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Ankle Muscle Stiffness Alone Cannot Stabilize Balance During Quiet Standing

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Morasso, Pietro G., and Vittorio Sanguineti. Ankle muscle stiffness alone cannot stabilize balance during quiet standing. *J Neurophysiol* 88: 2157–2162, 2002; 10.1152/jn.00719.2001. This communication addresses again the hypothesis that the stabilization of balance during quiet standing is achieved by the stiffness of ankle muscles without anticipatory active control. It is shown that a recently proposed method of estimating ankle stiffness directly from the analysis of the posturographic data is incorrect because it ignores the modulation of motoneuronal activity and grossly overestimates the real range of values in relation with the critical value of stiffness. Moreover, a new simulation study with a realistic model of ankle muscles demonstrates the mechanical instability of the system when there is no anticipatory control input. However, the simulations also suggest that in normal subjects the active stiffness mechanisms of stabilization have similar weights in determining the restoring forces that are necessary for preventing the body from falling.

INTRODUCTION

The hypothesis that the stabilization of balance during quiet standing is achieved by the stiffness of ankle muscles was formulated by Winter et al. (1998) on the basis of two arguments: the experimental observation that the oscillation on the support surface of the center of mass (COM) appears to be in phase with the center of pressure (COP) and the theoretical consideration that such phase lock is incompatible with the afferent and efferent delays associated with active control. This hypothesis was challenged by Morasso and Schieppati (1999), who demonstrated, on the basis of a simple biomechanical analysis of the human inverted pendulum, that the phase relation is a consequence of the dynamics of the plant and is independent of the stabilization mechanism; therefore it cannot be used as an argument for deciding whether the stabilization mechanism is predominantly due to stiffness or to active control. Moreover, they pointed out that there is a critical value of stiffness for the stabilization of the ankle: $K_{\text{critical}} = m \cdot g \cdot h$, where m is the mass of the body, g is the acceleration of gravity, and h is the distance from the ankle of the body center of mass. Results in the literature were quoted that show a range of values of ankle stiffness that are significantly lower than the critical level. To meet the criticism, Winter et al. (2001)[1] proposed a new method for estimating the ankle joint stiffness that yielded a value 8.8% greater, on average, than the critical level. They also formulated the hypothesis that this result might be related to the high nonlinear stiffness of the series elastic element of ankle muscles.

The purpose of this communication is to show that both lines of defense of the stiffness control model are incorrect for the following reasons: the proposed method of stiffness estimation cannot distinguish the effects of stiffness compensation from active control and thus overestimates the real level of stiffness and, second, the series elastic element of ankle muscles cannot provide enough stiffness to stabilize the body during quiet standing. These claims are supported by a methodological analysis of the experimental approach and by a new simulation study with a realistic model of ankle muscles that shows the mechanical instability of the system without an anticipatory control input. The simulations also suggest that in normal subjects the two stabilizing mechanisms, active control and stiffness, contribute about equal amounts of the restoring forces necessary to prevent falling.

In general, Winter et al.'s most serious flaw is the assumption that the nervous system does not change the level of motoneuron activation during stance. On the contrary, there is clear evidence (e.g., the study by Gatev et al. 1999) that central activation does change during quiet stance because electromyographic (EMG) activity of ankle muscles is modulated in anticipation of postural sway. However, this argument is not sufficient per se to rule out the stiffness control hypothesis: it might well be that stiffness control is the main mechanism and active anticipatory modulation is only a secondary phenomenon. For this reason, the correct evaluation of ankle stiffness is crucial for deciding the nature of postural stabilization.

Evaluation of ankle joint stiffness

With reference to Fig. 1, which is reproduced with permission from the original paper, the authors used the following equations for computing the ankle moment M_a and the sway angle ϑ_{SW}, to estimate the ankle stiffness

$$M_a = R \cdot \text{COP} = mg \cdot \text{COP} \quad (1)$$

$$\theta_{sw} = \text{COM}/h \quad (2)$$

$$K_a = \text{d}M_a/\text{d}\theta_{sw} \quad (3)$$

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[1] For simplicity we shall refer to Winter at al. (2001) as "the authors."

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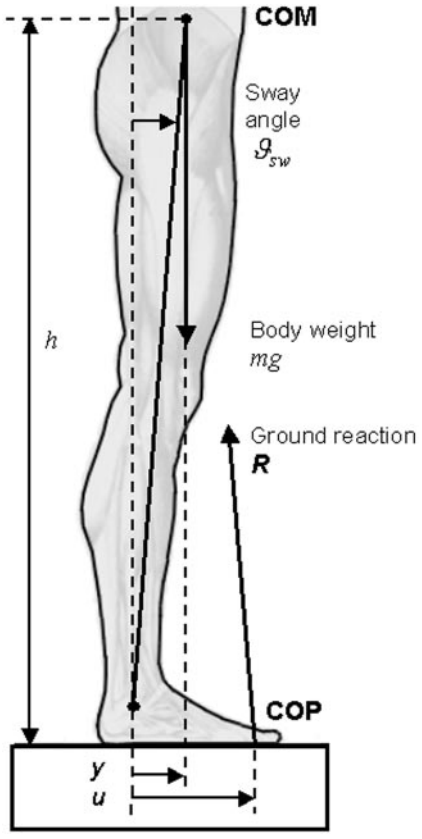

FIG. 1. Inverted pendulum model. It shows the relevant variables: the controlled variable (center of mass: COM); the control variable (center of pressure: COP); the body weight vector (mg); the ground reaction vector (*R*); the sway angle (ϑ_{SW}), which is proportional to the horizontal position of the COM for small oscillations; the height (*h*) of the COM. Reproduced, with permission, from Winter et al. (2001).

More specifically, COP and COM data were collected over a 10-s period of quiet standing and then, for each subject, the linear regression of M_a versus ϑ_{SW} was computed for the total set of samples, using the slope of the regression line as an estimate of K_a. By means of this analysis the authors could say that, on average, K_a is 8.8% greater than the critical value *mgh*.

We set aside the question of whether 8.8% is a sufficient margin for explaining the natural frequency of sway[2] and we focus on the main point: the measurement technique cannot render the true ankle stiffness but only the summed effect of stiffness compensation and active control. The flaw is subtle but critical. It is certainly true that the "stiffness" of a given body is the slope of the stress-strain characteristic curve, but the related measurements are valid if and only if the following two conditions are met: *1*) during the measurement the system operates in open-loop conditions, that is the only source of energy injected in the system is the known test disturbance and *2*) Stress and strain are measured in static or quasi-static conditions or time-dependent forces are specifically taken into account.[3]

During quiet standing, both conditions are violated for the following reasons. *1*) There is no reason to assume, a priori, that the level of activation of the motoneurons remains constant during the measurement time, which is not short but includes several postural oscillations. Thus changes in active muscle torque are not explicitly accounted for in the calculation of the total torque. *2*) The measured total ankle torque is inevitably "contaminated" by viscous and inertial components (which depend, respectively, on the velocity and acceleration of the sway angle) and which are not explicitly accounted for.

Given these conditions, the regression coefficient of the total ankle torque onto sway angle cannot be used to estimate ankle stiffness. In fact, the total ankle torque is the sum of three components: the torque generated by the "open loop stiffness," which is determined by the passive muscle properties and the muscle elasticity at the current activation level; the torque generated by the modulation of motoneuronal activity due to segmental reflexes, i.e., determined by "closed loop stiffness;" and the torque directly generated by descending motor commands in an anticipatory fashion. In principle, one might obtain the same torque-angle curve using only the third mechanism, i.e., with zero stiffness, and this means that the mere observation of the torque-angle curve does not say anything about the underlying ankle stiffness. The fact that the slope of the regression line is slightly greater than the critical stiffness simply says that the observed subjects were able to stand up against gravity by means of a suitable combination of the three mechanisms mentioned in the preceding text.

The problem is that measuring stiffness is a deceptively complex task. In an articulate review on the use and abuse of the notion of joint stiffness in biomechanics, Latash and Zatsiorsky (1993) emphasize the importance of elastic deformation and storage of potential elastic energy, determined by a well-controlled source of disturbance, when attempting a careful estimate of this parameter. In the case of the standing posture, the problem is complicated by the fact that there are two sources of potential energy: the energy due to gravity E_g and the energy due to the muscles E_m. Both variables are functions of the sway angle ϑ_{SW}, but E_g is a source of instability because it is bell-shaped (it has a point of maximum for $\vartheta_{SW} = 0$), whereas E_m is a source of stability because it is bowl-shaped. The total potential energy is the sum of the two, and it will be either bowl-shaped or bell-shaped depending on whether the ankle stiffness K_a is greater or smaller than the critical value $m \cdot g \cdot h$. In the former case, which is advocated by the authors, global stability is assured without any need of active intervention because the total potential energy has a point of minimum. In the latter case, the global system is unstable, and the only possibility for the nervous system is to dynamically stabilize it by means of anticipatory descending commands α, which have the effect of smoothly shifting the ankle angle ϑ_0 at which the elastic component of the ankle torque goes to zero. In particular, we can write the following

[2] In a spring-mass model, the following relation links the natural frequency ω_n, the moment of inertia *I*, and the total stiffness: $\omega_n^2 = K_{total}/I$. In the case of the standing posture $K_{total} = K_a - mgh$. For a value of the natural frequency of 0.5 Hz and a moment of inertia of 80 kg · m^2 we can see that the 8.8% margin of K_a with respect to the critical value *mgh* is largely insufficient.

[3] A priori one might think that during quiet stance the dynamic forces (inertial and viscous) are quite small and thus can be neglected. However, this is only partially true: such forces are small, but they have the same order of magnitude of the elastic forces due to the ankle stiffness. To check this point it is sufficient to consider the ankle dynamic equation: $M_a = I\ddot{\vartheta} + B\dot{\theta} + K_a\vartheta$. For example, at a frequency of 0.5 Hz the inertial, viscous and elastic terms are of the same order of magnitude, as it easy to calculate for realistic values of *I*, *B* and K_a (e.g., $I = 80$ kg·m^2, $K_a = 600$ N·m/rad and a damping factor of 0.3).

equation, which describes the dependence of this torque on the command α and the sway angle ϑ_{SW}[4]

$$-M_a(t) = B_a(\alpha)\dot{\vartheta}_{sw} + K_a(\alpha)[\vartheta_{sw} - \vartheta_0(\alpha)] \approx K_a(\alpha)[\vartheta_{sw} - \vartheta_0(\alpha)] \quad (4)$$

The equation clearly shows that if we compute the regression of M_a versus ϑ_{SW}, as the authors did, we are going to overestimate K_a because we should subtract from the measured values of the ankle torque the anticipatory active component, proportional to $\vartheta_0(\alpha)$. The point is that there is no reason, a prior, to neglect this component, since we know that the descending command α does change during sway.

The high correlation between ankle moment and sway angle can be explained by the dynamics of the inverted pendulum. In fact, it is remarkable that the regression is so good (the authors report a R^2 score of 0.918), and we should be able to account for it when rejecting the stiffness control model. To address this issue, we need to couple *Eq. 4,* which describes the relation between the ankle torque and the parameters of the ankle muscles, with the following equation which describes the dynamics of the body inverted pendulum

$$M_a = I\frac{d^2\vartheta_{sw}}{dt^2} - m \cdot g \cdot h \cdot \sin(\vartheta_{sw}) \quad (5)$$

where I is the moment of inertia of the human inverted pendulum, the ankle moment M_a is the control variable and the sway angle ϑ_{SW} is the controlled variable. The equation can be simplified by using the "small angle" approximation, i.e., $\sin(\theta) \approx \theta$, which is acceptable for small angles. As we demonstrated in the previous paper (Morasso and Schieppati 1999), the experimental observation that the control and controlled variables are phase-locked, although having different frequency bandwidths is a consequence of the intrinsic dynamics of the plant and not the nature of the motor controller. In particular, the phase relationship between M_a and ϑ_{SW} is independent of the value of the ankle stiffness. Consider now that the phase relationship between ϑ_{SW} and its second time derivative is, by definition, one of phase opposition, and thus the two terms on the right side of *Eq. 5* (with the small angle approximation) sum for each time instant and for each frequency component of the sway angle. The consequence is the following pair of results: in the population of data samples $\{M_a(t_k), \vartheta_{SW}(t_k); k = 1, 2, \ldots n\}$ the two variables appear to be linearly related and the regression coefficient is slightly greater than the critical value of stiffness $m \cdot g \cdot h$ by an amount that is proportional to the relative weight of inertial force versus gravity force.

The crucial point is that this has nothing to do with the value of the ankle stiffness but is fully explained by the dynamics of the inverted pendulum: in particular, the 8.8% excess of the regression coefficient with respect to the $m \cdot g \cdot h$ value does not measure the safety margin of the ankle stiffness but the relative weight of the inertia and gravity forces.

We wish to complement this discussion of the measurement of ankle stiffness by demonstrating that ankle stiffness can only account for about 60% of stabilization forces. For this purpose, we summarize, among the different studies that have addressed this problem, a set of experiments (Hunter and Kearney 1982; Weiss et al. 1988), which in our opinion are particularly relevant in this discussion on the stabilization of the standing posture. These studies cover the whole range of muscle activation up to maximum voluntary contraction for both plantarflexion and dorsiflexion. An actuator was used to generate pseudo-random joint perturbations, superimposed on a sustained bias torque from which it was possible to evaluate the dependence on such torque of the viscous and elastic components of the joint mechanical impedance. The main results can be summarized as follows. *1*) The ankle joint stiffness is linearly dependent on the level of bias torque for both dorsiflexion and plantarflexion. In the case of plantarflexion, which is more relevant in our case because sway movements occur around a slightly dorsiflexed posture (typically 3–4°), stiffness is well approximated by the following relation

$$K_a = 81.0 + 9.8 \cdot M_a \text{ Nm/rad} \quad (6)$$

2) The joint viscosity grows less linearly in such a way as to keep the damping parameter of the ankle joint fixed at a value of about 0.25.

With these data, we can evaluate the ankle stiffness around a typical standing posture and compare it with the critical stiffness. Let us consider a subject with a mass (m) of 80 kg, a distance of the COM from the ankle (h) of 1 m, and an average displacement of the COM (δ) of 5 cm forward with respect to the ankle. The average value of the total bias torque (for both ankles) is $Ma = m \cdot g \cdot \delta = 39.24$ Nm and the critical stiffness value is $K_{critical} = m \cdot g \cdot h = 784.5$ Nm/rad. From *Eq. 6* we obtain the following realistic value of total ankle stiffness in the standing posture

$$K_a = 2 \cdot (81.0 + 9.8 \cdot 39.24/2) = 546.5 \text{ Nm/rad} \quad (7)$$

This figure is considerably smaller than the critical value, and thus it rules out the possibility that the standing posture is maintained purely by stiffness stabilization. On the other hand, these data allow us to get at least a rough estimate of the synergistic action of stiffness compensation and anticipatory active control. In the data reported by the authors, the range of values of the ankle moment and the sway angle are as follows: $\Delta\vartheta_{SW} = 0.56°$, $\Delta M_a = 8.39$ Nm. Therefore from *Eq. 4,* which can be re-written as $\Delta M_a = K_a(\Delta\vartheta_{SW} - \Delta\vartheta_0)$ we can derive the following estimate of the range of variation of the equilibrium angle determined by the anticipatory modulation of the activity of ankle muscles

$$8.39 = 546.5 \cdot (0.56 - \Delta\vartheta_0) \cdot \pi/180 \Rightarrow |\Delta\vartheta_0| = 0.32^0$$

By multiplying the angular variations ($\Delta\vartheta_{SW}$ and $\Delta\vartheta_0$) by the ankle stiffness, we get the range of ankle torques due, respectively, to stiffness compensation and anticipatory active stabilization, and thus we can evaluate the relative weight of the active versus stiffness stabilization. It turns out that the credit for generating the postural stabilization forces is evenly shared between the elastic properties of muscle stiffness (about 60%) and the active mechanism that shifts the equilibrium point (about 40%).

Simulation study with a realistic muscle model

In their claim that muscle stiffness is sufficient to overcome the critical value necessary for stabilization, the authors also point to the high levels of stiffness of the series elastic element

[4] This is a linear approximation of a probably nonlinear relationship. It is acceptable because the range of motion is very small; moreover we can neglect the viscous component, as a first approximation.

of the plantarflexor muscles, evaluated by Winters and Stark (1988) among others. The problem is that the series elastic element is only one of the components of a realistic muscle model, although probably the main one responsible for the short-range stiffness. In general, it might be that in spite of the insufficient value of static stiffness, the complex nonlinear dynamics of the muscles, including the spinal reflexes, might provide additional stabilizing effects. However, this is a question which is difficult to answer analytically, due to the complexity of the system, and thus we carried out a simulation study (the details are in the master's thesis of M. Jacono[5]) with a realistic model of the muscles and segmental reactive mechanisms to check the intrinsic level of stability of the system in the absence of active control. The overall model consists of three parts: a muscle model, a stretch-reflex model, and an inverted pendulum model. The parameters of all the model elements are taken from the literature without any additional parameter adaptation.

Muscle model

The muscle model (extra-fusal fibers) is based on Winters (1995). It involves the following parts: a Hill-type contractile element (CE) controlled by the active state; a parallel element (PE), which models passive elasticity of connective tissue; and a series element (SE), which takes into account the instantaneous response of muscles, including tendons, to sudden load changes. The CE includes two components: a (nonlinear) elastic component characterized by a family of force-length curves and a (nonlinear) viscous component characterized by a force-velocity curve. SE and CE are modeled as nonlinear springs with exponential force-length curves. The model has two state-variables: the length of the SE and the level of the active state. The kinetics of the latter variable is modeled by a first-order dynamic equation, with a time-constant that depends on the level of activation and the activation/deactivation state of the muscle (Zahalak 1990).

Stretch reflex model

Intra-fusal fibers are modeled in the same way as extra-fusal fibers, except for the fact that they do not contribute to muscle force. Muscle spindle and Golgi tendon afferents are assumed to have a Gaussian response (Loeb 1984) with the peak in the middle of the physiological range. Spindle activation is a combination of a tonic and phasic component. Motor neuron activation is given by first-order linear dynamics, which introduces an additional state variable (neural excitation), with a descending input and a reflex input. The gains of the spindle and Golgi parts of the reflex input were calculated in such a way to take into account the constraint that each of the two signals contributes no more than 10% to the total neural excitation (Hogan 1984; Winters and Stark 1988). Spindle and Golgi afferences were affected by a 20-ms delay; the delay of the motor efferences was set to 30 ms.

[5] M. Jacono. *Modellamento dei muscoli scheletrici e dei riflessi spinali. Applicazione allo studio della stabilità della stazione eretta* (thesis for the electronic engineering degree). Genova, Italy: Faculty of Engineering, University of Genova, 2001. Thesis supervisors were V. Sanguineti and P. G. Morasso.

Global body model

The inverted pendulum model is based on *Eq. 5* (without the small-angle approximation). We assumed that the ankle is operated by a single dorsiflexor (tibialis anterior) and a single plantarflexor (soleus + gastrocnemius). Moment arms and physiological cross-section areas were determined from the literature (Dariush et al. 1998; Winter 1990; Yamaguchi et al. 1990).

Validation

First of all we tested the plausibility of the muscle + reflex model by examining how the viscous-elastic properties are affected by the presence/absence of the afferent signals. For this purpose, we considered the experiments by Lin and Rymer (1998) in which muscles, connected to an inertial load, were perturbed by force pulses. The results of these experiments show that if the reflex is present the mechanical response is dominated by elasticity, whereas if the reflex is absent the response is dominated by viscosity. The same qualitative behavior was duplicated in our model as regards timing and damping.

The second test, on a more general level, was related to the previously mentioned linear dependence of ankle stiffness on ankle moment, reported by Kearney and Hunter (1982) and Weiss et al. (1988). The global ankle + muscle + reflex model was stimulated with pseudo-random test disturbances similar to the experimental protocol and the qualitative results were quite similar (see Fig. 2).

We also examined the stabilizing effect of the short-range stiffness determined by the high stiffness value of the SE element. On this purpose, the ankle + muscle + reflex model was stimulated with test disturbances (truncated ramps) with different rise times and a fixed amplitude of 2 Nm. The resulting sway patterns were fitted with a linear spring-dash-pot-mass model. Figure 3 plots the estimated elastic coefficient (effective ankle stiffness) as a function of the rise time of the disturbance. The curve has a peak close to the origin, and then it settles to an asymptotic value that corresponds to the long-range stiffness. The initial peak, which is about twice the asymptotic value, corresponds to the short-range stiffness. What the figure demonstrates is that the extra-stiffness due to the series elastic element is only effective for very sharp postural disturbances, with durations less than 50 ms. However, physiological sway patterns are much slower and thus short-range stiffness is unlikely to have a significant stabilizing influence in the quiet upright posture.

Direct stability analysis

The goal of this analysis was to test whether or not the global model is able to maintain the upright posture in a stable way when the supraspinal command is kept constant with different baseline levels of muscle activation.[6] The global model is a nonlinear dynamical system of order 16 (with muscular, neural, and mechanical state variables). First we identified the equilibrium states of the system, i.e., the values of the state vector for which the corresponding vector of time derivatives goes to

[6] According to the data by Weiss et al. (1988), the bias torque in normal standing is $<20\%$ of maximum voluntary contraction.

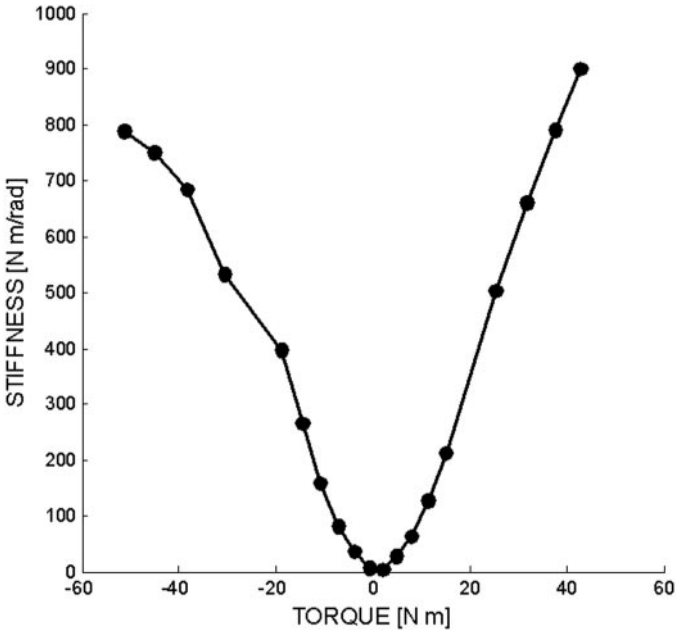

FIG. 2. Dependence of the ankle stiffness (ordinate: Nm/rad) on the bias torque (abscissa: Nm), estimated from the total simulation model by means of pseudo-random test disturbances. Such disturbances were applied to the model, and the input-output patterns were analyzed to estimate the stiffness coefficient. Dorsiflexor torque is positive; plantarfexor torque is negative. The range of values for the bias torque is up to the maximum voluntary contraction. Note that the dependence is approximately linear and is quite close to the experimental estimates reported by Hunter and Kearney (1982) and Weiss et al. (1988). For zero bias, the stiffness is not zero but has a value of about 14 Nm/rad, which is not visible at the larger scale factor. Note that in the case of the standing posture the bias torque, which is determined by the body weight and the horizontal displacement of the *COM*, is a small fraction of the maximum bias.

zero. The identified points correspond to totally unrealistic conditions with large values of the sway angle that bring the COM outside the support base and stretch the gastrocnemius beyond the physiological range. We also examined the behavior of the system in the neighborhood of the ideal standing posture ($\vartheta_{SW} = 0$, $\dot{\vartheta}_{SW} = 0$) by choosing different initial values of the mechanical state. Figure 4 plots in the phase plane (ϑ_{SW} vs. $\dot{\vartheta}_{SW}$) the local evolution of the mechanical state vector: this clearly shows that the ideal standing posture is not a point attractor but an unstable saddle point, and thus we can argue that the nonlinear dynamics and the stiff series element are not valid substitutes for the insufficient static stiffness.

DISCUSSION

In these concluding remarks, we address again the vexing question about the in-phase relationship between COP and COM and the argument made by the authors that this finding is incompatible with the afferent and efferent delays associated with active control. We wish to emphasize that this is a false argument because the zero lag is the straightforward consequence of the "biomechanical constraint" inherent in the structure of *Eq. 5.* Also, evidence that COP and COM are in phase in a model that includes time delays has been demonstrated by Peterka (2000). Therefore in a dynamically stabilized inverted pendulum the phase-lag will be zero whether K_a is supercritical, without any need of an active modulation of muscle activation, or is under-critical, thus requiring active muscle control. In the latter case, the muscle activation patterns must have an anticipatory nature, just enough to fulfill the biomechanical constraint and, at the same time, keep the COP slightly ahead of the COM to push it back to the dynamic equilibrium posture.

The support for the active stabilization of sway by anticipatory commands comes not only from a careful analysis of the ankle stiffness, which rules out super-critical levels, but also from other lines of evidence. The most direct one is the correlation between the COM and the EMG activity of the ankle muscles, which has been observed by Gatev et al. (1999): they found a significant cross-correlation between the rectified and integrated EMG of the lateral gastrocnemius and the anteroposterior motions of both the COP and the COM, with an anticipation of about 250 ms. Thus active control is perfectly compatible with the zero lag between COP and COM and requires a substantial amount of anticipatory sensorimotor processing.

Anticipatory active control is not in contrast with stiffness stabilization: on the contrary, they are synergistic mechanisms with a balanced sharing of the stabilization actions. At least, this is what appears from the data reported by the authors that only involve normal subjects.

On the other hand, the key for any successful anticipatory or feedforward control, to be carried out by a suitable internal model, is the availability of reliable sensory information to feed the predictive process and herein lies another line of evidence in support of active control, i.e., the clinical analysis of balance disorders. Without entering into the detailed comparison of the different pathological conditions, it is fair to say that in a variety of patients with smaller or greater modifica-

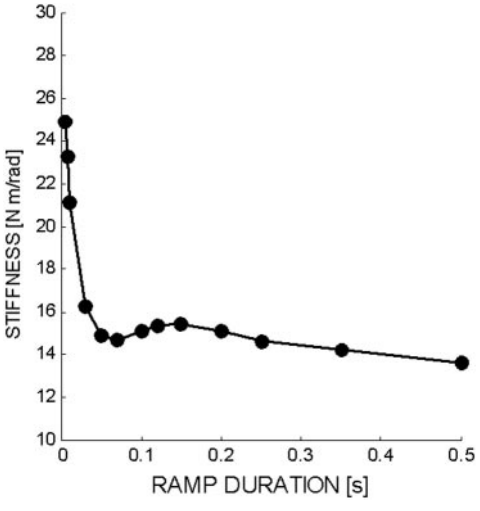

FIG. 3. Short- vs. long-range ankle stiffness estimated from the total simulation model by means of test disturbances (truncated ramps) with different rise times and a fixed amplitude of 2 Nm. Such disturbances were applied to the model and the input-output patterns were analyzed to estimate the stiffness coefficient. Abscissa: duration of the ramp (s); ordinate: estimated ankle stiffness (Nm/rad). For a ramp duration greater than about 50 ms, the estimated value is quite close to the long-range stiffness. For sharper disturbances the estimated coefficient is the short-range stiffness, related to the stiff series element. In these simulations, the bias torque was zero and thus the asymptotic value of the stiffness curve is quite close to the value plotted in Fig. 2 around the origin. Note that the twofold increase of ankle stiffness due to the stiff series elastic element is only effective for sharp disturbances with a rise time $<$50 ms.

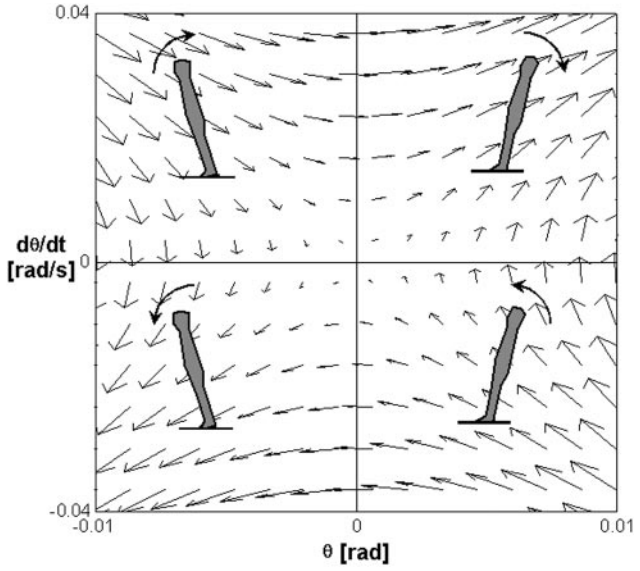

FIG. 4. Evolution in the phase-space (sway speed vs. sway angle) of the simulation model, in absence of anticipatory active control. Horizontal axis: sway angle (rad). Vertical axis: time derivative of the sway angle (rad/s). The supraspinal control variables of the ankle muscles are kept constant in such a way to provide physiological levels of the bias torque. The diagram is subdivided into 4 quadrants, which correspond to 4 different conditions. For example, the top right quadrant corresponds to backward-tilting postures (positive angle) and backward rotation velocities (positive velocity) as indicated by the sketched figure. The arrows in each quadrant show the evolution of the state vector, computed from the simulation model for different initial states. Note that in a stable system, with a point attractor, the vector field should be convergent toward the equilibrium configuration. The simulation model, without anticipatory control commands, is unable to stabilize the body, which falls forward or backward according to the different combinations of sway angle and sway speed.

tions of the posturographic patterns, including elderly subjects, the main problem is not a reduction of muscle force (and thus of muscle stiffness) but, rather, is a sensory deficit of one type or another. In other words, the reduced efficacy of predictive control, resulting from unreliable sensory information, is frequently compensated for by an increase of ankle stiffness via an exaggerated and energetically expensive coactivation of the ankle muscles. Thus in the stabilization of balance the exaggerated dependence on muscle stiffness seems to be a pathological sign not a physiological standard.

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