STATIC AND DYNAMIC BALANCE CONTROL IN CHILDREN WITH AUTISM SPECTRUM DISORDERS

By

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To my loving parents Judith and George Fournier

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Children with Autism Spectrum Disorders (ASD) typically exhibit impairments in three core symptom areas (deficits in communication, abnormal social interactions and restricted and/or repetitive behaviors). Within the third core category, symptoms related to stereotyped body movements and abnormalities in posture have been observed. Research suggests the postural control system in individuals with autistic disorder is immature and may never reach adult levels. Functional independence requires a postural control system that provides both postural stability during quiet stance and also dynamic stability as the body's center of mass (COM) moves away from its base of support. The purpose of this study was to identify postural control deficiencies associated with ASD during both static and dynamic postural challenges. Using functional tasks with increasing postural challenge, we investigated postural sway (movement of the center of pressure, COP) and separation of the COM from the base of support during quiet standing and displacements of the COP during gait initiation.

The hypothesis that children with ASD would have impaired postural control was supported. Statistical differences were detected between groups in all but one measure of postural sway. Further, the maximum separation between the COP and COM was on average 100% greater and more variable in children with ASD. These results seem to indicate an

immature control of posture during quiet standing. The hypothesis that children with ASD would have difficulty uncoupling the COP and COM during gait initiation was only partially supported. No statistical differences in posterior COP shift were detected between the groups, suggesting the COP shift mechanism for generating forward momentum is intact in children with ASD. However, significantly smaller lateral COP shifts were observed in children with ASD and may indicate instability or an alternative strategy for generating stance side momentum or lateral weight shifting.

CHAPTER 1
INTRODUCTION

Autism is a neurodevelopmental disorder diagnosed according to specific impairments in the areas of communication, reciprocal interaction and stereotypic behavior (DSM-IV., 2000). Autism Spectrum Disorders (ASD) is an inclusive term for individuals with autistic disorder and those diagnosed with Pervasive Developmental Disorder–Not Otherwise Specified (PDD–NOS) and Asperger's Syndrome (DSM-IV., 2000; Tanguay, Robertson, & Derrick, 1998). The primary impairments observed in children with ASD span across three broad categories: social interactions, communication and repetitive and restrictive behaviors. Associated features include diagnosis of mental retardation (~75%), uneven impairments in the development of cognitive skills, behavioral symptoms, altered responses to sensory stimuli, abnormalities in eating and mood and various non–specific neurological symptoms (primitive reflexes, delayed development of hand dominance and seizures) (DSM-IV., 2000). Within the third main category of symptoms (repetitive and restricted behaviors), previous research in the area of motor development has suggested that movement disturbances may be present during infancy and may be considered one of the earliest signs of autism (Teitelbaum, Teitelbaum, Nye, Fryman, & Maurer, 1998). Furthermore, motor problems have been the most frequently reported non–verbal deficits in children with autism (Noterdaeme, Mildenberger, Minow, & Amorosa, 2002). An immature postural control system can be a limiting factor on the emergence of other motor skills (such as coordinated hand/head movements and inhibition of reflexes), may constrain the ability to develop mobility and manipulatory skills (Shumway-Cook & Woollacott, 2001), and is of significant importance to quality of life. Therefore, systematically evaluating postural control in this population may be a first step towards determining the best approach for improving postural stability and related skills (mobility and manipulation).

With the etiology of ASD not being clearly defined, the specific pattern and source of motor deficits in this population remain unclear (Noterdaeme et al., 2002). However, the early onset of abnormal movements (within the first year of life) (Teitelbaum et al., 1998), perhaps even before communicative or social deficits manifest, suggests that motor deficits may be partially linked to some core characteristic of ASD (Leary & Hill, 1996; Nayate, Bradshaw, & Rinehart, 2005). Unfortunately, little attention has been given to motor deficits in the ASD population and have been referred to simply as a less important or a co–occurring syndrome (Noterdaeme et al., 2002). Leary & Hill (1996) in their review, however, suggest motor deficits likely impair the development of sufficient communicative and interactive skills (Leary & Hill, 1996). Indeed, Noterdaeme et al. (2002) suggested that motor disturbances in children with autism put an additional strain on the children's development; resulting in difficulty mastering daily activities such as eating with a knife and fork, stair climbing, writing, ball games and bicycling. Therefore, it appears that the occurrence of neuromotor deficits in autism may be a partial indication of the biological factors in the etiology of the disorder (Jones & Prior, 1985; Leary & Hill, 1996).

The ability to maintain an upright posture is a fundamental skill necessary for typical motor development in humans. Research suggests that individuals with autistic disorder have developmental delays in postural control with systems that never fully mature to adult levels (Kohen-Raz, Volkmar, & Cohen, 1992). The vestibular, somatosensory and visual systems are the afferent inputs involved in maintaining an upright posture. A deficit in any one of these systems or in the integration of information provided from these systems could affect the ability to maintain balance. Postural patterns employed by children with autistic disorder over the age of 6 years have been observed to differ from typically developing children, mentally retarded

children and adults with vestibular disorders. In addition, these older children with autistic disorder, when compared to typically developing children (ages 4 to 11 years), exhibited more variable and less stable postural control, particularly in the mediolateral direction (Kohen-Raz et al., 1992). Both children and adults with ASD have been observed to have impaired postural stability when compared to individuals with typical neuromotor development under conditions where one or more sensory inputs had been removed or modified. However, when afferent inputs were not modified, differences in postural sway are not as apparent (Gepner, Mestre, Masson, & de Schonen, 1995; Minshew, Sung, Jones, & Furman, 2004; Molloy, Dietrich, & Bhattacharya, 2003). In addition, when investigating age effects, data indicated a delayed development of the postural system in subjects with autistic disorder, which only began to improve at the age of 12 years, and never reached adult levels (Kohen-Raz et al., 1992; Minshew et al., 2004). These authors have therefore suggested that there is an involvement of the neural circuitry beyond the neural systems for social behavior, communication and reasoning. Furthermore, despite this information, there is still is a paucity of research investigating a wider range of functional tasks challenging both the static and dynamic postural control of this population.

Purpose of the Study

Functional independence requires a postural control system that provides both postural stability during quiet stance and also dynamic stability as the body's center of mass (COM) moves away from its base of support. The primary purpose of this study was to identify postural control deficiencies associated with ASD during both static and dynamic postural challenges. Using functional tasks with increasing postural challenge, we investigated postural sway and the separation of the COM from the base of support (center of pressure, COP) during quiet standing and manipulation of the COP during gait initiation.

Significance of the Study

The source and type of motor impairments in children with ASD remain unclear. The early onset of motor impairments suggests that motor deficits may indeed be related to some core characteristic of ASD. Delayed or abnormal balance control may constrain the ability for children with ASD to develop related stability or mobility skills. To date, limited research has focused on static balance in the ASD population. Furthermore, to the best of our knowledge, research in the area of dynamic balance in this population is non–existent. Using quantifiable measures of postural control that have been used successfully in other populations (elders, stroke, Parkinson's disease); findings from this investigation have the potential to provide important insight into static and dynamic control of children with ASD. Furthermore, by better characterizing these impairments, we can assist in the design of treatments that address postural instabilities early in development, which may help minimize or prevent subsequent emergence of deficits in other motor abilities.

Research Goals and Hypotheses

Specific Aim 1

To determine whether children with ASD have impaired postural control by quantifying COP measures during quiet stance.

Hypothesis 1

Based on previous research, we hypothesized that children with ASD would have increased postural sway. As a result, we believed the COP ranges (mediolateral and anteroposterior) and sway areas observed in children with ASD would differ from those observed in typically developing, aged–matched children. Specifically, we believed COP ranges and sway areas would be greater in children with ASD, indicating static balance instability.

Specific Aim 2

To determine whether children with ASD have impaired postural control by quantifying COP–COM distances during quiet stance.

Hypothesis 2

Based on previous research, we hypothesized that children with ASD would have increased postural sway. As a result, we believed the peak COP–COM distances observed in children with ASD would differ from those observed in typically developing, aged–matched children. Specifically, we believed peak COP–COM distances would be greater in children with ASD, indicating static balance instability.

Specific Aim 3

To determine whether children with ASD have impaired dynamic postural stability as the body transitions from a static to a changing base of support (gait initiation).

Hypothesis 3

We hypothesized that during gait initiation, children with ASD would have impaired abilities to uncouple the COP and COM, essential for propulsion in the forward and lateral directions. Specifically, we believed displacements of the COP would be decreased in children with ASD compared to typically developing, age–matched children, as seen in older individuals and those with disability.

CHAPTER 2
MATERIALS AND METHODS

Participants

Thirteen children with ASD (ages 8–16) were recruited from the Child & Adolescent Psychiatry Clinic at the University of Florida. Thirteen typically developing, chronologically aged–matched children (within one year) were recruited from the community and served as controls. We chose the age range of 8–16 years to ensure a relatively mature postural system in typically developing children, which in turn served as benchmarks for comparisons with children with ASD (Table 2–1). Descriptive data for children diagnosed with ASD (Leiter International Performance Scale-Revised (Leiter-R), Repetitive Behaviors Scales-Revised (RBS-R), Adaptive Behavior Assessment System (ABAS-II), and Brief Rating Inventory of Executive Functioning-Parent Form (BRIEF)) are presented in Tables 2-2, 2-3, 2-4 and 2-5 respectively.

Inclusion/Exclusion Criteria for Children with ASD

- Clinical diagnoses of ASD from a licensed professional (psychologist or physician) and confirmed with one of three scales (Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, Dilavore, & Risi, 1999), Social Communication Questionnaire (Rutter, Bailey, & Lord, 2003), Childhood Autism Rating Scale (Schopler, Reichler, & Renner, 1988)).
- No known genetic/medical conditions (fragile X syndrome, tuberous sclerosis, seizures) as confirmed by medical records/examinations.
- No known sensory deficits (not blind or deaf)
- Ambulatory with no significant physical impairments

Inclusion/Exclusion Criteria for Typically Developing Children

- No known sensory deficits (not blind or deaf)
- Ambulatory with no significant physical impairments
- No medical chart diagnoses of psychiatric (e.g. ADHD) or neurological (e.g. Tourette's Syndrome) disorders

Experimental Setup

All biomechanical testing was performed in the Biomechanics Laboratory, Center for Exercise Science at the University of Florida, Gainesville, Florida. Using a two adjacent forceplates (Type 4060–10, Bertec Corp., Columbus, OH), ground reaction forces (GRF) were recorded (360 Hz).

Testing Protocol

Static Balance (Quiet Stance)

All participants were asked to stand with their feet comfortably apart with one foot on each of the adjacent forceplates with a self–selected stance width. Foot positioning was marked on the initial trial and used for all subsequent trials. Participants were asked to stand as still as possible for 20 seconds with their arms comfortably at their side. Participants performed four experimental trials. Trials where voluntary movements were observed were rejected and additional trials were performed.

Dynamic Balance (Gait Initiation)

Participants were asked to stand on the adjacent forceplates as previously described. Participants began from a quiet stance position, and were given a verbal cue to initiate movement. Once the cue was given, participants were asked to take a short pause (approximately 2 sec) and then start walking with their preferred foot, at a self–selected speed, in the forward direction. Data collection began with the verbal cue and continued for several steps after both feet had left the forceplates. Participants were allowed several practice trials and data from 4 experimental trials were collected and used in subsequent analyses.

Data Reduction

Ground reaction forces and moments collected from the two forceplates were processed and the location of the COP for each forceplate was calculated (Appendix A). The location of the

COP_{net} from the two forceplates, in both the mediolateral (ML) or anteroposterior (AP) directions was calculated according to previously reported literature (Winter, Patla, Ishac, & Gage, 2003) (Appendix A). Once the COP was computed, the peak displacements in the mediolateral and anteroposterior directions were determined and the sway area was calculated. Because differences in stance width can influence postural control, specifically in the mediolateral direction (Rocchi et al., 2006), displacements during quiet stance and gait initiation trials were normalized to the individual's stance width and foot length.

Typically the position of the COM is calculated from positional data. This technique uses a weighted sum to estimate the COM for each participant using the 3D positional data from markers and anthropometric data. It is important to note however, that this technique requires that reflective markers (stickers) be affixed to an individual's skin in order to define body segments. This procedure was problematic in the ASD population, where most individuals did not like the sensations of stickers being affixed to their skin or clothing. As a result, segmental data could not be collected without markers present. The COM for the whole body was therefore determined via an alternative method. The accelerations obtained from ground reaction forces were doubly integrated using the trapezoidal method (Appendix A). Assuming the position of COP will coincide with that of the COM during quiet stance (Winter, Patla, Prince, Ishac, & Gielo-Perczak, 1998), estimates for COM displacement were obtained using the methodology described in the literature (Chan, 1999). Both methods (marker and integration) yield equivalent approximations of the whole body COM and can be used interchangeably (Chan, 1999; Lafond, Duarte, & Prince, 2004; Zatsiorsky & King, 1998).

Outcome Measures: Static Balance (Quiet Stance)

Characteristics of the individual's postural sway were assessed using traditional COP analyses including the range of anteroposterior and mediolateral sway and the overall sway area for each

of the quiet stance trials. Furthermore, the distance between the COM and COP in the transverse plane, defined as the COP–COM moment arm, was also calculated. As a result, peak moment arms ($COP–COM_{max}$) in the mediolateral and anteroposterior directions and subsequent resultant moment arms were also identified and analyzed for each of the quiet stance trials.

Outcome Measures: Dynamic Balance (Gait Initiation)

The COP trajectory during gait initiation was divided into 3 phases (S1, S2, S3) by identifying 2 landmark events previously described in the literature (Hass et al., 2004). Because the S1 phase represents the purposeful uncoupling of the COP and COM and thus is the initial balance challenge, peak displacement of the COP in the mediolateral (S1_ML) and anteroposterior (S1_AP) directions were calculated and analyzed for each of the gait initiation trials.

Data Analysis

Descriptive statistics (Mean and SD) were calculated for age, height, and weight (Table 2–1). Measures of central tendency and variability were calculated for the variables of interest. An individual's data from the four experimental trials in each condition were then averaged to provide one representative datum for each dependent variable. The representative datum was then submitted for statistical analyses.

Static Balance (Quiet Stance)

The primary hypotheses were that children with ASD would posses more postural sway during quiet stance than age matched controls. Initial exploration of the data revealed heterogeneity of variance between groups. Multivariate analyses of variance (MANOVA) have been reported to lack robustness in accurately testing for overall group differences when the assumption of homoscedasticity has been violated (Finch, 2005). As a result, non parametric Mann–Whitney ***U***–tests were used to test for group differences on COP trajectories and COP–

COM interactions during quiet stance. An á–priori alpha level of 0.05 was set for all statistical tests. All statistical tests were performed using SPSS 16.0 for Windows (Chicago, Illinois).

Dynamic Balance (Gait Initiation)

The primary hypothesis was that children with ASD would possess altered anticipatory postural adjustments resulting in reduced displacements of the center of pressure during gait initiation. Similarly to quiet stance data, the dependent variables of interest were submitted for Mann–Whitney ***U***–Tests to evaluate overall group differences. An á–priori alpha level of 0.05 was set for all statistical tests. All statistical tests were performed using SPSS 16.0 for Windows (Chicago, Illinois).

Table 2-1. Descriptive statistics (means and SD) for age, height and mass.

Group		Age (years)	Height (m)	Mass (Kg)
ASD (n=13)	Mean	11.1	1.45	50.2
	SD	2.3	0.17	21.8
TD (n=13)	Mean	13.1	1.57	48.2
	SD	2.2	0.12	10.3

Table 2-2. Leiter International Performance Scale-Revised (Leiter-R) means and ranges for all participants.

	ASD (N=13) Mean (range)	Control (N=13) Mean (range)
Leiter-R- Brief IQ	80.2 (36-124)	104.9 (67-129)

(Roid & Miller, 1997)

Table 2-3. Repetitive Behavior Scales-Revised (RBS-R) means and ranges for all participants.

	ASD (N=13) Mean (range)	Control (N=13) Mean (range)
RBS-R*- overall	20.6 (11-36)	0.9 (0-8)
RBS-R subscale 1 (stereotyped behavior)	3.8 (2-6)	0.2 (0-2)
RBS-R subscale 2 (self-injurious behavior)	1.5 (0-6)	0.0
RBS-R subscale 3 (compulsive behavior)	3.3 (0-6)	0.2 (0-1)
RBS-R subscale 4 (ritualistic behavior)	4.2 (1-6)	0.2 (0-1)
RBS-R subscale 5 (insistence on sameness)	5.2 (2-10)	0.4 (0-5)
RBS-R subscale 6 (restricted behavior)	3.6 (0-20)	0.1 (0-1)

(Bodfish, Symons, & Lewis, 1999; Bodfish, Symons, Parker, & Lewis, 2000)

Table 2-4. Adaptive Behavior Assessment System (ABAS-II) means and ranges for all participants.

	ASD (N=13) Mean (range)	Control (N=13) Mean (range)
Communication	3.9 (1-9)	11.1 (7-14)
Community Use	3.7 (1-8)	11.3 (8-16)
Functional Academics	5.9 (1-15)	12.0 (5-16)
Home Living	4.3 (1-11)	11.5 (6-17)
Health and Safety	4.3 (1-13)	11.3 (3-16)
Leisure	3.7 (1-9)	11.4 (7-15)
Self-Care	3.4 (1-11)	10.8 (5-15)
Self-Direction	2.1 (1-9)	10.6 (5-16)
Social	1.5 (1-4)	11.4 (7-14)

(Harrison & Oakland, 2003)

Table 2-5. Brief Rating Inventory of Executive Functioning-Parent Form (BRIEF) means and ranges for all participants.

	ASD (N=13) Mean (range)	Control (N=13) Mean (range)
Inhibit	68.2 (42-91)	47.6 (37-68)
Shift	75.4 (59-95)	44.8 (36-70)
Emotional Control	67.3 (45-91)	43.8 (37-80)
Behavioral Regulation Index	73.2 (53-99)	44.7 (36-76)
Initiate	64.3 (46-83)	50.8 (35-76)
Working Memory	65.2 (45-86)	52.5 (38-71)
Plan/Organize	63.2 (44-90)	48.6 (35-61)
Organization of Materials	55.3 (37-71)	47.6 (33-60)
Monitor	69.3 (50-84)	46.8 (33-70)
Metacognition Index	65.7 (49-87)	48.2 (34-63)
Global Executive Composite	70.2 (55-92)	47.0 (33-66)

(Gioia, Isquith, Guy, & Kenworthy, 2000)

CHAPTER 3
LITERATURE REVIEW

Autism and Autism Spectrum Disorders (ASD)

Five childhood disorders (autistic disorder, pervasive developmental disorder not otherwise specified (PDD–NOS), Asperger's disorder, Rett's disorder and childhood disintegrative disorder) are all grouped under the pervasive developmental disorders umbrella according to The Diagnostic and Statistical Manual for Mental Disorders (DSM–IV). Three of these disorders (autistic disorder, PDD–NOS and Asperger's syndrome) are often referred to as Autism Spectrum Disorders (ASD). These disorders are lifelong neurological conditions that can affect an individual's ability to communicate, understand language, play and socially interact with others and are often accompanied by restricted, repetitive and stereotyped patterns of behavior, interests and activities. Although the classical form of autism, (autistic disorder) which typically manifests itself in developmental delays before the age of 3, can be differentiated from other forms of ASD, the terms autism and ASD are often used interchangeably.

Although ASD affect functioning of the brain, their specific causes remain unknown. It is widely believed that their etiology are multifactorial with each factor possibly leading to the expression of different phenotypes within ASD. As a result, individuals with ASD vary widely in ability and functioning. Although individuals with ASD are typically characterized by developmental delays in verbal and non–verbal communication, social relatedness and leisure and play activities, these individuals also exhibit unusual, repetitive and perseverative movements. It is estimated that 1 in every 150 individuals is diagnosed with ASD, making it more prevalent than pediatric cancer, diabetes, and AIDS combined. It is not bound by any racial or social groups but is more likely to be diagnosed in boys than girls. Approximately 67 children are diagnosed with ASD each day with an estimated health care cost of 90 billion

dollars per year. With no medical detection or cure for ASD, research aimed at providing further insight into the etiology, early detection and treatment are of the utmost importance (Autism Society of America, Retrieved December 11, 2007).

Etiology of Autism

Autism spectrum disorders are some of the most heritable neuropsychiatric syndromes with a highly variable phenotype (Sykes & Lamb, 2007).Whole–genome linkage scans have implicated nearly every chromosome, although none have been consistently identified as contributory (Sykes & Lamb, 2007). There is growing evidence to suggest that as many as 3–15 genes are associated with ASD (Pickles et al., 1995; Risch et al., 1999). Although several loci have been identified in multiple studies (2q, 7q and 17q), lack of conclusive linkage results suggests the etiology of ASD is more complex and heterogeneous than original supposed (Sykes & Lamb, 2007). Many of these genes of interest have only accounted for a small fraction of the genetic variation in the disorder and knowledge of most of the genes remains incomplete (Sykes & Lamb, 2007). Other factors such as prenatal environmental exposures and gender are thought to exacerbate the matter further by increasing the likelihood of presenting with the disorders and influencing their severity (DiCicco-Bloom et al., 2006; Sykes & Lamb, 2007).

Given that ASD is primarily a genetic disorder involving multiple genes, understanding the underlying mechanisms will likely require a collaboration of multiple disciplines. Assessment of the earliest signs of ASD has helped guide this multidisciplinary research in the areas of abnormal brain growth, development and function (DiCicco-Bloom et al., 2006). Although ASD are neurodevelopmental disorders with a clinical onset between the ages of 2 and 4 years, very few studies have investigated the anatomical development of the brain during this critical age range (Courchesne et al., 2007), This period (2–4 years) is a crucial period in development as neural wiring patterns begin to lay the foundation for the development of higher order social,

emotional and communication functions (Courchesne et al., 2007). Unfortunately, most of the anatomical studies have focused on the older child, adolescent or adult with ASD (Cody, Pelphrey, & Piven, 2002). As a result, the abnormal brain growth responsible for the dysfunction in social, emotional and communication networks remains unclear (Courchesne et al., 2007).

Movement coordination is believed to be controlled by three important structures including the motor cortex, basal ganglia and the cerebellum. Although the cerebellum is integral in the coordination, it is not a significant contributor in either sensory or motor functioning (Shumway-Cook & Woollacott, 2001). Even if the cerebellum is destroyed, sensation and motor functioning are not lost. However, the quality of movement may be drastically altered (Shumway-Cook & Woollacott, 2001). Similarly, lesions of the basal ganglia do not result in paralysis; however, problems in coordinated movements emerge. The motor cortex interacts with the cerebellum and basal ganglia in order to identity the intended movement, the plan necessary for movement and the execution of the movement (Kandel et al., 2000).

The most consistent brain abnormalities related to ASD have been observed in the cerebellum (Bauman & Kemper, 2005). Regardless of age, sex or cognitive abilities, significant decreases in the number of Purkinje cells affecting the cerebellar hemispheres have been consistently observed (Arin, Bauman, & Kemper, 1991). Though the cerebellum only constitutes 10% of the total volume of the brain, it contains over half of all its neurons. The cerebellum functions as a relay station between intention and action by adjusting motor output in the cortex and brain stem during movement (Kandel, Schwartz, & Jessell, 2000). The cerebellum compares internal feedback signals which define the intended movement and the external feedback signals which define the actual movement. When the movement is repeated,

the cerebellum is able to perform corrections and gradually reduce error and therefore plays a key role in motor learning and adaptation (Kandel et al., 2000). Damage to the cerebellum may not necessarily affect the basic elements of perception and movement, but temporospatial coordination of movement is affected. Subsequently certain cognitive functions, motor learning and posture may be impaired (Kandel et al., 2000).

In addition to the cerebellum being implicated as a site of possible dysfunction in ASD, studies of locomotion have also suggested the involvement of the basal ganglia (Hughes, 1996; Minshew, Sung, Jones, & Furman, 2004; Vilensky, Damasio, & Maurer, 1981). It has been suggested that the basal ganglia are involved in the control of movement and movement disorders such as diminished movement in Parkinson's disease and excessive movement in Huntington's disease (Kandel et al., 2000). It is believed that the basal ganglia contribute to postural changes in the mediolateral direction during gait initiation and the maintenance of locomotion, which are necessary for providing stability during these movements (Bachevalier, 1994; Bronstein, Brandt, Woollacott, & Nutt, 1996; Ebersbach et al., 1999). Thus, it should not seem too surprising, that based on the role of the cerebellum and basal ganglia in coordinating movement, that children with ASD may posses altered postural control during both static and dynamic challenges.

Much of the morphological findings observed in mature individuals with ASD are in stark contrast to those observed in the relatively few studies that have investigated the developing brain in very young children with ASD (Courchesne et al., 2007). It is however important to note that much of the developing brain literature is based on single case studies and studies investigating larger samples are still necessary. Briefly, brain volumes have been reported to be 5–12% greater in very young children with ASD (2–4 years) relative to controls, where only an

average of 1% increase has been observed in adolescents or adults with ASD (Courchesne et al., 2001; Hazlett et al., 2005; Redcay & Courchesne, 2005; Sparks et al., 2002). Similarly, the amygdala has been observed to be larger in very young children with ASD relative to controls (Sparks et al., 2002), whereas decreased neuron numbers have been observed in the amygdalas of adults with ASD (Schumann & Amaral, 2006). Very young children with ASD have been reported to have average minicolumn size relative to controls (Courchesne et al., 2007). Conversely, older individuals with ASD present with reduced minicolumn size (Buxhoeveden et al., 2006). And finally, there does not appear to be cerebellar Purkinje cell loss in very young children with ASD relative to controls (Bailey et al., 1998), whereas a consistent loss has been observed in older individuals with ASD (Bailey et al., 1998; Kemper & Bauman, 1998; Lee et al., 2002; Vargas, Nascimbene, Krishnan, Zimmerman, & Pardo, 2005). Preliminary studies in very young children with ASD suggest the brain displays areas of overgrowth early in development but then shows degeneration, loss and size reduction in these very same areas later in life (Courchesne et al., 2007). These findings have resulted in a new hypothesis for brain growth pathology in ASD. This new hypothesis postulates two distinct phases, one of overgrowth during infancy and then one of arrest of growth during early childhood with a possible third phase of degeneration later in life (Courchesne et al., 2007). It is hypothesized that these areas of early abnormal overgrowth may display abnormal function later in development. However, it remains unclear whether the neural systems that fail to provide essential language, social, emotional and attention skills in children with ASD are also the same ones affected by overgrowth (Courchesne et al., 2007).

The three core symptoms associated with ASD (deficits in communication, abnormal social interactions and restricted and/or repetitive behaviors) are believed to involve widespread

neural systems. It appears that neural structures subserving perception of facial expression, joint attention, empathy and social cognition in ASD are compromised, as indicated by reduced neural activity in regions that govern these functions (Baron-Cohen et al., 1999; Critchley et al., 2000; DiCicco-Bloom et al., 2006; Pelphrey, Morris, & McCarthy, 2005; Pierce, Muller, Ambrose, Allen, & Courchesne, 2001). However, given that some skills such as basic perceptual skills and overall intelligence may be unaffected, it is thought that not all brain systems are affected equally (DiCicco-Bloom et al., 2006). It is theorized that excess neuron numbers resulting form the early overgrowth during infancy may profoundly disrupt circuit formation and thereby hinder the emergence of higher order behavioral skills (Courchesne et al., 2007). As a result, mapping of the networks may appear to be normal, however the long distance interactions of the networks would be weakened and possible "noisy".

Development of Postural Control

The development of postural control is traditionally defined as a series of predictable motor behaviors: crawling, sitting, creeping, pull–to–stand, independent stance and walking. These behaviors are generally referred to as motor milestones (Gesell, 1946; McGraw, 1931). Postural control theories describe these milestone behaviors and relate them to underlying neural structures (Shumway-Cook & Woollacott, 2001). Classic theories define postural control in terms of the emergence and integration of reflexes (Shaltenbrand, 1928). Reflex–hierarchy theory suggests that the inhibition and integration of reflexes is indicative of maturity within the lower levels of the CNS, allowing for more functional voluntary movements (Shumway-Cook & Woollacott, 2001; Woollacott & Shumway-Cook, 1990). Recent theories however, have alternatively suggested that the postural control system is a more complex interaction between musculoskeletal and neural systems, with control of movement being determined by both task and environment (Shumway-Cook & Woollacott, 2001).

The progression to bipedal stance is one of the major milestones of motor development and typically occurs during the first year of life, and continues to become refined throughout the course of development (Assaiante & Amblard, 1995). Given that the development of postural control is a complex process, involving much more than maturation of reflexes; new behaviors and skills are believed to emerge from an interaction between the maturing neural and musculoskeletal systems and the surrounding environment. Important milestones in the development of the neural and musculoskeletal systems are: 1) the development of muscle strength and changes relative to the different body segments, 2) the development of neuromuscular response synergies used to maintain balance, 3) the maturation of individual sensory systems, 4) the formation of sensory strategies to organize redundant inputs, 5) the development of internal representation important in the mapping of perception to action, and 6) the development of anticipatory postural adjustments (APA) mechanisms to allow modification in postural control (Shumway-Cook & Woollacott, 2001).

Although children appear to be smaller version of adults, they are proportionally different. Due to the relative size of the head compared to the body, the COM of a child is located roughly around the T12 vertebrae rather than the L5–S1 location seen in adults. The shifted location of the COM causes children to be "top heavy" and as a result, makes the task of static balance more challenging (Zeller, 1964). Under normal sensory conditions, children produce larger, more variable and oscillatory postural responses than adults (Forssberg & Nashner, 1982). As such, postural control has been found to be less efficient in children and only becomes essentially adult–like between the ages of 7 and 10, where amplitude, frequency and variability of sway begin to decrease (Figura, Cama, Capranica, Guidetti, & Pulejo, 1991; Hatzitaki, Zisi, Kollias, &

Kioumourtzoglou, 2002; Odenrick & Sandstedt, 1984; Rival, Ceyte, & Olivier, 2005; Shumway-Cook & Woollacott, 2001).

There is an extensive amount of literature in the area of postural control in children and according to Nougier (1998), the three main observations that emerge from these investigations are: a) children are less efficient at controlling static or dynamic balance, b) the contribution of vision in postural control appears to be less important in children than in adults and c) the development of balance and locomotion is non–monotonic, that is, there is a transition phase around 7 to 8 years in the development of the sensorymotor processes (Hay, 1984). Children 4–6 years have generally slower postural responses and more variable than those of children aged 7–10 yrs. Further, changes in body segment composition as a result of maturation are thought to contribute to the variability (Kugler, Kelso, & Turvey, 1982).

Young children under the age of 7 years have been shown to have difficulty controlling posture when somatosensory and visual inputs were altered (Forssberg & Nashner, 1982). However, children 6 years in age have been shown to maintain postural stability similarly to children 10 years in age (Nougier, Bard, Fleury, & Teasdale, 1998). In contrast, children 7 to 8 years old display increased sway due to an inability to minimize the magnitude of the COP displacements (Hay & Redon, 2001; Riach & Starkes, 1994) and begin to adopt more adult–like balance control strategies (Kirshenbaum, Riach, & Starkes, 2001). These results suggest that the ability to reorganize basic postural control under altered somatosensory information conditions is already present at the age of 6 years. These observations related to the use of sensory information during postural control are believed to support the existence of a critical phase, particularly around the age of 7 to 8 years, where there is a re–weighting of the different sensory systems involved in controlling balance.

Motor Deficits and Autism

Restricted, repetitive, and stereotyped patterns of behavior, interests and activities are among the core features of autistic disorder. Of these, stereotyped body movements (clapping, finger flicking, rocking, dipping, and swaying) and abnormalities in posture (walking on tiptoe, odd hand movements and body postures) are of particular interest However, the specific pattern and source of these motor deficits remain unclear (Noterdaeme et al., 2002). This is likely due to feelings that motor deficits may be a less important side effect rather than a concomitant symptom (Noterdaeme et al., 2002). Leary & Hill (1996) in their review, however, suggest that motor deficits likely impair the development of sufficient communicative and interactive skills. Indeed, motor disturbances have been reported to affect daily activities (playing, writing, drawing, games) and social integration (Kalverboer, 1993). In addition, children with fine motor and coordination disturbances have been reported to a have higher risk of developing learning and behavioral problems (Hadders-Algra & Touwen, 1992; Losse et al., 1991). In children with ASD, motor deficits have been reported to further tax development; resulting in difficulty with the full acquisition of functional skills related to manipulation, mobility and play (Noterdaeme et al., 2002). Therefore, it appears that the occurrence of neuromotor deficits in ASD may be a partial indication of the biological factors in the etiology of the disorder (Jones & Prior, 1985; Leary & Hill, 1996).

Static Balance (Quiet Stance) and Autism

The vestibular, somatosensory and visual systems are the afferent inputs involved in maintaining an upright posture. A deficit in any one of these systems or in the integration of information provided from these systems could affect the ability to maintain balance. Limited research has focused on the postural system in individuals with ASD during quiet stance. Postural patterns employed by children with autistic disorder over the age of 6 years have been

observed to differ from typically developing children, mentally retarded children and adults with vestibular disorders. Children with autistic disorder were observed to have a "paradoxical postural stress response" with equal or better stability under seemingly more difficult conditions, had an unusual distribution of body weight over their heels and toes, and tended to employ a more primary somatosensory, postural control system (even when visual cues were available). In addition, these older children with autistic disorder exhibited more variable and less stable postural control, particularly in the mediolateral direction when compared to typically developing children (ages 4 to 11 years) (Kohen-Raz et al., 1992). Both children and adults with ASD have been observed to have greater impairments in postural stability during times of altered sensory inputs when compared to individuals with typical neuromotor development. However, when afferent inputs were not modified, there appears to be conflicting results in the literature. Some research has detected differences in postural sway between the groups (Gepner et al., 1995; Kohen-Raz et al., 1992; Minshew et al., 2004), while one study detected no such difference (Molloy et al., 2003). In addition, when investigating age effects, results reveal a delayed development of the postural system in subjects with ASD, which appears to improve at the age of 12 years, however never reaches adult levels (Kohen-Raz et al., 1992; Minshew et al., 2004). These authors have, therefore, highlighted an involvement of the neural circuitry beyond the neural systems for social behavior, communication and reasoning. However, despite this information, there is still is a paucity of research investigating a wider range of functional tasks challenging both the static and dynamic postural control of this population.

Postural instability has often been analyzed through the assessment of COP movements only. It has been suggested, however, that there are inherent limitations to relying solely on traditional COP measures to quantify postural stability. During upright stance, with side by side

foot placement, the body has often been simplified to a 2 dimensional inverted pendulum with movement confined to the ankle joint (Winter et al., 1998). The basic assumption when using COP measures to evaluate postural control is that the shifting of weight and muscular control (measured by changes in COP) provides an estimate of body position or sway of the COM. Research suggest however, this may not be a valid assumption under all conditions (Panzer, Bandinelli, & Hallett, 1995). Although the COP is known to be controlled by ankle plantarflexors and dorsiflexors in the anteroposterior direction and hip abductors and adductors in the mediolateral direction, this simplification to a simple inverted pendulum has provided further insight into the dynamic relationship between the COP and COM. In order to keep the COM in a stable position, the COP must oscillate about the COM and as such, must have a larger dynamic range by comparison (Winter et al., 1998). Differences detected by the central nervous system (CNS) between the COP and the COM must then be corrected. As result, the difference between these two measures is often referred to as an "error" signal within the postural control system where the COM is the variable being controlled by the COP for the purposes of meeting the mechanical criterion for stability (Winter et al., 1998).

The mechanical criterion for stability during quiet stance is maintenance of the COM within the base of support (BOS). This criterion is generally believed to be achieved through the CNS's manipulation of the COP (Martin et al., 2002; Riley, Mann, & Hodge, 1990). Changes in the COP reflect forces that must be produced to return the COM to a more stable position (Martin et al., 2002). Consequently, an increase in COP sway has traditionally been believed to indicate increased COM sway. However, it has been suggested that an increase in COP sway may also indicate the adoption of a different postural control strategy (Panzer et al., 1995). According to these authors, an inverted pendulum model may be inadequate in describing

multilink systems such as the human body. Results from their investigation suggest that increases in COP sway do not have to coincide with increases in COM sway. During quiet stance, significant movements were observed in participants' individual body segments, indicating that not all movements were confined to the ankle. As a result, segmental accelerations associated with these movements were reflected in the COP as increased sway. However, no simultaneous changes in COM sway were detected. Therefore, the authors concluded that even with increased COP sway, the sum of the segmental accelerations successfully achieved their goal of stabilizing the COM. As a result, the observed increased sway in the COP was likely representative of an alternative strategy rather than instability. In addition, even though there may be some underlying dysfunction in postural control, it still may be possible for the COP movements to successfully stabilize the COM movements (Corriveau, Hebert, Prince, & Raiche, 2001).

Given that the COP and COM may yield different information and that measurements based solely on COP sway may be inadequate under certain conditions, researchers have become increasingly interested in the combined "error" measure of COP and COM separation (Corriveau et al., 2001; Corriveau, Hebert, Raiche, Dubois, & Prince, 2004; Hass, Waddell, Fleming, Juncos, & Gregor, 2005). This combined measure, defined as the distance at any given time between the COP and COM, is believed to provide better insight into postural control and efficacy than either measure taken separately (Winter et al., 1998). During quiet stance, the COP and COM should more or less coincide (Chang & Krebs, 1999; Jian, Winter, Ishac, & Gilchrist, 1993) in the transverse plane. This condition represents the individual's most stable standing position (Martin et al., 2002). When the body deviates from this position, the distance between the COP and COM increases and introduces an instability requiring active control by the

individual to return the COM to a more stable position within the BOS. The greater the COP–COM distance, the more inherently unstable the individual is and the more active postural control is needed. In taking both the COP and COM into account, this combined COP–COM distance has successfully identified small changes in postural control in other populations (stroke, elderly, diabetes with distal neuropathy) with know instability (Corriveau et al., 2001; Corriveau et al., 2004). Therefore, by combining the COP and COM measures and widening the range of task complexity to those including transition, we may be able to emphasize and clarify some of the more subtle and conflicting results currently reported in the literature focusing on postural control in ASD.

Dynamic Balance (Gait Initiation)

An intact postural control system is necessary to tolerate larger separations that occur between the COP and COM when movements transition from static conditions to ones that are more dynamic in nature. Gait initiation is a prerequisite to locomotion and has received considerable attention in the literature as a screening tool for dynamic balance control in elders and individuals with Parkinson's disease (Chang & Krebs, 1999; Hass et al., 2005; Martin et al., 2002). Gait initiation is defined as the transition phase between static balance in an upright position and the start of steady state walking (Burleigh, Horak, & Malouin, 1994; Jian et al., 1993; Mann, Hagy, White, & Liddell, 1979) and can therefore be considered a dynamic balance skill. It has been suggested that individuals with less effective postural control systems are inclined to reduce the separation between the COP and COM, particularly during transition movements, in an effort to reduce the amount of active control needed (Martin et al., 2002).

In order for gait to be initiated, there must be a separation of COP and COM (Jian et al., 1993). This separation represents a natural but deliberate destabilization during which momentum is generated and must be combined with a simultaneous maintenance of balance

(Breniere, Do, & Bouisset, 1987). These requirements however, are in conflict because momentum generation requires moving the COM outside of the BOS thereby creating instability (Polcyn, Lipsitz, Kerrigan, & Collins, 1998). The interaction between the COP and the COM therefore provides insight into how postural and intentional movement components are coordinated during locomotion (Chang & Krebs, 1999). As mentioned, gait initiation has been shown to be a sensitive indicator of balance dysfunction (Chang & Krebs, 1999) and is believed to be ideal for identifying changes in the postural and locomotor systems, including those less apparent deficits not typically identified with less sensitive measures taken during quiet stance and steady state walking (Hass et al., 2004).

The path traveled by the COP during gait initiation also discriminates between healthy adults, elders and elders with disability (Halliday, Winter, Frank, Patla, & Prince, 1998; Hass et al., 2004). The COP path can be divided into three phases (S1, S2, S3), with S1 being the initial phase beginning with the start command and ending with the most posterolateral position of the COP towards the initial swing leg. According to inverted pendulum theory, this combined displacement in the posterior and lateral directions (defined as the COP shift mechanism) will propel the COM in the forward and lateral directions respectively (Hass et al., 2004; Winter, 1995). S1 represents the purposeful uncoupling of the COP and COM and decreased COP displacements in either direction may be indicative of instability (Martin et al., 2002; Zettel, McIlroy, & Maki, 2002) or perhaps the use of an alternative, possibly less efficient strategy for generating momentum (Hass et al., 2004; Polcyn et al., 1998). S2 begins with the shifting of the COP towards the initial stance limb and ending with the point at which the COP begins to move anteriorly under the initial stance foot. During this phase, the COM continues to accelerate in the forward direction while the body weight and as a result COP, are shifted towards the stance limb.

Transferring the COP laterally allows for loading of the stance limb while simultaneously unloading of the swing limb, a process critical for initiating a step (Martin et al., 2002). Finally, S3 begins with the anterior movement of the COP under the stance foot and ends with toe off of the stance foot.

The magnitudes of temporospatial parameters such as COP displacement and average velocity reported during the different phases of gait initiation have, as a result, defined a continuum between stability and instability. With increasing age and disability, values are reported to be smaller, slower and more variable in both the anteroposterior and mediolateral directions (Hass et al., 2004; Martin et al., 2002). Even with deficits in speed and forcefulness, older individuals are still able control gait with similar muscle activation patterns and kinematic and kinetic patterns when compared to healthy, young individuals. These results suggest that quality or efficiency of movement is being sacrificed for stability and safety (Halliday et al., 1998). Although there appears to be a deterioration of the COP shift mechanism with increasing age and disorder, it seems this deterioration is not the only means by which insight into the control of dynamic balance can be achieved. There has also been a concurrent interest in research focusing of the emergence of this COP shift mechanism or basic anticipatory behavior prior to gait initiation.

The development of internal anticipatory postural adjustments (APA) related to gait initiation has been investigated in young children. Although APA are generally believed to safeguard against instability resulting from the impending movement, they may also serve as a necessary condition required for the intended movement (Ledebt, Bril, & Breniere, 1998). During gait initiation, uncoupling of the COP and COM is necessary to create propulsive forces required to reach intended gait speed (Breniere et al., 1987). Shifting of the COP prior to

movement is therefore necessary to accelerate the COM and ultimately generate the velocity of the subsequent steps. Understanding how these APA emerge and how they are refined during development may provide further insight into the maturation of neuromuscular systems in children with ASD.

The development of APA may be dependent on neural maturation, mastering of the task, or a combination of both (Ledebt et al., 1998). When compared to adults, observed differences in anticipatory behavior in children have been reported to be the result of immature neural functioning (peripheral nerve conduction and central processing) (Hirschfeld & Forssberg, 1994). Alternatively, having observed anticipatory behavior in children as young as 4 years of age, researchers suggest children will develop a feedforward control of posture as soon as they are able to sufficiently maintain posture through feedback control (Haas & Diener, 1988). Therefore, although general anticipatory behaviors appear in a less mature form at a very early age, these APA will then continue to be refined through experience. As a result Haas & Diener (1988) believe APA to be more task–dependent rather than age–dependent. This ability to anticipate upcoming instabilities and/or adjust current movements to facilitate future actions is an element essential to motor control (Ledebt et al., 1998). For this reason, the COP shift prior to gait initiation in children is of considerable interest, because it provides information on how feedforward control of movement is developed.

The COP shift mechanism prior to gait initiation has been reported in children as young as 4 years of age; however when compared to adults, appears to be less consistent and less refined (Ledebt et al., 1998; Malouin & Richards, 2000; Stackhouse et al., 2007). Although Malouin & Richards (2000) reported the presence of a COP shift in typically developing children 4–6 years, the magnitudes of the displacements were smaller in the posterior direction and larger in the

lateral direction when compared to adults (50–61 years). Ledebt et al. (1998) similarly observed anticipatory shifting of the COP prior to gait initiation in children 2–8 years. These authors reported an increase in shift magnitude with increasing age, suggesting the youngest participants (2.5 years) were the least effective at generating forward momentum. Anticipatory behavior prior to gait initiation has also been reported in children with cerebral palsy (CP) (Stackhouse et al., 2007). These authors reported that although the APA of children with CP were similar to those of typically developing children, children with CP appeared to demonstrate alternative strategies for developing forward momentum. Due to a decreased lateral shift of the COP towards the stance limb, Stackhouse et al. (2007) suggested there was an increased dependency on the stance limb to create the posterior shift of the COP necessary to generate forward momentum.

Research suggests the development of APA prior to gait initiation follows a progression of increased muscle activity and posterior displacement of the COP with increasing age, resulting in increased gait velocity (Stackhouse et al., 2007). An inability to fully develop or refine anticipatory movements associated with gait initiation may as a result, be indicative of delay or dysfunction. Ledebt et al. (1998) have suggested that the immature anticipatory behavior observed in very young children (2.5 years) during gait initiation may be the result of a less stable initial posture and/or a lack of cognitive control of movement. According to these authors, children may indeed have the ability to produce anticipatory movements at the muscular level, however, any effects of these movements on a global scale, may be obscured by postural instabilities. Further, as children acquire knowledge of new skills, they must develop cognitive structures and representations that allow them to negotiate new environments while attempting to

execute purposeful action. Therefore, in order for APA associated with gait initiation to emerge, young children must have a representation of their forthcoming velocity (Ledebt et al., 1998).

Evaluating the APA or COP shift mechanism during gait initiation may therefore be used as a means of quantifying motor coordination (Stackhouse et al., 2007). Analysis of the destabilizing movements may provide new insight into the dynamic control of posture in children with ASD. Further, by simultaneously evaluating static postural control, the results of this investigation have the potential to delineate subtle movement disturbances over a spectrum of tasks with increasing demands on postural control. The use of measures aimed at evaluating the relationship between the COP and the COM during both static and dynamic tasks may provide much needed information for interventions aimed and improving balance and subsequent movement coordination in children diagnosed with ASD.

CHAPTER 4
RESULTS

Static Balance (Quiet Stance)

Significant differences between groups were identified for all three traditional COP measures of postural control during quiet stance (Figure 4-1). Children with ASD produced significantly greater (>%400 greater, $\boldsymbol{U}$=48, N_{ASD}=13, N_{TYP}=13, $\boldsymbol{p}$=0.032) normalized mediolateral sway (COP_{ML}) than their age–matched controls. Similarly, the magnitude of the anteroposterior sway (COP_{AP}) was >100% greater in the ASD group ($\boldsymbol{U}$=18, N_{ASD}=13, N_{TYP}=13, $\boldsymbol{p}$<0.001). Consequently, sway area (COP_{SWAY}) was also significantly greater in the group with ASD ($\boldsymbol{U}$=25.5, N_{ASD}=13, N_{TYP}=13, $\boldsymbol{p}$=0.010, one–tailed). Furthermore, children with ASD had significantly greater variability from trial to trial when compared to typically developing children in the anteroposterior direction ($\boldsymbol{t}$=-2.83, df=23, $\boldsymbol{p}$=0.007) and sway area ($\boldsymbol{t}$=-1.89, df=23, $\boldsymbol{p}$=0.043). However, only a trend toward significance was detected for the mediolateral direction ($\boldsymbol{t}$=-1.63, df=23, $\boldsymbol{p}$=0.066) (Figure 4-2).

Significant differences between groups were also identified in two of the three COP–COM_{max} moment arms during quiet stance (Figure 4-3). The max separation between the COP and COM was significantly greater for the group with ASD in the anteroposterior (COP–$COM_{max–AP}$, > 90% greater, $\boldsymbol{U}$=37, N_{ASD}=13, N_{TYP}=13, $\boldsymbol{p}$=0.007) direction as well as the resultant (COP–$COM_{max–R,}$ >100% greater, $\boldsymbol{U}$=47.5, N_{ASD}=13, N_{TYP}=13, $\boldsymbol{p}$=0.029). No significant difference between groups was observed in the mediolateral direction (COP–$COM_{max–ML}$, $\boldsymbol{U}$=58.5, N_{ASD}=13, N_{TYP}=13, $\boldsymbol{p}$=0.093), in spite of the >100 % greater separation detected in children with ASD. Independent sample $\boldsymbol{t}$-tests also revealed that children with ASD had significantly greater variability from trial to trial when compared to typically developing children

in the mediolateral (***t***=-2.12, df=24, ***p***=0.028), anteroposterior (***t***=-3.03, df=24, ***p***=0.005) and resultant directions (***t***=-2.24, df=24, ***p***=0.023) (Figure 4-4).

Dynamic Balance (Gait Initiation)

While no differences were observed in the posterior direction (S1_AP, ***U***=30, N_{ASD}=13, N_{TYP}=13, ***p***=0.447), children diagnosed with ASD displaced their COP significantly less towards the swing leg (mediolateral direction) during gait initiation (S1_ML, >40 % less, ***U***=75, N_{ASD}=13, N_{TYP}=13, ***p***=0.004) (Figure 4-5). Children with ASD performed with significantly greater variability from trial to trial when compared to typically developing children in the anteroposterior direction only (***t***=-2.22, df=23, ***p***=0.018) (Figure 4-6).

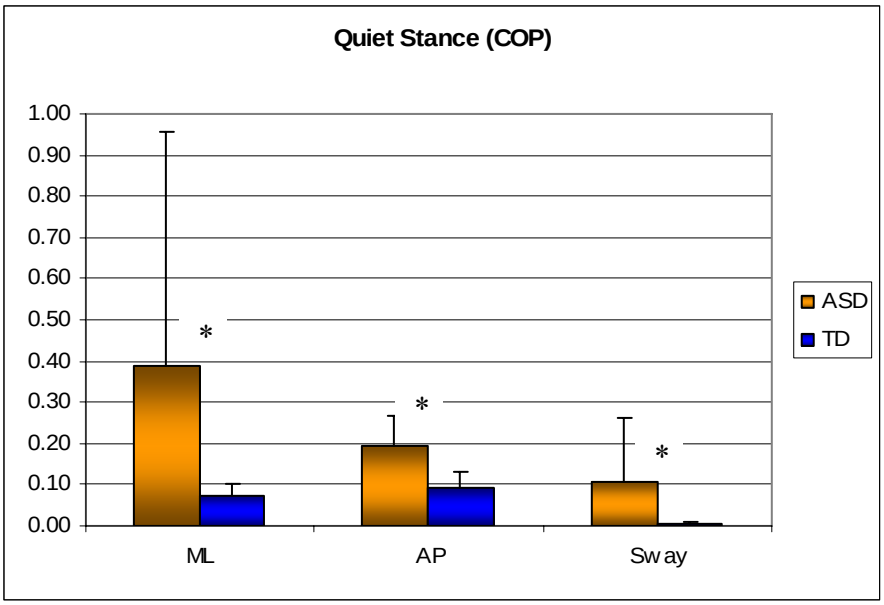
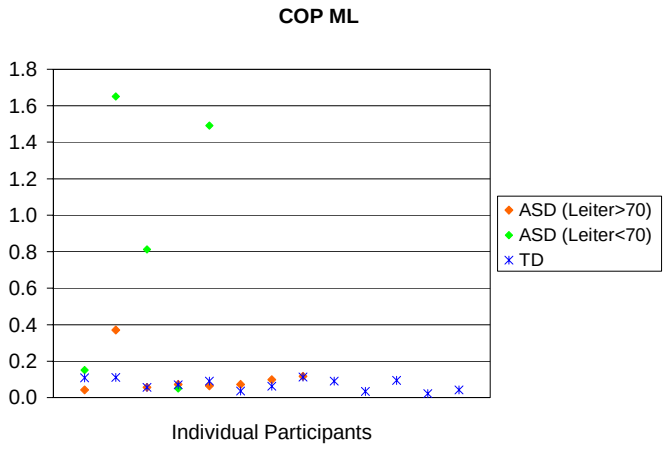


Figure 4-1. Means and SD for normalized COP measures (COP_{ML}, COP_{AP}, and COP_{SWAY}) during quiet stance (* p<0.05).

A



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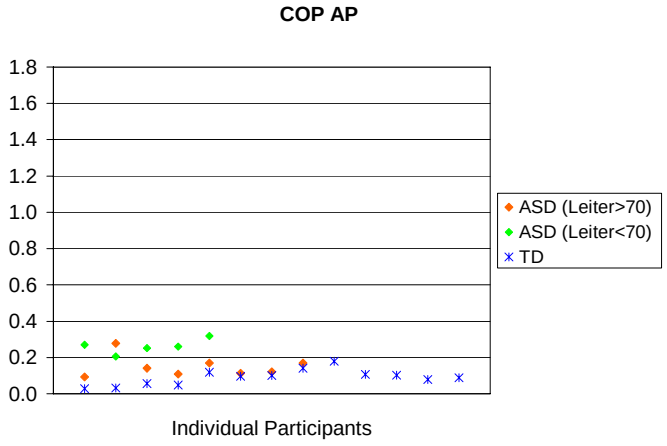

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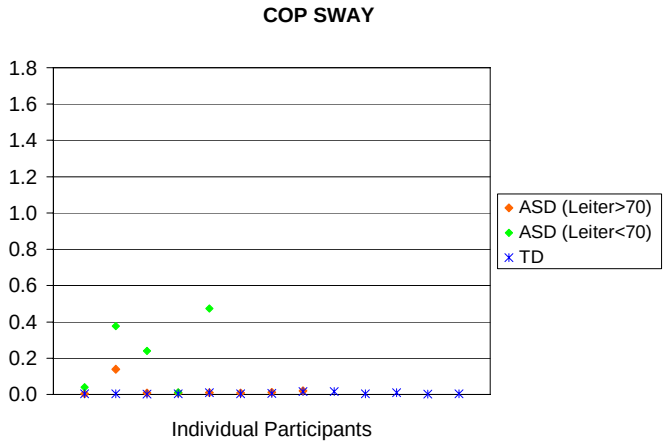


Figure 4-2. Scatter of normalized mean COP measures during quiet stance for each participant. A) COP movement in mediolateral direction. B) COP movement in anteroposterior direction. C) COP sway area.

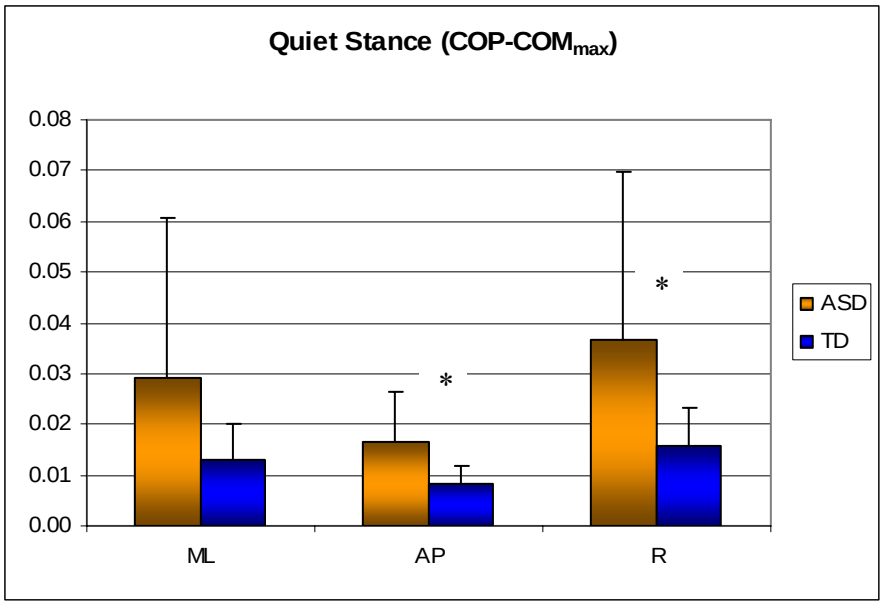
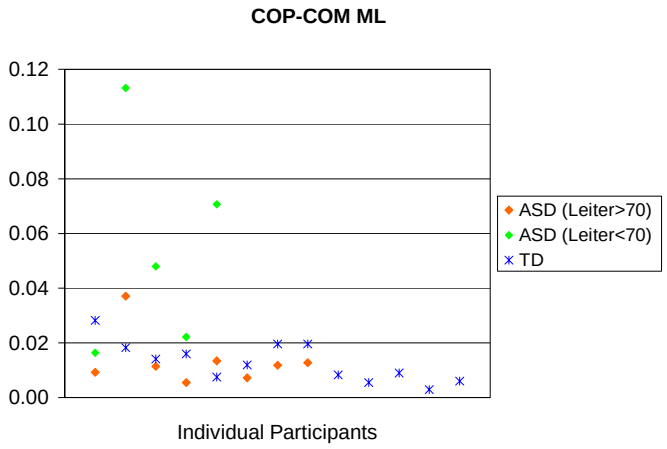


Figure 4-3. Means and SD for normalized peak COP–COM moment arms (COP–COMmax–ML, COP–COMmax–AP and COP–COMmax–R) during quiet stance (* $p<0.05$).

A



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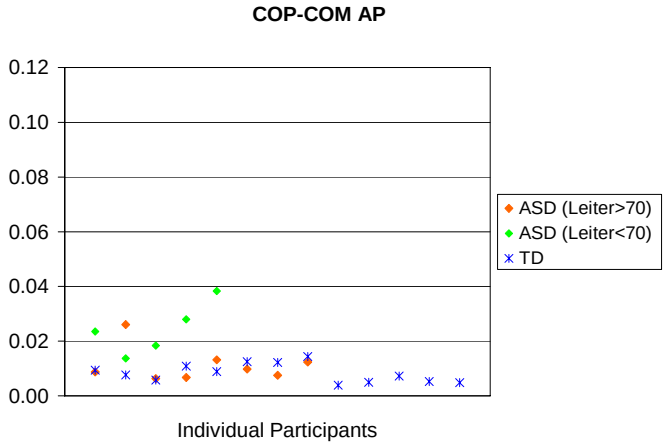

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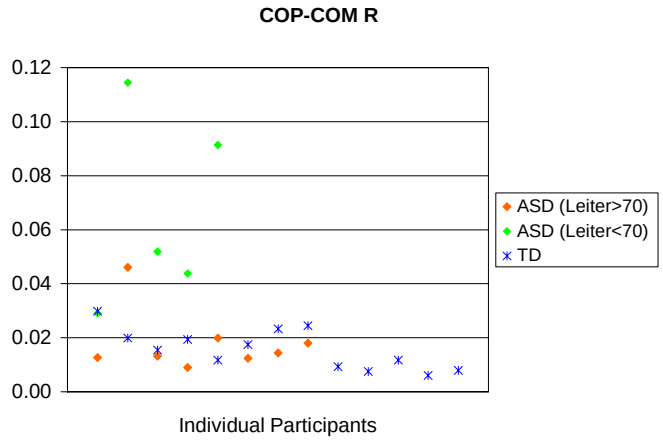


Figure 4-4. Scatter of normalized mean, peak COP–COM moment arms during quiet stance for each participant. A) COP–COM moment arm in mediolateral direction. B) COP–COM moment arm in anteroposterior direction. C) COP–COM resultant moment arm.

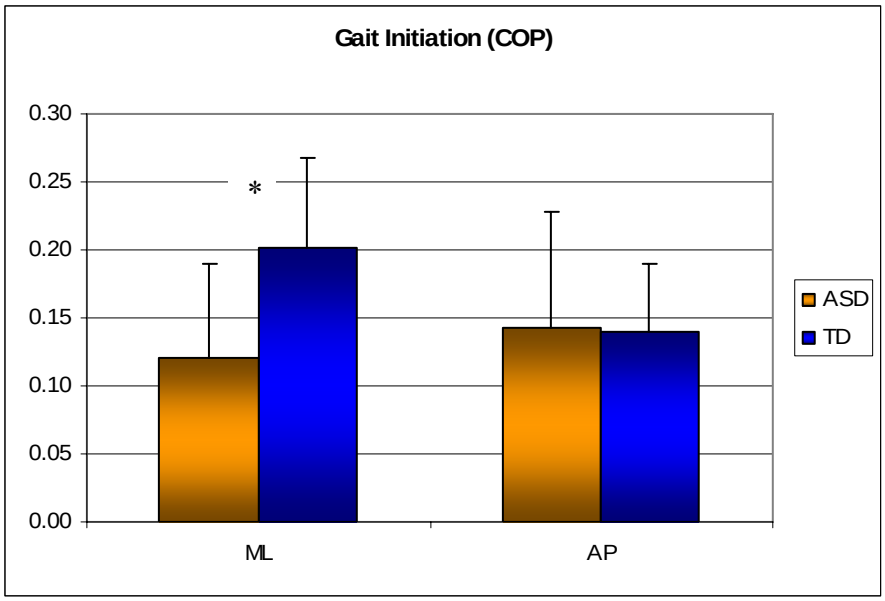
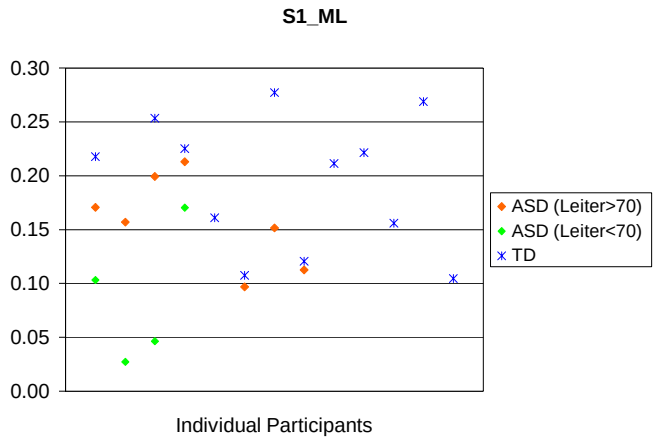


Figure 4-5. Means and SD for normalized COP displacements (S1_ML and S1_AP) during gait initiation for ASD (* p<0.05).

A



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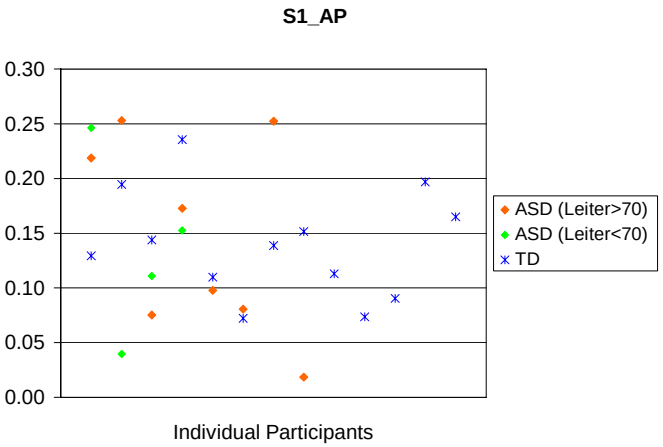

Figure 4-6. Scatter of normalized mean COP displacements during gait initiation. A) COP displacement in mediolateral direction. B) COP displacement in anteroposterior direction.

CHAPTER 5
DISCUSSION

An effective postural control system is a necessary foundation for individuals to acquire skills inherent to functional independence. Initially, there must be an ability to maintain equilibrium during static conditions where the COM remains within BOS such as during quiet stance; however, that ability must be further developed to include stability during dynamic conditions where the COM moves away from the BOS, such as during gait initiation. The current investigation has highlighted systematic postural instabilities in children with ASD using functional tasks representative of two different categories of postural challenges. Our hypothesis that children with ASD would have increased postural sway, indicated by larger traditional COP measures and greater separation of the COP and COM was supported by the data. Children with ASD exhibited significantly greater sway in the mediolateral and anteroposterior directions and subsequent sway area. Further, larger peak anteroposterior COP–COM moment arms and resultant COP–COM moment arms were observed in children with ASD. However, no differences were detected between the groups for the peak mediolateral separation. Furthermore, children with ASD shifted their COP significantly less in the mediolateral direction but not the anteroposterior direction during gait initiation. Thus, it appears that the sequella of ASD includes a retarded development or deterioration of postural control abilities during quiet stance.

Static Balance (Quiet Stance)

Using traditional measures of postural sway (COP) and a measure believed to provide further insight into postural efficacy (COP–COM moment arm), we were able to identify differences in postural stability between children with ASD and typically developing children during quiet stance. Although the outcome measures of COP sway and COP–COM moment arms were normalized to stance width in the mediolateral direction and foot length in the

anteroposterior direction for statistical analysis in the present investigation, the absolute values for these measures appear to be consistent with values reported elsewhere for various young adolescent populations. As a result, our findings appear to support prevailing reports that children with ASD have increased postural instability under conditions where sensory information has not been modified or removed (Gepner et al., 1995; Kohen-Raz et al., 1992; Minshew et al., 2004).

Sway area is a commonly reported outcome measure in the postural stability literature. It is a useful measure because it combines the postural sway in both the anteroposterior and mediolateral directions. In the present investigation, mean sway areas were 35.54 ± 49.70 cm^2 for children with ASD and 2.46 ± 1.80 cm^2 for TD children. These values are similar to those reported in children with postural deficits (2.74 to 10.1cm^2) and TD children (1.9 to 8.2 cm^2) (Bhattacharya, Shukla, Bornschein, Dietrich, & Keith, 1990; Geuze, 2003; Nault et al., 2002). Furthermore, median sway areas in the present investigation were 4.88 cm^2 for children with ASD and 1.83 cm^2 for TD children. These values are also similar to those reported previously in children with ASD (4.7 cm^2) and TD children (3.30 cm^2). When combining the findings of the current cross–sectional investigation with those of previously observed in ASD, it appears postural sway, as indicated by median sway area, decreases as age increases for TD children, but remains relatively unchanged for children with ASD. Although the currently investigation was not a longitudinal study, these observations appear to lend support to previous finding that the development of the postural system in children with ASD is delayed, and may never reach adult levels (Kohen-Raz et al., 1992; Minshew et al., 2004). Furthermore, in the current investigation, postural sway was observed to be over 400% greater in the mediolateral direction compared to a

100% increase in the anteroposterior direction for children with ASD, supporting the "directionally inconsistent and sporadic lateral sway" observed by Kohen–Raz et al. (1992).

The combined measure of COP–COM moment arm is believed to provide better insight into postural control and efficacy than either measure taken separately (Winter et al., 1998). The greater the COP–COM distance, the more inherently unstable the individual is and the more active postural control is needed. Children with ASD had larger peak COP–COM moment arms in the anteroposterior and resultant directions (0.31 ± 0.17 cm, and 0.72 ± 0.61 cm respectively) when compared to TD children (0.16 ± 0.06 cm, and 0.27 ± 0.12 cm respectively). COP–COM moment arms in TD children and children with scoliosis have been reported to range from 0.06 to 0.09 cm in the mediolateral direction, 0.08 to 0.10 cm in the anteroposterior direction and 0.1 to 0.13 cm in the resultant direction (Nault et al., 2002). Furthermore, COP–COM peak moment arms of 0.98cm have been reported for healthy adults 21–78 years of age (Panzer et al., 1995) during quiet stance. Peak COP–COM moment arms observed in the current investigation appear to be consistent with those previously published in various populations. Therefore, increased peak COP–COM moment arms observed in the current investigation further suggest postural instability in children with ASD under static conditions, particularly in the anteroposterior and resultant directions. Although no differences between groups were detected for peak COP–COM moment arms in the mediolateral direction, children with ASD were observed to have over 100% larger moment arms in this direction when compared to the TD children. Given the significant difference in COP sway but a non significant difference in peak COP–COM moment arms during quiet stance, it is speculated that the COM and COP may move together more consistently in the mediolateral direction in children with ASD.

Dynamic Balance (Gait Initiation)

The hypothesis that children with ASD would have difficulty uncoupling the COP and COM during gait initiation, indicated by decreased COP displacements was only partially supported by the data. The ability to uncouple the COP and COM is essential for initiating gait; however it simultaneously requires active postural control to counteract the instability created by the uncoupling (Hass et al., 2005). This active uncoupling creates momentum in both the anterior and stance side directions. Decreased COP displacements in either direction may be indicative of instability (Martin et al., 2002; Zettel et al., 2002) or the perhaps the use of an alternative, possibly less efficient strategy for generating momentum (Hass et al., 2004; Polcyn et al., 1998). S1_AP distances have been reported to range between 2.14 and 4.70 cm for young adults, 3.54–3.69 cm for older adults and 2.02–2.07 cm for individuals with Parkinson's disease (Halliday et al., 1998; Hass et al., 2004; Martin et al., 2002). Although absolute displacements in the posterior direction (S1_AP) did not differ significantly between TD children (2.77± 1.00 cm) and children with ASD (2.75 ± 1.50 cm), magnitudes for S1_AP in the present study appear to be similar to those reported elsewhere for various populations for children with ASD. The posterior shift mechanism creates an increased moment arm, allowing the ground reaction forces to accelerate the COM anteriorly. The non–significant results between groups for S1_AP indicate that the COP shift mechanism for generating forward momentum appears to be intact in children with ASD.

Stance side momentum is generated when the COP moves laterally towards the swing limb. This mediolateral momentum is believed to contribute to lateral stability during gait initiation. Stackhouse et al. (2007) observed decreased S1_ML distances in children with hemiplegic CP. According to these authors, children with hemiplegic CP had a tendency not to load their affected limb, resulting in decreased lateral displacements. Stackhouse et al. (2007)

further suggest this decreased lateral displacement characterized an alternative method for creating momentum. According to these authors, there was an increased reliance on the initial stance limb to create the posterior COP shift necessary for generating forward momentum. In the current investigation, S1_ML values were observed to be significantly smaller (2.18 ± 1.50 cm) for children with ASD when compared to typically developing children (3.72 ± 1.38cm). Decreased S1_ML distances have been observed in older adults with disability and those transitioning to frailty and have been reported to be the result of decreased hip muscle functions (Hass et al., 2004; Hass et al., 2005; Martin et al., 2002). Reported S1_ML distances have ranged between 3.63 and 4.38 cm for young adults, 2.19–4.12 cm for older adults and 2.02–2.30 cm for individuals with Parkinson's disease (Halliday et al., 1998; Hass et al., 2004; Martin et al., 2002). Just as posterior shift of the COP will propel the COM in the anterior direction, shifting of the COP in the lateral direction allows for the COM to move towards the stance limb. Thus, decreases in S1_ML observed in children with ASD will limit the extent to which the COM shifts towards the stance limb. As a result, when they transition from double limb support to single limb support, their COM will be further away from the base of support, resulting in less stable positioning during gait initiation. It therefore seems plausible that the significantly shorter S1_ML distances in the current investigation may indeed impart a dynamic postural instability.

In addition to further defining motor deficits in the ASD population, research investigating movement disturbances attempt to indirectly identify areas of the brain that may be responsible for these disturbances. Neuroanatomical and behavioral studies have implicated numerous areas of the brain responsible for impairments in this population, with an emphasis on the cerebellum (Courchesne, 1997; Courchesne et al., 1994; Pierce & Courchesne, 2001; Pierce et al., 2001; Ritvo et al., 1986) and the basal ganglia (Lewis, Tanimura, Lee, & Bodfish, 2007; McAlonan et

al., 2002; Nayate et al., 2005; Rinehart, Bradshaw, Brereton, & Tonge, 2001; Sears et al., 1999) for impairments observed in posture and gait. Both of these areas contribute to the tasks collected in the current investigation. The cerebellum is believed to integrate sensory information for balance control and the coordination of body movements (Kandel et al., 2000) whereas basal ganglia, although not as clearly defined, are reported to play an important role in normal voluntary movement and postural changes required for gait initiation and the maintaining of locomotion (Ebersbach et al., 1999; Kandel et al., 2000; Patchay, Gahery, & Serratrice, 2002) . Somatosensory (70%), vision (10%) and vestibular (20%) sensory information are the three main inputs in the control of posture (Horak, 2006). Several interconnected structures in the brain play a role in integrating this information, including the cerebellum and basal ganglia. The ability to re–weight sensory information is necessary for maintaining stability under changing environmental conditions (Horak, 2006). Limited studies of postural control and gait in ASD have lead to the belief that postural impairments observed in this population may be due to a dysfunction in sensory input integration occurring in the cerebellum (Hallett et al., 1993; Kohen-Raz et al., 1992). However, other findings suggest generalized postural dysfunction, gait initiation and gait patterns in ASD may be similar to those seen in Parkinson's disease, thereby implicating dysfunction in the basal ganglia (Hughes, 1996; Minshew et al., 2004; Vilensky, Damasio, & Maurer, 1981). While results from this investigation are unable to pinpoint specifically which anatomical structure or combination of structures are responsible for postural instabilities observed in children with ASD, these results do help confirm involvement of the cerebellum and/or basal ganglia.

Limitations and Future Directions

The current work has some limitations. Only two representative tasks were used in the current investigation to challenge postural control under static and dynamic conditions. A more

thoroughly defined spectrum of functional tasks with varying degrees of demands on the postural control system (including arm raise, sit to stand and sit to walk), is warranted. Tasks used in the current investigation are not directly related to one underlying control system (somatosensory, vision, and vestibular). Investigation of more tasks aimed at systematically manipulating each of these systems individually is needed to further identify the underlying causes of the postural deficits observed in this population.

It has been reported that an estimated 75% of children diagnosed with autistic disorder also possess mental retardation (MR). Of the limited literature investigating posture in the ASD population, some have controlled for MR (Minshew et al., 2004; Molloy et al., 2003) while others have not (Gepner et al., 1995; Kohen-Raz et al., 1992). Investigations containing individuals diagnosed with ASD and MR together, only ASD and only MR have reported that all of these have increased postural instability when compared to controls (Gepner et al., 1995; Kohen-Raz et al., 1992; Minshew et al., 2004; Molloy et al., 2003; Suomi & Koceja, 1994). It appears that both ASD and MR contribute to postural instability, but the extent of each individual contribution remains unclear. Evidence suggests however, that occurrence of abnormal repetitive behaviors is elevated in ASD when compared to MR (Bodfish et al., 2000). The distribution of the data in Figures 4-2, 4-4 and 4-6 appear to indicate that children with ASD and Leiter-R IQ scores of less than 70 (indicating MR), had larger values for the quiet stance variables measured and smaller values for the gait initiation variables measured, when compared to children with ASD and Leiter-R scores of greater than 70. These observations would suggest that MR contributed to the overall increased variability of the ASD group and as a result, may have contributing to the differences detected between groups. Therefore, it seems any

contribution the associated feature of MR has on postural instability in individuals with ASD should not be neglected.

Using one measure over a range of tasks is the inherent appeal in using the COP–COM moment arm for biomechanical analyses of postural control. Having one measure, not only allows comparisons across tasks, but also allows for comparison across ages, genders, disorders, and treatments. In order to compare COP–COM moment arms, the COP and COM must be collected. Although the COP is relatively easy to obtain in a laboratory and clinical settings via forceplates, the COM has traditionally been more cumbersome to obtain. Typically, motion analysis systems make use of marker data (defining body segments) to estimate the body's COM. It is a process that can be time consuming and requires expensive equipment and software. There are various methods for estimating the COM mathematically, however methods specific to gait initiation (as opposed to steady–state velocity) are still limited. As a result, the COP–COM moment could not be obtained for gait initiation trials. Therefore, choosing functional tasks where the COM has previously been obtained mathematically is suggested. Alternatively, for tasks where the COM cannot be obtained mathematically, it may be worth developing a plan aimed at increasing marker tolerance is this population.

CHAPTER 6
CONCLUSIONS

The findings of the current investigation suggest that motor differences in postural control are present in children diagnosed with ASD under both static and dynamic postural challenges. Larger COP movements in the mediolateral and anteroposterior directions and subsequent sway areas and larger peak COP–COM moment arms in the anteroposterior and resultant directions under static conditions (quiet stance) observed in children with ASD suggest increased postural sway and as result, increased postural instability. The development of anticipatory behavior characterized by the COP shift mechanism during gait initiation is not necessary for walking, but is required for efficient walking. Subtle differences in the ability to generate momentum while simultaneously maintaining balance under dynamic conditions (gait initiation) were observed between the two groups of children. Children with ASD shifted their COP significantly less towards the swing limb during gait initiation, suggesting instability in the mediolateral direction or possibly a less efficient strategy for generating momentum. No differences were detected between groups for posterior displacement of the COP, suggesting their ability to generate forward momentum during gait initiation is intact.

The results of the current investigation have systematically highlighted postural instabilities in children with ASD using functional tasks representative of two different categories of postural challenges. These findings have helped clarify some of the existing literature, indicating that children with ASD (8– 16 years) have postural instabilities, even under the most basic of conditions when no afferent or sensory information has been removed or modified. By further quantifying postural abilities over a wider range of tasks in this population, the extent to which deficits in motor control underlie core characteristics of the disorder will be further delineated. Given that motor control is mediated by brain structures that are consistently

reported to be compromised in this population, investigations into postural control provide a unique avenue for accessing altered functioning in these structures associated with ASD. Furthermore, by better characterizing the impairment associated with these disorders, behavioral treatments that include balance training, early in development, may help to prevent subsequent emergence of deficits in other motor abilities.

APPENDIX
CENTER OF PRESSURE (COP) & CENTER OF MASS (COM) CALCULATIONS

Calculating COP for One Forceplate

$$COP_i(x) = -M_{yi}/F_{zi} \quad (1)$$

$$COP_i(y) = M_{xi}/F_{zi} \quad (2)$$

Calculating COP_{net} for Two Forceplates

Method for calculating combined COP.

$$COP_{net}(x) = [(COP(x_1)*F_{z1})/(F_{z1}+F_{z2})] + [(COP(x2)*F_{z1})/(F_{z1}+F_{z2})] \quad (3)$$

$$COP_{net}(y) = [(COP(y1)*F_{z1})/(F_{z1}+F_{z2})] + [(COP(y2)*F_{z1})/(F_{z1}+F_{z2})] \quad (4)$$

Where,

i = forceplate of interest
COP (x) = COP location in the x direction
COP (y) = COP location in the y direction
x = M/L direction
y = A/P direction
M_y = moment about the y–axis
M_x = moment about the x–axis
F_z = ground reaction force in the vertical direction

(Winter et al., 2003)

Calculating COM via Integration

Trapezoidal Method of Numerical Integration

$$I_i = I_{i-1} + [(yi_{+1} - y_i)/2] *(x_{i+1} - x_i) \quad (5)$$

Where,

i = point of interest or i^{th} point
I_i = Integral of interest
y_i = y coordinate a i^{th} point
x_i = x coordinate at i^{th} point

Note: “i-1” term is required for even the first point. This is considered “the initial condition” for the integration.

Estimating COM Displacement

Method for estimating COM displacement described in Chan, 1999.

$a(t) = (g/W)\ f(t)$ horizontal acceleration (6)

$v(t) = \int [a(t) - a_0]\ dt$ integrated velocity (7)

$s(t) = \int [v(t) - v_0]\ dt$ integrated position (8)

$r(t) = s(t) - [u(t) - p(t)]$ estimated COM (9)

Where,

f(t) = horizontal ground reaction force
W= body weight
g = acceleration due to gravity
a_0 = mean of a(t)
v_0 = mean of v(t)
u(t) = moving average of s(t)
p(t) = moving average of COP
r(t) = estimated COM displacement

Note: moving averages are taken with a window of 4 seconds.

(Chan, 1999)

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BIOGRAPHICAL SKETCH

Kimberly Ann Fournier was born on August 31, 1973 in Moncton, New Brunswick, Canada. The second of three children, she grew up in Montreal, Canada where she graduated from Pierrefonds Comprehensive High School in 1990. She earned her B.S. in Athletic Training at Concordia University, Montreal, Canada in 1999 and her M.H.K. in Biomechanics at the University of Windsor, Windsor, Ontario, Canada. Upon completion of her Ph.D. program, Kimberly will be continuing her research in the area of autism, as a postdoctoral fellow with the support of funding obtained by Dr. Chris Hass and Dr. Krestin Radonovich through Autism Speaks™.