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Influence of duty cycle on the power-duration relationship: Observations and potential mechanisms

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ABSTRACT

The highest sustainable rate of aerobic metabolism [critical power (CP)] and the finite amount of work that can be performed above CP (W' [curvature constant]) were determined under two muscle contraction duty cycles. Eight men completed at least three constant-power handgrip tests to exhaustion to determine CP and W' for 50% and 20% duty cycles, while brachial artery blood flow ($\dot{Q}_{BA}$) and deoxygenated-[hemoglobin + myoglobin] (deoxy-[Hb + Mb]) were measured. CP was lower for the 50% duty cycle (3.9 ± 0.9 W) than the 20% duty cycle (5.1 ± 0.8 W; $p < 0.001$), while W' was not significantly different (50% duty cycle: 452 ± 141 J vs. 20% duty cycle: 432 ± 130 J; $p > 0.05$). At the same power output, $\dot{Q}_{BA}$ and deoxy-[Hb + Mb] achieved higher end-exercise values for the 20% duty cycle (9.87 ± 1.73 ml·s^{-1}; 51.7 ± 4.7 μM) than the 50% duty cycle (7.37 ± 1.76 ml·s^{-1}, $p < 0.001$; 44.3 ± 2.4 μM, $p < 0.03$). These findings indicate that blood flow influences CP, but not W'.

1. Introduction

The notion of an increase in exercise duration with progressively decreasing power outputs dates back at least to the early twentieth century (Hill, 1925, 1927) and potentially as early as the fourth century (Whipp et al., 1996; Whipp et al., 1998). This robust power-duration relationship is now commonly characterized using a hyperbolic mathematical model to obtain the asymptote (critical power, CP) and the curvature constant (W') (Hill, 1993; Jones et al., 2010; Whipp et al., 1982). CP demarcates the boundary between the heavy- and severe-exercise intensity domains, as it is the highest intensity in which a physiological steady-state can be achieved (i.e., for oxygen uptake ($\dot{V}_{O_2}$) (Poole et al., 1988); blood flow (Copp et al., 2010); intramuscular concentrations of phosphocreatine [PCr], inorganic phosphate [*P*i], and hydrogen ion [H^+] (Jones et al., 2008)). W' represents a finite work capacity that can be performed above CP and has traditionally been associated with 'anaerobic' metabolism (Coats et al., 2003; Ferguson et al., 2007; Fukuba et al., 2003; Miura et al., 1999; Miura et al., 2000; Monod and Scherrer, 1965). This interpretation is supported by [*P*i], [H^+], and [PCr] consistently achieving 'critical levels' upon exhaustion (Chidnok et al., 2013; Jones et al., 2008; Poole et al., 1988; Vanhatalo et al., 2010). Alternatively, W' may be determined by the magnitude of the severe-domain (i.e., the range between CP and $\dot{V}_{O_{2\,max}}$) (Burnley and Jones, 2007; Vanhatalo et al., 2010) and has been associated with the $\dot{V}_{O_2}$ slow component (Ferguson et al., 2007; Jones et al., 2003; Murgatroyd et al., 2011; Vanhatalo et al., 2011). This interpretation is supported by several studies that demonstrated a decrease in W' with interventions that increased CP (Jenkins and Quigley, 1992; Vanhatalo et al., 2008; Vanhatalo et al., 2010). It has been speculated that these decreases in W' were a result of the interventions increasing CP disproportionately to $\dot{V}_{O_{2\,max}}$ and therefore decreasing the magnitude of the severe-domain (Burnley and Jones, 2007; Vanhatalo et al., 2010). Although the mechanism(s) determining W' are not fully understood, it is clear that exercise tolerance for any activity performed at an intensity above CP is limited by the magnitude of W' with exhaustion ensuing upon complete utilization of W' if the power output is not reduced to an intensity equal to or below CP.

Monod and Scherrer (1965), in originally characterizing the power-duration relationship, suggested that CP is dependent upon the circulatory conditions in the muscle, while W' is determined by intramuscular 'anaerobic' (with the exception of O_2 stores) mechanisms. Subsequent experiments have revealed that CP is dependent upon the rate of aerobic ATP production (i.e., O_2 delivery and O_2 utilization) (Dekerle et al., 2012; Hill, 1993; Jones et al., 2010; Moritani et al., 1981; Vanhatalo et al., 2010), while W' (at least in part) is dependent upon 'anaerobic' ATP production (Heubert et al., 2005; Jenkins and Quigley, 1993; Miura et al., 1999; Miura et al., 2000; Smith et al., 1998). Thus, any intervention altering O_2 delivery (i.e., reduced blood flow) to the active skeletal muscle would be expected to alter CP, with presumably no (or little) affect on W'.

The increased intramuscular pressure accompanying muscle contraction can exhibit a profound influence on blood flow as a

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result of blood vessel compression, increased impedance to blood flow, and possible occlusion of blood flow (Hoelting et al., 2001; Lutjemeier et al., 2005; Robergs et al., 1997; Sadamoto et al., 1983). The muscle contraction-relaxation cycle yields rhythmic alterations in intramuscular pressure, and therefore blood flow, with the majority of blood flow occurring during the relaxation period when intramuscular pressure is low (Barcroft and Dornhorst, 1949; Folkow et al., 1970; Robergs et al., 1997; Walloe and Wesche, 1988). Robergs *et al.* (1997) suggesting that the relaxation period blood flow may be important in determining the attainment of a steady-state metabolic rate. The muscle contraction duty cycle (time under tension/total contraction time) directly impacts blood flow, such that with high duty cycles (longer time under tension relative to total contraction time) blood flow to the active skeletal muscle becomes limited (Bellemare et al., 1983; Buchler et al., 1985), while blood flow is not compromised at low duty cycles (shorter time under tension relative to total contraction time) even with increased contraction frequencies (Ferreira et al., 2006; Osada and Radegran, 2002; Sjogaard et al., 2002). Collectively, these results demonstrate that the muscle contraction duty cycle directly influences blood flow to the active skeletal muscle.

To the best of our knowledge there are no reports till date that have examined the influence of alterations in blood flow due to the muscle contraction-relaxation cycle on the parameters of the power-duration relationship. Therefore, the aim of the current study was to manipulate blood flow using muscle contraction duty cycles in order to assess the dependence of CP and W' on blood flow. We hypothesized that: 1) CP would be higher for the 20% duty cycle than the 50% duty cycle, while W' would remain unchanged, further, when the same power output was repeated at both duty cycles, 2) blood flow would be higher for the 20% duty cycle than the 50% duty cycle, but, 3) deoxy-[Hb + Mb] and EMG (electromyography) measurements would achieve similar end-exercise values for both duty cycles.

2. Methods

2.1. Subjects

Eight healthy men (age: 24.8 ± 2.5 years, height: 173.7 ± 4.6 cm; weight: 77.1 ± 14.6 kg) volunteered to participate in this study. Subjects reported to the Human Exercise Physiology Laboratory with at least 24 h between testing sessions and having abstained from vigorous activity within that 24 h period. All experimental procedures in the present study were approved by the Institutional Review Board of Kansas State University and conformed to the standards set forth by the *Declaration of Helsinki*. Prior to testing, each subject was informed of the overall protocol along with the potential risks involved. Each subject then provided written informed consent and completed a health history evaluation.

2.2. Experimental Protocol

All testing was performed on a custom-built handgrip ergometer. The handrail of the ergometer was attached to a pneumatic cylinder by means of a cable-pulley system and provided a fixed linear displacement of 4 cm. Resistance was set by pressurizing the pneumatic cylinder and work was accomplished by compressing the air within the cylinder when the handrail was moved. Power output was calculated as $P = Rdf \cdot k^{-1}$, where P is power in Watts (W), R is resistance in kg, d is displacement in meters, f is contraction frequency, and k is the constant 6.12 for the conversion of kg·m·min^{-1} to W. When seated at the ergometer the subject grasped the handrail so that the forearms were at approximately heart level and the elbows were slightly bent. A contraction

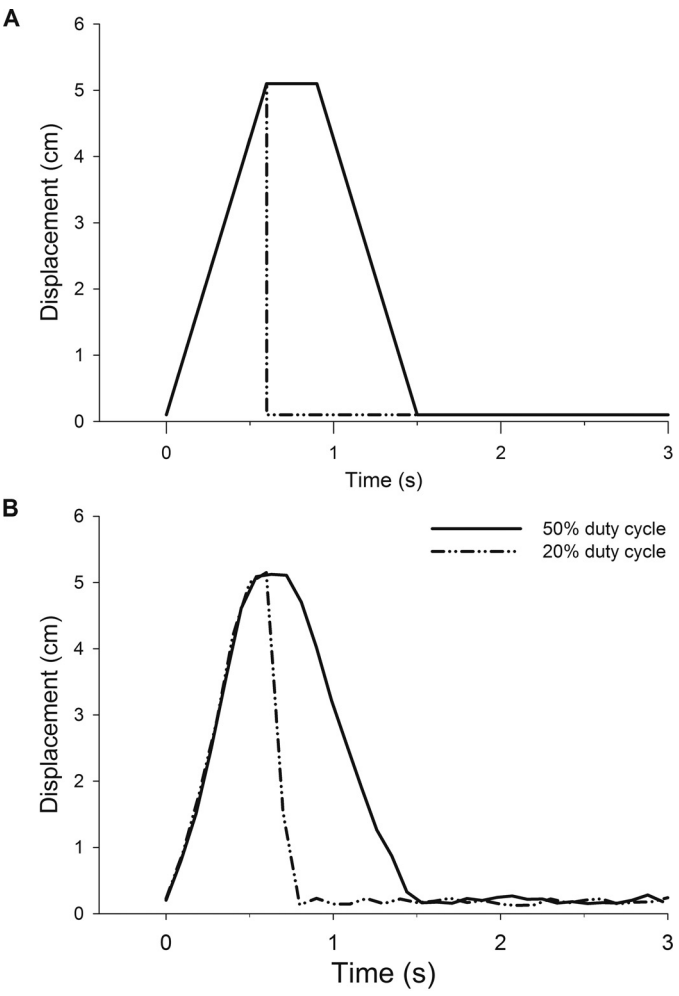

Figure 1. Displacement profiles for the 50% and 20% duty cycles. Schematic representation of the specific contraction components for each duty cycle (Panel A). The 50% duty cycle consisted of a 0.6 s concentric contraction period, a 0.3 s isometric transition period, a 0.6 s eccentric contraction period, and a 1.5 s relaxation period. The 20% duty cycle consisted of a 0.6 s concentric contraction period and a 2.4 s relaxation period. A displacement profile for a representative subject throughout a contraction cycle for each duty cycle is shown in Panel B.

frequency of 20 contractions·min^{-1} was utilized for both duty cycles so that each total contraction cycle duration was maintained at 3.0 s. Thus any set resistance would produce the same power output for both duty cycles. The 50% duty cycle consisted of a 1.5 s contraction period (in which the handrail was raised with concentric muscle contraction and lowered with eccentric muscle contraction) followed by a 1.5 s relaxation period. The 20% duty cycle consisted of a 0.6 s contraction period (in which the handrail was raised with concentric muscle contraction and then immediately released) followed by a 2.4 s relaxation period (Figure 1). Both duty cycles had the same duration of concentric contraction and total contraction cycle, while the 20% duty cycle had no eccentric contraction period and therefore a longer duration of time without muscle tension. The eccentric contraction period duration was altered specifically to minimize any metabolic differences between duty cycles, while emphasizing blood flow differences (see *Discussion*). Audio recordings set with the specific timing for each duty cycle were used along with feedback provided by an investigator monitoring the tests to ensure correct timing. Subjects completed three familiarization trials per duty cycle prior to data collection to aid in correct, consistent production of the contraction-relaxation timing. All testing