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The neuromuscular demands of toe walking: A forward dynamics simulation analysis

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Abstract

Toe walking is a gait deviation with multiple etiologies and often associated with premature and prolonged ankle plantar flexor electromyographic activity. The goal of this study was to use a detailed musculoskeletal model and forward dynamical simulations that emulate able-bodied toe and heel-toe walking to understand why, despite an increase in muscle activity in the ankle plantar flexors during toe walking, the internal ankle joint moment decreases relative to heel-toe walking. The simulations were analyzed to assess the force generating capacity of the plantar flexors by examining each muscle's contractile state (i.e., the muscle fiber length, velocity and activation). Consistent with experimental measurements, the simulation data showed that despite a 122% increase in soleus muscle activity and a 76% increase in gastrocnemius activity, the peak internal ankle moment in late stance decreased. The decrease was attributed to non-optimal contractile conditions for the plantar flexors (primarily the force–length relationship) that reduced their ability to generate force. As a result, greater muscle activity is needed during toe walking to produce a given muscle force level. In addition, toe walking requires greater sustained plantar flexor force and moment generation during stance. Thus, even though toe walking requires lower peak plantar flexor forces that might suggest a compensatory advantage for those with plantar flexor weakness, greater neuromuscular demand is placed on those muscles. Therefore, medical decisions concerning whether to reduce equinus should consider not only the impact on the ankle moment, but also the expected change to the plantar flexor's force generating capacity.

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1. Introduction

Toe walking is a gait deviation with multiple etiologies ranging from cerebral palsy to traumatic brain injury. It is often associated with premature and prolonged ankle plantar flexor electromyographic (EMG) activity (e.g., Colborne et al., 1994; Kalen et al., 1986), spasticity (e.g., Cahan et al., 1989; Perry et al., 1974) and contractures (e.g., Kelly et al., 1997; Stricker and Angulo, 1998). The increased plantar

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flexion posture can compromise walking stability and often results in decreased stride length and walking speed (Cahan et al., 1989; Davids et al., 1999; Hicks et al., 1988).

Recently, it has been proposed that toe walking provides a compensatory advantage over conventional heel-toe walking (Hampton et al., 2003; Kerrigan et al., 2000). Kerrigan et al. (2000) performed an inverse dynamics-based analysis of able-bodied subjects during heel-toe and toe walking and observed a significant decrease in the peak internal ankle plantar flexor moment and power generated in terminal stance and pre-swing during toe walking. They concluded that toe walking may provide a benefit for those with upper

motor neuron injury and distal lower extremity weakness by requiring lower ankle plantar flexor strength. Similarly, Hampton et al. (2003) performed a quasistatic analysis of the foot and tibia using data recorded from able-bodied subjects emulating toe walking postures of individuals with cerebral palsy. Their results showed that the increased equinus posture results in reduced plantar flexor force requirements. The reduced plantar flexor force (primarily from the gastrocnemius and soleus) was attributed to the closer proximity of the resultant ground reaction force vector to the ankle joint center with greater angles of plantar flexion. Similar to Kerrigan et al. (2000), they concluded that equinus walking is most likely a compensatory strategy for plantar flexor weakness.

While these studies noted a reduction in the plantar flexor force requirements during toe walking, Perry et al. (2003) demonstrated using fine wire electromyography that plantar flexor muscle activity during toe walking is greatly increased in late stance despite a reduced net plantar flexor moment. They hypothesized that the dichotomy between increased muscle activity and decreased joint moment was due to a reduction in force generating capacity of the ankle muscles because of greater plantar flexion angles during toe walking. Thus, although the peak plantar flexor force required during toe walking may be lower, the neuromuscular demand placed on the plantar flexors appears to be greater.

The isometric moment generation of the plantar flexors decreases with increasing plantar flexion angles (e.g., Gravel et al., 1988; Miyamoto and Oda, 2003; Nistor et al., 1982; Sale et al., 1982), despite an increase in the moment arm of the gastrocnemius and soleus about the ankle joint with increasing plantar flexion angles (Rugg et al., 1990). The decrease in moment output is attributed to the muscles operating at nonoptimal lengths on the muscle fiber force-length relationship. Thus, the increased ankle plantar flexion angle during toe walking likely reduces the forcegenerating capacity of the muscles. In addition, walking contains periods of both shortening and lengthening contractions of the plantar flexor muscles. Therefore, the muscle force generating capacity during walking could be impacted by both changes in fiber length as well as velocity. The poor contractile conditions associated with increased plantar flexion angles was recently highlighted in a modeling and simulation study showing that as ankle plantar flexion increases with walking speed, the force generating capacity of the plantar flexors becomes increasingly impaired (Neptune and Sasaki, 2005).

The goal of the present study was to explicitly test the hypothesis that greater angles of plantar flexion during toe walking are associated with a lower force generating capacity of the ankle plantar flexors compared to the normal posture in heel-toe gait. A detailed musculoskeletal model and forward dynamical simulations of ablebodied toe and heel-toe walking were developed to assess the force generating capacity of the plantar flexors by examining each muscle's contractile state. Specifically, we examined the muscle fiber length, velocity and activation relationships during toe and heel-toe walking to assess whether toe walking requires greater neuromuscular effort.

2. Methods

2.1. Forward dynamical simulations

Forward dynamical simulations of toe and heel-toe walking were generated using a previously described musculoskeletal modeling and dynamic optimization framework (e.g., Neptune et al., 2004) to analyze the contractile state and force production of the ankle plantar flexors. The sagittal-plane biped musculoskeletal model was developed using SIMM (MusculoGraphics Inc., Evanston, IL, USA) and the model's equations of motion were generated using SD/FAST (PTC, Needham, MA, USA). The equations of motion were then incorporated into simulation code generated by the Dynamics Pipeline (MusculoGraphics Inc., Evanston, IL, USA). Details of the musculoskeletal model and dynamic optimization that were used to produce the forward dynamical simulations emulating experimentally collected kinesiological data of able-bodied heeltoe and toe walking are described below.

2.2. Musculoskeletal model

The musculoskeletal model included a trunk (head, arms and torso combined as one segment) and right and left legs (each leg containing a femur, tibia, patella and foot) (Fig. 1). Degrees-of-freedom for the model included hip, knee and ankle flexion/extension for both legs, and trunk horizontal and vertical translation and anterior/posterior tilting. The knee flexion angle was used to prescribe two translational degrees-of-freedom of the knee joint (Yamaguchi and Zajac, 1989) and the position and orientation of the patella relative to the femur (Delp et al., 1990). The foot consisted of three segments: hindfoot, forefoot and toes. Flexion/extension was allowed between the hindfoot and forefoot and the forefoot and toes. Passive stiffness torques were applied at these joints so that realistic displacements were achieved during mid-stance. The model had a total of 13 degrees-of-freedom. Thirty visco-elastic elements were attached to the bottom of each foot segment to model the contact between the foot and ground. Details of the foot-ground contact model and parameter values are provided in Neptune et al. (2000).

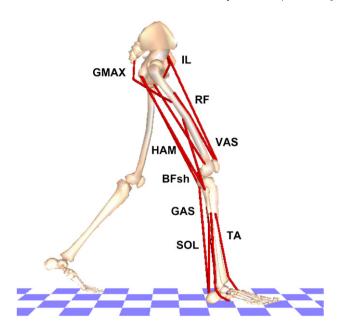


Fig. 1. The 2D-musculoskeletal model consisted of a HAT (head, arms and torso represented by the pelvis segment) and right and left legs (femur, tibia, patella and foot). The model was driven by seventeen Hill-type musculotendon actuators per leg that were combined into nine muscle groups based on anatomical classification, with muscles within each group receiving the same excitation pattern. The nine muscle groups were defined as IL (iliacus, psoas), GMAX (gluteus maximus, adductor magnus), VAS (three-component vastus), RF (rectus femoris), HAM (medial hamstrings, biceps femoris long head), BFsh (biceps femoris short head), SOL (soleus, tibialis posterior, peroneus longus), GAS (medial and lateral gastrocnemius) and TA (tibialis anterior). The muscle excitation-contraction dynamics were governed by Hill-type muscle properties.

Seventeen Hill-type musculotendon actuators including tendon compliance (Fig. 2) per leg were used to actuate the model. Individual muscle actuators were combined into nine muscle groups based on anatomical classification with muscles within each group receiving the same excitation pattern (Fig. 1). The muscle force generating capacity was governed by normalized forcelength and force-velocity relationships (Fig. 3), and a normalized non-linear tendon force-strain relationship to describe tendon force (Delp and Loan, 1995; Zajac, 1989). All musculotendon parameters were based on the work of Delp et al. (1990). Muscle activation-deactivation dynamics were represented with a first-order differential equation (Raasch et al., 1997) with activation and deactivation time constants of 50 and 65 ms, respectively. Passive joint torques representing ligaments and other connective tissues were used to limit the hip, knee and ankle joint range of motion at extreme joint angles (Davy and Audu, 1987).

2.3. Dynamic optimization

A simulated annealing optimization algorithm (Goffe et al., 1994) was used to fine-tune the muscle excitation

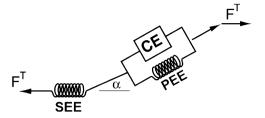


Fig. 2. Schematic of the musculotendon actuator used in the model. The properties of the musculotendon force generation $(F^{\rm T})$ were represented by an active contractile element (CE) in parallel with a passive elastic element (PEE). The muscle fiber was placed in series with tendon (SEE), which was represented by a non-linear elastic element. The pennation angle (α) denotes the angle between the muscle fibers and tendon. All musculotendon parameters, including the origin and insertion of each muscle, pennation angle, tendon slack length, resting fiber length and maximum isometric force were based on Delp et al. (1990).

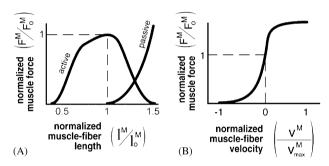


Fig. 3. Governing intrinsic muscle properties used in the model: (A) normalized force–length and (B) force–velocity relationships (Zajac, 1989). With deviations from the muscle fiber's optimal length (l_o^M) and increasing rates of shortening (V^M) , the ability of a muscle to produce force decreases. Note, negative velocity values indicate muscle shortening, F^M is the muscle force, F_o^M is the muscle's maximum isometric force, l_o is the fiber length and V_{\max}^M is the muscle's maximum shortening velocity.

patterns defined using block patterns by adjusting for each muscle the excitation onset, duration and magnitude until the difference between the experimental and simulated kinematic and kinetic data was minimized. The excitation magnitude was defined with four excitation nodes for all muscles, except the plantar flexors which were defined with six nodes to better represent their more complex EMG patterns (e.g., Perry et al., 2003). Constraints were placed on the excitation timing in the optimization to closely replicate the EMG timing in Perry et al. (2003) (i.e., EMG nominal values $\pm 10\%$ gait cycle) to assure that the muscles were generating force at the appropriate region in the gait cycle. The muscle excitation patterns were assumed to be symmetrical between the left and right legs. The specific tracking quantities used in the objective function included trunk translation and tilting, hip, knee and ankle joint angles and moments, and the anterior/ posterior and vertical ground reaction forces (GRF). The tracking data collected from the subjects' right side

was shifted 50% of the gait cycle to provide data for the left side.

2.4. Muscle contractile state

From the walking simulations, individual muscle fiber lengths and velocities over the gait cycle were determined from the model and normalized to the optimal fiber length and maximum contraction velocity of the muscle, respectively. The optimal fiber lengths were based on the work of Delp et al. (1990) and each muscle's maximum shortening velocity was estimated as 10 times the muscle fiber optimal length per second (Zajac, 1989). In addition, average musculotendon force and activation levels during each muscle's active region during stance and the average net internal ankle joint moment over the stance phase were quantified.

2.5. Experimental data collection

To provide initial conditions for the simulations and tracking data for the dynamic optimization, previously collected data from 10 able-bodied subjects (Perry et al., 2003; eight female, two male; age 36.9 ± 11.2 years; height 171.7 + 8.3 cm; mass 67.2 + 12.4 kg) were used and will be briefly summarized here. All subjects signed informed consent and all testing was conducted at the Pathokinesiology Laboratory at Rancho Los Amigos National Rehabilitation Center in Downey, CA. Each subject practiced toe walking until they were comfortable and achieved a consistent toe walking pattern. Each subject walked at their freely selected speed during toe walking while three-dimensional GRFs (Kistler Instrument Corp., Amherst, NY; 2500 Hz) and body segment motion data (Vicon Motion Systems, Oxford, UK; 50 Hz) were recorded from the right leg. Subjects then performed heel-toe walking at +5% of their toe walking speed while the same data were collected. The body segment motion data were measured using markers located over the sacrum, anterior superior iliac spine (bilaterally), greater trochanter, anterior thigh, medial and lateral femoral condyles, anterior tibia, medial and lateral malleoli, dorsum of the foot, first and fifth metatarsal heads, and posterior heel. All data were normalized over the gait cycle, averaged across trials, and then averaged across all subjects to produce a group average. Further details regarding the data collection and processing are provided in Perry et al. (2003).

3. Results

Consistent with our previous simulation analyses (e.g., Neptune et al., 2001; Neptune and Sasaki, 2005), the dynamic optimization was able to identify muscle excitation patterns such that the toe and heel-toe

walking simulations closely emulated the human subject body segment kinematic, ground reaction force and internal joint moment data. The walking speeds were 1.16 and 1.14 m/s for toe and heel-toe walking simulations, respectively. Of particular importance to the present study was the ability of the simulation to closely match the human subject ankle joint kinematics and internal moment profiles. The simulation was able to reproduce these quantities within ±2 standard deviations of the experimental data (Fig. 4). In both simulations, the timing of the plantar flexor activity compared well with the experimental EMG data (Fig. 4).

Despite a 122% increase in soleus (SOL) muscle activity during toe walking, the corresponding average SOL muscle force during stance decreased by 19% relative to heel-toe walking (Fig. 5). The gastrocnemius (GAS) muscle activity increased 76% during toe walking, however, this only resulted in a 37% increase in the average GAS muscle force (Fig. 5). The magnitude of the muscle forces was consistent with the net internal ankle joint moment. Despite the increase in plantar flexor activity during toe walking (Fig. 5), the peak ankle moment during late stance decreased slightly (Fig. 6). This reduction in the simulation ankle moment, despite the greater muscle activity, was attributed to a poor contractile state during toe walking, primarily due to the muscles operating at non-optimal muscle fiber lengths when the ankle was in greater plantar flexion (Fig. 4). The mean SOL fiber length normalized to its optimal fiber length during its active region in stance was 0.55 and 0.89 during toe and heel-toe walking, respectively (Fig. 7A). Similarly, the mean values for GAS were 0.59 and 0.90, respectively (Fig. 7A). Thus, both muscles were operating near the onset of the ascending limb of the force-length relationship (Fig. 3A), which greatly reduced their ability to generate force. In contrast, the mean fiber velocities were at low levels and similar in magnitude between the two walking tasks (Fig. 7B). Thus, the force-length relationship was the dominant intrinsic muscle property that decreased the force generating capacity of the plantar flexors during toe walking.

In addition to the greater intensity and duration of muscle excitation required for toe walking, maintaining the plantar flexed posture throughout stance required a 65% increase in the mean internal ankle moment during stance (Fig. 6: 1.11 and 0.68 N-m/kg for toe and heel-toe walking, respectively).

4. Discussion

The overall goal of this study was to use a detailed musculoskeletal model and forward dynamical simulations of able-bodied toe and heel-toe walking to

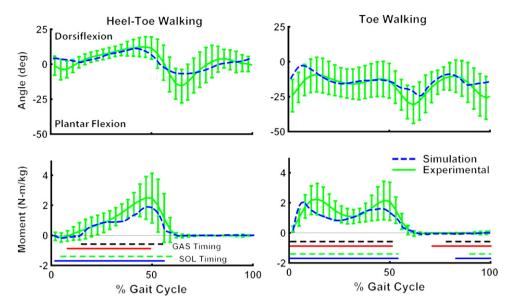


Fig. 4. Comparison of the simulation (dashed line) and experimental (solid line ± 2 standard deviations) ankle joint angle and internal moment profiles over the gait cycle. The horizontal bars indicate the soleus (SOL) and gastrocnemius (GAS) excitation timing in the simulation (dashed bar) compared to the experimental EMG timing (solid bar).

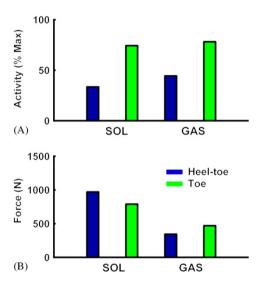


Fig. 5. (A) Average muscle activity from the soleus (SOL) and gastrocnemius (GAS) over the stance phase as a percentage of maximum. In both simulations, the muscles were active throughout the stance phase. (B) The corresponding average muscle forces during stance.

understand why, despite an increase in muscle activity in the ankle plantar flexors during toe walking, the internal ankle joint moment decreases. Specifically, we tested the hypothesis presented by Perry et al. (2003) that the dichotomy between increased muscle activity and decreased internal joint moment was due to a reduction in force generating capacity associated with shorter muscle fiber lengths at the increased plantar flexion angle observed during toe walking. To test this hypothesis, we assessed the force generating capacity

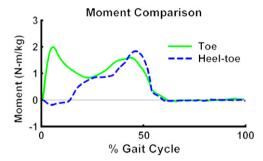


Fig. 6. Comparison of the internal ankle moment between toe and heel-toe walking over the gait cycle. Note, despite the substantial increase in plantar flexor activity, the peak moment in late stance decreased slightly.

of the plantar flexors by examining each muscle's contractile state (i.e., the muscle fiber length, velocity and activation) during toe and heel-toe walking.

Similar to Hampton et al. (2003) and Kerrigan et al. (2000), we found that the combined plantar flexor force and peak internal ankle plantar flexor moment were decreased during toe walking compared to heel-toe walking. Hampton et al. (2003), using a static decomposition model of the ankle joint moment derived from inverse dynamics, found that because the moment arm from the ground reaction force vector to the ankle joint center decreases with increasing plantar flexor angle, the required net force from the plantar flexor muscles also decreases. Kerrigan et al. (2000) used a similar biomechanical model and came to the same conclusion, that the reduced muscle force requirement in toe walking may provide a compensatory mechanism for those with plantar flexor weakness or other neuromus-

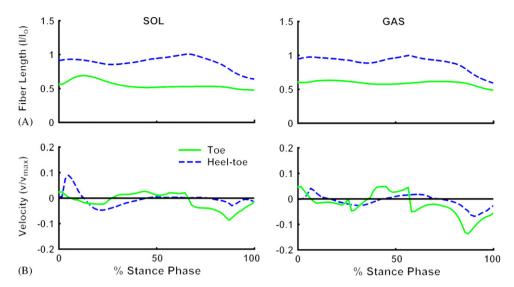


Fig. 7. Muscle fiber contractile state of the soleus (SOL) and gastrocnemius (GAS). (A) normalized muscle fiber length, and (B) normalized muscle fiber velocity. The muscle fibers (l) were normalized to their resting fiber length (l_o) and the fiber velocities (V) were normalized to their maximum shortening velocity ($V_{\text{max}}^{\text{M}}$), which was assumed to be $10*l_o/s$ (Zajac, 1989). Note, negative velocity values indicate muscle shortening.

cular disorders. Both of these studies, however, relied on inverse dynamics analysis which does not account for the contractile state of the muscles and its influence on the required neuromuscular demand.

In the current study, toe walking required greater muscle activation to achieve similar force and moment levels as those required during heel-toe walking. We observed a 122% increase in SOL activity and a 76% increase in GAS muscle activity (similar to the experimental EMG increases reported by Perry et al., 2003), despite documenting a decrease in the peak internal ankle plantar flexor moment during the propulsion phase in late stance (Fig. 6). In addition, toe walking required the duration of plantar flexor activity to increase over the entire gait cycle (Fig. 4; see also Perry et al., 2003; Rose et al., 1999), in order to provide the internal ankle moment necessary to maintain the plantar flexed posture from initial foot contact until the end of stance. Both experimental (e.g., Kerrigan et al., 2000; Perry et al., 2003) and our simulation data (Fig. 6) show that the sustained mean ankle moment over the stance phase was 65% higher in toe walking compared to heeltoe walking. The increased intensity and duration of muscle activity as well as the greater internal moment requirements for toe walking suggest that fatigue is another important factor that may contribute to inefficiency in toe walking.

The decrease in plantar flexor moment, despite an increase in muscle activity, is primarily the result of the shorter fiber lengths associated with the increased plantar flexion angle, rather than a change in fiber contraction velocities (Fig. 7). The decrease in force generating capacity with the increased plantar flexion angle is consistent with previous studies showing that,

despite an increase in the Achilles tendon moment arm as the foot moves from full dorsiflexion to full plantar flexion, the isometric moment generating capacity of the plantar flexor muscles decreases with increasing plantar flexion angle (e.g., Gravel et al., 1988; Miyamoto and Oda, 2003; Nistor et al., 1982; Sale et al., 1982).

Thus, our simulation analysis suggests that even though toe walking requires lower peak plantar flexor forces that might suggest a compensatory advantage for those with plantar flexor weakness, greater neuromuscular demand is placed on those muscles in order to overcome the poor contractile state and maintain the equinus posture. However, a potential limitation of our study is that we analyzed able-bodied subjects that could physically perform both heel-toe and toe walking tasks equally well to allow an objective comparison between the two walking tasks, rather than habitual toe walkers. It is possible that spasticity and prolonged equinus foot posture may alter the intrinsic muscle properties such that peak muscle force is generated at shorter fiber lengths. Previous studies have shown the muscle fibers in those with cerebral palsy are shorter than normal (Tardieu et al., 1982), have altered fiber types (Rose et al., 1994) and altered passive force-length relationships in subjects with chronic spasticity (Friden and Lieber, 2003). Thus, muscles may undergo significant remodeling such that the intrinsic force-length relationship may be significantly altered in subjects with chronic spasticity and equinus foot posture (Delp, 2003) that allows them to generate greater force at shorter fiber lengths. However, recent strength measurements in children with and without spastic diplegic cerebral palsy showed similar ankle torque-angle profiles with their maximum ankle torque values being generated at similar

plantar flexion angles (Engsberg et al., 2000; Ross and Engsberg, 2002). In contrast, earlier work by Tardieu and colleagues (Tardieu et al., 1982; Tardieu and Tardieu, 1986) demonstrated changes in the ankle torque–angle profiles. Thus, future work is needed to further assess muscle remodeling in chronic toe-walking patients and the extent intrinsic muscle properties are altered.

The results of the current study indicate that the force generating capacity of the plantar flexors is greatly reduced during toe walking due to non-optimal contractile conditions (primarily the force-length relationship). These results are of particular importance in light of recent modeling and simulation studies showing that the plantar flexors are important contributors to the body segment mechanical energetics in heel-toe walking by providing body support, forward propulsion and swing initiation during stance (e.g., Anderson and Pandy, 2003; Neptune et al., 2001; Zajac et al., 2003). However, it is unclear how equinus foot posture affects the ability of the plantar flexors to contribute to these important biomechanical functions and to what extent additional compensatory mechanisms are necessary to satisfy the energetic demands of toe walking. Further, it is not clear if toe walking provides any biomechanical advantage by allowing more elastic energy to be stored and released in the elastic structures crossing the ankle joint in such a way that it provides a biomechanical advantage relative to heel-toe walking (Kerrigan et al., 2000). Future work should address these questions by analyzing the biomechanical differences in muscle function between toe and heel-toe walking. Additionally, the impact of toe walking on walking stability also warrants further exploration.

As indicated by Perry et al. (2003), the clinical implications of understanding these relationships are critical. If toe walking increases the neuromuscular demand placed on the plantar flexors, then clinical decisions regarding the need to use therapeutic measures to reduce the equinus should consider not only the impact on internal joint moments, but also the expected change to the plantar flexor's force generating capacity.

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References

- Anderson, F.C., Pandy, M.G., 2003. Individual muscle contributions to support in normal walking. Gait Posture 17, 159–169.
- Cahan, L.D., Adams, J.M., Beeler, L.M., Perry, J., 1989. Clinical, electrophysiologic, and kinesiologic observations in selective dorsal

- rhizotomy in cerebral palsy. Neurosurgery State Art Review 4, 477-484.
- Colborne, G.R., Wright, F.V., Naumann, S., 1994. Feedback of triceps surae EMG in gait of children with cerebral palsy: a controlled study. Archives of Physical Medicine and Rehabilitation 75, 40–45.
- Davids, J.R., Foti, T., Dabelstein, J., Bagley, A., 1999. Voluntary (normal) versus obligatory (cerebral palsy) toe-walking in children: a kinematic, kinetic, and electromyographic analysis. Journal of Pediatric Orthopaedics 19, 461–469.
- Davy, D.T., Audu, M.L., 1987. A dynamic optimization technique for predicting muscle forces in the swing phase of gait. Journal of Biomechanics 20, 187–201.
- Delp, S.L., 2003. What causes increased muscle stiffness in cerebral palsy? Muscle Nerve 27, 131–132.
- Delp, S.L., Loan, J.P., 1995. A graphics-based software system to develop and analyze models of musculoskeletal structures. Computers in Biology and Medicine 25, 21–34.
- Delp, S.L., Loan, J.P., Hoy, M.G., Zajac, F.E., Topp, E.L., Rosen, J.M., 1990. An interactive graphics-based model of the lower extremity to study orthopaedic surgical procedures. IEEE Transactions of Biomedical Engineering 37, 757–767.
- Engsberg, J.R., Ross, S.A., Olree, K.S., Park, T.S., 2000. Ankle spasticity and strength in children with spastic diplegic cerebral palsy. Developmental Medicine and Child Neurology 42, 42–47.
- Friden, J., Lieber, R.L., 2003. Spastic muscle cells are shorter and stiffer than normal cells. Muscle Nerve 27, 157–164.
- Goffe, W.L., Ferrier, G.D., Rogers, J., 1994. Global optimization of statistical functions with simulated annealing. Journal of Econometrics 60, 65–99.
- Gravel, D., Richards, C.L., Filion, M., 1988. Influence of contractile tension development on dynamic strength measurements of the plantarflexors in man. Journal of Biomechanics 21, 89–96.
- Hampton, D.A., Hollander, K.W., Engsberg, J.R., 2003. Equinus deformity as a compensatory mechanism for ankle plantarflexor weakness in cerebral palsy. Journal of Applied Biomechanics 19, 325–330
- Hicks, R., Durinick, N., Gage, J.R., 1988. Differentiation of idiopathic toe-walking and cerebral palsy. Journal of Pediatric Orthopaedics 8, 160–163.
- Kalen, V., Adler, N., Bleck, E.E., 1986. Electromyography of idiopathic toe walking. Journal of Pediatric Orthopaedics 6, 31–33.
- Kelly, I.P., Jenkinson, A., Stephens, M., O'Brien, T., 1997. The kinematic patterns of toe-walkers. Journal of Pediatric Orthopaedics 17, 478–480.
- Kerrigan, D.C., Riley, P.O., Rogan, S., Burke, D.T., 2000. Compensatory advantages of toe walking. Archives of Physical Medicine and Rehabilitation 81, 38–44.
- Miyamoto, N., Oda, S., 2003. Mechanomyographic and electromyographic responses of the triceps surae during maximal voluntary contractions. Journal of Electromyography Kinesiology 13, 451–459.
- Neptune, R.R., Sasaki, K., 2005. Ankle plantar flexor force production is an important determinant of the preferred walk-to-run transition speed. Journal of Experimental Biology 208, 799–808.
- Neptune, R.R., Wright, I.C., van den Bogert, A.J., 2000. A method for numerical simulation of single limb ground contact events: application to heel-toe running. Computer Methods in Biomechanics and Biomedical Engineering 3, 321–334.
- Neptune, R.R., Kautz, S.A., Zajac, F.E., 2001. Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. Journal of Biomechanics 34, 1387–1398.
- Neptune, R.R., Zajac, F.E., Kautz, S.A., 2004. Muscle force redistributes segmental power for body progression during walking. Gait Posture 19, 194–205.

- Nistor, L., Markhede, G., Grimby, G., 1982. A technique for measurements of plantar flexion torque with the Cybex II dynamometer. Scandinavian Journal of Rehabilitation Medicine 14, 163–166.
- Perry, J., Hoffer, M.M., Giovan, P., Antonelli, D., Greenberg, R., 1974. Gait analysis of the triceps surae in cerebral palsy. A preoperative and postoperative clinical and electromyographic study. Journal of Bone and Joint Surgery America 56, 511–520.
- Perry, J., Burnfield, J.M., Gronley, J.K., Mulroy, S.J., 2003. Toe walking: muscular demands at the ankle and knee. Archives of Physical Medicine and Rehabilitation 84, 7–16.
- Raasch, C.C., Zajac, F.E., Ma, B., Levine, W.S., 1997. Muscle coordination of maximum-speed pedaling. Journal of Biomechanics 30, 595–602.
- Rose, J., Haskell, W.L., Gamble, J.G., Hamilton, R.L., Brown, D.A., Rinsky, L., 1994. Muscle pathology and clinical measures of disability in children with cerebral palsy. Journal of Orthopaedic Research 12, 758–768.
- Rose, J., Martin, J.G., Torburn, L., Rinsky, L.A., Gamble, J.G., 1999. Electromyographic differentiation of diplegic cerebral palsy from idiopathic toe walking: involuntary coactivation of the quadriceps and gastrocnemius. Journal of Pediatric Orthopaedics 19, 677–682.
- Ross, S.A., Engsberg, J.R., 2002. Relation between spasticity and strength in individuals with spastic diplegic cerebral palsy. Developmental Medicine and Child Neurology 44, 148–157.

- Rugg, S.G., Gregor, R.J., Mandelbaum, B.R., Chiu, L., 1990. In vivo moment arm calculations at the ankle using magnetic resonance imaging (MRI). Journal of Biomechanics 23, 495–501.
- Sale, D., Quinlan, J., Marsh, E., McComas, A.J., Belanger, A.Y., 1982. Influence of joint position on ankle plantarflexion in humans. Journal of Applied Physiology 52, 1636–1642.
- Stricker, S.J., Angulo, J.C., 1998. Idiopathic toe walking: a comparison of treatment methods. Journal of Pediatric Orthopaedics 18, 289–293.
- Tardieu, C., Huet de la Tour, E., Bret, M.D., Tardieu, G., 1982. Muscle hypoextensibility in children with cerebral palsy: I. clinical and experimental observations. Archives of Physical Medicine and Rehabilitation 63, 97–102.
- Tardieu, G., Tardieu, C., 1986. Mechanical evaluation and conservative correction of limb joint contractures. Clinical Orthopaedics and Related Research 219, 63–69.
- Yamaguchi, G.T., Zajac, F.E., 1989. A planar model of the knee joint to characterize the knee extensor mechanism. Journal of Biomechanics 22, 1–10.
- Zajac, F.E., 1989. Muscle and tendon: properties, models, scaling, and application to biomechanics and motor control. Critical Review of Biomedical Engineering 17, 359–411.
- Zajac, F.E., Neptune, R.R., Kautz, S.A., 2003. Biomechanics and muscle coordination of human walking. Part II: lessons from dynamical simulations and clinical implications. Gait Posture 17, 1–17.