Dynamical structure of center-of-pressure trajectories in patients recovering from stroke

Roerdink, M.; de Haart, M.; Daffertshofer, A.; Donker, S.F.; Geurts, A.C.; Beek, P.J.

published in
Experimental Brain Research
2006

DOI (link to publisher)
10.1007/s00221-006-0441-7

document version
Publisher's PDF, also known as Version of record

Exp Brain Res (2006) 174: 256–269
DOI 10.1007/s00221-006-0441-7

RESEARCH ARTICLE

M. Roerdink · M. De Haart · A. Daffertshofer
S. F. Donker · A. C. H. Geurts · P. J. Beek

Dynamical structure of center-of-pressure trajectories in patients recovering from stroke

Received: 31 August 2005 / Accepted: 12 March 2006 / Published online: 10 May 2006

Abstract In a recent study, De Haart et al. (Arch Phys Med Rehabil 85:886–895, 2004) investigated the recovery of balance in stroke patients using traditional analyses of center-of-pressure (COP) trajectories to assess the effects of health status, rehabilitation, and task conditions like standing with eyes open or closed and standing while performing a cognitive dual task. To unravel the underlying control processes, we reanalyzed these data in terms of stochastic dynamics using more advanced analyses. Dimensionality, local stability, regularity, and scaling behavior of COP trajectories were determined and compared with shuffled and phase-randomized surrogate data. The presence of long-range correlations discarded the possibility that the COP trajectories were purely random. Compared to the healthy controls, the COP trajectories of the stroke patients were characterized by increased dimensionality and instability, but greater regularity in the frontal plane. These findings were taken to imply that the stroke patients actively (i.e., cognitively) coped with the stroke-induced impairment of posture, as reflected in the increased regularity and decreased local stability, by recruiting additional control processes (i.e., more degrees of freedom) and/or by tightening the present control structure while releasing non-essential degrees of freedom from postural control. In the course of rehabilitation, dimensionality stayed fairly constant, whereas local stability increased and regularity decreased. The progressively less regular COP trajectories were interpreted to indicate a reduction of cognitive involvement in postural control as recovery from stroke progressed. Consistent with this interpretation, the dual task condition resulted in less regular COP trajectories of greater dimensionality, reflecting a task-related decrease of active, cognitive contributions to postural control. In comparison with conventional posturography, our results show a clear surplus value of dynamical measures in studying postural control.

Keywords Motor control · Posture · Non-linear dynamics · Stroke · Rehabilitation

M. Roerdink (✉) · A. Daffertshofer · S. F. Donker
A. C. H. Geurts · P. J. Beek
Faculty of Human Movement Sciences,
Institute for Fundamental and Clinical
Human Movement Sciences,
Vrije Universiteit, Van der Boechorststraat 9,
1081 BT Amsterdam, The Netherlands
E-mail: m.roerdink@fbw.vu.nl
Tel.: +31-20-5988516
Fax: +31-20-5988529

M. De Haart
Department of Rehabilitation, Amsterdam Medical Centre,
University of Amsterdam,
Amsterdam, The Netherlands

M. De Haart · A. C. H. Geurts
Department of Research, Development, and Education,
St. Maartenskliniek,
Nijmegen, The Netherlands

S. F. Donker
Department of Otorhinolaryngology,
Vrije Universiteit Medical Centre,
Amsterdam, The Netherlands

A. C. H. Geurts
Department of Rehabilitation Medicine,
University Medical Centre, St. Radboud,
Nijmegen, The Netherlands

Introduction

In quiet standing, the position of the center of mass varies continuously, resulting in changes in the forces exerted by the human body on the support surface and in the corresponding ground reaction forces. This postural sway can be studied by using force platforms that measure the displacement of the application point of the ground reaction force, that is, the center of pressure (COP). The time-evolution of the resulting COP trajectories is often viewed, usually implicitly, as a manifestation of random fluctuations in the postural control system. This view underlies the application of conventional averaging techniques in posturography that aim to

identify scalar values, like the mean COP velocity, by averaging out the assumed noisy or random character of postural sway. Such descriptive statistical values have been shown to change with pathologies and aging and to vary over a range of sensory and cognitive conditions. There are ample indications in the literature, however, that more detailed analyses of COP time-evolutions may provide further insight into postural control.

In this context, the nature of postural sway has been characterized both as deterministic chaotic (e.g., Newell et al. 1993; Yamada 1995; Pascolo et al. 2005) and stochastic (e.g., Collins and De Luca 1993; Newell et al. 1997; Delignières et al. 2003). Chaotic time series appear random and unpredictable but arise from deterministic non-linear processes, whereas stochastic time series are governed by chance alone (e.g., Brownian motion, see below) or by a combination of deterministic and random processes (e.g., biased random walk). Although conceptually different, these approaches both focus on the dynamical structure of COP trajectories, which may contain information about the postural control exerted. The 'smoothness' of the COP trajectories hints at strong deterministic components in the stochastic postural sway dynamics and comparatively weak influences of noise (see also Collins and De Luca 1993, 1995; Riley et al. 1999; Riley and Turvey 2002). Using linear systems theory and corresponding identification techniques, Kiemel et al. (2002) similarly concluded that COP trajectories reflect a mixture of deterministic and stochastic components. However, this more traditional approach tends to downplay the importance of the repeatedly demonstrated non-linear character of the COP dynamics. Hence, in order to identify the stochastic dynamics of postural sway, COP analyses that account for the non-linear and stochastic temporal evolution of postural sway appear appropriate. While it has already been demonstrated that analyses borrowed from the field of dynamical systems can be meaningfully applied to measured COP trajectories (e.g., Newell et al. 1993; Yamada 1995; Pascolo et al. 2005), we expected that they would also be valuable in evaluating the effects of health status, rehabilitation, and task manipulations in clinical studies (cf. Raymakers et al. 2005).

From this expectation, we reanalyzed the COP data that were collected and analyzed by De Haart et al. (2004) in an encompassing longitudinal study of the recovery of standing balance in stroke patients. Those data are particularly interesting from a clinical point of view because they cover over 30 patients whose postural control was assessed at five stages, during a 3 month recovery period under both sensory (eyes-open versus eyes-closed) and cognitive (standing with eyes open while performing an arithmetic dual task) manipulations. With conventional posturographic measures (e.g., mean position, sway amplitude, and sway velocity), De Haart et al. (2004) found that, compared to age- and gender-matched healthy elderly controls, stroke patients exhibited a severe weight-bearing asymmetry that was accompanied by increased sway amplitude and sway velocity, especially in the frontal plane. With follow-up assessments, the weight-bearing asymmetry reduced, as did the sway amplitude and velocity, suggesting that posture stabilized in the course of rehabilitation. Under the arithmetic dual task, COP velocity in the sagittal plane was greater, which was interpreted as an indication that patients 'stiffened up' to some degree, perhaps as a result of an increased alertness. Finally, without vision, a larger COP amplitude and velocity were observed.

We reanalyzed the data of De Haart et al. (2004) using a set of complementary measures borrowed from dynamical systems theory to evaluate our expectation that they would reveal new aspects of the data that were not disclosed by means of the conventional measures mentioned above. Since a plethora of measures may be used to analyze the temporal structure underlying COP trajectories, we restricted the analysis to four: the correlation dimension, the largest Lyapunov exponent, the sample entropy, and the Hurst exponent. This particular choice was motivated from the consideration that these measures are all defined operationally in terms of readily interpretable and complementary (i.e., deterministic and stochastic) properties of motor control, namely, number of degrees of freedom, local stability, regularity, and scaling behavior. More specifically, the correlation dimension (e.g., Grassberger and Procaccia 1983) measures the dimensionality of the COP time series (e.g., Newell 1998; Yamada 1995) and may provide an estimate of the number of (active) dynamical degrees of freedom involved in postural control (e.g., Kay 1988). Lyapunov exponents quantify the convergence or divergence of nearby points in the postural control state space, with the largest Lyapunov exponent characterizing a system's local stability (Wolf et al. 1985; Rosenstein et al. 1993; Yamada 1995), that is, the sensitivity of the postural control system to local perturbations. Sample entropy (Richman and Moorman 2000) quantifies the regularity (or predictability) of a time series, while scaling factors like the Hurst exponent [e.g., determined via detrended fluctuation analysis (DFA), Peng et al. 1994, 1995] quantify the extent to which a recorded COP time series exhibits long-range correlations. To date, these dynamical measures have rarely been used in clinical studies of upright standing (see, e.g., Newell et al. 1993; Pascolo et al. 2005, for exceptions), in spite of their capacity to provide new insights, as widely demonstrated in, for instance, physiology and the study of "dynamical diseases" (e.g., Goldberger et al. 1996, 1997, 2002; Glass 2001; Lipsitz 2002; Kyriazis 2003).

With our choice of measures, we abstained from using other techniques that have recently been applied in the study of postural control, such as recurrence quantification analysis (RQA, Riley et al. 1999), rambling–trembling component analysis (Zatsiorsky and Duarte 1999), sway-density analysis (Baratto et al. 2002), and stabilogram diffusion analysis (Collins and De Luca 1993, 1995). Although the various output measures of RQA are conceptually related to some of the measures used in the present study, especially those indexing

Table 1 Characteristics of the 33 stroke patients at the baseline assessment

Age (years)	61.2 (SD 13.0; range 27–82)
Time post-stroke (weeks)	9.8 (SD 5.2; range 3.3–24.1)
Type of stroke (infarction/haematoma)	26/7
Hemisphere of stroke (left/right)	10/23
Functional Ambulation Categories[a]	2 (range 1–4)
Lower-limb motor selectivity (Brunnstrom stage)[b]	IV (range II–VI)

[a]Values are median scores (range). Functional Ambulation Categories are defined as follows: 0, Non-functional (unable): patient cannot walk or requires help of two or more people; 1, Dependent (level 2): patient requires firm continuous support from one person who helps carrying weight and with balance; 2, Dependent (level 1): patient needs continuous or intermittent support of one person to help with balance and coordination; 3, Dependent (supervision): patient requires verbal supervision or standby help from one person without physical contact; 4, Independent (on level ground): patient can walk independently on level ground, but requires help on stairs, slopes, or uneven surfaces; 5, Independent: patient can walk independently anywhere (cf. Collen et al. 1990; Wade 1992)

[b]Values are median scores (range). Brunnstrom stages are defined as follows: I, flaccid paralysis; II, increased muscle tone without active movement; III, increased muscle tone with active movements mainly in rigid extension synergy; IV, increased muscle tone with alternating gross movements in extension and flexion synergies; V, muscle tone normalization with some degree of selective muscle control (i.e., combined active knee extension and foot dorsiflexion against some resistance); and VI, normal muscle tone and control (cf. Brunnstrom 1966; Fugl-Meyer et al. 1975)

stability and regularity, they are not identical and cannot be simply mapped onto each other. Unlike RQA, rambling-trembling component analysis, sway-density analysis, and stabilogram diffusion analysis have been constructed specifically to examine postural sway dynamics, but the aim of our study was to evaluate whether generally motivated measures derived from dynamical systems theory would have surplus value in the analysis of COP trajectories compared to standard posturographic measures.

Methods

Participants and procedure

We reanalyzed the COP data from 33 stroke patients (mean age 61.2 years, SD 13.0 years) out of an inception cohort of 37 stroke patients (viz. data from four patients were excluded due to missing values) and 22 healthy elderly (mean age 63.9 years, SD 9.3 years) that was used by De Haart et al. (2004).

In this study, stroke patients receiving standard rehabilitation training were evaluated five times over a 12 week period from the moment that each patient was able to stand without assistance for at least 30 s [i.e., grade 4 according to the standing balance scale described by Bohannon (1995)], the so-called baseline assessment, as well as 2, 4, 8, and 12 weeks after that moment. Stroke type (infarction/haematoma), location (left/right hemisphere), and the time post-stroke of the 33 included patients are provided in Table 1, together with an indication of the level of recovery at the baseline assessment, as quantified by the clinical evaluation of the patients' (1) walking skills, as rated according to the six-point (range 0–5) Functional Ambulation Categories (cf. Collen et al. 1990; Wade 1992), and (2) lower-limb motor selectivity, as scored according to the six motor stages defined by Brunnstrom (cf. Brunnstrom 1966; Fugl-Meyer et al. 1975).

During each assessment, two posturographic trials for three quiet-standing conditions [eyes open (EO), eyes open while performing an arithmetic dual task (DT), and eyes closed (EC)] were performed[1]. In each condition, the participants were asked to stand as still and symmetrically as possible (i.e., barefoot, arms alongside the trunk (if possible), feet placed with the heels 8.4 cm apart, with the toes pointing outward at a 9° angle from the sagittal midline), while their COP was recorded for 30 s at a sample frequency of 60 Hz. The healthy elderly underwent the same procedure (see Nienhuis et al. 2001). We refer to the original study by De Haart et al. (2004) for more detailed information about the participants, equipment, and the procedures.

Posturographic data analysis

Prior to all the analyses, each signal's mean was subtracted. Time series were sampled by means of $t \rightarrow t_i$, with $i = 1, 2, 3, \ldots, N$ and N indicating the total number of samples in the COP time series. The signals are denoted as $x(t_i)$, with $x = \mathrm{ML}$ (i.e., medio-lateral) = AP (i.e., anterio-posterior) COP displacements. We used the conventional standard deviation σ_x to quantify the variability of the postural sway dynamics $x(t_i)$. Besides the mean and variance of the time series, which, by definition, ignore the temporal structure of the COP trajectories, we assessed the COP dynamics by calculating its correlation dimension, largest Lyapunov exponent, sample entropy, and scaling behavior. In the following, we explain how those

[1]In the original study of De Haart et al. (2004), each balance assessment consisted of two consecutive test series, incorporating four quiet-standing tasks and one weight-shifting task, presented in a fixed sequence. This sequence was repeated in reverse order to control for time effects. Between the DT and EC condition, participants conducted a trial while looking at a vertical black bar, which served as a visual midline reference. The weight-shifting task was performed twice after (and preceding) the first (second) EC condition. A 1-min rest was given after each balance test, whereas a longer pause was allowed between the two test series. The arithmetic task in the DT condition consisted of a (varying) verbal sequence of eight single-digit additions (e.g., $7+4=11$ or $3+5=7$) equally timed over the 30-s period. The participants were instructed to verbally indicate the correctness of each summation by good or fault response.

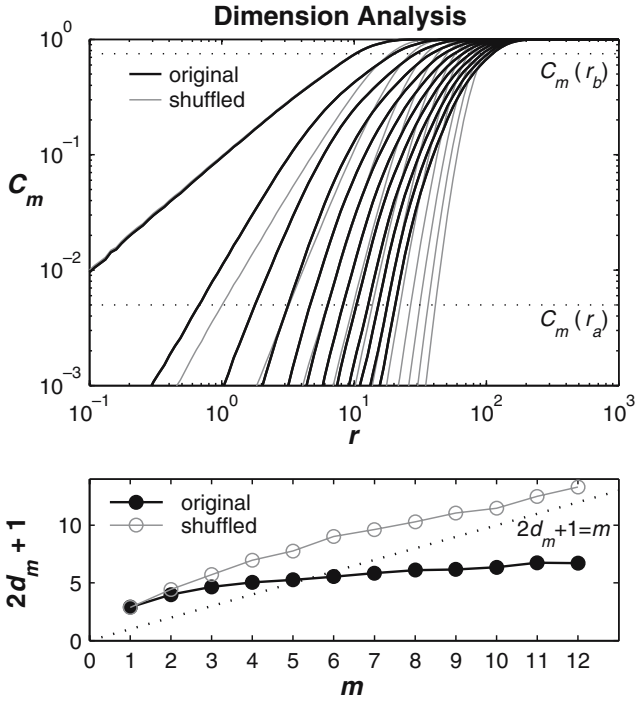

Fig. 1 Dimensionality analysis. Top panel: correlation sum C_m plotted for each embedding dimension m for original (*thick black lines*) and shuffled surrogate (*thinner gray lines*) COP time series (ML, EO, stroke patient). The *dotted horizontal lines* indicate the boundaries between which the slopes d_m were estimated (Kaplan et al. 1991; see text for further details). Lower panel: a plot of the slopes d_m for the original (*black solid circles*) and shuffled surrogate (*gray open circles*) correlation sums for increasing embedding dimension m. Note that at the sixth embedding dimension, the slope of the original COP time series satisfies the condition $m > 2d_m + 1$ (i.e., the corresponding dimension estimate D_2 is 2.26), whereas for the shuffled surrogate data, $C_m(r)$ scales with r^m

calculations were performed in such a manner that the analyses can be replicated if desired.

Correlation dimension

To calculate the correlation dimension of the time series, we followed Grassberger and Procaccia (1983) by conducting a phase space reconstruction of the COP dynamics (Takens 1981). To this end, the measured COP time series was embedded in a high-dimensional phase space by time-delaying the original time series as $x(t_i)$, $x(t_i+\tau)$, $x(t_i+2\tau)$, ... (see, for instance, Abarbanel 1996; Packard et al. 1980). The embedding delay τ corresponded to the first minimum of the mutual information function[2] (Fraser and Swinney 1986; see also Abarbanel 1996), that is, the time at which the original and delayed signals were maximally independent. As shown in Fig. 1, the modified correlation sum C_m of the COP time series $x(t_i)$ was finally computed for each embedding dimension

$$C_m(r) = \frac{1}{N_p} \sum_{i=1}^{N-W} \sum_{j=i+W}^{N} \Theta\left(r - \left|\mathbf{X}_i - \mathbf{X}_j\right|\right) \quad (1)$$

in which m refers to the embedding dimension and $\Theta(z)$ denotes the unit step function: $\Theta(z) = 1$ if $z \geq 0$, $\Theta(z) = 0$ otherwise. $\mathbf{X}_i$ is given as $[x(t_i), x(t_i+\tau), \ldots, x(t_i+(m-1)\tau)]$, $|\mathbf{X}_i - \mathbf{X}_j|$ is the Euclidean distance between $\mathbf{X}_i$ and $\mathbf{X}_j$, and r is a distance on a log scale. The cut-off parameter[3] W was defined as twice the first minimum in the mutual information function. N_p is the number of pairs i, j such that $|i\text{-}j| \geq W$, where $N_p = (N\text{-}W+1)(N\text{-}W)/2$ (Theiler and Rapp 1996). Importantly, for small distances r, the function $C_m(r)$ behaves as a power law, i.e.,

$$C_m(r) \propto r^{D_2} \quad (2)$$

where D_2 is the correlation dimension that is approximated via the linear region of the slope of the log–log display of $C_m(r)$ as a function of r. Notice that for very small r, estimating D_2 becomes difficult because usually the number of data points (samples) becomes very small, yielding inaccuracies similar to the case in which r becomes comparable to the attractor size. Hence, the slope was determined for an interval between r_a, i.e., the distance r capturing 0.5% of the pairs of points ($C_m(r_a) = 0.005$) and r_b, i.e., the distance r capturing 75% of the pairs of points ($C_m(r_b) = 0.75$) (see also Fig. 1), upper panel[4]. The slopes were determined per embedding dimension m according to:

$$d_m = \frac{\log_{10} C_m(r_b) - \log_{10} C_m(r_a)}{\log_{10} r_b - \log_{10} r_a} \quad (3)$$

[2]For our data, the first minimum of the mutual information occurred at 11 (mean 11.14, SE 0.15) and 10 (mean 9.91, SE 0.20) data samples for ML and AP sway components of the stroke patients, respectively. These minima did not change with *rehabilitation* or *condition* ($P > 0.05$). For the healthy controls, the first minimum of the mutual information occurred at 11 data samples for both ML (mean 11.10, SE 0.27) and AP (mean 10.54, SE 0.21) sway components. Again, no change with *condition* was found ($P > 0.05$). Note that the choice of the time delay τ was not based on these group averages but was determined independently for each trial.

[3]Correlations between consecutively sampled points can produce spurious indications of low-dimensional structure. With the introduction of the cut-off parameter $W > 1$, it is possible to minimize these correlations (Grassberger 1986; Theiler 1986). Therefore, all pairs of points that are closer together in time than some cut-off W were excluded. $W = 1$ returns the standard Grassberger and Procaccia (1983) formula.

[4]The dimension is often calculated by looking at the slope of the most linear segments of $C_m(r)$, requiring a means of evaluating a score for each plausible linear segment (i.e., based on the length of the segment or the goodness of fit to a line). The 'optimal' linear segment is chosen. In this way, these techniques emphasize the possible existence of strange attractors. A drawback of such methods is that the length scale chosen can depend discontinuously on the underlying signal, because a small change in the signal can change the relative ranks of the candidate linear segments and thereby change the calculated dimension substantially (Kaplan et al. 1991). Because the applied dimension analysis in this study did not involve examination of the linear scaling of $C_m(r)$, it would be incorrect to interpret the estimated dimension D_2 as *the* dimension of the attractor. Similarly, it would be incorrect to infer from this analysis that an attractor must exist.

We estimated the dimension D_2 by looking for cases in which the slopes d_m saturated with increasing embedding dimension m, with $m > 2d_m + 1$ (Fig. 1, lower panel). Finally, whenever $m > 2d_m + 1$ was fulfilled, the estimate D_2 could be considered reliable.

Largest Lyapunov exponent

We quantified the mean exponential divergence $d(t)$ at time t of initially close state-space trajectories by means of $d(t) \propto Ce^{\lambda_{\max} t}$ (e.g., Rosenstein et al. 1993). In this form, the exponent $\lambda_{\max}$ reflects a system's local stability, that is, its sensitivity to small and local perturbations and is referred to as the largest Lyapunov exponent. Briefly, if $\lambda_{\max}$ is negative, then any perturbation will exponentially damp out and initially close trajectories will stay close. In contrast, if $\lambda_{\max}$ is positive, the $d(t)$'s will diverge, i.e., the distance between trajectories will increase exponentially. Positive $\lambda_{\max}$ values indicate the presence of chaos, provided that the system stays in finite vicinity, implying that some attractor exists. In the latter case, nearby points diverge exponentially (local instability), causing a lack of predictability.

To calculate $\lambda_{\max}$, for each $\mathbf{X}_i$ of embedding dimension m ($m > 2d_m + 1$), the nearest neighbor was identified as the point closest to $\mathbf{X}_i$ with a temporal separation larger than twice the first minimum in the mutual information function (see above). Next, distances between neighboring trajectories in state space were calculated as a function of time (i.e., $j\Delta t = 3$ s) and averaged over all original pairs of nearest neighbors i. The $\lambda_{\max}$ were finally estimated from the slopes of

$$y(j) = \frac{1}{\Delta t}\frac{1}{N}\sum_{i=1}^{N} \ln\, d_i(j) \tag{4}$$

after fitting a range from $j\Delta t = 0$ to 0.75 s (Rosenstein et al. 1993).

Sample entropy

In addition to the aforementioned measures, we used more statistical descriptions to characterize not only the deterministic structure of the COP dynamics, but also its stochastic features. In general, entropy- or information-related measures are first candidates when analyzing a system's regularity or ordering. Since we investigated time series rather than arbitrary statistical ensembles, we used the sample entropy (Richman and Moorman 2000), which builds on the conditional probability that a signal of length N will repeat itself for M points, provided that it already repeated itself for M-1 points (within a tolerance range r and without allowing self-matches): the higher the entropy, the lower the time series' regularity.

In view of the limited length of the to-be-analyzed time series, we chose the window parameter as $M = 3$—note that the choice of r is limited since too small a tolerance yields low confidence, while the discriminative capacity drops when increasing the tolerance range (Pincus 1991; Pincus and Goldberger 1994). Recently, Lake et al. (2002) proposed to use the maximal value of the relative errors of sample entropy and conditional probability for a range of values of r and M to optimize the choice of r and M while enhancing the efficiency of the entropy estimate by penalizing conditional probabilities near 0 and near 1. Here, the sample entropy analysis parameters r and M were selected based on minima in the gray-scaled relative error map (Fig. 2)—cf. Lake et al. (2002) for further details.[5]

Scaling: detrended fluctuation analysis

The Hurst exponent H quantifies the leading order of the temporal change of a time series' correlation (or least squared displacements). Formally one may write

$$\sigma^2_{\Delta x}(\Delta t) \propto \Delta t^{2H} + \cdots \tag{5}$$

i.e., the variance σ^2 of the displacement Δx changes in time Δt according to a power law (with exponent $2H$). For the simplest diffusion process, the free Brownian motion or ordinary random walk, the Hurst exponent is $H = 0.5$. Other values in the range $0 < H < 1$ are typically referred to as fractional Brownian motion (Mandelbrot and van Ness 1968): $H > 0.5$ implies persistence, i.e., the trajectory tends to continue in its current direction and thus produces enhanced diffusion; $H < 0.5$ implies antipersistence, i.e., the trajectory tends to return from where it came and thus suppresses diffusion.

Importantly, COP trajectories are always bounded within the area of support, whereas (fractional) Brownian motion is, in principle, unbounded. Indeed, Eq. (5) does not hold for diffusion processes that remain limited in that the corresponding variance saturates beyond a critical time interval (see Delignières et al. 2003). Thus, in order to estimate the Hurst exponent using Eq. (5), some preprocessing needs to be applied. The basic idea is that if the original signal is bounded, then the integrated time series is unbounded and exhibits quantifiable scaling properties (Delignières et al. 2003; see also Eke et al. 2000, 2002). This integration is the first step in the so-called DFA (Peng et al. 1994, 1995), in which, subsequently, this integrated time series is divided into non-overlapping intervals. Within each interval, the time series is linearly detrended to remove trivial correlations (Fig. 3, center panel) and, finally, the root mean square fluctuations F_n of the residual are displayed as function of the interval size n (Fig. 3, lower panel). In the presence of a power law, the according log–log representation yields a scaling exponent α that relates to the Hurst exponent as $H = (2\alpha - 1)/4$.

[5] In agreement with, e.g., Lake et al. (2002) and Richman and Moorman (2000), time series were normalized to unit variance. Sample entropy software was obtained from PhysioNet (Goldberger et al. 2000).

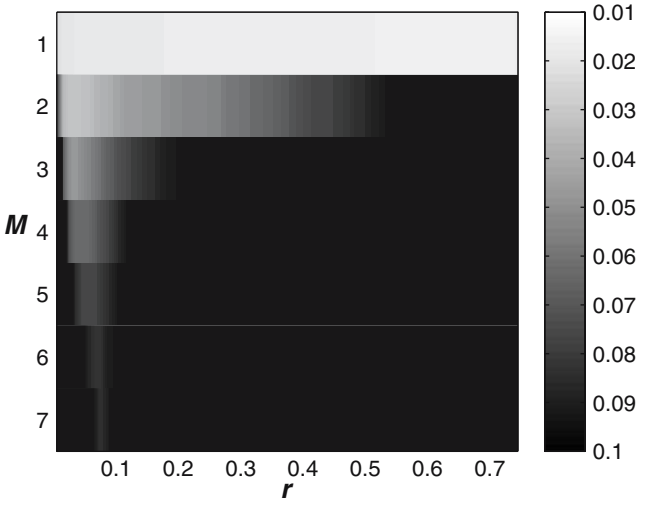

Fig. 2 Visual selection of the optimal tolerance r for estimating the sample entropy of 66 randomly chosen, normalized ML COP time series (i.e., $\sigma_{ML} = 1$) of healthy adults, based on the median value of the relative error. As can be seen from this gray-scaled map, a window length of $M = 1$ allows a confident estimation of entropy for a wide range of r. For $M \geq 2$, however, optimum values of r are clearly evident and lie generally between 0.02 and 0.1. The relative error rises very steeply for $r < 0.02$ and for larger r values, stressing the fact that many combinations of M and r are intolerable. Based on inspection of the gray-scaled visual relative error plot at the $M = 3$ trace, $r = 0.035$ was the optimal choice for the sample entropy parameter r (lowest relative error). Similarly, $r = 0.035$ and $r = 0.045$ were selected as optimal parameter values for ML and AP sample entropy estimates, respectively. These r values were the same for the stroke patients and the healthy controls

Detrended fluctuation analysis was applied to all $x(t_i)$ time series using logarithmically spaced interval lengths n from 10 to $N/2$ samples[6] ($N = 1{,}800$). The slope of the double-logarithmic plot of F_n versus n was estimated between $n = 0.26$ s and $n = 8.66$ s. The largest values of n were disregarded, since these points were based on only a few data segments, rendering the estimate unreliable.

Surrogate data

In order to guarantee the validity of the non-linear analyses applied here, we exploited surrogate data by means of both time- and phase-randomized COP trajectories (Theiler et al. 1992). We first generated surrogate data by randomly selecting samples of $x(t_i)$, i.e., we preserved the data's statistics (mean, variance, etc.), but 'destroyed' the data's temporal correlation by shuffling its temporal ordering (cf. Fig. 3, upper panel). Notice that, due to the absence of temporal correlations, shuffled surrogate data have a huge dimension (converging to infinity), a high sample entropy, and a Hurst exponent close to zero. In addition, we randomized the data's phase after Fourier transformation. In general, randomizing the phase does not alter the spectral power distribution and thus preserves the data's auto-correlation function (cf. Kantz and Schreiber 2004). Hence, Hurst exponents of phase-randomized and original data should match, while, as for the shuffled surrogates, phase-randomization is expected to result in high correlation dimension and sample entropy.

[6]Notice that a multivariate extension of the detrended fluctuation analysis algorithm yields identical results when applied to the embedded time series (see above) since we assumed stationarity.

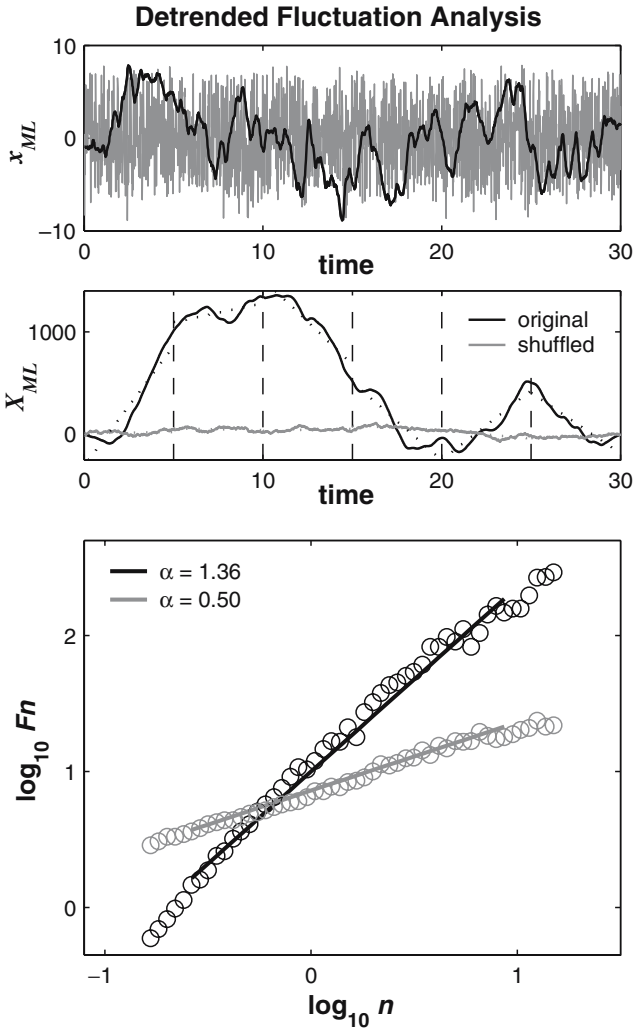

Fig. 3 Detrended fluctuation analysis. Upper panel: ML COP time series x_{ML} of a stroke patient with eyes open (*black line*) and its shuffled surrogate counterpart (*gray line*). Middle panel: integrated time series X_{ML}. The *vertical dashed lines* indicate an interval of length $n = 5$ s, the *dotted straight line* segments represent the trend estimated in each interval by a least-squares fit. Lower panel: double-logarithmic plot of fluctuation F_n versus interval length n. The slope α relates to the scaling exponent H according to $H = (2\alpha - 1)/4$. Note that for the shuffled data $H = 0$, indicating an uncorrelated random process, whereas for the COP time series $H = 0.43$, indicating long-range correlations

Statistics

All the dependent variables for the stroke patients were analyzed using a repeated measures analysis of variance (ANOVA) with within-subject factors *rehabilitation* (five

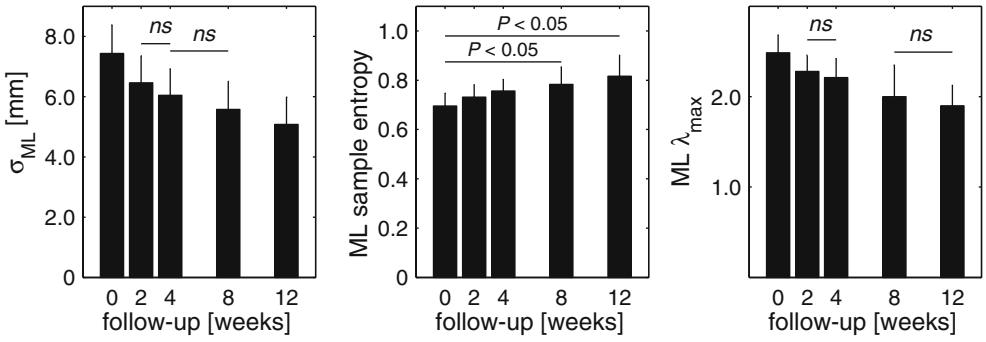

Fig. 4 *Rehabilitation* effects for σ_{ML}, ML sample entropy and ML λ_{max} values. *Error bars* denote the 95% confidence intervals for difference after Bonferroni adjustment for multiple comparisons. σ_{ML} and λ_{max} decreased significantly with follow-up assessments [except when explicitly stated so (*ns*)]. Sample entropy was significantly higher after 8 and 12 weeks when compared to baseline (0 weeks)

follow-up levels: baseline (week 0) and 2, 4, 8, and 12 weeks later) and *condition* (three levels: EO, DT, and EC). In the case of significant main or interaction effects, post-hoc analysis was performed by means of *t*-tests using the standard Bonferroni correction to maintain the family-wise error rate at 5%. The effect of *health status* was examined by means of a repeated measures ANOVA with *health status* (two levels: stroke patients (averaged over follow-up assessments) and healthy controls) as between-subject factor and *condition* as within-subject factor. Post-hoc *t*-testing with Bonferroni correction was performed for significant effects of *condition*, whereas for significant interaction effects, separate repeated measures ANOVAs with *condition* as within-subject factor were performed for the two groups. For main and interaction effects of *rehabilitation*, *condition*, and *health status*, Cohen's *f* was used as a measure of effect size; large effect sizes were operationalized by convention as $f > 0.4$ (Cohen 1988). To quantify the effects of *randomization*, we used a repeated measures ANOVA with *randomization* as within-subject factor (e.g., three levels: original COP data, shuffled surrogates, and phase-randomized surrogates), for stroke patients and healthy elderly, separately.

Results

The results (*F*, *P*, and *f* values) of the *rehabilitation* by *condition* ANOVA and the *health status* by *condition* ANOVA for the dependent COP variables are presented in Tables 2 and 3, respectively.

Rehabilitation

With follow-up assessments, COP variability (σ_{AP} and σ_{ML}) decreased significantly and local stability improved as evidenced by a significantly reduced λ_{max} in both ML and AP directions. In addition, COP regularity decreased in the ML direction as indexed by a significantly increased sample entropy (Fig. 4; Table 2). Scaling exponents[7] (*H*), dimension estimates (D_2), and sample entropy in the AP direction were not affected significantly by *rehabilitation* (Table 2).

Condition

Figure 5 displays *condition* effects for stroke patients (see also Table 2). The COP variability was significantly greater for EC as compared to both EO and DT. The COP regularity was clearly lower (significantly higher sample entropy) for DT than for EO and EC. The ML scaling exponents (*H*) were significantly lower for EC than for EO and DT. In contrast, AP scaling exponents were significantly higher for EO than for the two other conditions. The estimated dimension was significantly larger for DT than for EO. The largest Lyapunov exponent λ_{max} was significantly larger for EC (i.e., locally less stable) than for EO and DT. No significant *rehabilitation* × *condition* interaction was observed for either of the COP measures. For the healthy elderly, qualitatively similar *condition* effects were observed.

Health status

Stroke patients differed significantly from healthy elderly in all COP measures except for the ML scaling exponents (see also Table 3 and Fig. 6)[8]. COP variability

[7] To avoid false or spurious conclusions, Hurst exponents were also determined by means of a rescaled range analysis (Hurst 1965; Rangarajan and Ding 2000; Delignières et al. 2003; cf. Wing et al. 2004 for a related power spectral approach), yielding slightly higher estimates of the diffusion process than the DFA. To compare these two methods, the pair-wise two-tailed Pearson correlation coefficient between the scaling exponent based on the rescaled range analysis ($H \rightarrow H_{R/S}$) and the detrended fluctuation analysis ($H \rightarrow H_{DFA}$) was determined for all the trials of the stroke patients ($N = 990$). For both the AP and ML scaling estimates, the correlation analysis showed a good agreement between $H_{R/S}$ and H_{DFA} ($r = 0.918$, $P < 0.01$ and $r = 0.895$, $P < 0.01$, respectively).

[8] The significant results reported in Table 3 were all preserved when the averaged post-stroke values were replaced by the earliest post-stroke values.

Table 2 Main and interaction effects of *rehabilitation* (five levels) and *condition* (three levels) on standard deviation σ, Hurst exponent H, dimension estimate D_2, sample entropy, and largest Lyapunov exponent λ_{max} for ML and AP COP components of 33 stroke patients

	Rehabilitation			*Condition*			Interaction		
	$F^{a}_{(4,\ 128)}$	P	f	$F^{a}_{(2,\ 64)}$	P	f	$F^{a}_{(8,\ 256)}$	P	f
Standard deviation σ									
ML	24.29	<0.001	0.87	5.80	<0.01	0.43	1.67	*ns*	0.23
AP	11.97	<0.001	0.61	15.93	<0.001	0.71	1.42	*ns*	0.21
Hurst exponent H									
ML	0.65	*ns*	0.14	8.88	<0.001	0.68	1.57	*ns*	0.22
AP	0.89	*ns*	0.17	8.94	<0.001	0.53	0.55	*ns*	0.13
Dimension estimate D_2									
ML	0.32	*ns*	0.10	5.35	<0.01	0.41	0.73	*ns*	0.15
AP	0.36	*ns*	0.21	3.96	<0.05	0.39	1.17	*ns*	0.06
Sample entropy									
ML	5.70	<0.005	0.42	4.76	<0.05	0.38	1.14	*ns*	0.19
AP	0.66	*ns*	0.14	17.75	<0.001	0.75	1.31	*ns*	0.20
Lyapunov exponent λ_{max}									
ML	24.35	<0.001	0.87	8.30	<0.005	0.51	0.95	*ns*	0.17
AP	26.95	<0.001	1.02	5.94	<0.01	0.48	0.66	*ns*	0.16

ns not significant

[a]In case the sphericity assumption was violated, the number of degrees of freedom was adjusted using the Huynh–Feldt method. Missing values arose for the dimension estimate D_2 and the Lyapunov exponent λ_{max} due to the $m > 2d_m + 1$ criterion for six stroke patients for the AP COP component only (nine missing values in total). No missing value analysis was performed, resulting in a loss of six patients in the *rehabilitation* × *condition* repeated measures ANOVA

was significantly larger for stroke patients than for the healthy controls. COP regularity in the ML direction was higher (significantly lower sample entropy) for stroke patients, whereas COP regularity in the AP direction was significantly higher for the healthy controls. For the stroke patients, the scaling exponents for AP were significantly lower, while the estimated dimension and λ_{max} were significantly higher in both directions when compared to the healthy controls. There were two significant *health status* × *condition* interactions (Table 3), but these could not be interpreted as none of the post-hoc comparisons was significant.

Randomization

As expected, all Hurst exponents vanished for the shuffled surrogates (see above and Fig. 3, lower panel) and H did not differ from the original value after phase-randomization ($P > 0.05$; see Fig. 7) for both the stroke patients and the healthy elderly. This implies that strong correlations were present in the time evolution of the COP trajectories, suggesting a deterministic dynamical structure. Furthermore, ML and AP sample entropy estimates for the shuffled and phase-randomized surrogate time series were both significantly higher than their original counterparts for the stroke patients and the healthy elderly alike (Fig. 7). Higher entropy values imply that more information is required to describe the surrogate data due to the applied time- and phase-randomization, which again suggests a deterministic component in the original COP trajectories.

The dimension analysis revealed that $C_m(r)$ of shuffled surrogates scaled with r^m rather than r^{D_2} (see Fig. 1, lower panel), reflecting the presence of very high (or infinite) dimensional noise in the surrogate data that was absent in the original data. Irrespective of the health status and movement direction, the D_2 and λ_{max} of phase-randomized surrogates were significantly higher than their original counterparts (Fig. 7). In line with the previous results, this again indicates the presence of a pronounced deterministic component.

Discussion

In the present study, we reanalyzed the COP data of De Haart et al. (2004) using dynamical rather than conventional measures to examine whether this would lead to novel insights into the changes in postural control as a function of (a) health status (stroke patients versus healthy elderly), (b) rehabilitation (follow-up assessments), and (c) task conditions (EO, DT, EC). In the following, we systematically discuss to what extent new findings and insights were obtained vis-à-vis those reported by De Haart et al. (2004), using the three aforementioned independent variables as entry points. We then conclude with a general evaluation of the significance of studying postural sway dynamics.

Health status

As expected, we replicated the finding of De Haart et al. (2004) that stroke induced substantial differences in the global characteristics of the COP trajectories in comparison with the healthy controls (see also De Haart et al. 2004). Postural sway variability (i.e., deviation from the mean) was larger in patients than in healthy

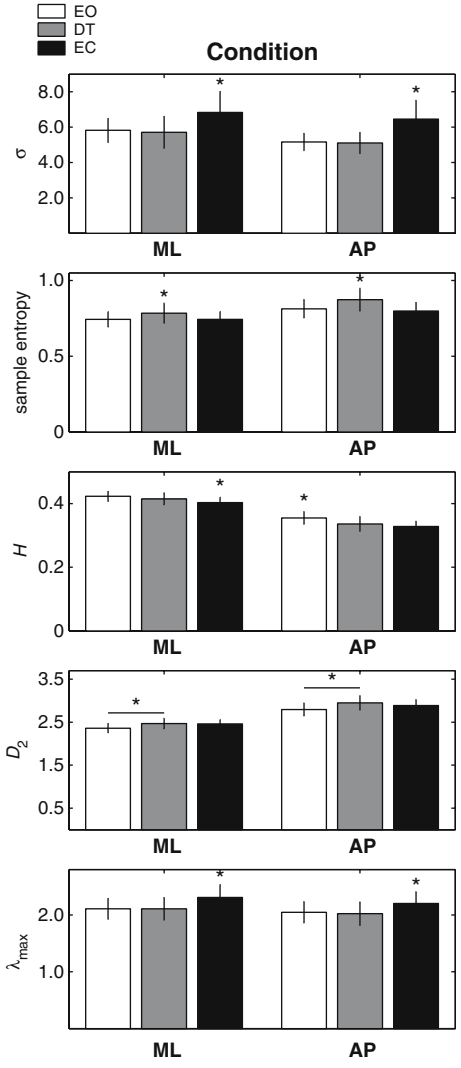

Fig. 5 *Condition* effects of ML and AP COP variability σ, sample entropy, Hurst exponent H, dimension estimate D_2, and largest Lyapunov exponent λ_{max}. Eyes open (EO: *white bars*), dual task (DT: *gray bars*), and eyes closed (EC: *black bars*) conditions for stroke patients (healthy adults showed qualitatively similar results) are depicted. *Error bars* denote the 95% confidence intervals. *Stars* placed at an error bar of a specific condition denote a significant difference from the other two conditions, whereas *stars* placed above connecting lines represent significant differences between the two connected conditions (see Table 2 for F, P, and f values for *condition* effects)

elderly. Analyses of surrogate data revealed, however, that the observed increase in variability was not due to an increase in noise. Unlike their shuffled and phase-randomized counterparts, the original data exhibited dynamical features other than pure randomness (see Fig. 7), suggesting the presence of a correlated dynamical structure in the recorded COP trajectories. Importantly, the structure (i.e., scaling behavior) of the correlations in the COP trajectories was preserved qualitatively in the stroke patients despite their impairment. That is, stroke did not lead to a breakdown of long-range correlations, as has been found for other pathologies (e.g., Goldberger et al. 1996, 1997, 2002). Our scaling analysis revealed Hurst exponents that varied between white noise and Brownian motion (i.e., $0 < H < 0.5$), indicating that the COP trajectories of both the healthy elderly and the stroke patients exhibited an anti-persistent behavior: a decreasing trend is followed by an increasing future trend (cf. Delignières et al. 2003, see also Collins and De Luca 1993; Frank et al. 2001). By definition, those results regarding the correlated dynamical structure in the recorded COP trajectories could only be obtained by applying dynamical methods of data analysis.

Marked differences between patients and healthy controls were found in the three other dynamical measures (Fig. 6). Dimensionality was increased in AP direction due to stroke (Fig. 6). This increased dimensionality may simply be interpreted as a result of increased noise, which would be consistent with the increased dimensionality and sample entropy after phase-randomization and shuffling (Fig. 7). However, the increased dimensionality may also be interpreted as a change in postural control. One option is to interpret the D_2 values literally in terms of chaos theory, which would imply that the data were chaotic because D_2 always saturated at a non-integer value, and that the dimension of the chaotic COP attractor was greater for the patients than for the healthy controls. However, claims about the presence of deterministic chaos have to be made with great caution because distinguishing between chaos and stochasticity on the basis of finite datasets might be difficult. Hence, we focused on the differences between groups and across conditions rather than the possibly chaotic nature of the data. Accordingly, the dimensionality findings may be interpreted to imply that the stroke patients recruited additional control processes (degrees of freedom), for instance to compensate for the reduced efficacy of ankle mechanisms on their paretic side (De Haart et al. 2004; Geurts et al. 2005). Alternatively, it could also be the case that the already present control structure (defined over essential degrees of freedom) was tightened so that (non-essential) degrees of freedom were released from control, resulting in greater dimensionality due to greater expression of noise along uncontrolled dimensions (i.e., the notion of uncontrolled manifold; cf. Schöner 1995; Scholz and Schöner 1999). Unfortunately, at the level of the COP variable, i.e., an output variable integrating many subsystems, it seems hard, if not impossible, to unambiguously relate the observed increased dimensionality to an increased noise level (be it directly or indirectly via an increasing number of released (or uncontrolled) degrees of freedom) versus an increased number of recruited control processes. As will become apparent in the following, however, the stability analysis and the regularity analysis provided clues to tentatively resolve this impasse.

The largest Lyapunov exponents (λ_{max}) of the COP trajectories were significantly greater for the stroke patients than the healthy elderly, demonstrating a

Table 3 Main and interaction effects of *health status* (between-subject factor: two levels) and *condition* (within-subject factor: three levels) on standard deviation σ, Hurst exponent H, dimension estimate D_2, sample entropy, and largest Lyapunov exponent λ_{max} for ML and AP COP components of 33 stroke patients and 22 healthy controls

	Health status			*Condition*			Interaction		
	$F^{a}_{(1,\ 53)}$	P	f	$F^{a}_{(2,\ 106)}$	P	f	$F^{a}_{(2,\ 106)}$	P	f
Standard deviation σ									
ML	63.35	<0.001	1.09	4.40	<0.05	0.29	3.34	<0.05	0.25
AP	20.62	<0.001	0.62	23.98	<0.001	0.67	0.44	*ns*	0.09
Hurst exponent H									
ML	0.02	*ns*	0.02	8.27	<0.001	0.40	0.03	*ns*	0.02
AP	15.11	<0.001	0.53	12.63	<0.001	0.49	1.04	*ns*	0.14
Dimension estimate D_2									
ML	3.67	$=0.061$	0.26	5.90	<0.005	0.33	1.13	*ns*	0.15
AP	24.69	<0.001	0.68	8.68	<0.001	0.41	0.51	*ns*	0.10
Sample entropy									
ML	7.38	<0.01	0.37	0.15	*ns*	0.05	1.37	*ns*	0.16
AP	9.60	<0.005	0.43	7.88	<0.005	0.38	0.40	*ns*	0.08
Lyapunov exponent λ_{max}									
ML	126.34	<0.001	2.77	14.36	<0.001	0.52	0.29	*ns*	0.07
AP	37.23	<0.001	0.83	30.53	<0.001	0.76	5.89	<0.005	0.33

ns not significant

[a]In case the sphericity assumption was violated, the number of degrees of freedom was adjusted using the Huynh–Feldt method. Due to the averaging of conditions with *rehabilitation*, no missing values for the factor *health status* were present (i.e., 33 stroke patients versus 22 healthy adults)

decreased local stability or a deteriorated neuromuscular control. Interestingly, Buzzi et al. (2003) found similar dynamical signatures (i.e., larger dimensionality and decreased local stability) in the variability of joint kinematics of gait with aging, which they attributed to deficiencies in the ability to actively control joint motion. Elderly walkers were unable to compensate for the natural stride-to-stride variations, which could increase the risk of falling (Buzzi et al. 2003). Similarly, stroke patients may have a reduced ability to compensate for small (internal and/or external) perturbations, forcing them to actively adjust postural control either by recruiting more degrees of freedom or by tightening the control of essential variables while releasing non-essential degrees of freedom. Thus, in all likelihood, the observed increase in dimensionality has functional significance and is not simply a reflection of an increased noise level.

Further evidence for this interpretation was found in the analysis of the regularity of the COP trajectories. In the stroke patients, the postural sway was more regular in the frontal plane (i.e., lower sample entropy), whereas it was more regular in the sagittal plane in the healthy controls (Fig. 6). Furthermore, the stroke patients showed greater sway variability in the frontal plane, whereas the healthy controls showed greater sway variability in the sagittal plane (Fig. 6). Hence, the direction with the largest sway variability also showed the greatest regularity in the COP movements, suggesting that postural sway was more tightly controlled along this direction. Notice that the studied rehabilitation cohort was characterized by severe impairments in frontal plane balance (De Haart et al. 2004; see also Paillex and So 2005). Furthermore, Brown et al. (2002) provided evidence for increased attention demands for quiet standing in stroke patients. Possibly, therefore, stroke patients actively (i.e., by means of increased cognitive control) compensated for the loss of accurate sensory information from the paretic leg and for other stroke-mediated impairments hampering balance control. The observed increased regularity of the medio-lateral COP trajectories in the stroke patients could therefore reflect an elevated cognitive contribution to postural control (see also below). Moreover, this finding excludes the possibility that the accompanying increased dimensionality is an expression of increased noise, since that would have produced higher rather than lower sample entropy, as was the case for the shuffled and phase-randomized surrogates (Fig. 7). All in all, it is fair to conclude at this stage of the discussion that using dynamical measures had surplus value in assessing the effects of health status on postural control, although not all of those effects could be interpreted yet in a conclusive manner.

Rehabilitation

With follow-up assessments the COP variability decreased significantly (Fig. 4), as did the corresponding regularity in the frontal plane (i.e., ML sample entropy increased, Fig. 4). Initially, the postural sway was very large and fairly regular, whereas 3 months later it was smaller and markedly less regular, which is in partial agreement with the findings and theoretical perspective of Goldberger et al. (1996, 1997, 2002). The long-range correlations (i.e., scaling exponents) in the COP trajectories, however, did not change over the five follow-up assessments.

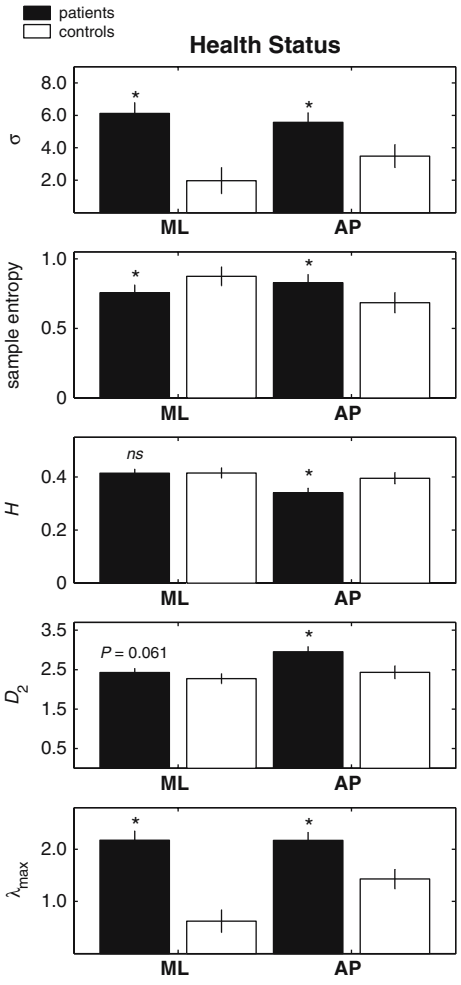

Fig. 6 *Health status* effects of ML and AP COP variability σ, sample entropy, Hurst exponent H, dimension estimate D_2, and largest Lyapunov exponent λ_{max}. The effect of *health status* is visualized by comparing stroke patients (*black bars*) and healthy controls (*white bars*). *Error bars* denote the 95% confidence intervals. *Stars* placed at the error bars denote significant differences between patients and controls (see Table 3 for F, P, and f values for *health status* effects)

The fact that the dimensionality of the COP trajectories was consistently higher for the stroke patients than for the healthy controls suggests that the reported differences in postural control persisted in time. However, in the course of rehabilitation, postural control improved in the stroke patients, as evidenced by increased local stability and decreased regularity. Both these novel findings are theoretically significant.

The observed increase in local stability is important in view of the suggestion of De Haart et al. (2004) that posture stabilized in the course of rehabilitation. By assessing postural stability directly by means of the largest Lyapunov exponent rather than by assuming that postural stability is inversely related to postural sway variability (which is not necessarily valid, cf. Newell et al. 1993), we demonstrated that postural stability increased in the course of rehabilitation. Thus, by reanalyzing the data using dynamical measures the proposition of De Haart et al. (2004) was confirmed empirically.

The observed decreased regularity with follow-up assessments is important in that it may reflect a reduction in the cognitive component directed to postural control in the course of rehabilitation, possibly due to improved multi-sensory integration and progressive internalization of altered body dynamics (see also Geurts et al. 2005). It could be speculated that, at the beginning of independent standing, stroke patients actively (i.e., cognitively) recruit additional control processes (e.g., additional strategies, co-contraction), while in the course of rehabilitation they learn to more automatically exploit the subspaces of controlled and uncontrolled variables. This possible trade-off between increased number of control processes and increased number of uncontrolled degrees of freedom can leave the observed dimensionality unchanged, despite marked changes in postural control. It should be noted in this context that after 3 months of follow-up, the frontal plane balance was still more regular and less stable in the stroke patients than in the healthy controls (Fig. 6), indicating that, congruent with the proposed relation between COP regularity and the cognitive contribution to postural control, the cognitive involvement in posture was still slightly elevated in the stroke patients.

Thus, in assessing the effects of rehabilitation, using dynamical measures was clearly beneficial in that they allowed for a confirmation of a tentative interpretation regarding the effect of recovery on postural stability, as well as a novel, albeit admittedly speculative, interpretation of the relationship between COP regularity and the cognitive regulation of posture. The cognitive dual task manipulation may provide a means to further assess this novel interpretation.

Task conditions

Without visual information, COP variability and λ_{max} increased significantly, while no difference in dimensionality was observed with or without vision. These findings agree with the study of Mégrot et al. (2002) on center-of-mass trajectories when standing on an unstable platform with eyes open and closed. In addition, sample entropy values did not differ with and without vision (Fig. 5), indicating that there was no change in postural sway regularity as a function of the availability of visual information. The scaling behavior indicated that successive data points were more negatively correlated with eyes closed than with eyes open. The corresponding increase in COP variability with eyes closed might have brought the postural control system close to its stability limits, which might have amplified the negative serial correlation between points in the COP time series in order to stay upright. It seems likely that other sensory systems (such as the vestibulum and muscle and joint receptors) may be facilitated to a greater extent in order

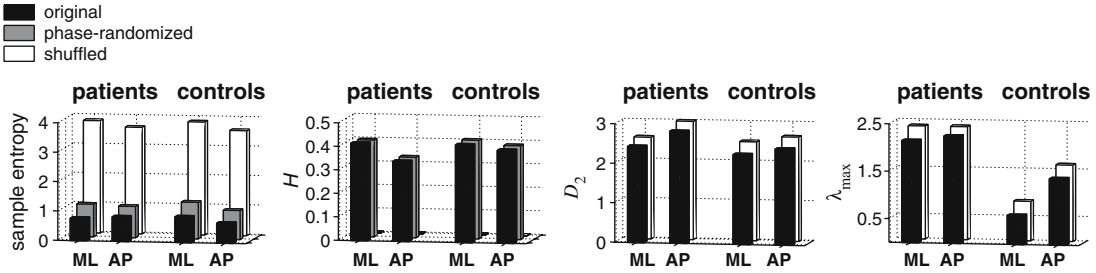

Fig. 7 *Randomization* effects for sample entropy, Hurst exponent H, dimension estimate D_2, and largest Lyapunov exponent λ_{max} depicted for both the stroke patients and the controls for ML and AP COP directions. Original COP data (*black bars*), phase-randomized (*gray bars*) and shuffled (*white bars*) surrogate data are depicted. Note that, apart from only H for original and phase-randomized surrogate data ($P > 0.05$), original, phase-randomized and shuffled surrogates all differed significantly from each other ($P < 0.005$). Note that all Hurst exponents vanished for shuffled surrogates, i.e., $H = 0$, whereas for D_2 and λ_{max} no shuffled surrogates values could be determined due to the $m > 2d_m + 1$ criterion (cf. Fig. 1, $C_m(r)$ scales with r^m)

to compensate for the visual deprivation. The fact that with eyes closed local stability strongly decreased indicates that vision plays a crucial role in modulating postural dynamics (e.g., Nienhuis et al. 2001; Mégrot et al. 2002; Geurts et al. 2005).

When the cognitive involvement in postural control was diminished by introducing an arithmetic dual task, the COP trajectories became less regular (sample entropy increased, see Fig. 5), which supports the relationship between postural sway regularity and cognitive contributions to postural control as proposed in the preceding: larger (smaller) cognitive involvement yields more (less) regular COP trajectories. De Haart et al. (2004) reported larger sway velocity related to a shift in COP median frequency in the DT condition without accompanying changes in the COP's spectral power (or variability). We observed increased dimensionality in the COP trajectories accompanied by less regularity when performing a dual task as opposed to standing with eyes open. It could be that the dual task led to a greater contribution or expression of noise to the COP dynamics (i.e., either direct or indirect). An increased noise level is traditionally viewed as detrimental for control, but the generality of this interpretation has recently been refuted by demonstrations of the beneficial effects of noise in sensori-motor control (e.g., Collins 1999; Cabrera et al. 2004; see also Wiesenfeld and Moss 1995; Thurner et al. 2002; Collins et al. 2003). Interestingly, under the DT condition, local stability did not differ from that in the EO condition, which suggests that the postural control system could have exploited noise in a like fashion.

In assessing the effects of sensory manipulations (i.e., EO versus EC conditions), the use of dynamical measures only yielded marginally novel findings: with increasing sensory difficulty stability diminished, as is well-known from previous studies using linear systems measures (e.g., Peterka 2002). In contrast, in assessing the effects of a cognitive dual task on postural control, the use of dynamical measures had surplus value as it led to the discovery of effects that were absent when using conventional posturographic measures. Moreover, those effects were fully consistent with the interpretation of the effects of health status and rehabilitation on postural control provided in the preceding: decreased cognitive involvement in postural control results in less regular COP trajectories.

Significance of studying postural sway dynamics

The present study was motivated from the expectation that, compared to the measures traditionally used in the study of COP trajectories, dynamical measures would have surplus value in studying postural control. We examined this expectation by reanalyzing an encompassing data set on the recovery of postural control following stroke, which not only included COP measurements while standing upright with eyes open, but also during sensory and cognitive manipulations. Notwithstanding the fact that the strength of statistical effects found for the dynamical measures were quite similar to those found for the standard deviation (see Tables 2, 3), this reanalysis led to several new important results and discoveries vis-à-vis the original analysis of the data using conventional posturographic measures. In particular, it was established that the data's variability was temporally structured, that postural stability increased during rehabilitation, and that postural sway regularity was positively related to the degree of cognitive involvement in postural control.

By combining the observed effects of health status, rehabilitation, and task conditions on the dynamical measures of interest, we arrived at a coherent theoretical interpretation that may be summarized in less scientific terms as follows. In stroke patients, maintaining balance is more difficult due to neuromuscular impairments, resulting in reduced postural stability. To cope with this reduced stability, postural control is actively (i.e., cognitively) increased, resulting in more regular yet higher dimensional COP trajectories. In the course of rehabilitation, postural stability improves, allowing the patients to relax their cognitive involvement in postural control, which leads to less regular COP trajectories. This process is reminiscent of that of automatization in skill

acquisition (cf. Huys and Beek 2002; Harbourne and Stergiou 2003; Huys et al. 2003, 2004; Milton et al. 2004). In line with this interpretation, the introduction of a cognitive dual task reduced the cognitive contribution to postural control, resulting in less regular COP trajectories of larger dimension but similar stability. Thus, by combining findings gathered from a complementary set of dynamics-related analyses under various task conditions and its recovery during rehabilitation after stroke, we could make readily interpretable inferences about (changes in) the underlying postural control. All in all, it is fair to conclude that the results of the present study supported our expectation that the use of dynamical measures would have surplus value in the analysis of COP trajectories. The implication of this overall conclusion is that, in future studies of postural control, it should be deemed worthwhile to incorporate both dynamical and conventional measures in the analysis of COP trajectories.

Acknowledgments This research was conducted while the first author was working on a grant of the Netherlands Organization for Health Research and Development (ZonMw grant 1435.0004).

References

Abarbanel HDI (1996) Analysis of observed chaotic data. Springer, New York

Baratto L, Morasso PG, Re C, Spada G (2002) A new look at posturographic analysis in the clinical context: sway-density versus other parameterization techniques. Motor Control 6:246–270

Bohannon RW (1995) Standing balance, lower extremity muscle strength, and walking performance of patients referred for physical therapy. Percept Mot Skills 80:379–385

Brown LA, Sleik RJ, Winder TR (2002) Attentional demands for static postural control after stroke. Arch Phys Med Rehabil 83:1732–1735

Brunnstrom S (1966) Motor testing procedures in hemiplegia: based on sequential recovery stages. Phys Ther 46:357–375

Buzzi UH, Stergiou N, Kurz MJ, Hageman PA, Heidel J (2003) Nonlinear dynamics indicates aging affects variability during gait. Clin Biomech 18:435–443

Cabrera JL, Bormann R, Eurich C, Ohira T, Milton J (2004) State-dependent noise and human balance control. Fluct Noise Lett 4:L107–L118

Cohen J (1988) Statistical power analysis for the behavioral sciences. Erlbaum, Hillsdale, NJ

Collen FM, Wade DT, Bradshaw CM (1990) Mobility after stroke: reliability of measures of impairment and disability. Int Disabil Stud 12:6–9

Collins JJ (1999) Fishing for function in noise. Nature 402:241–242

Collins JJ, De Luca CJ (1993) Open-loop and closed-loop control of posture: a random-walk analysis of center-of-pressure trajectories. Exp Brain Res 95:308–318

Collins JJ, De Luca CJ (1995) Upright, correlated random walks: a statistical-biomechanics approach to the human postural control system. Chaos 5:57–63

Collins JJ, Priplata AA, Gravelle DC, Niemi J Harry J, Lipsitz LA (2003) Noise-enhanced human sensorimotor function. IEEE Eng Med Biol Mag 22:76–83

De Haart M, Geurts AC, Huidekoper SC, Fasotti L, van Limbeek J (2004) Recovery of standing balance in postacute stroke patients: a rehabilitation cohort study. Arch Phys Med Rehabil 85:886–895

Delignières D, Deschamps T, Legros A, Caillou N (2003) A methodological note on nonlinear time series analysis: is the open- and closed-loop model of Collins and De Luca (1993) a statistical artifact? J Mot Behav 35:86–96

Frank TD, Daffertshofer A, Beek PJ (2001) Multivariate Ornstein-Uhlenbeck processes with mean-field dependent coefficients: application to postural sway. Phys Rev E 63:0011905/1–16

Fraser AM, Swinney HL (1986) Independent coordinates for strange attractors from mutual information. Phys Rev A 33:1134–1140

Fugl-Meyer AR, Jaasko L, Leyman I, Olsson S, Steglind S (1975) The post-stroke hemiplegic patient 1. A method for evaluation of physical performance. Scand J Rehabil Med 7:13–31

Geurts ACH, de Haart M, van Nes IJW, Duysens J (2005) A review of standing balance recovery from stroke. Gait Posture 22:267–281

Glass L (2001) Synchronization and rhythmic processes in physiology. Nature 410:277–284

Goldberger AL (1996) Non-linear dynamics for clinicians: chaos theory, fractals, and complexity at the bedside. Lancet 347:1312–1314

Goldberger AL (1997) Fractal variability versus pathological periodicity: complexity loss and stereotypy in disease. Perspect Biol Med 40:543–561

Goldberger AL, Amaral LAN, Glass L, Hausdorff JM, Ivanov PCh, Mark RG, Mietus JE, Moody GB, Peng C-K, Stanley HE (2000) PhysioBank, PhysioToolkit, and PhysioNet: components of a new research resource for complex physiologic signals. Circulation 101:e215–e220

Goldberger AL, Amaral LAN, Hausdorff JM, Ivanov PC, Peng C-K, Stanley HE (2002) Fractal dynamics in physiology: alterations with disease and aging. Proc Natl Acad Sci USA 99:2466–2472

Grassberger P (1986) Do climate attractors exist? Nature 323:609–612

Grassberger P, Procaccia I (1983) Characterization of strange attractors. Phys Rev Lett 50:346–349

Harbourne RT, Stergiou N (2003) Nonlinear analysis of the development of sitting postural control. Dev Psychobiol 42:368–377

Hurst HE (1965) Long-term storage: an experimental study. Constable, London

Huys R, Beek PJ (2002) The coupling between point-of-gaze and ball movements in three-ball cascade juggling: the effects of expertise, pattern and tempo. J Sports Sci 20:171–186

Huys R, Daffertshofer A, Beek PJ (2003) Learning to juggle: on the assembly of functional subsystems into a task-specific dynamical organization. Biol Cybern 88:302–318

Huys R, Daffertshofer A, Beek PJ (2004) Multiple time scales and multiform dynamics in learning to juggle. Motor Control 8:188–212

Kantz H, Schreiber T (2004) Nonlinear time series analysis. Cambridge University Press, Cambridge

Kaplan DT, Furman MI, Pincus SM, Ryan SM, Lipsitz LA, Goldberger AL (1991) Aging and complexity of cardiovascular dynamics. Biophys J 59:945–949

Kay BA (1988) The dimensionality of movement trajectories and the degrees of freedom problem: a tutorial. Hum Mov Sci 7:343–364

Kiemel T, Oie KS, Jeka JJ (2002) Multisensory fusion and the stochastic structure of postural sway. Biol Cybern 87:262–277

Kyriazis M (2003) Practical applications of chaos theory to the modulation of human ageing: nature prefers chaos to regularity. Biogerontology 4:75–90

Lake DE, Richman JS, Griffin MP, Moorman JR (2002) Sample entropy analysis of neonatal heart rate variability. Am J Physiol Regul Integr Comp Physiol 283:789–797

Lipsitz LA (2002) Dynamics of stability: the physiologic basis of functional health and frailty. J Gerontol Biol Sci 57A:B115–B125

Mandelbrot BB, van Ness JW (1968) Fractional Brownian motions, fractional noises and applications. SIAM Rev 10:422–437

Mégrot F, Bardy BG, Dietrich G (2002) Dimensionality and the dynamics of human unstable equilibrium. J Mot Behav 34:323–328

Milton JG, Small SS, Solodkin A (2004) On the road to automatic: dynamic aspects in the development of expertise. J Clin Neurophysiol 21:134–143

Newell KM, van Emmerik REA, Lee D, Sprague RL (1993) On postural stability and variability. Gait Posture 4:225–230

Newell KM, Slobounov SM, Slobounova ES, Molenaar PCM (1997) Stochastic processes in center-of-pressure profiles. Exp Brain Res 113:158–164

Newell KM (1998) Degrees of freedom and the development of center of pressure profiles. In: Newell KM, Molenaar PCM (eds) Applications of nonlinear dynamics to developmental process modeling. Erlbaum, Hillsdale, NJ, pp 63–84

Nienhuis B, Geurts AC, Duysens J (2001) Are elderly more dependent on visual information and cognitive guidance in the control of upright balance? In: Duysens J, Smits-Engelsman BC, Kingma H (eds) Control of posture and gait. NPI, Maastricht, pp 585–588

Packard NH, Crutchfield JP, Farmer JD, Shaw RS (1980) Geometry from time series. Phys Rev Lett 45:712–716

Paillex R, So A (2005) Changes in the standing posture of stroke patients during rehabilitation. Gait Posture 21:403–409

Pascolo PB, Marini A, Carniel R, Barazza F (2005) Posture as a chaotic system and an application to the Parkinson's disease. Chaos Solitons Fractals 24:1343–1346

Peng C-K, Buldyrev SV, Havlin S, Simons M, Stanley HE, Goldberger AL (1994) Mosaic organization of DNA nucleotides. Phys Rev E 49:1685–1689

Peng C-K, Havlin S, Stanley HE, Goldberger AL (1995) Quantification of scaling exponents and crossover phenomena in nonstationary heartbeat time series. Chaos 5:82–87

Peterka RJ (2002) Sensorimotor integration in human postural control. J Neurophysiol 88:1097–1118

Pincus SM, Goldberger AL (1994) Physiological time-series analysis: what does regularity quantify? Am J Physiol Heart Circ Physiol 266:H1643–H1656

Pincus SM (1991) Approximate entropy as a measure of system complexity. Proc Natl Acad Sci USA 88:2297–2301

Rangarajan G, Ding M (2000) Integrated approach to the assessment of long-range correlation in time series data. Phys Rev E 61:4991–5001

Raymakers JA, Samson MM, Verhaar HJJ (2005) The assessment of body sway and the choice of the stability parameter(s). Gait Posture 21:48–58

Richman JS, Moorman JR (2000) Physiological time-series analysis using approximate entropy and sample entropy. Am J Physiol Heart Circ Physiol 278:H2039–H2049

Riley MA, Balasubramaniam R, Turvey MT (1999) Recurrence quantification analysis of postural fluctuations. Gait Posture 9:65–78

Riley MA, Turvey MT (2002) Variability of determinism in motor behavior. J Mot Behav 34:99–125

Rosenstein MT, Collins JJ, De Luca CJ (1993) A practical method for calculating largest Lyapunov exponents from small data sets. Phys D 65:117–134

Scholz JP, Schöner G (1999) The uncontrolled manifold concept: identifying control variables for a functional task. Exp Brain Res 135:382–404

Schöner G (1995) Recent developments and problems in human movement science and their conceptual implications. Ecol Psychol 7:291–314

Takens F (1981) Detecting strange attractors in turbulence. In: Rand DA, Young LS (eds) Dynamical systems and turbulence. Springer, Berlin

Theiler J (1986) Spurious dimension from correlation algorithms applied to limited time-series data. Phys Rev A 34:2427–2432

Theiler J, Eubank S, Longtin A, Galdrikian B, Farmer JD (1992) Testing for nonlinearity in time series: the method of surrogate data. Phys D 58:77–94

Theiler J, Rapp PE (1996) Re-examination of the evidence for low-dimensional, nonlinear structure in the human electroencephalogram. Electroencephalogr Clin Neurophysiol 98:213–222

Thurner S, Mittermaier C, Ehrenberger K (2002) Change of complexity patterns in human posture during aging. Audiol Neurootol 7:240–248

Wade DT (1992) Measurement in neurological rehabilitation. Oxford University Press, Oxford

Wiesenfeld K, Moss F (1995) Stochastic resonance and the benefits of noise: from ice ages to crayfish and SQUIDs. Nature 373:33–36

Wing A, Daffertshofer A, Pressing J (2004) Multiple time scales in serial production of force: a tutorial on power spectral analysis of motor variability. Hum Mov Sci 23:569–590

Wolf A, Swift JB, Swinney HL, Vastano JA (1985) Determining Lyapunov exponents from a time series. Phys D 16:285–317

Yamada N (1995) Chaotic swaying of the upright posture. Hum Mov Sci 14:711–726

Zatsiorsky VM, Duarte M (1999) Instant equilibrium point and its migration in standing tasks: rambling and trembling components of the stabilogram. Motor Control 3:28–38