A nonlinear dynamical model of human gait

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We present a nonlinear stochastic 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 become 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. The 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.

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I. INTRODUCTION

Walking is a complex process which we have only recently begun to understand through the application of nonlinear data processing techniques [1, 2, 3] to study interval data. It has been known for over a century that there is variation of 3-4% in the stride interval of humans during walking, and only in the last decade did Hausdorff et al. [4] 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 [5, 6] 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 [7, 8].

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) [9], 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 [10]. Therefore a quadruped required 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 gait 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 regulate only the unperturbed inner frequency of a nonlinear forced Van der Pol oscillator [11] 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, like the Van der Pol oscillator, allows a more complex behavior that may be controlled also by an constraint that forces the oscillator to follow a particular fixed frequency.

The SCPG model is tested on a data set available in the public domain archives Physionet [12]. These data were originally collected and used by Hausdorff et al. [13] 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 10 healthy young men walking at a slow, normal and fast pace, for a period of one hour. The same individuals are then requested to walk at a pace determined by a metronome set at the average slow, normal and fast pace for 30 minutes to generate a second data set.

By estimating the Hölder exponents and their spectra using wavelet transform [14], it was shown that the stride interval time series is weakly multifractal with a main fractality close to that of the 1/f-noise. The time series is sometimes non-stationary and its fractal variability changes in the different gait mode regimes [15]. In particular, the persistence, as well as the multifractality of the stride interval time series, tends to increase for both slow and fast pace, 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. 2 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 stride interval time series. Sec. 3 reviews the stochastic properties of the normal and metronomic gait under different various pace velocities, slow, normal and fast. In Sec. 4 we present the mathematical details of the SCPG model. In Sec. 5 we compare the results of computation using the SCPG model with the phenomenological data. Finally, in Sec. 6 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, 14]. 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 on the walking cycle [13]. 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 [16], as well as in quadrupeds [4]. These characteristics, such as the switching among multiple gait patterns, is shown to not depend on the detailed dynamics of the constituent nonlinear oscillators, nor on their inter-oscillator 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 approximately 3-4% [8]. This random variability has been shown [7, 13] 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 operates in the unstable, that is, in the chaotic regime.

A stochastic version of a CPG was developed by Hausdorf et al. [7, 12] to capture the fractal properties of the inter-stride interval time series. This stochastic model was later extended by Ashkenazy et al. [7] 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 et al. [7] focused on explaining the changes in the gait time series during maturation, using the 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 et al. [7], 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 variability 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 data discussed in detail elsewhere [7]. We have analyzed the gait time series of 10 persons in the three different conditions of slow, normal and fast walking. Each time series is approximately one hour long for unconstrained walking, see, for example Fig. 1, for slow, fast and normal walking. Similarly, each time series is approximately 30 minutes long for metronically constrained walking, see, for example, Fig. 2, for slow, fast and normal walking. 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 years (range: 18-29 years); mean height 1.77 ± 0.08 meters 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 meters), approximately oval path and the stride interval was measured using ultra-thin, 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. More details regarding the collection of data can be found at Physionet [11] from where the data were downloaded and in Ref. [7, 12].

The fractal and multifractal analysis of the data is done by studying the estimated distribution of the local Hölder exponents using wavelet transforms [13, 18, 19, 20]. To better understand the meaning of the Hölder exponent $h$ we recall that the relation between the Hölder and Hurst exponents $H$ in the continuum limit of a monofractal noise is $h = H − 1$ according to the notation adopted in [3]. 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. Therefore, the fractal properties of the data can be studied by determining the mean value of the distribution of Hölder exponents and the multifractal properties are given by the width of the distribution itself. However, we stress that a time series of finite length will have a Hölder-exponent distribution with a non-zero width. The existence of such a non-zero 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 only be distinguished from a monofractal time series of the same length, if the width of its Hölder-exponent distribution is significantly larger than that of a correspondent monofractal time series [7].

Typical distributions of Hölder exponents, for unconstrained walking of a single individual, are depicted in Fig. 3. Fig. 4 shows the average distributions of the Hölder exponents for the cohort of 10 walkers. Figs. 3 and 4 show that stride interval time series for human gait are characterized by strong persistent fractal properties very close to that of 1/f-noise, $h ≈ 0$. However, normal gait is usually slightly less persistent than both slow and fast gait. The slow gait has the most persistent fluctuations and may present non-stationary properties, $h > 0$. The slow gait fluctuation may deviate most strongly from person to person. The higher values of the Hölder exponents for both slow and fast gait, 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 Hölder exponents for the different gait with the widths for a corresponding monofractal noise of the same length has proven that the stride interval of human gait is only weakly multifractal [5]. However, the multifractal structure is slightly more prominent for fast and slow gait than for normal gait.

Fig. 5 shows typical distributions of the Hölder exponents for metronome-constrained walking, which is little different from the histograms in Fig. 3. Fig. 6 shows the average distributions of the Hölder exponents for all 10 walkers. The figures clearly indicates that under the constraint of a metronome, the stride interval of human gait become 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/she walks more naturally. The fast gait appears to be almost uncorrelated noise because the distribution of 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 results 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 breaks the long-time correlation of the natural pace and generates a large fractal variability of the gait regime. In the next section we present a multifractal CPG model 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 [6] 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, that has an internal origin, increases the correla-
tion 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 regulate only the inner virtual frequency of a forced Van der Pol oscillator [8]. 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 [13], we can assume that the time series of the intensity of the impulses fired by the neural centers is associated to a time series of a virtual frequencies\(\{f_j\}\). So, in the spirit of the model suggested by Ashkenazy et al. [17], 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, that fires an impulse with a particular intensity that is associated to a particular virtual frequency. Ashkenazy et al. [17] 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 \(C\) 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 neural maturation and, therefore, the standard deviation \(C\) 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 et al. [17] 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 not only to describe 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 \(\pi\) 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] that is defined by the following equation

\[
\ddot{x} + \mu (x^2 - \mu^2) \dot{x} + (2\pi f_j)^2 x = A \sin(2\pi f_0 t) .
\]  

(1)

The parameter \(p\) controls the amplitude of the oscillations, \(\mu\) controls the degree of non linearity of the oscillator, \(f_j\) is the inner virtual frequency of the oscillator during the \(j-th\) cycle that is related to the intensity of the \(j-th\) 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 \(f\) if \(A = 0\).

We notice that the non linear 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 frequency \(f\). We assume that at the conclusion of each cycle, a new cycle is initiated with a new inner frequency \(f_j\) produced by the stochastic CPG model while all other parameters are kept constant. However, the simulated stride interval 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 frequencies \(\{f_i\}\) are centered around the driver frequency \(f_0\) according to the relation

\[
f_i = f_0 + \gamma X_i
\]  

(2)

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

\[
\frac{< X_i X_{i+r} >}{< X_i^2 >} = \exp \left[ -\frac{r}{r_0} \right].
\]  

(3)

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 [22], that is generated by the recursion equation

\[
X_i = a X_{i-1} + \varepsilon_i ,
\]  

(4)

where \(0 < a < 1\) is a constant and \(\{\varepsilon_i\}\) is a normalized zero-centered discrete Gaussian process. It is easy to prove [22] that the autocorrelation function of the chain \(\{X_i\}\) is given by

\[
\rho(r) = \frac{< X_i X_{i+r} >}{< X_i^2 >} = a^r .
\]  

(5)

A direct comparison between Eqs. [8] and [3] 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. [17], we assume that a frequency is activated by the position of a random walker given by the
function \( g(j) \) with \( j = 1, 2, \ldots \), whose jump sizes follow a Gaussian distribution of width \( C \). The width of this distribution, according to the interpretation of Ashkenazy et al. \([17]\), 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. Finally, the sequence of frequencies \( \{f_j\} \) is used in Eq. (6) recursively.

Normal gait, characterized by the frequency \( f_{0,n} \), is assumed to occur when the individual is relaxed, 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 increases. This increase in the stress is modeled by using the short-time correlation parameter \( r_0 \) of the stochastic CPG by assuming

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

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

We observe that in our experimental data set we measure the stride intervals of the gait. It is true that the frequency of walking may be associated with a long-time correlated neural firing activity that induces virtual pace 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.

\[\text{V. SIMULATED STRIDE INTERVAL GAIT}\]

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 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 second; the frequency of the slow and fast gait 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 seconds, respectively, that 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 \([17]\), that is, \( C = 25 \) 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. \([17]\). To generate an artificial sequence with a variability compatible to that of the experimental sequence, we chose \( B = 50 \) in Eq. (6) and, in Eq. (4), \( \gamma = 0.02 \), that is a value compatible to the average of the standard deviation of all the data analyzed by us \([7]\), however, the value of \( \gamma \) 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. (4). So, we choose a frequency \( f_0 \), calculate \( r_0 \) via Eq. (6) and the Markovian parameter \( \alpha \), then we generate a chain of frequencies \( \{f_j\} \) via Eqs. (4) and (5), and, 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 the oscillator \([11]\), \( \mu = 1 \) 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. (5), and the intensity \( A \) of the driver of the Van der Pol oscillator \([11]\).

Fig. 7 shows the stride interval time series for slow, normal and fast computer-simulated gaits using SCPG. For the simulation of normal gait we use \( A = 1 \) and for both slower and faster gait, we use \( A = 2 \). We suppose that the amplitude \( A \) of the driver of the Van der Pol oscillator \([11]\) should be smaller for 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 normal gait because normal gait is the most relaxed, spontaneous and, 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.

Fig. 8 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 SCPG used to produce the sequences of Fig. 7. We only change the intensity $A$ of the driver of the Van der Pol oscillator \( h \). We use for the normal gait $A = 4$ and for both slower and faster gait $A = 8$. Again we suppose that the intensity $A$ of the driver of the Van der Pol oscillator \( h \) should be smaller than that for both slower and faster gaits, because normal gait is the most relaxed and spontaneous. By comparing Figs. 7 and 8 we note the increase in randomness, the loss of persistence 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 phenomenologic data shown in Figs. 1 and 2.

Fig. 9 shows histograms of distributions of the Hölder exponents for the three computer-simulated gaits shown in Fig. 7. The calculation are done in the same way of 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 behavior of real data. By changing the gait mode from slow to normal, the mean Hölder exponent $\tilde{h}$ 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. (8). 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.

Fig. 10 shows the histograms of probability density estimations of the Hölder exponents for the three metronome-triggered computer-simulated gaits shown in Fig. 8. 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 \( h \), 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/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. Fig. 6 shows that the usually normal metronome-triggered gait is that with the highest persistent fractal properties. 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 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 Fig. 9 and Fig. 10 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 the more sensitive to a voluntary constraint and, so, and so the slower mode has the larger variability. In fact, our human experience and the superposition of the distributions of Hölder exponents for the 10 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 new kind of CPG model. One 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 data sets 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 regulate only the inner frequency of a forced Van der Pol oscillator. However, it is the 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 intensity are able to generate only a potential, but not an actual frequency. The chaotic behavior of such a nonlinear oscillator, like
the Van der Pol oscillator, and the possibility to force the frequency of the cycle with an external fixed frequency allows 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, Ref. [24] reports 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. (4). 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.

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[1] J.M. Hausdorff, C.-K. Peng, Z. Ladin, J.Y. Wei and A.L. Goldberger, “Is Walking a Random-walk - Evidence for Long-range Correlations in Stride Interval of Human Gait,” J. Appl. Physiol. 78 (1), 349-358 (1995).
[2] J.M. Hausdorff, L. Zemany, C.-K. Peng and A.L. Goldberger, “Maturation of Gait Dynamics: Stride-to-stride Variability and its Temporal Organization in Children,” J. Appl. Physiol. 86 (3), 1040 (1999).
[3] B.J. West and L. Griffin, “Allometric Control, Inverse Power Laws and Human Gait,” Chaos, Solitions & Fractals 10 (9), 1519-1527 (1999).
[4] B.J. West and L. Griffin, “Allometric Control of Human Gait,” Fractals 6 (2), 101-108 (1998).
[5] Vierordt, Ueber das Gehen des Meschen in Gesunden und Kranken Zustaenden nach Selb-stregistrieden Methoden, Tuebigen, Germany (1881).
[6] L. Griffin, D.J. West and B.J. West, “Random stride intervals with memory” J. Biol. Phys. 26, 185-202 (2000).
[7] N. Scafetta, L. Griffin and B. J. West, “Holder exponent spectra for human gait,” unpublished (2002), cond-mat/0208028.
[8] J.J. Collins and S.A. Richmond, “Hard-wired Central Pattern Generators for Quadrupedal Locomotion,” Biol. Cyb. 71 (5), 375-385 (1994).
[9] J.J. Collins and I.N. Stewart, “Coupled Nonlinear Oscillators and the Symmetries of Animal Gaits,” J. Nonlinear Sci. 3, 349-392 (1993).
[10] J. Guckenheimer and P. Holmes, Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields, Springer-Verlag, New York USA 2002.
[11] http://www.physionet.org/.
[12] J.M. Hausdorff, P.L. Purdon, C.-K. Peng, Z. Ladin, J.Y. Wei, A.L. Goldberger, “Fractal Dynamics of Human Gait: Stability of Long-range Correlations in Stride Interval Fluctuations,” J. Appl. Physiol. 80, 1448-1457, (1996).
[13] Z. R. Struzik, “Determining Local Singularity Strengths and their Spectra with the Wavelet Transform,” Fractals, Vol. 8, No. 2, 163-179 (2000).
[14] J. M. Winters and P. E. Crago, Biomechanics and Neural Control of Posture and Movements, Spring-Verlag, New York 2000.
[15] M.D. Mann, The Nervous System and Behavior, Harper & Row, Philadelphia (1981).
[16] A.H. Cohen, S. rossignol, and S. Grillner, Editors, Neural control of rhythmic movements in vertebrates, Wiley, New York (1988).
[17] Y. Ashkenazy, J.M. Hausdorff, P. Ivanov, A.L. Goldberger and H.E. Stanley, “A Stochastic Model of Human Gait Dynamics,” cond-mat/0103119 v1.
[18] S. G. Mallat, A Wavelet Tour of Signal Processing (2nd edition), Academic Press, Cambridge (1999).
[19] J. F. Muzy, E. Bacry, A. Arneodo, “The Multifractal Formalism Revisited with Wavelets,” Int. J. Bifurc. Chaos 4, No. 2, 245-302 (1994).
[20] P. C. Ivanov, M. G. Rosenblum, L. A. Nunes Amaral, Z. R. Struzik, S. Havlin, A. L. Goldberger and H. E. Stanley, “Multifractality in Human Heartbeat Dynamics,” Nature 399, 461-465 (1999).
[21] B.B. Mandelbrot, The Fractal Geometry of Nature, Freeman, New York, (1983).
[22] M.B. Priestley, Spectral analysis and time series, Academic Press, UK 2001.
[23] J.B. Bassighwaighte, L.S. Liebowitch and B.J. West, Fractal Physiology, Oxford University Press, Oxford (1994).
[24] J.M. Hausdorff, Y. Ashkenazy, C-K. Peng, P.C. Ivanov, H.E. Stanley and A.L. Goldberger, “When Human Walking Becomes Random Walking: Fractal Analysis and Modeling of Gait Rhythm Fluctuations,” Physica A 302 138-147 (2001).
FIG. 1: Stride interval for slow, normal and fast gait. The period of time over which measurements were done is approximately one hour.

FIG. 2: Stride intervals for slow, normal and fast gait for metronomic triggered walking. The total period of time is approximately 30 minutes.
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 ($h_0 = -0.012, \sigma = 0.056$) gait for a single individual. The fitting curves are Gaussian functions with average $h_0$ and standard deviation $\sigma$. 
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 gait. Each curve is an average over the 10 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 increasing of the width of the distribution $\sigma$ by moving from the normal to the slow or fast gait 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 ($h_0 = -0.035, \sigma = 0.081$) gait.
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 ($h_0 = -0.436, \sigma = 0.066$).

FIG. 6: Metronomic walking. Histogram estimation of the Hölder exponents for the three walking groups: slow-star, normal-triangle and fast-circle gait. Each curve is an average over the 10 cohorts in the experiment.
FIG. 7: Stride interval time series for slow, normal and fast computer-simulated gaits.

FIG. 8: Stride interval time series for slow, normal and fast gait for metronome-triggered computer-simulated gait.
FIG. 9: 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 \( h_0 = -0.015, \sigma = 0.063 \) gait for a single individual.

FIG. 10: 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 \( h_0 = -0.373, \sigma = 0.063 \) gait for a single individual.