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Effects of normobaric hypoxia on upper body critical power and anaerobic working capacity

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ABSTRACT

Purpose: To evaluate the effects of moderate normobaric hypoxia on the parameters of the work-time relationship in the upper body.
Methods: Twenty-one recreationally active men completed a graded exercise test in both normobaric hypoxia (H) and normoxia (N) to exhaustion on an arm ergometer to determine $\dot{V}O_2$peak and peak power output (PPO). Each participant completed four constant work-rate (CWR) arm-cranking tests at 90–120% PPO in both environmental conditions. Linear regression was used to estimate critical power (CP) and anaerobic working capacity (AWC) via work-time relationship during the CWR tests.
Results: H significantly reduced $\dot{V}O_2$peak (N: 2.34 ± 0.34 L vs. H: 2.27 ± 0.34 L, $p = 0.041$), PPO (N: 125.29 ± 17.98 W vs. H: 121.11 ± 17.98 W, $p = 0.001$), and CP (N: 90.22 ± 12.88 W vs. H: 85.26 ± 9.64 W, $p = 0.042$), but had no effect on AWC (N: 9.16 ± 2.81 kJ vs. H: 8.75 ± 3.23 kJ, $p = 0.600$).
Discussion: Moderate normobaric hypoxia appears to reduce CP in the upper body but has a variable effect on AWC.

1. Introduction

Understanding the development of fatigue has long been an interest of human physiologists (Noakes and St Clair Gibson, 2004). In particular, critical power (CP) is a marker of fatigue that has been used to classify the limit of severe intensity exercise (Poole et al., 2016). CP represents the highest aerobic intensity that can be sustained for a prolonged period of time and is a measure of the maximal rate of oxidative energy production (Poole et al., 2016). Alongside CP, anaerobic working capacity (AWC) signifies a finite amount of work that can be performed above CP, which is derived mainly from high-energy phosphates and muscle glycogen with a minimal amount of energy comprised of oxygen stores (Poole et al., 2016).

Altitude can have a significant impact on maximal and submaximal aerobic exercise performance, with sojourns to higher altitudes resulting in decrements in aerobic capacity (Fulco et al., 1998). Elevations in altitude decrease the availability of oxygen to the working muscles thereby limiting mitochondrial efficiency and, in turn, aerobic capacity (Villar and Hughson, 2017). Furthermore, a reduction in oxygen availability reduces maximal oxygen transport and $\dot{V}O_2$max (Bassett and Howley, 2000; Fulco et al., 1998). These altitude-related decrements in aerobic performance likely have a profound impact on other aerobic indices, e.g. CP (Shearman et al., 2016).

The earliest work on the effect of hypoxia on the work-time relationship done by Moritani and colleagues (1981) showed a significant reduction in CP as the inspired oxygen levels decreased, but very little change in AWC. More recent investigations have also shown that moderate levels of acute normobaric hypoxia (simulated altitudes of 2400–3900 m) decrease CP while having no effect on AWC during lower body cycling (Dekerle et al., 2012; Shearman et al., 2016; Simpson et al., 2015). However, Valli et al. (2011) found that severe hypoxia (altitude of 5050 m) elicited a reduction in both CP and AWC. CP serves as the lower boundary of the severe intensity domain, while $\dot{V}O_2$peak or peak power output (PPO) serves as the maximal limit of the severe intensity domain (Poole et al., 2016). The range of the severe intensity exercise domain above CP, or the $\dot{V}O_2$peak/PPO-CP difference, may be of particular interest when examining the effects of hypoxia on AWC since AWC is depleting when exercise is performed above CP or within the severe intensity domain. For example, Simpson et al. (2015) found a negative correlation between changes in the $\dot{V}O_2$peak-CP difference and AWC. The authors concluded that CP is reduced in moderate hypoxia, while the effect on AWC is variable and related to the extent in which hypoxia impacts $\dot{V}O_2$peak and CP.

The relationship between the PPO-CP difference and AWC may

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illustrate a compensation of anaerobic energy production when aerobic production is limited. A lower aerobic provision, at a given work rate, is likely compensated by anaerobic provision especially during intensities above CP. Therefore, AWC depletion with a concomitant development of the $\dot{V}O_2$ slow component, eventually results in exercise intolerance (Calbet et al., 2003b; Poole et al., 2016; Richardson et al., 1998). Townsend et al., (2017) demonstrated a curvilinear decrease in CP with increasing hypoxia; whereas, much greater simulated altitudes were required to induce significant reductions in AWC. Thus far, the effects of hypoxia on the work-time relationship in order to estimate CP and AWC have been limited to lower body cycling exercise.

The work-time relationship has been evaluated in the upper body (Capodaglio and Bazzini, 1996; Fukuda et al., 2014; Taylor and Batterham, 2002; Yang et al., 2017) and has been determined to be a valid assessment of upper body endurance (Belasco et al., 2010). The upper body is not capable of the same aerobic performance as the lower body (Martin et al., 1991). Specifically, the upper body musculature has a smaller muscle mass, smaller diffusion area, a larger diffusion distance, and a greater type II fiber distribution (Calbet et al., 2005; Sanchis-Moysi et al., 2010). Additionally, higher values of $\dot{V}CO_2$, respiratory exchange ratio, and V_E during arm cranking versus leg cycling suggest that the upper limbs may withdraw from anaerobic energy stores earlier than the lower limbs (Martin et al., 1991). Due to the upper body's earlier reliance on anaerobic metabolism to fuel exercise, hypoxia may induce an earlier reliance on glycolytic and phosphagen stores and in turn, elicit fatigue more quickly. Therefore, the aim of this study was to assess the effects of moderate normobaric hypoxia on the parameters of the work-time relationship in the upper body. We hypothesized that CP, relative to normoxia, would be reduced in hypoxia, but have little effect on AWC.

2. Methods

2.1. Participants

Twenty-one recreationally active men (21.4 ± 1.4 y; 175.5 ± 5.7 cm; 84.8 ± 11.7 kg; 18.0 ± 5.2% body fat) who resided at approximately 25 m above sea level volunteered for this study. Each participant was healthy and routinely exercised two to three days per week for at least 30 min. Participants were instructed to maintain their exercise routine except avoiding vigorous exercise 24–48 h before their respective testing visits. All participants completed a confidential medical health questionnaire, a physical activity readiness questionnaire, and provided written consent. This study was approved by the university's institutional review board.

2.2. Experimental design

Participants visited the human performance laboratory on seven separate occasions with at least 24 h separating each visit and no more than one week between visits. The first visit consisted of body composition analysis and an upper body graded exercise test (GXT) familiarization trial. The second and third visits consisted of one GXT under either normobaric normoxia (N) or normobaric hypoxia (H). The fourth, fifth, sixth, and seventh visits consisted of two constant work-rate tests with at least 30 min of rest between trials. Each pair of tests were randomized with respect to intensity (90/110% PPO or 100/120% PPO) and environment (N or H).

2.2.1. Body composition and familiarization trial

Body composition was assessed via multi-frequency bioelectrical impedance analysis (InBody 770, InBody, Seoul, Korea) to determine percent body fat and upper extremity lean mass. A familiarization of the GXT was provided to each subject with the arm ergometer (891E, Monark Upper Body Ergometer, Vansbro, Sweden) before testing sessions took place. Participants performed a five-minute warm-up at 50 W. As adapted from Hill et al. (2016), participants were required to maintain a cranking cadence of 50 revolutions per minute (RPM) at an initial workload of 50 W for three minutes. The workload was then increased 20 W every two minutes until the participant was unable to maintain a cadence above 50 RPM for five seconds despite verbal encouragement, or volitional fatigue. Each subject was seated with the crank arm lined up with the center of their glenohumeral joint and positioned so that their arms were extended but not fully locked out during cranking.

2.2.2. GXT

A GXT to volitional exhaustion was performed on the arm ergometer to determine peak power output (PPO), in watts (W), and peak oxygen consumption ($\dot{V}O_2$peak) in liters per minute ($L\,min^{-1}$). Prior to testing, each participant was fitted with a heart rate monitor (Garmin Ltd., Canton of Schaffhausen, Switzerland), to record the participants' heart rate (HR), and a mask that stabilized a flowmeter turbine around their mouth and nose to collect respiratory gases. All breath-by-breath gas exchange data was collected using a metabolic gas analyzer (Quark CPET, Cosmed, Rome, Italy) and all calibration procedures were carried out as per manufacturer's instructions. The gas calibration (mixture of 16% O_2, 5% CO_2, and Bal N_2) was performed with the metabolic gas analyzer's environmental sensor outside of the tent under normal atmospheric conditions within four hours of each test and the flowmeter calibration with a three-liter syringe was performed before each assessment. The same protocol executed in the familiarization trial was performed, and exhaustion was achieved when the participant could no longer maintain a cadence above 50 RPM for a duration of five seconds despite verbal encouragement or when volitional fatigue was achieved. Participants were kept blind to the environment and their performance, but not their cadence. A metronome set to 100 beats/min (50 RPM) was used in addition to the arm ergometer's digital RPM display to aid the participant's ability to maintain the prescribed cadence. The average of the last 30 s breath-by-breath oxygen consumption rate was recorded as $\dot{V}O_2$peak while PPO, using a staged protocol, was calculated as a percentage of the time spent in the last two-minute stage plus the power output from the preceding stage. For example, if a participant failed one minute into the two-minute stage, 50% of the difference in work rate between stages (i.e. 20 W) was added onto the previous stage's work rate to determine PPO (Kuipers et al., 1985). Participants performed all tests inside of a large transparent cubicle (At-Home Cubicle, Hypoxico, Inc., New York, NY, USA) connected to two external hypoxic generators (Everest Summit II Generator, Hypoxico, Inc., New York, NY, USA) to simulate the environmental condition. After gas calibration and prior to each assessment, the environmental sensor was placed within the tent to verify and measure FiO_2. Participants sat inside the enclosed tent for at least five minutes prior to the start of each test to acclimate to the environment (Dekerle et al., 2012). The environment for the second and third visits were randomized so that each participant would perform the GXT at a simulated altitude of 500 m (F_iO_2 = 20.1 ± 0.2%, normoxic conditions as measured by the environmental sensor) and 3250 m (F_iO_2 = 14.0 ± 0.1%, hypoxic conditions as measured by the environmental sensor). A dehumidifier (DH-35K1SJE5, Hisense International CO., LTD, Quingdao, Shandong, China) was used inside the tent to mitigate extreme increases in relative humidity. The testing environment was monitored via the metabolic gas analyzer's environmental sensor with average values as follows: temperature (24.5 ± 1.87 °C), relative humidity (37.7 ± 6.6%), and barometric pressure (756.8 ± 4.7 mmHg).

2.2.3. Constant work-rate test

When participants arrived for the final four days of testing, they were fitted with a heart rate monitor and then assessed for baseline oxygen saturation (CMD50D+, Contec Medical Systems, Qinhuangdao, China) to verify acclimation. Once the participant entered the enclosed tent, they were instructed to rest for two-minutes to establish resting