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RESEARCH ARTICLES

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Reduced plantar sensitivity alters postural responses to lateral perturbations of balance

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Abstract There is considerable evidence that lower-limb somatosensation contributes to the control of upright balance. In this study, we investigated the specific role of foot sole cutaneous afferents in the generation of balance corrections following lateral accelerations of the support surface. Participants were subjected to balance perturbations before and after targeted anesthesia of the cutaneous soles induced by intradermal injections of local anesthetic. Subject responses were quantified in terms of net joint torques at the ankles, hips and trunk. Contrary to the conclusions drawn in earlier studies, response torque impulses at the ankles and hips were clearly scaled with the perturbation impulse under both control and anesthetized conditions. Reduced plantar sensitivity produced a relative shift in compensatory torque production from the ankles and trunk to the hips. These findings demonstrate that plantar cutaneous afferents play an important role in the shaping of dynamic postural responses. Furthermore, the results suggest that loss of plantar sensation may be an important contributor to the dynamic balance deficits and increased risk of falls associated with peripheral neuropathies.

Keywords Foot sole · Cutaneous mechanoreceptor · Net joint torque · Automatic postural response · Cutaneous anesthesia

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Introduction

As many as 4.5 million Americans may suffer from peripheral neuropathies, largely as a consequence of diabetes mellitus (Apfel 1999; Richardson and Ashton-Miller 1996). Most common is a distal symmetric sensorimotor polyneuropathy, which is primarily confined to the axons of small and large-fiber sensory afferents. The result is a "stocking feet" pattern of sensory loss that begins in the toes and progresses proximally (Greene et al. 1999). These deficits in cutaneous and proprioceptive axons lead to reduced ankle position sensation (Simoneau et al. 1996; Van den Bosch et al. 1995; van Deursen et al. 1998). Peripheral neuropathy patients exhibit decreased stability while standing (Boucher et al. 1995; Geurts et al. 1992; Simoneau et al. 1994) as well as when subjected to dynamic balance conditions (Bloem et al. 2000; Inglis et al. 1994). Not surprisingly, epidemiological evidence has linked peripheral neuropathies with an increase risk of falling (Richardson and Ashton-Miller 1996; Richardson et al. 1992). It remains unclear, however, to what extent specific peripheral sensory systems contribute to the balance deficits seen in peripheral neuropathy patients.

In the present study, we address the role played by foot sole cutaneous mechanoreceptors in the maintenance of balance during dynamic situations. Several previous studies have used ischemia (pressure cuff above the ankles) to reduce sensation in the feet and ankles prior to studying dynamic postural responses (Diener et al. 1984; Horak et al. 1990). This method fails to isolate cutaneous sensation from intrinsic foot and ankle proprioception. More recently, Perry et al. (2000) found that corrective stepping behavior was modified after cooling of the feet. Again, this technique is not selective and can be expected to affect the deeper foot structures as well as cutaneous afferents. These earlier studies involved the measurement of ground reaction forces, body kinematics, and electromyographic activity from specific muscles. It is the torque produced at each joint, however, that represents the functional response to a mechanical perturbation of balance. Yet to our knowledge, no previous studies have

quantified the effects of reduced plantar sensation in terms of the body dynamics triggered by balance perturbations. Horak and colleagues found that hypoxic anesthesia of the feet and ankles resulted in a distal-to-proximal shift in the order of muscle activations in response to backward support surface translations (Horak et al. 1990). We therefore hypothesized that targeted anesthesia of the plantar soles would result in a distal-to-proximal shift in the production of corrective joint torques. Our preliminary evidence from quiet standing experiments after anesthesia of the forefoot soles suggested that plantar sensory loss produced predominantly mediolateral balance deficits (Meyer 2003; Meyer et al. 2004).[1] Consequently, we chose lateral support surface translations as the paradigm to test our hypothesis. Plantar cutaneous afferents were targeted by direct injections of local anesthetic into the skin of the foot soles, leaving foot and ankle proprioception and motor function unaffected.

Methods

Experimental subjects participated in two testing sessions separated by 1–7 days. During each test session, subjects were asked to maintain standing balance while experiencing a series of lateral support surface translations. Subjects' cutaneous foot soles were anesthetized during the second test session, while their first session served as control. The effects of plantar anesthesia on the control of unperturbed stance in the same subject group were described previously (Meyer 2003; Meyer et al. 2004).

Participants

The study group consisted of six healthy male volunteers aged 19–46 (mean 26, SD 10) years. Their masses ranged 60 to 82 kg (mean 72, SD 10 kg), and their heights ranged 168 to 181 cm (mean 174, SD 5 cm). Participants reported no history of neurological disorder, cardiac arrhythmia, or sensitivity to lidocaine. As an additional screening procedure, all subjects demonstrated the ability to remain standing with bare feet placed heel-to-toe and eyes closed for 30 s, indicating excellent balance control. The procedures used in this study were approved by the Boston University Charles River Campus Institutional Review Board and all subjects provided informed consent.

Anesthesia procedure

The use of a traditional "ankle block" was precluded in this study because it would affect foot and ankle proprioception as well as the intrinsic foot musculature. Sensation from the weight-bearing surface of the plantar soles was therefore reduced through multiple intradermal injections of an anesthetic solution by a board-certified anesthesiologist. A solution of 2% lidocaine HCL, 1:8 sodium bicarbonate, 1:200,000 epinephrine, and 12 U/mg hyaluronidase was delivered using 30 ga needles. Sodium bicarbonate served as a buffer to the acidic lidocaine, while epinephrine reduced the rate of washout and prolonged the effect of anesthesia. The use of hyaluronidase increased the area of skin affected by each injection, thereby reducing the total number of injections required. As shown in Fig. 1, anesthesia was produced in the weight-bearing skin overlying the metatarsal heads, the lateral soles, and the heels (the use of epinephrine in the digits is generally precluded; Dollery 1991; the toes therefore remained untreated.) Eight to fifteen 1 ml injections were required for each foot. In order to minimize discomfort to the subjects, injections of the anesthetic solution were performed slowly; the entire procedure required approximately 1 h to anesthetize both feet. To keep the soles clean after the injections, subjects wore thin, disposable slippers (Pillow Paws, Principle Business Ent. Inc., Dunbridge, OH) during all balance tests. Because the slippers collapsed to a thickness of less than 0.5 mm when loaded, the difference between barefoot stance and stance in these slippers was expected to be negligible. Nevertheless, identical slippers were worn during control testing.

Cutaneous pressure sensory thresholds were determined before and after anesthesia using a modified two-alternative forced-choice assessment adapted from previous studies (Holewski et al. 1988; Sosenko et al. 1990). The center of the area of skin to be anesthetized was stimulated with a series of Semmes-Weinstein nylon monofilaments (Stoeling Co., Wood Dale, IL). These filaments are designed to assess cutaneous pressure sensation and calibrated such that a specific longitudinal force is required to make them buckle. The filaments are labeled with a linear scale of perceived force (arbitrary units, or a.u.), corresponding approximately to a logarithmic scale of buckling force. Details of the pressure sensory threshold level (STL) testing procedure are provided elsewhere (Meyer and Oddsson 2003). The anesthesia procedure was considered successful if sensory thresholds were elevated above 5.07 a.u., a level normally indicative of peripheral sensory neuropathy (Holewski et al. 1988; Sosenko et al. 1990; Vinik et al. 1995). The depth of anesthesia was assessed again after the completion of balance testing.

A Disk-Criminator (Neuroregen L.L.C., Bel Air, MD) was used to determine subjects' ability to discriminate between two discrete points of pressure before and after anesthesia. This device consists of an octagonal disk with one or two stainless-steel prongs protruding from each edge. Spacing between the prongs ranges from 9 to 20 mm. The experimenter pressed the skin with the device twice in succession, alternating randomly between one and two prongs. The subject then reported how many prongs they had perceived at each application. Each spacing between pairs of prongs was tested three times. The closest spacing that could be correctly identified in at least two of three applications was designated the discrimination threshold distance for that site. Assessments were performed on the plantar skin overlying the third metatarsal and the heel of each foot.

The anesthesia procedure involved the infiltration of approximately 10 ml of fluid into the dermis of each foot sole. It was therefore important to rule out alterations in plantar skin compliance as the source of potential balance changes. The associated change in elastic skin compliance was estimated using a modified spring-loaded dial position indicator (McMaster-Carr Supply, Dayton, NJ). Stiffness of the position indicator was linearized by mounting an additional spring in series with the plunger. The modified position indicator was fixed to a precision instrumentation stage (Eric Sobotka, Farmingdale, NY) and positioned to press the plunger

Fig. 1 Anesthetized area of foot soles (*shaded*) after intradermal injection of local anesthetic

[1] The referenced articles describe two quiet stance experiments. The first, involving anesthesia of the forefoot soles, uncovered primarily mediolateral changes in balance. These results contributed to our decision to focus on mediolateral perturbation responses in the present study. The second quiet stance experiment, involving whole-sole anesthesia, was performed concurrently with the present study.

perpendicularly against the sole of the right heel. The stage was then advanced by increments of 1 mm and the corresponding position indicator measurements recorded. Elastic compliance of the skin was approximated by a linear regression of the relationship $C_{skin}=(s-d)/k_{spring}d$, where s is the stage displacement, d is the position indicator displacement, and k_{spring} is the elastic stiffness of the modified position indicator. Skin compliance was estimated before and after anesthesia. In order to minimize any difference in skin compliance between the two sensory conditions, subjects soaked their feet in warm tap water for 20 min prior to control testing.

In order to demonstrate that the anesthesia procedure had no effect on muscle proprioceptors, a brief test of toe position perception was administered before and after anesthesia. The experimenter manipulated the second or fourth toe up and down rapidly, stopping randomly in the up or down position. The subject then identified the position of the toe ("up" or "down") without seeing their feet. This procedure was repeated 10 times for each of the second and fourth toes, and any incorrect answers noted.

Balance testing

Set-up for the balance experiments is shown in Fig. 2. Lateral translations of the support surface were produced using BALDER (BALance DisturbER), a 1 kHz high-performance balance platform capable of making precise computer-controlled perturbations in the horizontal plane (Oddsson et al. 1999, 2004). Each perturbation consisted of a 25 cm lateral horizontal translation that produced an approximately trapezoidal velocity profile. All movements were initiated by a 600 cm/s^2 acceleration pulse followed by a period of constant velocity and a 980 cm/s^2 deceleration. The five different impulse magnitudes used were 1.2, 2.3, 3.5, 4.7, and 5.8 Ns, corresponding to acceleration pulses of 17, 33, 50, 67, and 83 ms in duration. The corresponding peak platform velocities were 10, 20, 30, 40, and 50 cm/s, respectively. Each combination of perturbation impulse and direction (left, right) were repeated three times in random order for a total of 30 perturbations during each testing session. Prior to the initiation of each data collection session, subjects underwent a brief training period involving a random series of five maximum perturbations in order to eliminate any startle responses (McIlroy and Maki 1995).

Subjects stood upon two Kistler 9284 multi-component force platforms (Kistler Instrument Co., Amherst, NY) mounted on the translating support surface. During the training period preceding control testing, subjects were asked to move their feet closer together after each perturbation. For each subject, the closest foot spacing that allowed them to keep both feet on the force plates during the largest magnitude perturbations was determined. This position of the feet was then marked on the force plates with tape and used during all subsequent control and anesthetized trials for that subject. Distances between the feet during recorded trials ranged from 23.2 cm to 29.7 cm (mean 25.9 cm, SD 2.9 cm). Forces and moments measured by the force plates were sampled at 100 Hz and stored on computer disk for later analysis.

Subject kinematics were measured using an Optotrak 3020 (Northern Digital Inc., Waterloo ON) motion analysis system. Subjects were viewed from behind. Infra-red markers were placed on the subjects' ankles (over the Achilles tendon at the midpoint between the maleoli), hips (greater trochanter), and shoulders (acromion). Markers at the hips and shoulders were mounted on Styrofoam cubes taped to the skin for visibility. Three additional markers were placed on each force plate in order to define the plane of movement and track the acceleration of the platform. Kinematic data were sampled at 100 Hz and stored on computer disk for later analysis.

Inverse dynamics model

Kinematic and kinetic data collected during lateral support surface translations were used to estimate the net joint torques produced at the ankles, hips, and trunk. An eight-segment inverse dynamics model (Fig. 3) was implemented using recursive Newton-Euler formulations of the joint reaction forces and moments (Winter 1990). Recursive estimation began with the force plates and progressed towards the trunk. Analysis was confined to forces and movements in the mediolateral plane. Kinematic measurements confirmed that body movements were largely confined to the mediolateral plane 70–500 ms after perturbation onset, the time interval of interest for this study. Model errors were quantified by the fictitious residual forces and moments that were applied to the upper body center-of-mass in order to maintain dynamic equilibrium.

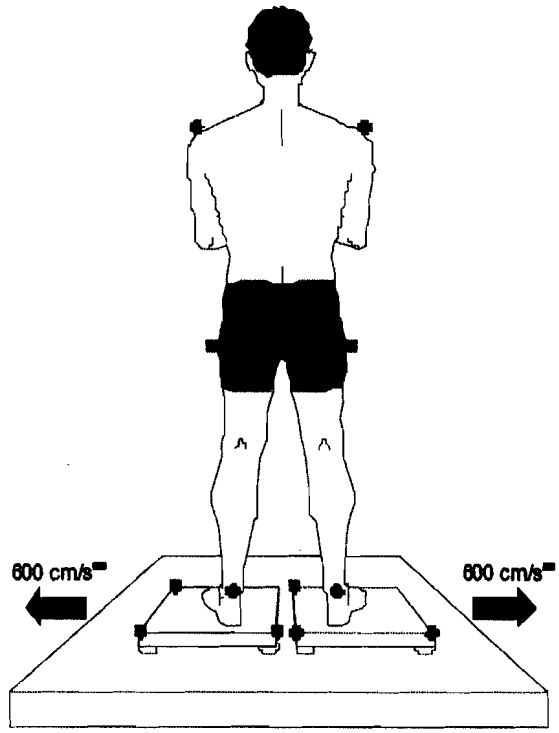

Fig. 2 Set-up for balance perturbation experiment. *Black circles* denote location of kinematic markers. Force plates measured ground reaction forces under each foot. Perturbations consisted of lateral accelerations of the support surface at 600 cm/s^2 for 17, 33, 50, 67, or 83 ms. In each case, total platform displacement was 25 cm

The inverse dynamics model involved the following assumptions. First, model segments were considered rigid and all joints were modeled as frictionless pin joints. Coriolis terms induced by measured changes in segment lengths were assumed negligible. Second, the positions of kinematic markers on the skin with respect to the underlying joint structures were assumed constant. Large lateral trunk flexion movements, which might lead to slippage between hip kinematic markers and the hip joint center, mainly occurred outside the time interval of interest (see Fig. 4). Third, the approximate locations of each segment's center-of-mass and moment of inertia were taken from the literature (Winter 1990; Zatsiorsky and Seluyanov 1983) based upon segment lengths derived from the kinematic data. Fourth, the subjects' foot soles were assumed to remain in contact with the force plates during the perturbations. This was confirmed by visual observation, although the heel of the foot ipsilateral to the perturbation direction was occasionally lifted a few millimeters during the largest perturbations. (In this paper, the terms "ipsilateral" and "contralateral" will refer to the side of the body with respect to the direction of platform translation.) Errors introduced by this assumption regarding the feet were minimal because the ipsilateral leg is almost totally unweighted during such events. Fifth, each leg was considered a single segment, allowing no lateral flexion of the knees. It is noteworthy that sagittal plane knee flexion would not violate this assumption. In any case,

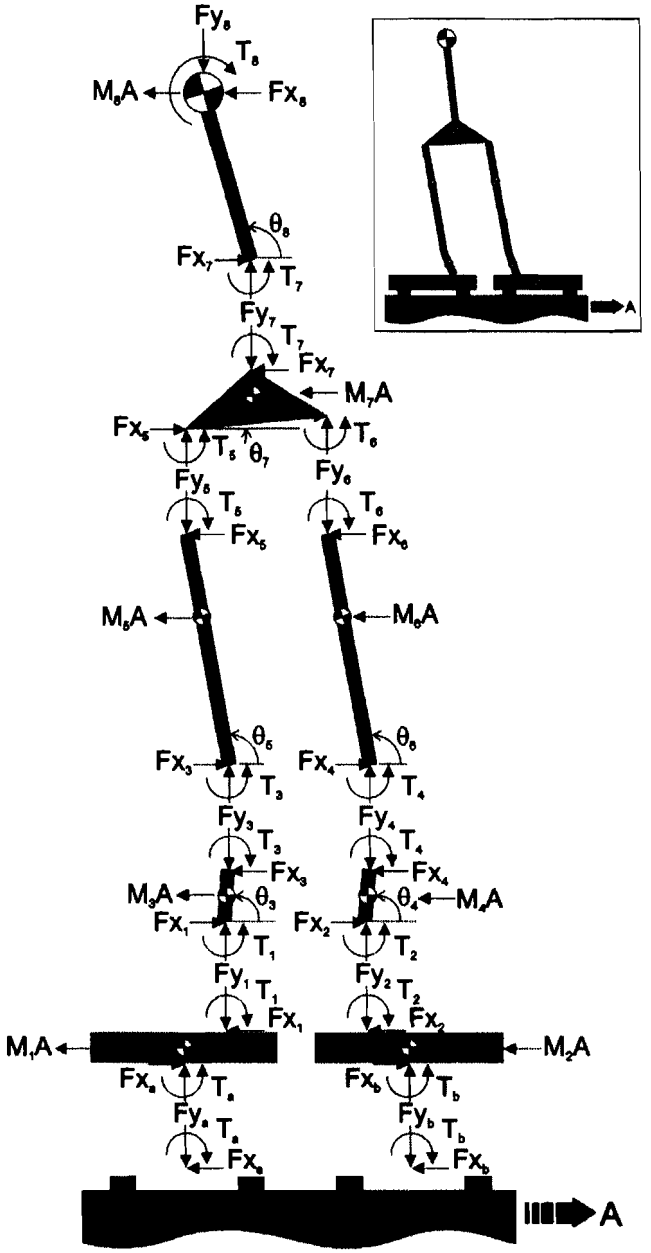

Fig. 3 Free-body diagram of the inverse dynamics model. The assembled model is shown in the *inset*. The model was perturbed by acceleration A of the support surface. The use of a non-inertial reference frame fixed to the moving platform necessitated the introduction of pseudo-forces at each segment. Forces Fx_a, Fx_b, Fy_a, Fy_b, and torques T_a, T_b were acquired from two force plates (segments 1 and 2)

measured variations in leg segment lengths did not exceed a few millimeters. Sixth, actual positions of the hip joint centers were inferred from kinematic markers over the greater trochanters and static measurements taken during manual palpation. Seventh, the trunk (including head, arms, neck, upper torso, and abdomen) was approximated by a single rigid segment with a rotation axis approximately concurrent with the lumbar spine L3/L4 joint. Subjects' arms were crossed across their chest (hands on elbows) to eliminate arm movements during the perturbations. Additional kinematic data collected in two subjects regarding head position confirmed that head movement with respect to the shoulders was negligible.

The model involved a non-inertial reference frame attached to the accelerating support surface (Fig. 3). A corresponding pseudo-force was therefore introduced for each model segment. Each pseudo-force is equal to the platform acceleration "A" multiplied by the segment mass, and opposite in direction to "A". Acceleration of the support surface was calculated by numerical double-differentiation of the position of a marker fixed to one force plate. The support surface acceleration and ground reaction forces were filtered using an adaptive quintic spline in order preserve the shape of the perturbation impulse.

Joint reaction forces and torques were expressed as changes from the initial, unperturbed state prior to perturbation. The sign and body-side of kinetic estimates derived from leftward perturbations were then adjusted to mimic rightward perturbations so that any directional asymmetries could be analyzed. The results of left and right perturbations were not pooled, however. Response torques for each trial were quantified by integrating the torque estimates over three time intervals from the onset of platform acceleration. The 70–100 ms interval reflected early reflexive responses.[2] The 100–200 ms interval included mainly automatic postural responses, as voluntary responses generally require >200 ms for implementation. The 200–500 ms interval can include both long-latency automatic responses as well as conscious behavior (Bloem et al. 2000; Nashner 1993; Slijper et al. 2002). Response torques occurring less than 70 ms after perturbation onset may include reflex activity rather than automatic postural responses and were not of interest in this study. Response torques occurring more than 500 ms after perturbation onset were contaminated by the deceleration of the support surface.

Four-factor repeated-measures analyses of variance (ANOVAs) were used to quantify the effects of perturbation magnitude, plantar sensory condition, perturbation direction, and trial number on subjects' torque responses. The perturbation magnitude factor had five levels, the sensory condition had two levels (control, anesthetized), the direction factor had two levels (left, right), and the trial factor had three levels (trials 1–3). The average magnitude of the resultant ground reaction force prior to the onset of support surface movement was used as a changing covariate. One ANOVA was performed for each combination of model joint (contra- and ipsilateral ankles and hips, trunk, model residual) and time interval (70–100 ms, 100–200 ms, 200–500 ms). The sum of torques produced at the ankles, hips, trunk, and residual was also of interest due to the anticipated reduction of intertrial variance resulting from the addition of random errors. We therefore investigated the effects of perturbation magnitude, sensory condition, perturbation direction, and trial number on the sum of torques using a 5×2×2×3 ANOVA for each time interval. The threshold for statistical significance was set at p=0.05 for all analyses. Significant main effects and interactions were investigated further using Tukey's honest significant difference test. Since sphericity could not be guaranteed in every case, a multivariate approach was used when the p value for Mauchley's test was less than 0.2. In addition, Spearman's rank correlation coefficient was used to quantify the correlation between perturbation and response torque impulse magnitudes. Statistical analyses were performed using the Statistica 5.5 software package (Statsoft Inc., Tulsa, OK).

Results

Subject responses under normal sensory conditions

Kinematics

Kinematics from a typical subject are shown in Fig. 4 for perturbation magnitudes 1, 3, and 5. The lowest perturba-

[2] Contamination of response torques by platform acceleration prevented the study of the 70–100 ms window during the two lowest perturbation magnitudes.

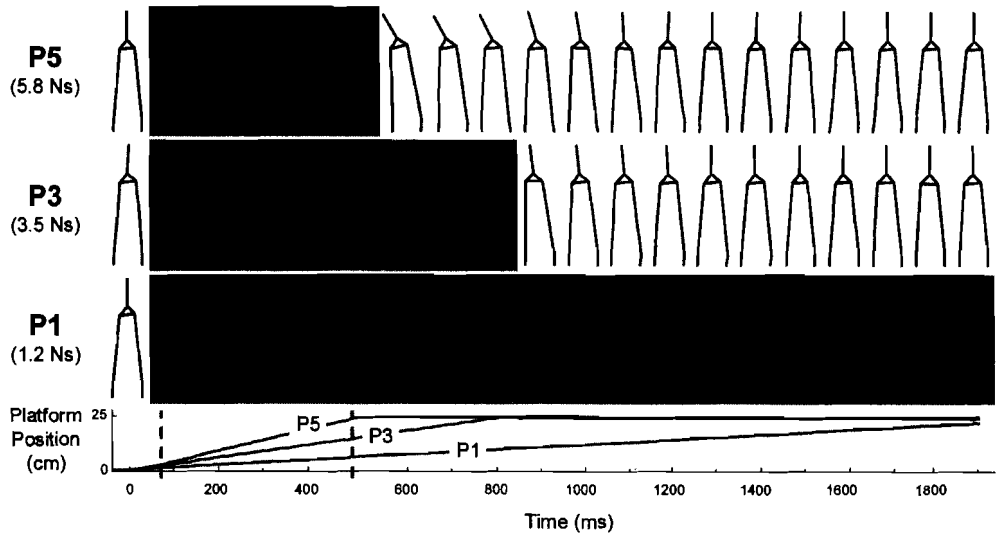

Fig. 4 Kinematics of a typical subject during three different perturbation magnitudes. *Stick figures* from representative trials are derived from kinematic measurements used in the inverse dynamics model. Figures are shown at 100 ms increments following the onset of support surface acceleration. *Shading* highlights periods of constant platform velocity. Platform positions in global (inertial) coordinates corresponding to the figures above are graphed in the *bottom panel*. Statistical analyses in this study were confined to the time interval 70–500 ms after the onset of support surface acceleration (between *dashed vertical lines in bottom panel*).

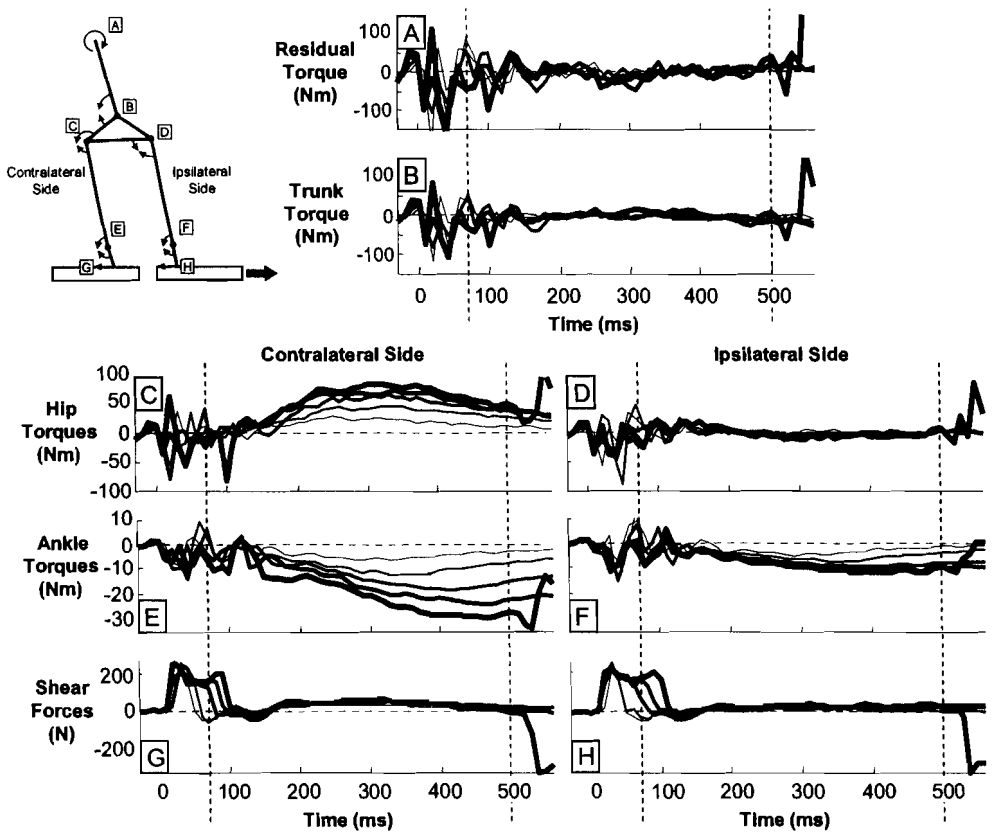

Fig. 5 Measured shear forces and net joint torque estimates for a typical subject under control sensory conditions. Torque amplitudes are expressed as changes from the resting torque prior to each perturbation. Time is measured with respect to the onset of support-surface acceleration. *Each line* represents an average of three rightward perturbations. *Increasing line thickness* denotes increasing perturbation impulse (note the general increase in response torque with perturbation magnitude). *Dashed vertical lines* bound the time interval of interest (70–500 ms). As in the text, the terms "ipsilateral" and "contralateral" are used in reference to the direction of support surface movement. A simplified version of the inverse dynamics model is shown in the *upper left*, with *arrows* denoting applied shear forces and how positive net joint torques (by the sign convention of Fig. 3) would cause adjacent segments to approach one another. *Panels G* and *H* depict the shear forces measured by force plates under each foot. *Panels E* and *F* depict ankle torques, while *C* and *D* show torque estimates for each hip. Estimated torques at the base of the trunk are shown in *panel B*. The fictitious residual torques in *panel A* represent the error present in the model

tion magnitude (P1) required very little movement by the subjects to retain standing balance. In contrast, the largest perturbation impulse (P5) involved dramatic orientation changes in the leg and trunk segments. In this case, complete recovery of vertical orientation in the trunk occurred >1 s after perturbation onset and was aided by deceleration of the support surface.

Net joint torques

Shear forces measured by the force platforms and the corresponding net joint torque estimates derived from the inverse dynamics model are shown for a typical subject in Fig. 5. Rightward perturbations are depicted; no differences between leftward and rightward perturbations were noted under control sensory conditions. The magnitudes of torques produced at both ankles (Fig. 5E, F) and the contralateral hip (Fig. 5C) increased with increasing perturbation impulse (increasing line thickness in the figure). In contrast, there was comparatively little torque produced at the ipsilateral hip (Fig. 5D) and lumbar trunk (Fig. 5B). In response to the support-surface acceleration, an abduction (clockwise) torque was produced at the contralateral hip, reaching its peak between ~250 and 350 ms after the perturbation onset (Fig. 5C). Hip abduction was aided by the counterclockwise ankle torque, which tended to invert the contralateral foot. The magnitude of the contralateral ankle torque reached its peak at around 300 ms for the smallest perturbation, but did not peak until ~475 ms after the largest perturbation impulse (Fig. 5E). The magnitude of torque produced at the ipsilateral ankle (Fig. 5F) was approximately one-third of that seen in the contralateral ankle. The residual torques, occurring 0–50 ms after perturbation onset (Fig. 5A), were related to the abrupt increase in platform acceleration. These errors occurred before the expected onset of automatic postural responses (minimum 70 ms) and therefore lay outside the time interval of interest.

Net joint torque impulse/average net joint torques

As anticipated from the data shown in Fig. 5, there was a statistically significant main effect of perturbation magnitude on the net joint torque impulse responses ($p<<0.001$ at all joints and time intervals). A minor training effect was seen in the contralateral ankles and hips, consisting of a small (<<1 Nm) shift in torque production from the 100–200 ms to 200–500 ms time interval (main effect of trial number, $p\leq0.033$). The only notable effect of perturbation direction was a slight increase (<1 Nm) in ipsilateral ankle torque production during rightward perturbations compared to leftward perturbations (100–200 ms and 200–500 ms intervals, $p\leq0.011$).

Since the time intervals of interest in this study were fixed, average torque within a given interval was proportional to the corresponding torque impulse. For clarity, the average torques produced at each joint under normal sensory conditions are depicted in Fig. 6. In the 200–500 ms range, there was a clear scaling of ankle (Fig. 6E, F) and contralateral hip (Fig. 6C) torques with increasing perturbation impulse. In contrast, trunk torques did not scale monotonically with perturbation magnitude (Fig. 6B). The average trunk torque reached its peak during perturbation P3 (3.5 Ns) and was reduced during

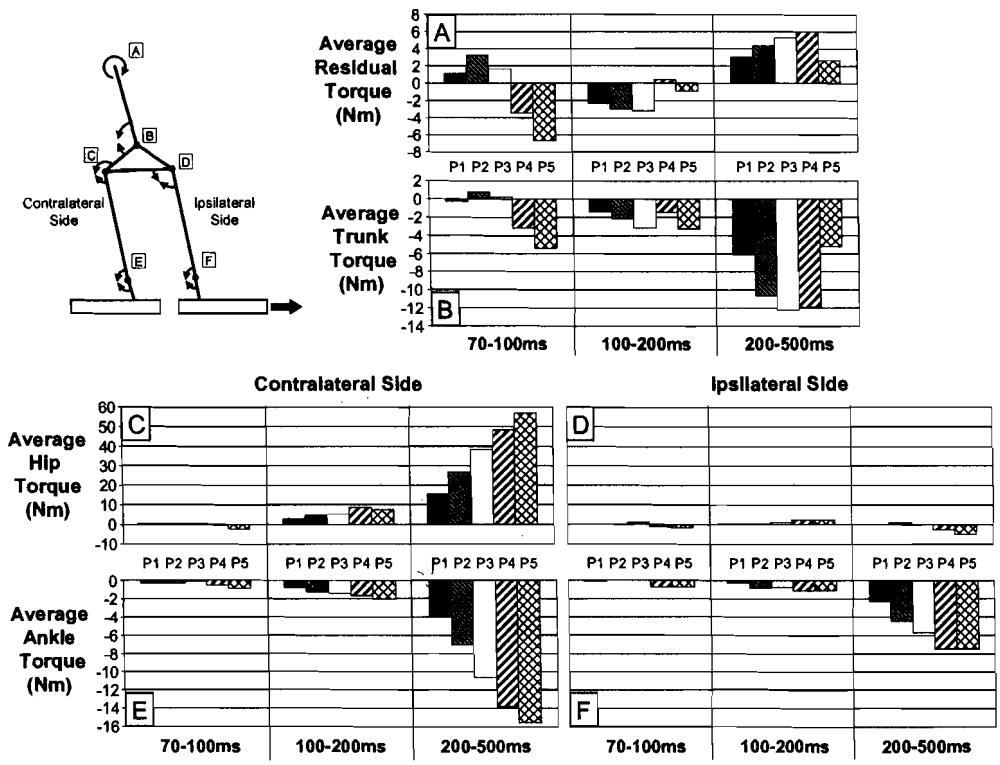

Fig. 6 Average net joint torques with normal plantar sensation. Torque amplitudes are expressed as changes from the resting torque prior to each perturbation. Each *bar* represents an average across subjects, perturbation directions, and repetition (statistical analyses described in the text involve within-subjects tests). See Fig. 5 for labeling scheme

Table 1 Correlation between perturbation impulse and response torque impulse. Rank correlation coefficients are averaged across subjects. Correlations that did not reach statistical significance ($p<0.05$) in at least five of the six subjects are noted with an *x*

	70–100 ms	100–200 ms	200–500 ms
Contralateral ankle	−0.42	−0.59	−0.88
Ipsilateral ankle	−0.51	−0.48	−0.74
Contralateral hip	−0.44	0.63	0.96
Ipsilateral hip	−0.51	−0.48	−0.74
Trunk	−0.58	x	x
Residual	−0.60	x	x

the largest perturbation. The average trunk torque was negative, corresponding to an ipsilateral flexion torque in the lower spine. Residual torque (Fig. 6A) was positive during the 200–500 ms interval, indicating that the estimated trunk torques could not fully account for the corrective clockwise rotation of the trunk segment. The repeated measures ANOVAs demonstrated no main effect of perturbation direction or trial number on torque integrals.

Correlation between response torque impulse and perturbation magnitude

A statistically significant main effect of perturbation magnitude on torque responses was further explored using Spearman's rank correlation. Correlations between perturbation and response torque impulse magnitudes within the three time intervals of interest are shown in Table 1. The Spearman rank correlation coefficients have been averaged across subjects and perturbation directions. Data from control trials are shown; there were no statistical differences between correlation coefficients from control and anesthetized trials. As expected from Fig. 6, correlations involving contralateral hip and ankle torques were quite high ($|R|>0.88$) within the 200–500 ms interval.

Effects of plantar anesthesia on perturbation responses

Efficacy of anesthesia

The intradermal injections of anesthesia caused only moderate discomfort and were well tolerated by all subjects. Unpleasant sensations were associated with the injection of fluid rather than the insertion of the 30 ga needles. Plantar skin compliance estimates averaged (±SD) 1.2±0.2 mm/N prior to anesthesia and 1.1 ±0.3 mm/N after the injections. A Wilcoxon test of compliance estimates confirmed that there was no significant change in elastic skin compliance associated with the injection of fluid into the dermis ($p>0.7$, average change 9 ±28%). Likewise, there were no changes in subjects' ability to perceive movements of the toes. Subjects reported a sensation that their soles had dramatically increased in thickness but exhibited no subjectively observable functional deficits after anesthesia.

Prior to anesthesia, average pressure sensory threshold levels under the heels and first metatarsal heads were 4.14 (±0.5) and 4.15 (±0.5) a.u., respectively. Immediately following the anesthesia procedure, STLs were elevated to

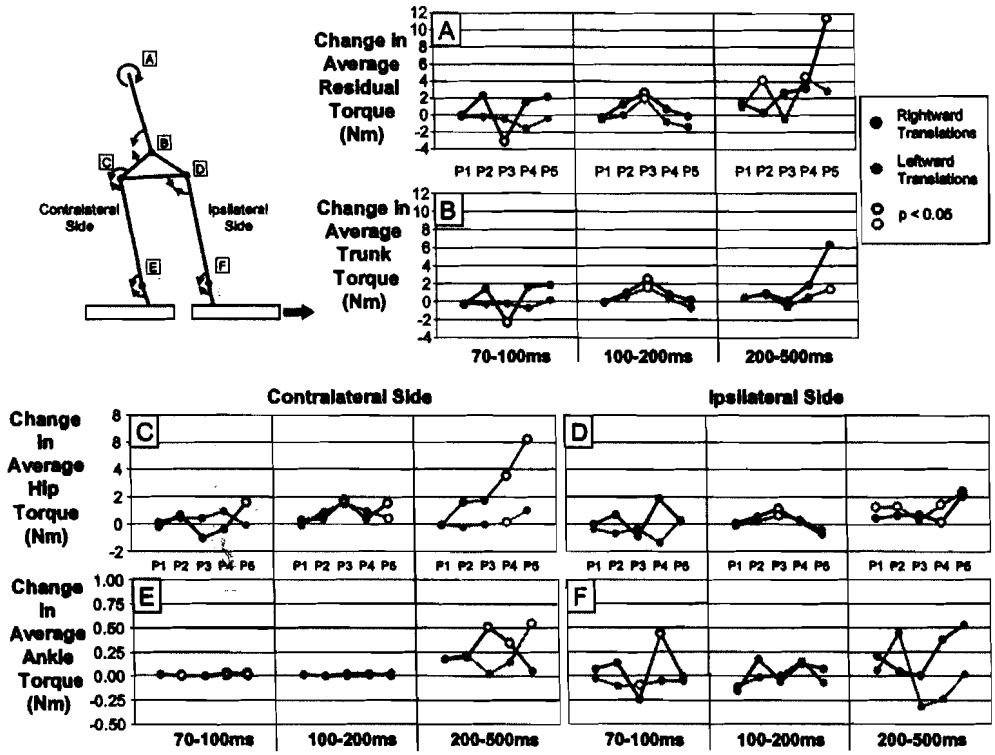

Fig. 7 Changes in average net joint torques associated with anesthesia of the foot soles. The effect of plantar anesthesia is quantified by the change in net joint torques from the control condition. For each subject, the difference between average anesthetized and control torques was calculated; *each point in the figure* is an average across subjects. *Dark lines* represent rightward support surface translations, while *gray lines* depict leftward translations. *Open circles* denote statistically significant ($p<0.05$) changes. *Error bars* have been omitted for clarity. For reference, the maximum between-subjects standard deviations were 3.7 Nm (ankles), 9.4 Nm (hips), 9.1 Nm (trunk), and 14.6 Nm (residual). See Fig. 5 for labeling scheme

or above 6.65 a.u. (the stiffest filament) in 23 of 24 test sites (96%). After approximately 1 h of standing, 17 sites (71%) remained at or above 6.65 a.u. (buckling of the 6.65 a.u. filament requires the application of approximately 4.4 N over 2 mm^2). All tested sites remained above the target threshold of 5.07 a.u. for the duration of the balance experiments. Prior to anesthesia, the average 2-point discrimination threshold under the third metatarsal heads was 12.5 mm. After anesthesia, all thresholds exceeded the device maximum of 20 mm and remained so for the duration of balance testing.

Changes in net joint torques with plantar anesthesia

Kinematics, torque responses, and average net joint torques from the anesthetized trials were qualitatively similar to the control data shown in Figs. 4, 5, and 6, respectively. Changes in average net joint torques associated with cutaneous anesthesia of the foot soles are shown in Fig. 7. The effect of anesthesia was not always equal between rightward and leftward support surface translations. This asymmetry may be related to the fact that all subjects were either right-handed or ambidextrous and preferred to kick with their right leg. For the most part, changes in net joint torques after anesthesia were small. For instance, the statistically significant changes in contralateral ankle torques (Fig. 7E, p=0.001) in the 70–100 ms interval constituted large relative increases, but their magnitudes (<1 Nm) were probably of little functional relevance. Within the 200–500 ms interval, there was a small decrease in the magnitude of contralateral ankle (inversion) torques (Fig. 7E, p=0.031). The increases in contralateral hip abduction torque (Fig. 7C) were larger in magnitude, reaching an average of 6 Nm (~11%) during the largest rightward perturbations ($p \leq 0.032$). While changes in trunk torque (Fig. 7B, $p << 0.001$) were similar in magnitude to those at the hip, they represent an average decrease in magnitude of stabilizing ipsilateral flexion of the trunk (see Fig. 6B for net torque magnitudes). The magnitude of this change approached 100% during the largest rightward perturbations, indicating approximately zero average lateral flexion of the trunk during the 200–500 ms interval with plantar anesthesia. The corresponding increase in residual torque magnitude (Fig. 7A, $p \leq 0.049$) suggests that the largest perturbations may have also induced subtle head or arm movements that were not explicitly included in the model.

Figure 8 depicts the average net body torques (i.e. sum of ankle, hip, trunk, and residual torques) under control and anesthetized conditions during the 200–500 ms interval. Plantar anesthesia was associated with a statistically significant increase (p=0.043) in net body torque within this interval. No significant difference was found within the 70–100 ms or 100–200 ms intervals.

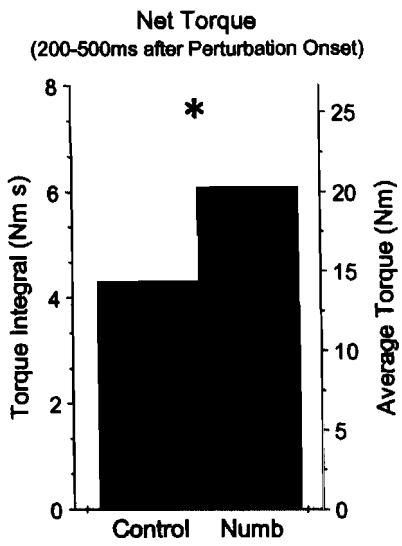

Fig. 8 Comparison of net torque integrals before and after anesthesia. Torque integrals over the range 200–500 ms after perturbation onset are measured on the *left vertical scale;* the corresponding average torque over this range is shown on the *right scale*. Net torque was calculated by summing the ankle, hip, trunk, and residual torques. Data is pooled across all perturbations and averaged across subjects. The difference between control and anesthetized conditions was statistically significant (p=0.043) using a repeated measures ANOVA

Discussion

To our knowledge, net joint torques produced in response to lateral support surface translations have not been previously reported. Our results from control conditions are qualitatively similar to the net joint torques previously seen in response to lateral shoulder pushes (Rietdyk et al. 1999). This similarity was expected, since acceleration of the support surface is equivalent to applying a pseudo-force to each body segment in the opposite direction (see Fig. 3). One of the major findings of Rietdyk et al. (1999), however, was that response torque impulse magnitudes were not scaled with perturbation impulse magnitude. They therefore concluded that subject responses were appropriately scaled to the largest *anticipated* perturbation, with sensory feedback providing little information regarding the perturbation magnitude. Another study of lateral hip pushes found that the sum of ankle and hip torque responses 500 ms after perturbation onset did not change with perturbation magnitude (Matjacic et al. 2001). In contrast to these earlier studies, we noted a very clear scaling of ankle and contralateral hip torque impulses with the perturbation impulse magnitude (see Fig. 6, Table 1). Correlation between perturbation impulse and contralateral hip torque impulse within the 200–500 ms interval averaged 0.95, and was never below 0.89 for any subject. This correlation was still considerable (R=0.65) within the 100–200 ms interval, while moderate correlations (R= −0.5) between perturbation impulse and ipsilateral limb torques were found in the 70–100 ms interval. Since the perturbation amplitude and direction were not known prior to the onset of acceleration, these results suggest that sensory cues regarding perturbation magnitude were

sufficiently informative and timely to modulate response torque production.

During lateral support surface translations, subjects produced contralateral ankle inversion, ipsilateral ankle eversion, contralateral hip abduction, and ipsilateral trunk flexion moments in order maintain stability. Using the sign convention adopted in Fig. 3, these corrective torques were negative at the ankles and trunk and positive in the contralateral hip. The net body torque (sum of ankle, hip, trunk, and residual torques using the aforementioned sign convention) within the 200–500 ms interval was increased by over 40% following plantar anesthesia. The net body *corrective* torque (sum of absolute joint torques), however, demonstrated no change with anesthesia over the same time interval. Together, these findings suggest that positive hip torques increased in magnitude while negative ankle and trunk torques decreased in magnitude. The changes shown in Fig. 7 support this assertion. During the larger perturbation magnitudes, anesthesia of the soles resulted in increased contralateral hip abduction torques. At the same time, contralateral ankle inversion and ipsilateral flexion of the trunk decreased in magnitude. Thus, plantar anesthesia induced an increased reliance on the hip abductors for the production of corrective torque.

This conclusion is consistent with several previous studies of anteroposterior dynamic balance after peripheral sensory loss. Hypoxic anesthesia of the feet and ankles caused an increased reliance on the so-called "hip strategy" to compensate for posterior support surface accelerations (Horak et al. 1990). Similar results were seen in diabetic peripheral neuropathy patients when subject to conflicting somatosensory, visual, and vestibular cues (Simmons et al. 1997). In a different study of diabetic peripheral neuropathy patients, however, Inglis et al. (1994) noted only a delayed "ankle strategy" response to posterior support surface translations. Since shear forces were not reported, it is unclear whether there were concomitant increases in hip flexion associated with peripheral neuropathy.

Our results also appear to be consistent with the only other study investigating the effects of plantar sensory loss on lateral perturbation responses. Perry et al. (2000) studied compensatory stepping responses after hypothermic anesthesia of the soles. Under control conditions, their subjects responded to lateral support surface translations by making a cross-over stepping maneuver. In contrast, their subjects tended to make shuffling side-step responses after plantar anesthesia. This change in stepping behavior may be a consequence of the relative shift of compensatory torque production from the ankles to the hips seen in the present study. Production of inversion torque in the ankle tends to increase vertical load on the lateral aspect of foot. However, after support surface translation, a side-step of the contralateral leg first requires a shift in weight to the ipsilateral leg followed by eversion of the contralateral ankle to produce the necessary ground clearance. When subjects have already responded to the perturbation with a strong ankle inversion torque, the remaining corrective torque generated by the hip abductors produces little shift in body weight to the ipsilateral foot. The cross-over step is then the easier response to implement. In contrast, increased corrective torque production from the contralateral hip may produce the shift in body weight necessary to implement a side step. The corresponding reduction in ankle inversion moment will also facilitate the eventual eversion of the (contralateral) swing foot. Thus a shift in relative torque production from the ankles to the hips may increase the likelihood of a side-step response to lateral support surface translations. It is therefore plausible that the changes in lateral stepping behavior seen by Perry et al. (2000) directly result from an increase in the relative contribution of hip torques to overall stability.

The relative shift in corrective torque production from the ankles to the hips may be due to a sensory conflict between intact lower limb proprioception and impaired plantar mechanoreception. Given the perceived ambiguity regarding the nature of the support surface when the soles are numb, it seems logical that the balance control system would place greater emphasis on more proximal sensors to maintain stability. Such a shift in sensory weighting need not involve a discrete switch in balance control strategy. Rather, it may be a result of the mechanics of the movement. In response to support surface tilts, studies suggest that foot somatosensation only triggers postural responses at the ankle joints, while more proximal triggers are responsible for upper leg and trunk muscle activity (Bloem et al. 2000, 2002). The support surface translations used in the present study produced a chain of successive joint angle changes beginning with the ankles. Sensory conflict between foot and ankle proprioceptors and the anesthetized soles may have decreased the magnitude of ensuing ankle responses. Alternatively, decreased activation of reflexive connections between cutaneous afferents and the peripheral musculature (McNulty et al. 1999) could be responsible for the reduced ankle muscle activation. Reduced ankle inversion torque would produce a concurrent reduction in leg abduction. Proprioceptors within the hip joints might then sense that normal leg abduction is reduced and consequently trigger compensatory increases in hip abductor activity. Greater hip abduction would tend to reduce the angle between pelvis and trunk, thereby reducing the required lateral trunk flexor activation. Thus, the reduction in ankle inversion torque caused by conflicting foot and ankle sensation may indirectly lead to the increased hip abduction and decreased lateral trunk flexion torques seen in our subjects.

Previous studies of anteroposterior support surface translations have demonstrated that subjects respond to small perturbations using predominantly the ankle musculature ("ankle strategy"). As the magnitude of the perturbation increases, there emerges a "hip strategy" in which substantial response torques are produced at the hips (Runge et al. 1999). With temporary loss of foot and ankle sensation induced by pressure cuffs, subjects adopt a hip strategy in response to support surface translations that normally warrant an ankle strategy response (Horak et al. 1990). Thus, loss of peripheral sensation caused subjects

to produce a response appropriate for a larger perturbation. In the present study, involving lateral perturbations, we too saw an increase in the relative importance of hip response torques after loss of foot sole sensation. However, there was no evidence within the range of perturbations studied that such a shift in response strategy normally occurs with increasing perturbation impulse. In fact, the ratio of hip torque integrals to ankle torque integrals was roughly constant across the range of perturbations implemented in this study. From the evidence available, it appears that the relative shift in response torque from the ankles and trunk to the hips seen in the present study represents an abnormal behavior rather than an overreaction to the perturbation magnitude. Further studies involving a larger range of lateral perturbation impulses are required to confirm this assertion.

While the effects of anesthesia did not cause our subjects to fall, it is clear that the loss of plantar sensation modified the normal postural responses of healthy subjects. By shifting torque production from the ankles to the hips, subjects adopted an abnormal postural strategy in response to lateral support surface translations. Since they were otherwise free of sensorimotor dysfunction and exhibited excellent balance, it is likely that this population was better able to compensate for deficits in a single sensory system than the general population. In any case, we can expect that the effects of plantar sensory loss would be more pronounced under more difficult balance conditions, such as the conflicting visual and vestibular information (e.g., vection) or the unstable footing often encountered in everyday life. Likewise, balance difficulties related to proprioceptive deficits are likely exacerbated by reduced sensitivity of the cutaneous foot sole. The fact that even healthy subjects were affected by foot sole anesthesia suggests that the loss of plantar sensation may be an important contributor to the dynamic balance deficits previously seen in diabetic peripheral neuropathy patients (Bloem et al. 2000; Inglis et al. 1994). An elderly population exhibiting subclinical peripheral sensory deficits may also be at increased risk for falls and fall related injuries. Further studies involving patients and matched controls are required to better understand the effects of peripheral neuropathy on dynamic postural responses.

Our results demonstrate that plantar cutaneous afferents mediate postural responses to lateral perturbations of balance. There is some evidence that foot sole sensation may play an even greater role in the maintenance of anteroposterior stability. Previous studies have shown that ankle torque responses to posterior support surface translations are modified by hypoxic anesthesia of the feet and ankles (Horak et al. 1990) or peripheral neuropathy (Inglis et al. 1994). Our own work has demonstrated that although cutaneous anesthesia of the forefoot soles produces predominantly mediolateral balance effects during unperturbed stance, deficits associated with whole-sole anesthesia are confined to the anteroposterior plane (Meyer 2003; Meyer et al. 2004). From a mechanical perspective, the human body is inherently more stable in the frontal plane than the sagittal plane due to the wider base of support. This fact suggests that anteroposterior stability may be more dependent upon sensory feedback than is mediolateral balance. Additional studies are required to determine if the loss of plantar sensation does indeed induce greater balance deficits during anteroposterior disturbances than during mediolateral perturbations.

This study produced two important findings. First, healthy subjects scale net response torques from the ankles and hips to the impulse associated with lateral support surface translations. These results demonstrate that subject responses are modulated by sensory input received during the course of the perturbation. Second, reduced sensitivity of the cutaneous foot soles results in a relative redistribution of corrective torques from the ankles and trunk to the hip joints. Both these findings support the notion that foot sole sensation is an important component of the human balance control system. Reduced plantar sensation may therefore be an important contributor to the balance deficits seen in peripheral neuropathy patients.

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