



Muscle and cerebral oxygenation during exercise performance after short-term respiratory work

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ABSTRACT

The purpose of the study was to investigate the effect of 30-min voluntary hyperpnoea on cerebral, respiratory and leg muscle balance between O₂ delivery and utilization during a subsequent constant-power test. Eight males performed a $\dot{V}O_{2\max}$ test, and two exercise tests at 85% of peak power output: (a) a control constant-power test (CPT), and (b) a constant-power test after a respiratory maneuver (CPT_{RM}). Oxygenated ($\Delta[O_2Hb]$), deoxygenated ($\Delta[HHb]$) and total ($\Delta[tHb]$) hemoglobin in cerebral, intercostal and vastus lateralis were monitored with near-infrared spectroscopy. The performance time dropped $\sim 15\%$ in CPT_{RM} ($6:55 \pm 2:52$ min) compared to CPT ($8:03 \pm 2:33$ min), but the difference was not statistically significant. The vastus lateralis and intercostal $\Delta[tHb]$ and $\Delta[HHb]$ were lower in CPT_{RM} than in CPT ($P \leq 0.05$). There were no differences in cerebral oxygenation between the trials. Thus, respiratory work prior to an exercise test influences the oxygenation during exercise in the leg and respiratory muscles, but not in the frontal cortex.

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1. Introduction

Whether exercise-induced respiratory demands for O₂ can influence exercise tolerance; and if so, what could be the potential mechanisms for this limitation, remains unresolved. Several experimental approaches have been used to determine the contribution of respiratory muscle fatigue to endurance exercise performance (Romer and Polkey, 2008). One of these approaches is the pre-fatigue of respiratory muscles through either voluntary hyperpnoea (global fatigue of respiratory muscles) or resistive external loads (fatigue of inspiratory or expiratory muscles) at rest, followed by a whole body exercise performance. Indeed, the findings from studies using such a protocol remain equivocal: some of them have observed significant reductions of exercise performance after pre-fatigue of the respiratory muscles (Mador and Acevedo, 1991; Martin et al., 1982; Verges et al., 2007), while others have not (Dodd et al., 1989; Sliwinski et al., 1996; Spengler et al., 2000).

It has been speculated that the mechanism by which pre-fatigue of respiratory muscles limits exercise tolerance is through activation of the respiratory muscle metaboreflex, resulting in reduced

blood flow to the exercising limb muscles (Dempsey et al., 2002, 2006). Likewise, it has been proposed that a competition for blood flow between the locomotor and the respiratory muscles exists, in such a way that respiratory muscle blood flow may increase at the expense of blood flow to working limb muscles (Harms et al., 1997, 1998). However, none of these studies, which used the experimental approach of the pre-fatigue of respiratory muscles followed by endurance exercise test, monitored blood flow or the levels of oxygenation of respiratory and locomotor muscles.

Aside from peripheral fatigue, previous findings have revealed that cortical deoxygenation in prefrontal regions has a potentially pivotal role in determining maximal exercise performance in healthy individuals exposed to hypoxia (Subudhi et al., 2007) and in patients with terminal lung disease (Jensen et al., 2002); but it does not limit normoxic exercise performance (Amann et al., 2007; Billaut et al., 2009). Nevertheless, it is still unknown whether the frontal cortex area becomes involved in the proposed competition of respiratory and locomotor muscles for blood flow in healthy individuals under normoxic exercise conditions preceded by respiratory muscle fatigue.

During exercise in humans it is not feasible to directly measure blood flow simultaneously in several muscle groups (Nielsen et al., 2001). However, near-infrared spectroscopy (NIRS) offers noninvasive, real-time assessment of local differences in the balance between O₂ consumption ($\dot{V}O_2$) and delivery (Van Beekvelt et al., 2001); and is popularly used to monitor cerebral (Amann

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et al., 2007; Billaut et al., 2009), intercostal (Nielsen et al., 2001; Vogiatzis et al., 2009) and leg muscle (Subudhi et al., 2007) oxygenation during exercise.

Accordingly, the purpose of the present study was to investigate the effect of a 30-min voluntary isocapnic hyperpnoea task on cerebral, respiratory and leg muscle oxygenation during a subsequent constant-power cycle ergometry test. We hypothesized that during the exercise performance test following the respiratory maneuver: (a) the leg muscle oxygenation will be reduced due to exaggerated respiratory muscle metaboreflexes in the pre-fatigued muscles, and (b) the cerebral oxygenation will be unimpaired confirming the notion that it does not constitute a limiting factor during normoxic exercise.

2. Material and methods

2.1. Subjects

Eight healthy, well-trained males (age 23.9 ± 4.6 years, body mass 72.56 ± 6.65 kg, stature 181.33 ± 5.30 cm, body fat $12.99 \pm 3.67\%$) took part in the study. All participants were non-smokers and free of heart and lung diseases and had normal resting pulmonary function, as assessed by a standard pulmonary function test (PFT). The subjects were informed in detail about the experimental procedures and risks involved with the experimental methodology, and gave their informed consent. They were instructed not to engage in any physical activity and not to drink or eat any caffeinated product on testing days. The experimental protocol was approved by the National Committee for Medical Ethics at the Ministry of Health of Republic of Slovenia and conformed to the Declaration of Helsinki.

2.2. Experimental protocol

On a preliminary visit to the laboratory, participants were thoroughly familiarized with the equipment and experimental procedures (cycle ergometer and respiratory maneuver). Thereafter, they participated in three separate trials. During the first session, they performed an incremental exercise test to exhaustion to determine their maximal oxygen uptake ($\dot{V}O_{2\max}$) and peak power output (PPO). On later occasions, each subject performed two constant-power tests in a counter-balanced order, at the same time of the day separated by at least 48-h of resting. Namely, they carried out: (a) a control constant-power test (CPT), and (b) a constant-power test after a 30-min respiratory maneuver (CPT_{RM}) (described below). All the exercise tests were performed on an electrically braked cycle-ergometer (Daun Electronic, Furbth, Germany). During the entire experimental period, the mean ambient temperature, relative humidity and barometric pressure were 20.3 ± 1.0 °C, $46.1 \pm 3.2\%$ and 906.2 ± 1.9 mm Hg, respectively.

2.2.1. $\dot{V}O_{2\max}$ testing

The $\dot{V}O_{2\max}$ test commenced with a 5-min rest period, followed by a 2-min warm up on a cycle-ergometer at a work rate of 60 W. Thereafter, the load was increased by 25 W min^{-1} until exhaustion. Attainment of $\dot{V}O_{2\max}$, defined as the highest $\dot{V}O_2$ averaged over 60 s, was confirmed according to the following classical criteria, listed in priority order: (a) severe fatigue or exhaustion resulting in an inability to maintain exercise at a given work rate (cycling cadence lower than 60 rpm), (b) a plateau in oxygen uptake, (c) a subjective rating of perception of effort at or near maximal, and/or (d) a respiratory exchange ratio >1.10 .

2.2.2. Constant-power tests

During both constant-power tests, the subjects were required to complete a 2-min warm-up on a cycle ergometer at an individ-

ualized work rate of 1.5 W kg^{-1} body weight. Subsequently, they cycled at 85% of PPO (mean power output = 304 ± 46 W). The participants selected their preferred pedal cadence (between 60 and 90 rpm) and they maintained it via visual and verbal feedback throughout the trial. The investigators terminated the test when the pedal cadence dropped below 70% of the self-selected cadence for ≥ 5 -s (task failure). During all tests, subjects remained seated on the cycle ergometer to minimize changes in muscle recruitment; and they received verbal encouragement always by the same investigators.

2.2.3. Respiratory maneuver (RM)

All participants used a respiratory endurance-training device (Spirotiger[®], Idiag, Fehraltorf, Switzerland), which consisted of a hand-held unit with a pouch and a base station (Keramidas et al., 2010). A two-way piston valve connected to a re-breathing bag permitted the addition of fresh inspired air into the bag in order to maintain a constant isocapnic end-tidal CO_2 fraction (Renggli et al., 2008). Personal target values were entered into the base unit that monitored the breathing frequency (f_R), set threshold limits for breathing patterns, and displayed visual and acoustic feedback to allow the subject to breathe within the threshold values for isocapnia.

The RM was performed in an upright standing posture. The duration of the RM was 30 min, and the participants allowed brief respites (30–60 s), when they were unable to maintain the requested f_R . The volume of the bag (V_{BAG}) was set at a value representing approximately 60% of the subject's slow vital capacity (SVC). The f_R was then determined by dividing 80% of maximum voluntary ventilation (MVV) by the bag volume such that $f_R = \text{MVV} (0.80)/V_{\text{BAG}}$. Finally, there was a 12–17-min interval between the RM and the subsequent exercise test.

2.2.4. Pulmonary function

Prior to the start of RM, the participants performed the PFT. Pulmonary function was assessed using a Cardiovit AT-2 plus (Schiller, Baar, Switzerland) spirometer, according to the criteria by Miller et al. (2005). The spirometer was calibrated before every test with a 2-L syringe (Schiller, Baar, Switzerland). Each subject performed each test three times and the highest of the three values was used for subsequent analysis. The PFT was used to obtain measures of forced vital capacity (FVC), forced expiratory volume in 1 s (FEV_1), peak expiratory flow (PEF), slow vital capacity (SVC) and maximum voluntary ventilation (MVV). The PFT was also repeated after the end of RM in order to assess the RM effect on MVV.

2.3. Instrumentation

Respiratory measurements. During the exercise tests, oxygen uptake ($\dot{V}O_2$), ventilation ($\dot{V}E$), carbon dioxide production ($\dot{V}CO_2$), tidal volume (VT) and f_R were measured on-line with a metabolic cart (Quark CPET, Cosmed, Rome, Italy). The gas analyzers and pneumotachograph were calibrated before each test with two different gas mixtures and a 3-L syringe (Cosmed, Rome, Italy), respectively. Data were averaged each minute.

Heart rate (HR) and PPO. HR was measured and recorded using a heart rate monitor (Polar S810i, Kempele, Finland). PPO was calculated by the equation: $\text{PPO} = \text{PO}_{\text{FINAL}} + (t/60 \times 25 \text{ W})$, where PO_{FINAL} refers to the last workload completed, and t is the number of seconds for which the final, uncompleted workload was sustained.

Arterial oxygen saturation (SpO_2). SpO_2 was monitored with a finger pulse oxymeter (BCI 3301, Wisconsin, USA), with an accuracy of ± 2 units across the range of 70–100% and an acceptable resilience to motion artifact (Langton and Hanning, 1990).

Ratings of perceived exertion (RPE). During the respiratory maneuver and the exercise tests, subjects were requested to pro-

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