Gait instability and fractal dynamics of older adults with a “cautious” gait: why do certain older adults walk fearfully?

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Abstract

Many older adults walk with a cautious and impaired gait of unknown origin, however, the relationship between fear of falling and the observed gait changes is not well understood. To better understand the “cautious” gait of the elderly, we tested the hypothesis that temporal gait variability, putatively a marker of intrinsic walking unsteadiness, is increased among older adults with a cautious gait and a higher-level gait disorder (HLGD), an altered gait that cannot be attributed to a well-defined cause. Twenty-five older adults (mean age: 78 years) with a HLGD were compared to healthy controls of similar age and sex (n = 28). The clinical characteristics (e.g., neurological status, fear of falling), the magnitude of the stride-to-stride variations in gait cycle timing (a measure of temporal gait variability), and a fractal index of gait (a measure of the stride dynamics independent of the magnitude of the variability) were studied in both groups. Gait variability was significantly increased (P < 0.0001) in HLGD subjects (52 ± 26 ms) compared to controls (27 ± 9 ms). Changes in frontal lobe and extra-pyramidal function were also found in the patient group. Among HLGD subjects, gait variability was not associated (P > 0.05) with age, gender, MMSE score, muscle strength, # of co-morbidities, balance, cerebellar signs, or pyramidal signs, but was significantly associated with scores on the Geriatric Depression Scale (r = 0.46, P < 0.02) and fear of falling (r = 0.69, P < 0.0001). Among HLGD subjects, only a fractal index was significantly different in fallers and non-fallers. These findings underscore the idea that the gait changes in older adults who walk with fear may be an appropriate response to unsteadiness, are likely a marker of underlying pathology, and are not simply a physiological or psychological consequence of normal aging.

Keywords: Gait; Aged; Falls; Fear of falling; Gait variability; Higher-level gait disorders

1. Introduction

Many older adults walk without any significant mobility impairment [1–3]. Among those older adults who do have a gait disturbance, the cause is often easily identifiable (e.g., Parkinson’s disease) [1–5]. There are, however, many older adults with an impaired gait that does not appear to be a result of any well defined disease [6]. In his review of patients attending a neurology clinic, Sudarsky found that the cause of the gait disturbance was “unknown”, even after neuro-imaging, in about 10–20% of older adults with a disturbed gait [3,7]. In a study of the “oldest old” (age range 87–97 years) in The Netherlands, Bloem et al. observed that about 20% of those studied had a normal gait, 69% had a gait disorder due to known disease, and about 11% of the subjects had an idiopathic “senile gait disorder”, i.e., a gait disorder of unknown origin [8]. Interestingly, those subjects with a gait disorder of unknown origin had a higher risk of mortality during a 5 year follow up period, compared to the group of age-matched subjects who had a normal gait [8]. Nutt et al. coined the term “higher-level” gait disorders (HLGD) to refer to an altered gait that is not a result of lower extremity or peripheral dysfunction and can not be attributed to well defined chronic disease [5,9]. One common example of a higher-level gait disorder is the idiopathic “cautious” gait of the elderly or the “senile gait” disorder [5,9]. A cautious gait is typically marked by mild to moderate slowing, reduced stride length, and mild widening of the base of support [5]. Fear of falling, common among many older adults [5,10–13], presumably plays an important role.
in this cautious gait [5,10], but the origin of the timid, reserved gait that is characteristic of so many elderly is not well understood. It is unclear if the changes in gait are related to a history of falls or a fear of future falls [12], whether these changes predispose to falls, or whether the appearance of a fearful gait is just coincidence (or secondary), i.e., the changes in gait are primary in origin and not just a result of fear. While a slow, guarded gait may be the response to a fear of falling, it does not, by itself, explain the exaggerated fear of falling seen in many older adults. Could other gait changes contribute to and explain the fear of falling?

In a study of a relatively heterogeneous group of older adults living in assisted living facilities, Maki observed an intriguing dichotomy [14]. Fear of falling was related to gait speed, while falls were related to gait variability (those who fell during a 1 year follow-up period had increased gait variability, regardless of gait speed). Maki explained that subjects with a fear of falling walked more slowly, but fall risk was independent of gait speed and was modulated instead by gait variability. Subjects unable to regulate the stride-to-stride fluctuations in walking were more unstable and unsteady and more likely to fall. Similar results, at least with respect to the relationships between gait speed, gait variability and falls, were obtained in a prospective study of community living older adults [15]. However, to date, gait variability in older adults with HLGD and a cautious gait has not been studied and the relationship between fear of falling and unsteadiness in this population is not known. Is it possible that older adults with a cautious gait walk fearfully because they have an unsteady gait? In contrast to the general elderly population, where fear of falling appears to be related to fall history [11,12], in this population, fear of falling may occur even in the absence of any fall history [10]. Perhaps increased unsteadiness could, in part, explain this fear of falling.

In the present study, we tested the hypothesis that gait variability, a marker of gait unsteadiness that is putatively unrelated to fear of falling [14], is increased among older adults with a HLGD and a cautious gait. Secondary aims of this study were: (1) to examine the factors associated with increased gait variability in order to better identify what contributes to fear of falling and the changes in gait dynamics in this population, and (2) to identify the factors that discriminate fallers and non-fallers in this population. As we describe below, our findings suggest that, in contrast to the general elderly population, a more complex relationship exists between fear of falling, gait instability, and falls in older adults with HLGD who walk fearfully.

2. Methods

2.1. Subjects

Twenty-five older adults who came to the Movement Disorders Unit at the Tel Aviv Sourasky Medical Center for evaluation of walking difficulties of unknown origin were studied. All patients were mobile and walked independently at the time of assessment and all underwent a thorough general and neurological examination.

Patients were excluded if the cause of their gait disturbance could be readily established. Thus, patients with a history of clinically established stroke, Parkinson’s disease, Alzheimer’s disease, possible normal pressure hydrocephalus or other diagnosed neurodegenerative disorder, and patients with rest tremor or pronounced bradykinesia were excluded. Patients who were taking anti-parkinsonian or anti-spastic medications, or had orthostatic hypotension were also excluded. We also excluded patients with significant visual, peripheral, or vestibular disturbances, as well as patients with significant orthopedic disturbances. Patients with dementia according to the DSM IV criteria [16], history of psychiatric disease, or past use of dopamine receptor blocking agents (anti-psychotic medications) were also excluded. In addition, we excluded patients with a history of traumatic head injury and/or loss of consciousness. Twenty-three patients underwent brain imaging (CT n = 20 and/or MRI n = 11). Patients with multiple brain infarcts or imaging suggestive of normal pressure hydrocephalus (NPH) were excluded.

No specific disorder could be diagnosed as a cause of the patients’ complaint about his or her walking difficulties prior to this study. In other words, all patients had self-reported walking difficulties that could not be attributed to any specific disease or medical condition. The objective was to study older adults who fulfilled the diagnosis of HLGD, i.e., walking difficulty not attributable to a chronic condition or disability, not necessarily patients with complaints about gait unsteadiness.

The patient population was compared to a group of 28 “healthy” controls of similar age. Control subjects were recruited from the community (n = 24) and from a nearby elderly housing facility. Subjects were included if they reported normal walking function, had no obvious clinical impairment, and did not have significant cognitive impairment (Mini Mental State Examination (MMSE) > 24). Subjects were excluded if they had any neurological disorder or any significant clinical history likely to affect their gait (e.g., stroke).

The study was approved by the Human Studies committee of the Tel-Aviv Sourasky Medical Center. All subjects provided informed written consent according to the declaration of Helsinki prior to entering the study.

2.2. Clinical evaluation and assessments

The evaluation included a detailed focused history and a complete, structured neurological exam including the motor part (part III) of the unified Parkinson’s disease rating scale (UPDRS) [17]. In addition, the Mini Mental State Examination (MMSE) [18] and the Geriatric Depression Scale (GDS) [19] were administered to assess mental state. Charl-
son co-morbidity index was used to quantify disease burden and general health status [20]. The Activities-Specific Balance Confidence scale (ABC) [21] was used to assess the level of fear of falling. A higher score reflects more confidence and less fear. Muscle strength was assessed at the quadriceps using a handheld digital dynamometer [22].

Average gait speed and stride length were evaluated by measuring the time and number of strides to walk 10 m. The average of three walking trials was used. Functional mobility and balance were assessed using the functional reach test (mean of three trials in cm) [23], the Pull test (item 30 on the UPDRS) and the Timed Up and Go test [24].

To quantitatively evaluate different neurological systems, we developed pyramidal, extra-pyramidal, frontal and cerebellar indices. The pyramidal index was considered positive if the subject had either a positive Babinski sign or clonus (or both), or if the subject had both spasticity and hyper-reflexia. To quantify extra-pyramidal involvement, we used the total score of the motor portion (Part III) of the UPDRS (higher scores worse) and an extra-pyramidal index based on items 22, 23, 24, 25, 26, 30, 31 of the motor portion of the UPDRS (i.e., gait was excluded from this index). The frontal index was considered to be positive if subjects had at least two of the following four signs: Meyerson reflex, snout reflex, palmo-mental reflex or facilitory paratonia [25]. Cerebellar function was assessed using the Romberg test, diadochokinesis, finger to nose and heel to shin tests. It was considered positive if any test was positive.

2.3. Walking protocol

Subjects were instructed to walk on level ground for 2 min at their normal pace in a 25 m long, 2 m wide hallway under usual lighting conditions. Subjects were told to turn around and continue walking when they reached the end of the hallway. All subjects were tested in the same environment. Subjects were “guarded” by a physical therapist who walked a few steps away from the subject, making sure not to interfere or set the pace. Study subjects were not aware of the specific questions of this investigation.

2.4. Assessment of gait dynamics

Previously described methods were used to quantify gait variability and evaluate gait dynamics during the 2 min walk [15,26–27]. Briefly, to measure the gait rhythm and the timing of the gait cycle (i.e., the stride time), force sensitive insoles were placed in the subject’s shoe. Two sensors were used, one under the heel and the other under the forefoot and the toes, and the data from both sensors were combined before the recording. These inserts produce a measure of the force applied to the ground during ambulation. A small, lightweight (5.5 cm × 2 cm × 9 cm; 0.14 kg) recorder was worn on the ankle and held in place using an ankle walle. An on board A/D converter (12 bit) sampled the output of the footswitches at 300 Hz and stored the digitized force record. Subsequently, the digitized data were transferred to a computer workstation for analysis using software that extracts the initial contact time of each stride [28]. From the force signal, the stride time or duration of the gait cycle (time from initial contact of one foot to subsequent contact of the same foot) was determined for each stride during the 2 min walk by applying a previously validated algorithm that locates initial contact times (and hence the stride time) by finding large increases and changes in the slope of the force [28].

To focus on the assessment of the dynamics of continuous, “normal” walking and each subject’s “intrinsic” dynamics and to insure that the analysis was not influenced by atypical strides (e.g., the turning at the end of the hallway), a median filter was applied to each subject’s time series to remove data points that were three standard deviations greater than or less than the median value [15,26]. There was general agreement between the values obtained before and after application of the median filter. Subsequently, stride time variability, the magnitude of the stride-to-stride fluctuations in the gait cycle duration, was calculated by determining the standard deviation (S.D.) and the coefficient of variation (CV) of each subject’s stride time [14,15,27]. In addition, we calculated the standard deviation of the stride time in overlapping, moving windows of 10 and 20 s and determined the lowest value for each subject (for each window size). The lowest standard deviation from among all of the moving windows reflects the “optimal performance”/lowest variability during the entire walking period. Stride-to-stride variability reflects gait unsteadiness and arrhythmicity and has been shown to prospectively predict falls [14,15,27,29].

The above measures quantify the magnitude of the stride-to-stride variability but are not sensitive to changes in the ordering of the stride times or the dynamics. Randomly re-ordering a time series will not affect the magnitude of the variability, but may dramatically alter the dynamic properties. To quantify how the dynamics of the stride time fluctuate over time during the 2 min walk, we applied detrended fluctuation analysis (DFA) to each subject’s sequence of stride times [26,30–32]. DFA was designed for evaluating the fractal scaling exponents and the degree of randomness in highly non-stationary physiological data. DFA eliminates trends in the time-series, and therefore can avoid the spurious detection of correlations that are artifacts of non-stationarities. 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 [26,30–32]. Briefly, each integrated time series is said to be self-similar and fractal-like 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 $F(n) \sim n^\alpha$ (a power-law relationship) where the fractal scaling index $\alpha$
(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 stride is completely uncorrelated with any previous values, i.e., white noise, the fractal index is 0.5. In contrast, long-range, persistent correlations are present if \( \alpha \geq 0.5 \), and \( \leq 1.0 \). Succinctly put, when \( \alpha \geq 0.5 \), lower values (closer to 0.5) indicate more randomness while higher values indicate more temporal structure and fractal-like order. In the present study, we calculated a short-range scaling index (\( n \leq 10 \)) to minimize data length issues [33]. Very generally, healthy physiologic systems have fractal scaling indices around 0.8–1.0 (de-}

Table 1

<table>
<thead>
<tr>
<th></th>
<th>Patients (( n = 25 ))</th>
<th>Controls (( n = 28 ))</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stride time standard deviation (ms)</td>
<td>52 ± 26</td>
<td>27 ± 9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Stride time CV (%)</td>
<td>4.2 ± 1.7</td>
<td>2.3 ± 0.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Lowest stride time standard deviation (in any 10 s period)</td>
<td>63 ± 4.7</td>
<td>46.6 ± 2.4</td>
<td>0.002</td>
</tr>
<tr>
<td>Lowest stride time standard deviation (in any 20 s period)</td>
<td>205 ± 73</td>
<td>104 ± 3.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>DFA Fractal Index</td>
<td>0.75 ± 0.18</td>
<td>0.84 ± 0.22</td>
<td>0.025</td>
</tr>
</tbody>
</table>

CV: coefficient of variation; HLGD: high level gait disorders; DFA: detrended fluctuation analysis.

The clinical characteristics of the patient group are described in detail elsewhere [34]. Briefly, cognitive function was slightly lower (MMSE: patients: 26.6 ± 3.4 controls: 28.8 ± 1.5; \( P < 0.009 \)) and depressive symptoms were slightly increased (GDS: patients: 11.9 ± 4.7; controls: 5.3 ± 3.6; \( P < 0.001 \)) in the patient group. Patients reported greater fear of falling (ABC score: patients: 60.3 ± 22.8, controls: 94.6 ± 6.4; \( P < 0.001 \)). The neurological examination revealed more frequent frontal lobe signs (88% versus 36%; \( P < 0.001 \)) among the patients. Although signs of tremor, bradykinesia, or rigidity were rarely seen in either group, increased extra-pyramidal involvement was observed in the patient group compared with the control group. This was true both in terms of part III of the UPDRS (which includes items on gait and balance: patients: 17.2 ± 7.3; controls: 4.3 ± 2.6; \( P < 0.001 \)) and in terms of the extra-pyramidal index (patients: 12.0 ± 5.5; controls: 3.8 ± 2.1; \( P < 0.001 \)). In both groups, there were no signs of pyramidal or cerebellar impairment. Patients tended to be weaker (quadriceps strength: patients: 147 ± 52 N; controls: 219 ± 58 N; \( P < 0.001 \)), had a shorter functional reach (patients: 21.6 ± 5.0 cm; controls: 33.5 ± 4.6 cm; \( P < 0.001 \)), performed worse on the Timed Up and Go test (patients: 1.6 ± 0.7; controls: 0.4 ± 0.5; \( P < 0.001 \)) and took longer to complete the Timed Up and Go test (patients: 18.1 ± 7.8 s; controls: 8.1 ± 1.3 s; \( P < 0.001 \)). Not surprisingly, gait speed was also reduced in the patient group (patients: 0.74 ± 0.22 m/s; controls: 1.24 ± 0.18 m/s; \( P < 0.001 \)). The lower gait speed in the patient group was due to both changes in cadence (patients: 103 ± 14 steps/s; controls: 114 ± 7 steps/s; \( P < 0.001 \)) and stride length (patients: 0.84 ± 0.20 m; controls: 1.27 ± 0.17 m; \( P < 0.001 \)).

An example of the stride-to-stride fluctuations observed in a patient is shown in Fig. 1. Stride-to-stride variations in gait cycle timing were markedly increased in this subject with a HLGD compared to the control subject. This inability to maintain a stable walking pattern was a consistent feature of the subjects with a HLGD. As summarized in Table 1 and Fig. 2, all measures of gait variability were significantly increased in the subjects with a HLGD compared to control subjects. All measures of the magnitude of the stride-to-stride variability tended to be twice as large in the
subjects with a HLGD compared to control subjects. The fractal scaling index of gait was also different in the two subject groups ($P = 0.025$).

Although the measures of gait variability were consistently higher in the subjects with a HLGD, there was a fairly large range among the subjects (much larger than among the control subjects; note the size of the standard deviations of the parameters shown in Table 1). Fear of falling explained much of this variance. As shown in Fig. 3, stride-to-stride variability was significantly correlated with fear of falling (as measured by the ABC scale); subjects with a more unsteady gait had less self-confidence in their ability to perform different activities without losing balance. The association between fear of falling and gait unsteadiness persisted after adjusting for gait speed in multiple regression analysis, suggesting that the relationship between fear of falling and gait variability was independent of gait speed.

### 3.1. Factors associated with gait variability

As noted, there was a wide range in the measures of gait unsteadiness in the subjects with a HLGD. Among the subjects with HLGD, the stride time CV was not associated ($P > 0.05$) with: age, gender, cognitive function (MMSE), the Charlson co-morbidity index, lower extremity muscle strength or power, or balance (functional reach, Pull test). Only three patients did not exhibit frontal signs. The motor portion of the UPDRS was associated with the stride time CV ($r = 0.42; P < 0.037$), however, when the balance and gait items were removed from the UPDRS score and we examined only the extra-pyramidal index, the association with the stride CV was no longer significant ($P > 0.15$). From among the other clinical characteristics, only

![Fig. 1. Example of the stride time fluctuations in an older adult with a HLGD and a healthy control subject. Note the increased stride-to-stride variability in the duration of the gait cycle (i.e., the stride time) in the patient compared to the control subject.](image1)

![Fig. 2. The standard deviation of the stride time (ms) was markedly increased in the subjects with a HLGD compared to the control subjects ($P < 0.0001$). The error bars reflect the standard error of the mean.](image2)

![Fig. 3. Fear of falling, as measured by the Activities-Specific Balance Confidence (ABC) Scale, was strongly associated with all measures of stride-to-stride variability. Shown is the relationship between the ABC scale and stride variability, specifically, the lowest stride time standard deviation during any 20 s period (recall Table 1). Subjects with greater gait stride-to-stride variability had lower ABC scores and were more fearful (Spearman’s correlation coefficient $r = -0.72; P < 0.0001$). (×) Controls; (○) patients. The linear regression line is shown.](image3)
Among the control subjects, stride time CV was not significantly associated with gait speed. However, among the patients, there was a significant association between gait speed and stride time CV ($r = 0.46; P = 0.022$) were significantly correlated with the stride time CV in the patient group.

The control subjects, stride time CV was not significantly associated with gait speed. However, among the patients, there was a significant association between gait speed and stride time CV ($r = 0.60; P = 0.002$). Patients who walked more slowly tended to have increased stride-to-stride variability. To further investigate the role of gait speed, we studied a subset of subjects from each group who walked with a similar gait speed. In this subset, mean gait speed was $0.99 \pm 0.06$ m/s and $1.06 \pm 0.07$ m/s in the patient ($n = 7$) and control ($n = 8$) groups, respectively ($P = 0.121$). Although there was no significant difference in gait speed, both the stride time standard deviation and the stride time CV were significantly larger in the patient group. For example, stride time CV was $3.2 \pm 0.5$ and $2.4 \pm 0.6$% in the patients and controls, respectively ($P = 0.021$). Consistent with the general finding, among this subset of subjects, fear of falling was significantly increased in the patient group compared to the controls ($P = 0.029$).

### 3.2. Factors associated with falling

Among the subjects with a HLGD, 10 subjects reported no falls in the previous year, while 15 subjects reported falling once or more (12 of these fell at least twice). In contrast, only 3 control subjects reported a fall in the previous year ($P < 0.001$) and none reported multiple falls. Thus, when all subjects (patients and controls) were stratified into fallers and non-fallers, many parameters were different in fallers and non-fallers, as when the patient and controls were compared (e.g., stride S.D. was $44 \pm 22$ ms in the fallers and $36 \pm 23$ ms in non-fallers, $P < 0.05$). In contrast, among the patients, fallers and non-fallers were similar with respect to all clinical characteristics and all measures of gait and balance (e.g., fear of falling, lower extremity strength, frontal lobe signs, functional reach, Pull test, stride CV), except for one aspect of gait dynamics: the fractal scaling index was different (more random) in fallers compared to non-fallers ($P < 0.009$) (see Fig. 4). Similar results were obtained if multiple-fallers (i.e., two or more falls) were compared to non-fallers ($P < 0.013$).

### 4. Discussion

In this first study of the gait dynamics of older adults with a HLGD, we find three key results: (1) gait variability is markedly increased among older adults with a HLGD and fear of falling compared to control subjects of similar age. (2) Physical factors (e.g., muscle strength, balance disturbances) are not associated with the level of gait variability among the older adults with HLGD. Instead, neuro-psychological factors, especially fear of falling, and depression are significantly related to stride-to-stride variability. (3) Among the older adults with a HLGD, fall history was not related to fear of falling, gait speed or other clinical measures. The fractal scaling index was the only measure that was related to fall history.

A description of the cautious gait of the elderly usually includes mention of a reduced gait speed, shorter stride length, and increased base of support [5]. These features were indeed present in the study subjects with a HLGD. Our results suggest that increased stride-to-stride variability is also a characteristic feature of these older adults. Interestingly, while the degree of gait variability in the subjects with a HLGD was not associated with typical measures of motor function (e.g., muscle strength, balance), gait unsteadiness was closely associated with fear of falling.

This finding could be explained by a common pathologic origin. The frontal lobe and extra-pyramidal signs observed in this patient group might be responsible for both the fear of falling and the gait instability. On the other hand, a different cause and effect relationship might be at work here. Dysrhythmicity, an unstable gait and an inability to maintain a steady walking rhythm may lead to or exacerbate a fear of falling and a lack of self-confidence. This possibility could suggest, for the first time, a gait-based reason why certain older adults, who do not have a specific neurodegenerative disease, walk fearfully. Consistent with this possibility, we note that previous studies in older adults with known neurological disease have observed that increased stride-to-stride variability is associated with frontal lobe dysfunction (e.g., Alzheimer’s disease) [29,35,36] and extra-pyramidal disease (e.g., Parkinson’s disease) [37,38].

One could argue, alternatively, that fear of falling (of largely unknown origin) leads to instability and increased gait variability. Although this possibility cannot be completely ruled out, Maki’s results suggest that fear of falling may be found without gait unsteadiness and gait instability.
unsteadiness may be found without fear of falling, at least in certain populations [14]. Previous findings which demonstrate that gait speed and gait unsteadiness may be dissociated [14,27,39,40] are also important in this regard. The increased gait unsteadiness observed in the older adults with a HLGD cannot simply be explained by the fact that they walked more slowly than the control subjects. Healthy older adults have been shown to walk with the same, small amount of stride-to-stride variability as healthy young adults, even though they walk slower than healthy, young adults [26]. In the present study, subset analysis revealed that even among subjects who walked with a similar gait speed, the patients were much more unsteady and more fearful. In the future, it may be helpful to further evaluate the influence of gait speed on stride variability in patients with HLGD, perhaps by asking the control subjects to walk very slowly and by asking the patients to walk as quickly as possible. Nonetheless, the present results suggest that something specific to this disorder apparently is responsible for the striking unsteadiness in the patient group. As noted, frontal lobe and extra-pyramidal dysfunction are the likely putative sources for the impaired mobility.

One could maintain that our finding of an altered gait pattern among these older adult is to be expected. Subjects with walking difficulties were selected and compared to a control group and walking difficulties were found. We note, however, that we did not identify potential study subjects based specifically on their stride-to-stride variability or gait unsteadiness. Older adults were selected if they fulfilled the criteria of HLGD. As noted above, a description of the cautious gait of the elderly typically does not even include gait unsteadiness as a characteristic feature. Visual observation does not allow estimation of fractal scaling indices. Thus, the finding of a consistently increased stride-to-stride variability and altered fractal scaling in the study subjects, compared to the control subjects, is not simply a result of the subject selection process. Moreover, the selection process did not any way guarantee, in advance, that there would be a significant association between fear of falling and stride-to-stride variability. Thus, we suggest that the present findings do indeed provide new insight into the HLGD in older adults.

Among relatively healthy older adults, gait variability distinguishes fallers from non-fallers [14,27,39]. The present findings are generally consistent with this. However, in the present study, gait variability was not related to fall status, among the subjects with a HLGD. In fact, in this patient group, almost all measures were similar in fallers and non-fallers (including fear of falling). Only the fractal scaling index was significantly decreased in fallers. It is important to note that the fractal index is independent of the magnitude of the stride-to-stride variability. Fallers and non-fallers both had increased gait variability (compared to the control subjects), but the change in the fractal index indicates that the walking pattern of the fallers was more random and less organized (on a temporal basis). The precise mechanisms that are responsible for the change in the temporal ordering of the stride pattern and the increased fall risk in the subjects with HLGD remain to be determined. Changes in the fractal pattern of physiologic signals have been shown to be sensitive markers of changes in aging and underlying disease and predictive of morbidity and mortality [26,30,39,41,42], suggesting the possibility that the change in fractal scaling reflects subtle intrinsic neurodegenerative distinctions. In a postmortem study, Whitman et al. observed prominent frontal atrophy in older adults with “disequilibrium of unknown cause” [6]. Our clinical examination also found frontal lobe dysfunction in the patient group, but it did not identify any differences between the fellers and non-fellers other than fall history. Perhaps sensitive functional neuro-imaging techniques may help to identify the source of the predisposition to falls and the reduced fractal index in a subset of older adults with a HLGD.

Why do many older adults walk fearfully? Although a history of falls by itself does not explain the gait changes or the fear of falling in this population, as it apparently does in others [12], the present results indicate that gait unsteadiness is a key feature of this disorder and that fear of falling and gait unsteadiness are closely related. Alterations in frontal lobe and extra-pyramidal function are likely causes of the unsteady gait. Thus, treatment of fear of falling without consideration of gait unsteadiness may not be the best approach to improve the gait of these patients. When investigating therapies for this common gait disorder that markedly affects functional status and quality of life, it may be helpful to consider the possibility that the fear of falling may be justified and even protective [43]. Furthermore, since fear of falling plays such an important role in this gait disorder, neuro-psychological interventions should also be considered. Given the ubiquity of gait changes of largely unknown origin and the role of gait unsteadiness as a modifier of fall risk and predictor of future disability [44], it may be especially important to determine if underlying pathology causes both increased gait variability and fear of falling, if the increased instability leads to fear of falling, or whether an unexplained fear contributes to the gait instability in this population. In the meantime, if an older adult comes in to the clinic with a gait disturbance, it might be wise to check for frontal and extra-pyramidal signs and not to assume that the impaired mobility and fear of falling are a physiological or psychological response to aging.

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