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Non-Asymptotical Postural Stabilization Strategy during Human Quiet Stance

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Abstract— **Postural sway, i.e., Center of Pressure and Center of Mass (CoM) and electromyograms (EMGs) from ankle muscles were measured during human quiet stance. It was confirmed that EMG activity of ankle extensors correlated with the CoM and CoM velocity, as reported earlier. However, the EMG activity was phasic and burst-like, compared to slow and smooth changes in CoM. It was demonstrated that the phasic EMG activity tended to appear only at specific periods mostly when the acceleration of CoM was close to zero and to terminate when CoM velocity changed its sign from positive to negative. We claimed that a simulated human stance model with a conventional PID feedback controller could not account for such phasic EMG activity. The phasic EMG activity was then associated with the active ankle torque, which is defined as the residual component of the stiffness torque in the total ankle torque. To this end, the active torque was estimated by assuming that the ankle stiffness was not large enough to make the upright posture asymptotically stable. The active torque and the phasic EMG activity were sharply correlated, implying that the phasic EMG activity might generate the active torque, and it might be produced based on a strategy other than the conventional PID feedback control.**

I. Introduction

HUMAN quiet stance control, which is maintained mostly by the ankle torque, is usually identified with a simple control problem similar to stabilization of inverted pendulum. A well known stiffness control hypothesis claims that the human central nervous system (CNS) tonically increases intensities of neural commands to the ankle muscles, and consequently the ankle joint impedance, in order to make upright posture asymptotically stable [1], [2]. This theory, however, requires a physiologically implausible large amount of neural noise to account for complicated postural sway during human standing [3]. The mechanism how the CNS solves this control problem is still under debate. In this study, we tried to show that a CNS strategy for maintaining human upright posture might not be a conventional PID control and discuss an alternative strategy.

II. A Model of Quiet Stance and Ankle Torque

The ankle joint torque, referred to as T, stabilizes the body during quiet stance. The torque T includes active and passive components. The passive component corresponds to the stiffness torque determined by muscle tonus and the stiffness of the joint tissues. Morasso and Schieppati [4] discussed that the stabilization of quiet stance by the passive stiffness torque alone would be far possible, and the active component is required to maintain quiet stance. The active torque component is produced by the CNS based on the feedback information on body kinematics [1], [5]. In this paper, the human body was modeled as a single link inverted pendulum that rotates about the ankle joint in the sagittal plane (Fig. 1). The equations of motion of the pendulum model are as follows:

$$I\ddot{\theta} = mgh\theta + T \qquad (1)$$

where θ is the sway angle, I is the moment of inertia of the body, m the body mass, h the distance from the ankle to the center of mass (CoM) of the body. The ankle torque during quiet stance may be represented approximately as

$$T \simeq f_v u \qquad (2)$$

where f_v is the vertical component of the total ground reaction force applied at the center of pressure (CoP) located at u representing the anterior-posterior (AP) displacement of CoP with respect to the ankle position projected on the ground.

Morasso *et al.* [3] discussed that the ankle torque may be decomposed into the following four components:

$$T = T_{ref} + T_{stiffness} + T_{active} + T_{noise} \qquad (3)$$

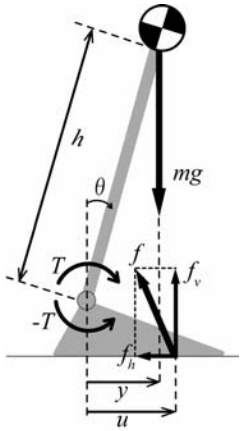

Fig. 1. An inverted pendulum model of human stance in the sagittal plane. y is the center of mass (CoM) position, u is the center of pressure (CoP) position, with respect to the ankle. f is the ground reaction force applied at CoP with f_h and f_v components. θ is sway angle, g is the gravity constant. T is the total ankle torque, and h is the distance from ankle to CoM.

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where $T_{ref} = mgh\theta_{ref}$ and $T_{stiffness} = P(\theta_{ref} - \theta)$ with θ_{ref} being the reference posture that could be determined in the CNS, and *P* the joint stiffness producing the passive torque. T_{noise} is the torque noise. In this study, we assumed $\theta_{ref} = 0$ and $P = p \cdot mgh$ where *p* is a positive constant. The upright stance is asymptotically stable if *p*>1 and unstable otherwise, since the stiffness torque with *p*>1 could compensate the gravity torque. Note that $T_{stiffness}$ should depend on muscle tonus, which means that it depends on the tonic component of the neural command controlling the muscle contraction. The active component, referred to as T_{active}, could thus be obtained as;

$$T_{active} = T - T_{stiffness} = T + P\theta = T + p \cdot mgh\theta \quad (4)$$

The purpose of this paper is to examine, qualitatively at least, whether or not this active component is determined based on the conventional PID-like strategy. If not, as Morasso *et al.* [3] proposed that the active component could be ballistic, basic characteristics of the active component would be described.

III. Method

A. CoP, CoM, and EMGs

The CoP during quiet stance with eyes open was measured for 65 sec using a force platform (AMTI, CA) from healthy young subjects. All subjects gave their informed consent to participate. Electromyograms (EMGs) were recorded by surface electrodes using a differential amplifier (bandwidth 5 Hz to 200 Hz). The electrodes were placed on muscles of the right tibialis anterior (TA), medial gastrocnemius (MG) and soleus (SO) with an interelectrode distance of 20 mm. A 12-bit A/D converter with 1-kHz sampling frequency was used. Measured EMGs were numerically rectified. Both force platform and rectified EMG data were low-pass filtered using the fourth-ordered zero-phase-lag Butterworth filter with the cutoff frequency 12 Hz. The rectified and smoothed EMGs, referred to as IEMGs, were considered to represent the level of muscle activity during quiet stance. The AP displacement of CoP with respect to the ankle position was defined as the filtered CoP, and referred to as *u*. The AP displacement of CoM, referred to as *y*, was estimated using the following filter [3]:

$$Y(j\omega) = \frac{\frac{g}{h\xi}}{\omega^2 + \frac{g}{h\xi}} U(j\omega)$$

Since the CoM sway was small, the sway angle of the body could be well approximated as $\theta = \arcsin(y) \simeq y$.

B. Model simulation with PID controller

A simple postural control model using PID feedback controller was simulated to compare its dynamics with the measured human postural sway. For the model simulation, we set $\theta_{ref} = 0$ so that $T_{ref} = mgh\theta_{ref} = 0$. *p*=0.7 was used for the stiffness torque. A small ankle joint viscosity torque $-D\dot{\theta}$ (*D*=10 Nm · sec/rad) was also introduced. The active torque was generated using a conventional PID controller with a feedback transmission delay $\tau = 100$ ms. The gains were set as $P_d = 200$ Nm/rad , $I_d = 5$ Nm/rad/sec , and $D_d = 200$ Nm · sec/rad . It was confirmed that smaller differential gains for the active torque could lead to instability due to the delay, suggesting that velocity information is important for the postural stability with PID control [6].

C. Timing of phasic EMG activation

CoM, CoM velocity which was obtained by the numerical differentiation, and IEMG waveforms were plotted together to see if one could identify conditions on the CoM and CoM velocity when the EMGs were phasically active, i.e., they were apparently distinguished from the tonic activity. The body sway kinematics were also plotted on a quasi-phase plane spanned by CoM and CoM velocity. The identified time intervals were then localized in the plane to represent the conditions on the CoM and CoM velocity for the phasic muscle activity. The cross correlation functions between CoM and IEMGs were calculated to quantify the time lags between the signals [6].

D. Deriving Active Torque and its correlation with IEMGs

The total ankle torque was obtained using Eq. (2) with the time series of *u* and f_v. The active torque was then estimated using Eq. (4). Although *p*=0.7 was used for the model simulation [7], we used *p*=0.98 for the estimation of stiffness

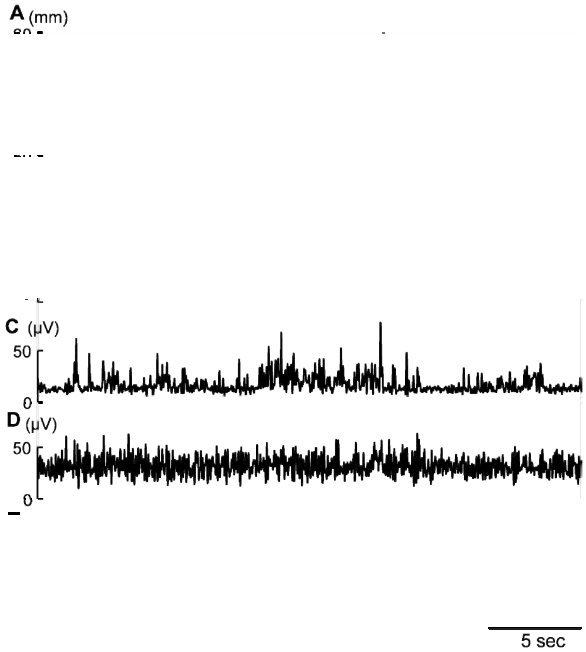

Fig. 2. Examples of measured time series during quiet standing. A: CoP (thin curve) and CoM (thick curve) in anterior-posterior (AP) direction with respect to the ankle position. B: CoM velocity. C: Smoothed Electricmyogram (IEMG) of medial gastrocnemius (MG). D: IEMG of soleus (SO). E: IEMG of tibialis anterior (TA).

torque $T_{stiffness}$. The use of larger value of *p* than that of used in the simulation reflects the large proportional gain ($P_d = 200$ Nm/rad) used for the delayed feedback control in the simulation. The cross correlation function between the estimated active torque (with its sign inversed) and the IEMGs was calculated. In order to show that the EMG phasic activity represents the active torque rather than the total ankle torque, the cross correlation function between the total ankle torque (with its sign inversed) and the IEMGs was also calculated.

IV. Result

A. CoM and EMGs

Fig. 2 exemplifies the measured time series during quiet standing. Those include CoP, *u*, CoM, *y* in AP direction with respect to the ankle position, CoM velocity, and IEMGs of MG, SO and TA. The cross correlation analyses between CoM and IEMGs of MG and SO, but not for CoM and IEMG of TA, confirmed the previous work published in [6] (figures not shown). That is, the ankle extensor activities (MG and SO) were well correlated with the CoM changes. The CoM changes were 100-150 ms behind the ankle extensor activities. The cross correlation between CoM velocity and IEMGs of MG and SO showed peak correlation close to zero time lag or even small positive time lag, which means that the CoM velocity was slightly ahead the EMG activity, was also confirmed (figures not shown). This observation was consistent with a role played by the high gain derivative (velocity) gain in the PID controller with the feedback transmission delay [6].

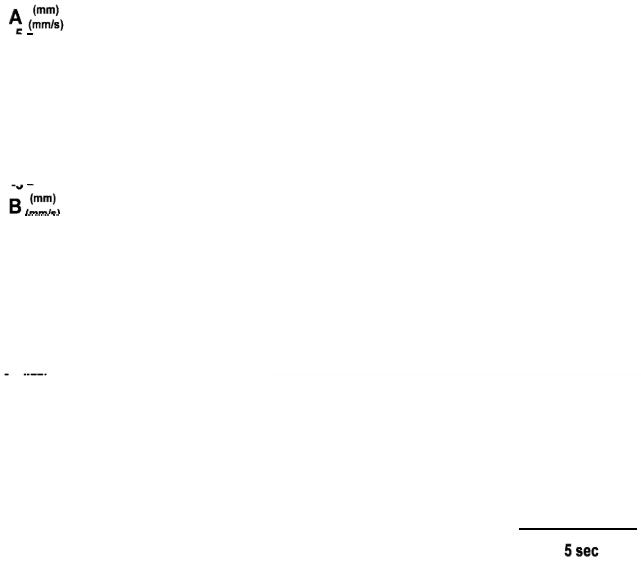

Fig. 3. A, B: Typical examples of measured time series during quiet standing in two subjects. CoM with respect to its mean value (thick line), CoM velocity (dotted line) in AP direction and IEMG of MG (thin line). Red portions represent phasic IEMG activity. C: A typical example of the model's behavior. Sway angle (thick line), sway velocity (dotted line) and active torque (thin red line).

B. Timing of phasic EMG activation

In Fig. 3, CoM, CoM velocity, and IEMG waveforms were superposed for an experimental time series. Sway angle, sway velocity and the active torque obtained from the PID model simulation are also plotted together. For the simulated data, it was natural that the active torque exhibited smooth curve and it was well correlated with the sway angle and sway velocity with time lags. That is, the active torque changed in accordance always with changes (both small and large, and positive and negative directions) in the sway angle and sway velocity. For the experimental data, phasic EMG activities, in particular for MG muscle activities, were not necessarily correlated with the CoM and CoM velocity. This could be confirmed both in Figs. 3 A and B. For example, the phasic EMG activity numbered as #1 at the beginning of Fig. 2A coincided with a large positive CoM velocity and with largely anterior positioning of CoM, which seemed to be consistent with the output signal of a PD controller. However, the phasic EMG activity was absent for the subsequent period despite that the CoM was largely deviated from its mean value and the CoM velocity was also largely negative. Such situations could be better confirmed by Fig. 4, in which the CoM and CoM velocity were plotted as a trajectory on the quasi-phase plane and several portions of the trajectory that were numbered in Fig. 3 with the phasic MG activities were marked by the thick curve segments. From this plot, one could observe that the phasic MG activities triggered when the acceleration of CoM became close to zero (i.e., when the CoM velocity became close to its peak) and terminated when the velocity changed its sign from positive to negative. However, there were some exceptions. For example, even the aforementioned conditions on the CoM acceleration and the sign of CoM velocity were satisfied, the phasic MG activity tended not to appear if the CoM was at near side of the ankle

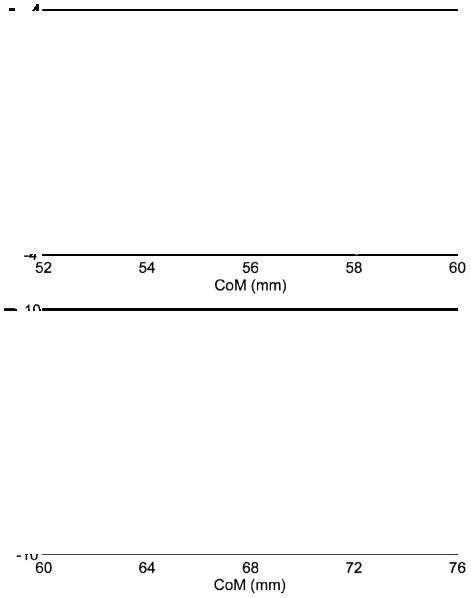

Fig. 4. A, B: CoM dynamics on the quasi-phase plane spanned by CoM and CoM velocity from two different subjects. Thick red portions represent segments of the trajectory in which the phasic IEMG activity of MG appeared. Each numbers besides the red portion corresponds to the number on phasic IEMG activity shown in Figs. 3A and B.

(leftward side of Figs. 4A and B), i.e., if the forward tilt angle of the body was small. Also, if the CoM was far from the upright, the phasic MG activities sustained for a second regardless the value of CoM acceleration and the sign of CoM velocity. As such, the appearance of the phasic MG activity was mostly conditional on the body sway acceleration and velocity sign, but there were some exceptions. This means that there might not be rigid CoM/CoM velocity thresholds for triggering of the phase EMG activity. A command torque generated by a conventional PID controller could not show these characteristics, implying that the neural commands controlling the phase EMG activities might not be determined by the conventional PID-like strategy.

C. Phasic EMG activity and active torque

Fig. 5A shows the estimated total ankle torque and the passive torque (stiffness torque). Since the stiffness torque was proportional to the CoM by its definition, it was smoother than the ankle torque. It cab be seen that the stiffness torque occupied relatively large ratio of the total ankle torque. Difference between the total ankle torque and the stiffness torque was the active torque (Eq. 4). In Fig. 5B, the estimated active torque with its sign inversed was plotted with the IEMG of MG to show that their waveforms coincided well qualitatively, not only for the periods with the phasic EMG activities but also for the remaining periods with the EMG activities of small amplitudes variations. This correlation was evaluated by the cross correlation function between the active torque with its sign inversed and the IMG of MG. and it was shown in Fig. 5C (thick curve). For comparison, the cross correlation between the total ankle torque with its sign inversed and the IMG of MG was also calculated (Fig. 5C thin curve). The correlation peak at a negative time lag about 100-150 ms was observed in the cross correlation function for both the active torque and the total ankle torque. However, the peak for the active torque was significantly sharper than that for the total ankle torque, implying that the phasic EMG activity of MG generated the active torque. The positive cross correlation peak with the wide width for the total ankle torque might be due to slow and smooth changes in the stiffness torque which was proportional the smooth and slow sway of CoM.

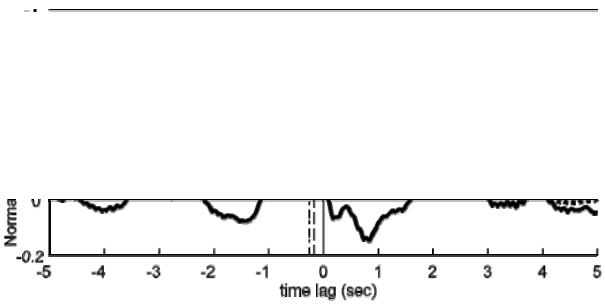

Fig. 5. A: The total ankle torque (thick line) and the estimated passive torque, i.e., stiffness torque (dotted line). Difference between these two was defined as the active torque. B: The estimated active torque with its sign inversed (thick gray line) and the IEMG of MG. C: The cross correlation function between the estimated active torque with its sign inversed and the IEMG of MG (thick curve), and between the total ankle torque with its sign inversed and the IEMG of MG (thin curve).

V. Discussion

The result shown here suggested that the following mechanism of postural control during quiet stance. Dominant degree of the ankle torque might be produced by the stiffness of the ankle joint to which the muscle tonus contributes by some amount, but the stiffness was not large enough to asymptotically stabilize the upright posture [4],[7]. The missing torque might be compensated by the active neural interventions producing the active torque to maintain the state point of the inverted pendulum, i.e., the point on the CoM-CoM velocity phase plane, around the reference posture, although its magnitude is not as large as the stiffness torque.

The phasic EMG tended to appear at specific periods (timings) mostly when the acceleration of CoM was close to zero and to terminate when CoM velocity changed its sign from positive to negative, but conditional upon the CoM position. Further study is required to elucidate how the CNS determines those timings.

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