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Inspiratory loading and limb locomotor and respiratory muscle deoxygenation during cycling exercise

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ABSTRACT

The aim of this study was to determine the effect of inspiratory loading on limb locomotor (LM) and respiratory muscle (RM) deoxygenation ([deoxy (Hb + Mb)]) using NIRS during constant-power cycling exercise. Sixteen, male cyclists completed three, 6-min trials. The intensity of the first 3-min of each trial was equivalent to ~80% $\dot{V}_{O_2\,max}$ ($EX_{80\%}$); during the final 3-min, subjects received an intervention consisting of either moderate inspiratory loading ($Load_{mod}$), heavy inspiratory loading ($Load_{heavy}$), or maximal exercise ($Load_{EX}$). $Load_{heavy}$ significantly increased LM [deoxy(Hb + Mb)] from 12.2 ± 9.0 μm during $EX_{80\%}$ to 15.3 ± 11.7 μm, and RM [deoxy(Hb + Mb)] from 5.9 ± 3.6 μm to 9.5 ± 6.6 μm. LM and RM [deoxy(Hb + Mb)] were significantly increased from $EX_{80\%}$ to $Load_{EX}$; 12.8 ± 9.1 μm to 16.4 ± 10.3 μm and 5.9 ± 2.9 μm to 11.0 ± 6.4 μm, respectively. These data suggest an increase in respiratory muscle load increases muscle deoxy(Hb + Mb) and thus may indicate a reduction in oxygen delivery and/or increased oxygen extraction by the active muscles.

1. Introduction

During moderate-intensity exercise the respiratory musculature require ~3–6% of total oxygen consumption, increasing to ~10–15% during maximal exercise (Aaron et al., 1992b; Harms et al., 1998), which requires a significant fraction of cardiac output to facilitate O_2 delivery. Elevating the work of breathing increases the metabolic demands of the respiratory musculature and during maximal exercise is suggested to promote competition between the respiratory muscles for its required percentage of cardiac output (Dempsey et al., 2006). Further, high respiratory workloads during high-intensity exercise have been shown to reduce limb perfusion by ~14–16% as a consequence of local vasoconstriction, to decrease O_2 consumption of the limb locomotor muscles (~10%), and reduce exercise tolerance by 15% (Harms et al., 1997, 2000). In contrast, it has been suggested that increasing respiratory muscle work during submaximal exercise intensities (50–75% $\dot{V}_{O_2\,max}$) does not present a sufficient stimulus to activate a sympathetically mediated vasoconstrictor reflex, and limb blood flow remains unaffected (Wetter et al., 1999).

The complex nature of the respiratory musculature has limited the ability to measure the O_2 delivery and utilization of these muscles in response to increased ventilatory demands. The measurement of simultaneous muscle groups during exercise has proven difficult, and has therefore tended to be focused on limb locomotor perfusion and $\dot{V}_{O_2}$. Near-infrared spectroscopy (NIRS) is a non-invasive method for assessing localized changes in the oxygenation status of hemoglobin (Hb) and myoglobin (Mb) at the level of the small blood vessels (e.g. arterioles, venules and capillaries) (Mancini et al., 1994), where muscle oxygenation is dependent on both O_2 delivery (blood flow and arterial O_2 content) and O_2 extraction ($a\text{-}v_{O_2\,diff}$). Typically, a change in tissue oxygenation is reflected by changes in the concentration of oxyhemoglobin and oxymyoglobin [oxy(Hb + Mb)], and can be used to infer changes in perfusion of the muscle tissue, whereas an increase in deoxyhemoglobin and deoxymyglobin concentration [deoxy(Hb + Mb)] is used to reflect an increase in muscle deoxygenation, and is dependent on changes in O_2extractionat the tissue (Ferrari et al., 1997).

During progressive incremental maximal exercise, high levels of ventilation (corresponding to ~85% $\dot{V}_{O_2\,max}$) have been shown to be associated with an accelerated decrease in respiratory muscle oxygenation and an attenuated decrease in limb locomotor oxygenation, suggesting that an increase in respiratory muscle demand may limit O_2 delivery (reduction in blood volume) to the limb locomotor muscles during exercise (Legrand et al., 2007). Moreover, NIRS has recently been used in combination with indocyanine

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green dye (a light absorbing tracer) to assess regional blood flow to the respiratory muscles under conditions of high ventilatory loads (Guenette et al., 2008; Vogiatzis et al., 2008). Specifically, Guenette et al. (2008) have shown that as ventilation increased during isocapnic hypernea, respiratory muscle blood flow was highly correlated ($r = 0.995$) with the work of breathing, while inactive muscle blood flow remained constant.

Importantly, the influence of increased ventilatory demand on limb locomotor perfusion during exercise appears to be intensity dependent, in particular where maximal, but not submaximal exercise compromised O_2 availability at the limb locomotor muscle (Harms et al., 1997; Wetter et al., 1999). Moreover, while increasing respiratory muscle work with resistive loading (inspiratory and expiratory), during moderate submaximal exercise (150 W) has been shown to increase limb and respiratory muscle deoxygenation (Nielsen et al., 2001), there is also evidence to suggest that limb locomotor deoxygenation is unaffected by an increase in respiratory load during near-maximal intensity exercise (~94% $\dot{V}_{O_2max}$) (Kowalchuk et al., 2002). Accordingly, the effect of increased ventilatory demands on limb locomotor muscle O_2 availability (delivery) and O_2 extraction during submaximal exercise remains unclear, while assessment of respiratory muscle response during whole-body exercise is limited. These equivocal findings may, in part be related to differing exercise intensities, respiratory muscle load, and ventilation rates used these studies (Kowalchuk et al., 2002; Legrand et al., 2007; Nielsen et al., 2001). Moreover, there appears to be a threshold or minimum intensity of work required to elicit fatiguing muscle contractions which activates this sympathetically mediated vasoconstrictor reflex. Specifically, evidence suggests that submaximal exercise at >80% $\dot{V}_{O_2max}$ provides a sufficient stimulus for fatiguing contractions of the diaphragm (Johnson et al., 1993).

Therefore, the aim of the present study was to evaluate the effect of inspiratory loading on limb locomotor and respiratory muscle deoxygenation during cycling exercise. We hypothesized that the inspiratory loading during submaximal exercise and maximal exercise with no additional inspiratory load would increase the metabolic demand of the respiratory musculature and consequently increase O_2 extraction by the tissue. Since previous studies have demonstrated that increased respiratory muscle work during submaximal exercise at 50–75% $\dot{V}_{O_2max}$ does not influence O_2 delivery to the limb locomotor muscle, we therefore hypothesized that combining an exercise intensity (80% $\dot{V}_{O_2max}$) which has previously been shown to elicit diaphragmatic fatigue (Johnson et al., 1993), and using an inspiratory loading protocol shown to reduce limb locomotor $\dot{V}_{O_2}$ (Harms et al., 1997, 1998) would increase limb locomotor O_2 extraction.

2. Methods

2.1. Subjects

Sixteen healthy, highly trained male competitive cyclists (mean ± SD; age 24 ± 5 years, stature 1.80 ± 0.06 m, mass 76.7 ± 7.7 kg, $\dot{V}_{O_2}$ 61.7 ± 7.3 mL/kg/min) volunteered to participate in this study. All subjects reported to be free from cardiovascular and pulmonary disease, with normal pulmonary function. All subjects were instructed to arrive at the laboratory fully hydrated, at least 4 h postprandial, and to avoid strenuous exercise 24 h prior to each visit. Subjects were also asked to refrain from alcohol and caffeine for at least 24 h and 6 h prior to each exercise session, respectively. All tests and procedures were approved by the Indiana University Institutional Review Board for Human Subjects, and all subjects provided informed written consent to participate in the study.

2.2. Study design

The subjects were required to visit the laboratory on 2 separate occasions in order to complete the exercise tests which were performed on a cycle ergometer (Monark, Model 828E, Varberg, Sweden), and which were separated by at least 48 h. During visit 1 to the laboratory all subjects completed an incremental exercise test to exhaustion to determine maximal oxygen consumption ($\dot{V}_{O_2}$) and peak power output ($\dot{W}_{max}$). During visit 2, all subjects completed an exercise session consisting of a warm-up and 3 experimental exercise trials: (1) a moderate inspiratory loading trial, (2) a heavy inspiratory loading trial, and (3) a maximal exercise loading trial.

2.3. Maximal incremental exercise testing

A maximal incremental cycling ergometer test was performed using a cycle ergometer to determine $\dot{V}_{O_2max}$. The incremental exercise test was initiated at a workload of 150 W, increasing by 50 W every 3 min until volitional exhaustion, or was terminated when the subject's cadence dropped by more than 10 rpm below their self-selected pedal rate. $\dot{V}_{O_2max}$ was determined as the highest 60-s average $\dot{V}_{O_2}$ value achieved prior to exercise termination. The configuration of the saddle and handlebar position was measured and recorded, and replicated in the subsequent exercise test.

2.4. Exercise trials and inspiratory loading

Upon arrival to the laboratory, the subjects were comfortably seated on the cycle ergometer and performed pulmonary and respiratory muscle function testing at rest. Subsequently, all subjects completed a 6-min warm-up, initiated at 150 W for 3 min, increasing to a power output equivalent to 80% $\dot{V}_{O_2max}$ for a further 3 min. Following the warm-up, all subjects completed a protocol modified from Harms et al. (1998) consisting of three, 6-min, randomized exercise trials separated by 20 min rest. The power output during the first 3-min of each exercise trial was conducted at a power output equivalent to 80% $\dot{V}_{O_2max}$, with no loading intervention ($EX_{80\%}$). The second 3-min of each trial consisted of an intervention designed to increase the inspiratory load during exercise which included either: (1) cycling at 80% $\dot{V}_{O_2max}$ while inspiring against a moderate resistive inspiratory load ($Load_{mod}$), (2) cycling at 80% $\dot{V}_{O_2max}$ while inspiring against a heavy resistive inspiratory load ($Load_{heavy}$), or (3) cycling at 100% $\dot{V}_{O_2max}$ ($Load_{EX}$). A 100% $\dot{V}_{O_2max}$ trial was included in the protocol to demonstrate the metabolic and ventilatory responses attained during maximal exercise and compare these responses to those obtained during the inspiratory loading trials.

Resistive inspiratory loading was achieved by placing a flow resistor in the inspiratory line, immediately prior to the mouthpiece. The flow resistor consisted of a rubber stopper with an opening of either 10 mm (moderate inspiratory loading) or 8 mm (heavy inspiratory loading) (Kowalchuk et al., 2002; Nielsen et al., 2001). The diameters used were designed to produce an inspiratory resistance of 6 cm H_2O/L/s during moderate loading and 9.5 cm H_2O/L s during heavy loading.

2.5. Metabolic and ventilatory measurements

During all exercise tests, ventilatory and metabolic data were continuously monitored using open-circuit, indirect calorimetry. Subjects breathed through a low-resistance two-way non-rebreathing valve (Hans Rudolph 2700, Kansas City, MO) which was connected on the expired side to a 5-L mixing chamber. Dried expired gases were continuously sampled at a rate of 300 mL min^{-1}