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Jonathan R. Knott
Utah State University

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ARTERIAL OXYGEN SATURATION AS A PREDICTOR OF ACUTE MOUNTAIN SICKNESS AND SUMMIT SUCCESS AMONG MOUNTAINEERS

by

Jonathan R. Knott

A thesis submitted in partial fulfillment
of the requirements for the degree

of

MASTER OF SCIENCE

in

Health and Human Movement

Approved:

Dale R. Wagner, PhD Major Professor	Edward M. Heath, PhD Committee Member
James Davis, MD Committee Member	Byron R. Burnham, EdD Dean of Graduate Studies

UTAH STATE UNIVERSITY
Logan, Utah

2010

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ABSTRACT

Arterial Oxygen Saturation as a Predictor of Acute Mountain Sickness and Summit Success among Mountaineers

by

Jonathan R. Knott, Master of Science

Utah State University, 2010

Major Professor: Dr. Dale R. Wagner
Department: Health, Physical Education and Recreation

The purpose of this study was to determine if arterial oxygen saturation (SaO_2), as measured by a finger pulse oximeter upon rapid arrival to 4260 m, could be predictive of acute mountain sickness (AMS) or summit success on a climb to 5640 m. In total 73 climbers volunteered to participate in the study. After excluding those taking drugs to counteract the effects of AMS and those with missing data, 48 participants (45 male, 3 female) remained. Climbers were transported from 2650 m to the Piedra Grande hut at 4260 m on Pico de Orizaba within 2 hr. After a median time of 10 ± 13 hr at the hut, they climbed toward the summit (5640 m) and returned with a median trip time of 13.3 ± 4.8 hr. The Lake Louise Self-assessment Questionnaire (LLSA) for AMS, heart rate, and SaO_2 from a finger pulse oximeter was collected upon arrival at the hut, repeated immediately before the climbers departed for their summit attempts, and immediately upon their return. The presence of AMS was defined as a LLSA score ≥ 3 with a

headache and at least one other symptom. Fifty-nine percent of the participants successfully reached the summit. Average SaO_2 for all participants at 4260 m prior to their departure for the summit was 84.2 ± 3.8%. Sixty percent of the participants met the criteria for AMS during their ascent. There was not a significant difference (p = .90) in SaO_2 between those who experienced AMS (SaO_2 = 84.3 ± 3.3%) and those who did not (SaO_2 = 84.2 ± 4.2%) during the ascent. Neither was there a significant difference (p = .18) in SaO_2 between those who reached the summit (84.8 ± 3.7%) and those who did not (83.3 ± 4.0%). Arterial oxygen saturation does not appear to be predictive of AMS or summit success.

(71 pages)

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Jonathan R. Knott

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DEFINITIONS OF TERMS

Acclimatization: Process of becoming physiologically adapted to hypoxic conditions.

Acute Mountain Sickness (AMS): Experiencing a headache along with several other symptoms such as dizziness, fatigue, gastrointestinal problems, and difficulty sleeping which result from the hypoxic conditions while at high altitude.

Arterial Oxygen Saturation (SaO_2): Percentage of oxygen bound to hemoglobin within the arterial blood.

Dalton's Law: The total pressure exerted by a gaseous mixture is equal to the sum of the partial pressures of each of the component gasses.

Finger Pulse Oximetry: Noninvasive method of measuring the percentage of arterial blood oxygen saturation by measuring the absorption of light-emitting diodes (LEDs) at different wavelengths.

Henry's Law: At constant temperature, the amount of a gas dissolved into a given volume is directly proportional to the partial pressure of the gas.

High Altitude: Generally considered to be 1500-3500 m; very high altitude is from 3500-5500 m; extreme altitude is > 5500 m.

Hypoxemia: Deficient oxygenation of the blood.

Hypoxic Ventilatory Response (HVR): Altered rate of ventilation induced by hypoxia.

Impaired Pulmonary Exchange (IPE): When a stimulus of internal or external origin elicits a reduction in gas exchange between the tissues of the pulmonary system.

Lake Louise Self-Assessment (LLSA): Questionnaire for determining the severity of AMS by quantifying the climber's self-reported perception of the severity of symptoms.

Visual Analogue Scale (VAS): Subjective continuous scale consisting of a solid line where one side represents "none" and the other represents "severe or maximal." Participants are asked to place a mark on the line that best represents their perceived level of discomfort.

CHAPTER 1

INTRODUCTION

Men and women have always been drawn to the power and mystery of the mountains. The greatest giant of them all, Mt. Everest, has stood as an inspiration to many as the ultimate accomplishment, though for many years it seemed insurmountable. It was not until 1953 that Edmund Hillary and Tenzing Norgay were able to take the final steps to reach the highest point on the planet. People are traveling to new and adventurous places at an ever-increasing rate. To satisfy this drive for adventure, the limits of the human body are constantly being pushed. Heights previously thought to be unconquerable are being conquered. These new ventures into the unknown are always greeted with unexpected trials that necessitate unique solutions.

In addition to the physical demands that excursions such as Hillary's present, there are also many physiological limitations to the human body as well. These negative effects have been recorded as early as Plutarch's writings of Alexander's army crossing into India in 326 BC. Associated with these and other writings have been descriptions of headache, intestinal discomfort, and fatigue. Today, a collective term used for the many adverse symptoms associated with high altitude travel is acute mountain sickness (AMS). As much as ever, AMS is a common diagnosis among those who reach high altitudes. Those unfortunate enough to experience AMS can expect to endure a great deal of discomfort. Due to these inherent consequences, AMS can seriously alter expedition plans and put an end to the possibility of human achievement.

Significance of Study

As a result of the severity of AMS and the wide spread potential for negative outcomes, it is essential that this syndrome be better understood. A great deal of recent research has been dedicated to this end. Much of this research is aimed toward the use of arterial oxygen saturation (SaO_2) as a means of predicting future susceptibility to AMS upon further ascent. By conducting further research, a better understanding of the physiological responses to altitude that directly attribute to this syndrome will be gained. If pulse oximetry can be found affective at predicting AMS it would provide a quantitative measurement rather than just a subjective measurement. This could potentially revolutionize the diagnosis of AMS. Results from previous research show potential for pulse oximetry in predicting AMS (Burtscher, Flatz, & Faulhaber, 2004; Burtscher, Szubski, & Faulhaber, 2008; Koehle, Guenette, & Warburton, 2010; Roach, Greene, Schoene, & Hackett, 1998; Tannheimer et al., 2009), but the relationship between SaO_2 and AMS is still not well defined.

Two potentially major influences in the relationship between SaO_2 and AMS are the altitude at which a study is conducted and the duration of hypoxic exposure. To date most SaO_2 research has been conducted at an altitude of around 4000 m while the duration of hypoxic exposure has varied. The majority of conclusions from this collection of efforts have stated that alterations in these two factors are what allow, or refuse to allow, SaO_2 predictions of AMS to be possible. A major roadblock for this theory however, is the lack of studies that focus on the alteration of these variables.

This study provided a unique opportunity to observe the changes in SaO_2 with increasing duration of hypoxic exposure while relating that change to AMS prediction. This required a distinctive environment where participants could rapidly gain elevation while measurements were collected over a period of several days. Few places in the world allow for this style of data collection; Pico de Orizaba in Mexico is one of these places. Not only does this location lend itself well to the study of SaO_2 and AMS, but it also allows for a more in depth look at the possible connection between SaO_2 measurements and their impact on the summit success of mountaineers. Conducting this unique study provided a great deal of new insight into these critical relationships that can potentially lead to better treatments of the direct physiological mechanisms attributed to AMS and to faster recoveries. In addition, the knowledge learned may translate into fewer cases and earlier predictions of this syndrome.

Purpose of Study

The purpose of this study was to travel to an atypical location, Pico de Orizaba Mexico, to study the relationship between AMS and SaO_2. In order to perform this study, the Lake Louise Self-assessment (LLSA) of AMS (Roach, Bartsch, Oelz, & Hackett, 1993) was utilized to compare AMS onset with SaO_2 measurements. These SaO_2 measurements were recorded prior to AMS onset by finger pulse oximetry at 4260 m. This comparison has led to a better understand of the relationship between AMS and SaO_2.

Research Questions

1. Is SaO_2 measured by finger pulse oximetry at 4260 m predictive of individual susceptibility to the later development of AMS?
2. Is SaO_2 measured by finger pulse oximetry at 4260 m predictive of summit success in mountaineers who are making a summit attempt the following day on a 5640 m peak?

Hypotheses

1. A low (< 84%) SaO_2 measured by finger pulse oximetry at 4260 m will predict a mountaineer's susceptibility to AMS upon further ascent.
2. A low (< 84%) SaO_2 measured by finger pulse oximetry at 4260 m will also predict a mountaineer's inability to successfully reach the summit on a 5640 m peak.

Limitations

1. Measurements made by the use of pulse oximetry directly reflect the percentage of blood oxygen. As a result, these measurements do not directly represent an individual's rate of ventilation, as there are a great number of other variables.
2. Pulse oximetry measurements do not give any indication as to the degree of oxygen metabolism on the cellular level. It is therefore possible for oxygen saturation levels to be high while cells are experiencing a reduced rate of oxygen metabolism in response to many possible variables.

3. Number of participants was limited to the number of willing mountaineers attempting to summit during the data collection period.
4. Questionnaire type assessments, such as the LLSA, have a strongly subjective influence that can interfere with AMS diagnosis.

Delimitations

1. Due to the harsh conditions of this study, assessments of AMS severity during the climbers' attempts to summit the mountain were not possible. As a result of this complication, post ascent questionnaires were rephrased to report how the participants felt during their ascent.
2. In addition to the inability to collect AMS assessments, measurements of SaO_2 were also not possible during the ascents above 4260 m. These measurements were, however, taken immediately upon the return of climbers from their attempts to summit the mountain.
3. As all who arrived to the mountain hut wished to make an attempt to reach the summit, participants of this study were their own control. There was no assigned group receiving no treatment.

Assumption

LLSA will correctly diagnose AMS.

CHAPTER 2

REVIEW OF LITERATURE

The increasing prevalence of high altitude adventure and travel has lead to a need for the improvement of AMS prediction and prediagnosis. This review will discuss the relevant background and research associated with AMS and the use of pulse oximetry as a diagnostic tool. The presentation of these findings has been structured into the following categories: (a) acute mountain sickness, (b) techniques for the assessment of acute mountain sickness, (c) pulse oximetry, (d) use of SaO_2 for the prediction of acute mountain sickness, and (e) summary.

Acute Mountain Sickness

Acute mountain sickness is frequently diagnosed in unacclimatized individuals who ascend to 2300 m of altitude or higher. Studies have shown upwards of 25% of travelers experience the symptoms of AMS (Basnyat, Lemaster, & Litch, 1999). The symptoms and their severity can vary to the degree of altitude to which they are trying to achieve and are a result of the many physiological responses to a hypoxic environment. At high altitude, there is a combined effect of Dalton's Law and Henry's Law. These laws describe the low partial pressure of oxygen that exists in the high altitude environment. Human exposure to these hypoxic conditions causes oxygen transport between blood and tissue to be reduced. This reduction is the result of the pressure gradient between oxygen in the alveoli, arterial blood, and tissues all approaching zero. The total effect of these processes means less oxygen available for utilization by the

body. These effects are collectively referred to as hypoxemia. Further responses to hypoxemia force the cardiopulmonary system to work harder so as to overcome the oxygen deficit by pumping a greater volume of blood through the lungs as well as moving more air in and out. This increase in the volume of air through the lungs is referred to as the hypoxic ventilatory response (HVR).

Initial responses to hypoxia often lead to further physiological changes that can result in the recognizable symptoms of AMS. These characteristic symptoms can include headache, fatigue, drowsiness, lack of appetite, and gastrointestinal distress. There are many specific internal and external factors that have been theorized to play important roles toward the onset of AMS. External factors include the rate and extent to which altitude is achieved. Ascending at faster rates and higher altitudes increases the prevalence of AMS. An ideal strategy for acclimatization involves ascending at a rate no greater then 600 m per 24 hr when above 2500 m and for every increase of 600 to 1200 m above this altitude, an extra day of acclimatization should be added (Hackett & Roach, 2001). This process of slow ascent allows for the body to adjust to the changing conditions with altitude in a gradual manner. Presently, research indicates that those with a history of AMS susceptibility may be at an increased risk while exposed to high altitude conditions (Wagner, Fargo, Parker, Tatsugawa, & Young, 2006). This theory suggests, with a high degree of probability, that individual internal, genetically influenced physiological characteristics common among those of similar ethnicities or among the same family or even population must play a significant role in the onset of AMS. Current epidemiological evidence from research studying the relationship of families and

populations to AMS susceptibility is in support of this idea (Rupert & Koehle, 2006; Wang, Koehle, & Rupert, 2009). Examples of genetically influenced internal factors that may lead to AMS susceptibility include predispositions to diseases such as sickle cell anemia or even an individual's cranial capacity to accommodate an increase in cerebral blood volume and brain water levels throughout the edemic response to hypoxemia (Hackett & Roach, 2001). If the symptoms of AMS are ignored and ascent is continued, climbers will risk developing more serious and life-threatening conditions. High altitude cerebral edema (HACE) is thought to be an end stage of AMS (Hackett & Roach, 2001) and can lead to severe impairment and death if untreated.

Current treatment for AMS is highly generalized, as the direct physiological mechanisms are not fully understood. Common resolutions involve ceasing ascent and often a descent in altitude until symptoms resolve. Often, oxygen therapy is implemented in hopes of overcoming the hypoxic conditions, so as to lead to a quicker resolution of symptoms. Additionally, acetazolamide and dexamethasone have been used in the treatment and prophylaxis of AMS. Other drug treatments for AMS include the use of nonsteroidal anti-inflammatory drugs. Such nonsteroidal anti-inflammatory drugs include aspirin for the prevention of headache and ibuprofen for the treatment of headache (Hackett & Roach, 2001). In one study, a dose of 400 mg of ibuprofen eliminated symptoms of headache associated with high altitude (Broome, Stoneham, Beeley, Milledge, & Hughes, 1994). In another study, prophylactic aspirin taken every four hours significantly reduced the occurrence of headache while at altitude (Burtscher, Likar, Nachbauer, & Philadelphy, 1998).

Techniques for AMS Assessment

In response to the need for diagnosis and ability to quantify the severity of individual cases of AMS, several assessments have been developed. Two common forms are the Lake Louise Self-assessment (LLSA) and the Environmental Systems Questionnaire (ESQ). The LLSA was first published in *Hypoxia and Mountain Medicine*, which was compiled by the collaborative effort of the 7th International Hypoxia Symposium held in Lake Louise, Canada in February of 1991. The contribution of the LLSA is due to the work of Robert Roach and colleagues (1993). In addition to a questionnaire of reported AMS symptom severity, this extensive version of the LLSA also involves the judgment of a physician. There is however, a simpler version of the LLSA that bases its diagnosis solely on the self-reported severity of headache, gastrointestinal symptoms, fatigue and/or weakness, dizziness / lightheadness, and difficulty of sleeping. Each symptom is rated on a scale of 0, meaning none at all, to 3, implying severe. AMS is diagnosed if an individual presents with a headache, at least one other symptom, and a total combined score of 3 or higher.

A second common questionnaire, the ESQ, was originally designed to be an inventory of the 52 physiological and psychological symptoms to expect when traveling to altitude and did not include a rating scale (Kobrick & Sampson, 1979). Later versions of the ESQ evolved to include a scale for scoring the individual severity of AMS cases (Sampson & Kobrick, 1980). The most recent revision termed the ESQ III (Sampson, Cymerman, Burse, Maher, & Rock, 1983) now includes 67 items and an AMS rating scale. Current research has suggested that the ESQ III may be equally effective in

diagnosing AMS if limited to items pertaining to cerebral function only (Beidleman, Muza, Fulco, Rock, & Cymerman, 2007); thereby simplifying the administration process of the ESQ III.

Consequences of the inherent characteristics of AMS susceptibility require expeditions to harsh conditions if accurate field studies are to be produced. The proliferation of questionnaire type assessments such as the LLSA and the ESQ have been in large part due to the ease of their field implementation. Assessments that utilize little to no equipment and can be done in a quick, safe manner are an obvious choice. Often the fulfillment of these requirements can outweigh inherent, minor, faults in methodology as obtaining some result is often better than no result. One such fault of these questionnaire-type assessments is the dependency on the subjective judgments of the participants. The process of quantifying self-reported symptoms can become extremely unclear when attempting to compare diagnosis between individuals due to their differences in perception of sensations such as pain. Medical professionals have dealt with the issue of subjectivity in their field by implementing tools such as the visual analog scale (VAS). Gallagher, Bijur, Latimer, and Silver (2002) reported the use of a VAS as a means of quantifying the subjective idea of pain. The use of scales such as this has begun to arise in altitude research for the assessment of headache severity while at altitude (Harris, Wenzel, & Thomas, 2003), but little is known as to the correlation of these scales with AMS. Recently, a novel idea proposed the implementation of a version of the VAS in conjunction with questionnaire-type assessments as an effort to remove some level of subjectivity and to better understand the correlation between the differing methods of

assessment (Wagner, Parker, Tatsugawa, & Young, 2007). Wagner et al. reported that the VAS does, in fact, significantly correlate with the LLSA for AMS. Future research in this area could greatly simplify and improve current methods of AMS assessment.

Pulse Oximetry

Pulse oximetry refers to the process of measuring the saturation levels of oxygen bound to hemoglobin within red blood cells. These measurements are made possible by passing infrared and red light wavelengths through an extremity of the body. The two wavelengths have different levels of absorption and transmission depending on the saturation level of hemoglobin. The desaturation of oxygen within the blood results in an increase of infrared light transmission and a decrease of red light transmission (Severinghaus & Honda, 1978). A comparison of these two rates of absorption provides a percentage of oxygen saturation within the arterial blood supply.

The idea of utilizing pulse oximetry for SaO_2 measurements began to appear in the early 1970s. Takuo Aoyagi is credited with the breakthrough idea of measuring changes in light transmission and using these measurements to calculate SaO_2. While relying on the differences between these transmissions rather than the intensity of individual transmissions in his calculations, he realized that these changes could only result from the variations within the blood (Severinghaus & Honda, 1978). This method eliminated the influence of different tissue densities between individuals on SaO_2 measurements and made accurate pulse oximetry possible.

Since its inception, pulse oximetry has become common practice in the medical profession. There has grown a great deal of dependence upon pulse oximetry in situations such as operating rooms. In cases such as this, patients are sedated and may be incapable of self-regulating oxygen saturation. These measurements allow for physicians to accurately monitor saturation levels and safely perform necessary procedures. In addition to its implementation in the field of medicine, the development of portable oximeters has led to the possible application of the oximeter in a diverse variety of situations.

SaO_2 Prediction of AMS

Exposure to high altitude creates hypoxic conditions, which have been shown to be directly related to SaO_2 levels (Burtscher et al., 2008). The occurrence of AMS is also a direct response to these hypoxic conditions. Based on this close association between SaO_2 and AMS, pulse oximetry measurements have often been thought of as a possible means of AMS prediction and diagnosis. Physiological responses to hypoxia such as a reduced HVR or impaired pulmonary exchange (IPE) would result in a theoretical decline of SaO_2. This decline could be easily and accurately measured with the use of a pulse oximeter. The critical implication is that the symptoms of AMS must be a secondary response directly attributed to the reduced SaO_2 and not simply occur in conjunction with a declining SaO_2 value. If a significant relationship were proven to exist, and the conditions which allow for such a relationship were explicitly known, then the measurement of SaO_2 would be a powerful predictor of future AMS development. These

measurements would also be a critical component in providing the quantifiable objectivity that is currently lacking from other AMS assessments.

The issue that still remains, however, is that the relationship existing between SaO_2 and AMS is highly uncertain. In a recently published article investigating this relationship, AMS susceptibility and SaO_2 were compared in 150 climbers (Burtscher et al., 2004). Of the 150, 63 were precategorized as susceptible to AMS (AMS+) by reporting to have experienced AMS on two or more previous occurrences while exposed to hypoxic conditions. The other 87 climbers were precategorized as nonsusceptible to AMS (AMS-) as a result of having multiple episodes of hypoxic exposure with no reported AMS symptoms. The researchers defined past diagnosis of AMS as experiencing a headache in addition to at least one other symptom from the LLSA scale during past hypoxic exposures. Altitude of exposure of the study ranged from 2000 m to 4000 m and was quickly induced through the use of helicopters, cable cars, and/or hypobaric chambers. Immediately upon hypoxic exposure, participants were seated in a resting position. Twenty to 30 min after initiating the exposure, SaO_2 measurements were taken using a pulse oximeter. The SaO_2 of the AMS+ group was compared to the SaO_2 of the AMS- group. The results of this study show that at low altitude (600 m) the SaO_2 of both groups were the same. Furthermore, after exposure to high altitude the SaO_2 of the AMS+ group was 4.9% lower than the AMS- group. Based on the derived model, 86% of the AMS+ group could be correctly predicted by their SaO_2 measurements.

From this study (Burtscher et al., 2004), it was also concluded that the decline of the HVR is not given significant consideration when exposure time is limited in study

design; therefore, implying the importance of studying the HVR in relationship to short term hypoxic exposure and the onset of AMS. Also reported was the finding of most significant differences between AMS+ and AMS- at induced altitudes between 3000 m and 4000 m, indicating an optimal altitude for future research to be conducted. Another interesting statement made by this study was that they did not attribute low SaO_2 to IPE, as can be a common conclusion under these conditions. Due, in part, to this implication the researchers were left to ask how much exposure time as well as what degree of altitude are necessary for an IPE response? Also suggested as the reason for the discrepancy between findings of a relationship verses no relationship between SaO_2 and AMS were the duration of hypoxic exposure. Summarized were the studies with a short duration of exposure (hours to days), which usually find no relationship, while studies of extended exposure (days to weeks) tended to be more apt at deciphering the existence of a significant relationship. A combination of these conclusions leads to the necessity of deciphering the AMS onset timeline in relation to the interconnected pathways of different physiological hypoxic responses.

Additional insight into the SaO_2 and AMS relationship was provided by Burtscher et al. (2008) who published a meta-analysis on the topic. A search of Pub Med indexed articles from 1976-2007 was conducted using the following search terms: intermittent hypoxia, simulated altitude, acute mountain sickness (AMS), prediction, mountaineering, trekking, and hypoxic ventilatory response (HVR). In addition to the search, the analysis included other relevant papers known to the author. In total, 16 papers were found, all of which looked at short-term hypoxic exposure. After review of

the studies, it was concluded that due to the differing conditions and parameters of the studies, no clear conclusions about SaO_2 and AMS could be clearly derived. However, it was stated that hypoxia was clearly responsible for AMS and that the degree of hypoxia, when acutely exposed to high altitude, varies markedly between individual HVR. Therefore, individual HVR may be considered as a predictor of tolerance to acute hypoxia. Also stated were the summarized results leading to SaO_2 values taken after prolonged exposure to hypoxia as being found to closely relate with AMS susceptibility. Upon compilation of these studies, it was concluded that a possible reason for the occurrence of significance among these studies was the influence of an IPE that may occur after prolonged hypoxic exposure. However, the question of how much time and altitude are needed for this impairment remained unanswered.

Hoping to better understand how much exposure time and altitude are necessary for SaO_2 prediction of AMS, Roach et al. (1998) performed a study of asymptomatic climbers coming through base camp at 4200 m on Denali with low SaO_2. The hypothesis presented was that these climbers would be more likely to develop AMS on further ascent. In all, 102 climbers volunteered to participate in the study. The climbers had flown to 2100 m, and then hiked to 4200 m in 9 ± 0.2 days to reach the camp. They stayed at 4200 m before testing for 2.3 ± 0.2 days. The total time spent by climbers at 4200 m before ascent was 4.5 ± 0.5 days. During the study, climbers were above 4200 m on summit attempt (6194 m) for 3 ± 0.2 days before returning for retesting. Upon returning, climbers were interviewed about their symptoms while above 4200 m. Roach et al. classified AMS using a clinical interview technique. A score of 2+ was classified as

AMS. Throughout the study, SaO_2 measurements were collected using ear oximetry. In addition to SaO_2, HVR rates were also measured. During the study 53% of climbers reached the summit. Reported reasons for not reaching the summit included inclement weather (45%), illness/other (35%), or involved in rescue (20%). Fifty-eight percent of climbers had no AMS during ascent while 42% were diagnosed with AMS during ascent. It was found that age related to AMS severity as 51% of those younger than 35 years had AMS and only 5% of those older than 35 years developed AMS. The SaO_2 at 4200 m for all participants was 81.5 ± 4.4. The HVR rates were not predictive of AMS susceptibility, leaving the relationship between these highly individualized responses and AMS in doubt and uncertain. Finally, AMS was found to be significantly and negatively correlated with SaO_2 ($r = -.48$, $p < .001$). It was concluded that SaO_2 as measured by ear oximetry was predictive of subsequent AMS development. Subjects with SaO_2 above 84% at this altitude appeared to be immune to subsequent AMS. Using the 84% SaO_2 cutoff predicted 100% of those who would succumb to AMS, but 55% of all participants were false positives, meaning that they were classified as susceptible and did not get sick (score of 3+). Lowering the cut off to a SaO_2 of 81.5% predicted 81% of those who would succumb to AMS, and reduced the false positives to 26%. Also concluded was that IPE may have been a cause of desaturation among participants and that this impairment begins prior to the onset of the clinical symptoms of AMS, leaving the possibility of IPE measurements to be highly related to AMS predictability.

In a study similar to Roach et al. (1998), SaO_2 was observed as a possible predictor for the occurrence of AMS (Koehle et al., 2010). This study looked at

individuals who had traveled to 4380 m, making the last 2200 m of travel within the last 1-2 days. The LLSA was used to diagnose AMS and SaO_2 was measured using a finger pulse oximeter. Results from the 41 participants showed significantly lower SaO_2 among those with AMS as well as the SaO_2 values being significantly correlated with the LLSA scores. In further support of these findings, Tannheimer et al. (2009) also reported a positive relationship between SaO_2 and AMS. Each of these studies lends further evidence toward the establishment of a relationship between SaO_2 and AMS.

Contrary to some of the current research, there are several studies that still leave the relationship between SaO_2 and AMS to question. O'Connor, Dubowitz, and Bickler (2004) conducted a study on Mt. Rainier with 169 participants, using the LLSA to score AMS 30 min after resting at 3080 m. Arterial oxygen saturation measurements from each participant were taken along with their heart rate. These two measurements, along with other recorded variables, were compared for correlation to the prevalence of AMS. Twenty-seven percent of the climbers were diagnosed with AMS during the study. The mean SaO_2 at 3080 m was reportedly 90 ± 4% and the mean heart rate at 3080 m was 87 ± 14 beats·min^{-1}. Heart rate was the only variable found to significantly correlate with AMS, as SaO_2 was not a significant predictor. These data support other studies where subject's exposure to altitude was limited by study design to hours/days where no significant relationship between SaO_2 and AMS was found (Roeggla, Roeggla, Podolsky, Wagner, & Laggner, 1996; Wagner et al., 2006).

Summary

According to the recent research, it is possible that there is a relationship between SaO_2 and AMS. This relationship however, if present, is not well understood. It would appear that SaO_2 measurements taken 20-30 min after hypoxic exposure at 2300-4200 m are a good predictor of AMS susceptibility and that HVR and IPE seem to be important in the complex pathophysiological acclimatization process responsible for AMS. If this relationship is to be better understood, researchers must decipher the pathway of physiological response to hypoxemia and the timeline of AMS symptom onset.

CHAPTER 3

METHODOLOGY

This chapter details the methods used in discovering how SaO_2 relates to AMS through the utilization of the LLSA and finger pulse oximetry. The chapter is organized as follows: (a) participants, (b) setting, (c) procedure, and (d) data analysis.

Participants

Every climber arriving to the Piedra Grande mountain hut (4260 m) on Pico de Orizaba intending to make a summit attempt was informed about the study and invited to participate. Each was given the letter of information to read, as this served as the informed consent. The letter was available in both English (Appendix A) and Spanish (Appendix B). Climbers who did not understand either English or Spanish were excluded from the study. Potential participants were encouraged to ask questions about the study before agreeing to participate. The institutional review board of Utah State University approved this study.

Setting

The study was conducted from the Piedra Grande climber's hut (4260 m) on the north side of Pico de Orizaba (5640 m). Data collection from the hut spanned 8 consecutive days. As a popular climbing destination with easy access, this location allowed for a large number of participants to take part in the study. This site was also chosen for its optimal altitude for SaO_2 measurements (Burtscher et al., 2008) as well as

to correlate with other research conducted at a similar altitude (Roach et al., 1998). In addition, this site provided a unique opportunity to study short-term field exposure to high altitude as a result of the rapid rate at which the majority of climbers were able to ascend. This unique timeline allowed for a short duration between the testing and retesting and provided a better understanding of the timeline associated with the onset of the physiological reactions that lead to AMS and their possible association with SaO_2.

Procedures

Climbers traveled by truck from the village of Tlachichuca (2650 m) to the Piedra Grande hut. As a result of this mode of travel they rapidly gained 2990 m of elevation within about 2 hr. After being informed of the study and if willing to participate, they were assigned an identification number. The date and time of each participant's arrival to the hut was noted. Within 30 min of exposure to 4260 m at the hut, data were collected.

The participants were seated while background and demographic information was collected (Appendix C). Demographic characteristics included age, gender, height, and weight. Historical information included altitude of residence, history of altitude illness, number of ascents above 3000 m in the 10 days prior to this ascent, time above 3000 m in the 10 days prior to this ascent, highest altitude achieved, and hours per week of training in the months leading up to this ascent. Following this collection of information, the LLSA for AMS (Appendices D and E) was administered. This was done by asking the participants to rate a series of 5 symptoms based on their current individual level of perceived severity. Each symptom was ranked on a scale ranging from "0 - None at all"

to “3 - Severe.” After LLSA administration, pulse oximetry measurements were taken. The finger pulse oximeter (SportStat, Nonin Medical Inc., Plymouth, MN) was placed on the right index finger, and the SaO_2 value remained stable for 1 min before being recorded. Also, the heart rate (HR) was measured by the pulse oximeter while noting the date and time of measurement. Following this initial data collection the climbers stayed in the hut until their summit attempt. Immediately prior to their departure SaO_2 and HR measurements and the LLSA questionnaire were repeated. Again, the time and date of the climbers’ departure was recorded. Immediately upon return from their summit attempt the date and time of return was recorded. All physiological measurements and the LLSA were repeated. This post ascent administration of the LLSA for AMS was phrased so as to describe the climbers’ perceived symptoms of sickness throughout the duration of their summit attempt above the hut (4260 m). In addition to the repetition of data collection as before, additional ascent data were collected. Climbers were asked whether they reached the summit, and whether they used acetazolamide or dexamethasone. If climbers were unsuccessful with their summit attempts, they were asked to report a reason for not reaching the summit. Possible reasons included: AMS, weather, fatigue, partner failure, or other.

Data Analyses

In the process of data analyses, the total hypoxic exposure times and median exposure times were calculated for the participants. Acute mountain sickness for each participant was scored for all three data collection periods (arrival at 4260 m, immediately prior to departure for the summit attempt, and immediately upon return from

the summit attempt) using the LLSA. Positive diagnosis involved participants presenting with a headache, at least one additional symptom, and a total symptom score ≥ 3. The means for all descriptive data were then calculated. Independent *t* tests were performed to determine if there was a significant mean difference in the preascent SaO_2 of those who did or did not experienced AMS during the ascent. Significance was based on $p \leq .05$. Following which, all collected variables were compared for significant difference between those with and without AMS development using independent variable *t* testing. Furthermore, an independent *t* test was conducted to determine if there was a significant mean difference in SaO_2, as measured preascent, between those who reached the summit and those who turned back. Significance was also based on $p \leq .05$. Next, independent variable *t* tests were conducted to compare all other collected variables for significant difference between those who reached the summit and those who did not. Then each of the mean SaO_2 measurements for participants with AMS and without for each of the three collections, were compared for statistical difference using an independent *t* test. Lastly, a change in SaO_2 was calculated between different periods of data collection. The first interval was from participants' arrivals to the hut until their departure for the summit. The second interval was from the participants' departures for the summit until their return. The third interval was calculated was from the participants' arrivals to the hut until their return from attempting to reach the summit. These differences were then compared between those who developed AMS and those who did not using an independent *t* test for statistical difference. All statistical analyses were done using the Statistical Package for the Social Sciences (SPSS, version 17.0) or Microsoft Excel (Version 2004).

CHAPTER 4

RESULTS

Throughout the duration of the data collection, 77 climbers arrived for their attempt to summit the mountain. Of those 77 climbers, 2 declined to participate for personal reasons, and 2 were omitted due to communication barriers. In total, 73 climbers volunteered to participate in the study. Twelve of the 73 climbers were missing relevant data. Missing data occurred due to unexpected complications such as an inability to contact participants post ascent. In addition, 13 reported using acetazolamide or dexamethasone to counter the effects of AMS. These individuals were excluded from the statistical analyses as the use of these drugs has been shown to reduce AMS symptom severity (Hackett & Roach, 2001). After all exclusions, there were 48 remaining participants with complete data; 45 were male and 3 were female. The descriptive statistics collected for the 48 remaining participants are summarized in Table 1.

In computing the time intervals between tests and retests, it was found that the median time participants stayed at the hut before departing for the summit was 10 ± 13 hr. The median time for participants to reach the summit and return to the hut was 13.3 ± 4.8 hr. Of the participants, 29 (60.4%) successfully reached the summit. For those unable to reach the summit, reasons given were; AMS being reported as the perceived cause in 8 (42.1%) cases, fatigue in 8 (42.1%) cases, partner failure in 2 (10.5%) cases, and other reasons were cited in 1 (2.1%) case. In measuring AMS; 2 (4.2%) had AMS (LLSA) upon arrival to the hut, 4 (8.3%) had AMS (LLSA) immediately prior to departure for the summit attempt, and 29 (60.4%) participants met the criteria for AMS (LLSA) during

the ascent. In addition, 23 (79.3%) of these AMS cases during ascent were severe as they scored ≥ 5 on the LLSA. The mean and standard deviations of the SaO_2, HR, and LLSA scores reported in Table 2 are separated between each of the three data collections. Table 3 identifies the number of participants diagnosed with or without AMS against those who did or did not summit.

Table 1

Descriptive Statistics (N = 48)

Descriptive Statistic	Min	Max	*M*	*SD*
Age (yr)	18	61	34.0	9.8
Height (cm)	148	190	176.0	7.6
Weight (kg)	45	100	75.2	11.2
BMI (kg/m^2)	18	32	24.3	3.4
Altitude of residence (m)	0	3000	1446.7	866.0
Ascents above 3000 m in last 10 days	0	10	1.4	1.9
Time (hr) above 3000 m in last 10 days	0	240	22.0	42.0
Highest altitude achieved prior to this attempt (m)	2200	8850	5466.0	1176.6
Hours per week training	0	25	8.6	7.1

Note. Data are self-reported and body mass index (BMI) is calculated from self-reported weight and height.

Table 2

Heart Rates, Oxygen Saturations, and LLSA Scores (N = 48)

Upon Arrival to hut	Min	Max	*M*	*SD*
Heart rate (beats·min^{-1})	53	127	83.9	17.5
SaO_2 (%)	74	94	86.3	4.3
LL AMS score	0	3	0.7	0.9
Departure for summit				
Heart rate (beats·min^{-1})	58	118	91.0	14.5
SaO_2 (%)	72	90	84.3	3.9
LL AMS score	0	8	1.8	1.7
Return from summit				
Heart rate (beats·min^{-1})	78	129	103.3	12.0
SaO_2 (%)	70	93	84.7	5.7
LL AMS score	0	13	5.3	3.3

Note. Scores and measurements are reported for the three periods of data collection that were conducted throughout the study.

Table 3

Acute Mountain Sickness and Summit Success Reported by Number of Climbers

	Summit	No Summit	Total
AMS	16	13	29
No AMS	13	6	19
Total	29	19	48

From the *t* tests conducted, there was no significant difference ($p = .90$) found in preascent SaO_2 between those who experienced AMS during ascent ($SaO_2 = 84.3 \pm 3.3\%$) and those who did not ($SaO_2 = 84.2 \pm 4.2\%$). Figure 1 displays the weak correlation ($r = -.14$) associated with the preascent SaO_2 measurements and subsequent AMS development. There was also no significant difference ($p = .18$) in preascent SaO_2 between those who reached the summit ($SaO_2 = 84.8 \pm 3.7\%$) and those who did not ($SaO_2 = 83.3 \pm 4.0\%$). After performing these comparison tests for significance, all demographic and historical variables were analyzed for any significant difference between AMS and summit success groups. There were several variables found to be significantly different ($p < .05$) between groups (Table 4). Age was found to be significantly higher ($p = .03$) among those without future development of AMS. Participant's altitude of residence was another variable significantly different ($p = .02$) between groups. Also, the highest altitude achieved prior to this attempt was found to be significantly greater ($p < .01$) for those who did not get sick.

Table 4

Mean Values of Significantly Different Characteristics Between Those With AMS and Those Without

	No AMS *M* ± *SD*	AMS *M* ± *SD*	*p* Value
Age (yr)	37.5 ± 10.5	31.4 ± 8.4	$p = .03$
Altitude of Residence (m)	1776 ± 718	1197 ± 881	$p = .02$
Highest Altitude Achieved (m)	6029 ± 1059	4912 ± 966	$p < .01$

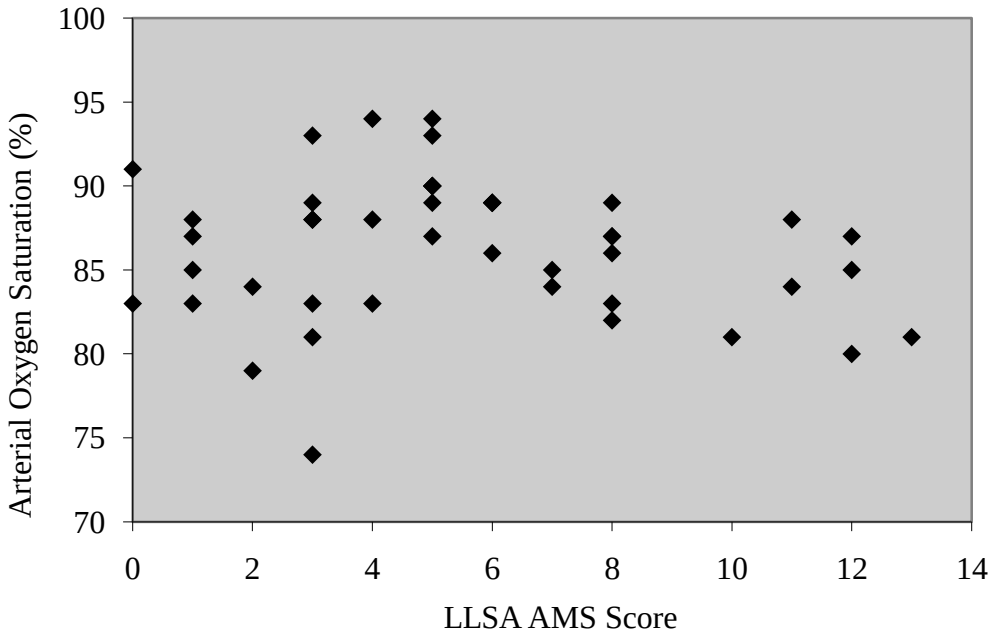

Figure 1. Relationship between arterial oxygen saturation prior to summit attempt and LLSA AMS severity during summit attempt ($r = -.14$).

While comparing all variables to participants' summit success, it was found that the number of ascents over 3000 m in the 10 days previous to this attempt was significantly greater (p = .02) for those who successfully reached the summit than those who did not. Participants who reached the summit had ascended an average of 1.8 ± 2.1 peaks while those who were unsuccessful had only ascended an average of 0.6 ± 1.2 peaks in the 10 days prior to this attempt. The number of hours spent above 3000 m in the 10 days previous to this study was another variable found to be significantly greater (p = .02) for those who found summit success. Participants who reached the summit had spent an average of 31.8 ± 49.6 hr above 3000 m while those who did not had only spent an average of 4.7 ± 14.5 hr. In addition, highest altitude ever achieved prior to this expedition was found to be significantly higher (p = .04) among those who successfully reached the summit. Those who had previously achieved an altitude of 5687.7 ± 1016.6 m were successful while those who were not had only achieved an altitude of 5028.1 ± 1276.3 m. Lastly, heart rate as measured at the time of departure for the summit attempt was significantly lower (p = .04) for those who would reach the summit. These participants had an average HR at the time of departure of 87.6 ± 14.2 beats·min^{-1} while those who did not reach the summit had an average HR of 96.1 ± 13.4 beats·min^{-1}.

In order to further analyze AMS development and summit success, each SaO_2 measurement was compared to a corresponding LLSA AMS score that was recorded at the same time. Table 5 and Figure 2 show the average SaO_2 measurements of those who developed AMS and those who did not as they changed over the course of the study. Arterial oxygen saturation and AMS were not significantly different (p = .94) when the

participants arrived to the hut. They were also not significantly different (p = .58) prior to participants' departures for their summit attempts. Arterial oxygen saturation was however significantly different (p = .04) between those with and without AMS in the measurements recorded after participants' summit attempts. Figure 3 shows this correlation between SaO_2 and AMS (r = -.21).

Following this comparison, the differences in a participant's initial, preascent, and post ascent measurements of SaO_2 were calculated so as to view how a participant's change in oxygen saturation (ΔSaO_2) over time would relate to AMS development (Figure 4 & Table 6). It was found that all participants showed desaturating ΔSaO_2 from the time of their arrival to the time at which SaO_2 was measured prior to ascent (ΔSaO_2 = -2.3 ± 3.3%). This desaturation was not significantly different (p = .71) between those with or without later AMS onset. However, the ΔSaO_2 between preascent and post ascent was significantly different (p = .03) between those who developed AMS and those who did not. The ΔSaO_2 over the entire course of hypoxic exposure (conclusion of climb – arrival at hut) was also significantly different (p = .03) between groups.

Table 5

Mean Arterial Oxygen Saturation for Those Who Developed AMS and Those Who Did Not

	AMS *M* ± *SD*	No AMS *M* ± *SD*	*p* Value
Arrival to the Hut (%)	86.5 ± 2.1	86.3 ± 4.3	.94
Departure for the Summit (%)	85.3 ± 5.5	84.1 ± 3.7	.56
Return from Summit Attempt (%)	82.9 ± 6.9	86.5 ± 3.6	.04

Note. Measurements are reported for the three periods of data collection that were conducted throughout the study.

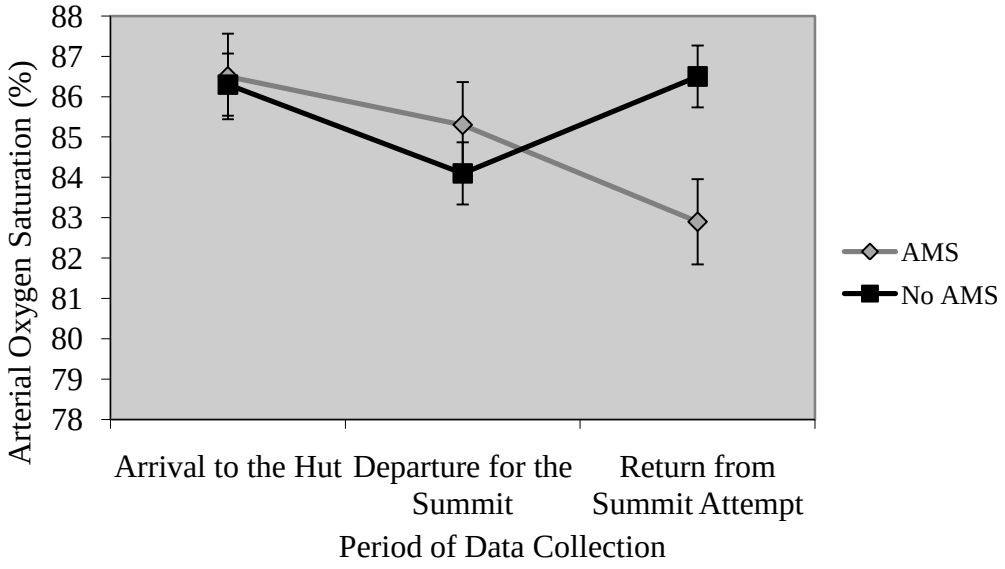

Figure 2. Progression of mean arterial oxygen saturations of participants who developed AMS versus the saturations of those who did not throughout the study.

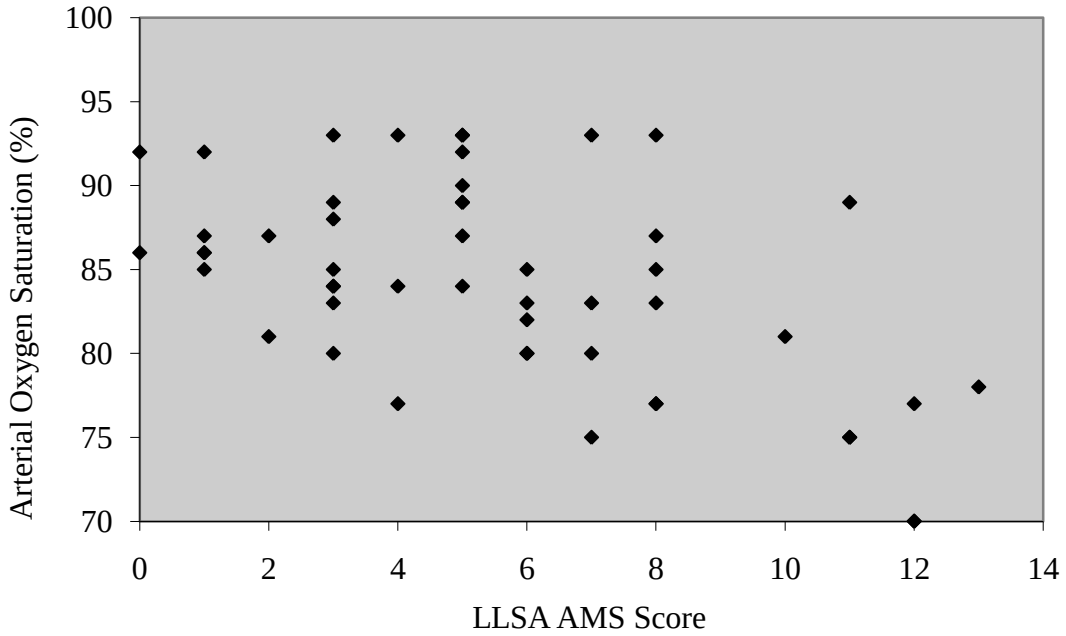

Figure 3. Relationship between post ascent arterial oxygen saturation and severity of AMS (r = -.21).

Table 6

Mean Values of the Change in Arterial Oxygen Saturation (ΔSaO_2) for Participants With and Without AMS

	AMS *M* ± *SD*	No AMS *M* ± *SD*
ΔSaO2 from Arrival to Departure for Summit (%)	-2.2 ± 3.5	-2.6 ± 3.0
ΔSaO2 from Departure for Summit to Return (%)	-0.7 ± 5.2	2.3 ± 3.2
ΔSaO2 from Arrival to Return from Summit Attempt (%)	-3.5 ± 5.8	0.2 ± 3.7

Note. ΔSaO_2 is calculated by taking the differences of SaO_2 measurements taken from two different data collections during the study.

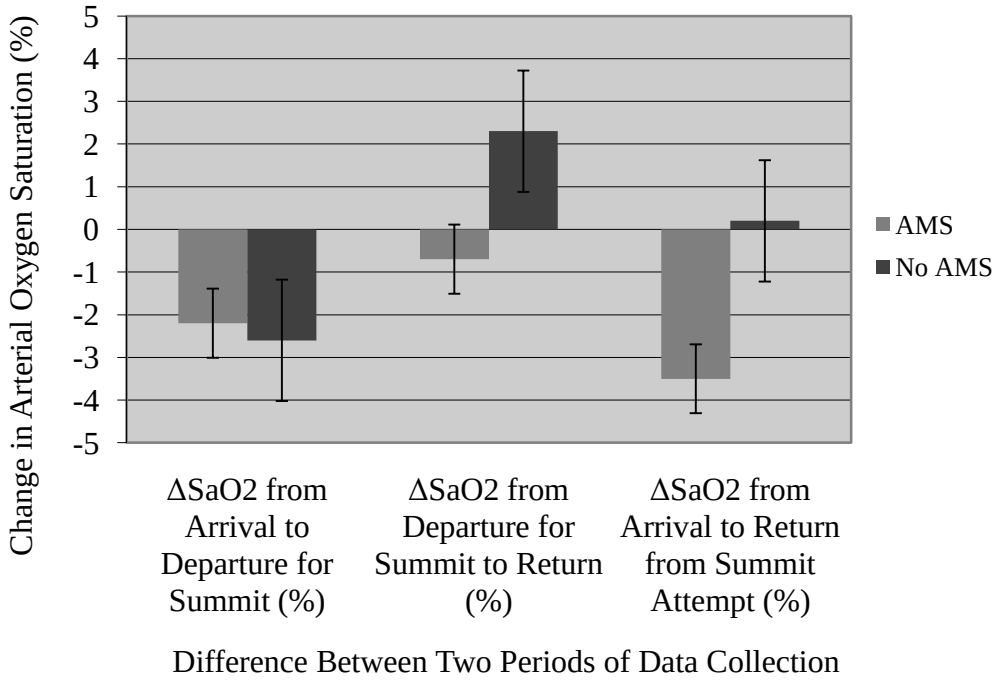

Figure 4. Change in arterial oxygen saturation (ΔSaO_2) of participants who developed AMS versus those who did not. There was a significant difference found between those with AMS and those without in the second (p = .03) and third (p = .03) comparisons of ΔSaO_2 measurements.

CHAPTER 5

DISCUSSION

SaO_2 and Summit Success

Of all the participants in this study, more than half of them reached the summit successfully, but the preascent SaO_2 of these participants was not significantly different from those who were unsuccessful. These results were unexpected. It is possible that a low SaO_2 may be overcompensated by a high level of physical fitness among climbers reaching the summit. Wagner et al. (2008) found that increasing the number of hours spent training per week was related to a greater likelihood of reaching the summit. In addition, the results of this study show heart rate was significantly lower, prior to ascent, among those who reached the summit. These findings correlate well, as a low heart rate can be a direct result of increased fitness. Each of these factors lends further support to the argument of increasing fitness as a means of overcoming a possible low SaO_2 during a summit attempt.

Significance of Other Variables to Summit Success

After finding no relationship between SaO_2 and summit success, it was logical to look to other variables to explain the difference between participants who reached the summit and those who did not. It was found that the number of ascents and the number of hours spent above 3000 m in the 10 days previous to this study were significantly different between participants who were successful and those who were not. This

difference could be attributed to the relationship between recent ascents and acclimatization, therefore, leading those with numerous recent attempts to be more acclimatized and less likely to become sick. However, as AMS and summit success were found to be unrelated, it is unlikely for this to be the solution. In addition to these findings, the highest altitude ever achieved by participants as well as their heart rate as measured at the time of departure for the summit attempt were also significantly different between successful and nonsuccessful participants. All of these results point toward participants' level of mountaineering experience as being directly related to their success on the mountain. This conclusion is strongly supported by current theories on successful mountaineering (Pesce et al., 2005; Wagner et al., 2008). The recent number of ascents, time above 3000 m in the 10 days previous to this study, and a greater personal record of highest altitude achieved all describe participants who are avid climbers and familiar with successful mountaineering.

In addition to finding a relationship between these variables and summit success, it was interesting to note that there was not a significant relationship between AMS and summit success. This lack of relationship implies that the symptoms of AMS did not stop the experienced climber from reaching the summit. Other research concludes that this occurrence is a result of the inherent characteristics of mountaineering causing it to be a highly driven sport. Therefore, the individuals involved in mountaineering must be equally driven. As a result of this drive, AMS does not seem to prevent motivated climbers from reaching the summit (Davies et al., 2009; Pesce et al., 2005; Tsianos et al., 2006; Wagner et al., 2008). This is not to say that ignoring the symptoms of AMS is the

only way to successfully reach the summit of a mountain. Another interpretation of this theory suggests that with experience, mountaineers become more familiar with knowing how hard and how far they can safely push their personal physical limitations. Although an experienced mountaineer may be aware of possible impending AMS symptoms, they could be more adept at assessing whether they would be able to reach the summit and safely return. Those lacking this experience may be more willing to err on the side of caution by heeding their symptoms of AMS and return short of their goal. Whatever the reasons for successfully reaching the summit, it is apparent from these results that neither SaO_2 nor AMS significantly influenced these participants' ability to reach the summit. It is also clear that individual level of fitness and experience played a crucial role in summit success.

SaO_2 and AMS

The results of the study indicate that there was no significant relationship between preascent SaO_2 measurements and the development of AMS upon further ascent. These results were unexpected as they differ from current theories that recognize a significant relationship between AMS and SaO_2. It is important to recognize that there are many variables, such as level of exertion, that have a strong tendency to reduce the reliability and validity of pulse oximetry measurements (Ontario Ministry of Health, 1992). Furthermore there are many variables that may have a significant influence on AMS assessment such as participant's level of machismo (Sobralske, 2006). These factors

could have made considerable contributions toward finding no significance between SaO_2 and AMS in this study.

Additional analysis of AMS and SaO_2 from each of the three data collections, however, revealed a bit more detail into this complicated relationship. Comparisons of concurrent AMS assessments and SaO_2 measurements for each of the first two data collections did not show any significant difference. The third data collection, which followed the participants' hypoxic exposure during their summit attempt, did however show a significant difference in the SaO_2 of those with AMS compared to those without. The linear comparison of these means is represented in Figure 2. The initial lack of a significant relationship between AMS and SaO_2 followed by the future progression of these means toward significance could be in part due to two different physiological responses to hypoxia. These two responses are the HVR and IPE. Bartsch, Swenson, Paul, Julg, and Hohenhaus (2002) conducted a study examining these acute ventilatory responses to hypoxia in relation to AMS. From their study, it was found that HVR attributed to a reduction in AMS scores from day 2 to 3 and was also associated with substantial improvement in oxygenation and ventilation. Furthermore, individuals with AMS showed lower, delayed HVR. From this study, IPE was reported to worsen with continued exposure and that severe cases of AMS resulted from a combined effect of HVR and IPE. Burtscher et al. (2004) also suggested HVR as a possible factor of influence in short term hypoxic SaO_2 changes as well as the role of IPE in long term hypoxic exposure. These points were further illustrated by showing that AMS susceptible individuals presented a low SaO_2 when initially exposed to hypoxic conditions to which

the researchers attributed a low HVR. To further support this theory, Roach et al. (1998) attributed IPE as a possible cause for oxygen de saturation among the participants of their study.

Each of these studies makes implications toward an important consideration for the use of IPE and HVR as an explanation for these findings. This implication is the potential difference in time for the onset of physiological oxygen saturation changes, which are measured by pulse oximetry, and the onset of the cerebral edema that causes the symptoms measured by the LLSA (O'Connor et al., 2004). This difference between these two times of onset for such necessary responses implies that if SaO_2 measurements are to be predictive of AMS assessments, the point at which oxygen saturation responses and cerebral responses to hypoxia overlap must be identified. The occurrence of an overlapping point in such symptoms would provide an optimal period for the SaO_2 prediction of future AMS susceptibility. This theory is also supported by conclusions backing the use of SaO_2 for AMS predictions and stating the necessity of adapting SaO_2 measurements to different locations with resulting changes to exposure time while at differing altitudes (Roach et al., 1998). Current research suggests that this duration of hypoxic exposure is a major factor of influence in the fluctuation of this critical point (Burtscher et al., 2008). Comparisons of the methods used in other recent studies reveals a great deal about the possible processes occurring with AMS and this timeline associated with symptom onset and exposure duration. The majority of studies reporting a significant relationship between SaO_2 and AMS were either conducted after a prolonged hypoxic exposure or were a result of comparing SaO_2 measurements with immediate

AMS assessments (Burtscher et al., 2004, 2008; Koehle et al., 2010; Roach et al., 1998; Tannheimer et al., 2009). Studies finding no relationship between AMS and SaO_2 involved the measuring of SaO_2 and AMS after a short hypoxic exposure (O'Connor et al., 2004; Roeggla et al., 1996; Wagner et al., 2006). These comparisons show that a longer duration of hypoxic exposure may lead to a higher probability of finding a significant relationship between SaO_2 and AMS.

A combination of the effects of HVR and IPE can be compiled through viewing the current theory of the physiological processes of AMS onset. Hackett and Roach (2001) provide a detailed description that lends a great deal of insight into the time line necessary for SaO_2 assessment of AMS. Apparent in their description, the authors showed two periods of hypoxia, the first resulting from the initial environmental exposure. This exposure elicits the immediate responses associated with hypoxia including the variable HVR. The second period of hypoxia is referred to as an exaggerated hypoxia. This level of hypoxia is apparently due to further compounding effects of physiological responses on top of the initial external, environmental exposure and is mostly limited to the pulmonary system causing IPE. As the duration of hypoxic exposure increases, these impaired pulmonary system responses progress toward the cerebral effects of AMS such as an increase in cerebral blood volume and brain water levels. This description seems to attribute initial symptoms of AMS to HVR and later symptoms to IPE. Further evidence from this paper reports cases of mild to moderate AMS showed no increased intracranial pressure. Therefore, initial AMS is not due to an

increase in intracranial pressure but more likely to mild oxygen deprivation. Moderate to severe AMS, however, showed cases of increased intracranial pressure.

Summarizing the combination of these ideas, it is possible to conclude that initial hypoxic exposure causes a HVR among mountaineers. As HVR differs between individuals (Burtscher et al., 2008), it can be assumed that those with a decline in HVR may incur the initial mild symptoms of AMS as a result of minor oxygen deprivation. As these initial symptoms presumably occur without the physiological cerebral edemic responses assessed by the LLSA, this period of AMS is unrelated to SaO_2 measurements. Additionally, increasing the duration of hypoxic exposure can lead to an IPE (Roach et al., 1998). This IPE may result in a concurrent oxygen desaturation measured by pulse oximetry along with the cerebral responses assessed by the LLSA. This degree of hypoxic exposure could represent the point at which symptom measurement and hypoxic response overlap, thereby allowing for SaO_2 measurements to be significantly related to AMS.

Significance of Other Variables to AMS

In addition to these results, it was also found that age of participants, altitude of residence, highest altitude ever achieved before this attempt by participants, and SaO_2 as measured after ascent were all significantly different between participants with and without the development of AMS. Age has often been associated with susceptibility to AMS (Roach et al., 1998; Wagner et al., 2006, 2008). Altitude of residence could relate with AMS development due to the effects of living and training at higher altitude toward

an increased base level of acclimatization and improved performance while at altitude (Levine & Stray-Gunderson, 1997; Stray-Gunderson, Chapman, & Levine, 2001). Highest altitude achieved prior to this attempt could relate in the same manner to AMS as was previously presented toward the association with summit success in that those with a history of high ascents tend to be the more experienced climbers. With experience comes a familiarity to the symptoms of AMS along with a possible desensitization to their effects. As a result, when asked about the severity of their symptoms, experienced climbers may respond lightly with the sense of having experienced a lot worse on previous trips. This familiarity could affect the assessment of AMS when using the LLSA.

Conclusion

Arterial oxygen saturation measurements were found to be unrelated to the onset of AMS and therefore, SaO_2 does not appear to be a reliable tool for the prediction of AMS susceptibility. In addition, SaO_2 measurements were not related to a mountaineer's ability to successfully reach the summit. Based on these findings, it can be concluded that SaO_2 is not useful for predicting summit success among mountaineers. The reliability and validity of pulse oximetry measurements and participant's level of machismo are most likely significant contributors to the complex relationship of SaO_2 and AMS found in this study. There are also a great number of other uncontrolled variables that create a complicated interplay of external environmental variables and internal physiological characteristics. Further research is needed to better understand these intricately

overlapping variables if SaO_2 is to be applied to the prediction and diagnoses of AMS. It may be that if this relationship does exist, there are simply too many factors to be controlled in order to make these measurements useful. It is recommended that other potential prediction methods and assessments be discovered.

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APPENDICES

Appendix A: Informed Consent (English)

UtahStateUniversity
Dept. of Health, Physical Ed., and Recreation
7000 Old Main Hill
Logan, UT 84322-7000
Voice: (435) 797-1497

Date Created: November 11, 2008; Page 49 of 60
USU IRB Approved: 11/00/2008
Approval terminates: 11/00/2009
Protocol Number: 2180
IRB Password Protected per IRB Administrator

LETTER OF INFORMATION

Arterial Oxygen Saturation as a Predictor of Next-day Acute Mtn. Sickness or Summit Success

Introduction/Purpose: Dr. Dale Wagner, assistant professor in the Health, Physical Education and Recreation Department at Utah State University (USU), and his research assistants (Jon Knott and Jack Fry) are conducting a study to determine if the arterial oxygen saturation measurement (amount of oxygen in your blood) from a finger pulse oximeter can predict acute mountain sickness or success in reaching the summit of Pico de Orizaba. We anticipate approximately 50-100 participants in this research study, and we invite you to participate.

Procedures: Upon arriving at the Piedra Grande Hut, you will be asked a few basic personal questions (e.g., age, height, weight). After sitting for about 3 minutes, your heart rate and oxygen saturation will be measured with a finger pulse oximeter. The oximeter is a noninvasive device that fits over the index finger similar to the machines that measure heart rate commonly used in shopping malls and gyms. The pulse oximeter measurement will be repeated 3 times: 6-hours after the first measurement, just before you depart for your summit attempt, and just after you return to the hut from your summit attempt. You will also be asked about acute mountain sickness symptoms and whether or not you reached the summit. The total time for your participation in this study will be approximately 10 minutes.

Risks: Data will be collected with an interview from a questionnaire and from the finger pulse oximeter; physical risks to participation in this study are minimal. The only foreseeable risk is to your privacy, however only the interviewer will know your personal information. Only the researchers associated with this study will have access to the data.

Benefits: Researchers hope to have a better understanding of what factors are advantageous to reaching high mountain summits as well as determining which factors predispose someone to acute mountain sickness. If we can tell by a number on a pulse oximeter whether or not someone is likely to develop acute mountain sickness if ascent is continued, then this would be a great benefit to the safety and success of future mountaineers.

Explanation & Offer to Answer Questions: Dr. Wagner or one of the other affiliated researchers has explained this study to you. You have been given the opportunity and encouraged to ask questions. If you have other questions or research-related problems, you may reach Dr. Wagner at (435) 797-8253 or by email at: dale.wagner@usu.edu

Extra Costs & Payments: There is no cost for your participation in this study. You will not be paid for participating in this study.

Voluntary Participation & Right to Withdraw: Your participation in this research is entirely voluntary. You may refuse to participate or withdraw at any time without consequence.

Confidentiality: Research records will be kept confidential, consistent with federal and state regulations. Your data will be identified by a number, not by your name. Only the research team will have access to the data, and it will be kept in a locked file cabinet in a locked room.

Approval Statement: The Institutional Review Board for the protection of human participants at USU has approved this research study. If you have any pertinent questions or concerns about your rights or a research-related injury, you may contact the IRB Administrator at (435) 797-0567 or email irb@usu.edu. If you have a concern or complaint about the research and you would like to contact someone other than the research team, you may contact the IRB Administrator to obtain information or to offer input.

Investigator Statement: "I certify that the research study has been explained to the individual, by me or my research staff, and that the individual understands the nature and purpose, the possible risks, and benefits associated with taking part in this research study. Any questions that have been raised have been answered."

_____ _____

Dr. Dale R. Wagner Date
Principal Investigator
(435) 797-8253

Appendix B: Informed Consent (Spanish)

UtahStateUniversity
Dept. of Health, Physical Ed., and Recreation
7000 Old Main Hill
Logan, UT 84322-7000
Voice: (435) 797-1497

Fecha Creada: 11 Noviembre 2008; Página 52 de 60
USU IRB Aprobación: 11/00/2008
Aprobación Termina: 11/00/2009
Palabra de Pase por IRB Administradora

La Carta de Información
La Saturación Arterial del Oxígeno
Como un Pronosticador de Día-Siguiente Enfermedad Aguda de Montaña (EAM) o Exito de Cumbre

Introducción/Propósito: Dr. Dale Wagner, el profesor agregado en el Departamento de Salud, Educación Física y Recreación de la Universidad del Estado de Utah (Utah State University) y sus ayudantes de investigación (Jon Knott y Jack Fry) estan haciendo un estudio para determinar si la medida arterial de la saturación de oxígeno (la cantidad de oxígeno en su sangre) de un oximeter de pulso de dedo puede predecir la Enfermedad Aguda de Montaña (EAM) o el éxito de alcanzar la cumbre del Pico de Orizaba. Anticipamos aproximadamente 50-100 participantes en este estudio de investigación, y le invitamos a usted a participar.

Procedimientos: Al llegar a la Cabaña de Piedra Grande, usted será preguntado algunas preguntas personales básicas (por ejemplo, su edad, estatura, y peso). Después de sentarse por 3-5 minutos, el ritmo de su corazón y la saturación de su oxígeno serán medidos con un oximeter de pulso de dedo. El oximeter es un aparato no invasor que se pone sobre el dedo índice, y es semejante a las máquinas que miden el ritmo del corazón comúnmente utilizado en centros comerciales y gimnasios. La medida de oximeter de pulso será repetida 3 veces: de 6 horas después de la primera medida, poco antes de partir por su tentativa de cumbre, y poco después de volver de su tentativa de cumbre a la cabaña. Usted también estará preguntado por si a tenido síntomas de EAM y si usted alcanzó la cumbre o no. El tiempo de su participación en este estudio será aproximadamente 10 minutos en total.

Riesgos: Los datos serán adquirido por medio de una entrevista con un cuestionario y del oximeter de pulso de dedo; los riesgos físicos de participación en este estudio son insignificantes. El único riesgo previsible es el de su privacidad. Sin embargo solamente el entrevistador sabrá su información personal. Solamente los investigadores asociados con este estudio tendrán acceso a los datos.

Beneficios: Por medio de esta investigación, esperamos obtener una comprensión mejorada de cuales factores son ventajosos al alcanzar las cumbres altas de las montañas y también determinar cuáles factores predisponen a alguien al EAM. Si podemos determinar por un número en un oximeter de pulso si alguien es probable o no a desarrollar EAM si la subida se continua, entonces esto sería un gran beneficio a la seguridad y el éxito de alpinistas en el futuro.

Explicación y La Oferta para Contestar Preguntas
Dr. Wagner o uno de los otros investigadores afiliados le han explicado este estudio a usted. A Usted se le ha dado la oportunidad y alentación al hacer preguntas. Si usted tiene otras preguntas o problemas relacionados a esta investigación, usted puede contactar al Dr. Wagner por teléfono a 435-797-8253 o por correo electrónico en dale.wagner@usu.edu.

Cuestas y Pagos Adicionales: No hay costo a usted para participar en este estudio. Usted no recibirá pago por su participación en este estudio.

Participación Voluntaria y El Derecho de Retirar
Su participación en esta investigación es enteramente voluntaria. Usted puede negarse a participar o retirarse en cualquier momento sin consecuencia.

Confidencialidad: Los registros de la investigación serán mantenidos confidencial, coherente con las regulaciones federales y del estado. Sus datos serán identificados por un número, no por su nombre. Sólo el equipo de investigación tendrá acceso a los datos, y serán mantenidos en un archivador cerrado con llave en un cuarto cerrado con llave.

Declaración de Aprobación: La Junta Institucional de la Revisión (IRB) para la protección de participantes humanos en la Universidad del Estado de Utah (Utah State University) ha aprobado a este estudio de investigación. Si usted tiene alguna pregunta pertinente o concierne acerca de sus derechos o una herida de esta investigación, usted puede contactar al Administrador de IRB en (435) 797-0567 o irb@usu.edu correo electrónico. Si usted tiene un concierne o la queja acerca de la investigación y usted querría contactar alguien de otra manera que el equipo de investigación, usted puede contactar al Administrador de IRB para obtener información o para ofrecer comentario.

Declaración de Investigador: "Certifico que el estudio de investigación le ha sido explicado al individuo, por mí o por mi personal investigador, y que el individuo comprende la naturaleza y el propósito, los riesgos posibles, y los beneficios asociados con tomar parte en este estudio de investigación. Todas las preguntas que se han hecho se han resolvidas."

_____ _____

Dr. Dale R. Wagner
Principal Investigador
435-797-8253

Fecha

Appendix C: Data Collection Form

Data Collection Form

_____
Participant #

Demographic Characteristics:

Age:_____ Gender:_____ Height:_____(in) Weight:_____(lb)

Acclimatization History:

Altitude of Residence:_____(ft) History of altitude illness:_____ (0 = no, 1 = yes)

Number of ascents above 3000 m (10,000') in 10 days prior to this ascent:_____

Approximate time above 3000 m (10,000') in 10 days prior to this ascent:_____
h:min

Highest altitude ever achieved prior to this ascent:_____(ft)

Hours/week of training in the month leading up to this ascent:_____

Pulse Oximetry & AMS Data:

Arrival at Piedra Grande Hut (4250 m): Date:_____ Time:_____

Pulse:_____ %SaO_2 :_____

Departure for summit attempt (4250 m): Date:_____ Time:_____

Pulse:_____ %SaO_2 :_____

Return from summit attempt (4250 m): Date:_____ Time:_____

Pulse:_____ %SaO_2 :_____

Ascent Data:

Summit:_____ (0 = no, 1 = yes) Use of Diamox/acetazolamide:_____(0 = no, 1 = yes)

Reason for not reaching summit:_____
(1 = AMS, 2 = weather, 3 = fatigue, 4 = partner failure, 5 = _____)

Arrival (30 min) at Piedra Grande

Lake Louise

Headache:

GI distress:

Fatigue:

Dizzy:

Sleep:

Return from summit attempt

Lake Louise

Headache:

GI distress:

Fatigue:

Dizzy:

Sleep:

Departure for summit attempt

Lake Louise

Headache:

GI distress:

Fatigue:

Dizzy:

Sleep:

Appendix D: Lake Louise AMS Self-assessment Scale (English)

Lake Louise AMS Self-Assessment

Headache:
0 None at all
1 A mild headache
2 Moderate headache
3 Severe headache, incapacitating

Gastrointestinal symptoms:
0 Good appetite
1 Poor appetite or nausea
2 Moderate nausea or vomiting
3 Severe, incapacitating nausea and vomiting

Fatigue and/or weakness:
0 Not tired or weak
1 Mild fatigue/weakness
2 Moderate fatigue/weakness
3 Severe fatigue/weakness, incapacitating

Dizziness/lightheadedness:
0 None
1 Mild
2 Moderate
3 Severe, incapacitating

Difficulty sleeping:
0 Slept as well as usual
1 Did not sleep as well as usual
2 Woke many times, poor night's sleep
3 Could not sleep at all

Appendix E: Lake Louise AMS Self-assessment Scale (Spanish)

Lake Louise AMS Self-Assessment

Dolor de cabeza:
0 Ninguno
1 Leve; un poco
2 Moderado
3 Grave; incapacitar

Síntomas gastrointestinales:
0 Apetito bueno
1 Apetito malo o náusea
2 Náusea moderado or vomitar
3 Grave; incapacitar con náusea o vomitar

Fatigue o la debilidad:
0 Ninguno
1 Leve; un poco
2 Moderado
3 Grave; incapacitar

Vértigos:
0 Ninguno
1 Leve; un poco
2 Moderado
3 Grave; incapacitar

La dificultad que duerme:
0 Normal; durmió usual
1 No durmió así como usual
2 Despertó muchos veces, durmió malo
3 No podría dormir en todo