Nonlinear dynamical model of human gait

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(Received 9 December 2002; published 20 May 2003)

We present a nonlinear dynamical model of the human gait control system in a variety of gait regimes. The stride-interval time series in normal human gait is characterized by slightly multifractal fluctuations. The fractal nature of the fluctuations becomes more pronounced under both an increase and decrease in the average gait. Moreover, the long-range memory in these fluctuations is lost when the gait is keyed on a metronome. Human locomotion is controlled by a network of neurons capable of producing a correlated syncopated output. The central nervous system is coupled to the motocontrol system, and together they control the locomotion of the gait cycle itself. The metronomic gait is simulated by a forced nonlinear oscillator with a periodic external force associated with the conscious act of walking in a particular way.

DOI: 10.1103/PhysRevE.67.051917 PACS number(s): 87.18.Sn, 05.45.−a, 05.45.Df, 87.17.Nn

I. INTRODUCTION

Walking is a complex process which we have only recently begun to understand through the application of nonlinear data processing techniques [1–4] to study interval data. It has been known for over a century that there is a variation of 3–4% in the stride interval of humans during walking [5], and only in the last decade did Hausdorff *et al.* [1] demonstrate that the stride-interval time series exhibits long-time correlation, suggesting that the phenomenon of walking is a self-similar, fractal activity. Subsequent studies by West and Griffin [3,4] supported the conclusion that the human gait time series is fractal. However, more recently it was determined that these time series, rather than being monofractal, are weakly multifractal [6,7]. In particular, in Ref. [7] the interested reader will find a detailed fractal and multifractal analyses of the stride-interval datasets that here we try to model.

Human locomotion is known to be a voluntary process, but it is also regulated through a network of neurons called a central pattern generator (CPG) [8], capable of producing a syncopated output. The early nonlinear dynamical models of CPGs for gait assumed that a single nonlinear oscillator be used for each limb participating in the locomotion process [9]. Therefore a quadruped requires the coupling of four nonlinear oscillators to determine the correct phase relations among the four legs in order to distinguish between various modes of locomotion, that is, walking, trotting, cantering, and galloping. More recent dynamical models, using the property of synchronization of nonlinear dynamical systems, allow for neurons within an assembly to become enslaved to a single rhythmic muscular activity. Thus, rather than having a separate nonlinear oscillator for each limb, it is possible to have a single CPG to determine how we walk.

The model that we present here, the super CPG (SCPG), assumes that the central nervous system is coupled to the motocontrol system, and together they control the locomotion of the gait cycle. We stress that it is the period of the gait cycle that is ultimately measured in these stride-interval experiments, and not the neural firing activity. The dynamics of human gate may also be voluntarily forced, for example, by following the frequency of a metronome. We model the complex gait system by assuming that the amplitude of the impulses of the correlated firing neural centers regulates only the unperturbed inner frequency of a nonlinear forced Van der Pol oscillator [10] that mimics the gait cycle. The stride interval is assumed to coincide with the actual period of the Van der Pol oscillator. In this way the gait frequency may differ slightly from the potential frequency induced by the neural firing activity. In fact, the chaotic behavior of nonlinear oscillators, such as the Van der Pol oscillator, allows a more complex behavior that may be controlled also by a constraint that forces the oscillator to follow a particular fixed frequency.

The SCPG model is tested on a dataset available in Ref. [11]. These data were originally collected and used by Hausdorff *et al.* [12] to determine the dependence of the fractal dimension of the time series on changes of the average rate of walking. These data contain the stride-interval time series for ten healthy young men walking at a slow, normal, and fast pace, for a period of 1 h. The same individuals are then requested to walk at a pace determined by a metronome set at the average slow, normal, and fast paces for 30 min to generate a second dataset.

The fractal and multifractal analyses of the data are done by studying the estimated distribution of the local Hölder exponents using wavelet transforms. The interested reader will find a detailed discussion about the analysis method, in particular, in Ref. [13], and in Refs. [14–16]. In Ref. [7] we also discuss this method in detail. To better understand the meaning of the Hölder exponent h, we recall that the relation between the Hölder and Hurst exponent H [17] in the continuum limit of a monofractal noise is $h=H-1$ according to the notation adopted in Refs. [7,13]. According to this definition, the autocorrelation function [18] of a fractal noise $\{\xi_i\}$ is related to the Hölder exponent h via the relation

$$C(r)=\frac{\langle \xi_i \xi_{i+r}\rangle}{\langle \xi_i^2\rangle}\propto r^{2H-2}=r^{2h}, \tag{1}$$

or, equivalently, in the power spectrum representation

$$S(f)=\int_{-\infty}^{\infty} C(r)e^{-i2\pi fr}dr \propto f^{1-2H}=f^{-1-2h}. \qquad (2)$$

Consequently, $h=0$ corresponds to pink or $1/f$ noise, $-1<h<-0.5$ corresponds to antipersistent noise, $h=-0.5$ corresponds to uncorrelated Gaussian noise, $-0.5<h<0$ corresponds to correlated noise, $h=0.5$ corresponds to Brownian motion, and $h=1$ corresponds to black noise [26].

By estimating the Hölder exponents and their spectra using a wavelet transform [13], we have shown [7] that the stride-interval time series is weakly multifractal with a main fractality close to that of $1/f$ noise. The time series is sometimes nonstationary and its fractal variability changes in the different gait mode regimes [7]. In particular, the persistence as well as the multifractality of the stride-interval time series tend to increase for both slow and fast paces, above that of the normal paces. Moreover, if the pace is constrained by a metronome, the stochastic properties of the stride-interval time series change significantly, from persistent to antipersistent fluctuations, but, in general, in each case there is a reduction in the long-term memory and an increase in randomness.

In Sec. II, we give a short introduction to the phenomenon of locomotion, the traditional methods for modeling using the CPG, and review the data processing used to establish the fractal behavior of the stride-interval time series. Section III reviews the stochastic properties of the normal and metronomic gaits under different various pace velocities, slow, normal, and fast. In Sec. IV, we present the mathematical details of the SCPG model. In Sec. V, we compare the results of computation using the SCPG model with the phenomenological data. Finally, in Sec. VI we draw some conclusions.

II. CENTRAL PATTERN GENERATOR AND LOCOMOTION

Walking consists of a sequence of steps. These steps may be partitioned into two phases: a stance phase and a swing phase. The stance phase is initiated when a foot strikes the ground and ends when it is lifted. The swing phase is initiated when the foot is lifted and ends when it strikes the ground again. The time to complete each phase varies with the stepping speed. A stride-interval is the length of time from the start of one stance phase to the start of the next stance phase.

Traditionally, the legged locomotion of animals is understood through the use of a CPG, an intraspinal network of neurons capable of producing a syncopated output [8,19]. The implicit assumption in such an interpretation is that a given limb moves in direct proportion to the voltage generated in a specific part of the CPG. Experiments establishing the existence of a CPG have been done on animals with spinal cord transections. Walking, for example, in a mesencephalic cat, a cat with its brain stem sectioned rostral to the superior colliculus, is very close to normal, on a flat, horizontal surface, when a section of the midbrain is electrically stimulated. Stepping continues as long as a train of electrical pulses is used to drive the stepping. This is not a simple linear response process because changing the frequency of the driver has little effect of the walking cycle [20]. However, since the frequency of the stepping increases in proportion to the amplitude of the stimulation, we can conclude that the variation in the stride-interval of humans is related to the fluctuation of the amplitude of the impulses of the firing neural centers.

As Collins and Richmond [8] point out, in spite of the studies establishing the existence of a CPG in the central nervous system of quadrupeds, such direct evidence does not exist for a vertebrate CPG for biped locomotion. Consequently, these and other authors have turned to the construction of models, based on the coupling of nonlinear oscillators, the *hard-wired* CPG, to establish that the mathematical models are sufficiently robust to mimic the locomotion characteristics observed in the movements of segmented bipeds [21], as well as in quadrupeds with the opportune symmetry properties [9,22]. These characteristics, such as the switching among multiple gait patterns, are shown to neither depend on the detailed dynamics of the constituent nonlinear oscillators nor on their interoscillator coupling strengths [8].

As we mentioned in the Introduction, it has been known for over a century that there is a variation in the stride-interval of humans during walking of $\approx 3-4\%$ [5]. This random variability has been shown [1,3,4,6,12] to exhibit long-time correlations, and suggested that the phenomenon of walking is a self-similar, fractal, activity. The existence of fractal time series better suggests that the nonlinear oscillators needed to model locomotion operate in the unstable, that is, in the chaotic regime.

A stochastic version of a CPG was developed by Hausdorff *et al.* [6,12] to capture the fractal properties of the interstride-interval time series. This stochastic model was later extended by Ashkenazy and co-workers [23,24] to describe the changing of gait dynamics as we develop from childhood to adulthood. The model is essentially a random walk on a correlated chain, where each node of the chain is a neural center of the kind discussed above, and with a different frequency. This random walk is found to generate a fractal process, with a multifractal width that depends parametrically on the range of the random walker's step size. Ashkenazy and co-workers [23,24] focused on explaining the changes in the gait time series during maturation, using their stochastic CPG model.

Herein we extend the previous models by assuming that gait dynamics are regulated by a stochastic correlated CPG similar to that of Ashkenazy and co-workers [23,24], coupled to the nonlinear oscillators needed to model locomotion in the unstable, forced, and chaotic regimes. We show that two parameters, the average frequency f_0 and the intensity A of the forcing component of the nonlinear oscillator, are sufficient to determine both the fractal and multifractal variabilities of human gait under normal, stressed, and metronomic conditions, using the SCPG model.

III. HUMAN GAIT ANALYSIS

In this section, we summarize the main fractal and multifractal characteristics of the stride-interval of the human gait

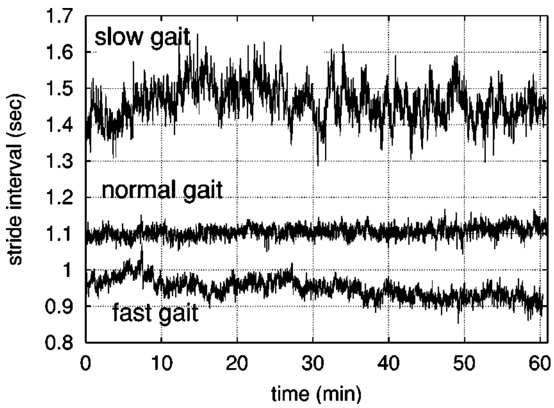

FIG. 1. Stride interval for slow, normal, and fast gaits. The period of time over which measurements were done is approximately 1 h.

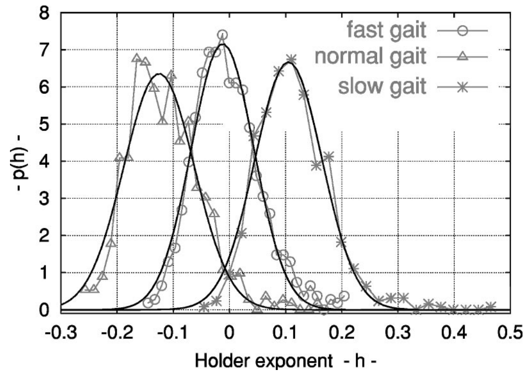

FIG. 3. Histogram and probability density estimation of the Hölder exponents: slow (star) ($h_0 = 0.105$, $\sigma = 0.060$), normal (triangle) ($h_0 = -0.125$, $\sigma = 0.063$), and fast (circle) ($h_0 = -0.012$, $\sigma = 0.056$) gaits for a single individual. The fitting curves are Gaussian functions with average h_0 and standard deviation σ.

data that we discussed in detail elsewhere [7]. More details regarding the collection of data can be found in Ref. [11] from where we downloaded the data and in Refs. [7,12].

The downloaded datasets analyzed in Ref. [7] consist of the gait time series of ten persons in the three different conditions of slow, normal, and fast walking. Each time series is $\approx$1-h long for unconstrained walking for slow, fast, and normal walking, see, for example, Fig. 1. Similarly, each time series is $\approx$30-min long for metronomically constrained walking for slow, fast, and normal walking, see, for example, Fig. 2. Participants in the study had no history of any neuromuscular, respiratory, or cardiovascular disorders. They were not taking any medications and had a mean age of 21.7 yr (range: 18–29 yr); mean height 1.77 ± 0.08 m and mean weight 71.8 ± 10.7 kg. All subjects provided informed written consent. Subjects walked continuously on level ground around an obstacle-free, long (either 225 or 400 m), approximately oval path and the stride-interval was measured using ultrathin, force sensitive switches taped inside one shoe. For the metronomic constrained walking, the individuals were told only once, at the beginning of their walk, to synchronize their steps with the metronome.

In Ref. [13], Struzik introduces a method to estimate the local Hölder exponents of a time series. This author shows

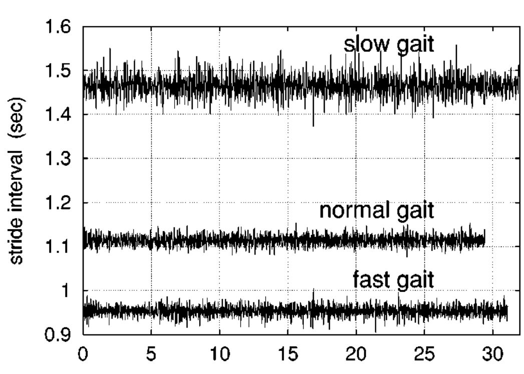

FIG. 2. Stride intervals for slow, normal, and fast gaits for metronomic-triggered walking. The total period of time is $\approx$30 min.

that the fractal properties of a dataset can be studied by determining the mean value $\overline{h}$ of the distribution of the Hölder exponents. The details of the method can be found in Refs. [7,13]. Moreover, Struzik also shows that a monofractal time series of finite length presets a nonzero width of the distribution of the Hölder exponents. Therefore, the existence of such a nonzero width can be a source of confusion between a monofractal time series of finite length and a truly multifractal time series. A multifractal time series can be distinguished from a monofractal time series of the same length only if the width of its Hölder-exponent distribution is significantly larger than that of a corresponding monofractal time series with the Hurst exponent $H = \overline{h} + 1$. To address this problem in Ref. [7] we suggest that given a dataset of length N, its distribution of the Hölder exponents estimated by using Struzik's algorithm can be approximately fitted by a Gaussian distribution of the type

$$g(h) = \frac{1}{\sqrt{2\pi}\sigma} \exp\left[-\frac{(h-h_0)^2}{2\sigma^2} \right], \tag{3}$$

where the value h_0 is a good approximation to $\overline{h}$. Usually, h_0 is slightly larger than $\overline{h}$ because the distribution of the Hölder exponents presents a slightly positive skewness. The standard deviation σ is considered a good indicator of the width of the distribution. Then, we generate many artificial datasets of fractal noise of finite length N characterized by a Hurst coefficient $H = \overline{h} + 1$ and study the distribution of the monofractal widths σ_F by using a fit with Eq. (3). Finally, if σ is larger than σ_F and this is statistically significant, we conclude that the original time series is multifractal.

By applying the above method [7] we determined that typical distributions of the Hölder exponents, for unconstrained walking of a single individual, are of the type depicted in Fig. 3. Figure 4 shows the average distributions of the Hölder exponents for the cohort of ten walkers. Figures 3 and 4 show that stride-interval time series for human gait are characterized by strong persistent fractal properties very close to that of the $1/f$ noise, $h \approx 0$. However, normal gait is

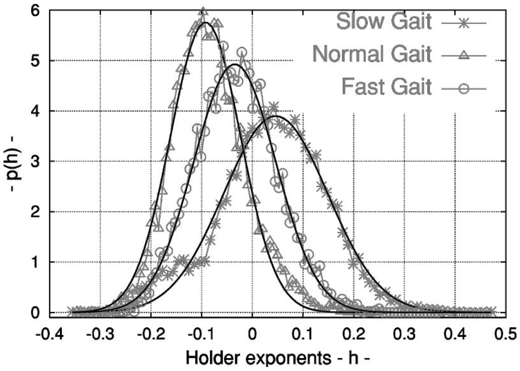

FIG. 4. Histogram and probability density estimation of the Hölder exponents for the three walking groups are shown: slow (star), normal (triangle), and fast (circle) gaits. Each curve is an average over the ten cohorts in the experiment. By changing the gate mode from slow to normal, the Holder exponents h decrease but from normal to fast they increase. There is also an increase of the width of the distribution σ by moving from the normal to the slow or fast gaits mode. The fitting curves are Gaussian functions: slow (star) ($h_0=0.046$, $\sigma=0.102$), normal (triangle) ($h_0=-0.092$, $\sigma=0.069$), and fast (circle) ($h_0=-0.035$, $\sigma=0.081$) gaits.

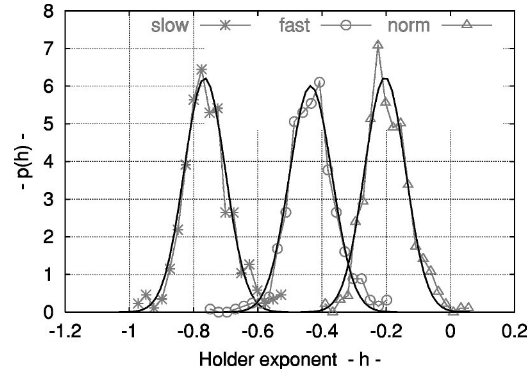

FIG. 5. Metronomic walking for a single individual. Histogram and probability density estimation of the Hölder exponents: slow (star) ($h_0=-0.765$, $\sigma=0.064$), normal (triangle) ($h_0=-0.204$, $\sigma=0.064$), and fast (circle) ($h_0=-0.436$, $\sigma=0.066$).

usually slightly less persistent than both slow and fast gaits. The slow gait has the most persistent fluctuations and may present nonstationary properties, $h>0$. The slow gait fluctuations may also deviate most strongly from person to person. The higher values of the Hölder exponents for both slow and fast gaits, relative to normal gait, may be explained as due to a stress condition that increases the persistency and, therefore, the long-time correlation of the fluctuations. Moreover, the regular curves of Fig. 4 show that unconstrained walking is characterized by fractal properties that do not change substantially from one individual to another. Finally, a careful comparison of the widths of the distributions of the Hölder exponents for the different gaits with the widths for a corresponding monofractal noise dataset of the same length has proven that the stride-interval of human gait is only weakly multifractal [7]. However, the multifractal structure is slightly more prominent for fast and slow gaits than for normal gait.

Figure 5 shows typical distributions of the Hölder exponents for metronome-constrained walking [7], which is little different from the histograms in Fig. 3. Figure 6 shows the average distributions of the Hölder exponents for all ten walkers. The figures clearly indicates that under the constraint of a metronome, the stride-interval of human gait becomes more random and the strong long-time persistence of the $1/f$ noise is lost for some individuals. The data present a large variability of the Hölder exponents from persistent to antipersistent fluctuations, that is, the exponent spans the entire range of $-1<h<0$. However, the metronome constraint usually has a relatively minor effect upon individuals walking normally, the second peak at low Hölder exponents in Fig. 6 being attributable to a single person, who has difficulty with the external cadence. Probably, by walking at a normal speed an individual is more relaxed and he or she walks more naturally. The fast gait appears to be almost an uncorrelated noise because the distribution of the Hölder exponents is centered close to $h=-0.5$ characteristic of Gaussian or uncorrelated random noise. Finally, the slow gait presents a large variability from persistent to antipersistent fluctuations.

We notice that some individuals may be unable to walk at a given cadence and their attempts to synchronize the pace result in a continual shifting of the stride-interval longer and shorter in the vicinity of an average. For these individuals the phasing is never right and this gives rise to a strong antipersistent signal for all three gait velocities.

In summary, the stride-interval of human gait presents a complex behavior that depends on many factors. Walking is a strongly correlated neuronal and biomechanical phenomenon which may be strongly influenced by two different stress mechanisms: (a) a natural stress that increases the correlation of the nervous system that regulates the motion at the changing of the gait regime from a normal relaxed condition to a consciously forced slower or faster gait regime, (b) a psychological stress due to the constraint of following a fixed external cadence such as a metronome. The metronome causes the breaking of the long-time correlation of the natural pace and generates a large fractal variability of the gait

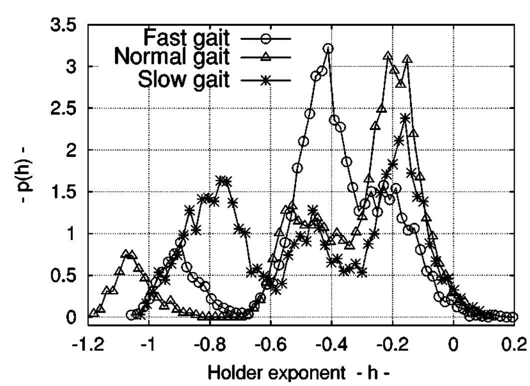

FIG. 6. Metronomic walking. Histogram estimation of the Hölder exponents for the three walking groups: slow (star), normal (triangle), and fast (circle) gaits. Each curve is an average over the ten cohorts in the experiment.

regime. In the following section, we present a SCPG model that is able to reproduce these properties.

IV. THE SCPG MODEL FOR HUMAN GAIT

In this section, we introduce a model of locomotion that governs the stride-interval time series for human gait. As anticipated in the previous sections the model has to simulate a CPG [8] capable of producing a syncopated correlated output associated with a motocontrol process of the gait cycle. Moreover, the model incorporates two separate and distinct stress mechanisms. One stress mechanism, which has an *internal* origin, increases the correlation of the time series due to the change in the velocity of the gait from normal to the slower or faster regimes. The second stress mechanism has an *external* origin and decreases the long-time correlation of the time series under the frequency constraint of a metronome. We model this complex phenomenon by assuming that the intensity of the impulses of the firing neural centers regulates only the inner virtual frequency of a forced Van der Pol oscillator [10]. The observed stride-interval is assumed to coincide with the actual period of each cycle of the Van der Pol oscillator; a period that depends on the unperturbed inner frequency of the oscillator, the amplitude of the forcing function, and the frequency of the forcing function.

Since the frequency of the stepping increases in proportion to the amplitude of the electric stimulation [20], we can assume that the time series of the intensity of the impulses fired by the neural centers is associated with a time series of virtual frequencies $\{f_j\}$. So, in the spirit of the model suggested by Ashkenazy and co-workers [23,24], we assume that the long-time correlated frequency of the SCPG is described by a random walk on a finite-size correlated chain, where each node of the chain is a neural center of the kind discussed above, which fires an impulse with a particular intensity that would induce a particular virtual frequency. Ashkenazy and co-workers [23,24] focused on explaining the multifractal changes in the gait time series during maturation from childhood to adulthood, assuming that neural maturation is parametrically associated with the range ρ of the Brownian process that activates the nodes of the finite-size correlated chain of frequencies.

Here, we adopt a different approach because we are interested in modeling the gait for human adults operating under different conditions. We assume that neural maturation and, therefore, the standard deviation ρ of the random walk process remains constant, whereas the strength of the correlation among the neural centers increases with the change of the velocity of the gait from the normal to the slower or faster regimes. The change of velocity is interpreted as a biological stress. Moreover, contrary to Ashkenazy and co-workers [23,24] we do not add any noise to the output of each node to mimic biological noise. The final output given by the actual frequencies of the gait cycle fluctuates due to the chaotic solutions of the nonlinear oscillators in the SCPG, here that being the forced Van der Pol oscillator. The advantage of using chaos in the model, rather than noise, is that chaos is an intrinsic property of the SCPG dynamics and therefore introduces variability in a controllable way.

We observe that nonlinear oscillators may present chaotic regimes and may be forced by an external frequency [10], so they may be useful in describing not only the change of phase from the walk, trot, canter, and gallop of the quadrupeds, but also the variability of the stride intervals observed in humans. In bipeds it is possible to mimic the movements of the two legs with two nonlinear coupled oscillators. However, because the geometry of the bipeds' gait, contrary to that for quadrupeds, is unique and the two legs must be shifted by π rad in phase, we can mimic the biped's gait with only one nonlinear oscillator. In our model we use a well-known neuronal oscillator model, that is, the forced Van der Pol oscillator [8,10] that is defined by the following equation:

$$\ddot{x}+\mu(x^2-p^2)\dot{x}+(2\pi f_j)^2 x = A\sin(2\pi f_0 t). \tag{4}$$

The parameter p controls the amplitude of the oscillations, μ controls the degree of nonlinearity of the oscillator, f_j is the inner virtual frequency of the oscillator during the jth cycle that is related to the intensity of the jth neural fired impulse, and A and f_0 are, respectively, the strength and the frequency of the external driver. The frequency of the oscillator would be $f=f_j$ if $A=0$.

We notice that the nonlinear term as well as the driver induce the oscillator to move around a limit cycle. The actual frequency of each cycle may differ from the inner virtual frequency f. We assume that at the conclusion of each cycle, a new cycle is initiated with a new inner virtual frequency f_j produced by the stochastic CPG model while all other parameters are kept constant. However, the simulated stride-interval is not $1/f_j$ but it is given by the actual period of each cycle of the Van der Pol oscillator.

We assume that the neural centers of the SCPG may fire impulses with different amplitudes that induce virtual frequencies $\{f_i\}$ with finite-size correlations. Here, therefore, we model directly the time series of virtual frequencies. The virtual frequencies $\{f_i\}$ are centered around the driver frequency f_0 according to the relation

$$f_i = f_0 + \gamma X_i, \tag{5}$$

where γ is a constant and X_i is a finite-size correlated variable, that is,

$$C_X(r) = \frac{\langle X_i X_{i+r}\rangle}{\langle X_i^2\rangle} = \exp\left[-\frac{r}{r_0}\right]. \tag{6}$$

The parameter r_0 measures the spatial range of the correlations of the neural network. The chain of frequencies $f_i = f_0 + \gamma X_i$ is generated by a first-order autoregressive process, also known as a linear Markov process [25], which is generated by the recursion equation

$$X_i = a X_{i-1} + \varepsilon_i, \tag{7}$$

where $0<a<1$ is a constant and $\{\varepsilon_i\}$ is a normalized zero-centered discrete Gaussian process. It is easy to prove [25] that the autocorrelation function of the chain $\{X_i\}$ is given by

$$C_X(r) = \frac{\langle X_i X_{i+r} \rangle}{\langle X_i^2 \rangle} = a^r. \quad (8)$$

A direct comparison between Eqs. (6) and (8) gives $a = \exp(-1/r_0)$, so, we can easily generate a data sequence with the desired finite-size correlation value r_0. Following Ref. [23,24], we assume that a frequency is activated by the position of a random walker given by the discrete function $g(j)$ with $j = 1,2, \ldots,$ whose jump sizes follow a Gaussian distribution of width ρ. The width of this distribution, according to the interpretation of Ashkenazy and co-workers [23,24], is associated with the human neural age maturation. This random walk mechanism allows us to obtain from the finite-time, correlated frequency series $\{f_i\}$, a new time series of frequencies $\{f_j\}$ with $i = g(j)$, characterized by long-time correlations, that is,

$$\{f_i\} \xrightarrow{i=g(j)} \{f_j\}. \quad (9)$$

Finally, the new sequence of frequencies $\{f_j\}$ is used in Eq. (4) recursively.

To establish the fractal properties of the SCPG model, we estimate the autocorrelation function of the new sequence of frequencies $\{f_j\}$. We have [18]

$$C_f(J) = \frac{\langle (f_j - f_0)(f_{j+J} - f_0) \rangle}{\langle (f_j - f_0)^2 \rangle} = \frac{\langle X_{g(j)} X_{g(j+J)} \rangle}{\langle X_j^2 \rangle}. \quad (10)$$

It is not difficult to deduce that

$$C_f(J) = \int_{-\infty}^{\infty} \exp\left[-\frac{|g - g(j)|}{r_0} \right] \frac{\exp\left[-\frac{[g - g(j)]^2}{2J\rho^2} \right]}{\sqrt{2\pi J \rho^2}} dg, \quad (11)$$

where the first term of the integral is the autocorrelation between the position $g(j)$ and a generic position g given by Eq. (6), and the second term of the integral is the Gaussian distribution of the generic position g after J steps of a random walker that starts from the position $g(j)$. Equation (11) can be solved, and gives

$$C_f(J) = \exp\left[\frac{Y}{2} \right] \operatorname{erfc}\left[\sqrt{\frac{Y}{2}} \right], \quad (12)$$

where

$$\operatorname{erfc}(x) = 1 - \frac{2}{\sqrt{\pi}} \int_0^x e^{-t^2} dt \quad (13)$$

is the complement of the error function and

$$Y = \left(\frac{\rho}{r_0} \right)^2 J. \quad (14)$$

Figure 7 shows the autocorrelation function of the stochastic CPG, Eq. (12). The variable Y is given by Eq. (14). The two straight lines correspond to the long-range autocorrelation function (1) with the Hölder exponents $h = -0.25$ and $h = -0.025$. The figure shows that for small Y, $C_f(J)$ is characterized by a slope with the Hölder exponent $h \approx 0$ typical of the pink noise and, for large value of Y, $C_f(J)$ asymptotically converges to a long-range fractal signal with $h = -0.25$ and the Hurst exponent $H = 0.75$. The inverse power-law character of the correlation function would lead one to conclude that the time series is a fractal stochastic process.

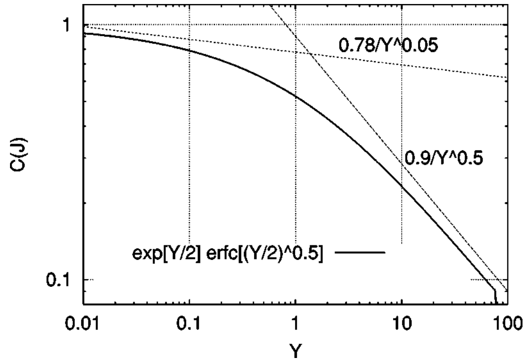

FIG. 7. Autocorrelation function of the stochastic CPG, Eq. (12). The variable Y $[Y = J(\rho/r_0)^2]$ is given by Eq. (14). The two straight lines correspond to the long-range autocorrelation function (1) with the Hölder exponents $h = -0.25$, and $h = -0.025$ which correspond to the Hurst exponents $H = 0.75$ and $H = 0.975$.

We assume that normal gait is characterized by the frequency $f_{0,n}$ and occurs when the individual is relaxed, and consequently the correlations between the neuronal centers are minimum. By implication, whether the gait increases or decreases in velocity, the correlations between the neuronal centers increase. This increase in the stress is modeled by using the short-time correlation parameter r_0 of the stochastic CPG by assuming

$$r_0 = r_{0,n}[1 + B(f_0 - f_{0,n})^2], \quad (15)$$

where $r_{0,n}$ is the short-range correlation among the firing neural centers at the normal frequency gait, f_0 is the mean frequency, and B is a positive constant that measures the increasing of short-range correlation at the anomalous frequency gait.

Figure 7 and Eqs. (14) and (15) suggest that the increase of the short-time correlation parameter r_0 leads a decrease of Y. Because we determine the fractal exponents by fitting a fixed number of steps J [7], a decrease of Y leads to a shift of the fitting range of the J steps toward a region where the curve of the autocorrelation function (12) is characterized by a higher curvature. A higher curvature of the autocorrelation function may be detected as an increase of the multifractal properties of the signal. So, we expect that our method of analysis gives a slight increase of the Hölder exponents as well as a slight increase of the multifractal properties when the gait increases or decreases in velocity according to Eq. (15).

In summary, our model is based upon the following assumptions. First, we have to observe that the experimental datasets are about the stride intervals of the gait. Second, the frequency of walking may be associated with a long-time correlated neural firing activity that induces virtual pace frequency, nevertheless, the walking is also constrained by the biomechanical motocontrol cycle that directly controls movement and produces the pace itself. Therefore, what we have to do is to incorporate both the neural firing activity given by a stochastic CPG and the motocontrol constraint that is given by a nonlinear filter characterized by a limit cycle. Therefore, we model our SCPG model such that it is based on the coupling of a stochastic with a hard-wired CPG model and depends on many factors. The most important parameters of the model are the short-correlation size r_0 of Eq. (6), which measures the correlation between the neuron centers of the stochastic CPG, the intensity A of the forcing driving component of the nonlinear oscillator of Eq. (4) and, of course, the mean frequency f_0 of the actual pace that distinguishes the slow, normal, and fast gait regimes. The other parameters, γ, ρ, μ, and p may be, to a first-order approximation, kept fixed.

While the numerical simulations are left to the following section, we can anticipate an interpretation of the two main parameters r_0 and A. In fact, the short-correlation size r_0 may be interpreted as a parameter that measures the natural correlation between the neural centers and such short-time correlation increases under particular stress, for example, when the velocity of the gait is slower or faster than the normal relaxed situation. The intensity of the forcing driving component A may be associated with the voluntary action of trying to follow a particular cadence and is expected to increase under a metronomic constraint.

V. SIMULATED STRIDE-INTERVAL GAIT

In this section we present and comment on our computer simulations of the stride-interval of human gait under a variety of conditions. For simplicity, we make use of the following values of the parameters. The frequency of the normal gait is fixed at the experimentally determined value of $f_{0,n}=1/1.1$ Hz, so that the average period of the normal gait is 1.1 s; the frequency of the slow and fast gaits are, respectively, $f_{0,s}=1/1.45$ Hz and $f_{0,f}=1/0.95$ Hz, with an average period of 1.45 and 0.95 s, respectively, which is similar to experimentally realized slow and fast human gaits shown in Fig. 1.

Also the hopping-range parameter is chosen equal to that for adults [23,24], that is, $\rho=25$ and kept constant. Moreover, we chose $r_{0,n}=25$ such that for $f_0=f_{0,n}$ we have $r_0=25$ that coincides with the corresponding value found in Ref. [23]. To generate an artificial sequence with a variability compatible to that of the experimental sequence, we chose $B=50$ in Eq. (15) and, in Eq. (5), $\gamma=0.02$, that is, a value compatible to the average of the standard deviation of all the data analyzed by us [7], however, the value of γ may be smaller and may decrease with an increase in the frequency f_0 and/or an increase in the intensity of the forcing amplitude A of Eq. (4).

So, we choose a frequency f_0, calculate r_0 via Eq. (15) and the Markovian parameter a, then we generate a chain of frequencies $\{f_i\}$ via Eqs. (5) and (7). Finally, by using the random walk process to activate a particular frequency of the short-time correlated frequency neural chain, we obtain the time series of the frequencies $\{f_j\}$ to use in the time evolution of the Van der Pol oscillator. For simplicity, we keep constant the two parameters of the nonlinear component of oscillator (4), $\mu=1$ and $p=1$. The only parameters allowed to change in the model are the mean frequency f_0 that changes also the value of r_0 via Eq. (15), and the intensity A of the driver of the Van der Pol oscillator (4).

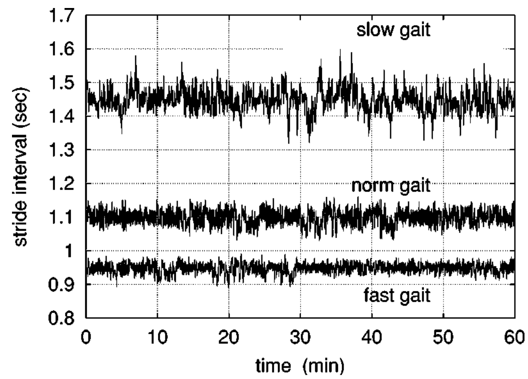

FIG. 8. Stride-interval time series for slow, normal, and fast computer-simulated gaits.

Figure 8 shows the stride-interval time series for slow, normal, and fast computer-simulated gaits using the SCPG. For the simulation of the normal gait we use $A=1$ and for both slower and faster gaits, we use $A=2$. We assume that the amplitude A of the driver of the Van der Pol oscillator (4) should be smaller for the normal gait than that for either the slower or faster gaits, because in our interpretation A measures the magnitude of the constraint to walk at a particular velocity. The amplitude A is smaller for the normal gait because the normal gait is the most relaxed, spontaneous, and consequently the most automatic of the three gaits. The figure shows that the SCPG model is able to reproduce a realistic persistence and volatility for the three gaits by simply changing the frequency of the gait itself. In particular, note the high volatility of the slow gait that is remarkably similar to that seen in Fig. 1.

Figure 9 shows the stride-interval time series for slow, normal, and fast metronome-triggered computer-simulated gaits. We use the same frequency series generated by the

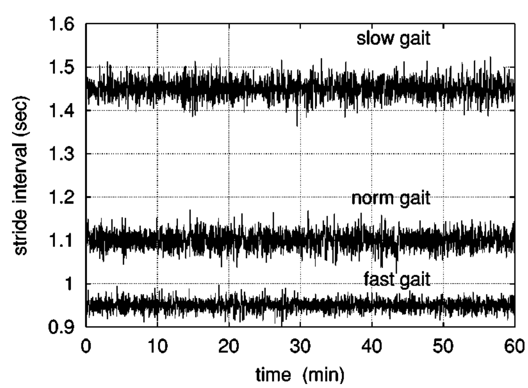

FIG. 9. Stride-interval time series for slow, normal, and fast gaits for metronome-triggered computer-simulated gait.

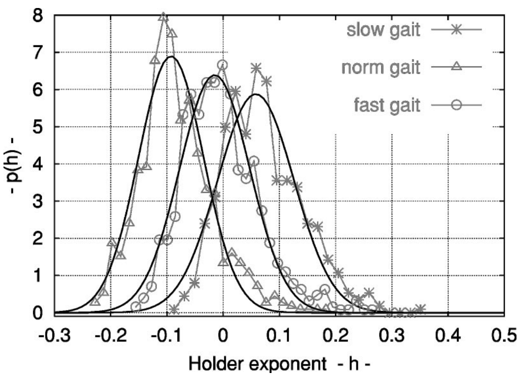

FIG. 10. Histogram of probability density estimation of the Hölder exponents for computer-simulated gait: slow (star) ($h_0 = 0.058$, $\sigma = 0.068$), normal (triangle) ($h_0 = -0.093$, $\sigma = 0.058$), and fast (circle) ($h_0 = -0.015$, $\sigma = 0.063$) gaits for a single individual.

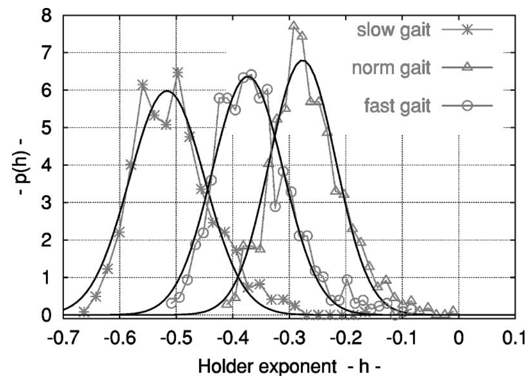

FIG. 11. Histogram and probability density estimation of the Hölder exponents for metronome-triggered computer-simulated gait: slow (star) ($h_0 = -0.516$, $\sigma = 0.067$), normal (triangle) ($h_0 = -0.276$, $\sigma = 0.059$), and fast (circle) ($h_0 = -0.373$, $\sigma = 0.063$) gaits for a single individual.

SCPG used to produce the sequences of Fig. 8. We only change the intensity A of the driver of the Van der Pol oscillator (4). We use for the normal gait $A = 4$ and for both slower and faster gaits $A = 8$. Again we suppose that the intensity A of the driver of the Van der Pol oscillator (4) should be smaller than that for both slower and faster gaits, because the normal gait is the most relaxed and spontaneous. By comparing Figs. 8 and 9 we note the increase in randomness, the loss of persistency, and the reduction in volatility; all effects that are induced in the latter time series by increasing the value of A and are found in the phenomenological data shown in Figs. 1 and 2.

Figure 10 shows histograms of distributions of the Hölder exponents for the three computer-simulated gaits shown in Fig. 8. The calculations are done in the same way as those used to produce the histograms in Fig. 3 for the experimental data, for details see Ref. [7]. The figure shows that the SCPG model is able to generate artificial stride-interval time series with statistical properties similar to the fractal and multifractal behaviors of the real data. By changing the gait mode from slow to normal, the center of the distribution of Hölder exponent h_0 decreases. In the same way by changing the gait mode from normal to fast, the mean Hölder exponent again increases, just as it does for the real data. According to the SCPG model, this increase in the scaling parameter is due to the increase of the inner short-time correlation among the neuronal centers, modeled by Eq. (15) as we have explained in the preceding section by commenting the behavior of the autocorrelation function $C_f(J)$ shown in Fig. 7 at small Y. Furthermore, this behavior is due to the biological stress of consciously walking at a speed that is different from the normal spontaneous speed. In addition, the multifractality of the gait time series slightly increases for a walking rate different from normal. Here again this effect is observed in the real stride-interval data and it is proven by a slight increase in the width of the histograms for fast and, in particular, slow gait.

Figure 11 shows the histograms of probability density estimations of the Hölder exponents for the three metronome-triggered computer-simulated gaits shown in Fig. 9. The calculated points show that the SCPG is able to generate artificial stride-interval time series that present similar fractal and multifractal behaviors to those of real stride-interval data taken under the constraint of a metronome. By increasing the intensity A of the driver of the Van der Pol oscillator (4), the randomness of the time series increases and it is possible to obtain a large variety of time series, from those having antipersistent to those with persistent fractal properties. In the SCPG, the parameter A measures the constraint of consciousness on the gait, and therefore the value of A has to increase if the walker is asked to synchronize his or her pace with the frequency of a metronome. The figure suggests that the SCPG model is able to explain a number of other properties of the metronome-triggered walking. Figure 6 shows that the usually normal metronome-triggered gait is that with the highest persistent fractal properties. The normal gait is also the most natural under the constraint of the metronome and, therefore, we should expect that the normal gait is the most automatic and the least constrained by human consciousness. This is the reason that we have chosen $A = 4$ for the normal metronome-triggered gait. For both slower and faster metronome-triggered gaits we have chosen $A = 8$ to indicate a higher conscious stress that constrains gait at anomalous speeds. Moreover, by comparing Figs. 10 and 11 and considering that in both simulations we have used the same value of the forcing parameter A for both slower and faster gaits, we notice that the largest fractal shift occurs for the slower gait. This increased shift implies that the slower gait is more sensitive to a voluntary constraint and, so, the slower mode has the larger variability. In fact, our human experience and the superposition of the distributions of the Hölder exponents for the ten cohorts in Fig. 6 show a large fractal variability of the slower gait. Finally, Fig. 6 reveals that few persons are characterized by a strong antipersistent pace when asked to follow a metronome. According to the SCPG model, some people are not able to find a natural synchronization and need to continuously adjust and readjust the speed of their pace to match the beat of the metronome. This changing of pace implies a very strong conscious act and, therefore, a very high value of the parameter A that would produce a strong antipersistent signal.

VI. CONCLUSION

We have introduced a kind of the SCPG model that is able to mimic the complexity of the stride-interval sequences of human gait under the several conditions of slow, normal, and fast regimes for both walking freely and keeping the beat of a metronome. The SCPG model is based on the assumption that human locomotion is regulated by both the central nervous system and by a motocontrol system. A network of neurons produces a correlated syncopated output that is correlated according to the level of physiological stress and this network is coupled to the motocontrol process. The combination of systems controls locomotion and the variability of the gait cycle. It is the period of the gait cycle that is measured in the datasets considered herein. Moreover, walking may be conditioned by a voluntary act as well, for example, walking may be consciously forced following the frequency of a metronome. We model the complex system generating the data by assuming that the correlated firing activity of the neural centers generated by a stochastic CPG regulates only the inner frequency of a forced Van der Pol oscillator that mimics the motocontrol mechanism of the gait cycle. The stride-interval is the actual period of each cycle of the forced Van der Pol oscillator. In this way the gait frequency is slightly different from the inner frequency induced by the neural firing activity whose impulse intensities are able to generate only a potential, but not an actual frequency. The chaotic behavior of such a nonlinear oscillator, such as the Van der Pol oscillator, and the possibility to force the frequency of the cycle with an external fixed frequency allow the SCPG model to generate time series that present similar fractal and multifractal properties to that of the human physiological stride-interval data in all situations here analyzed. Moreover, by implementing the SCPG with four coupled forced Van der Pol oscillators as in Ref. [8], it should be possible to simulate the change of phase between various modes of quadrupeds' locomotion, that is, walking, trotting, cantering, and galloping.

The variety of complex behaviors is regulated by two parameters, the average frequency f_0 and the amplitude A of the driver of the Van der Pol oscillator. The frequency f_0 regulates the speed and may be associated with a neuronal stress that increases the correlation among the neural centers. The amplitude A may be associated with the voluntary action of trying to track a particular frequency and it is expected to increase under a metronome constraint. Finally, Refs. [23,24] report that the stride-interval time series for elderly subjects and for subjects with Huntington's diseases are more random than for young healthy subjects. According to the SCPG model, this may be explained by a decrease of the normal short-range correlation among the neural centers that may be associated with a nervous degeneration caused by injury, disease, or aging. This decrease in correlation may be modeled through $r_{0,n}$ of Eq. (15). However, the decrease of correlation in the gait of those subjects may also be associated with an increase of the amplitude A of the driving force of the Van der Pol oscillator, Eq. (4). In fact, those subjects may also consciously choose to walk more carefully.

We emphasize that the selection of the van der Pol oscillator and of the particular stochastic CPG for the SCPG model is not unique. The two properties of the model necessary to capture the physiological properties of the interest in the gait-interval time series are the following: (1) the dynamics of the system unfolds on an attractor in phase space and (2) the natural frequency of the attractor is replaced by a random walk over a restricted set of frequencies. Property (1) is generic for relaxation oscillators, so the same behavior would result for a family of such nonlinear oscillators. Property (2) is also generic and leads to a multiplicative stochastic term in the nonlinear dynamical equation and to a multifractal output for the dynamical model.

ACKNOWLEDGMENT

N.S. thanks the Army Research Office for support under Grant No. DAAG5598D0002.

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