ELSEVIER

Gait and Posture 16 (2002) 238–248

GAIT & POSTURE

www.elsevier.com/locate/gaitpost

Reactive balance adjustments to unexpected perturbations during human walking

Reed Ferber [a,*], Louis R. Osternig [b], Marjorie H. Woollacott [b], Noah J. Wasielewski [b], Ji-Hang Lee [b]

[a] *Department of Physical Therapy, University of Delaware, 301 McKinly Lab, Newark, DE 19716-2591, USA*
[b] *University of Oregon, Eugene, OR, USA*

Received 30 November 2001; accepted 8 January 2002

Abstract

The purpose of this investigation was to determine the effect of unexpected forward perturbations (FP) during gait on lower extremity joint mechanics and muscle Electromyographic (EMG) patterns in healthy adults. The muscles surrounding the hip were found to be most important in maintaining control of the trunk and preventing collapse in response to the FP. Distinct lower extremity joint moment and power patterns were observed in response to the FP but an overall positive moment of support (M_s) was maintained. Therefore, reactive balance control was a synchronized effort of the lower extremity joints to prevent collapse during the FP.

Keywords: Human; Locomotion; Perturbation; Balance

1. Introduction

Human gait is one of the most common forms of human movement, yet the underlying neurological and biomechanical processes by which this movement occurs are complex and individualistic. The inherent instability involved in human locomotion results from a relatively small base of support, long single support phase, and the observation that 2/3 of the body's mass is located in the head–arm–trunk (HAT) segments [1]. Successful locomotion requires three essential elements, (a) the ability to generate and maintain fundamental locomotor patterns appropriate for moving toward an intended destination; (b) maintenance of basic dynamic equilibrium between a shifting center of mass (COM) and a constantly changing base of support; and (c) the ability to change locomotor patterns in response to external or internal inertial changes that threaten dynamic equilibrium [2]. While the first element is concerned with the generation of complex locomotor patterns, the latter two are critical in the detection of potential threats to balance and the subsequent reaction to either foreseen or unexpected perturbations during normal gait.

Often, individuals cannot anticipate external threats to dynamic equilibrium during gait and reactive mechanisms are required to act after the person experiences an unexpected perturbation [3]. Relatively few investigations have studied reactive postural adjustments during gait in response to unexpected perturbations [4–9]. Nashner [4] incorporated a moveable platform into a walkway to simulate unexpected perturbations during gait. Electromyographic (EMG) recordings from the gastrocnemius (GAS) and tibialis anterior (TA) muscles were measured along with lower extremity joint angles. Unexpected forward translation or downward rotation perturbations applied at HS produced increased TA EMG activity in the perturbed leg and unexpected backward translation or upward rotation produced increased GAS EMG activity. These recordings were similar to those obtained during standing perturbations [4,10]. Based on the observation that platform perturbations resulted in altered foot trajectory and stretching of the muscle, Nashner [4] hypothesized that alterations in

* Corresponding author. Tel.: +1-302-831-4646; fax: +1-302-831-4234
E-mail address: reedferb@udel.edu (R. Ferber).

0966-6362/02/$ - see front matter
PII: S0966-6362(02)00010-3

distal limb trajectories provided the principal sensory feedback input to the central nervous system to elicit reactive balance control strategies.

Tang et al. [5] hypothesized that proximal muscles (hip and trunk) may play as important a role as do distal muscles (TA and GAS) in balance recovery. Using a paradigm similar to Nashner [4], EMG recordings were analyzed from the TA, GAS, rectus femoris (RF), biceps femoris (BF), rectus abdominis (RA), and erector spinae (ES). Their results indicated that hip and trunk muscles (RA, ES) did not play a significant role in reactive balance adjustments during perturbed gait at HS since these muscles did not demonstrate more consistent activation, earlier onset latency, longer burst duration, or larger burst magnitude compared with distal leg and thigh muscles. Leg and thigh muscles (TA, GAS, RF, BF) did, however, demonstrate earlier onset, higher magnitude, and relatively longer duration of activity as compared with normal walking. It was, therefore, concluded that activity from leg and thigh muscles was the primary contributor to reactive balance control.

Winter [1,11] suggested that hip muscle activity is critical during gait for control of the HAT segment. It was estimated that an ankle moment about eight times greater than that of the hip was needed to control the HAT segment due to the combined moments of inertia of the rest of the body. Furthermore, it was suggested that during early stance, as the HAT segment accelerates anteriorly, the ankle does not intervene but undergoes a small dorsiflexor moment, and it is the large hip extensor moment that serves to directly control the displacement of the HAT segment to maintain dynamic equilibrium. Winter [11] also documented high intra-subject and inter-subject variability of the hip moments across trials and testing times. It was postulated that the high variability in hip moments was necessary on a stride-to-stride basis in an attempt to control the HAT segment and that changes in hip moment patterns were equally matched by alterations in knee moment patterns. It was hypothesized that such a deterministic trade-off between the hip and the knee indicated a stride dependent control of the HAT segment to maintain the total moment of support (M_s) to prevent the body from collapsing due to gravitational forces [11].

There is a controversy surrounding the postural control mechanisms in response to changes in dynamic equilibrium during gait and a paucity of literature serving to explain reactive balance mechanisms to unexpected perturbations. Of those investigations, only EMG and kinematic data have been presented and no studies have calculated the joint moments necessary for understanding the relative joint contributions in maintaining dynamic equilibrium. Therefore, the purpose of this investigation was to determine the effect of unexpected forward perturbations (FP) during gait on lower extremity joint moments and muscle EMG patterns in healthy subjects.

It was hypothesized that the forward perturbation would result in a greater knee and hip joint flexion and greater ankle plantarflexion. As a result of the alterations in lower extremity joint positions, it was also hypothesized that the perturbed limb would demonstrate a greater knee and hip extensor moment and a reduced ankle plantarflexion moment and increased ankle, knee, and hip joint extensor muscle EMG activity.

2. Methods

2.1. Participants

Ten (five males and five females) healthy young adults participated in the study. The mean age, body mass, and body height of subjects were 24.4 year ($\pm$3.1 year), 67.2 kg ($\pm$10.7 kg), and 170.1 cm ($\pm$9.3 cm), respectively. All subjects were physically active, participating in regular activity at least three times per week. No subject had a prior history of lower extremity infirmity or pathology, or was suffering from any osteoarthritic or musculoskeletal disease at the time of testing that may have affected the ability to perform the experiment. Prior to participation, each subject signed a consent form approved by the University's Human Subjects Compliance Committee.

2.2. Protocol

Unexpected perturbations were induced as subjects walked along a 5 m wooden walkway in which a force plate, capable of translational movement, was embedded. When preset, the force plate moved anteriorly or posteriorly a distance of 10 cm at a velocity of 40 cm/s upon heel contact. The selected velocity was based on previous literature reporting heel velocities during realistic slip movements when a person is walking on a slippery surface [12].

The subjects walked at a self-selected comfortable pace that was maintained throughout data collection via a metronome. Each subject began walking at a sufficient distance from the force plate so that the self-selected pace was attained prior to the foot of the test limb making contact with the center of the force plate. Muscle EMG, joint kinematic and kinetic data were collected while the subjects walked along the walkway for a 5 s period, which included the step prior to and following contact with the force plate.

Data were recorded from 48 trials using the subject's right limb. The first 12 trials consisted of 'true control' non-perturbations (NP) trials to establish normal walking gait and muscle activation patterns. Following the

NP true control trials, 36 additional trials were performed consisting of 12 FP, 12 NP 'catch' trials, and 12 backward perturbations (BP). The BP condition was not analyzed and, in addition to the random order of trials, was used to help prevent possible accommodation and anticipation of the FP condition. Subjects were not allowed to practice the perturbation trials.

There was a small risk that the subjects could fall when their balance was perturbed. To minimize the risk, the subjects wore a harness attached to an overhead track and were provided a handrail to grasp if needed.

2.3. Instrumentation

EMG data were collected using bipolar surface electrodes (DE-02, Delsys, Boston, MA, USA). The electrodes were placed on the skin overlying the muscle belly of the TA, medial head of the GAS, BF, and vastus lateralis (VL) of the test limbs. To achieve an optimal EMG signal and low impedance (<5 kΩ), three, 4 cm^2 areas of skin were sanded and cleaned, and electrode gel applied between the skin and electrodes in accordance to procedures outlined by De Luca et al. [13]. All raw EMG analog signals were on-line pre-amplified ($\times$ 7000), analog filtered (20–7000 Hz), and then converted into digital signals sampled at 1200 Hz for a 5 s duration via the Associated Measurement Laboratory (AMLAB) data acquisition system (AMLAB Inc., Sydney, Australia). Prior to data analysis, EMG signals were full-wave rectified and low-pass filtered at 6 Hz using a 4th order dual-pass Butterworth filter. EMG data for the FP and catch NP conditions were normalized to maximum EMG activity produced during the true control NP condition and expressed as the NP:FP ratio.

A six-degree of freedom custom-built force plate (Institute of Neuroscience Technical Service Group, University of Oregon, Eugene, OR, USA) equipped with strain gauges mounted underneath the four corners was used to measure the vertical (F_z), horizontal antero-posterior (F_x), and medio-lateral (F_y) ground reaction forces. Using a feedback electric circuit, the F_z forces also served as trigger signals to initiate the force plate movement when the signal registered approximately 40 N ($\sim$8% of body weight). During the forward perturbation condition, onset of force plate movement occurred at 3.1 ± 0.2% of stance (approximately 29.10 ± 0.19 ms after heel strike) and ended at 59.8 ± 2.5% of stance (approximately 543.21 ± 0.24 ms after onset). Kinetic data were recorded at 1200 Hz for a 5 s duration via the AMLAB system. Prior to analysis, kinetic data were low-pass filtered between 4 and 10 Hz using a 4th order dual-pass Butterworth filter. Selected filter frequencies were determined for each force signal based on specifications from the manufacturer.

Kinematic data were collected using a PEAK Performance Technologies Real-Time Data Acquisition System (Peak Performance Inc., Denver, CO, USA). Four cameras were positioned 4 m from the sagittal plane along the progression plane of the subject's gait path. The pre-determined criterion for tolerable error in space calibration was set at 0.2% (2 mm maximum error for a 1 m-long object). Five kinematic reflective markers were placed on the skin overlying the base of the fifth metatarsal, lateral malleolus, lateral condyle of the femur, greater trochanter of the femur, and acromion process of the scapula. A reflective marker was also placed on the force plate to register plate movement and serve as the point of reference for transformation of local center of pressure (COP) coordinates to global kinematic coordinates. Kinematic data were collected at 120 Hz for a 5 s duration with each of the four cameras synchronized with the AMLAB system. Each marker was then digitized for the entire collection period including the stride before and after the stance phase on the force plate. The digitized position data for all markers were then low-pass filtered between 4 and 8 Hz using a 4th order dual-pass Butterworth filter. Optimal filter frequencies were determined for each force signal based on power spectral analyses wherein 80% of the raw signal was retained after the filtering process. Linear and angular position, velocity and acceleration data were then calculated and exported for further analysis.

2.4. Inverse dynamics calculations

The magnitude of the segmental masses along with their moments of inertia were estimated using data reported by Dempster [14] and individual subject anthropometric data. COP was calculated from the ground reaction force data within the force plate local coordinate system. Joint moments were calculated through an inverse dynamics analysis using a custom written MATLAB (The MathWorks, Inc., Natick, MA, USA) computer program combining the anthropometric, kinematic, and kinetic data. Ankle, knee, and hip joint moments were expressed as a reaction moment to all external moments and represent the internal moments normalized to subject mass. All joint moments were expressed as positive values for extensor and plantarflexor moments. Extensor angular impulse (EAI) was calculated from the positive area under the joint moment curve. Joint powers were calculated as the product of the joint moments and angular velocities and normalized to subject mass.

2.5. Data analysis

Prior to analysis, each trial was partitioned for the stance phase of the gait cycle (heel strike to toe off), interpolated as a percent of stance, and an ensemble average was created by averaging the 12 trials for each condition. For the purpose of analyzing the temporal

relationship between the two time-series curves, each ensemble average curve was divided into five phases (P) and five discrete points (Pt) that were selected according to discrete kinetic events determined from vertical and anterior/posterior ground reaction forces (Fig. 1). Phase 1 (P1) ranged from heel strike to initial loading (Pt1), phase 2 (P2) from Pt1 to first acceptance of full body weight (Pt3), phase 3 (P3) from Pt3 to mid-stance (MS), phase 4 (P4) from MS to second acceptance of full body weight (Pt5), and phase 5 (P5) from Pt5 to toe off. Two other discrete points (Pt2, Pt4) denoted the troughs between Pt1and Pt3 and between Pt3 and Pt5, respectively. Comparisons were made for between condition differences, if any, in average joint moments, powers, positions, and muscle EMG activity for each of the five phases and five discrete points.

2.6. Statistical analysis

Two-way repeated measures Analysis of Variance (ANOVAs) (10×3; $\alpha = 0.01$) and a priori post-hoc tests were used to determine differences, if any, between the three conditions. The independent variables were, (1) the five phases and five discrete points of stance; and (2) condition (true control NP, catch NP, and FP).

3. Results

No significant ($P > 0.01$) differences were found between the true control blocked NP trials and the randomized catch NP trials for any lower extremity variable. The total time of stance was significantly ($P <$

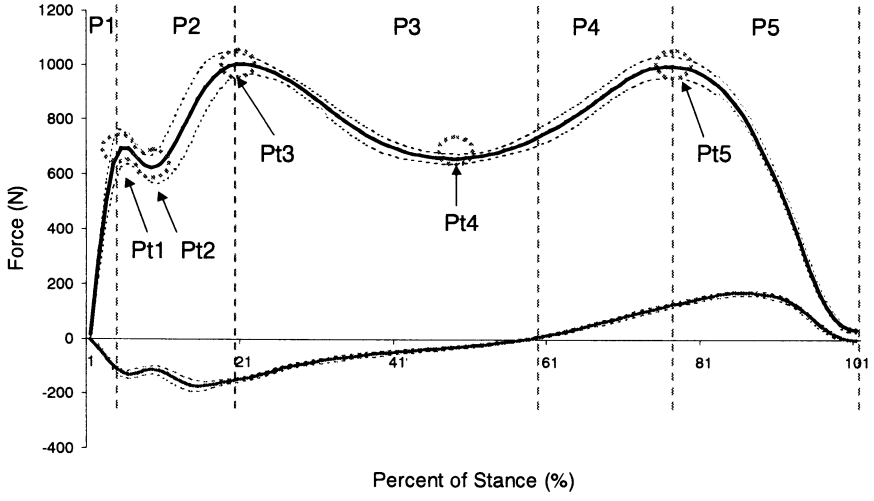

Fig. 1. Selection of five discrete points (dashed circles) and partitioning the stance phase into five phases (dashed vertical lines) according to discrete vertical and anterior/posterior kinetic events.

Table 1
Total time and percent of stance of the five phases and location of the five discrete points during stance phase of non-perturbation (NP) and forward perturbation (FP) conditions ($n = 10$)

Stance partition	NP		FP	
Phase (P)/Point(Pt)	Time (ms)	Percent of stance (%)	Time (ms)	Percent of stance (%)
P1	51.78 ± 10.44	6.34 ± 1.23	48.85 ± 9.06	5.02 ± 1.27
Pt1	51.78 ± 9.84	6.34 ± 1.21	48.85 ± 11.10	5.02 ± 1.33
Pt2	86.30 ± 11.39	10.22 ± 1.32	87.93 ± 15.04	9.64 ± 1.54
P2	129.45 ± 24.43	15.30 ± 3.33	185.63 ± 20.91	19.00 ± 2.14
Pt3	181.23 ± 14.07	21.64 ± 1.63	234.48 ± 21.10	24.02 ± 2.16
P3	284.64 ± 32.97	32.34 ± 3.82	341.95 ± 38.79	34.93 ± 3.97
Pt4	422.87 ± 12.69	49.22 ± 1.47	513.65 ± 18.26	52.61 ± 1.87
P4	207.27 ± 23.66	24.36 ± 3.07	185.63 ± 18.95	19.82 ± 1.94
Pt5	673.14 ± 28.22	78.34 ± 3.38	762.06 ± 34.68	78.77 ± 3.55
P5	190.07 ± 22.18	21.66 ± 2.65	215.76 ± 25.50	21.23 ± 2.27

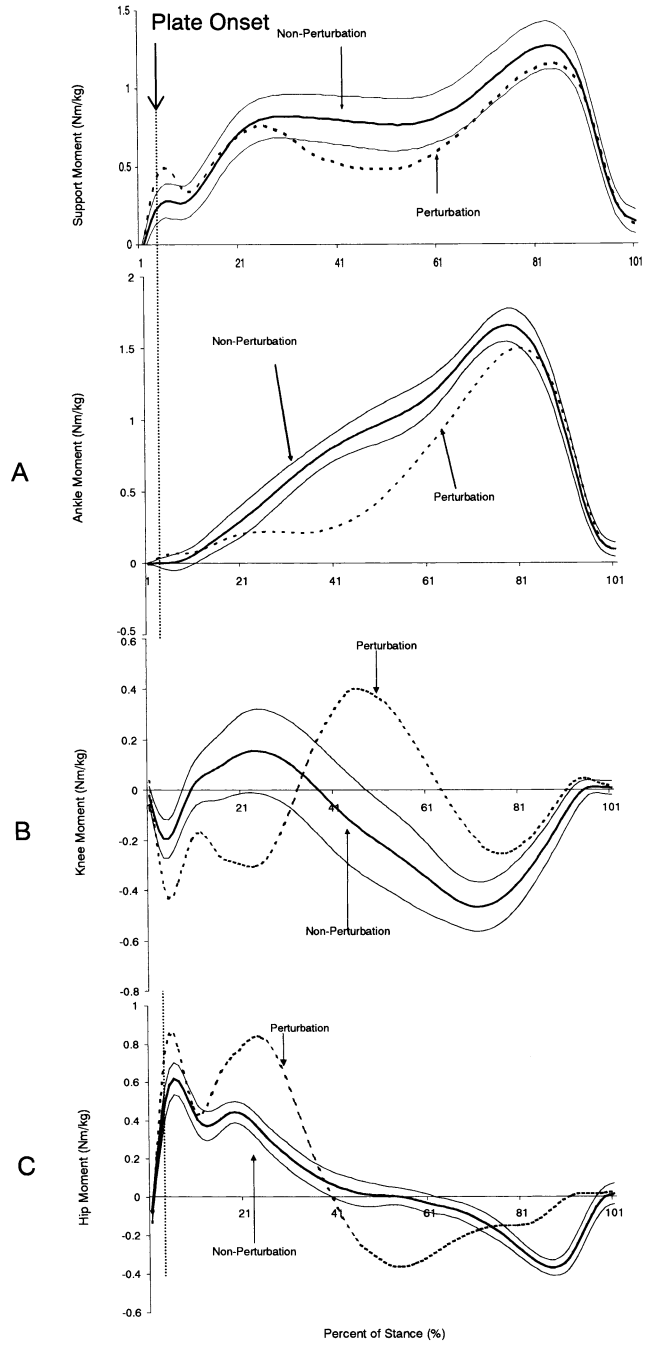

Fig. 2. Ankle (A), knee (B), and hip (C) joint moments and overall moment of support (M_s). Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick and thin lines are mean ± 1 S.D. for NP gait. Dashed thick line is mean of FP condition.

0.01) less for the NP condition (863.32 ± 77.27 ms) than the FP condition (977.59 ± 58.33 ms) and NP mid-stance occurred significantly ($P < 0.01$) earlier (49.6 ± 2.4% of total stance) than FP (59.8 ± 2.6% of total stance). Total time of the five phases and location of the five discrete points is summarized in Table 1.

3.1. Joint moments

The results revealed an overall positive M_s and a significantly ($P < 0.01$) reduced EAI (65.91 ± 5.61 N m/kg) for the FP condition compared with the NP (75.5 ± 6.78 N m/kg) condition (Fig. 2).

Table 2 presents lower extremity joint moments for each of the five phases (P) and five discrete points (Pt) of total stance during the NP and FP conditions as well as the total joint EAI for stance. The ankle exhibited significantly less EAI while the knee and hip exhibited significantly more EAI during the FP condition compared with NP (Table 2; Fig. 2A–C; $P < 0.01$).

The ankle NP plantarflexor moment rose steadily from heel strike through mid-stance to Pt5 before declining rapidly during the latter half of P5 (Table 2; Fig. 2A). The ankle FP plantarflexor moment remained relatively flat and was significantly smaller in magnitude that NP through the latter part of early stance (P2) and the first half of mid-stance (Pt3, P3, Pt4; Table 2; Fig. 2A; $P < 0.01$). The ankle FP moment paralleled but was significantly less than NP through the latter half of stance (P4, Pt5; Table 2; Fig. 2A; $P < 0.01$).

The knee NP generated an initial flexor moment in early stance and then followed a biphasic extensor–flexor–extensor moment pattern for early, mid-, and late stance periods, respectively (Table 2; Fig. 2B). The knee FP produced a significantly greater initial flexor moment than NP (P1, Pt1, Pt2) and, in contrast to NP, produced a flexor–extensor moment pattern during early (P2) and mid-stance (P2–Pt4) periods (Fig. 2B; $P < 0.01$). The knee FP moment paralleled NP for the remainder of stance although the knee produced a significantly smaller flexor moment during the latter half of mid-stance (P4) and a significantly greater extensor moment during late stance (Pt5, P5; Table 2; Fig. 2B; $P < 0.01$).

The hip NP extensor moment rose sharply in early stance and then decreased steadily until mid-stance after which a flexor moment was observed (Table 2; Fig. 2C). The hip FP moment paralleled but was significantly ($P < 0.01$) greater than NP immediately following the onset of force plate movement (P1, Pt1, Pt2, Table 2; Fig. 2C). In contrast to hip NP, the hip FP then produced an abrupt and significantly greater extensor moment (P2, Pt3), which rapidly decreased and became a large flexor moment through mid-stance (P3, Pt4, P4) that steadily declined through late stance (Pt5, P5; Table 2; Fig. 2C; $P < 0.01$).

3.2. Joint kinematics

Table 3 presents lower extremity joint position values during the NP and FP condition for P1–5 and Pt1–5 of total stance. The ankle NP position curve followed a plantarflexion–dorsiflexion–plantarflexion pattern over

early, mid-, and late stance periods, respectively (Table 3; Fig. 3A). The mean ankle FP position curve generally paralleled the NP condition curve through the stance phase (Table 3; Fig. 3A). However, the FP ankle position was significantly more plantarflexed than NP during the early (Pt2, P2) and mid-stance (Pt3, P3) periods (Table 3; Fig. 3A; $P < 0.01$).

The knee NP position curve followed a flexion–extension–flexion pattern over early, mid- and late stance periods, respectively (Table 3; Fig. 3B). The knee FP position curve paralleled NP prior to and immediately following force plate translation after which, in contrast to NP, the knee position remained in a relatively static position ($\sim 15^{\circ}$ flexion) until late stance (P2–P4; Table 3; Fig. 3B).

The hip NP position curve declined steadily from a flexed to extended position from early to mid-stance after which it followed a flexion–extension pattern from the latter half of mid-stance to late stance (Table 3; Fig. 3C). The hip FP position curve generally paralleled the NP condition curve throughout stance except the FP hip position was significantly more flexed during mid-stance (Pt3–P4) compared with NP (Table 3; Fig. 3C; $P < 0.01$).

3.3. Joint power

Table 4 presents lower extremity joint powers during the NP and FP conditions for P1–5 and Pt1–5 of total stance. The mean ankle NP ankle joint power curve revealed that small amounts of power were absorbed by the ankle during early stance and the first half of mid-stance after which the ankle sharply increased power generation until late stance (Table 4; Fig. 4A). The mean ankle FP joint power curve generally paralleled NP over the course of stance. However, ankle FP absorbed significantly less power during mid-stance (P2–P4) and produced significantly less power during late stance than ankle NP (Pt5, P5; Table 4; Fig. 4A; $P < 0.01$).

Table 2
Mean (±S.D.) of ankle, knee, and hip joint moments for non-perturbed (NP) and forward perturbation (FP) conditions ($n = 10$)

Stance partition	Ankle		Knee		Hip	
Phase (P)/Point(Pt)	FP	NP	FP	NP	FP	NP
P1	0.03±0.02	0.01±0.02	−0.26±0.06*	−0.13±0.06	0.49±0.11*	0.30±0.11
Pt1	0.01±0.04	0.01±0.04	−0.38±0.09*	−0.12±0.09	0.80±0.15*	0.39±0.17
Pt2	0.01±0.04	0.01±0.01	−0.27±0.07*	−0.09±0.08	0.59±0.14*	0.38±0.18
P2	0.17±0.06*	0.22±0.14	−0.14±0.12*	0.09±0.06	0.55±0.17*	0.24±0.13
Pt3	0.26±0.13*	0.50±1.16	0.22±0.23	0.17±0.11	0.14±0.31	0.16±0.14
P3	0.57±0.24*	0.83±0.09	0.02±0.14*	−0.08±0.12	−0.21±0.19*	0.04±0.13
Pt4	0.77±0.21*	0.97±0.11	0.09±0.12*	−0.23±0.08	−0.28±0.19*	−0.01±0.12
P4	1.28±0.16*	1.36±0.09	−0.22±0.11*	−0.41±0.09	−0.14±0.16*	−0.31±0.10
Pt5	1.51±0.12*	1.65±0.08	−0.27±0.13*	−0.44±0.07	−0.12±0.21*	−0.34±0.11
P5	0.91±0.08	0.98±0.01	−0.09±.009*	−0.18±0.04	−0.02±0.16*	−0.07±0.01
EAI	58.51±9.634*	78.88±4.49	8.39±2.74*	3.44±2.23	22.64±7.26*	12.93±7.19

Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments (N m/kg). *, Significantly different than corresponding NP condition ($P < 0.01$).

Table 3
Mean (±S.D.) of ankle, knee, and hip joint positions for non-perturbed (NP) and forward perturbation (FP) conditions ($n = 10$)

Stance partition	Ankle		Knee		Hip	
Phase (P)/Point(Pt)	FP	NP	FP	NP	FP	NP
P1	−3.79±3.61	−4.66±1.32	8.53±1.86	8.55±2.23	18.74±2.15	18.6±1.95
Pt1	−3.95±3.31	−5.47±1.85	9.46±1.65	10.31±2.35	18.15±2.33	17.94±1.92
Pt2	−3.46±1.01*	−5.45±1.65	10.47±2.24	10.86±2.68	17.55±2.25	17.77±1.97
P2	−2.03±1.32*	−1.08±1.74	11.76±2.02*	14.54±2.24	14.02±2.04	14.68±1.22
Pt3	2.17±1.60*	5.99±1.33	13.67±2.49*	16.13±2.18	12.84±2.03*	10.06±0.84
P3	6.04±1.15*	9.88±2.65	12.92±2.21	12.41±2.44	9.31±1.48*	7.45±1.35
Pt4	10.91±1.48	8.71±2.78	11.9±2.36*	9.52±2.01	7.96±1.44*	6.36±1.37
P4	11.35±2.77	10.48±3.20	10.74±2.34*	8.21±1.92	10.09±1.29*	8.46±1.88
Pt5	10.28±2.75	9.18±0.19	11.61±2.54	9.51±2.17	11.21±1.55	10.64±1.92
P5	1.05±2.92	−1.18±3.74	22.23±2.77	21.26±1.96	11.31±5.42	10.95±3.84

Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion (°). *, Significantly different than corresponding NP condition ($P < 0.01$).

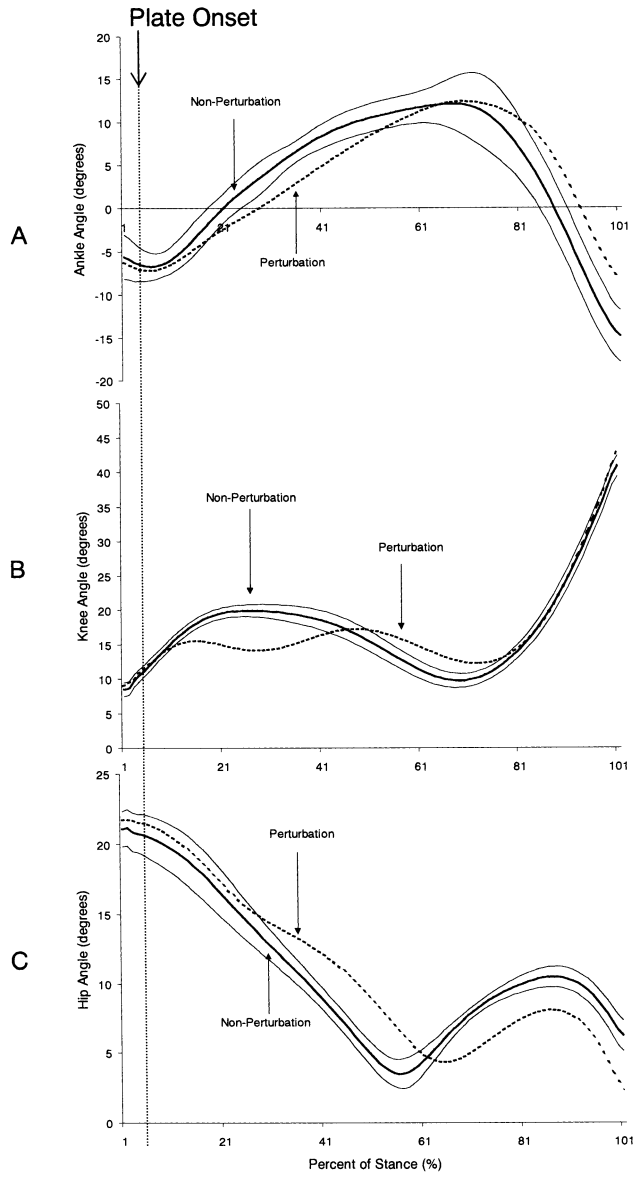

Fig. 3. Ankle (A), knee (B), and hip (C) joint positions. Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion. Solid thick and thin lines are mean ±1 S.D. for NP gait. Dashed thick line is mean of FP condition.

Knee NP and FP power curves differed markedly from one another. Both power curves were undulating in nature, with knee FP absorbing significantly more power following onset of force plate movement (Pt1; Table 4; Fig. 4B; $P < 0.01$). During mid-stance, knee NP and FP power curves were prominently opposed to one another with knee FP generating power early in mid-stance (P3) while knee NP absorbed power (Table 4; Fig. 4B; $P < 0.01$). Late in stance (Pt4) knee FP absorbed power while knee NP generated power (Table 4; Fig. 4B; $P < 0.01$). During late stance (Pt5, P5), knee FP power absorption was significantly less than knee NP (Table 4; Fig. 4B; $P < 0.01$).

The hip NP power curve exhibited power generation during early stance, power absorption during the first half of mid-stance followed by power generation for the latter half of mid-stance and late stance (Table 4; Fig. 4C). The hip FP power curve differed markedly from the hip NP power curve as significantly more power was generated by FP immediately following the onset of force plate movement (P1–Pt2) and throughout most of mid-stance (Pt3–P4; Table 4; Fig. 4C; $P < 0.01$). During late FP stance, (P5), the hip absorbed power in contrast to power generation demonstrated in the late NP condition (Table 4; Fig. 4C; $P < 0.01$).

3.4. Muscle EMG

Table 5 presents lower extremity muscle EMG values during the NP and FP conditions for P1–5 and Pt1–5 of total stance. Values expressed are the FP:NP ratio for the corresponding phase or discrete point of stance. The NP–TA EMG muscle response was characterized by strong activation during early stance followed by a rapid decrease to a low level for all of mid-stance and the first part of late stance, and then another surge of activity prior to toe-off (Fig. 5A). Compared with NP, FP–TA activity was significantly less during the first part of early stance (P1, Pt2) and significantly greater during the latter half of early stance (P2) and most of mid-stance (Pt3, P3; Table 5; Fig. 5A; $P < 0.01$). FP–TA muscle activity then paralleled NP–TA activity for the remainder of stance.

The NP–GAS EMG muscle response was characterized by a steady rise in activity from heel strike through mid-stance and then a rapid decrease during late stance (Fig. 5B). Compared with NP, FP–GAS produced significantly more EMG activity during early stance (P1, Pt1) followed by significantly ($P < 0.01$) less activity for the remainder of stance (P2–P5; Table 5; Fig. 5B; $P < 0.01$).

The NP–VL EMG muscle response was characterized by a burst of activity during early stance that steadily drops and remains relatively low throughout the remainder of stance (Fig. 5C). Compared with NP, FP–VL activity produced significantly less EMG activity during most of early stance (P1–Pt2), followed by significantly greater activity for the remainder of stance (P2–P5; Table 5; Fig. 5C; $P < 0.01$).

The NP–BF EMG muscle exhibited strong activation during early stance followed by a steady decrease for all of mid-stance and the first part of late stance and generated a surge of activity prior to toe-off (Fig. 5D). Compared with NP, FP–BF EMG produced significantly more EMG activity during early stance (P1–P2) and most of mid-stance (P2–Pt4) followed by reduced activity for the remainder of stance (P4–P5; Table 5; Fig. 5D; $P < 0.01$).

4. Discussion

The purpose of this investigation was to determine the effect of unexpected FP during gait on lower extremity joint moments and muscle EMG patterns in healthy subjects. Tang et al. [5] postulated that TA EMG activity served to restore the disrupted ankle joint trajectory and realign the leg segment of the perturbed limb. However, Tang et al. [5] also reported that subjects exhibited GAS EMG inhibition in response to the FP but the reason for this inhibition was not addressed in that study. The results of this investigation also demonstrated significantly reduced GAS EMG muscle activity during early stance in response to the FP. It is possible that suppressed GAS activation may attenuate the effect of the FP-induced ankle plantarflexion and help maintain balance. Another possible explanation for the increase in TA and suppression of GAS activity may come from examination of the moments and powers produced at the ankle joint during the FP. A small eccentric ankle dorsiflexor moment (power absorption) followed by a large eccentric ankle plantarflexor moment was observed for early and mid-NP stance, respectively (Fig. 2A, Fig. 4A). During FP, no ankle dorsiflexor moment was observed early in stance, in contrast to NP. Instead, the ankle produced a sustained, but significantly reduced, ankle plantarflexor moment throughout stance as compared with NP. Since the knee angle is under the control of moments of force at the hip and ankle, as well as the knee due to action of bi-articular muscles [15], a stronger than normal ankle plantarflexor moment can serve to slow down, or even reverse, forward rotation of the leg segment, resulting in a reduction in knee flexion [16,17]. The sustained reduction in the ankle plantarflexor moment during early FP stance, as observed in this study, may have contributed to the static knee flexion position during the FP as a possible reactive balance strategy necessary during early FP stance.

The motor patterns of the knee during early and mid-stance FP consisted of a large flexor moment during early stance which did not switch to an extensor moment until significantly later in mid-stance compared with NP (Fig. 2B). Significant fluctuations in power production (Fig. 4B) and a static knee flexion position (Fig. 3B) were also observed during early and mid-stance of FP. An initial suppression of VL EMG activity, relative to NP, was observed early in FP stance followed by a strong VL activation coincident with a large BF EMG burst (Fig. 5C and D). The EMG activity demonstrated by these two antagonistic muscles is indicative of co-contraction, possibly to maintain knee joint stability during early and mid-FP stance [5].

In the present study a reciprocal trade-off between the knee and hip was demonstrated during FP mid-stance when the knee exhibited an extensor moment in contrast to the knee flexor moment, observed during NP (Fig. 2B). At this same time, the FP hip produced a large flexor moment in contrast to the extensor moment during NP (Fig. 2C). The reciprocal trade-off between the knee and hip moments may be necessary to maintain a positive M_s and dynamic equilibrium in response to an unexpected FP. It has been shown that there is a reciprocal trade-off between the hip and knee joints such that dynamic balance and control of the HAT segment occurs via a coordination between posterior muscles (hip extensors/knee flexors) and anterior muscles (hip flexors/knee extensors) acting at either joint [1,11].

During the late phase of FP stance, a reduction in the peak ankle plantarflexor moment and ankle power generation was observed as compared with NP (Fig. 2A, Fig. 3A). Since the ankle power absorption is reduced during the first half of FP stance, the subsequent drop in the ankle plantarflexor moment and

Table 4
Mean (±S.D.) of ankle, knee, and hip joint powers for non-perturbed (NP) and forward perturbation (FP) conditions ($n = 10$)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/Point(Pt)	FP	NP	FP	NP	FP	NP
P1	0.06±0.23	0.01±0.01	−0.42±0.24	−0.40±0.19	0.56±0.22*	0.36±0.22
Pt1	0.08±0.42	0.002±0.19	−0.66±0.45*	−0.37±0.28	0.90±0.27*	0.51±0.32
Pt2	0.06±0.44	−0.02±0.18	−0.30±0.17	−0.26±0.20	0.79±0.26*	0.49±0.32
P2	−0.31±0.17*	−0.53±0.19	−0.10±0.07*	0.06±0.06	0.61±0.21	0.46±0.25
Pt3	−0.45±0.22*	−0.93±0.19	0.15±0.27	−0.03±0.11	0.01±0.20*	−0.33±.022
P3	−0.38±0.19*	−0.84±0.12	−0.07±0.11*	0.06±0.01	0.08±0.31*	−0.09±0.14
Pt4	−0.39±0.22*	0.94±0.51	−0.13±0.15*	0.22±0.01	0.33±0.03*	0.12±0.07
P4	0.16±0.81*	0.85±0.55	−0.19±0.12	−0.23±0.11	0.25±0.17*	0.04±0.09
Pt5	2.96±0.69	3.96±1.64	−0.49±0.26*	−0.92±0.07	0.11±0.27	0.01±0.13
P5	3.42±0.43*	4.14±0.81	−0.26±0.15*	−0.69±0.14	−0.01±0.01*	0.39±0.08

Positive values indicate power generation, negative values indicate power absorption (W/kg) *, Significantly different than corresponding NP condition ($P < 0.01$).

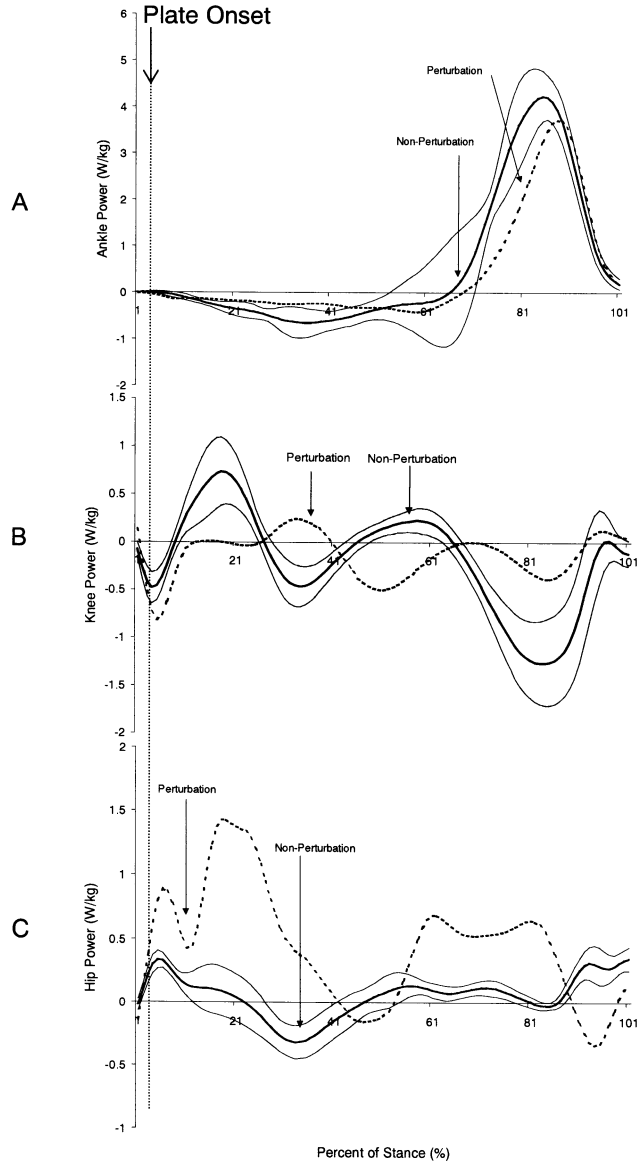

Fig. 4. Ankle (A), knee (B), and hip (C) joint powers. Positive and negative values are energy generation and absorption by the muscles. Solid thick and thin lines are mean ± 1 S.D. for NP gait. Dashed thick line is mean of FP condition.

power in late FP stance could possibly be attributed to a reduced storage of elastic energy in the plantarflexor muscles during the first half of stance. Elastic energy storage, however, was not directly measured in this study.

In the present investigation, postural adjustment responses observed during the stance phase of FP can be divided into three sections that are representative of the type of postural adjustment responses thought to occur within each. Within this conceptual model, postural responses observed from the time immediately following the onset of plate movement (< 70 ms; P1, Pt1, Pt2) are considered to be mechanical responses and can be solely attributed to viscoelastic changes in the tissues surrounding the lower extremity joints due to alteration of the body's COM relative to the base of support. It should be noted, however, that COM was not measured in this study. Responses observed between 70 and 250 ms (P2, Pt3) are considered to be a combination of mechanical and spinally-mediated (stretch reflex) neuromuscular responses, whereas responses observed after 250 ms (P3, Pt4, P4, Pt5, P5) are a combination of mechanical, spinally-mediated, and cortically-mediated neuromuscular responses [7,9,10,18–20]. The results from this investigation demonstrate that an increase in the hip extensor moment as a result of the FP begins approximately 9.7 ms (~ 1% after plate onset) after initiation of the force plate movement, whereas the reactive TA EMG burst follows after approximately 97 ms (~ 10% after plate onset). Tang et al. [5] reported TA EMG onset latencies of 91.2 ms concomitant with suppression of GAS EMG activity and postulated that the control of these occurrences resulted from polysynaptic spinal reflexes or supraspinal loops. Other investigations have also demonstrated spinally-mediated muscle onset latencies of 90–100 ms during standing perturbations [7,9,10,19] and suggest that postural reactions to unexpected FP prior to 70 ms are due to the mechanical viscoelastic stretching of muscle, tendon, and joint capsule [10,20]. Herman et al. [20] studied the gait initiation torque patterns of humans and reported that changes in muscle stiffness (torque development) immediately following gait initiation may be attributed to the inherent mechanical properties of muscle relative to lengthening of series-elastic tissue rather than changes in motor discharge patterns (EMG). Nashner [10] described a significant stabilizing effect due primarily to ankle joint stiffness prior to leg muscle EMG activation approximately 90 ms after plate onset. It was suggested that cortically-mediated responses would not begin until at least 200 ms following plate onset and that muscle EMG responses prior to 200 ms

Table 5
Mean (±S.D.) FP:NP ratio of muscle EMG activity ($n = 10$)

Stance Partition	TA	GAS	BF	VL
Phase (P)/Point (Pt)				
P1	0.87±0.09*	1.15±0.09*	1.24±0.12*	0.75±0.18*
Pt1	0.96±0.10	1.14±0.08*	1.26±0.08*	0.78±0.17*
Pt2	0.70±0.01*	1.01±0.04	1.17±0.06*	0.76±0.12*
P2	1.71±0.03*	0.92±0.03*	1.51±0.21*	1.23±0.07*
Pt3	1.63±0.15*	0.12±0.22*	1.39±0.01*	1.55±0.14*
P3	1.17±0.03*	0.80±0.03*	1.41±0.13*	1.51±0.14*
Pt4	0.97±0.03	1.10±0.12	1.28±0.07*	1.34±0.14*
P4	1.06±0.10	0.87±0.03*	0.66±0.01*	1.23±0.07*
Pt5	0.99±0.09	0.68±0.08*	0.74±0.08*	1.12±0.13
P5	0.98±0.03	0.85±0.06*	0.22±0.09*	1.04±0.01

Values greater than 1.0 indicate FP EMG activity greater than NP condition, values less than 1.0 indicate FP EMG activity less than NP condition *, Significantly different than NP condition ($P < 0.01$).

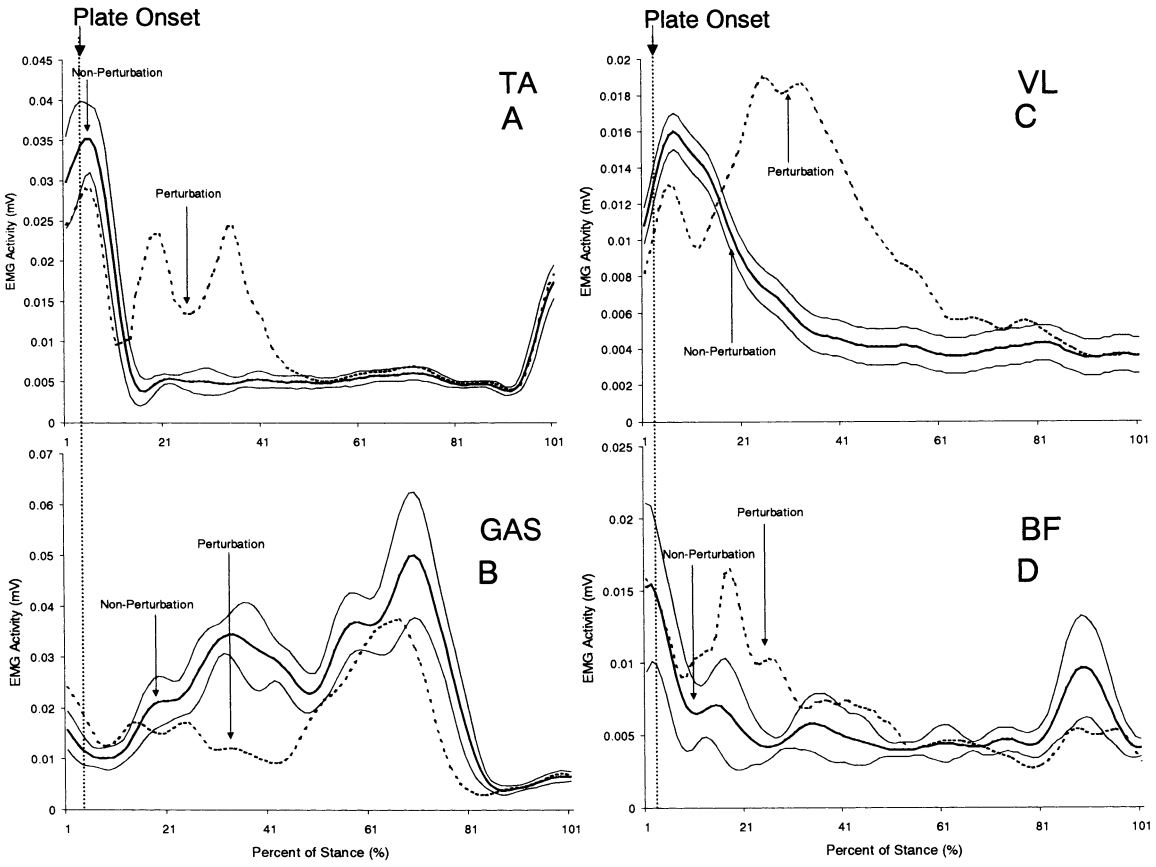

Fig. 5. Representative example of muscle EMG activity during FP (dashed thick line) and NP (solid thick and thin lines are mean ±1 S.D.) conditions for the tibialis anterior (TA: A), gastrocnemius (GAS: B), vastus lateralis (VL: C), and biceps femoris (BF: D).

are spinally-mediated. More recent studies have suggested that cortically-mediated EMG responses begin as early as 160 ms following treadmill-induced gait perturbations [21,22].

The results of this investigation suggest that the initial responses to plate movement are mechanical in nature, rather than a neuromuscular response, and can be attributed to a viscoelastic stretching of pre-loaded joint muscles. For example, prior to heel strike, the muscles surrounding the hip are programmed to produce a certain amount of tension to provide vertical support to the body and prevent forward acceleration of the HAT segment upon heel strike and early in stance. In response to the FP, a large extensor hip moment is observed almost immediately after force plate movement and is possibly due to the mechanical stretching of pre-loaded hip muscles. Since vertical support of the body is maintained until muscle EMG responses are observed approximately 90 ms after force plate movement, postural adjustment responses prior to 70 ms are likely mechanical in nature.

The increase in TA EMG activity and reduction of GAS EMG activity exhibited approximately 97 ms after onset of FP is consistent with previous literature [5,10] and has been attributed to spinally-mediated neurological motor reflexes as a result of mechanical muscle stretch and alterations in lower extremity joint trajectories. Although the ankle is the first joint to undergo alterations in trajectory as a result of force plate movement, it seems unlikely that the ankle is the only input to the CNS to initiate postural adjustments since the hip and knee joints also undergo significant mechanical postural adjustments. EMG responses were observed in the BF and VL approximately 120–190 ms after force plate movement and appear to correspond with the delayed alteration of knee and hip joint trajectories, relative to the ankle joint, and subsequent spinally-mediated neuromuscular responses.

Afferent input from mechanical postural responses may provide input to the CNS to initiate spinally-mediated postural adjustments first observed at approximately 70 ms, and cortically-mediated neurological responses observed 160–200 ms after plate onset. Mechanical postural responses that occur later in FP stance may serve to provide continued afferent feedback to the CNS to promote continued spinal and higher level motor responses in an effort to maintain dynamic equilibrium, a positive M_s, and forward propulsion of the body during FP gait.

5. Conclusion

The findings of the present research suggest that response to perturbed gait is a synchronized effort of the lower extremity joints to maintain dynamic equilibrium and the overall M_s during the stance phase of gait. The muscles surrounding the hip were found to be most important in maintaining control of the HAT segment and preventing collapse of the lower extremity as an initial response to the FP. Muscle EMG activity from the leg and thigh segments and lower extremity joint kinematics demonstrated similar patterns compared with previous investigations. These results indicate that healthy subjects, in response to an unexpected FP, demonstrate joint moment and power patterns that are distinct from NP gait in order to maintain dynamic equilibrium during locomotion.

Acknowledgements

This study has been supported, in part, by the International Society of Biomechanics Matching Dissertation Grant, the National Athletic Trainers Association Doctoral Research Grant, and the Eugene Evonuk Award for Environmental and Stress Physiology.

References

[1] Winter DA, Ruder GK, MacKinnon CD. Control of balance of upper body during gait. In: Winters JM, Woo S-L, editors. Multiple muscle systems: biomechanical and movement organization. New York: Springer, 1990:534–41.
[2] Shik ML, Orlovsky GN. Neurophysiology of locomotor automatism. Phys Rev 1976;56(3):465–501.
[3] Patla AE. Age-related changes in visually guided locomotion over different terrains: major issues. In: Stelmach GE, Homberg V, editors. Sensorimotor impairment in the elderly. Netherlands: Kluwer Academic, 1993:231–52.
[4] Nashner LM. Balance adjustments of humans perturbed while walking. J Neurophys 1980;44(4):650–64.
[5] Tang PF, Woollacott MH, Chong RKY. Control of reactive balance adjustments in perturbed human walking: roles of proximal and distal postural muscle activity. Exp Brain Res 1998;119:141–52.
[6] Brady RA, Pavo MJ, Owings TM, Grabiner MD. Foot displacement but not velocity predicts the outcome of a slip induced in young subjects while walking. J Biomech 2000;33:803–8.
[7] Dietz V, Quintern J, Berger W. Corrective reactions to stumbling in man: functional significance of spinal and transcorticol reflexes. Neurosci Lett 1984;44:131–5.
[8] Eng JJ, Winter DA. Strategies for recovery from a trip in early and late swing during human walking. Exp Brain Res 1994;102:339–49.
[9] Gollhofer A, Schmidtbleicher D, Quintern J, Dietz V. Compensatory movements following gait perturbations: changes in cinematic and muscular activity patterns. Int J Sports Med 1986;7:325–9.
[10] Nashner LM. Fixed patterns of rapid postural responses among leg muscles during stance. Exp Brain Res 1977;30:13–24.
[11] Winter DA. Sagittal plane balance and posture in human walking. Eng Med Bio 1987;9:8–11.
[12] Strandberg L, Lanshammar H. On the biomechanics of slipping accidents. In: Matsui H, Kobayashi K, editors. International series on biomechanics: biomechanics VIII-A, vol. 4A. Champaign: Human Kinetics, 1981:397–402.
[13] De Luca CJ. The use of surface electromyography in biomechanics. J Appl Biomech 1997;13:135–63.
[14] Dempster W. Space requirements of the seated operator. WADC Technical Report Ohio:L Wright-Patterson Air Force Base, 1959. 55–159.
[15] van Ingen Schenau GJ, Bobbert MF, van Soest AJ. The unique action of bi-articular muscles in leg extensions. In: Winters JM, Woo S-L, editors. Multiple muscle systems: biomechanical and movement organization. New York: Springer, 1990:639–52.
[16] Winter DA. Overall principle of lower limb support during stance phase of gait. J Biomech 1980;13:923–7.
[17] Winter DA, Olney SJ, Conrad J, White SC, Ouunpuu S, Gage JR. Adaptability of motor patterns in pathological gait. In: Winters JM, Woo S-L, editors. Multiple muscle systems: biomechanical and movement organization. New York: Springer, 1990:680–93.
[18] Bothner KE, Jensen JL. How do non-muscular torques contribute to the kinetics of postural recovery following a support surface translation. J Biomech 2001;34(2):245–50.
[19] Allum JHJ, Honegger F, Acuna H. Differential control of leg and trunk muscle activity by vestibulo-spinal and proprioceptive signals during human balance corrections. Acta Otol 1995;115:124–9.
[20] Herman R, Cook T, Cozzens B, Freedman W. Control of postural reactions in man: The initiation of gait. In: Stein, et al, editor. Control of posture and locomotion. New York: Plenum Press, 1973:363–88.
[21] Schillings AM, van Wezel BM, Mulder T, Duysens J. Muscular responses and movement strategies during stumbling over obstacles. J Neurophysiol 2000;83(4):2093–102.
[22] Schillings AM, Van Wezel BM, Mulder T, Duysens J. Widespread short-latency stretch reflexes and their modulation during stumbling over obstacles. Brain Res 1999;816(2):480–6.