ABSTRACT

THE EFFECTS OF CARDIORESPIRATORY FITNESS ON SYMPTOMS OF ACUTE MOUNTAIN SICKNESS

by Mathias Hieronymus

This paper addresses the effects of cardiorespiratory fitness on symptoms of acute mountain sickness (AMS), which is a pathological condition that is caused by acute exposure to high altitudes. After determining maximal oxygen consumption, body composition, and hematocrit at sea level, 14 subjects went to Nepal and ascended over the course of 14 days to Mount Everest Base Camp at 5380m. During these 14 days of ascent, participants were asked to fill out a Lake Louise AMS questionnaire every morning. Additionally, the participant’s blood oxygenation levels and heart rates were determined every morning using a pulse oximeter. The results indicate that the participants’ body mass was positively related to AMS while their relative VO$_2$max scores (in relation to lean mass) and their hematocrit levels were negatively related to AMS. Thus, the higher the individuals’ body mass and the lower their aerobic capacity and hematocrit levels, the higher their AMS scores.
THE EFFECTS OF CARDIORESPIRATORY FITNESS ON SYMPTOMS OF ACUTE MOUNTAIN SICKNESS

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Introduction

It is estimated that worldwide 100 million people each year spend some time in the mountains pursuing a wide variety of different sports (Burtscher, 1999). Acute Mountain Sickness (AMS) is a pathological condition that is caused by acute exposure to high altitudes. Symptoms include headache, fatigue, stomach illness, dizziness, and sleep disturbance. Symptoms typically occur six to 12 hours after ascent to a new altitude to more than 2500m (may occur sooner) and usually resolve over one to three days, providing that no further ascent is made (Barry & Pollard, 2003). Whether AMS occurs is determined by the rate of ascent, the altitude reached, the altitude at which an affected person sleeps and individual physiology. Although AMS is more prevalent at high altitude (>5000m/16400ft), it is not uncommon that people experience mild AMS at moderate to low altitude. Honigman et al. (1993) found that 25% of visitors to moderate altitude (1500–3500m/5000-11500ft) show symptoms of AMS whereas individuals who are younger, less physically fit, live at sea level or have a history of AMS more often develop these symptoms. Roach, Houston, Honigman, Yaron, Alexander, & Hultgren (1995) reported that the incidence of AMS was 16% in a group of 97 older women and men (aged 59 to 83 years) at an elevation of 2500m (8200ft). Wright (2006) reported that individual susceptibility to AMS varies considerably with a prevalence rate from 10-50% depending on the altitude achieved and the rate of ascent. In Wright’s opinion AMS occurs with moderate symptoms in about a third of a group, mild symptoms in another third and none in the remainder. It occurs more commonly in young and obese subjects but is unrelated to physical fitness and the risk for AMS is the same for both sexes.

The barometric pressure at sea-level is 760mmHg, which is decreasing with increasing altitude. For example, at Pikes Peak with an altitude of 4300m (14110ft) the barometric pressure is reduced to 440mmHg. Upon exposure to altitude there is a decrease in the partial pressure of inspired oxygen. In turn, this decrease leads to a reduction in the partial pressure of oxygen at the alveolar level of the lungs, where oxygen diffuses through the pulmonary capillaries to the blood. This results in fewer oxygen molecules binding to hemoglobin, that is, a decrease in arterial oxygen saturation (SAO$_2$). Burtscher, Szubski, & Faulhaber (2007) stated that when mountaineers ascend rapidly to 2500m, about 10% of them will suffer AMS, and when ascending to 4500m, the AMS incidence will exceed 60%. In their review they concluded that
values of SAO₂, determined 20-30min after exposure to simulated hypoxia equivalent to 2300-4200m, seem to be the most useful predictors of AMS susceptibility (>80% correct prediction). Muza, Pock, Zupan, Miller, Thomas, & Cymerman (2004) reported that the higher SAO₂ values above 2438m of residents at moderate altitude compared to residents of low altitude were related to the lower incidence of AMS.

Other studies have been conducted to determine susceptibility to AMS derived from measurements taken at sea-level. Savourey, Moirant, Eterradossi, & Bittel (1995) measured maximal oxygen consumption (VO₂max) and body composition before an expedition to the Andes (4500m) and tried to clarify the relationship between these parameters and AMS. They found no significant relationships between VO₂max, body composition and the occurrence of AMS. Concerning the relationship between VO₂max measured at sea level and severity of AMS Milledge, Beeley, Broome, Luff, Pelling, & Smith (1991) found, as well as Savourey et al. (1995), no correlation.

In order to assess symptoms of AMS a standardized scoring system, the Lake Louise AMS scoring system was developed in 1991. This system has since been used by many investigators and has been recommended to be adopted as the standard for AMS research. In this scoring system a constellation of symptoms (headache, nausea, dizziness, fatigue, and sleeplessness) is called AMS only when the victim has been exposed to altitude for more than two hours. AMS is defined as the presence of headache and at least one of the following symptoms: Gastrointestinal (anorexia, nausea or vomiting); fatigue or weakness; dizziness or lightheadedness; difficulty sleeping (Roach, Bärtsch, Oelz, & Hackett, 1993). Maggiorini, Muller, Hofstetter, Bartsch, & Oelz (1998) evaluated the Lake Louise AMS score questionnaire at different altitudes and compared it with the clinical score and the environmental symptoms questionnaire AMS-C score. They found that the Lake Louise consensus score is adequate and more effective for the assessment of acute altitude illness at different altitudes compared with the AMS-C score.

Most studies so far have been conducted at moderate altitude and it seems that there is a lack of literature related to the occurrence of AMS in a normal population at very high altitude (3500-5500m/11500-18000ft). The purpose of this study is therefore to examine the effects of exposure to very high altitude on SAO₂ and how SAO₂ is related to AMS. Additionally this study seeks to examine the relationship between VO₂max, Hematocrit (Hct) and body
composition measured at sea level and to symptoms of AMS.

Methods

Participants

Subjects in this study were recruited from a study abroad summer workshop sponsored by a Midwest university. The workshop consisted of a four-week trip to the Himalayas in Nepal including Laboratory visits prior to and after the trip. The participants started in Kathmandu and ascended over the course of 14 days to Mount Everest Base Camp. The total study sample included eight males and six females, all of whom were participants in the workshop. Descriptive characteristics for this sample are presented in Table 1. Participation in the research component of the workshop was voluntary for all subjects. No extra credit or compensation was offered, and approval for the study was obtained from the university’s Institutional Review Board.

Study Design

The study design was descriptive in nature and involved no experimental manipulation. Baseline data (body composition, Hct-levels, VO$_2$max) were obtained prior to subjects’ participation in the workshop. During the 14-day trek up the Himalayas, daily measures of subjects’ heart rate, blood oxygen levels, and level of altitude sickness were obtained.

Apparatus

Hct was determined by collecting 3ml of blood from a forearm vein using standard blood collection techniques and analyzed using standard microcapillary techniques. Body composition was measured using air displacement plethysmography (BodPod). VO$_2$max was determined using a Balke treadmill protocol. Oxygen saturation and heart rate were assessed via pulse oximetry. A Lake Louise AMS Questionnaire was used to assess symptoms of Acute Mountain Sickness (AMS). There have been several scoring systems used to diagnose and quantify mountain sickness in altitude research, most consisting of questionnaires and some with an
additional examination by a physician. Some such as the Environmental Symptom Questionnaire (ESQ) are long at 67 questions and an attempt has been made to simplify and standardize a scoring system to enable easy comparisons of results between studies. The Lake Louise Score is such a system arising from consensus meetings in Lake Louise in Canada in 1991 and 1993. Although primarily developed for research use, it is short, simple format, which is easy to complete in difficult situations, has led to its adoption by general trekkers and mountaineers. It is sensitive enough to detect AMS whilst having sufficient specificity not to lead to undue over diagnosis. It consists of a self report questionnaire including questions regarding headache, gastrointestinal symptoms, fatigue, dizziness, and sleeping difficulties. A diagnosis of AMS is based on a recent rise in altitude, the presence of a headache with the presence of at least one other symptom, and a total score of at least three. The Self Report score stands alone and is recommended for general mountain travelers. The Clinical Assessment score can be added to the Self Report score, in which case, in the context of a recent rise in altitude, a score of five or more would be taken as AMS. The Clinical Assessment questionnaire consists of additional questions regarding mental status, ataxia, and peripheral edema (Roach et al., 1993).

**Procedures**

Prior to their participation in the workshop, participants visited the Human Performance Lab in the Department of Kinesiology and Health. After arriving at the Lab Hct-values were obtained. To determine Hct-values a person trained in blood collection (phlebotomist) collected approximately 3 ml of blood from a forearm vein using standard blood collection techniques. Each blood sample was analyzed in triplicate using standard microcapillary techniques. Body fat percentage and the amount of lean body mass were determined using air displacement plethysmography (BOD POD). This procedure involves the assessment of body density by having the subject sit inside the BOD POD chamber while the measurement of the amount of air displaced occurs and body density is calculated. The assessment of body density will allow for the calculation of body composition (amount of lean mass and fat mass). After determining body composition and Hct participants performed a Balke walking treadmill protocol to assess VO₂max. For men the treadmill speed was set at 3.3mph, with the gradient
starting at 0%. After 1 minute it was raised to 2%, then 1% each minute thereafter. For women the treadmill speed was set at 3.0mph, with the gradient starting at 0%, and increased by 2.5% every three minutes.

Study participants, then, traveled to Nepal and spent the next 14 days ascending to Mount Everest Base Camp at 5380m (17600 ft). During these 14 days of ascent, participants were asked to fill out a Lake Louise AMS questionnaire every morning. Additionally, the participant’s blood oxygenation levels and heart rates were determined every morning using a pulse oximeter. The pulse oximeter is slid over the subject’s index finger and measures the level of oxygenation of the subject’s blood. A source of light originates from the probe at two wavelengths (650nm and 805nm). The light is partly absorbed by Hemoglobin (Hb), by amounts which differ depending on whether it is saturated or desaturated with oxygen. By calculating the absorption at the two wavelengths the processor can compute the proportion of Hb which is oxygenated.

**Statistical analysis**

All data were coded, collated, and entered into an SPSS data file. Statistical procedures were conducted using SPSS (Version 16.0). Data analyses began with calculation of descriptive statistics. Then, to determine whether study participants experienced significant changes in blood oxygenation levels, heart rate, and altitude sickness as a result of exposure to higher altitudes, a series of repeated measures analyses of variance (ANOVAs) were used. To measure relationships between blood oxygenation levels, heart rate, and altitude sickness levels, univariate correlational and multiple regression procedures were used. Similarly, to determine if the variables collected at baseline (body composition, VO$_2$max, Hct) could predict participants’ level of altitude sickness, a multiple regression analysis was used. Significance was assigned to values of p<.05.
Results

The first purpose of this study was to examine the effects of exposure to very high altitude on SAO₂ and to determine if SAO₂ and heart rate are related to AMS at these high altitudes. A second study purpose was to determine whether VO₂max, Hct and body composition as measured at sea level would be related to, or predictive of, AMS levels as experienced at high altitudes. The first hypothesis is that SAO₂ is negatively correlated to the occurrence of AMS. The second hypothesis is that VO₂max and Hct measured at sea level are negatively correlated to symptoms of AMS at altitude while body composition measured at sea level is positively correlated to symptoms of AMS at altitude. To test these hypotheses, a variety of statistical procedures were used. The results are presented in the following sections, beginning with descriptive statistics and continue to the main study analyses.

Descriptive statistics

Descriptive statistics for all baseline variables (assessed at sea level prior to the 14-day trek), including average Hct, body composition, height, mass, and rel. VO₂max (in relation to lean mass), are presented in Table 1.

<table>
<thead>
<tr>
<th>Age (m)</th>
<th>Height (kg)</th>
<th>Mass (kg)</th>
<th>% BodyFat</th>
<th>Lean mass (kg)</th>
<th>rel. VO₂max (ml/kg lean mass/min)</th>
<th>Hematocrit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average</td>
<td>24.75</td>
<td>1.82</td>
<td>85.00</td>
<td>15.80</td>
<td>70.79</td>
<td>57.1</td>
</tr>
<tr>
<td>S</td>
<td>6.92</td>
<td>0.06</td>
<td>13.70</td>
<td>7.61</td>
<td>6.58</td>
<td>7.78</td>
</tr>
<tr>
<td>Average</td>
<td>22.33</td>
<td>1.66</td>
<td>65.84</td>
<td>29.00</td>
<td>46.63</td>
<td>53.98</td>
</tr>
<tr>
<td>S</td>
<td>1.21</td>
<td>0.05</td>
<td>6.05</td>
<td>4.71</td>
<td>3.86</td>
<td>6.67</td>
</tr>
</tbody>
</table>

Table 1: Age, height (m), mass (kg), %bodyfat, lean mass (kg), relative VO₂max (ml/kg lean mass/min), and hematocrit for all baseline variables (n=14)

Descriptive statistics for all data collected over the course of 14 days at differing levels of altitude are presented in Table 2. These data includes daily measures of blood oxygenation (SAO₂), heart rate (HR), and altitude sickness (AMS). Graphs corresponding to this data are provided in Figures 1 and 2, respectively.
<table>
<thead>
<tr>
<th>Day</th>
<th>Altitude (m)</th>
<th>SAO₂</th>
<th>HR</th>
<th>AMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2700</td>
<td>93.79±2.04</td>
<td>73.86±12.96</td>
<td>0.14±0.53</td>
</tr>
<tr>
<td>2</td>
<td>3440</td>
<td>90.21±1.81</td>
<td>81.07±15.03</td>
<td>0.71±0.73</td>
</tr>
<tr>
<td>3</td>
<td>3440</td>
<td>91.21±1.63</td>
<td>77.50±14.25</td>
<td>0.21±0.43</td>
</tr>
<tr>
<td>4</td>
<td>3750</td>
<td>89.79±1.89</td>
<td>80.21±14.71</td>
<td>0.29±0.47</td>
</tr>
<tr>
<td>5</td>
<td>3780</td>
<td>89.71±1.77</td>
<td>80.00±14.76</td>
<td>0.86±1.41</td>
</tr>
<tr>
<td>6</td>
<td>4040</td>
<td>88.64±2.24</td>
<td>81.86±9.37</td>
<td>0.86±0.95</td>
</tr>
<tr>
<td>7</td>
<td>4480</td>
<td>87.29±1.90</td>
<td>80.79±12.17</td>
<td>0.79±0.97</td>
</tr>
<tr>
<td>8</td>
<td>4750</td>
<td>88.00±2.65</td>
<td>83.08±10.06</td>
<td>1.54±1.90</td>
</tr>
<tr>
<td>9</td>
<td>3850</td>
<td>91.31±2.25</td>
<td>76.23±13.04</td>
<td>0.69±1.32</td>
</tr>
<tr>
<td>10</td>
<td>4630</td>
<td>87.54±2.90</td>
<td>76.46±10.46</td>
<td>1.54±3.15</td>
</tr>
<tr>
<td>11</td>
<td>4930</td>
<td>85.67±3.26</td>
<td>77.17±8.65</td>
<td>2.08±2.75</td>
</tr>
<tr>
<td>12</td>
<td>5288</td>
<td>85.25±2.96</td>
<td>76.67±11.07</td>
<td>2.58±2.91</td>
</tr>
<tr>
<td>13</td>
<td>5288</td>
<td>85.58±2.02</td>
<td>76.17±8.07</td>
<td>1.58±2.31</td>
</tr>
<tr>
<td>14</td>
<td>3958</td>
<td>90.38±2.50</td>
<td>74.58±10.72</td>
<td>0.38±0.77</td>
</tr>
</tbody>
</table>

Table 2: Altitude, arterial oxygen saturation (SAO₂), heart rate (HR), and acute mountain sickness scores (AMS) as determined via the Lake Louise Mountain Sickness questionnaire over the course of 14 days (n=14)

Figure 1: Elevation profile (m) and acute mountain sickness scores (AMS) as determined via the Lake Louise Mountain Sickness questionnaire over the course of 14 days (n=14)
Assessment of Physiological Changes Across Altitude Levels

To determine if participants’ blood oxygenation, AMS, and heart rate levels changed significantly as a response to increased elevation or altitude levels, a series of three repeated measures ANOVAs were conducted. The dependent variables for these three analyses were SAO₂, heart rate, and AMS. From the 14 days of data, a set of eight days representing eight different levels of altitude were selected for the repeated measures ANOVAs (Table 3).

<table>
<thead>
<tr>
<th>Day</th>
<th>Altitude</th>
<th>SAO₂</th>
<th>HR</th>
<th>AMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2700</td>
<td>93.79 ± 2.04</td>
<td>73.86 ± 12.96</td>
<td>0.14 ± 0.53</td>
</tr>
<tr>
<td>4</td>
<td>3750</td>
<td>89.79 ± 1.89</td>
<td>80.21 ± 14.71</td>
<td>0.29 ± 0.47</td>
</tr>
<tr>
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<td>0.86 ± 0.95</td>
</tr>
<tr>
<td>7</td>
<td>4480</td>
<td>87.29 ± 1.90</td>
<td>80.79 ± 12.17</td>
<td>0.79 ± 0.97</td>
</tr>
<tr>
<td>8</td>
<td>4750</td>
<td>88.00 ± 2.65</td>
<td>83.08 ± 10.06</td>
<td>1.54 ± 1.90</td>
</tr>
<tr>
<td>11</td>
<td>4930</td>
<td>85.67 ± 3.26</td>
<td>77.17 ± 8.65</td>
<td>2.08 ± 2.75</td>
</tr>
<tr>
<td>12</td>
<td>5288</td>
<td>85.25 ± 2.96</td>
<td>76.67 ± 11.07</td>
<td>2.58 ± 2.91</td>
</tr>
<tr>
<td>14</td>
<td>3958</td>
<td>90.38 ± 2.50</td>
<td>74.58 ± 10.72</td>
<td>0.38 ± 0.77</td>
</tr>
</tbody>
</table>

Table 3: Altitude (m), oxygen saturation (SAO₂), heart rate (HR), and acute mountain sickness scores (AMS) as determined via the Lake Louise Mountain Sickness questionnaire for eight selected days (n=14)
The first repeated measures ANOVA for SAO2 revealed a significant main effect for elevation, $F(7, 84)=18.83$, $p<.00$, $\eta^2=.61$. This overall significant effect indicated that participants’ SAO2 levels did change significantly across the eight days at different altitudes. To identify specifically where significant differences occurred, the within-subjects contrasts were examined. These results revealed that participants’ SAO2 scores decreased linearly significantly from Day 1 to Day 6, then stabilized from Day 6 to Day 8. SAO2 scores then again decreased significantly from Day 8 to Day 12 followed by a significant increase from Day 12 to Day 14.

The second repeated measures ANOVA to test for change in participants’ HR showed a significant main effect for elevation, $F(7, 77)=2.182$, $p<.045$, $\eta^2=.17$. Examination of within-subjects contrasts revealed that participants’ HR increased significantly from Day 1 to Day 8, and then decreased significantly from Day 8 to Day 14.

The repeated measures ANOVA for AMS revealed a significant Mauchly’s effect ($w=.00$, chisquare=70.13, $p<.000$) suggesting that the standard deviations for the AMS scores across the eight days were not equivalent. Given this lack of sphericity, the Greenhouse-Geisser correction factor was used. Examination of these results revealed a significant main effect for elevation, $F(2.74, 35.62)=4.45$, $p<.01$, $\eta^2=.26$. Thus, participants’ AMS scores did vary significantly across the eight different levels of elevation. Within-subjects contrast revealed that subjects’ scores were stable from Day 1 to Day 4 but then increased linearly from Day 4 to Day 12. Participants’ AMS scores then decreased significantly from Day 12 to Day 14.

Assessment of Relationships Between AMS, Heart Rate, and SAO2

To determine if participants’ AMS scores at different levels of elevation were correlated with their heart rate and SAO2 scores, a series of Pearson Product-Moment correlational analyses between AMS, HR, and SAO2 at each of the 14 days were conducted. These results showed that HR was correlated with AMS at Day 6 ($r=.63$, $p<.01$) and at Day 8 ($r=.72$, $p<.00$). No other significant correlations were found. In addition, a multiple regression analysis was used to determine if participants’ heart rate and SAO2 scores on Day 12 (highest level of elevation) would be able to predict their AMS scores on that day. The results of this analysis revealed no significant predictive relationship between HR and SAO2 and AMS at Day 12.
Assessment of Relationships Between AMS and Baseline Variables

A multiple regression analysis was conducted to determine if a set of variables measured at baseline (or at sea level) could be used to predict participants’ altitude sickness levels at high elevation. The dependent variable for this analysis was AMS at Day 12 (highest elevation), and the independent or predictor variables were body mass (in kgs), aerobic capacity (relative VO$_2$max in relation to lean mass), and hematocrit level. The results indicated a significant predictive relationship, $R=0.86$, $R^2=0.73$, adjusted $R^2=0.632$, $F(3, 11)=7.30$, $p<.01$. Thus, it appears that the baseline variables as a group could explain a significant amount of the variation between participants in their altitude sickness scores at the highest level of elevation. Examination of the regression beta weights presented in Table 4 indicate the participants’ body mass was positively related to AMS while their relative VO$_2$max scores (in relation to lean mass) and their hematocrit levels were negatively related to AMS. Thus, the higher the individuals’ body mass and the lower their aerobic capacity and hematocrit levels, the higher their AMS score at the highest level of elevation.

<table>
<thead>
<tr>
<th>Predictor Variable</th>
<th>Beta</th>
<th>t-value</th>
<th>sig. level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass (kg)</td>
<td>1.36</td>
<td>4.61</td>
<td>.00</td>
</tr>
<tr>
<td>rel. VO2max (ml/kg lean mass/min)</td>
<td>-.55</td>
<td>-2.29</td>
<td>.05</td>
</tr>
<tr>
<td>hematocrit</td>
<td>-.69</td>
<td>-2.82</td>
<td>.02</td>
</tr>
</tbody>
</table>

Table 4: Body mass (kg), relative VO$_2$max (ml/kg lean mass/min), and hematocrit with corresponding beta weights, t-values, and significance levels at Day 12; Dependent variable is the acute mountain sickness score as determined via the Lake Louise Mountain Sickness questionnaire (n=14)

Discussion

The first hypothesis of this study was that SAO$_2$ is negatively correlated to the occurrence of AMS. This hypothesis was not supported. Although repeated measures ANOVA for SAO$_2$ showed a significant main effect for elevation, multiple regression revealed no significant predictive relationship between heart rate and SAO$_2$ and AMS at Day 12. The alveolar oxygen pressure level is reduced due to the reduced atmospheric pressure at high altitude. Thus, the SAO$_2$ level is lowered. This resulting lack of oxygen is regarded as a crucial starting mechanism in the development of AMS in healthy persons. That is, although low SAO$_2$ levels might initiate the development of AMS, our study failed to show a relationship between low SAO$_2$ levels and
the occurrence of AMS at very high altitude. Known sources of error in pulse oximetry are: poor pulse signal on cold extremities; fluctuation of saturation at higher altitudes due to Cheyne-Stokes breathing pattern; and a tendency to underestimate SAO\(_2\) under conditions of severe hypoxia (Tannheimer, Thomas, & Gerngroß, 2002). The above mentioned Cheyne-Stokes breathing pattern might be important in the etiology of AMS because this breathing pattern can be seen in otherwise healthy people during sleep. Burgess, Johnson, Edwards, & Cooper (2004) suggested that there is an important association between desaturation during sleep and AMS. AMS symptoms seem to have a greater correlation (R-squared=.66) to oxygen saturation in females in comparison to males (R-squared=.51).

Heart rate was correlated with AMS only on two days (Day 6 and 8). No other correlation was found between heart rate and AMS over the course of the 14-day trek. Repeated measures ANOVA also revealed no significant main effect for elevation regarding heart rate. However, females showed a greater heart rate response (p<.05) in six out of thirteen days. The reason for this increased heart rate response and the greater correlation between SAO\(_2\) and AMS as observed in females remains unclear but we suggest that underlying physiological mechanisms (e.g. hormonal differences) modulating the heart rate in females as compared to males might give an explanation.

The second hypothesis was that VO\(_2\)\(_{\text{max}}\) and Hct measured at sea level are negatively correlated to AMS while body composition is positively correlated to the occurrence of AMS. Multiple regression revealed that these variables as a group could explain a significant amount of the variation between the participants’ AMS scores. However, body mass and rel. VO\(_2\)\(_{\text{max}}\) seem to explain most of the variability between the participants’ AMS scores. Savourey et al. (1995) found no relationship between VO\(_2\)\(_{\text{max}}\), body fat content, BMI and AMS. Savourey and colleagues used skinfold thickness measurements to determine body fat content. In our study we used air displacement plethysmography. This might explain the differences in the findings of older studies and our study. Richalet et al. (1988) as well as Bircher et al. (1999) reported that VO\(_2\)\(_{\text{max}}\) does not seem to be related to AMS. However, both studies do not rule out the possibility that improvement of physical fitness may reduce the severity and incidence of AMS. This was reported by Gupta, Joseph, & Malhotra (1978) who showed that symptoms of AMS were less severe at 3500m in trained compared to untrained participants. On the other hand Kayser (1991) found a relationship between BMI and the occurrence of AMS. BMI does not take
into account body composition. In our study percent body fat can not be used to predict participants’ AMS scores at very high altitude (p<.08). However, body mass can be used to predict participants’ AMS scores at very high altitude (p<.00). This might explain the findings of Kayser (1991) although Kayser did not distinguish between lean mass and percent body fat in his participants. Lean mass, especially muscle mass needs oxygen to cover the metabolic demands. Therefore, it seems reasonable that lean mass, other than percent body fat is a strong predictor variable for the occurrence of AMS symptoms. The participants with the highest cardiorespiratory fitness in both genders developed the fewest AMS symptoms. However, participants with an average relative VO$_2$max showed mixed response with regard to AMS symptoms. Hematocrit measured at sea level can also be used to predict participants’ AMS scores at very high altitude (p<.02). The erythrocyte maturation process takes approximately five to seven days from the initial altitude-induced increase in serum EPO. A physiological threshold for increasing red blood cell mass and hemoglobin has been described to be at an altitude of 1600m (5250 ft) whereas there is great individual variability in the altitude-induced erythropoetic response (Chapman, Stray-Gundersen, & Levine, 1998). It seems reasonable that this individual variability may influence the development of AMS symptoms.

Limitations

The sample consisted mostly of college students with a mean age of 24 ± 5 years. As reported in the literature, age seems to have an influence regarding the development of AMS symptoms. Another limitation could be the small sample size with only 14 subjects (8 males & 6 females). We did not control for the menstrual cycle in the female group. This might explain some minor differences such as heart rate response.
References


