Development of Calcaneal Gait Without Prior Triceps Surae Lengthening: An Examination of Predictive Factors

Kenneth Huh, MD, Susan A. Rethlefsen, PT,† Tishya A.L. Wren, PhD,*†‡ and Robert M. Kay, MD*†*

Background: Although equinus is more common in cerebral palsy (CP), the prevalence of calcaneal gait (CG) has been reported at more than 30% among patients with CP, even in the absence of prior surgical intervention. The goal of this study was to identify patient characteristics predictive of the development of CG in patients without prior triceps surae lengthening.

Methods: Gait data were reviewed for 58 participants with bilateral involvement owing to CP (116 limbs) who had 2 gait analysis tests with no triceps surae lengthening between tests. None of the patients exhibited CG at the initial gait study. Patients were grouped according to whether or not they exhibited CG patterns at the second test. Factors potentially predictive of calcaneal gait patterns were compared statistically between groups.

Results: CG was shown by 24/116 extremities (21%) at the second study. The CG group experienced greater increase in body weight and body mass index between tests ($P = 0.006$ and 0.03 respectively). Passive dorsiflexion range with the knee flexed was significantly greater in the CG group ($P = 0.008$). The CG group also showed a tendency toward greater plantarflexor weakness, although this only approached statistical significance ($P = 0.08$) likely owing to small sample size. Age, CP subtype, time to follow-up, hamstring range, selective motor control, and gross motor functional level were not predictive.

Conclusions: Patients who undergo (or have potential to undergo) significant weight gain, and have tendencies toward excessive passive dorsiflexion with the knee flexed may be at risk for development of CG over time. In such patients, treatment regimens should include therapy to maintain or improve plantarflexor strength, and methods to prevent overstretching the plantarflexors. Nonsurgical treatments for triceps surae contractures, such as serial casting, may be preferable, to avoid hastening development of calcaneal crouch gait over time.

Level of Evidence: Prognostic study—Level III (case-control).

From the †Childrens Orthopaedic Center, Childrens Hospital Los Angeles; Departments of *Orthopaedic Surgery, Keck School of Medicine; and ‡Radiology and Biomedical Engineering, University of Southern California, Los Angeles, CA.

None of the authors received financial support for this study.

Reprints: Susan A. Rethlefsen, PT, Childrens Orthopaedic Center, Childrens Hospital Los Angeles, 4650 Sunset Blvd., M.S. 69, Los Angeles, CA 90027. E-mail: srethlefsen@chla.usc.edu.

Key Words: gait, calcaneus, cerebral palsy

(*J Pediatr Orthop* 2010;30:240–243)

Cerebral palsy (CP) is a term used to describe a group of nonprogressive conditions that cause physical disability in human development. Although it is considered to be a static lesion to the brain, the damage sustained by the central nervous system causes dynamic changes in gait patterns during growth. There are limited data on the development of gait in patients with CP, but the few studies available agree that there is deterioration of gait over time in patients with CP.

Bell et al[1] examined 28 patients with CP with 2 gait analyses done an average of 4.4 years apart with no prior history of surgeries and found a decrease in range of motion about the hip, knee, and ankle and declines in timing of toe off, cadence, and walking velocity between the 2 tests. Johnson et al[2] carried out 2 separate analyses an average of 32 months apart on 18 patients with spastic diplegia and also found a decrease in joint excursions of the pelvis, knee, and ankle and decreased single limb stance times. Norlin et al[3] carried out a single gait analysis on 50 patients with spastic CP and compared the results with age-matched controls. They found deterioration of gait velocity owing to shorter stride length, and an increase in stance time and double limb stance again indicating deterioration in gait over time.

Although there is agreement that gait deteriorates over time in patients with CP, data looking at the development of specific gait patterns are much more limited. Wren et al[4] examined 492 patients with pretreatment gait analysis done and assessed the prevalence of 14 specific gait abnormalities. Although equinus is more commonly associated with CP, there was greater than 30% prevalence of calcaneal gait (CG) among their participants. The study found increased likelihood of CG patterns in patients over time, regardless of whether or not they had undergone prior surgical intervention. Although CG is frequently mentioned as a complication resulting from overlengthening the heel cord for patients with equinus contracture, its spontaneous development over time has not been reported extensively in the literature. The reported prevalence of calcaneal deformity

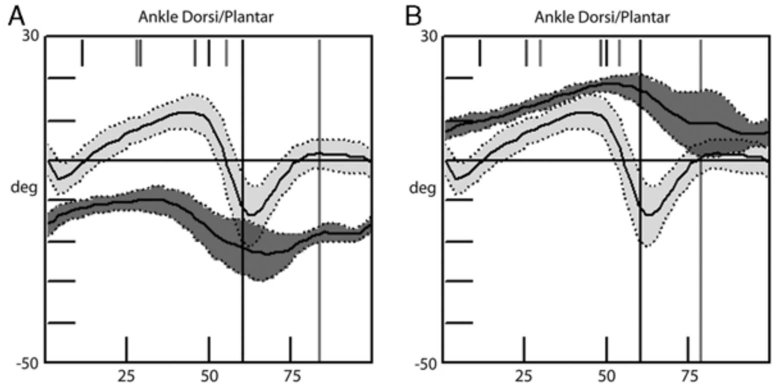

FIGURE 1. Sample kinematic data, participant from the calcaneal gait (CG) group (A, Initial test; B, Follow-up test). Light gray indicates normal; dark gray – patient data; positive values – dorsiflexion; negative values – plantarflexion.

after tendoachilles lengthening ranges from %0 to 36%.[5–8] Characteristics predictive of development of CG over time without prior surgery are unknown.

The goals of this study were to identify the patient characteristics predictive of the development of CG in patients with no prior history of heel cord lengthening, with an ultimate goal of facilitating the future development of treatment protocols to minimize calcaneal deformity and calcaneal gait.

METHODS

Gait analysis data were reviewed for all patients with CP who had undergone gait analysis testing in our laboratory from February, 1993 to September, 2008. Inclusion criteria were as: (1) diagnosis of CP, (2) minimum of 2 complete gait analysis assessments at our laboratory, (3) no history of triceps surae lengthening between gait studies, and (4) no evidence of midfoot breakdown. Sixty-five children fulfilled the inclusion criteria, but 7 patients with hemiplegia were excluded to allow the inclusion of 2 affected limbs in all participants. This resulted in the inclusion of 58 children with CP in the study, including 42 with diplegia and 16 with quadriplegia. Diplegia was defined as lower extremity involvement with minimal or no involvement of the upper extremities. Quadriplegia was defined as involvement of both upper and lower extremities in nearly equal fashion.

All participants had undergone gait analysis testing at our institution, including 3-dimensional kinematics (joint motions). The kinematic data were acquired using an 8-camera VICON (Oxford, UK) 3-dimensional motion analysis system. This system uses a set of 15 to 19 passive retroreflective markers attached over specific bony landmarks of the pelvis and lower extremities. Participants made several passes down a 15-meter path with the markers in place. Kinematic data from at least 3 trials were averaged, and the averaged data were included in the gait analysis report. An experienced gait laboratory physical therapist (SR) reviewed the gait data. CG was defined as excessive dorsiflexion (greater than 1 standard deviation above normal) during at least 50% of stance phase with or without diminished plantarflexion during push off (see sample patient data in Fig. 1). None of the participants studied had CG at the time of the initial gait analysis. Sixty-nine percent of the patients had undergone surgical intervention between gait studies, none of which included triceps surae lengthening.

Other participant measures had been taken during gait analysis testing, including examination of range of motion, selective motor control, and muscle strength. Range of motion was measured by a physical therapist using standard goniometry. Strength and selective motor control were tested manually by the physical therapist. Strength was assessed using the traditional scale of 0 to 5. Selective motor control was assessed using a scale of

TABLE 1. Demographics and Gait Velocity

Variable	NCG (N = 92)	CG (N = 24)	*P*
Age at initial study (y)	6.4 ± 1.9 (range: 3.5-12.4)	6.9 ± 1.8 (range: 3.6-12.4)	0.22
CP Subtype			
Diplegic	68 (74%)	14 (58%)	0.21
Quadriplegic	24 (26%)	10 (42%)	
Time between tests (y)	3.9 ± 2.8 (range: 0.9-13.7)	4.1 ± 2.4 (range: 1.1-11.5)	0.83
GMFCS level			
I/II	45 (51%)	11 (48%)	1.00
III/IV	44 (49%)	12 (52%)	
Gait velocity at initial study (% of normal)	65 ± 28 (range: 14-131)	55 ± 32 (range: 18-116)	0.13

TABLE 2. Change in Anthropometric Measures Between Tests

Variable	NCG (N = 92)	CG (N = 24)	*P*
Change in BMI (kg/m^2)	2.0 ± 2.8 (range: − 6.1-9.1)	4.0 ± 4.2 (range: − 2.0-11.0)	0.006
Change in height (cm)	20.1 ± 12.1 (range: 0.5-63.7)	23.1 ± 14.1 (0.5-63.7)	0.30
Change in body weight (kg)	11.5 ± 8.7 (range: − 2.5-46.1)	17.6 ± 13.2 (range: 0.1-46.1)	0.007

0 to 2, in which 2 = full selective movement out of a flexor or extensor pattern, 1 = partial selective movement out of a flexor or extensor pattern, and 0 = no selective movement out of a flexor or extensor pattern.

Patients were placed into 1 of 2 groups based on the development of CG between gait studies. The groups were CG and non CG (NCG). Statistical analysis (Student *t* test and Fisher Exact) was done to compare the 2 groups on these possible predictive variables: age at initial gait analysis, time between studies, CP subtype (diplegia or quadriplegia), ambulatory functional level as measured by the Gross Motor Function Classification System (GMFCS)[9] at initial gait analysis, gait velocity at initial gait analysis as a percentage of normal adjusted for age, and change in weight and body mass index (BMI) between studies. Comparisons were also made based on clinical examination at the initial gait study, including plantarflexion strength, plantarflexion selective control, quadriceps strength, quadriceps selective control, dorsiflexion range with the knee flexed, and extended and popliteal angle. The significance level was set at a $P < 0.05$.

RESULTS

Of the 116 extremities (58 test participants), 24 showed CG and 92 NCG at the second gait study. Sixteen of the 58 participants developed CG in at least 1 limb. The prevalence of CG in patients without prior history of heel cord lengthening was 28% of patients (16/58) and 21% of all limbs (24/116).

Demographics (age, CP subtype, time between tests, and GMFCS level) did not differ significantly between the 2 groups. Gait velocity at initial gait analysis also did not differ between groups (Table 1).

Change in BMI was greater in the CG group ($P = 0.006$). This was owing to a significant increase in body weight in that group. Patients in the CG group gained about 6 kg more than those in the NCG group ($P = 0.007$). Change in height was only 3-cm greater in the CG group, and was not significantly different between groups (Table 2).

Range of motion findings at the initial test in the 2 groups are reported in Table 3. Passive dorsiflexion range of motion with the knee flexed was significantly greater in the CG group ($P = 0.005$). However, dorsiflexion with the knee extended, popliteal angle, and hip extension range of motion did not differ significantly between groups.

There was no statistically significant difference in distribution of any of the strength or selective control variables between groups. Plantarflexor strength tended to be lower in the CG group (< 3/5 in 80% of limbs in CG compared with 46% in NCG), although the difference between groups was not statistically significant ($P = 0.08$) (Tables 4, 5).

DISCUSSION

This study is the first to look at the risk factors for CG in patients without prior history of triceps surae lengthening. In this study, 28% of the patients and 21% of the limbs studied were found to exhibit calcaneal positioning during gait. One of the statistically significant risk factors that predicted the development of CG over time without surgical lengthening was change in BMI between tests. The CG group experienced twice the increase in BMI shown by the NCG group. This was owing to a significant increase in body weight over time, with no significant difference in change in height between groups. Patients in the CG group gained about an average of 27% (of initial body weight) more than those in the NCG group. Change in height was only 2.6% (of initial height) greater in the CG group. These findings suggest that patients with potential to gain weight (such as those with more sedentary lifestyles, or with genetic predispositions to obesity) should be considered at risk for developing calcaneal crouch gait, and monitored appropriately.

The other factor predictive of development of CG without prior triceps surae surgery was increased passive dorsiflexion range of motion with the knee flexed (ie, soleus range of motion). This may be related to crouch during gait. Although hamstring range of motion was not

TABLE 3. Range of Motion at Initial Gait Study

	NCG (N = 92 Limbs)	CG (N = 24 Limbs)	*P*
DF with knee flexed (degrees)	16 ± 9 (range: − 2-35)	22 ± 11 (range: 0-40)	0.005
DF with knee extended (degrees)	6 ± 9 (range: − 10-35)	9 ± 10 (range: − 4-38)	0.11
Popliteal angle (degrees)	45 ± 12 (range: 10-75)	48 ± 15 (range: 18-90)	0.29
Hip extension (degrees)	− 8 ± 16 (range: − 20-0)	− 10 ± 8 (range: − 25-0)	0.07

TABLE 4. Strength Variables at Initial Gait Study

	Plantarflexors		Quadriceps	
	NCG N = 46	CG N = 10	NCG N = 68	CG N = 14
Strength < 3/5	21 (46%)	8 (80%)	5 (7%)	1 (7%)
Strength ≥ 3/5	25 (54%)	2 (20%)	63 (93%)	13 (93%)
P	0.08		1.00	

a predictive factor in this study, hamstring tightness was noted in both groups, and maintenance of hamstring range of motion may be especially important in patients with increased soleus range of motion. Excessive knee flexion during stance phase owing to hamstring tightness causes the ground reaction force to be directed posterior to the knee. To avoid toe-walking and to walk on a flat foot, the ankle must dorsiflex. As the knee flexion worsens over time, the amount of dorsiflexion needed to compensate would likely increase, thus further stretching the soleus and increasing calcaneus over time. In such patients, triceps surae lengthening might best be avoided or done as judiciously as possible. Nonsurgical interventions, such as serial casting, may be preferable in these cases. This would include patients with mild gastrocnemius contractures (limited dorsiflexion range of motion with the knee extended), as was present for some participants in both the CG and NCG groups in this study. There was a tendency toward greater plantarflexor weakness in the patients who developed calcaneal gait, although this only approached statistical significance likely owing to small sample size. Nevertheless, this suggests that plantarflexion strength in these patients should be preserved and weak plantarflexors protected, such as with appropriate bracing.

Although it was thought that older, more involved patients would be at higher risk for the development of calcaneal gait, this was not the case. Similarly, GMFCS level and CP subtype were not predictive for the development of CG suggesting that severity of involvement is not a factor. Wren et al[4] found increased odds over time of developing calcaneal gait. Similarly, in this study, development of CG occurred in nearly 30% of patients in an average of 4 years' time.

This study had several limitations. This was a retrospective study with a limited sample size. In addition, the data may have been biased toward a more involved population, as in our laboratory, it is primarily patients being considered for further surgery who are seen for follow-up gait analysis, and patients who were doing well are not referred for subsequent testing. The study excluded patients with hemiplegia. Therefore, the results cannot be extrapolated to the CP population in general. Sixty-nine percent of the participants had surgery at the hip and knee levels between gait studies, and the effect of these surgeries on gait development at the ankle was not ascertained. Finally, it is not known whether any of the participants had Botulinum toxin injections or serial casting treatments between studies, although these treatments have their primary effect in the short term.

As this is the first study to look specifically at the development of CG in patients with no prior history of triceps surae lengthening, the true prevalence is not known. However, it is felt that there are a significant number of such patients and if they can be identified early, treatment plans can be tailored to reduce the risk of calcaneal gait. For patients who undergo (or have potential to undergo) significant weight gain, who exhibit tendencies toward excessive dorsiflexion range of motion with the knee flexed, and tendencies toward plantarflexor weakness, treatment regimens should include therapy to maintain or improve plantarflexor strength, and methods to prevent overstretching of the plantarflexors (such as with use of dorsiflexion blocking AFOs). It is important to avoid early tendo-Achilles lengthening in these participants if possible, to prevent overlengthening of the heel cord, which may exacerbate the problem in the future. Nonsurgical treatments for triceps surae contractures, such as serial casting, may be preferable in these patients.

TABLE 5. Selective Control Variables at Initial Gait Study

	Plantarflexion Selective Control		Quadriceps Selective Control	
	NCG N = 65	CG N = 13	NCG N = 68	CG N = 16
Selectivity < 2	54 (83%)	13 (100%)	47 (69%)	12 (75%)
Selectivity 2	11 (17%)	0 (0%)	21 (31%)	4 (25%)
P	0.19		0.77	

REFERENCES

1. Bell KJ, Ounpuu S, DeLuca PA, et al. Natural progression of gait in children with cerebral palsy. *J Pediatr Orthop*. 2002;22:677–682.
2. Johnson DC, Damiano DL, Abel MF. The evolution of gait in childhood and adolescent cerebral palsy. *J Pediatr Orthop*. 1997;17:392–396.
3. Norlin R, Odenrick P. Development of gait in spastic children with cerebral palsy. *J Pediatr Orthop*. 1986;6:674–680.
4. Wren TAL, Rethlefsen S, Kay RM. Prevalence of specific gait abnormalities in children with cerebral palsy: influence of cerebral palsy subtype, age, and previous surgery. *J Pediatr Orthop*. 2005;25:79–83.
5. Damron TA, Breed AL, Cook T. Diminished knee flexion after hamstring surgery in cerebral palsy patients: prevalence and severity. *J Pediatr Orthop*. 1993;13:188–191.
6. Borton DC, Walker K, Pirpiris M, et al. Isolated calf lengthening in cerebral palsy. Outcome analysis of risk factors. *J Bone Joint Surg-British Vol*. 2001;83:364–370.
7. Segal LS, Thomas SE, Mazur JM, et al. Calcaneal gait in spastic diplegia after heel cord lengthening: a study with gait analysis. *J Pediat Orthoped*. 1989;9:697–701.
8. Craig JJ, van Vuren J. The importance of gastrocnemius recession in the correction of equinus deformity in cerebral palsy. *J Bone Joint Surg-British Vol*. 1976;58:84–87.
9. Palisano RJ, Hanna SE, Rosenbaum PL, et al. Validation of a model of gross motor function for children with cerebral palsy. *Phys Ther* 2000;80:974–985.

