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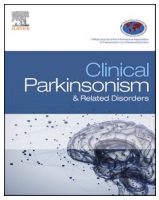

Spatial variability and directional shifts in postural control in Parkinson's disease

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ABSTRACT

Individuals with Parkinson's disease exhibit tremors, rigidity, and bradykinesia, disrupting normal movement variability and resulting in postural instability. This comprehensive study aimed to investigate the link between the temporal structure of postural sway variability and Parkinsonism by analyzing multiple datasets from young and older adults, including individuals with Parkinson's disease, across various task conditions. We used the Oriented Fractal Scaling Component Analysis (OFSCA), which identifies minimal and maximal long-range correlations within the center of pressure time series, allowing for detecting directional changes in postural sway variability. The objective was to uncover the primary directions along which individuals exerted control during the posture. The results, as anticipated, revealed that healthy adults predominantly exerted control along two orthogonal directions, closely aligned with the anteroposterior (AP) and mediolateral (ML) axes. In stark contrast, older adults and individuals with Parkinson's disease exhibited control along suborthogonal directions that notably diverged from the AP and ML axes. While older adults and those with Parkinson's disease demonstrated a similar reduction in the angle between these two control directions compared to healthy older adults, their reliance on this suborthogonal angle concerning endogenous fractal correlations exhibited significant differences from the healthy aging cohort. Importantly, individuals with Parkinson's disease did not manifest the sensitivity to destabilizing task settings observed in their healthy counterparts, affirming the distinction between Parkinson's disease and healthy aging.

1. Introduction

The typical manifestations of Parkinson's disease, including resting tremors, rigidity, and bradykinesia [16,97,109,121], all relate to variability. In support of this observation, the optimal movement variability hypothesis proposes that a robust and flexible postural control system exhibits a consistent yet intricate temporal structure in postural sway [42,127,126]. However, as individuals age or experience neurological disorders, their postural system loses its flexibility, resulting in a compromised state characterized by either persistent variations in postural sway, leading to rigid and highly predictable behavior, or irregular and random variations, resulting in erratic and unfocused behavior [26,46,122]. Notably, individuals with Parkinson's disease exhibit a highly deterministic structure in their postural sway variability compared to healthy individuals, indicating rigid and highly predictable behavior [84,93,112]. Consequently, individuals with Parkinson's disease face specific postural challenges, particularly in orientation (i.e., maintaining proper alignment with gravity) and stabilization, as well as balancing against external forces [12,63,131,130]. These difficulties arise from the inflexibility caused by the disease. The consequences of this inflexibility are substantial, as individuals with Parkinson's disease are at an increased risk of falls and injuries, significantly impeding their independence and mobility [1,41,92]. Therefore, it is crucial to explore the spatiotemporal structure of postural sway variability to identify a posture-based biomarker that can diagnose Parkinson's disease, track its progression, and assess the effectiveness of interventions. By understanding this aspect better, we can enhance the management and treatment of Parkinson's disease, ultimately improving the quality of life

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for affected individuals.

The healthy human postural center of pressure (CoP) exhibits variations from one sample to another that typically resemble fractional Brownian motion (fBm) [15,73]. In other words, these variations reflect a stochastic process characterized by long-range dependence and non-constant variance, exhibiting self-similarity and non-Markovian behavior. This long-range dependence manifests as a power-law in the autocorrelation (or equivalently, in the growth of standard deviation with time scale) with a power-law named *H* in memory of Harold Hurst [47]. We can use the variation in *H* to operationalize variation in the long-range dependence. Typically, the CoP is projected in orthogonal directions along the measuring force platform's anteroposterior (AP) and mediolateral (ML) directions. This projection assumes that postural control primarily occurs in these two directions, drawing inspiration from the inverted pendulum model [70–72]. Healthy posture exhibits fractal scaling patterns: self-similar structures with repeating patterns at different scales or magnifications [48]. Also, postural control modes may rely on an asymmetric distribution of fractal scaling along these two axes. For instance, in some cases, stronger fractal scaling is observed along the ML axis, while the AP axis exhibits weaker scaling [4,13,73]. In other cases, there may be less within-participant difference between the AP and ML axes. Instead, there may sooner be a group difference in fractal temporal correlations along one or another axis—for example, AP rather than ML; Duarte and Sternad [31].

The recognition of fractal temporal correlations in postural sway is an exciting development in movement science and motor control that has raised more questions than it has answered. Despite high-level conceptual comparisons between "temporal correlations" in fractal patterning and "sensory corrections" in terms of dexterous motor control, the relationship between fractal temporal correlations and traditional diagnoses or measures of stability has been highly ambiguous [4,30,33,36,64,77,94,98,104]. We can identify two significant obstacles to clarity. First, there has been little contact between the fractal modeling of posture and the theoretical models of motor control. Explanations of fractal-type patterns in movement variability vary widely. While some theories present laundry lists of physiological perturbations interacting across multiple timescales [20,32]. others appeal to the multitude of potentially independent contributing factors [27], including noise and delay implicit in physiology [19]. Furthermore, there are explanations attributing the collapsing variation of fractal temporal correlations to engaging feedback processes in response to independent task features [110,116–119,124]. Hence, explanations reflect a yet undifferentiated sense that fractal temporal correlations could reflect either task constraints, physiological adaptivity, or both—and some of these appeals risk conflating both ends of this spectrum of possible causes, for example, suggesting that task-dependent variation of fractal temporal correlations is a small-scale approximation of more global, clinical deficits in health or motor coordination (e.g., the "Conclusions" in Slifkin and Eder [119]). There is undoubtedly value in trying to infer from experimental results how destabilizing task settings might prompt healthy adults to generate fractal signals comparable to adults with a debilitating disease. However, there are diminishing returns from such comparison if we have somehow to load the variation of the single dimension "fractal temporal correlations" with fleeting task constraints and persisting, life-changing diagnosis. We see the rich potential value of drawing analogies about adaptivity [54], but no single one-dimensional observable should have to operationalize such a broad class of constructs.

The second obstacle to clarity about a judicious empirical accounting of how fractal temporal correlations matter to postural control is the traditional AP-vs.-ML framing of CoP. Of course, having just noted how heavily multiple constructs might hang upon a single 1D observable, it is fortunate that we have two dimensions on the support surface. Indeed, we have already seen that we might distinguish groups (e.g., old vs. young) on one or another of the axes of the force plate [31]. So, two orthogonal dimensions open the modeling space where we might hunt for more apparent dissociable coverage of task-sensitivity constructs or disease constructs. The axes of the force plate have resonated neatly with anatomical conventions for explaining upright posture as an inverted pendulum [70–72]. We can find empirical acknowledgments that the postural systems can flexibly reorient across these two axes, releasing postural variability in task-sensitive directions by turn [8]. However, the stricture of two orthogonal axes is already too diminishing of natural variability, for example, for statistical models to portray effects of different athletic training regimens on postural control in healthy upright bodies [37]. The fundamental nonlinearity implicit in CoP or other postural sway signals suggests a cascading variation of CoP across the 2D support surface that cannot be reduced to the sum of orthogonal components [35,59,58,87,90]. Ultimately, the pragmatic AP-vs.-ML framing of CoP has left motor control research with little accounting of time-varying heterogeneity in postural sway across multiple directions—even from nonlinear-type perspectives about intermittent motor control Cluff et al. [23], postural sway could embody more angles than two and along axes that may not be orthogonal. Maintaining postural balance requires continuous and comprehensive sensorimotor integration, allowing for orientations that extend beyond using only one specific direction. Therefore, presenting stability parameters exclusively along the AP and ML directions can give the impression that these directions are universally relevant to every individual when they might not be [40,123].

This manuscript aims to resolve both obstacles, using a novel empirical formalism to estimate features of a long-standing control theory in 2D CoP trajectory. In what follows, we first identify a blend of topological features in a class of theories on intermittent postural control. Next, we describe a novel analytical method that allows us to test whether fractal temporal correlations embody those topological features.

1.1. Resolving the first obstacle: Rephrasing intermittent control strategies as 2D distributions of fractal temporal correlations across the support surface

The impetus for a novel treatment of postural control generalizing beyond two anatomical and orthogonal axes may have, curiously, been in plain sight for a while—not hiding so much as the intuitions about orthogonal axes have eclipsed it. For instance, theories of intermittent control that promise to generate fractal temporal correlations appeal to non-orthogonal control principles [6,7,96]. Specifically, these theories highlight a capacity for control to switch between saddle-type and spiral-type control, two qualitatively different topologies. Specifically, task orientation can cultivate postural synergies that compress the potentially 2D sway patterns on the support surface to relatively 1D directions. Indeed, directing the attention forward or to the side can shift postural control to release most of its sway in the AP or the ML direction [8]. This predominantly unidirectional sway suggests saddle-like control (Fig. 1*a*), emphasizing stability in one direction while introducing instability in the orthogonal one. Shifting attention from the front to the side, or vice versa, cannot occur instantaneously; instead, it necessitates postural control accessing a spiral-type mode (Fig. 1*b*). Therefore, the saddle-type control must transition to a spiral-type control, relinquishing the relatively one-dimensional constraint to facilitate a two-dimensional spreading of sway, ultimately anchoring in a new one-dimensional task orientation. The conventional observation depicts postural sway adopting a saddle-type arrangement with stronger temporal correlations in one direction compared to the orthogonal one [4,13,73]. While fractal temporal correlations often support postural control, whether these correlations can manifest a spiral-type topology in sway remains unanswered.

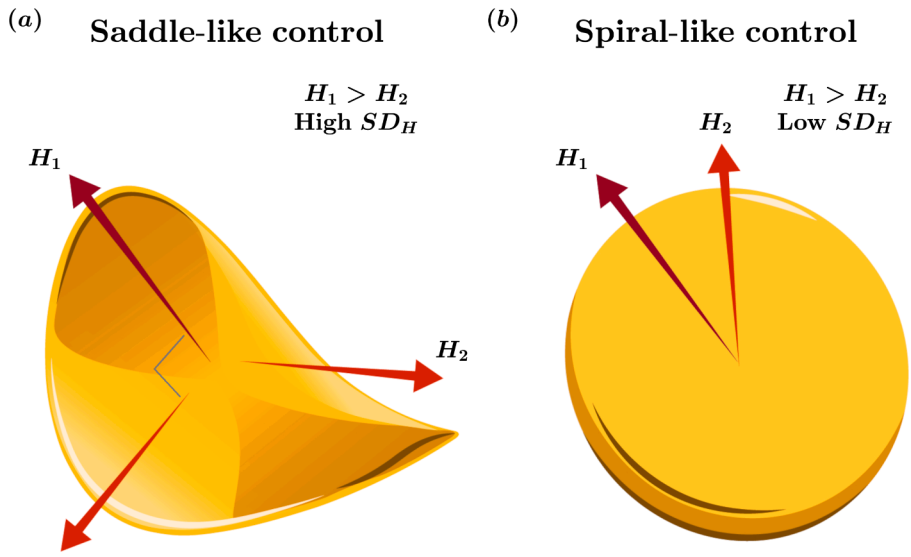

Fig. 1. Schematic illustration of the saddle- and spiral-like strategies for controlling posture along the 2D support surface. (*a*) A stable equilibrium could emerge when examining fractal temporal correlations predominantly in either the AP or ML axis but rarely in both, leading to a saddle-type control. For instance, heightened fractal scaling (i.e., large Hurst exponent H_1) may indicate strong temporal correlations in postural sway along the AP direction, coupled with a reduced scaling exponent along the orthogonal ML direction (i.e., $H_2 < H_1$). This strategy could implicate more proactive control strategies along the AP axis and passive strategies along the ML axis with large spatial variability in fractal temporal correlations (i.e., high SD_H). (*b*) Alternatively, releasing the postural synergy constrained across the AP or ML axes in postural control might, for example, under perturbations or changes in orientation, disengage saddle-type control and pave the way for a transition to 2D spiral-type behavior, accompanied by relatively smaller spatial variability in fractal temporal correlations (i.e., low SD_H).

1.2. Resolving the second obstacle: Estimating 2D distribution of fractal temporal correlations without necessarily orthogonal axes

A significant challenge for diagnosing spiral-type topology in intermittent complement to saddle-type topology is that the latter implies that a postural sway might depart from either canonical axis. No matter the fixity of the AP and ML axes of the force platform at angles $\theta = 90^{o}$ and $\theta = 0^{o}$ the directions of the strongest and weakest fractal scaling could now and then fall along axes separated by an angle $\Delta\theta < 90^{o}$. We might only see differences between groups in one or another (i.e., AP or ML) axis. For instance, in quiet standing, older adults show weaker temporal correlations (i.e., lower *H*) along the AP direction than young people do—without any other change between AP and ML [31]. Assuming the AP-vs.-ML axes are given, one might infer that older adults lose fractal temporal correlations in sway, unlike younger adults. While not inherently incorrect, this perspective overlooks the potential that prolonged strain on upright posture in older adults could prompt redirection of fractal temporal correlations in a different, non-orthogonal direction. These two possibilities reflect radically different expectations about the resources of a fractal-themed postural control. In the former case, postural control might increase or decrease its fractal temporal correlations (e.g., a global *H*) across the total of the orthogonal AP and ML axes (i.e., $H_{AP} + H_{ML}$). In the latter case, postural control might steer its fractal temporal correlations elsewhere (i.e., with a maximum H_{θ_1} or minimum H_{θ_2}) in other directions θ_1 and θ_2. These two options are not mutually exclusive. Postural control may enhance the total and average fractal temporal correlations along diverse non-orthogonal paths. The range of possibilities is extensive.

Rather than guessing what postural control could lie between the orthogonal axes, we can estimate the complete angular variety of fractal scaling. This possibility comes from a novel analysis from theoretical physics called the oriented fractal scaling component analysis (OFSCA) [113]. The OFSCA allows modeling a 2D random process by estimating fractal temporal correlations *H* along all directions from its midpoint, that is, from all angles $0^{o} \leqslant \theta < 180^{o}$. Thus, rather than projecting CoP along only AP and ML axes, the OFSCA estimates the fractal temporal correlations in CoP projections along all possible axes, that is, H_{θ_i} for each *i*th value of θ. It then identifies the angular position of the two axes with extremal values of *H*, that is, identifying a "major" axis and a "minor" axis located at the angles θ_1 and θ_2, respectively, along which the Hurst exponents H_1 and H_2 for the projection of CoP is maximal and minimal, respectively, relative to all other angles.

This new perspective on the potential suborthogonality of postural control may afford more rigorous falsifiability to vague proposals implicating fractal temporal correlations. Instead of simply presuming a saddle-type topology in which the maximum and minimum axial Hurst exponents H_1 and H_2 distribute such that $\theta_1 - \theta_2 = \Delta\theta = 90^{o}$, the OFSCA can identify axes with H_1 and H_2 separated by $\Delta\theta < 90^{o}$. Theoretically and now empirically [89,91], we have begun to build a case that narrower $\Delta\theta$ might correspond to the spiral-type topology that orthogonal axes had been inappropriate for showcasing. The previous uses of OFSCA on CoP fluctuations have shown, in sum, the following points. (i) Quiet standing in younger adults shows the classic orthogonal arrangement of H_1 and H_2, with healthy older adults showing reliably narrower angles between these major and minor axes, that is, $\Delta\theta < 90$. (ii) Experimental perturbations to stable posture (e.g., standing on foam or turning head) reduce $\Delta\theta$ below the pre-existing orthogonal or sub-orthogonal setting characteristic of healthy old and young participants. Still, there is no evidence that these perturbation-prompted narrowing of $\Delta\theta$ persist beyond the perturbation. (iii) The narrowing of $\Delta\theta$ corresponds to, on average, greater homogeneity in the angular variety of *H* in terms of its standard deviation SD_H. Hence, the wider or narrower angle $\Delta\theta$ corresponds to postural control potentially fanning out a more uneven or even distribution of fractal temporal correlations along most directions. Whereas we proposed wider $\Delta\theta$ to correspond to greater angular variability of *H* as suggested by the saddle-type topology, angular homogenization associated with narrower $\Delta\theta$ might correspond to the disc-like form of a spiral-type control regime. However, fractal temporal correlations do not appear to settle into an entirely flat spiral: despite the direct relationship between greater $\Delta\theta$ and greater SD_H, there is a tendency for the range of *H* (i.e., $H_1 - H_2$) to vary inversely with $\Delta\theta$. That is, change in $\Delta\theta$ seems to co-occur with the distribution of *H* values. Higher $\Delta\theta$ corresponds to a relatively wider distribution with shorter tails (e.g., more like a *t*-distribution than Normal distribution), and lower $\Delta\theta$ corresponds to a relatively narrow distribution with longer tails (e.g., more like *Normal*-distribution). Thus, as $\Delta\theta$ narrows, the

major and minor axes along θ_1 and θ_2 grow closer together in angular space but exhibit greater differences in Hurst $H_1 - H_2$ within this narrower angular region. However, there is a more homogeneous distribution on average across the wider remainder of the entire support surface.

We might integrate the foregoing observations into a proposal about intermittent postural control switching between saddle-type and spiral-type distributions of fractal temporal correlations across the support surface. Specifically, it may be that stable posture without perturbation primarily employs saddle-type distribution of fractal temporal correlations with the orthogonal orientation of most and least H. This saddle-type distribution would entail the greater average variability (e.g., SD_H) across angle, for example, because of the saddle's depressions of H at θ_1 roughly 90° from θ_2. The task-dependent perturbations may prompt postural control to switch from saddle-type to spiral-type, wherein the spiral's circular disk-like shape manifests in a more homogeneous dispersion of H outside of the narrower $\Delta\theta$ interval between the axes. Given the minimal influence of the vestibular relative to visual and proprioceptive information on posture [34,44,50,100,133], it is striking that subtle vestibular perturbation might produce this angular homogenization around the support surface. This effect might manifest both in the experimental manipulation of head orientation in the trained gymnasts with dexterous postural control [91] and in the healthy older adults [89] known to exhibit vestibular degeneration [5,51,81]. In both cases, the vestibular perturbation prompts the postural system to release its postural synergy from the saddle-type, distributing stronger fractal scaling (i.e., H_1) along the one-dimensional crest. Then, potentially searching for more stable task orientation [8], postural control distributes fractal temporal correlations into relatively more 2D form, into flatter, more circular and homogeneous spiral-type structures. Looser constraint on posture by vestibular information due to aging might entail greater sensitivity of $\Delta\theta$. The expected group difference in (i.e., reduction of) $\Delta\theta$ for healthy older adults might thus come with stronger dependence of $\Delta\theta$ both on the endogenous fractal temporal correlations (i.e., SD_H,H_1, and H_2) and task effects (i.e., closing eyes or destabilizing perturbations).

Across tasks and groups, we see a potential way to dissociate fleeting task effects from more stable group differences in the fractal-based control of posture. This point is essential for not conflating markers of adaptive response to task with markers of diseased postural control. It has been perfectly valid for fractal-themed rhetoric to find commonalities between the short-term perturbations of motor control due to tasks from persistent losses of functionality [38,39,76,78,119]. Indeed, a perturbed healthy posture could sometimes look like a more systematically diseased posture. The fractal scaling of postural sway can itself depend on task factors such as static vs. dynamic stance [14,69] and the participant's attentional state [23,28]. However, interest in disease biomarkers must not conflate postural responses that could be entirely adaptive in the short term with systematic group differences in the long term. For instance, the comparable $\Delta\theta$ reduction between perturbed gymnasts and quietly standing healthy older adults should not entail that older adults are more dexterous than trained gymnasts [91]. Excitement over the innovation of new fractal methods notwithstanding, it is essential to learn how to probe for nuanced patterns within relatively short signals. The efficacy of such methods for the diagnosis will hinge on our ability, within short-term measurements, to distill short-term task effects from clinically relevant, chronic, or systematic group effects [61]. Task and context variations could result in false positives when diagnosing Parkinson's disease using a biomarker derived from postural sway variability. Consequently, the biomarker we are currently exploring may not possess the specificity required to be a clinically helpful approach.

To adequately develop any diagnostic promise of the OFSCA, it is crucial to evaluate the impact of tasks and contextual factors on postural sway variability in groups of people with and without diseases like Parkinson's disease. The co-occurrence of a difference in $\Delta\theta$ between young and older adults with differing task effects on $\Delta\theta$ in each group is encouraging [89]. It suggests a step toward distinguishing short-term from long-term differences. However, a pending drawback in prior evidence of specificity to disease rather than the task is the absence of task effects on $\Delta\theta$ in older adults with Parkinson's disease. Here, we may add the fourth and final finding from previous research into OFSCA modeling of posture: Adults with Parkinson's disease exhibit a dramatic reduction of $\Delta\theta$ that has shown no task sensitivity to perturbations. We have estimated a group effect of healthy aging on $\Delta\theta$ and a task-dependent effect on $\Delta\theta$ in healthy older adults. However, the failure to find a task-dependent effect on $\Delta\theta$ of experimental perturbations in posture of older adults with Parkinson's disease is so far puzzling. It might mean that they show no sensitivity to tasks and that potentially their posture is permanently locked into the spiral-type control, permanently seeking a task orientation. However, complete task insensitivity seems unrealistic because individuals with Parkinson's disease show task effects in postural control through other analytical lenses [12,63,84,93,112,131,130].

1.3. The present reanalysis

The present work aimed to explain the reduced $\Delta\theta$ in adults with Parkinson's disease [89] and distill previously undetermined task effects on $\Delta\theta$ beyond simply group differences with diagnosis. More specifically, it aimed to resolve new nuance from our previous finding using three novel steps: (i) a wider variety of similarly destabilizing task manipulations, (ii) addressing the theoretical question of how well endogenous fractal fluctuations embody known topologies in intermittent postural control, and (iii) addressing clinical specificity to Parkinson's disease by pooling data from additional healthy controls and individuals with an alternate diagnosis of Stargardt's syndrome. Previous examinations of postural and suprapostural dexterity have widely shown two classes of interactions: group-by-task-effect interactions and group-by-endogenous-fractal-estimate interactions [17,11,52,57,59,58]. So, we expected that we could generalize usefully across slightly different variants of the same manipulations. We expected closing eyes and destabilizing quiet standing to create a more nuanced and principled contrast of these factors between Parkinson's disease and healthy aging.

1.3.1. Task effects

Although closing eyes was standard across most of the study protocols across the datasets, as described below, the perturbations of posture included standing on foam (rather than rigid) surfaces, tracking moving (rather than still) visual targets, and holding a tube filled with water (rather than sand). We coded these perturbations identically for comparison across the five datasets. This aggregation of task effects should clarify the task effects on $\Delta\theta$ for subsequent regression modeling of the OFSCA results. Testing task effects in a wider sample of participants without diagnosis might bring any task effects more clearly into relief. The regression modeling tested two classes of interactions: group membership with task parameters and group membership with endogenous fractal temporal correlations.

1.3.2. Effects of endogenous fractal temporal correlations

The foregoing proposals raise specific predictions about how changes in $\Delta\theta$ could operationalize intermittent reversals between the saddle- and spiral-type topologies of postural control explicitly in the fractal temporal correlations in sway [91]. Conceptually, we expected that clarifying the topologies of fractal temporal correlations implicit in intermittent postural control might allow us to partial out variation of $\Delta\theta$ and make the task effects more straightforward for each group.

1.3.3. Greater specificity about the effects of Parkinson's disease rather than another diagnosis on group effects as well as task-sensitive control

This reanalysis aimed to determine how specific the above-described

changes in $\Delta\theta$ might be due to Parkinson's disease rather than other diseased postures. It pools the Parkinson's disease dataset originally modeled using the OFSCA with several other datasets (Table 1) documenting various task effects on healthy young and older adults and young adults with Stargardt's syndrome—a visual disorder that can influence posture [111]. More specifically, we expected that modeling $\Delta\theta$ in terms of the interactions of diagnostic groups (i.e., Parkinson's disease, healthy aging, and Stargardt's syndrome) with the endogenous fractal parameters (i.e., H_1,H_2, and SD_H) might clarify the interactions of these diagnostic groups with task manipulations (i.e., closing eyes or overtly perturbing posture).

These interactions hold immense theoretical impact for understanding postural control with Parkinson's that would inform any clinical use of the angular or fractal biomarkers. Specifically, these contrasts allow posing specific questions about how well the endogenous fractal temporal correlations in individuals with Parkinson's disease embody the intermittent switching between saddle-type or spiral-type topologies. It is at least logically plausible that Parkinson's disease might manifest as a form of "advanced aging," predicting simply an accentuation of the effects on $\Delta\theta$ for older participants. This expectation would be at odds with the previous failure to find any task-sensitive change in $\Delta\theta$ in postural control with Parkinson's disease. If this reanalysis finds any task-sensitive changes in $\Delta\theta$ in individuals with Parkinson's disease, these changes should appear much weaker than in the case of healthy older participants. So, the alternative outcome here—and potential explanation for the reduced task-sensitive changes—may be that postural control with Parkinson's diasease fails to mold its endogenous fractal temporal correlations into the known adaptive topologies of intermittent control. Hence, two clear—and non-exclusive—possibilities for explaining the narrower $\Delta\theta$ are the following: participants with Parkinson's disease fail to show the perhaps adaptive task-responsive narrowing of $\Delta\theta$ that healthy adults (with and without gymnastic training) showed in Mangalam et al. [91] and participants with Parkinson's disease may also fail to distribute greater angular variability (i.e., higher H_{SD}) into the saddle-type arrangement of fractal temporal correlations with wider $\Delta\theta$.

Table 1
Participant groups and task manipulations submitted to the OFSCA, and the respective $M \pm SD$ values of $\Delta\theta$ yielded by the OFSCA.

Study	Population characteristics	Task manipulations	$\Delta\theta$
de Oliveira et al. [25]	Individuals with Parkinson's disease	Eyes, open, rigid surface	68.9 ± 18.1
		Eyes open, foam surface	63.2 ± 20.0
		Eyes closed, rigid surface	62.8 ± 20.2
		Eyes closed, foam surface	58.3 ± 20.6
dos Santos et al. [29]	Healthy young adults	Eyes open, rigid surface	85.9 ± 11.5
		Eyes open, foam surface	81.8 ± 10.5
		Eyes closed, rigid surface	85.2 ± 9.6
		Eyes closed, foam surface	75.3 ± 12.5
	Healthy older adults	Eyes open, rigid surface	81.3 ± 10.4
		Eyes open, foam surface	66.9 ± 14.0
		Eyes closed, rigid surface	77.0 ± 12.0
		Eyes closed, foam surface	66.3 ± 16.8
Sbrollini et al. [111]	Healthy young adults	Eyes closed	99.5 ± 15.6
		Eyes open, still target fixation	96.3 ± 13.2
		Eyes open, moving target tracking	98.4 ± 14.3
	Individuals with Stargardt's syndrome	Eyes closed	97.8 ± 13.5
		Eyes open, still target fixation	99.1 ± 14.6
		Eyes open, moving target tracking	96.4 ± 15.2
Lee et al. [75]	Healthy young adults	Eyes closed	88.4 ± 6.0
		Eyes open, fixated at 25 cm	89.4 ± 3.9
		Eyes open, fixated at 50 cm	90.7 ± 3.6
		Eyes open, fixated at 135 cm	88.9 ± 5.0
		Eyes open, fixated at 220 cm	90.3 ± 3.5
		Eyes open, fixated at 305 cm	89.6 ± 5.8
Furmanek et al. [35]	Healthy young adults	Balance a sand-filled tube	89.0 ± 19.0
		Balance a water-filled tube	100.6 ± 18.5

1.4. Specific predictions

We submitted the CoP trajectories from all of these datasets to the OFSCA. We expected that modeling the endogenous fractal estimates would allow statistical prediction of variation of $\Delta\theta$ due to intrinsic features of the topology of postural control strategies. We also expected that the task effects on $\Delta\theta$ for each group would appear more clearly in the model alongside the encoding of endogenous fractal temporal correlations. We made the following predictions that followed two main patterns across all groups: first, we predicted that stable posture would exhibit greater $\Delta\theta$ corresponding to the saddle-type control, and second, we predicted that perturbing posture would narrow whether with foam support surface, moving visual target, or fluid loading of the upper extremities. We anticipated the following:

- Healthy young adults would exhibit nonzero $\Delta\theta$ in quiet, unperturbed standing (Hypothesis 1a), suggesting the saddle-type control according to which $\Delta\theta$ might increase along with the endogenous fractal temporal correlations such as greater SD_H (Hypothesis 1b), lesser H_1 (Hypothesis 1c), and greater H_2 (Hypothesis 1d).
- Healthy young adults would exhibit narrower $\Delta\theta$ with eyes closed (Hypothesis 1e) and with the destabilizing perturbations (e.g., standing on the foam surface, tracking a moving visual target, and holding a liquid load; Hypothesis 1f).
- Healthy older adults would exhibit reductions in $\Delta\theta$ in quiet, unperturbed standing (Hypothesis 2a), suggesting the saddle-type control according to which $\Delta\theta$ might increase according to an interaction of healthy aging with the endogenous fractal temporal correlations such as greater SD_H (Hypothesis 2b), lesser H_1 (Hypothesis 2c), and greater H_2 (Hypothesis 2d).
- Healthy older adults would exhibit narrower $\Delta\theta$ according to interactions between healthy aging with eyes closed (Hypothesis 2e) and with postural-destabilizing perturbations (e.g., standing on the foam surface, tracking a moving visual target, and holding a liquid load; Hypothesis 2f).
- Young adults with Stargardt's syndrome would exhibit reductions in $\Delta\theta$ in quiet, unperturbed standing (Hypothesis 3a), suggesting the saddle-type control according to which $\Delta\theta$ might increase according to an interaction of Stargardt's syndrome with the endogenous fractal temporal correlations such as greater SD_H (Hypothesis 3b), lesser H_1 (Hypothesis 3c), and greater H_2 (Hypothesis 3d). Then again, it is also possible that Stargardt's syndrome might show reversals or attenuations of these changes in $\Delta\theta$ with these fractal effects, particularly if Stardgardt's syndrome does not resemble the loss of dexterity with aging (Hypothesis 3e).

- Young adults with Stargardt's syndrome would exhibit narrower $\Delta\theta$ according to interactions between Stargardt's syndrome with eyes closed (Hypothesis 3f) and with the destabilizing perturbation (e.g., tracking a moving visual target; Hypothesis 3g). Then again, as above, it is also possible that Stardgardt's syndrome might show reversals or attenuations of these changes in $\Delta\theta$ with task effect, particularly if Stardgardt's syndrome does not resemble the loss of dexterity with aging (Hypothesis 3h).
- Adults with Parkinson's diagnosis would exhibit reductions in $\Delta\theta$ in quiet, unperturbed standing (Hypothesis 4a). Suppose this effect was due simply to the status of Parkinson's disease as somehow accelerating the aging process. In that case, we should see the following accentuation of effects from Hypotheses 2b–d: saddle-type control according to which $\Delta\theta$ might increase according to an interaction of Parkinson's with the endogenous fractal temporal correlations such as yet greater SD_H (Hypothesis 4b), yet lesser H_1 (Hypothesis 4c), and yet greater H_2 (Hypothesis 4d) than we find in the healthy older participants. An important caveat here is that if Parkinson's is not simply accelerated aging, we may see individuals with Parkinson's disease show reversals of Hypotheses 2b–d (Hypothesis 4e).
- Adults with Parkinson's diagnosis would exhibit narrower $\Delta\theta$ than we find in the healthy older participants according to interactions between Parkinson's disease with eyes closed (Hypothesis 4f) and with the destabilizing perturbation (e.g., standing on a foam surface; Hypothesis 4g). However, again, we raise the same caveat as in Hypothesis 4e. If Parkinson's disease is not simply accelerated aging, we may see individuals with Parkinson's disease show reversals of Hypotheses 2e–f (Hypothesis 4h).

2. Methods

2.1. Participants and experimental procedures

We utilized an extensive collection of postural sway data that included a wide range of disease populations and task constraints. We present a summary of the key information below; however, we recommend referring to the original publications for comprehensive details regarding participants, task constraints, and experimental protocols.

2.1.1. Dataset 1

Thirty-two individuals with Parkinson's disease ($M \pm SD$ age: 66 ± 10 years; 8 women) participated in this study under both on-medication and off-medication conditions [25]. Table 2 provides details about individuals with Parkinson's disease. The patients were instructed to stand still on a force plate for 30 sec in four different conditions: standing on a rigid surface with eyes open, standing on a rigid surface with eyes closed, standing on a foam surface with eyes open, and standing on a foam surface with eyes closed. Each condition was repeated three times, and the order of the conditions was pseudorandomized for each participant. Ground reaction forces were recorded at 100 Hz. Before the experimental sessions, older adults with Parkinson's disease were required to maintain a stable dose of L-DOPA medication for at least one month. Two experimental sessions were conducted: one in the medication's ON condition and the other in the medication's OFF condition. In the ON condition, the participants were instructed to take their dopaminergic medication one hour before the session to stabilize the dosage. During the OFF condition, in contrast, the participants were required to abstain from using any medication for Parkinson's disease for at least 12 hours. The order in which the "ON" and "OFF" sessions were conducted was randomized across participants. These rigorous procedures were implemented to ensure the reliability and accuracy of the collected data in reflecting the effects of L-DOPA medication on individuals with

Table 2
Details about individuals with Parkinson's disease. See de Oliveira et al. [25] for further details.

Participant	Sex	Age	Disease duration	Hoehn Yahr	UPDRS II		UPDRS III	
					On	Off	On	Off
P1	F	53	4	1	1	2	7	10
P2	M	69	1	2	2	1	9	17
P3	M	68	19	3	5	10	33	48
P4	F	77	15	2	3	6	14	30
P5	M	65	15	3	11	9	32	36
P6	F	44	14	2	1	11	15	16
P7	M	60	5	2	4	6	15	30
P8	M	81	4	3	6	7	28	47
P9	M	76	11	2	1	3	12	20
P10	M	73	3	2	1	1	20	22
P11	F	66	10	4	12	12	35	38
P12	M	53	14	2	1	4	25	23
P13	M	46	8	1	1	10	2	6
P14	M	57	4	2	3	5	16	24
P15	M	74	3	3	7	4	15	29
P16	M	74	12	2	2	3	16	14
P17	M	50	5	2	7	8	19	22
P18	M	62	7	2	6	5	25	33
P19	M	70	4	2	0	4	28	26
P20	M	61	10	2	4	5	25	38
P21	M	60	7	3	3	4	17	32
P22	M	62	5	2	4	6	21	38
P23	M	77	13	2	8	10	7	17
P24	M	71	2	2	3	4	25	25
P25	M	68	4	3	1	1	12	22
P26	F	78	3	3	2	2	6	40
P27	F	82	5	3	3	4	21	21
P28	F	53	5	3	5	7	8	6
P29	M	53	8	2	7	7	14	19
P30	F	78	6	2	2	2	15	21
P31	M	69	4	2	8	9	31	32
P32	M	66	8	3	7	8	11	45

Parkinson's.

2.1.2. Dataset 2

A total of 22 healthy older adults (67 ± 8 years; 11 women) and 27 healthy young adults (28 ± 5 years; 12 women) participated in this study [29]. The protocol was the same as in Oliveira et al. [25]. Individuals were instructed to stand still on a force plate for 60 sec under four different conditions: standing on a rigid surface with eyes open, standing on a rigid surface with eyes closed, standing on a foam surface with eyes open, and standing on an unstable surface with eyes closed. Each condition was repeated three times, and the order of the conditions was pseudorandomized for each participant. Ground reaction forces were recorded at 100 Hz. Considering that the present study extended the findings of a previous investigation comparing OFSCA output between individuals with Parkinson's and healthy young and older adults, postural CoP data for only the initial 30 sec were utilized for the analysis.

2.1.3. Dataset 3

A total of 10 patients affected by the rare Stargardt's syndrome (38 ± 15 years; 4 women), all having the ABCA4 gene mutation, and 10 control healthy adults (38 ± 14 years; 4 women) participated in this study [111]. Stargardt disease is a genetic disorder characterized by progressive degeneration of the macula, leading to central vision loss in children and young adults. Individuals were instructed to stand still on a force plate for 60 sec in three different conditions: eyes closed; eyes open, still target fixation; eyes open, moving target tracking. Each condition was repeated five times, and the order of the conditions was pseudorandomized for each participant. Ground reaction forces were recorded at 2000 Hz and resampled to 20 Hz.

2.1.4. Dataset 4

A total of 16 healthy young adults (24 ± 4 years; 9 women) participated in this study [75]; also see Kelty-Stephen et al. [59]; Mangalam et al. [90]. Individuals were instructed to stand still on a force plate for 120 sec in six different conditions: eyes closed, eyes open, gaze fixated at a point projected at their eye level at distances of 25 cm, 50 cm, 135 cm, 220 cm, and 305 cm in front of them. More specifically, from behind the participant, a laser pen projected a static point-light on the center of a 5×5 in white tripod-mounted screen in front of a white visual-field-filling background. The 50 cm distance within the comfortable viewing range requires the least lens-accommodation effort, and the 25-cm distance and all longer distances served to destabilize posture [59]. Each condition was repeated three times, and the order of the conditions was pseudorandomized for each participant. Ground reaction forces were recorded at 100 Hz.

2.1.5. Dataset 5

A total of 10 healthy young adults (21 ± 1 years; 0 women) participated in this study [35]; also see Kelty-Stephen et al. [58]. Individuals were instructed to stand still on a force plate for 30 sec in two conditions: balancing either a sand-filled tube (stable condition) or a water-filled tube (unstable condition) using both hands. Each condition was repeated five times, and the order of the conditions was pseudorandomized for each participant. Ground reaction forces were recorded at 100 Hz.

2.2. Statistical analysis

2.2.1. Oriented Fractal Scaling Component Analysis (OFSCA)

Traditional CoP models rely on two key assumptions: (i) 2D CoP planar trajectory can be comprehensively characterized by the consideration of two independent fBm sample paths, denoted as $\{(x^{(1)}[i]\}$ and $\{x^{(2)}[i])\}$ ($i = 1, 2, \ldots, N$; with N being the length of the trajectory). (ii) These two components invariably maintain orthogonality. Consequently, the scaling property of each angular component (projection onto a rotated direction) remains consistent and impervious to any rotational transformation. Nevertheless, it is worth noting that this "isotropy" may not always hold and should be seen as the exception rather than the rule, as there is no inherent reason to believe that all natural trajectories exhibit isotropic behavior [53,103]. Our study employed the oriented fractal scaling component analysis [113] to delve into the anisotropic autocorrelation characteristics of 2D CoP planar trajectories. This approach commences by assessing the angle-dependent scaling properties of the trajectory using a higher-order detrending moving average (DMA), which we refer to as DDMA [129]. Subsequently, it dissects the observed 2D trajectory into two distinct components, each with varying orientations and scaling properties.

This approach reveals that CoP fluctuations exhibit a spatial distribution of long-range correlations. The directions with the strongest long-range correlations represent the primary directions of influence on posture control, as demonstrated by trajectories ϵ_1 and ϵ_2 at angles θ_1 and θ_2 relative to the horizontal reference direction in Fig. 2*a*. To uncover the inherent patterns within these original trajectories obtained from the observed 2D planar trajectory (represented here as fractional Gaussian noise or fGn), the OFSCA procedure begins with a transformation of the observed 2D trajectory. This extension encompasses all angles within the range of $0 \leqslant \theta < \pi$, as vividly depicted in Fig. 2*b*. Following this transformation, the DDMA analysis comes into play, quantifying the strength of long-range correlations present in these expanded trajectories at each angle. Here, Fig. 2*c* illustrates the directions linked to the lowest and highest values of the strength of long-range correlations, denoted as H_1 and H_2. To identify the original components, one must pinpoint the directions corresponding to these scaling exponents' maximum and minimum values, labeled as $\theta_{\min}$ and $\theta_{\max}$. Notably, these values consistently run orthogonal to the original orientations of the components, as demonstrated in Fig. 2d. Ultimately, the orientations of H_1 and H_2 are instrumental in reconstructing the actual 2D planar trajectory that comprises ϵ_1 and ϵ_2, along with their corresponding directions, as depicted in Fig. 2e.

For a more in-depth understanding of the OFSCA method, we recommend consulting the original article by Seleznov et al. [113]. Additionally, readers can find further insights into its application in individuals with Parkinson's disease in our previous study by Mangalam et al. [89]. We submitted each postural CoP planar trajectory to the OFSCA. We computed the angle between the major and minor directions of postural control reflecting the directions with the strongest and weakest temporal correlations θ_1 and θ_2, respectively, as $\Delta\alpha = \widehat{\theta}_1 \sim \widehat{\theta}_2$.

2.2.2. Linear mixed-effects modeling of $\Delta\theta$

A linear mixed-effects model was used to examine the fixed effects of Group and Postural conditions on $\Delta\alpha$. We included the random factor of participant identity by allowing the intercept to vary across participants. Statistical analyses were performed in *R* [105] using the packages "lme4" [10]. Significance was set at the two-tailed α level of 0.05. We detail the main effects and interactions below and indicate which foregoing hypotheses they addressed.

Group membership appeared as a main-effect covariate, with values *OlderAdults*, *Stargardt's*, and *Parkinson's* for fitting contrasts with the control value of *HealthyYoung*. Coefficients for the first three group labels corresponded to the average difference from the healthy young participants.

Other main-effect covariates encoded trial-by-trial endogenous fractal properties (the trial-by-trial angular variance of the Hurst exponents across the 2D CoP trajectory, maximum angular Hurst exponent, and minimum angular Hurst exponent, that is, SD_H, H_1, and H_2, respectively), and task settings (*EyesClosed* equaling 1 for all conditions with eyes closed in Datasets 1 through 4 allowed fitting contrast with the control value of 0 for all other task settings and *UnstablePosture* equaling 1 for the foam surface in Dataset 1; the foam surface in Dataset 2; the

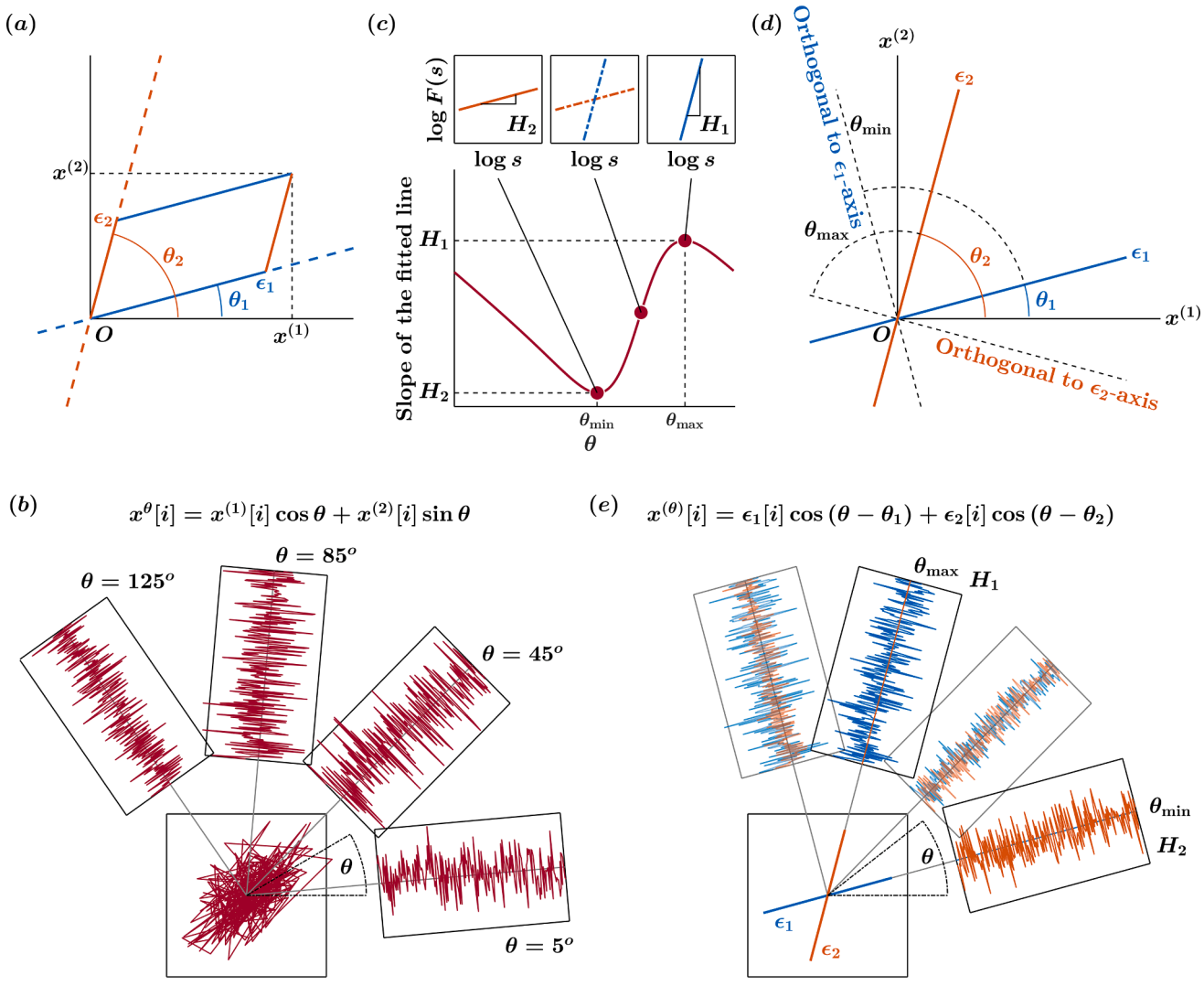

Fig. 2. Primary depiction of the detection of angle-dependent temporally correlated components $\{\epsilon_1[i]\}$ and $\{\epsilon_2[i]\}$ in the $(x^{(1)}, x^{(2)})$ plane. The OFSCA premise posits that the 2D CoP trajectory exhibits distributed long-range correlations. The primary directions with the strongest correlations, represented by angles θ_1 and θ_2 relative to the horizontal reference direction (*a*), influence posture control, as demonstrated by trajectories ϵ_1 and ϵ_2. To reveal inherent patterns within these initial trajectories, initially treated as fractional Gaussian noise (fGn), the OFSCA process begins by transforming the observed 2D trajectory (*b*). This transformation extends the trajectory, covering all angles within the $0 \leqslant \theta < \pi$ range. Then, DDMA analysis assesses the strength of long-range correlations across these extended trajectories for each angle (*c*). This framework identifies directions associated with the minimal and maximal values of long-range correlation strengths, referred to as H_1 and H_2. To determine the original components, one must pinpoint where these scaling exponents have their maximum and minimum values, designated as $\theta_{\min}$ and $\theta_{\max}$, respectively. Interestingly, these values consistently run perpendicular to the original component orientations (*d*). Ultimately, the orientations of H_1 and H_2 are used to reconstruct the actual 2D CoP trajectory, encompassing ϵ_1 and ϵ_2 (*e*). Reproduced from Mangalam et al. [89].

moving-target fixation in Dataset 3; fixations at 25,50,135,220, and 305 cm in Dataset 4; and the balancing of a water-filled tube allowed fitting contrasts with the control value of 0 for all other task settings).

We can align these main effects with the foregoing hypotheses. The intercept would allow testing Hypothesis 1a. The coefficients for *OlderAdults*, *Stargardt's*, and *Parkinson's* would allow testing Hypotheses 2a, 3a, and 4a, respectively. The coefficients for SD_H,H_1, and H_2, would allow testing Hypotheses 1b, 1c, and 1d, respectively. The coefficients for *UnstablePosture* and *EyesClosed* would allow testing Hypotheses 1e and 1f. We fit all *Group* × SD_H,H_1,H_2, all *Group* × *Task* interactions, and an effect of *Trial*. Although we included all main effects composing the significant interactions Allison [2], we removed all interaction terms that failed to improve model fit at $p < 0.05$. The only exception to this exclusion was *Trial*, which we kept to ensure that the linear-mixed effect model did not omit any chance to address order effects across the different tasks.

The alignment between interactions of the form *Group* × SD_H, H_1, H_2 aligned with the preceding hypotheses as follows. The coefficients for *OlderAdults* × SD_H (positive), *OlderAdults* × H_1 (negative), and *OlderAdults* × H_2 (positive) would allow testing Hypotheses 2b, 2c, and 2d, respectively. The coefficients for *Stargardt's* × SD_H (positive), *Stargardt's* × H_1 (negative), and *Stargardt's* × H_2 (positive) would allow testing Hypotheses 3b, 3c, and 3d, respectively. Hypothesis 3e was the prediction of a reversal of those coefficients. The coefficients for *Parkinson's* × SD_H (positive), *Parkinson's* × H_1 (negative), and *Parkinson's* × H_2 (positive), would allow testing Hypotheses 4b, 4c, and 4d, respectively. Hypothesis 4e was the prediction of a reversal of those coefficients.

The *Group* × *Task* interactions aligned with the preceding hypotheses as follows. The coefficients for *OlderAdults* × *EyesClosed* (negative) and *OlderAdults* × *UnstablePosture* (negative) would allow testing Hypotheses 2e and 2f, respectively. The coefficients for *Stargardt's* × *EyesClosed*, *Stargardt's* × *EyesClosed* (negative), and *Stargardt's* × *UnstablePosture* (negative), would allow testing Hypotheses 3f and 3g, respectively. The

coefficients for *Parkinson's* × *EyesClosed* (negative) and *Parkinson's* × *UnstablePosture* (negative) would allow testing Hypotheses 4f and 4g, respectively.

The policy of removing all higher-order terms that failed to improve model fit at $p < 0.05$ entailed removing *OlderAdults* × *EyesClosed*, *Stargardt's* × *EyesClosed*, *Stargardt's* × *UnstablePosture*, *Parkinson's* × *EyesClosed* thus leaving us without evidence supportive of Hypothesis 2e, 3f, 3g, 4f and 4g. Higher-order interactions likewise failed to improve model fit. We aimed to provide clean modeling separation between task effects and effects associated with endogenous fractal temporal correlations.

3. Results

We evaluated the angle dependence of $F^{(\theta)}(\tilde{s})$ for the original CoP planar trajectory over the range of $0 \leqslant \theta < \pi$ in increments of $\pi/179$ rad. We set the scaling range $1.2 < \log_{10}\tilde{s} < 2.5$ (from 0.16 to 3.2 sec) and estimated the slopes of linear regressions to find two representative orientations. We made noteworthy observations during the young adult's endeavor to maintain an upright stance while delicately balancing a water-filled tube using both hands. The most pronounced descent occurred at $\widehat{\theta}_{\min} = 179^{\circ}$, while the sharpest ascent was observed at $\widehat{\theta}_{\max} = 91^{\circ}$ (Fig. 3*a–d*). Consequently, we derived estimated orientations of $\widehat{\theta}_1 = 89^{\circ}$ and $\widehat{\theta}_2 = 1^{\circ}$. Notably, the scaling behaviors of the original components $\widehat{\epsilon}_1[i]$ and $\widehat{\epsilon}_2[i]$, reconstructed from these slopes, exhibited a distinct contrast (Fig. 3*d*, *e*, *f*). Within the scaling range of $1.2 \leqslant \tilde{s} < 2.5$, the scaling exponent $H = 0.72$ and $H = 0.98$ were indicative of healthy levels of long-range correlations along the major direction of control for the orientations $\widehat{\epsilon}_1$ and $\widehat{\epsilon}_2$, respectively. Remarkably, despite the challenging task of balancing the water-filled tube, which had the potential to destabilize the posture, both $\widehat{\epsilon}_1$ and $\widehat{\epsilon}_2$ aligned with the AP and ML directions, reflecting robust postural control along these two directions.

Figs. 4 and 5 present compelling visuals that reveal the deterioration of postural control, focusing on the distortions in orientation exhibited by an older adult and an untreated individual with Parkinson's disease. In their attempts to sustain an upright stance on an unstable surface, both participants undertook this challenging task with closed eyes. The deliberate selection of this postural condition for illustration was driven by its demanding nature, expected to elicit the most pronounced asymmetry in body sway.

During the older adult's attempt to maintain balance on an unstable surface with closed eyes, we observed that the steepest downward slope occurred at $\widehat{\theta}_{\min} = 117^{\circ}$, while the steepest upward slope was at $\widehat{\theta}_{\max} = 1^{\circ}$ (Fig. 4d). As a result, we derived estimated orientations of $\widehat{\theta}_1 = 27^{\circ}$ and $\widehat{\theta}_2 = 91^{\circ}$. Notably, the scaling behaviors of the original components $\widehat{\epsilon}_1[i]$ and $\widehat{\epsilon}_2[i]$ reconstructed from these slopes displayed a distinct contrast (Fig. 4e, f). Within the scaling range of $1.2 \leqslant \tilde{s} < 2.5$, the scaling exponent $H = 1.31$ for the orientation $\widehat{\epsilon}_1$ indicated that control along the major direction remained intact. Conversely, the scaling exponent $H = 1.08$ for the orientation $\widehat{\epsilon}_2$ pointed to a loss of long-range correlations along the minor direction of control. Notably, both $\widehat{\epsilon}_1$ and $\widehat{\epsilon}_2$ deviated significantly from the AP and ML directions, exhibiting a suborthogonal alignment of postural control along the two directions, potentially reflecting an adaptation to reduce fall along the AP direction.

In the Parkinson's patient who was not taking medication, an interesting phenomenon occurred when they attempted to maintain an upright stance on an unstable surface with their eyes closed. We observed the minimum and maximum slopes at angles of $\widehat{\theta}_{\min} = 168^{\circ}$ and $\widehat{\theta}_{\max} = 53^{\circ}$, respectively (Fig. 5d). This information allowed us to estimate the orientations of $\widehat{\theta}_1 = 78^{\circ}$ and $\widehat{\theta}_2 = 143^{\circ}$. Focusing on the scaling range $1.2 \leqslant \tilde{s} < 2.5$, we observed interesting differences in the scaling behaviors of the reconstructed original components, $\widehat{\epsilon}_1[i]$ and

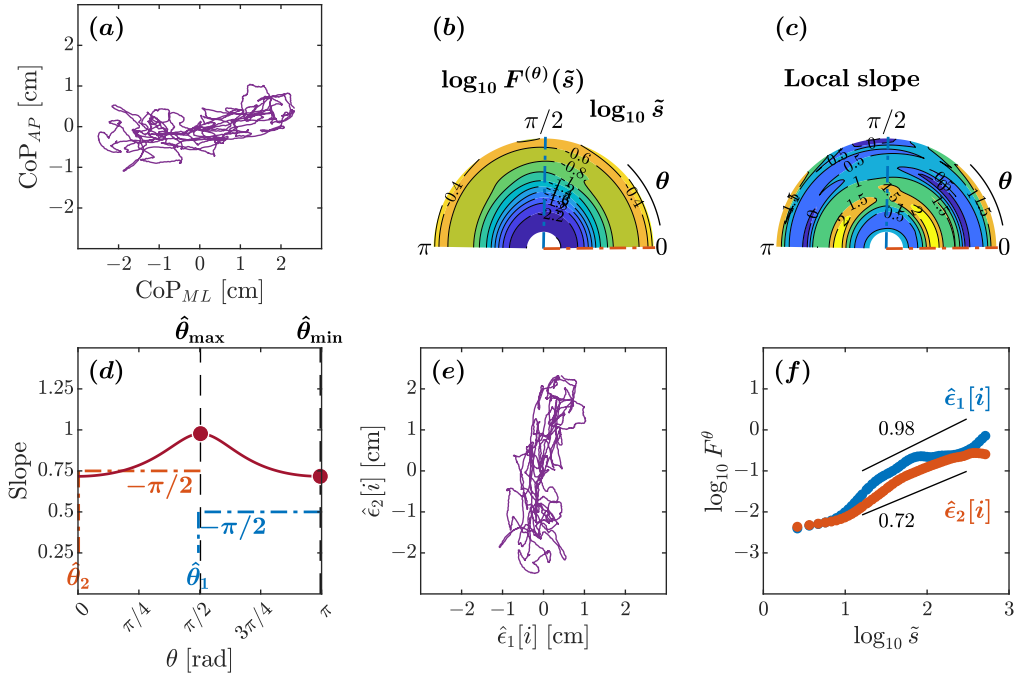

Fig. 3. Orientation decomposition of the CoP planar trajectory of a representative young adult maintaining an upright balance while balancing a water-filed tube. (*a*) CoP along the anatomical AP and ML directions. (*b*) θ-dependent heterogeneity in CoP fluctuations, indicated by the angle dependence of the fluctuation function quantifying heterogeneity or variability in CoP fluctuations across timescales, s, that is, $\log_{10}F^{(\theta)}(\tilde{s})$ vs. $\log_{10}\tilde{s}$, where $\tilde{s} \sim s/1.93$ in the second order DDMA. (*c*) θ-dependence of the local slopes of $\log_{10}F^{(\theta)}(\tilde{s})$ vs. $\log_{10}\tilde{s}$, indicating the spatial distribution of long-range correlations. (*d*) θ-dependence of the slope in the range of $1.2 < \log_{10}\tilde{s} < 2.5$. (*e*) Reconstructed CoP along the original directions of postural control, $\widehat{\epsilon}_1[i], \widehat{\epsilon}_2[i]$. (*f*) Fluctuation functions of CoP along the original directions of postural control, $\widehat{\epsilon}_1$ with $\widehat{\theta}_1 = 89^{\circ}$ and $\widehat{\epsilon}_2$ with $\widehat{\theta}_2 = 1^{\circ}$, with $\Delta\theta = 88^{\circ}$.

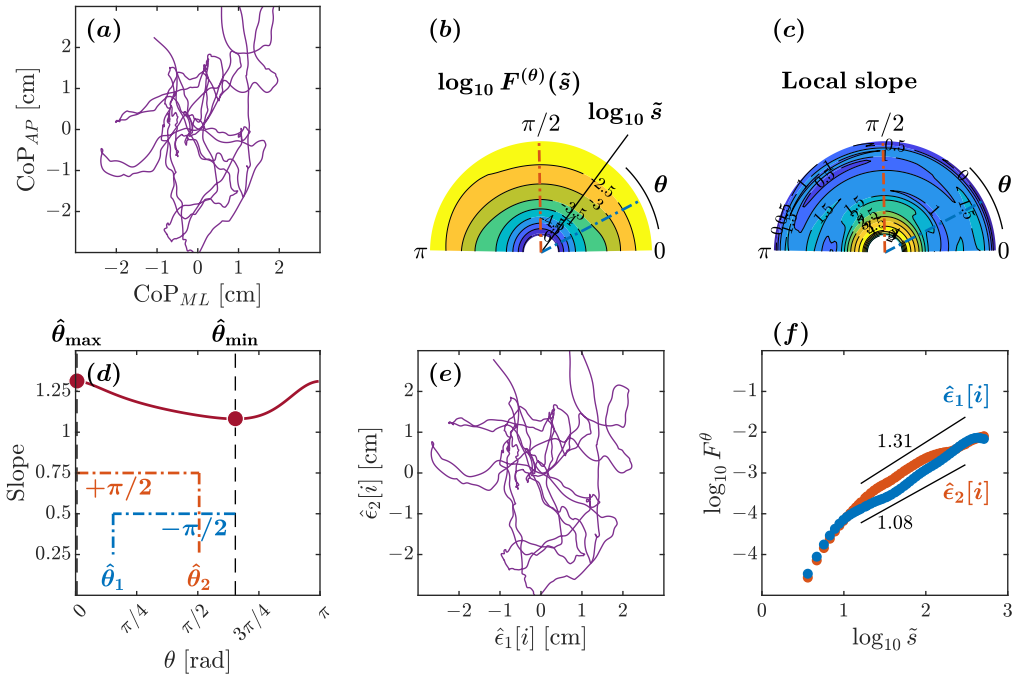

Fig. 4. Orientation decomposition of the CoP planar trajectory of a representative older adult maintaining an upright balance on an unstable surface with closed eyes. (*a*) CoP along the anatomical AP and ML directions. (*b*) θ-dependent heterogeneity in CoP fluctuations, indicated by the angle dependence of $\log_{10}F^{(\theta)}(\tilde{s})$ vs. $\log_{10}\tilde{s}$, where $\tilde{s} \sim s/1.93$ in the second order DDMA. (*c*) θ-dependence of the local slopes of $\log_{10}F^{(\theta)}(\tilde{s})$ vs. $\log_{10}\tilde{s}$, indicating the spatial distribution of long-range correlations. (*d*) θ-dependence of the slope in the range of $1.2 < \log_{10}\tilde{s} < 2.5$. (*e*) Reconstructed CoP along the original directions of postural control, $\hat{\epsilon}_1[i]$, $\hat{\epsilon}_2[i]$. (*f*) Fluctuation functions of CoP along the original directions of postural control, $\hat{\epsilon}_1$ with $\hat{\theta}_1 = 27^{\circ}$ and $\hat{\epsilon}_2$ with $\hat{\theta}_2 = 91^{\circ}$, with $\Delta\theta = 64^{\circ}$.

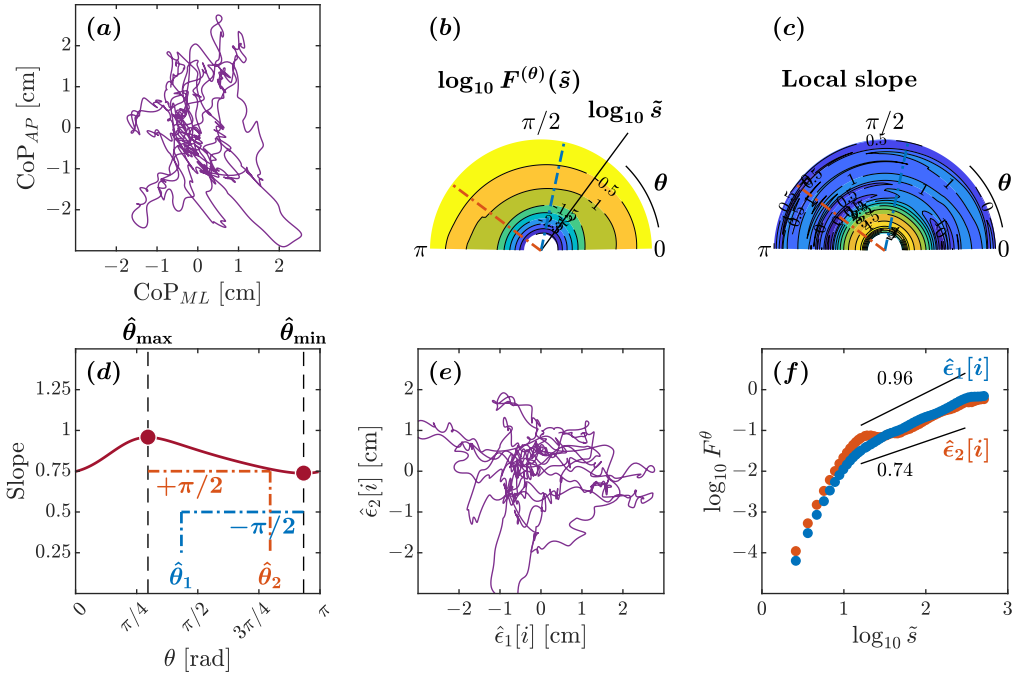

Fig. 5. Orientation decomposition of the CoP planar trajectory of a representative Parkinson's patient off medication maintaining an upright balance on an unstable surface with eyes closed. (*a*) CoP along the anatomical AP and ML directions. (*b*) θ-dependent heterogeneity in CoP fluctuations, indicated by the angle dependence of $\log_{10}F^{(\theta)}(\tilde{s})$ vs. $\log_{10}\tilde{s}$, where $\tilde{s} \sim s/1.93$ in the second order DDMA. (*c*) θ-dependence of the local slopes of $\log_{10}F^{(\theta)}(\tilde{s})$ vs. $\log_{10}\tilde{s}$, indicating the spatial distribution of long-range correlations. (*d*) θ-dependence of the slope in the range of $1.2 < \log_{10}\tilde{s} < 2.5$. (*e*) Reconstructed CoP along the original directions of postural control, $\hat{\epsilon}_1[i]$, $\hat{\epsilon}_2[i]$. (*f*) Fluctuation functions of CoP along the original directions of postural control, $\hat{\epsilon}_1$ with $\hat{\theta}_1 = 78^{\circ}$ and $\hat{\epsilon}_2$ with $\hat{\theta}_2 = 143^{\circ}$, with $\Delta\theta = 65^{\circ}$.

$\widehat{\epsilon}_2[i]$ (Fig. 5e, f). It became evident that the loss of coordinated control activity along both directions led to significant reductions in long-range correlations, as indicated by the estimated minimum and maximum slopes of 0.74 and 0.96, respectively. Furthermore, it is worth noting that the orientation of $\widehat{\epsilon}_1$ deviated considerably from the ML direction and was found to be suborthogonal to the $\widehat{\epsilon}_2$ orientation, reflecting an adaptation to reduce fall along the AP direction. In summary, this Parkinson's patient exhibited anisotropy in postural control and a more pronounced loss of long-range correlations in postural sway variability.

3.1. Linear mixed-effects modeling results for $\Delta\theta$

3.1.1. Group effects

At the group level, regression model coefficients supported Hypotheses 1a, 2a, and 4a but not 3a. According to the intercept, the young adults had $\Delta\theta$ of $1.070\times10^2\pm3.416\times10^0$ ($t=31.317, P<2.000\times10^{-16}$; Table 3; Hypothesis 1a)—slightly higher than 90° (notably, the model addresses random effect intercepts by participants, elucidating why the model intercept deviates from the more straightforward descriptive values presented in Table 1). The older adults exhibited significantly narrower $\Delta\theta$ of $-4.568\times10^1\pm9.623\times10^0$ ($t=-4.747$, $P<2.310\times10^{-6}$; Hypothesis 2a). No significant differences were observed between young adults with Stargardt's syndrome and healthy young adults, thereby failing to support Hypothesis 3a. Consistent with previous research [89], the older adults with Parkinson's disease exhibited significantly narrower $\Delta\theta$ of $-4.321\times10^1\pm4.789\times10^0$ ($t=-9.021, P<2.310\times10^{-16}$; Hypothesis 4a). However, whereas previous results showed a threefold narrowing of $\Delta\theta$ for older adults with Parkinson's disease compared to healthy older adults, we find now that this narrowing due to Parkinson's disease is a significant difference from healthy young adults but not from healthy older adults. That is, the novel inclusion of covariates involving endogenous fractal temporal correlation may have controlled for significant variation incorrectly attributed to simple differences in diagnosis. It remains for subsequent sections here to speak to how $\Delta\theta$ varied with fractal temporal correlations and whether older adults with Parkinson's disease did show a systematic difference in this shared variation.

Table 3
Outcomes of the LME[1] examining the influence of Parkinson's disease, task constraints, and other factors on $\Delta\theta$.

Factor	Estimate$\pm SE$	t	P[2]
Intercept	$1.070\times10^2\pm3.416\times10^0$	31.317	$<2.000\times10^{-16}$
OlderAdults	$-4.568\times10^1\pm9.623\times10^0$	−4.747	2.310×10^{-6}
Stargardt's	$-7.218\times10^0\pm7.267\times10^0$	−0.993	0.321
Parkinson's	$-4.321\times10^1\pm4.789\times10^0$	−9.021	$<2.000\times10^{-16}$
SD_H	$1.266\times10^2\pm4.077\times10^1$	3.105	1.929×10^{-3}
H_1	$-5.883\times10^1\pm1.251\times10^1$	−4.701	2.780×10^{-6}
H_2	$4.024\times10^1\pm1.181\times10^1$	3.407	6.700×10^{-4}
EyesClosed	$-2.729\times10^0\pm6.618\times10^{-1}$	−4.124	3.880×10^{-5}
UnstablePosture	$-1.940\times10^0\pm9.263\times10^{-1}$	−1.598	0.110
Trial	$9.400\times10^{-2}\pm3.146\times10^{-1}$	0.299	0.765
OlderAdults × SD_H	$1.208\times10^3\pm3.844\times10^2$	3.144	1.691×10^{-3}
OlderAdults × H_1	$-3.394\times10^2\pm1.288\times10^2$	−2.635	8.486×10^{-3}
OlderAdults × H_2	$3.682\times10^2\pm1.263\times10^2$	2.916	3.587×10^{-3}
OlderAdults × *UnstablePosture*	$-9.192\times10^0\pm2.165\times10^0$	−4.246	2.280×10^{-5}
Stargardt's × SD_H	$-2.083\times10^3\pm5.365\times10^2$	−3.884	1.060×10^{-4}
Stargardt's × H_1	$7.228\times10^2\pm1.816\times10^2$	3.979	7.150×10^{-5}
Stargardt's × H_2	$-7.000\times10^2\pm1.827\times10^2$	−3.831	1.310×10^{-4}
Parkinson's × SD_H	$3.472\times10^2\pm6.083\times10^1$	5.707	1.330×10^{-8}
Parkinson's × H_1	$-6.148\times10^1\pm1.757\times10^1$	−3.498	4.790×10^{-4}
Parkinson's × H_2	$7.509\times10^1\pm1.742\times10^2$	4.312	1.700×10^{-5}

[1] $\Delta\theta \sim OlderAdults^*(SD_H + H_1 + H_2 + UnstablePosture) + Stargardt's^*(SD_H + H_1 + H_2) + Parkinson's^*(SD_H + H_1 + H_2) + EyesClosed + Trial$.
[2] Boldfaced values indicate statistical significance at $P < 0.05$.

3.1.2. Endogenous fractal temporal correlations

The regression model supported the general prediction that changes in $\Delta\theta$ for healthy young adults changed alongside the topological features of the endogenous fractal temporal correlations. It supported all three of the specific Hypotheses 1b–d. $\Delta\theta$ increased with greater SD_H ($B=1.266\times10^2\pm4.077\times10^1, t=3.105, P=1.929\times10^{-3}$). $\Delta\theta$ was also inversely related to the range between the maximum and minimum Hurst exponents H_1 and H_2, with individual change in either of these Hurst exponents being sufficient to change $\Delta\theta$, which decreased with greater H_1 ($B=-5.883\times10^1\pm1.251\times10^1, t=-4.701, P=2.780\times10^{-6}$) and increased with greater H_2 ($B=4.024\times10^1\pm1.181\times10^1$, $t=3.407, P=6.700\times10^{-4}$).

Hence, the empirical portrait that begins to resolve is of two classes of topology: first, relatively wider-angled, more-orthogonal arrangement of axes with maximal and minimal Hurst exponents H_1 and H_2 appears to co-occur with more heterogeneity across angles of the support surface, and second, relatively narrower-angled, sub-orthogonal arrangement of axes with maximal and minimal Hurst exponents H_1 and H_2 appears to co-occurs with more disc-like homogeneity on average across angles of the support surface. In the former case, with greater average angular heterogeneity on the support-surface plane, we see narrower differences between extremes along the measured H continuum. In the latter case, with greater angular homogeneity on the support-surface plane, we see starker differences between extremes along the measured H continuum. This pattern of relationships between endogenous fractal temporal correlation and $\Delta\theta$ replicates previous findings in quiet standing with gymnasts [91], suggesting it is a signature of dexterous quiet stance. It is almost as if the angular distribution of H shows a trade-off between variance and range, resonating with age-old questions about how best to detect the excursions from Normality [24,99,128]

3.1.3. Task effects

We examined the impact of two distinct task manipulations: closing eyes and inducing postural destabilization using mechanical or visual instability. We anticipated that both conditions would reduce $\Delta\theta$, aligning with Hypotheses 1e and 1f, respectively. The regression model coefficients supported Hypothesis 1e but not Hypothesis 1f. The coefficient for *EyesClosed* indicated that closing eyes reduced $\Delta\theta$ by $-2.729\times10^0\pm6.618\times10^{-1}$ ($t=-4.124, P=3.880\times10^{-5}$). In contrast, the coefficient for *UnstablePosture* indicated that perturbing posture produced a negative trend, reducing $\Delta\theta$ by $-1.940\times10^0\pm9.263\times10^{-1}$ on average but not by a significant difference ($t=-1.598, P=0.110$). We retained the nonsignificant covariate for a judicious test of the subsequent interactions that did improve model fit [2]. We found contrasting effects compared with previous findings with a smaller cohort of healthy young gymnasts. Specifically, we observed no discernible

impact of closing eyes and a detrimental effect of postural instability on $\Delta\theta$. This discrepancy might stem from the nature of the postural instability task employed with the gymnasts, which involved a head-turn manipulation. It is plausible that head-turning acts more as a vestibular perturbation than a visual or mechanical one. Despite the relatively modest contribution of vestibular information to postural control [34,44,50,100,133], the significance of orientation in vestibular sensing could exert a more pronounced influence on vestibular perturbation and, consequently, on the angular information encoded by OFSCA.

3.1.4. Group interactions: Healthy older adults

The regression model showed that, compared to healthy young adults, the change in $\Delta\theta$ for healthy older adults showed stronger relationships with changes in their embodiment of topological features of the endogenous fractal temporal correlations, supporting Hypotheses 2b–d. Compared to healthy young adults, $\Delta\theta$ for healthy older adults showed even greater increase with greater SD_H ($B = 1.208 \times 10^3 \pm 3.844 \times 10^2, t = 3.144, P = 1.691 \times 10^{-3}$). Similar to the foregoing amplification of effects from young adults, $\Delta\theta$ for healthy older adults showed an accentuation of the healthy young adults' inverse relationship to the range between the maximum and minimum Hurst exponents H_1 and H_2. Again, individual change in either of these Hurst exponents was sufficient to change $\Delta\theta$, which decreased with H_1 ($B = -3.394 \times 10^2 \pm 1.288 \times 10^2, t = -2.635, P = 8.486 \times 10^{-3}$) and H_2 ($B = 3.682 \times 10^2 \pm 1.263 \times 10^2, t = 2.916, P = 3.587 \times 10^{-3}$). So, on all counts of fractal temporal correlations, postural control in healthy older adults appears only to have accentuated its commitments to the topologies apparent in young adult postural control. Notably, the coefficients for healthy older adults are roughly 10, 6, and 9 times larger than the corresponding baseline coefficients for healthy young adults. Hence, the variation of $\Delta\theta$ might be more sensitive in healthy aging, which could warrant future research to understand whether this heightened sensitivity is adaptive.

The regression model replicated known changes in $\Delta\theta$ due to task settings in healthy older adults. Specifically, it failed to support Hypothesis 2e but did support Hypothesis 2f. We omitted the interaction *OlderAdults* × *EyesClosed* because it failed to improve model fit. On the other hand, $\Delta\theta$ in healthy older adults decreased $-9.192 \times 10^0 \pm 2.165 \times 10^0$ in unstable task settings ($t = -4.246, P = 2.280 \times 10^{-5}$).

3.1.5. Group interactions: Stargardt's syndrome

The change in $\Delta\theta$ for young adults with Stargardt's syndrome radically reversed the relationships we found in healthy young adults with the embodiment of topological features of the endogenous fractal temporal correlations. Hence, the model failed to support Hypotheses 3b–d and specifically revealed significant effects in the opposite direction. Compared to healthy young adults, $\Delta\theta$ for young adults with Stargardt's syndrome showed a dramatic reduction with greater SD_H ($B = -2.083 \times 10^3 \pm 5.365 \times 10^2, t = -3.884, P = 1.060 \times 10^{-4}$). In a similar reversal of effects from young adults, $\Delta\theta$ for young adults with Stargardt's syndrome was directly related to the range between the maximum and minimum Hurst exponents H_1 and H_2. Once more, individual change in either of these Hurst exponents was sufficient to change $\Delta\theta$, which increased with H_1 ($B = 7.228 \times 10^2 \pm 1.816 \times 10^2, t = 3.979, P = 7.150 \times 10^{-5}$) and H_2 ($B = -7.000 \times 10^2 \pm 1.827 \times 10^2, t = -3.831, P = 1.330 \times 10^{-4}$). These results suggested that, whereas older adults exhibited an accentuation of the relationships between control topology and endogenous fractal fluctuations observed in healthy younger adults, young adults with Stargardt's syndrome showed a marked dissolution of these topologies and potentially embodying new ones not covered by the saddle-vs.-spiral-type duality. In sum, this evidence supports the more general Hypothesis 3e that postural control in Stargardt's syndrome does not resemble healthy aging—if anything, postural control in Stargardt's syndrome shows a pattern of fractal temporal correlations that is precisely opposite to what we find in healthy young adults or healthy aging.

Task effects highlight another failure of postural control in Stargardt's syndrome to resemble that in young adults or healthy aging. Specifically, there were no significant interactions of group membership in *Stargardt'ssyndrome* with either *EyesClosed* or *UnstablePosture*, failing to provide support for Hypotheses 3f and 3g. By default, the nonsignificant results for these task interactions with *Stargardt'ssyndrome* significantly differ from the significant interactions of the *OlderAdult* with *EyesClosed* or *UnstablePosture*. And so, by default, the regression model does support Hypothesis 3h. It is possible that subsequent testing could find a significant effect for one, another, or both of Stargardt's syndrome interactions with task settings.

3.1.6. Group interactions: Parkinson's disease

The effect of Parkinson's disease on $\Delta\theta$ did correlate with endogenous fractal temporal correlations and task effects. Previous modeling had found no task-effect differences between healthy young adults or healthy older adults and the change in $\Delta\theta$ for participants with Parkinson's disease [89]. Previous modeling also only tested the effects of endogenous fractal temporal correlations on $\Delta\theta$ in young-adult gymnasts Mangalam et al. [91]. The present modeling found that the interaction effects of *Parkinson's* with the endogenous fractal fluctuations and task settings were in the same direction as healthy young and healthy older adults. The main difference is that, whereas these interaction effects increased the sensitivity of $\Delta\theta$ in healthy older adults, these interactions increased $\Delta\theta$ *more* than for healthy young adults but *less* than for the healthy older adults. In this sense, although we found that Parkinson's disease accentuates some of the interaction effects from healthy younger adults, it appears that this accentuation is quite muted compared to healthy older adults. Postural control with Parkinson's patients showed smaller but significant accentuation than there was for healthy older adults.

The regression model coefficients supported Hypotheses 4b–d in all cases. They predicted an excess of $\Delta\theta$ variation *beyond* that in healthy young adults ($B = 3.472 \times 10^2 \pm 6.083 \times 10^1, t = 5.707, P = 1.330 \times 10^{-8}$). Once more, as in the case of the healthy older adults, $\Delta\theta$ for older adults with Parkinson's disease showed an accentuation of the healthy young adults' inverse relationship to the range between the maximum and minimum Hurst exponents H_1 and H_2. Like for the healthy older adults, individual change in either of these Hurst exponents was sufficient to change $\Delta\theta$, which decreased with H_1 ($B = -6.148 \times 10^1 \pm 1.757 \times 10^1, t = -3.498, P = 4.790 \times 10^{-4}$) and H_2 ($B = 7.509 \times 10^1 \pm 1.742 \times 10^2, t = 1.700, P = 1.700 \times 10^{-5}$). However, in addition to supporting Hypotheses 4b–d, the regression model supported Hypothesis 4e that the significant interactions of Parkinson's diagnosis with fractal temporal correlations would not simply resemble accelerated healthy aging. Indeed, the coefficients exhibited similar signs to those associated with the interactions of healthy older adults with endogenous fractal temporal correlations. However, in coding group membership into healthy *OlderAdults* versus *Parkinson's*, we maintained exclusivity, enabling an independent comparison of each group with the baseline of healthy young adults. To elucidate distinctions between healthy *OlderAdults* and those with *Parkinson's*, we implemented a slightly adjusted coding of group membership that identified participants with *Parkinson's* as belonging to the category of *OlderAdults*, which allowed coefficients fitting to encode differences from healthy older adults. Hence, although the coding of the group variable in the main text affirms the difference of the coefficients for *Parkinson's* from healthy young adults, the alternate coding produces a model on the same data that was able to confirm that these coefficients *Parkinson's* × SD_H, *Parkinson's* × H_1, and *Parkinson's* × H_2 were all significantly different from coefficients for *OlderAdults* × SD_H, *OlderAdults* × H_1, and *OlderAdults* × H_2. If Parkinson's disease were akin to accelerated aging, one would anticipate that older adults with Parkinson's disease would exhibit more pronounced effects compared to their healthy older counterparts.

However, the outcomes of this alternative coding present a contrasting picture; older adults with Parkinson's disease displayed *less* variability in association with endogenous fractal temporal correlations than their healthy older counterparts.

The regression model failed to support Hypotheses 4f–h. Older adults with Parkinson's disease showed no significant interaction with task effects on $\Delta\theta$. Also, we found no evidence of any reversal or attenuation of effects expected as part of healthy aging.

An interpretation of this pattern suggests that Parkinson's disease could signify a disconnection between $\Delta\theta$ and the saddle or spiral topologies observed in fractal temporal correlations. While individuals with Parkinson's disease do exhibit more pronounced effects of these fractal temporal correlations compared to their healthy counterparts, older adults with Parkinson's disease do not demonstrate a heightened engagement with these fractal temporal correlations in comparison to healthy older participants. Hence, whatever aspect of healthy aging entails this stronger dependence of $\Delta\theta$, older adults with Parkinson's disease do not show it. Prior work had shown a main effect of Parkinson's diagnosis with a reduction in $\Delta\theta$ [89]. However, the main effect appears attenuated now that we model the interaction of diagnostic group membership with endogenous fractal temporal correlations. Hence, the previous evidence of reduction might be partially contingent on the weakening of these topologies appearing in fractal temporal correlations [89]. However, this had not accounted for the dependence on endogenous temporal correlations until the subsequent work with gymnasts highlighted its potential adaptiveness for response to perturbation [91]. Older adults with Parkinson's disease may develop other topologies that follow from or contribute to diseased posture. However, as before [89], we found no task effects. This point suggests that, whatever alternate topologies they begin to explore other than healthy older adults, they did not show new coordination modes in a task-sensitive fashion.

4. Discussion

4.1. Summary of hypotheses and outcomes

Prior investigation examining the two-dimensional CoP trajectories by breaking it into directions representing the strongest and weakest fBm structure had shown that individuals with Parkinson's disease exhibit a suborthogonal control mechanism, which diverges from the orthogonal control observed in healthy young adults [89]. The current work aggregates multiple experiments' data to refine our understanding of how the deviation of this fractal patterning from the conventional anteroposterior and mediolateral directions changes with task conditions, group membership, and ongoing changes in fractal scaling along the directions of minimum and maximum strength of temporal correlations. Collectively, these investigations furnish compelling evidence supporting the notion that the OFSCA represents a promising, innovative method to develop a postural-sway-variability-based biomarker with the potential to facilitate early detection and ongoing monitoring of Parkinson's disease.

The present regression modeling focused on the OFSCA estimate of $\Delta\theta$, the angle between directions of strongest and weakest fractal temporal correlations as encoded by H. We modeled four major classes of hypotheses. The first class addressed the possible relationship of healthy young adults' $\Delta\theta$ to saddle- and spiral-type topologies for 2D distributions of fractal temporal correlations (Hypotheses 1a–d) as well as the sensitivity of healthy young adults' $\Delta\theta$ to manipulated task constraints (Hypotheses 1e–f). Results confirmed all of these hypotheses except for one. Healthy young adults showed posture with wide $\Delta\theta$ indicating close to the orthogonal arrangement of the major and minor axes, exhibiting strongest and weakest fractal temporal correlations, H_1 and H_2, respectively (Hypothesis 1a). Greater $\Delta\theta$ corresponded to greater angular variability as in saddle, rather than spiral-type topologies (Hypothesis 1b), as well as to lower H_1 (Hypothesis 1c) and to higher H_2 (Hypothesis 1d). This evidence is consistent with greater $\Delta\theta$ corresponding to a topology of greater average angular variability of fractal temporal correlations and lesser $\Delta\theta$ corresponding to a flatter, more homogeneous distribution of angular temporal correlations.

Task manipulations instructing participants to close their eyes narrowed $\Delta\theta$ (Hypothesis 1e), but manipulation destabilizing posture did not (Hypothesis 1f). Therefore, despite our observation that closing their eyes reduced the angle between the maximal and minimal fractal temporal correlations in healthy young adults, no similar narrowing effect was noted in the context of destabilized posture. This implies that the influence of visual information on the fractal topologies engaged in postural control might be more potent than that of proprioceptive information. Notably, this perspective is novel, as previous studies that simultaneously manipulated vestibular and visual perturbations found a significantly greater narrowing effect on $\Delta\theta$ with vestibular perturbations compared to visual perturbation, particularly in trained gymnasts [91]. It is possible that gymnastic training makes postural control more sensitive to vestibular perturbations and less sensitive to visual perturbations. Given the traditional understanding of visual and proprioceptive information having greater influence in postural control [22,83,108,132], this pattern of finding is surprising and warrants future study to examine visual, proprioceptive, and vestibular perturbations in one study and to determine when each type of perturbation may have the most effect.

A second set of hypotheses addressed healthy older adults. As indicated by previous results, we expected healthy older adults to show a reduction in $\Delta\theta$ (Hypothesis 2a). Healthy young adults with gymnastics training showed a task-sensitive reduction in $\Delta\theta$ due to the vestibular perturbation of a head turn [91]. We expected that the group difference of older adults might correspond to the vestibular degeneration [5,51,81]. Hence, we envisioned a similar patterning of results wherein the physiological changes across healthy aging might stabilize what healthy young gymnasts experience only as a fleeting task-dependent effect. We predicted that healthy aging might cultivate greater sensitivity of $\Delta\theta$ to both endogenous fractal temporal correlations (Hypotheses 2b–d) and task settings (Hypotheses 2e–f), yielding significantly larger amounts of variation in the same effects as observed in healthy young adults. The results supported all these predictions except that we supported Hypothesis 2f but not Hypothesis 2e, showing a different pattern of significance than for the healthy young adults. While closing eyes had an observable effect on healthy young adults, there was no interaction between healthy aging and the eyes-closed manipulation. In contrast, destabilizing task settings showed no significant effect in healthy young adults. Strikingly, destabilizing task settings led to a significant reduction in $\Delta\theta$ in healthy older adults.

The present work aimed to improve upon past investigations of possible clinical biomarkers (e.g., [89]) by ensuring greater specificity of the proposed biomarker. Therefore, including a second cohort characterized by clinical diagnosis enhances the overall balance and robustness of the experimental design. For instance, whereas we had previously only had healthy older adults as a control to compare patients with Parkinson's disease, we now have a separate diagnosis in younger adults to build a slightly broader portrait of diseased postural control. Hence, a third class of hypotheses aimed to test whether young adults with Stargardt's syndrome would show a comparable profile of changes in $\Delta\theta$ as healthy older adults. That is, it raised the question of whether this disease led young adults' postural control to resemble older adults in its group-average reduction (Hypothesis 3a), its dependence on endogenous fractal temporal correlations (Hypotheses 3b–d), and its task dependence (Hypothesis 3f–g). Alternatively, we were ready to find as an alternative that neither of these foregoing hypotheses would hold because disease—particularly disparate diagnoses—might fail to resemble any accelerated aging (Hypotheses 3e and 3h). Results failed to support Hypotheses 3a–d and 3f–g. Consistent with Hypothesis 3e, we found no group difference in $\Delta\theta$ and a complete reversal of the healthy young adults' positive, negative, and positive relationships of SD_H, H_1,

and H_2, respectively, with $\Delta\theta$. The negative, positive, and negative relationships of SD_H, H_1, and H_2, respectively, with $\Delta\theta$ in young adults with Stargardt's syndrome suggested that postural control in this diagnostic group might have entirely reshaped the control topologies to forms beyond the saddle- and spiral-types. Future research might explore other topologies these participants might have embodied in their postural control.

Consistent with Hypothesis 2h, we found no significant difference in how $\Delta\theta$ for participants with Stargardt's syndrome responded to task settings and so no resemblance to the significant interaction of *OlderAdults* with *UnstablePosture*. Certainly, although young adults with Stargardt's syndrome did not embody postural control that cancels the main effect of *EyesClosed*, the failure of $\Delta\theta$ to change with destabilizing task settings is characteristic of the failure for diseased postural control to make an adaptive response to contextual changes. The substantially different postural-control topologies may reallocate the control resources so much to undermine the task-sensitivity of the $\Delta\theta$ implicated in postural control.

Ultimately, our fourth set of hypotheses sought to examine whether older adults diagnosed with Parkinson's disease would manifest an amplification of the impacts on $\Delta\theta$ observed in healthy older adults. This inquiry was concerned with the possibility of Parkinson's disease mirroring an accelerated aging process, thereby accentuating the group-average reduction seen in healthy older adults (Hypothesis 4a), their reliance on endogenous fractal temporal correlations (Hypotheses 4b–d), and the task-dependent nature of these effects (Hypotheses 4f–g). Alternatively, we were ready to find as an alternative that neither of these foregoing hypotheses would hold because Parkinson's disease might fail to resemble any accelerated aging (Hypotheses 4e and 4h). The results supported Hypotheses 4e and 4h sooner than the other predictions. Specifically, the reduction of $\Delta\theta$ for participants with Parkinson's disease was significant compared to healthy young adults, but it was not significantly different from $\Delta\theta$ for healthy older adults. Also, although older adults with Parkinson's disease showed an accentuation of the positive, negative, and positive effects of SD_H, H_1, and H_2, respectively, on $\Delta\theta$ found in healthy young adults, this accentuation was significantly smaller than that found in healthy older adults. Similar to young adults with Stargardt's syndrome, older adults with Parkinson's disease did not show any significant difference in $\Delta\theta$ with task effects.

4.2. Specific directions for future inquiry

What is particularly noteworthy here is that prior research (i.e., [89]) had found a bigger reduction of $\Delta\theta$ for participants with Parkinson's disease than healthy older adults, which inspired our Hypothesis 4a. So, the previously reported difference between the reduction of $\Delta\theta$ in healthy older adults and that in older adults with Parkinson's disease might have been spuriously due to a failure to model this muted dependence on endogenous fractal temporal correlations in participants with Parkinson's disease. Articulating the topologies of fractal temporal correlations in postural control may thus better explain the apparent differences due to Parkinson's disease. It may be appealing for future research to shift from simply distinguishing outcomes based on qualitative diagnostic differences to modeling the development of disease and symptoms by referring to a common framework of topological features of control in temporal correlations. The latter approach might provide a less type-driven and more dimensional approach capable of modeling continuous differences across different patients and within the same patients over time, comparable to recent directions in psychopathology diagnosis [18].

The failure of older adults with Parkinson's disease to show only muted accentuation of $\Delta\theta$'s dependence on endogenous fractal temporal correlations compared to healthy older adults warrants further inquiry. This failure to show the same investment in these relationships between $\Delta\theta$ and SD_H, H_1, and H_2 as healthy older adults could suggest that older adults with Parkinson's disease embody other postural-control topologies that might better predict variation in $\Delta\theta$. Alternatively, an alternative interpretation posits that individuals with Parkinson's disease exhibit broader fluctuations in fractal temporal correlations, necessitating smaller coefficients to accommodate the variability observed in $\Delta\theta$ adequately. It could be the case that healthy older adults exhibit reduced variability in their endogenous fractal scaling. The regression model suggests that this diminished fractal variation may exert a more pronounced impact on healthy older adults. It is certainly possible that the stronger dependence of $\Delta\theta$ on fractal temporal correlations is evidence consistent with vestibular degeneration in healthy aging. However, Parkinson's disease entails only greater vestibular degeneration [120]. The stronger dependence on fractal temporal correlations in healthy older adults could reflect the compounding of two factors: vestibular degeneration due to healthy aging and the smaller variability of H that healthy older adults showed across the angular space of the support surface. It is also conceivable that other facets of the 2D distribution of CoP fractal temporal correlations might more accurately capture the structural changes associated with proprioceptive degeneration.

Another specific point warranting further inquiry is the absence of task effects on $\Delta\theta$ in both disease groups. We had previously found that the narrowing of $\Delta\theta$ could be an adaptive, fleeting task-sensitive response to perturbations [91]. The lack of specific task effects interacting with either of the disease groups seems to reinforce the simple point that diseased postural control is less dexterous, i.e., less capable of adapting to task constraints. They also show $\Delta\theta$ with muted dependence on fractal temporal correlations (e.g., for *Parkinson's* vs. *OlderAdults*) or outright-reversed dependence on fractal temporal correlations (e.g., for *Stargardt's* vs. *HealthyYoung*). Since healthy older adults show greater fractal dependence and task sensitivity of $\Delta\theta$, one may be related to the other. Perhaps task-dependence and fractal-dependence of $\Delta\theta$ could have mutual effects. Alternatively, exploratory modeling could find no significant interactions between task effects and endogenous fractal temporal correlation terms in our regression model. However, it is possible that either task or endogenous fractal temporal correlations could mediate the other, and this result would not appear in a regression model like the one reported here. However, future research might forgo the attention to $\Delta\theta$ and instead examine more direct relationships between them, for example, whether task manipulations might influence the 2D distributions of fractal temporal scaling and not simply the angle between θ_1 and θ_2. Then again, it is also possible that endogenous fractal temporal correlations could support task sensitivity. We could stimulate diseased postural systems with fractal temporal correlations [9,74,114]. In that case, it might be possible to bolster these endogenous fractal topologies that appear so diminished relative to age-matched controls (i.e., adults with Stargardt's syndrome compared to healthy young adults or adults with Parkinson's compared to healthy older adults). It would be therapeutically interesting to test whether such bolstering of endogenous fractal temporal correlations might make postural control in these diseased groups more task-sensitive.

4.3. Long-term entailments for the use of fractal scaling to investigate postural control

The present work elaborates on a long-running concern about the possibility that fractal temporal correlations might provide a biomarker-like window on the clinical status of patient physiology. This concern began with simplistic comparisons between young and older participants or between healthy and diseased participants, and the thesis has largely been the same, that is, that a loss of fractal-type complexity appears to go hand in hand with aging and disease [38,39,76,78,119]. This class of contrasts has had a heuristic value that has been positively inspirational and has pointed forward to many more modern elaborations [115]. The challenge has been maintaining a critical view of extending the heuristic value across the nuanced terrain of diagnosis and

health. For instance, a notable problem with this research area is the fundamental ambiguity about considering the full range of diagnoses and the diversity of healthy functioning. Although the role of complexity might afford a way to understand both between-group distinctions and within-group variations, very often, the noise-based stimulation interventions that aim to resuscitate the so-called healthy complexity have yet to disassociate clinically meaningful differences versus idiosyncratic differences within the group. For instance, while we aim to treat the clinically meaningful differences, we must also come to grips with the curious fact that, within any group with or without disease, the noise-based stimulation appears to depend on endogenous variability [9,45,74,101,102,114,125].

The present work achieves some early steps towards a more nuanced portrayal of how fractal-type complexity supports physiological coordination. Not only have we considered more than one disease, but we have also elaborated upon the intrinsic dynamics of endogenous fractal temporal correlation. This latter point has held, at times, more texture than early attempts at "loss of complexity" may have been ready to grasp in simpler heuristic form. For instance, appeals to "loss of complexity" have often appealed to an equally heuristic notion of "constraint." Presumably, "constraint" entails a rigid complement to "complexity," the value of such constraints has often appeared in different lights, making it unclear again which types of "complexity" is good and which might be the very disorder clinicians need to remove. In one line of research, we can see "constraint" (e.g., on the tails of skewed histograms in physiological data) appear as a healthy restraint on complexity, indicating lowered risk of disease [43,67,68,65,66,88]. In a distinct line of research, the imposition of "constraint" on the temporal correlations revealed by tailed autocorrelations emerges as a vulnerability to health and a potential risk factor for disease [80,79,78]. The challenge lies in recognizing that physiological functioning encompasses a greater nuance than a mere unidirectional continuum of increasing or decreasing "complexity." The hypothesis positing a reduction in complexity runs the peril of oversimplifying physiology, potentially transforming it into a monolithic construct. Clinicians, well-versed in the intricacies of the field, recognize the presence of extensive internal structures and contextual nuances influencing the trajectory of disease progression or health development throughout the lifespan.

In explicitly modeling the rich diversity of fractal patterning, the OFSCA represents an important step forward, making estimating losses of complexity more clinically valuable. Methodologies in biological and behavioral sciences are advancing, enhancing the characterization of heterogeneity in presumed complexity indicators. This includes exploring multiple fractal patterns rather than focusing solely on singular ones. The clinical payoff could be immense if we can tolerate the novel logical relationships (e.g., suborthogonal axes on the plane) and the florid-sounding terminology (e.g., "multifractality" to mean "multiple fractal patterns"). These new steps could help us chip away at the monolithic or too-dichotomous strictures of early notions of "complexity." This empirical flourishing of new methods unfolds in parallel with novel theoretical work exploring how anatomical-like constraints might complement and interact with fluid cascades of physiological processes. This line of theoretical reasoning allows us to envision how constraints might take on their clinical value—for good or for ill—based on their situation in a multi-scaled context, whether of tissues or patient histories. The most current evidence suggests, on the theoretical side, that the outcomes from such interplay between fluid physiology and contextual constraint should be "multifractal," as defined above, exhibiting variety in fractal scaling within the same process. Hence, the OFSCA joins a suite of preexisting multifractal methods (e.g., [21,55,95]), bringing the wholly novel insight of describing this diversity of fractal scaling in the angular space of postural control.

At the yet broader scale of understanding motor coordination, the present work embodies a novel proposal about posture control. In the present work, we have taken seriously the often loose proposal that fractal temporal correlations could be implicit in postural control. This proposal explains how we might interpret the temporal correlations in postural sway, for example, as though statistical correlations across time entail a mechanism for coordination across time. This gloss between statistical evidence and theoretical mechanism has strong heuristic value [3], but it is fundamentally hollow [49,62] and requires more diligent exploration of whether or not the fractal estimates of H precede and have effects on consequent postural outcomes [57,59,85,86]. The present work falls short of explicitly manipulating fractal scaling to stimulate postural control [107,106,56]. However, what it does instead is to model 2D endogenous fractal temporal correlations in terms of known topologies from intermittent postural control—specifically from postural control strategies whose intermittency is known to generate fractal scaling [6,7,96]. The OFSCA allows the new capacity to ask whether fractal temporal correlations embody these control topologies. If fractal scaling is important for postural control, how deeply can we situate them in the control process? If fractal scaling supports postural control, why could the control topologies not be the product of fractal scaling? Hence, we aim to test this concept of fractal-based control by inverting a traditional logic, for instance, that inverted-pendulum control modes oriented along the AP or ML axis might generate fractal scaling. This logic sits on the arbitrary premise that the AP and ML axes are privileged domains of postural control. However, the validity of and indeed the need for the OFSCA for studying postural control is that CoP may have systematic variability off of the standard vertical and horizontal (e.g., "AP" and "ML") axes of the force plate. So, there may be nothing privileged about variations along AP or ML, and rather, we may find the more generic capacity for fractal fluctuations to spread in orthogonal but also suborthogonal axes.

In summary, the present work offers new empirical reasons to raise two possibilities that have long gone unrecognized in fractal-themed rhetoric about postural control. The first and less controversial of these points is that much of the presumed evidence for or against the traditional "loss of complexity" perspective has been drastically underestimating the range of possible fractal differences that could manifest a loss of complexity. For instance, as noted in the Introduction, we often see older and younger adults compared only to the fractal temporal correlations along the AP and ML axes. Untold numbers of false positives and false negatives in the search for "loss of complexity" are hiding in the angular space between AP and ML axes in almost all modern postural research.

Second, and more controversially, we propose that, by searching for control topologies to manifest in terms of the angular distribution of fractal scaling, the Hurst exponent H itself may not be the simple incidental product of inverted pendulum control models. Contrarily, it is plausible that H functions as a control parameter and holds a legitimate position as a dimension on the phase planes used to model postural control. This consideration may extend to contexts involving inverted pendulum dynamics beyond the conventional upright-postural model. This point is out of step with most literature on postural control for which the θ of the inverted pendulum model is plotted against its derivative $\dot{\theta}$ [6,7,96]. The alternative proposition regarding the fractal-themed discourse on postural control remains inadequately defined for refutation or empirical testing. Significantly, the potential role of fractal scaling as a control parameter is pivotal in theory-driven approaches aimed at comprehending empirical evidence concerning cascade dynamics [82]. This approach would also align with recent findings suggesting that multifractal dynamics within postural and suprapostural variability play a pivotal role in facilitating the subsequent response to visual stimuli and mechanical perturbations [59,56,60]. We posit that the concept of OFSCA propels us to a heightened analytical plane, making this notion of H as a control parameter testable. Its capability to render saddle-type and spiral-type topologies as tangible and testable structures within 2D fractal processes is particularly noteworthy.

The present work opens the door to a more detailed inquiry into the

efficacy of fractal scaling as a control parameter for postural synergies. It has not fully tested the notion of fractal scaling as a control parameter for intermittent postural control. However, it has used the novel analytical approach of the OFSCA to reveal an immense store of internal texture in the 2D distribution of fractal temporal correlations corresponding to the quiet standing of adults from different diagnostic groups and under various task constraints. Subsequent investigations could leverage the OFSCA framework across a broader spectrum of experimental manipulations. Furthermore, a comprehensive exploration of the OFSCA output using an expanded array of geometric tools would benefit a more thorough analysis. For instance, we could elaborate on these proposals of saddle-vs-spiral topologies or discern what other topologies our groups with diseased posture might embody, such as the curvature of the steepness of descent of the *H* space. The OFSCA might also make it possible for future analyses to model the 2D distribution of fractal scaling in tandem with the 2D distribution of CoP. Such comparison could offer a rich means for testing any relevance that *H* might have for intermittent postural control and its characteristic topologies. A longer-range goal would be learning how best to reconcile and integrate OFSCA-type multifractal results with the prior and more prevalent case of multifractal results for univariate time series. The former describes an angular variety of fractal scaling. In contrast, the latter describes a variety of fractal scaling across time or fluctuation size.

Data availability

Data for individuals with Parkinson's disease, Dataset 1, were obtained from a publicly available gait dataset [25] (https://doi.org/10.6084/m9.figshare.13530587), and data for healthy younger and older adults, Dataset 2, were obtained from another publicly available dataset [29] (https://doi.org/10.6084/m9.figshare.4525082). Data for individuals with Stargardt's syndrome, Dataset 3, were obtained from another publicly available dataset [111]. Nevertheless, the link associated with this publication has expired; however, it can be obtained directly from the corresponding author. Data for healthy individuals from Lee et al. [75] and Furmanek et al.[35], Datasets 4 and 5, respectively, are not publicly available and can be obtained from the corresponding author Madhur Mangalam (`mmangalam@unomaha.edu`) upon reasonable request.

OFSCA code availability

The codes and instructions for implementing the OFSCA method can be accessed on https://osf.io/tcynf/. Any technical inquiries related to code usage or troubleshooting can be directed to the corresponding authors, Madhur Mangalam (`mmangalam@unomaha.edu`) or Ken Kiyono (`kiyono.ken.es@osaka-u.ac.jp`).

CRediT authorship contribution statement

Damian G. Kelty-Stephen: Validation, Formal analysis, Writing - original draft, Writing - review & editing. **Ken Kiyono:** Conceptualization, Methodology, Software, Writing - review & editing. **Nick Stergiou:** Writing - original draft, Writing - review & editing. **Madhur Mangalam:** Conceptualization, Methodology, Software, Validation, Formal analysis, Writing - original draft, Writing - review & editing, Visualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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