

# The importance of swing-phase initial conditions in stiff-knee gait

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## Abstract

The diminished knee flexion associated with stiff-knee gait, a movement abnormality commonly observed in persons with cerebral palsy, is thought to be caused by an over-active rectus femoris muscle producing an excessive knee extension moment during the swing phase of gait. As a result, treatment for stiff-knee gait is aimed at altering swing-phase muscle function. Unfortunately, this treatment strategy does not consistently result in improved knee flexion. We believe this is because multiple factors contribute to stiff-knee gait. Specifically, we hypothesize that many individuals with stiff-knee gait exhibit diminished knee flexion not because they have an excessive knee extension moment during swing, but because they walk with insufficient knee flexion velocity at toe-off. We measured the knee flexion velocity at toe-off and computed the average knee extension moment from toe-off to peak flexion in 17 subjects (18 limbs) with stiff-knee gait and 15 subjects (15 limbs) without movement abnormalities. We used forward dynamic simulation to determine how adjusting each stiff-knee subject's knee flexion velocity at toe-off to normal levels would affect knee flexion during swing. We found that only one of the 18 stiff-knee limbs exhibited an average knee extension moment from toe-off to peak flexion that was larger than normal. However, 15 of the 18 limbs exhibited a knee flexion velocity at toe-off that was below normal. Simulating an increase in the knee flexion velocity at toe-off to normal levels resulted in a normal or greater than normal range of knee flexion for each of these limbs. These results suggest that the diminished knee flexion of many persons with stiff-knee gait may be caused by abnormally low knee flexion velocity at toe-off as opposed to excessive knee extension moments during swing. © 2003 Elsevier Science Ltd. All rights reserved.

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## 1. Introduction

Stiff-knee gait is an abnormal movement pattern observed in many persons with cerebral palsy. It is characterized by diminished and delayed peak knee flexion during the swing phase of the gait cycle. This pattern inhibits toe clearance, causing patients to trip or adopt energy-inefficient compensatory movements (Sutherland and Davids, 1993). The diminished knee flexion is commonly thought to be caused by an excessive knee extension moment generated by inappropriate swing-phase activity of the rectus femoris muscle (Perry, 1987; Sutherland et al., 1990). Accordingly, stiff-knee gait is often treated surgically with a rectus femoris transfer, in which the insertion of the rectus femoris is released from the patella and

transferred to a location behind the knee (Gage et al., 1987; Perry, 1987). Unfortunately, the outcomes of this procedure are inconsistent; some patients benefit from significant improvements in knee flexion while others do not improve.

While the aim of current treatments for stiff-knee gait is to compensate for over-activity of the rectus femoris muscle during swing phase, there is evidence that other factors may be involved. Dynamic analysis of stiff-knee gait in individuals following stroke has shown that increasing the swing-phase hip flexion moment can significantly increase the range of motion of the knee (Kerrigan et al., 1998). This finding suggests that weak hip flexors may be a factor in stiff-knee gait. Abnormal foot kinematics at toe-off have also been linked to stiff-knee gait (Kerrigan et al., 1991). A case study of a stroke subject with stiff-knee gait described substantial improvement in knee flexion of the affected limb after prescription of an ankle-foot orthosis and strengthening of the plantar flexors (Kerrigan and Glenn, 1994). These

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findings indicate that abnormal kinematic conditions at toe-off, the initial conditions of swing phase, contribute to the diminished knee flexion associated with stiff-knee gait.

The importance of swing-phase initial conditions in generating knee flexion during normal gait has been demonstrated by Mochon and McMahon (1980) and Mena et al. (1981). These studies used computer models to simulate near-normal hip and knee kinematics in the absence of muscular joint moments by choosing appropriate kinematic conditions at toe-off. The influence of swing-phase initial conditions on knee flexion has also been demonstrated in models that include muscle activity. Piazza and Delp (1996) used a muscle-driven simulation of normal swing-phase dynamics to show that peak knee flexion in swing can be decreased by increased knee extension moment, decreased hip flexion moment, decreased knee flexion velocity at toe-off, and increased hip flexion velocity at toe-off.

The purpose of the current study is to clarify the role that knee flexion velocity at toe-off plays in stiff-knee gait associated with cerebral palsy. We hypothesize that some stiff-knee subjects do not have excessive knee extension moments during swing, but instead exhibit abnormal kinematic conditions at toe-off, particularly low knee flexion velocity. We tested this hypothesis by comparing kinematic conditions at toe-off and joint moments in early swing phase from stiff-knee subjects to those from able-bodied subjects. We also used subject-specific forward dynamic simulation of stiff-knee gait to determine the extent by which the combination of a subject's abnormal joint moments with a normal knee flexion velocity at toe-off could increase knee flexion during swing phase.

## 2. Materials and methods

The experimental gait data used in this study were collected at Connecticut Children's Medical Center in Hartford, CT as part of the routine treatment-planning process. Eighteen limbs in 17 stiff-knee subjects with cerebral palsy were analyzed. The inclusion criteria for this study required that each subject (i) exhibited a knee flexion range of motion from toe-off to peak flexion in the affected limb(s) that was less than 80% of normal, (ii) was a candidate for rectus femoris transfer surgery due to the affected limb exhibiting (a) a positive score on the Ely test for quadriceps spasticity (Bleck, 1987), (b) abnormal rectus femoris activity during swing, or (c) diminished or delayed peak knee flexion in swing (DeLuca et al., 1997), (iii) exhibited gait deviations primarily in the sagittal plane, (iv) was aged 6 years or older, (v) had not undergone a selective dorsal rhizotomy, and (vi) did not require orthoses or other assistance to walk. Gait deviations were determined to

be primarily in the sagittal plane if the hip abduction, pelvic tilt, and pelvic rotation of the affected limb remained within two standard deviations of normal for the period between toe-off and peak knee flexion. Control data were collected in the same laboratory from the right limbs of fifteen able-bodied subjects of approximately the same average age, height, and weight as the stiff-knee subjects (Table 1). All subjects gave informed consent for the collection of their gait data. Retrospective analysis of these data was performed in accordance with the regulations of both participating institutions.

All subjects underwent a routine clinical exam and gait analysis, including three-dimensional kinematic and kinetic analyses. Motion data were collected using a three-camera motion measurement system as described by Davis et al. (1991), and each subject's three-dimensional gait kinematics were computed using methods described by Kadaba et al. (1990). The kinematic conditions at toe-off were determined from these data. Toe-off was defined as the point at which the ground reaction force dropped below 5% of the subject's body weight.

A subject-specific model of the swing limb was constructed for each subject. The limb was modeled as a compound pendulum, consisting of thigh, shank, and foot segments, suspended from a translating hip (Fig. 1). All segments were connected by frictionless hinge joints, permitting motion in only the sagittal plane. A model was created for each subject by specifying the measured hip trajectory of the subject's pelvis during swing phase and by adjusting the segment lengths and inertial

Table 1  
Characteristics of able-bodied and stiff-knee subjects

Subject	Gender (m/f) <sup>a</sup>	Age (years)	Height (cm)	Weight (kg)
Able-bodied <sup>b</sup>	7m/8f	10.5 (3.0)	140 (19)	35.2 (13.7)
Stiff-knee <sup>b</sup> (SK)	11m/6f	10.6 (2.4)	137 (14)	32.0 (9.4)
SK-1	M	12.3	138	30.4
SK-2	F	8.8	126	20.6
SK-3	M	11.4	142	29.1
SK-4	M	14.5	160	51.4
SK-5	M	14.3	155	47.1
SK-6	F	9.1	127	32.2
SK-7	M	11.8	147	38.1
SK-8	M	14.3	161	47.8
SK-9	M	8.8	124	30.8
SK-10	M	6.1	110	20.4
SK-11	F	10.4	138	29.9
SK-12	M	8.2	124	22.2
SK-13	F	10.8	137	28.5
SK-14	M	7.9	126	24.9
SK-15	F	10.8	137	28.9
SK-16	M	9.3	130	25.4
SK-17	F	11.0	138	38.1

<sup>a</sup> Male/female.

<sup>b</sup> Values shown are the average (standard deviation).

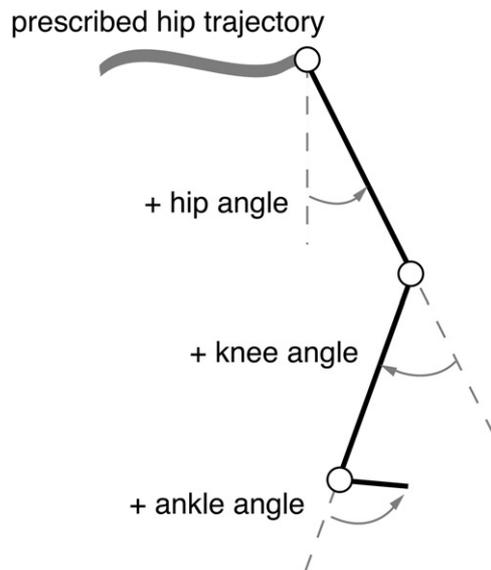


Fig. 1. Planar three-segment model of the swing limb. The limb was modeled as a compound pendulum suspended from a hip translating with a prescribed trajectory defined by the subject's measured kinematics. The model was made subject-specific by specifying the hip trajectory, segment lengths and inertial properties for each subject.

properties to match those of the subject. Segment lengths, estimated from marker positions, and subject weight were used to calculate the segment inertial properties based on the work of Jensen (1989) and Winter (1990).

Langrangian dynamics were used to formulate the equations of motion for the swing limb model, as published previously (Piazza and Delp, 1996). Quintic splines were fit to each subject's joint angle data so that the data could be differentiated to yield joint angular velocities and accelerations. These kinematics were used in an inverse dynamics analysis to calculate the moments about the hip, knee, and ankle. The average knee extension and hip flexion moments from the beginning of swing phase to the time of peak knee flexion were calculated for each limb. This time period, which we define as early swing phase, was chosen because it encompasses the portion of swing during which knee flexion occurs. These average moments were then normalized by subject weight. The resulting average early swing-phase knee extension moment ( $M_k$ ) for each stiff-knee subject was compared to the average of the  $M_k$  values for all able-bodied subjects.

A forward dynamic simulation was used to determine the kinematics that would result if the stiff-knee subjects walked with normal kinematic conditions at toe-off. When each limb's measured initial conditions and calculated moments were used as input to the forward dynamic simulation, the resulting kinematics

matched those measured experimentally (average root mean square difference for each joint angle was  $<0.005^\circ$ ), demonstrating the consistency of the inverse and forward simulations. For each of the stiff-knee limbs, the kinematic conditions at toe-off (knee flexion velocity, hip flexion velocity, knee flexion angle, and hip flexion angle) were sequentially adjusted to the average normal value, and the resulting knee kinematics were simulated. The range of knee flexion, from initial to peak flexion, was measured for the simulated kinematics and compared to the normal range. For all analyses, a parameter was considered above or below normal if it was more than one standard deviation from the average value for the able-bodied subjects.

### 3. Results

Only one of the subjects with stiff-knee gait exhibited an average early swing-phase knee extension moment that was larger than normal (Table 2, SK-1). Of the remaining 17 stiff-knee limbs, five exhibited average knee extension moments in the normal range and 12 exhibited values below normal. Average early swing-phase hip flexion moments were found to be within the normal range for 12 of the 18 stiff-knee limbs and below normal for five limbs.

Fifteen of the 18 stiff-knee limbs exhibited a knee flexion velocity at toe-off that was below normal (Table 2). Five of the stiff-knee limbs exhibited knee flexion velocities that were more than three standard deviations below normal. Most stiff-knee subjects exhibited other swing-phase initial condition values that were also more than one standard deviation below normal: 12 limbs exhibited a lower than normal initial hip flexion velocity, and initial hip and knee flexion angles were each below normal for 10 and four limbs, respectively. Our simulation studies showed that, of these kinematic conditions, the initial knee flexion velocity had the largest influence on range of knee flexion during swing, and thus further analysis focused on this variable.

Increasing the knee flexion velocity at toe-off to the average normal value resulted in a range of knee flexion, as measured from initial to peak flexion, that was normal or greater than normal for 15 of the 18 stiff-knee limbs (Table 2). For all of these 15 stiff-knee limbs, the magnitude of the increase in the range of knee flexion was larger than one standard deviation of the normal range ( $7^\circ$ ), with eight stiff-knee limbs exhibiting an increase of more than four times the normal standard deviation. While increasing the knee flexion velocity at toe-off did increase the range of knee flexion for all of these subjects, the timing of peak knee flexion remained delayed (Fig. 2).

Table 2  
Gait parameters of interest

Subject <sup>a,b</sup>	Limb	Average <sup>c</sup> early swing-phase knee extension moment, $M_k$ (Nm/kg)	Knee flexion velocity at toe-off, $V_k$ (°/s)	Experimental range <sup>d</sup> of knee flexion (°)	Simulated <sup>c</sup> range <sup>d</sup> of knee flexion (°)	Average <sup>c</sup> early swing-phase hip flexion moment, $M_h$ (Nm/kg)	Walking velocity (m/s)
Able-bodied <sup>f</sup>	Right	0.095 (0.063)	336 (70)	32 (7)	32 (7)	0.260 (0.140)	1.19 (0.13)
SK-1	Left	0.167	78	11	54	0.660	1.27
SK-2	Right	0.149	358	24	23	0.273	1.48
SK-3	Right	0.078	226	23	41	0.266	1.15
SK-4	Right	0.056	176	17	42	0.290	1.03
SK-5	Right	0.055	105	6	44	0.231	1.14
SK-6	Left	0.041	56	20	68	0.208	1.14
SK-7	Right	0.031	240	25	39	0.146	1.08
SK-8	Left	0.026	143	20	51	0.175	1.01
SK-9	Left	0.024	204	18	39	0.177	1.44
SK-10	Right	0.022	321	20	22	0.121	0.88
SK-11	Left	0.018	251	19	33	0.100	1.11
SK-12	Right	0.013	208	19	38	0.120	0.98
SK-13	Left	0.004	137	18	48	0.135	0.96
SK-9	Right	0.000	148	23	61	0.100	1.44
SK-14	Left	-0.003	341	18	18	0.110	1.17
SK-15	Left	-0.004	174	22	44	0.106	0.95
SK-16	Right	-0.009	63	22	65	0.133	0.89
SK-17	Left	-0.017	42	6	76	0.186	0.93

<sup>a</sup>SK indicates stiff-knee subject.

<sup>b</sup>SK subjects are listed in order of decreasing average early swing-phase knee extension moment.

<sup>c</sup>Averaged from time of toe-off to time of peak swing-phase knee flexion.

<sup>d</sup>Range measured from toe-off to peak swing-phase knee flexion.

<sup>e</sup>Simulation performed with a combination of normal knee flexion velocity at toe-off and subject's own joint moments.

<sup>f</sup>Values shown are the average (standard deviation).

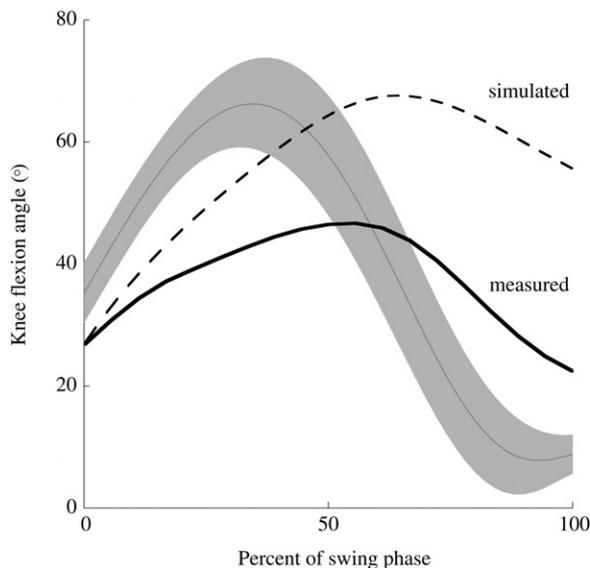


Fig. 2. An example of a subject's experimentally measured knee flexion compared to the result of a forward simulation in which the subject's knee flexion velocity at toe-off was raised to the average normal value. This change in initial knee flexion velocity resulted in a large increase in both the peak and range of knee flexion in swing. However, typical of most of our subjects, the peak remained delayed. The gray curve represents normal knee flexion kinematics  $\pm$  one standard deviation.

#### 4. Discussion

All of the stiff-knee limbs analyzed in this study demonstrated excessive rectus femoris activity during the swing phase and/or scored positively on the Ely test for quadriceps spasticity. Accordingly, all of the stiff-knee limbs were recommended to receive rectus femoris transfer surgery to alleviate the presumed excessive swing-phase knee extension moment produced by this muscle. However, we found that only one stiff-knee limb exhibited average knee extension moments that were larger than normal. Thus, the link commonly made between observed rectus femoris activity, excessive swing-phase knee extension moment, and stiff-knee gait may need to be refined. Since excessive knee extension moments in swing are thought to be the primary cause of stiff-knee gait, their absence in the majority of stiff-knee subjects we studied suggests that other factors contributed to diminished knee flexion in these cases. Our results show that low knee flexion velocity at toe-off was one of these factors.

All but three of the stiff-knee limbs exhibited a knee flexion velocity at toe-off that was more than one standard deviation below normal. A simulated increase in knee flexion velocity at toe-off to the normal value

resulted in a normal or greater than normal range of knee flexion for all 15 limbs that exhibited diminished knee flexion velocity at toe-off. Five limbs exhibited average early swing-phase hip flexion moments during swing that were below normal. Since the hip flexion moment promotes knee flexion (Piazza and Delp, 1996), insufficient hip flexion moments may also have contributed to diminished knee flexion for some subjects.

It is important to consider the limitations of our analysis. We used a planar model to represent the motion of the swing limb. While normal swing phase is often described as primarily a sagittal-plane motion, this approximation may not be appropriate for subjects with movement abnormalities. The stiff-knee subjects in this study were chosen such that they exhibited their primary kinematic deviations in the sagittal plane. However, some subjects did exhibit a compensatory circumduction of the hip, as is commonly associated with stiff-knee gait. Our model was unable to account for this motion.

Subjects' joint moments were calculated from their measured kinematic and kinetic data using inverse dynamics. This method requires the differentiation of measured joint angles, a process that is known to be sensitive to data smoothing techniques (Wood, 1982; Woltring, 1995). The implementation of spline fitting as our data smoothing technique may have affected the magnitude of the moment values we calculated. However, since both the able-bodied and stiff-knee subject data were processed using the same methodology, it is unlikely that the relative difference between the able-bodied and stiff-knee values was sensitive to the smoothing technique we chose.

We found that nearly all of the stiff-knee limbs exhibited knee flexion velocities below normal and knee extension moments that were normal or below normal. Our study examined only a small group of subjects, preventing us from being able to predict what percentage of the stiff-knee patient population follows this pattern. However, the consistency of our observations suggests that biomechanical factors other than an excessive knee extension moment during swing phase should be considered when treating stiff-knee gait.

We considered a parameter to be above or below normal if it was more than one standard deviation away from the average value for able-bodied subjects. It is worth examining whether our conclusions would have changed had we chosen either a more inclusive or more limited definition of normal. Had we chosen two standard deviations as our criterion, we would have classified all stiff-knee subjects as having an average early swing-phase knee extension moment within the normal range and ten of the eighteen limbs as exhibiting a knee flexion velocity that was below normal. Had we chosen half of one standard deviation as our criterion, one additional limb would have been characterized as having an early swing-phase knee extension moment

that was larger than normal. Thus, our definition of normal did not influence our general conclusion that reduced knee flexion often arises from lower than normal knee flexion velocity at toe-off as opposed to higher than normal knee extension moments.

Our finding that some stiff-knee subjects with cerebral palsy do not have excessive knee extension moments during swing phase is consistent with the findings of Riley and Kerrigan (1998) and Kerrigan et al. (1998), who used forward dynamic simulation of stroke subjects with stiff-knee gait to investigate the influence of the knee extension moment on knee flexion during swing. While Riley and Kerrigan (1998) concluded that swing-phase knee flexion range of motion is sensitive to changes in the knee extension moment, Kerrigan et al. (1998) found that simulations in which they halved the knee extension moment of their subjects did not result in a large change in knee flexion range of motion. This suggests that the measured knee extension moments were not large, thus decreasing them by 50% in the simulation did not result in a large change in knee flexion. Our observation that stiff-knee subjects exhibit a lower than normal knee flexion velocity at toe-off is consistent with observations made by Granata et al. (2000) in a study of 40 patients diagnosed with spastic cerebral palsy.

Stiff-knee subjects tend to walk more slowly than able-bodied subjects. It has been observed that both the knee flexion velocity at toe-off (from slopes in Fig. 3 of Murray et al. 1984; Winter, 1991) and the swing-phase knee moment (Prilutsky et al., 1998; Nene et al., 1999; Winter, 1991) decrease as walking speed decreases. In particular, data published by Winter (1991) show that a 25% decrease in walking speed corresponds to a 13% decrease in the knee flexion velocity at toe-off and a 45% decrease in the average early swing-phase knee extension moment (calculated from data in Fig. 3.33d and Tables 4.24a and 4.24b in Winter 1991). In this study the average walking speed of the stiff-knee subjects ( $109 \pm 18$  cm/s) was only 8% lower than that of the able-bodied subjects ( $119 \pm 14$  cm/s) (Table 2). If the normal knee flexion velocity at toe-off and normal average early swing-phase knee extension moment values are decreased by the percentages corresponding to an 25% decrease in walking speed, our general conclusions would not change; 12 (as opposed to 15) of the 18 stiff-knee limbs would exhibit knee flexion velocities at toe-off that are below normal and two (as opposed to one) limb would exhibit average early swing-phase knee extension moments that are larger than normal. No subject walked at a speed more than 25% below normal. Therefore, we feel that the conclusions drawn from our analysis were not affected by the walking speed of our subjects.

Our results indicate that many stiff-knee subjects with cerebral palsy do not walk with excessive swing-phase

knee extension moments. This suggests that the conventional rationale for performing a rectus femoris transfer, to eliminate excessive swing phase knee extension moment, is not appropriate for some subjects. Instead, we found that many stiff-knee subjects walk with insufficient knee flexion velocity at toe-off. Since kinematic conditions at toe-off are determined by stance-phase activity, stiff-knee gait, for some subjects, may be caused by abnormal muscle activity during stance phase. Thus, it may be excessive knee extension moments in stance, not swing, that are responsible for stiff-knee gait. Further, it is possible that excessive activity of the rectus femoris during stance contributes to diminished knee flexion velocity at toe-off, and that the rectus femoris transfer may be effective in addressing this cause of diminished knee flexion for some subjects. Our future work will investigate the link between stance-phase muscle activity, kinematic conditions at toe-off, and subsequent swing-phase knee flexion. This framework will enable clinicians to clearly identify the causes of diminished knee flexion and to design more effective treatments for stiff-knee gait.

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### References

- Bleck, E.E., 1987. Orthopaedic Management in Cerebral Palsy. Mac Keith, London, pp. 51–53.
- Davis, R.B., Öunpuu, S., Tyburski, D., Gage, J.R., 1991. A gait analysis data collection and reduction technique. *Human Movement Science* 10, 575–587.
- DeLuca, P.A., Davis, R.B., Öunpuu, S., Rose, S., Sirkin, R., 1997. Alterations in surgical decision making in patients with cerebral palsy based on three-dimensional gait analysis. *Journal of Pediatric Orthopaedics* 17, 608–614.
- Gage, J.R., Perry, J., Hicks, R.R., Koop, S., Werntz, J.R., 1987. Rectus femoris transfer to improve knee function of children with cerebral palsy. *Developmental Medicine and Child Neurology* 29, 159–166.
- Granata, K.P., Abel, M.F., Damiano, D.L., 2000. Joint angular velocity in spastic gait and the influence of muscle-tendon lengthening. *Journal of Bone and Joint Surgery A* 82, 174–186.
- Jensen, R.K., 1989. Changes in segment inertia proportions between 4 and 20 years. *Journal of Biomechanics* 22, 529–536.
- Kadaba, M.P., Ramakrishnan, H.K., Wootten, M.E., 1990. Measurement of lower extremity kinematics during level walking. *Journal of Orthopaedic Research* 8, 383–392.
- Kerrigan, D.C., Glenn, M.B., 1994. An illustration of clinical gait laboratory use to improve rehabilitation management. *American Journal of Physical Medicine and Rehabilitation* 73, 421–427.
- Kerrigan, D.C., Gronley, J., Perry, J., 1991. Stiff-legged gait in spastic paresis: a study of quadriceps and hamstrings muscle activity. *American Journal of Physical Medicine and Rehabilitation* 70, 294–300.
- Kerrigan, D.C., Roth, R.S., Riley, P.O., 1998. The modeling of adult spastic paretic stiff-legged gait swing period based on actual kinematic data. *Gait & Posture* 7, 117–124.
- Mena, D., Mansour, J.M., Simon, S.R., 1981. Analysis and synthesis of human swing leg motion during gait and its clinical applications. *Journal of Biomechanics* 14, 823–832.
- Mochon, S., McMahon, T.A., 1980. Ballistic walking. *Journal of Biomechanics* 13, 49–57.
- Murray, M.P., Mollinger, L.A., Gardner, G.M., Sepic, S.B., 1984. Kinematic and EMG patterns during slow, free, and fast walking. *Journal of Orthopaedic Research* 2, 272–280.
- Nene, A., Mayagoitia, R., Veltink, P., 1999. Assessment of rectus femoris function during initial swing phase. *Gait & Posture* 9, 1–9.
- Perry, J., 1987. Distal rectus femoris transfer. *Developmental Medicine and Child Neurology* 29, 153–158.
- Piazza, S.J., Delp, S.L., 1996. The influence of muscles on knee flexion during the swing phase of gait. *Journal of Biomechanics* 29, 723–733.
- Prilutsky, B.I., Gregor, R.J., Ryan, M.M., 1998. Coordination of two-joint rectus femoris and hamstrings during the swing phase of human walking and running. *Experimental Brain Research* 120, 479–486.
- Riley, P.O., Kerrigan, D.C., 1998. Torque action of two-joint muscles in the swing period of stiff-legged gait: a forward dynamic model analysis. *Journal of Biomechanics* 31, 835–840.
- Sutherland, D.H., Davids, J.R., 1993. Common gait abnormalities of the knee in cerebral palsy. *Clinical Orthopaedics*, 139–147.
- Sutherland, D.H., Santi, M., Abel, M.F., 1990. Treatment of stiff-knee gait in cerebral palsy: a comparison by gait analysis of distal rectus femoris transfer versus proximal rectus release. *Journal of Pediatric Orthopaedics* 10, 433–441.
- Winter, D.A., 1990. *Biomechanics and Motor Control of Human Movement*. Wiley, Waterloo, Canada, pp. 56–57.
- Winter, D.A., 1991. *The Biomechanics and Motor Control of Human Gait: Normal, Elderly, and Pathological*. Waterloo Biomechanics, Waterloo, pp. 1–52.
- Woltring, H.J., 1995. Smoothing and differentiation techniques applied to 3-D data. In: Allard, P., Stokes, I.A.F., Blanchi, J.-P. (Eds.), *Three-dimensional Analysis of Human Movement*. Human Kinetics, Champaign, IL, pp. 79–99.
- Wood, G.A., 1982. Data smoothing and differentiation procedures in biomechanics. *Exercise and Sport Science Review* 10, 308–362.