Oxygenation Responses While Wearing the Elevation Training Mask During an Incremental Cycling Test

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Abstract

Romero-Arenas, S, López-Pérez, E, Colomer-Poveda, D, and Márquez, G. Oxygenation responses while wearing the elevation training mask during an incremental cycling test. *J Strength Cond Res* 35(7): 1897–1904, 2021—The Elevation Training Mask 2.0 (ETM) is a commercial training mask that purportedly simulates altitude training, although their effects have not been conclusively demonstrated. Therefore, the purpose of this study was to evaluate the influence of wearing the ETM on muscle and brain oxygenation responses during a maximal incremental cycling test, as well as the influence of this device on the heart rate (HR) response, perception of effort (rating of perceived exertion [RPE]), arterial oxygen saturation (SaO_2), blood lactate (La^+), and performance (POpeak). Fourteen active males completed an incremental cycling test to volitional exhaustion in 2 separate and counterbalanced conditions, wearing the mask set at 9,000 feet (i.e., 2743 m) and a control condition (CTR, without ETM). During the trial, muscle and cerebral oxygenation were monitored continuously using near-infrared spectroscopy. Heart rate, RPE, and SaO_2 were also recorded from the beginning of the test until the volitional exhaustion. La^+ was measured at the end of each test. Wearing the ETM significantly reduced the POpeak by $-6.9 \pm 6.6\%$ ($p = 0.002$) and this was accompanied by lower La^+ values ($-12.8 \pm 21.6\%$; $p = 0.027$). SaO_2 was also significantly lower at maximal intensity in comparison with the CTR condition ($-1.5 \pm 0.3\%$; $p = 0.028$). However, both HR and RPE showed a similar trend during both sessions, as well as muscle oxygenation. Nevertheless, the mask caused an increase in brain oxygenation compared with the CTR condition ($p < 0.05$). In conclusion, our findings suggest that wearing the ETM causes a pronounced increase in O_2Hb and tHb in the frontoparietal cortex without any change in the muscle oxygenation.

Key Words altitude training mask, near-infrared spectroscopy, oxyhemoglobin, deoxyhemoglobin

Introduction

Athletes worldwide have been using altitude training in its many forms for many years (15). The physiological adaptations that occur as a result of a period of exposure to altitude may benefit subsequent performance at sea level. This idea has prompted the use of training camps based on altitude and also hypobaric chambers where the partial pressure of oxygen in the air can be manipulated to simulate altitude (8). The main physiological challenge caused by exercise at altitude is hypoxia (6); the air is less dense as ambient pressure decreases and, therefore, less oxygen is inspired. The decrease in alveolar oxygen tension results in a lowered oxygen delivery by the red blood cells to the active tissues (8). Consequently, the body demonstrates some adaptive responses that compensate for the relative lack of oxygen in the air. The results obtained from a recent meta-analysis (11) support the notion that a 2-week (336 hours) classic camp (>2,100 m) may be sufficient to increase hemoglobin concentration by a mean of ~3% and by at least 1% for 97.5% of athletes.

There are different devices that allow for simulating typical hypoxic conditions of a high-altitude training camp. Currently, the Elevation Training Mask 2.0 (ETM) (Training Mask LLC, Cadillac, MI) is a new product on the market that claims to invoke the same effects as altitude training to enhance athletic performance, but without a large economic outlay. This device covers the nose and mouth and has different-sized openings and flux valves (19) that can be adjusted to increase the resistance of respiration, making it more difficult to breathe while wearing the mask (12). However, to simulate altitude, the mask should have a mechanism to decrease partial oxygen pressure, inducing a hypoxic state during exercise (19) to increase hemoglobin mass.

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Porcari et al. (19) found that 6 weeks of training with ETM did not cause hematological changes, suggesting that training stimulus did not induce a hypoxic condition. Thus, the failure to significantly observe oxygen desaturation and changes in hematological variables suggested that the ETM works more like an inspiratory muscle training device rather than a simulator of altitude (19).

Limited research is available regarding oxygen delivery to the tissues (i.e., muscle and brain oxygenation) while wearing an ETM. Near-infrared spectroscopy (NIRS) is a noninvasive method for monitoring O_2 availability and utilization by the tissue (20). Thus, skeletal muscle oxygenation decreases in proportion to work rate, reflecting an increased NIRS-determined concentration of deoxyhemoglobin (HHb) (3). Near-infrared spectroscopy measurements at the level of the prefrontal cortex might provide information on the level of cerebral (de)oxygenation during exercise. Bhambhani et al. (2) showed during incremental ramp exercise that cerebral oxyhemoglobin (O_2Hb) increases up to a point at high-intensity exercise where a breakpoint occurs and a decline in O_2Hb is initiated. In addition, they found that HHb remained stable during submaximal exercise but then shows a rapid increase from hard to maximal intensity. These typical O_2Hb and HHb response patterns indicate that cerebral blood flow increases in accordance with the increase in work rate during incremental exercise (3).

In view of the above, there is a need to verify whether these typical O_2Hb and HHb response patterns are altered by wearing an ETM during an incremental exercise. Therefore, the purpose of this study was to evaluate the influence of wearing the ETM on muscle and brain oxygenation responses during a maximal incremental cycling test, as well as, peak power output, heart rate (HR), blood lactate (La^+) concentration, and arterial oxygen saturation (SaO_2). We hypothesized that due to the combination of the reduced breathing frequency on account of the mask's 3 resistance caps, and the rebreathing of expired CO_2 that had been accumulated in the mask's large dead space area (12), the peak power output during an incremental cycling test would be decreased. Furthermore, if the intensity of test is lower during the ETM condition during an incremental test, it could result in a diminished metabolic stress, leading to a reduced HR response and lower levels of La^+ concentration. In addition, we expect higher brain oxygenation response while wearing the ETM produced by an hypercapnic-induced state (17,18). However, based on previous findings (19), it was also hypothesized that both SaO_2 and muscle oxygenation would be similar during an incremental test performed with and without the ETM.

Methods

Experimental Approach to the Problem

A randomized, counterbalanced, within-subject procedure was performed. One week before tests, subjects completed one session to become familiar with the testing protocol and the ETM. Based on previous studies (12,14,19), the mask was set to simulate an altitude of 9,000 feet (i.e., 2,743 m). During this session, subjects sat in the laboratory for 5 minutes while wearing the ETM and then rode for 10 minutes at a self-selected pace on the same mechanically braked cycle ergometer used for testing. During the testing session, all subjects were asked to complete 2 testing conditions in a counterbalanced order. The testing session consisted of 2 incremental cycling tests: with and without ETM. During both tests, muscle and cerebral oxygenation were monitored continuously using NIRS. To quantify the physiological intensity of the testing sessions, HR, rating of perceived exertion (RPE), and SaO_2 were measured every minute during the whole test, in combination with La^+ values, measured after each incremental test. All subjects were tested by the same investigator, using the same procedure, at the same time of day, and in a similar ambient temperature (19–22° C). Subjects were asked to fast for 3–4 hours before testing, to refrain from consuming drinks containing caffeine or alcohol, and to avoid exercise 48 hours before testing sessions. Hydration was allowed at will.

Subjects

Fourteen healthy men volunteered to participate in this study. Subjects' mean ± *SD* age, height, body mass, and body mass index were 24.2 ± 3.0 years, 177.4 ± 6.0 cm, 74.8 ± 6.9 kg, and 23.6 ± 1.6 $kg \cdot m^{-2}$, respectively. The sample size was calculated using the G*Power software (version 3.1.9), which determined that a sample of $n = 13$ subjects would provide a statistical power of over 0.85 for all variables. Inclusion criteria for participation were: BMI between 20 and 25 $kg \cdot m^{-2}$, 18–30 years of age, a normal resting 12-lead electrocardiogram, willing to maintain their current level of physical activity, and physician clearance. Subjects were excluded if they had a history of cardiopulmonary diseases. Before testing, subjects read and signed a written informed consent document where they were informed about the design of the study and possible risks and discomforts related to the tests. Subjects were told that they were free to withdraw from the study at any time, without penalty. The study was conducted according to the Helsinki Declaration, and the Catholic University of Murcia (UCAM) approved all procedures used before the initiation of the study.

Procedures

Incremental Cycling Test. In each testing condition, subjects had to complete a maximal incremental test on a cycle ergometer (Cardgirus Medical, Bikemarc Sport Technology, Barcelona, Spain). After 5 minutes of warm-up, the test began at 0 W and power output was increased by 25 W every minute, while maintaining a cadence of 70–75 rpm, until exhaustion. The test was halted when subjects were no longer able to maintain their cadence over 65 rpm. During the ETM session, subjects wore the mask at the beginning of the incremental test. The mask was set to simulate an

altitude of 9,000 feet (i.e., 2,743 m). Each subject's peak power output (POpeak) was established as the wattage sustained during the last completed stage.

Physiological Measurements and Rating of Perceived Exertion. Heart rate was measured for the duration of the test using a PolarV800 (Polar Electro Oy, Finland). Rating of perceived exertion was assessed using the modified Borg CR-10 scale (4). Subjects reported their RPE 1 minute before (i.e., last minute of the warm-up) and every minute during the test until volitional exhaustion. All subjects were instructed to choose the number from the 0 to 10 scale that best described the level of exertion (0 = not exerted at all; 10 = maximally exerted). SaO_2 was estimated using a finger pulse oximeter (OXYM4000; Quirumed, Valencia, Spain) at 1-minute intervals throughout the exercise. La^+ was measured using Lactate Pro 2 (Arkray, Japan) from capillary blood samples drawn from a fingertip immediately after finishing each incremental test.

Near-Infrared Spectroscopy Measurements. Muscle and cerebral oxygenation were continuously monitored using a commercially available NIRS apparatus (Oxymon Mk III; Artinis Medical Systems, Elst, Netherlands). This device is a 2-wavelength (i.e., 760 and 850 nm), continuous wave system, which uses the modified Beer-Lambert law. Values for tissue oxyhemoglobin + oxymyoglobin, tissue deoxyhemoglobin + deoxymyoglobin, and total tissue hemoglobin (tHb) were reported as a change from baseline (60 seconds averaging before each test) in micromolar-centimeter units (μM-cm). To assess muscle oxygenation, the NIRS optodes were placed on the *vastus lateralis* muscle of each subject's dominant leg, approximately two-thirds of the way down between the greater trochanter and the lateral epicondyle of the femur. The optodes were fixed in a plastic holder, ensuring that the distance between the light source and the detector remained at 4 cm throughout all testing. A large percentage of adipose tissue over the site of interrogation can greatly influence light path length and make it difficult to quantify tissue oxygen; hence, thickness of the adipose layer of tissue overlying the quadriceps muscle was measured using a skinfold caliper (Slim Guide; Creative Health Products, Plymouth, MI), and it was calculated by dividing skinfold thickness by 2 (28), resembling subcutaneous fat and skin. Skinfold thickness at the site of measurement was 4.3 ± 1.6 mm, and considering that the penetration depth of the NIRS signal is half of the source-detector separation (4 cm in this study) and that the calculated values of skin and subcutaneous adipose tissue thickness were relatively low (10), the penetration depth of the NIRS signal thus reasonably reflected the hemodynamic change of the *vastus lateralis* muscle. To assess cerebral oxygenation, the NIRS optodes were placed on the right frontoparietal region at 3 cm from the middle line and 2–3 cm above the supraorbital crest, to avoid the sagittal and frontal sinus areas. The distance between the light source and the detector was 3.5 cm. To ensure that the optodes and detector did not move relative to the subject's skin, the optodes were fixed into position using an adhesive tape that was then secured with a black neoprene sports strapping. A surgical marker pen was used to mark probe placement to identify any device movement during testing. No sliding was observed at the end of any measurement in any subject. Care was taken to ensure that this method of fixation was sufficient to prevent movement of the device during testing without limiting the subject's movement in any way. During all tests, the NIRS system was connected to a personal computer for data acquisition (10 Hz). In the subsequent analysis, NIRS measurements were averaged every 60-second segment during the cycling test, including 1-minute recordings from the last minute of the warm-up, which served as the baseline. Measurements included a standard differential path length factor of 4.0 for the *vastus lateralis* muscle, as specified by the manufacturer, and 5.88 ± 0.10 for the prefrontal cortex (9).

Statistical Analyses

All variables are expressed in relation to the percentage of time to exhaustion, whereby 0% represents the beginning of the incremental cycling test and 100% represents the point of volitional exhaustion, which, in absolute terms, differed between subjects.

Data are presented as mean ± *SD* in the text and tables, and as mean ± *SE* in the figures. Each variable was examined using the Shapiro-Wilk normality test. Two-way repeated-measures analyses of variance were performed with session (ETM and control [CTR]) and time (baseline, start 0 W, 20, 40, 60, 80, and 100%) as factors for the following variables: HR, RPE, SaO_2, vastus lateralis and prefrontal cortex tHb, HHb, and O_2Hb. Post hoc analysis was performed using paired comparisons with Bonferroni correction. Regarding La^+ and POpeak, paired *t*-tests were used to compare both ETM and CTR conditions. Partial eta square (η_p^2) and Cohen *d* were calculated as effect sizes. All analyses were performed using SPSS 19.0 software for Windows (SPSS, Inc., Chicago, IL). Statistical significance was set at $p \leq 0.05$.

Results

POpeak and Blood Lactate

The results of the POpeak and La^+ are shown in Table 1. Wearing the ETM led to a significant reduction (−6.9 ± 6.6%; $p = 0.002$; $d = 0.46$) in the POpeak reached during the incremental test. Furthermore, La^+ values were lower (−12.8 ± 21.6%; $p = 0.027$; $d = 0.55$) after the ETM condition.

Heart Rate, Rate of Perceived Exertion, and Oxygen Saturation

With regard to the HR response during the incremental test, there was a main effect for INTENSITY ($F_{6, 72} = 420.2$, $p < 0.001$; $\eta_p^2 = 0.974$). Heart rate increased significantly from 0 to 100% of the peak power achieved at the end of the test

Table 1. Mean (*SD*) values of POpeak and La^+ during the CTR and ETM conditions.*

	CTR	ETM	Delta (%)	*d*
POpeak (W)	259.6 ± 45.1	240.4 ± 37.6†	−6.9 ± 6.6	0.46
La^+ ($mM \cdot L^{-1}$)	15.9 ± 3.1	13.9 ± 4.1*	−12.8 ± 21.6	0.55

*CTR = control condition; ETM = elevation training mask condition.
†$p < 0.05$; *d*: Cohen effect size.

in both conditions ($p < 0.001$ for all comparisons between baseline and 20, 40, 60, 80, and 100% POpeak; Figure 1A). Similar responses were observed in the RPE values. The analysis showed a main effect for INTENSITY ($F_{6, 72} = 410.1$, $p < 0.001$; $\eta_p^2 = 0.972$). As in the HR response, RPE values significantly increased from the baseline to the maximal intensity achieved during the test (100% POpeak) for both the CTR and ETM conditions ($p < 0.001$ for all comparisons; Figure 1B). Results from SaO_2 showed a main effect for INTENSITY ($F_{6, 72} = 8.9$, $p < 0.001$; $\eta_p^2 = 0.425$) and a CONDITION × INTENSITY interaction ($F_{6, 72} = 3.1$, $p < 0.01$; $\eta_p^2 = 0.205$). Post hoc comparison revealed that during both protocols, SaO_2 was significantly reduced at intensities of 80 and 100% of POpeak ($p < 0.05$ for all comparisons; Figure 1C). However, at maximal intensity (100% POpeak), SaO_2 was significantly lower during the ETM condition compared with the CTR condition (−1.5 ± 0.3%, $p = 0.028$; Figure 1C).

Muscle Oxygenation

Regarding the O_2Hb, the analysis revealed a main effect for INTENSITY ($F_{6, 72} = 23.7$, $p < 0.001$; $\eta_p^2 = 0.664$). Post hoc analysis showed a significant reduction in O_2Hb values from 40 to 100% POpeak in both conditions ($p < 0.01$ for all comparisons when comparing baseline, 0, 20% with 40, 60, 80, and 100% POpeak; Figure 2B). The results of the HHb showed a main effect for INTENSITY ($F_{6, 72} = 47.7$, $p < 0.001$; $\eta_p^2 = 0.779$). The HHb increased significantly from 20 to 80% of the incremental test in both conditions, CTR and ETM, when compared with baseline ($p < 0.01$ for all paired comparisons; Figure 2D). However, the increment of the HHb reached a plateau at 80% of the POpeak, because there were no differences with regard to the maximal intensity (100% POpeak) attained during the incremental test ($p = 0.164$). The tHb results showed a main effect for INTENSITY ($F_{6, 72} = 25.2$, $p < 0.001$; $\eta_p^2 = 0.677$). The tHB significantly increased from 20 to 60% of the POpeak during the incremental test in both conditions when compared with baseline ($p < 0.01$ for all paired comparisons). However, tHb values decreased significantly from 60% POpeak to 100% ($p < 0.05$ for comparisons between 60% and 80–100% POpeak; Figure 2F).

Brain Oxygenation

The analysis of the brain O_2Hb revealed a main effect for CONDITION ($F_{1, 12} = 17.4$, $p = 0.001$; $\eta_p^2 = 0.591$), INTENSITY ($F_{6, 72} = 44.9$, $p < 0.001$; $\eta_p^2 = 0.789$), and a CONDITION × INTENSITY interaction ($F_{6, 72} = 2.7$, $p = 0.022$; $\eta_p^2 = 0.181$). Post hoc analysis showed that O_2Hb during the CTR condition was significantly increased at intensities above 40% (i.e.,: 60–100%) of the POpeak ($p < 0.005$). However, during the ETM condition, baseline O_2Hb values were higher when compared with 0–100% of the POpeak ($p < 0.05$ for all comparisons). Furthermore, brain O_2Hb was

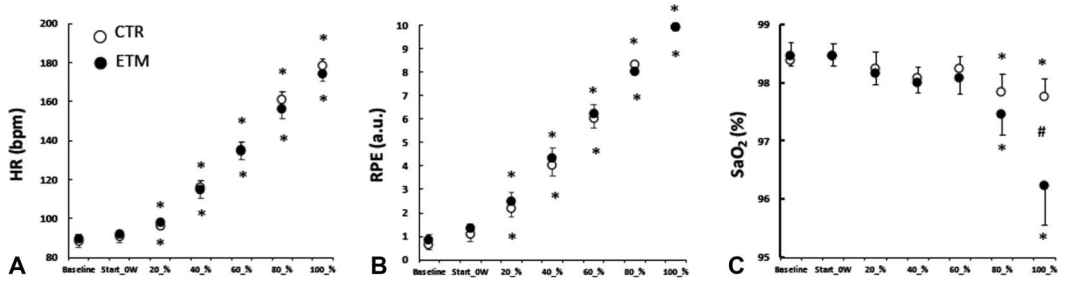

Figure 1. A) Heart rate (HR), (B) RPE, and (C) SaO_2 kinetics during the incremental cycling test during the CTR (open circles) and ETM (black circles) conditions. *Statistical differences in relation to the baseline values. #Statistical differences between both conditions (CTR and ETM). Data are presented as mean ± *SE*. RPE = rating of perceived exertion; CTR = control condition; ETM = elevation training mask condition.

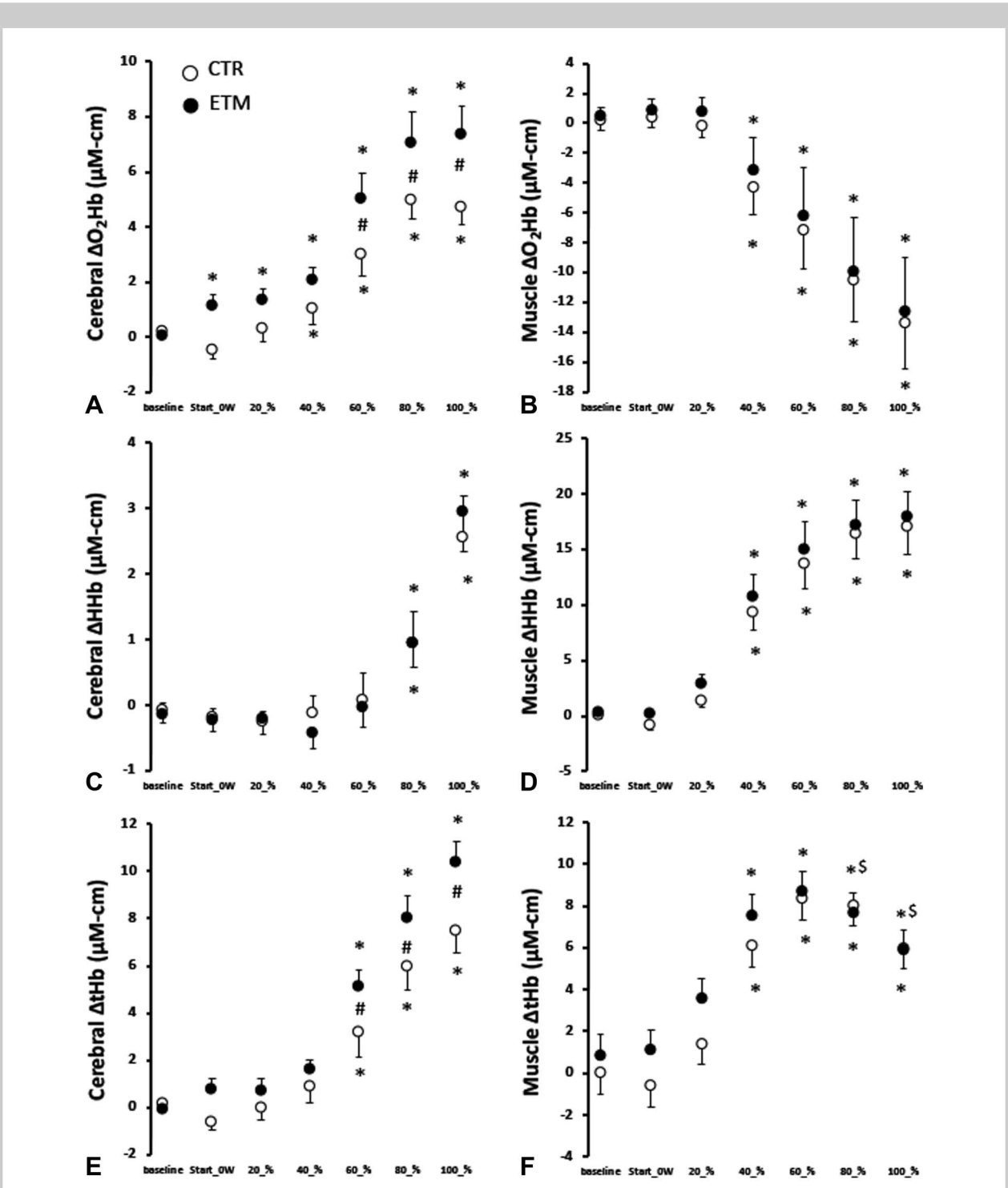

Figure 2. Brain and muscle oxygenation kinetics during the incremental cycling test during the CTR (open circles) and ETM (black circles) conditions. Left panel shows brain oxyhemoglobin, deoxyhemoglobin, and total hemoglobin (A, C, and E, respectively). Right panel shows muscle oxyhemoglobin, deoxyhemoglobin, and total hemoglobin (B, D, and F, respectively). *Statistical differences in relation to the baseline values. #Statistical differences between both conditions (CTR and ETM). $Statistical differences regarding the 60% POpeak. Data are presented as mean ± *SE*. CTR = control condition; ETM = elevation training mask condition.

higher during the ETM than in the CTR condition at intensities of 60–100% POpeak ($p < 0.05$ for all paired comparisons; Figure 1A). The results of the brain HHb showed a main effect for INTENSITY ($F_{6, 72} = 22.9$, $p < 0.001$; $\eta_p^2 = 0.656$). The HHb increased significantly at intensities of 80–100% of the POpeak in both conditions, CTR and ETM, when compared with baseline values ($p < 0.005$ for all paired comparisons; Figure 2C). The analysis of the brain tHb results showed a main effect for CONDITION ($F_{1, 12} = 9.2$, $p = 0.01$; $\eta_p^2 = 0.433$), INTENSITY ($F_{6, 72} = 68.5$, $p < 0.001$; $\eta_p^2 = 0.851$), and a CONDITION × INTENSITY interaction ($F_{6, 72} = 3.5$, $p = 0.004$; $\eta_p^2 = 0.226$). Post hoc analysis showed that tHb was significantly increased at intensities of 60–100% of the POpeak ($p < 0.005$ for all paired comparisons) during the CTR condition. However, during the ETM condition, tHb values were higher than baseline when compared with 40–100% of POpeak ($p < 0.005$). Furthermore, brain tHb values were higher in the ETM condition than in the CTR condition at intensities of 60–100% POpeak ($p < 0.05$ for all paired comparisons. Figure 2E).

Discussion

The principal purpose of this study was to evaluate the physiological responses of wearing the ETM during an incremental cycling test. Specifically, we hypothesized that due to the combination of the reduced breathing frequency on account of the mask's 3 resistance caps, and the rebreathing of expired CO_2 that had been accumulated in the mask's large dead space area (12), the peak power output during an incremental cycling test would be decreased, and O_2Hb and HHb response patterns would be different during incremental exercise with and without ETM. The present results clearly demonstrate that wearing an ETM during an incremental cycling test produced a decrease in POpeak and a pronounced increase in O_2Hb and tHb in the frontoparietal cortex.

An increase in the work of the respiratory muscles can limit exercise performance (24) due to increases in the sensation of breathlessness or due to competition between respiratory muscles and exercising leg muscles for blood flow at maximal intensity (5). Granados et al. (12), in a study designed using a constant workload, suggested that the addition of inspiratory and expiratory resistance, as well as dead space, contributes to reductions in respiratory rate and a small increase in the tidal volume. The net effect of these changes is a small reduction in the ventilation that reduces endurance exercise capacity and limits maximal exercise performance (12). The results of our study are consistent with the findings described above. When wearing the ETM, POpeak was reduced by 6.9%. In addition, La^+ values, which were lower in the ETM condition in comparison with the CTR condition, confirmed that the intensity of test was lower in the ETM condition. These results suggest that wearing the ETM during an incremental test may result in diminished metabolic stress, as evidenced by lower blood lactate levels. Jagim et al. (15) also observed lower La^+ values when subjects performed a resistance training session with the ETM when compared to the same protocol without ETM. These data are in agreement with the those of the study conducted by Chiappa et al. (8) who observed that addition of inspiratory resistance during recovery from intense exercise decreased La^+ values, without changes in arterialized blood gases. Although the reason is unclear, these authors suggested that inspiratory loading may increase respiratory muscle blood flow at the expense of leg blood flow. This augmented perfusion, superimposed with the high capillary density and oxidative capacity of the diaphragm and accessory respiratory muscles, would create a favorable condition for La^+ consumption by the aforementioned muscles. Therefore, the lower La^+ values during the loaded condition could be caused by an elevated uptake of La^+ by the inspiratory muscles (7).

In this study, both HR and RPE increased linearly with exercise load until volitional exhaustion (i.e., 100% of the POpeak). Although expiratory resistive work was not measured in our study, Granados et al. (12) found that the mask's 3 resistance caps impeded inspiratory and expiratory flow, leading to increased HR and RPE responses. This increase would be generated by the reductions in the ventilatory equivalents caused by the mask's resistance that would impede inspiratory and expiratory flow and would lead to reductions in peripheral oxygen saturation (12). In line with this possibility, Stark-Leyva et al. (25) found that during exercise with expiratory loading, the HR increased and the stroke volume decreased because of the higher intrathoracic pressure. However, the additional work of breathing attributed to ventilators is small and has not been shown to influence HR during submaximal exercise (12). However, our study found that wearing the ETM affected neither HR nor RPE. This could be explained by the exercise mode used. Although this study used an incremental cycling test until exhaustion (i.e.,: one-minute steps, 25-W increments), Granados et al. (12) tested these responses during a constant submaximal running test (i.e., 15 minutes at the velocity corresponding to the 65% $\dot{V}O_2$max intensity).

Regarding the SaO_2, present results revealed that arterial oxygen saturation tended to be the same from moderate to maximum intensities, although at 100% of the POpeak, the SaO_2 was 1.56% lower in the ETM condition compared with the CTR. These values represent a normal drop in SaO_2 during high-intensity exercise (22). Similarly, Porcari et al. (19) measured the SaO_2 during weeks 4 and 6, setting the mask to simulate 9,000 feet (i.e., 2,743 m) and 12,000 feet (i.e., 3,658 m), respectively, and the SaO_2 was ~2% lower in the mask group than in the control group (94 vs. 96%). Wehrlin and Hallen (27) evaluated the decrease of the SaO_2 in altitude, and they suggested that the decrease of the SaO_2 in nonacclimated people is about 4–5% for every 1,000 m ascended. Therefore, according to this study, ascending 2,743 m would imply a greater decrease in the SaO_2.

After measuring the concentration of oxyhemoglobin and deoxyhemoglobin with the NIRS, we did not observe differences in the muscle oxygenation responses between the ETM condition and the CTR condition. The results obtained are similar to those of previous NIRS studies during incremental exercise test performed without this device (1,3,13). In relation to the O_2Hb response, it initially remained constant near resting levels, finding a breakpoint around the 20% of the POpeak, where muscle oxygenation decreased more steeply. The HHb showed a triphasic response in both the conditions. In the first phase, the HHb increased slowly, then it showed a steeper slope during moderate-intensity exercise and finally, at the end of the incremental exercise, the HHb showed a plateau. In relation to the tHb, it initially increased slowly, plateaued from 60 to 80% of POpeak, and then decreased steeply. This oxygenation pattern might reflect a decreased blood flow due to a local vasoconstriction at the level of the locomotor muscles due to an increased metabolic load in the respiratory muscles (3). According to this concept, it would be reasonable to hypothesize that the ETM would increase the inspiratory muscle work and would hasten the decrease of the muscle tHB in the ETM condition. Nonetheless, Romer et al. (23) observed that incremental exercise did not elicit diaphragm fatigue because the time exercised by the subjects above 90% of $\dot{V}O_2max$ was possibly not enough to reach fatiguing levels. Therefore, the ETM does not seem to cause muscle oxygenation changes in our experimental design.

Contrary to what happens with the muscle oxygenation, the principal finding of the current study is that in frontoparietal cortex, the O_2Hb and tHb increased in the ETM condition during the incremental cycling exercise. These results are in accordance with the findings by Nielsen et al. (17), who found a similar increase in O_2Hb and tHb when subjects exercised using a resistive breathing device. These authors showed that even a small change in $PaCO_2$ was able to increase brain O_2Hb during exercise. These results indicate that CO_2 influences blood flow to the brain (17,18). Although we did not evaluate CO_2 concentration, our results and these previous findings suggest that rebreathing CO_2 could be occurring and the increase of the cerebral tHb observed in our study might be dominated by the arterial carbon dioxide tension (21). This is also supported by a study in mice that demonstrated how even a small rise in $PaCO_2$ (hypercapnia) leads to vasodilation of cerebral arterioles (18), which can explain the observed increase of the cerebral tHb and O_2Hb while wearing the ETM.

Regarding the question of whether the ETM simulates a hypoxic condition, the brain and muscle oxygenation kinetics observed in this study are different from those observed in the study from Subudhi et al. (26). These authors demonstrated that the incremental exercise to maximal exertion elicited a larger degree of cerebral deoxygenation in hypoxia compared with normoxia. Therefore, the regional cerebral oxygenation decreased progressively from start of the exercise to the point of maximal exertion. Nevertheless, in this study, O_2Hb response patterns were different during incremental exercise (i.e., ETM exacerbated the increase the O_2Hb).

Practical Applications

An important finding in this study was that wearing the ETM affected the pattern of frontoparietal cortex oxygenation during incremental exercise. Results demonstrated that wearing the ETM caused a marked increase in cerebral O_2Hb and tHb concentration. The increased cerebral blood flow (i.e., higher tHb levels) may be related to an increased intracranial pressure, which in turn is a major risk condition for cerebral autoregulation (16). Therefore, this cerebral perturbation should be considered when using this device as a training complement. In addition, the use of the ETM does seem to negatively influence cycling performance (i.e., peak power output), which may attenuate training outcomes over time. However, further studies are needed to determine the cause of this decreased performance in a more ecological setting (e.g., cycling time trial).

Acknowledgments

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