Interjoint dynamic interaction during constrained human quiet standing examined by induced acceleration analysis

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Interjoint dynamic interaction during constrained human quiet standing examined by induced acceleration analysis. J Neurophysiol 111: 313–322, 2014. First published October 2, 2013; doi:10.1152/jn.01082.2012.—Recent studies have demonstrated that human quiet standing is a multijoint movement, whereby the central nervous system (CNS) is required to deal with dynamic interactions among the joints to achieve optimal motor performance. The purpose of this study was to investigate how the CNS deals with such interjoint interaction during quiet standing by examining the relationship between the kinetics (torque) and kinematics (angular acceleration) within the multi-degree of freedom system. We modeled quiet standing as a double-link inverted pendulum involving both ankle and hip joints and conducted an “induced acceleration analysis.” We found that the net ankle and hip torques induced angular accelerations of comparable magnitudes to the ankle (3.8°/s² and 3.3°/s²) and hip (9.1° ± 3.2°/s² and 10.5° ± 3.5°/s²) joints, respectively. Angular accelerations induced by the net ankle and hip torques were modulated in a temporally antiphase pattern to one another in each of the two joints. These quantitative and temporal relationships allowed the angular accelerations induced by the two net torques to countercompensate one another, thereby substantially (~70%) reducing the resultant angular accelerations of the individual joints. These results suggest that, by taking advantage of the interjoint interaction, the CNS prevents the net torques from producing large amplitudes of the resultant angular accelerations when combined with the kinematic effects of all other torques in the chain.

quiet standing; interjoint coordination; biomechanics; induced acceleration analysis; multisegment model

THE HUMAN UPRIGHT STANCE is inherently unstable for purely mechanical reasons: the large body mass is maintained high above a relatively small base of support. In addition, experimental evidence indicates that the passive mechanical properties (stiffness and/or viscosity) of the ankle musculature are not enough to offset the toppling effect of gravity (Casadio et al. 2005; Loram and Lakie 2002b); therefore, asymptotic stability cannot be achieved even though spinal feedback mechanisms are incorporated (van Soest and Rozendaal 2008). Numerical simulation with a multi-degree of freedom (DoF) model of quiet standing has further demonstrated that the passive mechanical properties of the muscles around the three leg joints (i.e., ankle, knee, hip) are still insufficient to ensure stability even at maximum cocontraction of these muscles (van Soest et al. 2003). All of these factors demand an active stabilization by the central nervous system (CNS) to maintain the upright stance. The required active control mechanisms have attracted considerable attention from researchers and have been extensively studied.

It is well known that when relatively large perturbations are applied to a standing body, multiple coordination patterns between the ankle and hip joints (hip strategy) are observed (Horak and Macpherson 1996). For smaller perturbations, however, the ankle motion alone (ankle strategy) had long been considered to be sufficient to maintain balance. On the basis of these notions, most researchers model human quiet standing as a single-link inverted pendulum (SIP) that pivots at the ankle joint in the sagittal plane (Asai et al. 2009; Fitzpatrick et al. 1992; Loram et al. 2005a; Masani et al. 2003; Morasso and Schieppati 1999; Peterka 2000; Sasagawa et al. 2009b; Winter et al. 1998). These investigations based on the SIP model have the advantage of simplifying the control problem by reducing the number of variables, which then enables researchers to focus their analyses on the control mechanism around the ankle joint where the destabilizing effect of gravity is greatest.

The SIP model of quiet standing has recently been challenged on the basis of experimental evidence (Accornero et al. 1997; Aramaki et al. 2001; Creath et al. 2005; Günther et al. 2009, 2011a, 2011b, 2012; Pinter et al. 2008; Sasagawa et al. 2009a; Zhang et al. 2007). For example, Aramaki et al. (2001) and Sasagawa et al. (2009a) reported that the angular acceleration of the hip joint during quiet standing was even greater than that of the ankle joint. They also noted that these two angular accelerations were modulated consistently in antiphase to one another, thereby minimizing the horizontal acceleration of the body’s center of mass (CoM). A few recent studies have indicated that joints other than the ankle and hip (e.g., the knee) also contribute to controlling quiet standing (Günther et al. 2009, 2011a, 2011b, 2012; Hsu et al. 2007; Pinter et al. 2008). These findings clearly indicate that quiet standing is a multijoint movement rather than a single-joint movement, as assumed in the SIP paradigm. Investigations based on the multi-DoF model are then required to establish a physiologically plausible strategy adopted by the CNS for controlling quiet standing (Suzuki et al. 2012).

During a single-joint movement the kinetics-kinematics relationship is straightforward, because the magnitude of a torque is proportional to the angular acceleration it induces. During a multijoint movement, however, the relationship between kinetics (torque) and kinematics (angular acceleration) is complicated because of the dynamic interaction among the joints (Hirashima 2011; Hollerbach and Flash 1982; Scott 2004; Zajac and Gordon 1989). Torque at one joint induces angular acceleration not only at its corresponding DoF but also at remote joints. Such dynamic interaction among joints has
been intensively investigated, especially for upper limb movements. For example, when performing a horizontal reaching movement involving shoulder and elbow joints, the shoulder extension torque also accelerates the elbow into flexion (Gribble and Ostry 1999). Thus, to perform an optimal multijoint movement, it is necessary for the CNS to deal with such dynamic interjoint interaction.

Previous studies on healthy subjects demonstrated that the effects of such interjoint interactions are appropriately compensated for (Gribble and Ostry 1999; Hollerbach and Flash 1982) or used (Hirashima et al. 2003, 2007, 2008) during multijoint movements. During fast reaching movements, however, cerebellar ataxia patients exhibit a curved hand trajectory and target overshoot because of their inability to deal with the interjoint interaction (Bastian et al. 1996). Given that quiet standing is a multijoint movement, it has been hypothesized that interjoint interaction exerts a significant effect on postural dynamics during quiet standing and that such an effect is appropriately dealt with by the CNS for the efficient maintenance of balance.

The purpose of this study was to investigate how the CNS deals with the interjoint interaction during quiet standing. We studied this problem by examining the relationship between kinetics and kinematics within the multi-DoF system. As it is difficult to understand such a relationship intuitively, Zajac et al. (2002) proposed a mathematical method for addressing this issue. This method, “induced acceleration analysis,” enables us to quantify the mutual contribution of individual torques to accelerate the joint motions throughout the body. In this study, we modeled quiet standing as a minimum multi-DoF system—a double-link inverted pendulum (DIP)—involving both the ankle and hip joints. We then quantified the mutual contributions of ankle and hip joints to the angular accelerations of the two joints.

MATERIALS AND METHODS

Subjects. Eight men (mean ± SD: age 27.5 ± 1.6 yr, height 175.0 ± 6.0 cm, body mass 72.0 ± 8.5 kg) participated in this study. All subjects were healthy and had no history of known neurological or musculoskeletal disorders. They gave written informed consent for the study after receiving a detailed explanation of the purpose, potential benefits, and risks involved. The experimental procedures used in this study were in accordance with the Declaration of Helsinki and were approved by the Committee on Human Experimentation at the Graduate School of Arts and Sciences, The University of Tokyo.

Experimental protocol and measurements. Subjects were required to stand barefoot quietly on a firm surface with eyes open (EO) or eyes closed (EC). Five 60-s trials of each visual condition were alternately repeated, with sufficient rest between the trials. The subjects held their arms comfortably by their sides, with their feet at shoulder width and parallel to each other. Three wooden splints were used to restrict head-neck-trunk and knee motions. One splint (160 g) was strapped to the back of the subject at the forehead, chest, and pelvis. Other splints (80 g each) were strapped behind the knees of each leg. We used these splints to ensure correct DIP approximation of quiet standing by allowing motions to occur exclusively around the ankle and hip joints (Aramaki et al. 2001). Although such restriction could disturb the natural characteristics of quiet standing, we used it to measure the motion of a two-segmented body without ambiguity.

Joint motion data were obtained with a three-dimensional (3D) optical motion capture system (OptiTrack V100-R2; NaturalPoint, Corvallis, OR) composed of six infrared cameras in a semicircular arrangement. Spherical reflective markers, 11 mm in diameter, were affixed to the ankle (lateral malleolus), hip (great trochanter), and shoulder (acromion) on the right side of the subject’s body. The kinematic signals were sampled at a rate of 100 Hz and stored on the hard disk of a personal computer for later off-line analysis.

Data analysis. The standing body was modeled as a DIP consisting of two rigid segments: the leg and the head-arms-trunk (hereafter referred to as the “trunk”). In the analysis, the kinematics of the right leg was assumed to represent that of both legs. The kinematic signals were digitally smoothed with a dual-pass second-order low-pass Butterworth filter (filtfilt function in the MATLAB signal processing toolbox). The cutoff frequency was set to 2.0 Hz. We computed the segment lengths (l₁ and l₂) and joint angles (qₐ and qₕ), where subscripts “a” and “h” stand for ankle and hip, respectively, in the sagittal plane from the 3D coordinate data of the reflective markers for each data sample (Fig. 1). Joint angles and torques were defined as positive in extension. The angular velocities (q″ₐ and q″ₕ) and angular accelerations (q‴ₐ and q‴ₕ) were computed by numerically differentiating the angular displacement data with a three-point central difference formula (Winter 1990). The calculation procedures for the segment lengths and joint angles are presented in APPENDIX A.

Dynamic equations of motion for the DIP model of quiet standing were derived with Lagrange’s method (Alexandrov et al. 2005; Edwards 2007; van der Kooij et al. 1999; Winter 1990):

\[ \Theta(q, q\dot{}) = T + V(q, q\dot{}) + G(q) \]

In Eq. 1, \( q \) is the vector of joint angles, \( \dot{q} \) is the angular velocity, and \( \ddot{q} \) is the angular acceleration; \( T \) is the vector of joint torques; \( V(q, \dot{q}) \) is the vector of gravitational torques, which is a function of the hip joint angle \( \dot{q}_h \); \( G(q) \) is the vector of gravitational torques, which is a function of the two joint angles \( \dot{q}_a \) and \( \dot{q}_h \). The complete set of equations of motion for the DIP model and the details of the system inertia matrix are presented in APPENDIX B and APPENDIX C, respectively.

In the coordinate representation, Eq. 1 can be written more explicitly as:

\[ \begin{pmatrix} \Theta_{11} & \Theta_{12} \\ \Theta_{21} & \Theta_{22} \end{pmatrix} \begin{pmatrix} \dot{q}_a \\ \dot{q}_h \end{pmatrix} = \begin{pmatrix} T_a \\ T_h \end{pmatrix} + \begin{pmatrix} \frac{V_a(q, \dot{q})}{q_h} \\ \frac{V_h(q, \dot{q})}{q_h} \end{pmatrix} + \begin{pmatrix} G_a(q) \\ G_h(q) \end{pmatrix} \]

The joint torques at the ankle and hip joints were calculated from the kinematic data obtained in the experiment and standard anthropometric parameters (Winter 1990) as follows:

\[ T = \Theta(q, q\dot{}) - V(q, q\dot{}) - G(q) \]

Fig. 1. Double-link inverted pendulum (DIP) sagittal-plane model of human quiet standing. q, Joint angle; T, joint torque; r, distance to the center of mass from the distal joint of the segment; l₁, segment length. Subscripts a and h denote ankle and hip, respectively, and subscripts 1 and 2 reflect properties of the leg and trunk segments, respectively. The inclination of the body is exaggerated for the reader’s convenience.
By multiplying the inverse of the system inertia matrix \( \Theta(q)^{-1} \) on both sides of Eq. 1, we can distinguish the contribution of individual torque components (joint torque, \( T \); velocity-dependent torque, \( V \); and gravitational torque, \( G \)) of the ankle and hip joints to the ankle and hip angular accelerations (Zajac et al. 2002) (Fig. 2, phase A):

\[
\ddot{q} = \Theta(q)^{-1}(T + V(q, \dot{q}) + G(q))
\]

Fig. 2. Dynamics of the DIP model. Note that because velocity-dependent torques are negligible during quiet standing (\( \approx 10^{-3} \text{Nm} \)), they are not shown here. Left: torque components. Center: local joint kinematics. Right: global kinematics. In phase A, solid and dashed arrows denote direct and remote effects, respectively. \( k_1 \) and \( k_2 \) are weighting coefficients determined from anthropometric parameters (Sasagawa et al. 2009a).

As in Eq. 2, Eq. 4 can be expressed in the coordinate representation as:

\[
\begin{bmatrix}
\ddot{q}_a \\
\ddot{q}_h
\end{bmatrix} = \frac{1}{\det\Theta} \begin{pmatrix}
\Theta_{22} & -\Theta_{12} \\
-\Theta_{21} & \Theta_{11}
\end{pmatrix} \begin{bmatrix}
T_a \\
V_a(q, \dot{q}) + G_a(q)
\end{bmatrix} = \begin{bmatrix}
\sum_{i=a,h} c_i(q)T_i + \sum_{i=a,h} c_i(q)V_i(q, \dot{q}) + \sum_{i=a,h} c_i(q)G_i(q) \\
\sum_{i=a,h} d_i(q)T_i + \sum_{i=a,h} d_i(q)V_i(q, \dot{q}) + \sum_{i=a,h} d_i(q)G_i(q)
\end{bmatrix}
\]

In Eq. 5, \( c_i(q) \), for example, denotes \( \Theta_{i2}/\det\Theta \). Each of \( c_i(q) \) and \( d_i(q) \), which represents the “gain” from the torques to the angular acceleration (van Asseldonk et al. 2007), is a function of the hip joint angle \( \theta_h \) (see APPENDIX C). To clarify how the values (and signs) of \( c_i(q) \) and \( d_i(q) \) \((i \in \{a,h\})\) depend on \( \theta_h \), we present color-coded plots of them for a representative subject in Fig. 3. Vertical arrows in Fig. 3 appropriately indicate the mean hip joint angle \( (5.59^\circ) \) during a single trial. From this figure, we can see that \( c_a(q) \) and \( c_h(q) \) (each of the two is the “gain” from the ankle and hip torques, respectively, to the angular acceleration of the ankle joint) and \( d_a(q) \) and \( d_h(q) \) (each of the two is the “gain” from the ankle and hip torques, respectively, to the angular acceleration of the hip joint) at the mean joint configuration have opposite signs.

We can express Eq. 5 in another form:

\[
\begin{bmatrix}
\ddot{q}_a \\
\ddot{q}_h
\end{bmatrix} = \begin{bmatrix}
\sum_{i=a,h} \ddot{q}_i q_h T_i + \sum_{i=a,h} \ddot{q}_i V_i(q, \dot{q}) + \sum_{i=a,h} \ddot{q}_i G_i(q) \\
\sum_{i=a,h} \ddot{q}_i q_h T_i + \sum_{i=a,h} \ddot{q}_i V_i(q, \dot{q}) + \sum_{i=a,h} \ddot{q}_i G_i(q)
\end{bmatrix}
\]

where \( \ddot{q}_i q_h \), for example, denotes the contribution of the ankle joint torque \( (T_a) \) to ankle angular acceleration \( (\ddot{q}_a) \). A net contribution of a
single joint “i” to angular acceleration of its corresponding DoF or that of a remote joint “k” is the sum of the angular accelerations induced by the joint torque, velocity-dependent torque, and gravitational torque of the joint \( (\ddot{q}_{i, \text{NET}} = \ddot{q}_{i, \text{NET}}^T + \ddot{q}_{i, \text{NET}}^V + \ddot{q}_{i, \text{NET}}^G) \) (Fig. 2, phase B). This is equivalent to the angular acceleration induced by a net torque of the joint \( (\ddot{q}_{i, \text{NET}} = \ddot{T}_i + \sqrt{V_i} + G_i) \). This induced acceleration analysis has been used to scrutinize the contribution of individual muscle forces or joint torques to the accelerations of the body during pedaling (Zajac et al. 2002), walking (Zajac et al. 2003), standing (Challis 2011; van Asseldonk et al. 2007), and throwing (Hirashima and Ohtsuki 2008; Hirashima et al. 2008).

For the time series of each net torque, the power spectral density (PSD) function was calculated (Welch function in the MATLAB signal processing toolbox). The data for a single trial were divided into 10 segments (1,024 data points each) with 50% overlap. A 10-bit fast Fourier transform algorithm was applied to each segment to yield the power spectrum after being passed through a Hamming window. The power spectra of these segments were ensemble-averaged into the PSD function for a single trial. The mean power frequency (MPF) of each net torque for the 0- to 2-Hz bandwidth was calculated as follows:

\[
\text{MPF} = \frac{\int_0^2 \frac{f}{P} \, df}{\int_0^2 P \, df}
\]

where \( f \) and \( P \) denote the frequency and PSD, respectively.

The normalized cross-correlation \( (R_{xy}) \) gives the correlation between two time-domain signals \( [x(t)] \) and \( [y(t)] \) as a function of the time shift \( (\tau) \):

\[
R_{xy}(\tau) = \frac{x(t + \tau)y(t)}{\sqrt{x^2(t)\, y^2(t)}}
\]

where the overbar denotes an average over time \( (t) \). A normalized cross-correlation of 1 indicates that the two signals are in perfect positive correlation, \(-1\) indicates a perfect negative correlation, and 0 indicates no correlation. The value at which \( R_{xy} \) was statistically different from zero \((P < 0.001, n = 6,000)\) was calculated to be 0.04 in this case.

Because visual feedback did not have a significant influence on the results of this study, we analyzed the data for the EO and EC conditions together. The mean value across 10 trials (5 EO and 5 EC trials) was used as a representative value for each subject. The results are given as means \( \pm \) SD.

RESULTS

Torque components and net torques of ankle and hip joints.

Figure 4A shows the typical time series of the joint torques \( (T_a \) and \( T_b) \) and gravitational torques \( (G_a \) and \( G_b) \) of the ankle and hip joints during a 30-s EC trial (same trial as shown in Fig. 3). Because the magnitudes of the velocity-dependent torques \( (V_a \) and \( V_b) \) were negligible \((-10^{-5} \text{ Nm})\), they are not presented here. For all subjects and trials, \( T_a \) consistently had a positive sign (the plantar flexor torque was exerted), whereas the sign of \( T_b \) varied depending on the alignment of the trunk segment relative to earth vertical. In other words, in the subject whose trunk leaned forward (or backward) \( T_b \) had a positive (or negative) sign. The overall mean magnitudes of \( T_a \) and \( T_b \) were 28.9 \pm 11.9 and 5.9 \pm 3.2 \text{ Nm}, respectively. It is important to note that, except for a few high-frequency wiggles, the joint torque and gravitational torque were approximately mirror images of one another in each of the ankle and hip joints. That is, the ankle and hip joint torques always kept close to those required to offset the toppling effects of gravity. However, net torques existed in both joints because of instantaneous lack of balance between the joint torques and the gravitational toppling torques. Figure 4B shows the typical time series of the net ankle \( (\text{NET}_a) \) and hip \( (\text{NET}_b) \) torques during the same trial as shown in Fig. 4A.

Frequency-domain analysis was used to characterize the temporal features of the net torques of the ankle \( (\text{NET}_a) \) and hip \( (\text{NET}_b) \) joints. Figure 5 illustrates the PSD functions of \( \text{NET}_a \) (Fig. 5, top) and \( \text{NET}_b \) (Fig. 5, bottom). The PSD functions presented are the ensemble average across all trials and all subjects. Note also that for this ensemble-averaging procedure the PSD functions for each subject were calculated relative to the total power in the 0- to 2-Hz band. For both net torques, it can be seen that most of the power was distributed around 1 Hz. The MPFs of \( \text{NET}_a \) and \( \text{NET}_b \) were calculated to be \( 0.90 \pm 0.11 \text{ Hz} \) and \( 0.96 \pm 0.11 \text{ Hz} \), respectively.

Kinematic effects of individual torque components.

Figure 6A illustrates a typical time series of the angular acceleration components of the ankle (Fig. 6A, left) and hip (Fig. 6A, right) joints induced by the joint torque and the gravitational torque during the same trial as shown in Figs. 3 and 4. As in Fig. 4A, the contributions of the velocity-dependent torques were negligible \((-10^{-5} \text{ Nm/s})\), so they are not presented here. Although the magnitudes of the torques around the hip joint \( (T_b \) and \( G_b) \) were about one-fifth of those around the ankle joint \( (T_a \) and \( G_a) \), the angular accelerations induced by the hip torques are comparable to those induced by the ankle torques (compare
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It is also important to note that the contributions of NET\textsubscript{a} and NET\textsubscript{h} are consistently counterdirected to one another in both the ankle and hip joints. When a cross-correlation analysis was performed for each joint, the maximum negative correlation of statistical significance ($P < 0.001$) was found between $\tau = -0.02$ s and $+0.01$ s. Note that a positive value of $\tau$ means that the angular acceleration induced by the ankle net torque precedes that induced by the hip net torque. The correlation coefficients (at $\tau = 0$) between $\dot{q}_a^{\text{NET}_a}$ and $\dot{q}_a^{\text{NET}_h}$ and between $\dot{q}_h^{\text{NET}_a}$ and $\dot{q}_h^{\text{NET}_h}$ were both calculated to be $-0.95 \pm 0.01$. Because of the opposite signs within the rows of $\Theta(q)^{-1}$ (the opposite signs emerge when the matrix is inverted; compare Eq. 2 to Eq. 5 and see also Fig. 3), a phase relationship between the net torques is reversed when their kinematic effects are analyzed. That is, the reciprocal relationships between the net-torque-induced accelerations (Fig. 6B) were products of a consistent in-phase relationship between NET\textsubscript{a} and NET\textsubscript{h} (Fig. 4B). Such a consistent in-phase relationship between NET\textsubscript{a} and NET\textsubscript{h} can be seen more clearly when plotted on a torque plane (Fig. 7).

As a result of these quantitative and temporal relationships, the contributions of NET\textsubscript{a} and NET\textsubscript{h} are significantly counter-compensated by one another, leading to resultant joint angular accelerations whose amplitudes are about one-third of those induced by each net torque. The RMS values of $\ddot{q}_a$ and $\ddot{q}_h$ were calculated to be $1.2 \pm 0.5$ and $3.4 \pm 1.2$°/s², respectively (Fig. 2, phase C).

At the final stage (Fig. 2, phase D), the anterior/posterior CoM acceleration ($\ddot{X}_{\text{CoM}}$) is a linear summation of the individual joint angular accelerations (Aramaki et al. 2001; Kuo 1995; Sasagawa et al. 2009a):

$$\ddot{X}_{\text{CoM}} = k_1 \ddot{q}_a + k_2 \ddot{q}_h$$

where $k_1$ and $k_2$ are weighting coefficients determined from anthropometric parameters (Sasagawa et al. 2009a). The above-mentioned reductions in the individual joint angular accelerations are expected to result in decreased amplitude of the whole body’s horizontal acceleration during quiet standing.

DISCUSSION

How does the CNS deal with interjoint interaction during quiet standing? In the present study, we conducted an induced acceleration analysis to investigate how the CNS deals with interjoint dynamic interaction during quiet standing. Two major results were obtained: 1) The net torques of the ankle and hip joints induced angular accelerations of magnitudes comparable to the ankle ($\dot{q}_a^{\text{NET}_a}$, 3.8 ± 1.4°/s²; $\dot{q}_a^{\text{NET}_h}$, 3.3 ± 1.2°/s²) and hip ($\dot{q}_h^{\text{NET}_a}$, 9.1 ± 3.2°/s²; $\dot{q}_h^{\text{NET}_h}$, 10.5 ± 3.4°/s²) joints, respectively. 2) The angular accelerations induced by the net ankle and hip torques were consistently counterdirected to one another in each of the two joints [$R_{xy}(0) = -0.95 \pm 0.01$]. With such quantitative and temporal relationships between $\dot{q}_a^{\text{NET}_a}$ and $\dot{q}_a^{\text{NET}_h}$ and between $\dot{q}_h^{\text{NET}_a}$ and $\dot{q}_h^{\text{NET}_h}$, the resultant angular accelerations of the ankle ($\ddot{q}_a$) and hip ($\ddot{q}_h$) joints were substantially reduced (~70%).

As we show in Fig. 4B, net torques existed in the ankle and hip joints because of the instantaneous lack of balance between the joint torques and the gravitational toppling torques. A net torque can result, for example, from ballistic patterns of torque

Fig. 5. Power spectral density functions of NET\textsubscript{a} (top) and NET\textsubscript{h} (bottom). Lines indicate the ensemble average across all trials and all subjects. Gray areas represent SE across subjects. Mean power frequencies of NET\textsubscript{a} and NET\textsubscript{h} for the 0- to 2-Hz band were calculated to be 0.90 ± 0.11 Hz and 0.96 ± 0.11 Hz, respectively.

Fig. 4A to Fig. 6A). This was due to the human mechanical design [i.e., $\Theta(q)^{-1}$ in Eq. 4, determined from the length, mass, and moment of inertia of the segments and other factors]. That is, the second column of $\Theta(q)^{-1}$ [typically (~0.12 0.36)², in kg⁻¹·m⁻²], which determines the kinematic effects produced by the hip torques, is about three times the size of the first column [typically (0.05 –0.12)², in kg⁻¹·m⁻²], which determines the kinematic effects of the ankle torques (Fig. 3).

Kinematic effects of net torques. As mentioned above, net torques existed in the ankle and hip joints because of the instantaneous lack of balance between the joint torques and the gravitational toppling torques (Fig. 4B), which exert destabilizing effects on the stance by accelerating the joint motions directly or remotely. Figure 6B illustrates a typical time series of the mutual contributions of the net ankle (NET\textsubscript{a}) and hip (NET\textsubscript{h}) torques to accelerate the ankle (Fig. 6B, left) and hip (Fig. 6B, right) motions, respectively (same trial as shown in Figs. 3, 4, and 6A). The superimposed thin lines in Fig. 6B are the resultant, measured angular accelerations of the ankle ($\ddot{q}_a$) and hip ($\ddot{q}_h$) joints. Note that only 5 s of data from the 60-s trial is presented in this figure to isolate the signal features. As for the magnitudes of the net torque contributions to ankle joint, the root mean square (RMS) of $\dot{q}_a^{\text{NET}_a}$ and $\dot{q}_a^{\text{NET}_h}$ were calculated to be 3.8 ± 1.4°/s² and 3.3 ± 1.2°/s², respectively. Similarly, for the contributions to the hip joint, the RMS of $\dot{q}_h^{\text{NET}_a}$ and $\dot{q}_h^{\text{NET}_h}$ were 9.1 ± 3.2°/s² and 10.5 ± 3.5°/s², respectively. These results indicated that, in both joints, the magnitudes of the remote effects (angular acceleration induced by the net torque of its corresponding DoF).

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exertion used in an intermittent control strategy (Asai et al. 2009; Bottaro et al. 2008; Loram et al. 2005b; Loram and Lakie 2002a; Suzuki et al. 2012) or internal noise in the feedback control loop (Masani et al. 2003; Maurer and Peterka 2005; Peterka 2000). Loram and colleagues (Loram et al. 2005b; Loram and Lakie 2002a) showed that balance in upright standing is achieved by active adjustments of the ankle joint torque with a ballistic-like "drop and catch" pattern (if the pendulum is being raised, it is called a "throw and catch" pattern). During the drop phase, ankle joint torque decreases below that required for equilibrium (\( \tau_a^{\text{NET}} \)), which accelerates the CoM forward. The drop phase is temporally followed by the catch phase (\( \tau_a^{\text{NET}} \)), where the CoM is decelerated. This pattern of torque used to control the CoM is necessarily associated with calf muscle activity and muscle length adjustment. Indeed, the temporal features of the net torques characterized by the PSD functions (Fig. 5) mirror that of the muscle length adjustment demonstrated by Loram et al. (2005b). That is, both the net torques and muscle length adjustment during quiet standing have most of their power around 1 Hz.

Although we cannot identify the exact mechanism by which the net torque is produced, it results from the instantaneous lack of balance between a joint torque and the gravitational toppling torque and exerts destabilizing effects on quiet standing by accelerating joint motions directly or remotely. This study demonstrated, however, that in each joint the direct effect from the net torque of its corresponding DoF (\( \ddot{q}_i^{\text{NET}} \)) and the remote effect from the net torque of the adjacent joint (\( \ddot{q}_k^{\text{NET}} \)) were almost compensated by one another. This result suggests that, by taking advantage of the interjoint interaction, the CNS may prevent the net torques from producing large amplitudes.

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**Fig. 6.** A: typical time series of the angular acceleration components of the ankle joint (left) and hip joint (right) induced by joint torques (thin lines) and gravitational torques (bold lines) for a single EC trial (same trial as shown in Figs. 3 and 4). Although the magnitudes of the torques around the hip joint (\( T_h \) and \( G_h \)) are approximately 1/5th of those around the ankle joint (\( T_a \) and \( G_a \)), the angular accelerations induced by the hip torques are comparable to those induced by the ankle torques. B: typical time series of the contribution of the net ankle (\( \tau_a^{\text{NET}} \)) and hip (\( \tau_h^{\text{NET}} \)) torques to accelerate ankle (left) and hip (right) motions, respectively, for a single EC trial (same trial as shown in A and in Figs. 3 and 4). Note that only 5 s of data from the 60-s trial is presented to isolate the signal features. In both joints, the angular accelerations induced by the net ankle and hip torques are counterdirected to one another, thereby reducing the resultant angular accelerations (\( \ddot{q}_a \) and \( \ddot{q}_h \)).

**Fig. 7.** Example of a substantial correlation between \( \tau_a^{\text{NET}} \) and \( \tau_h^{\text{NET}} \) as a trajectory on the torque plane (same trial as shown in Figs. 3, 4, and 6). Note that the scale of the x-axis is approximately 3 times greater than that of the y-axis.
of the resultant angular accelerations when combined with the kinematic effects of all the other torques in the chain. In other words, the combined variance of the net-torque-induced accelerations \( q_i^{\text{NET}} + q_i^{\text{NET}} \) was constrained mostly to the uncontrolled manifold (UCM; Scholz and Schöner 1999) of each resultant angular acceleration \( q_i^{\text{NET}} \) in our case. Recently, Hsu et al. (2007), using UCM analysis, demonstrated that the angular motions of six major joints along the longitudinal axis of the body during quiet standing were coordinated such that their combined variance had minimal effect on the CoM and head positions. Although our present study and that of Hsu et al. (2007) differ in the number of DoFs modeled (2 vs. 6) and in the controlled variable (joint angular acceleration vs. CoM and head positions), our study is in agreement in that multiple variables in the chain are coordinated to minimize their combined effect on the critical controlled variables rather than being suppressed independently. Such a control scheme has been observed in various motor tasks such as sit-to-stand (Scholz and Schöner 1999), shooting (Scholz et al. 2000), and pointing (Tseng et al. 2002) and has been proven advantageous for achieving optimal motor performances in the face of signal-dependent noise (Todorov and Jordan 2002).

Origin of hip joint torque during quiet standing. In Fig. 4A, we demonstrated that the hip joint torque was modulated according to the requirement to offset the toppling effect of gravity, which is proportional to the trunk segment angle with respect to earth vertical (see Appendix B). Although previous studies have already shown a substantial amount of hip joint torque and its modulation during quiet standing (Günther et al. 2009, 2011a, 2011b), the mechanism responsible for such torque is still unclear. In this regard, Saffer et al. (2008) found a lack of coherence between the trunk segment angle and electromyogram (EMG) activities of the trunk muscles (rectus abdominis, erector spinae muscles) during quiet standing. On the basis of this finding, the authors concluded that the trunk muscles have no active control over the trunk’s segment motion. Because their examination was limited to the superficial trunk muscles, however, there is still a possibility that deep trunk muscles (e.g., ilioipsas muscles) actively contributed to generation of hip joint torque. In addition, Saffer et al. (2008) analyzed the “trunk segment” angle rather than the “hip joint” angle as adopted in the present study. Such a difference in the definition of the DoF may have had an influence on their conclusion (Günther et al. 2011b; Sasagawa et al. 2009a). Nevertheless, another possible factor contributing to hip joint torque lies in the passive mechanical properties of the hip musculature. That is, “joint torque” computed by inverse dynamics reflects the passive, mechanical (i.e., stiffness and/or viscosity) contributions by muscles, aponeurosis, tendons, and other connective tissues, as well as the active contribution of muscle contraction. Such passive contributions cannot be captured by EMG measurements.

Phase relationship between net-torque-induced angular accelerations. When looking at the phase relationship between the kinematic effects of NET\(_a\) and NET\(_h\) in each joint, we found that the induced accelerations were consistently counter-directed to one another (Fig. 6B). This reciprocal relationship between the net-torque-induced accelerations, by which the resultant angular acceleration was substantially reduced, was brought about by a consistent in-phase relationship between NET\(_a\) and NET\(_h\) (Fig. 7). Previous studies have suggested that kinetic and/or kinematic coupling between adjacent joints (i.e., ankle-knee, knee-hip) during quiet standing can be mediated by the biarticular muscles spanning the knee joint, such as the gastrocnemius, hamstrings, and rectus femoris muscles (Günther et al. 2009, 2011b; Rozendaal and van Soest 2007). Because there are no muscles spanning the ankle and hip joints simultaneously, however, instantaneous coupling between the net torques cannot be substantiated in a simple mechanical way.

Although it is beyond the scope of this study to illuminate the mechanism responsible for consistent phase coupling between the net torques, it is plausible that the extensor (and/or flexor) muscles of the ankle and hip joints receive temporally synchronized motor commands from the CNS. Such simultaneous activations of the ankle and hip joints can be achieved, in principle, by “neural coupling between muscles” as proposed by Rozendaal and van Soest (2007). More recently, Suzuki et al. (2012) demonstrated by a numerical simulation that the off-off/on-on reduced intermittent controller (i.e., simultaneous activation/inactivation of the active ankle and hip torques) successfully stabilized the DIP model of quiet standing by taking advantage of the dynamics on the two stable manifolds. These manifolds are associated with, respectively, a monotonic in-phase mode—where the ankle and hip angles change monotonically together in the same directions toward the upright position—and a stable, oscillatory antiphase mode—where the ankle and hip angles oscillate in opposite directions toward the upright position. Furthermore, Kiemel et al. (2008) experimentally showed nearly in-phase EMG responses of the ankle and hip muscles to visual scene movement during upright stance and considered these responses to be scaled versions of a single control signal.

It should be noted, however, that simultaneous activation of the ankle and hip joints is not the only solution to stabilize the DIP. In fact, Suzuki et al. (2012) demonstrated that a full intermittent controller (switching among off-off/on-off/off/on-on of the active ankle-hip torques) could also stabilize the DIP in the whole range of the passive hip stiffness. In addition, Kuo et al. (1998) and Creath et al. (2005) demonstrated, at least kinematically, that postural coordination between the ankle and hip joints during upright standing was altered when somatosensory information was disrupted by sway-referencing of the support surface. These results imply that the descending postural commands to individual joints are multivariate, rather than univariate, in nature, and that spatiotemporal coordination of these motor commands can be optimized in a task-specific manner.

The multi-DoF models of quiet standing may provide insight into underlying mechanisms responsible for the deterioration of postural control in some neurological disorders, such as sensory (LaFond et al. 2004) or cerebellar (Diener et al. 1984) ataxia. Previous studies have reported that patients without proprioception (Sainburg et al. 1995) and those with cerebellar lesions (Bastian et al. 1996) could not deal with interjoint interactions appropriately. For example, abnormally curved end-point trajectories may be produced during fast, multijoint arm movements. Such deficits were attributed to an inability of these patients to update an internal model of the limb’s dynamic properties (Sainburg et al. 1995). Because an internal model of the body’s inertial properties is necessary for precise
motor coordination, it is possible that the interjoint coordination during quiet standing is disturbed by neurological disorders. If, in an extreme case, the phase relationship between the ankle and hip net torques changes from the in-phase mode to an antiphase mode, the net torques would augment their contributions from one another, thereby increasing the resultant angular accelerations.

Control strategy of the multi-DoF human body. As we quantitatively demonstrated in this study, the human mechanical design [i.e., \(\Theta(q)^{-1}\)] determines how dynamic interaction among joints occurs within the multi-DoF system and at the same time requires the CNS to adjust the joint actuations to each other with appropriate strategies. Kuo (2005) suggested that the interjoint interactions make purely local feedback (controlling each joint depending solely on feedback from its own joint) an unsuitable stabilization strategy. To ensure stability under these dynamic interactions, it is necessary for the CNS to estimate the overall orientation of the body and limbs and then control each joint based on the estimate. An estimation of body dynamics can theoretically be obtained with the Kalman filter, which has been proposed as a model for the sensorimotor integration process in the CNS (Wolpert et al. 1995). The Kalman filter produces an optimal state estimation and then control each joint based on the estimate. An estimation of body dynamics with a motor command (“efference copy”) and sensory feedback (“afference copy”) with the internal model of the body and sensor dynamics. Several theoretical studies have been published in which the multi-DoF model of a standing human was balanced against internal or external perturbations with the use of an optimal estimator (Kuo 1995, 2005; van der Kooij et al. 1999, 2001).

Limitations. We should bear in mind that our present result (i.e., countercompensation of the net-torque-induced accelerations) was obtained when the standing body was modeled as a DIP. Although joints other than the ankle and hip were constrained with splints in our experiment, these joints essentially contribute to controlling balance during quiet standing (Günther et al. 2009, 2011a, 2011b, 2012; Hsu et al. 2007; Pinter et al. 2008). When the knee joint is released, for example, the spatiotemporal coordination pattern among the joints is complicated. That is, the net-torque-induced accelerations are expected to distribute near a UCM of a given joint angular acceleration in a 3D state space.

It should be also noted that knee joints might play a role in decoupling joint kinematics between both right and left legs during natural standing. In contrast, our application of constraints to the subjects’ knees more or less forced the two legs to act congruently. Although the subjects did not have any trouble maintaining balance while wearing splints, it is possible that the characteristics of natural quiet standing were partly lost because of the unnatural experimental condition. Although further study with a more complex and more accurate model of the body is needed to deepen our understanding of postural control during quiet standing, we believe that our analysis based on the DIP model gives us deeper insight than have previous studies based on the conventional SIP model.

Our present investigation focuses on the mechanical interaction occurring between the ankle and hip DoFs during quiet standing. If we want to go further and identify the dynamics of the neural feedback pathway in the closed-loop postural control system, we need to externally perturb the system along with system identification techniques to untangle the dynamics of both the controller and plant (van der Kooij et al. 2005). In particular, when identifying such dynamics in a multi-DoF system (such as DIP), multiple perturbations need to be applied (Boonstra et al. 2013).

Conclusions. In the present study, we investigated the manner in which the CNS deals with the interjoint, dynamic interaction during constrained quiet standing. Induced acceleration analysis revealed that the net ankle and hip torques induced angular accelerations of magnitudes comparable to the ankle and hip joints, respectively. It was also revealed that the angular accelerations induced by the two net torques were modulated in a temporally consistent antiphase pattern to one another in each of the ankle and hip joints. Based on these quantitative and temporal relationships, the net-torque-induced accelerations countercompensated one another in each joint. Hence, the resultant angular accelerations of the individual joints were substantially reduced. These results suggest that, by taking advantage of the interjoint interaction, the CNS may prevent the net torques—which result, for example, from the ballistic patterns of torque used in the intermittent control strategy or the internal noise in the feedback control loop—from producing large amplitudes of the resultant angular accelerations when combined with the kinematic effects of all other torques in the chain. The present argument has an important implication: Analyzing a single DoF cannot reveal the basis of dynamic control of the DoF when interacting with other DoFs. The CNS achieves the desired motor performances by smartly taking advantages of the redundant DoFs. Therefore, if we want to uncover the motor control strategies adopted by the CNS, more attention should be paid to coordination among the DoFs and the dynamic interaction that occurs among them.

APPENDIX A: CALCULATION OF SEGMENT LENGTHS AND JOINT ANGLES

\[ l_1 = \sqrt{(x_h - x_2)^2 + (z_h - z_2)^2} \]
\[ q_a = \tan^{-1}\left(\frac{z_h - z_2}{x_h - x_2}\right) \]
\[ q_b = \tan^{-1}\left(\frac{z_a - z_h}{x_a - x_h}\right) - q_a \]

where \( l \) is the length of the segment; \( (x, z) \) are two-dimensional Cartesian coordinates of the individual markers; \( q \) is angle of the joint; subscripts 1 and 2 reflect properties of the leg and trunk segments, respectively; and subscripts a, h, and s denote the ankle, hip, and shoulder, respectively.

APPENDIX B: DYNAMIC EQUATIONS OF MOTION FOR THE DIP MODEL

\[ T_a = \dot{q}_a[\Theta_1 + \Theta_2 + m_1 r_1^2 + m_2 r_2^2 + m_1 l_1^2 + 2m_2 l_1 r_2 \cos q_h] \]
\[ + \dot{q}_b[\Theta_2 + m_2 r_2^2 + m_2 l_2 r_2 \cos q_h] \]
\[ - \dot{q}_a[2m_2 l_2 r_2 \sin q_h] \]
\[ + \dot{q}_a[2m_2 l_2 r_2 \sin q_h] \]
\[ + \dot{q}_a[m_1 r_1 \cos q_h + m_1 l_1 \cos q_a + m_2 r_2 \cos (q_a + q_b)] \]

\[ T_b = \dot{q}_a[\Theta_2 + m_2 r_2^2 + m_2 l_2 r_2 \cos q_h] \]
\[ + \dot{q}_b[\Theta_2 + m_2 r_2^2] \]
\[ + \dot{q}_a[m_2 l_2 r_2 \sin q_h] \]
\[ + \dot{q}_b[m_2 l_2 r_2 \sin q_h] \]
\[ + \dot{q}_b[m_2 l_2 r_2 \sin q_h] \]

where \( \Theta \) is moment of inertia about the center of gravity; \( r \) is distance to the CoM from the distal joint of the segment; \( m \) is mass of the segment; subscripts 1 and 2 reflect properties of the leg and trunk.
APPENDIX C: SYSTEM INERTIA MATRIX

\[
\begin{bmatrix}
\theta_1 \\
\theta_2 \\
\theta_3 \\
\theta_4 \\
\theta_5
\end{bmatrix} = \begin{bmatrix}
\Theta_{11} & \Theta_{12} \\
\Theta_{21} & \Theta_{22} \\
\Theta_{31} & \Theta_{32} \\
\Theta_{41} & \Theta_{44} \\
\Theta_{51} & \Theta_{52}
\end{bmatrix} \begin{bmatrix}
\theta_1 \\
\theta_2 \\
\theta_3 \\
\theta_4 \\
\theta_5
\end{bmatrix} + \begin{bmatrix}
m_1 \gamma_1 + m_2 \gamma_2 \\
2m_1 \gamma_1 \cos \theta_k + m_2 \gamma_2 \\
\theta_1 \\
\theta_2 \\
\theta_3
\end{bmatrix}
\]

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