

# Can Muscle Stiffness Alone Stabilize Upright Standing?

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**Morasso, P. G. and M. Schieppati.** Can muscle stiffness alone stabilize upright standing? *J. Neurophysiol.* 83: 1622–1626, 1999. A stiffness control model for the stabilization of sway has been proposed recently. This paper discusses two inadequacies of the model: modeling and empiric consistency. First, we show that the in-phase relation between the trajectories of the center of pressure and the center of mass is determined by physics, not by control patterns. Second, we show that physiological values of stiffness of the ankle muscles are insufficient to stabilize the body “inverted pendulum.” The evidence of active mechanisms of sway stabilization is reviewed, pointing out the potentially crucial role of foot skin and muscle receptors.

## INTRODUCTION

Despite its apparent simplicity, the nature of the control mechanisms that allow humans to stand up is still an object of controversy. Visual, vestibular, proprioceptive, tactile, and muscular factors all contribute to the stabilization process. A model proposed by Winter et al. (1998) attributes muscle stiffness as the single factor involved in solving the problem. According to this theory, the intervention of the CNS is limited to the selection of an appropriate tonus for the muscles of the ankle joint, in order to establish an ankle stiffness that stabilizes an otherwise unstable mechanical system. Thus in this view the stabilization of quiet standing is a fundamentally passive process without any significant active or reactive component, except for the background setting of the stiffness parameters. In this paper we describe the flaws of the stiffness control model, review some of the relevant evidence in favor of an active intervention of the CNS in the stabilization process, and outline an alternative modeling framework.

### Misconceptions in the stiffness control model

The first misconception involves the relationships between the center of mass (COM) and the center of pressure (COP). The authors found that the oscillations of the two signals are in-phase and that there is a strong correlation between the acceleration of the COM and the COM-COP difference. However, as we show in the following section, such relationships are consequences of physical laws and cannot be used to prove one control theory over another. Moreover, the authors argue that the COM/COP correlation rules out the active/reactive control of balance because the delays in the sensory feedback would cause the COP to lag behind the COM, which is only partly correct. The main effect of the delay in the feedback loop is not the phase shift between the control and the con-

trolled variable but the global destabilization of the controlled system, which is intrinsically unstable. Therefore if we attribute functional importance to active mechanisms, because humans do succeed in stabilizing their balance, there must be something in the control circuitry that compensates for the original delays. Admittedly, this argument per se does not rule out the stiffness control model, simply cancels out one possible motivation for its “biological inevitability.” The phase-lock between COP and COM is a necessary consequence of physical laws, after the system has been stabilized.

The possibility of active/reactive control of balance is ruled out also from the sensory point of view, because vision is not functionally relevant and proprioceptive feedback signals are below physiological thresholds. However, the former observation is definitely contradicted by a large body of clinical evidence in adult and elderly subjects (e.g., Maki and McIlroy 1996; Paulus et al. 1984) and the latter fails to take into account the potential role of foot skin receptors (Kavounoudias et al. 1998; Wu and Chiang 1997). Thus the crucial question for assessing the feasibility of the stiffness control model is the following: are the muscles stiff enough to carry out the job of stabilizing the human inverted pendulum? To determine this we first outlined a theoretical framework of the system for expressing the conditions of stability and then evaluated the theoretical effectiveness of empiric stiffness levels.

### Theoretical framework

For an inverted pendulum (see Fig. 1) the system equation is

$$I_p \ddot{\theta} = mgh \sin(\theta) + \tau_{\text{ankle}} + z \quad (1)$$

where  $\theta$  is the sway angle,  $m$  and  $I_p$  are the mass and moment of inertia of the body (minus the feet),  $h$  is the distance of the COM from the ankle,  $g$  is the acceleration of gravity,  $\tau_{\text{ankle}}$  is the total ankle-torque, and  $z$  stands for the set of external or internal disturbances (such as respiration) that perturb the standing posture.<sup>1</sup> The ankle-torque must also satisfy an equilibrium equation for the foot:  $\tau_{\text{ankle}} + f_v u \approx 0$ , where  $f_v$  is the vertical component of the ground reaction force and  $u$  is the COP position. If we take into account that  $f_v \approx mg$  in quiet standing, then this equation tells us that variations of muscle torque are immediately and linearly translated into variations of the COP position. The two equations can then be combined

<sup>1</sup> The assumption of additive noise in the posture control model might seem artificial to the readers. However, there is experimental support for the role of noise in upright standing (Dijkstra et al. 1994; Paulus et al. 1984). Dijkstra et al. (1994) also provide arguments in favor of a contribution of visual input to stance.

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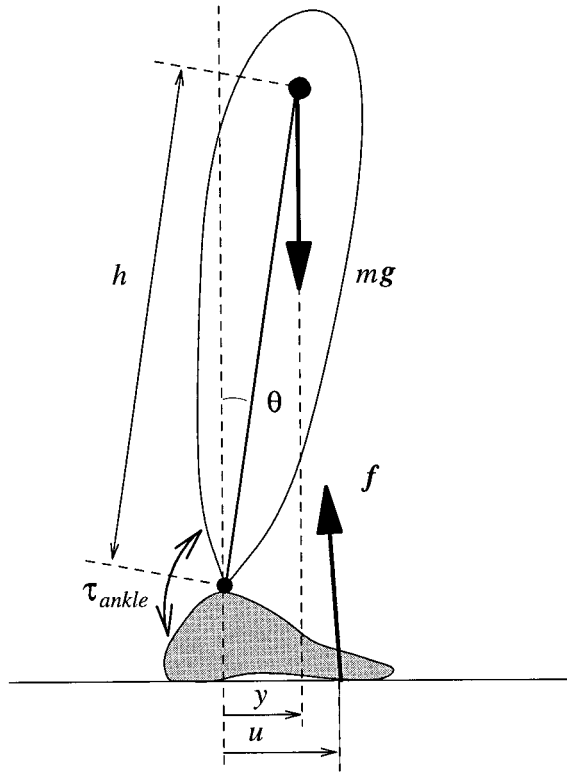


FIG. 1. Scheme of the human “inverted pendulum” for sway in the sagittal plane. Center of mass (COM), position is denoted by the variable  $y$ ; center of pressure (COP), position is denoted by the variable  $u$ ; ground reaction force, ( $f$ ); sway angle, ( $\theta$ ); acceleration of gravity, ( $g$ ); torque of the muscles around the ankle, ( $\tau_{ankle}$ ); and distance of the COM from the ankle, ( $h$ ).

into a single dynamic sway equation that relates the controlled variable  $y$  and the control variable  $u$ .<sup>2</sup>

$$\ddot{y} = \frac{g}{h_e} (y - u) + z' \tag{2}$$

In other words, the COM-COP difference is bound to be approximately proportional to the acceleration of the COM for purely mechanical reasons, because the postural noise  $z'$  is small in quiet standing. Moreover, the same equation tells us that for the horizontal component of the ground reaction force it must be  $f_H \propto (y - u)$  since  $f_H = m\ddot{y}$  according to Newton’s law. Therefore although no specific receptors exist that detect the COM, its position  $y$  can be indirectly estimated through measurements of  $u$  and  $f_H$  and some computational process that “fuses” them.

Let us now consider the phase relation implied by Eq. 2 during normal sway, using a simple method that can be applied to the different harmonic components of both oscillations. Let  $A_y, \Phi_y$  and  $A_u, \Phi_u$  be the amplitude and phase parameters of  $y$  and  $u$ , respectively. Then  $\Phi_y = \Phi_{y-u}$  is always 180° out of phase relative to  $\Phi_y$  which implies that  $\Phi_u = \Phi_y$  (and  $A_u >$

<sup>2</sup> Because the body sway angle is small, we can use the following approximations:  $\sin(\theta) \approx \theta$ ,  $\cos(\theta) \approx 1$ ,  $\dot{\theta} \approx \dot{y}/h$ . The moment of inertia can be expressed in general as  $I_p = mh^2k_s$  where  $k_s$  is a shape factor:  $k_s = 1$  if we assume that the body mass is concentrated in the COM and  $k_s = 1.33$  if it is uniformly distributed along a rod-like shape; for the human body the value of the coefficient is closer to the latter than the former estimate. We define an “effective” value of  $h$ :  $h_e = hk_s$ . Eq. 2 also includes the “noise” term  $z' = z/mh_e$ .

$A_u$ ). Again, this phase relationship is a physical necessity, not something that must be proved experimentally to discriminate the specific control action. Therefore the observation of null phase delay neither proves nor disproves the theory of stiffness control. Equation 2 also suggests an alternative way of calculating the COM from the COP with respect to the brute-force approach used by the authors that is based on a complex whole body model and the 3-D measurements of 21 markers: it is sufficient indeed to integrate Eq. 2 considering  $u(t)$  as the forcing input. Morasso and Spada (unpublished observations) developed an algorithm based on a variational approach and spline functions<sup>3</sup> (Fig. 2).

Stability

For analyzing conditions of stability of the postural control system, we consider the open loop transfer function (Fig. 3A) that is derived from the Laplace transform of Eq. 2:  $s^2Y(s) = g/h_e[Y(s) - U(s)] + Z'(s)$ . From this we get

$$Y(s) = \frac{1}{s^2 - g/h_e} [Z'(s) - g/h_e U(s)] \tag{3}$$

This clearly shows that the system is unstable because one of the roots of the denominator is positive. It is easy to demonstrate that the system can be stabilized by a simple PD (proportional + derivative) feedback linear controller, characterized by proportional and derivative gain factors:  $u = K_p y + K_d \dot{y}$ , whose Laplace transform is

$$U(s) = (K_p + sK_d)Y(s) \tag{4}$$

The closed loop transfer function (see Fig. 3B) becomes

$$Y(s) = \frac{1}{s^2 + sg/h_e K_d + g/h_e (K_p - 1)} Z'(s) \tag{5}$$

If the proportional gain of the controller is big enough ( $K_p > 1$ ) the system is then characterized by two complex conjugate roots with negative real part and this means that the response to small postural disturbances consists of damped oscillations. In particular, the natural frequency  $f_n$  and the damping coefficient  $\zeta$  can be computed as follows:  $f_n = 1/2\pi\sqrt{g/h_e(K_p - 1)}$ ;  $\zeta = K_d/2\sqrt{g/h_e(K_p - 1)}$ . Various estimates of both parameters are available in the literature (e.g. Gagey et al. 1980) in any case  $f_n$  is below 1 Hz and  $\zeta$  is rather small. For example, if we choose  $f_n = 0.5$  Hz and  $\zeta = 0.2$ , we get  $K_p \approx 2$  and  $K_d \approx 0.13$ . We performed a simple simulation experiment with a PD controller set according to such parameters and a persistent “noise” source  $z(t)$  (a combination of white noise and quasi-periodic spiky noise) that keeps the system away from its natural equilibrium (Fig. 2). In qualitative terms, the simulation shows that a simple linear controller is good enough to reproduce the morphology of the COM-COP relationship, although it does not rule out more complex nonlinear effects. Interpretation of the control law is as follows. 1)  $K_p > 1$  means that the COP position  $u$  must be driven to stay “ahead” of the corresponding COM position  $y$ ; 2)  $K_d > 0$  means that the anticipa-

<sup>3</sup> The solution  $y(t)$  of the equation, over a given observation time, is approximated by means of a B-spline function  $B(t)$ , which depends linearly on a set of parameters  $p$  as well as its second time derivative  $\ddot{B}(t)$ . Substituting the corresponding expressions into Eq. 2 we get a LSE (least square estimate) problem in  $p$  that can be solved with standard methods.

tion of the COP must be greater if the COM is falling away from the equilibrium and smaller if it is speeding back. It is important to note that this control mechanism is neutral with respect to the passive versus active alternative because the viscous-elastic properties can be approximately modeled by Eq. 4 and the PD feedback controller. The biological solution may be based on a mixture of the two modalities. We are mainly concerned with the question of estimating whether muscle stiffness alone is sufficient to solve the problem.

#### Stiffness control: stabilization by passive muscle properties

In the stiffness control paradigm, the two parameters of the control law can be associated with the elastic and damping coefficients of the ankle joint impedance, respectively. In particular, it is easy to show, by means of a change of coordinates from  $y$  to  $\theta$ , that the elastic parameter  $K_p$  is linked to the measurable ankle stiffness  $K_a$  by the following relation

$$K_a = mgh + I_p(K_p - 1)g/h_e \quad (6)$$

The limit case for stability ( $K_p = 1$ ) allows us to compute the minimum value of ankle stiffness:  $K_a = mgh$  ( $= 785 \text{ Nm/rad}$  for  $m = 80 \text{ Kg}$  and  $h = 1\text{m}$ ). The necessary value must be greater than that to fit the observed natural frequency. For example, with  $f_n = 0.5 \text{ Hz}$ ,  $m = 80 \text{ Kg}$ ,  $I_p = 107 \text{ Kg} \cdot \text{m}^2$ ,  $k_s = 1$ , we get the following estimate:

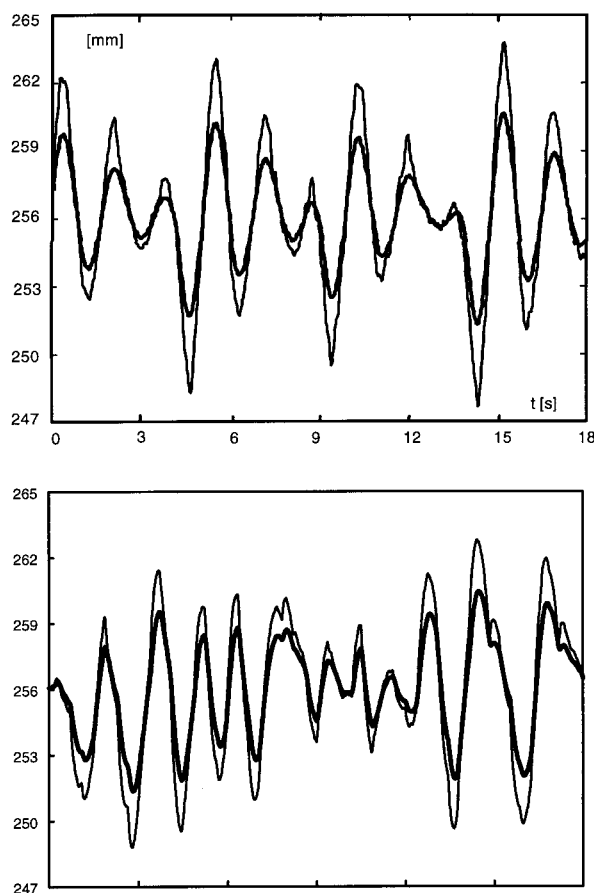


FIG. 2. *Top*: measured trajectory of the COP (—) and reconstructed trajectory of the COM (---). *Bottom*: simulated COM and COP trajectories with the control law  $u(t) = 2y + 0.13\dot{y}$  and a persistent disturbance source (white noise + impulsive quasi-periodic noise).

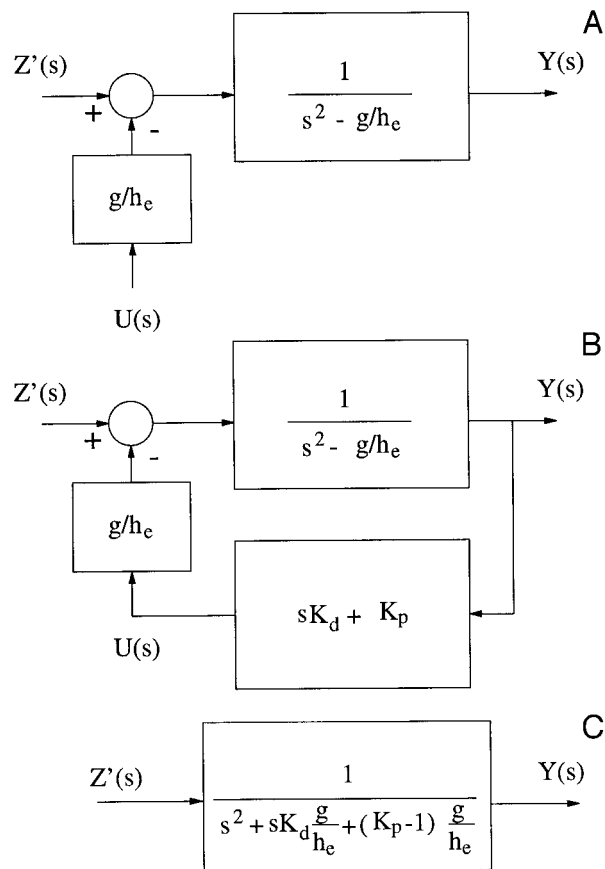


FIG. 3. *A*: block diagram of the open-loop postural system. *B*: block diagram of the closed loop postural control system. *C*: noise-to-sway transfer function of the closed loop control.  $U(s)$ ,  $Y(s)$ , and  $Z'(s)$  identify the Laplace transform of the COP signal, COM signal, and postural noise signal, respectively.

$$K_a = 785 + 1050 = 1835 \text{ Nm/rad} \quad (7)$$

Is this value compatible, at least as an order of magnitude, with available joint stiffness measurements and known muscle properties? Joint stiffness values have been estimated by Flash and Gurevich (1997) for the shoulder joint to vary in the range 45–90 Nm/rad for various levels of bias torque. The ankle stiffness is bigger because the total cross-sectional area of the muscles acting around the ankle is larger than that around the shoulder. Direct measurements of ankle stiffness by means of a special kind of ergometer have been performed by Hof (1998) showing a range of 250–400 Nm/rad, with a large bias torque (100 Nm), confirming previous estimates (Blanpied and Smidt 1991; Weiss et al. 1988). A similar conclusion can be obtained if we start with available estimates of muscle stiffness  $K_m$  (Winters and Stark 1988), transform them into the corresponding ankle stiffness according to the equation  $K_a = K_m \cdot r^2$  ( $r$  is the moment-arm of the muscle), and add up the stiffness parameters of all the ankle muscles. In any case, even using the most favorable estimates, we are still far away from the required value displayed in Eq. 6.<sup>4</sup> Therefore the available evi-

<sup>4</sup> The two elements of the “apparent” ankle stiffness  $K_a$  in Eq. 6 vary with the fourth power of the body size  $h$  since  $m$  goes with  $h^3$  and  $I_p$  goes with  $mh^2$ . Muscle strength and stiffness go with the cross-sectional area of the muscles and thus vary with  $h^2$  but the ankle stiffness due to the muscles goes with  $h^4$  since  $K_a = K_m r^2$ , as noted above. Thus as body size varies both the apparent



dence suggests that muscle stiffness alone<sup>5</sup> is insufficient to stabilize body sway, i.e., the parameters of the control law that are compatible with the observed natural frequency and damping are likely to be determined mainly by an active mechanism of stabilization that cannot have a reflex nature due to the intrinsic delays in the reflex pathways and the low-pass characteristics of muscle. However, it must be noted that actual estimates of ankle stiffness “during standing” are not yet available and this is a major deficiency in our current level of understanding of posture control, calling for the development of appropriate experimental approaches.

#### *Active mechanisms of stabilization*

The supporters of the passive stabilization of sway point out that the limited range of sway movements may not stimulate the different kinds of sway receptors beyond physiological thresholds. The experimental evidence, however, is somehow contradictory but certainly indicates that physiological levels of sway are very close to such thresholds in relation to vestibular, joint, and muscle receptors (see e.g., Fitzpatrick and McCloskey 1993; Konradson et al. 1993). Regarding muscle receptors, functional loss of group Ia spindle afferent fibers has been considered responsible for postural disequilibrium (Griffin et al. 1990; Weiss and White 1986). Spindle group II fibers, of smaller diameter but at least as numerous as the group Ia fibers, may be perhaps even more relevant as the origin of information utilized by postural control circuits. Both leg and foot muscles are the site of postural segmental reflexes (Schieppati et al. 1995), mainly because of spindle group II fibers (Schieppati and Nardone 1997). Where very slow movements of the body occur, such as during maintenance of quiet stance, it is conceivable that length signals coming from the less adaptable spindle secondaries provide an appropriate input to the CNS for detecting low-frequency displacements occurring mainly about the ankle (Gurfinkel et al. 1995) and for assisting foot and calf muscle reflex responses (Schieppati et al. 1995). The contribution provided by the plantar cutaneous receptors is frequently overlooked, with the exception of a few studies (Kavounoudias et al. 1998; Magnusson et al. 1990). These receptors do not measure sway but are related to different parameters of the ground reaction force  $f$  that are affected indirectly by the sway movements: 1) the vertical component  $f_v$ , 2) the horizontal component or shear force  $f_H$ , and 3) the point of application or *COP* position  $u$ . In principle,  $f_v$  can be derived by adding up the output of the receptors specifically affected by the vertical component of the contact forces. This parameter is not relevant for sway control because it is approximately constant during quiet sway. However, the *COP* position  $u$  can be computed by using the same receptor information but with a different computational process, which takes into account the position of the receptors as well as the intensity of the detected signals. This is a complex computational task that integrates information of a number of sensory channels with appropriate transduction characteristics, like the Ruffini and

ankle stiffness and the stiffness due to the muscles change in the same way and we can conclude that the calculation scheme is roughly independent of the body size.

<sup>5</sup> Please note that with this term we denote the intrinsic mechanical stiffness as well as the tonic part of the reflex stiffness because the mentioned experiments of stiffness estimate are in fact sensitive to the cumulative effect of the two elements.

Meissner terminations that are slowly or moderately fast adapting and have small receptive fields.

Shear force  $f_H$  is frequently ignored because it is small when compared with the weight force; however, it is in the Newton-range according to Eq. 2 and so is detectable by the plantar receptors as well as the foot muscle spindles. Moreover, because  $f_H$  is proportional to the COM-COP difference, it carries clear sway-relevant information; in the *A/P* direction, for example, if the force is directed forward it means that the COP is behind the COM and conversely if the force is directed backward. Therefore such shear contact forces have a dominant phasic component in contrast with the basically tonic nature of the vertical forces. This means that the appropriate receptors must be very fast adapting; moreover, there is no need of small receptive fields because localization in this case is not required. Pacinian corpuscles fit this description, although other receptors in the foot muscles might play a role as well.

Regardless of how the CNS processes this information, the indirect evidence that active mechanisms of stabilization underlie sway control is ample and multifaceted. Certainly information about mutual positions of body links, muscular torques, and interaction with the support has access to the CNS, and subjects can consciously evaluate the gross amplitude of their own sway during stance (Schieppati et al. 1999), possibly with respect to a reference position (Gurfinkel et al. 1995). Furthermore, the available data do not favor a constant level of antigravity leg and foot muscle activity during stance. Instead a relationship between the anteroposterior oscillations of the center of pressure and the profile of the rectified and integrated EMG of those muscles (Schieppati et al. 1994) indicates moment-to-moment action of a system of stance control based on timely produced muscle impulses.

In conclusion, having excluded a dominant effect of muscle stiffness, a plausible alternative computational scheme can be outlined that is based on the indirect estimate of a *postural state vector*  $x = [y, \dot{y}]^T$  obtained from the complex combination of a variety of sway-related sensory signals. Use of sensory signals in a feedback control mechanism that modulates the activity of the calf muscles is shown in Fig. 3B. The critical element of the scheme is the potentially catastrophic influence of even small delays in the feedback loop. (In the simulation model shown in Fig. 2 a delay of about 50 ms is sufficient to destabilize the control system.) Restabilization rules out the reflexive nature of the control mechanism and strongly suggests a central computational process that carries out two main functions: 1) integrating the multisensory information into a unifying estimate of the state vector and 2) compensating the transmission delays with an anticipatory action, i.e., a short-time prediction of the postural time series. From this point of view, the absence of short-latency reflexes of the muscles around the ankle, linked to the stimulation of cutaneous fibers of the foot (Abbruzzese et al. 1996), is paradoxically in favor of a supra-segmental role of such afferences in a more complex computational mechanism such as the one outlined previously. Detailed analysis of this point is beyond the scope of this paper, however, information on research in this area of motor control and sensory adaptation is available and is focused on the acquisition of “internal models” (Miall et al. 1993; Morasso and Sanguineti 1997; Morasso et al. 1999; Wolpert and Kawato 1998).

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