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Limit cycle oscillations in standing human posture

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

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 sensorimuscular 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., 2011, 2013; 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

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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}:

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

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, Eq. (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,

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

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),

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

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, 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 (Table 1). β was chosen by matching the amplitude of simulated LCOs using Eq. (1) to amplitudes seen in people with severe PD (Schmit et al., 2006) and is assumed to be 6000. As seen in Table 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}

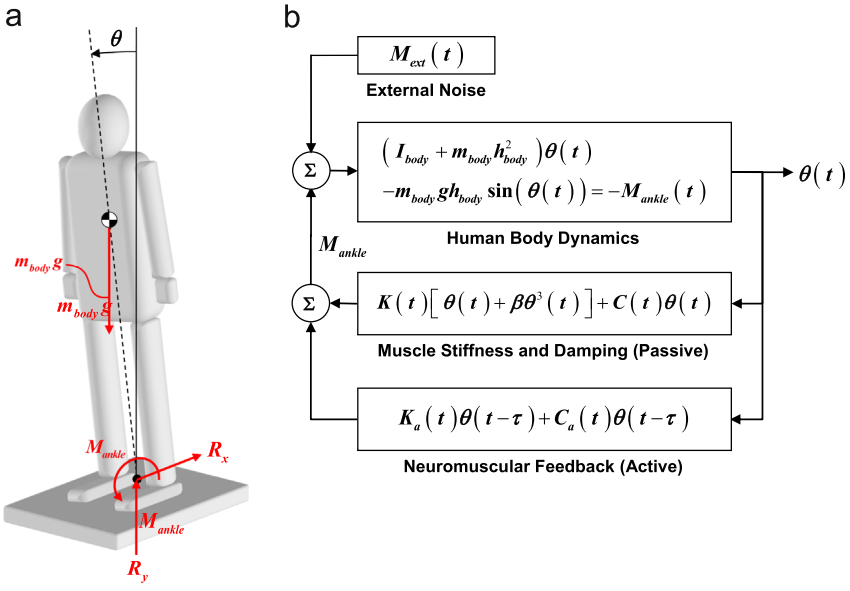

Fig. 1. (a) Diagram of inverted pendulum model for anterior-posterior postural sway with forces and correcting moments along with system geometric parameters, and (b) a block diagram of the postural control system.