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Effects of the GO2 Device on Blood Oxygen Saturation and Heart Rate at High Altitude: A Case Study

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Abstract- Lowered atmospheric pressures resulting from high altitudes (HA) reduce gas exchange in the alveoli and the respective capillaries. The heart and respiratory rates increase in an attempt to reestablish normal gas exchange during the hypoxic ventilatory response (HVR). Due to lowered atmospheric pressures of all gases, the HVR is often altered and inefficient, resulting in reduced blood oxygen saturation (SpO2). Low SpO2 is associated with HA illnesses like acute mountain sickness (AMS), High Altitude Cerebral Edema (HACE), and high altitude pulmonary edema (HAPE). The GO2 device is an ergogenic aide designed to create positive end expiratory pressure (PEEP), which is shown to increase SpO2. PURPOSE: The purpose of this study was to assess the effectiveness of the GO2 device as a means of inducing PEEP at HA in a case study that included a healthy, physically active adult that was not acclimatized to the a HA environment. METHODS: One healthy, fit male participant was taken to sea level (SL) for a period until acclimated. The participant's heart rate (HR) and SpO2 were measured at rest using a finger pulse oximeter (Innovo 430J/PE Deluxe Finger Pulse Oximeter with Plethysmograph and Perfusion Index) during 5, 4-minute intervals without the use of the GO2 device. Then the GO2 device was used and the 5 measurements were taken again in 4-minute intervals. Upon completion of the measure collected at SL, the participant traveled to a HA location (2573 meters elevation) and repeated the aforementioned measurements. RESULTS: At SL without the GO2 device, the mean HR was 71.0±2.5 beats per minute (bpm) and the SpO2 was 96.4%. At SL with the GO2 device the mean HR was 70.4±1.5 bpm and the SpO2 was 98.6%. At HA without the GO2 device, the mean HR was 88.4±1.7 bpm and the SpO2 was 91.8%. At HA with the GO2 device the mean HR was 80.4±1.1 bpm and the SpO2 was 98.0%. CONCLUSION: The GO2 device increased the SpO2 at both SL and HA by 2.2% and 6.8% respectively. HR was also affected by the GO2 device though virtually unchanged at SL, with a difference of 0.6 bpm, but was reduced by 8.0 bpm at HA.

Keywords- *Blood Oxygen Saturation, Heart Rate, GO2 Device, Acute Mountain Sickness, High Altitude Cerebral Edema, High Altitude Pulmonary Edema*

I. INTRODUCTION

Throughout history humans have progressively endeavored to accomplish the previously assumed physically impossible. Stretching the limits of speed, agility, endurance, and strength. Looking at the volcano that towers over the surrounding valley instills a desire to see the world from its perspective. Summiting iconic mountains throughout the world has, and likely will always be, a goal for many individuals wanting to push the limits of their physical, psychological, and physiological capabilities. However, despite physical training the human body imposes protective limitations that seem to be specifically designed to keep us from accomplishing some of these goals. High altitudes (HA) come with decreased partial pressures of oxygen that can cause debilitating and sometimes deadly illnesses [29]. For decades humans have explored ways to minimize the adverse effects of HA and thereby increase their physical ability to accomplish tasks. The development of ergogenic aids of all varieties have progressively improved endurance, strength, power, functionality, and health. Biomechanical aids have made it possible for people to run, that previously couldn't stand [32], or climb cliffs that were previously deemed unclimbable [25]. Physiological aids have helped people free dive for 20 minutes, when previously incapable of holding their breath for 30 seconds [3], and the use of pharmacological aids have made summiting high elevations at a quicker pace attainable for many whose physiology formerly struggled to acclimatize [35].

One ergogenic aid, positive end expiratory pressure (PEEP) has been documented in its ability to improve alveolar gas exchange [10, 21, 16]. The use of PEEP for this purpose is not new. In 1938 PEEP was described for its use in conjunction with mechanical ventilation [1]. However, the use of PEEP in healthy adults has relatively little research and even less regarding its use at HA as an ergogenic aid in the prevention or reduction of the adverse effects caused by low partial pressures of oxygen. The effectiveness of positive airway pressure in people suffering from cardiopulmonary illnesses has been studied for over a century. In 1970, 12 male subjects with chronic airway obstruction were selected and examined under exercise and resting conditions with and without the use of a technique called pursed lip breathing (PLB), which is a way to induce PEEP without the assistance

of a device. Results showed improvements in both arterial partial pressure of CO2 and O2 at rest, in the patients using PLB only [20]. In more recent decades, the potential benefits of positive airway pressure in healthy individuals has been examined under some conditions. Positive airway pressure can be created by either pressure breathing, such as the PLB, or using a device designed to create resistance during the expiratory phase. Typically, for healthy individuals, the emphasis for this additional pressure is on the expiratory phase due to the potential effect the additional pressure has in maintaining inflated alveoli for a longer period and therefore improving gas exchange [10]. PEEP also tends to slow and deepen the breathing, which potentially allows the lungs and heart to function more efficiently.

Atmospheric pressure is reduced as altitude increases, though the concentration of oxygen remains relatively constant at all altitudes. As the barometric pressure is lowered, so too is the body's ability to diffuse O2 into the blood [11]. Noticeable differences in physiology for most adults begin at what is considered HA (1,500-3,500 meters). The body begins to increase the rate and depth of ventilation in order to compensate for the lowered partial pressure of O2 in a process called the hypoxic ventilatory response (HVR), which simultaneously triggers an increasing heart rate to improve gas exchange [29]. The body's ability to utilize O2 in aerobic performance is decreased significantly, even for endurance trained athletes as shown by Wehrlin & Hallén [34] who recorded a 6% linear decrease in maximal oxygen uptake (VO2max) for every 1000 meters of elevation gain up to 2800 meters. More importantly than the apparent decrease in aerobic performance is the effect on health. If the HVR is impaired in some way, or if the alveoli collapse or are drastically reduced in size, gas exchange will be hindered and hypoxemia may lead to acute mountain sickness (AMS) or more fatal conditions such as high-altitude cerebral edema (HACE) and high-altitude pulmonary edema (HAPE) [29]. The adverse effects and illnesses caused by the lowered alveolar pressures can be partially reduced by increasing expiratory air pressure to maintain alveolar expansion and possibly prevent collapse, which improves gas exchange [10].

Initially the use of positive expiratory pressure in healthy individuals was performed by PLB and has been used by mountaineers for many decades. An increase in the amount of O2 absorbed from a given amount of air inhaled has been attributed to PLB [20]. In more recent years, interest has turned toward the use of ergogenic devices designed to induce PEEP. PEEP devices have been shown to improve the saturation of oxygen in the blood, decrease the symptoms of AMS and lead to increased ability to perform work at HA during the acute adaptation phase [21, 15]. The challenge with these devices is that they are sometimes awkward or uncomfortable in design or may struggle to function in the cold conditions often found at HA. The recently developed GO2 device used in this study is small (slightly larger than a football mouth guard), easy to use, and may serve better than its PEEP predecessors.

Hence the purpose of this study was to test the effectiveness of the GO2 device as a means of inducing PEEP at HA in a case study including one healthy, physically active adult. This study measured the effects the GO2 device had on blood oxygen saturation (SpO2) and heart rate (HR) as assessed with a finger pulse oximeter.

II. METHODS

A. Participant

Due to restrictions in place intended to reduce the risk of infection of COVID 19, this study was designed to be a case study with the single participant being a healthy, physically active, male, adult 40 years of age, body mass 77.3 kilograms, and a height of 172.7 centimeters. It should also be noted that the individual is a part-time mountain guide who normally lives at an elevation of 1450 meters. For the purposes of this study, the participant moved to SL for a period of 30 days to de-acclimate before taking measurements at SL.

Informed consent from the participating individual was obtained and approved by the Southern Utah University Institutional Review Board. The participant was informed of the procedures, any risks or discomforts that may be involved with the procedure, and that they may withdraw from the procedure at any time with no penalty.

B. Instrumentation

This experiment was conducted using the GO2 device, which is designed to be a simple mouthpiece, much like a typical sport mouth guard that you hold in your mouth Fig. 1. At the front of the device there is a unidirectional valve created by a rubber flap with tiny bands to create the resistance on the exhale. The resistance opposing the exhale is referred to as PEEP and the resistance is calibrated to a pressure of 4.5 cm H2O. The inhale offers no resistance. The ability of this device to create PEEP has been validated by Lambert [14].

Figure 1. GO2 Device

The SpO2 and HR were tested using an FDA approved finger pulse oximeter (Innovo 430J/PE Deluxe Finger Pulse Oximeter with Plethysmograph and Perfusion Index) Fig. 2. The Innovo is a FDA approved device [9]. Similar pulse oximeters have demonstrated acceptable levels of reliability and validity for the assessment of HR [17].

Figure 2. Innovo 430J/PE Deluxe Finger Pulse Oximeter with Plethysmograph and Perfusion Index

C. Procedures

In the 10 days leading up to the first phase of testing, the participant was trained in the use of the GO2 device and practiced using it at least 5 separate times (4 times is the suggested number of times it takes to learn to use the device efficiently, according to the device manufacturers). The first session was for 10 minutes during mild cardiorespiratory exercise (going for a walk on the beach). The second session was for the same amount of time. The third session was for 15 minutes, with moderate-intensity cardiorespiratory exercise (going for a jog). The fourth and fifth sessions using the device were for periods of 20 minutes each at a moderate exercise intensity. This process was to ensure that the participant was comfortable with the use of the device to avoid any issues like a learning effect during the assessment potion of the investigation.

The first phase of the experiment established a baseline and was conducted at or close to SL in Newport Beach California. The participant was tested at rest with the last intake of food being no closer than 3 hours prior to testing and no caffeine or other substances that may induce an abnormal heart or respiratory rate were ingested in the previous 5 days. The participant had his SpO2 and HR recorded 5 times within 20 minutes using the finger pulse oximeter.

Following those initial measurements, the participant began using the GO2 device for 1 minute before taking the first measurements of SpO2 and HR. Four more measurements were taken once every 4 minutes after. Nespoulet et al. [21] found it took 3 minutes for the PEEP device used in their study to have full effect on the physiology. Bilo et al. [5], found it took 5 minutes for the effects of slow deep breathing to be fully realized. This study used 4-minute intervals with 5 total measurements to establish time of onset of any effects and peak effects.

Travel to the next phase occurred the same day and took approximately 2 hours to drive to the Onyx Pass (2573 meters) near Big Bear Lake, California. After 4 hours of arrival at high elevation, the participant rated himself according to the Lake Louise Score for Acute Mountain Sickness (LLS) questionnaire provided to him [24] with no symptoms. The participant then began measurements of SpO2 and HR, which were then recorded in the same manner described in phase 1 without the GO2 device. As before, the measurements were taken 4 more times, once every 4 minutes for 5 total measurements. The participant then used the GO2 device for 1 minute before the assessments of SpO2 and HR were taken. Once again, the measurements were taken 4 more times, once every 4 minutes for 5 total measurements. Immediately after the final numbers were recorded the participant calculated his own LLS and was asymptomatic. The LLS is not intended to be part of the experimental procedure but was used only as a safety precaution.

III. RESULTS

The heart rate (HR) and blood oxygen saturation (SpO2) both with and without the use of the GO2 device at SL and HA are represented in Tables 1 and 2. The results are displayed graphically for comparison in Figures 3 and 4 for the SL and HA measurements.

At SL without the GO2 device, the mean HR was 71.0±2.5 beats per minute (bpm) and the SpO2 was 96.4%. At SL with the GO2 device the mean HR was 70.4±1.5 bpm and the SpO2 was 98.6%. At HA without the GO2 device, the mean HR was 88.4±1.7 bpm and the SpO2 was 91.8%. At HA with the GO2 device the mean HR was 80.4±1.1 bpm and the SpO2 was 98.0%.

The GO2 device increased the SpO2 at both SL and HA by 2.2% and 6.8% respectively. HR was also affected by the GO2 device though virtually unchanged at SL, with a difference of 0.6 bpm, but was reduced by 8.0 bpm at HA.

TABLE I. HEART RATE AND SPO2 MEASUREMENTS AT SEA LEVEL

	HR (bpm) w/o GO2 device	HR (bpm) With GO2 device	SpO2 (%) w/o GO2 device	SpO2 (%0 With GO2 device
0 min	72	71	97	98
4 min	71	71	97	99
8 min	74	70	96	98
12 min	71	68	96	99
16 min	67	72	96	99
Mean	71.0	70.4	96.4	98.6
SD	2.5	1.5	0.5	0.5

Heart rate (HR) and blood oxygen saturation (SpO2) without and with the use of the GO2 device at sea level. Measurements taken in 4-minute intervals.

TABLE II. HEART RATE AND SPO2 MEASUREMENTS AT ELEVATION (2573 METERS)

	HR (bpm) w/o GO2 device	HR (bpm) With GO2 device	SpO2 (%) w/o GO2 device	SpO2 (%0 With GO2 device
0 min	88	82	92	98
4 min	86	80	91	98
8 min	90	81	92	98
12 min	90	79	92	98
16 min	88	80	92	98
Mean	88.4	80.4	91.8	98.0
SD	1.7	1.1	0.4	0.0

Heart rate (HR) and blood oxygen saturation (SpO2) without and with the use of the GO2 device at 2573 meters above sea level. Measurements taken in 4-minute intervals

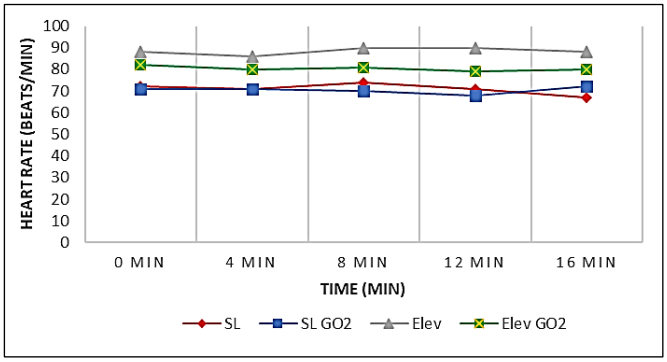

Figure 3. Comparison of resting heart rate under different conditions (SL- Heart rate at sea level; SL GO2- Heart rate at sea level while using the GO2 device; Elev- Heart rate at 2573 meters above sea level; Elev GO2- Heart rate at 2573 meters above sea level while using the GO2 device).

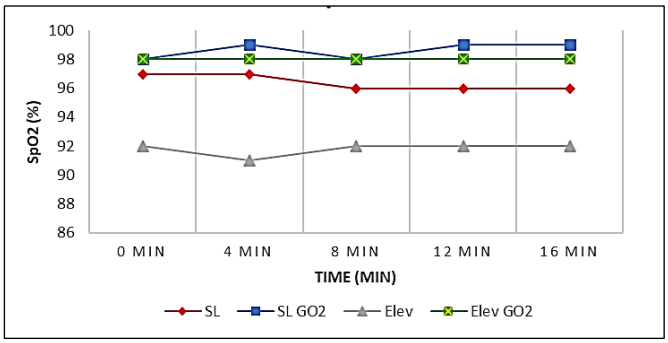

Figure 4. Comparison of blood oxygen saturation (SpO2) under different conditions (SL- SpO2 at sea level; SL GO2- SpO2 at sea level while using the GO2 device; Elev- SpO2 at 2573 meters above sea level; Elev GO2- SpO2 at 2573 meters above sea level while using the GO2 device).

IV. DISCUSSION

When an individual commits to climbing an iconic high mountain, there are considerable preparations required. Physical and technical training, along with a substantial financial commitment are often only secondary to the emotional investment. When an individual places that much time, money, and effort into a summit, only to have their own physiology keep them from accomplishing their goal, it is depressing to say the least. The greatest and most obvious benefits of a device like the GO2 device appears to be the capability of increasing SpO2, increase work capacity and decrease HR at HA among healthy, physically active adults. As such, the GO2 device may also be beneficial with respect to the prevention of illnesses such as AMS, HAPE, and HACE. These benefits will increase the likelihood of an individual accomplishing their goal of reaching the summit as well as potentially saving lives. Mountain guides often have multiple clients they are guiding up a mountain at the same time. If one individual suffers from symptoms caused by decreased SpO2 or has a decreased work capacity, it may lead to added danger for the guide or other clients as a rope team is dependent upon each other's health, coherence, and focus for safety along the often-treacherous routes over glacial crevasses.

Mountaineers are not the only individuals who could benefit from a device like the GO2 device. Athlete's often encounter higher altitudes than they are accustomed to when they travel for competitions. Lack of time to acclimatize can often be detrimental to the athlete's performance. A device capable of reducing the negative acute effects of HA would likely improve not only health, but performance in the competition as well.

Most of the positive effects that come from PEEP are dependent upon its ability to increase SpO2. Though the use of PEEP devices on healthy adults at HA has not been extensively studied, the majority of the studies using a PEEP device or PLB have shown an increase in SpO2 under either simulated or real HA conditions among healthy adults [5, 15, 21, 28], which are consistent with the findings of the current study. However, in contrast, no change in SpO2 due to 5 cm H2O PEEP at HA was found by Savourey et al. [26], nor in a subsequent study by Savourey et al. [27]. This difference may have been due to the specific PEEP device that was used. Launay et al. [15] explained that these early PEEP devices often created condensed water vapor creating discomfort and sometimes freezing (causing the valve to lock up). The GO2 device has such a simple construction that even though the conditions in this study were not freezing, these issues are not likely to occur and indeed there were no problems regarding its use and functionality in this experiment. Other possible reasons for differences in SpO2 results between the aforementioned studies and ours may be due to the ability of the participants to use the PEEP device properly, or more likely, the inaccuracy and/or improper use of the finger pulse oximeter. The papers by Savourey et al. [26, 27] are 2 decades old, and technology has improved considerably since that time. Incorrect readings may be due to poor positioning of the device, poor perfusion in the digit (ex. cold finger), skin pigmentation, patient movement, lighting around the device, or nail varnish. Modern devices, including the Innovo 430J/PE Deluxe Finger Pulse Oximeter with Plethysmograph and Perfusion Index, which was used in the current study, are often equipped with sensors allowing the user to know if the device is functioning or positioned properly, making it difficult for the user to get faulty readings.

Performance of any sort at HA, whether for a travelling athlete or mountaineer, has always been hindered by the effects of low oxygen pressures and the potential illnesses associated with those effects. Acclimation is not uniform for every individual. The acute physiological adaptations necessary for tolerance may be relatively quick for one individual and take days for another. The adverse effects caused by low atmospheric pressures can decrease physical abilities, create discomfort, and potentially cause syndromes like AMS, HACE, and HAPE. Of course, proper training, frequent visits to HA, and pharmaceuticals have all been shown to be effective methods in preventing or reducing many of these negative effects. The use of PEEP, however, as an ergogenic aid in HA situations does show promise. This study showed that using the GO2 device at both SL and HA, effectively increased the SpO2 from 96.4% to 98.6% at SL and 91.8% to 98.0% at HA providing evidence that it does create PEEP sufficiently to improve blood oxygen saturation in both conditions. This increase in SpO2 has the potential to decrease or eliminate the negative acute effects of altitude.

A. Physiological effects of high altitude

Traveling to HA causes the body to adjust to a lower partial pressure of oxygen in the atmosphere. Normal barometric pressure at SL is 760 mm Hg with a partial pressure of O2 around 159 mm Hg. The partial pressure of O2 in the alveoli of the lungs decreases to around 104mm Hg due to effects caused by displacement from CO2, dead space, and water vapor. By the time the O2 reaches the blood, the O2 saturation in the hemoglobin is around 95-98% [29]. The partial pressure of CO2 may be offset by hyperventilation, which could increase the partial pressure of O2 in the lungs. According to Taylor [29], as altitude increases and the barometric pressure decreases, so too will the subsequent partial pressures of O2 in the different components mentioned. The pressure of oxygen in the alveolar sacs of the lungs may reduce the surface area used for gas exchange, change the pressure gradient between the alveoli and blood, and may cause atelectasis. This results in reduced oxygenation of the blood as shown by a lower SpO2 from pulse oximetry. Healthy mountaineers or other athletes arriving at higher altitudes than they are accustomed to, will likely manifest a decreased SpO2 as an acute response. The heart will likely compensate by increasing cardiac output, stroke volume, and rate [31]. In addition, the body will increase the respiratory rate in an attempt to compensate for the lack of oxygen in the blood in order to sufficiently oxygenate the tissues throughout the body [29]. The current study demonstrated that the GO2 device effectively maintained or even reduced the cardiac effort, while still increasing the SpO2. This could potentially increase comfort and improve acclimation. Further, since the heart isn't working as hard there would likely be an associated increase in work capacity.

Normally the ventilatory response is initiated by increased CO2 in the blood. The increased partial pressure of CO2 in the blood (SpCO2) and decreased blood pH caused by the H+ ions, communicate with chemoreceptors in the medulla, initiating respiration to decrease the SpCO2 and increase blood pH. The subsequent reduction in SpCO2, along with the increased pH levels, communicate to the chemoreceptors of the medulla to decrease ventilation [29]. However, this normal ventilatory response is altered with altitude induced hypoxia since the partial pressure of CO2 is reduced as well as O2 partial pressure. This acute HVR varies in its effectiveness from individual to individual and is therefore unpredictable [29]. Indeed, the act of rhythmically slowing and deepening the breath to a rate of 6 breaths per minute, improved ventilation and SpO2 (80.2±7.7% to 89.5±8.2%) at acute HA (4559 m) conditions, in healthy adults [5]. Conversely, Nespoulet et al. [21] placed 16 healthy males under normobaric hypoxic conditions simulating 4300 meters altitude and noted no SpO2 changes as a result of PLB. However, they did see a decreased HR with PLB in comparison to the free breathing tests. The development of hypoxemia and possibly AMS may be due to a failure to increase the HVR [29]. The use of PLB and PEEP devices, including the GO2 device, require a slow, deep, and controlled breath. The valve on the GO2 device that activates on the exhale, requires a certain strength, depth, and rate of breath to initiate it. This effectively allows the individual to consciously slow and deepen their respiration, thus negating the potential individual differences in HVR at HA.

Taylor [29] explained that decreased alveolar partial pressure of oxygen as a response to HA is only slightly higher than the arterial partial pressure of oxygen at rest. As exercise is initiated, this difference is widened due to shortened transit time of the capillary blood over the alveoli, increased cardiac output, and greater venous oxygen desaturation. Under this altitude induced hypoxic condition, exercise exacerbates arterial oxygen desaturation. As a result, lowered SpO2 decreases endurance performance [29]. As mentioned before, the body's acute response to HA is to attempt to increase SpO2. Interestingly however, the prolonged adaptive response to lowered SpO2 may be to lower O2 utilization in the tissues instead [11]. The effectiveness and efficiency of the body's acute response will enhance an athlete/mountaineer's capability of performing at HA as well as reduce the risk for altitude related illnesses such as AMS [29]. Resting sufficiently and allowing the body time to adapt, is an option, but another option is to use ergogenic aids, like the GO2 device, designed to help increase SpO2.

B. Illnesses and concerns at high altitudes

Decreased physical capabilities are certainly a concern for the mountaineer/athlete performing at HA, but the detrimental effects caused by HA syndromes is of the greatest concern. During the first 2 to 3 days at HA (especially >2500 meters) an individual is at the highest risk of acute mountain sickness [4, 29]. According to the Lake Louise Acute Mountain Sickness Score revised in 2018, the symptoms of AMS always include a headache, but additionally include gastrointestinal issues (including poor appetite, nausea, and vomiting), and dizziness/lightheadedness [24]. Other authors have added peripheral edema, ataxia, change in mental status, and rales [29]. The challenge in diagnosing this syndrome is that it is largely subjective and therefore dependent on the patient's assessment of their own symptoms. Berger et al. [4] suggests that this challenge in diagnosing may be due, in part to the

idea that AMS should be divided into time courses associated with specific pathophysiologic factors. No matter what the type of AMS, it is usually self-correcting given time at a certain altitude and if necessary, decent will typically fix any issues. However, it is believed that AMS is a precursor to more deadly conditions like HACE or in some cases the subclinical pulmonary edema may lead to HAPE [29].

HACE is typically preceded by AMS symptoms but does not have to be. It is fairly rare and is less likely to occur at altitudes lower the 4000 meters, though it has been identified in individuals at altitudes around 2500 meters [12, 29]. The pathophysiological cause of HACE is not well understood and there is no current consensus as to its nature. One thought is that hypoxia-induced cerebral vasodilation over perfuses the microvascular cerebral beds. This creates hypertension and increases capillary pressures, leading to leakage. This leakage can increase pressure on the brain [12]. The onset of HACE usually occurs after 2 or 3 days of manifesting symptoms of AMS. HACE is potentially fatal and transportation to lower altitude should begin immediately [12, 29].

Another potentially deadly complication at HA is HAPE. As with HACE, HAPE is often preceded by symptoms of AMS for the 2 to 4 days prior to its onset. Much like the other altitude induced illnesses, HAPE most frequently occurs at altitudes >2500 m and is recognized as the most common altitude related cause of death [22, 29]. Though the actual cause of HAPE is largely unknown, Paralikar [22] describes the pathophysiological process stemming from inhomogeneous pulmonary vasoconstriction. In locations with low arterial vasoconstriction, this pulmonary vasoconstriction of the capillaries combined with hypoxic constriction of the pulmonary veins cause resistance downstream of the fluid filtration region, leading to hypertension. In addition to these hydrostatic mechanisms, Paralikar [22] adds the diminished capacity for reabsorption of alveolar fluids to the underlying mechanisms leading to HAPE. The main priority for individuals suffering from signs and symptoms of HAPE is a decrease in altitude immediately.

In the case of the travelling athlete, reduction in altitude may not always be an option. The real goal should be to avoid things going so far as to induce HACE or HAPE. A solution as simple as a PEEP device could be ideal. HA inevitably reduces the SpO2, the individual's physiologic response determines to what degree and how long it takes to adapt. Abnormally low SpO2 levels at HA have been linked in several studies to an individual's likelihood of manifesting symptoms of HA illnesses [2, 4, 28, 29]. Burtscher et al. [6] tested SpO2 levels, at altitudes of 2000 to 6000 meters, of 150 mountaineers, 63 of whom acknowledged having AMS in previous exposures to HA. The results showed a low ventilatory response and a lower SpO2 at all altitudes, in the individuals more susceptible to AMS. Karinen et al. [13] studied 8 expeditions with 83 ascents in total. They measured moderate exercise SpO2 (walking 50 meters, at a target HR of 150 bpm) and resting SpO2 during a 2400 to 5300-meter ascent. Results showed that both resting and exercise SpO2 levels were lower for the individuals who ended up with AMS, before and after the onset of symptoms. Clarenbach et al. [8] found that after ascending from 1130 to 4559 meters, 8 subjects were diagnosed with HAPE and had shown lower SpO2 levels than the control subjects from their time of arrival at 4559 meters until the end of the study.

The benefits that would come from eliminating or at least reducing the possibility of encountering any of these adverse effects are obvious. If an ergogenic aid like the GO2 device can increase SpO2 in the acute phase of adaptation to HA, it is likely that the symptoms of AMS would reduce in severity or be eliminated altogether. Since AMS is a common precursor to the more severe HACE and HAPE, their prevalence could potentially decrease as well. Additionally, the ability to perform work at HA would increase during this acute phase increasing the physical safety of all members of a rope team in mountaineering.

Pulse oximetry is the preferred method for testing SpO2 at altitude for its convenience and relative accuracy. According to the American Thoracic Society, most pulse oximeters are accurate to about ±2% [22]. There are several pitfalls and warnings regarding the use of pulse oximeters for predicting AMS or other illnesses. First, oximeters become even less accurate when SpO2 is less than 80%. Second, specific expected saturations for a given altitude may not be consistent. Third, ambient light, cold skin, and dyshemoglobinemias can all cause bad readings [18]. As long as the tester knows what to look for and proper experimental practices are followed, results can be relatively reliable and valid, as is shown in numerous studies including the ones mentioned above and even under severe physical exertion like ice climbing at HA [30].

C. Treatments PEEP/G02

One of the challenges with PLB is learning to do it properly to create the proper pressure in the lungs. Using a device to create PEEP is not only simpler to learn, but it has the potential advantage of creating a more specific and measured pressure and in turn, enhance gas exchange. In an early study of the use of PEEP on healthy individuals at HA (4400 meters), Schoene et al. [28] showed an increase in SpO2 in both resting and exercise conditions at levels of 5 and 10 cm H2O PEEP, and showed an increased HR at rest and exercise in the higher level of PEEP at 10 cm H2O. They also demonstrated its usefulness in increasing SpO2 in individuals suffering from HAPE at both 5 and 10 cm H2O of PEEP, though no significant change was seen in HR with these individuals. The authors attribute the increased SpO2 to the increased ventilation and modified breathing patterns. While not a variable that was focused on in the current study, the slowed rate and increased depth of respiration were also noted in the one healthy adult male at rest, while using 4.5 cm H2O set by the manufacturers of the GO2 device. Further, Savourney et al. [27] studied ventilation and breathing patterns in 22 healthy male subjects at SL under resting and exercise conditions using 0 and 5 cm H2O PEEP. The authors observed increases in tidal volume, inspiratory duration, and expiratory duration, along with a decreased breathing frequency under 5 cm H2O PEEP in both resting and exercise conditions.

As already established, healthy adults travelling from low to high altitudes have a distinct change in SpO2 and HR [14]. Measurements of SpO2, HR, and an LLS assessment for AMS were taken during two ascents of Mont Blanc (4810 meters). During one ascent 4 climbers used 5 cm H2O PEEP and the other 4 without. For the second ascent the groups were switched. The results showed an overall higher SpO2 for the 5 cm H2O PEEP group. The HR was unaffected, but the LLS was significantly lower for the PEEP group [15]. These results are promising for the use of PEEP at HA as a means of reducing the likelihood of experiencing AMS. It is likely that the lack of change in HR is due to the exertion put forth by the participants in climbing. Nespoulet et al. [21] found the use of PEEP devices set at 5 and 10 cm H2O increased SpO2 and decreased HR in 16 healthy adults under normobaric conditions simulating 4300 meters in altitude. Additionally, a field study of 9 healthy adults conducted at 4350 meters 10 cm H2O PEEP increased SpO2 significantly. These studies all support the hypothesis that the GO2 device could be successful in reducing or preventing HA syndromes such as AMS, HACE, and HAPE due to its ability to increase SpO2.

The main complaints and disadvantages to using a PEEP device in the past have been the inconvenience in transport or use, the bulkiness, and the tendency of certain devices to freeze up at higher, colder altitudes. The GO2 device is small, compact, easy to use and should function well no matter what the environmental conditions are. There has only been one study published on the effects of the GO2 device. The tests conducted by Lambert [14] were performed at SL. The first was under graded exercise conditions progressively increasing workloads until the self-selected cadence could no longer be maintained at 10 rpms. The other condition was under a steady state exercise performed until exhaustion. During moderate intensity exercise the HR was reduced but was elevated during recovery from the more intense paces. The GO2 device only influenced SpO2 under intense exercise. The results of the current study found an increase in SpO2 in resting conditions at both SL and HA. However, the results from Lambert's [14] report also show promise in the potential for the GO2 device to be used for improving the human body's performance and as an adaptive response to HA.

The greatest limitation of this study was sample size being a single 40-year-old male. There are many paths for future studies to focus on. Replicating the current study with a large participant pool randomly selected appears warranted. There is also a possibility that 30 days is not sufficient time to fully acclimate to SL since no studies were found suggesting any definitive length of time a body takes to de-acclimate. Thirty days at SL was selected because most evidence anecdotally suggests that performance begins to decrease at SL after about 10 to 14 days after returning from HA [7]. This may have skewed the results of the current study in that the participant may not have fully acclimated to SL. As a remedy, future studies could use participants that live at SL year-round. Another direction that would be interesting to pursue would be to focus on exercised conditions at HA rather than resting. The current evidence supports the use of the GO2 device as a method of increasing SpO2 at both SL and HA for male adults at rest.

V. CONCLUSIONS

Athletes travelling to higher altitudes than they are accustomed to, as well as mountaineers need to be aware of the adverse effects caused by the lower partial pressures of O2. The reduced performance capacity caused by low SpO2 and an impaired hypoxic ventilatory response can certainly be frustrating but can be partially countered by proper training for HA. The biggest concern, however, is the potential for illnesses that are induced by an impaired HVR and subsequently lowered SpO2. AMS can cause discomfort and weakness, but more importantly, if it is not resolved it can lead to HAPE or HACE. Acetazolamide can be taken to reduce the effects of AMS and both acetazolamide and dexamethasone are frequently taken to treat AMS and the more serious HAPE and HACE [29] but they do require a prescription. PLB has been taught for decades to mountaineers as a way to reduce the effects of AMS and improve gas exchange [21]. More recently, studies have focused on the use of PEEP to maintain open alveoli and possibly reduce atelectasis to improve gas exchange and increase SpO2 [10]. The use of PEEP at HA in healthy adults is by no means conclusive, but the majority of studies suggest physiological benefits that could prevent altitude illnesses and potentially increase performance at HA. Indeed, this study demonstrated that a healthy fit adult male can increase SpO2 effectively and quickly by using the GO2 device, while maintaining or even decreasing the HR at rest. This potentially increases the cardiorespiratory system's capacity to perform work at both SL and HA. The problem with PEEP devices has been the fact that there is little research on their use at altitude and their inconvenience in design and function. The GO2 device holds promise in providing the benefits of PEEP while being simple in function and use, as well as being compact in design.

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