RAPID REPORT

Paradoxical muscle movement in human standing

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In human standing, gravity causes forward toppling about the ankle joint which is prevented by activity in the soleus and gastrocnemius muscles. It has long been assumed that when people sway forwards the calf muscles are stretched and conversely that they shorten with backward sway. Consequently, for many years, two explanations for standing stabilization have flourished. First, tonic muscle activity itself may generate adequate intrinsic ankle stiffness. Second, if intrinsic ankle stiffness is inadequate, the resistance to stretch of the calf muscles may be augmented by stretch reflexes or by central control. These explanations require that the passive tissue (Achilles’ tendon, foot) transmitting the calf muscle tension is stiff. However, our recent measurements have indicated that this passive tissue is not stiff during standing. Accordingly, we predicted a counterintuitive mode of control where the muscles and body must, on average, move in opposite directions (paradoxical movements). Here we use dynamic ultrasound imaging in vivo with novel automated tracking of muscle length to test our hypothesis. We show that soleus and gastrocnemius do indeed move paradoxically, shortening when the body sways forward and lengthening when the body returns. This confirms that intrinsic ankle stiffness is too low to stabilize human standing. Moreover, it shows that the increase in active tension is associated with muscle shortening. This pattern cannot be produced by muscle stretch reflexes and can only arise from the anticipatory neural control of muscle length that is necessary for balance.

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For standing humans, the whole body centre of mass is typically maintained at a variable but short distance in front of the ankle joints. Gravity acts on the body to topple the person forwards. Two muscles in the back of the calf, soleus and gastrocnemius, actively oppose the toppling effect of gravity. The soleus muscle originates from the back of the lower leg bones and is attached to the heel via the Achilles’ tendon. The gastrocnemius muscle originates from above the back of the knee and joins the Achilles’ tendon, which is common to both muscles (Fig. 1A and B). When they generate force these muscles rotate the body towards the upright.

Producing the right amount of force to balance and stabilize the body at any ankle angle is a complicated business (Loram et al. 2001; Loram & Lakie, 2002a,b; Fitzpatrick, 2003). The unknown neuromuscular mechanism that achieves this result remains a subject of considerable debate (Fitzpatrick et al. 1992, 1994, 1996; Winter et al. 1998, 2001; Morasso et al. 1999; Morasso & Schieppati, 1999; Gatev et al. 1999; Loram et al. 2001; Morasso & Sanguineti, 2002; Loram & Lakie, 2002a,b; Fitzpatrick, 2003; Lakie et al. 2003). Several explanations have been proposed for the required modulation in calf muscle torque (Gurfinkel et al. 1974; Houk & Rymer, 1981; Fitzpatrick et al. 1992, 1994, 1996; Horak & MacPherson, 1996; Winter et al. 1998; Schieppati & Nardone, 1999; Gatev et al. 1999; Winter et al. 2001) and these explanations all assume that as the body sways forward the tonically active calf muscles are stretched. If the resulting passive rise in tension is inadequate, stretch of muscle spindles (sensory structures in the muscle that register muscle length) can generate extra tension by spinal or central reflex pathways. These theories demand a sufficiently rigid link transmitting force from the muscle to the ground. This link is composed of the Achilles’ tendon, other series elastic tissue (aponeuroses) and the foot, which is not rigid.

The stiffness of the Achilles’ tendon and foot and their relevance to energy storage/release in locomotion has been
known for some time (Alexander & Bennet-Clark, 1977; Ker et al. 1987; Fukunaga et al. 2001), but the relevance of tendon and foot stiffness to the control of standing had not been established. When the calf muscles are active, as in standing, this creates a certain intrinsic stiffness at the ankle joint. Measurements of ankle stiffness have previously been made (Kearney & Hunter, 1982; Hof, 1998; Mirbagheri et al. 2000; de Zee & Voigt, 2001; Maganaris, 2002) but it is only recently that the intrinsic stiffness present during the act of standing has been measured (Loram & Lakie, 2002a). This measurement is difficult to perform and full of uncertainties. The stiffness depends markedly on the size of the perturbation used to measure it (Kearney & Hunter, 1982) and it is difficult to be sure what structures surrounding the ankle joint are contributing to its value. If large perturbations are used the conditions of normal standing become impossible to sustain and if small perturbations are used the stiffness is difficult to measure unambiguously (Loram & Lakie, 2002a).

Small differences in the value of intrinsic ankle stiffness have profound differences for the mode of control necessary to sustain balance. If the stiffness is less than a critical value defined by the load stiffness (Fitzpatrick et al. 1992) the passive stiffness created by tonic muscle activity cannot stabilize balance (Morasso & Schieppati, 1999; Morasso & Sanguineti, 2002; Loram & Lakie, 2002b) and the nervous system is compelled to adopt a repetitive anticipatory control process (Morasso et al. 1999; Loram & Lakie, 2002b; Lakie et al. 2003). More particularly and less well known, low ankle stiffness predicts that balance can only be maintained if on average the soleus and gastrocnemius shorten when the person sways forwards and lengthen when the person sways backwards (Lakie et al. 2003). Using very small perturbations, our recent measurements of intrinsic ankle stiffness during standing have indicated that the stiffness is 91 ± 23% (mean ± s.d.) of the critical value (Loram & Lakie, 2002a). The ankle stiffness is likely to be greatest for small perturbations (Kearney & Hunter, 1982). Thus the stiffness in standing is likely to be less than 91% because on average ankle rotations during standing are slightly larger than the small perturbations that we used. Consequently we proposed a hypothesis that paradoxical movements of the calf muscle are the norm during standing sway (Lakie et al. 2003).

An incontrovertible test of our prediction is to observe changes in muscle length as they occur while a subject sways through a variety of angles. Accordingly we used a dynamic ultrasound scanner (ATL, HDI 3000)

Figure 1. Anatomy of standing
A, the calf muscles soleus and gastrocnemius connect the Achilles’ tendon to the back of the lower leg bones and the back of the knee, respectively. B, schematic view showing AT (Achilles’ tendon as a spring), G (gastrocnemius medialis), S (soleus), A (proximal aponeurosis of gastrocnemius medialis), B (distal aponeurosis of gastrocnemius), C (distal aponeurosis of soleus) and D (proximal aponeurosis of soleus), C, actual sonograph of the same view. B and C are morphologically distinct but moved as a unit and were tracked using a single set of markers. The size of the image is 7 cm × 7 cm.

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to view movement of the left soleus and gastrocnemius muscles in vivo. This novel combination of non-invasive high resolution moving images and automated tracking of muscle length enables the relationship of body (or limb) movement and muscle movement to be precisely determined.

**Methods**

Subjects stood with neither foot in front of the other at their customary distance apart. Subjects were asked to slowly sway forwards and backwards and they were asked not to flex at the knee or hip. Visual inspection confirmed that these instructions were carried out. For this experiment ankle angle provides a sufficient estimate of body angle.

Three healthy, male subjects, aged between 34 and 49, were tested. Relative to their own gravitational toppling torque per unit angle, one had average intrinsic ankle stiffness (Loram & Lakie, 2002) (mean ± 0.1 s.d.), one had higher than average ankle stiffness (Loram & Lakie, 2002a) (mean ± 0.6 s.d.) and one had a tendon stiffness of twice the population mean (Maganaris & Paul, 2000) (mean ± 2 s.d.). 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.

Ankle angle was measured by a laser range finder that was mounted on the support surface and reflected off the left shin. Left ankle torque was measured using a purpose built foot-plate with a single axis of rotation orientated with the ankle and a vertically mounted strain gauge. Surface EMG (IEMG, $\tau = 100$ ms) were recorded from the left soleus and gastrocnemius medialis. Details of the EMG recording method have been published previously (Loram et al. 2001)

An ultrasound probe was fixed along the calf to provide a parasagittal-plane view of the underlying muscles (Fig. 1A and B). In the typical sonograph provided (Fig. 1C), white streaks identify strands of intramuscular collagen. Thus fibres of gastrocnemius medialis descend from their proximal aponeurosis (A) to their distal aponeurosis (B), which is continuous with the Achilles’ tendon. Fibres of soleus can also be seen descending from their proximal aponeurosis (D), which is rather faint, to their distal aponeurosis (C), which is also continuous with the Achilles’ tendon. When either muscle shortens, the distal and proximal aponeuroses move approximately antiparallel relative to each other, and the angle of the fibres become more obtuse relative to the aponeurosis. By tracking and calculating the relative movement between both proximal and distal aponeuroses, any relative motion between the scanner probe and the muscle was eliminated and an estimate was formed of changes in muscle length. Unlike the method of observing both ends of complete muscle fascicles (Herbert et al. 2002), this method does not measure changes in fascicle length, which would actually be greater because of the pennation angle of the fascicles.

The muscle length tracking procedure was as follows. On the sonograph, eight points were identified along (i) the proximal ends of the gastrocnemius, (ii) the central aponeurosis, and (iii) the proximal ends of the soleus fibres (Fig. 1C). A square of pixels (typically 11 × 11) was centred on each point. One frame in the middle of the typically 1500 frame series (25 frames $s^{-1}$) was used as a base frame. 2-D cross-correlation was used to find the square of pixels in each of the other frames that best corresponded with each square in the base frame. Visual inspection of the tracking points was used to confirm that the points stayed with the muscle as the image moved. A movie (.avi) file is provided online for the reader to download and inspect (see Supplementary material).

For each muscle the vector displacement between arbitrary pairs of proximal and distal markers was calculated. Changes in displacement relative to the base frame gave the changes in interaponeurosis distance in both the vertical and horizontal directions. The vertical changes were much bigger than the horizontal changes. The central aponeurosis is visually parallel to the proximal aponeurosis of soleus and is parallel to the proximal aponeurosis of gastrocnemius to within 10 deg. Thus the orientation of the central aponeurosis was used to define the direction of changes in muscle length as opposed to the relatively small, perpendicular changes in muscle thickness. For each marker pair, changes in muscle length were calculated along this direction. The changes in muscle length were then averaged across all eight marker pairs to provide a mean. For each frame, the 95% confidence intervals in muscle length change were typically ± 0.24 mm and ± 0.28 mm for soleus and gastrocnemius, respectively.

**Results**

Figure 2 shows six, slow, sways for the subject with average ankle stiffness. As the subject slowly sways forward through about 4 deg, the gastrocnemius and soleus muscles shorten by about 4 and 3 mm, respectively, contrary to naïve expectation. The muscle shortening is accompanied by clear increases in muscle activation in gastrocnemius and soleus and also by clear increases in ankle torque. As the subject sways back to the vertical, the muscles lengthen by the same amount and the activation of all muscles...
decreases. These counterintuitive, paradoxical movements are the main result of the present work.

Figure 3 indicates that the paradoxical muscle movements are the norm for standing people. These contrary changes in muscle length and sway angle were clearly demonstrated by the subject with average ankle stiffness (subject 1) and the subject with higher than average ankle stiffness (subject 2). The subject with exceptionally high Achilles’ tendon stiffness (subject 3) showed little correlation between muscle length and sway angle with a clear tendency to paradoxical (negative correlation) rather than orthodox movements.

**Discussion**

When the subject leans forward, the ankle torque required for balance is increased and tension in the series elastic tissue and muscle rises. The increased tension stretches the tendon but it does not stretch the muscle fibres which are actively contracting. The rise in tension would be inadequate were it not for the active shortening of the muscle (bias), which produces additional elongation of the tendon. Since the subject is remaining in balance (the sways are slow and controlled with little acceleration), the subject is shortening the muscles by just the right amount to oppose the gravitational torque at every angle. When tension rises, muscle shortens actively and tendon is passively lengthened.

Theory and experiment (Lakie et al. 2003) predict that the largest bias adjustments would be required by subjects with the lowest series elastic stiffness. Conversely, with unusually high series elastic stiffness the paradoxical muscle movements would not occur or might even become orthodox. This is confirmed by our measurements. With the stiffest subject the length of the muscle was weakly but negatively correlated with ankle angle (Fig. 3). For this subject (No. 3) the stiffness is close to the critical value and the series elastic tissue closely compensates the increment in gravitational torque with angle so that little change in the length of the muscle element is required. With the other subject (No. 2) who more closely approximated ‘normal’ stiffness the muscle movements were clearly paradoxical as

![Figure 2. Slow continuous sways of representative subject showing ankle angle (A), left ankle torque (B), muscle length (C) for gastrocnemius medialis (continuous line) and soleus (dotted line), and integrated EMG (D) for gastrocnemius medialis (continuous line) and soleus (dotted line)](image-url)

For all EMG, \( \tau = 100 \text{ ms} \) and the background noise signal is around 0.04 V. Muscle lengths are shown relative to typical mean muscle belly lengths of 320 and 220 mm, respectively, for soleus and gastrocnemius.
shown by the negative correlation between muscle length and ankle angle (Fig. 3). These results accord with the prediction that in standing paradoxical muscle movements are the norm.

The paradoxical muscle movements in standing are a consequence of the low stiffness of the series elastic tissue. This low stiffness leads to three inescapable conclusions. (i) Intrinsic ankle stiffness alone cannot stabilize the human body, (ii) muscle stretch reflexes cannot enhance the intrinsic ankle stiffness, and (iii) anticipatory control of muscle length is necessary to maintain balance.

The ankle joint stiffness is less than the stiffness of the weakest link in the series chain (muscle, aponeurosis, tendon, foot) according to the relationship:

\[
\frac{1}{K_{\text{total}}} = \frac{1}{K_{\text{muscle}}} + \frac{1}{K_{\text{series elastic tissue}}}
\]

where \( K \) stands for stiffness. Therefore the passive ankle stiffness created by tonic muscle contraction (Winter et al. 1998, 2001; Horak & MacPherson, 1996; Gurfinikel et al. 1974) is less than the series elastic stiffness and this passive ankle stiffness is here demonstrated to be too low to prevent the body toppling forwards. If this passive stiffness provides an increment of ankle torque with sway angle that is 90% of the gravitational increment in torque with angle (Loram & Lakie, 2002), then the net increment of torque with angle is only a tenth of what it would be without the passive stiffness. Thus while the passive tissue cannot of itself maintain balance it does provide partial compensation for the effect of gravity on the body. It allows the nervous system to maintain balance by quite subtle torque adjustments.

Rapid rotation of the ankle that occurs for example when one’s foot turns on uneven ground may well stretch the calf muscles and stimulate a stretch reflex in the calf muscles. Rapid rotations of the foot about the ankle which are engineered in the laboratory may produce a similar result. However, in standing, the situation is different: forward sway is accompanied by a controlled shortening of these muscles rather than a stretch. The muscle movement is in entirely the wrong direction to cause a stretch reflex; accordingly muscle stretch reflexes cannot enhance ankle stiffness in quiet standing. Central, feedforward control of ankle stiffness by anticipatorily enhancing the resistance to stretch of soleus and gastrocnemius (Gatev et al. 1999) has seemed plausible. However, ‘lengthening of an already activated gastrocnemius muscle in a spring-like manner to absorb body weight’ was not observed; rather, the already activated gastrocnemius muscle shortened to ‘absorb body weight’. Ultimately, even if the muscle were made entirely rigid, ankle stiffness would still be low, limited by the low stiffness of the tendon and foot.

As explained above, the compliant Achilles’ tendon and foot leave the body intrinsically unstable and without recourse to stretch reflexes for stabilization (Rack, 1985). Moreover, because of the compliant tendon, the muscle spindles which register muscle length do not know the angle of the ankle joint (Rack, 1985; Herbert et al. 2002). Thus information concerning ankle angle must be used to control muscle length rather than muscle length used as a feedback signal to control body position.

To maintain balance at any angle the nervous system has to adjust muscle length by just the right amount to produce the appropriate bias of the tendon and the correct torque. In particular, previous experiments have predicted that the muscle and body must on average move in opposite directions (Lakie et al. 2003). Consequently the nervous system has no option but to operate in an anticipatory mode (Loram & Lakie, 2002b; Lakie et al. 2003). This is in agreement with the findings of Gatev et al. (1999) that the gastrocnemius muscle is activated in advance of body position. However, our result shows that the anticipatory control is of muscle length rather than muscle stiffness. Perhaps internal models are represented in the cerebellum (Morasso et al. 1999). Measurement of the low intrinsic ankle stiffness in standing (Loram & Lakie, 2002a), analysis of the ballistic character of sways

![Figure 3. Effect of ankle stiffness](image-url)

The correlation between muscle length and ankle angle is shown as for three subjects. A negative correlation indicates that the muscle shortens as the ankle angle increases. The centre and edges of the box are the median and interquartile correlations, respectively. The whiskers show the range. The soleus and gastrocnemius muscles are both included. Subject 1 has average series elastic stiffness (tendon, aponeurosis, foot). Subject 2 has higher than average series elastic stiffness and subject 3 has very high Achilles’ tendon stiffness. Two trials were performed for subjects 1 and 2 and three trials for subject 3.
(Loram & Lakie, 2002b) and investigations of balance in an analogous task using a weak spring (Lakie et al. 2003) provide increasing evidence that intermittent, ballistic-like adjustments in muscle length (the ballistic bias hypothesis) (Loram & Lakie, 2002b; Lakie et al. 2003) may be responsible for the apparently random sway pattern that is seen in quiet standing. The observations presented here are consistent with our ballistic bias hypothesis. The hypothesis needs to be tested by examining the dynamic control of muscle length during the small sways of quiet standing.

Since the time of the influential Sherrington School (Creed et al. 1932) posture has been thought of as something that is essentially static and distinct from movement. In this view, postural maintenance rests on the intrinsic properties of the muscles supplemented by variable amounts of integrated reflex activity. This schema, which originated from experiments on quadrupeds, has become rather generally accepted as underlying human standing.

Our result suggests, at least for slow voluntary sways of the body, that the muscles have to work dynamically and in anticipation of movement. Such postural adjustments cannot be brought about by changing the drive to a simple negative feedback control system such as segmental reflexes. They can only result from a higher order predictive controller. If our result can be shown to extend to natural quiet standing then many of the ideas which have been used to explain standing will be shown to be invalid. The question of muscle activity in quiet standing is presently under investigation.

References


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Supplementary material

The online version of this paper can be found at: DOI: 10.1113/jphysiol.2004.062398

An avi file shows real time moving ultrasound images of the gastrocnemius and soleus muscle, the tracking markers, and angle of sway of the subject.

This material can also be found at http://www.blackwellpublishing.com/products/journals/suppmat/tjp/tjp261/tjp261sm.htm/