Inspiratory Muscle Training, Altitude, and Arterial Oxygen Desaturation: A Preliminary Investigation

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Introduction: Specific inspiratory muscle training (IMT) has been shown to significantly attenuate the fall in arterial oxygen saturation (S_pO_2) during exhaustive exercise while breathing a hypoxic gas mixture of 14% oxygen. The aim of the current study was to assess the impact of IMT on resting S_pO_2 over a range of altitudes in healthy individuals. ***Methods:*** Resting S_pO_2 and the Borg Score were examined at altitudes of 0 or 400 m (0–400 m; 0–1312.3 ft), 1400 m, 4880 m, and 5550 m (4593 ft, 16,011 ft, and 18,209 ft) in 14 military personnel who were part of a climbing expedition to the Nepali Himalaya. Volunteer participants were randomly assigned to either a control ($N = 7$) or IMT ($N = 7$) group: IMT consisted of 1 set of 30 breaths twice daily at 50% maximal inspiratory mouth pressure (MIP) for 4 wk prior to departure. ***Results:*** MIP was similar between groups pre-IMT but increased significantly by 15% post-IMT. Baseline maximal expiratory mouth pressure was not different between groups. The Borg Score increased significantly from 1400 m, but was not different between groups at any altitude. Resting S_pO_2 declined significantly at ascending altitudes in both groups and was similar between groups at altitudes of 0-400 m and 1400 m. However, at altitudes of 4880 m and 5550 m, S_pO_2 was significantly higher (6%) in the IMT group. ***Conclusion:*** IMT can attenuate the fall in resting S_pO_2, but only at altitudes of 4880 m and above. Conversely, IMT had no effect on resting levels of dyspnea as measured by the Borg Score.

Keywords: hypoxia, SaO_2, IMT, MIP, MEP.

HYPOBARIC AND normobaric hypoxia both induce a number of acute and chronic physiological changes. One of the acute alterations is an increase in minute ventilation, which arises because the reduction in arterial oxygen partial pressure (P_aO_2) stimulates peripheral chemoreceptors. Given the influence of P_aO_2 on hemoglobin oxygen saturation (S_aO_2), it is unsurprising that S_aO_2 (or S_pO_2 if estimated using pulse oximetry) declines during ascent from sea level to altitude (9). However, the reduction in S_aO_2 does not go uncompensated and the hyperventilatory response to hypoxia causes pH to increase (8). This in turn promotes oxygen-hemoglobin binding in the lung and attenuates, although does not abolish, arterial desaturation (13).

The prolonged alkalosis occurring at altitude is harmful and is counteracted (in part) by an increase in 2,3-diphosphoglycerate, which shifts the oxygen dissociation curve to the right (8). Nevertheless, complete respiratory compensation does not occur (13). As a result, a balance is sought between enhancing oxygen loading in the lung, promoting oxygen unloading at the tissue, and minimizing pH disturbances (9).

The consequences of hypoxia on exercise performance have been examined extensively under normobaric hypoxic conditions. Studies have shown that breathing a gas mixture with a fractional inspired oxygen concentration (F_IO_2) of 0.15–0.13 can significantly shorten exercise time to exhaustion (12) and reduce maximal force output of the quadriceps (1). These changes are associated with significantly reduced exercising S_pO_2 values to around 76–82% (1,12). In comparison, breathing a hyperoxic gas mixture (F_IO_2 of 1.00), which preserves S_aO_2, can significantly reduce quadricep muscle fatigue at a given exercise intensity at sea level (1). Similarly, partial acclimatization to moderate altitude improves both S_aO_2 and exercise tolerance (5).

Inspiratory muscle training (IMT) has been shown to have a protective influence on exercising S_pO_2 during normobaric hypoxic conditions [$F_IO_2 = 0.14$, approximate altitude of 3050 m (10,006.6 ft)] by reducing the magnitude of arterial desaturation by approximately 5% (4). As a similar 'protective effect' was not observed in resting S_pO_2 post-IMT, one may postulate that the cardiopulmonary stress at rest was not severe enough for IMT to have a protective influence. If this was the case, IMT may exert a protective effect on resting S_pO_2 when a threshold level or range of hypoxia (and hence altitude) is reached. Therefore, it was the intention of this study to test the hypothesis that at altitudes in excess of 3050 m, IMT would significantly reduce the magnitude of arterial desaturation experienced at rest.

METHODS

The study protocol was approved in advance by the Biosciences Research Ethics Committee at the University of Portsmouth. Each subject provided written informed consent before participating. In total, 14 healthy service personnel from the British Armed Forces (12 men and 2 women) participated. All were part of an expedition to Mount Makalu in the Nepali Himalaya which took place in the summer of 2008. Subjects had no history of cardio-

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vascular or respiratory diseases, had passed a military medical within the past 12 mo, and were experienced mountaineers. Mean and SD for age, body mass, and stature was 37 (±5) yr, 72 (±8) kg, and 1.70 (±0.20) m, respectively.

Experimental Design

Maximal inspiratory and expiratory mouth pressures (MIP and MEP, respectively) were determined in accordance with the American Thoracic Society/European Respiratory Society guidelines (2) on respiratory muscle testing. Following the determination of MIP and MEP (RPM Micro Medical, Rochester, UK), subjects were randomly assigned to either an IMT (N = 7) or control (N = 7) group. The IMT group undertook 4 wk of pressure-threshold IMT (POWERbreathe, HaB International, Southam, Warwickshire, UK) consisting of 1 set of 30 breaths at 30 repetitions maximum (~50–60% MIP), twice daily, 7 days per week. The load on the trainer was adjusted weekly by the subjects to account for their increasing inspiratory muscle strength (7). The start date of the IMT was administered so that the final IMT session finished, at most, 3 d before departing the UK.

S_pO_2 (%) was assessed non-invasively using a pulse oximeter (Onyx 9500 finger probe, Nonin, Plymouth, MA) at sea level or 400 m [0-400 m (0–1312 ft); day 1] and at altitudes of 1400 m (4593 ft; day 1), 4880 m (16,011 ft; day 16), and 5550 m (18,209 ft; day 20; two subjects reached this camp on day 24), which corresponded to expedition camps in the Nepali Himalaya. To standardize data collection S_pO_2 was measured within 24 h of arrival at the camps and where possible at a similar time of day at each camp. Dyspnea was assessed at each camp using the Borg CR-10 scale. The number of subjects assessed at each altitude, along with barometric pressure (P_B; mmHg) and air temperature (°C) can be found in **Table I**.

Data Analyses

As parametric data assumptions were met (Shapiro-Wilks test), two-way repeated measures ANOVAs with between-subject effects (IMT vs. control) were used to assess S_pO_2, MIP, and dyspnea. Paired sample t-tests with Bonferonni adjustments were used to identify any significant within-group differences and independent sample t-tests were used to identify differences between groups. Baseline MEP was assessed using an independent samples t-test. An alpha level of 0.05 was set as a priori for statistical significance. Data are expressed as mean (SD).

TABLE I. BAROMETRIC PRESSURE, AIR TEMPERATURE, AND NUMBER OF CLIMBERS ASSESSED AT EACH ALTITUDE.

Altitude	P_B (mmHg)	Air Temperature (°C)	N
0–400 m	736 (26.9)	30 (0)	14
1400 m	/	26 (0)	14
4880 m	422 (0)	6 (5.7)	13
5550 m	381 (1.0)	2.5 (3.5)	10

Data are expressed as mean (SD). N = total number of subjects measured at the designated altitude.

RESULTS

Before IMT, mean MIP was 150 (25) cmH_2O for the control group and 147 (42) cmH_2O for the IMT group (P = 0.886). Following IMT, mean MIP increased by 15% to 169 (33) cmH_2O (P = 0.003), but was unchanged in the control group. Mean MEP values were similar between groups (P = 0.433), being 162 (26) cmH_2O and 177 (43) cmH_2O in the IMT and control groups, respectively.

S_pO_2 declined progressively with ascending altitude ($P < 0.01$), regardless of whether IMT was undertaken or not [baseline S_pO_2 was 99 (0.5) and 99 (1.0) for control and IMT groups, respectively]. S_pO_2 was 6% higher in the IMT group at altitudes of 4880 m (P = 0.001) and 5550 m (P = 0.050) (**Fig. 1**). Dyspnea was similar between groups (P = 0.462) despite increasing significantly as subjects ascended above 1400 m (dyspnea was identical at 0–400 m and 1400 m with a Borg Score of 0 for both altitudes per group). Specifically, dyspnea at 4880 m [0.6 (0.6) and 1.1 (1.0), IMT and control, respectively] and 5550 m [1.8 (1.1) and 2.0 (1.1), IMT and control, respectively] was significantly different to every other altitude ($P < 0.05$).

DISCUSSION

The main finding of the current study is that IMT, which increased inspiratory muscle strength by 15%, attenuated the fall in resting S_pO_2 at altitudes of 4880 m and 5550 m by 6% (Fig. 1), but had no impact on the Borg Score. As respiratory comfort was well maintained at each altitude (Borg Scores did not rise above 2.0 ± 1.0), the lack of difference between groups is not surprising and is consistent with previous reports that exhaustive exercise is required before hypoxia affects respiratory perception (4,12).

The role of IMT in modulating physiological variables during exercise has been well documented. Improvements in time trial performance, enhanced recovery rates, reduced levels of blood lactate, and respiratory

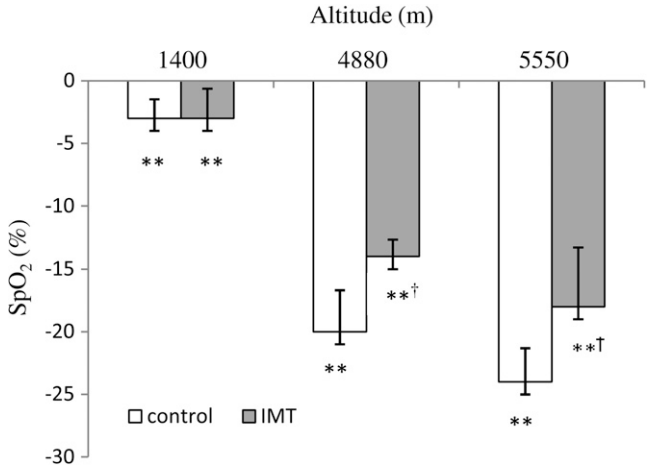

Fig. 1. Arterial oxygen saturation expressed as percentage change from baseline (0–400 m). Different at all altitudes within groups (**$P < 0.01$) and between groups at designated altitudes ($^{\dagger}P < 0.05$).

muscle fatigue as well as attenuated whole body and respiratory perceptions have all been reported [see McConnell and Romer (11) for a review of IMT]. With the exception of a study by Downey et al. (4), no studies have examined the impact of IMT on physiological variables under conditions of hypoxia. It is, therefore, not surprising that the mechanisms responsible for the IMT-mediated alterations in exercising S_pO_2 observed by Downey et al. (4) and at rest in the current study are yet to be fully resolved.

As Downey et al. (4) adopted a single acute bout of hypoxia and used an exercising model (although resting data was also reported), comparisons between their observations and those of the current study need to be made with caution. Nevertheless, the observed magnitude of resting arterial desaturation observed at altitudes of 4880 m and 5550 m in the current study (6%, see Fig. 1), was similar (5%) to that observed during submaximal hypoxic (F_IO_2 of 0.14) exercise to exhaustion by Downey et al. (4). Importantly, however, a protective effect was not observed at rest in the latter study or at altitudes of 1400 m and below in the current study (Fig. 1): this point will be returned to shortly. The reasons for Downey and colleagues (4) exercising observations are poorly understood, but are probably linked to alterations in pulmonary capillary red blood cell transit time, the ventilation-perfusion ratio, ventilation, and/or pulmonary diffusion capacity (3).

Specifically, Downey et al. (4) observed an increase in lung diffusing capacity of around 23% ($P < 0.05$) post exhaustive submaximal exercise (5 min) and a reduction in exercise minute ventilation of approximately 24% ($P < 0.05$) at a point when S_pO_2 was around 4% higher ($P < 0.05$) post- compared to pre-IMT. They suggested that a reduction in respiratory muscle work during exercise post-IMT led to a reduction in oxygen consumption and cardiac output. This in turn probably lengthened red blood cell transit time through the pulmonary capillary bed and hence S_pO_2 increased (4). The fact that IMT failed to impact resting S_pO_2 in their study but did in the present study may simply reflect differences in resting cardiopulmonary stress. Given that the fall in resting S_pO_2 was only attenuated at altitudes of 4880 and 5550 m in the current study, it is reasonable to assume that the resting cardiopulmonary stress would have been greater than that experienced by Downey and colleagues' (4) subjects, who were exposed to an F_IO_2 equivalent to an altitude of approximately 3050 m (10,007 ft).

As a positive relationship exists between ventilation and the oxygen cost of breathing (6), the role of ventilatory acclimatization and the associated work of breathing should not be overlooked. As ventilation increases at altitude, so too will the work of breathing, and in turn so too will the demand for respiratory muscle blood flow (6). Post-IMT, however, the work of breathing is likely to be lower and, therefore, so will be the respiratory muscles' demand for cardiac output (4). Although, an increase in minute ventilation is the typical response observed at altitude (8,9,13), an increase in exercising S_pO_2 without a concomitant increase in exercise minute ventilation has been reported and is consistent with the notion that either red blood cell transit time in the pulmonary capillary bed and/or lung diffusing capacity is enhanced (3). Thus, it is not inconceivable that IMT modified the natural hyperventilatory response to altitude, which may have, in turn, reduced respiratory muscle blood flow, although this is speculation. In addition, if as previously suggested IMT increases the concentration of inspiratory musculature monocarboxylate transporters, specifically MCT1 and MCT4 (10), the efflux of lactate and hydrogen ions from fibers in the respiratory musculature is likely to increase. Accordingly, one may surmise that blood flow to the respiratory musculature will be lower as the work of breathing is reduced post-IMT and so too will be the intramuscular acidity driven increase in blood flow; although it is hard to envisage how the latter would have a major effect on blood acidity at rest and in turn resting S_pO_2.

Although this study has presented novel data demonstrating that the magnitude of arterial desaturation during hypoxia can be ameliorated following IMT, it is limited by the lack of data relating to the physiological mechanisms underpinning such changes: for logistical purposes it was not possible to collect expired gas samples or undertake cardiac output measures during the sojourn. Rather, this study was a preliminary investigation to determine whether IMT might be useful in reducing the level of arterial desaturation in those subjected to altitude and the approximate altitude range required when at rest. It was not the aim to delineate underpinning mechanisms for any observed change. Nevertheless, there were several limitations to the present study, including the recruitment of a small heterogeneous sample, single measurements of S_pO_2 and an inability to standardize previous exercise habits of the subjects. In addition, the menstrual cycle stage of the two female climbers was not known. Although the ascent profiles were similar between subjects, it was not possible to standardize ambient temperature. Consequently, it is possible that changes in ambient air temperature (see Table I) may have affected body temperature and in turn the oxygen-dissociation curve and S_pO_2. However, there is no reason to assume that a reduction in body temperature would have occurred in the IMT group and not the control group. Similarly, the occurrence of acute mountain sickness, the severity of which is associated with arterial oxygen desaturation, was not measured in the current study. Clearly, such limitations should be addressed in future studies.

In conclusion, the aim of this preliminary investigation was to determine whether the level of arterial desaturation occurring during progressive altitude exposure could be attenuated in response to IMT. The finding that IMT exerted a protective effect on resting S_pO_2 at altitudes of 4880 m and 5550 m is novel. Furthermore, in light of Downey and colleagues (4) resting observations, it is tempting to speculate that an altitude of between approximately 3050 m and 4880 m is needed before the beneficial effect of IMT on resting S_pO_2 becomes apparent. The physiological mechanisms underpinning the

IMT-mediated alterations in $S_{p}O_2$ are not well understood and require investigation.

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