



Ankle and hip postural strategies defined by joint torques

C.F. Runge^{a,b,c}, C.L. Shupert^d, F.B. Horak^{d,*}, F.E. Zajac^{a,b,e}

^a Rehabilitation R & D Center (153), Veterans Affairs Palo Alto Health Care System, Palo Alto, CA 94304-1200, USA

^b Mechanical Engineering Department, Stanford University, Stanford, CA 94305-4021, USA

^c Biomechanics Department, Failure Analysis Associates, 2300 Chestnut St., Suite 150, Philadelphia, PA 19103, USA

^d Neurological Sciences Institute, Oregon Health Sciences University, 1120 NW 20th Avenue, Portland, OR 97209-1595, USA

^e Department of Functional Restoration, Stanford University, Stanford, CA 94305-4021, USA

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Abstract

Previous studies have identified two discrete strategies for the control of posture in the sagittal plane based on EMG activations, body kinematics, and ground reaction forces. The ankle strategy was characterized by body sway resembling a single-segment-inverted pendulum and was elicited on flat support surfaces. In contrast, the hip strategy was characterized by body sway resembling a double-segment inverted pendulum divided at the hip and was elicited on short or compliant support surfaces. However, biomechanical optimization models have suggested that hip strategy should be observed in response to fast translations on a flat surface also, provided the feet are constrained to remain in contact with the floor and the knee is constrained to remain straight. The purpose of this study was to examine the experimental evidence for hip strategy in postural responses to backward translations of a flat support surface and to determine whether analyses of joint torques would provide evidence for two separate postural strategies. Normal subjects standing on a flat support surface were translated backward with a range of velocities from fast (55 cm/s) to slow (5 cm/s). EMG activations and joint kinematics showed pattern changes consistent with previous experimental descriptions of mixed hip and ankle strategy with increasing platform velocity. Joint torque analyses revealed the addition of a hip flexor torque to the ankle plantarflexor torque during fast translations. This finding indicates the addition of hip strategy to ankle strategy to produce a continuum of postural responses. Hip torque without accompanying ankle torque (pure hip strategy) was not observed. Although postural control strategies have previously been defined by how the body moves, we conclude that joint torques, which indicate how body movements are produced, are useful in defining postural control strategies. These results also illustrate how the biomechanics of the body can transform discrete control patterns into a continuum of postural corrections. © 1999 Elsevier Science B.V. All rights reserved.

Keywords: Joint torque; Hip strategy; Postural control; Ankle strategy

1. Introduction

In 1985, Nashner and McCollum [1] hypothesized the existence of two discrete strategies that could either be used separately or be combined by the nervous system to produce adaptable control of the horizontal position of the center of mass (CM) in the sagittal plane. The ankle strategy repositioned the CM by moving the whole body as a single-segment inverted pendulum by production of torque at the ankle. The hip strategy, in contrast, moved the body as a double-segment inverted

pendulum with counterphase motion at the ankle and hip. They further suggested that hip strategy should be observed in situations that limit the effectiveness of ankle torque at producing whole-body motion (e.g. compliant or shortened support surfaces). The experimental observations that followed were consistent with this hypothesis, showing that ankle strategy was used to respond to translations during stance on a flat support surface, while hip strategy was observed during responses to backward translations during stance on a narrow (10 cm) beam [2].

In earlier experimental studies, postural control strategies were characterized primarily by muscle activation patterns and body kinematics [2]. Ankle strategy was characterized by early activation of dorsal ankle

* Corresponding author. Tel.: +1-503-4137471; fax: +1-503-4137229.

E-mail address: fay@otolith.lhs.org (F.B. Horak)

muscles followed by activation of dorsal thigh and trunk muscles (for responses to backward translations). These muscle activations were associated with the production of torque at the support surface, and kinematic analyses showed body movement predominantly at the ankle joint (although some small movement at the hip was also observed). The hip strategy (observed in response to translations of a narrow beam) was characterized by early activation of ventral trunk and thigh muscles associated with a relative increase of shear forces at the support surface and little phasic activation of ankle muscles. Kinematic analyses showed trunk flexion paired with ankle extension.

Mixed strategies containing components of both ankle and hip strategies (such as early activations in both dorsal ankle and ventral trunk muscles) were observed in subjects responding to translations of beams of intermediate size and when transitioning from trials on a narrow support surface to trials on a flat support surface, or vice versa. In those experiments mixed strategies observed during transition trials were hypothesized to be transient features of an adaptive process aimed at producing an optimal postural response for a given environmental condition [3]. However, Nashner and colleagues [1,2] predicted that mixed hip and ankle strategies would be observed in response to fast translations of a flat support surface. Subsequently, mixed postural control strategies in response to translations of a flat support surface were reported in control subjects with loss of somatosensation in the feet due to ischemia [4,5] and in patients with Parkinson's disease [6]. Henry et al. [7] also observed early hip and trunk muscle activations in controls in response to 9-cm, 35-cm/s translations of a flat support surface, not only for anterior/posterior translations, but also for lateral and diagonal translations.

More recent biomechanical models of postural control also suggest that hip strategy is a highly effective means of stabilizing body posture [8–10]. Based on estimates of maximal muscle force, Kuo's model [9,10] developed a 'feasible acceleration set' of all ankle and hip accelerations that can be produced when the feet are constrained to remain in place on the ground and the knee is constrained to be straight (both conditions consistent with previous experimental observations using slow platform translation velocities). Using optimization techniques, Kuo determined the ankle and hip accelerations that would require the least amount of muscle activity to return the body to a stable position in response to a perturbation. These calculations were carried out for two different postural goals: a 'position' goal (i.e. maintain upright body alignment), and a 'stability' goal (i.e. maintain the projection of the CM within the boundaries of stability). When the goal of maintaining upright body alignment was predominant, the model predicted the use of ankle strategy (defined

by Kuo as movement at the ankle joint without significant movement at the hip joint) to control posture. However, when either the goal of stability was optimized or the perturbation was large, thus requiring a fast, high-amplitude response, the model predicted the use of hip strategy (defined as the combined use of ankle and hip accelerations) to respond to postural perturbations on flat support surfaces [8,9]. Strategies involving hip acceleration only were not predicted by the model for any perturbation or any postural goal; this was due, at least in part, to the fact that many of the lower limb muscles act at more than one joint. Based on the assumptions of Kuo's optimization model, hip strategy appears to require less muscle activity than ankle strategy to effect the same movement of the CM on a flat surface [10]. The model suggests therefore that the choice of postural strategy depends both on the postural goal and on the environmental constraints.

Kuo's model predicts a continuum of responses as subjects increase their use of hip strategy with increasing platform velocity. However, this model is limited by the constraints of keeping the foot in contact with the floor and the knee straight. Further, the model is based on kinematics, as were many previous experimental observations. While kinematics describe the movement of the body, they provide little insight into the cause of the body movement; movement of a particular body segment can occur as a result of muscle activation at the joint, or can be a resultant effect of muscles acting on other segments. For example, hip flexion can result either from torque at the hip joint, or from ankle plantarflexion combined with gravitation forces acting on the trunk [11]. Information concerning muscle activation patterns from EMG recordings can provide additional insight into the cause of body movement, and previous experimental observations of body kinematics were paired with EMG recordings (see, for example Ref. [2]). Nevertheless, EMG recordings can still be misleading, as muscles may act concentrically or eccentrically and the activity of deep muscles cannot be recorded with surface electrodes.

While body kinematics and muscle activation patterns can provide some information about the motor control strategies underlying body movements, the calculation of the joint torques can provide more direct insight. Studies of joint dynamics have contributed significantly to the understanding of a variety of motor control phenomena, including paw shake responses in cats, the role of central pattern generators and peripheral feedback in bipedal and quadrupedal locomotion, the control of single limb movements, and the acquisition of limb movements during development (see Ref. [12] for an extensive review). For example, different patterns of joint torques at the ankle and knee during cat paw shake responses suggested different neural con-

trol strategies for the two joints, i.e. torques at the ankle appeared to produce the movement of the paw, while torques at the knee appeared to stabilize proximal limb segments during the movement [13]. Subsequent experiments confirmed this hypothesis by showing that casting the limb to prevent movement changed the pattern of torque generation at the knee, but not at the ankle [14].

The purpose of the experiment presented here was to examine the evidence for a continuum of postural control responses based on combinations of two discrete ankle and hip strategies. Joint torques at the ankle, knee, and hip were calculated in normal subjects responding to backward translations of a flat platform at a continuum of velocities. Muscle activation patterns and body kinematics are also presented in order to permit comparison of these results with those of previous studies and to provide as complete an analysis of the postural control strategies as possible.

2. Methods

The experimental protocol was approved by the Institutional Review Board of the Legacy Good Samaritan Hospital and Medical Center and was performed in accordance with the 1964 Helsinki Declaration. All subjects were volunteers who gave their informed consent prior to participation. Seven adults (mean age 31.3 ± 5.1 (S.D.)) with no history of otologic, neurologic, or orthopedic abnormality participated in the study.

Subjects stood on a servo-controlled, hydraulically-driven movable platform that was translated backward at one of a range of velocities on each trial. Subjects wore a safety harness attached to an overhead rail during testing; the harness was adjusted prior to the experiment to permit both full hip flexion and a step. Subjects were instructed to keep their arms crossed over their chests, to keep their eyes closed during all trials to eliminate the influence of vision and to maintain balance without taking a step if possible. The center of pressure (CP) under the subjects' feet was monitored on line during the experiment to prevent anticipatory leaning prior to the translation onset; shifts of ± 0.5 cm could be detected. Each translation was a backward ramp displacement of amplitude 20 cm, the duration of which determined the ramp velocity; the translation velocities chosen for this experiment were 5, 10, 15, 20, 25, 32, 40, 47 and 55 cm/s (programmed durations ranging from 4 s for the 5-cm/s translations to 364 ms for the 55-cm/s translations). The velocity profile of the translation was trapezoidal; each translation reached peak acceleration at 11 ms, and the peak of this acceleration varied linearly with the ramp velocity (e.g. 6.5 m/s² for 10 cm/s, 13.0 m/s² for 20 cm/s, up to 32 m/s²

for 55 cm/s). The duration of the constant velocity segment of the translation ranged from 3.9 s for the 5-cm/s translations to 300 ms for the 55-cm/s translations.

At the beginning of the experimental session, each subject was exposed to the range of platform velocities used in the experiment to determine the highest translation velocity that could be tolerated without steps or falls. The range of translation velocities tested for each subject was then chosen, and translation velocities were presented in blocks of three trials beginning with the highest tolerated velocity and decreasing with subsequent blocks. Occasional steps and heel-lifts did occur during the experiment, especially in response to the highest velocities tested, and subsequent analyses are confined to the trials without steps. All subjects experienced at least five different velocities, but some subjects (especially those who tolerated higher translation velocities) experienced more than five, up to a maximum of nine. Fig. 1 shows the velocities tested for each of the seven subjects.

Body positions in the sagittal plane were recorded using a Motion Analysis™ optical recording system. Two cameras recorded at 60 Hz the locations of two reflective markers placed on the support surface and one marker at each of the following body landmarks: the lateral malleolus (ankle), the lateral femoral condyle (knee), the greater trochanter (hip), and the seventh cervical vertebra (lower neck). Ankle angle was defined by the angle between the support surface and the shank (segment defined by ankle and knee markers), knee angle was defined by the angle between the shank and the thigh (segment defined by knee and hip markers),

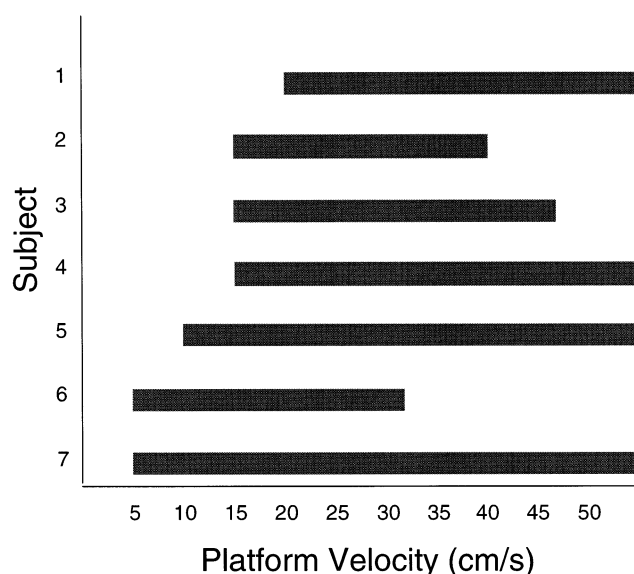


Fig. 1. Platform velocities tested for each experimental subject. The highest velocity tested for each subject was the highest velocity for which subjects could reliably maintain balance without stepping.

and hip angle was defined by the angle between the thigh and the trunk (segment defined by hip and C7 neck markers). Sagittal plane center-of-mass (CM) location was calculated as a weighted summation of individual segmental CM locations.

Muscle activity was recorded using pairs of 2.5 cm surface electrodes spaced 2–4 cm apart on eight muscles of the right leg, trunk, and neck: medial gastrocnemius (GAS), tibialis anterior (TIB), rectus femoris (QUA), biceps femoris (HAM), rectus abdominis at the level of the umbilicus (ABD), lumbar paraspinals at the iliac crest (PAR), sternocleidomastoid (STER), and upper trapezius (TRAP). EMG signals were band-pass filtered (70–2000 Hz) and full-wave rectified prior to sampling (360 Hz). They were then low-pass filtered (100 Hz) and stored for off-line analysis. Onset latencies were determined from single trials by a standardized computer routine that defined burst onset as the time the activation first exceeded the baseline level plus two standard deviations (where the baseline is defined as the average activation level for the 100 ms prior to translation onset), then remained above baseline for at least 25 ms. Burst sizes were computed as the integrated EMG for the first 150 ms following the burst onset. Visual inspection was used to check for false detections in all trials.

Force transducers at the anterior and posterior edges of the support surface measured vertical and horizontal (shear) reaction forces, sampled at 360 Hz. The vertical force measurements were used to compute a support surface reaction torque. Fore-aft shear force at the surface in the sagittal plane was used to detect onset of platform movement. The net muscular joint torques from each trial were estimated using a variation of the linear quadratic follower (LQF) method [15,16]. This technique calculates net joint torques by producing a forward simulation of each trial that reproduces the observed forces and kinematics. In this experiment, recorded ankle reaction torque data matched the simulated ankle torque data everywhere along its trajectory for the first 400 ms to within ± 10 N-m. Simulated and recorded segment angles matched to within $\pm 1^\circ$.

Statistical analyses of the data were performed using analysis of variance and linear regression techniques. To determine the effects of translation velocity on muscle latency, the mean latencies were analyzed with respect to velocity in a two-way repeated measures analysis of variance (velocity by muscle). Post-hoc Scheffe's *S*-tests were used to determine significance ($P < 0.05$). To determine the effects of translation velocity on muscle burst size, regressions of integrated EMG means on translation velocity were performed for each muscle, with slope significantly different from zero at $P < 0.05$. To determine the effect of translation velocity on peak torque values, a regression of mean peak torques on translation velocity was performed, with slope significantly different from zero at $P < 0.05$.

3. Results

3.1. Muscle activation patterns

At the faster translation velocities, muscle activations were larger, and some muscles that were silent at the slower velocities became active. Consistent with the distal-to-proximal EMG activity observed in past studies for ankle strategy responses to backward translations [2,5], slow translations (approximately 5–20 cm/s) induced corrective responses characterized by muscle activity on the posterior aspect of the body, beginning with gastrocnemius (Fig. 2, traces (a, b) of GAS, HAM, PAR). In response to 20 cm/s translations, for example, gastrocnemius activity began at 93 ± 2 ms (mean ± 1 S.E.) after the onset of the translation. Gastrocnemius latency was slightly longer for the 5-cm/s condition, but translation velocity had no other effect (Scheffe's *S*-test, $P > 0.05$ for all other velocities). In these slower trials, subjects also showed some activity in tibialis anterior, and two subjects showed very small, intermittent activity in quadriceps (similar to the subject shown in Fig. 2).

As the velocity of the translation increased beyond a subject-specific threshold (e.g. 20 cm/s for the subject shown in Fig. 2), muscle activations on the anterior aspect of the body appeared (Fig. 2, traces (c, d, e), esp. ABD, STER, QUA), beginning with rectus abdominis. The threshold velocity for activation in these muscles varied among subjects, but was always between 10 and 25 cm/s. For example, for responses to 40 cm/s translations, rectus abdominis (92 ± 4 ms) joined gastrocnemius in the earliest part of the automatic response (ABD and GAS latencies not significantly different; Scheffe's *S*-test, $P > 0.05$). The latency of rectus abdominis was similar at all suprathreshold velocities (Scheffe's *S*-test, $P > 0.05$). Rectus abdominis was joined by sternocleidomastoid (103 ± 6 ms; ABD and STER latencies not significantly different; Scheffe's *S* test, $P > 0.05$), followed by (Scheffe's *S*, $P < 0.05$) quadriceps (134 ± 4 ms) and trapezius (137 ± 9 ms). The amplitude of muscles already active at lower velocities also increased at higher velocities (regressions of the first 150 ms of GAS, HAM, PAR, and TIB bursts, positive slope, $P < 0.05$).

3.2. Kinematics of the postural responses

The hip and knee joint displacement trajectories associated with fast and slow translation velocities differed. Although there was some variability in the patterns across subjects at each translation velocity, the direction of the changes associated with velocity was similar for all subjects. The joint displacements consisted of an early, passive component, due strictly to the platform translation, and a later, active component that

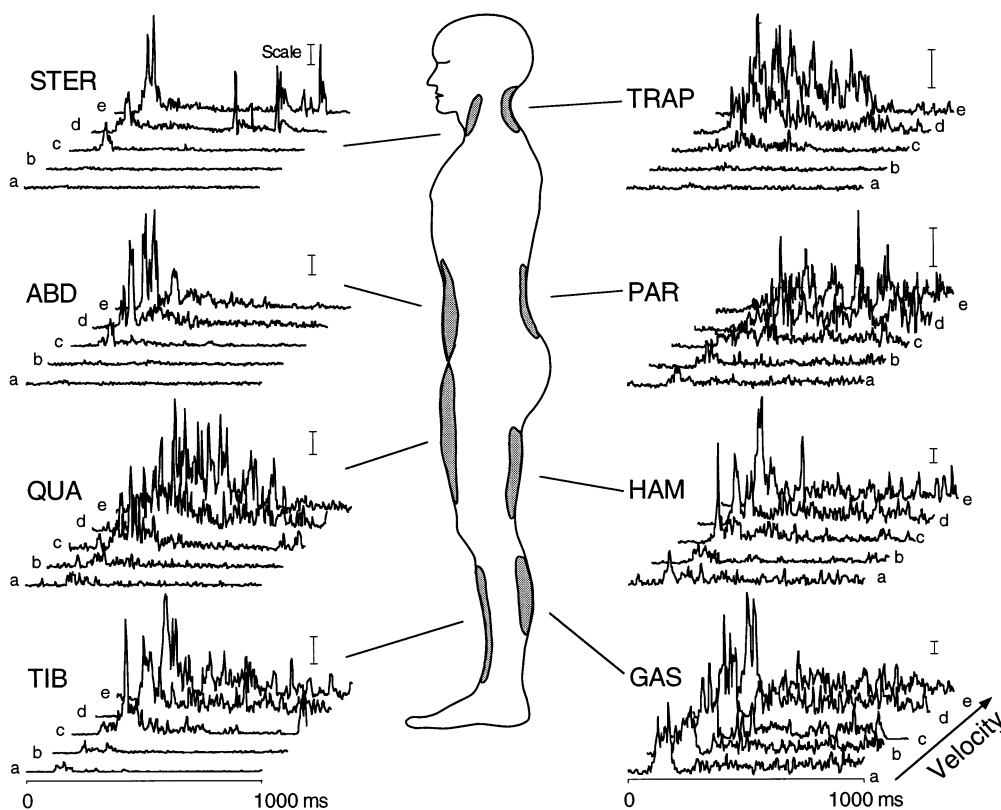


Fig. 2. EMGs from subject #2 in response to backward platform translations at five velocities. Each trace represents the average of three EMG responses to translations of the same velocity ((a) 15 cm/s; (b) 20 cm/s; (c) 25 cm/s; (d) 32 cm/s; and (e) 40 cm/s). The scale bars = 25 μ V for all muscles except GAS and TIB (= 100 μ V). At lower translation velocities (a), (b), muscle activity on the posterior aspect of the body appeared (gastrocnemius (GAS), hamstrings (HAM), paraspinals (PAR)), beginning with gastrocnemius (GAS). At higher translation velocities (c), (d), (e), activity in upper-body muscles on the anterior aspect of the body (rectus abdominis (ABD), quadriceps (QUA), sternocleidomastoid (STER)) was added, with ABD simultaneous to GAS in the earliest part of the response.

was a corrective response to the platform translation. The passive component occurred in the first 150 ms of each trial and consisted of slight ankle dorsiflexion, knee flexion, and slight hip extension (Fig. 3, 0–150 ms, ankle, knee, hip). For the corrective movements occurring after 150 ms, faster translation velocities produced not only increases in the magnitude of the movement at all joints, but also produced changes in the pattern of movement at the hip and knee. These changes in pattern were evident well before the platform began to decelerate (no earlier than 360 ms for the fastest velocity) and are therefore not due to the deceleration of the platform.

For slow translation velocities, postural corrections simply arrested the passive joint movement, with each joint gradually returning to equilibrium (Fig. 3, > 150 ms, ankle, knee, hip, for platform velocities \leq 20 cm/s). Postural corrections to faster translations, defined here as translations beyond a particular, subject-specific threshold (20 cm/s for subject shown in Fig. 3), consisted of quick hip and knee flexions to help return the body to equilibrium (Fig. 3, > 150 ms, velocities \geq 25 cm/s). Specifically, at faster velocities, a transient period

of active knee flexion (Fig. 3, knee, 150–500 ms, velocity \geq 25 cm/s) and a marked increase in the peak hip flexion emerge (Fig. 2, hip, velocity \geq 25 cm/s). The quick hip flexions reached peak magnitudes that correlated with translation velocity ($0.97 > r^2 > 0.63$, for each subject; $r^2 = 0.81$ for subject shown in Fig. 3), but varied considerably among subjects (e.g. range of maximum hip flexions: 16–98°; range of maximum knee flexions: 6–22°). In addition to flexions at the knee and hip, all subjects also showed some lifting of the heel at translation velocities of 25 cm/s or greater.

Although the pattern of muscle activations and joint displacements changed with increasing platform velocity, the whole body CM position changed only in magnitude as translation velocity increased (i.e. the shape of the trajectory of the CM was unaffected prior to platform stop; Fig. 4). The onset of the translation initiated forward CM movement with respect to the feet, due primarily to passive ankle dorsiflexion in the early, passive interval (Fig. 4, top panel, first 150 ms). After 150 ms, the forward displacement of the CM continued to increase for all velocities, eventually reaching a plateau or reversing direction during the active

response (Fig. 4, top panel, after 150 ms), with the peak displacement during the faster trials often coinciding with the stopping of the platform (Fig. 4, cf. CM peaks (top panel, arrows) with platform translation (bottom panel)).

3.3. Joint torques

Consistent with the discrete pattern changes demonstrated by the early muscle activations and kinematics, net joint torques also changed pattern at fast translation velocities. A hip flexor torque was added between 150 and 250 ms at a subject-specific velocity threshold

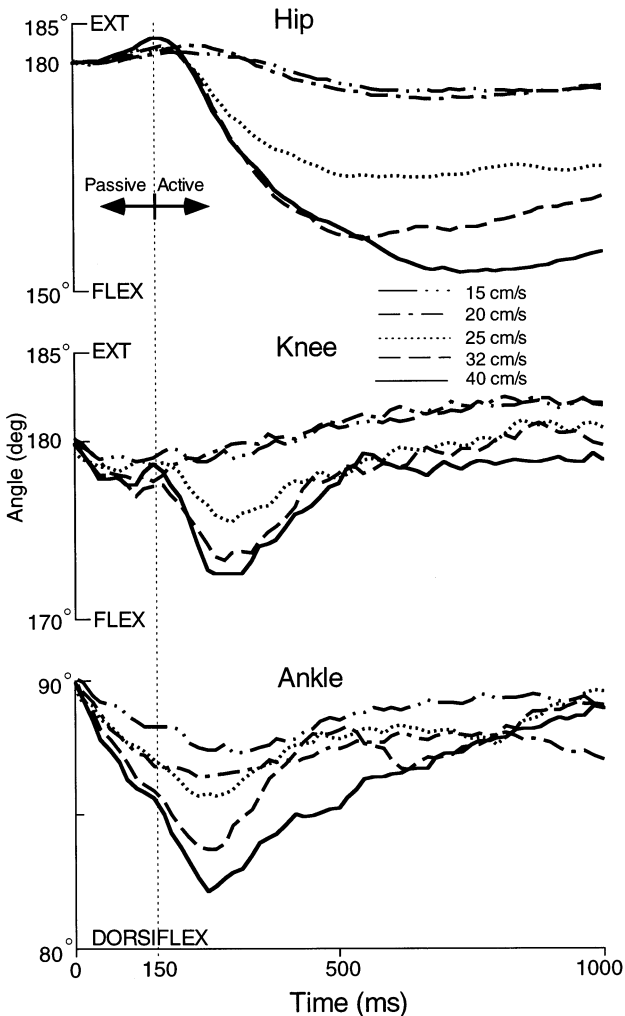


Fig. 3. Hip, knee, and ankle joint angles of subject #2 in response to backward platform translations. The 'active' response (i.e. motion coincident with translation-induced muscle activity) begins at about 150 ms. At slower translations (≤ 20 cm/s for this subject), the active response gradually arrests the previous passive body motion response to the platform translation (≤ 150 ms). In contrast, the active responses to faster translations (≥ 25 cm/s for this subject) includes quick hip flexion, often accompanied by quick knee flexion. The ankle dorsiflexes more with faster translation velocities, though the trajectory shape is rather velocity independent. All joint angles eventually return to those corresponding with upright posture.

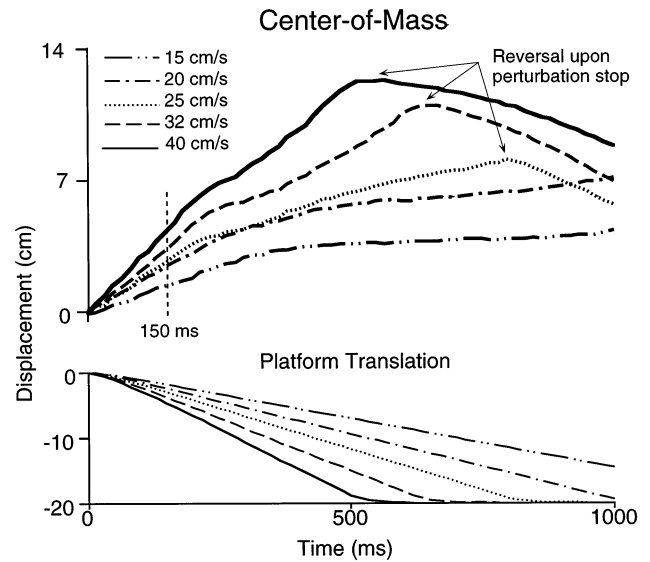


Fig. 4. Forward CM movement with respect to the feet of subject #2 in response to backward platform translations; onset of the platform movement is time 0. The 'passive' CM response (≤ 150 ms) increases gradually with platform velocity. The 'active' CM response (> 150 ms) eventually peaks and either plateaus or reverses, often at the time the platform stops moving (arrows).

(Fig. 5, velocities ≥ 20 cm/s) [17]. When the translation velocity exceeded the threshold, faster velocities caused peak hip flexor torques to increase (regression, $P < 0.05$). The velocity threshold for the appearance of hip torques was the same as that observed for the emergence of abdominal EMG bursts, and the hip torque was consistently low during this interval in trials without the bursts (Fig. 6, hip torque, see arrow). Although both the absolute magnitude of the flexor peak and the velocity at which hip torque and its concomitant abdominal burst emerged differed across subjects, all subjects demonstrated a threshold effect for hip torque. The range of hip torques during trials with ABD bursts was clearly higher than the range for trials without ABD bursts (Fig. 7). For the range of platform velocities without abdominal bursts, hip torques, while non-zero in some cases, were very small, and did not have the characteristic flexion peak between 150 and 250 ms. No corresponding threshold effect was observed for the net ankle or knee torques, which were well above zero and very consistent in pattern for all velocities, regardless of ABD activity (Fig. 6, knee torque, ankle torque).

4. Discussion

The results of this study verify that a continuum of mixed strategies is often employed by normal subjects to control balance on a flat support surface as the velocity of the postural perturbation increases [1,2]. Although the platform velocity at which hip strategy

was added to ankle strategy was different for different subjects, hip torques were always accompanied by ankle torques at higher platform velocities. The pure hip strategy that was observed and identified with EMG patterns during postural responses to translations of narrow support surfaces in previous studies was not observed in this experiment. The EMG records showed that when subjects added trunk flexor activation on a flat surface, they did not eliminate activity or show coactivation of ankle muscles as subjects typically do when standing across a narrow beam [2]. Although hybrid muscle responses have been previously observed

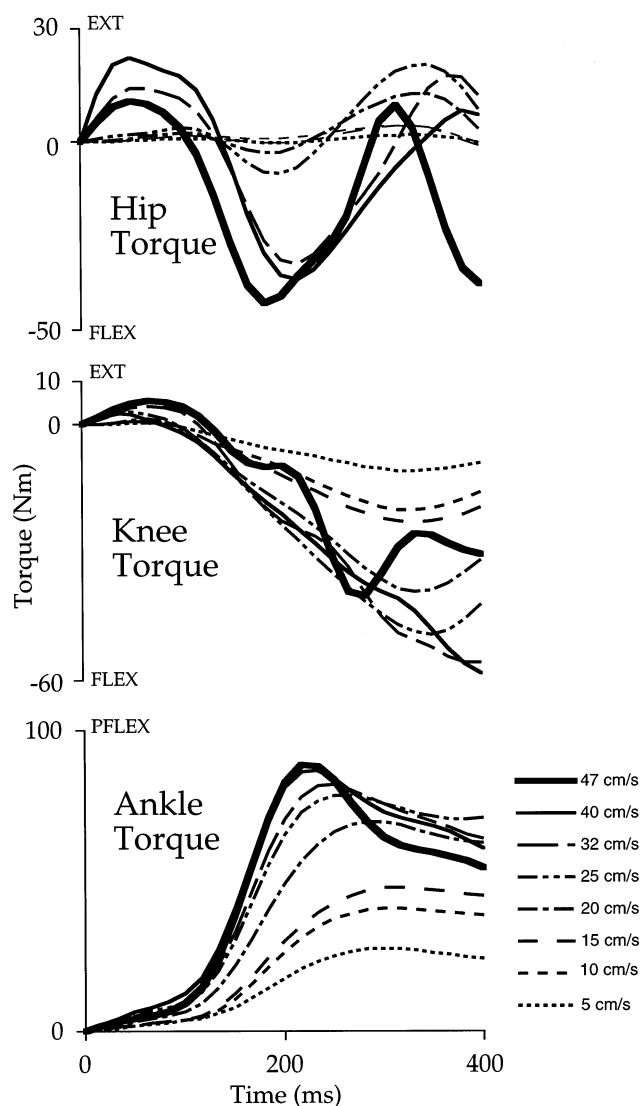


Fig. 5. Average hip, knee, and ankle torques for subject # 5 at eight velocities (5–47 cm/s). Each trace represents the average of three torque responses to translations of the same velocity. Hip torque changes pattern to include a hip flexor peak between 150 and 250 ms at a subject-specific velocity threshold, which for this subject was 20 cm/s. In contrast, ankle and knee torques showed a similar pattern for all velocities. Ankle and knee torques demonstrate magnitude increases with velocity, as does hip torque above subject-specific threshold for hip flexor torque.

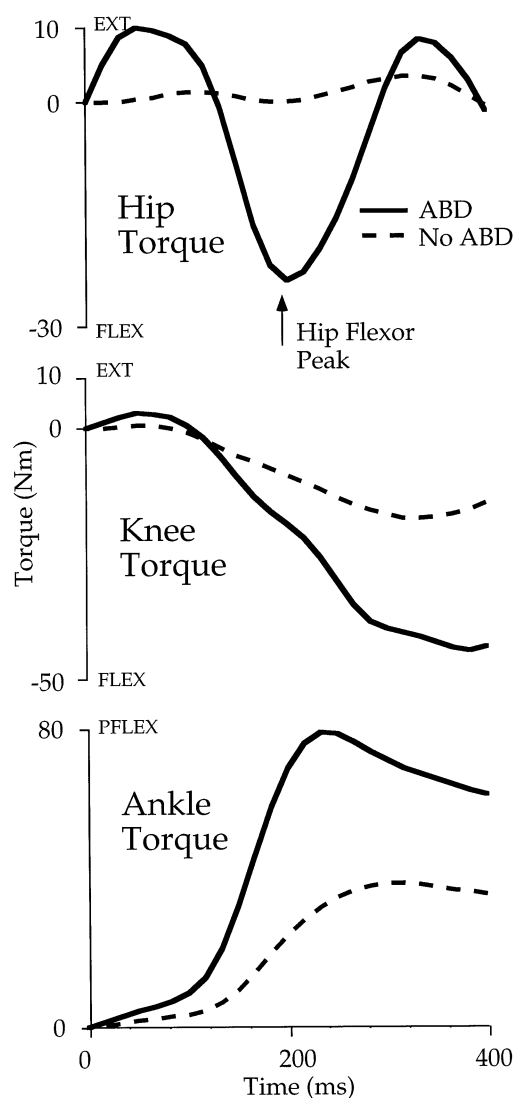


Fig. 6. Average net hip, knee, and ankle joint torques (calculated from velocity averages Fig. 5) corresponding to velocities in subject # 5 where ABD muscle activity was present (solid line; $n = 5$) or absent (dashed line; $n = 3$). Notice that a high peak hip flexor torque (arrow) is apparent when ABD activity appears but virtually zero when ABD is absent. Knee and ankle torques are non-zero in both sets and show no threshold effect.

in response to fast backward translations [7,18–21], the present study is the first to confirm, through joint torque analysis, that the EMG patterns observed during fast translations are indeed indicative of active control of combined ankle and hip action.

This muscle activation pattern is consistent with optimization models of posture that predict a combination of motion at the ankle and the hip at higher perturbation velocities [8–10]. Kuo's model predicts that the ratio of ankle acceleration to hip acceleration observed in these subjects should be approximately 1:–3. However, Kuo's model constrains the knee to be straight and the foot to remain in contact with the ground. In the experiments described in the present paper, subjects

showed flexion at the knee (up to 22° in some subjects at higher translation velocities), and all subjects also showed some lifting of the heel at higher translation velocities. Also, the subjects in these experiments were tested with higher translation velocities and amplitudes than those used in the experimental studies that formed the basis of Kuo's models [2,9,10]. The double-differentiated joint position data were somewhat noisy, but the ratios of ankle acceleration to hip acceleration were closer to 1:–1 than to Kuo's predicted 1:–3 ratio for most subjects. Despite these differences between the modeled and observed responses, the data from this experiment are consistent with the general assertion of the model; i.e., that hip and ankle accelerations are produced to control posture.

In these experiments, slow velocity translations induced responses similar to those previously described as ankle strategy [1,2,5]. These responses were characterized by activity in gastrocnemius, hamstrings, and paraspinals and relatively little knee and hip angles. The hip torques for these responses were also very low, and the pattern of the torques was variable between subjects. Because the hip torques and knee and hip joint displacements for slow translation velocities were not zero, it could be argued that the subjects were using a mixed strategy even at the lowest platform velocities. The experimental design could have contributed to the persistence of hip strategy at lower platform velocities; previous experiments have shown that subjects who adapt to using hip strategy to respond to translations of a beam persist in using hip strategy initially when they transfer onto a flat support surface [1], and in these experiments, platform velocities were tested in descending order, beginning with faster translation velocities that required hip strategy to maintain balance with feet

in place. However, as Kuo points out, proximal joint torques should not be expected to be zero even if the body is swaying rigidly as an inverted pendulum about the ankle, as the upper body segments must be stabilized to prevent motion due to referred forces from the motions at more distal joints and many muscles of the lower limb act at more than one joint [9]. The fact that significant increases in hip torque accompanied by bursts in rectus abdominis appeared at a characteristic velocity for each subject in this study also argues for a transition from a strategy in which the motion of the upper body is restricted to a strategy in which motion of the upper body is used to restore equilibrium.

At the faster translation velocities, hip strategy was added to the response, as demonstrated not only by rectus abdominis activity and increased hip flexion, but more importantly by an early hip flexor torque, which established active initiation of the hip flexion. The addition of a hip flexor torque to the postural response at faster translation velocities demonstrates a change in the control of balance to active generation of upper body flexion. When hip flexor torque is not used to maintain balance, the destabilizing gravity forces are countered by using plantarflexion torque generated about the ankle joints to arrest the forward body rotation and CM movement. However, while plantarflexion on a fixed surface rotates the lower leg backward, the same torques (if unopposed) flex the trunk forward. The multi-segmented human body can be controlled as a flexible, single-segment inverted pendulum only because passive anatomical structures and activations in proximal muscles (e.g. HAM, PAR) limit the relative movement between body segments to the small deviations typically observed in ankle strategy [2]. The postural control strategy is the same: muscles contributing to an ankle plantarflexion torque (e.g. gastrocnemius, recorded in this experiment, and soleus [22]) are activated to overcome the destabilizing torque of gravity on the whole-body CM. The stabilizing potential of ankle plantarflexion torque is quite limited, however, because the moment of inertia of the whole body about the ankle joint is quite high and heels will rise with significant plantarflexion torque [9]. Because relatively large ankle torques are required to produce relatively small corrections of the CM using ankle strategy, Kuo's optimization model predicts that mixed strategy would be used to correct for translations of all speeds on a flat surface if muscular effort is to be minimized. However, the subjects of this study corrected slow translations with little or no hip torque; this finding is consistent either with the hypothesis that the predominant postural goal during translations slow enough to not compromise stability is to maintain upright alignment or with the possibility that the difference in muscular effort to produce the two strategies is minimal for slow translations. Other explanations of

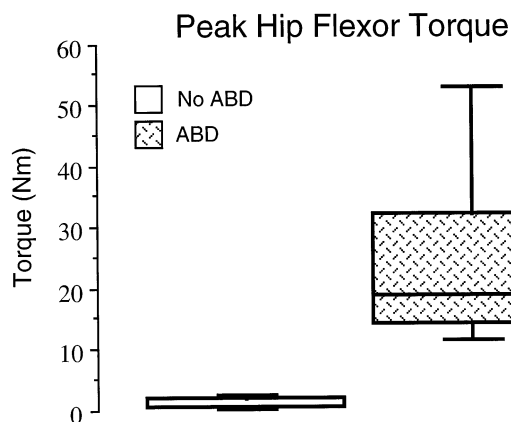


Fig. 7. Distribution of peak hip flexor torques among subjects (box-whisker plot; i.e. bottom tic mark indicates the 10% level, bottom of box the 25% level, the line in box the 50% level, top of box the 75% level, and the top tic mark the 90% level). In each subject the peak torques were averaged for trials with and without ABD activity. Notice that the hip torque is clearly higher in trials where ABD activity is present, even though the amount then varies much.

the data may also be possible. The torques calculated here are net joint torques, and the effects of agonist/antagonist co-contraction in muscles not recorded were not considered. Careful analysis of co-contraction would contribute to a more complete understanding of the range of postural control responses.

Although the magnitude of the hip flexor torque was generally much smaller than the magnitude of the corresponding ankle plantarflexion torque in the mixed strategy, the effects it has on balance control can be large. When a hip flexor torque is used, it works with gravity rather than in opposition, using the torque to effectively divide the body into two segments, resembling a double inverted pendulum. The activation of hip flexors propels the trunk forward, initiating forward rotation of the upper body. Sternocleidomastoid activity prevents passive whipping of the head backward, allowing the head and trunk to move as a single segment, and quadriceps activity assists hip flexion and straightens the knee, allowing the leg to move as a single segment. Gravity provides the downward force thereafter because the trunk is leaning forward. The natural fall of the upper body segment (head/arms/trunk) aids the rearward rotation of the straightened leg [23], which eventually brings the whole-body CM back to equilibrium.

When the body behaves as a double inverted pendulum, the CM location, a measure of static postural stability, is roughly a function of two angles, the leg angle and the upper body angle. Because the rotating upper body is falling forward in the early part of the hip strategy response, the whole-body CM will move back toward equilibrium only if the leg rotates backward. The ankle plantarflexion torque and the stopping of the platform are partly responsible for the leg reversal, but two properties inherent to the execution of the hip strategy greatly aid the swift backward rotation of leg. First, the moment of inertia of the body about the ankle joint decreases as the trunk falls forward and down, allowing a given ankle torque to effect a higher acceleration. Second, the clockwise rotation of the trunk produces a reactive force propelling the leg counterclockwise. It should be noted at this point that, although EMG activity in previous studies of responses on beams have suggested that hip strategy may be used in forward translations also [2], we cannot necessarily assume that hip strategy in response to forward translations will mirror the one described here for backward translations. The biomechanics of the body are not symmetric in the sagittal plane (especially at the knee), and thus a double-segment inverted pendulum control strategy may prescribe different kinematic and joint torque patterns for forward translations.

Optimization models have predicted that combined ankle and hip acceleration should produce faster correction of the CM than ankle acceleration alone [8–10].

However, a faster CM corrective response was not observed in these experiments. Instead the trajectory of the CM was highly similar for all platform velocities. This discrepancy may be due to the fact that the optimization models constrain the knee to remain straight. As was observed in this experiment, the knee actually undergoes significant flexion when hip torque is added to produce a mixed strategy. Flexion at the knee would limit the speed with which the leg can rotate backward and thus slow the correction of the CM position. This suggests that future experiments should measure knee angles and future models should account for it. Another possibility is that the blocked presentation of velocities in this experiment allowed the subjects to predict the time of platform deceleration. Together with the added hip torque, the effects of the platform deceleration at the end of the translation on body motion might be used to reverse CM motion [24]. The fact that the CM reversal coincided in time with the end of platform movement is consistent with this hypothesis.

Despite the fact that Kuo's model constrained both the knee to remain straight and the entire foot to remain in contact with the platform, his optimization model successfully predicted mixed strategies, rather than pure hip strategies, as the optimal postural response to fast translations of a flat support surface. Thus, as Nashner and McCollum [1] originally suggested, the use of a pure hip strategy may indeed be limited to a very narrow or compliant support surface. In fact, the results presented here clearly show that ankle torque was not only present at all velocities tested, but was highest at the fastest translation velocity. Furthermore, higher ankle torques were associated with larger CM displacements. This suggests that the purpose of the hip torque on a firm, flat surface may be to change the configuration of the body to facilitate torque at the ankle without lifting of the heels to effectively correct the CM position without taking a step. Thus, the biomechanics of the body may transform discrete neural control patterns into a continuum of postural corrections.

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