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Fatigue-induced spontaneous termination point – Nonequilibrium phase transitions and critical behavior in quasi-isometric exertion

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ABSTRACT

The aim of the present study was to unravel the integration among component processes that jointly lead to a fatigue-induced spontaneous termination point (FISTP), and to pinpoint possible mechanisms underlying this phenomenon. On 5 days during 2 weeks, six participants, who were familiar with the task, performed a quasi-isometric arm-curl exercise holding an Olympic bar with an initial elbow flexion of 90° to the point of spontaneous termination of the exercise due to exhaustion. The repeated measurements ANOVA of windowed variance measures based on the time series of the elbow angles revealed a highly significant effect of the exertion time on the intra- and inter-trial enhancement of the elbow-angle variability when approaching a FISTP. Spectral analysis showed that the variability was generated predominantly in time spans of 1 to about 10 s suggesting that slower and hence higher order control loops are destabilized close to termination points. There was a significant, positive correlation between termination angle and angle variability. The discontinuous change of the elbow angle at the moment of exercise termination was preceded by an enhancement of intra- and inter-trial fluctuations of the elbow angle. This may hint at a nonlinear coupling between the participating neuromuscular components or, more generally, a nonlinear dynamical process underlying the FISTP phenomenon. This dynamical characteristic may indeed explain why other accounts based on separable sites of local, physiological limitations fail to elucidate the occurrence of FISTP.

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1. Introduction

A fatigue-induced spontaneous termination point (FISTP)[1] is a behavioral event defined at a kinematic level as an instant of a spontaneous movement change toward lower energy expenditure levels. This change occurs rapidly, that is, on time scales much shorter than the activity of the system under study. Given its relationship with optimization of energy expenditure, it has been defined as an evolutionary stabilized, protective neuro-metabolic inhibition (e.g., Yakovlev, 1979). As such, it is a ubiquitous phenomenon which is experienced with varying frequency in humans as well as in animals, e.g., a predator terminating the chase of a prey. Given its biological significance, it can be considered an important class of psycho-physiological behavior. Despite its importance, however, the FISTP phenomenon has yet to be explained in detail and literature only contains incidental accounts that usually agree with hybrid linear activation-threshold models of early cybernetics (e.g., Gandevia, 2001; Noakes, St. Clair Gibson, & Lambert, 2005).

Given that fatigue and hence the FISTP are characterized by impairment of the muscles' ability to produce sufficient force for the task under study, most studies focused on the level of muscular skeletal (sub)systems in search for causes of FISTP, be they metabolic (Cairns, 2006; Cairns, Knicker, Thompson, & Sjøgaard, 2005; Hargreaves, 2008; Westerblad, Allen, & Lannergren, 2002) or non-metabolic (Allen, Lamb, & Westerblad, 2008; Ferreira & Reid, 2008; McKenna, Bangsboo, & Renaud, 2008; McKenna & Hargreaves, 2008). Motor spinal and supraspinal levels of the CNS, which fail to drive the motoneurons, as well as more central neurotransmitter mechanisms have also been considered as specific origins of fatigue, or "sites of impairment" (Davis & Bailey, 1997; Gandevia, 2001; Klass, Levenez, Enoka, & Duchateau, 2008; Taylor & Gandevia, 2008). In consequence, with the so-called task dependency model (TDM) it has been suggested that there is no single cause of muscle fatigue, and that the site of impairment and the dominant mechanisms are specific to the task being performed (Enoka & Duchateau, 2008; Maluf & Enoka, 2005). Over the years, the "site of impairment approach" has been challenged, more recently via the so-called "central governor model" (CGM) (Lambert, St. Clair Gibson, & Noakes, 2005; Noakes et al., 2005; St. Clair Gibson & Noakes, 2004). The CGM considers the control of exercise intensity via a single, domain-specific central controller exerting an integrative, homeostatic control over the participating physiological systems. That is the CGM postulates a generic, integrative but central "programmer" that controls motor output (see Atkinson, Peacock, St. Clair Gibson, & Tucker, 2007). Interestingly, other initial ideas about interactive approaches have recently been introduced but still await further development in dedicated research programs (Amann & Calbet, 2008; Amann & Dempsey, 2008; Nybo, 2008; Taylor, Todd, & Gandevia, 2006).

In fact, locally "specialized" as well as central "generic" control seem to contradict more established ideas about self-organizing control that can readily account for transition phenomena like FISTP. This line of thought is advocated here as it starts off with the dynamic interaction between participating component processes. The integration between component psycho-physiological processes is indeed becoming a central issue in fatigue research. For example, although the anatomical, i.e., structural and functional/regulatory integration of the neuromuscular system is known, its dynamics remains to be explored. In a complex system like the neuromuscular system, the hybrid dynamics may be nonlinear. Determining the general class of dynamics of the neuromuscular integration close to the FISTP event can provide important constraints for the very nature of future models and, by extension, for our understanding of exercise-induced fatigue and FISTP.

Nonlinear dynamics has specific but many hallmark features. In the current study the focus is on the presence of critical behavior close to transition points, on account of the analogy in thermodynamics referred to as "phase transitions". Phase transitions occur when (external) control parameters are tuned so that a stable state becomes unstable (Haken, 1983; Nicolis & Prigogine, 1977; Patashinskii &

[1] Fatigue-induced task failure has been defined as a cessation of a bout of exercise (Gandevia, 2001). This cessation, established by convention, coincides with the inability to satisfy some previously defined performance criteria. However, in persistence exercises, participants are able to continue the exertion at relatively high intensity levels far beyond the point when these criteria fail to be met. This phase of exercise is of utmost importance for recognizing the nature of fatigue-induced exercise termination. We chose the expression "fatigue-induced spontaneous termination point" to distinguish this from the determination of task failure by convention.