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Decrease in Hurst exponent of human gait with aging and neurodegenerative diseases*

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In this paper the decrease in the Hurst exponent of human gait with aging and neurodegenerative diseases was observed by using an improved rescaled range (R/S) analysis method. It indicates that the long-range correlations of gait rhythm from young healthy people are stronger than those from the healthy elderly and the diseased. The result further implies that fractal dynamics in human gait will be altered due to weakening or impairment of neural control on locomotion resulting from aging and neurodegenerative diseases. Due to analysing short-term data sequences rather than long datasets required by most nonlinear methods, the algorithm has the characteristics of simplicity and sensitivity, most importantly, fast calculation as well as powerful anti-noise capacities. These findings have implications for modelling locomotor control and also for quantifying gait dynamics in varying physiologic and pathologic states.

Keywords: dynamics, nonlinearity, Hurst exponent, locomotion
PACC: 0545, 0547, 8770

1. Introduction

A hallmark of physiologic systems is their extraordinary complexity. The nonstationarity and nonlinearity of signals generated by living organisms defy traditional mechanistic approaches based on homeostasis and conventional biostatistical methodologies. Recognition that physiologic time series contain 'hidden information' has fuelled growing interest in applying concepts and techniques from statistical physics,[1,2] including chaos theory,[3] fractal theory [4,5] and methods based on entropy,[6−9] to a wide range of biomedical problems from molecular[10] to organismic levels.[11−13]

The dynamics of a kind of physiologic control system, human gait, has also been studied in recent years. Largest Lyapunov exponent[14] was used to detect the dynamics of human gait. Multifractal and cross correlation[15] also became the tools for quantifying the dynamics of gait and its alteration resulting from neurodegenerative diseases. Another method used to compare the difference in complexity of gait between different walking speeds was an approach based on entropy, named multiscale entropy (MSE).[16] However, apart from the complication of the algorithms themselves, these methods normally require a large dataset with several thousands of points or even more, and a strict preprocess of filtering in order to get a better result, all of which affect the calculation of speed heavily, and thus such methods have difficulty in practical application. The normal human gait time series is extremely inhomogeneous and nonstationary, which fluctuates around the mean value in an irregular and complex manner.[17] The presence of scaling properties suggests that the fractal analysis may provide a method to recognize aging and diseased states by studying changes in the scaling properties of physiological signals. Detrended fluctuation analysis (DFA), a scaling technique, which was introduced by Hausdorff *et al* to study the dynamics of human gait under different walking rates, is proved to be effective.[17] It had been observed that scaling was degraded in certain diseased states such as Parkinson and Huntington in their later work.[18−21]

Another fractal scaling technique, rescaled range (R/S) analysis featuring Hurst exponent, was initially

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proposed by Hurst H E,[22] an English hydraulician, who worked on the optimal reservoir based on record of overflows of the Nile River Dam, and later this technique was applied to analyse time series in many fields[23] including biomedicine.[24] Similar to DFA, which not only can help deal with the ubiquitous biologic 'complication' of nonstationarity[25,26] but also has no requirement of a long dataset, a modified R/S method was introduced in the paper, and applied in detecting and quantifying the alteration in fractal dynamics of human gait with aging and neurodegenerative diseases.

2. Improved rescaled range (R/S) analysis

Given a time series of N points as follows,

$$\{R(i) : 1 \leq i \leq N\},$$

we equally partition it into A consecutive sections with the box size n. Each box is labelled as I_a, $a = 1, \cdots, A$, and each element within each box is denoting by $R_{k,a}$, $k = 1, \cdots, n$. For a given n, we calculate the running means E_a, using

$$E_a = \frac{1}{n} \sum_{k=1}^{n} R_{k,a}, \tag{1}$$

then get the accumulated deviation from the mean $X_{k,a}$, according to the equation below,

$$X_{k,a} = \sum_{i=1}^{k} (R_{i,a} - E_a). \tag{2}$$

The range is defined as a distance between the minimum and the maximum value of $X_{k,a}$,

$$R_{I_a} = \max_k (X_{k,a}) - \min_k (X_{k,a}). \tag{3}$$

The rescaled range (R/S) is obtained by dividing R_{I_a} with the standard deviation S_{I_a},

$$S_{I_a} = \sqrt{\frac{1}{n} \sum_{k=1}^{n} (R_{k,a} - E_a)^2}. \tag{4}$$

Finally, the averaged rescaled range (R/S) is obtained for a given n, using

$$(R/S)_n = \frac{1}{A} \sum_{a=1}^{A} \left(\frac{R_{I_a}}{S_{I_a}} \right). \tag{5}$$

Repeating the calculating process described above, we can get a function relation between averaged R/S and the box size n. Averaged R/S is expected to show a power-law dependence on the box size n:

$$(R/S)_n \propto n^H, \tag{6}$$

Where H is the Hurst exponent. For convenience when plotting, we logarithmize the above function, namely,

$$\log_{10} (R/S)_n \propto \log_{10} n. \tag{7}$$

A resulting slope, or Hurst exponent H is obtained from the double-log graph by a least square fit. The relationship between the fractal dimension D and H is,

$$D = 2 - H. \tag{8}$$

The time series can be divided into three distinct categories according to different values of H: $H < 0.5$, $H = 0.5$, $H > 0.5$. The case $H = 0.5$ corresponds to random or uncorrelated data. If $H > 0.5$, the data are persistent and characterized by long-term correlations or 'memory' effects on all time scales, that is, the data element at any moment would depend (at least in a statistical sense) on the element at relatively remote times. The strength of the persistence increases as H approaches 1.0. The time series with $H < 0.5$ is antipersistent, which means that the time series data are negatively correlated. Our modification relative to the standard Hurst R/S analysis consists of using only one box whose width increases from the lower bound to the upper bound, so that in this way the ordering in the initial series is preserved. Another alteration what we get is the averaged R/S for a given box size n in order to get a smooth result.

3. Analysis for human gait using improved R/S analysis

The stride interval is a measure of the gait rhythm and is typically defined as the time from heel strike (initial contact) to next heel strike of the same foot. Our previous studies using improved R/S analysis demonstrated that human gait from healthy people under free walking situation, whatever walking rate is usual, fast or slow, all exhibited long-range correlations, namely, the stride interval at any instant would depend on the interval at relatively remote times, and this dependence would decay in a scale-invariant (fractal-like), power-law fashion, whereas the

long-term correlations of the stride interval series during metronomically-paced walking will degrade or disappear. Thus, one might speculate further that supraspinal neural control (e.g., the cerebral functional region of movement control) is critical in generating and maintaining these long-range correlations. Once locomotion system is influenced or damaged, the long-range fractal dynamics of human gait will degrade. In order to further investigate the factors affecting our locomotion system and alteration in fractal dynamics of human gait resulting from those factors, we studied two databases of human gait from website www.physionet.org. which has to use is introduced in Ref.[27].

3.1. Influence of aging and Parkinson's disease on the fractal dynamics of human gait

Walking stride interval time series included in the first database are from 15 subjects: 5 healthy young adults (23–29 years old), 5 healthy old adults (71–77 years old), and 5 older adults (60–77 years old) with Parkinson's disease. Subjects were asked to walk continuously on level ground around an obstacle-free path. The stride interval was measured by using ultra-thin, force sensitive resistors placed inside the shoes. The analogue force signal was sampled at 300 Hz with a 12 bit A/D converter, using an ambulatory, ankle-worn microcomputer that also recorded the data.

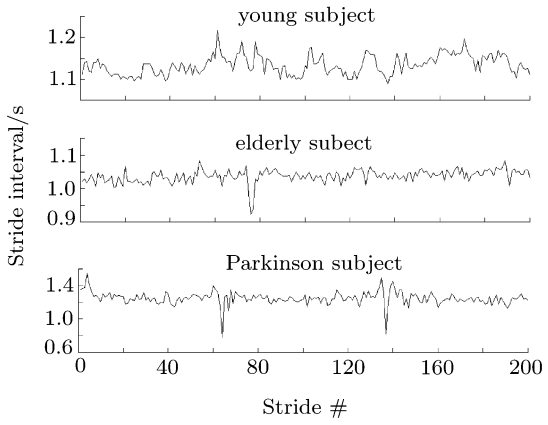


Fig.1. An episode of stride interval series from one healthy young subject, one healthy elderly subject, and one Parkinson subject, respectively.

Subsequently, the time between foot-strikes was automatically computed. An episode of stride interval series from one healthy young subject, one healthy elderly subject, and one Parkinson subject respectively is shown in Fig.1.

Improved R/S method was applied to analyse all the stride interval time series obtained from three groups: healthy young adults, healthy old adults and older adults with Parkinson's disease with no preprocessing of filtering. The length of data record for each calculation was chosen as $N = 200$, and box size n increased from the lower bound 4 to the upper bound $N/5$. The resulting Hurst exponents of three groups were listed in Table 1, and all the parameters here were expressed as mean (SD). As illustrated in Table 1, the Hurst exponent of gait from the healthy young adults is obviously greater than that from the old healthy adults and older adults with Parkinson's disease (t test, $P < 0.05$). Although the difference of Hurst exponents between the older adults with Parkinson's disease and the healthy old adults is not statistically significant (t test, $P < 0.20$), there still seems to be a trend that the Hurst exponent of the former is smaller than that of the latter. A representative example of the Hurst exponent calculated from their corresponding stride interval fluctuations for one healthy young adult, one healthy old adult and one older adult with Parkinson's disease respectively is shown in Fig.2.

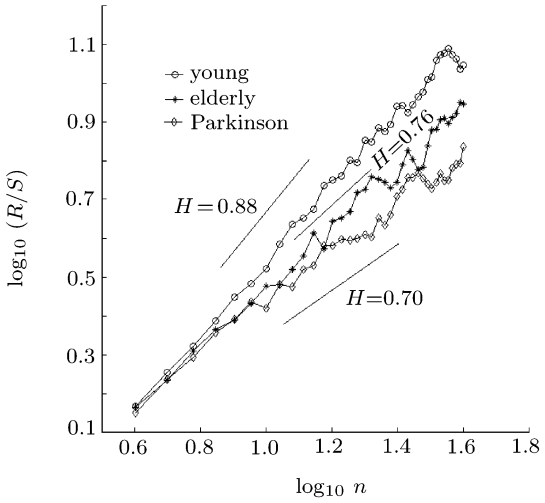


Fig.2. A representative example of the Hurst exponent calculated from their corresponding stride interval series for one healthy young adult, one healthy old adult and one older adult with Parkinson's disease, respectively.

Table 1. The resulting Hurst exponents of human gait from three groups.

group	age/year	stride interval times/s	Hurst exponent
healthy young subjects	24.4(3.1)	1.10(0.06)	0.87(0.04)
healthy elderly subjects	74.6(2.3)	1.04(0.09)	0.77(0.08)
Parkinson subjects	–	1.16(0.16)	0.72(0.07)

3.2. Influence of Huntington' disease on the fractal dynamics of human gait

Neurodegenerative disease often affects gait and mobility. To better understand the pathophysiology of these diseases and to improve the ability to measure responses to therapeutic interventions, it may be helpful to quantify gait dynamics accurately. The records in the neurodegenerative disease database are from patients with Parkinson's disease ($n = 15$), Huntington's disease ($n = 20$), or amyotrophic lateral sclerosis ($n = 13$). Records from 16 healthy control subjects are also included here as the comparison group. The raw data were obtained by using force-sensitive resistors, with the output roughly proportional to the force under the foot. Stride-to-stride measures of footfall contact times were derived from these signals. In order to satisfy the requirement for the data length ($N \geq 200$), we just selected the records from 16 patients with Huntington's disease and 14 healthy control subjects. Moreover, the stride time series of both right foot and left foot extracting from gait record were analysed by our improved R/S algorithm. An episode of stride interval series from one healthy control subject and one Huntington subject is shown in Fig.3.

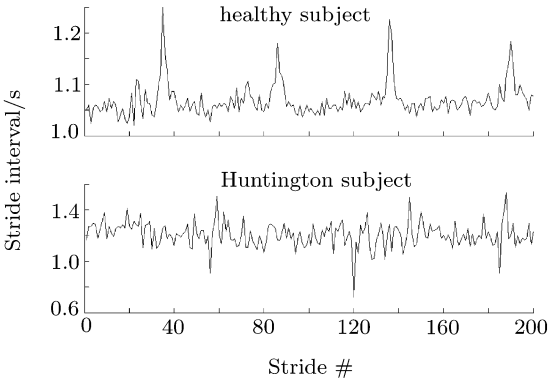


Fig.3. An episode of stride interval series from one healthy control subject and one Huntington subject.

Improved R/S method was then applied to analyse all the stride interval time series from two groups: patients with Huntington's disease and healthy control subjects with no preprocessing of filtering. The length of data record for each calculation was chosen as $N = 200$, and box size n increased from the lower bound 4 to the upper bound $N/5$. The resulting Hurst exponents of two groups were listed in Table 2, and all the parameters here were expressed as mean (SD). As illustrated in Table 2, the Hurst exponents of both the right and left stride interval series from the healthy subjects are obviously greater than those from subjects with Huntington's disease (t test, $P < 0.001$). A representative example of the Hurst exponent calculated from their corresponding stride interval fluctuations for one healthy control subject and one patient with Parkinson's disease respectively is shown in Fig.4.

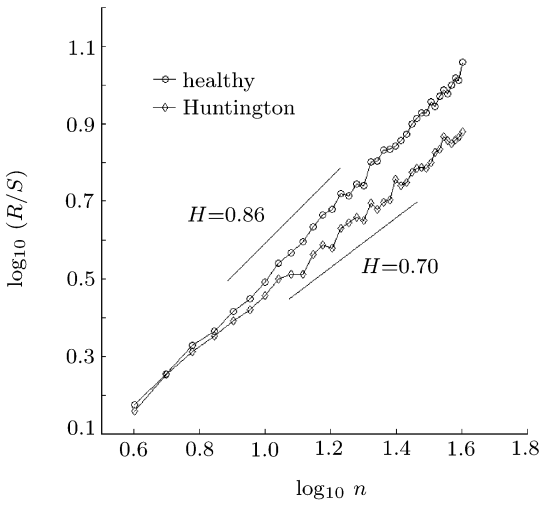


Fig.4. A representative example of the Hurst exponent calculated from their corresponding stride interval series for one healthy control subject and one patient with Huntington disease.

Table 2. Hurst exponents of human gait from healthy subjects and patients with Huntington's disease.

group	age/year	stride interval times/s	Hurst exponent	
			left foot	right foot
healthy subjects	40.1(19.0)	1.35(0.16)	0.83(0.04)	0.84(0.04)
Huntington subjects	46.1(12.1)	1.22(0.31)	0.71(0.10)	0.72(0.10)

4. Results and discussion

The decrease in Hurst exponent of human gait with aging and neurodegenerative diseases such as Parkinson and Huntington was observed by using an improved rescaled range (R/S) analysis method in this paper. It indicates that the long-range correlations of human gait from healthy young people are stronger than those from the elderly and the diseased. The result further implies that fractal dynamics of human gait will be altered due to weakening or impairment of neural control on locomotion resulting from aging and neurodegenerative diseases.

Due to analysing short-term data sequences other than long datasets required by most nonlinear methods, the R/S algorithm has the characteristics of simplicity and sensitivity, most importantly, fast calculation as well as powerful anti-noise characteristic. It is proved to be effective in differentiating short-term nonstationary human gait by Hurst exponent. These findings have implications for modelling locomotor control and for quantifying gait dynamics in physiologic and pathologic states.

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