The Consequences of Short-Range Stiffness and Fluctuating Muscle Activity for Proprioception of Postural Joint Rotations: The Relevance to Human Standing

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Loram ID, Lakie M, Di Giulio I, Maganaris CN. The consequences of short-range stiffness and fluctuating muscle activity for proprioception of postural joint rotations: the relevance to human standing. J Neurophysiol 102: 460–474, 2009. First published May 6, 2009; doi:10.1152/jn.00007.2009. Proprioception comes from muscles and tendons. Tendon compliance, muscle stiffness, and fluctuating activity complicate transduction of joint rotation to a proprioceptive signal. These problems are acute in postural regulation because of tiny joint rotations and substantial short-range muscle stiffness. When studying locomotion or perturbed balance these problems are less applicable. We recently measured short-range stiffness in standing and considered the implications for load stability. Here, using an appropriately simplified model we analyze the conversion of joint rotation to spindle input and tendon tension while considering the effect of short-range stiffness, tendon compliance, fluctuating muscle activity, and fusimotor activity. Basic principles determine that when muscle stiffness and tendon compliance are high, fluctuating muscle activity is the greatest confounding factor. The unstable human “inverted pendulum” (Loram et al. 2007a), for load stabilization, which is to increase the time constant of postural movements: short-range muscle stiffness, tendon compliance, and task-related fluctuating muscle activity.

Much of the knowledge concerning proprioception comes from studies related to locomotion, perturbed balance, or laboratory preparations where movements are relatively large, i.e., of several degrees magnitude (Gandevia 1996; Prochazka 1996; Windhorst 2007). During postural tasks such as human standing, postural control at the wrist and elbow or postural tremor, the regulated joint movements are very small, i.e., a few tenths of a degree (Chew et al. 2008; Hore et al. 1990; Loram et al. 2005a,b), yet it is known that muscle proprioceptors can register these small movements (Fitzpatrick and McCloskey 1994; Fitzpatrick et al. 1994; Hall and McCloskey 1983). Proprioception of such small movements is problematic for reasons that are less relevant and usually ignored in studies related to locomotion or perturbed balance. Three issues complicate muscle proprioception of small postural movements: short-range muscle stiffness, tendon compliance, and task-related fluctuating muscle activity.

Functionally, any impairment in ability to sense such small joint movements is important for the regulation of balance and fine motor control. For all populations, healthy, aged, and those in a neurologically compromised state, understanding the physiological principles of proprioceptive impairment is inherently valuable.

Short-range stiffness

The muscle tendon unit comprises contractile tissue (muscle) in series with tendinous tissue: thus joint rotation is transmitted mechanically to elongation of the series contractile tissue (muscle) and to changes in tension of the muscle tendon unit. Muscle is well known to have substantial short-range stiffness (Campbell and Lakie 1998; Hill 1968; Rack and Westbury 1974) and tendion stiffness is low at low forces (de Zee and Voigt 2001; Hof 1998; Ker 1981; Maganaris and Paul 2000). For small postural ankle rotations, muscle stiffness is much higher than tendon stiffness, whereas normally for larger rotations the tendon and muscle are approximately equal in stiffness (Herbert et al. 2002; Hoang et al. 2007; Loram et al. 2007b). Short-range muscle stiffness is approximately proportional to muscle tension (Rack and Westbury 1974); so, consistent with similar examples (Gurfinkel et al. 1997; Halaki et al. 2006), in normal standing with moderate muscle activity, one would expect short-range muscle stiffness to be similar to or only slightly higher than that for passive muscle. Previously we considered one implication of short-range muscle stiffness for load stabilization, which is to increase the time constant of the unstable human “inverted pendulum” (Loram et al. 2007a).

INTRODUCTION

It is well known that muscle sensory organs, such as spindles and Golgi tendon organs, which are sensitive to changes in fascicle length and muscle tension, respectively, play a key role in the proprioception of movement. Moreover, although the role of Golgi tendon organs and nonmuscular afferents should not be forgotten, the longstanding view is that joint rotation and thus the proprioception of movement are mainly sensed by muscle spindles registering changes in fascicle length (Gandevia 1996; Hall and McCloskey 1983; Matthews 1981; Proske 2006).
Here we analyze in detail the role of short-range muscle stiffness in the proprioception of small postural movements.

**Nonlinear spindle gain in relation to short-range stiffness for passive muscle**

As muscle stiffness increases in relation to tendon stiffness, the proportion of joint rotation transmitted to muscle elongation decreases. For small movements <0.5°, the ratio of muscle elongation to joint rotation is approximately ten times less than that for movements >0.5° (Loram et al. 2007b) (see also Figs. 1 and 2). The elongation of extrafusal and parallel embedded intrafusal fibers would be approximately equal (Mileusnic et al. 2006). For passive muscle (i.e., no motor units are neurally stimulated), spindles are beautifully and complementarily adapted to transduce these small movements. Muscle spindles have an inherent nonlinear response to displacement with enhanced receptivity to small displacements (Matthews and Stein 1969). Larger displacements evoke a reduced ratio of afferent firing to muscle elongation and this ratio can be adapted by altering the activity of the fusimotor neurons ($\gamma_{\text{dynamic}}$ and $\gamma_{\text{static}}$) (Matthews 1981). In effect, the gain of the spindles is variable so that passive rotations can be transduced over a wide range of sizes and velocities.

**Adjustment of bias for tonically active muscle**

What happens when the muscle is active (i.e., motor units are stimulated by $\alpha$ motoneurons)? Spindle firing is related to stretch of the intrafusal fibers embedded in parallel with extrafusal fibers. Tonic $\alpha$ motoneuron activity results in shortening of extrafusal and intrafusal muscle fascicles, causing intrafusal fibers to approach their slack length below which there is insufficient intrafusal stretch to depolarize the afferent Ia and II fiber endings. Normally, efferent fusimotor activity ($\gamma_{\text{dynamic}}$ and $\gamma_{\text{static}}$) is increased, which stretches the sensory portion of the intrafusal fibers (Mileusnic et al. 2006), resulting in a change, usually an increase, in the tonic firing of the primary and secondary spindle afferents (Aniss et al. 1990; Crowe and Matthews 1964; Matthews 1981). This constant $\gamma$ activation allows muscle spindles to retain their sensitivity to muscle stretch across a wide range of levels of tonic muscle activation. Following Mileusnic et al. (2006) and the anatomical understanding that fusimotor activity stretches the springlike sensory portion of intrafusal fibers, we describe constant fusimotor activity as applying a constant bias to the intrafusal fibers. For this study, we define intrafusal bias as lengthening of the sensory portion of the intrafusal fibers caused by fusimotor activity. For a measuring instrument it is equivalent to applying an offset to keep the measurement centered about zero or the most sensitive region of its “operating range.”

**Varying muscle activity**

As mentioned earlier, through fusimotor activity ($\gamma_{\text{dynamic}}$ and $\gamma_{\text{static}}$), the nervous system can vary the gain (ratio of firing to muscle elongation) and bias (muscle length that evokes zero firing) of the spindle response to changes in muscle length. Is this adaptable bias and gain approach adequate when the task requires muscle activity to be modulated?

For example, during quiet standing the muscle activity is moderate, perhaps 10% of maximum voluntary contraction. However, the postural load is unstable and thus balance is achieved only by appropriate modulation of muscle activity (Casadio et al. 2005; Lakie et al. 2003; Loram and Lakie 2002; Loram et al. 2005a). Variation in muscle activity causes the muscle fibers to shorten and lengthen. The crucial point is that changes in extrafusal fiber length represent a sum of active and passive components—i.e., active shortening/relaxation resulting from neural control plus passive elongation/shortening caused by joint rotations produced by body sway. We are considering the passive elongation/shortening caused by joint rotation to be the signal of interest and this signal is only a partial component of the changes in extrafusal fiber length. Both these components are transmitted mechanically to the embedded intrafusal fibers and thus the signal of interest is obscured at the source before transmission to the muscle spindle. The key question is whether altering the gain and bias of the muscle spindles can extract the signal of interest.

Changing the spindle gain will not alter the ratio of the passive and active contributions to changes in extrafusal length, which are transmitted to the muscle spindle. Both the signal of interest and the active shortening will be magnified equally.

A constant intrafusal bias will not alter the ratio of these two components, active and passive, that are transmitted to the muscle spindle. Elongation of the intrafusal sensory region represents a sum of extrafusal elongation and intrafusal bias. Only by changing the intrafusal bias synchronously with extrafusal active shortening can the active component be removed at the source from the mechanical input to the spindle. This latter process would be called a rigid $\alpha$–$\gamma$ linkage, which denotes a precise, dynamic coupling between $\alpha$ and $\gamma$ motor neuron activity, whereas constant $\gamma$ activation, sometimes called $\alpha$–$\gamma$ coactivation, is generally taken to mean that $\gamma$ activity is raised only when $\alpha$ activity is raised, thus maintaining a constant bias associated with tonic muscle activity. Physiologically a rigid fusimotor linkage may be supplied both by the pervasive minority of $\beta$ skeletal fusimotoneurons, which innervate both extra- and intrafusal fibers (Maltenfort and Burke 2003; McWilliam 1975) and by appropriately modulated $\gamma$ motor neurons. To compound the problem of modulating the fusimotor neurons precisely, there are timing differences between extrafusal and intrafusal action due in part to differences in axon conduction velocities ($\alpha$ vs. $\gamma$ and $\alpha$ vs. $\beta$) (Boyd et al. 1977; McWilliam 1975) and also to a lower contraction speed of intrafusal compared with extrafusal fibers (Boyd et al. 1977). The conduction velocity issue applies less for $\beta$ skeletal fusimotoneurons, which are simply (slower) collaterals of $\alpha$ motoneurons.

**What is known about intrafusal activity?**

Currently, relatively little is known about $\gamma$ activity in locomotion and postural control (Windhorst 2007). It is known that $\gamma$ efferents are activated to adjust the gain of spindles and also that $\gamma$ efferents are coactivated with $\alpha$ motoneurons to remove the effect of muscle activity, at least in a relatively gross manner. The most recent and sophisticated modeling of muscle spindles confines itself to constant firing rates in the $\gamma$ efferents (Mileusnic et al. 2006). Recent evidence has shown modulation of $\gamma$ activity during locomotion (Taylor et al. 2000), although these are relatively large movements and large modulations in $\gamma$ activity.
Primary problem: modulated muscle activity and small joint rotations

When changes in muscle length resulting from joint rotation are large with respect to active variations in muscle length, the passive component would dominate the combined active and passive components of spindle input. When joint rotations are small (as in normal standing), the passive component might be lost in comparison with the larger active fluctuations in fascicle length. Qualitatively, one would expect the signal-to-noise ratio to depend on the amount of joint rotation transmitted to the muscle in relation to the amount of active shortening/lengthening of the muscle.

A key question is how large are the active fluctuations in fascicle length in relation to those caused by postural joint rotations? In human standing, balance of an inverted pendulum via a compliant linkage dictates that the amount of active muscle shortening/lengthening in relation to the amount of joint rotation is determined by the stiffness of the Achilles tendon relative to the load stiffness and the moment of inertia of the human “inverted pendulum” (Lakie et al. 2003; Loram et al. 2005a). Load stiffness represents the ratio of gravitational toppling torque to center-of-mass angle from the vertical. Observation of the paradoxical changes in muscle length associated with quiet standing sway (Loram et al. 2005a,b) implies that changes in muscle length are dominated by active modulation rather than passive transmission of joint rotation (Figs. 2 and 4; Loram et al. 2005a). However, conclusions are complicated by the fact that standing balance is a closed-loop process in which fluctuations in muscle activity are also related to bodily sway by postural feedback mechanisms. Thus apart from knowing that paradoxical muscle movements have to be predominantly active rather than passive, determining the full extent of the active modulation requires knowledge of the ratio of muscle to tendon stiffness, which was not available at that time.

What proprioceptive mechanisms can register ankle joint rotation during quiet standing?

These previous observations leave open the question of which proprioceptive mechanisms can transduce ankle joint rotation under such circumstances. Attention was drawn briefly to four possible mechanisms (Loram et al. 2005a): 1) spindles in the active agonist muscle with constant activity, 2) spindles in the active agonist muscle with rigid \( \alpha-\gamma \) linkage, 3) spindles in the passive antagonist muscle, and 4) Golgi tendon organs in the active agonist muscle. The purpose of this report is to combine what has been recently discovered about short-range muscle stiffness and tendon compliance in relation to human standing to analyze and quantify the relative merits of the four proposed mechanisms for transducing joint rotation into a proprioceptive signal. For understanding muscle proprioception, this comparison illuminates fundamental physiological questions (Windhorst 2007). Using an appropriately simplified open-loop model of muscle and spindles we quantify the transmission of joint rotation to muscle spindle input and Golgi tendon organ input. Herein, muscle spindle input refers throughout to displacement of the equatorial sensory portion of the intrafusal fibers and thus includes mechanical and fusimotor contributions. Tendon organ input refers to muscle–tendon tension. Specifically, we ask the following questions.

1 During human standing, what is the ratio of actively modulated to passively transmitted changes in extrasynaptic muscle length?

2 For registration of joint rotation, what is the relative effectiveness of i) spindles in passive muscle, ii) spindles in active agonist with noisy coactive but otherwise constant \( \gamma \) activity, iii) spindles in active agonist with rigid \( \alpha-\gamma \) coactivation, and iv) muscle–tendon tension?

3 What testable predictions regarding spindle afferent firing will discriminate between constant \( \gamma \) activity versus rigid \( \alpha-\gamma \) coactivation?

4 Does short-range muscle stiffness lead to an effective dead zone in the proprioception of joint rotations?

METHODS

Ultrasound measurement of changes in muscle length

The questions and analysis in this study are derived from previous observations of human quiet standing (Loram et al. 2005a,b) and passively applied rotations of the ankle joint (Loram et al. 2007b) during which we used ultrasound to measure continuous changes in muscle length of the gastrocnemius and soleus muscles. In this previous work we measured changes in length of the muscle (contractile element) along the longitudinal line of the muscle tendon unit, as shown in Fig. 1. Thus in these previous studies, and in the analysis of this study (Fig. 3A), the muscle tendon unit is regarded as a tendon in series with a muscle; contractile element (muscle) elongation \( L_{ce} \) refers to longitudinal changes such as those measured by this ultrasound method. For these small length changes in which the geometry of the muscle changes very little, we assume that the architectural gear ratio (the ratio of longitudinal strain to fascicle strain) remains constant. Thus we assume changes in fascicle length, “seen” by spindles, are linearly related to changes in muscle length measured by ultrasound.

Calculation of stiffness ratio \( r = k_{se}/k_{ce} \)

In previous work, we applied constant speed, 0.35 deg s\(^{-1}\), ankle rotations of 7° to participants who were supported in a vertical standing position (Loram et al. 2007b). Since they were supported, these participants were able to keep the calf muscles passive. From these experiments we obtained measurements of the contractile element (muscle) elongation \( L_{ce} \) and series elastic (tendon) elongation \( L_{se} \) of the passive calf muscles soleus and gastrocnemius for displacements from 0.03 to 7°: these measurements are presented again for the convenience of the reader in Fig. 2, A and B. From these measurements, and for the analysis of this study, we calculate the ratio

\[
r = k_{se}/k_{ce} \tag{1}
\]

where \( k_{se} \) is the contractile element (muscle) stiffness and \( k_{ce} \) is the series elastic (tendon) stiffness. For passive muscle in which there is no active tension and the muscle tendon unit is effectively two springs in series, the stiffness ratio is calculated using

\[
r = L_{ce}/L_{se} \tag{2}
\]

to give values for all displacements from 0.05 to 7° of ankle rotation. The change in stiffness ratio (tenfold, mostly between 0.3 and 0.4°) is shown in Fig. 2.
Muscle model

We aim to represent the primary characteristics of muscle, which is that it shortens actively and is elongated passively by tendon tension. We use the simplest possible model of muscle that allows for the fact that muscle–tendon tension is a sum of active and passive components. We use a linear Hill-type model comprising a tendon spring in series with a contractile element consisting of an active tension generator and passive spring. Although muscle behavior is clearly nonlinear, our analysis applies to movements within a small range.
within which model coefficients may be assumed to be constant. We omit damping elements from the tendon and contractile element since behavior is almost entirely springlike during the small, slow movements present under postural conditions (Figs. 5 and 6; Loram et al. 2007b). After writing equations for tension in the contractile element and tension in the series elastic element, we substitute for tension in the series elastic element. The model is then defined by two equations that relate the output’s tension $T$ and contractile tissue length $L_{c,t}$ to the input’s active tension $\alpha$ generated by alpha motoneurons and muscle–tendon unit length $L$ generated by joint rotation

$$T = \alpha + Lk_{c,t}\ell(r + 1)$$

(3)

$$L_{c,t} = -\alpha/k_{c,t} + L(r + 1)$$

(4)

In this model and all the models that follow, quantities are regarded as calculated relative to their mean values.

**Spindle model**

Muscle spindles contain several parallel intrafusal fibers embedded as a group in parallel with extrafusal (skeletal) muscle fibers and provide afferent information in response to elongation of the intrafusal (and associated extrafusal) fibers (Boyd et al. 1977; Matthews 1981). Intrafusal fibers contain a passive sensory region in series with a polar region that can actively shorten as well as be passively elongated. Primary afferents are wrapped around the sensory region and respond to stretch of this region. Although some secondary endings are likely to lie entirely in the polar region (Banks et al. 1982), in general secondary afferents straddle the boundary of the sensory and polar regions and respond to stretch of both regions (Matthews 1981; Mileusnic et al. 2006). Activity in the $\gamma_{\text{static}}$, $\gamma_{\text{dynamic}}$, and $\beta$ fusimotor neurons causes the polar regions to shorten and stretches the equatorial sensory region (Boyd et al. 1977). Thus fusimotor activity stimulates the primary afferents (Boyd et al. 1977) and has a more ambiguous effect on the secondary afferents, although these are usually stimulated (cf. Fig. 5; Mileusnic et al. 2006). Fusimotor activity also modulates the damping stiffness of the polar region, which affects velocity sensitivity of the primary afferent (Matthews 1981).

The purpose of this analysis is to model the changes in spindle input in the simplest possible way. When reduced to its simplest form, spindle input can be represented as changes in length of equatorial sensory region ($S$), which depends on two main inputs, contractile tissue length $L_{c,t}$ and a generalized fusimotor activity $\gamma$ and may be represented as

$$S = k_1 L_{c,t} + k_2 \gamma$$

(5)

where the coefficients $k_1$ and $k_2$ are determined by spindle anatomy and physiology (McMahon 1984). For small movements, these coefficients are assumed to be constant.

**Derived model for changes in spindle input**

Substituting $L_{c,t}$ from Eq. 4 into Eq. 5 gives the model for calculating spindle input $S$

$$S = -k_1 \alpha/k_{c,t} + k_2 L(r + 1) + k_2 \gamma$$

(6)

Thus spindle input is determined by muscle–tendon length $L$ (which itself is determined by joint angle), extrafusal muscle activity $\alpha$, and fusimotor activity $\gamma$.

Each of these inputs is weighted by variables of physiological significance. The extrafusal muscle activity variable is weighted by tendon compliance $1/k_{c,t}$. The “joint rotation” input $L$ is inversely weighted by $r$, the ratio of muscle stiffness relative to tendon stiffness. Fusimotor drive is weighted by unmodeled variables within the muscle spindle system. The unmodeled relationship between spindle input $S$ and afferent firing is inherently nonlinear but is also influenced by fusimotor activity.

Two special cases of fusimotor activity are considered.

**Case 1. Constant fusimotor activity**

All quantities represent variation from the mean value. Thus when fusimotor activity is constant $\gamma$ is zero and so Eq. 6 reduces to

$$S = k_1[-\alpha/k_{c,t} + L(r + 1)]$$

(7)

Spindle input is determined by joint angle and extrafusal muscle activity. The dominant contribution is determined by the extent of muscle activity and “joint rotation” weighted inversely by tendon stiffness and contractile (relative to tendon) stiffness, respectively.

**Case 2. Rigid $\alpha-\gamma$ linkage ($\beta$ linkage)**

Fusimotor activity $\gamma$ is modulated and contains a component of rigid $\alpha-\gamma$ coactivation that is an exact copy of alpha muscle activity. This rigid coactivation may include input from $\beta$ skeletal fusimotor neurons, which innervate both extra- and intrafusal fibers (Maltenfort and Burke 2003). Fusimotor activity $\gamma$ contains an additional second component $n$, which is everything independent of $\alpha$ activity. We can construct a model in which fusimotor activity is the sum of these two components. The coefficients of each component are written in a way that is convenient to give

$$\gamma = (\beta k_1 k_{c,t} + n) k_2$$

(8)

This model is entirely general; $\nu$ might be related to some other variable, be constant, or be random. The coefficients for the $\alpha$ component of $\gamma$ are arranged so that coefficient $\beta$ determines whether the coactive component is scaled precisely ($\beta = 1$) or $\gamma$ (imprecisely ($\beta \neq 1$). Substituting Eq. 8 into Eq. 6 gives

$$S = k_1[(\beta - 1)\alpha/k_{c,t} + L(r + 1)] + n$$

(9)

If the cancellation is precise ($\beta = 1$) spindle input change is determined only by “joint motion” $L$ and independent gamma activity $n$

$$S = k_1[L(r + 1)] + n$$

(10)

If $n$ is random, then with ensemble averaging by a sufficient number of spindles the “joint rotation” signal could in principle be extracted from the sensory intrafusal displacement in the presence of fluctuating muscle activity. Such ensemble averaging is likely, even at the level of the $\alpha$ motoneuron, since each $\alpha$ motoneuron receives terminals from all or nearly all the primary afferent fibers in that muscle (Mendell and Henneman 1971).

**Simulation investigations**

The purpose of the simulations is to provide quantitative predictions and visualizations of the factors governing individual and ensemble-averaged spindle input in relation to small postural ankle rotations against a background of postural fluctuations in muscle activity. The muscle and spindle models were implemented as blocks in the MATLAB, Simulink environment (Fig. 3C) and normal values from quiet standing were taken for tendon stiffness ($c = 0.85$), the ratio of muscle to tendon stiffness ($r = 10$), and a moment arm of 5 cm (Loram et al. 2007b).

In previous work (Loram et al. 2005a,b) we studied ten subjects standing quietly with three trials each of eyes open and eyes closed. From simultaneous force plate, laser range finder, soleus and gastrocnemius electromyography (EMG), and ultrasound measurements we calculated changes in the sagittal location of the center of gravity.
ankle joint rotation, and muscle length ($L_{ce}$). The mean size of individual sways measured as rotation about the ankle joint was 0.13 and 0.23° with eyes open and eyes closed, respectively. The mean sway frequencies were 0.39 and 0.4 Hz, respectively.

Thus for simulation analysis, sinusoidal ankle joint rotations of peak-to-peak (pk-pk) amplitude 0.2° at 0.4 Hz would appropriately match normal standing. For these simulated ankle rotations, the corresponding pk-pk muscle–tendon length displacement is 175 μm, given the moment arm of 5 cm. Moreover, given a prevailing short-range muscle to series elastic stiffness ratio of 10 (Loram et al. 2007b), the amount of joint rotation passively transmitted to muscle elongation would be about 10 and 18 μm, respectively, for eyes open and eyes closed, hardly contributing to the observed changes in muscle length.

From the previous measurements of ten quietly standing subjects (Loram et al. 2005a) the mean fluctuations in muscle length of gastrocnemius and soleus were ±209 and 133 μm (±SD), respectively. These fluctuations were predominantly paradoxical (negatively correlated with respect to joint rotation) and thus actively rather than passively produced. From those data (Loram et al. 2005a) and also using recent measurements of short-range muscle stiffness (Loram et al. 2007b), the expected ratio of actively modulated to passively produced changes in muscle length is calculated in the Appendix. These calculations support the idea that the observed ±209- and ±133-μm fluctuations are almost wholly caused by modulations in muscle activity.

For the following simulation investigations we chose to use the soleus muscle as representing a constantly active and constantly modulated agonist. Rather than use an active muscle length (soleus muscle), we use a representative (median SD) 40-s record of soleus muscle to series elastic stiffness ratio of 10 (Loram et al. 2007b), the expected ratio of actively modulated to passively modulated muscle activity (Fig. 4). Sinusoidal joint rotations (2, 1, and 0.2°, pk-pk) are applied relative to the soleus muscle modulated with the representative pattern described earlier. No modulation is applied to the γ motoneurons. The ratios of contractile to series elastic stiffness $r$ are 1, 1, and 10, respectively, for the 2, 1, and 0.2° inputs.

Representative effect of fluctuating muscle activity and rigid α–γ coactivation (Fig. 5). Sinusoidal joint rotations (0.2°, pk-pk, equivalent to 175 μm pk-pk change in muscle–tendon length), are applied to three muscle cases including:

1. Passive muscle, with no active α or γ modulation.

2. Active muscle with active extrafusal fluctuations (α) of ±133 μm (±SD) and constant intrafusal activity (γ) containing additional low-frequency noise (±50 μm, ±SD).

3. Like 2) but with additional rigid α–γ coactivation (β = 1) applied to γ.

The intrafusal (γ) noise is randomly seeded, white noise limited to 100 Hz and low-pass filtered with a first order time constant of 2 s. In all, 100 spindles are used to provide a convenient display in Fig. 5.

Effect of number of spindles and γ noise on ensemble averaged spindles (Fig. 6, A and B). Sinusoidal joint rotations (0.2°) are applied to the three muscle cases cited earlier. The amplitude of the random γ noise is varied: the SD of the γ noise (measured as intrafusal length modulation) relative to the ±133 μm (±SD) extrafusal modulation varied from 0.1 to 5 (i.e., ±13 to ±660 μm). The ensemble-average changes in spindle input are calculated from 500 spindles.

Effect of rigid α–γ gain (β) on ensemble averaged spindles (Fig. 6, C and D). Sinusoidal joint rotations (0.2°) are applied to passive muscle and the actively modulated muscle with rigid α–γ coactivation. The gain of the α–γ coactivation is varied from β = 0 to β = 2. The relative amplitude of the random γ noise is 0.5 and 500 spindles are averaged for the ensemble effect.

Effect of short-range stiffness and tendon compliance on ensemble-averaged spindles (Fig. 6, E–G). Sinusoidal joint rotations (0.2°) are applied to the three preceding muscle cases. The activity input (α) for the muscle is kept the same as that for previous muscles, which would result in 133 μm (±SD) for the case where relative tendon stiffness $c = 0.85$. Where $c = 0.85$ the muscle length fluctuations resulting from the same activity are greater and vice versa. In the rigid α–γ muscle, the relative amplitude of the random γ noise is 0.5 and the gain of the α–γ coactivation $β = 1$. Tendon stiffness relative to the load stiffness $c$ is varied from 0 to 1.8. Contractile element relative to tendon stiffness $r$ is set at 1 and 10 for long-range and short-range stiffness, respectively. In all, 500 spindles are averaged.
Effect of size of ankle rotation and muscle activity on individual and ensemble averaged spindles (Fig. 7). Sinusoidal joint rotations ranging from 0.05 to 4° (pk-pk) (44–3,500 μm, pk-pk) are applied to muscle in which the active extrafusal modulation (α) is varied from 0 to 1,330 μm (±SD). For each rotation angle the contractile element length Lce (i.e., muscle length) is taken from Fig. 2D for soleus. Rigid α–γ intrafusal modulation is applied: the relative amplitude of the random γ noise was 0.5 and β was 1. For the ensemble response, 500 spindles are averaged.

RESULTS

Joint rotation is poorly transmitted to muscle elongation during postural movements

The nonlinear transmission of ankle joint rotation to elongation of the passive soleus and gastrocnemius muscles is shown in Fig. 2. The elongation of the muscle rises from about 50 μm deg⁻¹ for 0.05° rotations to about 500 μm deg⁻¹ for 2° rotations with a transition at around 0.4° (Fig. 2, A and B). This nonlinear transmission is associated with a ratio of muscle to series elastic stiffness, which decreases from about 20 for 0.05° rotations to about 1 for rotations >0.4° (Fig. 2, C and D). The transition from the short-range region to the long-range region at around 0.4° means that the normal, small sways of quiet standing lie within the region of short-range stiffness with a muscle to series elastic stiffness ratio of approximately 10.

The following simulation results show the behavior of the model (Fig. 3) using sinusoidal ankle rotations and measured fluctuations in muscle length (Loram et al. 2005a). Postural fluctuations in muscle length and tension reflect muscle activity rather than joint rotations

As the size of ankle rotations decreases in relation to a fixed background of modulated activity (Fig. 4, A–C), the changes in muscle length (Fig. 4, D–F) and muscle–tendon tension (Fig. 4, G–J) progressively reflect active postural modulation (Fig. 4, A–C, dashed) rather than joint rotation (Fig. 4, A–C, solid). The modulated α activity is representative of quiet standing. Ankle rotations of 2 and 1° are clearly transmitted to modulation in muscle length and tension. For ankle rotations of 0.2°, muscle length and tension reflect active modulation rather than the...
joint rotation. If spindle input in muscle spindles reflects changes in muscle length, then during small postural movements of 0.2°, spindles would register active modulation rather than joint rotation (Fig. 4, J–L).

Two sensory solutions: passive muscle and rigid $\alpha$–$\gamma$ coactivation

Postural ankle joint rotations of 0.2° (Fig. 5A), applied to passive muscle with no extrafusal or intrafusal activity (Fig. 5B), produce small changes in extrafusal contractile length (Fig. 5C) and spindle input (Fig. 5D). These changes in length are uncomplicated by fluctuations in muscle activity and have a high correlation with joint rotation (Fig. 5E).

The same joint rotations (Fig. 5F) applied to active muscle with postural fluctuations in muscle activity (Fig. 5G) cause changes in muscle length that are largely obscured by muscle activity as previously (Fig. 5H). When $\gamma$ fusimotoneurons are coactive with $\alpha$, but showing constant but noisy activity (Fig. 5G), then a population of sensory intrafusal fibers experiences changes in length highly correlated with extrafusal, $\alpha$, muscle activity (Fig. 5, I and J) and poorly correlated with joint rotation (Fig. 5K).
For the same joint rotations (Fig. 5L) and active muscle (Fig. 5M), when γ input includes a component of rigid α–γ coactivation (Fig. 5M) that on average is tuned to produce changes in polar intrafusal length that perfectly match active extrafusal shortening (β = 1), then the correlation between spindle input and extrafusal activity is lost (Fig. 5, O and P). The spindles are weakly associated with joint rotation (Fig. 5Q) complicated by the noise of imperfect matching of intrafusal and extrafusal activity (Fig. 5M). Thus using rigid α–γ coactivation, spindles are correlated with joint rotation with a low signal to noise ratio (Fig. 5Q).

**Ensemble averaging of spindles with rigid α–γ coactivation increases the signal-to-noise ratio**

The coefficient of variation (R^2) presented shows the variance in ankle joint rotation predicted by the ensemble-averaged spindle input.

For passive or unmodulated muscle, the R^2 is very high (R^2 \approx 1) for any number of spindles. Thus the signal-to-noise ratio for sensory intrafusal fibers registering 0.2° joint rotations is extremely high and ensemble averaging is not needed (Fig. 6A).

For actively modulated muscle with γ coactivation but no rigid α–γ linkage (β = 0), the R^2 is very low (R^2 \approx 0) for any number of spindles. Ensemble averaging does not improve the signal-to-noise ratio (Fig. 6A) because the “noise” is not random but is associated with extrafusal muscle activity.

For actively modulated muscle with a rigid α–γ linkage (β = 1), the R^2 increases with the number of spindles averaged in the ensemble. Thus averaging increasing numbers of spindles produces a signal that predicts the ankle joint rotation more highly (Fig. 6B). This is because the mismatch between extrafusal and intrafusal activation (γ–α, Fig. 5M) is random and reduced by averaging. The number of spindles required to produce a signal of high predictive value depends on the extent of random mismatch between γ and α modulation. Intrafusal mismatch (±SD) equal to postural modulation in activity (α ±SD) required 500 spindles to produce a signal with an R^2 approaching 0.5. When the mismatch is fivefold α modulation, the R^2 never reaches 0.05 with 500 spindles.

**Gain of rigid α–γ coactivation needs to be tuned precisely for small postural rotations**

When the gain β of the rigid α–γ coactivation deviates from one, the R^2 between ensemble-averaged spindle input and joint rotation decreases (Fig. 6C). This is because fusimotor-driven polar intrafusal shortening does not perfectly cancel active extrafusal shortening of contractile tissue. When β is low, corresponding to undercancellation of active extrafusal modulation, spindle input is negatively associated with extrafusal activity α (Fig. 6D). When β is high, corresponding to overcancellation of active extrafusal modulation, spindle input change is positively associated with extrafusal activity α (Fig. 6D).
6D) and this could form the basis of a positive feedback muscle activity amplifying mechanism via the Ia and II feedback loops. A high ratio of muscle to series elastic stiffness increases the sharpness of the decrease in \( R^2 \) when \( \beta \) is not equal to one (Fig. 6C).

High tendon compliance decreases the correlation between spindles and postural joint rotations

As tendon compliance increases (c decreases), the \( R^2 \) between ensemble-averaged spindle input and joint rotation decreases (Fig. 6, E and F). This is because increasing tendon compliance increases scaling of active changes in muscle length relative to the passively transmitted component (Eqs. 5 and 6). When the muscle is stiff and the ratio \( r \) of muscle to series elastic stiffness is high, then muscle spindles become poorer indicators of joint rotation (Fig. 6, E and F), although muscle tension becomes a better indicator of joint rotation (Fig. 6G).

For passive muscle, tendon compliance is not a factor influencing tension or muscle length as a indicator of ankle rotation (Fig. 6, E–G). Generally, passive muscle length and tension always provide a considerably better prediction (\( R^2 \)) of joint rotation than active muscle (Fig. 6, A–G).

Registration of small postural movements by active and passive muscle

When the size of ankle rotations increases from 0.05 to 4°, the mean correlation between individual spindles and muscle activity is largely independent of the size of ankle rotation (Fig. 7, A and B). If the \( \gamma \) input is constant with random noise, then individual spindles are on average highly correlated with \( \alpha \) activity apart from the largest ankle rotations and lowest muscle activity (Fig. 7A). If the \( \gamma \) input contains a rigid \( \alpha-\gamma \) linkage (\( \beta = 1 \)), then individual spindles are on average not correlated with \( \alpha \) activity (Fig. 7B) and this is not altered by muscle activity or size of rotation.

For passive or unmodulated muscle (Fig. 7C, thinnest line), the \( R^2 \) between ankle rotation and ensemble-averaged spindle input is very high at all amplitudes of rotation.

For muscle with postural modulation of activity, with \( \gamma \) independent of \( \alpha \) (Fig. 7C), the \( R^2 \) is very low for rotations <0.5° but much higher for those >0.5°. As muscle activity increases, there is an increased threshold for the increase in \( R^2 \) and the extent of the rise in \( R^2 \) is reduced.

For actively modulated muscle with postural activity and a rigid \( \alpha-\gamma \) linkage (Fig. 7E) the threshold is reduced to 0.1°. The rigid \( \alpha-\gamma \) linkage substantially decreases the threshold for higher levels of muscle activity and substantially increases the \( R^2 \) for all ankle rotations beyond the threshold.

For actively modulated muscle, muscle tension provides a similar \( R^2 \) with joint rotation for ensemble averaged spindles without \( \alpha-\gamma \) coactivation.

In summary, for passive muscle the \( R^2 \) between ensemble-averaged spindle input (or muscle tension) and joint rotation is far higher than that in active muscle. Modulation of muscle activity substantially reduces the \( R^2 \), although this can be
partially improved by a rigid $\alpha\gamma$ linkage combined with averaging more spindles.

**DISCUSSION**

**Summary of results**

Combining recent measurements of the nonlinear ratio of muscle to series elastic stiffness (Loram et al. 2007b) and of the stiffness of the agonist tendon during human standing (Loram and Lakie 2002; Loram et al. 2005a,b) we have modeled the probable transduction of ankle joint rotations during quiet standing into proprioceptive signals of spindle intrafusal displacement and muscle–tendon tension. An appropriately reduced, the open-loop model of muscle and spindles has led to the following results, which are applicable to ankle joint rotations during quiet human standing and to analogous postural tasks (Chew et al. 2008).

1. Active agonist fluctuations in extrafusal muscle length are one order of magnitude higher than passively transmitted contributions (Eqs. A4 and A5, Figs. 4 and 5). These extrafusal fluctuations are reproduced in intrafusal fibers and obscure passively transmitted input to the muscle spindles (Fig. 5).

2. For registering postural ankle joint rotations passive and unmodulated muscle is much more effective than actively modulated muscle: spindle input and muscle–tendon tension are highly correlated with joint rotation, unlike in the active agonist muscle (Figs. 6 and 7).

3. For the normally modulated, active agonist, spindle input with rigid $\alpha\gamma$ coactivation provides the best registration of joint angle. Muscle–tendon tension is a poor signal of postural rotations and spindle input with constant $\gamma$ activity provides no register of postural rotations (Figs. 6A and 7, C–E).

4. The active agonist muscle shows an effective dead zone in the registration of joint rotations that is related to the short-range stiffness of muscle. For joint rotations $>0.5^{\circ}$, spindle input and muscle–tendon tension become useful signals of joint rotation (Fig. 7, C–E).

5. Using microneurography, the existence of rigid $\alpha\gamma$ coactivation during quiet standing can be tested by the mean correlation of individual spindles with muscle activity. Providing cases of covarying ankle rotation are excluded, a mean correlation of zero would indicate the presence of rigid $\alpha\gamma$ coactivation (Figs. 7, A and B and 6D).

**Physiological validity of the results**

Real muscle and muscle spindles are in reality very complex. However, we are not focusing on the complexities here. Rather we are asking what signal the equatorial part of the spindle might reasonably be expected to “see” when joint rotations are small and task-related active changes in muscle length are relatively high. These results are based partly on recent physiological data and partly on model calculations. What physiological conclusions can and cannot be drawn from such an analysis?

The muscle model is used to calculate changes in muscle–tendon tension and extrafusal muscle length (Fig. 3, A and C). For passive muscle, this model has been previously shown to provide a good description of changes in muscle length and ankle torque during small applied ankle rotations (Fig. 5; Loram et al. 2007b) and is consistent with the well-established principle that muscle is very stiff for small movements (Hill 1968; Rack and Westbury 1974). The model values of muscle and tendon stiffness are derived from these recent experiments and the size of the applied ankle rotations and the active postural fluctuation in muscle length is also derived from recent previous observations of quiet standing (Loram et al. 2005a,b). It can be concluded that active postural fluctuations in muscle length dominate passively transmitted changes from postural joint rotations. Thus muscle-based proprioceptive signals of postural joint rotation (muscle length and tension) are confounded by modulated muscle activity (Figs. 4 and 5) and this cannot be resolved by increasing the gain of sensory organs embedded in the muscle–tendon unit. Proprioception in passive and unmodulated muscle is not complicated by this problem of fluctuating muscle activity.

The spindle model assumes that changes in intrafusal length are the same as changes in extrafusal length (Mileusnic et al. 2006). Since intrafusal length is a sum of active modulation and passively transmitted components, the proprioceptive signal of joint rotation is obscured at the mechanical input to the spindle. The model assumes that fusimotor action shortens the polar intrafusal region and maintains stretch of the sensory region during active extrafusal shortening (Matthews 1981; McMahon 1984; Mileusnic et al. 2006). It is understood that normally under tonic conditions this prevents the spindle afferents from falling silent (Matthews 1981). However, in principle a mechanism based on fusimotor action could cancel the transmission of active extrafusal length fluctuations to the sensory region of the intrafusal fibers. Since fusimotor action during standing is not known, it is reasonable to predict the effect of different mechanisms such as constant $\gamma$ activity or a rigid $\alpha\gamma$ linkage. Since the fusimotor “noise” level is not known, we assume that noise in polar shortening follows the rule of signal-dependent noise (Harris and Wolpert 1998) and is proportional to extrafusal activity modulation. With this assumption we can compare active muscle with passive muscle and with various degrees of modulation in active muscle. Since we do not know the real $\gamma$ noise level, we cannot calculate the actual spindle behavior but can predict it for a range of noise levels (Fig. 7, A and B). Moreover, the effect of $\gamma$ noise can be reduced by ensemble averaging of spindles. By assuming a slightly generous but physiologically realistic number of 500 spindles in the soleus muscles (Voss 1971) we can predict average spindle behavior for different noise levels.

**What factors are not included in the models that might alter the conclusions drawn?**

The muscle model ignores use of a damper since this contributed little to the predicted response (Loram et al. 2007b). The nonlinear effects of larger joint rotations have been ignored since a mean value of muscle stiffness relevant to the size of rotation captured well the muscle–tendon tendon and muscle length for small rotations (Fig. 5; Loram et al. 2007b). The change in resting tension with stretch and other time-dependent effects have been ignored for the same reason. Also for small rotations we have ignored the fact that the intra- and extrafusal muscles may change properties differently at different muscle lengths, giving different sensitivities. Also, we have assumed that the muscle acts homogeneously with respect to activity and stretch. Inhomogeneous behavior is
likely to become more important at low activity levels (Proske and Morgan 1984). Inhomogeneous response to stretch may result in a distribution of intrafusal elongations. Ensemble averaging would extract the mean response from this distribution. Inhomogeneous or partial activation of the whole muscle might in theory leave some populations of extrafusal and intrafusal fibers uncomplicated by active extrafusal shortening. Current ultrasound observation of changes in muscle length during standing support (e.g., Supplementary Material in Loram et al. 2005a) supports the view that active shortening is integrated across the whole muscle (Loram et al. 2005a, 2006b). These factors are likely to increase variability in the organs that sense muscle–tendon tension and muscle length but not alter the conclusions of principle drawn from the model.

The spindle model ignores the relationship between spindle input and afferent firing is concerned with mechanical input or “stretch” of the afferent fibers. It is assumed that sensory intrafusal displacement is an adequate description of that spindle input. Even though secondary afferents span the sensory (70%) and polar regions (30%) of the intrafusal fibers, it is assumed that secondary firing is a linear sum of sensory and polar stretch in which sensory stretch dominates the response and that primary afferent causes a net increase in firing of all afferents (Mileusnic et al. 2006). Similarly, we ignore the observation that primary and secondary afferents may have different meanings for the nervous system (Boyd et al. 1977). It is possible that primary afferents may signal equatorial sensory stretch as we have described here, whereas secondary afferents may signal changes in muscle length that, in quiet standing, largely reflect motor output of the nervous system.

All γ afferents have been collapsed to a single γ input whose only effect is to cause intrafusal shortening. The effect of fusimotor input in changing dynamic and static gain has been ignored. Likewise it has been assumed that during small movements about a mean position, nonlinear effects including slackening of the intrafusal fibers can be ignored. It is unlikely that these omitted factors alter the conclusions of this analysis.

The analysis of this study focuses on the mechanical transmission of joint rotation to spindle input under postural conditions: however, other factors may also influence the proprioception of joint rotations. The intrinsic electrical properties of spinal motoneurons affect the relationship between muscle–stretch–related afferent firing and motoneuron activity. As a consequence of diffuse, neuromodulatory excitation from the brain stem, both animal and human motoneurons, particularly for slow twitch muscle fibers relevant to postural activity, have been shown to exhibit persistent inward currents and persistent firing following activation by stretch-related afferents (Alaburda et al. 2002; Collins et al. 2002; Heckman et al. 2008; Hultborn et al. 2004). When motoneuron firing is persistent, the motoneuron becomes unresponsive to changes in stretch–related afferent input (Heckman et al. 2008). This might be regarded as a temporal refractoriness to sensory input. However, if the persistent state of the motoneuron could be overcome by a sufficiently large change in afferent input, this mechanism could also contribute significantly to a proprioceptive dead zone.

Physiological implications of this analysis

Little is known about fusimotor modulation in human movement and balance. The existence of an effective rigid α–γ linkage in quiet standing is supported by the prevalent presence of β skeletal fusimotoneurons in addition to independent γ fusimotoneurons (Maltenfort and Burke 2003; Matthews 1981; Mileusnic et al. 2006). The predictions made by this analysis enable testing of this idea using microneurography. With no effective rigid α–γ linkage (β = 0), individual spindle firing should be negatively correlated with muscle activity during quiet standing (Fig. 6C). As activity increases, the muscle shortens and spindles also shorten. An effective linkage (β = 1) would reduce this association to zero (Fig. 7C). As activity increases, muscle shortens but the intrafusal fibers also actively shorten by an equal amount so there is no correlation between sensory intrafusal stretch and muscle activity. Note that muscle activity in quiet standing would have to isolate and study changes in muscle activity that are unassociated with covarying ankle rotations. A third, interesting possibility (β > 1), a positive correlation between spindle firing and muscle activity, would indicate positive muscle activity feedback (Fig. 6C). As muscle activity increases, muscle shortens and the intrafusal fibers shorten even more, thus stretching the sensory intrafusal region. During quiet standing there is tantalizing evidence that phasic spindle activity may be positively associated with phasic fluctuations in EMG (Aniss et al. 1990), which would support the third case that β > 1. Further experiments following these authors are especially required.

Registering small postural movements is clearly important for the nervous system. This analysis predicts that passive muscle or muscle with little modulation in activity will be best suited to a sensory role. We think the sensory role of passive muscle has been underrated in the analysis of spinal and higher mechanisms that regulate human posture (Roberts 1978; Rothwell 1994; van Soest and Rozendaal 2008). More specifically, what does this imply for stretch reflex mechanisms?

Stretch reflexes can be considered as feedback pathways using afferent information from muscle spindles in the agonist muscles to modulate activity in those same agonist muscles. When that afferent information correlates highly with sway, one would expect those feedback pathways to make an effective contribution to the task. Figure 7, C and D predicts that autogenic stretch reflexes will be progressively more useful during large imposed ankle rotations, such as perturbed balance, but not during quiet standing where sways are a few tenths of a degree (Loram et al. 2005b). During quiet standing, in the absence of accurate, rigid α–γ coactivation (Fig. 7A), it is more likely that spindle afferents in the active agonist calf muscles will signal muscle activity (e.g., Fig. 2; Loram et al. 2005a); we predict (Fig. 7, C and D) that pathways using afferents from passive antagonists (reciprocal inhibition) or passive synergists will provide more effective peripheral modulation of agonist activity than autogenic stretch reflexes (Di Giulio et al. 2009).

This analysis resonates with recent research into the electrical excitability of cat spinal motoneurons, which shows that reciprocal inhibition from passive antagonist muscles powerfully suppresses persistent inward currents in the agonist ankle extensor (Hyngstrom et al. 2007). These authors have formulated the hypothesis that the focused effects of Ia reciprocal
inhibition in suppressing the persistent inward current allows precise differentiated control patterns to be sculpted from the diffuse neuromodulatory excitation provided by the brain stem (Heckman et al. 2008). Combined with this study, these points strengthen the importance of passive and quiet unmodulated muscle in learning and maintaining fine postural control.

For quiet standing and related tasks there is a need to reexamine the role of passive and quiet muscles as listeners in combination with active agonists. Recent evidence supports the idea that agonist activity in the calf muscle may be intermittent, thus allowing intervening periods of passive sensory function (Di Giulio et al. 2009). More generally, this analysis may explain the findings of previous researchers that muscle sense is attenuated during and before movement (Collins et al. 1998) and also that movement sensitivity is reduced during cocontraction when subjects attributed this to “more noise in the system” (Wise et al. 1998).

The insight that quiet muscles have an important sensory function implies that excessive muscle activity and associated signal-dependent variability is detrimental for postural control and balance. This insight has clinical and behavioral significance. Normal subjects with unnecessary coactivation of muscles and a whole-body pattern of muscle activity that is higher than necessary are predicted to have higher sensory thresholds for postural movements. Likewise, elderly, parkinsonian, cerebellar, and dystonic subjects are predicted to have higher proprioceptive thresholds as a consequence of their increased coactivation of muscles, which are normally inactive in younger or healthy people.

Different muscles have different muscle–tendon properties that are related to the normal biomechanical and physiological demands made on them. However, in addition to the usual considerations, these differing properties have implications for their sensory role during quiet standing.

Soleus, gastrocnemius, and tibialis anterior rank in order of decreasing tendon stiffness (Maganaris 2002). The higher tendon stiffness of soleus lessens the effect of muscle activity in obscuring the passive transmission of joint rotations (Fig. 6, E and F). Soleus also has about 408 spindles (Voss 1971). In combination with a rigid α–γ linkage, this large number of spindles could reduce the threshold for registering joint rotations (Figs. 6, A and B and 7, A and B). Ensemble averaging of a large number of spindles would be required to compensate for variability in the rigid α–γ linkage.

Gastrocnemius has about 156 spindles, which is less than that of soleus (Voss 1971). This analysis predicts that with a smaller number of spindles and lower tendon stiffness, gastrocnemius may have a role different from that of soleus, which includes more passive listening and less active “agonizing.” Previous analysis and observation of soleus and gastrocnemius (Lakie et al. 2003; Loram et al. 2005a,b) have focused on their common role as active agonist. More recent, ultrasound observation during quiet standing has shown differences in behavior, supporting the idea that gastrocnemius has longer durations of passive listening than those of soleus (Di Giulio et al. 2009).

Tibialis anterior has about 284 spindles (Voss 1971), which is a large number relative to its mass, and has a relatively low tendon stiffness (Maganaris 2002). These properties predict that tibialis anterior would be best suited for a passive listening role since passive muscles with compliant tendons do not require a large number of spindles to extract the joint rotation signal. Recent ultrasound observation supports the idea that changes in muscle length of tibialis anterior provide relatively good transduction of ankle rotations during quiet standing (Di Giulio et al. 2009).

It is thought that joint movements are mainly sensed by muscle spindles (Gandevia 1996; Matthews 1981; Prosko 2006). However, muscle–tendon tension also conveys joint rotation information. In passive muscle, tendon tension conveys the same information as muscle length and is uncomplicated by muscle activity (Figs. 6G and 7E). Golgi tendon organs are usually regarded as transducing muscle tension and these organs were once regarded as being insensitive to passive stretch because of their relatively high threshold (Pierrrot-Deseilligny and Burke 2005). However, evidence shows a clear response to passive stretch beyond a relatively low tension threshold, supporting the view that tendon organs appear to be remarkably reliable in signaling whole muscle tension, whether passive or active (Gregory et al. 2002). Thus for passive and unmodulated muscle, it is likely that spindles and Golgi tendon organs contribute harmoniously to a sensation of joint rotation. When activity is modulated, tendon tension reflects activity modulation (Fig. 4) and unlike spindles, Golgi tendon organs have no mechanism for subtracting active modulation. Thus as for spindles with no rigid α–γ linkage, Golgi organs are predicted to be unable to register small postural joint rotations (Fig. 7D), although short-range muscle stiffness would convey an advantage on muscle tension over muscle length information (Figs. 6, G vs. E and 7, D vs. C).

In effect we have considered whether fusimotor activity may be transiently modulated to eliminate transient changes in spindle firing due to transient changes in extrafusal activity. In theory, a similar subtraction process might also occur centrally using an efference copy of muscle activity (cf. Fig. 7; McCloskey et al. 1983). If such a central process occurs, then the same principles apply as for the muscle spindle and one would expect the same conclusions to be drawn. The error in the subtraction process would require reduction by averaging and the error would be relatively large for small joint rotations where muscle stiffness is high. Thus in the actively modulated agonist muscles, these factors predict a proprioceptive dead zone. We speculate that β skeletal fusimotorneurons might provide a more direct and faithful “efference copy” than could be generated centrally. This peripheral “efference copy” might more easily accommodate changes such as muscle fatigue and uncompensated activity from other muscles.

In conclusion, during quiet standing and related postural tasks, proprioception of small regulated movements has problems not normally considered when studying locomotion or perturbed balance. Short-range muscle stiffness reduces the transmission of joint rotations to the extrafusal fibers and high tendon compliance amplifies the fluctuations in muscle length related to homonymous activity. The operation of muscle spindles determines that fluctuating homonymous activity is the greatest factor in preventing registration of postural movements and in combination with short-range stiffness lead to a proprioceptive dead zone for small movements. Passive and unmodulated muscle, uncomplicated by fluctuations in muscle activity, enables a better register of postural joint rotations. It is predicted that a rigid α–γ coactivation could enable active agonist muscles to register postural rotations, but this mechanism would require and would explain the need for a large
number of spindles in active agonist muscle. This mechanism would produce spindle firing during standing that is unassociated with fluctuating muscle activity and this prediction is testable using microneurography.

APPENDIX

Relative proportions of active and passive components of contractile displacement during human standing

The purpose of this appendix is to use recent measurements of short-range muscle stiffness and tendon compliance during standing (Loram et al. 2005a, 2007a,b) to calculate expected ratio of actively modulated to passive changes in muscle length.

Generally, using Eq. 4, contractile displacement (changes in muscle length) consists of the sum of an active component \( L_{ce,a} \) and passive component \( L_{ce,p} \), where \( L_{ce,a} = -\alpha L_{0} \) and \( L_{ce,p} = M(\theta(r + 1)), \) where \( \theta \) is joint rotation and \( M \) is the moment arm

\[
L_{ce} = L_{ce,a} + L_{ce,p}
\]

(A1)

For quiet standing which involves balancing the human “inverted pendulum” through a compliant tendon linkage (the Achilles tendon), the equation in Laplace form relating contractile displacement \( \theta \) expressed in angular units to ankle joint rotation \( \theta \) (Loram et al. 2005a) is

\[
\theta(t) = \frac{\theta_0}{(1 + \tau_2 s + \tau_1 s)}/(c - 1) c / (c - 1) c / (c - 1) c / (c - 1) c / (c - 1) c / (c - 1)
\]

(A2)

Thus after rearranging, the ratio of active to passive modulation in contractile length is

\[
\frac{L_{ce,a}}{L_{ce,p}} = \frac{(r + 1)(1 + \tau_2 s + \tau_1 s)}/(c - 1) c / (c - 1) c / (c - 1) c / (c - 1) c / (c - 1) c / (c - 1)
\]

(A4)

Using representative values applicable during quiet standing of \( m = 70 \) kg, \( h = 0.92 \) cm, \( \theta = 77 \) kg·m² (Loram and Lakie 2002; Loram et al. 2006a), \( c = 0.85 \) (Loram et al. 2005a,b), and \( r = 10 \) (Loram et al. 2007b), where \( s = 2 \pi f \) and \( i = \gamma / (1 - 1) \), for sustained sinusoidal oscillation at frequency \( f \) in Hertz, the ratio becomes

\[
\frac{L_{ce,a}}{L_{ce,p}} = -62 \gamma^2 - 2.9
\]

(A5)

which at a typical mean sway frequency of \( 0.4 \) Hz (Loram et al. 2005b) gives a value of 13. Active shortening is one order of magnitude higher than passively transmitted elongation.

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