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Research Article

Gait Patterns of Children with Idiopathic Hypotonia

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Abstract The characteristics of idiopathic hypotonic gait are poorly understood. The purpose of this study was to identify biomechanical parameters that differentiate between children with hypotonia and an age-matched control group. Twelve children with idiopathic hypotonia, aged 6–13 years, participated in the study. Twenty-two children with no known disorders, aged 6–13 years, served as a control group. A 6-camera Vicon MCam and three force plates were used to collect kinematic and kinetic data during gait. Significant differences in the mean kinematic and kinetic values between groups were tested using a MANOVA. No significant group differences were found for any temporal-spatial variables. Significant group differences ($P < .05$) were found for sagittal ankle angle and moment, sagittal knee angle and moment, and sagittal hip angle. The majority of deviations appear to be related to impairments in the gastrocnemius complex. A greater awareness of gait deviations in this population will increase our understanding of the disorder and aid in treatment planning.

Keywords clinical gait analysis; idiopathic hypotonia

1 Introduction

Hypotonia refers to a decreased resistance to passive movement and is a common diagnosis in infants and children [8]. Hypotonia may be caused by peripheral and central nervous system disorders and metabolic, neuromuscular, and connective tissue disorders [8]. Disorders associated with hypotonia include Down syndrome, cerebellar ataxia and muscular dystrophy (MD). In some cases, the underlying cause of hypotonia is unknown and is referred to as idiopathic hypotonia. In conjunction with decreased muscle tone, children with hypotonia may also exhibit decreased strength, joint hypermobility, increased flexibility, delayed acquisition of independent walking and abnormal walking patterns [2, 16]. Despite these serious orthopaedic and motor problems, there are a limited number of studies that provide an objective, three-dimensional analysis of the walking patterns of children with hypotonia.

To date, studies of hypotonic gait have tended to focus on children with Down syndrome (DS). DS is typically associated with central hypotonia, in which global or whole body abnormalities in muscle tone are present [19]. Reported deviations in kinematic gait parameters in DS include flat foot contact, reduced sagittal ankle angles [18], increased stance phase dorsiflexion [11], increased hip and knee flexion postures [10, 11, 17, 18], decreased hip extension in terminal swing [11, 17], increased hip abduction in swing [17], increased hip adduction in swing [11], increased pelvic rotations [1], external foot rotation [1] and longer duration in stance phase [17, 18]. Reported deviations in kinetics included reduced sagittal ankle moments [1, 6, 9, 11], reduced ankle power [1, 6], absence of the sagittal knee extensor moment peak [10] and decreased sagittal hip joint moments and hip power [1].

Generalizing the results of DS studies to children with idiopathic hypotonia is problematic for several reasons. First, in conjunction with decreased muscle tone, children with DS typically exhibit ligamentous laxity, hyperflexibility of the joints and orthopaedic problems such as pes planovalgus, hip and patella dislocation and genu valgum [2]. Such disorders could contribute to gait abnormalities and may not be present in children with idiopathic hypotonia. Second, idiopathic hypotonia may stem from central and/or peripheral causes [12]. Depending on the origin of the hypotonia, the gait patterns of children with idiopathic hypotonia may not resemble those of children with Down syndrome, who typically exhibit central hypotonia. This is due to the differential clinical signs between peripheral and central hypotonia related to, among others, weakness, deep tendon reflexes, cognition, antigravity and movements [12]. Research that quantifies the gait patterns of children with idiopathic hypotonia is warranted.

Studies examining gait in children with idiopathic hypotonia are needed to increase our understanding of the movement patterns associated with this disorder. Three-dimensional gait analysis provides valuable clinical information that cannot be obtained by observational gait analysis alone. For example, measurements of net muscle activity (joint moments and powers) and electromyography data can provide insight on the underlying neuromuscular activity

Gender (Male/female)	Age (years)	Weight (kg)	Height (cm)	Age at onset of independent gait (months)	Distribution of hypotonia
M	6.0	23.4	120.5	15	Generalized
M	6.5	18.2	111.0	24	Distal
M	5.6	21.4	115.0	23	Generalized
M	7.6	22.1	121.1	24	Generalized
M	7.7	31.4	130.5	20	Generalized
M	12.4	24.1	127.8	84	Generalized
F	7.5	23.6	116.2	26	Generalized
M	13.0	39.6	154.9	43	Generalized
M	11.0	85.5	146.0	26	Generalized
F	10.0	21.8	128.0	19	Distal

Table 1: Participant characteristics for the hypotonic group.

that generates movement. These analyses also provide important information for treatment planning and treatment evaluation (e.g. orthoses, pre- and post-surgery). Currently, little is known about the gait patterns of children with idiopathic hypotonia. Therefore, the purpose of this study was to quantify and assess the gait patterns of children diagnosed with idiopathic hypotonia versus age-matched controls.

2 Method

2.1 Participants

Twelve children, aged 6–13 years, participated in the study, having been diagnosed with idiopathic hypotonia between birth and 3 years of age by a physician. Participants were recruited from a local rehabilitation clinic, which provides physiotherapy services to children with hypotonia. Measures of hypotonia were conducted by a physiotherapist using a Modified Ashworth Scale. One child was noncompliant, and another child required assistance during walking trials. This resulted in a total of ten ($N = 10$) independent walkers (8 males, 2 females; mean age = 8.7 years; mean height = 127.1 cm; mean weight = 31.1 kg). Further characteristics of the hypotonic group of children are provided in Table 1. Twenty-two ($N = 22$) children, aged 6–13 years, were recruited from local schools to serve as a control group (14 males, 8 females; mean age = 8.2 years; mean height = 130.3 cm; mean weight = 30.7 kg). Exclusion criteria for the control group included a history of musculoskeletal injury to the lower extremities and premature birth. Parental consent and child assent were obtained prior to the study. This study was approved by the Research Ethics Board at the University of New Brunswick.

2.2 Instrumentation/apparatus

A six-camera Vicon 512 motion capture system (Oxford Metrics Group, UK) was employed to track the three-dimensional trajectories of reflective markers placed on the subjects' skin. All gait trials were collected at a sampling frequency of 60 Hz. In addition, three force plates (Kistler Instruments, Winterthur, Switzerland) collected the three-dimensional ground reaction forces and moments during each gait cycle. Force plate data was sampled at 600 Hz during walking. A 22-foot walkway allowed each child to attain steady state velocity through the recording area. Two digital cameras, a weight scale and calipers were used to obtain anthropometric measures from each participant.

Marker location for dynamic trials
Left and right anterior superior iliac spine
Left and right mid-thigh wand
Left and right lateral femoral condyle
Left and right mid-shank wand
Left and right malleolus
Left and right heel
Left and right 2nd metatarsal head
Sacral wand
Left and right shoulder (midway between neck and acromion process)
C7, base of neck
Additional markers for static trials
Left and right greater trochanter

Table 2: Marker locations for gait trials.

2.3 Procedures

Each participant was asked to visit the motion analysis laboratory for a single testing session. Twenty reflective markers, representing key anatomical landmarks, were placed directly on the skin of each participant (Table 2). To ensure accurate placement of markers, participants were asked to wear minimal clothing or bathing suits during data collection. Several "warm-up" trials were conducted to allow the participants to adjust to the markers and the walkway. Twelve to twenty gait trials were collected for each participant at a self-selected walking speed. The number of total trials collected depended on fatigue and/or compliance. Successful trials were those with visible markers and clean force plate strikes (one foot fully on plate).

Following the completion of the gait trials, the reflective markers were removed and a new segment inertia marker set was applied. This latter marker set aided in the identification of joint centers and segment outlines during the digitization process. Participants were then asked to stand in the anatomical position within a calibration frame, while simultaneous front and side digital photographs were taken. Anthropometric data such as joint width, height and mass were also measured.

2.3.1 Data analysis

All data analysis was performed using custom software in Matlab (Mathworks, Natick, Massachusetts, USA). A comprehensive kinematic and kinetic analysis of each child's gait was performed. The body was modeled as a series of rigid links joined by 3 degree-of-freedom articulations. The model consisted of the left and right foot, shank, thigh and the pelvis and trunk. Joint center locations were estimated in accordance with Davis et al. [7]. Temporal-spatial measures and joint angles were calculated from the three-dimensional coordinates produced by the Vicon 512 motion analysis system. 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 a *yxz* sequence, corresponding to flexion/extension, adduction/abduction and internal/external rotation.

Representative gait cycles for the left and right limb were selected for analysis based on temporal-spatial data. For each participant, cadence, velocity and percent of cycle spent in single stance were calculated for each available gait cycle for the right and left limb. The mean of each of these three gait parameters was computed for both the left and right limb. Using a least-squares method, the single left and right gait cycle that most closely approximated the respective mean of these three measures was selected as the final trial for analysis.

A mathematical model (elliptical cylinder method) of the human body was used to estimate the segment inertial properties of each child. This model has been used previously in adult and paediatric research [3, 13, 14, 15]. This technique requires the digitization of the front- and right-side full body images obtained from the digital photographs of each participant in the anatomical position. The model consists of 16 segments and each segment is assumed to consist of elliptical cylinders created at 1–2 cm intervals in the transverse plane. Given that the volume and density of each elliptical cylinder is known, the segment mass, center of mass location and three-dimensional moments of inertia can be estimated from the stacked elliptical cylinders representing each segment. This technique is advantageous as it permits the estimate of individually tailored estimates of segment inertial data based on each participant's body.

Net joint moments and joint power for the hip, knee and ankle joints were estimated using the inverse dynamics approach. This technique combines the motion data, force plate data and segment inertial data. The required absolute linear and angular velocities and accelerations were calculated from the embedded coordinate systems using a five-point derivative. These data were filtered using a 6 Hz low-pass Butterworth filter.

2.3.2 Statistical analysis

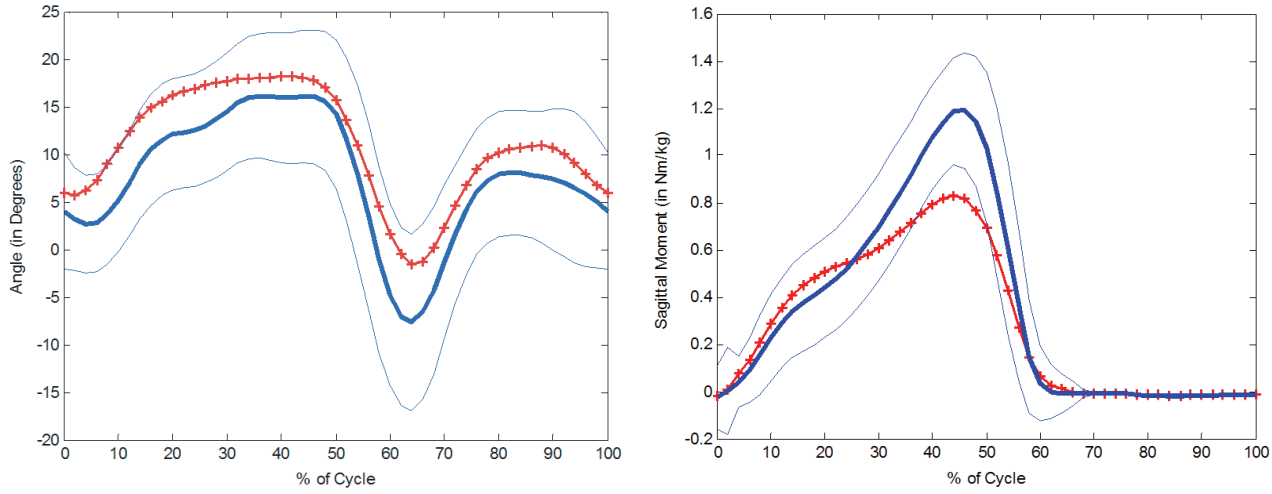
Data reduction involved the extraction of discrete parameters (e.g. maximum and minimum values) from each individual's left- and right-gait waveforms. Significant ($P < .05$) differences in the mean temporal-spatial and discrete parameters (Table 3) between the control and hypotonia group were tested using a multivariate analysis of variance (MANOVA). All statistical tests were performed using SPSS (SPSS Inc.).

3 Results

Descriptive data for the discrete gait parameters tested are provided in Table 3. No significant differences were found between groups for any of the temporal-spatial parameters. Significant group differences ($P < .05$) were found for five of the discrete gait parameters (Table 3). At the ankle joint, the hypotonic group demonstrated significantly decreased peak plantarflexion angles during the gait cycle [hypotonia: mean −5.28 degrees (SD 7.54); control: mean −9.86 degrees (SD 8.84)] and significantly decreased peak plantarflexor moments in stance [hypotonia: mean 0.88 Nm/kg (SD 0.21); control: mean 1.25 Nm/kg (SD 0.25)]. Graphs of the mean sagittal ankle angle and moments are provided in Figure 1. At the knee joint, the hypotonic group demonstrated significantly larger peak knee flexion angles during the stance phase [hypotonia: mean 33.84 degrees (SD 11.26); control: mean 25.45 degrees (SD 10.69)] and significantly decreased peak knee flexor moments in stance [hypotonia: mean −0.29 Nm/kg (SD 0.13); control: mean −0.41 Nm/kg (SD 0.17)]. Graphs of the mean sagittal knee angle and moments are provided in Figure 2. The hypotonic group demonstrated significantly larger peak hip flexion angles during the stance phase [hypotonia: mean 40.68 degrees (SD 8.30); control: mean 35.47 degrees (SD 9.65)]. Graphs of the mean sagittal hip angle are provided in Figure 3.

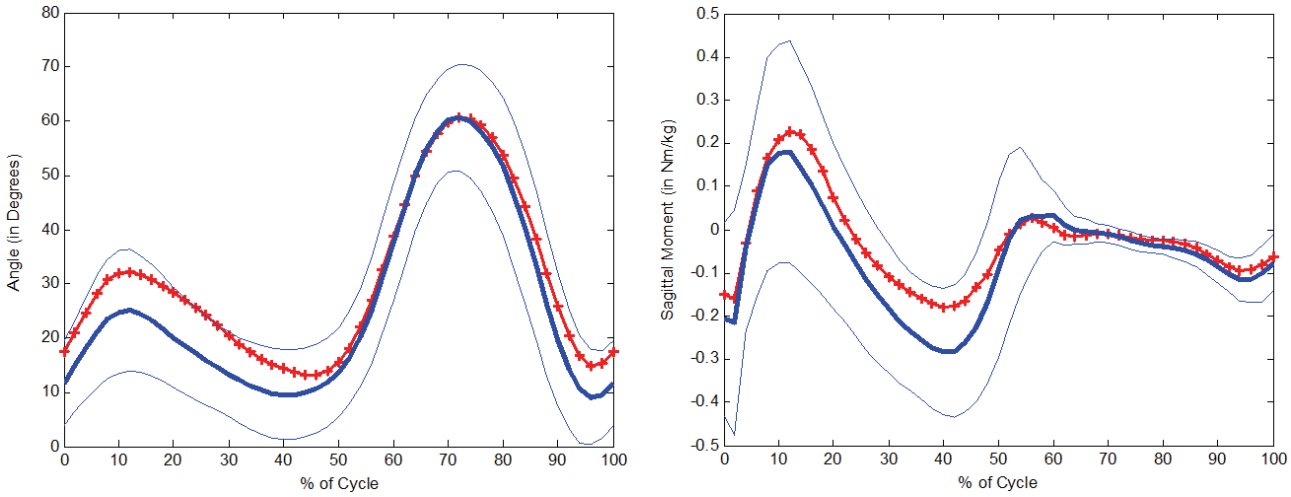
4 Discussion

The purpose of this study was to identify differences in gait parameters between the hypotonic and control groups. No significant differences ($P < .05$) were found



(a) (b)

Figure 1: (a) Mean sagittal ankle angle (+ dorsiflexion/ − plantarflexion) for hypotonic group (mean = red +) and control group (mean = solid thick blue line, ±1 SD = thin blue lines); (b) Mean sagittal ankle moment (+ plantarflexor/ − dorsiflexor) for hypotonic group (mean = red +) and control group (mean = solid thick blue line, ±1 SD = thin blue lines).



(a) (b)

Figure 2: (a) Mean sagittal knee angle (+ flexion/ − extension) for hypotonic group (mean = red +) and control group (mean = solid thick blue line, ±1 SD = thin blue lines); (b) Mean sagittal knee moment (+ extensor/ − flexor) for hypotonic group (mean = red +) and control group (mean = solid thick blue line, ±1 SD = thin blue lines).

for any of the temporal-spatial parameters. However, significant differences were found for five of the discrete gait parameters, namely, peak ankle plantarflexion angle and moment in stance, peak knee flexion angle and moment in stance, and peak hip flexion angle in stance. As no biomechanical studies of idiopathic hypotonic gait exist, findings were compared to the available DS studies. In contrast to previous research [1,5,6,10], we did not observe significant differences in joint powers.

Significantly different patterns of motion were observed at the ankle joint between the hypotonic and control groups. The hypotonic group had a significantly decreased mean peak plantarflexion angle and mean peak plantarflexion moment during stance. As shown in Figure 1, the mean

		Hypotonia		Control	
Variables	Phase	Mean	SD	Mean	SD
Cadence (steps/min)	Cycle	137.42	13.83	134.29	18.32
Walking speed (cm/s)	Cycle	89.69	24.81	98.00	22.57
Stride length (cm)	Cycle	52.91	5.42	54.60	7.51
Double stance (%)	Cycle	20.33	6.22	20.03	3.82
Toe-off (%)	Cycle	59.48	4.08	60.17	2.61
Cycle time (s)	Cycle	0.88	0.09	0.91	0.13
Maximum pelvic tilt (deg)	Cycle	14.27	4.57	12.75	6.25
Minimum pelvic tilt (deg)	Cycle	8.85	4.10	8.28	5.85
Maximum pelvic obliquity (deg)	Cycle	4.73	3.37	3.98	2.97
Minimum pelvic obliquity (deg)	Cycle	-4.02	3.34	-3.32	2.77
Maximum pelvic rotation (deg)	Cycle	6.57	4.17	6.90	6.27
Minimum pelvic rotation (deg)	Cycle	−5.95	5.43	−5.95	6.05
Maximum hip flexion (deg)	Stance	**40.68***	8.12	35.47	9.65
Maximum hip flexion (deg)	Swing	42.30	7.37	38.55	9.58
Maximum hip extension (deg)	Stance	−4.94	9.21	−8.60	8.71
Sagittal hip range of motion (deg)	Cycle	47.68	9.83	47.73	10.85
Maximum hip adduction (deg)	Stance	9.66	5.59	8.92	4.90
Maximum hip abduction (deg)	Stance	−2.61	4.66	−1.51	5.08
Maximum hip extension moment (Nm/kg)	Stance	0.86	0.28	0.91	0.34
Maximum hip flexion moment (Nm/kg)	Stance	−0.32	0.19	−0.38	0.19
Maximum hip power (W/kg)	Cycle	1.47	0.69	1.45	1.10
Maximum knee flexion (deg)	Stance	**33.84***	11.26	25.45	10.69
Maximum knee flexion (deg)	Swing	62.88	11.90	63.50	10.05
Maximum knee extension (deg)	Stance	11.67	12.79	8.99	8.35
Sagittal knee range of motion (deg)	Cycle	54.92	8.48	57.89	9.66
Maximum knee extension moment (Nm/kg)	Stance	0.30	0.24	0.27	0.20
Maximum knee flexion moment (Nm/kg)	Stance	**−0.29***	0.13	−0.41	0.17
Maximum knee power (W/kg)	Cycle	0.94	0.53	0.93	0.54
Maximum ankle dorsiflexion (deg)	Stance	21.42	6.89	18.46	6.66
Maximum ankle dorsiflexion (deg)	Swing	13.17	7.02	9.93	5.90
Maximum ankle plantarflexon (deg)	Stance	**−5.28***	7.54	−9.86	8.84
Sagittal ankle range of motion (deg)	Cycle	26.82	7.12	28.37	8.48
Maximum foot rotation (deg)	Cycle	−2.86	9.18	3.21	15.77
Maximum dorsiflexion moment (Nm/kg)	Stance	−0.09	0.13	−0.09	0.12
Maximum plantarflexion moment (Nm/kg)	Stance	**0.88***	0.21	1.25	0.25
Maximum concentric ankle power (W/kg)	Stance	1.80	0.80	2.71	1.02
Maximum eccentric ankle power (W/kg)	Stance	−0.78	0.30	−0.88	0.46

Table 3: Group descriptive statistics for temporal-spatial data and gait variables (* refers to significant differences between groups, $P < .05$). Phase refers to the portion of the cycle the data was extracted from [Cycle = entire gait cycle; Stance = stance phase; Swing = swing phase].

plantarflexion moments of the control group were greater for almost a third of the gait cycle. During gait, plantarflexor moments are generated to (1) oppose the passive dorsiflexor moments generated by ground reaction forces, (2) control the forward advancement of the tibia during the stance phase and (3) aid in forward progression through segmental power transfers. Significantly decreased plantarflexor moments in the hypotonic group may compromise these functions. It is likely that the decreased moments are due to weakness of the gastrocnemius and/or soleus leading to poor tibial control. Previous research has also reported decreased sagittal ankle angles and moments in children with Down syndrome [6,9,11,17]. Similar findings have also been reported in immature walkers [4].

The hypotonic group demonstrated a significantly increased mean peak knee flexion during the loading response compared to the control group. The loading response is associated with stabilization of the knee joint and shock absorption. Increased knee flexion during this portion of the gait cycle is associated with inappropriate

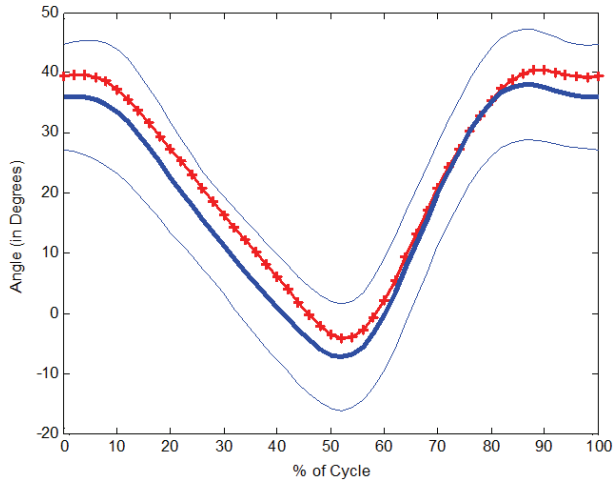


Figure 3: Mean sagittal hip angle (+ flexion/ − extension) for hypotonic group (mean = red +) and control group (mean = solid thick blue line, ±1 SD = thin blue lines).

knee extensor muscle activity and/or gastrocnemius/soleus muscle weakness. Compared to the control group, the hypotonic group showed increased mean knee flexion for the majority of the stance phase (Figure 2). The hypotonic group also showed significantly decreased mean peak knee flexor moments during terminal stance, which was mainly a function of decreased plantarflexor activity. Galli et al. [11] also found increased knee flexion orientation at initial contact in children with DS. These findings at the knee and ankle joints may be important in terms of treatment planning for children with hypotonia (e.g. strength training and orthotics).

The hypotonic group demonstrated a significantly larger mean peak hip flexion at initial loading. Given the lack of significant findings in the hip kinetic data or pelvic angle data, the increased hip flexion was likely the result of increased knee flexion and is therefore not clinically relevant. Increased hip flexion at initial loading was also reported by Cimolin et al. [5], but was considered a function of increased anterior pelvic tilt. Compared to the control group, the hypotonic group showed small differences in sagittal hip angles for most of the gait cycle, with the exception of midswing (Figure 3). Previous work has identified several hip deviations not found in the present study. These include increased hip adduction in swing [11], increased hip abduction in swing, decreased hip extension in swing [18] and decreased sagittal hip joint moments and power [1].

Gait patterns of children with idiopathic hypotonia show some similarities to those of children with DS. However, numerous gait deviations identified in children with DS were not found in the present study. While the idiopathic hypotonic group showed a mainly generalized distribution of hypotonia (Table 1), the results of the gait analyses showed a common distal involvement (e.g. gastrocnemius-soleus complex). The more proximal hip joints and pelvis showed compensatory deviations or normal motion, respectively.

This study has limitations that must be considered. First, all of the children who volunteered for this study were receiving, or had received, treatment in the form of physiotherapy and/or orthotics. Therefore, it is possible that the natural gait patterns of the children have been modified as a function of treatment. Second, EMG data, which was not included in this work, could provide valuable information on individual muscle activity during gait.

5 Conclusion

This study has found several gait measures that were significantly different between the hypotonic and control groups. A greater awareness of deviations in gait patterns will increase our understanding of hypotonia and aid in treatment planning and evaluation. Future work should increase sample sizes to test the validity of the current findings. In addition, incorporating EMG with the kinematic and kinetic parameters of gait will allow for the quantification of individual muscle activity during the gait cycle and a greater understanding of the underlying causes of movement deviations.

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References

[1] K. Byung-Sik, D. Y. Bang, and K. Bong-Ok, *Gait characteristics in Down syndrome*, Gait Posture, 3 (1995), 84.

[2] M. A. Caselli, E. Cohen-Sobel, J. Thompson, J. Adler, and L. Gonzalez, *Biomechanical management of children and adolescents with Down syndrome*, J Am Podiatr Med Assoc, 81 (1991), 119–127.

[3] V. L. Chester and R. K. Jensen, *Changes in infant segment inertias during the first three months of independent walking*, Dyn Med, 4 (2005), 1–4.

[4] V. L. Chester, M. Tingley, and E. N. Biden, *A comparison of kinetic gait parameters for 3–13 year olds*, Clin Biomech, 21 (2006), 726–732.

[5] V. Cimolin, M. Galli, G. Grugni, L. Vismara, G. Albertini, C. Rigoldi, et al., *Gait patterns in Prader-Willi and Down syndrome patients*, J Neuroeng Rehabil, 7 (2010), 28–35.

[6] M. Cioni, A. Cocilovo, F. Rossi, D. Pacci, and M. S. Valle, *Analysis of ankle kinetics during walking in individuals with Down syndrome*, Am J Ment Retard, 106 (2001), 470–478.

[7] R. B. Davis, S. Ounpuu, D. Tyburski, and J. R. Gage, *A gait data collection and reduction technique*, Hum Mov Sci, 10 (1991), 575–587.

[8] V. Dubowitz, *Evaluation and differential diagnosis of the hypotonic infant*, Pediatr Rev, 6 (1985), 237–243.

[9] M. Galli, G. Albertini, N. Tenore, and M. Crivellini, *Gait analysis in children with Down syndrome: progress report-intern*, Rev Med Sci, 13 (2001), 21–27.

[10] M. Galli, C. Rigoldi, R. Brunner, N. Virji-Babul, and A. Giorgio, *Joint stiffness and pattern evaluation in children with Down syndrome*, Gait Posture, 28 (2008), 502–506.
[11] M. Galli, M. Romei, N. Tenore, M. Crivellini, and G. Albertini, *Gait analysis in children with Down syndrome: the effect of flatfoot*, in Proceedings of the 8th Annual GCMAS Meeting, University of Delaware, USA, 2003.
[12] S. R. Harris, *Congenital hypotonia: clinical and developmental assessment*, Dev Med Child Neurol, 50 (2008), 889–892.
[13] R. K. Jensen, *Estimation of the biomechanical properties of three body types using a photogrammetric method*, J Biomech, 11 (1978), 349–358.
[14] R. K. Jensen, S. Doucet, and T. Treitz, *Changes in segment mass and mass distribution during pregnancy*, J Biomech, 29 (1996), 251–256.
[15] R. K. Jensen, H. Sun, T. Trietz, and H. E. Parker, *Gravity constraints in infant motor development*, J Mot Behav, 29 (1997), 64–71.
[16] K. Martin, J. Inman, A. Kirschner, K. Deming, R. Gumbel, and L. Voelker, *Characteristics of hypotonia in children: a consensus opinion of pediatric occupational and physical therapists*, Pediatr Phys Ther, 17 (2005), 275–282.
[17] A. W. Parker and R. Bronks, *Gait of children with Down syndrome*, Arch Phys Med Rehabil, 61 (1980), 345–351.
[18] A. W. Parker, R. Bronks, and C. W. Synyder, *Walking patterns in Down's syndrome*, J Ment Defic Res, 30 (1986), 317–330.
[19] C. Wusthoff and D. Hyder, *Hypotonia*, in Schwartz's Clinical Handbook of Pediatrics, J. J. Zorc, ed., Lippincott Williams and Wilkins, Philadelphia, PA, USA, 4th ed., 2008, 504–512.