Voluntary performance

Guy Van Orden

Center for Cognition, Action, and Perception, Department of Psychology, University of Cincinnati, USA

Key words: *intentionality; fractal behavior; dynamical disease; mind and body; self-control.*

Summary. *Will, purpose, and volition have long been viewed as either causes of behavior or of no direct consequence to behavior. In this essay, volition affects a flexible direct coupling of participant to task, modulating the degrees of freedom for kinematics in action, a point of view first introduced in theories of motor coordination. The consequence is an explanation consistent with present knowledge about involuntary and voluntary sources of control in human performance, and also the changes of the body expressed in aging and dynamical disease. Specifically, this view explains how tradeoffs between sources of overly regular versus overly random dynamics change the structure of variability in repeated measurements of voluntary performance.*

Introduction

Voluntary control is a willful control of behavior, distinguishing a *wink* from a *blink*, as famously illustrated by Alicia Juarrero (1). One legacy of 20th century cognitive and behavioral science was its widespread failure to accommodate concepts like willful control, purpose, or volition; lacking a reasonable scientific understanding of these concepts as a basis for control of human activities. Yet, ordinary human experience requires these concepts to make sense of acting with purpose, achieving one's goals, or the spontaneous intention to reach out and touch someone. In addition, society at large sets these ideas in stone, judging the intentionality of actions that may bring a person before the law, as in a judgment of *involuntary manslaughter*.

The culprits of the widespread failure are the elaborate faux-causal methods and analyses, based on the general linear model, that scaffold most empirical studies of human behavior. Assuming the general linear model, manipulations of behavior in experimental factors will appear to have sufficient causal powers to produce their reported effects – so we talk about the effects of psychological factors. However, this causal inference forgets a very old lesson of psychology: first and foremost, the necessary source of the data from a laboratory performance is a willing participant's intention to participate (2). Laboratory studies of cognitive and motor activities require a participant to take on the experimenter's instructions as intentions in performing the task at hand (3). So all data speak to human intentions and initiative, originat-ing as they do in each participant's intentions to perform the task as instructed.

Consequently, the intentional nature of voluntary performance must have been there all along, waiting to be discovered in the details of variability of the laboratory data. An emphasis on close examination of the details of the data is an emphasis that all scientists share, which is why behavioral scientists study laboratory measurements of behavior so closely, becoming experts in the process, in descriptive and inferential statistics to analyze and explain the observed variability in measurements. This variability in the data is the thing explained by the theories offered about human performance. In fact, a scientist who says the words *behavior* or *performance* is actually talking about changes in some particular measurements, the details of variability in behavioral measurements.

To this center of scientific gravity came the discovery that the variation in repeatedly measured human behavior is scale-free, meaning that the magnitude of variation is proportional to the scale on which it is measured (see Fig. 1). Scale-free variation lacks a stable central tendency because variance grows with sample size, bringing into question previous inferences relying exclusively on observed means (4). Scale-free variation is understood using fractal geometry and it is called *fractal time*, *fractal behavior*, and *fractal noise* due to that fact. I will use an equivalent term *pink noise* from this point forward, however, because it is a better fit to the upcoming illustrations. The term *pink noise* comes from a resemblance between the spectral portraits of fractal noise to those of pink light,

Correspondence to G. Van Orden, Center for Cognition, Action, and Perception, Department of Psychology, University of Cincinnati, Cincinnati, OH 45221-0376, USA
E-mail: guy.van.orden@uc.edu

Adresas susirašinėti: G. Van Orden, Center for Cognition, Action, and Perception, Department of Psychology, University of Cincinnati, Cincinnati, OH 45221-0376, USA
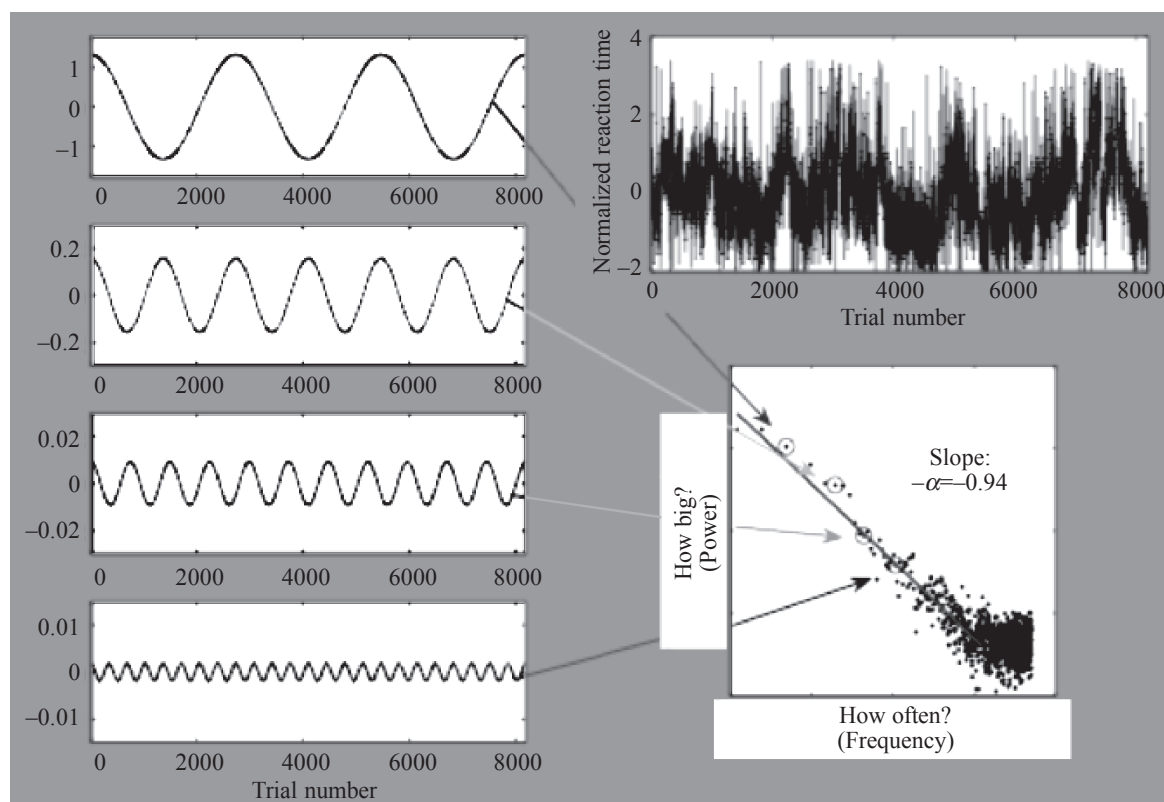El. paštas: guy.van.orden@uc.edu

Fig. 1. One person's response time data

The left side of the figure presents specific frequencies and amplitudes of the regular sine waves that approximate the irregular aperiodic data series (in the upper right of the figure). The arrows connect the sine waves to corresponding points of the spectral plot. Each point plotted represents a particular size or amplitude of change (power) across the data values and how often changes of that size occur (frequency). The spectral slope $=-0.94$, which is approximately $\alpha\approx1$. Note that the y-axes in the sine wave illustrations have been adjusted to make smaller amplitude sine waves visible.

more power in the lower frequency "red" regions, less power in the higher frequency "blue" regions.

Fractal geometry is the mathematics of heartbeats, galaxies, trees, lightning, nervous systems, arteries and capillaries, and it is the mathematics of variation in the repeated measurements of human behavior. Human performance is fractal, whether it is called cognitive or motor, physiological or behavioral. Fractal geometry is necessary to understand the variability in fractal data, in which the "noise" is the informative signal of human performance. A human performance is any task-related behavior that can be measured repeatedly; for example, the repeatedly measured step lengths of human gait on a treadmill (5), or a repeatedly measured amplitude at a particular frequency in identically repeated speech (6), or in the repeated estimates of when a second has passed or estimates of an inch produced in drawn line lengths (7).

Fractal behavior as a fractal pink noise is often estimated using a spectral plot. To construct a spectral plot, begin by decomposing a time-ordered data series into sine waves of different amplitudes (see Fig. 1).

Slow large changes in the data series are captured by the slow-frequency large-amplitude sine waves (top left of Fig. 1), and fast changes are captured by fast-frequency small-amplitude waves (bottom left of Fig. 1). The amplitude or *power* (amplitude squared) concerns the size of particular changes S(f) and appears on the y-axis of the power spectrum. Size of change S(f) is plotted against the frequency (f) of changes, which is also an estimate of how often changes of that size occur (on log-log scales). The slope of the regression line between how often (f) and how big S(f) in the spectral plot estimates the scaling relation between size and frequency of change. In Fig. 1, the size of change S(f) is inversely proportional to its frequency (f): $S(f)=1/f^{\alpha}=f^{-\alpha}$, with scaling exponent $\alpha\approx1$, the scaling exponent of pink noise.

The first reports of the fractal patterns in human performance, in the United States at least, came to be known as *Bills' Blocks* after the psychologist, Arthur Bills, from the University of Cincinnati (see Fig. 2). Professor Bills could not have known what he had discovered; fractal geometry did not exist then as a

Normal (20.9% Oxygen) Record:
Subj: Geldreich   Time Responses In Color Naming

15% Oxygen Record:

12% Oxygen Record:

10.5% Oxygen Record:

8.8% Oxygen Record:
Subj: Geldreich
Break-down after 20 minutes
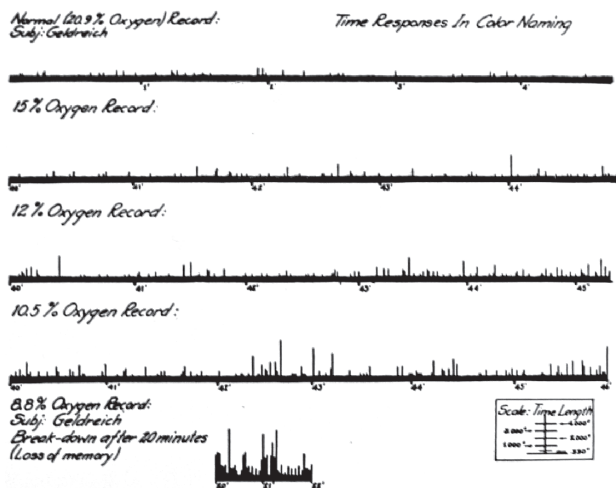(Loss of memory)   Scale: Time Length

PLATE 4.—Showing the increase in length and frequency of blocks as the oxygen supply is reduced. Each vertical line represents the time taken for one response. The long vertical lines are blocks. The short horizontal line at the bottom, showing almost continuous blocking, was terminated by loss of consciousness. (From an unpublished study by Geldreich and Bills.)

Fig. 2. Bills' Blocks are illustrated in this Plate 4 from (8) portraying data from a color-naming task as the oxygen supplied to a participant is reduced

By the term *blocks*, Bills meant extreme response times, which he imagined to imply that the capacity of the participant to respond was prevented, or blocked, as though by a barrier to responding. In the Plate, the blocks are vertical lines that correspond to extreme response times, rising from the horizontal lines as though on a graph with a y-axis of response time. The numbers on the x-axes refer to the number of minutes into the recording of color naming performance. The illegible numbers in the text box labeled: Scale: Time Length are 1000 and 3000 on the left and 350, 2000, and 4000 on the right, which presumably correspond to milliseconds.

frame of reference. Nonetheless, the pattern he described was notably intermittent, and he described a nested wavy pattern of waxing and waning performance in the repeatedly measured task performances. Bills and his contemporaries also noticed several facts about the pattern that remain patently counterintuitive, such as that larger samples amplify the wavy pattern and variance, which is the basis of the fractal properties (8).

Bills' interpretation of the pattern was understandably incorrect relying as he did on the intuitive sums of waxing and waning processes of the body and mind, which he believed would "block" or delay a laboratory response (8, 9). Yet, similar interpretations of this fractal behavior have been offered at some point in every contemporary discipline that has confronted this fractal behavior. For instance, a few years ago, I saw Benoit Mandelbrot's speak about fractal behavior at the U.S. National Science Foundation. The first question from the audience was whether the fractal patterns could not be captured by simple sums of component patterns! My anecdote is only noteworthy in this

context because Mandelbrot (10, 11) had much earlier spelled out the paradoxes that inevitably follows on this kind of solution; some examples of these paradoxes, as they appear in human performance data, are illustrated in Van Orden et al. (12).

Arthur Bills made his discovery in the early decades of the 20th century, and in the final decades of that century, the reports of fractal patterns began to trickle in. Presently, the fractal pattern of pink noise has been observed in virtually every kind of conventional laboratory performance prompting some scientists to put forward the idea of universality (13–17). The reviews cited in this essay span classic human performances including almost all of, or at least a representative sample of, the 150 years of textbook response-time tasks inherited by cognitive psychology – any task that has presented a stimulus to which participants responded, consistent with task instructions, yielding a "trial" response time, the outermost estimate of the duration in time of response processes. However, there are conceptual difficulties to overcome before a widespread acceptance that human behavior is fundamentally fractal behavior.

The conceptual dilemma, at the heart of the difficulties, is that pink noise is simultaneously a regular and irregular pattern, which is a recently introduced concept for science at large and an entirely novel concept for the behavioral sciences, so novel that it even contradicts the conventional statistical axioms (17, 18) – in other words, a tough pill to swallow for any scientist. On one horn of the conceptual dilemma is the highly regular spectral slope of a power spectrum, characterizing the scaling relation. It is tempting, and consistent with a long tradition, to imagine that the separate points that compose the slope of the power spectrum, in fact, correspond to separate processes, differing in their amplitude and frequency of variation. This common mistake ignores the fact of the regular spectral slope itself; however, a slope that changes in a coherent unity, keeping its component points in line, even as a manipulation causes it to get steeper or to become shallower (19). On the other horn of the dilemma, pink noise as it appears in a time-ordered data series is an aperiodic, highly irregular waveform, the product of complexity and nonlinear dynamics. In truth, though pink noise is neither regular nor irregular, and it is both extremes simultaneously. This conundrum exists because pink noise is a third kind of phenomenon, different from both regular and random (12, 20–22).

Yet, what does pink noise say about voluntary control of behavior? For one thing, the universality of fractal behavior in human performance and other

system behaviors gives credence to the complexity thesis that common dynamical organizations will appear in systems of different material construction, in living as well as nonliving matter. Even if true though the complexity thesis does little to satisfy curiosity about how pink noise figures in voluntary control of human behavior. To satisfy this curiosity, Geoff Hollis, Heidi Kloos, Sebastian Wallot, and me attempted to synthesize the previous reports of fractal behavior in human performance within a single control parameter of voluntary performance. This essay describes the resulting synthesis and the data that motivated it (see also 12, 23, 24).

A control parameter of variation

The observed noise is not identical in every human performance. In more difficult or less familiar tasks, the variation in the data can depart from the pink noise complexity, sometimes toward a random pattern of white noise with a flat spectral slope and a scaling exponent α=0 (e.g., 25–30). Difficulty and novelty may, in this instance, be two ways (among others) of injecting unsystematic change into human performance, from one trial to the next. Unsystematic changes in trial-to-trial task demands are sources of unsystematic perturbations to measurements. Unsystematic perturbations change a spectral analysis in the direction of random noise (19). However, not all examples of the effects of task demands are so intuitive, as scaling exponent α's can range between the α=0, flat, spectral slope of white noise and the α=2 steeper spectral slope of Brownian noise, at least (see Fig. 3).

These reliable changes are powerfully constraining how to think about pink noise behavior, greatly reducing the wiggle room for a theory of fractal behavior in voluntary performance. In addition, it has long been a dream to tell the same story for all kinds of behavior, whether the changes in the fractal pattern come from changes in motor coordination or cognitive activity. Our goal is such a unifying principle, a universal principle, bridging the dualist convention of distinctions between the body and the mind in task performances.

For instance, motor task performance is often discussed as though it refers primarily or even exclusively to the more material aspects of the mind-body interaction, and cognitive activity as though it refers to mental aspects alone. The distinction has for a long time appeared arbitrary however. All human performances are the intentional actions of participating human beings, as when a participant takes on the experimenter's instructions as his/her mental intentions about how to perform a "motor" task, for instance,
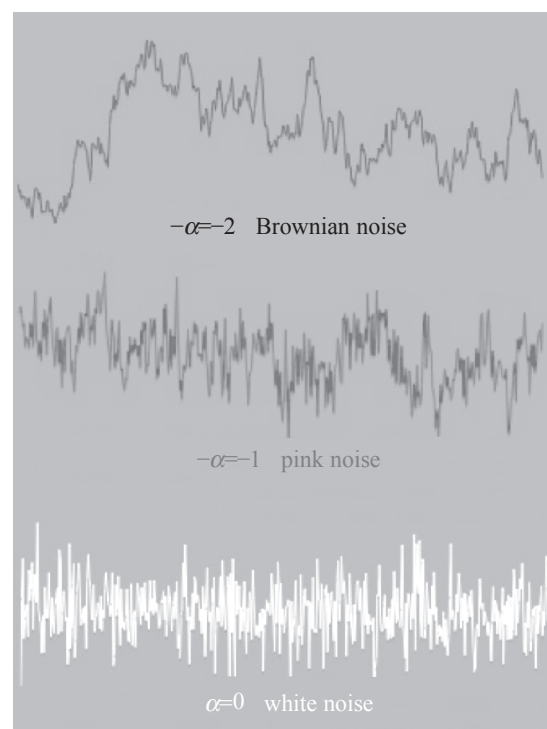

Fig. 3. Illustrations of overly regular variation in behavior as Brownian noise, overly random variation in behavior as white noise, and the third kind of variation, pink noise

and all laboratory performances require motor coordination of measurable behavior to produce "mental" data. Consequently, unless body and mind are strictly independent in how they interact, which they are not, how could we get the mind out of the motor or the motor out of the mind?

Concerning the control parameter, though, after several false starts, it appeared that tradeoffs within a kind of ratio between voluntary and involuntary control could give an adequate account of the available data (12). Sources of voluntary control became a denominator of sorts, and sources of involuntary control became a numerator, and all sources of control were evaluated with respect to the degrees of freedom (DoF) afforded by a task environment. In this idealized ratio, voluntary and involuntary constraints reduce the DoF available for behavior to complement the controllable DoF of the task environment, insuring that behavioral kinematics successfully performs the task (31).

The idea for a ratio did not come out of the blue. It was inspired by work in physics on *self-organized criticality*. The guiding metaphor in physics came from intuitions of Per Bak and his colleagues about sand piles that self-organize critical behavior in the size and timing of avalanches (32, 33). A critical state is something like an up-coming choice not yet made, an unbroken symmetry among poised options, a limbo

of potentialities. Critical behavior is predicted near critical states of the sand pile, in which critical behavior is seen in the time between the occurrences (the frequency of occurrences) of large and small avalanches, portrayed in a spectral plot, yielding a scaling relation like the $\alpha \approx 1$ scaling relation visible in Fig. 1.

The theoretical work concerning self-organized criticality was done imagining avalanches of sand, but the first successful recipe for observing the predicted critical behavior included rice kernels, chosen to have an aspect ratio favoring length over width (34). In the model system of the rice pile, extra long kernels of rice created extra friction between kernels, allowing the slowly growing rice pile to build up elaborate structure – one kernel added at a time.

Small local piles of rice within the growing pile would each build up to a point that one more fortuitous kernel would topple the small pile in a small local avalanche. The extra friction between extra long kernels of rice made these local tipping-points common, building local piles throughout the rice pile all poised at thresholds to topple. Once enough local piles became poised together at threshold, rare and extremely large landslides became possible, as predicted for critical behavior (i.e., corresponding to points in a spectral plot like those in the upper left quadrant of the spectral plot in Fig. 1).

A ratio was inspired by the Reynolds numbers of fluid dynamics and heat transfer that plus a contrast between the rice pile successes and the reported sand pile failures (35). In the failed attempts, sand piles did not build sufficiently structured local piles, and avalanches never became of sufficient magnitude to include extreme avalanches and satisfy the scaling relation. The aspect ratio of grains of sand yielded too little friction to compensate for the inertia of grains falling to the pile. Consequently sand pile avalanche behavior was overly random, dominated by the inertial behavior of grains of sand.

Reynolds numbers are a ratio of inertia over viscosity/friction (36), and we proposed that the control parameter of avalanche behavior is also a ratio of inertia over friction, illustrated in Formula (1). Too much inertia yielded overly random sand pile avalanches, while too much friction would yield overly regular avalanches (as in mudslides perhaps). Only a proper balance of inertia to friction yielded the fractal avalanche behavior of the rice pile, predicted by the hypothesis of self-organized criticality.

Variation in avalanche behavior:

$$\frac{\text{Overly random}}{\text{Overly regular}} = \frac{\text{Inertia}}{\text{Friction}} \qquad (1)$$

The extended analogy between the control parameters of fluid dynamics, avalanche behavior, and human performance is represented by Formula (2). The respective numerators are equated as sources of overly random variation and denominators are equated as sources of overly regular variation. For human performance, overly random variation is further equated with *fractional Gaussian noise* and scaling exponents between 0 and 1. Likewise, overly regular variation is equated with *fractional Brownian motion* and scaling exponents between 1 and 2 (compare Fig. 3).

Variation in human performance:

$$\frac{\text{Overly random}}{\text{Overly regular}} = \frac{\text{Involuntary control}}{\text{Voluntary control}} \qquad (2)$$

As the source of overly random variation, a task environment supplies the overall available DoF, which will be independent of and more numerous than the available DoF that the movements of the body can exploit. Consider the task of tapping a key to match a metronome beat; the DoF that the task makes available are indifferent to the participant unless by design. It matters not at all to the task environment whether the tap that presses a key to match a metronome beat comes from a person's finger or from a mindless pebble bounced off a passing truck. The measurement outcomes will be the same, so long as successive taps or passing trucks are spaced the same in time.

Embodied control and fractal behavior

In human performance, embodied control divides between voluntary or involuntary sources, both of which reduce the task environment DoF to the available DoF for possible motor kinematics. Some embodied constraints reflect lasting relationships among the body's components. For example, lasting constraints insure that a finger cannot take an excursion, by itself and separate from the hand, up around the head and down the spine along its pathway to tap a key. The range of motion for tapping is limited by the relation between finger and hand, and among finger joints, muscles, fasciae, and the nervous system, limiting each finger to a range of motion to move in some ways but not in others. The DoF of muscles are also linked such that the force that a given muscle length will generate depends on the muscle position within the context of neighboring muscles and connective tissues (37).

Synergies are temporary couplings among these constraints across the tensegrity structure of the body. Like the cytoarchitecture of a cell (38, 39), the human body is an integrated tensegrity structure (37). In the

body, skeletal struts coupled tautly by muscle and fascia cables allow force transmission at the speed of sound to tell each component of the body what the other components are doing. Zero-delay transmission of this information is manifest in "large and immediate restoring forces... by mechanical impedances from a... moving limb's stiffness and viscosity" (37, p. 686). Fluid performance by highly skilled actors requires synergies that are perpetually integrated tailor-made suits, so to speak, with respect to how well the kinematics of the body will exploit available DoF to meet the flow of changes in task demands.

Synergies transform the high-dimension noise of the task environment into low-dimension white noise, also limited in dimension by measurement itself. In the tapping task, for instance, embodied synergies limit the DoF to become the kinematics that keeps the beat. Residual sources of variation and perturbation, present in the task environment, fluctuate across measurement trials as random variables. These sources of overly random control are sources of involuntary control, although embodied sources of involuntary control reduce (but cannot eliminate) the DoF of random variation in behavioral measurements.

Sources of voluntary control are different. Voluntary control tightens or loosens the coupling between task and participant (beyond mindless entrainment). Voluntary control is also a temporary and flexible source of control, bringing constraints into and out of existence, as task performance requires. However, the changes in voluntary control unfold more slowly than the kinematics of measured behavior, so sources of voluntary control can exaggerate overly regular, slower oscillations of variation across the measured trial values. The slower timescales of voluntary control amplify more slowly changing variation to the data, which on that basis can sometimes resemble Brownian noise with $\alpha \approx 2$ in the spectral portrait.

In this view of human performance, the pink noise scaling relation comes about because changes in any part of the mind and body are coupled to changes in every other part. The components of mind and body are coupled in *interaction-dominant dynamics* via positive feedback loops, insuring that the changes of each component are reflected throughout and insuring that every component knows at the same time how the others are changing (17, 37, 40–43). Fractal behavior emerges in the frustrated compromise among the coupled components. Compromise is perpetually frustrated by components' tendencies to change independently of one another – the tendency toward overly random behavior – and each component's

tendency to dominate the dynamics of the system as a whole – the tendency toward overly regular behavior. Statistically self-similar, aperiodic, fractal, pink noise falls out of this stream of frustrated compromises due to the overly random and overly regular nature of interaction-dominant dynamics.

The ratio that defines the value of a control parameter implies that any single spectral plot or scaling exponent will be ambiguous, due to the simple fact of it being the value of a ratio. The ratio of voluntary and involuntary constraints makes this ambiguity explicit because summary changes in uncontrolled DoF could be due to specific changes in either the numerator or the denominator. Specific changes in the numerator alone or specific changes in the denominator alone, either one, can move the observed variation away from pink noise or toward pink noise. For instance, all other things equal, adding uncontrolled DoF to a numerator increases disorder in the coordination and moves the scaling exponent toward $\alpha \approx 0$, but so does decreasing the controllable DoF of a denominator.

Variation in scaling exponents

Positive feedback and interaction-dominant dynamics also dictate the recipes for laboratory methods to produce pristine examples of pink noise. The best empirical demonstrations ($\alpha \approx 1$) have used repeated measures of simple task performances – repeated estimates of time intervals or spatial quantities, or the repeated articulation of the same word – otherwise holding constant the stimulus, the response, and other trial factors across all the measurement trials (28). Systematic or unsystematic differences among trials would otherwise be amplified in positive feedback. Amplified differences from trial to trial are trial-to-trial changes in the numerator of involuntary control, and perturbations to measured values. A method that purposefully builds in unsystematic perturbations, for instance, increases uncontrolled DoF and moves the scaling exponent toward random white noise and $\alpha \approx 0$ (19).

The several decades of reports of scaling exponents in human performance have produced enough examples of changes in scaling exponents to provide a first glimpse of task and participant differences in fractal behavior. Unfortunately, only a few reports correspond to exaggerated voluntary control, as when a toddler locks out DoF in knees, hips, and torso to avoid falling while taking his/her first steps, and only gradually relaxes rigid voluntary control with age and experience (5), or as when a Parkinson's sufferer must exert

voluntary control to sustain upright posture (44). In these few cases, though, exaggerated voluntary control yielded scaling exponents different from the typical scaling exponents of gait or posture, changing in the predicted direction toward the $\alpha \approx 2$ of over regular Brownian noise.

Willful control of eye movements also produces scaling exponents closer to $\alpha \approx 2$ and Brownian noise. In this eye-movement task, participants moved their eyes from the center of a display to its periphery and back again, systematically moving their gaze around the display. Imagine moving your eyes around a clock face, returning the eyes back to the clock center after each hour on the clock – back and forth, hour to center, next hour to center, and so on. When the participants controlled the timing of their eye movements, the task emphasized voluntary control and produced overly regular variation in the participants' eye movements and scaling exponents nearer to $\alpha \approx 2$. In another condition, voluntary control was reduced as the experimenter controlled the timing of eye movements, which yielded scaling exponents closer to $\alpha \approx 1$ and pink noise (Sebastian Wallot and Charles Coey, personal communication, July 27, 2010).

Tradeoffs between voluntary control and involuntary control are also observed using manipulations of involuntary control, which are much more common. Laboratory experiments usually manipulate sources of involuntary control. In the example of a finger, tapping to a beat, the audible beat of the metronome is an exogenous task constraint that can entrain behavior, reducing task DoF and the need for voluntary control. Like all constraints, entrainment is defined in a relation, this time between a task demand (metronome beat) and a task participant (entrainable actor). Entrainment reduces the need for voluntary control, in turn, and increases the presence of overly random variation in tapping performance, which departs toward white noise with $\alpha \approx 0$. Variation moves back again toward pink noise and $\alpha \approx 1$ (45), if voluntary control is reintroduced, by tapping to a *remembered* beat without a metronome.

A different way to reintroduce voluntary control is to require syncopated tapping between the beats of the metronome. Syncopated tapping requires more voluntary control than synchronized tapping to resist the pull of the entrainment by the metronome beats, to keep the taps between the beats. Reintroducing the need for voluntary control increases the prominence of overly regular against overly random control. Correspondingly, instead of scaling exponents closer to $\alpha \approx 0$ and white noise, as in synchronized tapping to

the beat, the scaling exponents move back in the direction of $\alpha \approx 1$ and pink noise (45, 46).

The prominence of voluntary control can be changed in the same way in human gait. A metronome entrains gait and reduces the need for voluntary control, so the scaling exponent of step frequency changes in the direction of white noise with $\alpha \approx 0$ (47). Step length is unaffected by the metronome however (48). The reduction in voluntary control is specific to frequency of gait, because the metronome constrains frequency directly, impacting step length only indirectly, although one can imagine manipulations to entrain step length (49).

Even without a metronome, the preferred pace of adults on a treadmill is a pace indicating less voluntary control, compared to the pace of walking across ordinary terrain for instance. A scaling exponent closer to white noise indicates less voluntary control, and adult participants' treadmill gaits yield scaling exponents centered on a value closer to whiter variation than the scaling exponents for their nonpreferred gaits. Gaits slightly faster or slower than a preferred pace induce more voluntary control and move scaling exponents toward pink noise with $\alpha \approx 1$ (47). This is true for wide ranging deviations from the preferred treadmill pace, in both walking and running, and across a variety of measurements (*stride interval*, *stride length*, *step interval*, *step length*, and *impulse*; impulse = force × change in time), measured first at the preferred pace (50, 51).

Accuracy feedback is another way to reduce the demands of voluntary control. Task feedback is a source of involuntary control – like a metronome beat – reducing the need for voluntary control. In line with this fact, accuracy feedback provided in a time estimation task following each trial's time-estimate yielded scaling exponents closer to the $\alpha \approx 0$ of white noise, compared to time estimation without feedback (Nikita Kuznetsov and Sebastian Wallot, personal communication, December 15, 2009). Time estimation without feedback typically yields scaling exponents close to the $\alpha \approx 1$ of pink noise.

Over-training can enhance involuntary control. An elite ballerina over-trains to insure her torso to remain upright over her body's center of balance, creating the appearance in motion of gliding across the stage. Torso posture has a trainable basis in the endogenous constraints of the body and enhanced involuntary control from overtraining reduces the need for voluntary control. Variation in a dancer's posture is measured by deviations around a center-of-pressure on a force plate. Scaling exponents of elite dancers' posture

express reduced voluntary control with values closer to the $\alpha \approx 0$ of random white noise, when compared to the values of scaling exponents produced by ordinary adults or elite athletes who are not dancers (52).

Self-organized criticality

So far, I have described an ad hoc account of otherwise baffling results from the literature on fractal human performance (24). The previous data had stopped all the false starts we had made early on, in our attempt to formulate a control parameter. It is a fair question to ask of an ad hoc account how can we know with greater confidence that a task performance is the product of emergent coordination and the critical states of a complex system?

For instance, how would we recognize an instance of self-organized criticality? Self-organized criticality prominently features positive feedback, allowing minuscule changes to be amplified in their effects (53). This prominent role of positive feedback also tells us what kind of evidence should be present when a system is near criticality. In particular, the interdependence created by positive feedback in systems near to their critical states predicted explicitly the coherent fractal pattern of pink noise that has been observed so widely in human behavior.

Self-organized criticality predicts other phenomena as well. As discussed, critical states exist in symmetry (as unmade choices or potentialities). Symmetry implies that critical states are by nature unstable; the smallest relevant contingency will collapse the symmetry into one or another option. Unstable critical states are comparable to the *repellers* in chaos theory, which are impossible states. Yet, fractal behavior is common in nature. How can critical states be impossible and at the same time be so commonly observed in evidence as pink noise? The solution is that living systems actively acquire constraints and it is a balance among conflicting constraints that gives the appearance of being attracted toward an otherwise, repellant, critical state (32, 33).

Indirect evidence suggesting attraction to critical states was found in an experiment in which participants repeated the English word "bucket" aloud very many times (6). Each successive pronunciation of "bucket" was recorded, and each recording was parsed identically into dozens of frequency bins. The amplitude of each frequency bin could then be tracked across a participant's series of recorded pronunciations. This resulted in dozens of separate data series per participant, each with a spectral slope and scaling exponent. Aggregating the resulting scaling exponents in

a histogram revealed a normal Gaussian distribution around the scaling exponent value of $\alpha \approx 1$ (shifted slightly toward white noise). In other words, the "attractive" central tendency of variation in scaling exponents in a repetitive speech task was very close to pink noise.

Direct evidence of attraction to criticality came from an experiment that trained adult performance and from two cross-sectional studies of development. The training study observed adult performance in a Fitt's tracing task. In thousands of trials, participants traced a stylus between two dots on an electronic tablet, and the time required to trace from dot to dot was repeatedly measured. In the earliest practice block, the scaling exponents of the trace times were reliably below $\alpha \approx 1$, closer to $\alpha \approx 0$, but across the training blocks, the pattern of variation in performance showed the predicted attraction toward the scaling exponent $\alpha \approx 1$. Across five practice blocks, 5500 trace trials total, the central tendency for the participants' scaling exponents approached $\alpha \approx 1$ (54).

Evidence of self-organized criticality was also observed across a cross-section of ages of children who performed a temporal estimation task. In the task, a child pressed a button to estimate when a designated time interval had passed, yielding a data series of times between button presses. Scaling exponents of the data series showed a progression with age, from scaling exponents of overly random control in younger children, closer to $\alpha \approx 0$, and toward scaling exponents closer to pink noise and $\alpha \approx 1$ in older children and adults (55).

Human gait develops toward pink noise from the other direction. Young children produce scaling exponents for treadmill gait dispersed widely and including overly regular behavior, encroaching on $\alpha \approx 2$. Older children produce scaling exponents less widely dispersed, and teenage children and adults produce scaling exponents that are narrowly dispersed around the values observed in the preferred treadmill gaits of adults (5).

These studies of human development provide the necessary evidence to motivate a hypothesis of self-organized criticality. The observed attraction to critical states is attraction to the best of both kinds of control, combining stability and flexibility to accommodate both familiar and surprising changes in our relation to the world around us. Compare this to the fact that overly rigid control will produce stable behavior in a highly predictable environment but will perseverate, pathologically, as the environment becomes less predictable. Or that overly random control will make

flexible changes in a less predictable environment but will fail to track predictable regularities.

Parkinson's and other dynamical diseases

Development and training reveal human performance attracted over time toward criticality, sometimes resulting in more constrained and skilled voluntary control and sometimes resulting in accrued constraints that relax over-zealous voluntary control. Enhanced voluntary control emerges at the scale of willful organisms interacting with each other and with the objects of their worlds, within the contexts and events composing lifetimes. Overly willful control of task performances in healthy participants produces overly regular variation and scaling exponents near $\alpha \approx 2$. Generally, the $\alpha \approx 2$ of Brownian noise coincides with a loss of flexibility in dynamics, an emerging dominance of system dynamics by more slowly changing constraints. Congestive heart disease is an example, as congested arteries rigidly constrain the dynamics of blood flow and heartbeats (see Fig. 4).

Parkinson's disease is also a loss of flexibility in overly regular control. Equating voluntary control with constraints that reduce DoF in behavior explains the

Learning and Memory Decline (56–58)
Deficit in Lying/Inability to Follow Instructions (59)
Comprehension/Attention Deficits (60, 61)
Browner Variation in Physical Activity (44, 62)

Frozen Movement (77)

Odor Descrimination (63)
Deficits in Color Perception (64)
Dysfluent Word Pronunciations (65)
Deficits in Prosody Productions and Perception of Emotional Tone (66, 67)
Vowel Production Deficits (68, 69)
Limb Tremor (70, 71)
Vocal Tremor (68, 69)
Deficit in Gait (72–75)
Brown EEG patterns (68, 69)
Brown Heart Beat (76)
Brown Variation in Physical Activity/Posture (44, 62)

Power

Slow ← Timescale of Constraint → Fast

Ordered Schematic of Progressive Dysfunction in Parkinson's Disease

Fig. 4. Approximate progression of Parkinson's disease estimated from the cited descriptions of patients with pink noise and brown noise scaling relations as a backdrop. Parkinson's first erodes the capacity for change on the fastest timescales and then intermediate and slow changing timescales. Eventually sufferers appear frozen in time although they continue to move on the timescales of very slowly changing constraints

widely observed Parkinson's symptom in which palsy tremor is visibly dampened by voluntary control, eliminating the tremor early in Parkinson's disease and at least partly damping the tremor oscillations in later stages. The bodies of Parkinson's patients no longer produce smooth kinematics, and patients have difficulty initiating and controlling motion. I mention the Parkinson's symptoms in particular because a complexity account of Parkinson's symptoms is plausible and because the conventional story of Parkinson's is riddled with gaps, originating as it does in the gradual decline of areas of the brain that produce the neurotransmitter dopamine (4, 12).

For instance, why are fine-grain capacities most vulnerable early in Parkinson's disease? The essential neural conduction among modules is intact in Parkinson's disease, and the conduction rate across neurons is plenty fast to move fast-changing information throughout the nervous system. Why then do the early Parkinson's symptoms include the disruption of the faster and finer aspects of perception and action? On top of that, how do the gradual changes in dopamine availability produce the qualitative changes in mobility and perception? Moreover, why does Parkinson's disease erode cognition along with mobility and why do the cognitive symptoms appear to be idiopathic? Conventional stories uncritically blame the missing "functions" on the missing dopamine (78), appealing to the superficial faux-causal logic that failed to recognize intentionality (79).

The complexity explanation is subtle and compelling. The emphasis shifts from a "faulty isolated component" (such as a faulty dopamine-uptake system) to the reduced coupling among the components and the erosion of the system-wide capacities to flexibly coordinate the mind and the body with the environment (80–82). Like any neurotransmitter, dopamine bridges the synaptic gaps between the neurons to perpetuate the electrochemical waves of action potentials. Action potentials create feedback loops of neuronal activity and larger traveling waves. Traveling waves self-organize across the cortex; they are an observable realization in the brain of the emergent motor coordination, perception, and cognition (23, 83–86).

Damage that reduces the dopamine in the brain reduces the capacity to coordinate the traveling waves reflecting a broader deficit in coordinating cognitive and motor activities as well as the dynamics of physiology. However, Parkinson's disease is systematically progressive, as Fig. 5 illustrates. The most vulnerable constraints early on are those that change on the fastest timescales, the dynamical range expressing
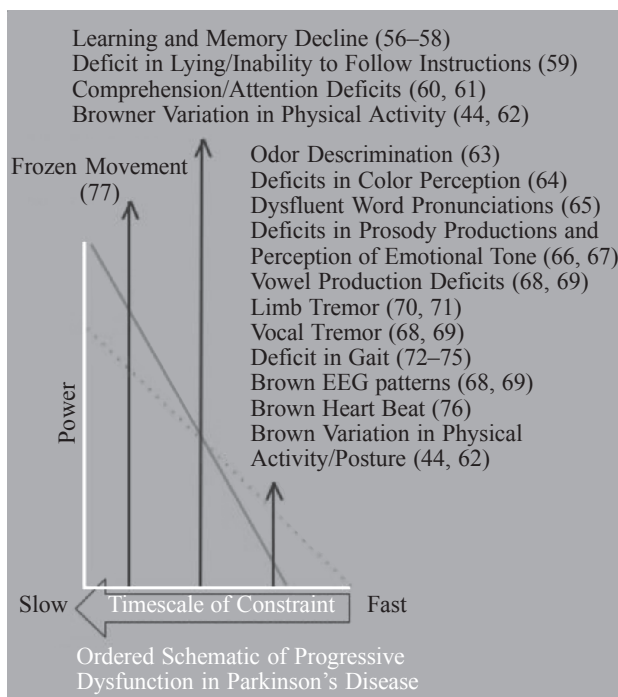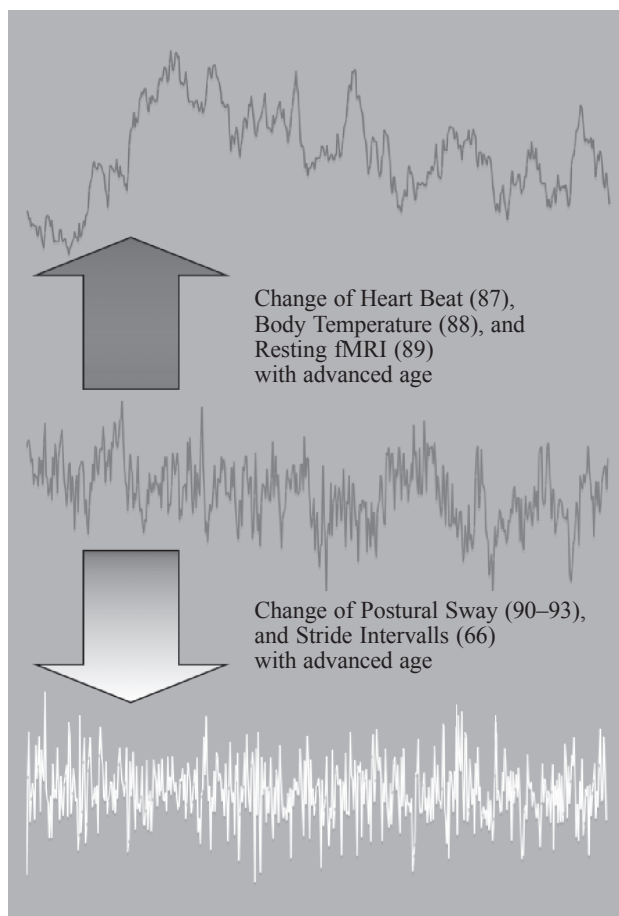
Fig. 5. Departures from complexity and $\alpha \approx 1$
due to advanced age

the late-stage Parkinson's sufferers can appear to be frozen in place. In truth, they are moving still, on the glacial timescale of their remaining capacity to change behavior.

The coarser-grain of slower timescale willful control is thus revealed in Parkinson's coarse-graining of relevant constraints. With the increased needs for voluntary control, the variation and scaling exponents of gait, arm movements, and speech all depart from healthy complexity toward the $\alpha \approx 2$ of Brownian noise and overly regular control. A ranked distance from $\alpha \approx 1$ and toward $\alpha \approx 2$ predicts very well the severity of all other Parkinson's symptoms (62).

Lost cognitive capacities appear idiopathic, which is the fancy way a scientist says, "I do not know why." Yet, cognition, more than perception-action, expresses idiosyncratic contingencies of a person's history. Contingencies of history, education, language, work, hobbies, travel, and health shape the strengths and stabilities of cognitive capacities well before Parkinson's disease begins. Parkinson's disease dissects this maze of idiosyncratic strengths and stabilities of history and interest, with its scalpel of instability and frequency of change. Slower timescales of cognition, including volition, are relatively preserved throughout, while faster timescale cognition, including the subtler shades of interpersonal dynamics, are more vulnerable. The protracted unraveling of timescales eventually erodes altogether the coordination among the brain, the body, and the world, eroding the mind as collateral damage.

Advanced age and its associated dynamical diseases exaggerate involuntary control and overly random behavior. Involuntary control, as the name suggests, is defined by default, inclusive of all sources of control except voluntary control. This includes sources of control within the systems of the body that, taken on their own terms and on their own timescales retell the present fractal story of tradeoffs between overly regular and overly random behavior. In atrial fibrillation, a rare form of heart disease, the scaling exponents of heartbeats depart toward $\alpha \approx 0$ and the overly random behavior of white noise (4). With ordinary advanced age, the scaling exponents of posture and gait also depart toward $\alpha \approx 0$, as does gait in Huntington's disease. In addition, as was the case for Parkinson's disease, the ranked distance from $\alpha \approx 1$ toward $\alpha \approx 0$ predicts as well the severity of all other Huntington's symptoms (94). These departures from complexity are summarized in Fig. 4.

Overly random white noise suggests loss of structure in dynamics, the presence of unsystematic perturbations among the organ systems, and a reduced

the smallest amplitude and least power in the spectral portrait. If the amplitude of dynamics gauges the resilience of dynamics as the capacity for coordination unravels, then it gauges the vulnerability of fast-changing constraints as well. Fast-changing constraints are the constraints that come from small and subtle changes in an emotional tone or in social alliances, the constraints that support fine-grained perceptual distinctions, and the constraints that are necessary to initiate sudden or rapid movements. In other words, Parkinson's disease first destabilizes the capacity for subtle, fine-grain, and fast-moving self-organization of perception, action, and cognition.

Erosion of constraints on fast timescales also explains the unwelcome palsy in Parkinson's disease. The palsy symbolizes a less refined, less precise coordination in control. The eroded capacity to simply bring the hands to rest is due to a lack of the fine-grain, fast changing, involuntary, "dithering" control that would ordinarily insure a stable resting point. Parkinson's disease eventually erodes intermediate and slow timescale capacities for change as well, such that

capacity for coupling among the organ systems. The extreme failure of coupling dynamics may explain the catastrophic cascade of failing organ systems in multiple-organ dysfunction syndrome – the usual cause of death in a hospital's intensive care unit. In multiple-organ dysfunction syndrome, the organs of the body seem to separate from each other in the sense that they no longer support each other's functions. An overly random perturbation of a system's interaction-dominant dynamics among its component processes would also collapse the interdependence among the component systems, compromising "the well-being of the body's system-of-systems" (4, p. 311).

Summary conclusions

Vast sums in many currencies have been spent in recent years to finance a quest for reliable correlates of mental functions in the images of brain activity. Success in this quest would require straightforwardly that the basis functions of behavior also exist in the brain (79, 95). And yet, the methods to parse behavior and brain piecewise, into commensurate basis functions, stem from the same faux-causal logic that previously closed the door to the willful control that distinguishes *winks* from *blinks* (1). And in any case, "Which parts of the brain correlate with what kinds of behavior?" is the wrong question to start with.

In a system analysis, the first question to ask is, "How do the components of the system interact?" (86, 96–98). The answer to this question decides what kind of research efforts will be appropriate from there on out, everything from the designs of experiments to the tools of the data analyses (19, 40, 96, 99). Fractal behavior provides a conclusive answer to how the components of a system interact. They change each other's dynamics in their interaction, while respecting the constraints available to their interaction.

It is a good bet then that the brain and the body coalesce in interaction-dominant dynamics, given the ubiquity of fractal dynamics in human performance. Ergo a person allowed willful control over what to think, while his or her brain is imaged, shows a brain busy in fractal activity. Brain activity flickers with the same pink noise signal as behavior. And task demands push the signal toward overly random activity or in the opposite direction toward overly regular activity, just as they do in behavior (100, 101).

And yet most brain science still clings to a logic of piecewise functional decomposition – even work in neurodynamics – failing to fully respond to the meaning of the fractal behavior for the system's dynamics. In addition, most brain and behavioral scien-tists remain ignorant of reported multifractal dynamics, which will assuredly be widely explored (102, 103). Multifractal dynamics could even turn out to be the decisive counter-evidence to the 20th century faux-causal logic. Ihlen and Vereijken explain that multi-fractal dynamics supplies the conclusive evidence for interaction-dominant dynamics, which requires a generalization of causal logic to include interdependence and emergence (103).

Lastly, a contrast between the timescales of behavior versus the timescales of the brain upends the too long unexamined idea that the brain causes behavior. For the most part, brains change on timescales orders-of-magnitude faster than the timescales of behavior. In a control hierarchy, the components that change on the slower timescales supply the constraints to control the components changing on faster timescales (104–106). For example, the relatively slowly changing BOLD signal represents the relatively slowly changing metabolic constraints, which supply limits to possible brain activity on faster changing timescales (83).

Pursuing this idea, behavior must in some sense control the brain, which is not a too far-fetched notion. On-going behaviors have inertial properties, which supply constraints and stability to the fractal activity of the nervous system (107). And I noted already that brain activity is skewed toward overly regular or overly random activity by slower timescale changes in task demands, i.e., task behavior. Perhaps, then, the "functions" of behaviors are not inherited from the brain activity; it is the other way around. "Functions" emerge in the fine-grain details of the kinematics of the brain and behavior (37, 108–110), while coarser-grain brain functions do not exist. The so-called brain functions exist only fleetingly in the flow of behavior's entailments at the fractal interface of the nervous system and world.

Behave as instructed encapsulates the single universal competence of voluntary performance. *Fractal behavior* is the performance universal that results. Scaling exponents of fractal behavior range widely nevertheless, spanning fractional Brownian motion and fractional Gaussian noise, at least. The directions of change that are observed inform us about tradeoffs between overly regular and overly random control, which reveal how development, skills, health, and wellness affect the coupling between task and person. Altogether, this essay has laid out a fresh wager on the tired question of how the mind and body interact. If the answer is to be found in human performance – if the question can be answered in the dynamics of the brain and body – then the answer is interaction-

dominant dynamics. Interaction-dominant dynamics unites body and mind, and mind and body.

Valingi judesiai

Guy Van Orden

*Cincinačio universiteto Psichologijos katedros Pažinimo, elgsenos ir suvokimo centras,
Jungtinės Amerikos Valstijos*

Raktažodžiai: tikslingumas, fraktalinė elgsena, dinaminė liga, protas ir kūnas, savikontrolė.

Santrauka. Valia, tikslas ir ketinimas ilgai buvo vertinami arba kaip elgsenos priežastis, arba kaip netiesioginiai elgsenos veiksniai. Požiūris, kad valia pasireiškia kaip adaptatyvi tiesioginė jungtis tarp subjekto ir užduoties, moduliuojanti laisvės laipsnius kinematinėje veikloje, pirmą kartą buvo pasiūlytas motorinio koordinavimo teorijose. Pasekmė yra paaiškinimas, apimantis turimas žinias apie valingus ir nevalingus žmogaus judesio kontrolės šaltinius, taip pat apie kūno pokyčius, atsirandančius senėjimo ir ligos dinaminiuose procesuose. Tiksliau šis požiūris paaiškinamas kompromisu tarp pernelyg reguliarių ir pernelyg atsitiktinių šaltinių dinamikos, sąlygojančios kintamumo struktūros pokyčius pakartotinių valingo judesio matavimų metu.

References

1. Juarrero A. Dynamics in action: intentional behavior as a complex system. Cambridge (MA): MIT Press; 1999.
2. Lindworsky J. Experimental psychologie. (Experimental psychology.) translated by DeSilva HR. 1931. London: George Allen & Unwin Ltd.; 1923.
3. Vollmer F. The control of everyday behaviour. Theory Psychology 2001;11:637-54.
4. West BJ. Where medicine went wrong. Rediscovering the path to complexity. London: World Scientific; 2006.
5. Hausdorff JM, Zemany L, Peng C-K, Goldberger AL. Maturation of gait dynamics: stride-to-stride variability and its temporal organization in children. J Appl Physiol 1999;86: 1040-7.
6. Kello CT, Anderson GG, Holden JG, Van Orden G. The pervasiveness of 1/f scaling in speech reflects the metastable basis of cognition. Cogn Sci 2008;32:1217-31.
7. Gilden DL, Thornton T, Mallon MW. 1/f noise in human cognition. Science 1995;267:1837-9.
8. Bills AG. The psychology of efficiency. New York: Harper; 1943.
9. Bills AG. General experimental psychology. New York: Longmans, Green and Co.; 1934.
10. Mandelbrot BB. The fractal geometry of nature. San Francisco: Freeman; 1982.
11. Mandelbrot BB. Multifractals and 1/f noise. New York: Springer-Verlag; 1998.
12. Van Orden G, Kloos H, Wallot S. Living in the pink: intentionality, wellness, and complexity. In: Hooker C, editor. Philosophy of complex systems. Handbook of the philosophy of science. Vol. 10. Amsterdam: Elsevier. In press.
13. Gilden DL. Cognitive emissions of 1/f noise. Psychol Rev 2001;108:33-56.
14. Gilden DL. Global model analysis of cognitive variability. Cogn Sci 2009;33:1441-67.
15. Kello CT, Van Orden G. Soft-assembly of sensorimotor function. Nonlinear Dynamics Psychol Life Sci 2009;13:57-78.
16. Riley MA, Turvey M T. Variability and determinism in motor behavior. J Mot Behav 2002;34:99-125.
17. Van Orden G, Holden JG, Turvey MT. Self-organization of cognitive performance. J Exp Psychol Gen 2003;132:331-50.
18. Brown C, Liebovitch L. Fractal analysis. Los Angeles: Sage; 2010.
19. Holden JG, Choi I, Amazeen PG, Van Orden G. Fractal 1/f dynamics suggest entanglement of measurement and human performance. J Exp Psychol Hum Percept Perform. In press.
20. Nicolis G, Rouvas-Nicolis C. Complex systems. Scholarpedia 2007;2:1473. [accessed April 24, 2009] Available from: URL: http://www. scholarpedia.org/article/Complex_systems
21. Sporns O. Complexity. Scholarpedia 2007;2:1623. [accessed April 24, 2009] Available from: URL: http://www.scholar pedia.org/article/Complexity
22. Tsonis AA. Randomicity: rules and randomness in the realm of the infinite. London: Imperial College Press; 2008.
23. Hollis G, Kloos H, Van Orden G. Origins of order in cognitive activity. In: Guastello SJ, Koopmans M, Pincus D, editors. Chaos and complexity in psychology. Cambridge: Cambridge University Press; 2009. p. 206-41.
24. Kloos, H, Van Orden G. Voluntary behavior in cognitive and motor tasks. Mind Matter 2010;8:19-43.
25. Correll J. 1/f noise and effort on implicit measures of bias. J Pers Soc Psychol 2008;94:48-59.
26. Clayton K, Frey BB. Studies of mental "noise". Nonlinear Dynamics Psychol Life Sci 1997;1:173-80.
27. Gilden DL. Fluctuations in the time required for elementary decisions. Psychol Sci 1997;8:296-301.

28. Kello CT, Beltz BC, Holden JG, Van Orden G. The emergent coordination of cognitive function. J Exp Psychol General 2007;136:551-68.
29. Kiefer AW, Riley MA, Shockley K, Villard S, Van Orden G. Walking changes the dynamics of cognitive estimates of time intervals. J Exp Psychol Hum Percept Perform 2009;35: 1532-41.
30. Ward L. Dynamical cognitive science. Cambridge (MA): MIT Press; 2002.
31. Bernstein NA. The coordination and regulation of movements. London: Pergamon Press; 1967.
32. Bak P. How nature works: the science of self-organized criticality. Oxford: Oxford University Press; 1997.
33. Bak P, Tang C, Wiesenfeld K. Self-organized criticality: an explanation of 1/f noise. Phys Rev Lett 1987;59:381-4.
34. Frette V, Christensen K, Malthe-Sorenssen A, Feder J, Jossang T, Meakin P. Avalanche dynamics in a pile of rice. Nature 1996;379:49-52.
35. Jensen HJ. Self-organized criticality. Emergent complex behavior in physical and biological systems. Cambridge: Cambridge University Press; 1998.
36. Iberall AS. On the general dynamics of systems. General Systems 1970;XV:7-13.
37. Ingber DE. Tensegrity I. Cell structure and hierarchical systems biology. J Cell Sci 2003;116:1157-73.
38. Ingber DE. Tensegrity II. How structural networks influence cellular information processing networks. J Cell Sci 2003;116: 1397-408.
39. Turvey MT. Action and perception at the level of synergies. Hum Mov Sci 2007;26:657-97.
40. Holden JG, Van Orden G, Turvey MT. Dispersion of response times reveals cognitive dynamics. Psychol Rev 2009;116:318-42.
41. Turvey MT, Moreno M. Physical metaphors for the mental lexicon. Ment Lex 2006;1:7-34.
42. Van Orden G, Holden JG. Intentional contents and self-control. Ecol Psychol 2002;14:87-109.
43. Van Orden G, Holden JG, Turvey MT. Human cognition and 1/f scaling. J Exp Psych General 2005;134:117-23.
44. Schmit JM, Riley MA, Dalvi A, Sahay A, Shear PK, Shockley K, et al. Deterministic center of pressure patterns characterize postural instability in Parkinson's disease. Exp Brain Res 2006;168:357-67.
45. Chen Y, Ding M, Kelso JAS. Origins of timing errors in human sensorimotor coordination. J Mot Behav 2001;33:3-8.
46. Deligniéres D, Torre K, Lemoine L. Long-range correlation in synchronization and syncopation tapping: a linear phase correction model. PLoS One 2009;4(11):e7822.
47. Hausdorff JM, Purdon PL, Peng C-K, Ladin Z, Wei JY, Goldberger AL. Fractal dynamics of human gait: stability of long-range correlations in stride interval fluctuations. J Appl Physiol 1996;80:1448-57.
48. Terrier P, Turner V, Schutz Y. GPS analysis of human locomotion: further evidence for long-range correlations in stride-to-stride fluctuation of gait parameters. Hum Mov Sci 2005; 24:97-115.
49. Jordan K, Newell KM. The structure of variability in human walking and running is speed-dependent. Exerc Sport Sci Rev 2008;36:200-4.
50. Jordan K, Challis JH, Newell KM. Speed influences on the scaling behavior of gait cycle fluctuations during treadmill running. Hum Mov Sci 2007;26:87-102.
51. Jordan K, Challis JH, Newell KM. Walking speed influences on gait cycle variability. Gait Posture 2007;26:128-34.
52. Schmit JM, Regis D, Riley MA. Dynamic patterns of postural sway in ballet dancers and track athletes. Exp Brain Res 2005;163:370-8.
53. Camazine S, Deneubourg JL, Franks NR, Snweyd J, Theraulaz G, Bonabeau E. Self-organization in biological systems. Princeton (NJ): Princeton University Press; 2001.
54. Wijnants ML, Bosman AMT, Hasselman F, Cox RFA, Van Orden G. 1/f scaling in movement time changes with practice in precision aiming. Nonlinear Dynamics Psychol Life Sci 2009;13:79-98.
55. Kloos H, Kiefer AW, Gresham L, Shockley K, Riley MA, Van Orden G. Response time dynamics of children and adults. Proceedings of the 15th International Conference on Perception and Action; Minneapolis; 2009.
56. Allain H, Lieury A, Thomas V, Reymann JM, Gandon JM, Belliard S. Explicit and procedural memory in Parkinson's disease. Biomed Pharmacother 1995;49:179-86.
57. Howard LA, Binks MGB. The contribution of apraxic speech to working memory deficits in Parkinson's disease. Brain Lang 2000;74:269-88.
58. Price A, Shin JC. The impact of Parkinson's disease on sequence learning: perceptual pattern learning and executive function. Brain Cogn 2009;69:252-61.
59. Abe N, Fujii T, Hirayama K, Takeda A, Hosokai Y, Ishioka T, et al. Do Parkinsonian patients have trouble telling lies? The neurobiological basis of deceptive behaviour. Brain 2009; 132:1386-95.
60. Peron J, Vicente S, Leray E, Drapier S, Drapier D, Cohen R, et al. Are dopaminergic pathways involved in theory of mind? A study in Parkinson's disease. Neuropsychologia 2009; 47:406-14.
61. Grossman M, Kalmanson J, Bernhardt N, Morris J, Stern MB, Hurtig HI. Cognitive resource limitations during sentence comprehension in Parkinson's disease. Brain Lang 2000;73: 1-16.
62. Pan W, Ohashi K, Yamamoto Y, Kwak S. Power-law temporal autocorrelation of activity reflects severity of Parkinsonism. Mov Disord 2007;22:1308-13.
63. Double KL, Rowe DB, Hayes M, Chan DKY, Blackie J, Corbett A, et al. Identifying the pattern of olfactory deficits in Parkinson disease using the brief smell identification test. Arch Neurol 2003;60:545-9.
64. Diederich NJ, Raman R, Leurgans S, Goetz CG. Progressive worsening of spatial and chromatic processing deficits in Parkinson disease. Arch Neurol 2002;59:1249-52.
65. Goberman AM, Blomgren M, Metzger E. Characteristics of speech disfluency in Parkinson disease. J Neurolinguistics 2008:23:470-8.
66. Ariatti A, Benuzzi F, Nichelli P. Recognition of emotions from visual and prosodic cues in Parkinson's disease. Neurol Sci 2008;29:219-27.
67. Lloyd AJ. Comprehension of prosody in Parkinson's disease. Cortex 1999;35:389-402.
68. Hertrich I, Lutzenberger W, Spieker S, Ackermann H. Fractal dimension of sustained vowel productions in neurological dysphonias: an acoustic and electroglottographic analysis. J Acoust Soc Am 1997;102:652-4.
69. Zhang Y, Jiang JJ. Nonlinear dynamic mechanism of vocal tremor from voice analysis and model simulations. J Sound Vib 2008;316:248-62.
70. Aly NM, Playfer JR, Smith SL, Halliday DM. A novel computer-based technique for the assessment of tremor in

Parkinson's disease. Age Ageing 2007;36:395-9.

71. Jankovic J, Schwartz KS, Ondo W. Re-emergent tremor of Parkinson's disease. J Neurol Neurosurg Psychiatry 1999; 67:646-50.

72. Blin O, Ferrandez AM, Serratrice G. Quantitative analysis of gait in Parkinson patients: increased variability of stride length. J Neurol Sci 1990;98:91-7.

73. Frenkel-Toledo S, Giladi N, Peretz C, Herman T, Gruendlinger L, Hausdorff JM. Treadmill walking as an external pacemaker to improve gait rhythm and stability in Parkinson's disease. Mov Disord 2005;20:1109-14.

74. Hausdorff JM, Cudkowicz ME, Firtion R, Wei JY, Goldberger AL. Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. Mov Disord 1998;13:428-37.

75. Hausdorff JM, Peng C-K, Ladin Z, Wei JY, Goldberger AL. Is walking a random walk? Evidence for long-range correlations in stride interval of human gait. J Appl Physiol 1995; 78:349-58.

76. Haapaniemi TH, Pursianinen V, Korpelainen JT, Huikuri HV, Sotaniemi KA, Myllylä VV. Ambulatory ECG and analysis of heart rate variability in Parkinson's disease. J Neurol Neurosurg Psychiatry 2001;70:305-10.

77. Hausdorff JM, Balash Y, Giladi N. Time series analysis of leg movements during freezing of gait in Parkinson's disease: akinesia, rhyme or reason? Physica A 2003;321:565-70.

78. Previc FH. Dopamine and the origins of human intelligence. Brain Cogn 1999;41:299-350.

79. Van Orden G, Pennington BF, Stone GO. What do double dissociations prove? Cogn Sci 2001;25:111-72.

80. Edwards R, Beuter A. Parkinsonian tremor and simplification in network dynamics. Bull Math Biol 1999;51:157-77.

81. Goldberger AL, Amaral LAN, Hausdorff JM, Ivanov PCh, Peng C-K, Stanley HE. Fractal dynamics in physiology: alterations with disease and aging. Proc Natl Acad Sci U S A 2002;99:2466-72.

82. Goldberger AL, Peng C-K, Lipsitz LA. What is physiologic complexity and how does it change with aging and disease? Neurobiol Aging 2002;23:23-6.

83. Davia CJ. Life, catalysis and excitable media: a dynamic systems approach to metabolism and cognition. In: Tuszynski J, editor. The physical basis for consciousness. Heidelberg: Springer; 2005. p. 229-60.

84. Freeman WJ. How brains make up their minds. New York: Cambridge University Press; 2000.

85. Ito J, Nikolaev AR, van Leeuwen C. Spatial and temporal structure of phase synchronization of spontaneous alpha EEG activity. Biol Cybern 2005;92:54-60.

86. Kelso JAS. Dynamic patterns: the self-organization of brain and behavior. Cambridge (MA): MIT Press; 1995.

87. Beckers F, Verheyden B, Aubert AE. Aging and nonlinear hear rate control in a healthy population. Am J Physiol 2006; 290:2560-70.

88. Varela M, Jimenez L, Farina R. Complexity analysis of the temperature curve: new information from body temperature. Eur J Appl Physiol 2003;89:230-7.

89. Wink AM, Bernard F, Salvador R, Bullmore E, Suckling J. Age and cholinergic effects on hemodynamics and functional coherence of human hippocampus. Neurobiol Aging 2006; 27:1395-404.

90. Duarte M, Sternad D. Complexity of human postural control in young and older adults during prolonged standing. Exp Brain Res 2008;191:265-76.

91. Lin D, Deol H, Nussbaum MA, Madigan ML. Reliability of COP-based postural sway measures and age-related differences. Gait Posture 2008;28:337-42.

92. Norris JA, March AP, Smith IJ, Kohut RI, Miller ME. Ability of static and statistical mechanics posturographic measures to distinguish between age and fall risk. J Biomech 2005; 38:1263-72.

93. Thurner S, Mittermaier C, Ehrenberger K. Change of complexity patterns in human posture during aging. Audiol Neurotol 2002;7:240-8.

94. Hausdorff JM, Mitchell SL, Firtion R, Peng C-K, Cudkowicz ME, Wei JY, et al. Altered fractal dynamics of gait: reduced stride-interval correlations with aging and Huntington's disease. J Appl Physiol 1997;82:262-9.

95. Van Orden G, Paap KR. Functional neuroimages fail to discover pieces of mind in the parts of the brain. Philos Sci 1997;64:85-94.

96. Kelso JAS. Cognitive coordination dynamics. In: Tschacher W, Dauwalder J-P, editors. The dynamical systems approach to cognition. River Edge (NJ): World Scientific; 2003. p. 45-67.

97. Lewontin RC. The analysis of variance and the analysis of causes. Am J Hum Genet 1974;26:400-11.

98. Sternberg S. The discovery of processing stages: extensions of Donders' method. Acta Psychol 1969;30:276-315.

99. Riley MA, Van Orden G, editors. Tutorials in contemporary nonlinear methods for the behavioral sciences 2005. [accessed May 1, 2009] Available from: URL: http://www.nsf.gov/sbe/bcs/pac/nmbs/nmbs.jsp.

100. Bhattacharya J. Increase of universality in human brain during mental imagery from visual perception. PLoS One 2009;4: e4121.

101. Bullmore E, Barnes A, Bassett DS, Fornito A, Kitzbichler M, Meunier D, et al. Generic aspects of complexity in brain imaging data and other biological systems. Neuro Image 2009;47:1125-34.

102. Wink AM, Bullmore E, Barnes A, Bernard F, Suckling J. Monofractal and multifractal dynamics of low frequency endogenous brain oscillations in functional MRI. Hum Brain Mapp 2008;29:791-801.

103. Ihlen EAF, Vereijken B. Beyond $1/f\alpha$ fluctuation in cognitive performance. J Exp Psychol Gen. In press.

104. Buzsáki G. Rhythms of the Brain. New York: Oxford; 2006.

105. Newell A. Unified theories of cognition. Cambridge (MA): Harvard University Press; 1990.

106. Simon HA. The organization of complex systems. In: Pattee HH, editor. Hierarchy theory: the challenge of complex systems. New York: George Braziller; 1973. p. 1-27.

107. Hollis G, Van Orden G. The blue collar brain. (submitted).

108. Järvilehto T. The theory of the organism-environment system: I. Description of the theory. Integr Physiol Behav Sci 1998; 33:321-34.

109. Kloos H, Van Orden G. Soft-assembled mechanisms for the unified theory. In: Spencer JP, Thomas M, McClelland J, editors. Toward a unified theory of development: connectionism and dynamic systems theory re-considered. New York: Oxford University Press; 2009. p. 253-67.

110. Van Orden G, Kloos H. The question of phonology and reading. In: Snowling ML, Hulme C, editors. The science of reading: a handbook. Oxford: Blackwell Publishing; 2005. p. 61-78.