

Treatment of Acute Mountain Sickness: Hyperbaric Versus Oxygen Therapy

Study objectives: To compare the benefits of simulated descent in a hyperbaric chamber with those of supplementary oxygen for the treatment of acute mountain sickness.

Design: A prospective study.

Setting: The Snake River Health Clinic in Keystone, Colorado, which has an altitude of 2,850 m (9,300 ft).

Type of participants: Twenty-four patients who presented with acute mountain sickness.

Interventions: A simulated descent of 1,432 m (4,600 ft) was attained by placing the patients in a fabric hyperbaric chamber and pressurizing the chamber to 120 mm Hg (2.3 PSI) above ambient pressure. Patients were randomly assigned to either the hyperbaric treatment or treatment with 4 L of oxygen given by facemask; both treatments lasted for two hours.

Measurements and main results: Mean arterial oxygen saturation (SaO_2) increased 7% ($84 \pm 2\%$ to $91 \pm 1\%$) with pressurization and 14% ($83 \pm 4\%$ to $96 \pm 1\%$) with oxygen during treatment over pretreatment levels. Symptoms of acute mountain sickness decreased as rapidly with pressurization as with oxygen treatment, despite significantly higher SaO_2 in the oxygen-treated group during treatment. Symptomatic improvement was retained in both groups at least one hour after treatment.

*Conclusion: Simulated descent in a fabric hyperbaric chamber is as effective as oxygen therapy for the immediate relief of acute mountain sickness. [Kasic JF, Yaron M, Nicholas RA, Lickteig JA, Roach R: Treatment of acute mountain sickness: Hyperbaric versus oxygen therapy. *Ann Emerg Med* October 1991;20:1109-1112.]*

INTRODUCTION

High-altitude illnesses are caused by the hypobaric hypoxia experienced by visitors to a high altitude. These are classified as acute mountain sickness (AMS), high-altitude pulmonary edema (HAPE), and high-altitude cerebral edema (HACE). Although AMS is usually mild and self-limited, it often is associated with or may progress to the potentially fatal forms of HAPE or HACE. AMS is effectively treated with descent, oxygen, or medication.

Simulated descent has recently become a practical treatment option because of the development of lightweight, portable hyperbaric chambers.¹ Mountaineers have reported several cases of dramatic improvement with this treatment while they were high on a mountain where neither descent nor oxygen was immediately available.² Takei and associates were the first to report the use of hyperbaric therapy for the treatment of altitude illness.³ They found that the majority of their 14 patients had complete relief of their symptoms. No control group was used in this initial study. Taber evaluated hyperbaric treatment in patients with AMS, HAPE, and HACE.⁴ Hyperbaria was found to be effective, although no control group was included, and many patients had either oxygen or dexamethasone treatment concurrent with hyperbaria.⁴

Hackett and colleagues evaluated the effectiveness of hyperbaria versus equal oxygen therapy in the treatment of moderate-to-severe HAPE.⁵ They concluded that oxygen and hyperbaric therapy were equally effective in the resolution of symptoms. To date, no controlled comparison of hyperbaria

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TABLE. Comparison of entry characteristics

Diagnosis	Treatment	Gender		Age (Yr)	Height (cm)	Weight (kg)	Travel Time (hr)
		Male	Female				
AMS	Pressure	7	3	46 ± 4	173 ± 5	74 ± 5	11 ± 3
	Oxygen	5	3	37 ± 3	179 ± 4	76 ± 7	11 ± 2
AMS and HAPE	Pressure	3	0	30 ± 8	180 ± 3	78 ± 7	14 ± 3
	Oxygen	2	1	36 ± 3	174 ± 1	82 ± 5	8 ± 1

No significant differences between treatment groups.

with oxygen for the treatment of AMS has been reported. Therefore, we compared the efficacy of simulated descent with that of oxygen therapy in patients with AMS and AMS associated with mild HAPE.

MATERIALS AND METHODS

Study patients were recruited from those entering the Snake River Health Clinic in Keystone, Colorado (altitude, 2,950 m; barometric pressure, 544 ± 7 mm Hg), for medical treatment of altitude illness. Patients who received a clinical diagnosis of AMS were evaluated for the presence and severity of headache and nausea. A mild headache was assigned one point, and two points were given for a severe headache. Nausea was given one point. Patients with nausea or headache who had arrived to altitude within 72 hours were considered to have AMS and qualified for entry into the study. AMS patients with mild HAPE as diagnosed by chest radiography and clinical examination also were recruited into the study.⁶⁻⁸

Exclusion criteria included severe altitude illness (requiring prompt evacuation to a lower-altitude treatment facility); previous treatment with oxygen, acetazolamide, or dexamethasone; acute or chronic heart or lung disease (not including HAPE); less than 18 years of age; pregnancy or nursing mother; or evidence of acute upper respiratory infection.

On acceptance into the study, the methods and rationale were explained. Patients agreeing to participate signed informed consent and then were randomly assigned to oxygen or hyperbaric treatment protocols. The study was approved by the Human Subjects Review Committee of the University of Colorado.

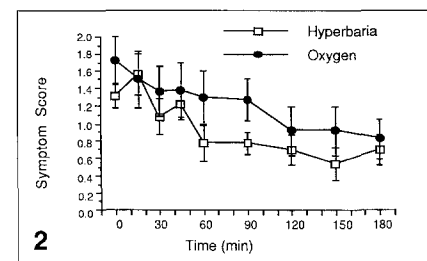
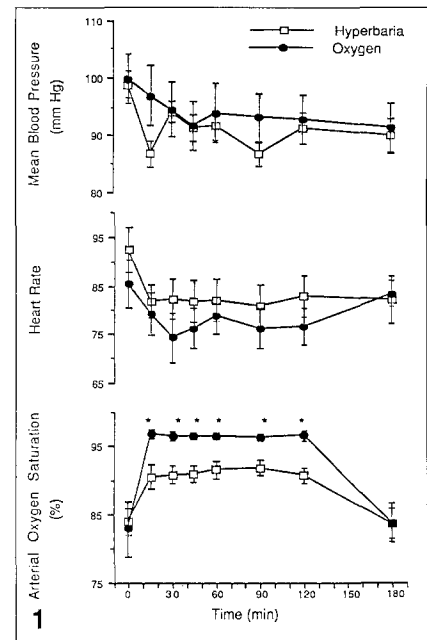
Physiologic parameters and symptom responses were recorded by one of the investigators. Baseline measurements of pulse rate, blood pressure, and arterial oxygen saturation (SaO₂) were recorded. Supine blood

FIGURE 1. Mean blood pressure, heart rate, and arterial oxygen saturation versus time. *P < .05 between treatments.

FIGURE 2. Combined symptom scores of headache and nausea. Significant improvement occurred with both therapies by the end of treatment. P < .05.

pressure and heart rate (measured with a Physio-Control VSM3 monitor, Physio-Control Corp, Redmond, Washington), SaO₂ (measured with an Ohmeda 4700 pulse oximeter [Ohmeda Monitoring Systems, Louisville, Colorado] by finger probe), and symptoms of headache and nausea were monitored at 15-minute intervals for the first hour and at 30-minute intervals for an additional hour of treatment and for one hour after treatment. Symptom scores were monitored using the same point system that was used for entry of patients into the study. SaO₂ values were calculated by averaging measurements stored at six-second intervals over a three-minute period at each 15-minute interval. Normal values for SaO₂ at the clinic altitude were obtained by pulse oximetry in 47 normal, healthy, acclimatized volunteers.

A 5.5-kg, 0.81-m-diameter by 2.1-m-long cylindrical fabric hyperbaric chamber (Gamow Medical Tent, Portable Hyperbarics Inc, Ilion, New York) with an internal support frame was used as the pressure chamber. The chamber is large enough to easily accommodate a supine patient, and plastic windows permit observation of the chamber interior by the clinician. The chamber was pressurized (over three to four minutes) with a 4-lb bellows-type foot pump to 100 mm Hg above ambient atmospheric pressure. Once pressurized, an oil-free diaphragm compressor (model 607CE, Thomas Industries



Inc, Sheboygan, Wisconsin) circulated air through the chamber at 1.5 ft³/min and increased the pressure to 120 mm Hg (2.3 PSI) above ambient atmospheric pressure (equal to a descent of approximately 1,432 m [4,600 ft] at the Keystone location). In earlier studies with the chamber, this volume was found adequate to keep the steady-state oxygen and carbon dioxide concentrations above 20% and below 1%, respectively.⁹

The oxygen-treated patients were given oxygen through a rebreather facemask at a flow rate of 4 L/min (fraction of inspired oxygen, FiO₂, 30% to 35%). This oxygen-flow rate represented the standard of care for AMS therapy at the Snake River Health Clinic. Therapy lasted two hours in each treatment modality.

Differences between treatment groups for heart rate, SaO₂, blood pressure, age, height, weight, and travel time to Keystone were evaluated using a two-sample Student's t test. Symptom responses between groups and genders were compared

using the Mann-Whitney *U* test. The log-rank survival comparison was used to evaluate time to resolution of symptoms between the groups. Data presented are given as mean \pm SEM, with $P < .05$ considered significant.

RESULTS

Twenty-nine patients were randomized into the study. Because of mechanical and technical errors, complete data were available in only 24 of the subjects, and the remainder was excluded from data analysis. These errors occurred in the monitoring equipment, not with the hyperbaric chamber. Missing data were distributed between the two groups (two in hyperbaric and three in oxygen). Of the 24 with complete data, 18 were diagnosed with AMS alone, and six had mild HAPE in addition to AMS. Ten AMS patients received hyperbaric treatment, and eight received oxygen treatment. Three AMS and HAPE patients received hyperbaric treatment, and three received oxygen treatment. Treatment groups were similar with regard to gender, age, height, weight, travel time to Keystone, and symptom severity at presentation (Table).

Oxygen saturation on room air was measured in normal acclimatized volunteers; mean value was $94 \pm 2\%$. Before treatment, both treatment groups in the present study had SaO_2 values significantly below this normal control value ($P < .05$). Pretreatment values for SaO_2 , heart rate, and blood pressure were similar between groups. During treatment, SaO_2 was significantly higher in the oxygen-treated group ($83 \pm 4\%$ to $96 \pm 1\%$) than in the hyperbaric treatment group ($84 \pm 2\%$ to $91 \pm 1\%$). This represents a 14% increase over pretreatment values for the oxygen-treated group and a 7% increase for the hyperbaric treatment group. At one hour after treatment, SaO_2 in each treatment group returned to pretreatment values.

Heart rate and mean arterial blood pressure decreased significantly in both groups during treatment compared with pretreatment values, with no significant differences between the groups (Figure 1). Both treatments significantly improved symptoms by the end of treatment ($P < .05$). Symptom responses and speed of symptom resolution did not differ significantly between the groups throughout two

hours of treatment and one hour after treatment (Figure 2).

Analysis of the data for AMS and HAPE patients alone revealed results similar to those of the AMS-only patients.

DISCUSSION

This study demonstrated that hyperbaric therapy was as effective as oxygen therapy for the immediate treatment of AMS. Both methods were successful in resolving or improving the symptoms of AMS in the majority of patients.

The long-term effects of our treatments are unknown because our patients were monitored for only one hour after treatment. Two patients (one in each group) were unresponsive to treatment. Both of these patients may have been misdiagnosed initially. One patient (oxygen treatment) may have had a migraine headache, and one patient (hyperbaric treatment) was thought to have a viral syndrome.

No complications occurred as a result of hyperbaric therapy. The chamber offered sufficient room for the patients to stretch out and rest. No patient had to be released from the chamber because of claustrophobia. Although the pressure change in the chamber can cause a pressure gradient across the tympanic membrane, no patients had difficulty clearing their ears when the chamber was pressurized slowly. No other complaints were made by the hyperbaric patients except that the chamber tended to get warm as a result of pressurization and the presence of monitoring equipment.

Bartsch and associates recently reported improvement in AMS symptoms in persons breathing room air only, suggesting that a significant placebo effect exists in the treatment of AMS.¹⁰ In our study, the aim was to compare two treatments as they are used in the clinical setting. We did not attempt to blind either the oxygen or the hyperbaric therapy.

AMS is a clinical syndrome consisting of headache, nausea, vomiting, shortness of breath, dizziness, and malaise. Symptoms and severity of AMS vary in different persons. AMS is generally more severe with rapid ascent to altitude. Headache and nausea are the cardinal symptoms of AMS (and thus were chosen for the monitoring parameters in this

study).^{7,8,11} HAPE is characterized by increasing dyspnea, a dry cough progressing to a productive one, prostration, and, if untreated, death.^{7,8}

As more people travel to high altitudes, AMS has become more common. A recent unpublished study by the Colorado Altitude Research Institute has shown that as many as 24% of the 10 to 15 million visitors to the Rocky Mountain high country (altitude, 8,000 to 10,000 ft) experience some form of AMS (Ben Honigman, MD, personal communication, 1990). It is estimated that the high-altitude resort industry may lose \$50 to \$75 million a year because of altitude illness among visitors.¹² In mild cases of AMS, rest, frequent small meals, avoidance of alcohol, and over-the-counter medications for headache may be all that is needed for treatment. In our clinical experience, most people recover in a few days and are able to gradually resume normal activity.

However, in more severe cases of high-altitude illness, descent is the treatment of choice and may indeed be life saving. Dramatic improvement often accompanies a modest, 300-m reduction in altitude.¹³ Descent reverses hypobaric hypoxia by increasing the partial pressure of inspired oxygen (PI_{O_2}). Oxygen therapy reverses hypoxia by raising the fraction of oxygen in the inspired gas (FI_{O_2}). Dexamethasone and acetazolamide have been used to treat AMS patients effectively,^{14,15} but neither medication works all the time, each can have unpleasant side effects, and both have a significant delay of onset of action.¹⁶

In rough mountainous terrain, during bad weather conditions, and with very ill patients, descent may not be feasible, and supplemental oxygen may not be available. Simulated descent in a hyperbaric chamber may be the treatment of choice under these conditions. In contrast to bottled oxygen, which is heavy to carry and limited in supply, fabric hyperbaric chambers are lightweight, and the duration of treatment is not limited.

Although the role of hypoxia in the pathophysiology of altitude illness is of primary importance, it is unclear whether hypobaria plays an additional role. Decompression-induced platelet aggregation,¹⁷ intravascular microbubble formation,¹⁸ and alter-

ation of the forces governing transvascular fluid fluxes across the pulmonary vasculature¹⁹ have been suggested as mechanisms.

In our study, the SaO₂ values in the oxygen group were significantly higher throughout the two hours of treatment compared with those of the hyperbaric group. This reflects the increased P_iO₂ in the oxygen group (P_iO₂, 149 to 173 mm Hg with oxygen vs 123 mm Hg with hyperbaria). We note with interest that increased oxygen saturation associated with oxygen therapy did not result in better symptom resolution than hyperbaric treatment. This finding suggests the possibility that a threshold exists beyond which further improvement in oxygenation is of no additional benefit. It remains unclear exactly what roles oxygen and elevation of barometric pressure play or interplay in the treatment of AMS.

CONCLUSION

Oxygen and simulated descent resulting from pressurization in a hyperbaric chamber are equally effective in the immediate treatment of AMS. Two hours of simulated descent of more than 1,400 m is as effective as breathing 4 L/min of supplemental oxygen for the relief of the symptoms of AMS. Two hours of treatment resulted in a greater SaO₂ with oxygen therapy; however, AMS symptoms decreased as rapidly with

pressurization as with oxygen therapy. Further studies are needed to help elucidate the role of hyperbaria and its synergistic effects with oxygen in the treatment of AMS.

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