ORIGINAL INVESTIGATION (ARTIGO ORIGINAL)

HYPEROXIA DURING RECOVERY IMPROVES PEAK POWER DURING REPEATED WINGATE CYCLE PERFORMANCE.

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Abstract
KAY, B.; STANNARD, S. R.; MORTON, R. H. Hyperoxia during recovery improves peak power during repeated Wingate cycle performance. Brazilian Journal of Biomotricity, v. 2, n. 2, p. 92-100, 2008. Purpose: We have used a random order single blind crossover design to assess the effect of breathing 21% O_2, 60% O_2, and 100% O_2 during a four-minute recovery from a 30s maximal cycle exercise protocol on repeat performance of the exercise. All pairs of Wingate tests were undertaken by participants breathing ambient air, the O_2 percentage was manipulated only during recovery between the exercise bouts. Participants: Participants were 12 males: age 20 or 21, height 181 ± 11 cm, body mass 79 ± 7 kg [means ± SD]). Results: Peak power output was higher during the second Wingate test after 100% O_2 breathing than after both 21% and 60% O_2 breathing, however the rate of fatigue was closely correlated with peak power. Total work was similar for all second tests. Conclusions: 100% O_2 breathing during a four-minute recovery from maximal exercise appears to improve absolute power output in a subsequent exhaustive exercise test, however the rate of fatigue is also increased and the transient ergogenic effect is therefore short lived: perhaps 1-2 seconds. The utility of such an intervention to most sports and physically demanding situations is therefore likely to be limited.

Key Words: Intermittent exercise, maximal exercise, muscle fatigue, ergogenic aids.

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Introduction

In humans, during muscular exercise of a relative intensity exceeding the individual 'critical power or velocity' (CP); acute muscular fatigue sooner or later develops (MORITANI et al., 1981; MORTON, 1996; MORTON & BILLAT, 2004; JONES et al., 2008). This fatigue can be directly detected in the laboratory setting in several ways, including either a time limit to performance at a set power output exceeding the individual critical power (MORITANI et al., 1981; MORTON, 1996; MORTON & BILLAT, 2004; JONES et al., 2008), or a reduction in maximum volitional power output as a function of time (ENOKA & DUCHATEAU, 2007), for example during a supra-maximal exercise test such as the 30s Wingate cycle protocol. It remains unclear what the specific loci of causality are with respect to acute muscular fatigue; however it is clear that these are multi factorial, complex, and task specific (ALLEN & WESTERBLAD, 2001; BARTON, 2002; HEPPLE, 2002; MACINTOSH & RASSIER, 2002; WESTERBLAD & ALLEN, 2003; GREEN, 2004; ABBISS & LAURSEN, 2005; COOKE, 2007).

Recently however, it has been confirmed (JONES et al., 2008) that when exercise exceeds the individual CP, the concentration of phosphocreatine (P-Cr) progressively falls and concomitantly the concentration of inorganic phosphate (Pi) progressively increases. Conversely, when exercise does not exceed CP, these effects are absent: i.e. resting concentrations of these metabolites are not perturbed. This inference could be construed as circumstantial evidence that a reduced concentration of P-Cr is one determinant of exercise induced muscle fatigue. Indeed, it has been shown (HOGAN et al., 1999) that volitional fatigue (i.e. time limit to performance at set work rate) occurs at a given concentration of P-Cr irrespective of work rate or exercise time (those authors manipulated the concentration of P-Cr as a function of exercise intensity and time by imposing normobaric hypoxia during 'ramp testing', and 'steady state' testing). Given its role as an energy shuttle and capacitor (WALLIMANN et al., 1998), especially during intense muscle work, this appears to make sense. Concomitantly, the increase in concentration of Pi may lead to Pi entering the sarcoplasmic reticulum and precipitating out with Ca^{2+}, thus interfering with muscle contraction under certain circumstances (ALLEN et al., 2008; ALLEN & WESTERBLAD, 2001; WESTERBLAD et al., 2002).

The re-phosphorylation of creatine acts to replenish the concentration of P-Cr, and concomitantly reduces the concentration of Pi. This re-phosphorylation of creatine is entirely oxygen dependant (HASELER et al., 1999; HOGAN et al., 1999; WALLIMANN et al., 1998) and the time-constant for P-Cr replenishment is apparently improved during recovery from intense exercise bouts when participants breathe hyperoxic gas (HASELER et al., 1999; HOGAN et al., 1999). Extended to a sporting arena or other physically challenging work situation, these observations may encourage the practice of hyperoxic gas inhalation in order to expedite recovery between strenuous bouts of work, competition, or training. In previous studies regarding P-Cr, exercise performance, and hypoxia and/or hyperoxia (HOGAN et al., 1999 ;HASELER et al., 1999; HASELER et al., 2004; HASELER et al., 2007; TUCKER et al., 2007;

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JONES et al., 2008) participants also breathed hyperoxic gas during the exercise, a practice which is likely to be impractical in many circumstances, and almost certainly disallowed during sporting participation.

No empirical evidence for improved exercise performance coincident with increased fraction of inspired oxygen during recovery from exhaustive exercise *only* appeared to be available in the literature. Therefore, the purpose of this investigation was to assess whether increased fraction of inspired oxygen during recovery from high-intensity exercise (30s Wingate cycle ergometer test), is associated with improved practical performance in a subsequent high-intensity exercise bout (repeated Wingate test) initiated 4-minutes after the previous bout. We hypothesize that 60% O_2 breathing during recovery would be associated with improved repeat Wingate performance as compared to normoxia (21% O_2), and further, that 100% O_2 breathing would be associated with still further improved repeat Wingate performance.

Methods

- Participants / selection

Participants (n=12 recreationally active males, age 20-21 years [range], height 181 ± 11 cm, body mass 79 ± 7 kg [means ± SD]) volunteered to take part and provided written informed consent. The study was approved by the Massey University Human Ethics Committee. Participants first completed a medical history and physical activity questionnaire to assess suitability for participation.

- Experimental design

The effect of breathing normobaric gas with three different oxygen fractions on various physical performance markers were assessed using a random order, single blind crossover design. After several familiarization sessions, participants underwent physical performance testing on three occasions, each separated by at least 48-hours. On each occasion, two consecutive 30-s Wingate cycle ergometer tests (subjects undertook 6 Wingate tests in all): were used to determine peak and minimum power outputs (Watts) whenever they occurred during the tests (minimum power was defined as the lowest power output after peak power had been achieved), time taken to reach peak power (seconds), mean power over the 30-s test duration (Watts), total work over the 30-s test duration (Joules), and finally the rate of fatigue (Watts.second^{-1}).

Each pair of Wingate tests was separated by a 4-minute period of active recovery (cycling at 50W). Participants were required to breathe normobaric gas with three different fractions of oxygen (21%, 60%, and 100% O_2; balance N_2) during the active recovery period only, i.e. all exercise was complete while breathing ambient air. Subjects were not advised as to the gas mixture they were receiving.

- Experimental Protocol

Participants were asked to refrain from consuming alcohol or other recreational drugs throughout the duration of the testing period. Further, participants were asked to refrain from consumption of food and beverage, other than water, in

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the two hours prior to testing. Finally participants were asked to maintain their normal sporting and exercise activities except to refrain from strenuous exercise in the twenty four hours prior to testing. Participants underwent several familiarization trials in the seven days prior to the experimental protocol. Prior-to exercise, the electro-magnetically braked cycle ergometer (Lode, USA) was setup according to individual requirements (seat and handlebar heights, etc). A 5-minute warm up period was completed (cycling at 50W, self-selected cadence). This was followed immediately by the first of two Wingate tests. Participants were instructed to cycle as 'hard and fast' as possible for 30-s, against a resistance based on body weight (0.075 kg·kg body mass^{-1}). Participants received strong verbal encouragement. All output variables were calculated by the Lode ergometer hardware/software and outputted to a linked personal computer. A large gas impermeable bag was filled to normobaric pressure prior to each trial with the appropriate pre-prepared gas mixture (BOC gasses, New Zealand). A two way rotary valve was used to direct whether the prescribed gas or ambient air was inhaled. A plastic tube was attached to the valve, which lead to a non re-breathing mouthpiece (Hans-Rudolph, USA). Participants breathed ambient air during each Wingate test and the prescribed gas during the 4-minute recovery period on each occasion. The second Wingate test began immediately following the recovery period, and this was an exact repeat of the first test. Participants were fed and hydrated, and also closely monitored during a period of 30 minutes post exercise to ensure that no ill effects of the testing resulted.

- Statistics

We used SPSS 16.0 for analysis of variance with repeated measures (general linear model with Huynh-Feldt adjustments to the degrees of freedom where appropriate). Significance was set at an alpha level of 0.05. Least significant difference (LSD) was used post hoc for pair wise comparisons of all means.

Results

There was a significant increase in peak power output achieved during the second Wingate test when 100% O_2 was inspired during the recovery period, as compared to the second Wingate test when 60% O_2 was breathed during the recovery ($P = 0.006$), and also when compared to the second Wingate test when 21% O_2 was inspired during the recovery period ($P = 0.02$). There were no significant differences in time to reach peak power between any of the 6 tests ($P = 0.15$). In terms of minimum power output, all second tests returned lower values than all first tests ($P < 0.03$). Mean power over the 30 second tests was lower in all second tests than all first tests ($P < 0.005$), and the second Wingate test following 100% O_2 breathing returned a higher mean power output value than the second Wingate test following 60% O_2 breathing ($P = 0.02$), but not higher than the second Wingate test following 21% O_2 breathing ($P = 0.60$). Total work during all second Wingate tests was lower than all first tests ($P < 0.001$), and the second Wingate test following 100% O_2 breathing returned a higher total work value than the second Wingate test following 60% O_2 breathing ($P = 0.03$), but not higher than the second Wingate test following 21%

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O_2 breathing (P = 0.66). The rate of fatigue was well correlated with the peak power output value across all tests (r = 0.88, p < 0.01), and was higher for the second Wingate test following 100% O_2 breathing than all other tests (P < 0.02), except the second test following 21% O_2 breathing, which was nearly significant (P = 0.054).

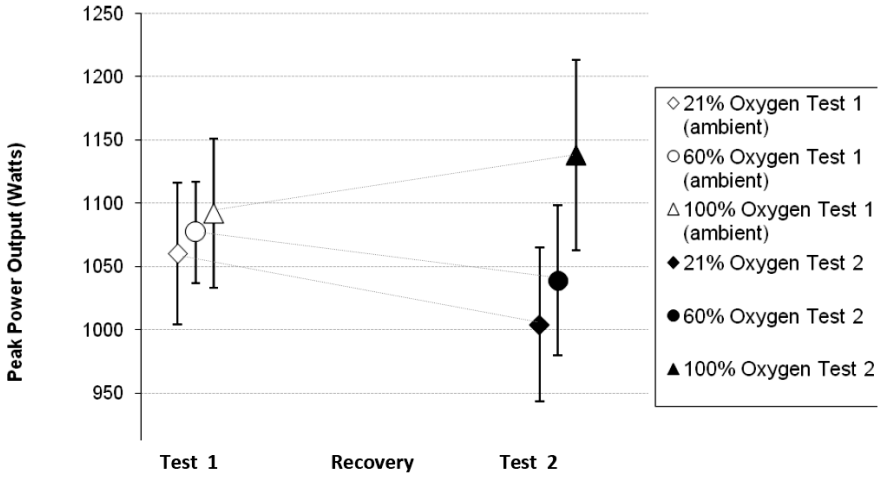

Figure 1 - Peak power (error bars show standard error, n=12) achieved during Wingate cycle tests: All test ones were after breathing ambient air, Test twos were conducted with subjects having breathed either ambient air (21%) or different oxygen enriched mixtures in random crossover fashion (60% and 100%).

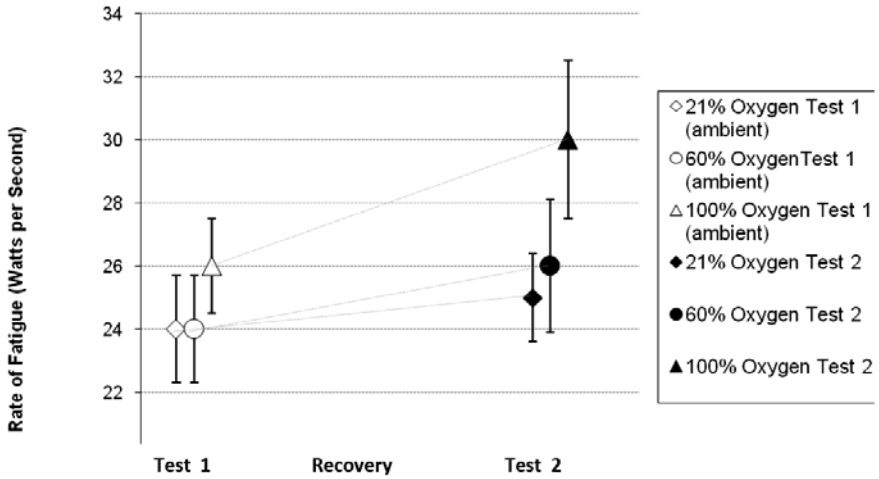

Figure 2 - Rate of fatigue (error bars show standard error, n=12) during Wingate cycle tests: All test ones were after breathing ambient air, Test twos were conducted with subjects having breathed either ambient air (21%) or different oxygen enriched mixtures in random crossover fashion (60% and 100%).

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Discussion

The primary findings of this investigation were that breathing 100% O_2 during the recovery period between Wingate cycle tests was associated with a statistically higher subsequent peak power output than breathing either 60% or 21% O_2. Peak power occurred typically around 3s into each test, and thereafter power declined rapidly for the first 2-3s, and then more gradually to minimum power output by 30s. With respect to rate of fatigue, the observation that there was a correlation with peak power leads us to conclude that the intervention (hyperoxia during recovery between Wingate cycle tests separated by 4 minutes) is apparently not particularly utilitarian, as the benefit of the recovery hyperoxia is quickly expunged (~2-3s) via more rapid fatigue development. Given this very transient ergogenic effect, hyperoxic breathing during recovery only is likely to be of little benefit to most sports and physical work situations. It should be noted that the difference between the value for total work following 60% O_2 breathing as compared to 21% O_2 breathing was -787.5 J (95% confidence limits -104 to -1471 J). This means the difference between these means may equate to a difference in mean power output over the 30s of as low as -3.5W, or as high as -49W. Further research is required to establish whether this observation is an anomaly or whether a 'J shaped' relationship exists when mean and total power are plotted against PO_2.

This investigation has focused on the systemic, practical effect of hyperoxia during recovery from exertion on immediately subsequent exercise performance. Our outcome measures are unable to detect the loci of acute muscular fatigue development, as these are multifaceted as previously cited, and may be of a central and/or of a peripheral nature (NOAKES et al., 2001; TUCKER et al., 2007; AMANN & DEMPSEY, 2008). As performance measures were the only outcome measures taken, we can only speculate as to the mechanisms behind our observations. However, in relation to the muscle itself, the near horizontal nature of the oxyhemoglobin dissociation curve at normal alveolar partial pressure of O_2 (i.e. 95%+ O_2 saturated at ~95 mmHg); makes it appear unlikely that the large increase in alveolar partial pressure of O_2 caused by breathing 100% O_2 (667% over ambient air) would be effective in raising actual oxygen delivery to the mitochondria by a useful margin. However, others (HASELER et al., 1999; HOGAN et al., 1999) have showed that increasing the inspired O_2 percentage to 100% during exercise performance was associated with delayed point of volitional fatigue in a 'ramp test', and that the time constant (half-time) for P-Cr repletion was improved (20-s vs. 25-s, $p<0.05$) during passive recovery when 100% O_2 was inspired. The inverse effects were noted when hypoxia was imposed. Given that P-Cr repletion is dependent on mitochondrial ATP (WALLIMANN et al., 1998), these findings provide surety that O_2 delivery to, and uptake by the mitochondria is indeed usefully increased during passive recovery by breathing 100% O_2 as compared to 21% O_2, possibly via increased plasma borne O_2. Despite this, those authors could not detect a statistically significant increase in VO_2 under hyperoxia during either the exercise or the subsequent recovery (when compared to normoxia).

Alternatively, an increased partial pressure of oxygen may have had some central effect which may help elicit a greater effort during the second bout. It

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has been argued that central governance may limit muscular activation during exercise, in order to protect both the skeletal muscles and the myocardium from ischemia (NOAKES et al., 2001; AMANN et al., 2006; AMANN & CALBET, 2007; AMANN et al., 2007; TUCKER et al. 2007). During hyperoxic exercise, this limitation is apparently attenuated, and during hypoxia it is possibly exacerbated (NOAKES et al., 2001; AMANN et al., 2006; AMANN & CALBET, 2007; AMANN et al., 2007; TUCKER et al. 2007). However, participants in the current study undertook all exercise under normoxic, normobaric conditions. Some participants in this study did report a transient and short-lived sense of euphoria and slight intoxication when given 100% O_2, which may have psychologically predisposed them to exert still more effort than what they considered to be maximal before the hyperoxic intervention. However, because local factors may have remained limiting, the greater peak power achieved was perhaps, at a trade off for a more rapid fatigue and therefore no effect on total work.

Conclusion

This study is the first we are aware of that shows normobaric, hyperoxic breathing during recovery from maximal exertion undertaken in normobaric ambient conditions may provide for a short-lived improvement in peak power development during a second bout of maximal exertion initiated soon after the first. Given the lack of statistically identifiable differences between mean power, total work, and minimum power during the 30s efforts, this transient 'power boost' appears to be of little utility during most competitive and training situations. Future research is required in order to elucidate any clinical benefit or otherwise in given situations.

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