Human postural sway results from frequent, ballistic bias impulses by soleus and gastrocnemius

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It has been widely assumed for nearly a century, that postural muscles operate in a spring-like manner and that muscle length signals joint angle (the mechano-reflex mechanism). Here we employ automated analysis of ultrasound images to resolve calf muscle (soleus and gastrocnemius) length changes as small as 10 μm in standing subjects. Previously, we have used balancing of a real inverted pendulum to make predictions about human standing. Here we test and confirm these predictions on 10 subjects standing quietly. We show that on average the calf muscles are actively adjusted 2.6 times per second and 2.8 times per unidirectional sway of the body centre of mass (CoM). These alternating, small (30–300 μm) movements provide impulsive, ballistic regulation of CoM movement. The timing and pattern of these adjustments are consistent with multisensory integration of all information regarding motion of the CoM, pattern recognition, prediction and planning using internal models and are not consistent with control solely by local reflexes. Because the system is unstable, errors in stabilization provide a perturbation which grows into a sway which has to be reacted to and corrected. Sagittal sway results from this impulsive control of calf muscle activity rather than internal sources (e.g. the heart, breathing). This process is quite unlike the mechano-reflex paradigm. We suggest that standing is a skilled, trial and error activity that improves with experience and is automated (possibly by the cerebellum). These results complement and extend our recent demonstration that paradoxical muscle movements are the norm in human standing.

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The physiological paradigm of postural maintenance has changed little for nearly a century (Massion *et al.* 2004). Sherrington and Magnus established the existence of reflex mechanisms in the spinal cord and brainstem and showed their ability in mammals to maintain standing by tonic reflexes, to adjust postural configuration by attitudinal reflexes and to restore disturbances of normal posture by righting reflexes (Sherrington, 1906; Magnus, 1925; Creed *et al.* 1932). The existence of these mechanisms in mammals generally presents an impressive argument that in humans the nervous system maintains upright balance by utilizing these low level reflex systems (Grillner & Wallen, 2004; Massion, 1998). Indeed, most contemporary analysis of human standing has proceeded on the basis that control of the centre of mass (CoM) is largely delegated to this ancient subsystem (Gurfinkel *et al.* 1974, 1995; Shadmehr & Arbib, 1992; Fitzpatrick *et al.* 1992; 1994, 1996; Horak & MacPherson, 1996; Winter *et al.* 1998; Schieppati & Nardone, 1999; Fitzpatrick, 2003) or to a correspondingly simple, reflex-like, negative feedback system with time delays of up to 200 ms (Peterka, 2000, 2002; Masani *et al.* 2003; Maurer & Peterka, 2004; Peterka & Loughlin, 2004).

More recently, an alternative hypothesis has been developed. This argument, initially proposed by Morasso *et al.* (1999), suggested that quiet standing is no different from any other form of movement, in that it requires planning, anticipation and internal models for its accomplishment just like moving a limb in a controlled manner. In standing, it is not so obvious that this is the case because the movements of the body and movements of the muscles are so small. One has a false impression that the system is static and that nothing very exciting is going on. In reality, bipedal human standing is highly precarious and somewhat different from standing in four-legged mammals. In normal stance the CoM of a four-legged mammal is almost inevitably within the base of support and there is a postural requirement to maintain adequate stiffness in the limbs. For human standing, the CoM is high, the base of support is small, and the stiffness of the

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ankle joint is low (Loram & Lakie, 2002*b*; Casadio *et al.* 2005) which together demands a more advanced system for controlling balance and which requires a longer period of learning before it is mastered. Humans are the only mammals to sustain bipedal stance as their normal posture and thus it is entirely possible that standing is controlled by those more developed facilities of the nervous system that we do not have in common with other mammals.

Morasso *et al.* (1999) originally based their argument for internal models on the low value of ankle stiffness derived from the literature of biomechanical measurements. However these measurements were obtained with large ankle perturbations and with subjects seated or holding on to a rail. More recent, ecologically sensitive measurements of the intrinsic ankle stiffness present during standing have shown that the mean intrinsic stiffness ranges from 91% (Loram & Lakie, 2002*b*) to 64% (Casadio *et al.* 2005) of the load stiffness of the human 'inverted pendulum'. This stiffness was hypothesized to lie in the series elastic component (SEC) of the calf muscles (Loram & Lakie, 2002*b*), namely the Achilles tendon, aponeuroses and also the flexible foot, which together transmit force generated from the ground to the body (Fig. 1). This means that if the calf muscles are maintained at constant activation then a person standing with feet side by side will inevitably topple forwards. The implications of this low stiffness, spring-like linkage in series with the muscles and the body were predicted by an experiment in which an inverted pendulum was manually balanced using a range of stiffness of series springs (Lakie *et al.* 2003). The muscle and the load were shown to be decoupled: they are not mechanically constrained to do the same thing at the same time. From this experiment, two main predictions emerged: (i) on average the muscle movements would be paradoxical, that is the calf muscles should move in the opposite direction to the stretch imposed by the sway of the body; and (ii) control was achieved by active, dynamic changes in muscle length that regulate the acceleration and change in position of the CoM. There were predicted to be approximately two to three of these active adjustments in muscle length for each sway of the body and these muscle movements were predicted to average 120 μm in size for a typical adult.

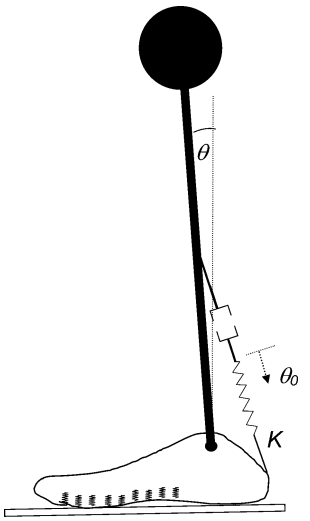

Figure 1. The dynamic bias model
The body is represented by an inverted pendulum with its centre of mass (CoM) indicated. The gastrocnemius and soleus muscles together are represented by the contractile element (CE). These muscles act through a spring-like element which connects them to the ground through the foot. The total stiffness of this elastic link is represented by K. The system operates by dynamically altering the length of the CE thus altering the position of one end of K. We refer to the length of the CE as the bias of the spring. In angular terms, the length of the spring is given by the angle of the CoM relative to the vertical (θ) minus the length of the bias (θ_0). Ankle torque is then given by $T = K(\theta - \theta_0)$. All quantities are expressed in angular terms.

The actual movements of the postural muscles during standing have only very recently been observed (Loram *et al.* 2005). We have used a novel non-invasive technique that enables us to resolve changes in muscle length as small as 10 μm and without disturbing the standing process. We used an ultrasound scanner to view the muscles and automated analysis of the images to compute the continuous changes in muscle length. We have already shown that, in accordance with a low stiffness SEC, muscle and body move on average in opposite directions (paradoxical muscle movements) both in exaggerated voluntary sways (Loram *et al.* 2004) and during the much smaller, involuntary sways of quiet standing (Loram *et al.* 2005). Here in this paper, we examine the dynamic adjustments in muscle length that are used to control acceleration and position of the CoM during postural sway. Specifically we are testing the second prediction of our inverted pendulum experiment, which is that sagittal standing is controlled by two to three, small (120 μm), active adjustments in muscle length per sway of the body. Having confirmed their existence and frequency bandwidth, we describe the way in which these active adjustments in muscle length can control standing. We then examine the source of sagittal instability in quiet stance. Having presented our results, we briefly outline the processes that might be responsible for the generation of these active muscle length adjustments. We suggest that the neural controller requires a level of flexibility and sophistication that is higher than has often been supposed.

Methods

Subjects and procedure

Ten healthy subjects, one female and nine male, aged between 25 and 50 years, stood quietly, with neither foot in front of the other and feet at a normal distance apart. Subjects were asked to stand for six trials of 40 s in

short succession in which three trials with eyes open were alternated with three trials with eyes closed. The subjects gave informed consent, and the study was approved by the local human ethics committee and conformed to the principles of the Declaration of Helsinki.

Signal measurement and recording

As reported previously (Loram *et al.* 2005), combined ankle torque from both legs was measured using a purpose-built foot plate. Surface EMG signal was recorded from the left soleus and gastrocnemius medialis, amplified (10 000 ×) and band-pass filtered at 60–500 Hz. All signals were sampled at 1000 Hz and recorded to 16-bit resolution. The EMG signals were digitally rectified and were either presented raw, or integrated ($\tau = 200$ ms) as appropriate. The position of the body CoM was calculated by filtering the combined torque signal (Loram & Lakie, 2002*b*). We also measured ankle angle using a laser range finder (YT25MGV80; Wenglor Sensoric, Germany) that was mounted on the support surface and reflected off the left shin. An ultrasound probe (Esaote Biomedica AU5 scanner, 7.5 MHz linear-array probe) was fixed along the left calf to provide a parasagittal-plane view of the underlying muscles. Images from the ultrasound scanner were digitized at 25 frames s^{-1} using a frame grabber (DT3120; Data Translation) and synchronously recorded on computer using MATLAB software. The method for tracking and calculating changes in muscle length has already been reported (Loram *et al.* 2004, 2005). From the 60 trials, two were subsequently discarded when it was realized that the ultrasound scanner had frozen during the trial.

Methods of data analysis

Coherence analysis. For each trial a coherence spectrum (Schwarzenbach & Gill, 1992) was calculated between the following pairs of signals: (i) CoM angle *versus* soleus muscle length; (ii) CoM angle *versus* gastrocnemius muscle length; (iii) CoM angle *versus* soleus integrated EMG activity; (iv) CoM angle *versus* gastrocnemius integrated EMG activity; (v) soleus integrated EMG activity *versus* gastrocnemius integrated EMG activity; and (vi) soleus muscle length *versus* gastrocnemius muscle length. Coherence at different frequencies measures the extent to which two signals x and y are phase locked and is calculated as:

$$C_{xy}(f) = \frac{|P_{xy}(f)|^2}{P_{xx}(f)P_{yy}(f)}$$

where P_{xx} is the power spectrum of x and P_{xy} is the cross power spectrum of x and y.

Calculating CoM sway and bias movements from the velocity spectrum. Standing sway is irregular and is composed of individual, relatively independent sways. A CoM sway is defined as a unidirectional movement of the CoM and a bias movement is defined as a unidirectional lengthening or shortening movement of the muscle. The mean duration of these movements was determined using frequency analysis. We have found that frequency analysis gives a more robust measure of the mean duration than the mean value of inter-reversal durations which is dominated by the more numerous, small amplitude, short duration bias movements. CoM velocity and muscle velocity were calculated from CoM angle and muscle length using a REMEZ, FIR, differentiating filter with a pass band of 12 Hz. The power spectrum of CoM velocity and muscle velocity were calculated and the mean frequency for the postural bandwidth 0–3 Hz was calculated using:

$$\overline{f} = \frac{\sum f P_{vv}}{\sum P_{vv}}$$

where f is the frequency and P_{vv} is the velocity power spectrum. This value represents the mean frequency at which the CoM or muscle velocity oscillates. During each velocity cycle there are two unidirectional CoM sways. Thus the mean CoM sway duration, T, is calculated using the formula:

$$\overline{T} = \frac{1}{2\overline{f}}$$

where f is the mean frequency of CoM velocity. Similarly, the mean bias movement duration was calculated using the same formula:

$$\overline{s} = \overline{T} \times |\overline{v}|$$

where f is the mean frequency of the muscle velocity. The mean size of sway and bias movement was calculated using:

$$\overline{s} = \overline{T} \times |\overline{v}|$$

where $|v|$ is the mean absolute velocity (speed) of the CoM or muscle length.

The dynamic bias model. The dynamic bias model, shown in Fig. 1, is used as a conceptual aid to interpreting the data. This model predicts that changes in muscle length (θ_0), CoM angle (θ) and CoM acceleration α are related by the following equation,

$$\theta_0 = (I/cmgh) \times \alpha + ((c-1)/c) \times \theta$$

where I is the moment of inertia of the inverted pendulum, mgh is the toppling torque per unit angle (load stiffness) of the inverted pendulum and c is the stiffness of the SEC relative to the load stiffness

(Loram *et al.* 2005). All quantities, including muscle length, are expressed in angular terms. The linear equivalent can be calculated by dividing by the moment arm of the Achilles tendon.

Quantifying the relative SEC stiffness. The relative stiffness, *c*, of the SEC was calculated using the normalized cross correlation between muscle length and CoM angle and using the values of load stiffness and moment of inertia determined previously (Loram *et al.* 2005). The relative SEC stiffness was calculated for each muscle, soleus and gastrocnemius, and the trial stiffness was calculated as the mean of these two values.

Statistical analysis of CoM sway and bias movements. The possible correlation between SEC stiffness and each of CoM sway duration, CoM sway size, bias duration and bias size was tested using the Pearson correlation coefficient. A one-way ANOVA was used to test for significant differences between eyes open and eyes closed conditions in the size and duration of sway and bias movements. To test for a combined effect of SEC stiffness and the eyes open/eyes closed condition on the size of bias movements, a MANOVA was used. For all tests, 58 trials from 10 subjects were available.

Time-locked averaging of micro falls and bias reversals. Because the events of significance occur irregularly during standing sway, we used time-locked averaging to identify the common features by averaging out randomly occurring changes. We systematically identified events of interest and averaged the data 1.5 s either side of those instants. We wanted to investigate the source of instability in human standing and so we focused on destabilizing rises in velocity which we call micro falls. These micro falls can be identified by a rise in velocity which reaches a maximum and which then reduces while the subject is moving forwards. So, for our averaging points, we used velocity maxima (zero acceleration) occurring when the velocity was positive. We also wanted to examine the impulsive nature of changes in bias and so we focused on reversals in muscle length. So for our averaging points we determined the instants when the muscle velocity changed from positive to negative, or from negative to positive. To prevent cases arising from noise in the velocity record, we low-pass filtered the CoM and muscle velocity with a cut off at 3 Hz and we calculated the CoM acceleration by differentiating the CoM velocity using a REMEZ, FIR differentiating filter with a pass band of 3 Hz. Having identified the averaging points, the averaging process was applied to unsmoothed data.

These instants were averaged for each trial and the six trials for each subject were averaged to produce a subject average. Finally, the pattern from each of the 10 subjects was averaged. We averaged rectified EMG rather than integrated EMG so as not to compromise the timing information in the EMG signals.

Results

The bandwidth of postural control in standing

Coherence measures the extent to which two signals maintain a constant phase relationship. A value of one means that the two signals are perfectly phase locked at that frequency, and a value of zero means that components at that frequency are initiated and terminated entirely randomly in one signal with respect to the other. Thus, an entirely random fluctuation would have a coherence value of zero with any other signal.

Figure 2 shows that during standing balance, interaction between the CoM and the calf muscles soleus and

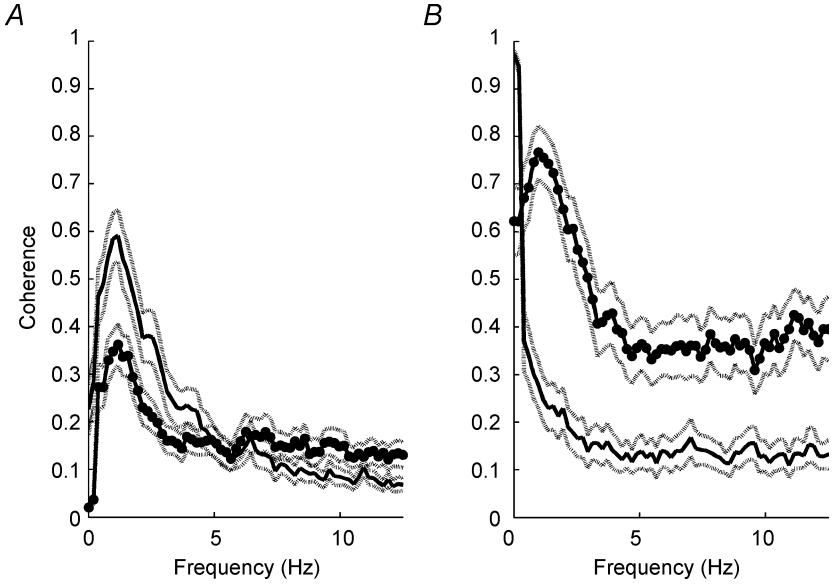

Figure 2. The bandwidth of postural control
A, shows the coherence between the CoM angle and muscle length (continuous line) and between CoM angle and EMG activity (●–●). Both soleus and gastrocnemius muscles have been averaged together for muscle length and EMG activity. *B*, shows the coherence between soleus EMG and gastrocnemius EMG activity (continuous line) and between soleus muscle length and gastrocnemius muscle length (●–●). In both *A* and *B* the lines represent the combined average of 10 subjects. The dashed lines represent 95% confidence intervals in the mean values. Muscle length was sampled at 25 Hz and thus the frequency range shown is 0–12.5 Hz. A coherence of one means that the two signals are perfectly phase locked at that frequency. A coherence of zero means that at that frequency, sinusoids in one signal are initiated and terminated entirely randomly with respect to the other signal.

gastrocnemius occurs in the frequency bandwidth 0–3 Hz. Figure 2*A* shows the coherence between muscle length and the CoM angle, and between EMG signal and the CoM angle. The Figure shows two distinct regions. Above 3 Hz there is a region which shows approximately constant low coherence. This is due to the presence of some coherent noise in both signals and it sets the noise floor. Below 3 Hz, the coherences are outside the 95% confidence intervals of the noise floor. However, even in the region of 0–3 Hz, the coherences are relatively low. This indicates that while not entirely random and purposeless, the changes in muscle length and EMG activity are irregular and only partly synchronized with changes in CoM angle. Muscle length is generally more coherent with CoM angle than is EMG activity. This reflects the mechanical coupling between muscle length and body angle via the Achilles tendon and the noisier nature of the EMG signal.

Figure 2*B* shows the coherence between soleus and gastrocnemius for muscle length and for EMG signal. Again there are two distinct regions. Above 3 Hz, there is a region of approximately constant moderate coherence. These two muscles are mechanically coupled by the Achilles tendon at their distal end, and so noise like fluctuations in one muscle are inevitably recorded in the other muscle. This means that the background coherence (noise floor) is quite high. The two muscles have a coherence that is greater than the 95% confidence limits of the noise floor in the range 0–3Hz (Fig. 2*A*) and shorten and lengthen most coherently (0.77) at a frequency of 1.0 Hz. It is interesting that the integrated EMG activities of these muscles are highly coherent (0.97) at tonic levels (0–0.2 Hz), but remarkably incoherent at higher frequencies. Thus the two muscles are activated independently above tonic frequencies and the synchronous changes in muscle length results almost entirely from the mechanical coupling of the muscles. In other words an external stretch of one muscle or an active contraction of one muscle has a synchronous effect on the other muscle. At around 1 Hz, the two muscles effectively function as a single unit.

Muscle movements and body sways

Frequency analysis was used to examine the overall pattern of body sway and muscle length alteration. Figure 3*A* shows that alterations in muscle length are considerably more frequent than alterations in CoM angle. Muscle lengthening or shortening reverses direction with a peak frequency of 1 Hz and a mean frequency of 1.3 Hz whereas body sway reverses direction with a peak frequency of 0.25 Hz and mean frequency of 0.45 Hz (Fig. 3*A*). These values indicate that reversals in muscle shortening/lengthening are on average 2.8 times more frequent than reversals in CoM sway.

The SEC is the linkage through which changes in muscle length and tension exert their effect on the body. We examined the influence of SEC stiffness on body sway and muscle movement (Fig. 3*B*–*E*). Unidirectional sways of the body are generally a few tenths of a degree (Fig. 3*B*). This magnitude is not affected by the value of SEC stiffness (Pearson correlation coefficient, $n = 58$, $P = 0.27$), though the sways were larger with the eyes closed condition (mean, 0.23 deg) compared with the eyes open condition (mean, 0.13 deg) (ANOVA, $n = 58$, $P = 0.014$). Unidirectional changes in muscle length range from mean values of 30 μm to 300 μm (Fig. 3*C*). There is clear, dramatic increase in the size of muscle movement as the stiffness of the SEC decreases (Pearson correlation coefficient, $n = 58$, $P = 0.00006$). In Fig. 3*C* it looks as though there may be a combined effect of SEC stiffness and size of muscle movement separating the eyes open from the eyes closed cases; however, this distinction is not justified statistically (MANOVA, $n = 58$, $P = 0.07$). The duration of body sways is generally around 1–1.5 s with no effect from SEC stiffness (Pearson correlation coefficient, $n = 58$, $P = 0.3$) or whether the eyes are open or closed (ANOVA, $n = 58$, $P = 0.6$; Fig. 3*D*). The duration of muscle shortening/lengthening is 0.41 ± 0.06 s (mean $\pm$ S.D) (Fig. 3*E*). Unlike the variation in the size of muscle movements with SEC stiffness, there is no such variation in the duration of muscle movements either resulting from SEC stiffness (Pearson correlation coefficient, $n = 58$, $P = 0.7$) or from eye closure (ANOVA, $n = 58$, $P = 0.7$). This result raises the interesting question of what limits the control bandwidth of these postural muscles.

Illustration of key results using a representative subject

In order to determine the effect and possible purpose of these changes in muscle length it is necessary to examine the irregular sway pattern and changes of muscle length in the time domain.

Figure 4 shows data from a representative subject standing with eyes open. This Figure shows all of the key points that will be made more clearly by the time-locked averaging analysis that follows. The CoM angle varies through several tenths of a degree over 20 s and irregularly reverses direction (Fig. 4*A*). Within this sway pattern speed is regulated to remain less than 0.3 deg s^{-1} (Fig. 4*B*) and acceleration less than $\sim$1 deg s^{-2} (Fig. 4*C*). Muscle length is constantly changing (Fig. 4*D*) with a peak amplitude of several hundred micrometres with both muscles, soleus and gastrocnemius, following a similar pattern. The changes in muscle length show clear similarities with the CoM acceleration (Fig. 4*C*). As acceleration is a measure of the lack of balance between ankle torque and the torque generated on the body by gravity (Loram &

Lakie, 2002*a*), this indicates that changes in muscle length are associated with changes in balance.

The alternations in muscle length are at a different, higher, frequency than the sway frequency. The low frequency drift in muscle length is paradoxical; that is, muscle length increases as the CoM angle decreases. These paradoxical changes can be seen most clearly over the first 8 s. For this subject, the muscle length appears to fluctuate with some regularity and it is the size more than the timing of muscle movements that is modulated.

In Fig. 4, the asterisks show all the falling cases of a destabilizing rise in velocity, i.e. where the velocity of the CoM rises to a maximum while the person is swaying forwards. These asterisks mark the averaging

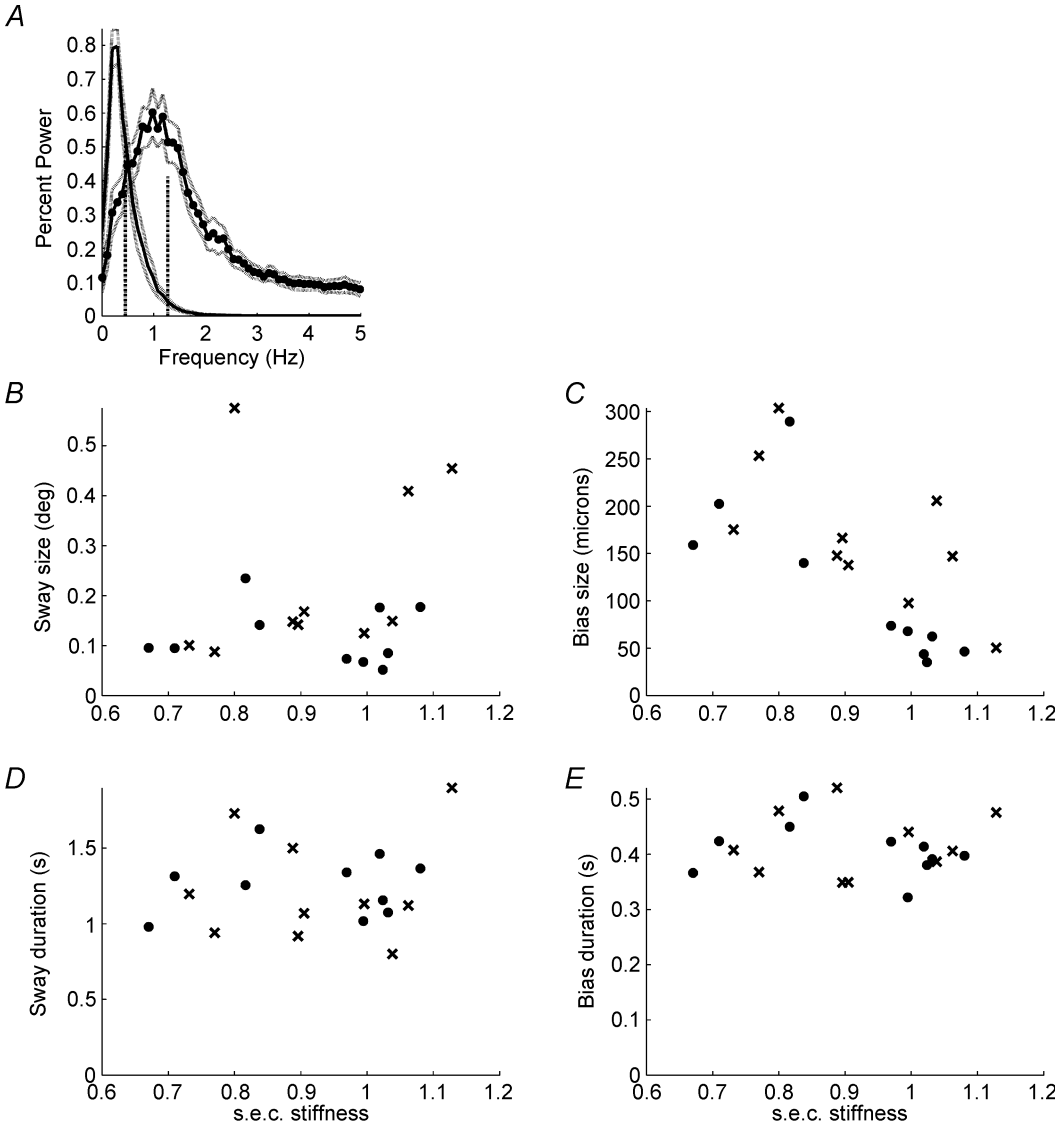

Figure 3. CoM sway and bias movements
A, shows the velocity power spectrum of CoM angle (continuous line) and muscle length (●–●). The power spectra are calculated relative to their maximal values. Muscle length is the average of soleus and gastrocnemius which were very similar across the range shown. The lines represent the combined average of 10 subjects. The dashed lines represent 95% confidence intervals in the mean values. For all 10 subjects, mean values plotted against SEC stiffness are shown for sway size (*B*), bias movement size (*C*), sway duration (*D*) and bias movement duration (*E*). The filled circles are a mean of the three eyes open trials and the crosses are a mean of three eyes closed trials. A sway is defined as a unidirectional movement of the CoM. A bias movement is defined as a unidirectional change in muscle length. Relative stiffness is defined as the stiffness of the SEC divided by the load stiffness of the human inverted pendulum and has been calculated by the cross correlation between CoM angle and muscle length, averaged for soleus and gastrocnemius muscles (Loram *et al.* 2005). The mean range and standard deviation of the relative stiffness for an individual subject are 0.35 and 0.14, respectively.

points for micro falls and they amount to an instantaneous attainment of equilibrium (zero velocity gradients). Here, using the representative subject, we preview the averaged information that we will subsequently show in Fig. 7. The destabilizing rise in velocity before the asterisk is usually closely preceded by an increase in muscle length and the muscles usually shorten immediately before balance is attained and the speed of the micro fall reduces. Thus the shortening of calf muscles is associated with the regulation of speed and balance at the position of the asterisk. As a result of this muscle shortening, usually the direction of sway reverses (e.g. $t = 11$ s) and sometimes (e.g. $t = 8.5$, 9 and 13.5 s) it does not. For this subject, whole body sagittal sway is mechanically both caused and regulated by the soleus and gastrocnemius muscles.

In the next analysis (Figs 5 and 6), reversals in muscle length are used as averaging points. In this Fig. 4, these points would be located at all the local maxima and minima of the muscle length records in Fig. 4*D*.

Time locked averaging of transient changes in muscle length

For individual subjects, Fig. 5 shows the impulsive effect (Fig. 5*A*) that is associated with muscle shortening (Fig. 5*B* and *C*). Time-locked averaging of all minima in muscle

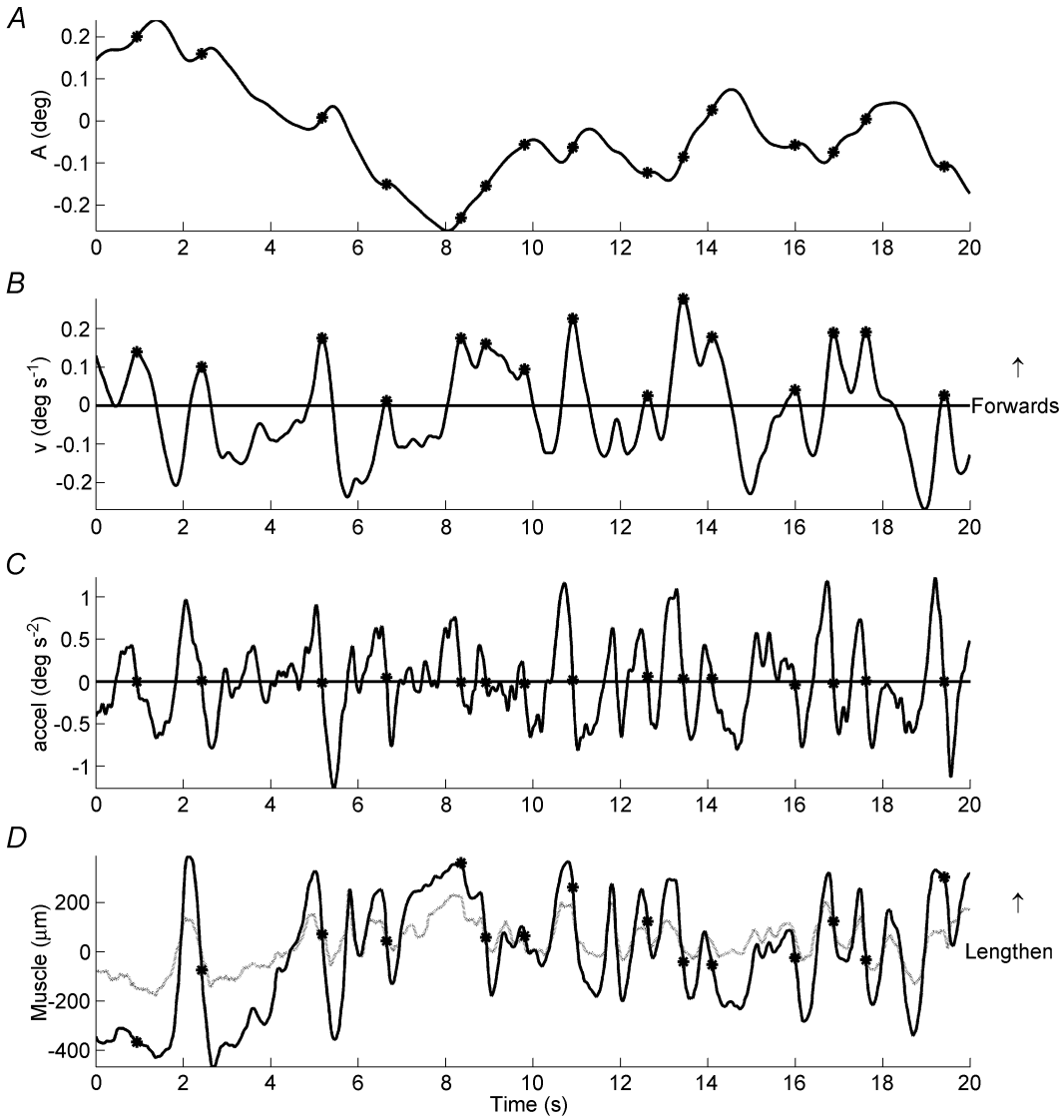

Figure 4. Locating micro falls and bias reversals
Time records are shown for a representative subject of CoM angle (*A*), CoM velocity (*B*), acceleration (*C*) and soleus (dashed line) and gastrocnemius (continuous line) muscle length (*D*). The asterisks identify micro falls when the CoM speed rises to a maximum value while the subject is swaying forwards. All values are shown relative to a mean of zero. Positive angle, velocity and acceleration are forwards, away from the vertical. Positive changes in muscle length indicate lengthening. All quantities are expressed relative to their mean value.

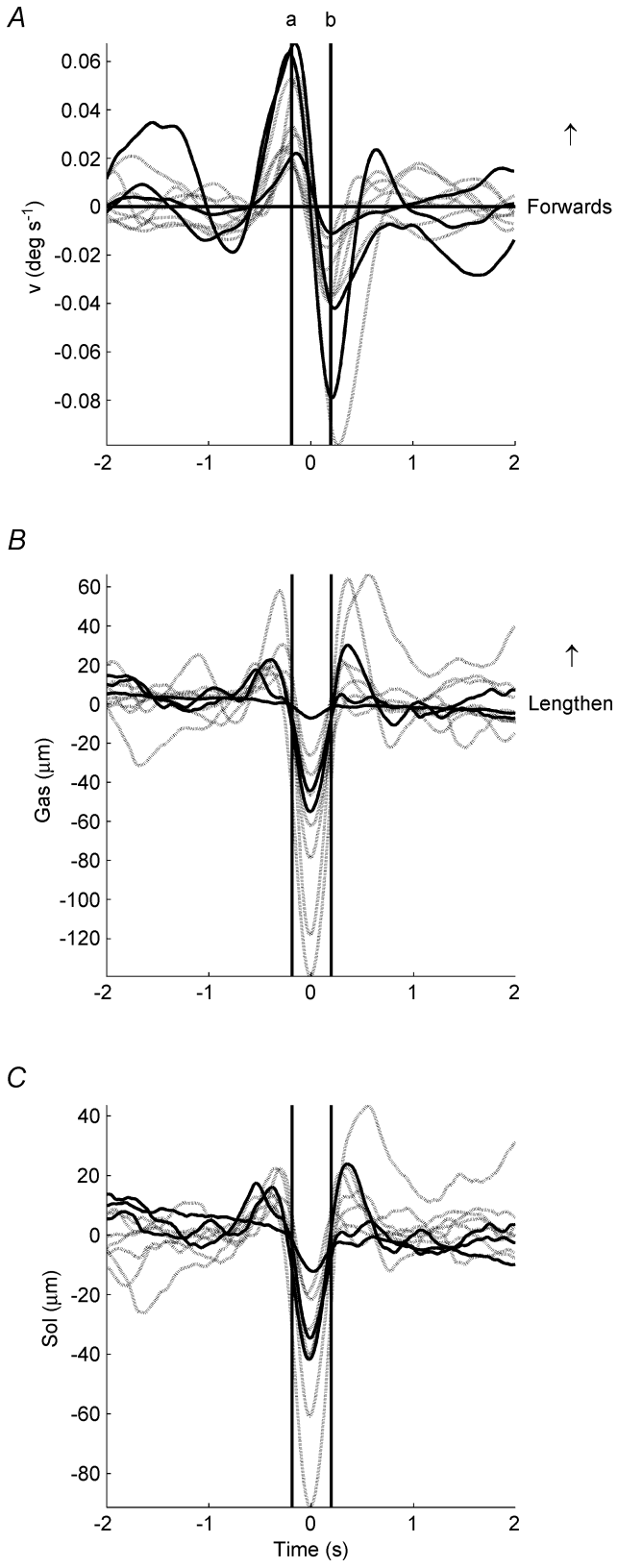

Figure 5. Averaged bias reversals – variation with subject
For each subject, all cases of bias reversals from six trials have been averaged. An average $n = 297$ events per subject for six trials. Time zero represents the averaging point which is the local minimum in muscle length. *A*, shows CoM velocity. *B* and *C*, show gastrocnemius and soleus muscle length, respectively. Velocity, angle and muscle length all increase positively. The marker lines, a and b, indicate the beginning and end of the impulse. The continuous lines indicate subjects with a SEC stiffness greater than 100%. All quantities are expressed relative to their mean value.

length (Fig. 5*B* and *C*) shows that the mean transient shortening in gastrocnemius muscle length varies in size from 20 μm to 130 μm for the different subjects. The changes in length are smaller for soleus. These transient shortenings are associated with a change in CoM velocity (peak to peak, a–b in Fig. 5) of 0.02–0.15 deg s^{-1}. An impulse is defined as a change in momentum which in this context is equivalent to a change in velocity of the CoM. Measured from a to b, the duration of these impulses is 383 ± 55 ms (subject mean ± s.d.). Thus, amongst all 10 subjects, there is little variation in the duration of their muscle shortenings (Fig. 5*B* and *C*), and the duration of the associated impulsive effect (Fig. 5*A*).

The symmetrical impulsive effect of muscle shortenings (Fig. 6*A*–*D*) and muscle lengthenings (Fig. 6*E*–*H*) is shown in Fig. 6. More importantly, Fig. 6 reveals how the transient changes in soleus and gastrocnemius EMG activity and muscle length are related to the velocity of the CoM (Fig. 6*A*). Using cross correlation between averaged EMG signal (Fig. 6*C*, *D*, *G* and *H*) and muscle length (Fig. 6*B* and *F*), changes in rectified EMG signal precede changes in muscle length by 167 ms and 203 ms for soleus and gastrocnemius, respectively. What stimulus causes the changes in the EMG? The changes in rectified EMG signal show a very similar pattern to changes in CoM velocity (Fig. 6*A*). Could changes in the EMG derive from a simple feedback reflection of the velocity signal as recently proposed (Masani *et al.* 2003)? Cross correlation between averaged EMG signal (Fig. 6*C*, *D*, *G* and *H*) and averaged CoM velocity (Fig. 6*A* and *E*) show that soleus and gastrocnemius EMG activity leads CoM velocity by 58 ms and 28 ms, respectively. However, the duration of this lead of EMG activity over CoM velocity is not constant. Moving along the time axis through Fig. 6, EMG activity becomes progressively further in advance of CoM velocity. Before a, the EMG marginally lags CoM velocity; at a, EMG and velocity are broadly simultaneous; and at *b* EMG is noticeably ahead of velocity. The completion of each impulse is anticipatory of changes in velocity. This result occurs symmetrically for shortening (Fig. 6*A*–*D*) and lengthening (Fig. 6*E*–*H*) impulses.

Time-locked averaging of micro falls

As we shall show (Fig. 7) as an averaged result for all subjects, micro falls, destabilizing increases in speed while swaying forwards (* in Fig. 4), are associated

with closely preceding increases in calf muscle length. Regulation of the micro fall, reduction in speed, results from reacto-predictive modulation of calf muscle activity. The inter-relationship between ankle torque, CoM angle, muscle length and SEC stiffness is excellently explained by the dynamic bias model.

In detail, the 'timetable' of an averaged micro fall is as follows (Fig. 7). The line a represents the start of the micro fall and it can be seen that preceding changes in all quantities average around zero. At a ($t = -0.70$ s), the gastrocnemius activity decreases (Fig. 7*H*), muscle length increases by 30 and 50 μm for soleus and gastrocnemius, respectively (Fig. 7*E*), ankle torque decreases below that required for balance (Fig. 7*A*) and the CoM velocity increases as the CoM accelerates forwards (Fig. 7*C* and *B*, respectively). Using cross correlation between the two traces, increases in ankle angle lag increases in CoM angle by 35 ms overall (Fig. 7*D*). This shows that the CoM falls forwards before movement occurs at the ankle joint and so toppling occurs at the knee and/or hip before the ankle joint. At *b* ($t = -0.33$ s), 370 ms after a, soleus (*G*) and gastrocnemius (*H*) activity increases. At *c* ($t = -0.23$ s), 107 ms later still soleus and gastrocnemius begin to shorten (by 80 μm and 130 μm, respectively) and ankle torque begins to rise. At the asterisks ($t = 0$ s), CoM velocity reaches a maximum and the velocity cancelling impulse begins. At *d* ($t = 0.23$ s), soleus and gastrocnemius start to lengthen, and ankle torque begins to fall. At *e* ($t = 0.51$ s), the velocity of the CoM is reduced to zero. At *f* ($t = 0.77$), balance is restored, acceleration reaches zero and the velocity cancelling impulse ends. (An averaged plot of rising, destabilizing increases in speed shows the same pattern of events though with reversed polarity).

The changes in muscle length (Fig. 7*E*) are predicted well by the dynamic bias model (Fig. 7*F*). Using a moment arm of 5 cm and a best fit relative stiffness of 89% and 76% for soleus and gastrocnemius, respectively, the changes in muscle length can be predicted with a variance accounted for (VAF) of 91.3% and 99.1%. To produce a VAF of more than 70%, the best fit relative stiffness could vary by $\pm$ 14% and $\pm$ 23% for soleus and gastrocnemius, respectively.

Contemporaneous changes in soleus and gastrocnemius muscle activity (Fig. 7*G* and *H*), length (Fig. 7*E*) and tension (Fig. 7*A*) (a to b) are associated with loss of balance in quiet standing. The calf muscles that control balance contribute wholly or partially to the loss of balance. When a destabilizing fall occurs the corrective reaction begins (b) after a delay. This associated effect of this active reaction (b–e) is to reverse the acceleration, limit the increase in CoM velocity and reduce the CoM velocity to close to zero (e). The shift in CoM angle is limited to about 0.1 deg. The amount of muscle shortening and the timing of the transition between shortening and subsequent lengthening determines whether the CoM speed is reduced and the CoM caries on falling, or whether the direction of the CoM is reversed. The velocity cancellation is almost perfect, though the average result is to reverse the direction of the CoM (Fig. 7*C* and *D*).

The irregularity of the balance process is shown by the fact that this forward micro fall is not associated with a

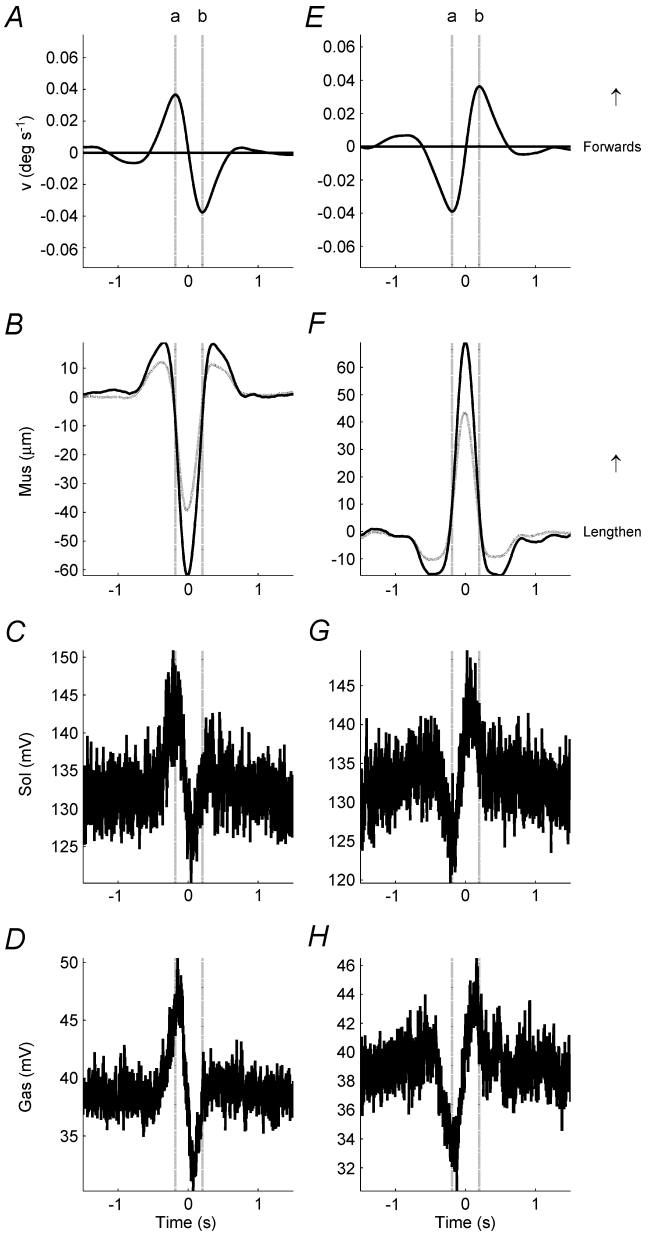

Figure 6. Averaged bias reversals
For each subject, all cases of bias reversals from six trials have been averaged. The time-locked patterns from all 10 subjects have been averaged to produce this Figure which incorporates 2870 and 2867 events for the minima and maxima, respectively. *A–D*, show muscle length minima. *E–H*, show muscle maxima. Time zero represents the averaging point. Velocity and muscle length increase positively and are expressed relative to their mean value. *A*, CoM velocity; *B*, gastrocnemius (continuous line) and soleus (dashed line) muscle length; *C*, soleus, rectified EMG signal; *D*, gastrocnemius, rectified EMG signal. *E–H*, as in *A* to *D*. The marker lines a and b indicate the beginning and end of the impulse.

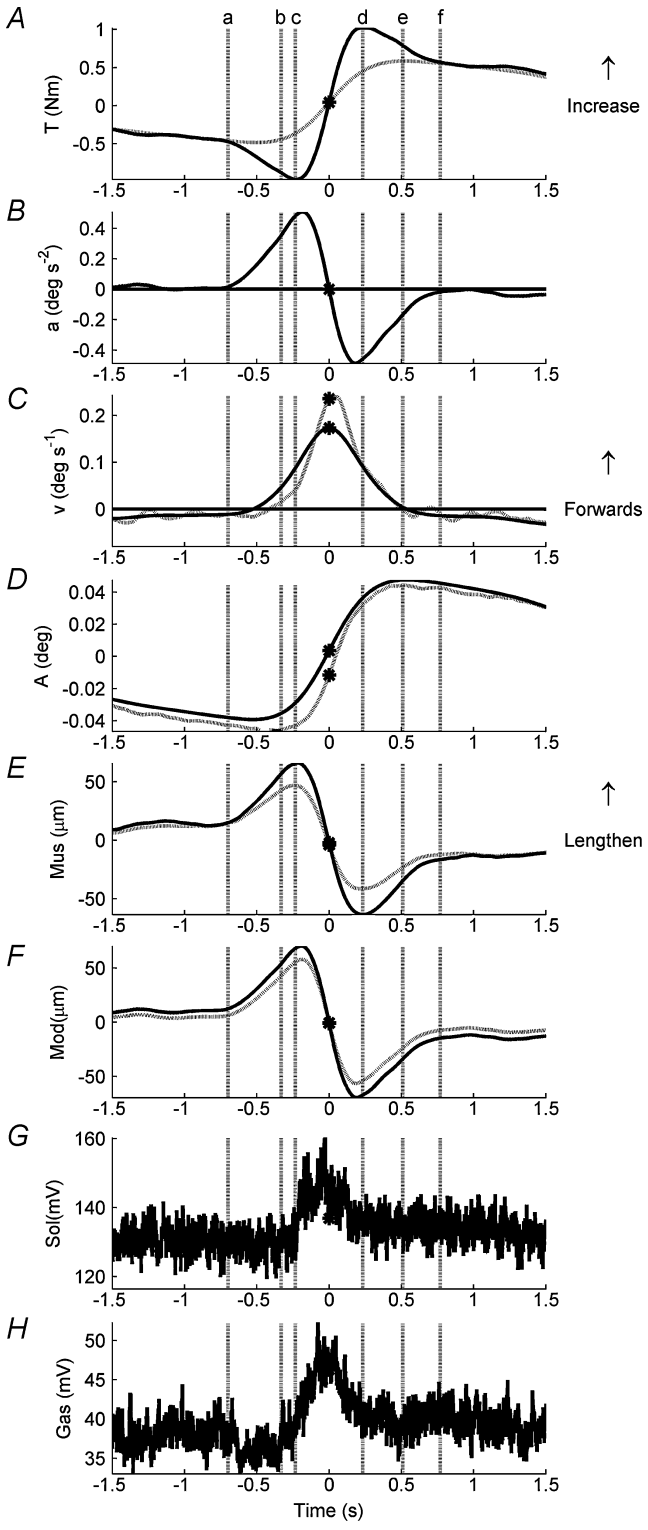

Figure 7. Averaged micro falls
For each subject, all cases of micro falls from six trials have been averaged. The time-locked pattern from all 10 subjects have been averaged to produce this Figure which incorporates 1541 events into the average. A micro fall is a rise in speed of the CoM to a maximum while the subject is swaying forwards. Time zero represents the averaging point which is a velocity maximum while falling forwards. Positive changes in torque represent increases. Positive angle and velocity are forwards from the vertical. Positive changes in muscle length indicate lengthening. All quantities except EMG activity are expressed relative to their mean value. *A*, ankle torque from both legs (continuous line) and ankle torque required to balance the CoM (dashed line); *B*, CoM acceleration; *C*, CoM velocity (dashed line) and ankle joint velocity (continuous line); *D*, CoM angle (dashed line) and ankle angle (continuous line); *E*, gastrocnemius (continuous line) and soleus (dashed line) muscle length; *F*, muscle length predicted by the dynamic bias model, gastrocnemius (continuous line), soleus (dashed line); *G*, soleus, rectified EMG signal; *H*, gastrocnemius, rectified EMG signal. The marker lines indicate the preceding loss of balance (a), the initial increase in EMG activity (b), the consequent decrease in muscle length (c), the subsequent increase in muscle length (d), the cancellation of CoM velocity (e) and the attainment of zero acceleration (f).

previous forward micro fall (Fig. 7*B*–*D*). Because they occur at random instants, preceding CoM movement averages zero. This lack of regularity in motion of the CoM reduces the timescale over which prediction of a micro fall can occur. This raises the question of what information stimulates the rise in muscle activity at b. By inspection one can see that the activity of soleus and gastrocnemius does not copy the pattern of any one signal, e.g. muscle length, ankle torque, CoM velocity, ankle velocity, CoM angle or ankle angle. The pattern of rectified EMG signal is closest to velocity. Overall, using cross correlation, soleus and gastrocnemius rectified EMG activity (Fig. 7*G* and *H*) precedes ankle velocity by 53 ms and 38 ms, respectively, and lags CoM velocity by 4 ms and 17 ms, respectively. Between the asterisks ($t = 0$) and e, the EMG activity is modulated in advance of CoM velocity which demonstrates the predictive element to the velocity cancelling impulsive change in muscle length (asterisk to f). Between a and b, gastrocnemius EMG activity decreases which does not reflect the velocity signal. The earliest information concerning the destabilizing rise in velocity comes from CoM acceleration and the simultaneous changes in muscle length associated with that acceleration. In summary, the pattern of EMG activity is initially reactive to the loss of balance, and is subsequently predictive of the damping impulse.

In this process of regulating balance and velocity, muscle length is actively controlled in a counter spring-like manner, i.e. muscle length decreases as muscle tension increases (Fig. 8). This behaviour is the complete opposite of the spring-like behaviour that has traditionally been assumed and it can only be achieved by neural modulation of muscle length. This Figure illustrates how changes in muscle length are equivalent to changes in ankle torque.

The averaged changes in muscle length associated with a destabilizing rise in velocity were shown by all 10 subjects (Fig. 9). The subjects with stiffest SEC showed smaller changes in muscle length and larger destabilizing rises in velocity. For one subject (SEC stiffness > 1), there was little

or no velocity damping modulation in gastrocnemius and all the velocity damping behaviour occurred in the soleus muscle. The result was also shown in the raw data for this subject as well as the averaged results (Fig. 9*B*–*C*).

Discussion

In this study we have observed quiet standing as a closed loop process.

Changes in EMG activity will produce alterations in muscle length which will produce movement of the body. The motion of the body will also stimulate modulation of the EMG activity. The advantage of this approach is that we observe standing as it is, without interference; so it allows the physiological sequencing of events to be precisely determined. The limitation is that our conclusions regarding cause and effect need to be compared with further, more artificial, experiments which attempt to study each causal pathway in isolation.

Human standing requires active, adjustments in muscle length

We have used a new technique based on ultrasound imaging to provide measurements of changes in length of the calf muscles soleus and gastrocnemius during quiet standing. Before this study (Loram *et al.* 2005), these tiny changes in length have never been observed. We have found that each irregular, unidirectional sway of the CoM is accompanied by on average 2.8 purposive, unidirectional, adjustments in the length (bias) of soleus and gastrocnemius. The size of these active adjustments depends on the stiffness of the SEC of the calf muscles, varying from an average of 30 μm for stiffer subjects (relative stiffness, 1–1.1) to 300 μm for less stiff subjects (relative stiffness, 0.7–0.85). The size and duration of body sways was unrelated to the SEC of the subject. The results presented here confirm the prediction derived from our previous work in which subjects manually controlled a real inverted pendulum using a low-stiffness spring (Lakie *et al.* 2003). We predicted an average of two to three unidirectional adjustments in muscle length for each unidirectional sway of the CoM with a mean adjustment size of 120 μm for a person of average SEC stiffness (90%).

These findings confirm recent theories (Morasso *et al.* 1999; Morasso & Schieppati, 1999; Morasso & Sanguineti, 2002) and are consistent with recent evidence (Loram & Lakie, 2002*a*,*b*; Lakie *et al.* 2003; Loram *et al.* 2004; Casadio *et al.* 2005) that humans cannot maintain bipedal stability in the sagittal plane through unchanging muscular activity in the calf muscles. The SEC of the calf muscles has a stiffness less than the load stiffness of the human inverted pendulum (Loram & Lakie, 2002*a*,*b*). Thus, without proactive control of the calf muscles (Lakie *et al.* 2003), the person would inevitably fall forwards until they have to take a step.

What limits the bandwidth of postural control?

We found that on average the direction of muscle movement was reversed 2.6 times per second which corresponds to a mean frequency of 1.3 Hz (Fig. 3). Unlike the size of muscle movements, the mean duration of muscle movements was unaffected by the stiffness of the SEC and it was also unaffected by whether the eyes were open or closed. Correspondingly, the mean duration of impulsive muscle shortenings (or lengthenings) was 383 $\pm$ 55 ms (subject mean $\pm$ S.D.) (Fig. 5). The frequency profile of muscle velocity (Fig. 3) was consistent with the bandwidth of postural control by the calf muscles of 0–3 Hz established from coherence data (Fig. 2) and is also consistent with the postural bandwidth established by Fitzpatrick *et al.* (1992). This control bandwidth of 0–3 Hz is not restricted to postural control in standing. It is replicated in tracking experiments where spectral analyses of coherent tracking response signals show negligible power above 2–4 Hz (Neilson *et al.* 1988*a*).

The relative invariance of bias durations poses an interesting question. What limits the bandwidth of postural control? Is it the maximal rate at which the muscle length can be alternated? Is it neural factors such as the rate at which the nervous system can plan and initiate actions?

When the foot and ankle joint are rhythmically oscillated by an external input, the calf muscles can demonstrate reflex modulated alterations in length, considerably out

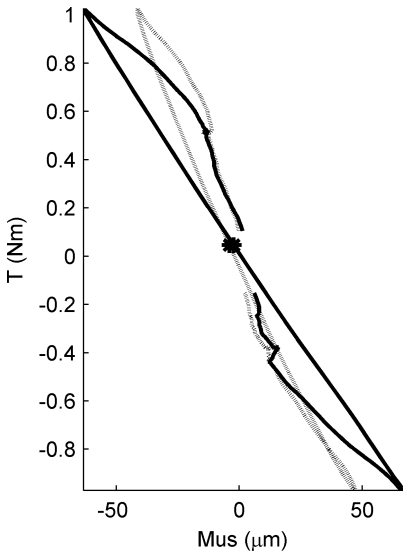

Figure 8. Averaged micro falls show paradoxical length changes
This Figure shows the same averaged micro falls data as Fig. 7. Averaged ankle torque has been plotted against averaged muscle length. The asterisk shows the averaging point. Torque and muscle length both increase positively.

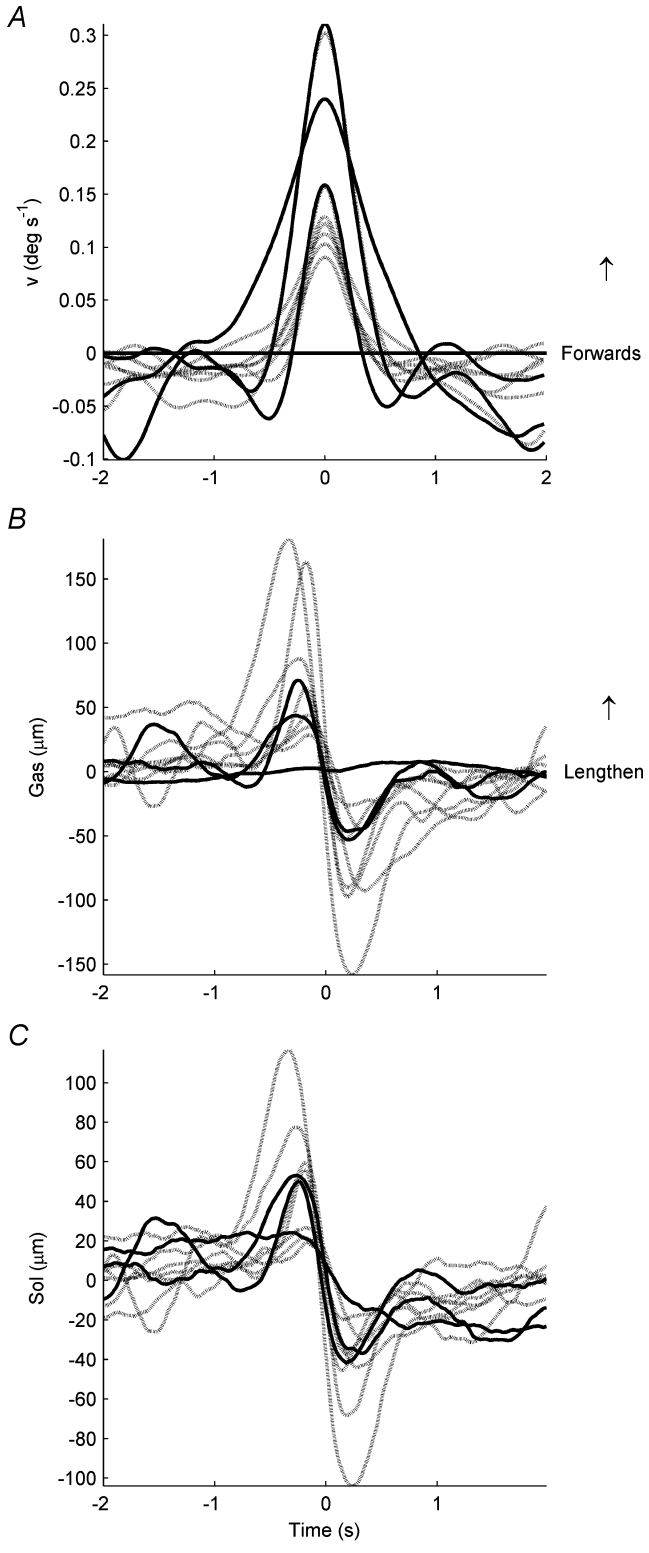

Figure 9. Averaged micro falls - variation with subject
For each subject, all cases of micro falls from six trials have been averaged. An average $n = 159$ events per subject for six trials. A micro fall is a rise in speed of the CoM to a maximum while the subject is swaying forwards. Time zero represents the averaging point which is a velocity maximum while falling forwards. *A*, shows CoM velocity. *B* and *C*, show gastrocnemius and soleus muscle length, respectively. Velocity and muscle length increase positively and are expressed relative to their mean value. The continuous lines indicate subjects with a SEC stiffness greater than 100%.

of phase with the mechanical input, at up to 8 Hz or more (Rack *et al.* 1983; Evans *et al.* 1983). Thus the nervous system is capable of modulating soleus and gastrocnemius muscle length at considerably higher frequencies than the mean and range (1.3 Hz, 0–3 Hz) that are observed under postural conditions. This indicates that a longer duration of neural processing is associated with each muscle length adjustment that we observe. A mean frequency of 1.3 Hz, indicates a mean time period of ∼800 ms which indicates a mean processing time per unidirectional muscle movement of 400 ms (Fig. 3). This duration is longer than the reflex initiated response time (65 ms for a monosynaptic reflex; Evans *et al.* 1983), and is comparable to a human reaction time requiring some choice and predictive planning (Craik, 1947; Vince, 1948).

The finding of modulated responses occurring every 400 ms resonates with results from investigations into visually guided pursuit tracking of a continuously moving target. It has been demonstrated that the human operator behaves as an 'intermittent correction servo' by making ballistic movements at a mean interval of approximately 500 ms (Craik, 1947). The intermittency results from a psychological refractory period, equal in duration to the reaction time, during which the operator is unable to respond to a second stimulus, similar to the first (Vince, 1948). In continuous tracking, inverse internal models are employed to transform desired trajectories into motor commands. There is evidence that the nervous system requires a finite time period for planning and it does not commence planning a new movement until planning of the old movement has been completed (Neilson *et al.* 1988*a*,*b*). We hypothesize that standing balance is a process of this type. The relatively long duration of 400 ms allows plenty of opportunity for predictive, planning mechanisms to shape the amplitude and timing of bias adjustments.

At low frequencies, this process resembles a sampled, negative feedback process (Neilson *et al.* 1988*a*). This would explain why simple negative feedback circuits can characterize the mean parameters of standing sway well (Maurer & Peterka, 2004) even if the instantaneous balancing process consists of intermittent, ballistically executed movements. After all, position is regulated in quiet standing, and so is velocity, so is it not inevitable that this process can be represented by a proportional differential (PD) controller (Peterka, 2000; 2002; Loram *et al.* 2001; Masani *et al.* 2003; Peterka & Loughlin, 2004; Maurer & Peterka, 2004)?

The correspondence between muscle length and ankle torque predicted by the dynamic bias model

Standing is usually studied using a force plate and the centre of pressure (CoP), the point of application of the ground reaction force, is usually recorded. Fluctuations in CoP forwards and backwards are entirely equivalent to fluctuations in ankle torque. By applying the dynamic bias model (Fig. 1) to the averaged micro falls (Fig. 7*F*) we have shown that these averaged, short duration changes in muscle length, ankle torque and CoM angle are very well explained by this model using a mean SEC stiffness of 83% relative to the load stiffness of the subject. This simple model aids interpretation, in that short duration muscle movements are equivalent to simultaneous torque fluctuations via the spring-like (Loram *et al.* 2005) SEC of the calf muscles. In individual cases there are other factors that influence muscle length, such as ankle co-contraction, varying muscle modulation between the two legs, tendon creep and tendon hysteresis. These other factors mean that at any one time, muscle length does not correspond as well to acceleration and CoM angle as is shown here in these averaged plots. But on average these other factors cancel out.

A unidirectional muscle movement is equivalent to a change in torque. Does this mean that the nervous system is modulating muscle length rather than ankle torque in its control of balance? We don't know. We also don't know whether there is any real distinction between the two ideas. But we can see that there is a difference between internally and externally generated changes in ankle torque. While a change in muscle length is equivalent to a change in ankle torque according to the stiffness of the SEC (mainly the Achilles tendon), an externally applied change in ankle torque does not necessarily produce the same change in muscle length. The change in muscle length produced by an external change in torque depends on the muscle stiffness which may not be at all the same thing as the SEC stiffness. Thus by examining changes in muscle length, we gain insight into the working of the controlling actuator, that is the internally generated changes in torque that are controlled by the nervous system.

Because the SEC is not stiff, muscle length is not mechanically constrained to follow ankle angle. And, due to the complexity of the nervous system, changes in muscle length can be driven by stimuli other than CoM movement and its linear, time invariant derivatives. Thus muscle movements can be generated independently of CoM motions. The dynamic bias model predicts that muscle movements cause changes in CoM acceleration. The muscle may alternate between shortening and lengthening while the inertia of the CoM means that CoM motion need not alternate between rising and falling. Thus there is no mechanical or control objection to the muscle velocity alternating independently of, and more frequently than, CoM velocity.

The ballistic bias impulse mechanism

The integral of the torque change through the duration of the adjustment produces an impulse, effectively a change in velocity, given to the CoM. It is appropriate to think in impulse terms because the effect of the bias change is given in a short timescale relative to the motion of the CoM. As there are on average 2.8 bias adjustments for each CoM sway, the velocity change is delivered in approximately one-third of a unidirectional CoM sway. The changes in muscle activation are delivered in a shorter time still (Fig. 6). As the impulsive effect is discharged by the nervous system in a short timescale relative to the effect on CoM position, and before feedback of the result can be received, this process is properly described as ballistic. For example, after a bias action, the nervous system will not know, and will have to wait to find out whether or not the direction of CoM motion will be reversed. Small differences in impulse will result in completely different motion sequences for the CoM. Instants when the CoM is finely balanced and moving at low speed are effectively bifurcation points where alternative small changes in ankle torque could result in opposite motions of the CoM (Loram & Lakie, 2002*a*). These bifurcation points create unpredictability in the motion of the CoM. The delay between the initiation of a destabilizing rise in velocity and the corrective reaction (Fig. 7) is evidence that these micro falls are not perfectly predicted. The summated effect of these ballistic bias, impulse actions is regulation of position and velocity. This interpretation is consistent with previous evidence of the ballistic nature of balance derived from pedal balancing of a real inverted pendulum (Loram & Lakie, 2002*a*).

It has been previously hypothesized that postural control of standing operates in an open loop mode over durations less than approximately 1 s, and in a closed loop mode over durations more than 1 s (Collins & DeLuca, 1993, 1995). This hypothesis was based on the observation that the direction of motion of the CoM correlates positively with itself for durations up to 1 s (persistent motion) and correlates negatively with itself (antipersistent motion) for longer durations. The CoM sway durations that we observe (Fig. 3*D*) are consistent with those observed by Collins and DeLuca and our ideas of impulsive, ballistic control are sympathetic to their idea of open loop/closed loop control. Our observations of muscle length show that over a sway timescale of 1 s, there are several ballistic-like attempts to control CoM motion. The existence of bifurcation points which are extremely sensitive to small changes in ankle torque, the unstable nature of the human 'inverted pendulum' (Loram & Lakie, 2002*a*) and the time taken to respond to

unpredicted losses of balance (a to e in Fig. 7) all account for the 1 s timescale of persistent motion.

The physiological origin of postural sway

There has been some debate whether standing sway results from internal perturbations such as breathing or the heart beat (Sturm, 1980; Conforto *et al.* 2001; Hodges *et al.* 2002; Gandevia *et al.* 2002), whether it results from 'noise' from some unattributed source (Winter *et al.* 1998; Peterka, 2000; Masani *et al.* 2003) or whether it results from inaccuracies in the modulation of calf muscle activity (Loram *et al.* 2001; Loram & Lakie, 2002*a*). For the 10 subjects studied, a very clear answer can be given. On average, the CoM sway is very closely related to fluctuations in muscle length and ankle torque via the dynamic bias model (Figs 1 and 7). This indicates that modulation of the ankle musculature largely explains (91–99% VAF, Fig. 7*F*) control of the CoM. CoM sways and corresponding acceleratory ankle torques are clearly related to simultaneous fluctuations in calf muscle length. The 370-ms delay between the onset of the loss of balance, and the start of the corrective reaction indicates that on average, the destabilization represents a deviation from what was predicted. Because the system is unstable, the predictive error in torque provides a perturbation which grows into a sway which is reacted to and corrected. The correction is itself imperfect and thus the source of subsequent sway. This process is consistent with the idea that sensory, computational and motor noise place limits on the ability of the subject to produce a perfect torque response (Jeka *et al.* 2004).

Thus, CoM sagittal sway results from fluctuations in calf muscle activity acting through the low stiffness SEC. Any sway resulting from heart beat or respiration must be much smaller than the average sway pattern reported here.

Neural mechanisms of postural control

Time-locked averaging of micro falls (Fig. 7) and changes in muscle length (Fig. 6) indicate that the nervous system responds to an increase in CoM velocity with a time delay of around 370 ms. The latency of a group I stretch reflex is 65 ms (Evans *et al.* 1983) or 42 ms (Schieppati & Nardone, 1999) and that of the group II, medium latency stretch reflex is 75 ms (Schieppati & Nardone, 1999). Thus, local stretch and other reflexes are ruled out on the basis of timing. As the response pattern of the EMG activity does not follow any one signal (e.g. ankle angle, ankle velocity, muscle length, ankle torque) it probably draws on composite sources. In this case, CoM acceleration (a composite signal) and the simultaneous changes in calf muscle length provide the earliest information of the loss of balance. It is well known that the brain will use any information from any meaningful source to solve the task at hand. Thus the most natural explanation, consistent with the timing information, is that the nervous system uses all global sources of information available that contribute to knowing the motion of the CoM. This explanation is consistent with the results derived from relatively large balance perturbations that show that global rather than local sources of proprioception are used to control the ankle musculature during standing (Bloem *et al.* 2000; Allum *et al.* 1998). This explanation is also consistent with research that shows that integrated information from multiple sense organs is readily combined and reweighted in the maintenance of balance (Peterka, 2002; Oie *et al.* 2002; Peterka & Loughlin, 2004).

The nervous system acts predictively during quiet standing as the EMG pattern producing an impulse terminates well before velocity cancellation is complete (Figs 6 and 7). It has been recently proposed that velocity feedback can explain the predictive modulation of calf muscle activity (Masani *et al.* 2003). These authors argue that the nervous system acts as a PD controller with a time delay of 100 ms such that the control signal (EMG) reflects the CoM velocity and angle signals. Our results show that the EMG signal controlling muscle impulses generally anticipates CoM velocity by 58 ms and 28 ms, for soleus and gastrocnemius, respectively (Fig. 6), and when losses of balance (rather than all impulses) are sampled (Fig. 7), EMG signal anticipates ankle velocity and fractionally lags (4–17 ms) CoM velocity. Given that a short latency reflex requires about 42 ms (Schieppati & Nardone (1999)), even this fractional lag during losses of balance is inconsistent with the idea that the control signal follows CoM velocity after a time delay of 100 ms.

Generally, the timescale (400 ms) of the observed impulses (Fig. 6) and reaction to loss of balance (Fig. 7) means the brain has time to use its own internal models of CoM motion and muscle activity to modulate the amplitude and timing of the ballistic bias activity. Arguably the earliest knowledge the nervous system has of an impending micro fall, comes from the knowledge of previous changes in muscle length combined with an internal model of the effect of those changes in muscle on the motion of the CoM. As the impulses are delivered ballistically, the nervous system will almost certainly refine its internal models with the immediate feedback of results that is received from the velocity signal. This leads to the speculation that a supervising learning network is utilized. Such updating of internal models is thought to occur in the cerebellum (Imamizu *et al.* 1998; Wolpert *et al.* 1998; Morasso *et al.* 1999; Kawato, 1999). This postulated role of the cerebellum in standing might explain why patients with cerebellar ataxia suffer impaired postural control (Sanguineti *et al.* 2003). Our interpretation identifies standing balance in humans as an automated, trial and error, skilled, learned activity more than a low level reflex process.

Paradigms of posture

The mechano-reflex understanding of postural mechanisms has derived much evidence from investigations on cats and other quadrupeds. The animal stands on limbs which are flexed by the animal's weight. Collapse is prevented by constant activity of the extensor muscles. An external perturbation on the animal causes the extensor muscles to be elongated as the joints flex beyond their set position. The perturbation is resisted by intrinsic mechanical joint impedance supplemented by local reflexes.

In human standing, forward toppling about the ankle joint is resisted by near constant activity in the soleus and gastrocnemius muscles. Much contemporary analysis has extended the mechano-reflex paradigm to human standing (Gurfinkel *et al.* 1974, 1995; Shadmehr & Arbib, 1992; Fitzpatrick *et al.* 1992, 1994, 1996; Horak & MacPherson, 1996; Winter *et al.* 1998; Schieppati & Nardone, 1999; Fitzpatrick, 2003; Masani *et al.* 2003). A common view is that in man, as with other mammals, the posture-preserving system is phylogenetically old and operates relatively autonomously (van Ingen Schenau *et al.* 1996; Massion *et al.* 2004). If the cerebellum is intimately involved in the automation of standing balance then the process is phylogenetically more recent than previously thought and if the anticipatory impulsive adjustments of soleus and gastrocnemius required for standing are the same process as the anticipatory postural adjustments required as a preliminary to general movement, then the posture-preserving system may in fact be much more integrated into the movement control scheme than has been recognized.

The evidence presented here shows that in quiet standing, the sagittal motion of the CoM is controlled by an active, impulsive, ballistic, process operating at a rate of 2.6 modulated actions per second consistent with complex sensorimotor integration and predictive planning. This process is a good candidate for automation by the cerebellum. The CoM stands nearly perfectly balanced by the SEC of the calf muscles as a well-sprung, mobile mechanism that is ponderously unstable. By delivering alternating impulses via the gastrocnemius and soleus muscles, the nervous system keeps the velocity of the CoM low and controls the position of the CoM. As well as controlling CoM position and velocity the bias adjustments are themselves the major source of postural sway. Because their magnitude is rarely if ever precisely correct they act as perturbations which if left uncompensated, would lead to a fall.

Here is a simple analogy that illustrates the impulsive ballistic nature of the process. Imagine trying to maintain a heavy ball as still as possible on a hillside. The ball is controlled by striking it with a bat at a relatively fixed rate. The motion of the ball will be caused by the blows themselves. It will move sometimes up the hill (because the effect of the blows are greater than gravity) and sometimes down the hill (effect of blows less than gravity), but not in any regular way. It can be maintained near the top of the hill or near the bottom or at any point in between. To do this, the batter has to judge the size of each blow. We suggest that in essence it is this never ending, trial and error process which has to be carried out in human standing. The process of loss of balance and regaining balance has to be repeatedly solved under the ever changing conditions of balance and we suggest that this is a skilled, trial and error activity that improves with experience rather than a reflex process.

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