Altered fractal dynamics of gait: reduced stride-interval correlations with aging and Huntington's disease

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[1]*Gerontology Division, Beth Israel Hospital, Boston 02115; and Division on Aging, Harvard Medical School, Boston 02115;* [2]*Hebrew Rehabilitation Center for the Aged, Boston 02131;* [3]*Biomedical Engineering Department, Boston University, Boston 02215;* [4]*Cardiovascular Division, Beth Israel Hospital/Harvard Medical School, Boston 02215; and* [5]*Neurology Department, Massachusetts General Hospital/Harvard Medical School, Boston, Massachusetts 02114*

Hausdorff, Jeffrey M., Susan L. Mitchell, Renée Firtion, C. K. Peng, Merit E. Cudkowicz, Jeanne Y. Wei, and Ary L. Goldberger. Altered fractal dynamics of gait: reduced stride-interval correlations with aging and Huntington's disease. *J. Appl. Physiol.* 82(1): 262–269, 1997.—Fluctuations in the duration of the gait cycle (the stride interval) display fractal dynamics and long-range correlations in healthy young adults. We hypothesized that these stride-interval correlations would be altered by changes in neurological function associated with aging and certain disease states. To test this hypothesis, we compared the stride-interval time series of *1*) healthy elderly subjects and young controls and of *2*) subjects with Huntington's disease and healthy controls. Using detrended fluctuation analysis, we computed α, a measure of the degree to which one stride interval is correlated with previous and subsequent intervals over different time scales. The scaling exponent α was significantly lower in elderly subjects compared with young subjects (elderly: 0.68 ± 0.14; young: 0.87 ± 0.15; $P < 0.003$). The scaling exponent α was also smaller in the subjects with Huntington's disease compared with disease-free controls (Huntington's disease: 0.60 ± 0.24; controls: 0.88 ± 0.17; $P < 0.005$). Moreover, α was linearly related to degree of functional impairment in subjects with Huntington's disease ($r = 0.78$, $P < 0.0005$). These findings demonstrate that stride-interval fluctuations are more random (i.e., less correlated) in elderly subjects and in subjects with Huntington's disease. Abnormal alterations in the fractal properties of gait dynamics are apparently associated with changes in central nervous system control.

nonlinear dynamics; human locomotion; long-range correlations; power-law scaling

THE DURATION OF THE GAIT CYCLE fluctuates from one stride to the next in a complex fashion (14, 30, 43). Recently, these apparently "noisy" variations have been shown to display a hidden and unexpected fractal property (14, 15). In healthy young subjects, this variability is not simply attributable to random fluctuations (white noise) or solely to short-range influences. Instead, gait cycle duration, i.e., the stride interval, exhibits long-range power-law correlations. Fluctuations in the stride interval are statistically correlated with variations in the stride interval hundreds of strides earlier, and this influence decays in a scale-invariant, fractal manner. This behavior appears to be intrinsic to the healthy locomotor system; it persists regardless of walking speed and disappears in healthy subjects only during metronomically paced walking (14, 15).

From a neurophysiological control viewpoint, this behavior is of interest because it signifies the presence of long-term dependence. The mechanism(s) responsible for these stride-interval correlations are largely unknown. They may be a consequence of peripheral input or lower motorneuron control, or they may be related to higher nervous system centers that control walking rhythm. Although the breakdown of long-range correlations during metronomic walking suggests that supraspinal influences (e.g., a metronome) can override the normally present long-range correlations, their origin and function remain to be determined. To gain insight into the basis for this long-term dependence, we investigated the effects of advanced age and of a neurodegenerative condition, Huntington's disease, on stride-interval correlations.

Aging is associated with a number of neurophysiological changes that may alter the locomotor system's ability to generate stride-interval correlations. These include diminished nerve conduction velocity, deafferentation, loss of motorneurons, decreased reflexes, reduced proprioception, and decreased muscle strength, as well as decreased central processing capabilities (9, 18, 20, 21, 23, 26, 28, 29, 35, 36, 41). The magnitude of these age-related changes depends to a great extent on an individual's comorbid medical conditions. Nevertheless, to some degree, these changes appear to be a part of even "normal" aging. Thus healthy elderly subjects may serve to model subtle changes in neuromuscular control.

Huntington's disease is an autosomal-dominant neurodegenerative disease of the central nervous system. Primary clinical features are chorea and cognitive and personality changes (33). Most of the pathological changes are seen in the basal ganglia, with a loss of neural projection in the striatum (caudate nucleus and putamen) (33). Multiple neurochemical markers (e.g., dynorphin, enkephalin, substance P, and γ-aminoleutynic acid) are depleted in striatum of patients with Huntington's disease (6). However, the mechanisms through which these changes affect the ability of the basal ganglia to regulate motor control are still being elucidated (1, 6). The net result of these changes is that patients with Huntington's disease often display uncontrolled "dancing" (choreiform) movements and gait ataxia, although these features are not necessarily always linked (24). Huntington's disease generally affects people in their 30s and 40s who are typically

free from concomitant disease and age-related physiological changes. With impairment limited primarily to the central nervous system, Huntington's disease offers a contrast to aging for the study of the conditions necessary for stride-interval correlations.

We hypothesized that the locomotor system's ability to produce stride-interval correlations would be diminished in elderly subjects and in subjects with Huntington's disease. To test this hypothesis, we compared the stride-interval dynamics of subjects with advanced age or Huntington's disease with healthy control subjects.

METHODS

This study took place in two parts at two locations. In *part 1*, the effect of aging was examined, and in *part 2*, the effect of Huntington's disease was investigated.

Elderly population. The Harvard Cooperative Program on Aging maintains a database of elderly subjects in the community and their medical history. From this database, we searched for persons 70 yr or older, free from disease that might directly affect gait, including any neurological, musculoskeletal, cardiovascular, or respiratory disorders; rheumatoid arthritis; or diabetes. Subjects were also excluded if they were taking any of the following drugs that might affect gait: benzodiazepines, neuroleptics, antidepressants, diuretics, or vasodilators. Ten subjects met these criteria and agreed to participate in our study. Subjects had no history of falls and appeared to be in excellent health. A targeted neurological examination was performed by a trained geriatrician (S. L. Mitchell). Testing included assessment of sensory and motor function, vision (Snellen score), cognitive function (10), and depression (37). Twenty-two young healthy adults (aged 24.6 ± 1.9 yr), with no evidence of any gait-related disorder, were recruited from among our colleagues and the community to serve as controls. All subjects provided informed written consent as approved by the Committee on Clinical Investigations of Beth Israel Hospital.

Huntington's disease population. Subjects with Huntington's disease were recruited from the Neurology Outpatient Huntington's Disease Clinic at Massachusetts General Hospital (MGH). Subjects were selected based only on their ability to walk independently for 5 min. Seventeen subjects with Huntington's disease participated in the study. The degree of neurological impairment was assessed by a neurologist using the total functional capacity (TFC) (38) score of the Unified Huntington's Disease Rating Scale (0 = most severe; 13 = healthiest) (17). This clinical measure of function has been shown to correlate with positron emission tomorgraphy (PET) scan indexes of caudate metabolism (44). Medications and comorbidities were reviewed, and signs of depression, confusion, and dementia were evaluated clinically (17). Ten control subjects, free from any known neurological or cardiovascular disease, were recruited from the general community. Their age was 34.5 ± 13.4 yr. All subjects provided informed written consent as approved by the MGH Institutional Review Board.

Stride-interval measurement. In *part 1* of this study, elderly subjects and young controls were instructed to walk at their self-determined, usual rate for 6 min without stopping (unless necessary) on level ground around a large, 160-m-long, roughly circular path. In *part 2* of this study, subjects with Huntington's disease and controls were instructed to walk up and down a 77-m-long, straight hallway at their self-determined rate for 5 min without stopping (unless necessary) on level ground. The protocol differed slightly because of the use of two locations.

To measure the stride interval, ultrathin, force-sensitive switches were placed inside each subject's right shoe (12). The output of these foot switches provides a measure of the force applied to the floor. The signal was sampled at 300 Hz and stored in a lightweight, ankle-worn recorder. Subsequently, the recorded signal was automatically analyzed (12) to determine initial contact time and, hence, the stride interval (the time from initial contact, typically heel strike, to the next initial contact of the same foot) for each gait cycle of the walk.

Quantifying stride-interval correlations. To determine the degree of stride-interval correlations, we removed the first 30 s of each subject's time series (to minimize any start-up effects) and then applied detrended fluctuation analysis (DFA) (14, 32) to each median-filtered stride-interval time series. (The median filter was applied to allow study of the "intrinsic" stride-to-stride dynamics and not those due to turning, for example, at the end of one trip down the hall; the filter simply deletes those points that are far away from the median value). DFA is a modified random-walk analysis that makes use of the fact that a long-range (power-law) correlated time series can be mapped to a self-similar process by simple integration. Methodological details have been provided elsewhere (14, 15, 32). Briefly, each integrated time series is self-similar if the fluctuations at different observation windows [F(n)] scale as a power-law with the window size n (i.e., the number of strides in a window of observation or the time scale). Typically, F(n) will increase with window size n. A linear relationship on a double log graph indicates that $F(n) \approx n^{\alpha}$, where the scaling index α (also called the self-similarity parameter) is determined by calculating the slope of the line relating log F(n) to log n. For a process where the value at one step is completely uncorrelated with any previous values, i.e., white noise, $\alpha = 0.5$. In contrast, long-range, persistent correlations are present if $0.5 < \alpha \leq 1.0$. An $\alpha < 0.5$ signifies antipersistent correlations (a large stride interval is more likely to be followed by a small one and vice versa over different time scales).

In general, α is equivalent to Hurst's exponent (3, 40). The Hurst's exponent is a measure that has been widely used to evaluate the self-similarity and correlation properties of fractional Brownian noise, the time series produced by a fractional (fractal) Gaussian process. Both Hurst's exponent and α are used to evaluate the presence or absence of long-range dependence and its degree in a time series. However, local trends (nonstationarities) are often present in physiological data and may compromise the ability of some methods to measure self-similarity. The DFA method (α) was used here because it was designed to be insensitive to these trends by removing local nonstationarities from the analysis (32). It is important to note that, like other measures of self-similarity, α depends on the sequential ordering of the fluctuations in the time series but not on the overall magnitude of the fluctuations (i.e., the variance of the time series). (Indeed, for all the stride-interval time series in this study, we confirmed that α was unchanged even if the time series was normalized to its SD value.) Theoretically, a time series can display self-similarity and fractal scaling with relatively small overall variance or large variance, and, conversely, a time series can have no correlations while having either small or large overall variance.

Because it is difficult to obtain walking data for an extended period of time in clinical patients, we asked subjects to walk for 5 (or 6) min. This does not allow for testing of true long-range correlations (thousands of strides) but still enables evaluation of stride-interval correlations. To determine the degree and nature of stride-interval correlations, we calculated α over the region $10 \leq n \leq 20$. This region was

chosen as it provides a statistically robust estimate of stride-interval correlations[1] that are most independent of finite size effects (length of data) (31).

DFA has typically been used on relatively long data sets (thousands of points). For long data sets, it has been shown that DFA provides a fairly accurate measure of the true scaling exponent (13, 40). To be able to better assess the present results, we generated artificial fractional Brownian noise time series of known α and applied DFA over the region $10 \leq n \leq 20$. Software obtained from the National Simulation Resource of the University of Washington's Center for Bioengineering[2] was used to generate the time series of known α by using a previously validated method (5, 7). For $0.5 \leq \alpha \leq 1.0$, the mean of 10 simulated realizations was within 6% of the true value for time series of lengths 128, 256, and 512. The accuracy fell off somewhat for $\alpha < 0.5$.

Other measures of gait and balance. For each subject's stride-interval time series, we also calculated the average stride interval and the stride-interval coefficient of variation (100 × SD/mean), a measure of the magnitude of stride-to-stride variability and gait unsteadiness (11). In addition, the mean gait speed of each walking trial was determined by counting the number of laps around the track (of known length) and dividing by the time of the walk. The "up-and-go test" was also performed as a gross measure of functional balance and mobility (34). This test measures how long a subject needs to rise from a chair, walk 3 m, turn around, and return to a seated position. "Normal" elderly subjects require between 7 and 10 s to perform this task (34). Test time has been correlated with the Berg balance scale and the Barthel index of activities of daily living (34).

Statistical analysis. For continuous variables, the Wilcoxon rank sum test was performed to compare each study group and its control group. This nonparametric test makes no assumptions about the underlying distribution of the data being compared. For categorical data, Fisher's exact test was used to test for group differences. Group differences were considered statistically different if $P \leq 0.05$. Linear regression was performed to determine whether group differences in α persisted after adjusting for any potentially confounding clinical variables (e.g., height) that differed significantly between the control and study groups. Correlations between two continuous variables (e.g., α and Huntington's disease impairment) were measured by using the Spearman correlation coefficient (r). Statistical analysis was performed by using SAS software, release 6.04 (Cary, NC). Group results are reported as means ± SD.

RESULTS

Aging and stride-interval correlations. The mean age of the 10 (5 women) elderly subjects was 75.7 ± 3.2 yr, height was 1.64 ± 0.08 m, and weight was 72.0 ± 11.2 kg. There was no clinical evidence of peripheral sensory loss or neurological impairment in any subject. Vision (corrected Snellen score) was within normal range (<20/60) in all but one subject (20/70). There were no indications of depression or cognitive impairment. The age of the young control subjects (11 women) was 24.6 ± 1.9 yr. Height and weight was 1.70 ± 0.08 m and 65.6 ± 11.8 kg, respectively. Height, weight, and gender were not statistically different in the two groups.

Figure 1 shows an example of the stride-interval time series for a young and an elderly subject. Visual inspection suggests a possible subtle difference in the dynamics of the two time series. Fluctuation analysis reveals a marked distinction in how the fluctuations change with time scale for these subjects. The slope of the line relating log F(n) to log n is less steep and closer to 0.5 (uncorrelated noise) for the elderly subject. This indicates that the stride-interval fluctuations are more random and less correlated for the elderly subject than for the young subject. Similar results were obtained for other subjects in these groups as well. The scaling exponent α was 0.68 ± 0.14 for the elderly subjects vs. 0.87 ± 0.15 for the young subjects ($P < 0.003$).

Table 1 summarizes the measures of gait in these two groups. Elderly and young subjects had similar average

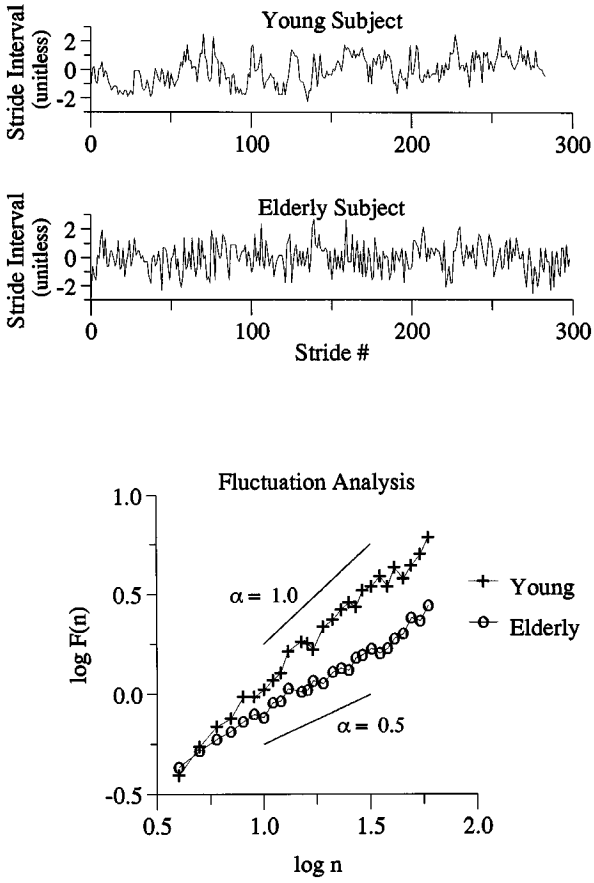

Fig. 1. Example of effects of aging on fluctuation analysis of stride-interval dynamics. Stride interval time series are shown in *top* and fluctuation analysis is shown in *bottom panel* for a 71-yr-old elderly subject and a 23-yr-old young subject. For illustrative purposes, each time series is normalized by subtracting its mean and dividing by its SD. This normalization process highlights any internal "structure" in the time series but does not affect our analysis. Thus, in this figure, stride interval (s) is unitless. For this elderly subject, fluctuation analysis shows that stride-interval fluctuations [F(n)] increase more slowly with time scale n. This indicates a more random and less correlated time series. Indeed, scaling index (α) is 0.56 for this elderly subject and 1.04 for this young subject.

[1] Although one cannot establish long-range correlations and fractal fluctuations of the stride interval with only 5 min of walking data, it is still possible to perform the fluctuation analysis over more limited time scales. This analysis is, in turn, sufficient to indicate changes in fractal-related scaling exponents.

[2] The software was obtained from http://nsr.bioeng.washington.edu/NSR/NSR.html.

stride intervals, and, hence, the time series length was also similar in the two groups (young: 315 ± 22 strides; elderly 316 ± 28 strides). Both groups required comparable amounts of time to perform the up-and-go test. The magnitude of stride-to-stride variability (i.e., stride-interval coefficient of variation) was also very similar in the two groups (and the percentage of points deleted by the median filter was not different in the two groups; young: 2.5 ± 1.0%; elderly: 2.6 ± 1.3%). These results show that, although α was different in the two age groups, the gross measures of gait and mobility function of these elderly subjects were not significantly affected by age. Average gait speed of elderly subjects was similar to that observed in other studies of "healthy" elderly adults (2) and was slightly less than that of the young subjects. Elderly subjects were also slightly shorter (1.64 vs. 1.70 m; $P = 0.06$). However, univariate analysis showed that α was not associated with gait speed ($r = -0.07$; $P > 0.7$) or height ($r = 0.04$; $P > 0.8$). Multivariate analysis techniques were employed to confirm whether speed and height were confounding the association between age and α. After adjustment for these potential confounders (speed and height), age still remained independently associated with α ($P < 0.0005$).

Huntington's disease and stride-interval correlations. The age of the 17 subjects (11 women) was 46.3 ± 12.8 yr, height was 1.85 ± 0.09 m, and weight was 75.6 ± 15.8 kg. The degree of neurological impairment as assessed by using the TFC score was 7.4 ± 3.6 (range: 2–12). Signs of depression, confusion, and dementia were present in six, four, and four subjects, respectively. Antidepressants and neuroleptics were used by seven and three subjects, respectively. Subjects were free of other neurological disease or comorbidities and were not taking other medications that might affect gait. The mean age of 10 control subjects (9 women) was 34.5 ± 13.4 yr, height was 1.83 ± 0.09 m, and weight was 69.8 ± 11.4 kg. On average, subjects with Huntington's disease were slightly older than control subjects ($P < 0.04$) but were not statistically different in terms of gender, height, or weight.

Figure 2 shows the stride-interval time series and the fluctuation analysis of a subject with Huntington's disease and a control subject. The stride-interval fluctuations of the subjects with and without Huntington's disease have a visual appearance similar to that of the stride-interval data of the elderly and young subject (Fig. 1), respectively. For the subject with Huntington's disease, stride-interval fluctuations F(n) increase slowly with time scale n (α = 0.40). This indicates increased randomness and reduced stride-interval correlations as compared with the control subject (α = 0.92). Similar results were obtained for other subjects in these groups as well.[3] The scaling component α was 0.60 ± 0.24 for the subjects with Huntington's disease and 0.88 ± 0.17 in the control subjects ($P < 0.005$).

Among the subjects with Huntington's disease, α was inversely correlated with disease severity as assessed by the TFC score ($r = 0.78$, $P < 0.0005$) (see Fig. 3). The scaling component α was significantly lower ($P < 0.005$), indicative of more random stride-interval fluctuations, in subjects with the most advanced stages of Huntington's disease (TFC ≤ 5: α = 0.44 ± 0.18), as compared

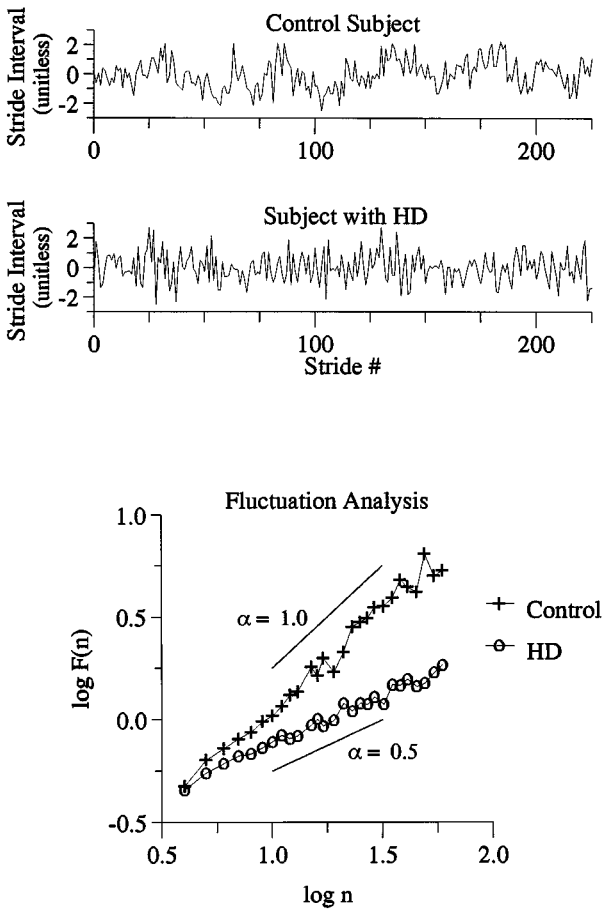

Fig. 2. Example of effects of Huntington's disease (HD) on fluctuation analysis of stride-interval dynamics. Normalized (see Fig. 1 legend for explanation) stride-interval time series are shown in *top panel* and fluctuation analysis in *bottom panel*. For subject with Huntington's disease (41 yr old), stride-interval fluctuations F(n) increase more slowly with time scale n. This indicates a more-random and less-correlated time series. Indeed, α is 0.40 for this subject with Huntington's disease and 0.92 for this 23-yr-old control subject.

Table 1. *Effect of aging on gait measures*

	Elderly	Young Controls
Stride-interval correlations, α	0.68 ± 0.14	0.87 ± 0.15†
Mean stride interval, s	1.05 ± 0.10	1.05 ± 0.07
Stride-interval CV, %	2.0 ± 0.7	1.9 ± 0.4
Gait speed, m/s	1.24 ± 0.18	1.42 ± 0.21*
Up-and-go time, s	7.6 ± 1.0	7.4 ± 1.3

Values are means ± SD. CV, coefficient of variation. *$P < 0.05$; †$P < 0.003$.

[3] Because our analysis focused on the scaling region $10 \leq n \leq 20$, where n is the number of strides in the given window of observation, we would not expect to see much difference if steps were analyzed rather than strides. (Local compensations from one step or stride to the next are outside the region of our analysis.) In some subjects, we had data from the left and right foot and we were able to confirm this.

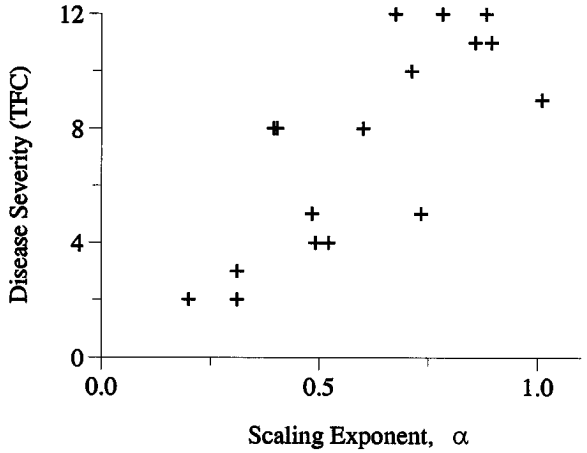

Fig. 3. Relationship between disease severity and degree of stride-interval correlations (α) among subjects with Huntington's disease. Disease severity is measured by using total functional capacity (TFC) score of Unified Huntington's Disease Rating Scale (0 = most impairment; 13 = no impairment).

with subjects in the early stages of the disease (TFC ≥ 9: $\alpha = 0.83 \pm 0.12$). In a few subjects with the most severe impairment, α was <0.5, suggesting the presence of anticorrelations.

With regard to other clinical features of the disease (measured by using the Unified Huntington's Disease Rating Scale), α was not associated with the amount of chorea (of any body segment or total), whole body bradykinesia, dystonia, degree of abnormalities of eye movements, the speed of alternating arm and hand movement, or gait and balance scores. However, α was significantly associated with dysarthria score ($r = -0.66$, $P < 0.001$). When subjects were stratified based on medication use and cognitive and behavioral functions, there were no differences in α, except that α was slightly lower in the four subjects with signs of dementia ($P < 0.04$). (The average of α of the subjects without signs of dementia was still significantly less than that of control subjects.) Nevertheless, several subjects without signs of dementia also had small α values (near 0.4). Finally, among the subjects with Huntington's disease, α was not significantly associated with age ($P > 0.15$).

Table 2 summarizes other measures of gait in these two groups. Mean stride interval was not statistically different in subjects with Huntington's disease and in controls. Subjects with Huntington's disease walked more slowly, took more time to complete the up-and-go test, and had increased stride-to-stride variability. Consistent with these results, the number of strides taken during the walk was slightly larger in control subjects (subjects with Huntington's disease: 234 ± 32 strides; controls: 252 ± 12 strides; $P > 0.05$). The percentage of points deleted by the median filter was similar in the two groups (subjects with Huntington's disease: 4.8 ± 1.4%; controls: 5.6 ± 1.3%; $P > 0.05$). The scaling exponent α was not significantly correlated with gait speed, up-and-go time, or gender, whereas it was correlated with age ($r = -0.39$; $P < 0.05$) and stride-interval variability ($r = -0.62$; $P < 0.001$). Multivariate analysis showed that even after adjustment for these potential confounders α remained independently associated with the presence of Huntington's disease ($P < 0.005$). In contrast, age and stride-interval variability were not independently associated with α.

Table 2. *Huntington's disease effect on gait measures*

	Huntington's Disease Subjects	Controls
Stride-interval correlations, α	0.60 ± 0.24	0.88 ± 0.17†
Mean stride interval, s	1.15 ± 0.17	1.06 ± 0.04
Stride interval CV, %	7.4 ± 5.9	2.0 ± 0.4‡
Gait speed, m/s	1.17 ± 0.36	1.43 ± 0.10*
Up-and-go time, s	11.3 ± 3.5	7.5 ± 1.0‡

Values are means ± SD. *$P < 0.05$; †$P < 0.005$; ‡$P < 0.0005$.

Median filter effects on scaling exponent α. Deleting points from a time series of fractional Brownian noise may alter the correlation properties of the time series. To test the extent to which the deletion of turning points affected the estimate of α in stride-interval time series, we deleted corresponding points on simulated data of known α and measured the effects of the deletions. Specifically, for known α ranging from 0.50 to 0.95, we generated *x* simulated data time series (each with a different seed number) using the Davies and Harte algorithm (7), where *x* is the number of subjects in a given group (e.g., 10 elderly subjects or 17 subjects with Huntington's disease). From each of these simulated time series, we then deleted the points that corresponded (in time) to the deleted points in each of the stride-interval time series. The length of the original (and "filtered") simulated time series was also matched to the corresponding experimental data set. After deleting these points, we then calculated α. The average α was within 4% of the true value, no matter which group's data provided the template for deletions (for the young and old groups who walked for an extra minute and had less deletions, the difference was <2%).

It is, therefore, unlikely that application of the median filter significantly altered the estimation of the "intrinsic" self-similarity in each group's walking pattern. In fact, it is interesting to note that after application of the median filter both groups of control subjects had almost identical gait measures, despite minor differences in the walking protocol (6 vs. 5 min; circular path vs. back-and-forth hallway) and the 10-yr difference in the average age of the two groups (no subjects participated in both studies). The median filter applied to each subject's time series was apparently effective in minimizing any effects due to the walking protocol (turning around). Before the filter, α was 0.83 ± 0.15 and 0.95 ± 0.14 in the aging and Huntington's disease control subjects, respectively. After the filter, the group values were essentially identical to each other, even if

we calculated an average α in two different ways.[4] This indicates the robustness of estimates of mean α and may also suggest that the median filter was effective in preserving the underlying correlation properties while removing extraneous data points associated with changes in gait direction.

DISCUSSION

This study demonstrates that a seemingly intrinsic mathematical property of one output of the healthy locomotor system changes both with aging and with Huntington's disease. In healthy control subjects, stride-interval fluctuations were consistent with our previous findings of fractal scaling (14, 15). In contrast, in the elderly and in subjects with Huntington's disease, fluctuations tended to be more random, and the correlations of one stride with nearby strides were reduced. Moreover, the degree of stride-interval correlations was inversely associated with the degree of functional impairment in subjects with Huntington's disease. In fact, in the most impaired subjects with Huntington's disease, the stride-to-stride fluctuations became either completely uncorrelated, like white noise, or anticorrelated.

The finding of reduced stride-interval correlations with aging and with Huntington's disease parallels other findings of changes in fractal scaling and long-range correlations with age and disease (25). For example, alterations in the fractal properties have been observed in the fluctuations in breathing during hyperoxia and hypoxia (16) and in the variations of the electroencephalographic-evoked potentials with aging and disease (4, 27, 39). One of the most widely studied examples of fractal physiology is the beat-to-beat fluctuations in cardiac dynamics. The correlations properties of heart rate are diminished in the elderly (19) and in certain subjects with cardiovascular disease (e.g., heart failure) (22, 32). Intervention studies have demonstrated that vagal blockade alters this fractal scaling of the heart beat and its dependence on intact autonomic neural function (42).

The alterations in the fractal dynamics of the stride interval are not simply attributable to reduced gait speed or increased stride-to-stride variability (unsteadiness) with aging or disease. Previous findings (15) showed the presence of stride-interval correlations (α close to 1.0) even when healthy subjects walked slowly (i.e., 1.0 ± 0.2 m/s). In the present study, stride-to-stride variability was virtually unchanged in the elderly subjects compared with young controls, yet the stride-interval correlations were significantly reduced. Furthermore, the magnitude of the stride-interval correlations was independent of gait speed and stride-to-stride variability in both studies. Apparently, stride-interval correlations depend on some aspect of the neuromuscular control system that is not directly related to walking velocity or gait unsteadiness.

Given the reduced stride-interval correlations in subjects with Huntington's disease, especially in the most impaired subjects, it is likely that the areas of the cerebrum that are affected by Huntington's disease play an important role in generating stride-interval correlations. While some pathological changes have been found in the cortex (6), the primary pathology is in the basal ganglia. Perhaps, the striatal pathology that leads to a decrease in fine motor control in Huntington's disease also impairs the "long-term dependence" and fine control necessary for stride-interval correlations.

Other factors that may have contributed to the differences between the stride-interval correlations of control and Huntington's disease subjects include age (control subjects were slightly younger), medication use, and cognitive and personality changes. Multivariate analysis suggests that the small age effect is not significant. This is supported by *1*) findings of identical degrees of stride-interval correlations in the two (young adults vs. middle-aged subjects) control groups, despite an almost 10-yr difference in mean age and by *2*) the fact that older control subjects showed no reduction in α (e.g., α was 1.11 and 0.89 in a 57- and 52-yr-old subjects, respectively), whereas some young Huntington's disease subjects showed large reductions in α (e.g., α was 0.31, 0.40, and 0.40 in Huntington's disease subjects who were 34, 41, and 42 yr old, respectively). Interestingly, α was not significantly altered in the subjects who showed signs of depression or confusion or those who were using neuroleptics. The independence of α and neuroleptic use is consistent with findings of Koller and Trimble (24). They found that haloperidol therapy decreased chorea but did not change gait impairment of subjects with Huntington's disease. The scaling exponent α tended to be lower in patients with signs of dementia. However, compared with control subjects, α was still significantly lower in those subjects with no signs of dementia, and several subjects without signs of dementia also had small α values. Subjects with Huntington's disease were free of other comorbidities and peripheral disease that would be likely to affect gait. Thus the decrease in stride-interval correlations with Huntington's disease is probably not simply due to secondary factors associated with this condition but is most likely related to the underlying central neuropathology.

In our group of healthy elderly subjects, the reduction in stride-interval correlations is less than that seen in the subjects with Huntington's disease. The reduced correlations in the elderly gait may be due in part to comorbidities that we did not detect. Alternatively, the alterations in gait dynamics in the elderly may be due to subtle changes in neural control that were not revealed by our clinical evaluation. These elderly subjects were free from overt neurological disease. However, even in "normal" elderly adults, there is an age-related decline in dopamine content in the basal ganglia, and it has been suggested that age-related changes in gait may result from subtle changes in

[4] In the first, we simply calculated α for each individual subject and took the average. In the second, we normalized each subject's time series to its SD (after subtracting the mean) so that individual records could be analyzed as if they originated from the same underlying dynamic system. We then averaged $F(n)$ for each box size across all subjects in the group and extracted α.

striatal dopamine mechanisms (8). The reduction in stride-interval correlations may also be a manifestation of these changes in central processing. However, although the primary pathology of Huntington's disease is also in the basal ganglia, it is unlikely that the same mechanisms are affecting the elderly and subjects with Huntington's disease. Huntington's disease is primarily hyperkinetic, whereas these putative age-related changes are hypokinetic. Furthermore, the affected regions within the basal ganglia are also different. Nonetheless, it is interesting to speculate why measures of stride-to-stride variability and gross measures of mobility (up-and-go time) are unchanged in these elderly subjects, whereas stride-interval correlations are diminished. Perhaps, the ability to produce a correlated gait is a more demanding locomotor challenge. The diversity of the age-associated neuromuscular changes (e.g., pyramidal, extrapyramidal, and peripheral) and the absence of overt neurological disease in our subjects make it difficult to precisely determine the cause(s) of the changes in stride-interval correlations in these elderly subjects.

Precise elucidation of the factors affecting the fractal dynamics of gait remain to be determined. Stride-interval correlations are decreased with advanced age and with Huntington's disease and are probably dependent on intact central nervous system (basal ganglia) processing but are independent of walking speed and variability. Future studies that examine subjects with pathologies involving different portions of the extrapyramidal and pyramidal systems may provide additional insight into this unexpected property of normal walking. Finally, we note that the analysis of stride-interval dynamics may have practical utility in neurological diagnosis and in the quantitative assessment of therapeutic interventions.

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