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Original Article

Clinical gait analysis in older children with autism spectrum disorder

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ABSTRACT

Individuals with autism spectrum disorder (ASD) often exhibit motor deficits that increase their risk of falls. There is a lack of understanding regarding gait biomechanics demonstrated by older children with ASD. The purpose of the study was to determine differences in gait patterns between older children with ASD and typically developing children. Eleven children with ASD and 11 age- and gender-matched typically developing children were recruited for the study. Participants walked on a force-instrumented treadmill at a constant speed (1.1 m/s - 1.2 m/s) for five minutes (min). Participants performed maximal voluntary contractions to assess their knee muscular strength. Differences between individuals with ASD and matched control participants were examined through paired *t*-tests with a significance level of $p \leq 0.05$. Individuals with ASD demonstrated a smaller knee extensor torque compared to controls ($p = 0.002$). Participants with ASD exhibited a shorter stride length ($p = 0.04$), a greater cadence ($p = 0.03$), and a higher variation in stride width ($p = 0.04$) compared to control participants. The individuals with ASD experienced a greater braking ground reaction force ($p = 0.03$) during loading response. The results indicate older children with ASD develop a unique gait pattern signified by a reduced stride length, increased cadence, and an increase of variation in stride width. This unique gait pattern may represent a movement strategy used by the individuals with ASD to compensate for the weakness associated with their knee extensor muscles. Individuals with ASD who demonstrate these unique gait deviations may face reduced postural stability and an increased risk of fall-related injuries.

1. Introduction

Autism spectrum disorder (ASD) is a type of neurodevelopmental disorder commonly diagnosed in the early years of childhood.[1] ASD is defined by impairments in communication, deficits in social interaction, and restricted or repetitive behavior.[2] In the United States, according to the Center for Disease Control and Prevention, as many as 1 in 36 children have been diagnosed with ASD.[3] Although the etiology of ASD is unclear, it is generally accepted that environmental and/or genetic factors contribute.[4] Disrupted neurodevelopment may also lead to other symptoms in individuals with ASD, including movement system impairments. Previous research found children with ASD exhibited abnormal motor function as early as their first year of life.[5] For example, infants who were later diagnosed with ASD showed gross motor deficits, such as delayed motor development, abnormal muscle tone, and posture asymmetry.[6] In addition, toddlers who were later diagnosed with ASD tended to exhibit abnormal gait patterns such as toe walking.[7] Children that walk with altered gait may face an increased risk of falling-related injuries during daily activities.[8] Furthermore, motor deficits could result in reduced participation in physical activity for children with ASD.[9]

Laboratory gait analysis for individuals with ASD is necessary to improve understanding and provide a basis for the development of rehabilitation programs. However, previous research on quantitative gait analysis on children with ASD is very limited and inconsistent. Such research primarily focused on gait patterns of younger children with ASD, with mean ages typically ranging between two and 11 years.[8,10,11] Among these studies, children with ASD were observed to exhibit a shorter stride length,[12–14] greater stride width,[10–12] and higher cadence compared to typically developing controls who achieved developmental milestones according to their ages.[7] Besides changes in temporo-spatial parameters, ground reaction forces (GRF) were found to be different between children with ASD and their controls.[11] Children with ASD were observed to have greater braking GRF during loading response and lower vertical GRF during push-off than their typically developing peers.[11] This is potentially problematic because it may suggest lower stability and shock absorption during weight acceptance, as well as insufficient control of body weight during terminal stance.[11]

However, there were also studies reporting no differences in gait

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Abbreviations

ASD – Autism Spectrum Disorder
IRB – Internal Review Board
BMI – Body Mass Index
GRF – Ground Reaction Force
AP – Antero-posterior
ML – Medio-lateral
ASIS – Antero-superior Iliac Spine
MVIC – Maximal Voluntary Isometric Contraction
Co-var – Coefficient of Variation

parameters including stride length,[7,15,16] stride width,[16,17] and cadence between children with ASD and the controls.[15] The inconsistencies between these findings highlight the complex influence of ASD. It is reported that children with typical development adopt a mature gait pattern after age eight,[18,19] but children with ASD could experience a greater delay in gait development.[20] Additionally, children with ASD develop their mature gait at different rates, so the high degree of variation in age ranges in previous research has made direct comparison between studies difficult. In order to better characterize ASD-related gait patterns, it is necessary to study children with ASD after they have developed a mature gait. There is currently a general absence of gait data from older children with ASD.

Thus, the primary purpose of the study was to examine gait patterns exhibited by older children with ASD in comparison to a group of age- and gender-matched controls. It was hypothesized that older children with ASD would exhibit alterations in gait parameters and ground reaction forces when compared to their controls. Additionally, as the ASD condition could impact physical condition and limit the ability to perform physical activities, the secondary purpose of the study was to examine the knee muscular strength of individuals with ASD in comparison to their controls. It was hypothesized that individuals with ASD would present a deficit in knee strength compared to their controls.

2. Methods

2.1. Participants

Eleven older children age ranged from nine to 17 years old with ASD were recruited for the study. Mature gait has been shown in children with ASD around the age of eight years.[20] Eleven age- and gender-matched typically developing children were also recruited as controls. The sample size was estimated by using G*Power software (large effect size, significant level: 0.05, power: 0.8). It was performed to determine that a sample size for the participants in each group was eight.[14] To participate in the study, individuals with ASD were required to present proof of a clinical diagnosis of the disorder from a medical professional, which was also verbally confirmed by each individual's legal guardian. In addition, in order to be able to apprehend the instructions of performing laboratory testing activities, ASD individuals must have high functioning Autism.[21] Each participant with an ASD diagnosis was age- and gender-matched to a typically developing participant without an ASD diagnosis. This study excluded individuals with ASD who exhibited toe walking, which is an abnormal gait without the involvement of heel strikes.[22] Individuals were excluded if they had comorbidities affecting their motor developments such as heart diseases and obesity. Moreover, individuals with neuromuscular and orthopedic impairments that affect gait movement were excluded. Finally, we also received parents' confirmations of noticeably delayed motor developments attributed to ASD during early childhood for 8 of the 11 ASD participants.

2.2. Ethical approval

Approval from the university's Institutional Review Board (IRB) was obtained prior to the commencement of the study [IRB#1243649]. Signed informed consent documents were obtained from participants' parents and assent was obtained from participants.

2.3. Procedures

This study was conducted in a controlled laboratory setting. Upon arrival, participants were asked to change into tight-fitting clothing and standardized athletic shoes. Anthropometric measurements were recorded for height, weight, inter-anterosuperior iliac spines (ASIS) distance, knee length, knee width, and ankle width. The researcher placed twenty-two retro-reflective markers and six marker clusters on the surface of participant's compression clothing by using a modified Plug-in Gait marker set. Motion capture was performed with a 15-camera Vicon system (MX40, Oxford Metrics, Oxford, UK) sampling at 100 Hz. The walking task took place on an AMTI force-instrumented treadmill (Advanced Mechanical Technology, Inc., Watertown, MA, USA) with force plates used to collect three-dimensional ground reaction forces at 2000 Hz. A harness was used to fully support the participant while on the treadmill if a loss of balance occurred.

Following preparation procedures, the participants were given a 3-min (min) warm up trial on the treadmill to acclimate to the instrument and lab environment. The treadmill speed was gradually increased to a target speed for each participant. For participants aged 12 years old and under, the treadmill speed was 1.1 m/s.[18,19] For participants aged above 12 years old, the treadmill speed was 1.2 m/s.[18,19] Participants then performed the walking task. During this task, they were asked to walk on the treadmill at a constant speed for 5 min. Participants were then given a 5-min break before they performed maximal voluntary isometric contractions (MVIC) on a Cybex dynamometer (Cybex International, NY, USA) to measure knee muscular strength. The MVIC testing for knee flexors and extensors were performed at 30° and 60° of knee flexion, respectively. Knee strength values were normalized to the participant's body mass. This protocol was verified as a reliable way to measure quadriceps and hamstring strength.[23]

2.4. Data processing and analysis

VICON Nexus 2.7 (Oxford Metrics, Oxford, UK) was used to track and reconstruct marker trajectories during walking trials. Visual 3D v.6 (C-Motion, Germantown, MD, USA) was used to process biomechanical data. Specifically, a fourth-order Butterworth low-pass filter was used to smooth marker trajectory data at 8 Hz and ground reaction force data at 40 Hz. Temporo-spatial parameters including stride length, stride width, cadence, and double-limb support time were computed. Ground reaction force variables including peaks of antero-posterior (AP), medio-lateral (ML), and vertical GRF were extracted. The coefficient of variation (*Co-var*) was calculated for stride width in order to investigate gait variability.[24] Statistical analyses were performed using the SPSS software (Version 25.0 for Windows, IBM Corp., Chicago, IL, USA). Differences in knee strength, temporo-spatial parameters, and ground reaction forces during walking between individuals with ASD and matched control participants were examined through paired t-tests (significance level set at $p < 0.05$). Cohen's *d* was calculated to assess effect size (0.2 = small effect, 0.5 = moderate effect, 0.8 = large effect).[25]

3. Results

There were no significant differences between groups for age ($p = 0.72$), height ($p = 0.23$), mass ($p = 0.84$), and body mass index (BMI) ($p = 0.71$) (Table 1). Individuals with ASD demonstrated a significantly shorter stride length (6 % less) ($p = 0.03$) and greater cadence (8 % more) than the control group ($p = 0.02$) (Table 2). Although there was no

Table 1
Participant demographics.

Variables	Control Group ($n = 11$) Mean (***SD***)	ASD Group ($n = 11$) Mean (***SD***)	*p* value
Age (year)	13 (2)	13 (2)	0.72
Body Mass (kg)	58.55 (11.40)	56.93 (24.01)	0.84
Height (m)	1.66 (0.10)	1.59 (0.16)	0.23
BMI (kg/m^2)	21.06 (2.44)	21.51 (4.19)	0.71

Note: ASD = Autism Spectrum Disorder; BMI = Body Mass Index; *SD* = standard deviation.

Table 2
Temporo-spatial parameters of the ASD group and the control group.

Temporo-spatial Parameters	Control Group Mean (***SD***)	ASD Group Mean (***SD***)	Cohen's *d*	*p* value
Stride length (m)	1.26 (0.06)	1.18 (0.11)	0.73	0.04*
Stride width (m)	0.12 (0.02)	0.11 (0.04)	0.21	0.50
Cadence (steps per minute)	113 (4)	121 (8)	0.76	0.03*
Percentage of double limb support (%)	27.89 (1.53)	26.53 (3.84)	0.37	0.24
Coefficient of variation in stride width (%)	17.51 (7.08)	23.05 (10.98)	0.71	0.04*

Note: ASD = Autism Spectrum Disorder; *SD* = standard deviation; *$p < 0.05$.

significant difference in stride width between the two groups ($p = 0.33$), the individuals with ASD demonstrated a significantly greater (32% more) *Co-var* of stride width than the control group ($p = 0.04$). Furthermore, the GRF profile was found different between the two groups (Table 3). It was observed that the individuals with ASD experienced a significantly greater AP braking GRF (14% larger) than the control group ($p = 0.03$). Though it was not statistically significant ($p = 0.07$), the individuals with ASD also tended to apply a smaller vertical GRF (4 % less) than the controls during the push-off phase with a medium effect size. Individuals with ASD exhibited a significantly smaller knee extensor torque (15% less) than the control group ($p = 0.000\ 7$) (Table 4). There was no significant difference in knee flexor torque between groups ($p = 0.11$).

4. Discussion

The primary purpose of this study was to examine characteristics of gait parameters of older children with ASD in comparison to age- and gender-matched controls. The secondary purpose of the study was to examine lower-extremity strength in individuals with ASD, represented by knee muscle torques. As hypothesized, the results of the study indicated that the participants with ASD demonstrated significant deficits in knee strength and significant differences in walking patterns compared to their age-and gender-matched typically developing controls.

Table 3
Normalized peak GRFs of the ASD group and the control group.

Normalized Peak GRFs	Control Group Mean (***SD***)	ASD Group Mean (***SD***)	Cohen's *d*	*p* value
Vertical GRF in the first half of stance (BW)	1.11 (0.07)	1.10 (0.24)	0.045	0.88
Vertical GRF in the second half of stance (BW)	1.04 (0.06)	1.00 (0.07)	0.58	0.07
Antero-posterior braking GRF (BW)	0.14 (0.02)	0.16 (0.04)	0.23	0.03*
Antero-posterior propulsive GRF (BW)	0.15 (0.02)	0.16 (0.03)	0.71	0.45
Medio-lateral GRF (BW)	0.01 (0.08)	0.01 (0.08)	0.36	0.25

Note: GRF = ground reaction force; ASD = Autism Spectrum Disorder; BW = body weight; *SD* = standard deviation; *$p < 0.05$.

Table 4
Normalized knee strength of the ASD group and the control group.

Normalized Knee Strength	Control Group Mean (***SD***)	ASD Group Mean (***SD***)	Cohen's *d*	*p* value
Extensor torque ($N{\cdot}m{\cdot}kg^{-1}$)	2.92 (0.52)	2.49 (0.67)	1.10	0.0007*
Flexor torque ($N{\cdot}m{\cdot}kg^{-1}$)	1.50 (0.42)	1.30 (0.48)	0.50	0.11

Note: ASD = Autism Spectrum Disorder; *SD* = standard deviation; *$p < 0.05$.

In this study, the ASD group exerted 15% less knee extensor torque than their typically developing controls. Although not tested, other lower extremity extensors controlling the ankle and hip joints may also possess weaknesses within the individuals with ASD. Weaknesses in lower extremity extensor muscles could limit the ability of the individuals with ASD to counteract forces when performing weight bearing physical activities such as normal walking. The mechanism of developing muscle strength deficit is not clear. It is possible that the delayed motor development commonly shown in ASD children (8 out of 11 children confirmed in this study) could play a critical role affecting the strength development of the muscles. It is also possible that a combination of the motor deficits and insufficient level of physical activities due to ASD conditions could undermine the muscle strength. Future studies are warranted to investigate the impact of motor deficits and physical activity level on ASD individuals' physical conditions including muscle strength.

Significant differences in temporo-spatial parameters and ground reaction forces were observed between the individuals with ASD and their controls. The individuals with ASD walked in shorter strides with a higher cadence than the controls. Employing shorter strides reduces the need for large forces from leg extensor muscles to advance the body forward at a given speed. The peak vertical GRF during the push-off phase was found to be reduced in the group with ASD. Although this reduction was not statistically significant ($p = 0.07$), a moderate effect size (0.6) was observed. Considering the significant strength deficit in knee extensors of the individuals with ASD, it is suggested that the shorter stride length observed was adopted as a compensatory movement strategy.

At a set speed, it is expected that smaller steps would result in increased cadence. In this study, the individuals with ASD exhibited an 8% increase in cadence compared to their controls. Although it is suggested that this strategy is adopted to compensate for deficits in muscular strength, this increase in demand for cadence could place more stress on lower extremity musculature. As walking distance increases, individuals with ASD may be susceptible to accelerated lower extremity muscular fatigue. Furthermore, the individuals with ASD in this study demonstrated a 14% larger braking AP GRF during loading response than that of their controls. Elevated braking forces applied at the heel during foot strike would force the foot to be rapidly dropped to the ground. In order to avoid slapping the foot on the ground, ankle dorsi-flexors must work harder to control the lowering motion of the foot at foot strike. This increased demand on ankle dorsi-flexors could stress and fatigue these muscles. Additionally, if this compensation pattern response is not adequately adapted in the ankle dorsi-flexors, it could increase stress further up the kinetic chain at the knee joint.

This study also examined the level of variation in stride width. Increasing stride width improves stability and balance, while decreasing stride width helps improve efficiency of the walking movement. When walking at a constant speed, participants in this study were expected to maintain a preferred stride width for optimal stability and efficiency. However, the participants with ASD showed inconsistency in stride width and frequently adjusted their stride width while walking. Specifically, the coefficient of variation of the individuals with ASD was 32% greater than that of their controls. Varying stride width seems to be a strategy employed by the individuals with ASD to balance between maintaining

dynamic stability while optimizing walking efficiency. Weaknesses in knee extensor muscles exhibited in the participants with ASD along with potentially poorer lower-limb coordination may contribute to this variability.

Due to the neurodevelopmental nature of ASD, previous research has largely focused on understanding gait in younger children with ASD.[12] However, previous findings were inconsistent. For example, some studies showed that younger children with ASD demonstrated reduced stride length when they walked,[12,13] while other studies reported no difference in stride length between children with ASD and typically developing children.[15,16] Walking cadence in children with ASD was found to be faster or similar to typically developing peers.[7,12,15,17] After reviewing over 20 years of research in ASD gait, Lum and coauthors recognized the inconsistent findings regarding ASD individuals' gait profiles.[26] But they reported that in general, shorter step length, increased cadence, and wider step width were common among ASD individuals.[26] Our findings largely agree with the gait patterns mentioned. However, the existing inconsistent findings strengthen the theory that the effect of ASD on gait development is highly complex. Individuals with ASD are certainly affected by motor deficits during the formation of mature gait patterns, but the extent of these effects varies. Ultimately, gait development could be delayed in individuals with ASD compared to their typically developing peers.

In addition to alterations in temporo-spatial parameters, ASD individuals were found to display age related changes in joint kinematics.[27] Older children with ASD could achieve more energy efficient gait kinematics by showing less ankle dorsiflexion and knee flexion at heel strike and greater plantar-flexion at toe-off than those of younger children.[27] The current study does not emphasize on examining ASD individuals' joint kinematics, future studies could be expanded to determine whether there are age-related changes in lower-extremity joint kinematics exhibited by older children and adolescents with ASD in comparison to age- and gender-matched healthy individuals.

Delayed or altered gait development could have significant indications for the ability of individuals with ASD to live a healthy life. Not only can it negatively affect participation in physical activity during youth, but this trend is also likely to continue into adulthood. The implications of a sedentary lifestyle on comorbidities, such as obesity and heart disease, stress the importance of continued gait development even after early childhood. It is also suggested that deficits in motor development should be considered as a standard diagnostic tool, along with cognitive and psychosocial evaluations.

The intention of this study was to quantify fully developed gait patterns of older children with ASD and the quality of their knee muscular strength. Individuals with ASD possessed unique gait patterns signified by reduced stride length, increased cadence, greater variation of stride width, and higher braking forces when compared to their typically developing peers. They also demonstrated a tendency towards a decreased vertical GRF at push-off with borderline significance and a medium effect size. In addition, individuals with ASD displayed physical deficits, displayed by weakness in their knee extensors. Alterations in gait parameters seen in the participants with ASD are likely compensatory movement patterns developed to account for the strength deficit in their lower extremity extensors.

Individuals with ASD may be exposed to increased risk of fall-related injuries. At any given speed, an increased cadence combined with high braking forces could lead to overuse of lower extremity muscles. Muscular fatigue is likely to be aggravated in these individuals as walking distance increases. Lower extremity muscular fatigue combined with high variation in stride width may reduce gait stability and elevate fall risk. Risks related to muscular and coordination deficits may be exacerbated as movement difficulty increases (e.g., running, stepping stairs, carrying school bags). Biomechanical gait analysis to identify movement deficits is necessary to develop adequate exercise intervention programs. Focusing on strengthening lower extremity muscles and improving dynamic stability may be beneficial and ultimately decrease risks of fall-related injuries in individuals with ASD.

In this study, we did not assess participants' sensory profile and do not know the extent of ASD individuals' sensory impairments. It is possible that severe sensory impairments could have an impact on ASD individuals' gait patterns.[28] Future studies could focus on understanding the effect of sensory impairment on ASD individuals' gait and explaining how and why sensory impairments affect walking. Another potential limitation of this study was the inability to avoid medication interference. Nine of the eleven ASD participants took medications for their ASD treatments. Some medications may influence ASD individuals' gross motor behaviors. The effect of the medications on gait performance could not be controlled for in this study. Future studies may focus on gait characteristics of ASD participants without the influence of medications.

5. Conclusions

Older children and adolescents with ASD examined in this study showed unique gait patterns characterized by a short stride length, fast cadence, and high variation of stride width. These ASD individuals also experienced high braking forces during walking. Knee extensor weakness may have led to the observed compensatory movement strategies. Individuals with ASD living with these unique gait deviations may face increased risk of fall-related injuries. Understanding gait pattern deviations in individuals with ASD could help practitioners design appropriate intervention programs to improve physical ability and gait quality, and ultimately decrease the potential risk of fall-related injuries.

Submission statement

All authors confirm that this work has not been previously published, nor is it currently under consideration for publication elsewhere. All authors approve this work for publication and if accepted, it will not be published elsewhere without written consent of the copyright-holder.

Ethical approval statement

Approval from the university's Institutional Review Board (IRB) was obtained prior to the commencement of the study [IRB#1243649]. Signed informed consent documents were obtained from participants' parents and assent was obtained from participants.

Authors' contributions

Xinye Wu: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Writing – original draft. **D. Clark Dickin:** Conceptualization, Data curation, Methodology, Writing – review & editing. **Laura Bassette:** Conceptualization, Data curation, Investigation, Methodology, Writing – review & editing. **Caroline Ashton:** Formal analysis, Writing – original draft, Writing – review & editing. **He Wang:** Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Supervision, Writing – original draft, Writing – review & editing.

Conflict of interest

All authors declare no conflict of interest.

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