Maturation of gait dynamics: stride-to-stride variability and its temporal organization in children

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Hausdorff, J. M., L. Zemany, C.-K. Peng, and A. L. Goldberger. Maturation of gait dynamics: stride-to-stride variability and its temporal organization in children. *J. Appl. Physiol.* 86(3): 1040–1047, 1999.—In very young children, immature control of posture and gait results in unsteady locomotion. In children of ~3 yr of age, gait appears relatively mature; however, it is unknown whether the dynamics of walking change beyond this age. Because stride dynamics depend on neural control, we hypothesized that motor control would continue to develop beyond age 3. To test this hypothesis, we measured the gait cycle duration on a stride-by-stride basis in 50 healthy 3- to 14-yr-old children (25 girls). Measurements of stride-to-stride variability were significantly larger both in the 3- and 4-yr-old children, compared with the 6- and 7-yr-old children, and in the 6- and 7-yr-old children, compared with the 11- to 14-yr-old children. Measurements of the temporal organization of gait also revealed significant age-dependent changes. The effects of age persisted even after adjusting for height. These findings indicate that mature stride dynamics may not be completely developed even in healthy 7-yr-old children and that different aspects of stride dynamics mature at different ages.

age; walking; spectral analysis; fractal analysis

WHEN YOUNG CHILDREN first begin to walk, immature control of posture and gait results in large stride-to-stride fluctuations and frequent falls (5, 23). By the time children are ~3 yrs old, their gait is relatively mature (26), and the visually apparent unsteadiness has been replaced by a more stable walking pattern. Nonetheless, subtle changes in the development of neuromuscular control and locomotor function continue well beyond age 3 (2, 19, 23, 25, 26). Some studies suggest a decrease in walking variability after this age (21, 24). However, a key unanswered question is whether subtle changes in gait unsteadiness and stride-to-stride dynamics also occur beyond this age.

Even in healthy young adults, the gait cycle duration (the stride time) fluctuates from one stride to the next in an apparently random, "noisy" manner (11, 16). However, in young adults with intact neural control, the magnitude of these fluctuations is relatively small. Although the stride-to-stride changes appear to fluctuate randomly, with no correlation between present and future stride times, the healthy adult locomotor system actually possesses "memory," such that the change from one stride to the next displays a subtle, "hidden" temporal structure that has been associated with long-range, fractal organization (11, 12). In contrast, in persons with neurological disease and in older persons, especially those with a history of falls, stride-to-stride variability increases, and the temporal organization of stride time dynamics is altered as well (3, 4, 7, 8, 10, 14).

These studies suggest that analysis of the stride time dynamics may also provide a window into the development of neuromuscular control in children. Given the apparent parallels between the immature gait of children and the unsteady gait of older persons and persons with neurological impairment (23), along with the subtle continued development of neural control beyond age 3, we hypothesized that stride time dynamics will not be fully matured at this age. In the present study, we tested this hypothesis by measuring stride-to-stride fluctuations in the gait cycle duration of healthy 3- to 14-yr-old children. More specifically, we sought *1*) to characterize the development of mature stride dynamics, *2*) to determine at what ages changes in gait dynamics occur, and *3*) to compare the gait dynamics of children to those of adults.

METHODS

Subjects

Fifty boys and girls participated in this study. Parents of 3- to 14-yr-old children were asked if they would be willing to have their child participate in this study. If the child and parent were willing to participate, parents were asked to provide informed written consent and to fill out a questionnaire describing the child's medical history. Most children were attending a local day camp or day care center. Children were excluded if they had any disorders likely to affect gait, if they were unable to walk independently for 8 min, or if they were born prematurely. Children were classified into three age groups: 3- and 4-yr-old ($n = 11$), 6- and 7-yr-old ($n = 20$), and 11- to 14-yr-old ($n = 12$) children. A few 5-yr-old ($n = 3$), 8-yr-old ($n = 1$), and 10-yr-old ($n = 3$) children were also studied. Height and weight of the youngest, middle, and oldest age groups were 105 ± 2, 125 ± 1, and 155 ± 10 cm and 17.3 ± 0.7, 25.3 ± 0.9, 44.4 ± 2.7 kg, respectively. There were equal numbers of boys and girls, and there were similar numbers of boys and girls in each age group. For comparison, we used data from historical controls [specifically, 10 healthy young adults (age, 18–29 yr) who walked for 1 h around a large track under conditions similar to those in the present study (12)]. All of the analysis methods performed on the children's data were applied to the first 8 min of the longer data segments in this adult control group.

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Protocol

Subjects walked at their self-determined, normal pace for 8 min around a 400-m running track. All subjects wore their own shoes or sneakers. An investigator walked slightly behind each subject during the test. A recently developed technique was used to measure the stride time dynamics during this relatively long walk on level ground (9, 11, 12). Two force-sensitive switches were placed inside the subject's right shoe: one underneath the heel of the foot and the other underneath the ball of the foot. The output of these foot switches, which provides a measure of the force applied to the floor, was sampled at 300 Hz and stored in a small ($5.5 \times 2 \times 9$ cm), lightweight (0.1 kg), ankle-worn recorder.[1] Subsequently, the recorded signal was automatically analyzed to determine initial contact time (heel strike) of each stride throughout the walk, and, hence, the stride time (the time from one heel strike to the next heel strike of the same foot) time series (9). The average walking speed was determined by measuring lap time.

Stride Time Dynamics

To study the effects of age on the intrinsic stride-to-stride dynamics, some preprocessing was performed on each time series. The first 60 s and the last 5 s of each time series were not included to eliminate any start-up or ending effects and to allow the subject to become familiar with the walking track. The time series were also processed to remove any pauses (stride time >2 s and the 5 s before and after any pauses) as well as any large spikes or outliers. These outliers, which occurred infrequently, were removed so that the intrinsic dynamics of each time series could be more readily analyzed. This was accomplished by using previously established methods (8, 10) by *1*) determining the mean and SD of the stride time while excluding the 5% of the data with the lowest and highest values and then *2*) removing from the original time series all data that fell >4.0 SD away from this mean value. The number of pauses (typically 0) and the number of strides excluded (typically 2%) were similar in all three age groups.

As shown in Table 1 and summarized below, we applied several measurements to analyze the variability and temporal structure of the stride time dynamics.

Stride-to-Stride Variability Measures

To estimate the overall stride-to-stride variability, we calculated the SD of each time series and the coefficient of variation (CV) ($100 \times$ SD/mean), an index of variability normalized to each subject's mean cycle duration. Both the SD and the CV provide a measure of overall variations in gait timing during the entire walk, i.e., the amplitude of the fluctuations in the time series with respect to the mean. However, these measures may be influenced by trends in the data (e.g., due to a change in speed) and cannot distinguish between a walk with large changes from one stride to the next and a walk in which stride-to-stride variations are small and more long-term, global changes (e.g., a change in average value) result in a large SD. Therefore, to estimate variability independent of local changes in the mean, we quantified successive stride-to-stride changes (i.e., the difference between the stride time of one stride and the previous stride) by determining the first difference of each time series. The first difference, a discrete analog of the first derivative, is one standard method for removing slow varying trends and is calculated by subtracting the previous value in the time series from the current value. The SD of the first-difference time series provides a measure of variability after detrending.

Table 1. *Stride time analysis methods*

Variability Measures: Fluctuation Magnitude
Standard deviation (SD)
Coefficient of variation (CV)
SD of detrended (first difference) time series
Temporal Structure Measures: Fluctuation Dynamics
Fourier (spectral) analysis
Autocorrelation decay time
Detrended fluctuation analysis

Unlike variability measures, temporal structure measures are sensitive to the order of the data points in the time series.

Temporal Structure Measurements

To study the temporal organization, we applied three methods to analyze different aspects of the dynamic structure of the time series of the stride time.

Spectral analysis. Fourier spectral analysis is a standard method for examining the dynamics of a time series. To ensure that these measures of the dynamics were independent of the average stride time or the stride time variability, we studied the first 256 points of each subject's time series (after the 60-s start-up period) by first subtracting the mean and dividing by the SD. This produces a time series centered at 0 with a SD of 1.0. Subsequently, standard Fourier analysis, with the use of a rectangular window, was performed on each time series. To quantify any differences in the spectra, we calculated the percentage of power in the high-frequency band (0.25–0.50 strides^{-1}) and the ratio of the low-frequency (0.05–0.25 strides^{-1}) to high-frequency power. This ratio excludes the power in the lowest frequencies and thus is independent of very large-scale changes in the stride time. By computing the ratio of the fluctuations over relatively long time scales (i.e., low frequencies) to short time scales (i.e., high frequencies), an index of the frequency "balance" of the spectra is obtained. A large low-to-high ratio is indicative of nonstationarity. Therefore, to the extent that the gait of the younger children is more nonstationary, one would expect this spectral ratio to decrease with maturation.

Autocorrelation decay. As a complementary method for analyzing the temporal structure of gait dynamics, we examined the autocorrelation properties of the stride time. The autocorrelation function estimates how a time series is correlated with itself over different time lags and provides a measure of the memory in the system, i.e., for up to how many strides is the present value of the stride time correlated with past values? After direct calculation of the autocorrelation function in the time domain (20), we calculated two indexes of autocorrelation decay: $\tau_{37\%}$ and $\tau_{67\%}$, the number of strides for the autocorrelation to decay to 37% ($1/e$) or 63% ($1 - 1/e$) of its initial value, respectively. To minimize any effects of data length, mean, or variance, we applied this analysis to the first 256 strides and normalized each time series with respect to its mean and SD. This autocorrelation measure emphasizes the correlation properties over a very short time scale, in

[1] Previous studies have shown that lower-extremity loading on the order of 1–2% of body weight can have subtle effects on gait. For the lightest child studied here, the gait monitor was ~0.8% of body weight. For most 6- and 7-yr-old children (15 of 20) and all of the oldest children, the monitor was <0.5% of body weight. Thus a very small loading effect cannot be excluded in the 3- and 4-yr-old children. However, it seems unlikely that this influenced the comparisons between the two older groups, in which, as shown below, there were still significant age-related differences.

which the correlation decays most rapidly. If the memory of the system increases with maturity, one would expect to see longer decay times in older children.

Stride time correlations. To further study the temporal structure of the stride time dynamics (independent of the overall variance), we also applied detrended fluctuation analysis (DFA) (11, 18) to each subject's time series. DFA is a modified random walk analysis that can be used to quantify the long-range, fractal properties of a relatively long time series or, in the case of shorter time series (i.e., the present study), it can be used to measure how correlation properties change over different time scales or observation windows (10, 18). Methodological details have been provided elsewhere (10–12, 18). Briefly, the root-mean square fluctuation of the integrated and detrended time series is calculated at different time scales, and the slope of the relationship between the fluctuation magnitude and the time scale determines a fractal scaling index (α). To determine the degree and nature of stride time correlations, we used previously validated methods (10) and calculated α over the region $10 \leq n \leq 20$ (where n is the number of strides in the window of observation). This region was chosen because it provides a statistically robust estimate of stride time correlation properties that are most independent of finite size effects (length of data) (17) and because it has been shown to be sensitive to the effects of neurological disease and aging in older adults (10). Like the autocorrelation method, the DFA method quantifies correlation properties. However, the DFA method assumes that, within the scale of interest, the correlation decays in a power-law manner and, therefore, a single exponent (α) can quantify the scaling. Whereas the autocorrelation method was applied to examine the dynamics over very short time scales, the DFA method, as applied here, examines scaling over relatively longer time periods. If the stride-to-stride fluctuations are more random (less correlated) in younger children, one would expect that α would be closer to 0.5 (white noise) in this group. In contrast, an α value closer to 1.5 would indicate fluctutations with a brown noise quality, indicating the dominance of low-frequency, slowly changing trends (18).

Statistical Methods

The nonparametric Kruskal-Wallis test was used to test for statistical differences among the three age groups. If this test showed significant differences, multiple Wilcoxon rank sum tests were performed to compare two groups at a time. These nonparametric tests make no assumptions about the underlying distribution of the data being compared. $P \leq 0.05$ was used as the level for statistical significance in detecting univariate group differences. Statistical analysis was performed by using SAS software release 6.12 (SAS, Cary, NC). Group results are reported as means $\pm$ SE.

RESULTS

Stride Time-Variability Measurements

Representative examples of the effects of age on the stride time fluctuations are shown in Fig. 1. The stride-to-stride variability is largest in the 4-yr-old, lower in the 7-yr-old, and smaller still in the 11-yr-old child. As summarized in Table 2, there was a highly significant effect of age on variability ($P < 0.0001$). Both the SD and CV were significantly larger in the 3- and 4-yr-old children compared with the 6- and 7-yr-old children ($P < 0.0001$). In addition, these measures were significantly larger in the 6- and 7-yr-old compared with the 11- to 14-yr-old children ($P < 0.005$). Of note, the stride-to-stride variability of the 11- to 14-yr-old children was closest to the values obtained in healthy young adults (CV = 1.3 $\pm$ 0.1% in the young adults and 2.1 $\pm$ 0.1% in the 11- to 14-yr-old children).

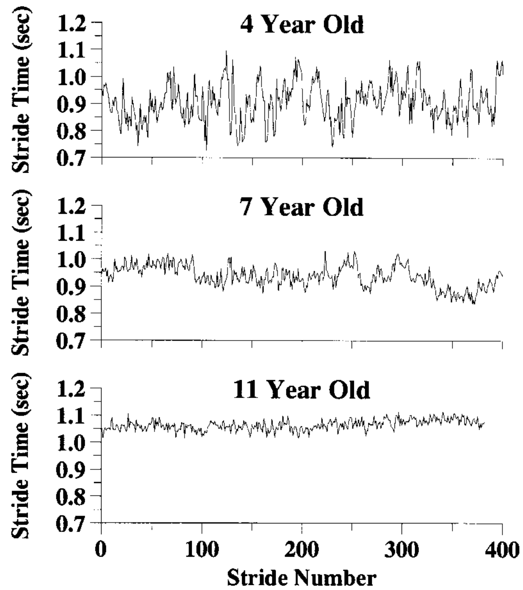

Fig. 1. Representative walking time series of 4-, 7-, and 11-yr-old children. Stride-to-stride fluctuations are largest in 4-yr-old child and smallest in 11-yr-old child. Coefficient of variation (CV), a measure of variability, was 8.4, 4.3, and 1.9% in 4-, 7-, and 11-yr-old children, respectively.

In the representative examples shown in Fig. 1, the local average of the stride time of the oldest child is relatively constant throughout the walk. In contrast, for the two younger children, the local average appears to change from time to time. Therefore, we next addressed two questions. *1*) Is the increased variability in the younger children simply due to fatigue during this walk? *2*) Is this increased variability due to a change in rate during the walk (e.g., long-term slowing down or speeding up) and not indicative of short-term, stride-to-stride unsteadiness per se?

Table 2. *Stride time variability*

	3–4 Yr Olds	6–7 Yr Olds	11–14 Yr Olds
SD, ms			
Original time series	55 ± 5†	31 ± 2	23 ± 1*‡
After detrending	48 ± 4†	28 ± 1	22 ± 1*‡
First 30 strides	47 ± 5†	25 ± 1	17 ± 1*‡
CV, %			
Original time series	6.1 ± 0.5†	3.3 ± 0.2	2.1 ± 0.1†‡
Lowest 30-stride segment	3.1 ± 0.2†	1.8 ± 0.1	1.2 ± 0.1†‡
First 30 strides	5.1 ± 0.5†	2.6 ± 0.1	1.6 ± 0.1†‡

Values are means $\pm$ SE; n = 11, 20, and 12 in 3- to 4-yr-old, 6- to 7-yr-old, and 11- to 14-yr-old age groups, respectively. Kruskal-Wallis tests detected significant differences among the 3 groups for all measures ($P < 0.0001$). Significant differences compared with 6- and 7-yr-old group, *$P < 0.005$; †$P < 0.0001$. ‡Significant difference between oldest and youngest groups, $P < 0.0001$.

To evaluate these questions, we detrended each time series to minimize the effects of any local changes in average stride. Figure 2 shows the results for the time series shown in Fig. 1. Even after detrending, variability is largest for the 4-yr-old child and smallest for the oldest child. This inverse relationship between variability and age after detrending was found in general for all subjects as well. The SD of the detrended time series, a measure of the dispersion or variability, was significantly larger in the 3- and 4-yr-old children compared with the 6- and 7-yr-old children ($P < 0.0001$) and in the 6- and 7-yr-old children compared with the oldest children ($P = 0.004$).

As a further test of these findings, we analyzed subsections of each subject's time series to find the 30 consecutive strides with the lowest CV. (A data-analysis window was moved forward five strides at a time across the time series, and in each window the CV was calculated.) Variability during this segment should be largely independent of a subject's speeding up or slowing down during the trial and reflects the "best-effort" of the neuromuscular control system. For the data shown in Figs. 1 and 2, the CV calculated in this manner was 3.8, 1.9, and 1.1% for the 4-, 7-, and 11-yr-old children, respectively. Figure 3 shows the results of this lowest variability time segment for all subjects. Even during a relatively short time period, the fluctuations from one stride to the next were significantly increased in the 3- and 4-yr-old children compared with the 6- and 7-yr-old children ($P < 0.0001$) and in the 6- and 7-yr-old children compared with the oldest children ($P < 0.0001$). In fact, the CV of each of the oldest children was lower than that of all of the 3- and 4-yr-old children.

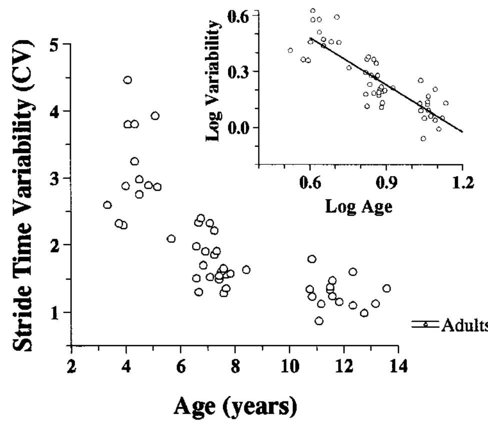

Fig. 3. Stride time variability as function of age. Shown is CV calculated during 30-stride subsection of each subject's time series with the lowest variability. Even during this period of relatively steady walking, gait variability decreases with age. *Inset*: data points replotted on log-log axes. Slope of best-fit line is close to -1.0, indicating that CV decreases inversely with age (CV $\approx$age^{-1}). Note how the stride time variability observed in oldest children approaches that of healthy adults (12). Eror bars, mean ± SE for young adults.

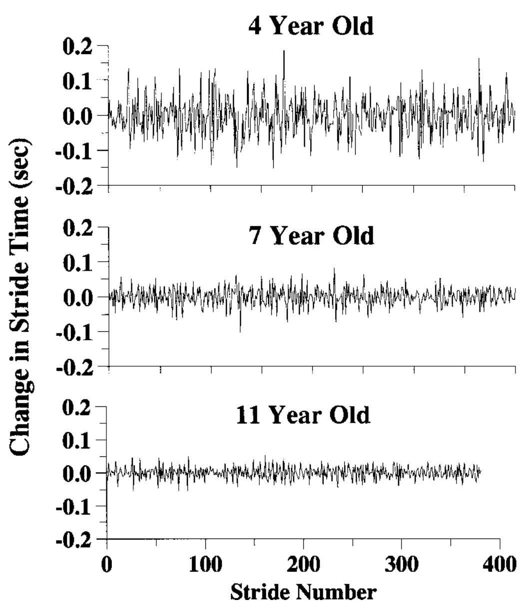

Fig. 2. Representative time series after detrending (for same data sets shown in Fig. 1). Even after detrending, which minimizes effects of local changes in mean value, stride-to-stride fluctuations in stride time are still largest in 4-yr-old child and smallest in 11-yr-old child. SD was 60, 27, and 20 ms for time series of these 4-, 7-, and 11-yr-old subjects, respectively. (For graphing purposes, 2 off-scale data points with values between 0.2 and 0.3 s are not shown for 4-yr-old subject.)

Finally, to confirm that the increased variability in the younger children was not simply due to fatigue or a change of speed during the walk, we studied the variability of only the first 30 strides. As was the case for the entire walk, both the SD and CV were significantly larger in the 3- and 4-yr-old children compared with the 6- and 7-yr-old children ($P < 0.0001$) and in the 6- and 7-yr-old children compared with the oldest children ($P < 0.0003$; Table 2).

Temporal-Structure Measurements

Spectral analysis. The above results demonstrate that the magnitude of stride-to-stride variability decreases with maturation in healthy children. The question we next address is whether the temporal structure of the stride time dynamics is also age dependent. Figure 4 shows the results of spectral analysis for the time series shown in Fig. 1. As expected, there appears to be a change in the frequency spectrum with age. The power in the higher frequency ranges appears to be slightly larger in the oldest child and smaller in the two younger children. Conversely, low-frequency power appears to be reduced in the 11-yr-old child compared with the two younger children. For the entire group in general, the percentage of high-frequency power was increased and low-frequency power was decreased in the oldest children compared with the other two groups (Table 3). Although these trends were not significant, there was a significant dependence of the low-to-high ratio on the age group ($P < 0.002$). This spectral ratio was significantly larger in the oldest children compared with the 6- and 7-yr-old children ($P < 0.02$), and it also tended to be larger in the 6- and 7-yr-old children compared with the youngest children ($P = 0.06$). In other words, the ratio of the stride time fluctuations on relatively large time scales to the fluctuations on shorter time scales decreased with age.

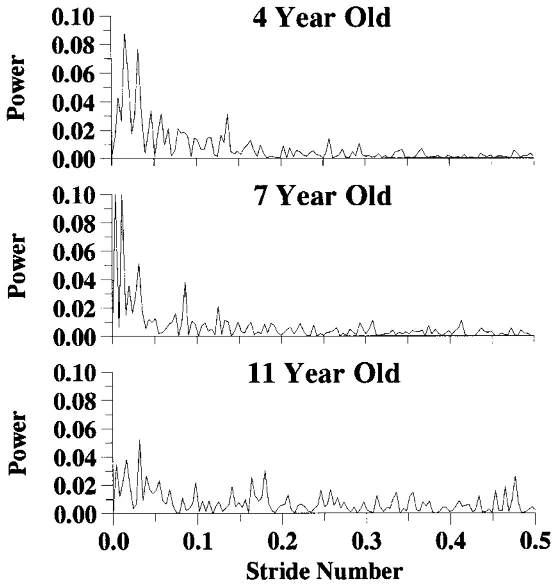

Fig. 4. Representative results of spectral analysis (for data sets shown in Fig. 1). Time series were normalized so that total power is same in each of the spectra. Note subtle decrease in low-frequency power and increase in high-frequency power with age. Ratios of low-frequency power (0.05–0.25 stride^{-1}) to high-frequency power (0.25–0.50 stride^{-1}) were 9.0, 4.6, and 1.5 for the 4-, 7-, and 11-yr-old subjects, respectively.

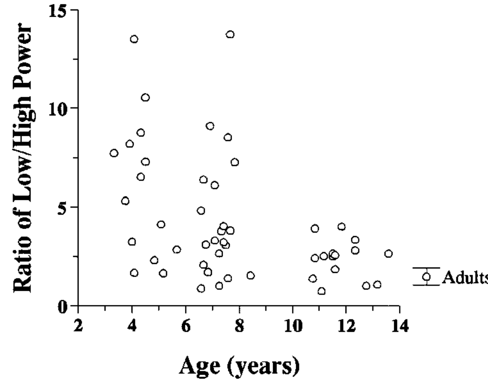

Fig. 5. Ratio of power in the relatively low-frequency (0.1–0.2 stride^{-1}) to relatively high-frequency (0.3–0.4 stride^{-1}) bands decreases with age. Note that this index of stride dynamics excludes highest and lowest frequencies. In oldest children, ratio approaches that of healthy adults. Error bars, mean ± SE for young adults. See also Table 3.

To confirm that this difference in spectral balance was not due to any simple large-scale trends in the data, we performed spectral analysis of each time series after detrending each time series (by taking the first difference). The results were similar to those for the original time series (Table 3); this suggests that there is a change in spectral balance independent of large-scale trends in the data. Moreover, we confirmed that this effect persisted even if we changed (somewhat arbitrarily) the way in which the spectra were divided. For example, when the high-frequency band was redefined as 0.3–0.4 stride^{-1} and the low-frequency band as 0.1 to 0.2 stride^{-1}, a similar effect of age on the balance of spectral power was observed (Table 3 and Fig. 5).

Autocorrelation measurements. As expected, measurements of the decay of the autocorrelation function also varied with age. For the younger children, $\tau_{63\%}$ decayed rapidly (after 2 or 3 strides), whereas this decay time was generally larger in the two older groups. Specifically, $\tau_{63\%}$ was 2.5 ± 0.2 and 4.8 ± 0.6 strides in the 3- and 4-yr-old and the 6- and 7-yr-old children, respectively ($P < 0.0005$). $\tau_{63\%}$ was slightly, but not significantly, larger in the 11- to 14-yr-old children (5.6 ± 1.1 strides) compared with the 6- and 7-yr-old children. Similar results were obtained for $\tau_{37\%}$. This measure of the decay of the autocorrelation function was also lowest in the 3- and 4-yr-old children (5.8 ± 1.0 strides), larger ($P < 0.06$) in the 6- and 7-yr-old children (11.4 ± 3.3 strides), and tended to be slightly larger in the 11- to 14-yr-old children (19.0 ± 9.8 strides; $P < 0.01$ compared with the youngest children).

Table 3. *Spectral analysis*

	3–4 Yr Olds	6–7 Yr Olds	11–14 Yr Olds
% High-frequency power, 0.25–0.5 stride^{-1}	0.054 ± 0.010	0.064 ± 0.012	0.100 ± 0.022
Low-to-high ratio	6.8 ± 1.2	4.1 ± 0.5	2.3 ± 0.5*†
Low-to-high ratio after detrending	0.30 ± 0.02*	0.20 ± 0.03	0.15 ± 0.03†
% High-frequency power, 0.3–0.4 stride^{-1}	0.022 ± 0.005	0.023 ± 0.004	0.038 ± 0.009
Low-to-high ratio	6.8 ± 1.1	4.5 ± 0.7	2.3 ± 0.3*†
Low-to-high ratio after detrending	0.41 ± 0.06	0.29 ± 0.04	0.18 ± 0.02†

Values are means ± SE; n = 11, 20, and 12 in 3- to 4-yr-old, 6- to 7-yr-old, and 11- to 14-yr-old groups, respectively. Kruskal-Wallis tests detected significant differences among the 3 groups for all measures except high-frequency power. Significant differences between groups; * compared with 6- to 7-yr-old group, $P < 0.05$; †oldest compared with youngest group, $P < 0.005$.

Stride time correlations. The fractal-scaling index α was similar in the two youngest age groups and tended to decrease in the oldest children (α = 0.93 ± 0.04, 0.93 ± 0.03, 0.88 ± 0.04, in the 3- and 4-yr-old, 6- and 7-yr-old, and 11- to 14-yr-old children, respectively). When this analysis was performed on the first difference of the time series (i.e., after removing any large trends), the effect of age became more pronounced and statistically significant ($P < 0.01$ and $P < 0.05$ comparing the 11- to 14-yr-old children to the 6- and 7-yr-old and the 3- and 4-yr-old children, respectively).

The DFA method automatically "detrends" the data by determining the fluctuations about the least-squares, best-fit straight line in each window of observation. Nonstationarities (trends) that are not well characterized by a straight line could possibly give rise to an inaccurate scaling exponent. Therefore, to further examine the dynamical properties, we also computed the scaling index α by using higher order DFA detrending. Specifically, we detrended each window of box size n by using second-order polynomials instead of the first-order linear detrending (12).

With second-order detrending of the time series, the age effect was apparent both before (see Fig. 6) and after taking the first difference of the time series. Among the younger subjects (<11-yr-old subjects), 10 subjects (~25%) had scaling indexes >1.0, whereas all

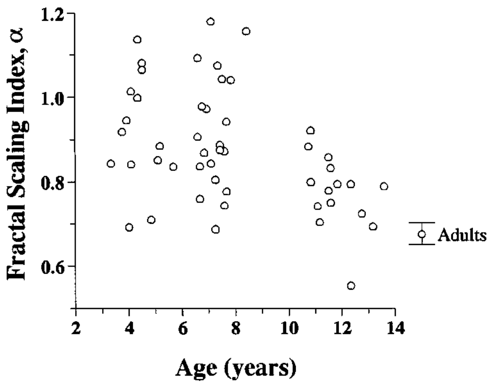

Fig. 6. Fractal scaling index (α) decreases with age. This finding suggests an age-related change in stride-to-stride dynamics over the range of 10–20 strides. Scaling exponent shown here was determined after 2nd-order detrending of each window of observation. Note how α observed in oldest children approaches that of healthy adults. Error bars, means ± SE for young adults.

of the scaling exponents were <1.0 in the oldest subjects. Although the scaling properties were similar in the 3- and 4-yr-old and the 6- and 7-yr-old subjects, α was significantly lower in the oldest children compared with the 6- and 7-yr-old children and compared with the 3- and 4-yr-old children ($P < 0.05$). The mean α of the oldest children comes closest to the mean value obtained in young adults (Fig. 6).

Relationship of Stride Dynamics to Height

In children, many aspects of gait depend on body size. For example, over certain age ranges, stride length increases linearly with age, but the relationship between stride length and age becomes constant after adjusting for height or leg length (2). Similarly, we observed a significant increase ($P < 0.0001$) in velocity in the 6- and 7-yr-old children (1.20 ± 0.03 m/s) compared with the 3- and 4-yr-old children (1.00 ± 0.03 m/s). However, relative velocity (velocity/height) was essentially identical in these two groups ($P = 0.92$). To begin to evaluate whether the changes in stride dynamics were only a function of changes in biomechanics related to growth, we normalized the dynamical measurements with respect to height. In the present study, height increased linearly with age ($r = 0.96$; $P < 0.0001$). Variability is inversely related to age (Fig. 3). Thus we normalized the dynamical variables (either by appropriately dividing or multiplying by height) and reexamined the relationship with age. The age dependence persisted when the measures of variability and dynamics were adjusted for height or if leg length was used instead of height as the normalizing factor. For example, the CV normalized with respect to leg length was 128 ± 10, 83 ± 5, and 67 ± 3 in the 3- and 4-yr-old, 6- and 7-yr-old, and the 11- to 14-yr-old subjects, respectively. The difference between the 6- and 7-yr-old and the 11- to 14-yr-old children was significant ($P < 0.05$). The CV in the 3- and 4-yr-old children was significantly increased ($P < 0.0005$) compared with both older groups.

Finally, we note that, consistent with previous findings (26), the average values of stride time and walking velocity were age dependent. Mean stride time was 900 ± 14 ms in the 3- and 4-yr-old children, increased to 955 ± 12 ms in the 6- and 7-yr-old children ($P < 0.01$), and increased further in the 11- to 14-yr-old group (1.072 ± 18 ms; $P < 0.0001$). Walking velocity was 1.00 ± 0.03 m/s in the 3- and 4-yr-old children, 1.20 ± 0.03 m/s in the 6- and 7-yr old children, and 1.28 ± .0.03 m/s in the 11- to 14-yr old children. The difference between the youngest and the two older groups was significant ($P < 0.0001$); however, walking velocity was not significantly different in the 6- and 7-yr-old and the 11- to 14-yr-old groups.

DISCUSSION

This quantitative study of stride variability and dynamics reveals several interesting new findings. *1*) Stride-to-stride variations in gait cycle duration are significantly larger in healthy 3- and 4-yr-old children compared with 6- and 7-yr-old children and in 6- and 7-yr-old children compared with children ages 11 to 14. *2*) The temporal structure of gait fluctuations is not fully developed in 7-yr-old children, whereas in older children (11- to 14-yr-old children), stride dynamics approach the values observed in adults. *3*) Different features of stride dynamics do not develop at the same time (Table 4). Thus, whereas visual observation might suggest that the stride dynamics of children are not different from those of adults, quantitative measurement of gait dynamics indicates that stride-to-stride control of walking is not fully mature even in 7-yr-old children.

A number of similarities have been reported in the gait patterns of children and elderly adults (5, 6, 23). This finding may reflect a reappearance of primitive reflexes or simply diminished control of balance (23). The present study demonstrates that parallels also exist with respect to stride dynamics. As we observed in children, alterations of stride dynamics have been seen in older adults and persons with neurological impairment (3, 4, 7, 8, 10, 14).

Although the stride dynamics of young children share some characteristics of the unstable dynamics of the elderly and those with neurological dysfunction, there appear to be important differences as well. For

Table 4. *Effects of age on stride time dynamics*

	3–4 Yr Olds	6–7 Yr Olds	11–14 Yr Olds
Variability	↑↑	↑	—
Low- to high-frequency power	↑↑	↑	—
Autocorrelation decay time	↓	—	—
Fractal scaling exponent	↑	↑	—

Arrows indicate comparisons with oldest children ($n = 12$), in whom stride time dynamics are most like those of adults. Note how different aspects of the temporal structure of the stride dynamics tend to mature at different ages. The low- to high-frequency power ratio was not statistically different in the 2 youngest groups; however, this representation reflects the observed trend toward a decreased ratio in 6- and 7-yr-old children ($n = 20$) compared with 3- and 4-yr old children ($n = 11$).

example, the present findings suggest that the fractal-scaling index changes monotonically throughout the lifespan (highest in children, lower in adults, and lowest in the elderly and persons with neurological disease). In contrast, stride variability likely changes in a U-shaped fashion (high in children, lower in adults, and higher with disease and perhaps also in very advanced age). Thus, from the perspective of stride time dynamics, the changes in gait of older persons do not simply reflect a return to an immature gait pattern.

The alterations of the dynamics of the stride time in the younger children may be caused by a number of factors. The increased variability may in part be related to decreased walking velocity and decreased postural stability at lower speeds (23). However, whereas adjustment for height minimized the effects of age on velocity, the age-related differences in both the magnitude of the variability and in the dynamics persisted after controlling for height. A number of factors also suggest that the observed age-related changes in the temporal organization of stride dynamics are most likely not simply attributable to reduced height, gait speed, change in concentration during the walk, or increased stride-to-stride variability (unsteadiness). For example, fractal scaling indexes were similar in the 3- and 4-yr-old children compared with the 6- and 7-yr-old children, despite significant differences in stride-to-stride variability, velocity, and height. Age-related differences in stride dynamics were evident in dynamical metrics even after detrending to minimize the effects of changes in speed or local average stride time. Moreover, an age-related effect was observed in the ratio of spectral balance, a measure that was derived independently of stride-to-stride variability and very-low-frequency changes likely to be associated with change of speed or loss of concentration.

Future study of children who are walking at different speeds may help elucidate the role of velocity on stride dynamics in children. In addition, studies that include assessment of motor control and balance as well as other aspects of the locomotor control system may also help to clarify the role of potential contributing factors to the development of mature gait dynamics. Perhaps differences in motor control development account for some of the observed heterogeneity in stride dynamics within each age group (e.g., Fig. 1). An intriguing possibility is that these dynamical metrics may provide a means of quantifying the stage of maturational development. In any case, it seems that *1*) stride time dynamics most likely depend on some aspect of the neuromuscular control system that is not merely related to walking velocity or gait variability, and *2*) the immature gait dynamics in children may reflect the subtle, ongoing development of more than one component of motor control. The dynamic action theory of motor control postulates that locomotor function can be viewed as a complex system with multiple degrees of freedom, the collective behavior of which is governed in part by the principle of self-organization (13, 23, 27). Perhaps, therefore, mature locomotion dynamics emerge only when all of the interacting individual components are fully developed. The change in scaling exponents with age, a measure associated with a nonequilibrium dynamical system with multiple degrees of freedom (1, 22), may reflect this emergent behavior. Candidate elements that could affect stride dynamics include biomechanical and neural properties that are known to mature only in older children (e.g., electromyogram recruitment patterns are more variable in children who are <7 yr of age) (15, 23).

Additional studies will be needed to explain these complex age-related changes in the magnitude and temporal structure of stride dynamics. Nonetheless, the present findings have potentially important implications for the understanding and modeling of the integrative control of locomotor function and neural development. Furthermore, the results suggest the possibility that quantitative measures of stride dynamics may be useful in augmenting the early detection and classification of gait disorders in children.

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REFERENCES

1. **Bak, P., and M. Creutz.** Fractals and self-organized criticality. In: *Fractals in Science*, edited by A. Bunde and S. Havlin. New York: Springer-Verlag, 1994, p. 27–47.
2. **Beck, R. J., T. P. Andriacchi, K. N. Kuo, R. W. Fermier, and J. O. Galante.** Changes in the gait patterns of growing children. *J. Bone Joint Surg. Am*. 63: 1452–1457, 1981.
3. **Blin, O., A. M. Ferrandez, J. Pailhous, and G. Serratrice.** Dopa-sensitive and dopa-resistant gait parameters in Parkinson's disease. *J. Neurol. Sci*. 103: 51–54, 1991.
4. **Blin, O., A. M. Ferrandez, and G. Serratrice.** Quantitative analysis of gait in Parkinson patients: increased variability of stride length. *J. Neurol. Sci*. 98: 91–97, 1990.
5. **Breniere, Y., and B. Bril.** Why do children walk when falling down while adults fall down in walking? *Can. R. Acad. Sci. III* 307: 617–622, 1988.
6. **Forssberg, H., B. Johnels and G. Steg.** Is Parkinsonian gait caused by a regression to an immature walking pattern? *Adv. Neurol*. 40: 375–379, 1984.
7. **Hausdorff, J. M., M. E. Cudkowicz, R. Firtion, J. Y. Wei, and A. L. Goldberger.** Gait variability and basal ganglia disorders: stride-to-stride variations in gait cycle timing in Parkinson's and Huntington's disease. *Mov. Disord*. 13: 428–437, 1998.
8. **Hausdorff, J. M., H. E. Edelberg, S. L. Mitchell, A. L. Goldberger, and J. Y. Wei.** Increased gait unsteadiness in community dwelling elderly fallers. *Arch. Phys. Med. Rehabil*. 78: 278–283, 1997.
9. **Hausdorff, J. M., Z. Ladin, and J. Y. Wei.** Footswitch system for measurement of the temporal parameters of gait. *J. Biomech*. 28: 347–351, 1995.
10. **Hausdorff, J. M., S. L. Mitchell, R. Firtion, C. K. Peng, M. E. Cudkowicz, J. Y. Wei, and A. L. Goldberger.** Altered fractal dynamics of gait: reduced stride interval correlations with aging and Huntington's disease. *J. Appl. Physiol*. 82: 262–269, 1997.
11. **Hausdorff, J. M., C.-K. Peng, Z. Ladin, J. Y. Wei, and A. L. Goldberger.** Is walking a random walk? Evidence for long-range

correlations in the stride interval of human gait. *J. Appl. Physiol.* 78: 349–358, 1995.

12. **Hausdorff, J. M., P. L. Purdon, C.-K. Peng, Z. Ladin, J.-Y. Wei, and A. L. Goldberger.** Fractal dynamics of human gait: stability of long-range correlations in stride interval fluctuations. *J. Appl. Physiol.* 80: 1448–1457, 1996.
13. **Kelso, J. A. S., and B. Tuller.** A dynamical basis for action systems. In: *Handbook of Cognitive Science*, edited by M. S. Gazanniga. New York: Plenum, 1984: 321–356.
14. **Miller, R. A., M. H. Thaut, G. C. McIntosh, and R. R. Rice.** Components of EMG symmetry and variability in Parkinsonian and healthy elderly gait. *Electroencephalogr. Clin. Neurophysiol.* 101: 1–7, 1996.
15. **Myklebust, B. M.** A review of myotatic reflexes and the development of motor control and gait in infants and children: a special communication. *Phys. Ther.* 70: 188–203, 1990.
16. **Pailhous, J., and M. Bonnard.** Steady-state fluctuations of human walking. *Behav. Brain Res.* 47: 181–190, 1992.
17. **Peng, C.-K., S. V. Buldyrev, A. L. Goldberger, S. Havlin, M. Simons, and H. E. Stanley.** Finite size effects on long-range correlations: implications for analyzing DNA sequences. *Phys. Rev. E* 47: 3730–3733, 1993.
18. **Peng, C.-K., S. Havlin, H. E. Stanley, and A. L. Goldberger.** Quantification of scaling exponents and crossover phenomena in nonstationary heartbeat time series. *Chaos* 6: 82–87, 1995.
19. **Preis, S., A. Klemms, and K. Muller.** Gait analysis by measuring ground reaction forces in children: changes to an adaptive gait pattern between the ages of one and five years. *Dev. Med. Child Neurol.* 39: 228–233, 1997.
20. **Press, W. H., B. P. Flannery, S. A. Teukolsky, and W. T. Vetterling.** *Numerical Recipes*. Cambridge, UK: Cambridge Univ. Press, 1987, p. 381–453.
21. **Rose-Jacobs, R.** Development of gait at slow, free, and fast speeds in 3- and 5-year-old children. *Phys. Ther.* 63: 1251–1259, 1983.
22. **Schoner, G., and J. A. S. Kelso.** Dynamic pattern generation in behavioral and neural systems. *Science* 239: 1513–1520, 1988.
23. **Shumway-Cook, A., and M. H. Woollacott.** *Motor Control: Theory and Practical Applications*. Baltimore, MD: Williams & Wilkins, 1995.
24. **Slaton, D. S.** Gait cycle duration in 3-year-old children. *Phys. Ther.* 65: 17–21, 1985.
25. **Sutherland, D. H., R. Olshen, L. Cooper, and S. L. Woo.** The development of mature gait. *J. Bone Joint Surg. Am.* 62: 336–353, 1980.
26. **Sutherland, D. H., R. A. Olshen, E. N. Biden, and M. P. Wyatt.** *The Development of Mature Walking*. Oxford, UK: MacKeith, 1988.
27. **Thelen, E., J. A. S Kelso, and A. Fogel.** Self-organizing systems and infant motor development. *Developmental Rev.* 7: 39–65, 1987.