Benefit of Acclimatization to Moderate Altitude on Arterial Oxygen Saturation Following Rapid Ascent to 4300 M

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Introduction

During long-term exposures (days-to-weeks) to high altitudes, humans compensate for the decreased inspired oxygen partial pressure (PIO₂) by progressively increasing ventilation (for a review see reference (4,14). For example, following rapid ascent to 4,300 m elevation, ventilation increases during the first 6-8 days (7). The rise in ventilation produces a decrease in arterial carbon dioxide partial pressure (PaCO₂) and a concomitant increase in PaO₂ (12).

The time course and magnitude for acquiring altitude acclimatization has been well described for unacclimatized lowlanders rapidly ascending to high altitudes. However, the magnitude of altitude acclimatization developed in lowlanders residing at moderate elevations (1,000 - 2,000 m) has not been well documented. Moreover, there is no comprehensive database that describes the degree to which acclimatization to moderate altitudes improves arterial oxygenation upon rapid ascent to higher altitudes. We propose that lowlanders acclimatized to moderate altitudes will maintain a higher level of arterial oxygenation when rapidly ascending to higher altitudes compared to lowlanders residing at low altitudes.

Numerous military installations housing large numbers of military personnel are located at moderate altitudes. Development of a database that describes the distribution of arterial oxygen saturation in lowlanders acclimatized to a range of moderate altitudes would provide commanders with ascent timetables to higher elevations that take full advantage of the personnel’s acclimatization status. Furthermore, current limits on the time that aircrews of unpressurized aircraft may fly above 3,048 m without supplemental oxygen are based on studies of unacclimatized lowlanders. Altitude-acclimatized aircrews may be able to safely operate beyond these limits, thus enhancing operational capability.

The purpose of this study was to determine the distribution of arterial oxygen saturation following rapid ascent to high altitude (4,300 m) in military personnel residing at moderate (~2,000 m) altitude. These data were compared to similar measurements previously collected on men and women residing near sea level.

Materials and Methods

Studies were conducted on 38 military personnel (25 men, 13 women) assigned to the U.S. Air Force Academy (AFA group). All subjects had resided in the Colorado Springs, CO metropolitan area for at least 3 months prior to the study. All the subjects had passed their most recent military physical performance test, and were in good health. Women volunteers tested negative for pregnancy. The subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to U.S. Army Regulation 70-25 on the use of volunteers as subjects of research.
On the day of testing, subjects reported to an indoor test site located at the USAFA (terrestrial elevation 2,255 m, pressure altitude 1,860 m). At that site, several test procedures were performed: administration of an Environmental Background Survey and the Environmental Symptoms Questionnaire, and measurement of resting ventilatory parameters and forced vital capacity (FVC). After completion of these procedures, subjects entered a vehicle (van) and were transported to the U.S. Army Pikes Peak Research Facility on the summit (4,300 m terrestrial elevation) of Pikes Peak, CO via the Pikes Peak Highway. The vehicle stopped at the following pressure altitudes according to the prevailing barometric pressure: 2,438 m, 3,048 m, 3,658 m and on the summit 4,043 m. At each stop, the subjects remained in the vehicle at rest for approximately 5 min after which their resting \( \text{SaO}_2 \) and heart rate were recorded. Upon arriving at the summit, the same 5 min measurements were made after which the subjects entered the U.S. Army Pikes Peak Research Facility. The subject's resting ventilatory parameters were immediately measured. After about 1 h on the summit, the subjects were administered the Environmental Symptoms Questionnaire. The subjects then returned to the USAFA and were released from the study. The ascent profile is illustrated in Figure 1. Between 2,438 and 4,043 m, the average ascent rate was 66 m/min.

For each subject, all testing was completed on one day. The following test procedures were performed. At the USAFA lab, each subject’s height and weight were measured. Then each subject completed the Environmental Background Survey (EBS). The EBS is a 57-item questionnaire designed to elicit information on test volunteer’s previous experience in stressful climatic conditions in addition to epidemiologic, and medical history data. The presence of hypoxic-induced symptoms (dizziness, shortness of breath, alertness, etc) and the incidence of AMS were determined from information gathered using the Environmental Symptoms Questionnaire (ESQ). The ESQ is a self-reported, 68-question inventory used to document symptoms induced by altitude and other stressful environments (9). A weighted average of scores from cerebral symptoms (headache, lightheaded, dizzy, etc.) designated AMS-C and from respiratory symptoms (short-of-breath, hurts-to-breathe, etc.) designated AMS-R were calculated. AMS-C scores greater than 0.7 and AMS-R scores greater than 0.6 are defined as indicating the presence of AMS (9). Also, an alertness factor was calculated from the questionnaire (9). The ESQ was administered at the USAFA lab prior to starting the ascent and after about 1 hour on the summit.

Figure 1. Ascent profile from USAFA laboratory to U.S. Army Pikes Peak Research Facility. Elapsed time includes a 5 minute stop at each of the 3 intermediate altitudes.
Following completion of the questionnaires, each subject’s resting minute ventilation (\( \dot{V}E \)), and end-tidal oxygen and carbon dioxide partial pressure (PETCO\(_2\) and PETO\(_2\)) were measured using an open-circuit metabolic measurement system (SensorMedics Vmax229). Simultaneously, blood oxygen saturation (SaO\(_2\)) and heart rate (HR) were measured by pulse oximetry (Nellcor N-200), and blood pressure by auscultation. The subjects were studied after having fasted for at least 2 hr and having been seated at rest for 10 min. Resting ventilation was measured once at the USAFA and once upon arrival on the summit of Pikes Peak. The same measurement system was used to measure the subject’s FVC and forced expired volume-1 s (FEV\(_1\)) after completing the resting ventilatory measurements at the USAFA.

Resting SaO\(_2\) and heart rate were measured in the vehicle during each 5 min stop along the Pikes Peak Highway at pressure altitudes of 2,438 m, 3,048 m, 3,658 m and 4,043 m. The same finger pulse oximeter described in the resting ventilation studies was used.

**Results**

All data are reported as the group mean (\( \bar{X} \)) ± standard deviation (S.D.). The 38 subjects’ age, height, weight, FVC and FEV\(_1\) were: 35 ± 8 y, 176 ± 8 cm, 74.9 ± 13.8 kg, 5.1 ± 0.8 l and 4.2 ± 0.7 l. Based on analysis of the EBS, all subjects regularly participated in aerobic physical conditioning and nearly half in strength conditioning.

The resting ventilatory parameters are illustrated in Table 1. At the USAFA test site (1,860 m), PETO\(_2\), PETCO\(_2\) and SaO\(_2\) were significantly (\( p<0.05 \)) lower than normal values reported for lowlanders residing at sea level (PETO\(_2\) 104 mmHg, PETCO\(_2\) 40 mmHg and SaO\(_2\) >96% (5). During the approximately 2 h ascent to 4,043 m, resting SaO\(_2\) progressively decreased (Figure 2). The decrease in SaO\(_2\) with increasing altitude was significant (\( p<0.001 \)) at and above the 3,048 m elevation. There was no significant change in resting HR (Figure 2) during the ascent to 4,043 m.

**Table 1: Resting ventilatory parameters at residence altitude (USAFA) and during rapid ascent to 4,043 m.**

<table>
<thead>
<tr>
<th>Pressure Altitude (m)</th>
<th>( \dot{V}O_2 ) (l/min)</th>
<th>( \dot{V}E ) (BTPS) (l/min)</th>
<th>PETO(_2) (mmHg)</th>
<th>PETCO(_2) (mmHg)</th>
<th>SaO(_2) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,860</td>
<td>0.296 ± 0.051</td>
<td>10.7 ± 2.3</td>
<td>75.4 ± 4.9</td>
<td>33.6 ± 2.8</td>
<td>94 ± 1</td>
</tr>
<tr>
<td>4,043</td>
<td>0.284 ± 0.053</td>
<td>10.5 ± 2.6</td>
<td>51.5 ± 5.7</td>
<td>32.1 ± 4.5</td>
<td>86 ± 2</td>
</tr>
</tbody>
</table>

\( \bar{X} \) ± S.D.

After arrival at 4,043 m, resting PETO\(_2\), PETCO\(_2\) and SaO\(_2\) were significantly (\( p<0.001 \)) lower compared to the measurements made at 1,860 m a few hours earlier (Table 1). The subjects’ PETCO\(_2\) at their residence altitude (1,860 m) correlated significantly (\( r = 0.70, p<0.001 \)) with their PETCO\(_2\) (Figure 3) and to a lesser degree with their SaO\(_2\) (\( r = 0.36, p<0.05 \)) measured at 4,043 m.
None of the subjects reported ESQ symptom scores indicative of developing Acute Mountain Sickness. There were also no significant changes in the scores for alertness or fatigue following 1 h at 4,043 m.

Figure 3. Relationship between residence altitude resting PETCO₂ and resting PETCO₂ following rapid ascent to 4,043 m pressure altitude.
Discussion

This study demonstrated that military personnel residing at moderate altitude (~2,000 m) for greater than 90 days are mildly hypoxic at their residence altitude, and that the interindividual differences in the ventilatory response at higher altitude is related to differences among individuals at their residence altitude.

Previous studies have reported a large variation among individuals in the degree of ventilatory acclimatization at high altitude (10,11). Reeves et al., (8) reported that the variability in the degree of ventilatory acclimatization at high altitude, was related to the individual's sea level end-tidal PETCO₂. That is, the lower the individual's PETCO₂ at sea level, the greater their ventilation at high altitude. The current study extends this relationship to subjects residing a moderate altitude. It is clear that there is considerable inter-individual variability in the acute response to hypoxia, the rate of acclimatization to altitude and the vulnerability to mountain sickness. The latter finding may be potentially useful in predicting an individual's ventilatory response and subsequent well-being to a future high altitude exposure. Our study did not examine the relationship between arterial oxygen content at high altitude and susceptibility to high altitude illness, or physical and cognitive performance decrements. However, it is reasonable to expect that individuals maintaining higher arterial oxygen content will be less affected by the high altitude environment.

Another goal of this study was to determine the extent to which lowlanders acclimatized to moderate altitudes maintain their arterial oxygenation when rapidly ascending to higher altitudes compared to lowlanders residing a low altitudes. Numerous studies have reported the ventilatory response on arrival and during residence on the summit of Pikes Peak (2,3,6,7,13). The results of two of these studies (6,7), and our current study are illustrated in Figure 4. When sea level residents rapidly ascend to a pressure altitude of 4,043 m, on arrival their PETCO₂ is 34.9 ± 2.8 mmHg and their SaO₂ is 81 ± 5%. It takes 9-12 days of continuous residence at high altitude for the SaO₂ to rise to 88 ± 2%. By comparison, upon rapid ascent to 4,043 m, the AFA subjects resting SaO₂ was 86 ± 2%. These data suggest that personnel residing at ~2,000 m elevation for more than 90 days have acquired a level of ventilatory acclimatization equivalent to residing at 4,043 m for a week or more.

Figure 4. The USAFA personnel resting SaO₂ upon arrival to 4,043 m pressure altitude is illustrated by the two dotted-lines representing the mean ± 1 standard deviation. Also shown for comparison is the sea level residents resting SaO₂ upon arrival and the effect of ventilatory acclimatization on SaO₂.
Given the degree of ventilatory acclimatization achieved by personnel residing at the moderate altitude studied, we would expect such personnel to be less susceptible to Acute Mountain Sickness and decrements in cognitive and physical performance during rapid ascent to higher altitudes. Lowlanders who have achieved the level of acclimatization seen in our AFA group usually are not impaired by AMS symptoms, have complete restoration of cognitive performance and substantial improvements in physical work performance (1,14). Thus, we conclude that military personnel residing at moderate altitudes for a period of at least 90 days can be rapidly deployed to higher altitudes of up to 4,043 m with a low probability of developing AMS and experiencing significant performance decrements.

References


