determined empirically. The description of the dorsal visual stream as involving vision for action is generally in keeping with the requirements of these tasks. However, further research, particularly functional neuroimaging work is necessary in confirming the brain systems and structures associated with these tasks. As mentioned above, the ventral and dorsal visual streams include a great deal of 'cross-talk' and are highly interconnected, making the attribution of a visual perception task to one and only one visual stream difficult.

Another limitation of the current study was the lack of a developmental approach. The sample spanned the ages of 7 and 23 years, through which there is considerable developmental change in movements. Issues in the measurement of movement planning, for example, did seem to be undermined by developmental effects. To correct for this problem participants were matched on the basis of age to minimize group differences, but it is possible that the results may be affected by qualitative changes in development. If, for example, the younger children completed tasks differently that the older adolescents and young adults, group differences might be less apparent. This limitation may be reflected in a higher frequency of Type II errors than Type I errors, however. Developmental effects were probed on several occasions; however, these were limited in

their statistical power by small sample sizes, and more detailed analyses of the effects of development may be required in future studies.

Another limitation was the use of the Dean-Woodcock as a criterion of clumsiness. The Dean-Woodcock is a combination of a neurological and neuropsychological assessment of motor abilities, which is a relatively less advanced method of assessing motor skills than a more ecological approach that assesses the child's ability to carry out meaningful goal-oriented movements (Tupper & Sondell, 2004). However, the Dean-Woodcock was selected because it is norm-referenced which was useful in establishing the severity of motor symptoms. In addition, it was the only norm-referenced measure of motor ability available at the time of data collection that spanned the adolescent and young-adult age range. Finally, it is also a published measure, which is either known to, or available to clinicians addressing motor problems with their patients.

Conclusion

Erratic timing and abnormalities in skills theoretically associated with dorsal visual stream functions are both correlated with motor dysfunction in AS, and both are plausible causes, though this quasi-experimental study does not address causation directly. The most conspicuous question left unanswered by this study is whether the clumsiness in AS represents two problems – dorsal visual stream dysfunction and timing problems – or a more parsimonious single core deficit that causes both problems. Dysfunction in the dorsal visual stream might appear to implicate cortical systems in AS. However, it would be difficult to imagine that visual deficits could lead to motor timing

deficits or the pattern of dysfunction seen across tests on the Dean-Woodcock. There is evidence that generally clumsy children may also have some dorsal stream dysfunction (Sigmundsson et al. 2003) to some extent. Because the dorsal stream may be intimately connected with the ability to perform movements, it may be that clumsiness or motor dysfunction undermines the ability of this visual system to function properly. Dorsal stream dysfunction, may, therefore, be a symptom of clumsiness, rather than a cause. Further research is required.

That a single core deficit was not immediately apparent may not be surprising, given that motor dysfunction is seen in so many developmental disorders (Dewey, Crawford, Wilson, & Kaplan, 2004). What is needed is a systematic comparison of the quality of motor dysfunction across developmental conditions. The motor system is an integral, if difficult to measure, part of the nervous system. Patterns of dysfunction, may, like patterns of cognitive functions, lead to a method of evaluating the validity of our nosological diagnostic system. While the present study does provide insight into a small number of functions in a single disorder, what is needed is an assessment of a greater number of movement-related variables across a greater number of psychiatric and neurodevelopmental disorders.

Indeed, it is possible that replication of the results of the present study with an AS group and an autistic control group may provide evidence of their distinctness. Gepner and Mestre (2002) found in their very small sample that AS participants were less stable in the face of virtual motion, while autistic participants were more stable; the present study replicated the findings for the AS sample. It would be informative to replicate the findings of the autism sample. In addition, several studies in autism have noted global

motion deficits; however, the AS sample in the present study did not have global motion deficits. If these findings were replicated in a study directly comparing AS and autism, it would provide the first distinctions between AS and autism that do not reflect differences in severity, and the strongest evidence to date of the theory that autism and AS do not lie on a spectrum of severity, but have underlying qualitative differences.

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Appendix I: Consent Forms

UNIVERSITY OF VICTORIA
OFFICE OF THE VICE-PRESIDENT, RESEARCH
HUMAN RESEARCH ETHICS COMMITTEE

Participant Consent Form
For participants with AS

Motor Skills in Asperger's Disorder

You are being invited to participate in a study entitled "Motor Skills in Asperger's Disorder" that is being conducted by Kelly Price, M.Sc. and Dr. Kimberly Kerns. I, Kelly Price am a Graduate Student in the department of Psychology at the University of Victoria and you may contact me if you have further questions by phone (250-472-4195) or email (kprice@uvic.ca).

As a Graduate student, I am required to conduct research as part of the requirements for a doctoral degree in Clinical Neuropsychology. This is being conducted under the supervision of Dr. Kimberly Kerns. You may contact my supervisor by phone (250-721-7553) and by email (kkerns@uvic.ca).

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equipment, force plates that measure the pattern of forces you put on the floor, and other measures of motor skills. Your parents will also be asked to complete a questionnaire that addresses symptoms of Asperger's Disorder.

There are no known or anticipated risks to you by participating in this research. You will be asked to participate for up to four hours, with a break in the middle if you would like. Testing will occur at the University of Victoria, in L-Hut, room 41 and/or 50.

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The researcher may have a relationship to some participants as a volunteer from the Asperger's Group. To help prevent this relationship from influencing your decision to participate, the following steps to prevent coercion have been taken: no information about your involvement in this study will be shared, nor will your participation in any way affect any of the services offered by the Mental Health Program or the Vancouver Island Health Authority.

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Name of Participant	*Signature*	*Date*
Kelly Price, M.Sc.		
Name of Investigator	*Signature*	*Date*

A copy of this consent will be left with you, and a copy will be taken by the researcher.

UNIVERSITY OF VICTORIA
OFFICE OF THE VICE-PRESIDENT, RESEARCH
HUMAN RESEARCH ETHICS COMMITTEE

Participant Consent Form
Control Group

Motor Skills in Asperger's Disorder

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You are being asked to participate in this study because you expressed interest in participating and because you are similar in terms of gender and age to one of the youths with Asperger's Disorder. Your participation allows us to determine how people without Asperger's perform on these measures, and allows us to determine if there are any differences between people with and without Asperger's.

If you agree to voluntarily participate in this research, your participation will include completing a questionnaire regarding symptoms of Asperger's (some of which may seem unusual to you), and completing measures of reasoning, visual perception of movement, movement planning, timing, force control, and motor skills. Many of these activities will be completed on a computer, though others will involve using virtual reality equipment, force plates that measure the pattern of forces you put on the floor, and other measures of motor skills. Your parents will also be asked to complete a questionnaire that addresses symptoms of Asperger's Disorder.

There are no known or anticipated risks to you by participating in this research. You will be asked to participate for up to four hours with a break in the middle if you would like. Testing will occur at the University of Victoria, in L-Hut, room 41 and/or 50.

The potential benefits of your participation in this research include helping people to better understand the development of motor skills in Asperger's and what differences there may be in how the nervous system processes this information. In addition, upon completion of the study, you will be compensated for your time with a $15 honorarium. It is important for you to know that it is unethical to provide undue compensation or inducements to research participants and, if you agree to participate in this study, this form of compensation to you must not be coercive. If you would not otherwise choose to participate if the compensation was not offered, then you should decline.

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Kelly Price, M.Sc.		
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UNIVERSITY OF VICTORIA
OFFICE OF THE VICE-PRESIDENT, RESEARCH
HUMAN RESEARCH ETHICS COMMITTEE

Parent Consent Form
Parents of Participants with AS

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This consent form, a copy of which will be provided to you and your son/daughter for your records and as a reminder of your right to withdraw from this research at any time, is only part of the process of informed consent. It will give you important details of what the research is about, and what your participation will involve. If you would like more details about something mentioned here, or about information not included here, please do not hesitate to ask – all of your questions are important to us and we will answer them to the best of our ability. Please take the time to read this consent form carefully to understand any accompanying information.

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applied to the floor, and other measures of motor skills. Parents will also be asked to complete a questionnaire that addresses symptoms of Asperger's Disorder.

There are no known or anticipated risks to you or your child by participating in this research. Your child will be asked to participate for up to four hours with a break in the middle. Testing will occur at the University of Victoria, in L-Hut, room 41 and/or 50.

The potential benefits of participation in this research include helping to better understand the development of motor skills in Asperger's and what differences there may be in how the nervous system processes this information. In addition, upon completion of the study, your child will be compensated for your time with a $15 honorarium. It is important for you to know that it is unethical to provide undue compensation or inducements to research participants and, if you and your child agree to participate in this study, this form of compensation to you must not be coercive. If you and your child would not otherwise choose to participate if the compensation was not offered, then you should decline.

Your participation and your child's participation in this research must be completely voluntary. If you and your child do decide to participate, you may withdraw at any time without any consequences or any explanation. If you do withdraw from the study your data and your child's data will not be used in subsequent analyses.

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_____	_____	_____
Name of Participant	*Signature*	*Date*

Please retain a copy of this consent form, and return the other (signed, if you agree to complete the questionnaire) to the researcher.

UNIVERSITY OF VICTORIA
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Your participation and your child's participation in this research must be completely voluntary. Please complete the attached questionnaire only if you feel comfortable doing so.

In terms of protecting your anonymity no information regarding your identity will be released or recorded in the data set. Your confidentiality and the confidentiality of the data will be protected by storing records independently of the identifying information used to contact participants. All data will be stored in a locked file cabinet at the University of Victoria. No identifying information will be included in the database.

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_____	_____	_____
Name of Parent	*Signature*	*Date*

Please retain a copy of this consent form, and return the other (signed, if you agree to complete the questionnaire) to the researcher.

Appendix II: The screening questionnaire for Asperger Syndrome and other high functioning autism spectrum disorders in school age children (ASSQ, Ehlers *et al.*, 1999),

ASSQ	Participant Code Number:

This child/youth stands out as different from others of his/her age in the following way:

		No	Somewhat	Yes
1	Is old fashioned or precocious	ρ	ρ	ρ
2	Is regarded as an "eccentric professor" by the other children	ρ	ρ	ρ
3	Lives somewhat in a world of his/her own with restricted idiosyncratic intellectual interests	ρ	ρ	ρ
4	Accumulates facts on certain subjects (good rote memory) but does not really understand the meaning	ρ	ρ	ρ
5	Has a literal understanding of ambiguous and metaphorical language	ρ	ρ	ρ
6	Has a deviant style of communicating with a formal, fussy, old-fashioned, or "robotlike" language	ρ	ρ	ρ
7	Invents idiosyncratic words and expressions	ρ	ρ	ρ
8	Has a different voice or speech	ρ	ρ	ρ
9	Expresses sounds involuntarily; clears throat, grunts, smacks, cries, or screams	ρ	ρ	ρ
10	Is surprisingly good at some things and surprisingly poor at others	ρ	ρ	ρ
11	Uses language feely but fails to make adjustments to fit social contexts or the needs of different listeners	ρ	ρ	ρ
12	Lacks empathy	ρ	ρ	ρ
13	Makes naïve and embarrassing remarks	ρ	ρ	ρ
14	Has a deviant style of gaze	ρ	ρ	ρ
15	Wishes to be sociable but fails to make relationships with peers	ρ	ρ	ρ
16	Can be with other children but only on his/her terms	ρ	ρ	ρ
17	Lacks best friend	ρ	ρ	ρ
18	Lacks common sense	ρ	ρ	ρ

		No	Somewhat	Yes
19	Is poor at games: no idea of cooperating in a team, scores "own goals"	ρ	ρ	ρ
20	Has clumsy, ill coordinated, ungainly, awkward movements or gestures	ρ	ρ	ρ
21	Has involuntary face or body movements	ρ	ρ	ρ
22	Has difficulties in completing simple daily activities because of compulsory repetition of certain actions or thoughts	ρ	ρ	ρ
23	Has special routines: insists on no change	ρ	ρ	ρ
24	Shows idiosyncratic attachment to objects	ρ	ρ	ρ
25	Is bullied by other children	ρ	ρ	ρ
26	Has markedly unusual facial expressions	ρ	ρ	ρ
27	Has markedly unusual posture	ρ	ρ	ρ

Appendix III: Asperger Syndrome Diagnostic Interview (ASDI, Gillberg *et al.*, 2001)

ASDI	Participant Code Number:

Relation to individual rated: ρ Mother
ρ Father
ρ Other (Please specify: _____)

Please complete all of the following questions. If a symptom is applicable to any noticeable extent, please check "does apply".

		Does Not Apply	Does Apply
1	Does he/she exhibit considerable difficulties interacting with peers?	ρ	ρ
2	Does he/she exhibit a low degree of concern or a seeming lack of interest in making friends or interacting with peers?	ρ	ρ
3	Does he/she have problems appreciating social cues, i.e., does he/she fail to note changes in the social conversation/interaction or to take account of such changes in his/her interaction with other people?	ρ	ρ
4	Does he/she exhibit socially or emotionally inappropriate behaviours?	ρ	ρ
5	Is there a pattern of interest or a specific interest which takes up so much of his/her time that time for other activities is clearly restricted?	ρ	ρ
6	Is there some repetitive quality to his/her interest patterns or specific interests?	ρ	ρ
7	Are his/her interest patterns based more on rote memory than on true meaning?	ρ	ρ
8	Does he/she try to introduce and impose routines, rituals or interests on himself/herself in such a way as to produce problems for himself/herself?	ρ	ρ
9	Does he/she try to introduce and impose routines, rituals or interests on himself/herself in such a way as to produce problems for others?	ρ	ρ
10	Was his/her language development delayed?	ρ	ρ

		Does Not Apply	Does Apply
11	Is his/her language 'superficially perfect' regardless of whether or not there are comprehension problems or other speech and language problems?	ρ	ρ
12	Is his/her language formal, pedantic, or 'overly adult'?	ρ	ρ
13	Is there any characteristic about his/her voice (pitch, volume, quality, intonation, word stress, 'prosody etc) which you find peculiar or unusual?	ρ	ρ
14	Are there any comprehension problems (including misinterpretations of literal/implied meanings)?	ρ	ρ
15	Does he/she make limited use of gestures?	ρ	ρ
16	Is his/her body language awkward, gauche, clumsy, strange, or unusual?	ρ	ρ
17	Are his/her facial expressions limited to a rather small repertoire?	ρ	ρ
18	Is his/her general expression (including facial) sometimes inappropriate?	ρ	ρ
19	Is his/her gaze stiff, strange, peculiar, abnormal, or odd?	ρ	ρ
20	Has he/she been noted to perform poorly on neurodevelopmental examinations (motor clumsiness) in the past?	ρ	ρ

Please include any other comments you have below:

Appendix IV: Autism-Spectrum Quotient (AQ, Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001)

Take the AQ Test	Participant Code Number:

Check the box that matches the extent to which you agree with the following statements. If you're not sure, just check the box that is closest to how you would answer. Please complete ***all*** the questions.

		Definitely Agree	Slightly Agree	Slightly Disagree	Definitely Disagree
1	I prefer to do things with others rather than on my own.	ρ	ρ	ρ	ρ
2	I prefer to do things the same way over and over again.	ρ	ρ	ρ	ρ
3	If I try to imagine something, I find it very easy to create a picture in my mind.	ρ	ρ	ρ	ρ
4	I frequently get so strongly absorbed in one thing that I lose sight of other things.	ρ	ρ	ρ	ρ
5	I often notice small sounds when others do not.	ρ	ρ	ρ	ρ
6	I usually notice car number plates or similar strings of information.	ρ	ρ	ρ	ρ
7	Other people frequently tell me that what I've said is impolite, even though I think it is polite.	ρ	ρ	ρ	ρ
8	When I'm reading a story, I can easily imagine what the characters might look like.	ρ	ρ	ρ	ρ
9	I am fascinated by dates.	ρ	ρ	ρ	ρ
10	In a social group, I can easily keep track of several different people's conversations.	ρ	ρ	ρ	ρ
11	I find social situations easy.	ρ	ρ	ρ	ρ
12	I tend to notice details that others do not.	ρ	ρ	ρ	ρ
13	I would rather go to a library than to a party.	ρ	ρ	ρ	ρ
14	I find making up stories easy.	ρ	ρ	ρ	ρ

		Definitely Agree	Slightly Agree	Slightly Disagree	Definitely Disagree
15	I find myself drawn more strongly to people than to things	ρ	ρ	ρ	ρ
16	I tend to have very strong interests, which I get upset about if I can't pursue.	ρ	ρ	ρ	ρ
17	I enjoy social chitchat.	ρ	ρ	ρ	ρ
18	When I talk, it isn't always easy for others to get a word in edgewise.	ρ	ρ	ρ	ρ
19	I am fascinated by numbers.	ρ	ρ	ρ	ρ
20	When I'm reading a story, I find it difficult to work out the characters' intentions.	ρ	ρ	ρ	ρ
21	I don't particularly enjoy reading fiction.	ρ	ρ	ρ	ρ
22	I find it hard to make new friends.	ρ	ρ	ρ	ρ
23	I notice patterns in things all the time.	ρ	ρ	ρ	ρ
24	I would rather go to the theatre than to a museum.	ρ	ρ	ρ	ρ
25	It does not upset me if my daily routine is disturbed.	ρ	ρ	ρ	ρ
26	I frequently find that I don't know how to keep a conversation going.	ρ	ρ	ρ	ρ
27	I find it easy to 'read between the lines' when someone is talking to me.	ρ	ρ	ρ	ρ
28	I usually concentrate more on the whole picture, rather than on the small details.	ρ	ρ	ρ	ρ
29	I am not very good at remembering phone numbers.	ρ	ρ	ρ	ρ
30	I don't usually notice small changes in a situation or a person's appearance.	ρ	ρ	ρ	ρ
31	I know how to tell if someone listening to me is getting bored.	ρ	ρ	ρ	ρ
32	I find it easy to do more than one thing at once.	ρ	ρ	ρ	ρ
33	When I talk on the phone, I'm not sure when it's my turn to speak.	ρ	ρ	ρ	ρ
34	I enjoy doing things spontaneously.	ρ	ρ	ρ	ρ
35	I am often the last to understand the	ρ	ρ	ρ	ρ

	point of a joke.				
		Definitely Agree	Slightly Agree	Slightly Disagree	Definitely Disagree
36	I find it easy to work out what someone is thinking or feeling just by looking at their face.	ρ	ρ	ρ	ρ
37	If there is an interruption, I can switch back to what I was doing very quickly.	ρ	ρ	ρ	ρ
38	I am good at social chitchat.	ρ	ρ	ρ	ρ
39	People often tell me that I keep going on and on about the same thing.	ρ	ρ	ρ	ρ
40	When I was young, I used to enjoy playing games involving pretending with other children.	ρ	ρ	ρ	ρ
41	I like to collect information about categories of things (e.g., types of cars, birds, trains, plants).	ρ	ρ	ρ	ρ
42	I find it difficult to imagine what it would be like to be someone else.	ρ	ρ	ρ	ρ
43	I like to carefully plan any activities I participate in.	ρ	ρ	ρ	ρ
44	I enjoy social occasions.	ρ	ρ	ρ	ρ
45	I find it difficult to work out people's intentions.	ρ	ρ	ρ	ρ
46	New situations make me anxious.	ρ	ρ	ρ	ρ
47	I enjoy meeting new people.	ρ	ρ	ρ	ρ
48	I am a good diplomat.	ρ	ρ	ρ	ρ
49	I am not very good at remembering people's dates of birth.	ρ	ρ	ρ	ρ
50	I find it very easy to play games with children that involve pretending.	ρ	ρ	ρ	ρ

Appendix V: Timing Perception Task (Time Comparison)

"Next you'll hear a pair of rhythmic beats, and I'd like you to decide whether the second rhythm is playing at a faster or slower speed than the first. The first rhythm will go at a regular speed, that is, it will stay the same for the whole game, but you'll hear it each time, just to remind you how fast it goes. The second rhythm of each pair though, will change throughout the game. Sometimes it will be faster than the first, regular speed, and sometimes it will be slower than the regular speed. Listen very carefully to decide whether it is faster or slower.

"Don't be distracted by how long the beats play for. Some of them play for about 5 or 10 seconds, while others only play just long enough to hear a few beats.

"The game starts with two practice trials. Some people have found this one to get boring, but remember that it only takes about 5 to 10 minutes."

Let the participant continue. Body movements and humming are allowed. Encourage the participant to pay attention if they appear not to be. Ensure that no clocks or watches are present.

Appendix VI: Timing Production Task

"Here I would like to see how well you can time your movements. You will hear a tone repeated at a regular speed. What I'd like you to do is press the zero key on this keypad whenever you hear the tone. Try to time your keypresses so that you are pressing the button at the exact same time that you hear the tone. After about a dozen tones, they will suddenly stop, but I would like you to keep pressing the button at the exact same speed until I tell you to stop."

Start with trial ###37.

"Great! Lets try that a few more times. Try to time your button presses so that they are the exact same speed or tempo as the tones that you hear."

Proceed from Trial ###01. After trial ###10, skip to trial ###51.

"Now we'll do the exact same thing again, but this time using a different tempo. This time you'll be pressing the button slower."

Continue until ###60, then discontinue task.

Appendix VII: Coloured Rod Test Instructions

Coloured Rod Test (adapted from Hughes, 1996)	**Participant Code Number:**	

Dominant Hand: **L R** Colour-blindness: Circle incorrect: **Red Blue Black Green**
All correct: ☐

- Say **"Now I'd like to see how well you can stand this rod in these two wells. When I ask you to grasp the rod, please hold it towards the end, not the middle. Let's practice."** While demonstrating, say, **"Remember that you wrap your hand completely around the rod; don't just use your fingers."**
- Allow participant to put the rod into each of the discs so that it stands upright. Allow up to 5 trials, or until they feel confident.
- Use dominant hand for all trials (ask participant to sit on hand if it is difficult to avoid using the non-dominant hand, but note this).
- Return hand to lap between trials.
- **"Now put your hands in your lap. Please listen to the colours I tell you carefully. First,..."**

"I want you to pick up the ____ half of the rod and place it in the ____ disc."

Colour on Dominant Side	**Rod Colour**	**Disc Colour**	**Grasp**	**Thumb Position**
Blue	Blue	Green	Under **Over**	⇧ ⇩
Blue	Red	Black	**Under** Over	⇧ ⇩
Red	Blue	Black	**Under** Over	⇧ ⇩
Blue	Blue	Black	Under **Over**	⇧ ⇩
Blue	Red	Green	**Under** Over	⇧ ⇩
Red	Red	Green	Under **Over**	⇧ ⇩
Blue	Red	Black	**Under** Over	⇧ ⇩
Red	Red	Black	Under **Over**	⇧ ⇩
Red	Red	Green	Under **Over**	⇧ ⇩
Red	Blue	Black	**Under** Over	⇧ ⇩
Red	Blue	Green	**Under** Over	⇧ ⇩
Blue	Blue	Black	Under **Over**	⇧ ⇩
Blue	Blue	Green	Under **Over**	⇧ ⇩
Red	Red	Black	Under **Over**	⇧ ⇩
Red	Blue	Green	**Under** Over	⇧ ⇩
Blue	Red	Green	**Under** Over	⇧ ⇩

Number of "unders" correct in first half: ___ second half: ___ total: ____

¾ "unders" correct in the 1st half: **Y N** 2nd half: **Y N** ¾ for whole test (6/8): **Y N**

Appendix VIII: Motion perception task instructions

RUNNING THE SUBJECT: MOTDX PROGRAM

1. The program is run from the "Program" menu. This menu has 2 options DEMO (for demonstration) and RUN. Choose DEMO to explain the task to the child. To aide the explanation of the task, DEMO mode stimuli have infinite stimulus durations.

Explain to the children that they will be see two patches of dots, one will always have completely random motion, the other will always have some dots that move back and forth together. Their task is to identify which panel ("one" or "two") contains the dots that are moving together. NOTE: You might want to put some stickers on the frame of the computer above each patch to denote the "first" and "second" panels.) Point to the appropriate patch to show the child the motion that they are looking for.

Because the coherent signal gets harder to see as the tests progresses there will be times when the child will be unsure which panel contained the coherent motion. In this case they MUST guess. Explain this to them. Give them a few Demo trials until you are sure that they understand the task (this shouldn't take more than 5 demo trials).

The experimenter should enter the child's responses by hitting the key appropriate key to the child's judgement. In this case the keys are "." (full stop "one") and "/" (forward slash; for "two"). These keys were chosen for ease of use by the experimenter who must sit to the right-hand side of the child during the experiment. Note: the motion program will accept a button press only after the stimulus has terminated (i.e., the motion stimuli have left the screen).

2. Four estimates of motion detection threshold will be collected. At the end of the first there will be a screen that gives the threshold estimate in %. Explain to the child what this means; the percentage represents the proportion of dots that need to be moving together in order to just detect the coherent motion, low numbers are good and to try to get a lower number for the second trial. Press Return to begin the second trial and run as above until the program terminates.

3. You can manually terminate the program (the same holds true for the Form task) at any time by pressing the ESC key and then exiting the program by typing "ALT-X" at the bottom left-hand side of the Program Menu. (The Alt-X combination terminates the program similarly).

Appendix IX: Instructions for the Form Program

RUNNING THE FORM PROGRAM

1. The program is run from the "Program" menu. This menu has 2 options DEMO (for demonstration) and RUN. Choose DEMO to explain the task to the children. Give them a few Demo trials until you are sure that they understand the task (again this shouldn't take more than about 3 DEMO trials).

2. Explain to the children that they will be see two patches of line segments, one will always have them completely randomised, the other will always have some that make a circle in the centre of either patch. Their task is to identify which panel ("one" or "two" as for the motion) contains the circle. The experimenter should enter the response by pressing the appropriate key (also same as for motion). Because the coherent signal gets harder to see as the tests progresses, there will be times when the children will be unsure which panel to choose. In this case they MUST guess. Explain this to them.

3. As for the MOTDX program, four threshold estimates will be collected. At the end of the first there will be a screen that gives the threshold estimate in %. Explain to the child what this means. It denotes the percentage of line segments that need to be coherently oriented (to concentric circles) in order to just see the motion. Press "Return" to begin the second trial and run as above until program terminates. NOTE: Program can be terminated at any time in the same way as for the Motion test by hitting the "ESC" key.

Appendix X: Instructions for the biological motion perception task

The Walkers Test	**Participant Code Number:**	

Show Video 1. Let me explain how to do this. First, we asked people to walk through the room, while our computers watched them. If you look carefully, you can see markers on the hips, knees, and ankles of this little girl. Show Example 1. **The computer turned these markers into dots, and here we can see how the girl moved. These dots represent her hips, knees, and ankles.** Show Video 2. **Then we asked this girl to walk through the room again, and this time she walked a little slower. Then we took the markers from one leg in the first video** (point) **and one leg from the second video** (point) **and put them together to make this.** Show Example 2. **Here you see that the legs are moving at different speeds, and it doesn't really even look like a person.**

Some of the people that we have in our videos have normal gaits or ways of walking, while others have odd or unusual gaits, including limps or movements that are different on each side of their body.

As you watch each of these trials, I want you to figure out two things. First, figure out whether there are two walkers overlaid, or just one walker. As soon as you can guess, press the button on the bottom. Then, decide whether the person is someone who is walking normally, or if they have an unusual gait. Any questions?

Trial #	Code Number				Time (s)	Number of Walkers	Normal/ Atypical	Comments, if any
1	D	N	3	C		1 **2**	**N** A	
2	D	N	2	A		1 **2**	**N** A	
4	S	A	3	E		**1** 2	N **A**	
5	D	A	2	B		1 **2**	N **A**	
6	S	N	3	A		**1** 2	**N** A	
7	S	N	3	B		**1** 2	**N** A	
8	S	N	1	B		**1** 2	**N** A	
9	S	N	1	C		**1** 2	**N** A	
10	S	A	3	A		**1** 2	N **A**	
12	S	N	3	C		**1** 2	**N** A	
13	S	N	1	E		**1** 2	**N** A	
14	S	A	3	B		**1** 2	N **A**	
15	D	A	1	E		1 **2**	N **A**	
16	S	N	2	C		**1** 2	**N** A	
17	S	A	2	D		**1** 2	N **A**	
18	S	A	2	B		**1** 2	N **A**	
19	D	N	3	A		1 **2**	**N** A	
20	S	N	3	D		**1** 2	**N** A	

21	S	A	2	E		**1** 2	N **A**	
22	D	N	1	A		1 **2**	**N** A	
23	S	A	1	A		**1** 2	N **A**	
24	D	N	3	D		1 **2**	**N** A	
25	S	N	2	D		**1** 2	**N** A	
26	D	N	3	E		1 **2**	**N** A	
27	D	N	2	C		1 **2**	**N** A	
28	D	N	2	E		1 **2**	**N** A	
29	S	A	1	B		**1** 2	N **A**	
30	S	A	1	D		**1** 2	N **A**	
31	D	A	3	D		1 **2**	N **A**	
33	D	A	1	C		1 **2**	N **A**	
34	D	A	2	D		1 **2**	N **A**	
35	S	A	3	D		**1** 2	N **A**	
36	D	A	1	D		1 **2**	N **A**	
37	D	A	1	A		1 **2**	N **A**	
38	D	N	1	D		1 **2**	**N** A	
40	S	N	2	A		**1** 2	**N** A	
41	S	N	2	E		**1** 2	**N** A	
42	S	A	2	A		**1** 2	N **A**	
43	D	A	3	A		1 **2**	N **A**	
44	D	A	3	B		1 **2**	N **A**	
45	D	A	1	B		1 **2**	N **A**	
46	S	N	3	E		**1** 2	**N** A	
47	D	A	2	C		1 **2**	N **A**	
48	S	A	3	C		**1** 2	N **A**	
49	S	A	1	C		**1** 2	N **A**	
50	S	N	1	A		**1** 2	**N** A	
51	D	A	3	E		1 **2**	N **A**	
52	D	N	2	D		1 **2**	**N** A	
53	S	A	1	E		**1** 2	N **A**	
54	D	A	2	A		1 **2**	N **A**	
55	S	N	1	D		**1** 2	**N** A	
56	D	A	2	E		1 **2**	N **A**	
57	D	N	1	E		1 **2**	**N** A	
59	S	A	2	C		**1** 2	N **A**	
60	D	N	1	C		1 **2**	**N** A	

Total Time		
Number of 1s correct		
Number of 2s correct		
Number of Ns correct		
Number of As correct		

Note that 5 trials were erased, so there are only 55 trials remaining. The order given above is still accurate.

Appendix XI: Balance Test Instructions

Balance Test

Instructions:
Record BOS points. Remember to zero plate between each trial (S must stand down).

Trial 1: "**I am going to get you to stand on this plate a total of 7 times. After the first few trials, we'll be using the virtual reality equipment. First, I'd like you to stand on the plate, and remain as still as you can until I tell you to step off the plate. This will take about 45 seconds.**

Trial 2: **"Now I'd like you to stand on the plate with your eyes closed, and again, remain as still as you can until I tell you to step off.**

Trial 3: **"Now I'd like you to wear this virtual reality equipment."** Ensure that the person is comfortable, able to see the screen, and aware that they are able to lift the screen if they are feeling ill. Reassure them that if they are about to fall at any point, you will be there to ensure that they are safe. **"Now, stay as still as you can, with your eyes open, until I tell you to step off."**

Trial 4: **"Now I am going to show you a moving hallway. Watch the screen and try to remain as still as you can, until I tell you to step off.**

Trial 5: **"Now I am going to show you another moving hallway. Again, watch the screen and try to remain as still as you can until I tell you to step off.**

Trial 6: **"This time you won't see anything on the screen except the desktop. I would like you to look left and right, moving your head, but keeping your eyes fixed on the desktop. When I tap your shoulder, look in that direction. Try to keep the rest of your body as still as you can, until I tell you to step off."**

Start tapping (on the right side) 3 seconds after the motion begins, and continue tapping every 3 second until the motion ends.

Trial 7: **"Last one. We'll do exactly what we did last time, except this time you'll see a hallway instead of the desktop. Remember to turn your head in the direction of the shoulder that I tap. Otherwise, keep your body as still as you can".**

Summary

Trial	Eyes	HMD	Display	Head
1	Open	No	-	Still
2	Closed	No	-	Still
3	Open	Yes	Desktop	Still

4	Open	Yes	Hallway with No Person	Still
5	Open	Yes	Hallway with Person	Still
6	Open	Yes	Desktop	Rotating L/R every 3 s
7	Open	Yes	Hallway with Person	Rotating L/R every 3 s