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Gait patterns in children with autism

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ABSTRACT

Background: Very few studies have examined the gait patterns of children with autism. A greater awareness of movement deviations could be beneficial for treatment planning. The purpose of this study was to compare kinematic and kinetic gait patterns in children with autism versus age-matched controls.
Methods: Twelve children with autism and twenty-two age-matched controls participated in the study. An eight camera motion capture system and four force plates were used to compute joint angles and joint kinetics during walking. Parametric analyses and principal component analyses were applied to kinematic and kinetic waveform variables from the autism (n = 12) and control (n = 22) groups. Group differences in parameterization values and principal component scores were tested using one-way ANOVAs and Kruskal–Wallis tests.
Findings: Significant differences between the autism and control group were found for cadence, and peak hip and ankle kinematics and kinetics. Significant differences were found for three of the principal component scores: sagittal ankle moment principal component one, sagittal ankle angle principal component one, and sagittal hip moment principal component two. Results suggest that children with autism demonstrate reduced plantarflexor moments and increased dorsiflexion angles, which may be associated with hypotonia. Decreased hip extensor moments were found for the autism group compared to the control group, however, the clinical significance of this result is unclear.
Interpretation: This study has identified several gait variables that were significantly different between autism and control group walkers. This is the first study to provide a comprehensive analysis of gait patterns in children with autism.

1. Introduction

Autism is a developmental disorder characterized primarily by impaired social interactions and communication, and restricted, repetitive, and stereotyped patterns of behaviour (APA, 1994). Persons with autism also demonstrate a variety of motor symptoms including: alterations in motor milestone development (Provost et al., 2007; Teitelbaum et al., 1998), hypotonia, muscle rigidity, akinesia, and bradykinesia (Damasio and Maurer, 1978; Kohen-Raz et al., 1992), and postural control impairments (Kohen-Raz et al., 1992; Minshew et al., 2004). These motor symptoms can compromise a child's ability to perform activities of daily living, such as walking. An abnormal walking pattern can lead to pain, fatigue, and joint stress, which in turn, may affect a child's functional capabilities and quality of life. To ensure optimal functionality, independence, and improved quality of life, researchers and clinicians must further examine the neuromuscular and biomechanical function of children with autism.

Although it has been shown that autism is associated with abnormal gait patterns and postures in children (Vilensky et al., 1981; Kohen-Raz et al., 1992; Vernazza-Martin et al., 2005), few studies have examined gait patterns associated with this disorder using quantitative methods. Of those available, most have focused solely on kinematics (Vilensky et al., 1981; Vernazza-Martin et al., 2005; Rinehart et al., 2006a,b) or force plate data (Fournier et al., 2006). To date, the only comprehensive study of the three-dimensional kinematics and kinetics of gait in autism is for adults (Hallett et al., 1993). To our knowledge, there are no studies on the three-dimensional kinematics and kinetics of gait patterns in children with autism.

Gait patterns of children with autism have been previously investigated. Vilensky et al. (1981) reported increased stance phases, shorter stride lengths, increased hip flexion at toe-off, decreased knee extension and altered foot contact patterns (toe or foot-flat contact) in twenty-one children with autism (3.9–11.3 years, mean age 7.1 years) compared to fifteen controls (3.3–10.0 years, mean age 6.1 years). In this study, the children with autism had displayed various autistic behaviours before the age of 30 months and were hospitalized in a psychiatric facility at the time of testing. Vernazza-Martin et al. (2005) also reported shorter step length in nine children with autism (4–6 years) versus six controls (4–6 years), along with increased upper body oscillations. Children with Rett syndrome, Asperger's disorder, and disintegrative disorders of childhood were excluded from the study. Contrary to Vernazza-Martin et al. (2005), Rinehart et al. (2006a), found increased stride length variability but not

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reduced stride length in ten high-functioning children with autism (6.75–14.4 years, mean age 10.6 years) compared to ten individually matched controls (where possible) on IQ, sex, and age. Similar findings were reported by Rinehart et al. (2006b) in eleven children with autism (4.3–6.75 years, mean 5.8 years) versus eleven controls matched on IQ, sex, and age.

A consistent and comprehensive account of gait in autism has not been established. More research is needed to further understand gait patterns associated with this disorder. In addition, studies that measure joint kinematics and kinetics could provide further information on the causes of differences in gait patterns in this population. For children with autism who may require therapy, a greater understanding of their gait patterns can also aid in the development of optimal treatment plans. Effective treatment programs lead to improved long-term functionality for children and reduced health care costs.

The purpose of this research was to compare the gait patterns of children with autism to age-matched controls. To aid in recognition of differences in patterns of motion, parameterization and waveform analyses were performed on the biomechanical data and results were compared.

2. Methods

2.1. Participants

Twelve children (n = 12) aged 5 to 9 years with autism participated in the study (10 males, 2 females; mean age = 6.3 years; mean height = 121.03 cm; mean weight = 29.31 kg). This study excluded children who were: 1) diagnosed with Asperger's or pervasive developmental disorder not otherwise specified, and/or 2) toe-walkers. Previous research has shown that the gait patterns of children with Asperger's disorder are relatively normal (Rinehart et al., 2006a). While children with autism may toe walk, this gait pattern is markedly different than other gait patterns. Pooling gait data from toe-walkers and non-toe-walkers could result in a loss of meaningful data for either group. Two children were excluded from the study due to toe-walking, resulting in 12 participants. Further participant characteristics are provided in Table 1. Hypotonia was confirmed in 33% and gross motor delay was confirmed in 25% of the children. Assessments were conducted by their physiotherapist during their routine physical examinations.

A portion of previously published control data was used for data comparisons (Chester et al., 2006) and consisted of twenty-two (n = 22) children aged 5–9 years (10 males, 12 females; mean age = 6.2 years; mean height = 119.42 cm; mean weight = 28.66 kg). Both the control and autism group were recruited from the local Fredericton area. One-way ANOVAs were used to test for significant differences ($P<0.05$) in height, weight, and age between the autism and control groups. No significant differences were found for height [$F(1,32) = 0.17, p = 0.68$], weight [$F(1,32) = 1.71, p = 0.20$], or age [$F(1,32) = 0.04, p = 0.84$]. While no significant between-group differences were found, it should be noted that Mervis and Robinson (2003) suggested that groups should only be considered matched on a control variable if between-group differences have a P value of at least 0.5. In the present study, we consider the characteristics of the control group to *approximate* those of the autism group.

Ethical approval for this study was obtained from the University of New Brunswick Research Ethics Board. Parental consent and child assent was obtained prior to each child's participation in the study.

Table 1
Participant characteristics for the autism group.

Subject	Age (years)	Height (cm)	Weight (kg)	Diagnoses
Male	5.25	118.5	23.1	No known diagnoses
Female	5.50	113.0	26.5	No known diagnoses
Male	5.83	116.0	25.4	Generalized mild hypotonia Gross motor delay
Male	8.42	136.5	45.4	No known diagnoses
Male	5.33	113.0	20.2	Generalized mild hypotonia
Male	5.33	124.0	30.6	Generalized mild hypotonia
Male	5.67	113.0	22.7	Gross motor delay
Male	5.75	108.5	21.3	Joint hypermobility
Male	8.25	135.0	51.9	No known diagnoses
Male	5.91	115.5	22.0	Mild central hypotonia Gross motor delay
Female	5.67	99.1	14.5	No known diagnoses
Male	9.08	160.2	48.1	No known diagnoses

2.2. Instrumentation/apparatus

An eight camera Vicon MCam motion capture system (Oxford Metrics Group Ltd.), sampling at 60 Hz, was used to track the three-dimensional trajectories of reflective markers placed on the subjects' skin. Four force plates (Kistler 9281B21, 9281CA, Kistler Instruments, Winterthur, Switzerland) embedded in the lab floor measured the three-dimensional forces and moments during gait. A weight scale, measuring tape, and calipers were used to obtain anthropometric measures from each subject.

2.3. Procedures

Twenty reflective markers were placed directly on the skin of each participant in accordance with Davis et al. (1991). Several 'warm-up' trials were conducted to allow the participants to adjust to the markers and the lab environment. Children were then encouraged to perform at least 20 trials, if possible.

2.4. Data analysis

Data was analyzed using custom software created in Matlab (Mathworks, Natick, Massachusetts, USA). For each participant, trial selection involved the computation of cadence, velocity, and percent of cycle spent in single stance for each gait cycle. The single gait cycle that most closely approximated the individual mean of all gait cycles on these three measures was selected as the single trial for analysis for each participant. The rigid body model consisted of the left and right foot, shank, thigh and the pelvis and trunk. The locations of the three non-collinear markers on each body segment were used to create embedded coordinate systems at the joint centers. Joint angles were computed from the relative orientations of the embedded coordinate systems using Euler angles in an *yxz* sequence, corresponding to flexion/extension, adduction/abduction, and internal/external rotation. Joint center locations were estimated in accordance with Davis et al. (1991). The required absolute linear and angular velocities and accelerations were calculated using a five-point derivative. Displacement data were filtered using a 6 Hz low-pass Butterworth filter prior to differentiation. Net joint moments and joint power for the hip, knee, and ankle joints were estimated using an inverse dynamics approach. Joint moments and joint power were normalized to body weight, and all data were normalized to 100% of the gait cycle.

2.5. Statistical analysis

Significant ($P<0.05$) differences in temporal–spatial parameters and discrete parameters (Table 2) between the control and autism group were tested using a series of one-way ANOVA's with Bonferroni adjustments. For the principal component scores, a test of normality (Shapiro–Wilk) and equality of covariance matrices (Box's Test) revealed that the assumptions of multivariate normality and equality of variances for a MANOVA could not be met. Therefore, a series of one-way ANOVA's with Bonferroni adjustments were used to test for significant differences for the principal component scores that were

Table 2
Group descriptive statistics for temporal–spatial data and gait variables obtained by parameterization methods (* refers to significant differences between groups, $P<0.05$). Phase refers to the portion of the cycle the data was extracted from [Cycle = entire gait cycle; Stance = stance phase; Swing = swing phase].

Variables	Autism			Control	
	Phase	Mean	SD	Mean	SD
Cadence (steps/min)	Cycle	134.27*	18.25	125.50	14.40
Walking speed (cm/s)	Cycle	99.89	17.20	99.45	21.96
Stride length (cm)	Cycle	54.71	7.93	58.15	7.18
Double stance (%)	Cycle	21.31	3.37	20.32	3.03
Toe-off (%)	Cycle	61.06	2.35	60.10	2.16
Cycle time (s)	Cycle	0.91	0.13	0.97	0.12
Maximum pelvic tilt (deg)	Cycle	15.23	4.82	12.93	6.50
Minimum pelvic tilt (deg)	Cycle	10.62	4.97	8.41	6.03
Maximum pelvic obliquity (deg)	Cycle	4.28	2.60	3.90	2.88
Minimum pelvic obliquity (deg)	Cycle	−5.10	2.71	−3.13	2.76
Maximum pelvic rotation (deg)	Cycle	7.74	6.41	6.85	−5.65
Minimum pelvic rotation (deg)	Cycle	−7.78	6.28	6.59	6.37
Maximum hip flexion (deg)	Stance	41.05*	8.50	35.07	9.00
Maximum hip flexion (deg)	Swing	42.66*	8.55	37.18	8.90
Maximum hip extension (deg)	Stance	−6.06	7.81	−7.52	8.79
Sagittal hip range of motion (deg)	Cycle	49.53*	7.12	46.96	11.09
Maximum hip adduction (deg)	Stance	8.76	4.04	8.60	5.24
Maximum hip abduction (deg)	Stance	−4.12	4.11	−2.03	5.37
Maximum hip extension moment (Nm/kg)	Stance	0.92	0.40	0.89	0.32
Maximum hip flexion moment (Nm/kg)	Stance	−0.29*	0.11	−0.38	0.18
Maximum hip power (W/kg)	Cycle	1.42	0.63	1.29	0.89
Maximum knee flexion (deg)	Stance	27.25	8.08	24.20	10.62
Maximum knee flexion (deg)	Swing	60.82	9.67	61.62	9.41
Maximum knee extension (deg)	Stance	9.38	10.14	7.64	8.17
Sagittal knee range of motion (deg)	Cycle	53.29	7.11	57.15	10.06
Maximum knee extension moment (Nm/kg)	Stance	0.29	0.16	0.28	0.20
Maximum knee flexion moment (Nm/kg)	Stance	−0.39	0.18	−0.41	0.18
Maximum knee power (W/kg)	Cycle	0.93	0.63	0.90	0.54
Maximum ankle dorsiflexion (deg)	Stance	18.07	7.31	9.03	10.19
Maximum ankle dorsiflexion (deg)	Swing	10.19	6.53	2.14	10.25
Maximum ankle plantarflexon (deg)	Stance	−9.12	9.19	−17.26	12.10
Sagittal ankle range of motion (deg)	Cycle	27.26	8.75	26.60	7.16
Maximum foot rotation (deg)	Cycle	−1.01	10.75	−2.73	7.23
Maximum dorsiflexion moment (Nm/kg)	Stance	−0.06	0.06	−0.09	0.13
Maximum plantarflexion moment (Nm/kg)	Stance	1.12*	0.21	1.27	0.25
Maximum concentric ankle power (W/kg)	Stance	2.54	0.74	2.63	1.10
Maximum eccentric ankle power (W/kg)	Stance	−0.64	0.29	−0.87	0.44

normally distributed with equal covariance matrices, while the Kruskal–Wallis Test was used to analyze the principal component scores that failed to meet these assumptions. All statistical tests were performed using SPSS (SPSS Inc).

For the waveform analyses, principal component analysis (PCA) was applied to the 14-kinematic and kinetic waveform variables from each group (equivalent to the number of different waveforms used in the parameterization method, see Table 2). Each waveform was normalized to 51 time points, one for every 2% of the gait cycle. Each waveform variable was stored in a separate matrix with dimensions 34×51 (# of subjects X # of time points), for a total of 14 matrices. The first 22 rows of subjects were the control group data, while the remaining rows were for the autism group data. All waveform data was transformed into principal components using an eigenvector analysis of the covariance structure. This facilitated the assessment of both magnitude and pattern differences across the gait cycle. Based on the small sample sizes in this study, the first two principal components were retained for each kinematic and kinetic waveform variable. The first principal component represents the maximum amount of variability accounted for in the original data. The second principal component explains the maximum amount of variability not accounted for by the first principal component.

For each waveform variable, differences between the autism and control group were examined using the principal component scores, principal component coefficients, and principal components scaled to the proportion of variability accounted for (Jackson, 1991). The scaled principal component represents the correlation between the *i*th principal component and the *j*th time sample using the associated standard deviation. The scaled principal components are then squared and represent the proportion of variability accounted for by the principal component at each portion of the gait cycle. Principal component scores provide a measure of distance indicating how closely each waveform conforms to the pattern of variability captured by each principal component. Principal component coefficients provide measures of magnitude and direction of deviation from the mean curve, while scaled principal components provide information on where in the gait cycle the principal component loads the greatest. Mean principal component scores were used to illustrate the patterns of variation captured for each waveform variable. Only principal components associated with significant between-group differences in PC scores were presented. All PCA calculations were performed using Matlab (Mathworks, Natick, Massachusetts, USA). A more detailed account of this statistical analysis can be found in Wrigley et al. (2006).

3. Results

3.1. Parameterization

A significant difference ($P<0.05$) in temporal–spatial data was found between the group of children with autism (autism group) and the age-matched control group (Table 2). Cadence was significantly higher for the autism group compared to the control group. No other significant differences in temporal–spatial data were found.

Several differences in joint kinematics were observed between the two groups (Table 2). Compared to the control group, the autism group showed significant differences in ankle kinematics, namely, increased peak ankle dorsiflexion in swing phase and decreased peak plantarflexion angles. Significant differences were also found for ankle joint kinetics (Table 2). Peak plantarflexor moments were significantly smaller in the autism group. Significant group differences ($p<0.05$) were also observed in hip joint kinematics and kinetics (Table 2). The autism group showed increased peak hip flexion angles in stance, and swing phase. In terms of hip angles, the autism group also showed a greater mean range of joint motion. In addition, the autism group demonstrated significantly decreased peak hip flexor moments.

3.2. Waveform analysis

Significant differences ($P<0.05$) were found for three of the principal component scores: sagittal ankle moment principal component one, sagittal ankle angle principal component one, and sagittal hip moment principal component two. Descriptive data for these scores are provided in Table 3. For each waveform variable, the number of principal components retained for comparison was limited to a maximum of two, due to the small sample size of the group of

Table 3
Group descriptive data for waveform variables found to be significantly different ($P<0.05$) using PCA.

PC Scores	Autism			Control		
	Mean	SD	Range	Mean	SD	Range
Sagittal ankle angle PC1	−40.31	71.74	298.26	18.2	39.03	251.83
Sagittal ankle moment PC1	0.28	1.08	3.96	−0.13	0.85	3.29
Sagittal hip moment PC2	−0.23	0.6	2.5	0.11	0.65	3.33

children with autism. On average, these principal components accounted for 86.88% of the variation in the biomechanical data.

3.3. Sagittal ankle moment

Sagittal ankle moment principal component one (Fig. 1a) accounted for 55.2% of the total variation and captured the change in pattern of the waveforms (from first either adding then subtracting from the mean waveform or vice versa). A positive PC score (Table 3), along with a negative PC coefficient indicate that during this first half of the cycle, indicates that the autism group deviates by subtracting from the mean sagittal ankle moment waveform. From 50–70% of the gait cycle, the PC coefficient becomes positive and the ankle moment for the autism group adds to the mean, indicating an increased plantarflexor moment.

To assess the relative importance of principal component coefficients, sagittal ankle moment principal component one was scaled to the percentage of variation explained and plotted in Fig. 1a. From this plot, it is evident that the majority of variability captured by sagittal ankle moment principal component one occurs during the first half of the cycle with approximately 90% of the total variability accounted for at approximately 32% of the gait cycle. From midcycle to early swing (50–70% of the cycle), a maximum of 48% of the total variation is explained at 58% of the cycle. For the remainder of the gait cycle, a smaller peak accounts for a maximum of 12% of the variation during swing phase (76%). However, since the magnitude of this portion of the curve is very low, any variations within this phase are of little practical relevance.

To illustrate the pattern of variability captured, mean principal component scores for sagittal ankle moment principal component one were transformed into the original coordinate space to create representative waveforms. Fig. 1b shows the representative waveforms for each group. The autism group shows decreased plantarflexor moments during the first half of the cycle, and then increased moments until the swing phase.

3.4. Sagittal ankle angle

Sagittal ankle angle principal component one accounts for 66.83% of the total variation and captured differences in the magnitude of the waveforms during the gait cycle. For the autism group, the positive PC coefficient (Fig. 2a) and the positive PC score (Table 3) indicates that this group deviates by consistently adding to the mean ankle angle waveform (Fig. 2b), while the negative score for the control group leads to their waveform subtracting from the average. From the plot of the principal component coefficients scaled to the percentage of variation explained (Fig. 2a), it was evident that the majority of variability captured by sagittal ankle angle principal component one occurs during the early to midstance and late swing phase. Approximately 63% of the

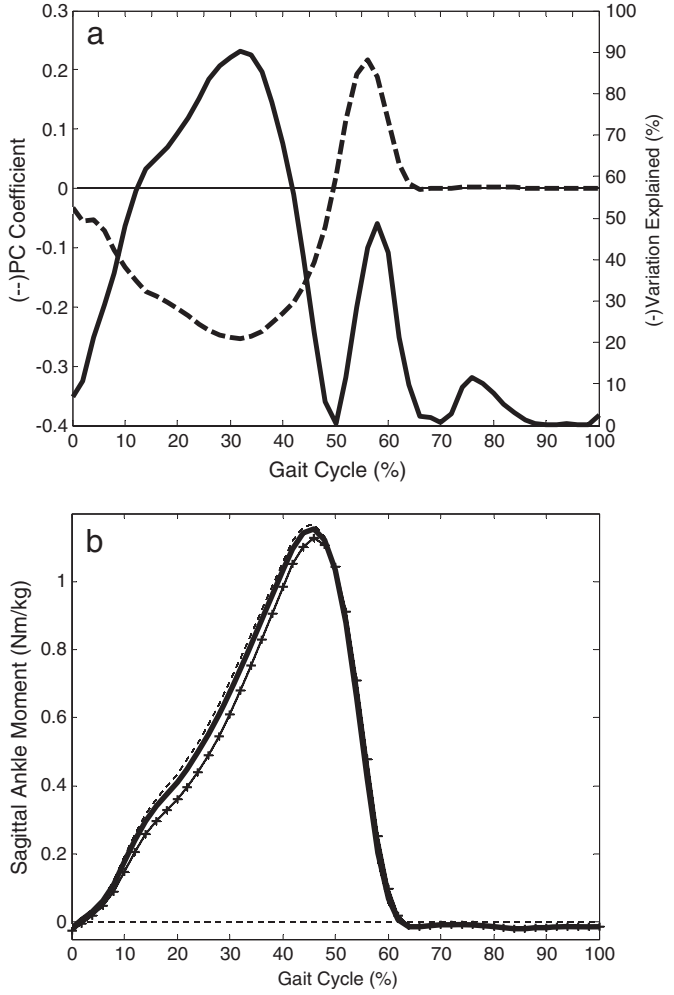

Fig. 1. (a) Sagittal ankle moment principal component (PC) one plotted in the original form (dashed line) and as a percentage of variation explained (solid line). (b) To illustrate the mode of variability captured, the transformed mean curve (thick line), along with transformed waveforms that correspond to the control group (dashed line) and the autism group (crosses), were plotted.

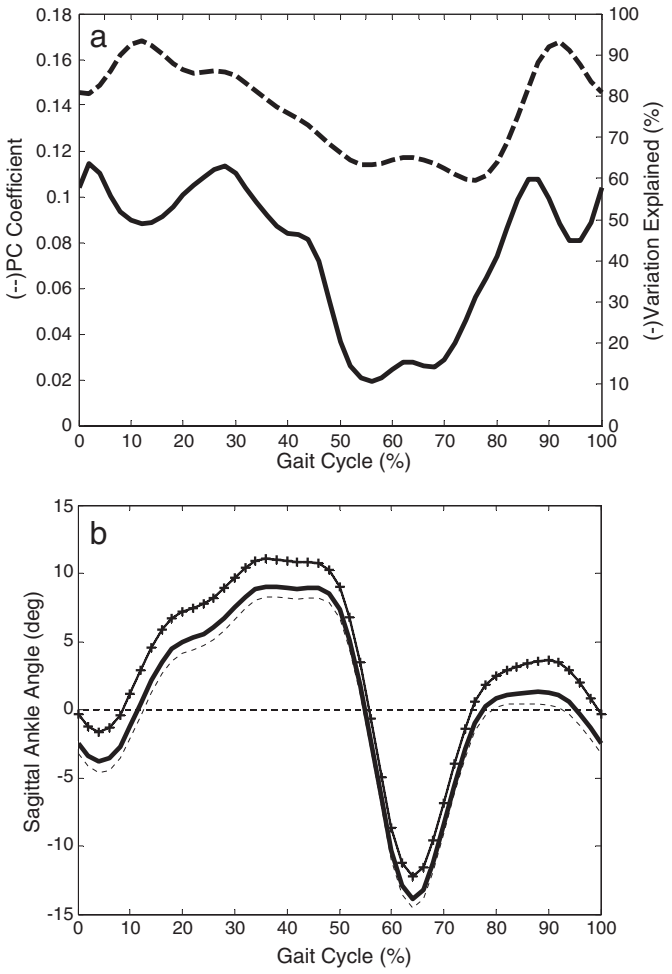

Fig. 2. (a) Sagittal ankle angle principal component (PC) one plotted in the original form (dashed line) and as a percentage of variation explained (solid line). (b) To illustrate the mode of variability captured, the transformed mean curve (thick line), along with transformed waveforms that correspond to the control group (dashed line) and the autism group (crosses), were plotted.

total variability was accounted for during initial loading (Fig. 2a). Smaller portions of variability were captured during preswing and early to midswing. The representative curves plotted in Fig. 2b display this pattern of variability quite clearly. The autism group shows an angle waveform that is shifted upwards across the entire cycle, resulting in decreased plantarflexion angles at the beginning of the cycle, followed by increased dorsiflexion angles until preswing, decreased plantarflexion in preswing and early swing, and then increased dorsiflexion for the remainder of the cycle.

3.5. Sagittal hip moment PC2

Sagittal hip moment principal component two accounted for 25.51% of the total variation and captured the change in pattern of the waveforms. Fig. 3a reveals three fluctuations in the pattern of variation. The first area occurs from 0 to 20% of the gait cycle, with a peak of 31% of the total variability explained at 10% of the gait cycle. During initial loading, the positive principal component coefficient and the negative PC score (Table 3) for the autism group leads to a lower sagittal hip moment compared with the average waveform (Fig. 3b). The principal coefficient then becomes negative between 20–70% of the cycle, which results in the autism group adding to the mean curve (Fig. 3b). The majority of the variability captured by the principal component concentrates between 20 and 70% of the gait cycle, with 70% of the maximum variability accounted for at midcycle. Therefore, principal component two captures the difference in the magnitude of the sagittal hip moments in this region, with the autism group demonstrating a smaller peak hip flexor moment. The principal component captures smaller portions of variability for the remainder of the cycle (70–100% of the gait cycle), with approximately 20% of the variation being explained at 95% of the gait cycle.

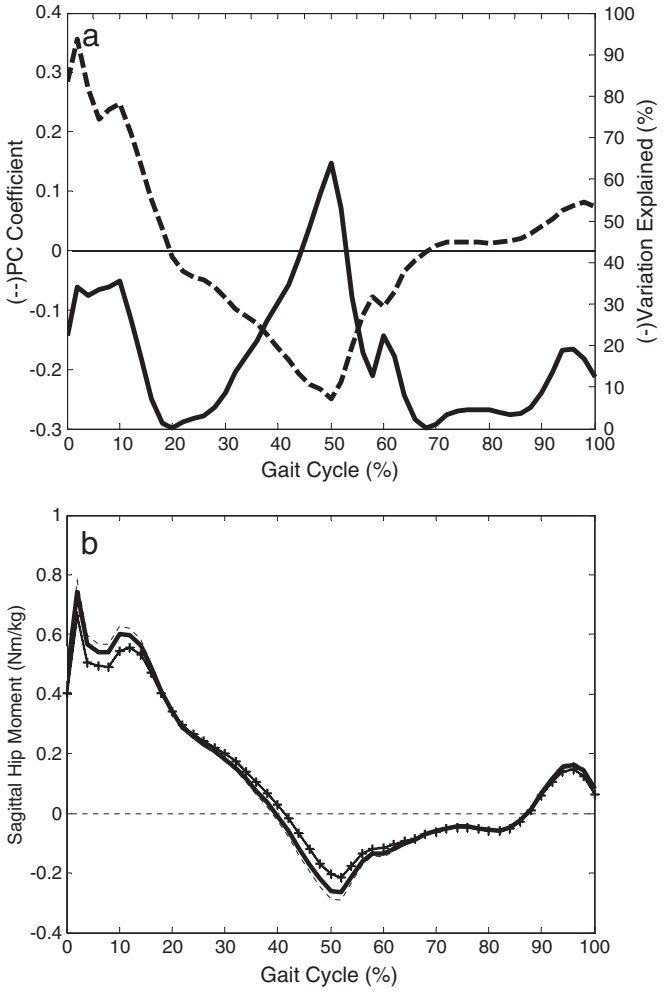

Fig. 3. (a) Sagittal hip moment principal component (PC) two plotted in the original form (dashed line) and as a percentage of variation explained (solid line). (b) To illustrate the mode of variability captured, the transformed mean curve (thick line), along with transformed waveforms that correspond to the control group (dashed line) and the autism group (crosses), were plotted.

4. Discussion

The purpose of this research was to compare the gait patterns of children with autism to age-matched control data. To identify differences in the magnitude and pattern of kinematic and kinetic waveforms between the two groups, the data was analyzed using parameterization techniques and a principal component analysis. The results of the two analyses were then compared. The parameter-based analysis found significant group differences ($P<0.05$) for cadence, peak ankle dorsiflexion in swing, peak plantarflexion angle and moment, hip range of motion, peak hip flexion in stance and swing, and peak hip flexor moments. Significant group differences ($P<0.05$) were found for three of the principal component scores, namely the sagittal ankle moment principal component one, sagittal ankle angle principal component one, and sagittal hip moment principal component two.

A significantly higher cadence was found for the autism group compared to the control group. Unlike previous work, no significant differences were observed in stride length (Vilensky et al., 1981; Vernazza-Martin et al., 2005) or in the percentage of cycle spent in the stance phase (Vilensky et al., 1981). The findings of the present study are similar to those of Rinehart et al. (2006a), who found no significant differences in the mean values of temporal–spatial data between groups of children with high-functioning autism, Asperger's disorder, and controls. Similar results were found in a subsequent study of younger children with autism versus controls (Rinehart et al., 2006b). Despite the lack of significance in mean values of temporal–spatial data, greater stride length variability was observed in children with autism compared to those children with Asperger's disorder and controls (Rinehart et al., 2006a). Greater variability in walking velocity, stride time, and stride length was also found for children with autism versus controls (Rinehart et al., 2006b). While the present study did not assess variability, the results suggest that the autism and control groups are similar in terms of temporal–spatial gait measures.

As described by the principal component scores and coefficients, significant differences in the first principal component scores for the sagittal ankle moment pattern were observed between the two groups. A maximum of 90% of the total variability was accounted for at 32% of the gait cycle (Fig. 1a). The autism group showed decreased plantarflexor moments during the first half of the gait cycle. These results were similar to the parameterization technique, which also found significantly smaller peak plantarflexion moments in the autism group (Fig. 1b). However, the PCA technique was able to identify decreased plantarflexion moments for the first half of the cycle, providing an indication of when the autism group began to diverge. The retention of temporal and pattern data in the PCA analysis was important for discriminating between the two groups of children.

It has been shown that plantarflexor moments are associated with walking speed (Stansfield et al., 2001), however, no significant differences were found for velocity between the autism and control group [autism: mean 0.99 m/s (SD 0.22); control: 1.0 m/s mean (SD 0.17)]. In the present study, it is likely that the decreased plantarflexor moment is related to hypotonia, confirmed in 33% of the individuals in the autism group (Table 1). During the stance phase of gait, plantarflexor moments are generated to oppose the passive dorsiflexor moments generated by ground reaction forces and to control

the forward progression of the tibia. The results suggest that there are differences in the ability to counteract passive dorsiflexor moments and/or control tibial advancement. The decreased plantarflexor moments in the autism group may lead to decreased forward progression and support as less power would be transferred to the trunk and swing limb (Neptune et al., 2001). These findings may be important in terms of treatment planning for children with autism (e.g. plantarflexor strength training, stretching, and orthotics).

Significant differences in sagittal ankle angles between the autism and control group were found by both the parameterization and waveform techniques. For the autism group, the parameterization method showed decreased peak plantarflexion angles and increased peak ankle dorsiflexion angles in swing. Significant differences in the first principal component scores for the sagittal ankle angle pattern were observed between the autism and control groups. Approximately 63% of the total variability was accounted for at initial loading (Fig. 2a). Unlike the plantarflexed ankle position shown by the control group at initial contact, the autism group demonstrated a neutral ankle position followed by reduced plantarflexion angles until 8% of the gait cycle (Fig. 2b). In contrast, Vilensky et al. (1981) estimated sagittal ankle angles at two instances of the gait cycle, namely foot strike and toe-off, and found increased plantarflexion angles at initial contact in children with autism compared to a control group. These contradictory results may be partially due to considerable differences in motion capture technology and techniques between the two studies. These contradictory results may be partially due to considerable differences in motion capture technology and techniques between the two studies. Vilensky et al. used two 50 Hz cameras to record data on film, projected the images, and visually estimated joint center locations. Two-dimensional angles were then measured using a protractor. These manual measurements would rely on the experience of the analyzer and may have been prone to error. In addition, the two-dimensional angles obtained from this technique may lead to underestimations of the true joint angles. To measure ankle angles, Vilensky et al. placed a marker on the distal hallux, which may have cause errors in ankle angle values (hallux can move independently). Therefore, marker placement can contribute to error and cause differences in results between studies. Further research, with modern equipment and standardized protocols and data processing techniques, is needed to improve our understanding of gait patterns in autism.

Across the gait cycle, the majority of the variability captured by sagittal ankle angle principal component one was related to the increased dorsiflexion in the autism group from early stance to preswing and also during the swing phase (Fig. 2a). The increased dorsiflexion angles in stance coincided with the decreased plantarflexion moments (Fig. 2b). In contrast to the parameterization technique, less than 20% of the variability was explained at toe-off when peak plantarflexion occurs. Parameterization techniques typically extract parameters from gait waveforms in a subjective and a priori manner. While the results of the PCA analysis must be confirmed with greater sample sizes, they suggest that peak plantarflexion angles do not differentiate between autism and control gait patterns. The statistical method used to analyze gait patterns may be an important factor in our understanding of gait in autism.

Significant differences in sagittal hip joint kinematics and kinetics between the autism and control group were found by the parameterization technique. For the autism group, this method of analysis showed increased range of motion, increased peak hip flexion angles in stance, and decreased peak hip flexor moments compared to the control group (Fig. 4). While findings of the waveform analysis were similar for hip joint kinetics, no significant differences in hip kinematics were found using PCA. Vilensky et al. (1981) also examined hip kinematics in children with autism compared to a control group and found increased hip flexion at toe-off. In the present study however, significantly increased hip flexion angles were found

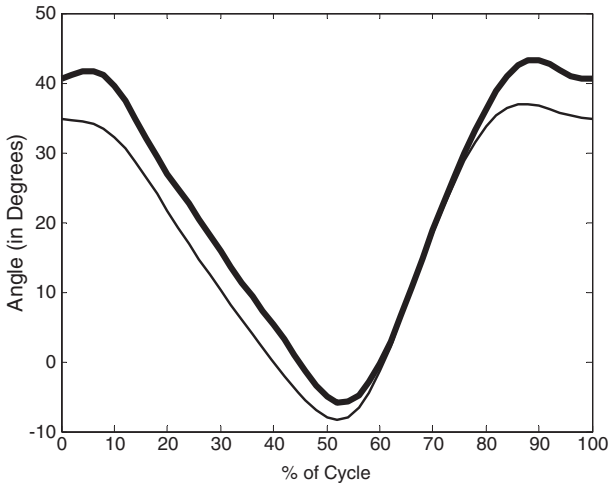

Fig. 4. Mean sagittal hip angle for the autism (thick line) and control group (thin line) versus percentage cycle.

in early stance and mid-to-late swing. In the absence of any significant differences in pelvic or knee angles, the relevance of this finding is unclear. More research is needed to investigate hip movement in children with autism and its relation to hypotonia and decreased plantarflexor moments.

Significant differences in the second principal component scores for the sagittal hip joint moment pattern were observed between the autism and control groups. The principal component accounted for 25.51% of the total variation and captured the change in pattern of the waveforms (Fig. 3a). The autism group showed a decreased extensor moment during the initial 20% of the cycle. However, the majority of variability explained occurs between 20 and 68% of the cycle with the peak variability explained (70%) occurring near the peak hip flexor moment at midcycle. The autism group showed decreased peak hip flexor moments compared to the control group (Fig. 3b). The initial hip extensor moment is used to generate hip extension, while the peak hip flexor moment at midcycle facilitates the reversal of hip motion from extension to flexion. The decreased hip flexor moment may be related to the decreased hip extension at midcycle. The lack of significant differences in walking velocity and pelvic and knee kinematics suggests that this is not a major gait deviation.

As autism is a heterogenous disorder often involving a spectrum of motor symptoms, we excluded children with autism who were toe-walkers or diagnosed with Asperger's disorder. As such, the results of this study focused on high-functioning children with a history of normal tone, low tone, and/or gross motor delay. Therefore, these results should not be generalized to toe-walkers or children with hypertonicity. Further, many of the children who volunteered for this study (8 out of 12) were either receiving or have received physiotherapy treatment. Therefore, it is possible that the natural gait patterns of the children have been modified as a function of treatment.

5. Conclusions

No previous studies have examined the kinematic and kinetic gait patterns of children with autism. This study found that the gait patterns of children with autism are significantly different from control data based on 1) plantarflexor moments and angles, and 2) hip flexor moments and angles. Ankle angles and plantarflexor moments indicate that plantarflexor weakness is one possible cause of altered gait patterns. At the hip, decreased hip extensor moment activity may be partially due to increased hip flexion during gait. However, the cause of the increased hip flexion is unclear. This study analyzed the gait data using parameterization methods, as well as PCA. PCA, which

requires no a priori decisions on gait parameter importance, provide additional information on gait patterns in autism. Future work should use larger sample sizes to validate the findings in the present study. Future studies should also focus on gait analyses in those groups that were excluded from this study (toe-walkers, Asperger's disorder, and otherwise-non specified disorder). In addition, the use of electromyography to measure individual muscle activity would increase our understanding of the identified gait deviations in these populations.

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