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Non-invasive Positive Pressure Ventilation during Sleep at 3800m: relationship to Acute Mountain Sickness and sleeping oxyhemoglobin saturation

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Abstract

Background and objectives—Ascent to high altitude results in hypobaric hypoxia and some individuals will develop Acute Mountain Sickness, which has been shown to be associated with low oxyhemoglobin saturation during sleep. Previous research has shown that positive end-expiratory pressure by use of expiratory valves in a face mask while awake, results in a reduction in AMS symptoms and higher oxyhemoglobin saturation. We aimed to test whether pressure ventilation during sleep would prevent AMS by keeping oxyhaemoglobin higher during sleep.

Methods—We compared sleeping oxyhemoglobin saturation and the incidence and severity of Acute Mountain Sickness in seven subjects sleeping for two consecutive nights at 3800m above sea level using either non-invasive positive pressure ventilation that delivered positive inspiratory and expiratory airway pressure via a face mask, or sleeping without assisted ventilation. The presence and severity of Acute Mountain Sickness was assessed by administration of the Lake Louise questionnaire.

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Role of each author: Pamela Johnson. This work comprised a Chapter of her PhD “Sleep and Breathing at High Altitude” which was awarded in August 2008. Conduct and analyse all sleep studies; apply non invasive ventilation to each subject at the Barcroft Lab. on White Mountain. Write up the research as a chapter of PhD and as an original article for publication in a peer reviewed journal. Daniel Popa. Recruitment of all subjects. Participation in all the procedures carried out in San Diego and Barcroft Lab. Perform and analyse PETCO₂ and SaO₂ sampling on each subject morning and evening. Presentation of part of the findings at a scientific meeting in USA.

Kim G Prisk. Supervision of Dan Popa. Facilitation and communication between University of Sydney and University of California, San Diego participants.

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Results—We found significant increases in the mean and minimum sleeping oxyhemoglobin saturation and decreases in AMS symptoms in subjects who used positive pressure ventilation during sleep. Mean and minimum sleeping SaO₂ was lower in subjects who developed AMS after the night spent without positive pressure ventilation.

Conclusion—The use of positive pressure ventilation during sleep at 3800m significantly increased the sleeping oxygen saturation; we suggest that the marked reduction in symptoms of AMS is due to this higher sleeping SaO₂. We agree with the findings from previous studies that the development of AMS is associated with a lower sleeping oxygen saturation.

Keywords

hypobaric hypoxia; acute mountain sickness; positive pressure ventilation; Lake Louise Score; sleeping oxyhemoglobin saturation

Introduction

Acute mountain sickness (AMS) can affect people who travel from near sea level to altitudes above 3000m but can affect some people at altitudes as low as 2000m.

Hypoxia is believed to be the cause of AMS although the pathophysiologic mechanisms are poorly understood (Ward et al, 2000; Bartsch et al, 2004).

Pressure support via a face mask improves alveolar minute ventilation on inspiration and, by maintaining expiratory pressure, prevents upper airway and alveolar collapse during expiration. Expiratory positive pressure or PEEP may reduce shunting recruiting atelectatic lung. The application of positive end-expiratory pressure (PEEP) via a face mask with an expiratory valve has been shown to increase the oxyhemoglobin saturation and decrease AMS symptoms in awake subjects at high altitude (Launay 2004; Oelz et al 1983; Savourey et al 1999; Schoene et al 1985). The mechanism by which PEEP improves oxyhaemoglobin saturation is thought to be improved ventilation caused by recruitment of microatelectatic alveoli and breathing pattern changes (Wayne 1976). However, PEEP has not been tested during sleep at high altitude and the mechanism responsible for improvement in AMS symptoms is unknown. The use of continuous positive airway pressure (CPAP), after ascent to 3205m on Mount Cook in New Zealand (Davis et al 1999), has been shown to improve oxyhemoglobin saturation and improve symptoms of high altitude pulmonary edema (HAPE).

The combination of inspiratory and expiratory pressures used in positive pressure ventilation, because of its capacity to augment ventilation, would theoretically have a more beneficial effect on oxyhaemoglobin saturation than PEEP alone and thus reduce AMS.

There appears to be a central role of hypoxemia causing AMS (Bartsch et al, 1990; Sutton et al, 1979; Roach et al, 1998). The low barometric pressure at high altitude results in low inspired P_{O2} and hence low alveolar P_{O2} leading to oxygen deprivation. Oxyhemoglobin saturation is known to be lower during sleep at high altitude (West et al 1986; Masuyama et al, 1989) and the development of AMS has been shown in some studies to be associated

with low mean sleeping oxyhemoglobin saturation (Burgess et al., 2004. Erba et al., 2004) while others have not found this relationship (Eichenberger et al 1996., Nickol et al 2006).

Research has shown that oxygen enrichment of room air by use of concentrators is a very useful method of eliminating AMS and improving the general well being of people working at high altitude (West, 1995) and improves well-being the following day and sleep quality, both subjectively and objectively, in people sleeping at 3800m with room oxygen enriched to 24% (Luks et al, 1998); this comes as no surprise as AMS is due entirely to the hypoxia of high altitude.

Hypothesis and Aims

There have been no studies using either PEEP, CPAP or other positive airway pressure devices during sleep at high altitude to improve sleeping oxyhemoglobin saturation. Acute Mountain Sickness has been shown to be associated with a low mean SaO₂ during sleep. The purpose of this study was therefore to determine if the use of positive airway pressure via a face mask during sleep at an altitude of 3800m would result in a higher mean SaO₂ during sleep and reduce the incidence and symptoms of AMS.

Materials and Methods

Subjects and study design

Seven healthy subjects (ages 22-24, four male and three female) participated in the study. Each subject gave informed consent and the protocol was approved by the University of California, San Diego Human Research Protection Program. None had traveled to high altitude in the previous six months.

Sleep studies were performed at sea level in San Diego to exclude the presence of sleep-disordered breathing in these young, healthy subjects, and on each of the two nights spent at the White Mountain Research Station, California which is 3800 metres above sea level. The subjects did not ascend to higher altitude nor descend to lower altitude during the two day sojourn at 3800m.

The subjects arrived by car at 3800m in late afternoon before the first sleep study. The last experiments (morning end tidal CO₂ and SaO₂) were conducted no more than 40 hours after arrival at high altitude.

The subjects slept one night using positive pressure ventilation and one night without; the order was randomised. We used a variable positive airway pressure device (VPAP® 111 ST A, ResMed, San Diego, California) that delivers positive pressure during inspiration which falls to a lower pressure during expiration. Thus, positive pressure is delivered throughout the breathing cycle: inspiratory pressure providing assistance to inspiration and expiratory pressure acting to maintain a distending pressure during the expiratory phase of ventilation.

The VPAP machine was connected via two metres of tubing to a firmly fitted full face mask (ResMed Ultra Mirage or Respironics Comfort Full) with a constant flow expiratory port. The VPAP machine was used on spontaneous mode i.e. the subject triggered the inspiratory

positive airway pressure (IPAP) when breathing in and the expiratory positive airway pressure (EPAP) was maintained when breathing out. The pressures were titrated for each individual according to comfort. The pressures used for the subjects were from 9-12cms H₂O IPAP and 4-6cms H₂O EPAP. Only subject # 1 had IPAP 12cms and EPAP 6cms, the other six subjects had IPAP 9cms and EPAP 4cms. This swing between IPAP and EPAP provided minor mechanical ventilation of 5-6cms H₂O. The subjects had not been familiarized with VPAP before its use overnight at White Mountain.

Sleeping oxyhemoglobin saturation, chest and abdominal inductive plethysmography and nasal airflow using oxygen cannula and pressure transducer were recorded in all subjects at sea level and both nights at 3800m. Sleep was recorded using a portable polysomnographic device, the Siesta (Compumedics, Melbourne, Vic, Australia).

The Lake Louise Questionnaire (Roach et al., 1993) was administered to each subject in the morning within an hour of waking from sleep at 3800m. The Lake Louise Score (LLS) derived from the questionnaire was used to assess the presence and severity of Acute Mountain Sickness (AMS). The subjects were asked if the following symptoms were present and to rate the severity: headache, loss of appetite or nausea, fatigue or weakness, dizziness or lightheadedness and difficulty sleeping. Each symptom was given a score from 0 to 5, where 0 equals no symptom and 5 or higher equals severe symptoms. The presence of headache, plus a score of 4 or more derived from the abovementioned symptoms was diagnostic of AMS.

Five minute measurement of SaO₂ using pulse oximetry (Nellcor N-200 oximeter, Nellcor Puritan-Bennett Inc; Pleasanton, CA, USA) and end tidal CO₂ using a Beckman capnograph (model LB-2; Beckman, Sciller Park, IL, USA) were performed in the evening and in the morning after sleep studies on both days at 3800m.

The subjects did not consume caffeine, acetazolamide, alcohol or other respiratory stimulants or respiratory depressants during the research period. The female subjects were not taking hormonal contraceptive medication.

Analysis

Respiratory events during sleep were scored using the American Academy of Sleep Medicine (AASM) 1999 criteria. Respiratory analysis was performed to obtain the periodic breathing apnea/hypopnea index (number of central apneas/hypopneas per hour of sleep) and the mean and minimum oxyhemoglobin saturations during sleep. Mean and minimum oxyhemoglobins were compared for use of VPAP on the first or second night at 3800m as well as for the overall differences.

Statistical Analysis

Paired t tests were used to compare the mean and minimum sleeping oxyhemoglobin saturations and the Lake Louise Score for the two nights spent at 3800m, and the mean and minimum SaO₂ of subjects who did or did not develop AMS after a night's sleep without VPAP. One way analysis of variance with repeated measures was used to compare the

means of those using or not using VPAP on the first or second night at 3800m. Wilcoxon Signed Ranks test was used to compare LLS on and off VPAP.

Lake Louise Score is presented as the median $\pm$ standard deviation. All other data are presented as the mean $\pm$ standard deviation.

Statistical significance is considered to be $p < 0.05$

Results

Lake Louise Scores and Sleeping Oxygen Saturations on nights using VPAP and not using VPAP. Tables 1 and 2

The use of positive pressure ventilation via a face mask during sleep at 3800m had significant effects on the symptoms of Acute Mountain Sickness (AMS), measured by the Lake Louise Score (LLS), and the sleeping oxygen saturation (SaO_2). The LLS was significantly lower ($p = 0.04$) and the mean and minimum sleeping SaO_2 were significantly higher ($p = 0.002$ and 0.005) when using VPAP.

Although this was true for the group as a whole, the subjects who developed AMS appeared to benefit more from the use of VPAP. The three subjects without AMS had LLS of zero on both mornings, whereas the four subjects with AMS had LLS of 4, 5, 7 and 7 without VPAP and 0, 0, 1 and 1 when using VPAP ($p = 0.006$). Table 2.

There were also differences in the sleeping SaO_2 in the four AMS subjects and the three subjects without AMS, both on and off VPAP, with the subjects without AMS having a higher sleeping mean and minimum SaO_2 on both nights.

The four subjects who developed AMS had significantly lower mean ($p = 0.02$) and minimum (<0.001) sleeping SaO_2 than the three subjects without AMS on the nights they slept without assisted ventilation.

The AMS subjects had a similar sleeping SaO_2 when using VPAP to the subjects without AMS sleeping without VPAP ($p = 0.2$ for mean and 0.1 for min.). Thus, subjects who developed AMS had sleeping SaO_2 increased by the use of VPAP to the level of the subjects without AMS when not using VPAP. The AMS subjects had significant improvement in mean SaO_2 when using VPAP ($p = 0.005$) but not the minimum ($p = 0.06$); while the subjects without AMS did not demonstrate significant improvement in either mean or minimum SaO_2 when using VPAP ($p = 0.06$ and 0.2).

There were no differences in either the mean or minimum SaO_2 in subjects using VPAP on the first or second night at 3800m ($p > 0.1$). Table 3.

Morning and Evening End Tidal CO_2 and Oxyhemoglobin Saturation. Table 4

The tests were performed with the subject sitting in an upright position. There was no evidence of acclimatisation in morning and evening measurements of SaO_2 and ETCO_2 in any subject during the two day sojourn at 3800m i.e. these measurements did not

significantly change over the two day period. Morning and evening SaO₂ and ETCO₂ were not significantly different after sleeping with or without VPAP.

Discussion

This research has demonstrated that sleeping at high altitude using a positive pressure ventilator (VPAP) improves sleeping oxygen saturation and alleviates or abolishes the symptoms of Acute Mountain Sickness (AMS). We also demonstrated that those subjects who developed AMS had lower overnight oxygen saturation than those subjects who did not develop AMS. When VPAP was used during sleep by subjects with AMS their oxygen saturation was similar to the subjects without AMS on the nights they slept without VPAP. This research also demonstrates that the subjects who developed AMS appeared to derive a greater benefit from the use of VPAP during sleep as the degree of improvement in sleeping SaO₂ was greatest in them.

In our subjects 57% developed AMS, with Lake Louise Scores from 4-7; this prevalence is in agreement with previous work on AMS e.g. Hackett's 1976 finding of 53% of trekkers at Pheriche in the Nepal Himalaya at an altitude of 4200m and Kayser et al in 1993 with 69% in the Alps at 4360m.

AMS has been shown to be associated with a low mean sleeping SaO₂ (Burgess et al 2004; Erba et al 2004), and keeping the SaO₂ higher during sleep by the use of VPAP appears to have effectively prevented AMS symptoms.

The mechanism of this improved sleeping oxygen saturation and associated improvement in AMS symptoms is unknown but may involve increased alveolar minute ventilation by the application of inspiratory positive airway pressure, and increased functional residual capacity (FRC), with prevention of upper airway and alveolar collapse during expiration and consequent improved gas exchange. VPAP inspiratory pressures in our subjects were from 9-12cms H₂O and expiratory pressures were from 4-6cms H₂O. These are relatively low pressures but were obviously effective in increasing sleeping SaO₂.

The major shortcoming of this research was that only seven subjects were studied. The power of the statistical analyses would be increased by studying a larger group. This was also an unblinded study, with no control e.g. sham VPAP being used. However, the results, particularly for the AMS subjects, clearly demonstrate a strong case for the use of positive pressure ventilation at high altitude to prevent AMS. This study would have benefitted, also from the addition of measurements of minute ventilation, arterial pH or overnight ETCO₂.

The three female subjects did not develop AMS, and overnight SaO₂ was higher than the four male subjects on both nights at 3800m. This may be due to the respiratory stimulatory effects of ovarian hormones. Some studies have found women to be less susceptible to AMS (Murdoch, 1995 and 1998; Kayser 1991) while others have found that men and women are equally susceptible (Honigman 1993; Hackett 1976). This finding may also be a type 2 error due to the small number of subjects.

We used VPAP rather than continuous positive airways pressure (CPAP) because we believed it would be more effective in increasing sleeping SaO₂. VPAP delivers a higher inspiratory pressure, presumably increasing tidal volume, then the pressure falls to a lower level during expiration, making breathing more comfortable than with CPAP.

Positive end-expiratory pressure (PEEP) is known to improve gas exchange and increase oxyhemoglobin saturation in pulmonary edema (Wayne, 1976) and at high altitude (Schoene et al, 1985; Larson, 1985) but it is also thought to increase the work of breathing, the risk of barotrauma, worsen concomitant cerebral edema by decreasing venous return (Oelz et al, 1983) and may reduce cardiac output in low volume states. The method used to deliver PEEP in the field at high altitude was via a tightly fitted face mask with a valve that allowed inspiration at barometric pressure and expiration at increased pressure.

PEEP has also been used during ascent of Mont Blanc at 4810m (Launay et al, 2004), in hypobaric chamber experiments (Savoirey et al, 1998) and was found to reduce the incidence of AMS and improve oxyhemoglobin saturation.

Continuous positive airways pressure (CPAP) was used by a group studying its effect at Mount Cook (3205m) in New Zealand (Davis et al, 2002. Abstract). Fourteen subjects ascended Mt Cook rapidly; the use of a specially designed CPAP machine that operated at low gas flows increased oxyhemoglobin saturation and decreased the respiratory rate with no apparent fall in cardiac output. This work was presented as an abstract only.

This study demonstrates the efficacy of positive airway pressure in improving oxyhemoglobin saturation at high altitude.

There is no published work to report VPAP or CPAP use during sleep at high altitude to prevent the occurrence of AMS. The findings from two studies (Erba et al 2004; Burgess et al 2004), demonstrating the link between low sleeping oxyhemoglobin saturation and AMS, suggest that more research into the use of the above methods during sleep is needed.

In conclusion, this research demonstrated that the use of a positive pressure ventilator that delivers both positive inspiratory and expiratory pressures, during sleep at high altitude reduces or abolishes the symptoms of AMS. The mechanism for the improvement may be through improvement in sleeping SaO₂ consequent upon inspiratory pressure assistance acting to increase ventilation and/or positive pressure associated alveolar recruitment acting to improve gas exchange.

The results from this small study should be considered as preliminary research only; more work is needed to determine if VPAP could be a useful tool for treating AMS in the field.

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Summary at a glance

Overnight oxyhaemoglobin desaturation is related to Acute Mountain Sickness (AMS). AMS can be debilitating and may require descent. Positive pressure ventilation during sleep at high altitude may prevent AMS and therefore be useful in people travelling to high altitude, who are known to suffer from AMS.

Table 1

The mean and minimum sleeping oxyhemoglobin saturations (SaO₂) and the Lake Louise Scores (LLS) were compared when non-invasive positive pressure ventilation (VPAP) was used during sleep at 3800m and when it was not used (spontaneous breathing). There were significant improvements in the mean ($p = 0.002$) and the minimum ($p = 0.005$) SaO₂ when VPAP was used. Acute Mountain Sickness (AMS) symptoms, measured by the LLS, were significantly decreased when VPAP was used during sleep ($p = 0.04$).

Subject (sex)	Mean SaO ₂ % spontaneous breathing	Mean SaO ₂ % VPAP	Min. SaO ₂ % spontaneous breathing	Min. SaO ₂ % VPAP	LLS post-spontaneous breathing	LLS post-VPAP	VPAP 1 st or 2 nd night at 3800m
1(m)	76	82	69	75	4	0	2
2(m)	72	80	63	69	5	1	1
3(m)	73	79	67	72	7	1	2
4(m)	74	76	59	65	7	0	1
5(f)	80	82	73	74	0	0	2
6(f)	80	83	74	74	0	0	1
7(f)	82	87	74	80	0	0	2
Mean ± SD	76.7 ± 3.9	81.3 ± 3.5	68.4 ± 5.8	72.7 ± 4.8	3.3 ± 3.3	0.3 ± 0.5	
		$p = 0.002$		$p = 0.005$		$p = 0.04$	

Table 2

When the four subjects who developed AMS were compared with the three subjects who did not develop AMS it was found that the mean SaO₂ was significantly higher on both nights in the subjects without AMS ($p < 0.001$ spontaneous breathing, and $p = 0.03$ with VPAP). The minimum SaO₂ was also higher in the subjects without AMS on both nights but reached significance only during the night of spontaneous breathing ($p = 0.009$). IPAP and EPAP pressures were 9cms and 4cms H₂O in all but subject #1 whose setting were 12cms and 6cms H₂O. These pressures gave a swing of 5-6cms H₂O, which delivered a minor degree of mechanical ventilation.

AMS Subjects	Mean SaO ₂ % spontaneous breathing	Mean SaO ₂ % VPAP	Min. SaO ₂ spontaneous breathing	Min. SaO ₂ VPAP	LLS post-spontaneous breathing	LLS post-VPAP	IPAP/EPAP pressures cmsH ₂ O
1 (m)	76	82	69	75	4	0	12/6
2 (m)	72	80	63	69	5	1	9/4
3 (m)	73	79	67	72	7	1	9/4
4 (m)	74	76	59	65	7	0	9/4
Mean ± SD	73.8 ± 1.7	79.3 ± 2.5	64.5 ± 4.4	70.3 ± 4.3	5.8 ± 1.5	0.5 ± 0.6	
No-AMS Subjects							
5 (f)	80	82	73	74	0	0	9/4
6 (f)	80	83	74	74	0	0	9/4
7 (f)	82	87	74	80	0	0	9/4
Mean ± SD	80.7 ± 1.2	84 ± 2.6	73.7 ± 0.6	76 ± 3.5	0	0	
P value	< 0.001	0.03	0.009	0.06	< 0.001	0.1	

Table 3

Subjects used VPAP during sleep on either the first or second night at 3800m.. When comparing the mean and minimum SaO₂ and AMS symptoms (LLS) on the two nights a significant difference was found only in the minimum SaO₂, which was significantly lower when VPAP was used on the first night at 3800m ($p = 0.05$).

Subjects using VPAP 1st night	mean SaO ₂ % spont.breathing	mean SaO ₂ % VPAP	min. SaO ₂ % spont.breathing	min.SaO ₂ % VPAP
2	72	80	63	69
4	74	76	59	65
6	80	83	74	74
Mean ± SD	75.3±4.2	79.7±3.5	65.3±7.8	69.3±4.5
Subjects using VPAP 2nd night	mean SaO ₂ % spont.breathing	mean SaO ₂ % VPAP	min. SaO ₂ % spont.breathing	min.SaO ₂ % VPAP
1	76	82	69	75
3	73	79	67	72
5	80	82	73	74
7	82	87	74	80
Mean ± SD	77.8±4.0	82.5±3.3	70.8±3.3	75.3±3.4
	<i>ns</i>	<i>ns</i>	<i>ns</i>	$p = 0.05$

Table 4

Five minute measurements were taken of end tidal carbon dioxide and oxyhemoglobin saturation in the mornings after sleeping at 3800m. There was no evidence of acclimatisation during the 36-40 hours spent at 3800m i.e. ETCO₂ and SaO₂ were not significantly different on the first and second mornings

Subjects	Morning SaO ₂ post-spontaneous breathing	Morning SaO ₂ post-VPAP	Morning ETCO ₂ post-spontaneous breathing	Morning ETCO ₂ post-VPAP
	%	%	mmHg	mmHg
1 (m)	89	89	29.7	31.3
2 (m)	91.4	88.1	32.1	33.7
3 (m)	94.3	92.6	28.6	27.1
4 (m)	86.5	86.5	31.2	32.5
5 (f)	88.9	92.2	30.7	33.5
6 (f)	95.4	94	26.5	28.5
7 (f)	93	91.4	28.8	29
Mean ± SD	90.5 ± 2.7	91.2 ± 3.2	29.7 ± 1.9	30.8 ± 2.6
	<i>p</i> = 0.3		<i>p</i> = 0.2	