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Nicholas Stergiou

University of Nebraska at Omaha, nstergiou@unomaha.edu

Leslie M. Decker

University of Nebraska at Omaha

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1 **Human Movement Variability, Nonlinear Dynamics, and Pathology: Is There A Connection?**

2 Nicholas Stergiou^{1,2,*} and Leslie M. Decker¹

3 ¹ Nebraska Biomechanics Core Facility, University of Nebraska at Omaha, 6001 Dodge Street, Omaha,
4 NE 68182-0216, USA.

5 ² Department of Environmental, Agricultural and Occupational Health Sciences, College of Public
6 Health, University of Nebraska Medical Center, 987850 Nebraska Medical Center, Omaha, NE 68198-
7 7850, USA.

8 *Corresponding author. Tel.: 402-5543247; fax: 402-5543693.

9 E-mail address: nstergiou@unomaha.edu (N. Stergiou).

10

11 **Abstract**

12 Fields studying movement generation, including robotics, psychology, cognitive science and
13 neuroscience utilize concepts and tools related to the pervasiveness of variability in biological systems.
14 The concept of variability and the measures for nonlinear dynamics used to evaluate this concept open
15 new vistas for research in movement dysfunction of many types. This review describes innovations in
16 the exploration of variability and their potential importance in understanding human movement. Far
17 from being a source of error, evidence supports the presence of an optimal state of variability for
18 healthy and functional movement. This variability has a particular organization and is characterized by
19 a chaotic structure. Deviations from this state can lead to biological systems that are either overly rigid
20 and robotic or noisy and unstable. Both situations result in systems that are less adaptable to
21 perturbations, such as those associated with unhealthy pathological states or absence of skillfulness.

22

23 **Highlights**

24 > Exploration of variability using measures for nonlinear dynamics opens new vistas for research and
25 treatment for movement dysfunction. > Chaos could be a powerful component of the locomotive
26 system and its structure can be controlled by the nervous system. > An optimal state of variability that
27 exhibits chaos is important for health and functional movement. > Loss of this optimal state of
28 variability renders the system more predictable and rigid. > Increases beyond optimal variability render
29 the system more noisy and unpredictable

30

31 **Keywords**

32 Chaos, Stability, Anterior Cruciate Ligament, Cerebral Concussion, Passive Dynamic Walker, Infant
33 Motor Development

34

35

36 **1. Introduction**

37 One of the most common features of human movement is its variability. Human movement
38 variability can be described as the normal variations that occur in motor performance across multiple
39 repetitions of a task (Stergiou, Harbourne, & Cavanaugh, 2006). This variability is intrinsic in all
40 biological systems and it can be observed quite easily. If a person tries to repeat the same movement
41 twice, the two actions will never be identical. Bernstein (1967) used an expression “repetition without
42 repetition” whereby each repetition of an act involved unique, non-repetitive neural and motor
43 patterns. Recently, the role of movement variability in motor control has become an object of study in
44 its own right (Bates, 1996; Newell & Corcos, 1993). Therefore, a number of questions have been
45 raised (Stergiou et al., 1996). Examples of such questions are “How variability is controlled while
46 learning a new skill?”, “Is variability associated with disease/health?”, and “What are the sources of
47 variability, and how do they interact in the production of the observed variation in movement?”

48 In the past, variability in motor performance has been considered from a variety of theoretical
49 perspectives (e.g., Newell & Corcos, 1993). A prominent theory is the Generalized Motor Program
50 Theory (GMPT; Summer & Anson, 2009). This theory considers variation in a given movement
51 pattern to be the result of error. This error in the ability to predict the necessary parameters for
52 employing the underlying motor program results in variation in motor performance (Schmidt, 2003;
53 Schmidt & Lee, 2005). With task-specific practice, prediction error is gradually eliminated or
54 minimized, thereby optimizing the accuracy and efficiency of the movement pattern.

55 Another prominent theory is the uncontrolled manifold (UCM) hypothesis. Practically, motor
56 variability has been associated with motor redundancy. Motor redundancy refers to having more
57 elements than necessary to solve a task, resulting in the existence of multiple solutions to a given
58 motor problem (Scholz & Schöner, 1999). Latash, Scholz, & Schöner (2002) described the UCM
59 hypothesis to address this problem of motor redundancy. According to this hypothesis, when a multi-
60 element system changes its state within a UCM computed for a particular performance variable (e.g.,

61 total force produced by a set of fingers), this variable is kept at a constant value. As long as the system
62 does not leave the UCM, the hierarchically higher controller (e.g., central nervous system) does not
63 need to interfere and, in that sense, the system of elemental variables does not need to be controlled
64 within that manifold. If the system leaves the UCM and shows an acceptable error in the performance
65 variable, the controller may have to interfere and introduce a correction (Latash, 2008). The UCM
66 approach has been applied to several motor tasks such as maintaining quiet stance, finger force
67 production, bimanual pointing, sit-to-stand, and pistol shooting (Domkin, Laczko, Jaric, Johansson, &
68 Latash, 2002; Latash, Scholz, Danion, & Schöner, 2001; Scholz, Kang, Patterson, & Latash, 2003) to
69 discover coordination strategies of apparently redundant motor systems and uncover the functional
70 purposes that variability plays in those motor tasks.

71 A third theoretical perspective briefly presented here is the Dynamical Systems Theory (DST)
72 which proposes that biological systems self-organize according to environmental, biomechanical, and
73 morphological constraints to find the most stable solution for producing a given movement (Clark &
74 Phillips, 1993; Hamill, van Emmerik, Heiderscheit, & Li, 1999; Kamm, Thelen, & Jensen, 1990;
75 Kelso, 1995; Thelen, 1995; Thelen & Ulrich, 1991). Increased variability in a movement pattern
76 generally indicates loss of stability, while decreased variability generally indicates a highly stable
77 behavior. The GMPT, UCM, and DST perspectives are similar in that they all recognize that decreased
78 variability results from the efficient execution of a given movement pattern. DST focuses more on
79 behavioral transitions and provides tools to describe such phenomena. Specifically, DST suggests that,
80 in certain dynamical systems and under certain conditions, when variability increases and reaches a
81 specific critical point, the system becomes highly unstable and switches to a new, more stable
82 movement pattern (with less variability). This proposition is a significant step forward because it
83 explains transitions between behavioral states and implies that a persistent lack of movement
84 variability may indicate rigid, inflexible motor behaviors with limited adaptability to changing task or
85 environmental demands. However, a significant limitation of DST is that it does not account for the

86 observation that some behaviors, which appear to be highly stable, paradoxically are performed in
87 variable ways. This is especially evident when we observe elite sports players or musicians performing
88 (e.g., Michael Jordan taking a jump shot or Yo-Yo Ma playing the cello). Not only is their
89 performance more consistent than that of less capable individuals, but they also seem to have
90 developed an infinite number of ways of performing. If we actually consider fundamental motor skills
91 (i.e., gait) as activities when applied in “real life” contexts, we can actually say that every single one of
92 us is a Michael Jordan in our abilities to walk through crowds or on diverse and challenging terrains.
93 Therefore, it seems that in this sense, variability is closely related with a rich behavioral state.

94 The idea that variability decreases with skill acquisition in one context (motor learning paradigm)
95 and increases with skill acquisition in another context (the development of a behavioral repertoire) is
96 readily explained by the way in which variability is measured. Typical motor learning curves are
97 constructed using traditional variability measures of skill performance to capture error in performance.
98 Such linear statistical measures quantify the magnitude of variation in a set of values independently of
99 their order in the distribution. The magnitude of variability continuously decreases and eventually
100 plateaus as motor learning occurs. In contrast, variation in how a motor behavior emerges in time is
101 best captured by measures where the temporal organization in distribution of values is the facet of
102 interest. Temporal organization (or structure) of variability is quantified by the degree to which values
103 emerge in an orderly manner, often across a range of time scales. Therefore, recent theoretical
104 approaches perspectives have suggested that variability contains important information about
105 movement (Amato, 1992; Cavanaugh, Guskiewicz, & Stergiou, 2005; Harbourne & Stergiou, 2009;
106 [Newell & Corcos, 1993](#)). These approaches have now propagated in the human movement literature
107 and lead the development of alternative theoretical frameworks and methodology to study human
108 movement related injuries and treatments.

109 Much of the controversy that exists in the literature with respect to human movement variability
110 stems from the methodology used. Traditional linear measures, such as the standard deviation or the

111 range, are measures of centrality and thus provide a description of the amount or magnitude of the
112 variability around a central point (Fig. 1). From a human movement perspective, this approach in
113 evaluating variability has led to several practitioners and scientists to believe that the mean is the
114 standard of performance and everything away from the mean is error. From a statistical standpoint, the
115 valid usage of traditional linear measures to study variability assumes that variations between
116 repetitions of a task are random and independent (of past and future repetitions) (Lomax, 2007).
117 However, previous studies have shown that such variations are distinguishable from noise (Delignières
118 & Torre, 2009; Dingwell & Cusumano, 2000; Dingwell & Kang, 2007; Stergiou, Buzzi, Kurz, &
119 Heidel, 2004). In addition, several studies have indicated that these variations have a deterministic
120 origin (Dingwell & Cusumano, 2000; Dingwell & Kang, 2007; Harbourne & Stergiou, 2009; Miller,
121 Stergiou, & Kurz, 2005). Thus, they are neither random nor independent. For instance, although
122 variations between strides during walking appear to vary randomly, with no correlation between the
123 present and future strides, the healthy adult locomotor system actually possesses “motor memory”,
124 such that the fluctuations from one stride to the next display a subtle, “hidden” temporal structure.
125 Mathematical tools, such as entropic or fractal measures or tools developed for the study of
126 deterministic chaos have enabled the evaluation of this temporal structure of variability. From this
127 approach, how human movement evolves over time becomes of importance. Therefore, the focus is not
128 on the standard of performance represented by the average but rather on the exploratory nature of
129 movement, which enhances practice and quality of performance.

130 [Insert Fig. 1 about here, 100%]

131 From an evaluation perspective, these two approaches are complimentary since each explores
132 different aspects of variability (Harbourne & Stergiou, 2009; Stergiou et al., 2004). As mentioned
133 above, conventional statistical tools quantify the magnitude of variation in a set of values
134 independently of their order in the distribution; this works properly for linear systems. In contrast,
135 variation in how a motor behavior emerges in time is best captured by tools developed for the study of

136 nonlinear systems. These tools that have been used in the literature for this purpose include
137 approximate entropy, sample entropy, correlation dimension, largest lyapunov exponent, and detrended
138 fluctuation analysis (Bruijn, van Dieën, Meijer, & Beek, 2009; Cavanaugh, Kochi, & Stergiou, 2010;
139 Delignières, Deschamps, Legros, & Caillou, 2003; Donker, Roerdink, Greven, & Beek, 2007; Gates &
140 Dingwell, 2007, 2008; Hausdorff, 2009; Liao, Wang, & He, 2008; Kurz & Hou, 2010; Kurz,
141 Markopoulou, & Stergiou, 2010; Sosnoff, Valentine, & Newell, 2006; Sosnoff & Voudrie, 2009; Stins,
142 Michielsen, Roerdink, & Beek, 2009; Vaillancourt, Sosnoff, & Newell, 2004; Yang & Wu, 2010).

143 **2. Further theoretical developments**

144 There is a growing body of literature showing that the cycle-to-cycle variation seen in a wide
145 variety of physiological systems is nontrivial and may offer insight into the control of these systems
146 (Bassingthwaight, Liebovitch, & West, 1994). This intrinsic movement variability is highly
147 suggestive of a fundamental feature of the neural control of movement. Cai et al. (2006) provided some
148 evidence with respect to this issue by studying the ability of spinal mice to learn to step. In their
149 protocol, variation was permitted by applying an assist-as-needed mode of control of a robotic arm
150 attached to the ankle of each hindlimb. The results showed that when the intrinsic variability was
151 overridden (e.g., when a fixed pattern is imposed with no variability allowed), learning of a task was
152 suboptimal relative to the condition when the training is assist-as-needed. The authors suggested that
153 training with robotic control algorithms that provide a soft assist-as-needed control permits the
154 intrinsic variability that characterized any neural controlled movements. This study provided strong
155 evidence that a fundamental strategy of the neural control of a given motor task (stepping) is to
156 incorporate a degree of variability in the sensorimotor pathways. Importantly, when the system is
157 forced to adapt a rigid behavior, it produces suboptimal results. From a clinical point of view, these
158 findings highlight the importance of variation of stepping kinematics as a feature of optimizing
159 relearning to step.

160 Further evidence for the association of variability and health comes from research on higher neural
161 functions and their association with gait. To better understand the underlying mechanisms of gait
162 variability in community-dwelling older adults, [Rosano, Brach, Studenski, Longstreth, & Newman](#)
163 [\(2007\)](#) investigated the relationships between the variability of different aspects of gait and subclinical
164 brain vascular abnormalities in adults who are free of neurological diseases. Increased variability of
165 step length was associated with greater prevalence of infarcts, including infarcts in the basal ganglia. It
166 was also associated with greater white matter hyperintensities severity, independent of age, gender,
167 cognitive function and cardiovascular disease. Importantly, these brain abnormalities were associated
168 with increased movement variability in comparison with optimal healthy behavior. This is in the
169 opposite direction than what was presented in the previous paragraph. Here we do not have rigidity and
170 absence of movement variability, which is undesirable, but we have too much variability, which is also
171 undesirable. Can it possible then that healthy movement variability is associated with an optimal state,
172 which is in between too much and too little? Interestingly, [Rocchi, Chiari, & Horak \(2002\)](#)
173 demonstrated that variability of postural sway was larger than normal in patients with Parkinson's
174 disease without the effects of drugs and even larger with levodopa. However, with deep brain
175 stimulation these patients exhibited smaller than normal variability of postural sway. Practically, the
176 normal healthy controls were in between all these conditions suggesting that too much or too little
177 were not optimal. Similarly, in [Brach, Berlin, VanSwearingen, Newman, & Studenski \(2005\)](#), elderly
178 individuals with extreme step width variability (either low or high step width variability) were more
179 likely to report a fall in the past year than those with moderate step width variability. Therefore, either
180 too little or too much step width variability was associated with falls.

181 Recently, it has been demonstrated that temporal variations in biological signals, even though they
182 appear no different from random noise, exhibit deterministic patterns. These patterns have been
183 defined as chaotic (Fig. 2: middle panel) and can have significant implications for medicine. For
184 example, heart rhythms in which the variation in the time interval between subsequent QRS waves is

185 either periodic or random (Fig. 2) have been associated with heart attacks ([Denton, Diamond, Helfant,](#)
186 [Khan, & Karagueuzian, 1990;](#) [Glass & Mackey, 1988](#)). Conversely, chaotic heart rhythms are related
187 to healthy states. Similar results have been found in other biological signals. These studies employed
188 more advanced tools to describe conditions in which more conventional, linear techniques appeared
189 inadequate, confounding scientific study and the development of meaningful therapeutic options.
190 Research along these lines include investigations of heart rate irregularities, sudden cardiac death
191 syndrome, blood pressure control, brain ischemia, epileptic seizures, and several other conditions
192 ([Amato, 1992;](#) [Buchman, Cobb, Lapedes, & Kepler, 2001;](#) [Faure & Korn, 2001, 2003;](#) [Garfinkel,](#)
193 [Spano, Ditto, & Weiss, 1992;](#) [Goldberger, Rigney, Mietus, Antman, & Greenwald, 1988;](#) [Goldstein,](#)
194 [Toweill, Lai, Sonnenthal, & Kimberly, 1998;](#) [Korn & Faure, 2003;](#) [Lanza et al., 1998;](#) [Orsucci, 2006;](#)
195 [Slutzky, Cvitanovic, & Mogul, 2001;](#) [Toweill & Goldstein, 1998;](#) [Wagner, Nafz, & Persson, 1996](#)),
196 aiming to understand their effect on the human physiology and eventually develop prognostic and
197 diagnostic tools. Based on such investigations, the presence of chaotic temporal variations in the steady
198 state output of a healthy biological system can represent the underlying physiologic capability to make
199 flexible adaptations to everyday stresses placed on the human body ([Lipsitz & Goldberger, 1992;](#)
200 [Lipsitz, 2002](#)). Importantly, there are certain benefits for the nervous system for adopting chaotic
201 regimes allowing a wide range of potential behaviors. This leads to healthy biological systems that are
202 adaptable and flexible in an unpredictable and ever-changing environment ([Faure & Korn, 2001,](#)
203 [2003](#)). But what happens in diseased states? In which way are these deterministic properties of a
204 healthy system and, in consequence, its behavior is affected? Two main propositions have been
205 developed recently to address these questions.

206 [Insert Fig. 2 about here, 100%]

207 The first proposition has been stated by [Lipsitz and Goldberger \(1992\)](#) and proposed that healthy
208 systems are characterized with physiologic capability to make flexible adaptations to everyday stresses
209 placed on the human body. In the case of human gait this is demonstrated as we observe variations

210 over small time scales (i.e., a few strides) being statistically similar to those over larger and larger time
211 scales (i.e., hundreds and even thousands of strides). The use of scaling analysis techniques (e.g.,
212 detrended fluctuation analysis) revealed that fluctuations in stride interval time series display long-
213 range correlations (Hausdorff, Peng, Ladin, Wei, & Goldberger, 1995), and these correlation properties
214 evolve during childhood (Hausdorff, Zermany, Peng, & Goldberger, 1999) and degrade both with
215 physiologic aging and with certain degenerative neurological diseases (Hausdorff, 2009; Hausdorff et
216 al., 1997). This breakdown in this physiologic capability may be associated with the degradation of
217 these properties (Peng, Hausdorff, & Goldberger, 2000). Thus, it is proposed that there is a positive
218 correlation between this physiologic capability and healthy motor performance. On the other hand, it is
219 proposed that there is a negative relation between physiologic capability and aging (Lipsitz &
220 Goldberger, 1992).

221 However, not all studies are consistent with this proposition. It appears that the task goal plays a
222 critical role in shaping the nature of the differences that arise with aging and disease (Vaillancourt &
223 Newell, 2002, 2003). For instance, postural studies showed that the direction of change as a function of
224 aging is opposite in the actions of posture and locomotion (Newell, 1998; Hausdorff et al., 1997). This
225 contrasts with the above proposition. Rather it is the loss of adaptability of the intrinsic dynamics that
226 is the key feature of change as a function of age. In an empirical examination of the “*loss of*
227 *adaptability hypothesis*”, Vaillancourt and Newell (2003) examined whether age-related differences in
228 the time and frequency structure of force output are dependent on task demands. They found that the
229 specific direction of the change is dependent on the task demands and reflects the role of intentions and
230 goals in organizing the dynamics of the motor output (Vaillancourt & Newell, 2002).

231 More recently and in an effort to bring together the above propositions, Stergiou et al. (2006) and
232 as refined in Harbourne & Stergiou (2009) proposed a new theoretical model to explain movement
233 variability as it relates to motor learning and health. This model is based on the idea that mature motor
234 skills and healthy states are associated with optimal movement variability that reflects the adaptability

235 of the underlying control system. The principle of optimality in movement variability is pioneering in
236 the sense that it relates in an inverted U-shape relationship the presence of chaotic temporal variations
237 in the steady state output of a healthy biological system with the concept of predictability (see Fig. 3).
238 Practically at this optimal state of movement variability the biological system is in a healthy state and
239 is characterized by exhibiting chaotic temporal variations in the steady state output (i.e., the uppermost
240 point along the inverted U-shaped function), attaining high values only in the intermediate region
241 between excessive order (i.e., maximum predictability) and excessive disorder (i.e., no predictability).
242 Thus, this variability has deterministic structure and reflects the adaptability of the system to
243 environmental stimuli and stresses. Decrease or loss of this optimal state of variability renders the
244 system more predictable, rigid and with a robotic type of motor behavior. This is fairly similar to the
245 Lipsitz and Goldberger hypothesis. However, our research group also added that increases beyond
246 optimal variability render the system more noisy and unpredictable, similarly to what is observed for
247 example in a very frail elder or a drunken sailor walking. Both situations result in decreased
248 adaptability to perturbations and are associated with lack of health (see Fig. 3).

249 [Insert Fig. 3 about here, 100%]

250 Recent empirical research in motor control supports the theoretical model of optimal movement
251 variability. Among the most recent investigations, the study of [Cignetti, Schena, & Rouard \(2009\)](#)
252 gives an illustration of the flexibility capabilities of the neuromuscular system to counteract the fatigue
253 induced by a cross-country skiing effort. The study exemplifies the model developed by [Stergiou et al.](#)
254 [\(2006\)](#). In this investigation, both the inter-cycle variability in cross-country skiing gait and its
255 evolution with fatigue were examined to understand the flexibility capabilities of the neuromuscular
256 system. The fluctuations of the limb movements of the skiers were not random but displayed a chaotic
257 behavior, reflecting flexibility to adapt for possible perturbations present during skiing. This behavior
258 degraded with fatigue through increased and more random fluctuations.

259 In the theoretical model of optimal movement variability, it is also proposed that motor
260 development and learning processes obey these principles. In other words, the development of healthy
261 and highly adaptable systems relies on the achievement of the optimal state of variability.
262 Alternatively, abnormal development may be characterized by a narrow range of behaviors, some of
263 which may be rigid, inflexible and highly predictable or, on the contrary, random, unfocused and
264 unpredictable. Motor disabilities many times are described as such. In accordance with this proposition
265 the authors also suggest that the goal of neurologic physical therapy and performance in sport activities
266 should be to enhance the development of this optimal state movement variability by incorporating a
267 rich repertoire of movement strategies, which can be achieved by implementing a multitude of
268 experiences. Several such examples are given later in the review.

269 **3. Variability does not equate with stability**

270 Before we will continue with the presentation of our experimental work, which is based on the
271 above proposition, we would like to address an issue where we believe that there is confusion in the
272 literature. As mentioned above, variability was interpreted traditionally as noise superimposed upon a
273 signal, where the signal is the intended movement and the variability is random noise about this
274 intended movement (Newell & Corcos, 1993). The focus of this approach was to quantify the amount
275 of variability associated with the movement of interest. Typically, the amount of variability was
276 assessed by the standard deviation. Increased amount of variability found in postural sway as well as in
277 gait has been linked to an increased risk of falling in the elderly (Demura, Kitabayashi, & Aoki, 2008;
278 Maki, 1997). As increased amount of variability has been reported as a predictor of risk of falling, it
279 has been assumed that variability and stability are negatively correlated, where increases in the amount
280 of variability were assumed to equate with increases in instability. However, evidence shows that a
281 moving system (e.g., a swaying body during posture or a moving body during gait) with large
282 variability implies neither a highly stable system nor poor stability (Cavanaugh et al., 2005, 2006;
283 Cavanaugh, Guskiewicz, & Stergiou et al., 2005). For instance, a trained athlete can balance without

284 falling while standing one-legged on a fully inflated soccer ball. Clearly, this demonstrates exceptional
285 stabilizing capacity despite the fact that center-of-pressure measurements under the ball will
286 demonstrates large movement variability. This simple example illustrates that variability does not
287 necessarily predict instability.

288 As recently stated by [Granata and England](#) (Reply to the Letter to the Editor from [Beauchet, Allali,](#)
289 [Berrut, & Dubost, 2007](#)), “it is incorrect to assume that variability can be equated to the biomechanics
290 of stability”. According to their view, “variability” refers to the ability of the motor system to reliably
291 perform in a variety of different environmental and task constraints, while “stability” refers to the
292 dynamic ability to offset an external perturbation. Thus, variability and stability represent different
293 properties within the motor control process. For these authors and others, variability is quantified using
294 measures derived from linear statistics, such as the standard deviation of the mean ensemble curve ([Li,](#)
295 [Haddad, & Hamill, 2005](#)), whereas stability is quantified using measures derived from nonlinear
296 dynamics. Specifically, local stability is commonly defined as the “inverse of the rate of divergence
297 from the intended trajectory after a small perturbation”, as quantified by the use of the largest
298 Lyapunov exponent ([Buzzi, Stergiou, Kurz, Hageman, & Heidel, 2003](#); [Dingwell et al., 2000, 2001](#);
299 [Dingwell & Cusumano, 2000](#); [Dingwell & Kang, 2007](#); [Hurmuzlu & Basdogan, 1994](#); [Hurmuzlu,](#)
300 [Basdogan, & Stoianovici, 1996](#); [Kang & Dingwell, 2006a, 2006b, Stergiou et al., 2004](#)). By the same
301 token, stability can be inferred via the “presence of long-range, fractal correlations”, as quantified by
302 the use of fractal analysis (e.g., detrended fluctuation analysis) ([Hausdorff, 2009](#); [Hausdorff et al.,](#)
303 [1995, 1996, 1997, 1999, 2000](#); [Jordan et al., 2006](#)). Thus, stability covers different aspects, that of
304 deviations from deterministic orbits quantified through trajectory divergence (local stability) and that
305 of temporal statistics quantified through correlations and entropies (self-similarity and regularity; see
306 also below).

307 Presently and after significant work in this area, we humbly believe that the terminology used by
308 human movement scientists to describe their findings should be more specific and straightforward.

309 This is necessitated by our interactions with clinicians and practitioners where simplicity is important
310 in order to establish proper communication and efficient collaboration. To date, there is general
311 agreement that measures for linear systems (indexed by either the standard deviation in absolute terms,
312 or the coefficient of variation in relative terms) quantify the amount or magnitude of the variations
313 present in a time series (e.g., center of pressure oscillations or gait fluctuations), whereas the measures
314 for nonlinear systems (e.g., approximate entropy, sample entropy, correlation dimension, largest
315 Lyapunov exponent, and detrended fluctuation analysis) quantify the structure or organization of the
316 variations present in a time series (i.e., changes observed in gait fluctuations or postural sway
317 oscillations over time). But, there is no reason to infer that stability is uniquely related to any measure,
318 since each of those measures quantifies different aspects of the time-dependent structural
319 characteristics embedded in a given time series. For instance, the largest Lyapunov exponent quantifies
320 the rate at which nearby trajectories from a time series in state space diverge over time; this equals the
321 so-called local stability, i.e. deviation from a certain orbit (Wolf, Swift, Swinney, & Vastano, 1985;
322 Rosenstein, Collins, & De Luca, 1993; Abarbanel, 1996); the approximate entropy quantifies the
323 regularity of a time series (Pincus, 1991; Pincus, Gladstone, & Ehrenkranz, 1991); the detrended
324 fluctuation analysis quantifies the presence of long-range correlations in a time series (Hausdorff et al.,
325 1995). In conclusion, we suggest that interpretation of findings derived from nonlinear dynamics
326 should not be made beyond what the nonlinear measures actually quantify, unless correlated with other
327 measurements (e.g., electromyographic analysis for quantification of muscle fatigue,
328 magnetoencephalographic analysis for direct quantification of cortical activity, etc.).

329 **4. Experimental work from our laboratory exploring the above theoretical frameworks**

330 Armed with the above tools a great number of investigators have explored important questions on
331 variability and sought to provide support for or against the above-mentioned theoretical frameworks.
332 Here we will present some of our work including posture and gait from healthy and pathological
333 populations at different stages of the lifespan.

334 *4.1. Infant motor development*

335 [Harbourne and Stergiou \(2003\)](#) explored the development of independent postural control in
336 sitting. Specifically, they investigated whether developing postural control in sitting has deterministic
337 origins, and if so, how this can be characterized using measures for nonlinear dynamics. Normal
338 infants were examined longitudinally. Postural sway data were collected for ten seconds while the
339 child attempted to maintain sitting postural control on a force platform. The resulting center of pressure
340 (COP) time series during sitting were analyzed which revealed largest Lyapunov exponent values that
341 were significantly different from their surrogate counterparts. This result indicated that the fluctuations
342 observed in the COP time series were not randomly derived, and reflect deterministic processes by the
343 neuromuscular system. The fluctuations in the time series were not noise, but had a structure or order
344 that needed further investigation and description. Results further indicated differences in the largest
345 Lyapunov exponent (LyE) and approximate entropy (ApEn) across the three stages of sitting
346 development, reflecting changes in the dynamics of sitting postural control. The LyE values decreased
347 as the sitting behavior emerged, indicating less divergence in the movement trajectories of the COP.
348 Therefore, as the infants had more experience exploring the sitting position, they increasingly occupied
349 trajectories that were closer together within the state space. The ApEn values also decreased, indicating
350 that the child develops more repeatable movement patterns of the COP, which are most successful to
351 maintain sitting postural control. Based on the findings the authors suggested that a centrally
352 determined program of specific muscle responses is unlikely to provide successful postural control
353 within the changing context of a growing infant. The findings of this study add to the evidence that
354 infants dynamically assemble the sitting posture by originally organizing movement strategies that are
355 more regular and repeatable, thus first allowing control of the degrees of freedom to approximate the
356 skill, and then to explore adaptations to function in the environment.

357 Toward the goal of better understanding the control strategies that are involved in evaluating
358 postural control during sitting in infants, [Harbourne, Deffeyes, Kyvelidou, and Stergiou \(2009\)](#)

359 performed a principal component analysis as a data reduction method. Four factors were identified: the
360 postural sway area and the amount of variability of that area (Factor 1: range and root-mean-square in
361 the anterior-posterior and medial-lateral directions); the divergence of the sway movement trajectories
362 and the regularity of the sway front-to-back (Factor 2: LyE and ApEn in the anterior-posterior
363 direction); the speed and coordination of the postural sway (Factor 3: postural sway velocity and
364 frequency dispersion); and the divergence of the sway movement trajectories and regularity of the
365 sway side-to-side (Factor 4: LyE and ApEn in the medial-lateral direction). Thus, Factors 1 and 3
366 included measures of the amount of variation in the postural sway, and Factors 2 and 4 included
367 measures of the temporal organization of these variations. The isolation of the two types of measures
368 into separate factors indicated possible separate features of postural control during sitting in typically
369 developing infants. For example, the measures of the amount of variation did not exhibit loading into
370 different factors by direction (anterior-posterior vs. medial-lateral) suggesting that during development
371 infants may concentrate in a specific direction for exploration. Furthermore, the authors stressed that
372 different measures taken together offer a more comprehensive description of postural control, with the
373 ability to understand specific characteristics in the system. Problems in the system may occur when
374 one or more of several components are compromised. The authors also stressed the importance of this
375 approach in determining the health of the developing postural control system in infants as well as for
376 early diagnosis of postural disorders.

377 The above two studies suggested that learning of the sitting skill appears to be in line with
378 theoretical suggestions and empirical results obtained when examining the acquisition of motor skills
379 (e.g., [Mitra, Riley, & Turvey, 1997](#); [Newell & Vaillancourt, 2001](#)). Specifically, the maintenance of
380 equilibrium in the sitting skill is based on a problem of compressing a high-dimensional system
381 composed of many components (e.g., neural, muscular and segmental components) into a low-
382 dimensional system (such as in chaos which arises specifically in very low-dimensional nonlinear
383 systems that are deterministic; [Strogatz, 1994](#)) with only few macroscopic or collective variables that

384 need to be controlled. This could allow the attainment of the postural performance since these variables
385 govern the coordination of the different system components. In this way, it is then possible to assume
386 that the rationale of decreasing the degrees of freedom and then the number of controlled collective
387 variables makes the achievement of the postural control easier for the infants.

388 Our methodological approach was further used by [Deffeyes, Harbourne, Kyvelidou, Stuberg, &](#)
389 [Stergiou \(2009b\)](#) to investigate how sitting postural sway in typically developing infants differs from
390 developmentally delayed infants. Infants in the developmentally delayed group were diagnosed with
391 cerebral palsy, or else were developmentally delayed and at risk for cerebral palsy. Motor development
392 in infants with cerebral palsy is delayed, meaning that developmental milestones such as sitting,
393 standing, or walking may occur later than in infants with typical development, and in severe cases
394 these milestones may never be met ([Wu Day, Strauss, & Shavelle, 2004](#); [Fedrizzi et al., 2000](#)). The
395 results from [Deffeyes et al. \(2009b\)](#) showed that the LyE was the only parameter of COP time series
396 that revealed significant differences ($p < 0.000$) between infants with typical versus delayed
397 development. The authors suggested that the infants with delayed development appear to further
398 minimize the fluctuations that are present in their postural sway patterns indicating more rigid control
399 than infants with typical development. If it is assumed that the infants with typical development have
400 better motor control, then it can be suggested that these infants are exploring a wider variety of
401 solutions to postural control. It can also be assumed that infants with delayed development are further
402 freezing degrees of freedom to have fewer control parameters to manipulate as they maintain upright
403 posture. These results may seem contradictory with the results presented by the study of [Harbourne](#)
404 [and Stergiou \(2003\)](#). However, this is not the case if we consider them under the prism of the
405 theoretical framework of optimal state of variability. The infants with delayed development behave in
406 a more robotic and periodic fashion (Fig. 3) than healthy typically developing infants. Furthermore, the
407 healthy infants seem to “live” or move between randomness and optimal variability as they explore
408 effective strategies for postural control. Importantly, the nonlinear measure of LyE has the potential to

409 add the specificity of diagnosis in the early months of life, when most standardized tests of infant
410 development have little predictive value.

411 Most recently, [Deffeyes et al. \(2009a\)](#) examined the utility of different entropy algorithms to
412 further explore if different control strategies exist between typically developing infants and
413 developmentally delayed infants. Postural sway data were acquired while infants were sitting on a
414 force platform. Two types of entropy measures were used: (1) symbolic entropy (SymEn), (2) a new
415 asymmetric entropy (SymEn) measure, and (3) ApEn. For each method of analysis, parameters were
416 adjusted to optimize the separation of the results between the infants with delayed development and the
417 infants with typical development. The method that gave the widest separation between the two groups
418 was the asymmetric SymEn method, which [Deffeyes et al. \(2009a\)](#) developed by modification of the
419 SymEn algorithm. The ApEn algorithm also performed well, using parameters optimized for the infant
420 sitting postural sway data. As in the previous study the infants with delayed development were found
421 to have more regular patterns of postural sway, while the infants with typical development are seen to
422 have more information entropy in their movement. This further supported the conclusions from the
423 previous study and suggested that the development of a postural control strategy involves an
424 exploration of many possible solutions to arrive at a control strategy with an optimal state of
425 variability. The authors further suggested that infants with typical development appear to be exploring
426 more motor strategies, giving rise to the development of chaotic temporal variations in their postural
427 sway.

428 Critical to the above studies, was to establish the reproducibility of these measures for infant sitting
429 posture. Therefore, [Kyvelidou, Harbourne, Stuberg, Sun, & Stergiou \(2009\)](#) and [Kyvelidou,
430 Harbourne, Shostrom, & Stergiou \(2010\)](#) investigated the intra-session and inter-session reliability of
431 linear and nonlinear measures when used to analyze COP time series during the development of infant
432 sitting postural control in both typically developing and developmental delayed infants. Overall, the
433 results showed that the evaluation of COP time series using linear and nonlinear measures is a reliable

434 method for quantifying incremental change across the development of sitting postural control in both
435 typically developing infants and in infants with or at risk for cerebral palsy, and therefore the efficacy
436 of therapeutic interventions directed at improving the sitting postural abilities in infants with motor
437 developmental delays.

438 Taken together, the findings from the motor development investigations conducted by our research
439 group identified control strategies that point towards a new approach with respect to therapy
440 (Harbourne & Stergiou, 2009). In this approach, the therapist assumes that the general rule for the
441 patient is to optimize variability of movement for improving functional mobility and therefore health.
442 This will include keeping the patient in a state of dynamic equilibrium during therapy sessions.
443 Additionally, the therapist uses the strategy of providing only information for the patient on how to do
444 a task if the patient does not have a way to get the information. The rationale is that variability is
445 encouraged if the patient seeks information independently, and the patient is kept in a dynamic state.
446 The therapist does not focus on a particular movement form or strategy, but rather allows the patient to
447 discover that enhanced deterministic variability of various movements has an inherent value in
448 promoting success during a task. Importantly, the findings from our motor development studies
449 recommend that measures for studying nonlinear dynamics reveal that exhibiting chaotic temporal
450 variations is probably inherent in normal variations, indicating features of motor control that are
451 important for physical therapists to measure as they implement intervention. The application of
452 principles based on our theoretical framework capitalize on concepts and measures of nonlinear
453 dynamics to provide with innovative approaches to guide physical therapist practice and research in
454 motor development.

455 *4.2. Sports medicine*

456 Over the past ten years, we have conducted several studies on musculoskeletal injuries such as
457 anterior cruciate ligament injury or brain injury such as cerebral concussion.

458 *4.2.1. Anterior cruciate ligament: A sport-related injury to the neuromuscular system*

459 Anterior cruciate ligament (ACL) is the most commonly injured ligament in sports (Zarins &
460 Adams, 1988) and its reconstruction is a common operation among orthopaedic surgeons who are
461 involved in sports medicine. The purpose of ACL reconstruction and subsequent rehabilitation is to
462 restore complete and normal functionality of the knee joint in terms of muscular strength and stability
463 (Chmielewski, Rudolph, & Snyder-Mackler, 2002). The ability of the surgical procedure to achieve
464 complete and normal functionality of the knee joint is assessed with either static measures (i.e., KT-
465 1000, pivot-shift test) or questionnaires (i.e., Lysholm score) or combinations of both (i.e.,
466 International Knee Documentation Committee score). However, none of these measures is a true
467 assessment of the dynamic functionality of the reconstructed knee under low demanding activities,
468 such as walking, or higher demanding activities, such as those encountered in sports. To address this
469 critical knowledge gap, our research group conducted explored variability during gait to assess the
470 efficacy of anatomical ACL reconstruction for restoring normal knee mechanics and preserving long-
471 term joint health.

472 In our first study, Stergiou, Moraiti Giakas, Ristanis, & Georgoulis (2004) investigated the effect of
473 walking speed on the dynamic function of the ACL deficient knee using nonlinear measures. Dynamic
474 function of the knee was assessed in terms of the structure of the variations that exist in the natural
475 stride-to-stride movements of the knee. Individuals with unilateral deficiency walked on a treadmill at
476 different speeds while kinematics was collected. The deficient knee was found to have significantly
477 larger LyE values than the intact contralateral knee. Furthermore, increases in walking speed did not
478 affect these differences in the LyE values. However, these results were limited because comparisons
479 with healthy controls were not included. It is quite possible that the intact contralateral knee is not
480 absolutely healthy and several compensations occur leading to contradictory results.

481 Therefore, Moraiti, Stergiou, Ristanis, & Georgoulis (2007) extended the above research by
482 investigating the temporal structure of the variations present in the ACL deficient knee as compared to
483 that of a healthy control knee during walking. Individuals with unilateral ACL deficiency and healthy

484 controls walked at their self-selected speed on a treadmill, while lower extremity kinematics was
485 collected for 80 consecutive strides. The ACL deficient knee exhibited smaller LyE values than a
486 healthy control knee, indicating less divergence in the flexion-extension movement trajectories of the
487 deficient knee. The results also verified the hypothesis made in the previous study that the intact
488 contralateral knee is not absolutely healthy. The fact that the ACL deficient knee exhibited smaller
489 LyE values than the healthy control is likely non-desirable since it may represent a decrease or loss of
490 the optimal state of variability (which is exhibited by the healthy controls) rendering the system more
491 predictable, periodic and with a rigid type of motor behavior. Neurologically this can explained if we
492 consider that the ACL plays an important role in knee function because of its mechanical properties
493 and the mechanoreceptors that exist in it (Johansson, Sjölander, & Sojka, 1991; Solomonow &
494 Krogsgaard, 2001). For instance, it has been shown that activations of the ACL mechanoreceptors
495 induce hamstring contraction resisting anterior tibial translation (ACL-hamstring reflex), in both
496 animals and humans (Dyhre-Poulsen & Krogsgaard, 2000; Fujita, Nishikawa, Kambic, Andrish, &
497 Grabiner, 2000; Tsuda, Okamura, Otsuka, Komatsu, & Tokuya, 2001). It has been proposed that the
498 loss of proprioceptive input from the mechanoreceptors that exist in the ACL may lead to changes in
499 the central nervous system which, in turn, leads to the development of altered muscle patterns and
500 postural synergies (Courtney, Rine, & Kroll, 2005; Di Fabio, Graf, Badke, Breunig, & Jensen 1992;
501 Valeriani et al., 1996). It has been further suggested that this kind of injury might be regarded as a
502 neurophysiological dysfunction, not being a simple musculoskeletal injury (Kapreli & Athanasopoulos,
503 2006). Therefore, ACL deficiency can lead to altered somatosensory input, which results in decline in
504 the system's flexibility and narrowed functional responsiveness reflected as rigidity.

505 Importantly, degeneration of the knee joint and eventually development of osteoarthritis has been
506 associated with ACL deficiency. Longitudinal follow-up studies have shown that ACL deficiency
507 leads to the development of chondral injuries (Mankin, 1982), meniscal tears, degeneration of the
508 articular cartilage and eventually post-traumatic arthritis (Fithian, Paxton, & Goltz, 2002; McDaniel &

509 [Dameron, 1983; Noyes, Matthews, Mooar, & Grood, 1983; Noyes, Mooar, Matthews, & Butler, 1983](#)).
510 Therefore, it is possible that the increased behavioral rigidity found in these patients could lead to
511 continuous systematic loading of the same areas on the articulating surfaces of the bones resulting over
512 time in these pathological results. The absence of flexibility in the system does not practically allow
513 for the loading to be more dispersed and over time the result is osteoarthritis. This hypothesis,
514 however, needs to be further explored via additional research. Nonetheless, from this theoretical
515 standpoint, initial experimental work has demonstrated the ability of nonlinear analysis to provide
516 insight on specific causal physiological mechanisms of motor pathology.

517 Based on the above, it can then be asked if ACL reconstruction can restore the LyE values to
518 normative levels. Thus, [Moraiti et al. \(2010\)](#) investigated the functional outcome after ACL
519 reconstruction using bone-patellar tendon-bone (BPTB) and quadrupled semitendinosus and gracilis
520 tendon (ST/G) autografts by evaluating the stride-to-stride fluctuations present in the knee flexion-
521 extension time series. Patients with BPTB and patients with ST/G ACL reconstruction, two years
522 postoperatively, and healthy controls walked on a treadmill at their self-selected pace, while lower
523 extremity kinematics was collected for 100 consecutive strides. Both the BPTB and the ST/G groups
524 had significantly larger LyE values than the healthy controls, even though clinical outcomes indicated
525 complete restoration. No differences were found between the BPTB and the ST/G LyE values.
526 Practically, the ACL reconstruction using either BPTP or ST/G renders the system more noisy and
527 unpredictable as compared to healthy controls. This is probably because ACL reconstruction cannot
528 restore the proprioceptive pathways found in a healthy knee ([Solomonow et al., 1987; Johansson,
529 Sjölander, & Sojka, 1990](#)). These results may indicate that the current reconstruction techniques or the
530 grafts used are not sufficient in restoring knee kinematic variability to normal (i.e., absence of
531 complete reinstatement of the actual anatomy of the ACL; [Arnoczky, Tarvin, & Marshall, 1982](#)).

532 Behaviorally, the findings from the above studies indicate that the ACL deficient individual
533 exhibits a more predictable and rigid behavior with respect to their knee movement variability. On the

534 other hand, after ACL reconstruction the knee demonstrates a more noisy and unpredictable behavior.
535 Clinically, these results can be explained as follows. An individual that knows that the ACL is
536 reconstructed feels “secure” to increase and add extra movement. However, since the proper
537 proprioceptive channels are not there, the temporal structure of the stride-to-stride variations of the
538 knee is not restored to normative levels. On the contrary, the rigidity found in the ACL deficient knee
539 signifies that ACL deficient patients are more “careful” in the way they walk trying to eliminate any
540 extra movements. These behavioral phenomena are well described by the theoretical proposition of the
541 optimal movement variability. Specifically, healthy gait is characterized by an optimal state of
542 movement variability. This state allows for flexibility, adaptability, and ability to respond to
543 unpredictable stimuli and stresses. In our above experiments this is the state that is exhibited by our
544 healthy controls. Decrease or loss of this optimal state is associated with a system that is more rigid
545 and very repeatable, as in the ACL deficient knee. Increase beyond optimal variability is associated
546 with a system, which is noisy and irregular, as in the ACL reconstructed knee (Fig. 3).

547 Furthermore, the impaired variability noted in the reconstructed knee using either graft could be the
548 reason that ACL reconstruction is still linked to susceptibility to further sports injury and development
549 of future pathology without alleviating the problems that were mentioned above for the ACL
550 deficiency. Specifically, long-term follow-up studies have shown an increased incidence of
551 osteoarthritis in ACL-reconstructed knees ([Pinczewski et al., 2007](#)). The studies described above used
552 ACL reconstruction techniques representative of the standard of care for the last fifteen years (i.e.,
553 single graft bundle, typically transtibial drilling of femoral tunnel). Cadaver and in vivo studies have
554 highlighted limitations of this approach for restoring normal knee anatomy and function, and led to a
555 surge of interest in anatomical ACL reconstructions that attempt to better reproduce its actual two-
556 bundle anatomy and insertion sites. The approach used here can provide similar insights for the
557 efficacy of these new surgical techniques for ACL reconstruction for restoring normal knee movement
558 patterns and preserving long-term joint health.

559 *4.2.2. Cerebral concussion: An example of sport-related injury to the brain*

560 Complete recovery of postural control after cerebral concussion is an important determinant of an
561 athlete's readiness to return to competitive activity. Athletes who return to competitive activity too
562 early after injury are potentially more vulnerable to injury recurrence, the consequences of which can
563 be dramatic (Kelly et al., 1991). The assessment of postural control provides an indirect means of
564 identifying concussion-related neurophysiological abnormality (Guskiewicz, Ross, & Marshall, 2001).
565 Postural control traditionally has been characterized according to a biomechanical framework as
566 postural balance. Changes in postural control in athletes after cerebral concussion previously have been
567 measured with a metric known as the equilibrium score resulting from the Sensory Organization Test
568 (SOT) (Guskiewicz, Riemann, Perrin, & Nashner, 1997; Guskiewicz et al., 2001; Guskiewicz, 2002).
569 This score estimates the maximum anterior-posterior angular displacement of the whole body center of
570 gravity based on the range of the anterior-posterior COP displacement. Higher equilibrium scores are
571 derived from lower amplitude COP displacement, thereby assuming greater postural stability. Several
572 studies have raised the possibility that traditional postural stability measures (such as the SOT
573 equilibrium scores; Guskiewicz, 2002) may not be capable of detecting subtle changes in postural
574 control.

575 In response to this concern, Cavanaugh et al. (2005a) recently investigated whether ApEn could
576 detect changes in postural control in athletes with normal postural stability after cerebral concussion.
577 COP data were collected from NCAA Division I (USA) athletes prior to and within 48 hours after
578 injury. After injury, athletes displayed normal postural stability equivalent to preseason levels. For
579 comparison, COP data also were collected from healthy non-athletes on two occasions. Compared to
580 healthy controls, COP oscillations among athletes generally became more regular (lower ApEn value)
581 after injury despite the absence of postural instability. For anterior-posterior time series, declines in
582 ApEn values were much larger in SOT conditions 1 (eyes open on a firm surface) and 2 (eyes closed
583 on a firm surface) than for all other conditions. For medial-lateral time series, ApEn values declined

584 after injury in all sensory conditions. Thus, if an investigator's goal is solely to assess changes in the
585 variability of COP oscillations after cerebral concussion, standing quietly with eyes open and eyes
586 closed on a stable platform may be the only sensory condition that would need to evaluate. Overall,
587 these findings provided preliminary evidence that ApEn could be a sensitive indicator of change in
588 postural control in the acute stage after concussion. However, the authors also wanted to identify why
589 ApEn seems to be sensitive to these changes.

590 Therefore, [Cavanaugh et al. \(2006\)](#) examined the post-concussion recovery of postural control
591 using ApEn. Collegiate athletes from whom COP and symptom data were collected at preseason, less
592 than 48 hours after injury, and 48 to 96 hours after injury, were included in the analysis. Compared
593 with the healthy preseason state, ApEn values for the anterior-posterior and medial-lateral time series
594 declined immediately after injury in both steady and unsteady injured athletes. The decline in ApEn
595 values after concussion reflects changes in the neurophysiological and mechanical constraints on
596 postural control. Diffuse axonal injury reduces and distorts the interactions among neurons in the brain
597 ([McCrorry, Johnston, Mohtadi, & Neeuwisse, 2001](#)), thereby increasing the regularity of cortical
598 oscillations ([Pincus, 1995](#)) that are subsequently manifested in more regular patterns of COP
599 oscillation. Increased co-contraction of the lower extremity musculature is also generated by injured
600 athletes in an attempt to gain control over postural sway. This mechanism can also result in more
601 regular COP oscillations. Above and beyond, the positive relationship between ApEn values and
602 equilibrium scores indicated that larger amplitude COP oscillations (diminished postural control
603 reflected in a lower equilibrium score) tended to be more regular (lower ApEn values), whereas lower
604 amplitude COP oscillations (better postural control reflected in a higher equilibrium score) tended to
605 be more noisy (higher ApEn values). It appears, therefore, that effective postural control is achieved
606 through relatively unconstrained, more irregular patterns of motor output. The ApEn and the
607 equilibrium score have distinct theoretical constructs. ApEn quantifies regularity in the system output
608 to provide clues to underlying system organization ([Pincus & Goldberger, 1994](#)). The ApEn algorithm

609 is a highly iterative process that analyzes the recurrent nature of short sequences of data points
610 considered incrementally throughout a time series. In contrast, the equilibrium score provides little
611 insight into the evolving patterns of variation in postural control performance during the course of a
612 trial. Equilibrium scores are calculated using only two COP data points, the maximum and the
613 minimum, regardless of when they occur. As a biomechanical measure, the resulting range of COP
614 displacement reflects only the amount of variability in the system output.

615 Importantly, the ApEn provides a theoretically distinct and valuable measurement alternative that
616 may prove effective for reducing uncertainty in the return-to-play decision. Another very interesting
617 finding of the above study was that, at 48 to 96 hours after injury, ApEn values for the medial-lateral
618 time series remained significantly depressed, even among athletes whose initial postural instability had
619 resolved. In other words, the effects of cerebral concussion on postural control appeared to persist for
620 longer than 3 to 4 days, even among athletes with no clinical signs of unsteadiness. This finding
621 contrasts with the SOT equilibrium score data that demonstrated that postural instability generally
622 resolves within that time frame allowing athletes to return to sports (Riemann & Guskiewicz, 2000,
623 Guskiewicz et al., 1997, 2001; McCrea et al., 2003). It is then possible that the documented recurrence
624 of cerebral concussions is due to undetected pathology that ApEn is more sensitive to identify
625 (Cavanagh et al., 2005b).

626 Collectively, the above findings support the theoretical model of optimal movement variability,
627 indicating that effective postural control in quiet standing is achieved via relatively unconstrained
628 patterns of motor output. As sensory information was withdrawn or degraded, COP oscillations
629 became more regular. Pincus (1994) and Pincus & Keefe (1992) gave heuristic support for the idea that
630 systems with a relatively limited number of viable interconnections among components may generate
631 more regular output. Newell (1998) proposed a similar idea using a degrees of freedom theoretical
632 framework. Accordingly, either fewer or more poorly organized degrees of freedom reduce the
633 adaptive capability of the individual (Newell, van Emmerik, & Sprague, 1993). Together, these

634 hypotheses suggest that a healthy postural control system, because of numerous interconnections
635 among its components, is capable of adapting to a wide variety of task and environmental demands.
636 Hence, when the system is allowed to operate with minimal constraints (e.g., at rest during quiet
637 standing under normal sensory conditions), the system (i.e. COP oscillations) output appears to
638 fluctuate in relatively random fashion, presumably reflecting the readiness of the system to rapidly
639 respond to perturbation. In the presence of injury, however, normal interconnections among system
640 components would be compromised, thereby reducing the motor flexibility and adaptability of the
641 system. As a result, fluctuations in the system output at rest would be more constrained, appearing
642 more regular. Consistent with the aforementioned hypotheses, the removal of accurate sensory
643 feedback not only made it more difficult for individuals to precisely control body position, but also
644 artificially constrained interactions among control system components, producing more predictable
645 oscillations in system output (Cavanaugh et al., 2005a, 2005b).

646 Based on the above presented studies, future investigations in the postural control after cerebral
647 concussion could explore the following questions: “How long ApEn values remain depressed after
648 injury?”, “Which factors correlate with the eventual return of ApEn values to pre-injury levels?”, and
649 “Which specific neurophysiological or mechanical mechanisms explain the changes in regularity of
650 postural sway after concussion?”. These investigations can lead to the determination of whether the
651 changes in the ApEn values after injury are associated with an increased risk of recurrence of cerebral
652 concussion.

653 *4.3. Chaos in passive dynamic gait models*

654 Full and Koditschek (1999) suggested that the multifactorial nature of locomotion can be
655 approached by using simple models or templates that can be made to resolve the redundancy of
656 multiple legs, joints and muscles by seeking synergies and symmetries. Using this approach, our group
657 sought to identify a template that can exhibit chaos in its gait variability. Such a template can verify

658 that chaos can be present in the fluctuations that are present from one step to the next during
659 locomotion, and then can be used to investigate how chaos in gait can be controlled.

660 A relatively simple model that has been used as a template to address questions about the
661 biomechanical requisites and energetics of bipedal human locomotion is the passive dynamic walking
662 model that walks down a slightly sloped surface (Garcia, Chatterjee, Ruina & Coleman, 1998;
663 Goswami, Thuirot & Espiau, 1998; Kuo, 2001, 2002; McGeer, 1990). Garcia et al. (1998)
664 demonstrated that a simple passive dynamic walking model can exhibit a cascade of period doublings
665 in the walking pattern. They noted that the distances between consecutive period doublings appear to
666 converge to the Feigenbaum constant (4.669201...). This suggested that a passive dynamic walking
667 model might exhibit a chaotic bipedal locomotive pattern (Alligood, Sauer, & Yorke, 1997). However,
668 Garcia et al. (1998) did not examine or prove the presence of chaos per se in the model's locomotion.
669 Nor did they identify which ramp angle is associated with the onset of a chaotic walking pattern. Kurz,
670 Stergiou, Heidel, & Foster (2005), using simulations of the model, were the first to identify that as the
671 ramp angle was increased, a cascade of bifurcations were present in the model's locomotive pattern
672 that lead to a chaotic attractor from $0.01839 \text{ rad} < \text{ramp angle} < 0.0189 \text{ rad}$. These results provided
673 evidence that such a model can be used as a template for exploring the biomechanical control
674 parameters responsible for chaos in human locomotion.

675 Subsequently our group proceeded to investigate how the presence of chaos on our template can be
676 controlled. In two subsequent studies, Kurz and Stergiou (2005, 2007a) demonstrated that
677 implementing "muscles" in the model in the form of hip joint actuations during the swing phase can
678 provide slight perturbations to the unstable manifolds of points in a chaotic system that will promote
679 the transition to new stable behaviors embedded in the rich chaotic attractor. Stable behavior here is
680 when the passive walker does not fall down. The simulations indicated that systematic alterations of
681 the hip joint actuations resulted in rapid transitions to any stable locomotive pattern available in the
682 chaotic locomotive attractor (Kurz & Stergiou, 2007a). Based on these findings, they investigated the

683 benefits of having a chaotic gait with a biologically inspired artificial neural network (ANN) that
684 employed this chaotic control scheme. The ANN was robust and capable of selecting a hip joint
685 actuation that transitioned the passive dynamic model to a stable gait embedded in the chaotic attractor.
686 Additionally, the ANN was capable of using hip joint actuations to accommodate environments that
687 were previously unstable and to even overcome unforeseen perturbations. These simulations provided
688 with an understanding of the advantages that exist when we have a locomotive system that exhibits
689 chaos and provide insight as to how chaos can be used as an advantageous control scheme for the
690 nervous system (Kurz & Stergiou, 2005).

691 Similar results as with the hip joint actuation were produced using toe-off impulses that assist the
692 forward motion of the center of mass (Kurz & Stergiou, 2007b). Furthermore, results from human
693 experiments supported the model's prediction that the control of the forward progression of the center
694 of mass influences the gait dynamics. More recently, Kurz et al. (2010) and Kyvelidou, Kurz, Ehlers,
695 & Stergiou (2008) used the passive bipedal walking model to relate attractor divergence and walking
696 balance. Their simulations revealed that attractors that have a greater amount of divergence are more
697 susceptible to falls from external perturbations. They supplemented these results with human
698 experiments where they demonstrated that elderly and patients with Parkinson's disease have walking
699 patterns that are more noisy with increased LyE values than their young healthy counterparts. These
700 results suggested that elderly and patients with Parkinson's disease may have a higher likelihood of
701 falling as predicted by the theoretical framework of the optimal movement variability.

702 Together, these studies demonstrated that chaos could be a powerful component of the locomotive
703 system. As we mentioned earlier in this review, chaos is necessary for the control of locomotion by
704 allowing the nervous system to rapidly transition to new gaits that are embedded within the chaotic
705 attractor demonstrating healthy flexibility and adaptability. This is reflected in our optimal movement
706 variability model where deterioration of these properties results in lack of health. As demonstrated in
707 the above studies, joint actuations and mechanical perturbations could be used to rapidly transition to

708 any gait available in the bifurcation map of the passive dynamic walking model. In a similar fashion,
709 humans demonstrate predictable scaling in the chaotic structure of the gait pattern as the dynamics of
710 the locomotive system are assisted and the mechanics of the locomotive system are altered. The above-
711 presented experimental results demonstrate that the presence and the way chaos is being exhibited
712 could be controllable which is fundamentally important for the nervous system.

713 **5. Concluding comments**

714 In conclusion, using analysis for nonlinear dynamical systems to human behavior provides a better
715 understanding of variability and how relates with pathology. In this context, the theoretical model of
716 optimal movement variability developed by our research group provides the framework for interpreting
717 both simulated and empirical results. Fields studying movement generation, including robotics,
718 psychology, and neuroscience have utilized concepts and tools related to the pervasiveness of
719 variability in biological systems. The concepts of variability and chaotic variations in human
720 movement along with the advanced tools used to measure these concepts open new vistas for research
721 in movement dysfunction and pathology. In this review we described innovations in the exploration of
722 variability and their potential importance in understanding human movement. Far from being a source
723 of error, evidence supports the necessity of an optimal state of variability for health and functional
724 movement. Concepts of and methods used for nonlinear dynamics offer significant application
725 possibilities to guide rehabilitation practice and research in human movement.

726

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1232 **Figure Captions**

1233 **Fig. 1.** Complementary linear and nonlinear measures from different signals; six signals are displayed,
1234 with the respective values for range and largest Lyapunov Exponent (LyE). The first two time series
1235 are periodic and have been generated using the sine function $15\sin(t/24)$ and the cosine function
1236 $40\cos(t/24)$. The following two time series are chaotic and have been generated using the Rössler and
1237 Lorenz systems, respectively. The final two time series are random and correspond to uniformly and
1238 Gaussian distributed white noise, respectively. All time series contain 4000 data points. The figure
1239 demonstrates that signals can have the same range but differ in terms of temporal structure (LyE) or
1240 they can have different ranges but the same LyE.

1241

1242 **Fig. 2.** Periodic, chaotic, and random time series and their corresponding three-dimensional phase
1243 space plots. The phase space plot is obtained by plotting the original time series and its time delayed
1244 copies. This figure provides with an illustration of a chaotic signal and how is different from other
1245 signals.

1246

1247 **Fig. 3.** Theoretical model of optimal movement variability illustrated using the signals from Fig. 2. For
1248 clarification purposes, the signals presented ("Periodic", "Chaotic", and "Random") are not the only
1249 three possibilities. Behavior in terms of variability should be viewed in a continuum as being more or
1250 less predictable (on the X-axis) or exhibiting or not chaos (on the Y-axis). Thus, there are many other
1251 possibilities.