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Children with Heavy Prenatal Alcohol Exposure Exhibit Atypical Gait Characteristics

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Abstract

BACKGROUND—Impaired motor function in children with histories of prenatal exposure to alcohol has been previously reported but, to date, no studies using quantitatively-based analyses have been performed to assess gait in these children.

METHODS—Gait of children with ($n = 18$) or without ($n = 26$) prenatal alcohol exposure was assessed using an electronically instrumented walkway. Children completed blocks of trials traversing the walkway with different combinations of walking condition (increased, self-paced, and decreased cadence) and direction (forward and backward). Gait velocity, cadence, stride length, step width, foot angle, and double support time, as well as the variability of these temporal-spatial markers, were used to assess gait.

RESULTS—Results indicated that, in comparison to typically developing children, alcohol-exposed children produced exaggerated foot angle and increased step width. Additionally, alcohol-exposed children produced greater intra-subject variability of gait velocity and walking cadence while walking forward and backward, and greater variability in step width when walking backward and for all three walking conditions.

CONCLUSIONS—The results indicate that selected gait markers are adversely affected by prenatal exposure to alcohol. Clinicians and front line personnel (e.g., teachers) should provide movement enriched experiences to help ameliorate these alcohol-related deficits.

Keywords

Fetal alcohol spectrum disorder; gait; GAITrite®; teratogenesis

INTRODUCTION

Fetal Alcohol Spectrum Disorders (FASD) is a non-diagnostic umbrella term conceptualized as a continuum of developmental effects stemming from prenatal alcohol exposure. At the extreme end of the continuum is Fetal Alcohol Syndrome (FAS), which was clinically defined in 1973 by the presence of central nervous system (CNS) damage or dysfunction, pre- and/or post-natal growth retardation, and distinct dysmorphic facial anomalies (Jones and Smith, 1973). Estimates of the prevalence of FAS vary from 2 to 9 children per 1,000 live births in a mid-western American city (May et al., 2014), to 4 to 12 per 1,000 children in Italy (May et al., 2011), to 136 to 209 per 1,000 children in South Africa (May et al., 2013) and 120 per 1,000 in Western Australia (Fitzpatrick et al., 2015). However, these estimates do not include children without the facial dysmorphology associated with FAS (Viljoen et al., 2005). When dysmorphic and non-dysmorphic children are considered together, prevalence estimates increase considerably (Roozen et al., 2016), as do the economic and social costs associated with FASD (Easton et al., 2015).

Alcohol-induced insult to the CNS of both dysmorphic and non-dysmorphic alcohol-exposed children can be expressed as volumetric reductions in the temporal, frontal, and parietal lobes as well as the total brain; the corpus callosum may be reduced or subject to partial or complete agenesis; the cerebellum, hippocampus, basal ganglia and thalamic structures can be undersized, and grey and white matter volume is disproportionately reduced relative to total brain volume (Glass et al., 2014). Prenatal alcohol exposure is also associated with slower nerve conduction velocity (Avaria et al., 2004), atypical neuromuscular innervation and muscle development (David and Subramaniam, 2005) and fewer and smaller motor neurons.

The combined effect of these compromised neural and neuromuscular systems may result in motor skill dysfunction in children with prenatal alcohol exposure, which can have significant long-term practical ramifications. For example, gait is an essential component of many activities of daily living, and an inability to appropriately initiate and regulate this motor response can lead to decreased skill performance that in turn, erodes a child's self-confidence, self-esteem, and self-efficacy (Schoemaker & Kalverboer, 1994).

While our current understanding of gait control in young alcohol-exposed children is based on a limited number of studies, motor anomalies can be seen as early as infancy. One series of investigations assessed maturational delays in alcohol-exposed infants using the Psychomotor Development Index (PDI) of the Bayley's Scales of Infant Development. The PDI comprises a subset of posture and locomotor tests that are rater-scored and converted into a composite standardized score. Results from these studies indicate prenatal alcohol exposure results in a lower PDI (i.e., reduced gait control) (Coles et al., 2015; Golden et al., 1982; Jacobson et al., 1993). In contrast to these studies, other investigations using the PDI failed to detect any differences between the gait of alcohol-exposed and typically developing infants (Chandler et al., 1996; Forrest and Florey, 1991; Fried and Watkinson, 1988; Fried and Watkinson, 1990; Fried et al., 1992; Gusella and Fried, 1984). The reasons for the equivocal results are unclear but could be related to the use of small sample sizes,

inconsistent application of scoring criteria across research projects and different levels of prenatal alcohol exposure.

Information about gait control in prenatally alcohol-exposed children of school age is based on anecdotal evidence and a single investigation that also used a standardized score (the dynamic gait index: DGI) as a metric of walking. Olegård et al., (1979) reported clumsy and uncoordinated gross motor movements in alcohol-exposed children; Marcus (1987) noted that children with prenatal alcohol exposure experience difficulty with tandem walking (i.e., the toes of the back foot touch the heel of the front foot at each step), have clumsy gait, and adopted a wide-based support when walking. Similar to the PDI, the dynamic gait index is calculated from several locomotor tasks that are rater-scored and converted into a standardized index of performance. Children prenatally exposed to alcohol produced a lower DGI value than typically developing children (Lubetzky-Vilnai et al., 2011).

Anecdotal evidence is open to subjective bias and while the DGI is sufficiently sensitive to detect reduced gait control in alcohol-exposed children, the index represents a composite score generalized across several gait tasks that could mask anomalies in specific gait characteristics (e.g. stride length, gait velocity). To date, this type of gait analysis has not been completed with children with histories of prenatal alcohol exposure. Therefore, this study investigated the gait of children and adolescents with histories of *in utero* alcohol exposure using multiple quantitatively based spatial and temporal markers of locomotion.

Six markers are consistently used when assessing gait of asymptomatic and symptomatic children (Lythgo et al., 2011; Weiss et al., 2013). The markers are: gait velocity (cm/s), step cadence (steps/min), stride length (cm) (i.e., the distance between two successive placements of the same foot), foot angle (deg) (i.e., the angle of the foot measured relative to the line of the direction of travel), step width (cm) (i.e., the distance between right and left heel placements on successive steps), and double support time (ms) (i.e., during walking the time both feet are in contact with the ground). The present study used these markers to assess gait in children with or without histories of heavy prenatal alcohol exposure while walking with different combinations of stepping rate (i.e., self-paced cadence, increased cadence, and decreased cadence) and direction (i.e., forward or backward). Inconsistency of motor responses in alcohol-exposed children is a frequently reported characteristic (Simmons et al., 2009) and, therefore, variability (i.e., within-subject standard deviation across 5 trials for each selected gait marker) was also computed and statistically analyzed to evaluate whether excessive variability of gait is associated with the clinical group.

Using multiple markers to index gait performance is commonly reported in the gait literature and it is not unusual that markers are selectively affected by the clinical condition under investigation (Hackney and Earhart, 2010; Pickett et al., 2012). The advantage of this approach lies in the ability to identify which spatial and/or temporal characteristics of walking deviate from control group values, thus allowing rehabilitation therapists, clinicians, and practitioners to design therapies and rehabilitation programs that specifically target the identified deficits in gait.

Additionally, there are neural correlates for some of the gait markers. The supplementary motor area (de Laat et al., 2012), cerebellum (Rao and Louis, 2016) and prefrontal cortex (Burhan et al., 2015) are implicated in gait cadence, whereas gait velocity is related to altered activity in the cerebellum (Matsushima et al., 2015), basal ganglia (Chastan, et al., 2009), corpus callosum (Ryberg et al., 2011), prefrontal cortex (Burhan et al., 2015) and motor cortex (Annweiler et al., 2013). Step length involves basal ganglia structures (Chastan et al., 2009) and step length variability is related to hippocampal involvement (Shimada et al., 2013). Step width is dependent on hippocampal (Shimada et al., 2013), callosal (Fling et al., 2016), and inferior parietal lobe (de Laat et al., 2012) engagement. Given that the structural integrity of many of these neural systems can be compromised by gestational exposure to alcohol, it is predicted that gait of alcohol-exposed children will be significantly different, as indexed by specific gait markers and variability of these markers, from that produced by typically developing children.

MATERIALS AND METHODS

Participants

Prenatal Alcohol Exposure (PAE group)—Children aged 7–17 years old with histories of heavy prenatal alcohol exposure ($n = 18$) were recruited from the Center for Behavioral Teratology (CBT) at San Diego State University. Alcohol exposure history was confirmed using available social service, adoption agency records, legal and medical records, and responses to questionnaires completed by the legal guardian or biological mother. Due to the retrospective nature of the study, the precise timing and amount of maternal alcohol consumption were unknown, but for the sample included in this study, heavy alcohol exposure was defined as more than 4 alcohol drinks/occasion at least once/week or greater than 14 drinks on average/week. Additionally, children in the PAE group were evaluated by a dysmorphologist with expertise in ethanol teratogenesis using criteria defined by the Dysmorphology Core of the Collaborative Initiative on Fetal Alcohol Spectrum Disorders (CIFASD), which included physical growth deficiencies (height or weight < 10 percentile), defining facial features (the presence of two of the following: short palpebral fissures, smooth philtrum, thin vermilion) and microcephaly (head circumference < 10 th percentile) or deficient structural brain growth (Mattson et al., 2010). Three participants met criteria for FAS and the remaining 15 participants were classified as non-dysmorphic alcohol-exposed children.

Control (CON group)—CON children ($n = 26$) with minimal or no prenatal alcohol exposure were recruited through referral (i.e., word of mouth) to the CBT, electronic and non-electronic public notices, and through community events such as health fairs. Minimal prenatal exposure to alcohol was defined as maternal consumption of ≤ 1 alcohol drinks per week on average and never more than 2 drinks on any 1 occasion. *In utero* alcohol exposure was determined via direct report from the biological mother.

General Procedures—Prior to gait testing, all children were assessed using different scales of performance including full-scale IQ (FSIQ), as determined by the Wechsler Intelligence Scale for Children – Third (Wechsler, 1991) or Fourth Editions (Wechsler,

2003) or the Differential Ability Scales- General Conceptual Ability (DAS-GCA), language, learning, memory, visual-spatial and visual-motor ability, academic performance, and executive function (Mattson et al., 2011). Exclusionary criteria for all participants were: any language other than English as a primary language; FSIQ < 70; and below 7 or above 17 years of age. Children in the PAE and CON groups were matched as closely as possible on age and socio-economic status. Demographic information for the PAE and CON groups is presented in Table 1.

Apparatus and Methods

Written legal guardian consent and participant assent was obtained in accordance with the ethical standards and approval of the San Diego State University Institutional Review Board. Gait markers were assessed using a Platinum GAITRite Walkway system (CIR Systems Inc., Sparta, NJ) located in a quiet testing area. The walkway consisted of a carpet (90 cm × 7 m × 3.2 mm), embedded with 13,824 sensors that provided spatial and temporal resolution of 1.27 cm and 8 ms, respectively. Data were recorded in real time at a sampling rate of 80 Hz. The walkway was connected to a laptop computer running MS Windows XP by a serial interface cable.

Participants completed a total of six blocks of walking with each block consisting of seven trials. The inter-trial interval was approximately 20 s. The first two trials in each block were considered practice trials and were not included in any statistical analyses. The first block of trials involved walking forward along the length of the walkway at a self-paced cadence. This testing was followed by two blocks of trials walking forward in synchrony with a metronome signal set at 20% higher (increased walking cadence) and lower (decreased walking cadence) than the average self-paced cadence. The order of the increased and decreased conditions was counterbalanced across participants. Participants repeated this sequence of testing while walking backward. Children wore regular shoes for all test conditions.

To reduce acceleration and deceleration effects during each trial, children began walking from a strip of blue tape positioned 1m from the front edge of the walkway and continued walking until passing a second strip of tape located 1m from the back edge of the walkway. Additionally, children were encouraged to establish an increased and decreased walking cadence by stepping in place synchronously with the metronome until the appropriate rhythm was established, at which point they were free to begin walking.

Assessment of gait was completed as part of a larger study in which participants also performed a drawing task on the same day. Approximately 30 minutes was required for gait assessment and 20 minutes for the drawing task. The two tasks require different motor skills, but to eliminate any potential for practice or fatigue effects, the order of completing the two tasks was counterbalanced across participants and an inter-task break of approximately 10 minutes was provided. At the conclusion of testing, each child was given a small monetary reward.

Data Analysis

Participant demographic information of age, height, FSIQ, and socioeconomic status (SES) were independently assessed using one-way analysis of variance (ANOVA).

For each trial of walking, the six gait markers were recorded and analyzed. Data were averaged across trials within each trial block and examined using a 2 (Group) $\times$ 2 (Walking Direction) $\times$ 3 (Walking Condition) repeated measures analysis of covariance (ANCOVA), with age as a covariate. Group (PAE and CON) was a between-subjects variable, Walking Direction (forward and backward) and Walking Condition (self-paced cadence, increased cadence and decreased cadence) were within-subjects variables. Significant interactions of two or more variables were further analyzed using univariate ANOVAs, and Bonferroni corrections were applied for post-hoc comparisons. Assumptions of sphericity and homogeneity of variance were examined using Mauchly's and Levene's test, respectively. Violations of sphericity prompted the use of the Greenhouse-Geisser conservative estimate of degrees of freedom, and violations of homogeneity of variance were addressed by using Games-Howell procedures for post hoc comparisons. FSIQ was not included as a covariate in the analyses given the inappropriateness of covarying a variable that is an inherent characteristic of a clinical condition (Dennis et al., 2009) and the potential removal of group-specific variance that would invalidate between group comparisons of gait performance (Miller & Chapman, 2001). Data were analyzed using SPSS version 22.0 (SPSS Inc., Chicago).

RESULTS

Demographic Data

Contrasts between group age, height and SES were not significant ($p > .05$). Analysis of FSIQ revealed a significantly lower FSIQ for the PAE group ($p < .0001$), which is consistent with reports of impaired intellectual functioning in children with prenatal alcohol exposure.

Gait Dependent Variable Data

Group mean values (standard error in parentheses) and variability (indexed by the standard deviation) of each dependent variable as a function of walking direction and walking condition are presented in Tables 2 and 3, respectively.

Gait velocity—With data collapsed across walking direction and condition, there was no significant difference between group means (PAE = 99.75 cm/s; CON = 99.79 cm/s) for gait velocity $F(1, 41) = .001$, $p = .991$, $\eta^2 = .01$). A main effect of Walking Direction, $F(1, 41) = 13.18$, $p = .001$, $\eta^2 = .24$, indicated both groups walked forward with a greater velocity (mean = 119.4 cm/s) than walking backward (mean = 80.3 cm/s). Additionally, a significant effect of Walking Condition, $F(1.67, 68.68) = 10.71$, $p = .0001$, $\eta^2 = .21$, revealed that when collapsed across groups, mean velocity was 80.3, 99.6 and 119.4 cm/s for decreased, self-paced and increased cadence walking, respectively. Differences between these mean values were significant ($p < .05$).

Analysis of gait velocity variability indicated a significant main effect of Group, $F(1, 41) = 12.5$, $p = .001$, $\eta^2 = .23$, Walking Direction, $F(1, 41) = 16.7$, $p = .001$, $\eta^2 = .29$, and Walking Condition, $F(1.95, 80.1) = 5.0$, $p = .009$, $\eta^2 = .10$, together with a significant Group $\times$ Walking Condition interaction, $F(1.95, 80.1) = 6.21$, $p = .003$, $\eta^2 = .13$. For both directions of walking, the PAE group produced significantly greater variability in gait velocity than the CON group and both groups had significantly less variability in gait velocity when walking backward compared to gait variability while walking forward. Analysis of the Group $\times$ Walking Condition interaction revealed that the PAE children produced greater walking variability during the increased cadence condition compared to CON children, but not when walking with decreased or self-paced cadence. Mean variability of gait did not change for CON children across cadence conditions ($p > .05$), but the PAE group produced significantly greater gait variability during the increased cadence walking condition ($p = .04$) compared to the decreased cadence walking condition.

Cadence—A main effect of Walking Direction, $F(1, 41) = 5.56$, $p = .02$, $\eta^2 = .12$, revealed both groups produced a significantly greater stepping cadence when walking forward (forward vs. backward = 115.6 vs. 107.7 steps/minute). A main effect of Walking Condition, $F(1.43, 58.7) = 87.6$, $p = .0001$, $\eta^2 = .68$, and a Group $\times$ Walking Condition interaction, $F(1.43, 58.7) = 4.3$, $p = .03$, $\eta^2 = .10$ were also found. However, follow-up analyses revealed no significant difference in stepping rate between the two groups when walking with decreased, self-paced, or increased cadence.

Analysis of cadence variability produced a main effect of Group, $F(1, 41) = 6.13$, $p = .02$, $\eta^2 = .13$, and Walking Condition, $F(1.89, 77.7) = 4.67$, $p = .02$, $\eta^2 = .10$. The PAE group produced significantly greater cadence variability across all walking conditions and directions when compared to the CON group. With data collapsed across groups, cadence variability was 3.2, 4.5 and 3.9 steps/minute for the decreased cadence, self-paced cadence and increased cadence conditions, respectively. All differences between mean values were significant ($p < .05$) with the exception of the comparison between self-paced and increased cadence walking.

Stride length—There were no significant main effects or interactions for stride length ($p > .05$). Analysis of stride length variability revealed a significant main effect of Walking Condition, $F(1.9, 81.5) = 4.02$, $p = .02$, $\eta^2 = .09$, and a Group $\times$ Walking Condition interaction, $F(1.9, 81.5) = 3.42$, $p = .04$, $\eta^2 = .08$. Pairwise analysis of the interaction revealed that stride length was significantly more variable for the PAE group when walking with an increased cadence. There were no group differences during decreased cadence or self-paced walking ($p > .05$). Stride length variability did not significantly change for either group across walking condition ($p > .05$).

Foot angle—A significant main effect of Group, $F(1, 41) = 6.37$, $p = .02$, $\eta^2 = .13$, indicated that for both walking directions and three walking conditions the PAE group produced a positive foot angle (i.e., foot ‘turned out’) (5.3 deg), whereas the CON group averaged a relatively neutral foot angle (i.e., foot angle approached zero) (0.2 deg). There were no significant main effects or interactions for foot angle variability.

Step width—A significant main effect of Group, $F(1, 41) = 4.40, p = .04, \eta^2 = .10$, revealed that for the decreased, self-paced and increased cadence conditions, the PAE group produced significantly greater step width than the CON group. A Group $\times$ Walking Direction interaction, $F(1, 41) = 6.94, p = .01, \eta^2 = .15$, was also found. Follow-up analysis of the interaction indicated similar step width for both groups when walking forward ($p > .05$) and increased step width in both groups when walking backward compared to walking forward ($p = .0001$). However, when walking backward step width was significantly greater in the PAE group compared to the CON group ($p = .005$).

Analysis of step width variability revealed a significant main effect of Group, $F(1, 41) = 7.16, p = .01, \eta^2 = .15$, with the PAE group producing significantly greater step width variability during decreased, self-paced and increased cadence than the CON group. A significant main effect of Walking Direction, $F(1, 41) = 5.46, p = .03, \eta^2 = .12$, and a Group $\times$ Walking Direction interaction was also detected, $F(1, 41) = 4.50, p = .04, \eta^2 = .10$. The interaction resulted from both groups producing similar step width variability when walking forward ($p > .05$), but the PAE group produced significantly greater step width variability walking backward ($p = .004$).

Double support time—A main effect of Walking Direction, $F(1, 41) = 8.56, p = .005, \eta^2 = .18$, revealed that, regardless of group assignment, children spent more time in the double support phase when walking backward ($p = .01$). A significant main effect of Walking Condition, $F(1.67, 68.6) = 3.28, p = .05, \eta^2 = .07$, and a Group $\times$ Walking Condition interaction, $F(1.64, 68.6) = 7.2, p = .003, \eta^2 = .12$, were indicated. Post hoc analyses indicated no differences between the two groups for any of the three walking conditions, although double support time significantly decreased when walking condition changed from decreased to self-paced to increased cadence.

Analysis of double support time variability revealed a single Group $\times$ Walking Condition interaction, $F(1.69, 69.4) = 4.54, p = .02, \eta^2 = .10$. There was no difference in double support time for the two groups during decreased cadence and increased cadence (p values $> .05$). However, for self-paced cadence, the PAE produced significantly greater double support time variability ($p = .04$). Pairwise comparisons between each walking condition revealed that, for the PAE group, variability during the decreased and self-paced cadence conditions was significantly greater than during the increased cadence condition ($p < .05$). Double support time variability for the CON group significantly increased as cadence changed from decreased to self-paced to increased pace (p 's $< .05$).

DISCUSSION

The present study indicated selected differences in the gait of children with and without prenatal alcohol exposure. One result revealed a significant difference between the PAE and CON groups with regard to the angle of the foot while walking forward and backward. Foot angle associated with typically developing children remained neutral (i.e., foot pointing in the same direction as the line of travel) regardless of walking direction, whereas alcohol-exposed children produced a significantly greater foot angle (feet 'turned out'). Foot angle for the PAE and CON groups was unaffected by walking cadence. The accentuated foot

angle for alcohol-exposed children partially corroborates the splayed foot position observed during forward walking in alcohol-exposed rats (Hannigan and Riley, 1988) and in rats subjected to a binge drinking condition at a developmental age corresponding to human adolescence (Forbes et al., 2013). Accentuated foot angle in the gait of typically developing children is mainly attributed to abnormal twisting of the femur (thigh bone), the tibia (shin bone) or curvature of the foot, all of which typically resolve within the first decade of life. These pathologies cannot be discounted in the present study with regard to the PAE group, but Forbes and colleagues provide some evidence that atypical foot angle is associated with alcohol-induced neurodegeneration of the cerebellum. However, details of the mechanism underpinning this cerebellar-foot angle link in relation to FASD have not been identified to date and the functional significance of atypical foot angle remains unclear for all children.

Another result indicated that gait of PAE children was marked by significantly greater step width during backward walking and for all three conditions of walking cadence. There are two possible explanations for this difference in step width that are not mutually exclusive. First, increased step width is associated with reduced volume of the pallidum, which is involved in maintaining postural stability, and parietal cortex, which coordinates movement within the context of the surrounding environment (Rosano et al., 2006). Both structures can experience atrophy in response to gestational alcohol exposure (Nardelli et al., 2011; Woods et al., 2015), and consequently, a wider stepping action is a likely outcome for this clinical group. Second, our results indicated that PAE children experienced deficits in selected gait parameters, which may reflect reduced postural control relative to CON children. Therefore, as a means of compensating for this locomotor instability, PAE children increase step width, which has the biomechanical effect of increasing the area that defines the base of support. A similar increase in stability could result from accentuating foot angle, which was also observed in the PAE children.

A third result of the present study revealed increased variability for the PAE group for some gait markers. Specifically, for all cadence conditions and for both walking directions, cadence variability was significantly higher in the PAE group and step width variability was greater for the three cadence conditions and when walking backward. These results align with previous studies reporting increased motor variability for PAE children during force regulation and motor timing responses (Nguyen et al., 2013; Simmons et al., 2009). Furthermore, these findings have serious practical implications. For instance, step width variability is associated with falls and postural instability (Brach, Berlin, VanSwearingen, Newman, & Studenski, 2005), which stems from cerebellar damage, often a result of prenatal alcohol exposure (Glass et al., 2014). The source of motor variability is open to debate, but at least one model of motor control proposes that some variability is necessary to facilitate adaptation to changes in the local external environment (Smits-Engelsman & Wilson, 2013). However, the same model also proposes that excessive motor variability distorts the temporal and spatial characteristics of a response, thereby leading to impaired execution of daily activities. In the case of a prenatally alcohol-exposed child, the observed excessive variability in cadence and step width could have the practical effect of reducing coordination and negatively impacting skill performance in games and activities that require precision in gait (e.g., running and kicking a ball, climbing steps), and thus would be an important target for intervention.

An exception to demonstrating increased variability was seen in gait velocity. Counter-intuitively, both groups produced less variability in gait velocity when walking backward in comparison to walking forward, which can occur independent of changes to other gait markers, such as cadence variability (Laufer, 2005). More consistent gait velocity when walking backward may be related to the amount of visual feedback information available to the child during walking. During forward walking, visual information is readily available and used to make on-line modifications to walking speed, thereby increasing step-to-step variability. In contrast, during backward walking, children were instructed not to turn their heads in the direction of travel, which limited visual input. For this situation, children appear to exhibit stereotypical steps executed with minimal visually directed corrections resulting in a relatively constant velocity.

Research testing the efficacy of rehabilitation therapies designed to ameliorate gait deficits in children with prenatal alcohol exposure has yet to be fully delineated. Consequently, the work of therapists, clinicians, and research scientists who work with this clinical group might be guided by gait interventions that have proven effective with other clinical syndromes. For example, Seo and Park (2016) demonstrated that step width decreased in individuals with lower back pain following completion of an exercise regimen designed to increase range of motion and functional strength through rhythmic, flowing movement sequences. Given the readily available contemporary technology, it would be comparatively easy to determine if a similarly designed exercise program would reduce step width in alcohol-exposed children. In a similar vein, Im and colleagues (2016) found that gait variability decreased in patients with cerebellar ataxia as a result of engaging in a sustained program of locomotor training centered on establishing proper gait mechanics. This outcome suggests that for cerebellar patients at least, exercise induces plastic changes in the cerebellum similar to the morphological changes in cerebellar tissue of alcohol-exposed rats completing a program of complex motor training (Klintsova et al., 2002). Clearly, additional work is required to confirm whether specific therapies remediate atypical gait characteristics in children prenatally exposed to alcohol and to what extent the functional capacity and/or structural integrity of neural correlates is impacted by exposure to these therapies.

The study has some potential limitations. First, the relatively small number of participants may adversely affect statistical power. However, given the significant findings for step width, foot angle, cadence variability and step width variability, we are confident that our statistical procedures reliably indicate differences in gait performance for the two groups. Second, the comparatively large age range of participants and the maturation of gait with developmental age may violate the statistical assumption of group homogeneity. However, in a comprehensive gait analysis study of a large cohort of children, Sutherland et al., (1988) reported that a mature gait pattern, as indexed by markers of walking velocity, cadence, step length and double support time, is well established by three years of age. This age is well below the lowest age of seven years for our youngest participants.

In summary, the results indicate that selected markers of gait performance are differentially affected by prenatal alcohol exposure. Step width and foot angle were accentuated and cadence variability and step width variability were significantly increased in the PAE group. These selective differences in gait are consistent with motor performance deficits reported

for these children when performing a range of motor tasks and should be targeted via appropriate interventions.

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Table 1

Demographic information for the PAE and CON groups.

	PAE	CON	
Sex [M:F (M:F%)]	8:10 (44:56)	18:8 (69:31)	
Age (years) [M (SD)]	10.9 (3.1)	11.8 (3.4)	$p > .05$
Height (cm) [M (SD)]	138.2 (39.4)	146.4 (19.4)	$p > .05$
FSIQ ¹ [M (SD)]	84.3 (13.1)	112.1 (15.4)	$p < .001$
SES ² [M (SD)]	44.3 (16.1)	49.2 (10.4)	$p > .05$

¹ Intelligence scores were derived from either the Wechsler Intelligence Scale for Children-III, the Wechsler Intelligence Scale for Children-IV or the Differential Ability Scales- General Conceptual Ability (DAS-GCA) depending on the time the child enrolled at the CBT.

² Socioeconomic status (SES) was estimated using the Hollingshead Four Factor Index of Social Status (Hollingshead 1975, unpublished data)

Table 2

Gait parameter means (standard error) for the PAE and CON groups when walking forward and backward. Data are collapsed across walking conditions to facilitate group comparisons.

	Backward Walking		Forward Walking	
	PAE	CON	PAE	CON
Velocity (cm/s)	81.2(3.4)	79.36(2.79)	118.4(3.1)	120.2(2.5)
Velocity Variability	5.9(0.4) *	4.0(0.4)	7.2(0.5) *	5.5(0.6)
Cadence (steps/m)	110.2(3.1)	105.2(2.6)	115.5(2.5)	115.7(2.1)
Cadence Variability	4.7(0.4) *	3.4 (0.4)	4.2 (0.5) *	3.1(0.4)
Stride Length (cm)	89.3(3.3)	91.1 (2.8)	123.5(2.7)	125.9(2.3)
Stride Length Variability	8.0 (0.5)	6.9(.4)	6.3(.5)	5.7(.4)
Foot Angle (degs)	6.1(2.2) *	0.7(18)	4.7(1.4) *	0.3(11)
Foot Angle Variability	3.6(0.3)	3.2(0.2)	2.4(0.3)	2.5(0.3)
Step Width (cm)	15.7(0.9) *	12.2(0.8)	8.6(0.7)	8.7(0.6)
Step Width Variability	2.2(0.1) *	1.6(0.1)	1.6(0.1)	1.5(1)
Double Support Time (ms)	295(13.0)	299(11.0)	230(9.0)	225(7.0)
Double Support Variability	37(4.0)	36(3.0)	25(2.0)	21(3.0)

* = significantly different from controls, $p < .05$

Table 3

Gait parameter means (standard error) for the PAE and CON groups when walking with decreased, self-paced and increased cadence. Data are collapsed across walking direction to facilitate group comparisons.

	Decreased			Cadence Self-Paced			Increased		
	PAE	CON		PAE	CON		PAE	CON	
Velocity (cm/s)	81.8(2.8)	78.9(2.3)		98.2(2.9)	100.9(2.4)		119.3(4.1)	119.4(3.4)	
Velocity Variability	5.3(0.4)	4.7(0.3)		6.5(0.6)	5.6(0.5)		7.7(0.6)*	4.9(0.5)	
Cadence (Steps/m)	95.7(2.1)	90.6(1.8)		112.2(2.7)	110.4(2.2)		130.7(3.1)	130.4(2.6)	
Cadence Variability	4.0(0.4)*	2.4(0.3)		5.0(0.5)*	3.9(0.5)		4.5(0.5)*	3.4(0.5)	
Stride Length (cm)	103.4(3.1)	104.9 (2.6)		105.5(2.5)	109.9(2.1)		110.2(3.6)	110.7(2.9)	
Stride Length Variability	7.5(0.6)	7.2(0.5)		6.2(.5)	5.5(0.4)		7.8(0.4)*	5.9(0.3)	
Foot Angle (degs)	6.7(18)*	0.9(15)		4.1(14)*	0.1(1.3)		5.4(15)*	-0.5(1.2)	
Foot Angle Variability	2.9(0.3)	2.5(0.2)		2.0(0.4)	3.2(0.3)		3.1(03)	2.8(0.2)	
Step Width (cm)	12.3(0.7)*	10.9(0.7)		11.9(0.6)*	10.1(0.5)		12.2(0.6)*	10.4(0.5)	
Step Width Variability	1.9(0.2)*	1.4(0.1)		1.9(0.2)*	1.5(01)		2.0(0.2)*	1.7(0.1)	
Double Support Time (ms)	308(12.0)	327(10.0)		266(10.0)	257(8.0)		213(8.0)	203(7.0)	
Double Support Variability	34(4.0)	39(3.0)		35(3.0)*	26(3.0)		24(2.0)	21(2.0)	

* = significantly different from controls, $p < .05$