Upright, correlated random walks: A statistical-biomechanics approach to the human postural control system

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The task of maintaining erect stance involves a complex sensorimotor control system, the output of which can be highly irregular. Even when a healthy individual attempts to stand still, the center of gravity of his or her body and the center of pressure (COP) under his or her feet continually move about in an erratic fashion. In this study, we approach the problem of characterizing postural sway from the perspective of random-walk theory. Specifically, we analyze COP trajectories as one-dimensional and two-dimensional random walks. These analyses reveal that over short-term intervals of time during undisturbed stance the COP behaves as a positively correlated random walk, whereas over long-term intervals of time it resembles a negatively correlated random walk. We interpret this novel finding as an indication that during quiet standing the postural control system utilizes open-loop and closed-loop control schemes over short-term and long-term intervals, respectively. From this perspective, our approach, known as stabilogram-diffusion analysis, has the advantage that it leads to the extraction of COP parameters which can be directly related to the steady-state behavior and functional interaction of the neuromuscular mechanisms underlying the maintenance of erect stance. © *1995 American Institute of Physics.*

I. INTRODUCTION

Upright stance is regulated by a complex control system that involves a number of different sensory systems, i.e., the visual, vestibular, and somatosensory systems. The net output of this control system can be highly irregular. For example, during quiet standing, the center of pressure (COP) under an individual's feet continually moves about in an erratic fashion. A plot of the time-varying coordinates of the COP is known as a stabilogram (Fig. 1). For several decades, researchers have studied the human postural control system by using force platforms to measure quiet-standing COP trajectories (Fig. 1). To date, however, the motor control insights gained from static posturography have been meager. This is due largely to the fact that, in most cases,[1–4] the analyses of the posturographic data have been limited to summary statistics, e.g., sway path length, average radial area, etc., which, in general, cannot be interpreted in a physiologically meaningful way. This situation is confounded by the fact that the COP is a measure of whole-body dynamics, and thereby represents the summed effect of a number of different neuromusculoskeletal components acting at a number of different joints. As a consequence of these factors, the utility of static posturography in the laboratory and clinic has been severely limited. Thus, there is a clear need to develop a reliable approach for extracting physiologically meaningful information from stabilograms.

We approach the problem of characterizing COP trajectories from a different perspective, namely, that of random-walk theory. In particular, we hypothesize that the output of the human postural control system under quiet-standing conditions can be modeled as a system of bounded, correlated random walks. Our approach, which is called *stabilogram-diffusion analysis*, is based on the assumption that the postural control system involves both deterministic and stochastic components. One of our aims for this work is to develop a modeling framework that can be used to formulate and test hypotheses concerning the relative contributions of different sensorimotor subsystems and strategies to "quasistatic" postural control. Accordingly, in the present paper, we show that stabilogram-diffusion analysis leads to a series of COP parameters that can be directly related to the resultant steady-state behavior and functional interaction of the neuromuscular mechanisms underlying the maintenance of undisturbed stance. In this paper we review some of the main findings of our earlier studies.[5,6] A more complete description of stabilogram-diffusion analysis and our interpretation of its associated parameters can be found in Collins and De Luca.[5]

II. METHODS

A. Experimental methods

Ten healthy subjects—five males and five females—of similar age (19–24 yr, mean: 22±2 yr), height 1.60–1.80 m, mean: 1.69±0.08 m), and body weight (54.5–77.1 kg, mean: 64.3±8.4 kg) were included in the study. The subjects had no evidence or known history of a gait, postural, or skeletal disorder. Informed consent was obtained for each subject prior to participation, after the nature and possible consequences of the studies were explained. Postural sway was evaluated by using a Kistler 9287 multicomponent force platform and signal conditioner to collect COP trajectories under a subject's feet. Each subject was instructed to stand in an upright posture in a standardized stance on the platform. In the standardized stance, the subject's feet were abducted 10° and their heels were separated mediolaterally by a distance of 6 cm. During the testing, the subjects stood barefoot with their arms comfortably at their sides and their eyes open and fixed on a point in front of them. A series of five 90 s

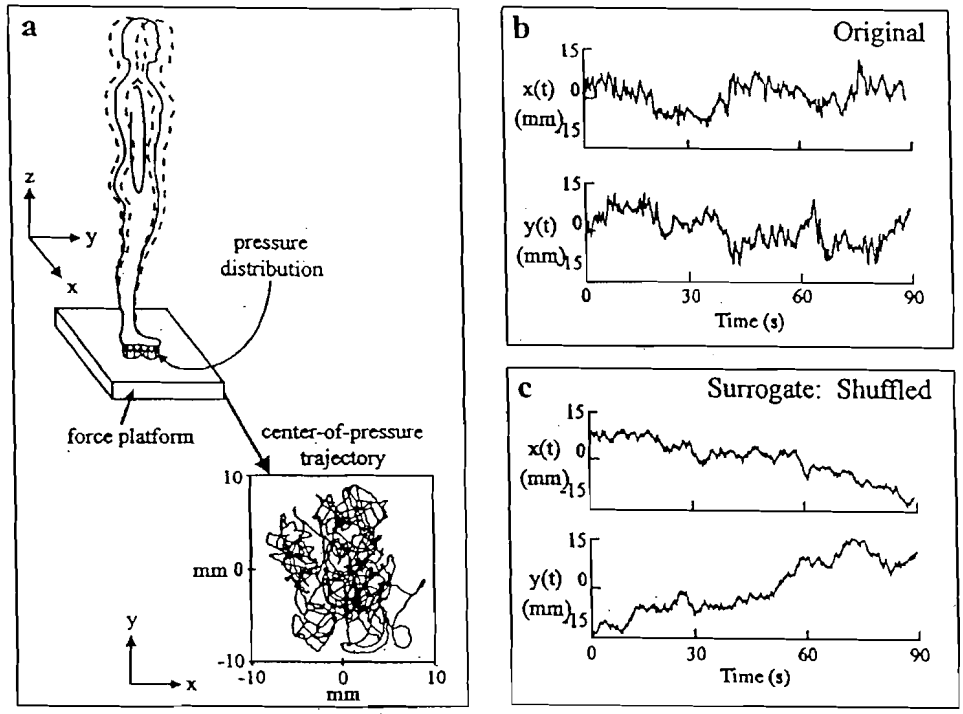

FIG. 1. (a) A schematic diagram of the experimental setup for examining quiet-standing postural stability. Shown also is a typical 90 s stabilogram for a healthy young individual. (b) The x-coordinate and y-coordinate time series corresponding to the stabilogram in (a). (c) Shuffled surrogate random-walk data sets that were generated from the original COP time series in (b).

trials was conducted for each subject. Rest periods of 2 min were provided between each trial. The COP signals were anti-aliased using a second-order lowpass filter (with a cutoff frequency of 34.1 Hz), and subsequently sampled at a rate of 100 Hz. The time series were then downsampled to a rate of 50 Hz, and the mean was subtracted from each time series to give an average COP position of (0,0).

B. Data analysis: Random-walk approach

The COP trajectories were studied as one-dimensional and two-dimensional random walks. In a classical random walk or, more generally, ordinary Brownian motion, past increments in displacement are uncorrelated with future increments, i.e., the system has no memory. In such cases, the mean square displacement of a random walker is linearly related to the time interval Δt by the general expression[7]

$$\langle \Delta j^2 \rangle = 2D_j \, \Delta t, \qquad (1)$$

where $\langle \Delta j^2 \rangle$ is mean square displacement, D_j is the diffusion coefficient, and $j=x,y,r$. (In the present paper, x, y, and r denote mediolateral, anteroposterior, and planar displacements and measures, respectively.) In words, the diffusion coefficient is an average measure of the stochastic activity of a random walker, i.e., it is directly related to its jump frequency and/or amplitude.

In a correlated random walk or, more generally, fractional Brownian motion,[8] past increments in displacement are correlated with future increments, i.e., the system has memory. In such cases, the relation given by Eq. (1) is generalized to the following scaling law:

$$\langle \Delta j^2 \rangle \sim \Delta t^{2H_j}, \qquad (2)$$

where H_j is the scaling exponent, which can be any real number in the range $0<H_j<1$. Scaling exponents quantify the correlation between the step increments making up the trajectory of a random walker. This is best illustrated by considering the correlation function C for fractional Brownian motion, which is given by the expression[9]

$$C=2(2^{2H_j-1}-1).$$

Note that for $H_j=0.5$, the increments in displacement are statistically independent, i.e., $C=0$. This is the result expected for classical Brownian motion. For $H_j>0.5$, past and future increments are positively correlated, i.e., $C>0$. In this case, a random walker moving in a particular direction for some t_0 will tend to continue in the same direction for $t>t_0$. In general, an increasing (decreasing) trend in the past implies an increasing (decreasing) trend in the future. This type of behavior is known as *persistence*.[9,10] For $H_j<0.5$, on the other hand, the stochastic process is negatively correlated, i.e., $C<0$. In this case, increasing (decreasing) trends in the past imply on the average decreasing (increasing) trends in the future. This type of behavior is referred to as *anti-persistence*.[9,10]

In order to calculate diffusion coefficients and scaling exponents from COP trajectories, we first generated plots of

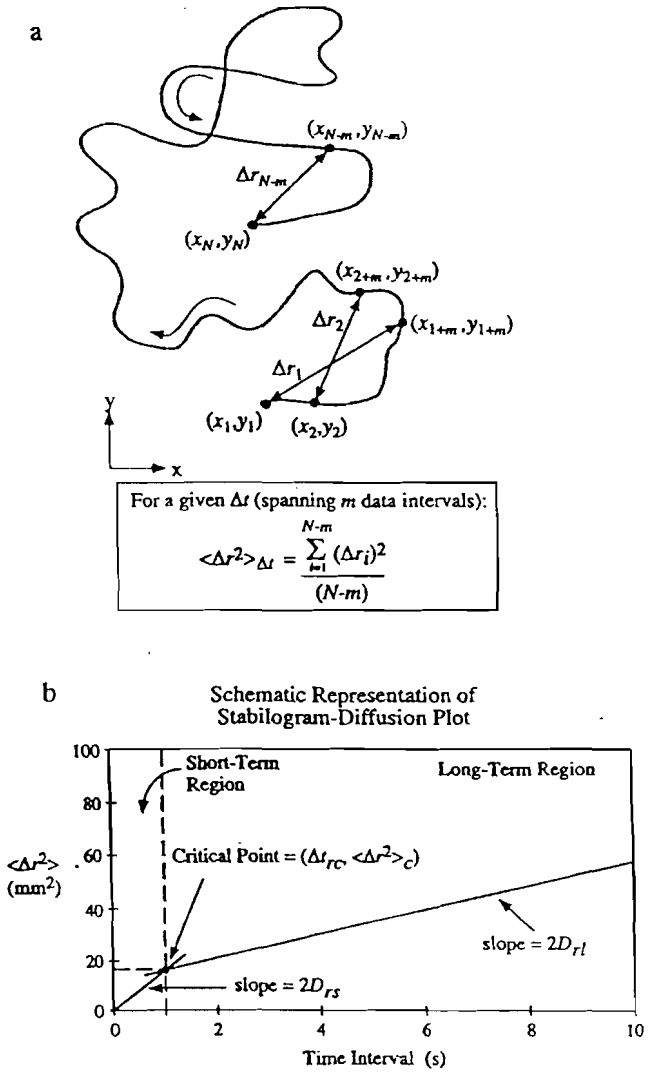

FIG. 2. (a) Diagram showing the method for calculating mean square planar displacement $\langle \Delta r^2 \rangle$ as a function of time interval Δt for a COP trajectory made up of N data points $(x_1, y_1; x_2, y_2; \ldots; x_N, y_N)$. In this case, Δt does not represent the sampling interval; instead, Δt represents a moving time window spanning m data intervals. (b) A schematic representation of a typical resultant planar stabilogram-diffusion plot ($\langle \Delta r^2 \rangle$ vs Δt) generated from COP time series according to the method shown in (a). The diffusion coefficients D_{rs} and D_{rl} are computed from the slopes of the lines fitted to the short-term and long-term regions, respectively. The critical point, $(\Delta t_{rc}, \langle \Delta r^2 \rangle_c)$, is defined by the intersection of the lines fitted to the two regions of the plot. The scaling exponents H_{rs} and H_{rl} are calculated from the slopes of the log–log plots of the short-term and long-term regions, respectively (adapted from Collins and De Luca[5]).

mean square COP displacement versus Δt. (Such plots will be referred to as stabilogram-diffusion plots.) The displacement analysis was carried out by calculating the square of the displacements between all pairs of points separated in time by a specified time interval Δt [Fig. 2(a)]. The square displacements were then averaged over the number of Δt making up each COP time series. This process was repeated for increasing values of Δt.

Experimental studies concerned with diffusion-like processes typically analyze either long time series of data measurements or a large number of smaller time series of such measurements.[11,12] In a posturographic investigation, it would be impractical, however, to have subjects stand on a force platform for extended periods of time. Physiological factors such as fatigue would tend to obscure the results. In the present study, it was therefore decided to collect a reasonable number of 90 s trials for each subject and analyze averaged sets of the results derived from these tests. Specifically, stabilogram-diffusion plots were computed for each subject trial, and then five such curves were averaged to obtain a resultant stabilogram-diffusion plot for a particular subject.

Diffusion coefficients [Eq. (1)] were calculated from the slopes of the resultant linear–linear plots of mean square COP displacement versus Δt. Similarly, scaling exponents [Eq. (2)] were computed from the resultant log–log plots of such curves. In all cases, the slopes were determined by utilizing the method of least squares to fit straight lines through defined portions of the aforementioned plots. All parameters were determined by a single investigator.

III. RESULTS

Resultant linear–linear and log–log plots of mean square planar COP displacement versus Δt for a representative subject are shown in Figs. 3(a) and 3(b), respectively. It should be noted that the stabilogram-diffusion curves changed slope after a critical point (or period) at some small Δt. This general feature was found in the resultant plots for all ten subjects who participated in this study. In order to parametrize such plots, two regions were identified—a short-term region and a long-term region [Fig. 2(b)]. These regions were separated by a transition period over which the slope of the stabilogram-diffusion plot changed considerably. (A third, distinct region over which mean square COP displacement saturates to some constant value is also expected after a sufficiently large time interval, given the fact that COP displacements are bounded by the base of support defined by an individual's feet, i.e., for bounded motion, $\langle \Delta j^2 \rangle$ saturates to a constant value after a sufficiently large Δt.[13] Unfortunately, the time series considered in this study were not long enough to characterize this region reliably.) Diffusion coefficients and scaling exponents were computed for each region [Fig. 2(b)].[14] Subscripts s and l will be used throughout the manuscript to denote the short-term and long-term regions, respectively. (The lines fitted for computation of D_{js}, D_{jl}, H_{js}, and H_{jl} had r^2 values that ranged from 0.91 to 1.00, 0.70–1.00, 0.97–1.00, and 0.74 to 1.00, respectively; the r^2 values for most data sets were all typically greater than 0.97.) An estimate for each critical point (or period) was determined as the intersection point of the regression lines fitted to the two regions of the linear–linear plots of $\langle \Delta j^2 \rangle$ vs Δt [Fig. 2(b)].[15]

The group means and standard deviations of the diffusion coefficients, scaling exponents and critical point coordinates for the ten subjects are given in bar-plot form in Fig. 4. Several general points should be noted. First, the short-term diffusion coefficients were considerably greater than the respective long-term diffusion coefficients [Fig. 4(a)]. In addition, the anteroposterior diffusion coefficients were larger than the respective mediolateral diffusion coefficients [Fig. 4(a)]. (This latter result was not unexpected, given that

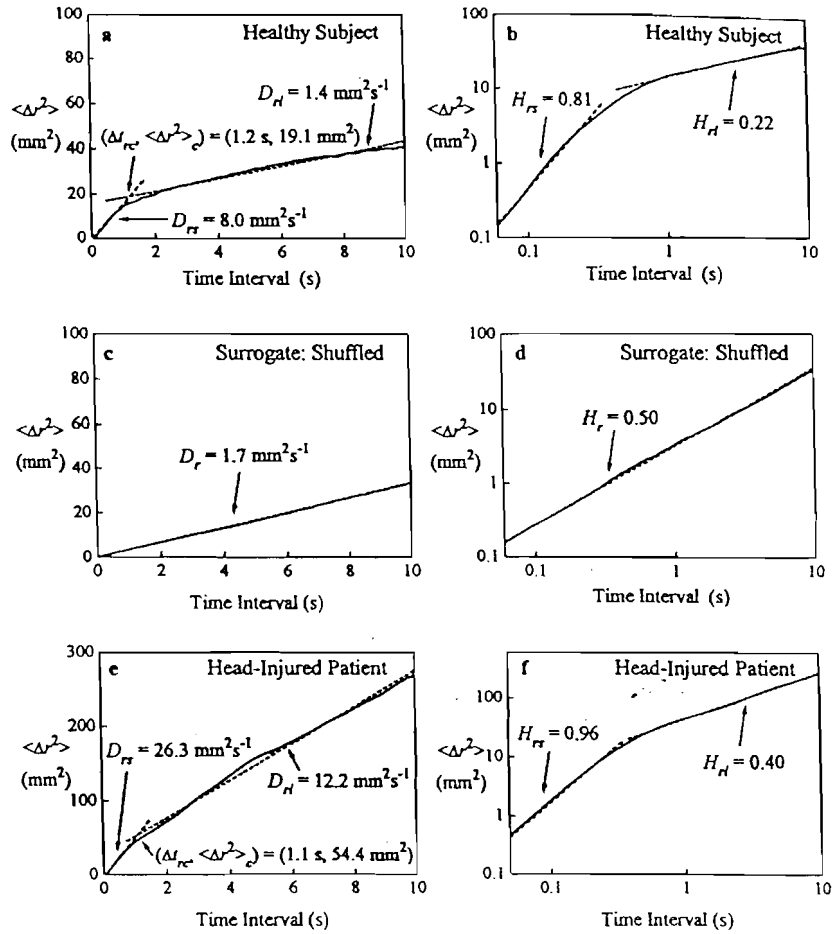

FIG. 3. Resultant (a) linear–linear and (b) log–log plots of mean square planar COP displacement versus time interval for a representative subject. Also shown in (a) and (b) are the fitted regression lines (dashed lines) for the short-term and long-term regions and the computed values for the respective stabilogram-diffusion parameters. (c) As in (a), but for shuffled surrogate random-walk data sets that were generated from the original COP time series. (d) As in (b), but for the surrogates in (c). (e) As in (a), but for a patient with a mild head injury. (f) As in (b), but for the patient in (e).

upright bipedal stance is considerably more stable in the frontal plane than in the sagittal plane.) Second, the short-term scaling exponents were considerably greater than 0.5 [Fig. 4(b)]. Thus, over short-term intervals of time during quiet standing, the COP behaves as a positively correlated random walk, i.e., the system exhibits persistence. On the other hand, the long-term scaling exponents were considerably less than 0.5 [Fig. 4(b)]. Thus, over long-term intervals of time, the COP behaves as a negatively correlated random walk, i.e., the system exhibits anti-persistence. Finally, the critical period occurred over relatively small time intervals, i.e., $\Delta t_{j_c} \approx 1.0$ s [Fig. 4(c)], and mean square displacements, i.e., $\langle\Delta j^2\rangle_c$ was typically less than 20 mm^2 [Fig. 4(d)].

In order to determine whether the aforementioned correlations in the COP time series were artifacts of the data-set size and/or the amplitude distribution of the increments in displacement, we randomly shuffled the temporal order of the increments[16] making up the COP time series and then recombined the increments to form surrogate random-walk sequences [e.g., see Fig. 1(c)]. For each subject, an ensemble of ten different shuffled surrogate sets were generated from each of the five original COP time series and subsequently analyzed. (The regression lines fitted for computation of the respective surrogate scaling exponents had r^2 values that ranged from 0.97 to 1.00.) We calculated the significance of the differences between the computed H_j values for the original COP time series and the surrogates according to the method described by Theiler *et al.*[17] (We also used techniques described therein to estimate error bars on the significance.) With this approach, the significance is defined by the difference between the value of H_j for the original COP time series and the mean value of H_j for the surrogates, divided by the standard deviation of the H_j values for the surrogates. We found that the double-logarithmic plots of mean square displacement versus Δt for the shuffled surrogates displayed only a single scaling region [e.g., see Fig. 3(d)], as would be expected for an uncorrelated random walk. The H_j values for the surrogates (range: 0.47–0.54, mean: 0.50±0.02) were also similar to those expected for a classical random walk [Figs. 5(a), 5(c), and 5(e)], and they were significantly different from those computed for the original COP time series [Figs. 5(b), 5(d), and 5(f)]. Thus, we were able to reject the

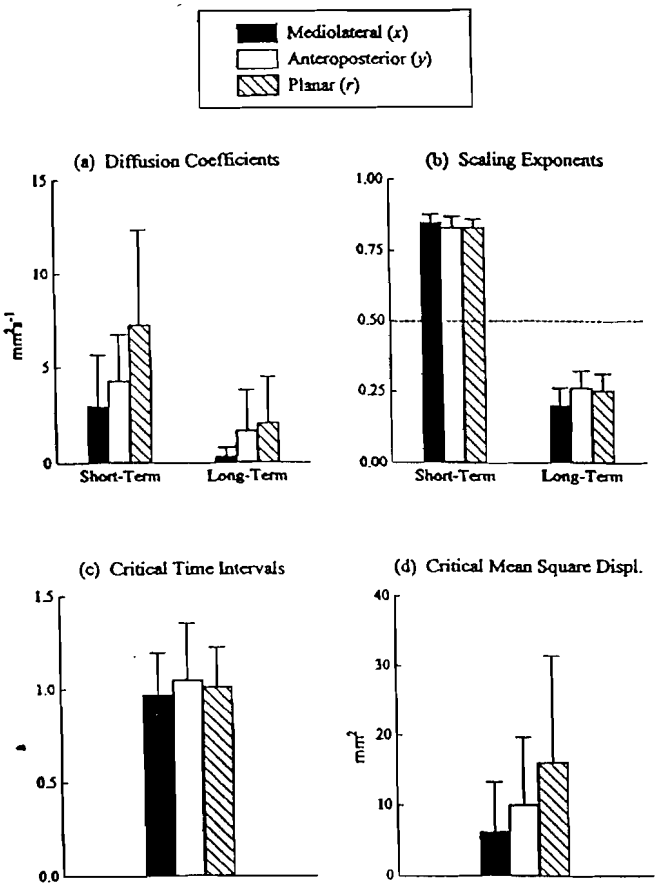

FIG. 4. Group means and standard deviations for the ten subjects: (a) diffusion coefficients, (b) scaling exponents, (c) critical time intervals, and (d) critical mean square displacements. In (b), a dashed line is drawn at the value expected for classical Brownian motion, i.e., $H_{ji}=0.50$.

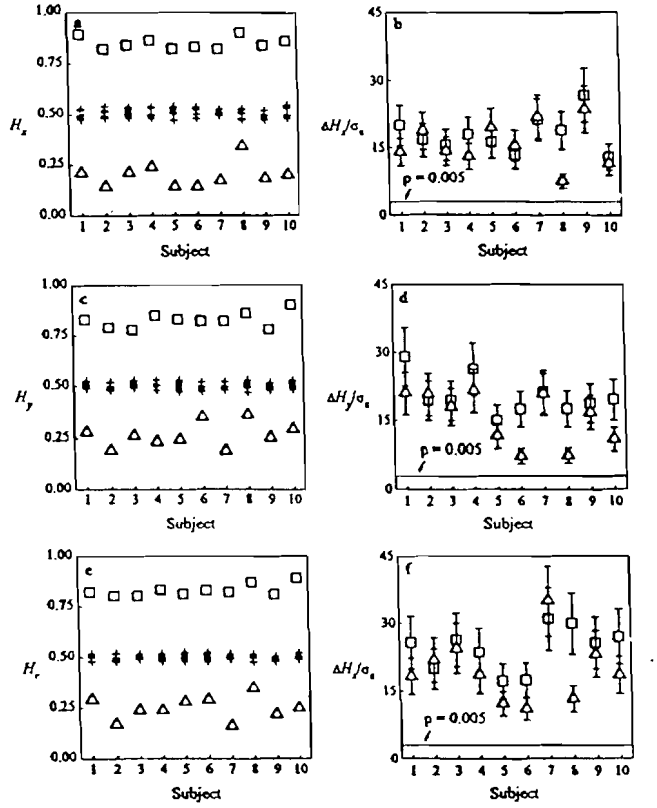

FIG. 5. Random-walk analyses of COP time series and shuffled surrogate data sets. (a) Calculated values of H_x for the short-term (□) and long-term (△) scaling regions of the original COP time series and for the shuffled surrogates (+) for each of the ten subjects. (b) The significance of the differences between the computed H_x values for the original COP time series and the surrogates in (a). The significance values and error bars were calculated according to the techniques described by Theiler *et al.*[17] Here σ_x is the standard deviation of the H_x values for the surrogates, and ΔH_x is the difference between the value of H_x for the original COP time series and the mean value of H_x for the surrogates. A dashed line is plotted at the significance level that corresponds to a p value of 0.005. (c) As in (a), but for the anteroposterior scaling exponents (H_y). (d) As in (b), but for the computed values of H_y in (c). (e) As in (a), but for the planar scaling exponents (H_r). (f) As in (b), but for the computed values of H_r in (e).

null hypothesis that postural sway is an uncorrelated random walk. Importantly, these results suggest that the correlations in the COP time series were due to underlying dynamic processes and that they were not artifacts of the analysis.

Stabilogram-diffusion analysis may eventually be used to identify and/or diagnose individuals with balance disorders. As a preliminary indication of the possible clinical utility of this technique, we have included in Figs. 3(e) and 3(f) resultant planar stabilogram-diffusion plots for a patient (age: 36 yr) with a mild head injury. It is clear that some of the computed results for this patient were significantly different from those for the healthy subjects of the present study. For instance, the patient's short-term and long-term planar diffusion coefficients were more than three standard deviations greater than the respective group means given in Fig. 4(a). It can also be seen from Fig. 3(e) that the long-term region for the patient was characterized by small-amplitude, low-frequency oscillations. This finding suggests that some (or all) of the COP trajectories for this patient contained similar such oscillations. [It should be noted, however, that we have observed similar oscillations in the stabilogram-diffusion plots for a small number of healthy subjects, e.g., see Fig. 3(d) in Collins and De Luca.[5]] Further work with well-defined patient populations is obviously needed.

IV. DISCUSSION

In this work, we demonstrated that quiet-standing postural sway can be modeled as a system of bounded, correlated random walks. Specifically, we showed that over short-term intervals of time during undisturbed stance the COP behaves as a positively correlated random walk, whereas over long-term intervals of time it resembles a negatively correlated random walk. From a physiological standpoint, we interpret the presence of short-range positive correlations in the COP data as an indication that the postural control system utilizes open-loop control mechanisms over short-term intervals of time ($\Delta t<1$ s) and small displacements. That is, the system allows the COP to "drift" for some time and/or displacement. This novel finding, which suggests that the system allows a certain amount of "sloppiness" in balance control, challenges the generally accepted notion that erect stance is always regulated by the action of feedback mechanisms.[18,19] It is important to note, however, that our analyses do not exclude the role of feedback mechanisms,

such as the visual, vestibular, and proprioceptive systems, in the regulation of upright stance. In fact, the presence of longer-range negative correlations in the COP data suggests that closed-loop control mechanisms are utilized over long-term intervals of time ($\Delta t > 1$ s) and large displacements. That is, after some time and/or displacement, the postural control system shifts the COP back toward a relative equilibrium position. Within this conceptual model, the central nervous system still continually receives afferent information from peripheral sensory organs; however, such information is not used to modify the efferent signals transmitted to postural muscles unless, for example, some threshold value is exceeded. The integration of open-loop control schemes with closed-loop feedback mechanisms for balance regulation may have evolved to account for feedback-loop delays and inherent noise in the system (e.g., due to inherent muscle force fluctuations[20]), and to simplify the task of integrating vast amounts of sensory information when the body is not in jeopardy of instability.

Within the context of the above postural control hypothesis, the short-term and long-term diffusion coefficients approximate the effective stochasticity of the open-loop and closed-loop postural control mechanisms, respectively. The finding that the short-term diffusion coefficients were substantially greater than the long-term diffusion coefficients thus suggests that the open-loop control schemes (the output of which may take the form of descending commands to different postural muscles) exhibit an effectively higher level of stochasticity than the closed-loop feedback mechanisms. The short-term stochastic effects are likely due to the noise-like fluctuations that are produced across various joints of the body by the aforementioned open-loop activation signals. (Such fluctuations are a consequence of the fact that skeletal muscles are incapable of producing constant forces.[20]) The long-term stochastic effects may be related to the fact that the human body in upright stance can assume a number of different positions that are "statically stable." During quiet standing, an individual may switch between these different equilibrium positions in a stochastic manner.

Likewise, in light of the above hypothesis, the critical point coordinates approximate the temporal and spatial coordinates of the transition region over which the postural control system switches from open-loop control to closed-loop control. As noted in our earlier paper,[5] the position of the critical point may be set by a number of physiological/biomechanical factors and/or mechanisms, including (1) a proprioceptive "dead zone," i.e., a region over which slight variations in body-segment position and orientation are left unchanged; (2) a "dead zone" that arises from the interaction of postural responses with the body's inertia; or (3) fixed, preprogrammed central commands that are utilized in quiet stance. This issue requires further study.

Fractal measures, such as fractal dimensions, have been applied to several physiological systems and processes,[21,22] including the human postural control system.[23–25] However, in most cases, these measures have not been linked to the underlying physiology in a meaningful way; instead, their use has been limited primarily to describing and classifying time-series patterns and the shapes of biological objects. The novelty of the present approach is that fractal-type parameters, i.e., scaling exponents, are interpreted from a motor control standpoint, i.e., they are linked in a mechanistic fashion to the dynamic characteristics of the postural control system. It would be erroneous to assume, however, that postural sway, given its fractal nature, is an instance of deterministic chaos. In a recent study,[6] we used surrogate-data techniques and algorithms from dynamical systems theory to show that COP trajectories are indistinguishable from correlated noise. We did not find any evidence that postural sway reflects a dynamical system with a low-dimensional attractor. We therefore concluded that balance regulation, as viewed through COP measurements, is better represented as a stochastic process, as opposed to a chaotic one. This work is consistent with our present findings.

In summary, stabilogram-diffusion analysis has the advantage that it leads to the extraction of three sets of COP parameters—diffusion coefficients, scaling exponents, and critical point coordinates—that can be directly related to the resultant steady-state behavior and functional interaction of the neuromuscular mechanisms underlying the maintenance of upright stance. It should also be noted that the majority of the stabilogram-diffusion parameters exhibit "good" to "excellent" reliability, as measured by intraclass correlation coefficients.[5] Thus, individuals typically have their own "signature" stabilogram-diffusion plots. This statistical-biomechanics approach therefore can be used to formulate and test hypotheses concerning the relative contributions of different sensorimotor subsystems and strategies to quiet-standing balance control.

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