Statistical quantifiers of memory for an analysis of human brain and neuro-system diseases

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1. Introduction: Time series analysis to study human movement

The challenge of identifying statistical memory effects occurring in time series analysis of discrete, temporal data sets of medical disorders continues to attract considerable attention in various cross disciplines. This is so because the ultimate goal is always the construction of suitable statistical quantifiers that carry the potential to indicate and quantify the underlying medical disorder. In this context, the time series analysis presents an important tool, which can be put to work to characterize the complex behavior in medical physics.

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With this work we shall demonstrate the relevance and the critical role of statistical memory effects for the functioning of healthy physiological systems. We will show that the enduring existence of statistical memory in discrete stochastic dynamics of living systems indicates a reliable manifestation of pathological (or catastrophical) abnormalities. Particularly, the manifestation of memory effects in human locomotion dynamics can be related to different brain and central nervous system disorders.

Historical notes

Locomotion refers to the variety of human or animal mobility, related to their forward movement in physical space. In a more narrow sense the term *locomotion* is used to quantify biomechanical movements of objects [1]. This topic has its roots in the ancient Greeks (Socrates, Plato, Aristotle), who produced a record of human behavior and action in relationship to our powers of perception. More specifically, the first biomechanical studies were undertaken by the famous scientist Leonardo da Vinci, who studied human motions on the basis of anatomy and mechanics. The Italian naturalist Giovanni Alfonso Borelli made an important contribution to biomechanics. In his book, "*De motu animalium*" (1680–1681) he analyzed the mechanical motions occurring in humans and in animal joints while walking, running and swimming [2]. The first experimental studies of human walking were conducted in Germany by Weber and Weber as early as in 1836 [3] and also by Braune and Fischer (1895) [4,5], in France by Marey (1863) [6], in America by W.O. Fenn (1935) [7], H. Elftman (1938) [8,9] and others (for example, E. Harless, H. von Helmholtz, E. Muybridge, H. von Meyer). In Russia the development of biomechanics is connected with works in theoretical anatomy by P.F. Lesgaft (1905) and the book of I.M. Sechenov "*Ocherk rabochih dvizeniy cheloveka*" (1901), containing the summary of the important biomechanical characteristics of human motions. The detailed studies of human locomotion were done by Bernstein et al. [10] who with collaborators has analyzed the walking of healthy children and elderly people.

Human gait dynamics

Walking is one of the most prominent type of human locomotion. The analysis of human walking is of salient interest to biomedicine, biomechanics and physiology. Characterizing locomotion dynamics is essential for understanding the neuro-muscular control of locomotion. In particular, quantifying the dynamic stability during walking is important for identifying people who have a greater risk of stumbling and falling [11,12]. Despite its well-developed experimental methods this field still lacks good and reliable theoretical quantitative indicators for human gait disorders. Interesting results in this field have been derived before by Hausdorff et al. [13–21]. They studied the dynamical changes of human gait, connected with various diseases [13–16], the increasing instability of gait in elderly people [13,16–18], presence of long-range correlations in stride interval fluctuations [19,20], stride-to-stride variability and its temporal organization in children [21]. Walking indeed constitutes a complex process which we only recently started to understand through the application of non-linear data processing techniques [12,22–27].

In this work we propose a study of gait disorders by the use of statistical methods that are based on a so-termed memory function approach, as has been put forward in a different context in a recent work [28].

2. Theoretical description of memory effects in discrete stochastic dynamics of complex systems

Statistical physics makes available a wealth of methods which are suitable and effective in analyzing the statistical memory effects inherent in discrete time stochastic dynamics of complex systems. Such typical concepts include the generalized master equations and their corresponding statistical quantifiers [29–33], Lee's recurrence relation method [34–37], the generalized Langevin equation [38–41], the tailored generalized master equation approach for studying the ageing effects in systems with non-Poisson distribution [42], to name only a few. Likewise, the kinetic approaches of Zwanzig and Mori [43–47] are one of the most simple analytical methods for studying the statistical memory effects in various systems such as the ones being considered here.

An important feature of complex systems is the great number of degrees of freedom that underlie their coarse grained, relevant dynamics. The Zwanzig–Mori projecting formalism allows one to derive the exact equations of motion for the relevant dynamical variables. The dynamics of other irrelevant degrees of freedom are hidden in the so-called form of a memory term which is related statistically to fast fluctuating forces. Although there exist other projection techniques as well, such as those in Refs. [48–50], the functional structure of the Zwanzig–Mori kinetic equations is independent of the considered complex system [51].

Memory function formalism

Time correlation functions are one of the most convenient tool for the analysis of the time correlations in complex systems. In prior works [52,53] the chain of interconnected finite-difference equations for the discrete time correlation function (TCF):

$$a(t) = \frac{\langle \mathbf{A}_k^0 \cdot \mathbf{A}_{m+k}^m \rangle}{\langle |\mathbf{A}_k^0|^2 \rangle}, \tag{1}$$

has been obtained. Here, the normalized TCF $a(t)$ can be represented as a scalar product of vectors of the initial set:

$$\mathbf{A}_k^0 = \{\delta x_0, \delta x_1, \ldots, \delta x_{k-1}\} = \{\delta x(T), \delta x(T+\tau), \ldots, \delta x(T+(k-1)\tau)\} \tag{2}$$

and final set:

$$\begin{aligned} \mathbf{A}_{m+k}^m &= \{\delta x_m, \delta x_{m+1}, \ldots, \delta x_{m+k-1}\} \\ &= \{\delta x(T+m\tau), \delta x(T+(m+1)\tau), \ldots, \delta x(T+(m+k-1)\tau)\} \end{aligned} \tag{3}$$

system states. The stochastic discrete process is described by the time series $\{x_j\}$ of a certain stochastic variable:

$$X = \{x(T), x(T+\tau), \ldots, x(T+(N-1)\tau)\}, \tag{4}$$

where T is the start time of signal registration, $(N-1)\tau$ is the total registration time, τ is the discretization time step, $x_j = x(T+j\tau)$ is the value of X and $\delta x_j = x_j - \langle X \rangle$ is the fluctuation on the jth time step, $\langle X \rangle = 1/N \sum_{j=0}^{N-1} x_j$ is the mean value of X. The initial time correlation function $a(t) = M_0(t)$ is related to the memory functions of higher order $M_n(t)$, where $n = 1, 2, \ldots$, by means of the finite-difference kinetic equations:

$$\frac{\triangle M_{n-1}(t)}{\triangle t} = \lambda_n M_{n-1}(t) - \tau \Lambda_n \sum_{j=0}^{m-1} M_n(j\tau) M_{n-1}(t-j\tau). \tag{5}$$

Here, λ_n is the eigenvalue and Λ_n denotes the relaxation parameter of Liouville's quasi-operator $\hat{L}$:

$$\lambda_n = \mathrm{i}\frac{\langle \mathbf{W}_{n-1} \hat{L} \mathbf{W}_{n-1} \rangle}{\langle |\mathbf{W}_{n-1}|^2 \rangle}, \qquad \Lambda_n = \frac{\langle |\mathbf{W}_n|^2 \rangle}{\langle |\mathbf{W}_{n-1}|^2 \rangle}. \tag{6}$$

$\mathbf{W}_n$ are the dynamic orthogonal variables, defined by the Gram–Schmidt orthogonalization procedure $\langle \mathbf{W}_n, \mathbf{W}_m \rangle = \delta_{n,m} \langle |\mathbf{W}_n|^2 \rangle$ ($\delta_{n,m}$ is Kronecker's symbol), which is used for the vectors of the system state by the following way:

$$\begin{aligned} &\mathbf{W}_0 = \mathbf{A}_k^0(0), \qquad \mathbf{W}_1 = (\mathrm{i}\hat{L} - \lambda_1)\mathbf{W}_0, \qquad \mathbf{W}_2 = (\mathrm{i}\hat{L} - \lambda_2)\mathbf{W}_1 - \Lambda_1 \mathbf{W}_0, \ldots, \\ &\mathbf{W}_n = (\mathrm{i}\hat{L} - \lambda_n)\mathbf{W}_{n-1} - \Lambda_{n-1}\mathbf{W}_{n-2} - \cdots. \end{aligned} \tag{7}$$

These equations constitute the finite-difference analogue of Zwanzig–Mori kinetic equations, derived from the microscopic equations of motion. The initial TCF, the memory functions, the kinetic coefficients λ_n and relaxation parameters Λ_n determine the diversity and characteristics of the memory effects in the discrete time evolution of complex systems. It is worthwhile to point out that the full time dependence of the TCF, the memory functions, the kinetic and the relaxation parameters are all evaluated from the discrete time series.

3. Experimental data — the duration of the gait cycle

As a first case we start out from the experimental data set for the stride-to-stride variations of the gait cycle timing in Parkinson's disease, Huntington's disease, amyotrophic lateral sclerosis and compare those with healthy control subjects (for further details, see in Refs. [13–15] and *Research Resource for Complex Physiologic Signals*: http://www.physionet.org).

Each group contains 10 people. The average ages of the control subjects, HD subjects, PD subjects and ALS subjects were at 37 (range, 20–69), 50 (range, 34–66), 70 (range, 57–79) and 52 (range, 36–70), respectively. The

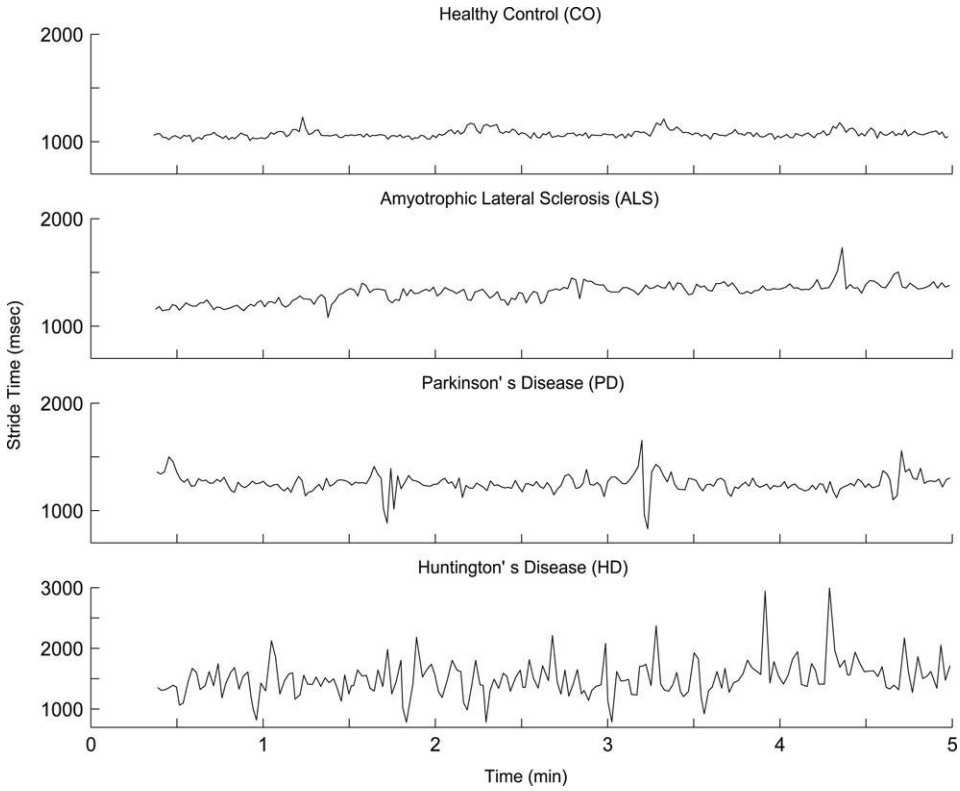

Fig. 1. Examples of typical time series for the stride time dynamics (gait cycle duration) from four members of the group of healthy, ALS, PD, and HD patients. The values of mean-squared amplitude $\langle A \rangle$ for locomotion dynamics of these time series are 1.06, 1.32, 1.26 and 1.49 (a.u.), respectively. The dispersion σ^2 for the stride time dynamics of these four subjects are 0.033, 0.181, 0.163 and 0.286 (a.u.), respectively.

control and the HD subjects were predominantly female (9 of 10 control subjects and 8 of 10 HD subjects), while subjects with PD and ALS were predominantly male (7 of 10 and 8 of 10, respectively). The heights and the weights of the subjects in the four groups were not significantly different (and the heights and the weights were not significantly correlated with any of the measures of gait dynamics [13–15]).

All subjects were instructed to walk at their normal pace along a 77 m-long hallway for 5 min (the first 20 seconds of the recorded data were excluded to minimize start-up effects). To measure the gait rhythm and the timing of the gait cycle, force-sensitive insoles were placed in the subject's footwear [54]. These inserts produce a measure of the force applied to the ground during ambulation. A small, lightweight ($5.5 \times 2 \times 9$ cm; 0.1 kg) recorder was worn on the ankle and held in place using an ankle wallet. An on-board analog-to-digital converter (12 bit) sampled the output of the foot switches at 300 Hz and stored the data. Subsequently, the digitized data were transferred to a UNIX workstation for analysis by using software that extracts the initial contact time of each stride [54]. With this information, the stride time or duration of the gait cycle (time from initial contact of one foot to subsequent contact of same foot) was determined for each stride.

Fig. 1 depicts the stride time dynamics of control subject, ALS, PD, and HD subjects 57, 50, 74, and 70 years old, respectively, in equal time and amplitude scales (for more details, see *Research Resource for Complex Physiologic Signals*: http://www.physionet.org). The mean-square amplitudes $\langle A \rangle = \{\sum_{j=0}^{N-1} x_j^2/N\}^{1/2}$ for the locomotion dynamics of four people groups on average are 1.11, 1.31, 1.21 and 1.27 (arbitrary units), respectively. The dispersion $\sigma^2 = (1/N)\sum_{j=0}^{N-1}(x_j - \langle X \rangle)^2$ for the stride time dynamics of four people groups on average are 0.046, 0.165, 0.212 and 0.183 (a.u.), respectively.

4. Results of experimental time series analysis by simple measures of memory

The results of comparative analysis for the locomotion dynamics of healthy people and patients with different brain and CNS diseases are presented below. The first subsection represents some spectral and phase-space characteristics,

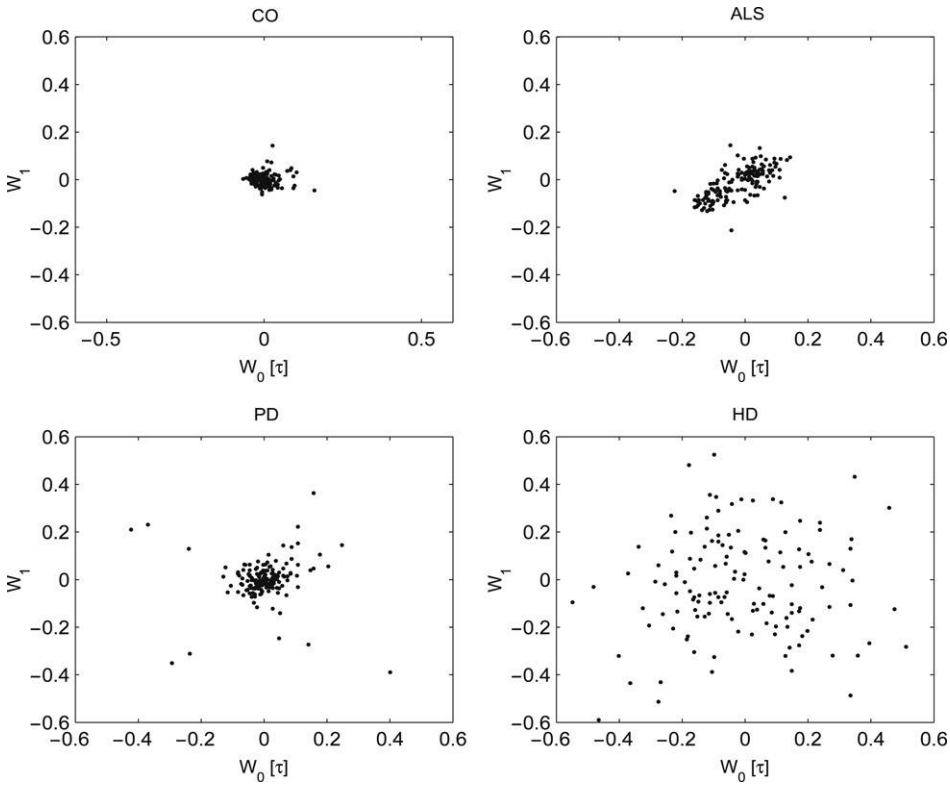

Fig. 2. The plane projections of phase portraits for the first two dynamical orthogonal variables ($\mathbf{W}_0$, $\mathbf{W}_1$) for a control healthy subject, ALS, PD, and HD subjects, respectively. The abnormal locomotion features in the different diseases result in evident deformations and changes in the scale of the corresponding phase clouds. Being typical for each presented disease, the forms of these phase portraits allow one to identify by simple graphical means the corresponding medical disorder.

regarding healthy people and patients. We shall demonstrate that the locomotion dynamics abnormalities for different diseases result in distinct deformations of the initial TCF power spectra $\mu_o(\nu)$, particularly, in the change of the fractal features, in the stratification of the phase clouds. The second subsection contains the simple measures of memory which are effective for analyzing the human gait rhythm. We will determine the way memory effects undergo some changes as a result of human locomotion dynamics abnormality. All calculations have been obtained by using the right foot.

4.1. Alteration of spectral characteristics at breakdown of human locomotion dynamics

Fig. 2 presents the plane projections of phase portraits of two dynamic orthogonal variables $\mathbf{W}_0$, $\mathbf{W}_1$, for control subject, ALS, PD, and HD subjects. All figures are derived from the initial time series. The phase-portrait structure is determined by fluctuations in the initial time signals. The most significant fluctuations result in prominent deformations of phase clouds. For the gait of a healthy subject the phase portrait consists of symmetrically centralized nucleus containing a high concentration of the phase points. Such a form of the phase cloud can be related to the small fluctuations near the zero value, as derived from the time dependence of $\mathbf{W}_0$, $\mathbf{W}_1$). Another form is observed in brain and CNS disease (including PD, HD, ALS). Small fluctuations in stride-to-stride variations of gait cycle timing, which are typical for healthy subject gait dynamics, are now changed by more pronounced fluctuations. These deviations lead to large deformations of the central nucleus in the phase clouds. The corresponding phase clouds are formed by the dynamic intermittency in the initial time series. The phase cloud for ALS subject also contains the centralized nucleus. The nuclear scale is larger than in the case of the healthy subject. For the PD's patient we observe the centered nucleus with smaller density of phase points. The large fluctuations, typical for patient's locomotion dynamics, result in a stratification of the same phase points on a plane. The nuclear scale becomes smaller than in the case of the ALS subject. An even distribution of phase points appears on the phase plane in the case of HD. Thus the

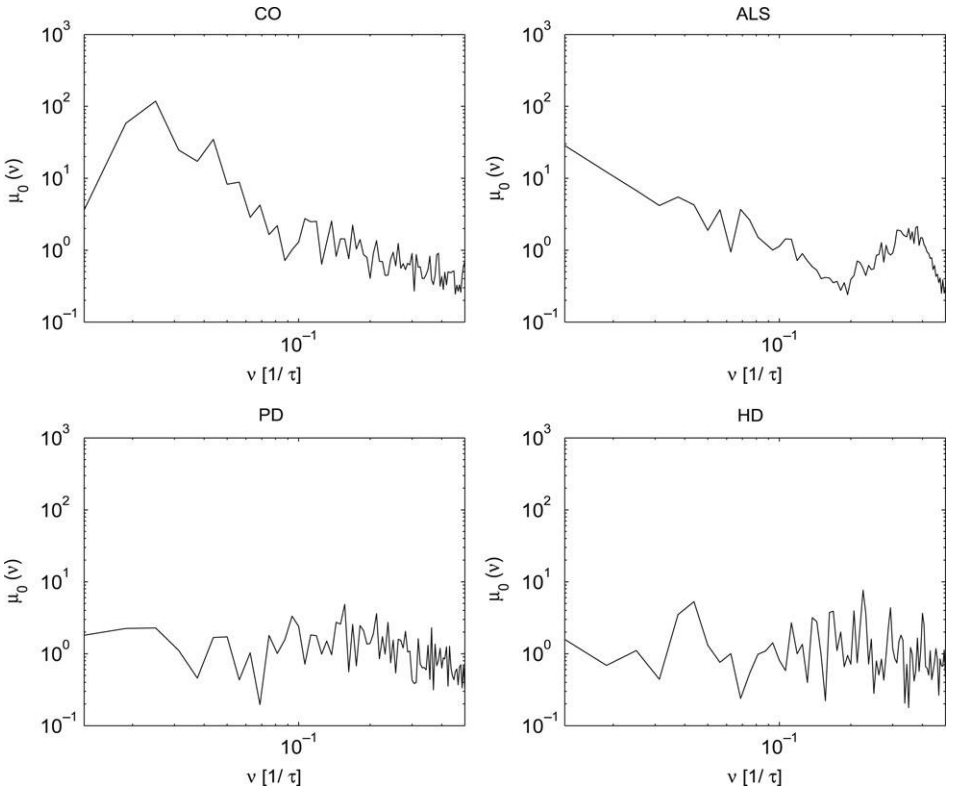

Fig. 3. The initial TCF power spectra $\mu_o(\nu)$ for gait cycle duration dynamics of a healthy control subject, ALS, PD, and HD subjects. All figures are presented on a double logarithmic scale. In the initial TCF power spectra $\mu_o(\nu)$ (CO) the fractal dependence $\mu_0(\nu) \sim 1/\nu^{\beta}$ is manifested. Such fractal behavior is typical for locomotion dynamics of healthy people. Depending on the type of brain or CNS disease a drastic change in the fractal dynamics of the human gait behavior takes place.

human gait abnormality with certain brain and CNS diseases leads to distinct deformations of the structures in phase space. The typical form of phase portraits for every disease allows one to make their identification and to illustrate the differences by simple graphical means for differentiation.

Fig. 3 depicts the initial TCF $\mu_o(\nu)$ power spectra for the gait cycle duration dynamics of control subject, ALS, PD and HD subjects. For a detailed analysis all the figures are presented on a log–log scale. In these power spectra the fractal dependence $\mu_0(\nu) \sim 1/\nu^{\beta}$ versus frequency is drawn. A finding is that every human brain and CNS disease (in particular, PD, HD, ALS) leads to a specific behavior of the fractal dynamics of human gait. Similar results have been obtained by Hausdorff et al. (see for example Refs. [13,20]). In particular, our analysis which is based on statistical physics and non-linear dynamics, reveals that these stride time dynamics for healthy people possess a fractal (self-similar) organization: stride-to-stride variations over small time scales are statistically similar to those over extended time scales.

4.2. Simple measures of memory as statistical quantifiers for singularities of human gait rhythm

A first measure of memory

As a first measure of memory we study next the relaxation time scales of the initial TCF $a(t)$ and memory functions of the ith order $M_i(t)$. Originally this non-Markovian parameter, characterizing the degree of non-Markovianity of an arbitrary relaxation process, was introduced for analyzing the irreversible phenomena in condensed matters [55,56]. The relaxation times of the initial TCF (the existence of duration of correlations in the considered system) and memory functions of the ith order (the duration of existence of memory) are determined as follows: $\tau_a = \Delta t \sum_{j=o}^{N-1} a(t_j)$, $\tau_{M_1} = \Delta t \sum_{j=o}^{N-1} M_1(t_j)$. The simplest criterion for the quantitative estimation of memory in this relaxation process is determined as: $\varepsilon_1 = \tau_a/\tau_{M_1}$. When $\varepsilon_1 \gg 1$, the relaxation time of the memory function of the first order is much smaller than the relaxation time of the initial TCF. In this case the process is characterized by a

very short memory; in the limit $\varepsilon \to \infty$ it reduces to a Markovian behavior. Particularly, our prior work [57] proposes an identifier for the transition from strong memory, non-Markovian-like kinetic equations to memoryless, Markovian-like ones; this occurs when this memory parameter tends to infinity. A decrease of the parameter ε_1 characterizes the relative memory lengthening and strengthening of memory effects. Thus, the presented quantitative criterion characterizes the degree of memory and strength of memory present in the underlying relaxation process.

In Refs. [58,59] the concept of a non-Markovian parameter spectrum ε and Markovization degree for non-equilibrium processes in disordered condensed matter was introduced. These parameters are related to the properties of the system as well as to the memory function, the memory life time of the process by the initial TCF. The spectrum of non-Markovian parameter $\{\varepsilon\} = \{\varepsilon_1, \varepsilon_2, \ldots, \varepsilon_{n-1}\}$ presents a set of dimensionless ε_i values, reading:

$$\varepsilon_1 = \tau_a/\tau_{M_1}, \qquad \varepsilon_2 = \tau_{M_1}/\tau_{M_2}, \ldots, \qquad \varepsilon_{n-1} = \tau_{M_{n-1}}/\tau_{M_n}.$$

Here, τ_i denotes a relaxation time of the memory function of ith order, the number i defines the relaxation level.

In recent works [52,53] the notions of the frequency-dependent non-Markovian parameter have been generalized:

$$\varepsilon_i(\nu) = \left\{ \frac{\mu_{i-1}(\nu)}{\mu_i(\nu)} \right\}^{\frac{1}{2}}, \tag{8}$$

where $i = 1, 2, \ldots$, and $\mu_i(\nu)$ describes the frequency power spectrum of the correlation function of the ith order.

Below it will be revealed that from the frequency dependence of $\varepsilon_1(\nu)$ and the value at zero frequency of this parameter $\varepsilon_1(0)$, one is able to determine the quantitative estimation of the locomotion dynamics of patients and healthy people. We show that the values of $\varepsilon_1(0)$ allow one to differentiate the various human diseases of brain and CNS.

Fig. 4 presents the frequency dependence of the non-Markovian parameter $\varepsilon_1(\nu)$ for the stride time dynamics of control subject, ALS, PD, and HD subjects. The authors' recent studies (see, for example, Ref. [60]) allows one to reveal the peculiar parameter $\varepsilon_1(0)$, seemingly identifying the anomalous (pathological) functioning of complex systems. The information measure of memory $\varepsilon_1(0)$ allows one to estimate quantitatively the statistical memory effects in discrete time dynamics of complex systems. The values of parameter $\varepsilon_1(0)$ for four subject groups on average are 2.47 (CO), 1.63 (ALS), 1.37 (PD), 1.2 (HD). The derived values show strong memory in the gait cycle duration dynamics of patients. Particularly, in HD subjects' gait dynamics, the strong non-Markovian ($\varepsilon \to 1$) and the strong memory ($\tau_{M_1} \sim \tau_a$) effects are being observed. Thus the drastic increasing of memory effects in various brain and CNS diseases accurately reveals the serious abnormalities in human fractal locomotion dynamics.

A second measure of memory

A specific role of strong memory in patients' gait dynamics is distinctly manifested in the behavior of a second measure of memory, via the parameter δ_i at level i. In Ref. [28] this fundamental measure of the memory effects in discrete dynamics of complex systems has been defined as:

$$\delta_i(\nu) = \left| \frac{\tilde{M}_i'(\nu)}{\tilde{M}_{i+1}'(\nu)} \right|, \tag{9}$$

where $\tilde{M}_i'(\nu) = \mathrm{d}\tilde{M}_i(\nu)/\mathrm{d}\nu$ and $\tilde{M}_i(\nu)$ is the Fourier transform of the ith order memory function. This characterization of memory is based on the set of dimensionless statistical quantitative quantifiers, determining the memory effects in the time evolution of the system. The set of parameters $\delta_i(\nu)$ proves useful for quantifying the amplification of relative memory effects occurring on different complexity levels. This second measure provides a statistical criterion for the comparison of the relaxation time scales and memory time scales of the process under study. When this parameter obeys $\delta \gg 1$, the complex system dynamics can be described by the short-range temporal memory scale. In the extreme case this process can be characterized by a δ-like memory with parameter $\delta \to \infty$. In the case of $\delta > 1$ one deals with a situation of moderate memory strength, and the case where $\delta \sim 1$ typically constitutes a more regular behavior, possessing the strong memory features. Particularly, the informational parameter $\delta_1 = \delta_1$ ($\nu = 0$) is useful for analyzing the different possible stages of a complex dynamics inherent in physiological systems.

For example, in a recent work of the authors [60] it was shown, that the appearance of the strong memory in MEG signals of the patient with photosensitive epilepsy, and the transition from the chaotic to robust regime,

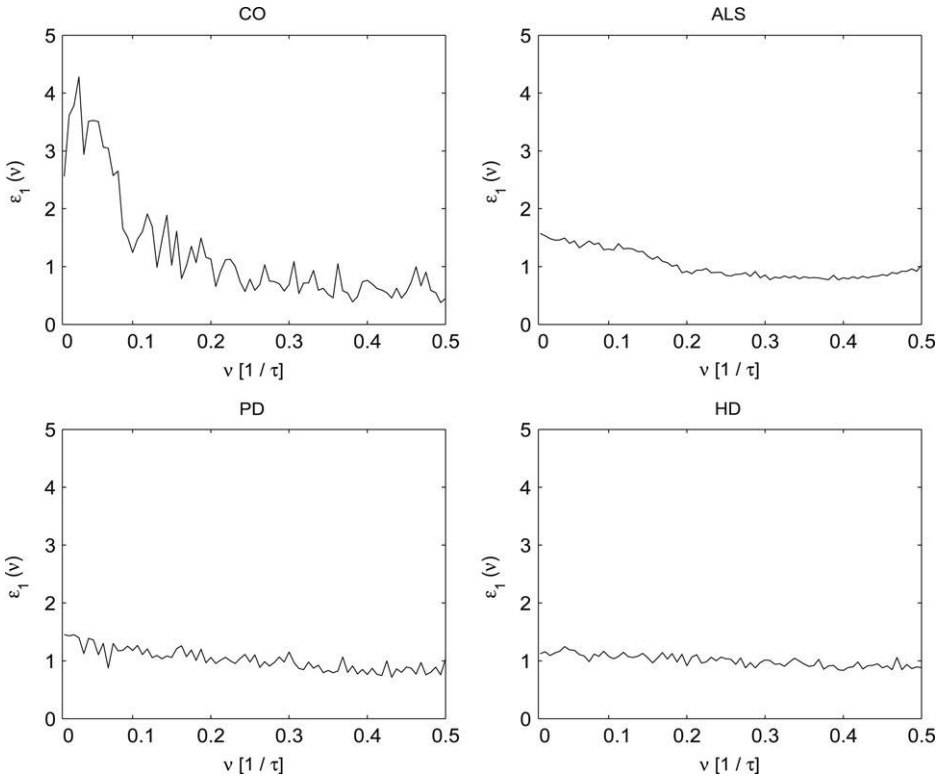

Fig. 4. The frequency dependence of the first level of the non-Markovian parameter $\varepsilon_1(\nu)$ for the stride time dynamics of control healthy subject, ALS, PD, and HD patients. This informational measure of memory allows one to estimate quantitatively the memory effects in discrete time dynamics of complex live systems. These differences become most pronounced at zero frequency. The values of parameter $\varepsilon_1(0)$ for the considered people are 2.56 (CO), 1.57 (ALS), 1.45 (PD), and 1.12 (HD). Thus, the locomotion dynamics of a healthy subject are characterized by moderate memory effects. Strong memory features occur in the gait dynamics of patients suffering from disease. In particular, strong memory is detectable in the stride time dynamics of Huntington's disease.

allow the detection of the cerebral cortex areas, forming epileptic seizure at photosensitive epilepsy. The complex relations, existing between the non-linear effects and the statistical memory effects, determine a high stability in brain functioning against certain negative influences. A prompt interaction between the different brain areas prevents the development of the collective neurons' activity, typical for the photosensitive epilepsy. Thus, the dynamics of physiological systems are manifested in many spatial and temporal scales, and the pathological states in live systems result in the changes of these spatio-temporal structures. At present special attention is given to problems of distinguishing and analyzing the stochastic and regular components of the experimental time series from biological and live systems.

Fig. 5 depicts the frequency dependence of the memory measure $\delta_1(\nu)$ for gait dynamics of four different groups of subjects. This informational measure differs significantly at zero frequency for the differing groups. This fact is connected with the stable long-range correlations in gait time duration dynamics. The parameter $\delta_1(0)$ assumes on average for the four groups the values 8.02 (CO), 2.68 (ALS), 1.96 (PD), and 1.67 (HD). This underpins the fact that the critical role of strong memory is manifested in the gait stochastic dynamics for different groups of patients. Remarkably, the frequency dependence of $\delta_1(\nu)$ (PD, HD) contains considerable amplitude bursts which are very specific for the corresponding disease.

Statistics of the measures of memory

Table 1 presents the values of the parameters $\varepsilon_1(0)$ and $\delta_1(0)$ for subjects from these four groups together with the mean values for each group. These informational measures of memory exhibit distinctly the critical role of the strong memory present in different brain and CNS diseases. The characterization of the influence of memory effects on the time evolution of complex systems, including the functioning of live systems, is possible in this way. Each of these high

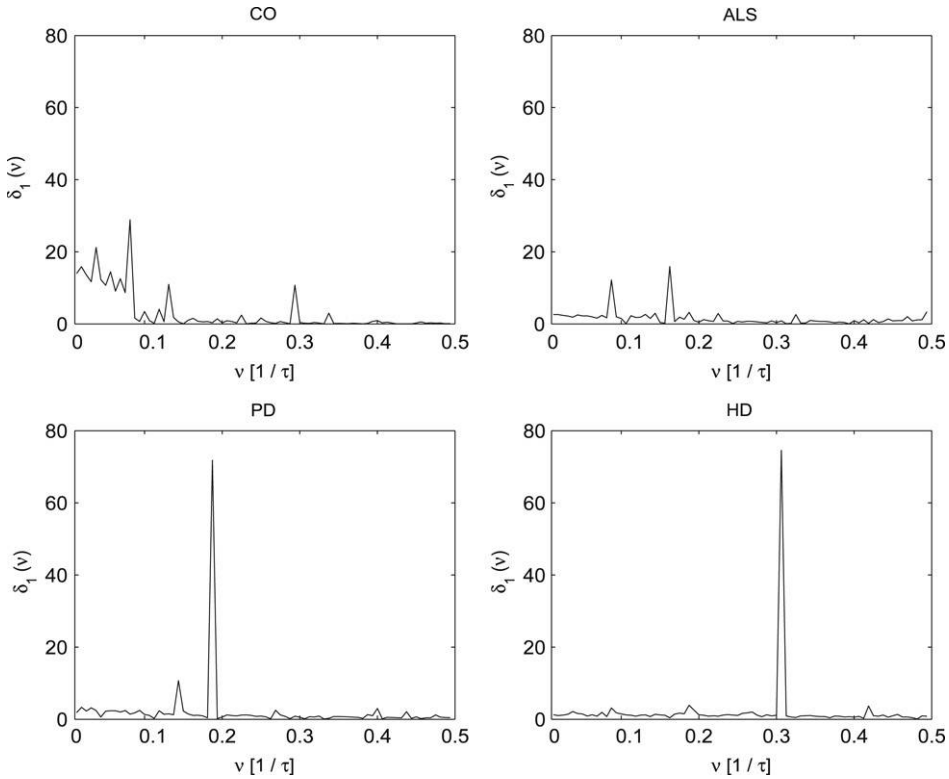

Fig. 5. The specific role of strong memory in patients' gait dynamics becomes more pronounced when studying the behavior of the second informational measure of memory $\delta_1(\nu)$. The largest differences in the numerical values of this measure for stride time dynamics in control subject, ALS, PD, and HD subjects occur at zero frequency $\delta_1 = \delta_1(0)$. The values of the parameter δ_1 for the considered subjects are 15.98 (CO), 2.56 (ALS), 1.82 (PD), and 1.22 (HD). It is evident, that the strong memory indicates the abnormalities in fractal dynamics of human gait in presence of different brain and CNS diseases.

Table 1
The values of parameters $\varepsilon_1(0)$, $\delta_1(0)$ for 10 subjects from each group (CO — control healthy group, ALS — patients with amyotrophic lateral sclerosis, PD — patients with Parkinson's disease, HD — patients with Huntington's disease)

Parameter	$\varepsilon_1(0)$				$\delta_1(0)$			
Subject	CO	ALS	PD	HD	CO	ALS	PD	HD
1	2.56	1.23	1.13	1.16	15.98	1.68	1.01	1.28
2	2.08	1.21	1.45	1.57	4.98	1.48	1.82	2.59
3	2.61	1.07	1.93	1.04	5.09	1.16	1.29	0.97
4	1.86	2.04	1.99	1.61	4.74	4.73	4.05	2.8
5	4.71	2.1	1.71	1.3	15.2	4.32	3.05	1.79
6	1.9	1.92	1.19	1.35	9.13	1.51	1.16	2.67
7	2.58	1.57	0.99	0.8	4.7	2.56	0.89	0.62
8	1.88	2.12	1.19	1.12	5.25	4.23	4.09	1.22
9	2.36	1.57	1.04	0.86	7.05	1.21	1.1	0.87
10	2.2	1.51	1.05	1.23	9.07	3.92	1.13	1.88
Mean value	2.47	1.63	1.37	1.2	8.02	2.68	1.96	1.67

Listed are also the mean values of the parameters $\varepsilon_1(0)$, $\delta_1(0)$ for each group.

dimensional complex systems possesses a great number of degrees of freedom. In a real-life situation only a minor part of these variables remain independent. The remaining continuous or discrete variables, characterizing the states of complex systems, are mutually correlated. As a rule, under normal conditions, such a dependence can be described in terms of Markovian stochastic processes. In the case of natural catastrophes, social crises or human diseases

the strong outside influence results in the particular regularization of natural chaotic behavior of complex systems and their self-organized reorganization. In this case the complex system behavior becomes more robust and more regular. This feature is accompanied by a strengthening of the non-Markovian statistical effects, i.e. the appearance of strong memory. The presented measures of memory $\varepsilon_1(\nu)$ and $\delta_1(\nu)$ provide therefore valuable quantitative criteria, characterizing serious locomotion dynamic abnormalities, connected with brain and CNS diseases.

Further we can characterize the relaxation properties in gait cycle duration dynamics with the help of the values of the relaxation parameter $\lambda_1 = \mathrm{i}\langle \mathbf{A}_k^0(0)\hat{L}\mathbf{A}_k^0(0)\rangle / \langle |\mathbf{A}_k^0(0)|^2\rangle$. It itself is an eigenvalue of Liouville's quasi-operator $\hat{L}$ [52,53]. The parameter λ_1 determines the relaxation rate of the studied process. On average the values of λ_1 for each group assume on average the values: 0.49 (CO), 0.68 (ALS), 0.82 (PD), and 0.87 (HD). The distinctions between these relaxation rates reflect the abnormalities of the fractal human gait dynamics for different brain and CNS diseases. Let us note that the locomotion dynamics of healthy people are characterized by a lower value of the relaxation rate.

5. Discussion

In this paper we have carried out a comparative analysis of discrete stochastic gait dynamics of patients suffering from Parkinson's disease, Huntington's disease and amyotrophic lateral sclerosis and compared our findings with a group of healthy subjects. Our statistical method is based upon the conception of the time correlation function and corresponding memory functions of the underlying time series, as well as upon specific statistical indicators which derive from these time series. The proposed informational characteristics for such statistical memory features are the parameters $\varepsilon_1(0)$ and $\delta_1(0)$, defined above in Eqs. (8) and (9). These quantifiers allow for an accurate quantitative assessment of the dynamical states of patients and healthy subjects. Particularly, we have identified a significant role of the statistical memory effects in human locomotion dynamics. Strong memory effects and a transition from the chaotic behavior to a robust and regular regime typically occur for the gait of the patients suffering from different brain and CNS diseases. Even a small increase of these statistical memory effects may indicate the pathological changes occurring in human brain and CNS functioning. As a result, one can observe a drastic change in the behavior of these statistical indicators and structures of the corresponding phase clouds, inherent to the patients' locomotion dynamics.

Finally, the change of human locomotion dynamics in Parkinson's, Huntington's diseases and in amyotrophic lateral sclerosis results in a characteristic variation of our statistical quantifiers. Particularly, these changes reflect the dynamical characteristics of the corresponding time series, in values and forms of phase portraits of the orthogonal variables, as well as in the initial TCF power spectra $\mu_0(\nu)$, in the power dependence $\mu_0(\nu) \sim 1/\nu^{\beta}$, the relaxation parameter λ_1, and in the behavior of the informational memory measures $\varepsilon_1(\nu)$ and $\delta_1(\nu)$.

The presented conception of time series analysis provides a useful toolkit made up of both, a set of numerically derived statistical quantifiers and a graphical scheme that can be put to work to identify and to differentiate human locomotion disorders occurring in different brain and CNS diseases. This work thus presents a first step towards the understanding of the physiological processes in human organisms. In addition to the applications described in Section 4, the presented method for analyzing the states and dynamics of complex systems may also be applied to other problems in which some fluctuation characteristics are recorded in the form of time series. Apart from the application taken from medicine in this work other important applications that come to mind are the monitoring changes in climatic systems and ecosystems, astrophysics (analysis of variations in the activity of stellar and quasi-stellar objects), geophysics (prognostication of atmospheric events, seismic activity), to name a few. In conclusion, abnormalities of human fractal-like locomotion dynamics occurring in different brain and CNS diseases (including Parkinson's disease, Huntington's disease and amyotrophic lateral sclerosis) become reflected in characteristic changes of a set of spatio-temporal memory criteria.

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