Title: Limit cycle oscillations in standing human posture

Authors: James R Chagdes^a, Shirley Rietdyk^b, Jeffrey M Haddad^b, Howard N Zelaznik^b,
Michael E Cinelli^c, Luke T Denomme^c, Kaley C Powers^c, Arvind Raman^d

Affiliations: ^aDepartment of Mechanical and Manufacturing Engineering, Miami University,
650 East High Street, Oxford, Ohio 45056.

^bDepartment of Health and Kinesiology, Purdue University, 800 West Stadium
Avenue, West Lafayette, Indiana 47907-2046.

^cDepartment of Kinesiology & Physical Education, Wilfrid Laurier University, 75
University Avenue West, Waterloo, Ontario, Canada N2L 3C5.

^dSchool of Mechanical Engineering, Purdue University, 585 Purdue Mall, West
Lafayette, Indiana 47907-2088.

Corresponding Author: Arvind Raman^d, raman@purdue.edu

Abstract: Limit cycle oscillations (LCOs) are a hallmark of dynamic instability in time-
delayed and nonlinear systems such as climate change models, biological
oscillators, and robotics. Here we study the links between the human
neuromuscular system and LCOs in standing posture. First, we demonstrate
through a simple mathematical model that the observation of LCOs in posture is
indicative of excessive neuromuscular time-delay. To test this hypothesis we
study LCOs in the postural sway of individuals with multiple sclerosis and
concussed athletes representing two different populations with chronically and

21   acutely increased neuromuscular time-delays. Using a wavelet analysis method
22   we demonstrate that 67% of individuals with multiple sclerosis and 44% of
23   individuals with concussion exhibit intermittent LCOs; 8% of MS-controls, 0% of
24   older adults, and 0% of concussion-controls displayed LCOs. Thus, LCOs are not
25   only key to understanding postural instability but also may have important
26   applications for the detection of neuromuscular deficiencies.

27   **Keywords:**   Balance; model; time-delay; bifurcations; limit cycle; neuromuscular disease

28   **Word Count:** 4000

1. Introduction

Limit cycle oscillations (LCOs) are self-sustained periodic motions that mark the onset of dynamic instability in nonlinear and time-delayed systems (Insperger and Stépán, 2011). LCOs are an important focus of nonlinear systems research including physiological control (Mackey and Glass, 1977), chemical reactions (Epstein, 1992; Epstein and Luo, 1991), aeroelasticity (Dowell, 1975; Holmes, 1977), machining (Porter, 1967; Tobias, 1965), robotics (Insperger and Stepan, 2000), climate change (Degregorio et al., 1992), biological oscillators (Batzel and Tran, 2000; MacDonald, 1978), traffic (Orosz et al., 2009), and predator-prey models (Albrecht et al., 1973; May, 1972; Stepan, 1986). In many of the systems listed above, the appropriate combination of nonlinearities and time-delays drive the system away from a static equilibrium, resulting in LCOs.

Nonlinearities and time-delays are also present in sensori-muscular feedback loops that control upright posture (Winters and Stark, 1985) raising the question whether and how LCOs might arise in human postural sway. The presence of LCOs in posture, if confirmed, would imply a pathological loss in the capacity to regulate and stabilize upright stance. Thus LCO mechanisms in human balance could be fundamentally important in the understanding of postural instability and provide important insights into neuromuscular health and rehabilitation.

Previously dynamics of balance has focused on nonlinear analysis of experimental data and not on specific mechanisms of instability such as LCOs. Typically, time-dependent measures such as Lyapunov exponents (Collins and Deluca, 1994; Donker et al., 2007; Ladislao and Fioretti, 2007; Roerdink et al., 2006; Yamada, 1995), entropy measurements (Cavanaugh et al., 2005; Donker et al., 2007; Haddad et al., 2013; Haddad et al., 2011; Roerdink et al., 2006), and recurrence quantification (Haddad et al., 2008; Kinsella-Shaw et al., 2006; Rhea et al., 2011;

Schmit et al., 2006) are used to characterize the dynamics of postural sway. Although these nonlinear methods provide important insights into how postural adaptability and flexibility change with age and neuromuscular disease, insights into the mechanisms responsible are not identified. Additionally, questions about the relationship between changes in time-dependent measures such as entropy and stability of upright posture remain unanswered.

Although LCO behavior has been observed in complex models of postural dynamics that included intermittent controllers (Asai et al., 2009; Gawthrop et al., 2014; Kowalczyk et al., 2012), smooth switch controllers (Eurich and Milton, 1996; Yao et al., 2001), or models that include several muscle groups (Verdaasdonk et al. (2004) the mechanisms for developing LCOs are clouded by the complexity of such models. Stepan (2009) demonstrated the appearance of LCOs in a simple model of upright balance, however passive feedback was neglected.

Thus there is a need to understand the mechanisms of LCOs in simple, yet realistic mathematical models of upright balance which are commonly used (Peterka, 2002). Moreover LCOs need to be measured and detected robustly in specific populations with neuromuscular deficits. In this article we study the onset of LCOs in upright human posture through a bifurcation analysis of a commonly used biophysical-mathematical model and a novel wavelet analysis of experimental posture data of individuals with multiple sclerosis (MS), athletes with concussions, age-matched healthy controls, and older adults.

2. Theoretical Modeling Methods

We first describe the mechanism by which LCOs can arise in a widely used biophysical-mathematical model of human stance where such behavior has not previously been observed. Specifically we adapted the inverted pendulum model from Peterka (2002) to simulate the

anterior-posterior sway angle θ (Fig. 1(a)) where posture is perturbed by external torque M_{ext} and regulated by corrective ankle torque M_{ankle} :

$$\left[I_{body} + m_{body} h_{body}^2 \right] \ddot{\theta}(t) - m_{body} g h_{body} \sin(\theta(t)) = M_{ankle}(t) + M_{ext}(t). \quad (1)$$

The body has mass m_{body} with center-of-mass (CoM) a distance h_{body} from the ankle joint (Fig. 1(a)). M_{ankle} is the sum of passive and active torques $M_{ankle} = M_{passive} + M_{active}$, the former representing torque due to “passive” muscle stiffness and damping, and the latter being torque due to “active” time-delayed neuromuscular feedback through proprioceptive, vestibular, and visual sensory information. The center-of-pressure (CoP) is bounded within the base-of-support. Since the location of the ankle relative to the foot does not change the underlying dynamics of the inverted pendulum model, we assume the ankle is directly above the center of the foot.

Therefore, equation (1) holds valid when $|CoP| \leq \frac{1}{2} L_{foot}$, where L_{foot} is the length of each foot (see appendix A1 for CoP calculation).

Passive control is modeled as a nonlinear proportional-derivative controller,

$$M_{passive}(t) = K(t) [\theta(t) + \beta \theta^3(t)] + C(t) \dot{\theta}(t), \quad (2)$$

where $K(t) = K + n_K(t)$ is the linear muscle stiffness of nominal gain K with noise $n_K(t)$ and β is a dimensionless parameter which represents the nonlinearity in the force-extension/compression response of the muscles involved in postural control (Winters and Stark, 1985). $C(t) = C + n_C(t)$ represents the linear muscle damping (Barauskas and Krusinskiene, 2007; Fukuoka et al., 2001; Maurer and Peterka, 2005; Peterka, 2002, 2003; Vette et al., 2010) of nominal gain C with noise $n_C(t)$. Active control generated from sensory feedback is modeled

as a linear time-delayed proportional-derivative controller (Maurer and Peterka, 2005; Peterka, 2002),

$$M_{active}(t) = K_a(t)\theta(t-\tau) + C_a(t)\dot{\theta}(t-\tau) \quad (3)$$

where $K_a(t) = K_a + n_{K_a}(t)$ is the linear neuromuscular position-feedback of nominal gain K_a and noise $n_{K_a}(t)$, while $C_a(t) = C_a + n_{C_a}(t)$ is the linear neuromuscular velocity-feedback of nominal gain C_a and noise $n_{C_a}(t)$. The proprioceptive, vestibular, and visual systems each estimate the sway angle θ . However, because there is finite time $\tau \in (0, \infty)$ for a postural correction to occur after sensing a deviation, the corrective torque at time t depends on the sway angle at a delayed time $\theta(t-\tau)$ (Masani et al., 2003; Masani et al., 2006; Maurer and Peterka, 2005; Peterka, 2000, 2002, 2003; Ting et al., 2009; Vette et al., 2010).

The appropriate model parameters were non-dimensionalized by the muscle stiffness required to stabilize the upright equilibrium with no additional feedback ($K^{cr} = m_{body}gh_{body}$). K is assumed to be 75% of K^{cr} , consistent with prior work (Tab. 1). β was chosen by matching the amplitude of simulated LCOs using equation (1) to amplitudes seen in people with severe PD (Schmit et al., 2006) and is assumed to be 6000. As seen in Tab. 1, there is a lack of consensus on the appropriate values for C and C_a . While this is true, we will later show that the choice of using C or C_a has very little effect on the stability of upright balance; ultimately what matters is the sum of the two. For this study, we have chosen to assume C to be 20% of K^{cr} while C_a is 0% of K^{cr} (for the exception of the calculation of bifurcation curves only where we show the case of (i) $C = 0.2K^{cr}$ with $C_a = 0$, and (ii) $C = 0$ with $C_a = 0.2K^{cr}$). M_{ext} was chosen to have a

1/f structure (Duarte and Zatsiorsky, 2001) with zero mean and one standard deviation of 0.375% of K^{cr} , consistent with prior work (Asai et al., 2009; Peterka, 2000). The noise in each of the control parameters were chosen to have a 1/f structure with zero mean and one standard deviation of 5% of their respective parameters (i.e. one standard deviation of the noise for K was 5% of K), well below the variation of stiffness gain reported in Winter et al. (1998).

Equation (1) is a time-delayed nonlinear differential equation and its steady solutions and bifurcations in the absence of external or parametric noise were calculated using the DDE-BIFTOOL MATLAB package (Engelborghs et al., 2001). The passive control parameters K , β , and C were held constant while the active control parameters K_a and τ were varied to obtain bifurcations in upright asymptotic stability and mimic the effects of neuromuscular impairment (i.e. MS) (Cameron et al., 2008). Steady state solutions and bifurcation were verified analytically following the work of Campbell et al. (1995) (see appendix A2) and numerically using the MATLAB R2012b (The MathWorks, Inc.) function *dde23* to produce 45 s time series realizations.

The bifurcation analysis shows that upright posture is stable when the parameter combination (K_a, τ) lies in the grey region of Fig. 2. When K_a is decreased such that $K + K_a < K^{cr}$ upright posture loses asymptotic stability for all values of τ via a supercritical pitchfork bifurcation (Fig. 2) and two stable (forward/backward) leaning positions emerge (Fig. 2). On the other hand, upright posture loses asymptotic stability via a Hopf bifurcation when the parameter combination (K_a, τ) lies in the right, white region of Fig. 2 (shown for the case when $C = 0.2K^{cr}$ with $C_a = 0$ (solid line) and for the case when $C = 0$ with $C_a = 0.2K^{cr}$ (dashed lines)). As seen the difference between the two cases is not significant. The reason for this, is that

the contribution of corrective torque arising from position-feedback terms will dominate the system for typical angular rotations (± 0.0044 rad) and velocities (± 0.0028 rad/s) (Winter et al., 1998). Given the lack of consensus in literature, we have chosen to assume $C = 0.2K^{cr}$ and $C_a = 0$ for the remainder of this study. Following the work of Campbell et al. (1995) the Hopf bifurcation can be shown to be supercritical; thus, there co-exists a stable limit cycle: for small perturbation from the limit cycle, the CoP will return to the limit cycle resulting in LCOs.

To better understand the characteristics of these LCOs and their relationship to the control parameters K_a and τ we performed a continuation on the merging LCOs using the DDE-BIFTOOL. This analysis shows that large values of K_a and small values of τ will result in large amplitude and higher frequency LCOs (Fig. 3). Furthermore, this analysis predicts that LCOs will be less than three degrees in amplitude and between 0.2 Hz and 1.2 Hz, within the range of reported postural sway values.

We also present time series simulations of equation (1) with the inclusion of external and parametric noise to better understand how CoP trajectories might evolve over time. When the parameter combination (K_a, τ) is in the region resulting in a stable upright position, CoP realizations are attracted to the upright position with small noise induced fluctuations (Fig. 2(b)). When K_a is such that $K + K_a < K^{cr}$ and the parameter combination (K_a, τ) is in the region left of the pitchfork boundary, CoP realizations are attracted the forward/backward leaning positions but the noise causes CoP to drift between the two (Fig. 2(a)). When the parameter combination is beyond the Hopf boundary, CoP realizations predict a large amplitude oscillation (Fig. 2(c)).

LCOs also occur intermittently, especially at or near the stability boundaries. When K_a and τ are such that the system lies near the supercritical Hopf bifurcation of Fig. 2, the noise in

the control parameters, specifically n_c and n_{K_a} causes the parameter combination (K_a, τ) to transition, slowly and randomly between the grey region (asymptotically stable upright equilibrium) and the right, white region (LCOs). This results in intermittent LCOs as represented by the corresponding CoP time series realization (Fig. 2(d)).

3. Experimental Materials and Methods

To validate the theoretical predictions, we examine CoP time series for the presence of LCOs in people with MS and athletes with concussion, who likely have longer time-delays and, in the case of MS, increased stiffness due to their neurological impairments (Benedetti et al., 1999; Eckner et al., 2012; Rao et al., 1989; Warden et al., 2001). While Benedetti et al. (1999) found an increased ankle stiffness in individuals with MS during gait, they did not differentiate between K and K_a . It is likely that the reported increase in stiffness was a result of an increase in both K and K_a . These two impairments are ideal since they have different etiologies with similar symptoms, allowing us to examine if LCOs are common across disorders with longer time-delays.

3.1. Participants

We analyzed data from two previously published studies. The first data set (reported in Denommé et al., 2014) consisted of 12 individuals with MS (44 ± 9.4 yrs), 12 age-matched healthy controls (45 ± 9.9 yrs), and 12 older adults (68 ± 4.5 yrs) (Tab. 2). The participants with MS had Expanded Disability Status Scale (EDSS) scores (Kurtzke, 1983) of 1.0-3.0 (scale 1-10), reflecting mild cognitive and motor impairments, and no overt signs of instability. The second data set (reported in Powers et al., 2014) examined nine varsity football players with concussion (20 ± 1.2 yrs) (Tab. 3) and nine age- and position-matched healthy teammates who served as

controls (20 ± 1.5 yrs) (Tab. 4). Exclusion criteria included any medication or injury that would affect normal balance control or gait, controls did not have a concussion within the previous 12 months. It was reported that all recruitment and consent procedures were approved by an appropriate IRB.

3.2. Task

Participants were instructed to step onto a force platform (Bertec Legacy Balance System^{TM}, Columbus, OH), place their feet together, and stand with their hands at their sides. Participants completed three trials with eyes open and three trials with eyes closed. Each trial lasted 60 s and the order of visual conditions (eyes open and eyes closed) was randomized. Concussion participants were tested twice: at the acute phase (symptomatic yet able to perform task) and at return-to-play (Tab. 3). The concussion-control testing occurred at the point in time that coincided with the acute phase of their concussed counterparts. In addition, both concussion and concussion-control participants completed a health history questionnaire that included the SCAT2 (2009) symptom evaluation checklist on each testing day.

3.3. Data Processing

Ground reaction forces and moments were sampled at 50 Hz and used to calculate CoP (Winter, 1995). The first 15 s of data were removed to ensure that each participant settled into a quiet standing position after stepping onto the force platform. For consistency, each time series was truncated to 30 s. Data were not filtered.

3.4. Experimental Data Analysis

A LCO is defined as large amplitude periodic motion at a dominant frequency occurring in a system without external, periodic forcing. The developed method to detect LCOs is based on

the observation that LCOs could occur intermittently (Fig. 4(a) & 4(c)); i.e., localized in both time and frequency. Thus, identifying intermittent LCOs requires fine resolution in both time and frequency. Wavelet analysis is useful for determining timescale content at different instances in time and has been used for analyzing postural data (Chagdes et al., 2009; Kirchner et al., 2012; Liang et al., 2014; Suarez et al., 1999, 2001). The continuous wavelet transform allows for the calculation of the wavelet coefficient at any combination of time instance and timescale; whereas, the discrete wavelet transform will only calculate the wavelet coefficients at pre-defined increments (Addison, 2002). This is advantages in the present study as the time instances and timescales of most interest are unknown as the characteristics of LCOs could vary between participants.

For LCO detection using the continuous wavelet transform, we chose to use the Mexican hat wavelet, $\psi(t) = [1 - t^2]e^{-t^2/2}$, because it resembles a smooth cycle of oscillation. The wavelet function is typically normalized by $1/\sqrt{a}$ to have unit energy at all scales

$$\psi_{a,b}(t) = \frac{1}{\sqrt{a}} \psi\left(\frac{t-b}{a}\right). \quad (4)$$

While the frequency content within each wavelet function varies, the frequency at which power spectral density is largest, known as the center frequency, f_c , can be used to better understand how the function will correlate with different signals ($f_c = \sqrt{5/2}/2\pi \approx 0.251$ [Hz] for the Mexican hat mother wavelet). Furthermore, f_c can be used to understand how a change in the scale of the mother wavelet will affect the timescale content captured by the wavelet transform

using the formula $t_a = \frac{a}{f_c f_{\text{sampling}}}$, where f_{sampling} is the sampling frequency of the signal. The

correlation of the signal to the wavelet is represented by the wavelet coefficient, $T(a, b)$.

$T(a, b)$ is calculated by correlating at lag zero the signal, $x(t)$, with the normalized wavelet,

$\psi_{a,b}(t)$, at various timescales, t_a , and time instants, b (Addison, 2002),

$$T(a, b) = \int_{-\infty}^{\infty} x(t) \psi_{a,b}^*(t) dt, \quad (5)$$

where the symbol $*$ represents the complex conjugate. Because the wavelet function is typically normalized by $1/\sqrt{a}$ the magnitude of wavelet coefficients will not only be a factor of signal amplitude, but of scale as well (Addison, 2002). As a result, a short timescale fluctuation will have a larger wavelet coefficient than a longer timescale fluctuation with the same amplitude and shape. This characteristic must be taken into account when interpreting the amplitude of a signal by evaluating the wavelet coefficients at different timescales.

Several parameters were selected as important in the detection of intermittent LCOs.

Local maxima and minima of wavelet coefficients must be of at least magnitude

$T^{cr}(a) = A\sqrt{a/E}$ mm-s ($E = 3\sqrt{\pi}/4$ is the total energy of the Mexican hat wavelet) which

identifies the presence of an oscillation with A mm or greater amplitude at any timescale. By

allowing T^{cr} to be a function of the scale a , the method has the ability to identify LCOs at any

timescale. For this study we chose a strict requirement of $A = 15$ mm to ensure LCOs in CoP

were well above the typical fluctuations of a healthy individual at all timescales (Fig. 4(b) &

4(d)). These maxima and minima must repeat by alternating along the time axis more than $2n$

times and must not drift more than $\pm m$ seconds in timescale per every second. We have chosen

to require $n = 2$ representing two complete oscillation cycles and $m = 1$ to allow for slowly

evolving changes in LCO frequency. Maxima and minima wavelet coefficients are considered to

repeat if the spacing in time between them is within χ percent of their mean timescale. We chose to let $\chi = 10$ to ensure that the oscillation frequency corresponded to the timescale of the maxima and minima wavelet coefficients. Detector performance was tested on realization of the mathematical model and shown to be robust for various magnitudes of external noise and control parameters within the physiological range of quiet stance (see appendix A3).

4. Experimental Results

The wavelet analysis identified LCOs in two of the 12 individuals with MS and none of the MS-controls or older adults when standing with eyes open (Tab. 2). An example of the wavelet analysis method identifying intermittent LCOs (Fig. 5(a)) in an individual with MS can be seen in Fig. 5(c) with the corresponding CoP time series of intermittent LCOs seen in Fig. 5(e). When standing with eyes closed, LCOs were identified in eight of the 12 individuals with MS, one of the 12 MS-controls, and none of the older adults (Tab. 2).

During the symptomatic testing, LCOs were identified in two of the nine concussed athletes when standing with eyes open and in four of the nine concussed athletes when standing with eyes closed (Fig. 6(c); Tab. 3). LCO behavior was not identified in any of the concussion-control athletes in either condition (Fig. 6(d); Tab. 4). When concussed athletes returned-to-play, none of the athletes displayed LCOs when standing with their eyes open; however, one concussed athlete displayed LCOs when standing with their eyes closed (Tab. 3).

5. Discussion

The theoretical analyses demonstrate that a wide range of (K_a, τ) parameter combinations yield a single stable upright equilibrium (Fig. 2(b)), as would be exhibited in a

healthy individual or individual with minimal neuromuscular impairment. However, when the combination of K and K_a is sufficiently small in magnitude ($K + K_a < K^{cr}$), the model predicts the emergence of two stable equilibria that deviate from upright (Fig. 2(a)) resulting from a supercritical pitchfork bifurcation (Fig. 2). This is a *static* instability and the resulting “leaning” postures have been observed in certain clinical populations such as camptocormia (Jankovic and Azher, 2006) and pleurothotonus or “Pisa Syndrome” (Suzuki and Matsuzaka, 2002).

On the other hand, when both K_a and τ are too large, a predicted *dynamic* instability of the upright equilibrium and the emergence of a stable LCO about the upright position via a supercritical Hopf bifurcation (Fig. 2). The emergence of LCOs is determined when neuromuscular health is compromised, specifically, when K_a and τ are modified and τ becomes of the same order as the natural timescale of postural sway. The effects of other control parameters were not investigated and are therefore a limitation of this study.

The predictions of the simple model help to interpret the experimental observations of LCO behavior in people with MS or concussion, who likely have longer time-delays. Unfortunately, these increases in time-delays could not be verified as the data were collected prior to the development of this work. LCOs were detected in 67% of the participants with mild MS, even though they did not demonstrate overt signs of instability, or more subtle changes in the CoP root mean square (Denommé et al., 2014). These experimental findings are similar to the model predictions, such that longer τ and increased K_a will result in LCOs in upright stance. LCOs were only identified in 8% of the MS-control participants and none of the older adults (Tab. 2). This latter observation suggests that LCOs are a signature of postural sway in individuals with neuromuscular impairment that exceeds age-related deficits. The increased time-delay in MS results from increased time needed to process sensory information in higher

brain centers, as well as neural transmission and muscle activation time (Cameron et al., 2008). Increased stiffness associated with MS may also have increased K_a , which would also increase the likelihood of LCO behavior. The individuals with MS who did not demonstrate LCOs likely had different neuromuscular parameters than those with LCOs, consistent with the varying effects of MS on each individual (Kurtzke, 1983). However, even though LCOs were not identified in the postural sway of all individuals with MS, it is clear that the population of individuals with MS displayed more LCOs than the populations of MS-controls and older adults.

Increased time-delay can also be observed following traumatic brain injury, as in concussion. During symptomatic testing, LCO behavior was observed in 22% of the concussed athletes while standing with eyes open and 44% of the concussed athletes while standing with eyes closed (Tab. 3). None of the concussion-control athletes displayed LCOs (Tab. 4). It is interesting to note that LCOs were identified in four of the five athletes with the highest symptom scores (Tab. 3). We had predicted that LCOs would not be observed when athletes return-to-play; this was true for three of the four athletes with LCOs. Concussed player one still demonstrated LCOs at return-to-play (Tab. 3). We argue that the presence of LCOs at return-to-play indicates that a neuromuscular impairment remained in player one. The absence of LCOs in the remainder of the athletes at return-to-play provides strong support for the model.

Interestingly, LCOs were more prevalent when individuals stood with their eyes closed. Based on our model, removal of visual information could be interpreted as increased time-delay. Peterka (2002) also estimated that time-delay increased when vision was removed by fitting a model to experimental data. In the latter study, balance was perturbed in young healthy adults with a support surface rotation. The similar finding across studies – with different perturbations and populations – lends support to the interpretation that removing vision increases time-delay.

We speculate that time-delay increases with eyes closed in order to accrue more information from the somatosensory and vestibular systems to identify the current state of balance before they act, effectively increasing time-delay.

LCOs in postural data have likely been reported in the existing literature without being identified as LCOs. For example, individuals with Parkinson's disease have been reported to show abnormally large periodic oscillations (Maurer et al., 2004; Maurer et al., 2003; Rocchi et al., 2002; Schmit et al., 2006). There are many different interpretations for this behavior (i.e., Schmit et al. (2006) suggest a loss of complexity); however we suggest these individuals are displaying LCOs as a result of their neuromuscular impairments. The transient 1 Hz oscillations identified in Peterka and Loughlin (2004) are potentially a result of a LCO instability.

6. Conclusions

In summary, we have shown that LCOs arise in a commonly used model of human stance when the combination of neuromuscular position-feedback gain and time-delay exceed a threshold. A novel wavelet analysis was used to discover intermittent LCOs in CoP data of eight of the 12 people with MS and four of the nine athletes with concussion. The absence of LCOs in CoP data of the older adults affirms that dynamic instability in quiet stance arises specifically due to chronic or acute neurological deficit and not simply due to aging. The possible evidence of LCOs in CoP data is fundamental for understanding dynamic instabilities and has four potential applications. First, LCO behavior can be used in the detection of neuromuscular impairment. Second, LCO characteristics may differ between disorders, facilitating diagnosis of a specific disorder. Third, LCO characteristics may pinpoint the specific neuromuscular parameters that are compromised in an individual, and facilitate the development of therapeutic interventions. Fourth, changes in LCO characteristics may be used to assess the efficacy of

interventions. The clinical application is especially promising as the assessment takes less than five minutes, requires no subject instrumentation, and can be conducted by a nurse or assistant. In future research we intend to understand the sensitivity of parameter changes in an effort to develop criteria for detection more subtle LCOs, which may facilitate early detection of MS and quantitative assessment of concussion.

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10. Figure Captions

Fig. 1 (a) Diagram of inverted pendulum model for anterior-posterior postural sway with forces and correcting moments along with system geometric parameters, and (b) a block diagram of the postural control system

Fig. 2 Upright equilibrium ($\theta^* = 0$ deg) stability regions of posture where stable regions (gray) and unstable regions (white) are bounded by a supercritical pitchfork bifurcation (blue line) and a supercritical Hopf bifurcation (red line). The Hopf bifurcation curve is shown for two different combinations of passive damping and delayed velocity feedback gains: $C = 0.2K^{cr}$ with $C_a = 0$ (solid line) and for the case when $C = 0$ with $C_a = 0.2K^{cr}$. Above and below the graph are CoP time series realizations of the different stability regions showing the changes in system dynamics where limit cycle trajectories are shown in red

Fig. 3 Characteristics of (a) amplitude and (b) fundamental frequency of limit cycle oscillations in the parameter space of neuromuscular position feedback gain and neuromuscular time-delay

Fig. 4 Experimental CoP time series of a (a) participant with MS showing sustained LCOs, (b) MS-control participant not showing LCOs, (c) participant with concussion showing intermittent LCOs, (d) concussion-control participant not showing LCOs, and (e) older adult participant not showing LCOs

Fig. 5 (a)-(b) Continuous wavelet transform, (c)-(d) continuous wavelet transform with wavelet coefficient of at least magnitude $T^{cr}(a)$ [mm-s], and (e)-(f) corresponding CoP time series, of

536 (left column) an individual with MS showing intermittent LCOs and (right column) an MS-
537 control individual not showing LCOs

538 **Fig. 6** (a)-(b) Continuous wavelet transform, (c)-(d) continuous wavelet transform with wavelet
539 coefficient of at least magnitude $T^{cr}(a)$ [mm-s], and (e)-(f) corresponding CoP time series, of
540 (left column) an athlete with concussion showing intermittent LCOs and (right column) a
541 concussion-control athlete not showing LCOs

542 **11. Table Captions**

543 **Tab. 1** Postural control parameters of similar bipedal models

544 **Tab. 2** MS, MS-control, and older individual characteristics and percentage of LCOs identified
545 in CoP time series

546 **Tab. 3** Individual characteristics and percentage of LCOs identified in CoP time series for
547 athletes with concussions

548 **Tab. 4** Individual characteristics and percentage of LCOs identified in CoP time series for
549 athletes without concussions

Figureg

Fig. 1

Fig. 2

(a)

(b)

Fig. 3

(a)

(b)

(c)

(d)

(e)

Fig. 4

Fig. 5

Fig. 6

Tab. 1

Author	K^{cr} (Nm)	K/K^{cr} (–)	C/K^{cr} (s)	K_a/K^{cr} (-)	C_a/K^{cr} (s)	τ (ms)
Asai (2009)	588.60	0.80	0.01	0.80	0.46	200
Maurer et al. (2005)	648.64	0.90	0.26	0.88	0.03	177
Peterka (2002)	732.19	0.13	0.03	1.32	0.45	100
Vette et al. (2010)	713.81	0.73	0.01	0.18	0.22	80
This Paper	659.92	0.75	0.20	0-1.50	0.00	0-500

Tab. 2

Pair	MS				MS-Controls			Older Adults		
	Age (yrs)	EDSS (1-10)	EO-LCOs	EC-LCOs	Age (yrs)	EO-LCOs	EC-LCOs	Age (yrs)	EO-LCOs	EC-LCOs
1	23	1.0	-	2.9%	24	-	-	61	-	-
2	29	3.0	-	-	32	-	-	62	-	-
3	38	2.0	-	18.1%	38	-	-	63	-	-
4	43	2.0	-	8.3%	42	-	-	65	-	-
5	45	2.0	-	14.8%	44	-	-	66	-	-
6	47	3.0	15.9%	81.2%	46	-	-	69	-	-
7	49	3.0	29.5%	53.4%	48	-	-	70	-	-
8	49	3.0	-	-	49	-	-	70	-	-
9	50	2.5	-	-	51	-	-	72	-	-
10	51	2.0	-	37.0%	52	-	-	72	-	-
11	51	2.5	-	-	55	-	10.2%	73	-	-
12	53	3.0	-	18.4%	59	-	-	74	-	-

Tab. 3

Player	# of Previous Concussions	Symptom Score	Symptom Count	Symptomatic			Return to Play		
				Days Since Concussion	EO-LCOs	EC-LCOs	Days Since Concussion	EO-LCOs	EC-LCOs
1	0	37	16	1	16.0%	11.7%	15	-	10.2%
2	0	4	3	8	-	-	34	-	-
3	0	67	19	13	24.8%	57.4%	41	-	-
4	1	44	19	11	-	39.2%	34	-	-
5	1	5	4	3	-	-	11	-	-
6	0	29	11	3	-	-	10	-	-
7	2	21	14	4	-	-	30	-	-
8	2	51	21	4	-	-	48	-	-
9	0	56	17	1	-	15.6%	15	-	-

Tab. 4

Player	# of Previous Concussions	Symptom Score	Symptom Count	EO-LCOs	EC-LCOs
1	0	0	0	-	-
2	1	0	0	-	-
3	0	0	0	-	-
4	4	28	15	-	-
5	3	0	0	-	-
6	0	1	1	-	-
7	0	0	0	-	-
8	0	0	0	-	-
9	1	6	2	-	-