Nonlinear time series analysis of normal and pathological human walking

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Characterizing locomotor dynamics is essential for understanding the neuromuscular control of locomotion. In particular, quantifying dynamic stability during walking is important for assessing people who have a greater risk of falling. However, traditional biomechanical methods of defining stability have not quantified the resistance of the neuromuscular system to perturbations, suggesting that more precise definitions are required. For the present study, average maximum *finite-time* Lyapunov exponents were estimated to quantify the local dynamic stability of human walking kinematics. Local scaling exponents, defined as the local slopes of the correlation sum curves, were also calculated to quantify the local scaling structure of each embedded time series. Comparisons were made between overground and motorized treadmill walking in young healthy subjects and between diabetic neuropathic (NP) patients and healthy controls (CO) during overground walking. A modification of the method of surrogate data was developed to examine the stochastic nature of the fluctuations overlying the nominally periodic patterns in these data sets. Results demonstrated that having subjects walk on a motorized treadmill artificially stabilized their natural locomotor kinematics by small but statistically significant amounts. Furthermore, a paradox previously present in the biomechanical literature that resulted from mistakenly equating variability with dynamic stability was resolved. By slowing their self-selected walking speeds, NP patients adopted more locally stable gait patterns, even though they simultaneously exhibited greater kinematic variability than CO subjects. Additionally, the loss of peripheral sensation in NP patients was associated with statistically significant differences in the local scaling structure of their walking kinematics at those length scales where it was anticipated that sensory feedback would play the greatest role. Lastly, stride-to-stride fluctuations in the walking patterns of all three subject groups were clearly distinguishable from linearly autocorrelated Gaussian noise. As a collateral benefit of the methodological approach taken in this study, some of the first steps at characterizing the underlying structure of human locomotor dynamics have been taken. Implications for understanding the neuromuscular control of locomotion are discussed. © *2000 American Institute of Physics.* [S1054-1500(00)00804-1]

Falls and their resulting injuries have a significant impact on the rates of morbidity and mortality among older adults as well as on health care costs. As many as 23% of falls among community dwelling elderly persons result in serious injury[1] and more than half of these falls result from trips that occur during locomotion.[2] Therefore, quantifying dynamic stability during walking is very important for assessing people who may be at greater risk of falling. A number of biomechanical indices have been proposed to define locomotor ''stability,'' all of which rely on quantifying some measure of variability in locomotor patterns.[3–6] However, these methods do not relate the notion of stability to the resistance of the neuromuscular system to perturbations, suggesting that more precise definitions of dynamic locomotor stability are required. Analytical models of locomotor patterns based on systems of coupled nonlinear oscillators[7–12] and simulations of passive dynamic walking machines exhibiting chaotic behavior[13,14] suggest that nonlinear analysis techniques may provide insights into the neuromuscular processes that govern locomotion. However, few efforts have been made to apply experimental techniques from nonlinear dynamics to locomotion. While some authors have used Floquet multipliers to quantify locomotor stability,[15,16] this method assumes purely periodic motion. Methods for estimating maximum *finite-time* Lyapunov exponents from experimental data[17,18] provide a promising means of directly quantifying local dynamic stability during locomotion. The present research computed maximum finite-time Lyapunov exponents from human walking kinematics to examine how the locomotor control system adapts to walking on a motorized treadmill and to degradation of peripheral sensory feedback. The present analyses demonstrate that the concepts of variability and stability must be carefully distinguished in studies of human locomotion and, more generally, that dynamical systems approaches can be used to precisely characterize local dynamic stability in such studies.

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I. INTRODUCTION

Traditional methods of describing human locomotion involve collecting gait data for a number (typically 5 to 10) of independent and isolated strides, time-normalizing the data from each stride to a standard length (100%), and then averaging the data across strides. This approach seeks to examine the nature of gait patterns exhibited during a ''typical'' stride, and how such patterns differ between healthy subjects and patients with various locomotor pathologies. Variables examined in this manner may include kinematics, ground reaction forces, muscle activation patterns, and/or moments and powers at individual joints computed from inverse dynamics (e.g., Ref. 3). While this approach has allowed researchers to address many important clinical questions related to locomotor disorders, it ignores the inherent dynamical nature of locomotion. Any information about how the neuromuscular system controls locomotion from one stride to the next is lost in this approach. The techniques used to analyze nonlinear dynamical systems may therefore provide unique insight into these ongoing neuromuscular control processes, particularly as they relate to the control of dynamic stability during continuous walking.

Time-series analysis methods typically require large sets of data collected continuously over many cycles of motion. The most convenient means of collecting such data is to have subjects walk on a motorized treadmill. However, while several studies have reported biomechanical differences between overground and treadmill walking,[19–21] other studies have reported minimal or no differences.[22–24] In particular, walking on a motorized treadmill appears to reduce the stride-to-stride variability of locomotion compared to overground walking.[19,25,26] Most of these previous studies, however, did not examine the continuous nature of walking, but focused merely on differences between single averaged stride patterns. The first objective of the present study was to test the hypothesis that walking on a motorized treadmill artificially stabilizes natural walking patterns compared to walking over level ground by driving the locomotor system at a nearly constant externally enforced speed.

Peripheral neuropathy is a secondary consequence of diabetes mellitus that results in a gradual dying back of nerves from the fingers and toes to more proximal regions.[27,28] Neuropathic patients are as much as 15 times more likely to report an injury during standing or walking and have a greater risk of repetitive falls than healthy subjects,[29,30] independent of other co-morbidities.[31] However, standard biomechanical comparisons of averaged single-stride gait characteristics have shown few differences between the locomotion patterns of neuropathic patients and matched controls beyond a reduction in self-selected walking speed.[32–34] These slower walking speeds have led some to suggest that these patients may be adopting a ''more conservative'' locomotor control strategy.[33] However, these slower speeds are also associated with increased locomotor variability.[25,34] This suggests that slowing one's walking speed should increase one's risk of falling if, as traditionally assumed, greater variability were indicative of loss of stability. However, the fact that young healthy subjects also exhibit increased variability during slow walking,[35,36] in a context where stability is clearly not an issue, calls this assumption into question. If increased variability were indicative of loss of stability, then slowing down, as a locomotor control strategy, would be completely antithetical to the goal of maintaining stability. Thus, it remains unclear why neuropathic patients choose to walk more slowly in the absence of any obvious physiological impairments (e.g., loss of strength, etc.) that would prevent them from walking faster.

The principal reason for such apparently paradoxical results arises from the fact that statistical measures of variability do not address the nature or the source of the increased instability that leads to the increased risk of falling in these patients. Standard deviations only quantify the average magnitude of the variations that occur between strides, and therefore cannot quantify how the locomotor control system responds to perturbations. In the present context, ''local stability'' refers to the sensitivity of a system to infinitesimally small perturbations. The natural variations that occur from stride to stride during locomotion reflect precisely these types of perturbations and it is presumably the effects of such perturbations that measures of gait variability are attempting (unsuccessfully) to quantify. The second objective of this study was therefore to compare the local dynamic stability of continuous overground walking kinematics in diabetic patients with severe neuropathy to those of gender-, age-, height-, and weight-matched nondiabetic controls. The relative contributions of the loss of peripheral sensation and changes in self-selected walking speeds to the local dynamic stability of walking patterns in these subjects were also examined.

Traditional methods of describing human gait inherently assume that stride-to-stride variations in these movement patterns arise solely from noise in the system. However, no efforts have been made to fully characterize the exact nature of this noise or to locate it along the deterministic/stochastic continuum. Thus, potentially important information about the precise nature of the stochasticity or any possible deterministic influences that might be contributing to these fluctuations is essentially ignored. It has been hypothesized that sensory feedback plays a role in adjusting step-to-step limb trajectories to maintain balance during locomotion,[37] and/or in smoothing unintended irregularities that occur during unperturbed movements.[38] Therefore, the fluctuations overlying the nominal cyclic movements in human walking may reflect valuable information about the neuromuscular processes contributing to the generation of both normal and pathological locomotor patterns.

Stride interval data from human walking exhibit $1/f$ noise.[39–41] It has been suggested that these long range correlations in walking are the result of complex deterministic processes in the central nervous system that play an active role in regulating locomotor dynamics.[39,40] However, analytical models of a similar rhythmic sensory motor coordination task demonstrate that such correlation structures might also arise as a natural byproduct of the presence of stochastic noise and sensory feedback delays in neural circuitry.[42] Since $1/f$ noise can be modeled by a number of either deterministic or stochastic processes and since there is no gener-

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ally accepted interpretation of such findings,[42,43] the underlying source of the $1/f$ noise observed in locomotor patterns remains speculative. Therefore, the third objective of this study was to use appropriately generated phase-randomized surrogate data[44] to test the hypothesis that the fluctuations exhibited in continuous walking kinematics can be generated by a specific class of stochastic processes: linearly autocorrelated Gaussian noise.

II. DATA COLLECTION

For the overground/treadmill (OG/TM) comparisons, 10 young healthy subjects (5 males and 5 females; mean age $=27.10\pm3.25$ yrs, height$=1.71\pm0.09$ m, and weight$=64.85\pm12.47$ kg) participated. For the neuropathic/control (NP/CO) study, 14 diabetic patients with significant peripheral neuropathy (NP Group; 12 males and 2 females, mean age $=61.0\pm6.6$, height$=1.77\pm0.07$ m, weight$=95.2\pm14.1$ kg), and 12 healthy controls with no history of diabetes or neuropathic illness (CO Group; 10 males and 2 females, mean age$=57.6\pm7.7$, height$=1.76\pm0.08$ m, weight$=91.1\pm9.8$ kg) participated. Subjects in the NP and CO groups were matched on marginal distributions (i.e., approximately the same mean and variance within each group) according to gender, age, height, and weight.[34]

Each subject was required to pass an extensive screening exam to ensure that no subject had a history of medications, illnesses (other than peripheral neuropathy, where appropriate), surgeries, or other injuries that may have affected their walking patterns. Physical exam included quantifying passive ranges of motion (ROM) at the knee, ankle, and great toe and an assessment of lower extremity strength at the knee and ankle. NP and CO subjects were also tested for their level of peripheral sensation using a standard touch/pressure sensation (TPS) testing protocol at four locations on the bottoms of both feet: the hallux, first and fifth metatarsal heads (MTH-1 and MTH-5), and the heel. The TPS test determined the minimum detectable response to first-order bending of a series of wirelike nylon monofilaments of increasing diameter. Each monofilament was calibrated to produce a known applied force (range: 0.04 to 300 N) on the bottom of the foot under first-order bending. Detailed descriptions and results of these tests are reported elsewhere.[26,34]

A portable ''data-logger'' was built to collect kinematic data during continuous walking. The data-logger consisted of a programmable microprocessor and an 8-channel 12-bit A/D converter interfaced to a 15-Mb Flash RAM card. The data-logger was attached to a lightweight harness (<2.5 kg total weight) that did not interfere with the subject's normal walking (Fig. 1). Three electrogoniometers were placed across the approximate joint centers of the hip, knee, and ankle joints of the right leg to measure sagittal plane movements of these joints. A triaxial accelerometer was mounted at the base of the sternum to measure upper body motions. The ''Y''-accelerometer was oriented approximately vertically, with the ''X'' and ''Z'' accelerometers approximately horizontal (anterior-posterior and mediolateral, respectively). The baseline electrical noise generated by each of these transducers was between 0.76 and 1.11 mV root mean square (rms), which was on the order of the minimum resolution of the A/D converter (±1 mV). The data-logger was programmed to collect data for 10 min at 66.7 Hz (40 000 samples).

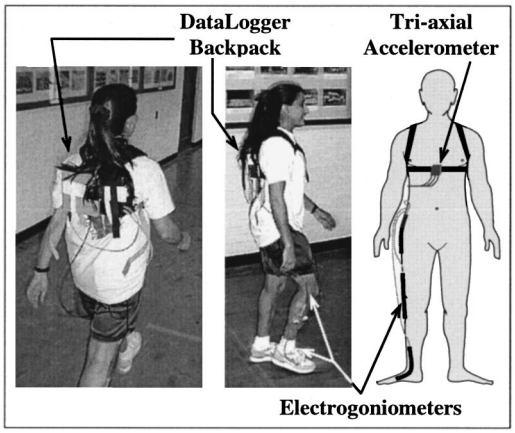

FIG. 1. Setup of data-logger data collection instrumentation. The triaxial accelerometer quantified three-dimensional (3D) movements of the upper body. Electrogoniometers measured movements of the joints of the right leg in the sagittal plane (i.e., in the plane of forward progression).

During overground walking trials, each subject walked around a roughly rectangular level indoor walking track approximately 7 m wide and 200 m long at his/her own self-selected comfortable speed. Average walking speeds were calculated by directly measuring the total distance walked and dividing by 10 min. For treadmill walking trials, the speed of the treadmill was set to the average walking speed calculated from the overground trial for each subject and data collection was repeated. For the NP and CO subjects, data were collected only during overground walking.

III. NONLINEAR ANALYSES

A. State space reconstruction

State space reconstructions of the raw kinematic data were performed based on standard embedding techniques.[45–47] For each time series, an appropriate state space was reconstructed from the original time series and its time delayed copies,

$$X(t)=[x(t),x(t+T),\ldots,x(t+(d_E-1)T)], \tag{1}$$

where $X(t)$ is the d_E-dimensional state vector, $x(t)$ is the original one-dimensional data, T is the time delay, and d_E is the embedding dimension. Time delays (T) for the reconstructions were calculated from the first minimum of the average mutual information (AMI) function,[48] which evaluates the amount of information (in bits) shared between two data sets over a range of time delays. Choosing the first minimum of the AMI function provides adjacent delay coordinates with a minimum of redundancy.

Embedding dimensions (d_E) were computed from a global false nearest neighbors (GFNN) analysis,[49] which compares the distances between neighboring trajectories at successively higher dimensions. ''False neighbors'' occur when trajectories that overlap in dimension d_i are distinguished in dimension d_{i+1}. As i increases, the total percentage of false

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neighbors declines and d_E is chosen where this percentage approaches zero. GFNN analysis was performed using values of $R_{\text{tol}}=17$ and $A_{\text{tol}}=2$ as recommended in Ref. 49.

B. Stationarity

The stationarity of all walking patterns was established by evaluating recurrence plots[50–52] of each embedded time series. Recurrence plots were generated by calculating the Euclidean distances ($\delta_{i,j}$) between all pairs of points $X(i)$ and $X(j)$ in the embedded state space and then plotting those points in the (i,j) plane where $\delta_{i,j}$ was less than a specified radius, r,

$$\delta_{i,j}=\|X(i)-X(j)\|<r. \tag{2}$$

Since i and j are points in time, the recurrence plots convey natural and subtle information about temporal correlations in the original time series.[51,52] Nonstationarities in the time series are manifested as gross inhomogeneities in the recurrence plot. All recurrence plots were generated using a radius value of $r=5\%$ of the total data set size for each time series.

C. Local scaling structure

The correlation sum, $C(r,N)$, quantifies the way in which the density of points in state space scales with the size of the volume containing those points.[18,53–55] Thus, $C(r,N)$ provides insight into the geometric structure of an embedded time series. In the present study, $C(r,N)$ was computed from[17,18,55]

$$C(r,N)=\frac{1}{(N-n)(N-n-1)}\times\sum_{i=1}^{N}\sum_{j=1+n}^{N}H(r-\|X(i)-X(j)\|), \tag{3}$$

where r was the radius of the volume of points being considered, H was the Heaviside step function, N was the total number of samples in the time series, and n was chosen to be the average stride time for each subject (typically 70-80 samples). Using this formulation, neighboring points which where correlated in time (i.e., $|i-j|<n$) were omitted from the calculation to ensure that $C(r,N)$ captured only *geometric* correlations in each of the embedded time series.[18,54,55]

The computation of $C(r,N)$ is susceptible to noise and nonstationarities,[18,54,56] both of which are common in physiological data.[55] To address these concerns, instrumentation noise was minimized by careful design of the data-logger and the stationarity of each time series was established from the recurrence plot analysis. Furthermore, the GFNN algorithm ensured that each time series was embedded in a state space with a sufficiently large d_E to guarantee saturation of $C(r,N)$. Finally, each 10-min time series was divided into five intervals of 2 min each ($N=8000$ samples ≈ 100 strides). This allowed for the evaluation of the variance in each estimated $C(r,N)$ curve, while retaining a sufficiently large N to ensure accurate computation.[55]

The scale-invariant ''self-similarity'' that is one of the hallmarks of low-dimensional deterministic ''chaos''[18,53–55] results in a linear variation of $\log[C(r,N)]$ with respect to $\log[r]$ as $r\to 0$. In cases where such self-similar dynamics exists, the correlation *dimension*, D_2, is defined as

$$D_2=\lim_{N\to\infty}\lim_{r\to 0}\frac{\log[C(r,N)]}{\log[r]}. \tag{4}$$

The linearity of the relationship between $\log[C(r,N)]$ and $\log[r]$ can be determined by computing local slopes, or ''local scaling exponents'' (LSE)[18,55] directly from plots of $\log[C(r,N)]$ vs $\log[r]$ (Fig. 2). In the present study, LSEs were computed for each embedded time series using a standard three-point difference formula,

$$\text{LSE}_{(i)}=\frac{\log[C(r,N)]_{(i-1)}-\log[C(r,N)]_{(i+1)}}{2\cdot\Delta\log[r]}. \tag{5}$$

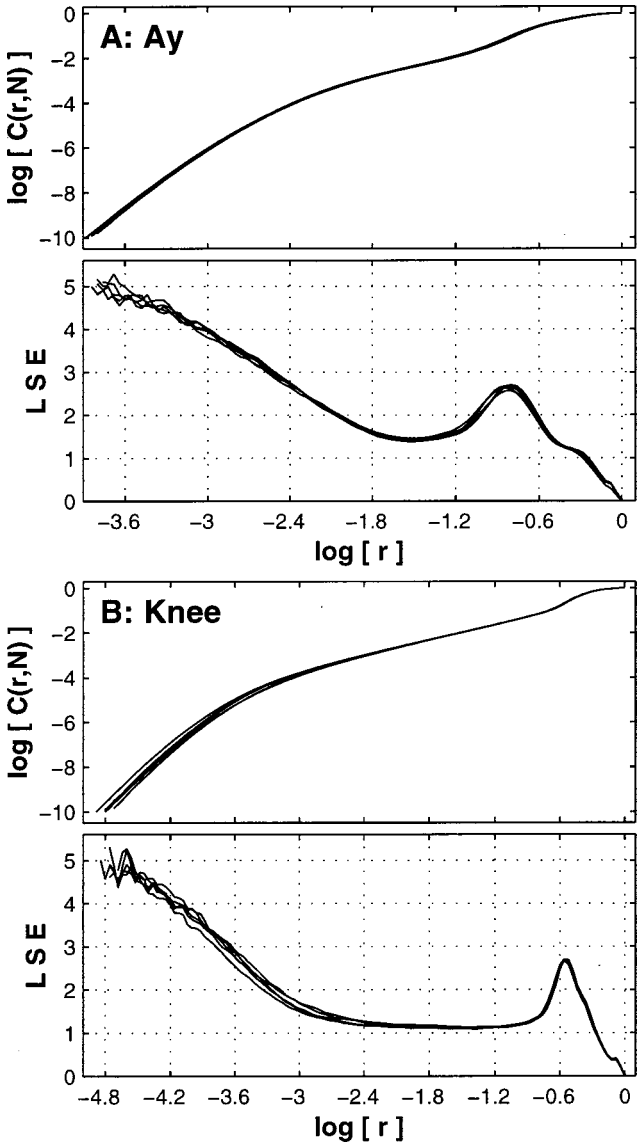

FIG. 2. Correlation sums and local scaling exponents (LSE) for the Ay (vertical) acceleration (A) and knee joint angle (B) time series taken from a typical subject. Five curves are shown in each plot: one for each of the five 2-min intervals of data analyzed. An embedding dimension of $d_E=5$ was used for all calculations. Vertical gridlines on the LSE plots denote points along $\log[r]$ where statistical comparisons were made.

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Correlation sums from biological data sets rarely exhibit the strictly linear behavior that is required to make an unambiguous determination of D_2.[18,55,57] The data sets from the present study were no exception (Fig. 2). Nevertheless, even in these cases, the LSEs themselves still provide a quantitative description of the geometric scaling behavior of these embedded time series across a wide range of length scales. Figure 2 shows that these data sets in fact exhibit different scaling behaviors across different scaling ranges in $\log[r]$.[18,55,57] For very large r values, (approximately $\log[r] \geqslant -0.6$ in Fig. 2), the calculation of $C(r,N)$ is dominated by the finite length of the data set[55] and the LSEs approach zero. For the knee data of Fig. 2(b), there is a second region (approximately $-2.4 \leqslant \log[r] \leqslant -0.6$) where the LSEs remain relatively constant with a value of approximately 1, corresponding to the nominal one-dimensional limit cycle structure. This phenomenon was much less prevalent in the upper body acceleration data sets [e.g., Fig. 2(a)], although some structure in these curves was still visible. For smaller values of r (approximately $\log[r] \leqslant -1.8$), data points begin to move off of the limit cycle and spread apart across the available state space.[18] This process ends at the smallest length scales (approximately $\log[r] \leqslant -3.6$), where the LSEs approach the value of the embedding dimension ($d_E = 5$), indicating where the noise regime lies.[18,55]

Similar LSE curves were generated for all six kinematic variables to determine if treadmill walking or diabetic neuropathy produced significant changes in local scaling behavior. To test for statistical differences in local scaling behavior, individual LSE values were extracted from these curves at several locations along $\log[r]$. Five LSE estimates were obtained at each value of $\log[r]$ for each subject for each data set: one LSE for each of the five 2-min intervals examined. These LSE values were then used as repeated measures in a series of multifactor analyses of variance (ANOVA) for randomized block design[58] to test for differences between OG and TM walking and between NP and CO subjects. ANOVA results were adjusted using a Bonferroni correction[58] to account for the ''compounded uncertainty'' that arises when making multiple comparisons within the same data set. Thus, a value of $\alpha = 0.05/k$ was chosen to delineate statistically significant differences, where k was the number of comparisons made across each set of LSE curves. For acceleration variables, ANOVA's were performed at evenly spaced intervals of $\Delta \log[r] = 0.6$ from $\log[r] = -3.6$ to $\log[r] = -0.6$ (i.e., $k = 6$; $\alpha = 0.008$). LSEs for lower extremity time series were tested at evenly spaced intervals of $\Delta \log[r] = 0.6$ from $\log[r] = -4.8$ to $\log[r] = -0.6$ (i.e., $k = 8$; $\alpha = 0.006$). These points of comparison are indicated by the vertical grid lines shown in Fig. 2.

D. Finite-time Lyapunov exponents

Lyapunov exponents quantify the average exponential rate of divergence of neighboring trajectories in state space, and thus provide a direct measure of the sensitivity of the system to infinitesimal (i.e., ''local'') perturbations.[17,18] The maximum Lyapunov exponent (λ_1) for a dynamical system can be determined from

$$d(t) = d_o e^{\lambda_1 t}, \tag{6}$$

where $d(t)$ is the mean divergence between neighboring trajectories in state space at time t and d_o is the initial separation between neighboring points.[17] *Finite-time* exponents (λ^*) are distinguished from true Lyapunov exponents (λ_1), which are strictly defined only in the dual limit as $t \to \infty$ and $d_o \to 0$ in Eq. (6).

A previously published algorithm[17] was used to provide estimates of average maximum finite-time Lyapunov exponents (λ^*) for each time series examined. This algorithm is robust to changes in T, d_E, data set size, and measurement noise. Therefore, each 10-min time series was first divided into five intervals of 2 min each ($N = 8000$ samples ≈ 100 strides) to allow for the calculation of within-subject as well as between-subject variances in the λ^* estimates. Taking the log of both sides of Eq. (6), λ^* was defined by

$$\ln[d_j(i)] \approx \lambda^*(i\Delta t) + \ln[d_{oj}], \tag{7}$$

where $d_j(i)$ was the Euclidean distance between the jth pair of nearest neighbors after i discrete time steps (i.e., $i\Delta t$ s).[17] Euclidean distances between neighboring trajectories in state space were calculated as a function of time and averaged over all original pairs of nearest neighbors. The λ^* exponents were then estimated from the slopes of linear fits to curves defined by[17]

$$y(i) = \frac{1}{\Delta t}\langle \ln[d_j(i)] \rangle, \tag{8}$$

where $\langle \cdot \rangle$ denotes the average over all values of j. Since the intrinsic time scales were different for each subject (i.e., each subject had a different average stride time), these curves were rescaled in time to the average stride number (i.e., number of attractor orbits) for each subject. For the present study, the λ^* were estimated from best-fit linear slopes of these local divergence curves over scaling regions corresponding to the time between four and ten strides (see Figs. 7 and 13).

The five estimates of each λ^* calculated for each subject were taken as repeated measures in a series of multiple-factor ANOVAs for a randomized block design[58] to test for differences between OG and TM walking and between NP and CO subjects. A significance level of $\alpha = 0.05$ was chosen to delineate statistically significant differences between each pair of test groups.

While differences in local dynamic stability between NP and CO subjects may have been due to differences in sensory status, they might also have been due to group differences in walking speed, ROM, and/or lower extremity strength. Furthermore, walking speed is not an independent predictor, since intrinsic differences in sensation, ROM, and/or strength may also affect self-selected walking speeds. Therefore, a multivariate regression analysis[58–60] was performed to determine if intrinsic differences in sensory status, ROM, and/or strength directly influenced local dynamic stability, or if these effects were in fact mediated by changes in self-selected walking speeds. This procedure was performed on all λ^* variables where statistically significant between-group differences were indicated by the ANOVA.

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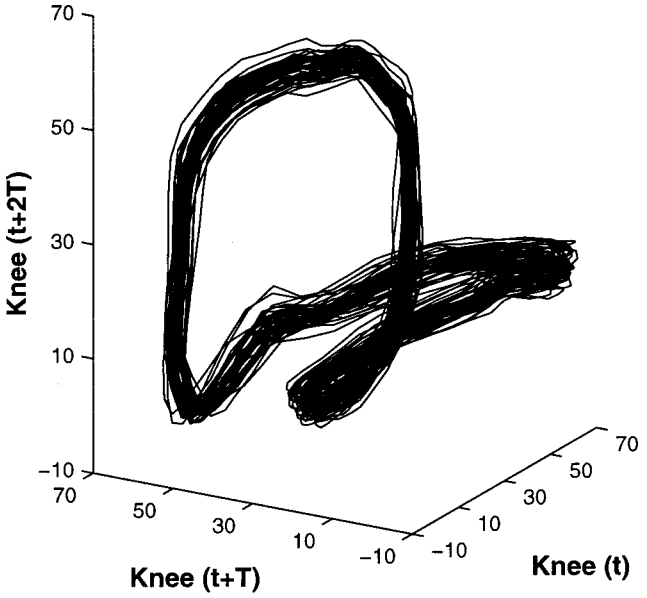

FIG. 3. Example of a representative set of 4000 samples (1 min) of knee joint angle data embedded in a 3D state space ($T=16$ samples). Note that stride-to-stride variations in the data add a noiselike complexity to the underlying nominally periodic behavior.

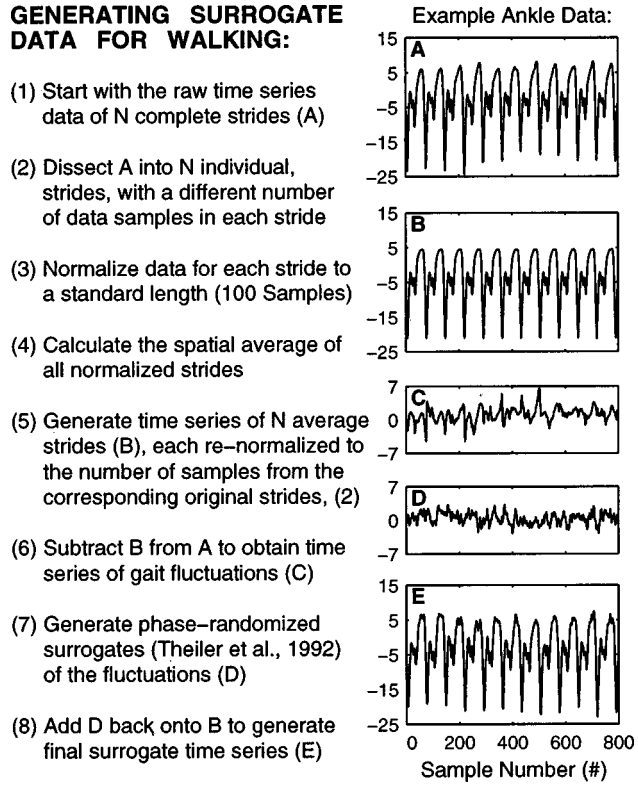

FIG. 4. Outline of the procedure used for generating phase-randomized surrogate (S1) time series for nominally periodic locomotion data.

E. Surrogate data

Human locomotion is strongly (but not exactly) periodic. Locomotor kinematics exhibits both temporal and spatial variations within and across strides. These naturally occurring variations generate movements that are nominally periodic, while adding a noiselike complexity to the underlying periodic structures in the embedded state spaces (Fig. 3). The exact nature of these fluctuations is presently unknown and was the focus of interest in the present study. Therefore, a modification of the method of surrogate data[44] was developed to examine the nature of the fluctuations overlying the nominally periodic movement patterns in these human walking data. The null hypothesis was tested that the variations about the mean walking patterns in the original data could have been generated by a linearly autocorrelated Gaussian process.[44]

Surrogate data for testing this hypothesis were generated by the procedure outlined in Fig. 4. The average kinematic pattern for each time series was computed and subtracted from each stride of the original time series, leaving only the overlying fluctuations [Fig. 4(c)]. Phase-randomized surrogates of these fluctuation signals were generated by computing the Fourier transform of the original fluctuation signal, randomizing the phase spectrum, and computing the inverse Fourier transform. This procedure generated stochastic fluctuation signals [Fig. 4(d)] with the same means, variances, autocorrelations, and power spectra as the original fluctuation signals.[44] These fluctuation signals were then added back onto the mean kinematic signals to generate the final surrogates, which were termed ''S1'' [Fig. 4(e)]. Thus, these resulting surrogate signals had the same underlying nominal periodicity as the original data, except that the overlying fluctuations about the means were now randomized. It should be noted that while the spatial fluctuations were averaged out when generating the mean walking signals [Fig. 4(b)], the temporal variations were not. Therefore, these surrogates should be viewed as providing a conservative test for the null hypothesis.

The utility of this procedure was tested with simulated time series. An appropriately scaled baseline periodic signal was superimposed with fluctuation signals generated from the standard Lorenz equations[61] and with phase-randomized surrogates of the same Lorenz signal. These Lorenz and surrogate Lorenz fluctuations were clearly distinguishable, with or without being superimposed on the baseline signal.[62]

As an additional control condition, phase-randomized surrogate data sets were generated directly from the original time series [i.e., from Fig. 4(a)], without first subtracting off the nominally periodic components of these signals. These surrogates were termed ''S2.'' Thus, in these surrogates not only were the fluctuations about the mean periodic orbits randomized, but the periodic components themselves were phase-randomized, resulting in surrogate signals with no discernable periodic structure. These surrogates assume a null hypothesis that the entire nominally periodic signals might be generated by linearly autocorrelated Gaussian noise. While human locomotion is obviously not generated by random movements, these surrogates were used to judge the influence of phase-randomizing only the overlying fluctuation signals [Fig. 4(c)] relative to the entire signal [Fig. 4(a)].

A second modification of the surrogate data method was in the statistical analysis of the surrogates. In the customary formulation,[44] multiple surrogates are generated for a single original time series. A test statistic calculated from the origi-

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nal time series is then compared to a variance of statistics calculated from the surrogates to establish statistical differences. A multiplicity of surrogates is required when no estimate of the variance in the value of the test statistic is available for the original time series. In the present study, there were five original time series for each variable for each subject, and between 10 and 14 subjects in each test group, providing ample estimates of the variance in the test statistics computed from each set of original time series data.

Surrogates (S1) were generated for all trials where subjects walked over level ground. Estimates of λ^* were computed for each surrogate time series in the same manner as those calculated for the original data sets. The original and surrogate exponents from each set of time series data were statistically compared using a two-factor repeated measures ANOVA[58] to test for differences between original (OR) and surrogate (S1) data types for each of the three subject groups (OG, NP, and CO). Although only one surrogate was generated for each original time series, the ANOVA yielded valid statistical discriminations between the original and surrogate data sets by making comparisons between multiple original-surrogate pairs of λ^* values across multiple subjects.

IV. RESULTS

A. OG/TM comparisons

Results of the GFNN analysis were nearly identical for both OG and TM walking and reached minimum plateau values at around $d_E=5$ for all six time series measures (Fig. 5). Horizontal accelerations in the anterior-posterior (Ax) and mediolateral (Az) directions did not reach 0% false neighbors, but began to increase slightly at higher embedding dimensions. This pattern is typical of signals with added noise[49] and, given that identical instrumentation was used to collect all three acceleration signals, may indicate increased levels of dynamical noise[43,49] in these time series variables. A value of $d_E=5$ was used for all signals for all subsequent analyses.

Recurrence plots exhibited no evidence of nonstationarity for any of the upper body acceleration signals recorded from any of the subjects tested. Some evidence of mild nonstationarity, in the form of very low frequency drifting, was observed in the lower extremity movements for a couple of subjects during the OG walking condition [Fig. 6(a)]. In all cases, however, these nonstationarities were substantially reduced or eliminated in the TM walking condition [Fig. 6(b)]. This finding is consistent with the notion that motorized treadmills suppress such low-frequency drifting behavior by externally fixing the mean walking speed.

Correlation sums and local scaling exponents (LSE) revealed some small differences in local scaling structure between OG and TM walking for a few time series variables for a few subjects. However, these differences varied widely between subjects and between time series and more than half of all of the data sets examined exhibited no discernable differences at all. There were no overall statistically significant differences in the LSEs between OG and TM walking for any of the time series variables examined. Therefore, it was concluded that TM walking had no significant effects on the local scaling structure of walking kinematics.

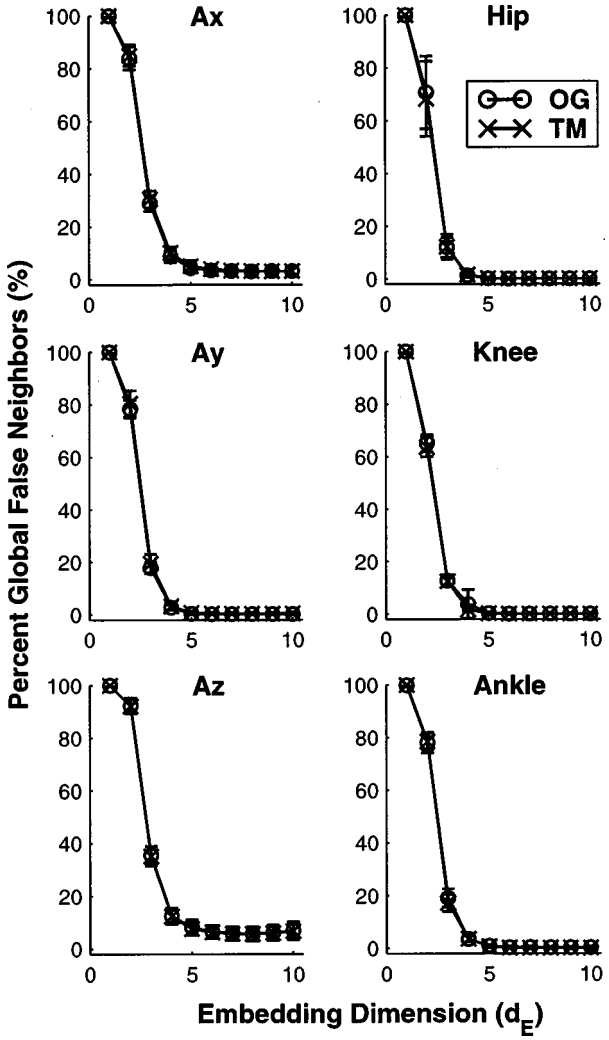

FIG. 5. Averaged results of GFNN analysis for OG and TM walking. Within each subplot, two curves are drawn corresponding to the mean results for the OG and TM conditions, respectively. Error bars denote between-subject standard errors. Results indicated an appropriate embedding dimension of $d_E=5$ for all time series.

In contrast, distinctive differences between the OG and TM walking conditions were evident from plots of the logarithmic divergence curves for each of the six time series measures (Fig. 7). On average, there were decreases of between 3% and 18% for five of the six λ^* exponents quantified during TM walking compared to OG walking (Fig. 8). These differences were statistically significant ($p<0.05$) for all three lower extremity joint movement variables, and nearly significant ($p=0.051$) for vertical accelerations of the upper body. These results confirm the hypothesis that, compared to OG walking, TM walking is associated with statistically significant improvements in local dynamic stability of locomotor kinematics, particularly for movements of the lower extremity.

B. NP/CO comparisons

Limited joint mobility is a common complication associated with diabetic neuropathy. NP patients from the current

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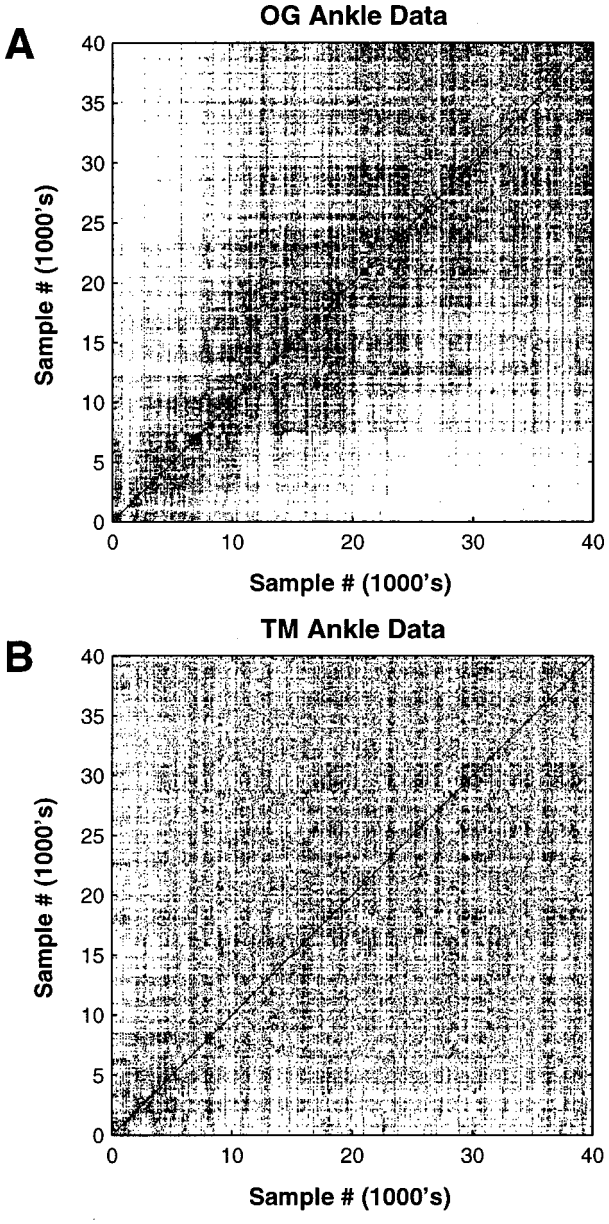

FIG. 6. Example recurrence plots for the ankle joint movements of a typical subject during both OG walking (A) and TM walking (B). Note that the evidence of mild nonstationarity exhibited during the OG walking trial was eliminated during TM walking.

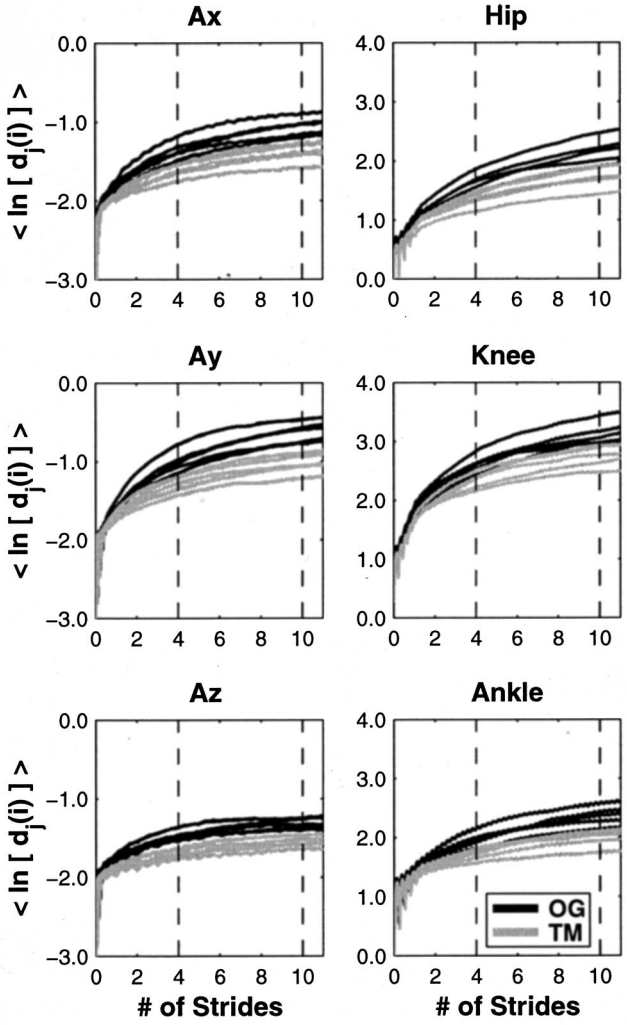

FIG. 7. Logarithmic divergence curves for a typical subject for both OG and TM walking. Five curves are drawn for each condition; one for each of the five 2-min intervals of data that were analyzed. Vertical lines delineate the region across which estimates of λ^* were calculated. Similar results were obtained for all ten subjects.

study showed significantly reduced ROM at both the knee ($p=0.047$) and great toe ($p=0.000$), but not at the ankle ($p=0.298$).[34] NP patients also exhibited some decreases in muscle strength; however, these differences did not reach statistical significance. The minimum buckling forces detectable by NP patients were between 35 and 400 times greater than for CO subjects (Fig. 9). All differences in sensory status were highly statistically significant ($p=0.000$) and demonstrate the severity of the peripheral neuropathy in these patients. Average self-selected walking speeds were also significantly slower for NP patients than CO subjects (NP $=1.24\pm0.21$ m/s; CO$=1.47\pm0.19$ m/s; $p=0.008$), which was consistent with previous findings.[32,33]

GFNN analyses of the time series data from the NP and CO subjects produced results that were strikingly similar to those obtained from the OG and TM comparisons (refer to Fig. 5). The total % GFNN again reached minimum plateau values at $d_E=5$ for all six time series variables and the % GFNN for the Ax and Az accelerations did not completely reach zero. Additionally, the % GFNN for these data were greater for the NP group than for the CO group (Fig. 10). Since the same instrumentation was used to collect the data from all subjects, this pattern may indicate a greater level of dynamical noise[43,49] in these horizontal upper body accelerations for the NP patients than for the CO subjects. A value of $d_E=5$ was used for all time series for all subsequent analyses.

Recurrence plots exhibited no obvious nonstationarities for any of the upper body acceleration data for any of the NP or CO subjects. Mild nonstationarities (similar to Fig. 6) were found in some of the lower extremity time series. In general, these trends were most prevalent at the ankle joint and occurred more often in NP patients than in CO subjects. It should also be noted that a number of subjects in both groups displayed no evidence of nonstationarity in any of the

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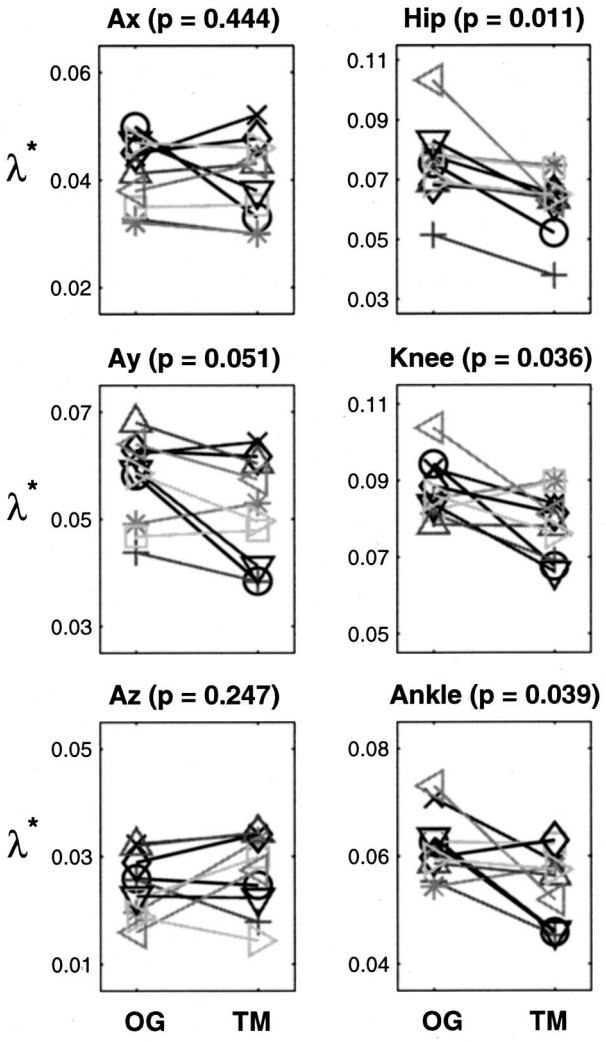

FIG. 8. Average λ* exponents ($<\ln[d_j(i)]>$/stride) for each subject for OG and TM walking. Each line and marker represents the mean values for one subject. Values of $p<0.05$ indicate statistically significant differences between OG and TM walking.

time series collected. Two NP patients, however, displayed marked nonstationarities in their ankle joint movement patterns (Fig. 11). While the underlying causes of these nonstationarities are not known, there were no indications of technical difficulties during data collection, and it is worth noting that this degree of nonstationarity only occurred in these two neuropathic patients and only at the ankle joint, where the neuropathy was most severe. These two time series were deleted from further analyses to avoid having the results be adversely affected by the presence of such strong nonstationarities.

Statistically significant differences between NP and CO subjects for the LSEs were found for four of the six time series measures examined, including all three lower extremity time series variables (Fig. 12). In each of these cases, NP patients exhibited slightly greater LSEs than CO subjects. Furthermore, these differences were exhibited across intermediate length scales that were *above* the noise floor, but *below* the level where the global limit cycle dynamics dominated the scaling behavior. These results are in agreement

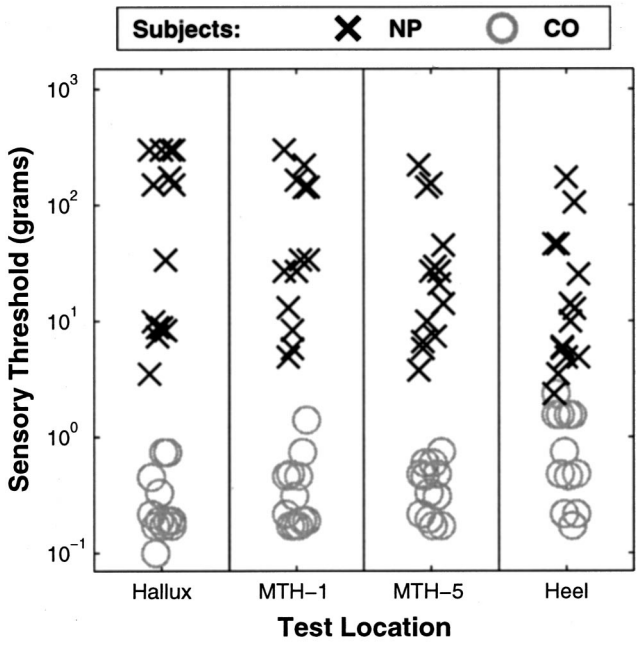

FIG. 9. Sensory thresholds (grams) for NP and CO subjects for each of the four locations tested on the bottoms of the feet: the hallux, first metatarsal head (MTH-1), fifth metatarsal head (MTH-5), and the heel. Horizontal displacements of markers within each column were made only to distinguish between multiple subjects having the same scores.

with the notion that sensory feedback plays a role in adjusting and/or fine-tuning locomotor patterns,[37,38] rather than generating the more global features of those patterns. These more global features are thought to arise largely from loco-

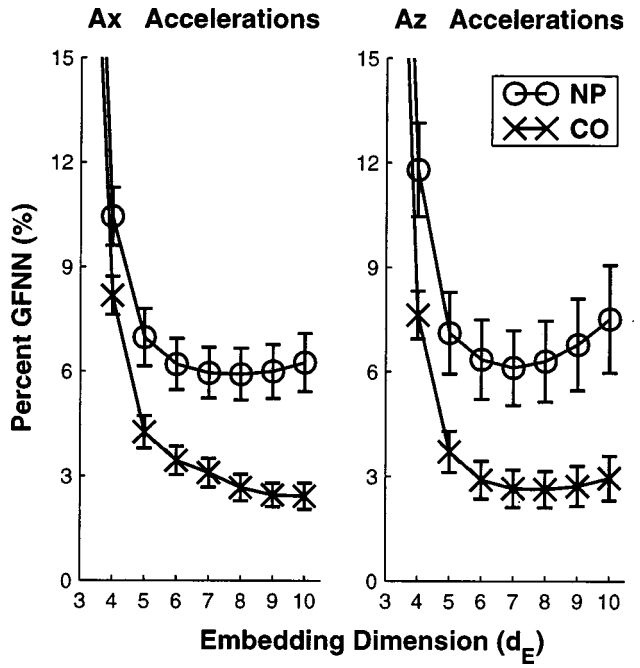

FIG. 10. Averaged GFNN analysis results for NP and CO subjects for anterior-posterior (Ax) and mediolateral (Az) accelerations. Within each subplot, the two curves correspond to the mean results for NP and CO subjects, respectively. Error bars represent between-subject standard errors. Results for all other time series measures were similar to those shown in Fig. 5.

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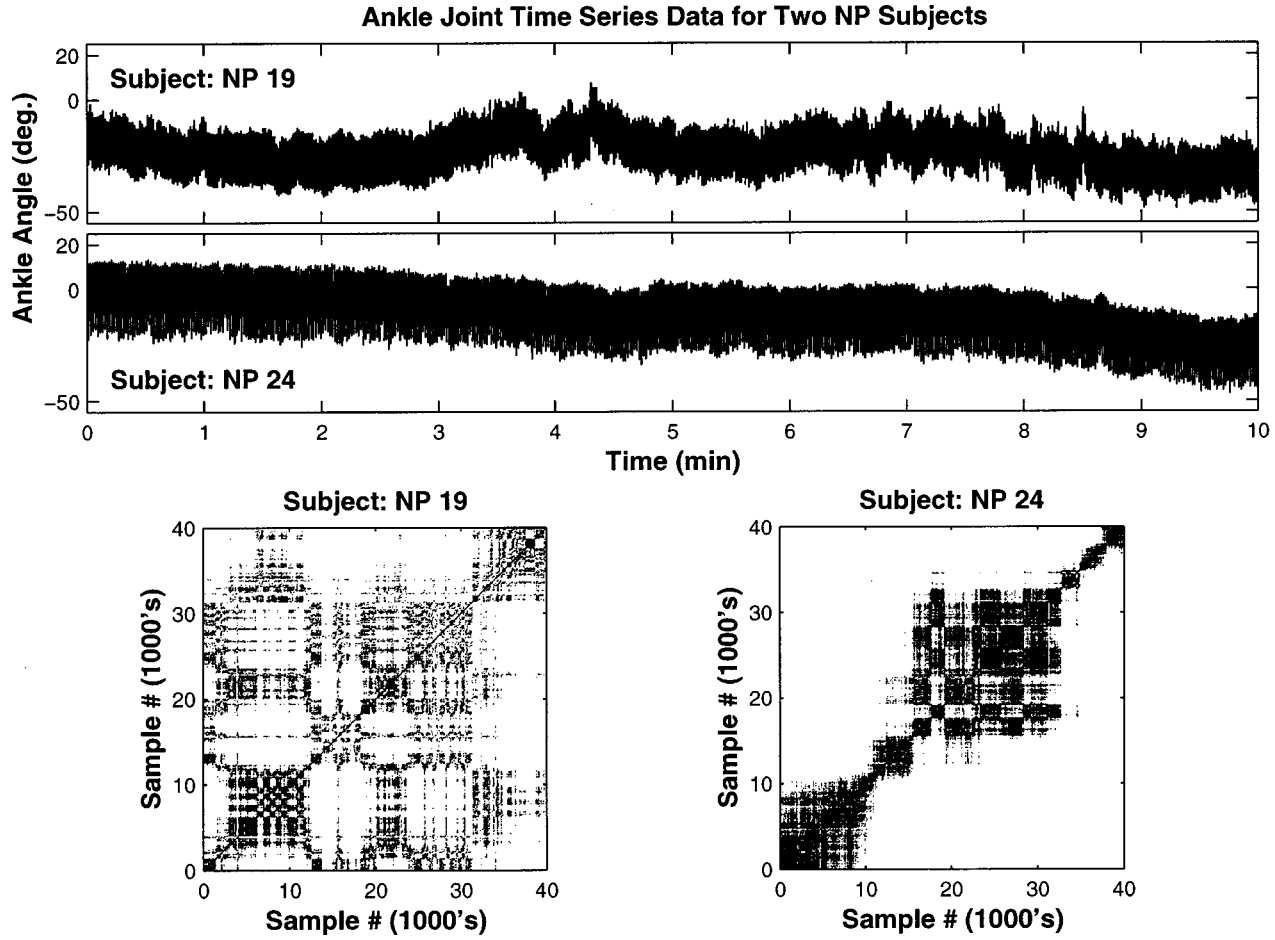

FIG. 11. Time series data and recurrence plots for the ankle joint movements of two of the NP subjects (#19 and #24) where evidence of gross nonstationarities was found. These time series were deleted from further analysis to avoid adversely affecting the results.

motor mechanics and/or central nervous system control, both of which remain intact in the NP patients. It was therefore concluded that loss of peripheral sensation was associated with significant differences in the local scaling structure of walking kinematics at those length scales where it was anticipated that sensory feedback would play the greatest role.

Differences between NP patients and CO subjects for λ^* were statistically significant for upper body accelerations in the horizontal plane in both the anterior-posterior (Ax, $p=0.001$) and mediolateral (Az, $p=0.007$) directions (Fig. 13), and for knee joint movements ($p=0.022$). The multivariate regression analysis[60] demonstrated that walking speed significantly predicted local dynamic stability in the horizontal plane (Fig. 14), but that sensory status did not contribute directly to differences in local dynamic stability, after differences in walking speed and other variables had been accounted for. However, loss of sensation was significantly associated with decreased walking speed, consistent with previous findings.[32–34] Therefore, these results indicate that NP patients, as an indirect result of slowing their self-selected walking speeds, and not as a direct effect of the neuropathy itself, adopt locomotor patterns that result in small but statistically significant improvements in the local dynamic stability of upper body movements in the horizontal plane.[60] This finding supports the clinical hypothesis that by slowing down, NP patients adopt a ''less destabilizing and more conservative'' walking strategy than CO subjects.[33]

C. Surrogate data comparisons

LSE curves exhibited consistent differences between original and surrogate time series (Fig. 15) across all subjects and time series variables examined. At intermediate to small length scales (approximately $\log[r] \leqslant -1.2$ in Fig. 15), the surrogate fluctuations (S1) distorted the local scaling behavior of each time series, resulting in consistently larger LSEs than were obtained for the original (OR) fluctuations. However, the local scaling behavior at larger length scales (from approximately $\log[r] \geqslant -1.8$ to approximately $\log[r] \geqslant -1.2$ in Fig. 15) remained largely intact. This was not surprising, as these structures are determined by the same nominally periodic signal [e.g., Fig. 4(b)] that underlies both the OR and S1 time series. Figure 15 also demonstrates that generating phase-randomized surrogates from the original time series [e.g., from Fig. 4(a)], without first subtracting off the underlying nominally periodic component (S2), destroys the local scaling behavior on *all* length scales, as anticipated. Thus, it was concluded that the local scaling properties at medium-to-small length scales (approximately $\log[r] \leqslant -1.2$ in Fig. 15) in these signals cannot be ascribed to linearly autocorrelated Gaussian noise.

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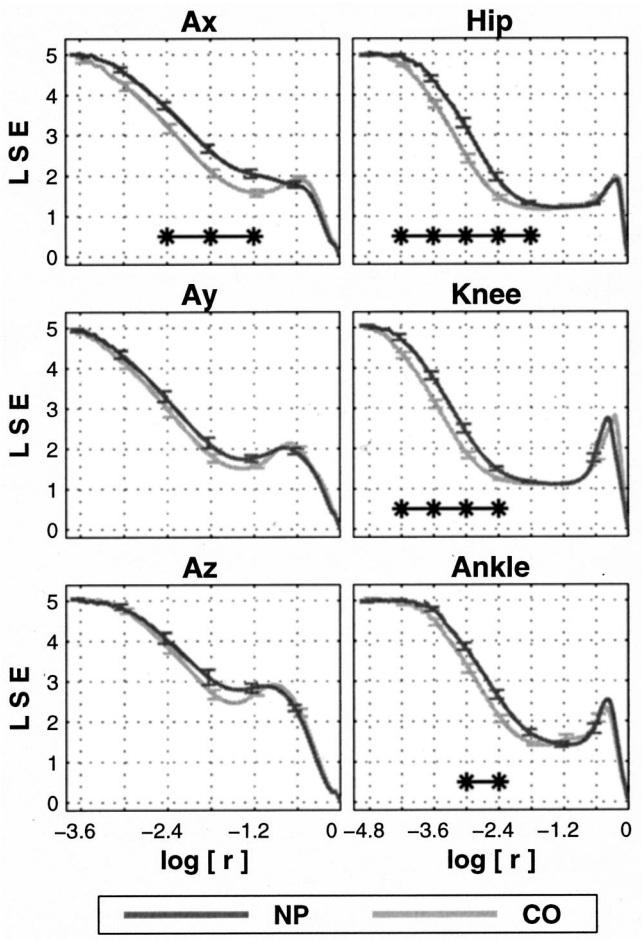

FIG. 12. Average local scaling exponents (LSE) for each time series for NP and CO groups. Error bars denote between-subject standard errors within each group. Vertical gridlines denote points along log[r] where statistical comparisons were made. Stars (*) denote points along log[r] where differences between NP and CO groups were statistically significant ($p<0.008$ for Ax; $p<0.006$ for hip, knee, and ankle).

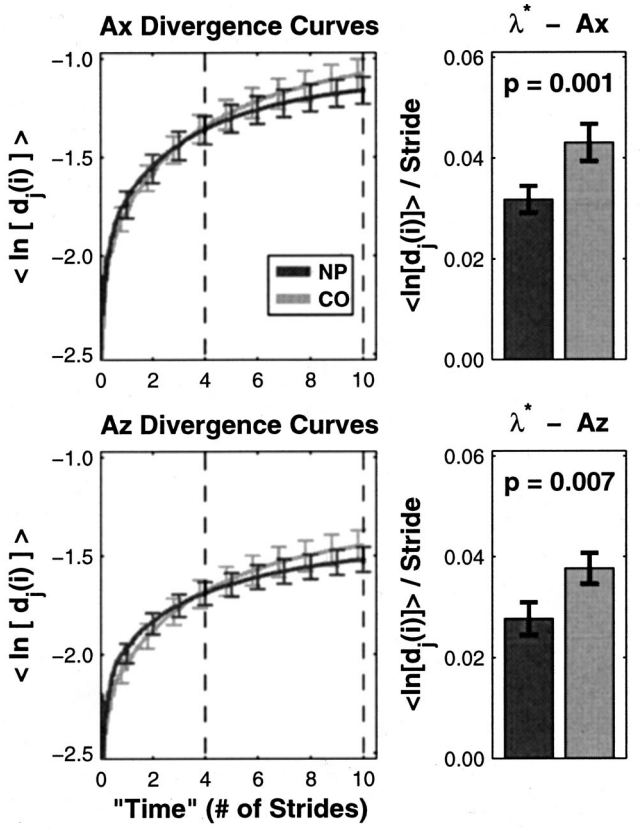

FIG. 13. Average logarithmic divergence curves and λ^* values for anterior-posterior (Ax) and mediolateral (Az) accelerations for NP and CO groups. Error bars denote between-subject standard errors within each group. Vertical lines delineate the region across which estimates of λ^* were calculated.

Furthermore, these surrogate data sets consistently exhibited greater magnitudes of divergence than their corresponding original time series. This can be seen in Fig. 16(a), where the divergence curves for the surrogates (S1) are shifted up, relative to their original (OR) counterparts. As expected, these differences were further magnified by generating phase-randomized surrogates directly from the original time series [S2 in Fig. 16(a)]. Similar shifts occur when stochastic noise is added to a deterministic signal,[17] indicating the progressively increased levels of stochasticity in these surrogate time series (S2>S1>OR). Similar shifts occurred in these curves for all subjects [Fig. 16(b)] for all six time series measures.

The error bars shown on the curves of Fig. 16(b) indicate that there were substantial variations across subjects. However, there was a highly consistent trend within all subjects for the S1 λ^* exponents to be different from the OR λ^* exponents [Fig. 16(c)]. Similar trends were observed for all time series variables for all subject groups. The differences between original (OR) and surrogate (S1) λ^* were statistically significant ($p<0.015$) for all six time series measures evaluated for all three groups of subjects. Therefore, these results further demonstrate that the overlying fluctuations in these nominally periodic human walking data can be clearly distinguished from a linearly autocorrelated Gaussian noise process.[44]

V. DISCUSSION

A. Local dynamic stability of human walking

The primary objectives of the present study were to examine the effects of both walking on a motorized treadmill and the loss of peripheral sensation on the dynamics and local stability of human walking. The results of these analyses demonstrate that walking on a motorized treadmill artificially stabilizes locomotor patterns compared to walking over level ground by small but statistically significant amounts. However, walking on the motorized treadmill also had no effect on the local scaling structure of locomotor kinematics, and in some cases reduced or eliminated subtle nonstationary behavior by enforcing a constant average walking speed. Therefore, the relative impact of these various factors should be taken into account when considering the use of motorized treadmills in future studies on locomotor dynamics and walking stability.

Biomechanical studies have traditionally assumed that walking variability can be equated with stability.[3–6] However, statistical measures of variability do not account for the spatio-temporal structure of time series data and in no way

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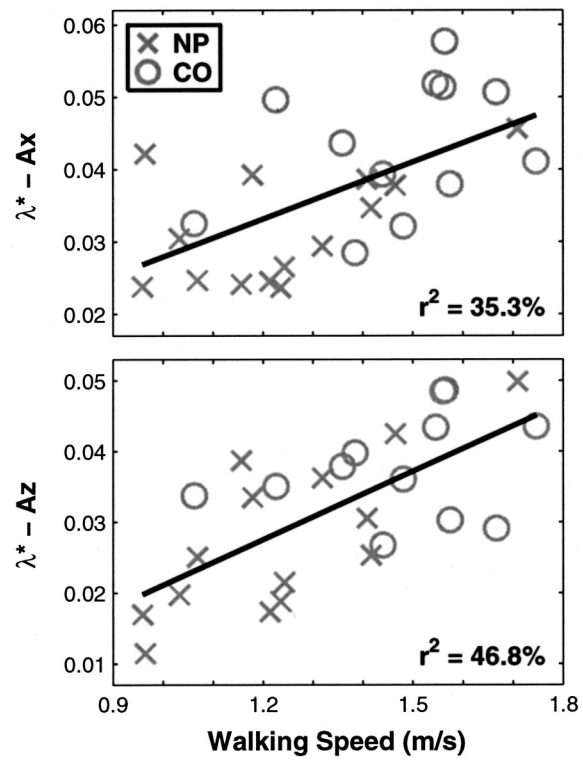

FIG. 14. Relationships between walking speed and local dynamic stability of upper body movements in the horizontal plane (Ax and Az), demonstrating that these correlations were largely independent of diagnostic group (NP vs CO). Straight lines represent linear least squares fits to each data set.

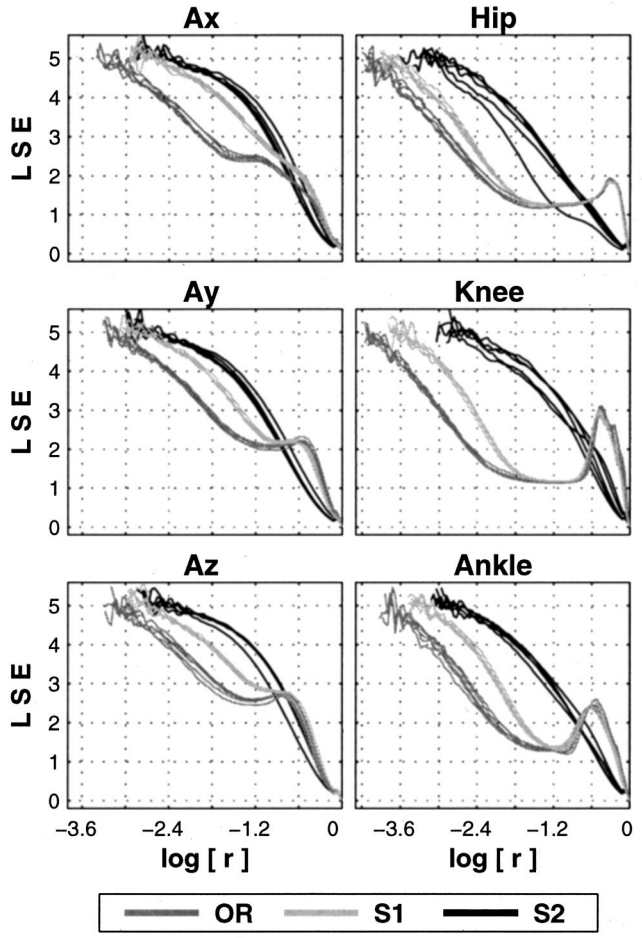

FIG. 15. Local scaling exponents (LSE) for original (OR) and surrogate (S1) time series from a typical subject. Results of generating phase-randomized surrogates directly from the original time series (S2) are also shown for comparison. Five curves are shown for each data set: one for each of the five 2-min intervals of data analyzed. Similar results were obtained for all subjects in all subject groups, indicating distinct differences in local scaling behavior between all three signal types.

quantify the sensitivity of the locomotor system to perturbations. Stride-to-stride standard deviations of locomotor kinematics were also calculated for the subjects examined in the present study, and are reported in detail elsewhere.[26,34] Walking on a motorized treadmill led to reductions in the variability of lower extremity kinematics of between 4% and 17% compared to overground walking as well as enhanced local dynamic stability.[26] However, correlations between these measures of variability and λ^* were largely negative and generally weak ($-23\% < r^2 < +6\%$). NP patients demonstrated no differences in upper body variability and between 2% and 16% *greater* variability for lower extremity kinematics compared to CO subjects.[34] However, local dynamic stability measures in these subjects simultaneously exhibited *improvements* for upper body movements (Fig. 13) and no significant differences for lower extremity kinematics. Correlations between variability measures and λ^* in these subjects were again largely negative and generally weak ($-28\% < r^2 < +7\%$). These results demonstrate that measurements of stride-to-stride variability and local dynamic stability quantify fundamentally different aspects of locomotor behavior and that the concepts of variability and local dynamic stability must be carefully distinguished in studies of human locomotion.

Furthermore, when applied to patients with diabetic neuropathy, this approach of equating variability and stability has resulted in a paradox between the hypothesis that slower walking speeds should lead to improved stability[33] and the finding that slower speeds in fact result in increased variability.[25,34–36] It must be noted that all of the NP subjects in the present study had been living with significant sensory loss for many years. Therefore, the measurements made on these patients reflected not only the direct effects of the neuropathy itself, but also of any adaptations these subjects had developed over the years to compensate for their sensory loss. In the present study, the decreases in λ^* for upper body accelerations in the horizontal plane of the NP patients were significantly predicted by reductions in self-selected walking speeds in these patients (Fig. 14). Furthermore, these NP patients consistently exhibited lower λ^* values than CO subjects for all six time series measures quantified (although not all of these trends were statistically significant). The results of the present study therefore confirm the hypothesis that decreases in walking speed represent a compensatory strategy used by NP patients[33] that results in an improvement in the local dynamic stability of upper body movements during unimpeded level walking.

Thus, the increased risk of falling in NP patients does not appear to be due to the inability of these patients to generate locally stable locomotor rhythms. All of the NP

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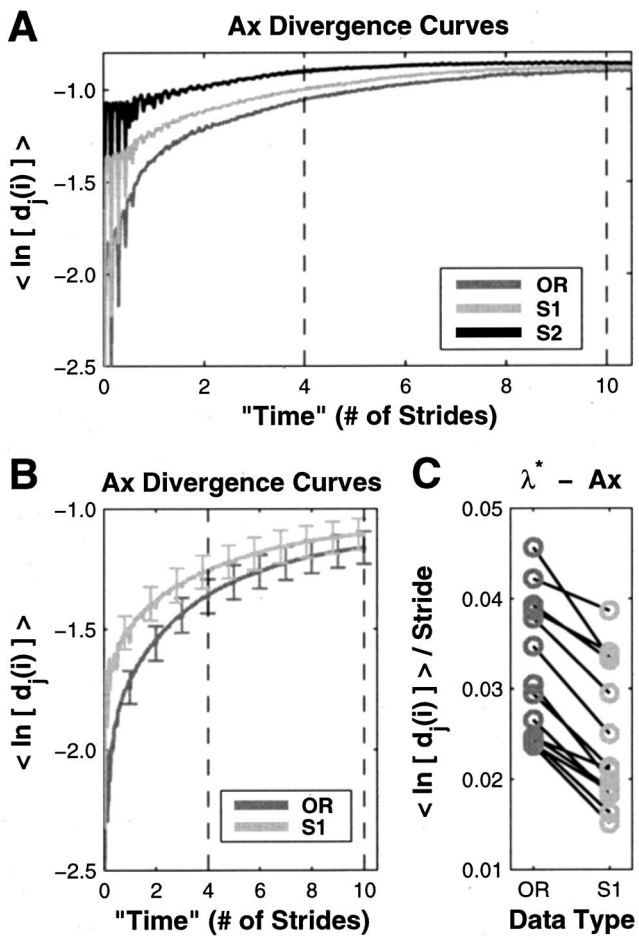

FIG. 16. (a) Average logarithmic divergence curves for original (OR) and surrogate (S1) data sets for the anterior-posterior (Ax) accelerations for a typical subject. Results of generating phase-randomized surrogates directly from the original time series (S2) are shown for comparison. (b) Average logarithmic divergence curves for OR and S1 data sets for Ax accelerations for all NP patients. Error bars represent between-subject standard errors within each group. (c) Corresponding λ^* exponents for OR and S1 data sets for Ax accelerations for all NP patients. Each line and marker set denotes the average result for one NP subject. Overall differences between OR and S1 exponents were highly statistically significant ($p=0.000$). Similar results were obtained for all time series measures for all subject groups.

patients in the present study were easily able to walk without incident in the open, well-lit, and unobstructed environment used in the study protocol. Their increased risk of falling is therefore more likely due to their inability to develop and execute appropriate avoidance and/or response strategies when faced with unexpected obstacles or large-scale perturbations during locomotion. Such large-scale perturbations would necessarily challenge these patients' *global* limits of stability. Some researchers have tested these global stability limits by physically tripping subjects.[63,64] However, such studies are difficult to undertake and potentially hazardous to the subjects involved. Future research on the dynamic stability of patients who have significantly increased risks of falling should focus on carefully varying the types and magnitudes of perturbations given during locomotion. Such experiments would allow researchers to gain a better understanding of how patients with different pathologies explore the stable regions within their state spaces and of where the global stability boundaries of their movement patterns lie.

B. Dynamical structure of human walking

In addition to making possible the precise characterization of local dynamic stability, the dynamical systems approach adopted in the present paper promises the additional benefit of shedding new light on the underlying structure of the human locomotor system. As a result of the analyses carried out in this study, the first steps were taken in this direction. It is important to remember, however, that in undertaking this project there is little *a priori* knowledge of the minimal model structure needed to generate the observed walking dynamics. Thus, unlike the situation in many branches of mathematical physics where known, first-principles balance laws provide detailed knowledge about model structure, one can not hope as the result of this one study to definitively situate the model in the deterministic-stochastic, linear-nonlinear plane.

One of the fundamental questions in neuromuscular control research is Bernstein's historical (1935) *degrees of freedom problem*[65]: How are the very many degrees of freedom of the human body coordinated to produce smooth, rational movements? Perhaps the most basic property of the locomotion data observed in the present study was its apparent low dimensionality. An embedding dimension of $d_E=5$ was found to be adequate for all of the time series measures quantified, for all subjects, and for all test groups. Thus, it was concluded that, over the scales observable in this experiment (as defined by the overall signal-to-noise ratio and precision of the data across all subjects), the global locomotor dynamics is effectively quite low dimensional and, if predominantly deterministic, should be expected to be well modeled by a dynamical system with as few as five state space dimensions. While not specifying precisely *how* it occurs, this result demonstrates that human walking involves a substantial ''collapse of dimension''[66] from the very high dimensional state space of all possible movements to an approximately five-dimensional subspace.

Furthermore, the LSEs of Fig. 2 initially suggested that a reasonable value for the correlation dimension for this system might be $D_2 \approx 1$, with the scaling structures at smaller length scales being approximated as noise. This implies that locomotor kinematics (particularly for lower extremity movements) might be satisfactorily described as a noisy limit cycle process. These findings are in line with recent findings that rhythmic movements generated by pathological tremors might be adequately modeled by a nonlinear stochastic second-order process.[57] However, the surrogate (S1) data results from the present study demonstrate that the ''noise'' that generates the drifting of the LSEs away from linearity is not simply linearly autocorrelated Gaussian noise. Thus, while some form of a stochastically driven nonlinear oscillator may provide an appropriate model of global locomotor dynamics, more complex stochastic inputs may be required to adequately capture the details of the scaling behaviors observed at smaller length scales. Initial experiments in rhythmic movement tasks suggest that such complex noise

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patterns might be related to the inherent noise and time delays in the central nervous system.[42] However, the exact nature and underlying biological source of these locomotor fluctuations remains unknown.

The question of to what extent the minimal model required for the observed locomotor behavior needs to be, on the one hand, linear or nonlinear, or on the other hand, deterministic or stochastic, is much more difficult to answer. In the present context, it is essentially impossible to conclusively answer this question in the absence of supporting *a priori* (theoretical) information. However, it is possible in specific cases, to show the degree to which proposed model structures are capable of capturing observed data. Existing mathematical models of human locomotion range from extremely simple mechanical models[13,14,66] to highly complex neuro-musculo-skeletal models.[10,11] Continued development of methods similar to those employed in the present study will enable researchers to test the necessity and validity of each of these models by directly comparing the dynamical structure of the model output with that of human walking.

C. ''Noise'' and ''chaos'' in human walking

In the study of human locomotion, the natural variations that occur from stride to stride have most often been viewed simply as noise. The results of the present study demonstrate that these fluctuations can be clearly distinguished from the output of a linear system driven by Gaussian noise. It should be kept in mind that the method developed for generating the surrogate data in the present study was designed to randomize only the spatial fluctuations in these signals, and not the temporal variations, thus producing a conservative test of the null hypothesis. Even from this conservative test, substantial and highly consistent differences were exhibited between the original (OR) and surrogate (S1) time series (Figs. 15 and 16).

However, the rejection of the null hypothesis does not prove that these fluctuations are ''chaotic.'' In fact, the nonconstant values of the LSEs over the length scales where these fluctuations dominate the dynamical behavior of the system suggest the opposite. The present analysis does not, for example, preclude the possibility that these fluctuations may have resulted from a static nonlinear transform of a linear Gaussian process.[44] Such a situation might arise if the original data were passed through a nonlinear filter, or if a nonlinear measurement function were used (e.g., if $y(t) = \arctan[x(t)]$ were analyzed instead of $x(t)$ itself[43]). In the present experiments, the measurements examined were linear measures of joint angle and acceleration, the data were collected while operating well within the linear range of the electronics, and the data were analyzed in their raw and unfiltered form. Thus, while it is possible that these fluctuations resulted from a static nonlinear transform of a linear Gaussian process, we conclude that any such transformation must have been the consequence of physiological processes within the locomotor system itself. The question of whether or not the natural physiology of human movement incorporates such nonlinear filtering properties is indeed an interesting question. However, such pursuits are left to future research.

While the positive λ^* computed in the present study may seem to suggest the possibility of an underlying chaotic mechanism for these fluctuations, experimental data always contain noise (from the system itself and/or from the instrumentation). Such noise also produces sensitivity to initial conditions and can fool many algorithms into signaling chaos where it does not in fact exist.[43,44] Since no purely deterministic *a priori* model for human locomotor control exists, specific conclusions about the underlying source of these positive λ^* exponents are difficult to make. However, the fact that these fluctuations could be clearly distinguished from a linearly autocorrelated Gaussian process suggests that stride-to-stride fluctuations in locomotor output may be at least partly under the control of deterministic central nervous system processes. Such fluctuations may reflect corrective adjustments generated to maintain balance and smooth movements during ongoing gait.[37,38] The nature and origin of these stride-to-stride fluctuations therefore deserve much further study.

VI. CONCLUSIONS

This paper has demonstrated the utility of using a dynamical systems approach to analyzing data from human locomotion. In particular, the estimation of average maximum *finite-time* Lyapunov exponents (λ^*) enabled a more precise characterization of the local dynamic stability of human walking than previously achieved. Small but statistically significant reductions in λ^* were found for TM walking compared to OG walking. Thus, the more constrained artificial environment imposed by the motorized treadmill slightly stabilizes natural OG walking patterns. The careful analysis of local dynamic stability further enabled the present study to resolve a paradox that arose in previous studies from using stride-to-stride variability as a measure of walking stability in NP patients. By slowing their self-selected walking speeds, NP patients are, by a small but statistically significant amount, more locally stable than CO subjects, even though they continue to exhibit greater stride-to-stride standard deviations in their locomotor kinematics.[34,60] Thus, this paper conclusively demonstrates that in the context of human locomotion, the concepts of variability and local dynamic stability must be clearly differentiated. More importantly, the work presented here demonstrates that the greater propensity of NP subjects to fall must be due to changes in global rather than local stability limits.

As a collateral benefit of the state space approach taken in the present study, an initial characterization of the locomotor dynamical system was carried out. The required effective state space dimensionality was found to have the same low value of $d_E = 5$ in all cases. Examination of the local scaling behavior within these state spaces revealed that these locomotor patterns did not exhibit the scale-invariant fractal structure associated with deterministic chaos. Differentiating ''chaotic'' from ''nonchaotic'' dynamics can be helpful in situations where such distinctions are anticipated by *a priori* deterministic models. However, such distinctions can be ''unnecessary and misleading''[67] when applied to systems where no such model exists, as is the case with human loco-

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motion. In fact, it was anticipated that locomotor dynamics would be affected by a number of both deterministic and stochastic influences. The present analyses have confirmed this expectation and have revealed a number of unique features of these different influences. Furthermore, using a modified surrogate data method, it was shown that the stride-to-stride variations in the time series from these experiments could be statistically distinguished from the output of a simple linear system driven by Gaussian noise. This contradicts the traditional assumption of most gait analyses that stride-to-stride variations are merely ''noise.'' While preliminary, the results of these initial experiments have clearly demonstrated that nonlinear time series methods based on state space reconstruction can provide significant and useful insight into the neuromuscular control of locomotion in ways that traditional linear statistical approaches cannot.

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