

## Understanding the complexity of human gait dynamics

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(Received 6 March 2009; accepted 6 May 2009; published online 29 June 2009)

Time series of human gait stride intervals exhibit fractal and multifractal properties under several conditions. Records from subjects walking at normal, slow, and fast pace speed are analyzed to determine changes in the fractal scalings as a function of the stress condition of the system. Records from subjects with different age from children to elderly and patients suffering from neurodegenerative disease are analyzed to determine changes in the fractal scalings as a function of the physical maturation or degeneration of the system. A supercentral pattern generator model is presented to simulate the above two properties that are typically found in dynamical network performance: that is, how a dynamical network responds to stress and to evolution. © 2009 American Institute of Physics. [DOI: 10.1063/1.3143035]

**Walking is thought to be a consequence of the two-way interaction between the neural networks in the central nervous system plus the intraspinal nervous system on one side and the mechanical periphery consisting of bones and muscles on the other. That is, while the muscles receive commands from the nervous system, they also send back sensory information that modifies the activity of the central neurons. The coupling of these two networks produces a complex stride interval time series that is characterized by particular symmetries including fractal and multifractal properties that depend on several biological and stress constraints. It has been shown that (a) gait phenomenon is essentially a rhythmic cycle that obeys particular phase symmetries in the synchronized movement of the limbs, (b) the fractal and multifractal natures of the stride interval fluctuations become slightly more pronounced under faster or slower paced frequencies relative to the normal paced frequency of a subject, and (c) the randomness of the fluctuations is higher for children than for adults and increases if subjects are asked to synchronize their gait with the frequency of a metronome or if the subjects are elderly or suffering from neurodegenerative disease. Here we review a supercentral pattern generator (SCPG) model, which is able to reproduce these known properties of walking and briefly discuss the physiological and psychological interpretations of the model parameters.**

### I. INTRODUCTION

Locomotion is the process by which animals move from one geographic position to another. It includes starting, stopping, changes in speed, and alteration in directions. These events are superimposed to a basic pattern that can be defined as a rhythmic displacement of body parts that maintains the animal in constant forward progression.<sup>1,2</sup>

Human locomotion is both a voluntary and an automated process and its rhythmic movements are regulated by both

feedforward and feedback control.<sup>2</sup> The automated part of legged locomotion of animals is understood through the use of a central pattern generator (CPG). A CPG consists of a network of firing neurons capable of producing a synchronized output that cause the muscles of the limbs to move in a given succession.<sup>3,4</sup> Because the movement of each limb almost identically cyclically repeats itself,<sup>5</sup> its dynamics can be described by a nonlinear oscillator for each limb participating in the locomotion process.<sup>6</sup> For example, a quadruped requires the coupling of four nonlinear oscillators to determine the correct phase relations among the four legs in order to distinguish among various modes of locomotion, that is, walking, trotting, cantering, and galloping. The four coupled nonlinear oscillators can also be thought of as phases of a single central synchronized nonlinear dynamical network that regulates the overall rhythm of muscular activity.

The existence of a CPG has been experimentally established by transecting the spinal cord of animals. 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 a little effect on the walking cycle.<sup>7</sup> 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.

Human and animal locomotion have been described as three distinct stages: (1) development stage (from rest to some velocity), (2) rhythmic stage (some constant average velocity), and (3) decay stage (coming back to rest).<sup>8</sup> Many observations have confirmed that the rhythmic stage of human walking is very consistent and directly related to the optimal efficiency for any individual. Human walking is

herein studied as a temporal sequence of steps. A step can be partitioned in 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.<sup>9</sup> The sequences we are interested in are made of successive stride intervals, that is, the length of time from the start of one stance phase to the start of the next stance phase.

By applying nonlinear data processing techniques to stride interval sequences some researchers<sup>10–16</sup> have begun to disclose the intrinsic complexity of the process of walking, which is not just a simple cyclical periodic phenomena. Although it has been known for over a century that there is a variation of 3%–4% in the stride interval of humans during walking,<sup>17</sup> only in the last decade did Hausdorff *et al.*<sup>10,11</sup> demonstrate that the stride-interval time series exhibits a long-time correlation, suggesting that the phenomenon of walking is a self-similar long-range correlated phenomenon that can be described by fractal Brownian motion.<sup>18</sup> This particular erratic motion can be described by a given fractal exponent. Subsequent studies<sup>12–15</sup> supported the conclusion that the human gait time series is not only fractal but also weakly multifractal.

The discovery that locomotion is a complex cyclical phenomenon involving both order and randomness in different degrees has suggested the development of a correlated stochastic version of a CPG for the purpose of capturing the fractal properties of the interstride interval sequences. This kind of model was first suggested by Hausdorff *et al.*<sup>19</sup> and was later extended<sup>20,21</sup> to describe the changing of gait dynamics as humans develop from childhood to adulthood. This stochastic model essentially consists of a random walk on a correlated chain, where each node of the chain is assumed to be a neural center of the kind discussed above firing with a different frequency. This random walk is found to generate a fractal process. More recently, West and Scafetta<sup>15</sup> developed a SCPG that reproduced both fractal and multifractal properties of the gait dynamics, which will be described in detail subsequently.

The potential utility of these models is that they are capable of reproducing some aspects of walking dynamics. Stride time interval sequences of children, adults, elderly, and people with degenerative diseases are characterized by different fractal and multifractal exponents. These exponents can be used as a measure of the degree of maturation or degeneration of the neural network that regulates human movement. Also a different kind of stress, for example, walking faster or slower than normal, or following a given frequency of a metronome, alters the fractal exponents of stride intervals. The fractal dependency of these sequences is quite a common behavior and it is found in a multitude of quasi-periodic physiological signals, such as, for example, inter-heartbeat and breath intervals,<sup>13</sup> indicating that physiological signals under several condition can be modeled with nonlinear oscillators working in the unstable, forced, and chaotic regimes.

We briefly describe the major observed pattern in human gait dynamics and describe SCPG as a model describing human dynamics. 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 fractal and multifractal variabilities of human gait under several conditions.

## II. BACKGROUND

In this section we summarize the main fractal and multifractal characteristics observed in the stride interval sequences for different individuals under different conditions. Details regarding the collection of the data herein analyzed can be found at Physionet ([www.physionet.org](http://www.physionet.org)) from where the data can be downloaded.

The analysis of the data is performed with alternative algorithms to measure the fractal exponents. In his seminal work Mandelbrot<sup>18</sup> showed that many natural phenomena are described by self-affine, correlated, time series generally known as fractal noise. These signals are characterized by an exponent that he called  $H$  in honor of Hurst who first used one of these techniques<sup>22</sup> for measuring the persistence of the Nile's flood cycles.

In general a fractal is defined in the following way. If  $X(t)$  is a fractal process with Hurst exponent  $H$  and  $c$  is a constant, then  $X(t)=X(ct)/c^H$  is another fractal process with the same statistics. Fractal noise is characterized by a spectrum satisfying the power law

$$P(f) \propto f^{-\beta}, \quad (1)$$

where  $f$  is the frequency, the exponent  $\beta=(2H-1)$ , and  $H$  is the Hurst exponent. The self-affine property expressed by Eq. (1) and the relation between  $\beta$  and  $H$  are theoretically valid only for an infinitely long monofractal noise. The interpretation of the exponent  $H$  is as follows: if  $H=0.5$  the signal is random noise; if  $0 < H < 0.5$  the noise is antipersistent, that is, a large value is likely followed by a small value and vice versa; if  $0.5 < H < 1$  the noise presents long-range persistent correlation, that is, a large value is likely followed by another large value and vice versa; the value  $H=1$  corresponds to what is commonly known as *pink* noise or  $1/f$  noise; values of  $H > 1$  describe fractal Brownian motion, among them the *random walk*, which has  $H=1.5$ . Two of the techniques commonly used for the analysis of fractal time series are detrended fluctuation analysis<sup>23</sup> and the diffusion entropy analysis and the standard deviation analysis.<sup>24</sup>

The multifractal spectrum is analyzed by a methodology introduced by Struzik<sup>25</sup> and then extended by Scafetta *et al.*<sup>12</sup> for estimating the local Hölder exponents of a time series. We use this methodology herein. The multifractal properties of a sequence can be determined from studying a distribution of Hölder exponents that is centered at a given mean value  $\bar{h}$  with a width given by the standard deviation  $\sigma$ . The exponent  $\bar{h}$  is related to the Hurst exponent by  $H=\bar{h}+1$ . The standard deviation  $\sigma$  is an indicator of the possible multifractal nature of the time series. A distribution of Hölder exponents can be approximately fitted by a Gaussian distribution of the type

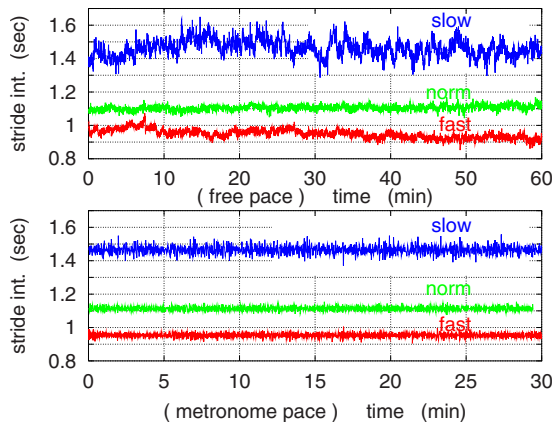


FIG. 1. (Color online) Typical stride interval time series in the free and metronome paced conditions for normal, slow, and fast paces. The histograms are fitted with Gaussian functions.

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

where the value  $h_0$  is often a good approximation to  $\bar{h}$ . Usually,  $h_0$  is slightly larger than  $\bar{h}$  because the distribution of Hölder exponents presents a slightly positive skewness.

Note that a monofractal time series of finite length presents a nonzero width of the distribution of Hölder exponents that vanishes as the time series becomes infinitely long. 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 correspondent monofractal time series.

### A. Natural and metronomically constrained walking

Figure 1 shows samples of stride interval sequences under different conditions. Each time series is approximately 1 h long for natural slow, normal, and fast walking and about 30 min long for metronomically constrained walking for slow, fast, and normal walking. Participants in the study had no history of neuromuscular, respiratory, or cardiovascular disorders. They were not taking medications and had a mean age of 21.7 yr (range: 18–29 yr), mean height of  $1.77 \pm 0.08$  m, and mean weight of  $71.8 \pm 10.7$  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.

Basic statistical analysis of ten sequences gives the mean stride intervals  $1.32 \pm 0.05$ ,  $1.12 \pm 0.02$ , and  $1.00 \pm 0.01$  for, respectively, unconstrained slow, normal, and fast paces. For metronomically constrained slow, normal, and fast paces we get, respectively,  $1.36 \pm 0.03$ ,  $1.12 \pm 0.02$ , and  $1.00 \pm 0.01$ .<sup>12</sup>

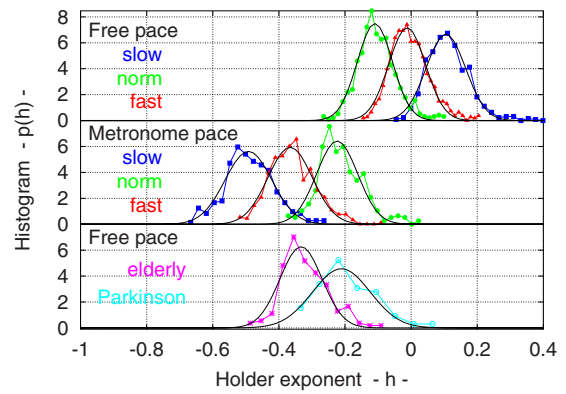


FIG. 2. (Color online) Typical Hölder exponent histograms for the stride interval series in the freely walking and metronome conditions for normal, slow, and fast paces, for elderly, and for a subject with PD. In the test are reported the average properties. The histograms are fitted with Gaussian functions.

Scafetta *et al.*<sup>12</sup> determined that typical distributions of Hölder exponents, for unconstrained walking of a single individual, are of the type depicted in Fig. 2. By estimating the distribution of Hölder exponents, it has been shown that the stride interval time series for natural gait shows fractal properties similar to  $1/f$ -noise and is weakly multifractal. The time series may be nonstationary and its fractal variability changes in different gait mode regimes. In particular, the persistence, as well as the multifractality of the stride interval time series, tends to increase slightly for both slow and fast paces above that of the normal pace. By averaging the results for ten subjects walking naturally at normal, slow, and fast paces we get  $h_{0,n} = -0.092$ ,  $\sigma_{0,n} = 0.058$ ;  $h_{0,s} = 0.035$ ,  $\sigma_{0,s} = 0.061$ ; and  $h_{0,f} = -0.045$ ,  $\sigma_{0,f} = 0.058$ , where the subscripts  $s, n, f$  refer, respectively, to slow, normal, and fast paced gaits, and  $h_0$  and  $\sigma_0$  refer to the average and standard deviations of the distribution of the Hölder exponents. The Hölder exponent distribution width  $\sigma$  was found to be  $\sim 5\%$  larger than the width relative to a monofractal noise of equal length, which indicates that the analyzed sequences are weakly multifractal.

Figure 2 shows that stride interval time series for human gait are characterized by strong persistent fractal properties very close to that of  $1/f$ -noise,  $h \approx 0$ . However, a normal gait is 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. A careful comparison of the widths of the distributions of Hölder exponents for different gaits with widths for a corresponding monofractal noise data set of the same length has been established that the stride interval of human gait is only weakly multifractal. However, the multifractal structure is slightly more prominent for fast and slow gaits than for a normal gait.

If the pace is constrained by a metronome, beating at the average rate of the cohort of walkers, the stochastic properties of the stride interval time series change significantly in a wide range from persistent to antipersistent. In general, in each case there is a reduction in the long term memory and an increase in randomness as the shift in the Hölder exponent histogram in Fig. 2 shows. By averaging the results for ten subjects we get  $h_{0,n}=-0.37$ ,  $\sigma_{0,n}=0.063$ ;  $h_{0,s}=-0.48$ ,  $\sigma_{0,s}=0.066$ ; and  $h_{0,f}=-0.36$ ,  $\sigma_{0,f}=0.059$ . The figure clearly indicates that under the constraint of a metronome, the stride interval of human gait increases its randomness because the distribution of Hölder exponents is centered more closely to  $h=-0.5$ , that is, the characteristic value of Gaussian or uncorrelated random noise. The data present large variability in the values 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 on individuals walking normally. Probably, by walking at a normal speed an individual is more relaxed and s/he walks more naturally. The fast gait appears to be almost uncorrelated noise while the slow gait presents a large variability from persistent to antipersistent fluctuations with an average that is close to random noise.

Scafetta *et al.*<sup>12</sup> also noticed 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 and (b) a psychophysical 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 regime.

## B. Children

Very young healthy children have an immature control of posture. Their gait is characterized by large and irregular stride-to-stride fluctuations. Their attempts at walking are often interrupted by frequent falls. As is well known, with the growth of children their walk becomes more stable and similar to the adult one. These changes are a consequence of the development of human neuromuscular control and locomotor function. Some previous studies<sup>26,27</sup> emphasized that the neuronal and muscular developments of the child locomotor function are gradual. Usually, as is also indicated by the fact that energy expenditure in walking children gradually approaches the adult one throughout the development,<sup>28</sup> a decrease in walking variance is observed with aging.

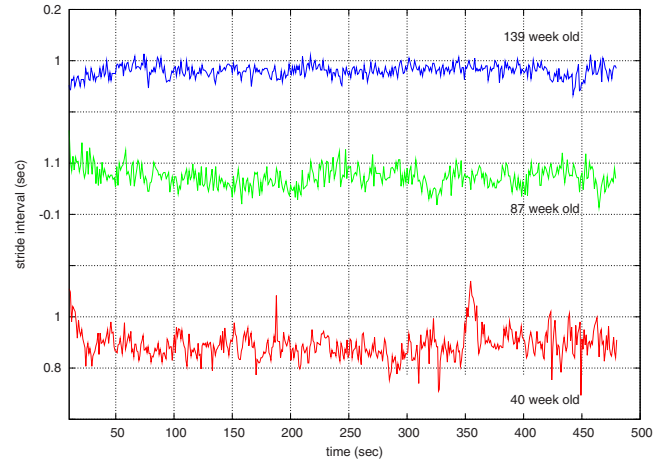


FIG. 3. (Color online) Typical stride interval time series in the free paced conditions for children of different age.

Although the variance of these sequences decreases, the gait of children continues to be characterized by stride-to-stride fluctuations that appear to be “random” or “noisy.” Hausdorff *et al.*<sup>11</sup> investigated whether the maturation of children gait dynamics could be detected and measured in the subtle changes in the temporal organization of the child’s stride-to-stride variability. The biological and physiological importance of such a measure is that it can be used to evaluate the development of neuromuscular control in children.

Hausdorff *et al.*<sup>11</sup> studied an 8 min long stride-to-stride sequences from 3- to 14-yr-old healthy children; there were similar numbers of boys and girls in each age group. They found that the stride-to-stride variability decreases steadily with age, as Fig. 3 shows: for three children aged 4, 7, and 11 yr, their coefficients of variation, a relative measure of the standard deviation of the variability, were 8.4%, 4.3%, and 1.9%, respectively. The decrease in variability refers mostly to the high frequency component of the stride-to-stride sequences and, therefore, is not related to accidental slow changes in the pace rate due, for example, to fatigue or environmental distractions. Thus, these changes are indicative of the development of the neuromuscular control network.

The temporal structure of these stride-to-stride sequences was evaluated with spectral analysis while the strength of long-range correlations was determined through the Hurst scaling exponent as measured with detrended fluctuation analysis. The power spectrum analysis revealed that there is a change in the frequency spectrum with age. Figure 4 shows our Hölder exponent distribution analysis of these data referring to different children from age 3 to 12 walking for a few minutes. The data are fit with a quadratic polynomial which suggests that the average Hölder exponent  $h_0$  increases with the age of the children. Very young children are characterized by stride intervals that appear less persistent than the walking of older children, which approaches that of the adults. Sometime the data for younger children appear interrupted by stride intervals significantly longer than the average: this suggests momentary losses of posture control, as would be expected for these very young individuals.

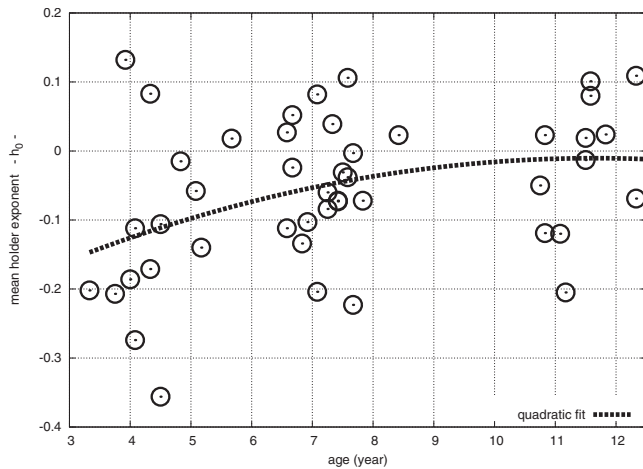


FIG. 4. Typical average Hölder exponent for stride interval time series in the free paced conditions for children of different ages. The data fit with a second order polynomial and suggest a maturation effect. Younger children walk more randomly than older children.

The above findings suggest that humans experience a gradual maturation of the neuromuscular control system. Although a visual observation of a walking child might suggest that the stride dynamics of children are not substantially different from those of adults, quantitative measurements based on both amplitude of the variation in the stride-to-stride length and long-range memory of the sequences of stride amplitudes indicate that the stride-to-stride control of walking does not reach a fully mature stage until adolescence. This evidence adds to the results obtained by studies on walking energy costs<sup>28</sup> and analyses of temporal and distance parameters of gait.<sup>29,30</sup> These studies indicate that inefficient ventilation, faster stride rates, shorter stature, more distal distribution of mass in the lower limbs, poor motor control, and interlimb coordination in children cause differences in gait dynamics between children and adults.

Most of the maturation process occurs between the ages of 3 and 8 and stabilized around the ages of 10–12, as one would expect a transition from childhood to adolescence. In fact, as Fig. 4 shows, the average Hölder exponent increases from a range of about  $-0.3 < h_0 < -0.1$ , which is indicative of a weak persistent noise, for very young children to a more persistent noise for older children, which is comparable to those found for adults at  $-0.1 < h_0 < 0.1$ .

Clearly, further research is needed to establish if this kind of analysis is a robust and sensitive indicator of gait maturation. The study of gait dynamics does seem to contribute to our understanding of the development of the control of gait and may also be of interest in a clinical context when the potential success of intervention is related to the maturity of the patient.

### C. Aging and neurodegenerative diseases

Changes associated with aging that negatively affects balance and gait include declining strength, muscle mass, and bone density; impaired respiratory capacity; selective atrophy of central nervous system components controlling balance and gait; and deterioration of peripheral sensory func-

tions. Central nervous system age-related changes include shrinkage of neural soma and processes of the central cortex<sup>31</sup> and in the vermis of the cerebellum.<sup>32</sup> Neurodegenerative diseases, including Parkinson's disease (PD), Huntington's disease, and amyotrophic lateral sclerosis (ALS), also produce changes in altered neuromuscular control. See Fig. 2.

Parkinson and Huntington's diseases are typical disorders of the basal ganglia and are associated with characteristic changes in gait rhythm. Because the neurons of the basal ganglia likely play an important role in regulating muscular motor control such as balance and sequencing of movements, it is reasonable to expect that the stride-to-stride dynamics, as well as the gait cycle duration, is affected by these neurodegenerative diseases.

Typical observed walking pattern alterations include double support timing (when both feet are in contact with the ground), increased variability of gait cycle duration, significant increase in stride length variability, variable walking speed, and variable step length.<sup>33</sup> The variability of the stride time, swing time, double support time, and step time of patients with Huntington's disease is on average double the corresponding values of patients with PD, which, in turn, is on average double with those of healthy individuals.<sup>34</sup> The greater the degree of neurologic impairment, the larger the gait variability. In any case, while stride length, gait variability, and fractal scaling of gait are all impaired in PD, distinct mechanisms likely contribute to and are responsible for the regulation of these disparate gait properties.<sup>35</sup>

On the contrary, average gait cycle duration, which is the stride time, is not significantly different in Parkinson and Huntington's disease patients. This indicates that the neurological disorder caused by these diseases primarily affects the control of the stride-to-stride variability of gait timing more than average gait cycle timing. Therefore, it is the dynamical evolution of successive strides that is mostly affected.

The walking of these patients, in particular for those with a more severe impairment, is characterized by sequences of disconnected random strides rather than by single continuous motion of healthy individuals. This behavior may be caused by multiple causes: impairment in anticipatory reflexes, diminished capacity to perform automatic sequential steps, disruption in the normal internal cueing required to string together submovements, inability to generate muscle forces at a constant level, impaired reflexes, and changes in the basal ganglia's effectiveness in integrating sensory stimuli. Thus, there may be both an increase in randomness in the functioning of basal ganglia and/or in the peripheral neural-muscular activity.<sup>36</sup>

A number of scientists<sup>13,37,38</sup> showed that neurodegenerative disease patients present altered fractal dynamics of gait characterized by reduced stride-interval correlations, that is, the walking of these individual becomes more random. The randomness increases with the severity of the neurodegenerative impairment, as Fig. 5 shows for several patients with Huntington's disease. In fact, the average Hölder exponent decreases from a normal range (about  $-0.1 < h_0 < 0.1$ ) indicative of a  $1/f$  noise for patients with a less severe im-

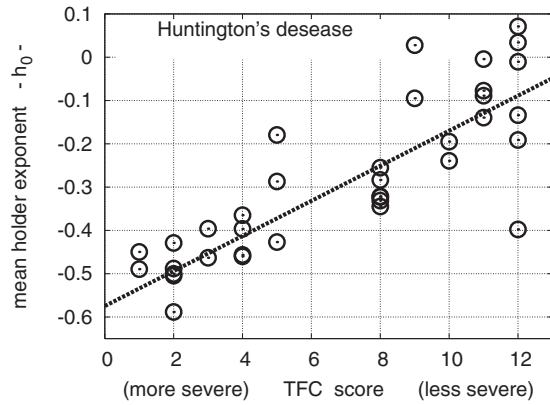


FIG. 5. Relationship between the mean Hölder exponent and the total functional capacity score of Unified Huntington's Disease Rating Scale (0 = most impairment; 13 = no impairment). The mean Hölder exponent decreases (that is, the sequences become more random) as the disease severity increases. The two measures are highly correlated ( $r^2=0.64$ ,  $P<0.005$ ).

pairment to a range of about  $-0.6 < h_0 < -0.4$  for patients with a more severe impairment. By repeating the Hölder exponent distribution analysis for five elderly subjects and a different set of five subjects with PD, we find on average  $h_{0,eld} = -0.28 \pm 0.10$ ,  $h_{0,PD} = -0.23 \pm 0.19$ , which is a range we found very close for very young children.

ALS is a disorder marked by loss of motor neurons. Major consequences of this disorder to walking are that the average stride time is significantly longer than normal, the average walking speed is significantly slower than normal, the measures of the magnitude of stride-to-stride variability are significantly increased over and above normal, and the fluctuation dynamics of normal gait are perturbed.<sup>39</sup> These kinds of gait alterations may be caused by muscle weakness, decreased endurance, and muscle fatigability. Other neuromuscular properties are also affected such that the motor cortex is hyperexcitable, muscle fiber conduction velocity decreases, mechanical efficiency is reduced, and muscle activation distal to the muscle membrane may be impaired.

Liao *et al.*<sup>40</sup> studied gait rhythm asymmetry in left and right legs in patients with PD, Huntington's disease, and ALS. They found that gait symmetry in subjects with PD and Huntington's disease is disturbed less than in subjects with ALS.

### III. MODEL

West and Scafetta<sup>15</sup> introduced a model of locomotion that governs the stride interval time series for human gait, which was called the SCPG. The SCPG model incorporates two separate mechanisms simulating "stress" into the generated stride interval time series. For stress we intend any mechanism or cause that induces an alteration of the stride dynamics relative to that observed for adult normal locomotion.

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-range time correlation of sequences as under the frequency constraint of a metronome. We mod-

eled the time series for walking assuming that the intensity of impulses of the firing neural centers regulates only the inner virtual frequency of a forced Van der Pol oscillator. A Van der Pol oscillator is herein adopted because it is a prototypical nonlinear oscillator capable of producing stable oscillations (known as limit cycles) which describe the quasi-periodic gait dynamics very well. 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, and frequency of the forcing function.

Since the frequency of stepping increases in proportion to the amplitude of the electric stimulation, 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 a way similar to the model suggested by Hausdorff *et al.*<sup>21</sup> and Ashkenazy *et al.*<sup>20</sup> we assume that the long-time correlated frequency of the SCPG is described by a random walk moving on a finite-size correlated chain of virtual firing nodes. 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 *et al.*<sup>20</sup> focused on explaining the fractal changes in the gait time series during maturation from childhood to adulthood. They assumed that neural maturation is parametrically associated with the short-range coefficient  $\rho$  of the chain of virtual firing nodes on which the Brownian process moves for activating the nodes of the finite-size correlated chain of frequencies. The smaller is the coefficient  $\rho$ , the more random is the resulting process.

This same mechanism may describe the fractal transition for elderly and patients with neurodegenerative diseases as well. In fact, the finite-size correlated chain of virtual firing nodes simulates the neural motor control network which would become less correlated because of the physiological impairment.

The SCPG was introduced to model the gait for human adults operating under a variety of conditions. We assume that neural maturation and, therefore, the standard deviation  $\rho$  of the random walk process remains constant, whereas the strength of the correlation among the neural centers increases with the change in the velocity of gait from normal to slower or faster regimes. The change in velocity is interpreted as a biological stress.

We can mimic the human gait with a single nonlinear oscillator. In our model we use a forced Van der Pol oscillator which is defined by the following equation:

$$\ddot{x} + \mu(x^2 - p^2)\dot{x} + (2\pi f_j)^2 x = A \sin(2\pi f_0 t). \quad (3)$$

The parameter  $p$  controls the amplitude of the oscillations,  $\mu$  controls the degree of nonlinearity of the oscillator,  $f_j$  is the inner virtual frequency of the oscillator during the  $j$ th cycle which is related to the intensity of the  $j$ th neural fired impulse, and  $A$  and  $f_0$  are, respectively, the strength and 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, induces the oscillator to move around a limit cycle. The actual frequency of each cycle may differ from the inner virtual

frequency  $f_j$ . 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 SCPG model, while all other parameters are kept constant. However, the simulated stride interval is not  $1/f_j$  but is given by the actual period of each cycle of the Van der Pol oscillator. We found this mechanism more interesting than that proposed by Hausdorff *et al.*<sup>21</sup> and Ashkenazy *et al.*<sup>20</sup> who added noise to the output of each node to mimic biological variability. In fact, we noticed that the so-called biological noise is naturally produced because of the chaotic solutions of nonlinear oscillators in the SCPG, here being the forced Van der Pol oscillator, while fluctuating around its limit cycle.

We assume that the neural centers of the SCPG may fire impulses with different voltage amplitudes that would 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

$$f_i = f_0 + \gamma X_i, \tag{4}$$

where  $\gamma$  is a constant and  $X_i$  is a finite-size correlated variable, that is,

$$C_X(r) = \frac{\langle X_i X_{i+r} \rangle}{\langle X_i^2 \rangle} = \exp\left[-\frac{r}{r_0}\right]. \tag{5}$$

The parameter  $r_0$  measures the spatial range of the correlations of the neural network. The chain of frequencies (4) is generated by a first-order autoregressive process, also known as a linear Markov process, which is generated by the recursion equation

$$X_i = aX_{i-1} + \varepsilon_i, \tag{6}$$

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

$$C_X(r) = \frac{\langle X_i X_{i+r} \rangle}{\langle X_i^2 \rangle} = a^r. \tag{7}$$

A direct comparison between Eqs. (5) and (7) gives  $a = \exp[-1/r_0]$ , so we can easily generate a data sequence with the desired finite-size correlation value  $r_0$ . We assume that a frequency is activated by the position of a random walker given by the discrete function  $g(j)$  with  $j = 1, 2, \dots$ , whose jump sizes follow a Gaussian distribution of width  $\rho$ . This random walk mechanism allows us to obtain from the finite-time, correlated frequency series  $\{f_j\}$  a new time series of frequencies  $\{f_{ij}\}$  with  $i = g(j)$  characterized by long-time correlations, that is,

$$\{f_{ij}\} \xrightarrow{i=g(j)} \{f_j\}. \tag{8}$$

Finally, the new sequence of frequencies  $\{f_{ij}\}$  is used in Eq. (3) recursively.

To establish the fractal properties of the SCPG model, we estimate the autocorrelation function of the new sequence of frequencies  $\{f_{ij}\}$ . We have

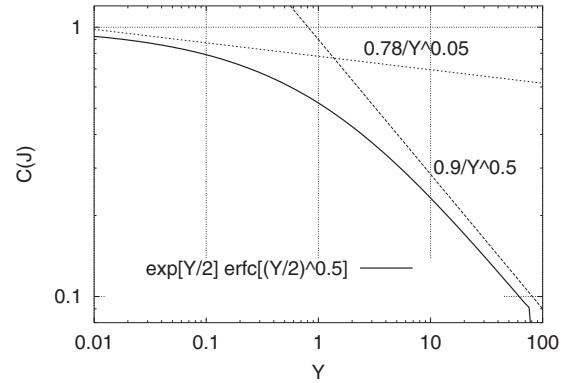


FIG. 6. Autocorrelation function of the stochastic CPG, Eq. (11). The variable  $Y$  ( $Y = J(\rho/r_0)^2$ ) is given by Eq. (13). The two straight lines correspond to the long-range autocorrelation function with the Hölder exponents  $h = -0.25$  and  $h = -0.025$  that correspond to the Hurst exponents  $H = 0.75$  and  $H = 0.975$ , respectively.

$$C_f(J) = \frac{\langle (f_j - f_0)(f_{j+J} - f_0) \rangle}{\langle (f_j - f_0)^2 \rangle} = \frac{\langle X_{g(j)} X_{g(j+J)} \rangle}{\langle X_j^2 \rangle}. \tag{9}$$

It is not difficult to deduce that

$$C_f(J) = \int_{-\infty}^{\infty} \exp\left[-\frac{|g - g(j)|}{r_0}\right] \frac{\exp\left[-\frac{(g - g(j))^2}{2J\rho^2}\right]}{\sqrt{2\pi J\rho^2}} dg, \tag{10}$$

where the first factor in the integral is the autocorrelation between the position  $g(j)$  and a generic position  $g$  given by Eq. (5), and the second factor in 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 (10) can be solved and yields

$$C_f(J) = \exp\left[\frac{Y}{2}\right] \operatorname{erfc}\left[\sqrt{\frac{Y}{2}}\right], \tag{11}$$

where

$$\operatorname{erfc}(x) = 1 - \frac{2}{\sqrt{\pi}} \int_0^x e^{-t^2} dt \tag{12}$$

is the complementary error function and the scaled variable of interest is

$$Y = \left(\frac{\rho}{r_0}\right)^2 J. \tag{13}$$

Figure 6 shows the autocorrelation function of the SCPG, Eq. (11). The variable  $Y$  is given by Eq. (13). The two straight lines correspond to the long-range autocorrelation function 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 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 Hurst exponent  $H = 0.75$ . The inverse power-law character of the correlation function implies that the time series is a fractal stochastic process.

A normal gait is characterized by a given basic frequency  $f_{0,n}$ . In this situation a person is relaxed and conse-

quently the correlations of the motor control network simulated by the SCPG are a given value. When the gait pace increases or decreases in velocity, the motor control network becomes more correlated because under stress the network focuses on a given task. This increase in the stress is modeled by using the short-time correlation parameter  $r_0$  of the SCPG by assuming

$$r_0 = r_{0,n}[1 + B(f_0 - f_{0,n})^2], \quad (14)$$

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 6 and Eqs. (13) and (14) suggest that the increase in the short-time correlation parameter  $r_0$  leads to a decrease in  $Y$ . Because we determine the fractal exponents by fitting a fixed number of steps  $J$ , a decrease in  $Y$  leads to a shift in the fitting range of the  $J$  steps toward a region where the curve of the autocorrelation function (11) is characterized by higher curvature. A higher slope coefficient may be interpreted as a higher fractal dimension and a higher curvature of the autocorrelation function may be interpreted as an increase in multifractal properties of the signal. Therefore, we expect that the SCPG model gives a slight increase in the Hölder exponents, as well as a slight increase in multifractal properties, when the gait frequency deviates from normal. This change in the Hölder exponent is the behavior observed in the data for natural walking.

#### IV. RESULTS

The SCPG can be used to simulate the stride interval of a human gait under a variety of conditions.<sup>15</sup> We use the average experimentally determined value of the basic frequency  $f_{0,n}=1/1.1$  Hz so that the average period of the normal gait is 1.1 s; the frequencies of slow and fast gaits are chosen to be, 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 the experimentally realized slow and fast human gaits shown in Fig. 1.

We use  $\rho=25$  and kept it 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 by Ashkenazy *et al.*<sup>20</sup> To generate an artificial sequence with a variability compatible to that of the experimental sequence, we chose  $B=50$  in Eq. (14), and in Eq. (4),  $\gamma=0.02$ , which is a value compatible to the average of the standard deviation of all the data we analyzed; however, the value of  $\gamma$  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. (3).

So, we choose a frequency  $f_0$ , calculate  $r_0$  via Eq. (14) and the Markovian parameter  $a$ , then we generate a chain of frequencies  $\{f_i\}$  via Eqs. (4) and (6). 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 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 the oscillator (3),  $\mu=1$  and  $p=1$ . The only parameters allowed to

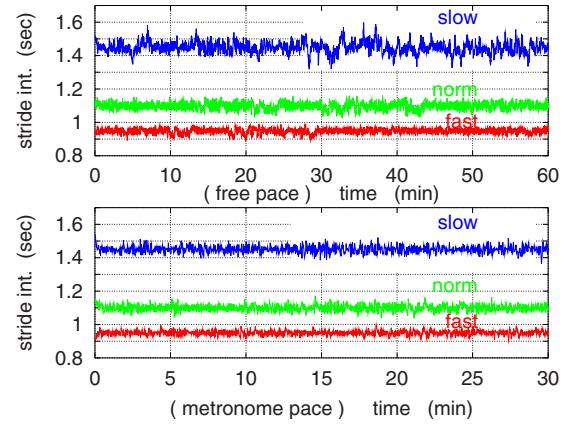


FIG. 7. (Color online) Typical computer-generated stride interval time series using the SCPG model in the freely walking and metronome paced conditions for normal, slow, and fast paces. The simulated data appear qualitatively similar to those depicted in Fig. 1.

change in the model are the mean frequency  $f_0$ , which changes also the value of  $r_0$  via Eq. (14), and the intensity  $A$  of the driver of the Van der Pol oscillator (3).

Figure 7 shows the simulated stride interval time series for slow, normal, and fast gaits using SCPG for both unconstrained and constrained walks. For the simulation of normal gait we use  $A=1$ , and for both slower and faster gaits we use  $A=2$ . For the metronomically constrained gait we use  $A=4$  for normal gait and  $A=8$  for both slower and faster gaits. We suppose that the amplitude  $A$  of the driver of the Van der Pol oscillator (3) should be smaller for normal gait than that for either 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 normal gait because normal gait is the most relaxed, spontaneous, and, consequently, the most automatic of the three gaits. For the same reason the amplitude  $A$  is increased in the metronomically constrained gait because this condition increases the stress on a subject. 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. By comparing the top and bottom panels of the figure we note the increase in randomness, the loss of persistency, and the reduction in volatility; all effects are induced in the latter time series by increasing the value of  $A$  and are found in the phenomenological data shown in Fig. 1.

Figure 8 shows the histograms of distributions of the Hölder exponents for the six computer-simulated gaits depicted in Fig. 7. The graphs in Fig. 8 reveal patterns similar to those shown in Fig. 2 for the empirical data. The center of the distribution of Hölder exponent  $h_0$  and the width of the distribution increases for both slow and fast paces relative to the unconstrained walking, as observed in the real stride interval data. For the metronomically constrained gait the SCPG model also simulates rather well the observations shown in Fig. 2, in particular, the shift toward lower Hölder exponents implies that the neuronal response to the external stimulus is to produce more randomness in sequences. Also for both slow and fast paces the sequences become more random than that of the normal pace, as is also found in the

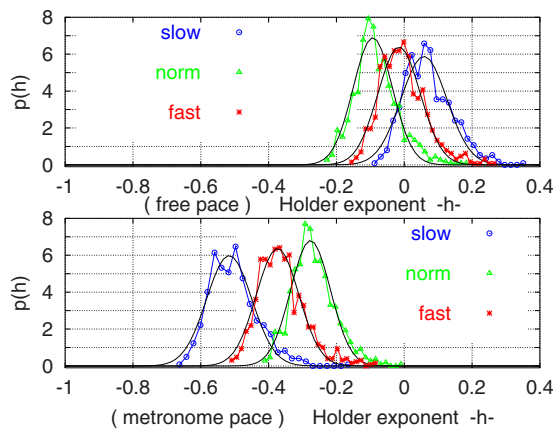


FIG. 8. (Color online) Typical Hölder exponent histograms for computer-generated stride interval series using the SCPG model in the freely walking and metronome paced conditions for normal, slow, and fast paces. The parameters of the SCPG model were chosen in such a way to approximately reproduce the average behavior of the fractal and multifractal properties of the phenomenological data. The histograms are fitted with Gaussian functions. The results appear qualitatively similar to those depicted in Fig. 2.

data, as a consequence of the fact that under anomalous fast or slow paces, an external stress, such as a metronome, can significantly disrupt the gait dynamics.

## V. DISCUSSION

An important question our study raises is which aspects of bipedal locomotion are passively controlled by biomechanical properties of the body and which aspects are actively controlled by the nervous system. It is evident that the rhythmic movements are regulated by both feedforward and feedback control.<sup>2</sup> Thus, there is no simple answer to the above question because both the biomechanical properties of the body and the nervous system are entangled and both can contribute to the peculiar variability patterns we have observed. Whether some degree of stride variability can also occur in an automated passive model for gait, for example, a walking robot, is a realistic expectation, in particular, if the robot can adjust its movements according to the environment. However, human locomotion may be characterized by additional peculiar properties that emerge from its psychobiological origin that naturally generates  $1/f$  scaling and long-range power-law correlated outputs.<sup>41</sup>

The stride interval of human gait presents a complex behavior that depends on many factors. The interpretation of gait dynamics that emerges from our SCPG model is as follows: the frequency of walking may be associated with long-time correlated neural firing activity that induces a virtual pace frequency, nevertheless, the walking is also constrained by the biomechanical motor control system (MCS) that directly controls the movement and produces the pace itself. Therefore, we incorporate both the neural firing activity given by a stochastic CPG and the motor control constraint that is given by a nonlinear filter characterized by a limit cycle. Therefore, we model our SCPG 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 length  $r_0$  of Eq.

(5), 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. (3), and, of course, the mean frequency  $f_0$  of the actual pace that distinguishes the slow, normal, and fast gait regimes. Other parameters  $\gamma$ ,  $\rho$ ,  $\mu$ , and  $p$  may be to a first-order approximation fixed by the background conditions.

Walking is also significantly influenced by two different stress mechanisms: (a) a natural stress that increases the correlation of the nervous system and regulates the motion at the changing of the gait regime from a normal relaxed condition to a consciously forced slower or faster gait regime and (b) a psychophysical stress due to the constraint of following a fixed external cadence such as a metronome. A psychophysical control, like that induced by a metronome, breaks the long-time correlation of the natural pace and generates a large fractal variability of the gait regime.

The SCPG model is able to mimic much of the complexity of the stride interval sequences of human gait under several conditions of slow, normal, and fast regimes for both walking freely and keeping the beat of a metronome. The model is based on the assumptions that human locomotion is regulated by both the central nervous system (NCS) and the intraspinal nervous system (INS). A network of neurons produces a fractal output that is correlated according to the level of physiologic stress and this network is coupled to the INS that generates the rhythmic process of the pace. The combination of the two networks controls walking and the variability of the gait cycle. It is the period of the gait cycle that is measured in the data sets and the SCPG model faithfully reproduces the stochastic and fractal characteristics of those phenomenological data. The correlation length in the SCPG determines the natural stress discussed in (a), whereas the amplitude of the driver models the psychological stress of the metronome in (b). Finally, the SCPG correctly prognosticates that the decrease in average of the long correlation of the stride interval time series for children and for the elderly or for those with neurodegenerative diseases can be understood as a decrease in the correlation length among the neurons of the MCS due to neural maturation and neurodegeneration, respectively.

## ACKNOWLEDGMENTS

N.S. thanks the Army Research Office for support (Grant No. W911NF-06-1-0323).

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