including overflow motor activity, poor segmental dissociation, impaired balance, absence of myostatic deformities, and other extra-pyramidal signs. (1,2,3,4) This study applied computer-based analysis of gait to objectively assess for the presence of significant dyskinesia in children with cerebral palsy. Two questions were addressed: (1) which gait parameters are distinct between spastic and dyskinetic children? and (2) can these parameters be used to generate a predictive model for dyskinesia?

METHODOLOGY:

Three dimensional kinematic and kinetic analysis (VICON 370, AMTI Force Platforms, Vicon Clinical Manager) was performed on 18 normal children (NL), 17 children with principally spastic cerebral palsy (CPS), and 23 children with significantly dyskinetic cerebral palsy (CPD). Children were assigned to the CPS or CPD groups prospectively, based upon clinical analysis by an experienced physician and physical therapist. The three groups were comparable with respect to age (p=0.67,one way ANOVA). Selected gait parameters were analyzed by using mixed model ANOVA, and Tukey's HSD post hoc. A predictive model was developed using logistic regression analysis with non-parametric independent variables.

RESULTS:

The analysis of selected gait parameters is shown in Table 1. The normalized dynamic base of support (stride width/pelvic width) was significantly wider, and more variable, for the CPD group than the CPS and NL groups. The stride profile (stride length/stride width) was significantly different between all three groups, with CPD < CPS < NL. The maximum lateral acceleration (meters/seconds^2) was significantly greater and more variable for the CPD group than the CPS and NL groups.

The following logistic regression equation was developed: $p = e^x/1+e^x$, where $x = -1.16 + 1.8842$ (stride profile) + 2.18 (max. lat. accel.). The results of the predictive model are summarized in Table 2. The predicted probability of dyskinesia is shown for each value of the non-parametric variables included in the model. For example, the model predicts that children with a stride profile < 3.1 and a maximum lateral acceleration > 1.25 m/s^2 have a 95% probability of being clinically classified as dyskinetic. The model correctly classified 20 of 23 children as dyskinetic, exhibiting 87% concordance with the clinical classification.

TABLE 1:
CP: SPASTIC VS. DYSKINETIC VS. NORMAL GAIT PARAMETERS

PARAMETER		NORMAL NL	SPASTIC CPS	DYSKINETIC CPD	1 WAY ANOVA (P)	TUKEY POST HOC
BASE OF SUPPORT	Mean	.46	.59	.80	.0001	CPD > CPS, NL
	Std. Dev.	.12	.19	.31		
STRIDE PROFILE	Mean	6.5	4.3	2.9	.0000	CPD < CPS < NL
	Std. Dev.	2.1	1.9	1.4		
MAX. LAT. ACCEL.	Mean	.76	.90	1.34	.0000	CPD > CPS, NL
	Std. Dev.	.22	.27	.45		

Table 2:
CP: SPASTIC vs. DYSKINETIC PREDICTIVE MODEL

Stride Profile Greater Than or Less Than 3.1	Maximum Lateral Acceleration (m/s^2) Greater Than or Less Than 1.25	Predicted Probability of Dyskinesia
> 3.1	< 1.25	.24
< 3.1	< 1.25	.67
> 3.1	> 1.25	.74
< 3.1	> 1.25	.95

DISCUSSION:

Children with dyskinetic cerebral palsy have a significantly wider and more variable dynamic base of support, a shorter stride length, and greater maximum lateral acceleration than children with spastic cerebral palsy. A predictive model using these parameters exhibited excellent sensitivity in correctly classifying children as dyskinetic, suggesting that objective assessment of dyskinesia is possible with computer-based analysis of gait. Incorporation of additional gait parameters and clinical subjects may further improve the accuracy of this method.

REFERENCES:

1. Bax. *Dev Med Child Neurol*, 6:295-307, 1965.
2. Blair, et al. *Dev Med Child Neurol*, 27:615-622, 1985.
3. Minear. *Pediatrics*, 18:841-852, 1956.
4. Phelps. *J Bone Joint Surg*, 14:773-782, 1932.

LIMITS TO PASSIVE RANGE OF JOINT MOTION AND THE EFFECT ON CROUCH GAIT IN CHILDREN WITH CEREBRAL PALSY

Michael S. Orendurff, MS, James S. Chung, BS, and Rosemary A. Pierce, PT
Gait Analysis Laboratory, Shriners Hospital for Children, Portland, Oregon

Introduction

Individuals with cerebral palsy may develop joint contractures which limit both passive and active range of motion. Limitations in active range of motion (ROM) may be due to a wide variety of causes including deficits in motor control and perception, strength, spasticity, bony configuration of the joint, ligamentous and musculotendinous length. In order to sort out the cause of reduced active ROM in a task such as walking, detailed physical examination is often used to determine the extent of ligamentous and musculotendinous restrictions (passive ROM). Orthopedic interventions are often based on passive ROM limitations when they coincide with recognized patterns as quantified by computerized gait analysis. For example when tight hamstrings result in crouch gait, surgical intervention may be warranted. Choosing appropriate surgical interventions is a complex task. Since there are multiple solutions to overcome the limitations imposed by various shortened musculotendinous units, ligaments and bony malformations, adaptations to restricted motion at a specific joint may be compensated for in a wide array of strategies. In addition limitations are rarely limited to just one joint and the interaction of multiple limitations of joint ROM complicates compensation mechanisms further. In order to approach the simplest level of joint interaction, it was the goal of this project to examine the passive limitations of sagittal joint motion at the hip, knee and ankle and determine the influence upon hip and knee flexion and ankle kinetics during gait.

Methods

One hundred and six children diagnosed with cerebral palsy (mean age 11.4 ± 4.2 years, range 3.9 to 20.5 years; diagnosis: 63 spastic diplegia, 30 spastic hemiplegia, 5 spastic quadriplegia, 3 spastic triplegia, 3 hereditary spastic paraplegia, 1 spastic paraplegia, 1 hypotonic cerebral palsy) were evaluated by physical examination with a goniometer to determine joint contractures of the hips, knee and ankles in the sagittal plane. Maximum hip extension was measured using a Thomas test (HF_{cont}). Passive range of knee extension was measured supine with the hip flexed to 90° and recorded as the decrement from full knee extension (full extension being 0°). Both a rapid extension of the knee was used (popliteal grab, POP_{grab}) and slow stretch maximum extension (popliteal angle, POP_{angle}). Ankle dorsiflexion was measured in full hip and knee extension (PF_{Kext}), and in hip and knee flexion beyond 90° (PF_{Kflex}).

Computerized gait analysis was performed on each subject using a 6-camera VICON 370 system with two AMTI force plates. 13 reflective markers were placed on the lower extremities in accordance to the model described by Vicon Clinical Manager (VCM). Data were the processed with VCM and three representative trials were averaged together. Five variables were extracted from the kinematic and kinetic data from one side chosen at random for each individual. These were maximum hip extension (HE_{max}), knee extension in single limb stance (KE_{max}), knee extension in terminal swing ($KEtsw_{max}$), plantarflexor moment in early stance (PFM_{early}) and ankle push-off energy (A2E).

The effect of the contractures determined from the physical exam on the active motion of the joints during gait was determined using simple and multiple linear regression using StatView. One limb was randomly chosen to be evaluated from each individual's barefoot appliance-free gait. The alpha level was set at $p < .05$ a priori.

Results

Significant relationships were detected for various passive ROM measurements, but no more that 20% of the variance could be explained for any dynamic gait variable. The relationships worsened when restricting the analysis to only children with spastic diplegia. The data is summarized in table form below.

		Dynamic Gait Parameters				
Passive ROM		HE_{max}	KE_{max}	$KEtsw_{max}$	PFM_{early}	A2E
HF_{cont}	p	<.0001	.0102	.0178	.6548	.0017
	R^2	.185	.065	.056	.002	.096
	n	100	100	100	99	100
POP_{angle}	p	.3322	.2647	.0003	.0016	.0304
	R^2	.009	.012	.119	.094	.045
	n	105	105	105	104	105
POP_{grab}	p	.1482	.0622	.0443	.0016	.2285
	R^2	.020	.033	.039	.094	.014
	n	105	105	105	104	105
PF_{Kext}	p	<.0001	.1319	.0006	.0052	.0005
	R^2	.197	.023	.111	.077	.115
	n	101	101	101	100	101
PF_{Kflex}	p	<.0001	.6703	.0778	.0218	.0021
	R^2	.159	.002	.030	.051	.089
	n	104	104	104	103	104

Discussion

It is clear from the data that no passive ROM measurement definitively predicts dynamic motion in gait. Although significant probabilities were derived in many cases indicating a relationship between variables, R^2 values were extremely low, suggesting that other factors had larger influences on dynamic motion than did contractures of the hip, knee or ankle in the sagittal plane.

In determining treatment for gait abnormalities, it is insufficient to simply measure the contracture and assume its effect on dynamic posture in gait. For example, crouch gait has been attributed to hip flexion contractures, tight hamstrings, weak plantarflexors, or some combination of these. However when adding all these variables into a model which predicts knee flexion in single limb stance, only 10.3% of the variance was explained. While it is clear that these factors may be the cause of crouch in some individuals, not all crouch can be accounted for by these factors. Apparently, many other factors are influencing knee flexion in single limb stance, and other possible causes must be evaluated in order that appropriate treatment choices are made.

Effectiveness of Strength Training in Spastic Cerebral Palsy

Diane L. Damiano, PhD PT, Mark F. Abel, MD
Motion Analysis and Motor Performance Laboratory
Kluge Children's Rehabilitation Center, University of Virginia

Introduction

Muscle strength is an essential component of normal motor control that is deficient in cerebral palsy (CP), yet until recently strength training programs were not advocated in this population. Previous studies have documented positive effects from strengthening(1,2); however, these focused solely on the knee musculature in ambulatory children and adolescents with mild to moderate spastic diplegia. Hence the clinical effectiveness of strengthening across multiple muscles and diagnostic categories in CP has not yet been established. The goal of this project was to expand the exercise program to include more and different muscles selected on the basis of weakness. Two distinct clinical sub-groups were targeted: children with spastic hemiplegia or moderate/severe spastic diplegia. We hypothesized that resistance training would increase strength and thereby improve gait and overall motor function. For the children with hemiplegia, it was additionally hypothesized that correcting strength asymmetry through training of the more affected side would reduce asymmetric use of the extremities during gait.