Oxygen Saturation In High-Altitude Pulmonary Edema

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High altitude, defined as elevations greater than or equal to 8000 feet (2438 m) above sea level, is responsible for a variety of medical problems both chronic and acute. The spectrum of altitude illness ranges from the common, mild symptoms of acute mountain sickness, such as insomnia, headache, and nausea, to severe and potentially fatal conditions, such as high-altitude pulmonary edema (HAPE) and high-altitude cerebral edema (HACE).¹

HAPE is a noncardiogenic form of pulmonary edema that predominantly affects young, physically active, previously healthy individuals who arrived at high altitude between 1 and 4 days before developing symptoms. Symptoms of early, milder cases include dry nonproductive cough, decreased exercise tolerance, and dyspnea on exertion. In more severe cases the victim experiences severe dyspnea at rest, orthopnea, and cough productive of pink, frothy sputum. In the most severe (and rare) cases, HAPE can be complicated by ataxia, lethargy, or coma indicative of concomitant HACE. Symptoms are typically accompanied by physical findings of rales, temperature of up to 38.8°C (102°F), tachypnea, tachycardia, and possibly cyanosis. Rales often begin in the right middle lobe and then spread diffusely.1-3

A chest radiograph is the reference standard for confirming the diagnosis of HAPE. Radiographic findings show a normal cardiac silhouette with asymmetrical, fluffy, patchy infiltrates.^{1,3,4} Demonstration of sufficiently low arterial oxygen saturations, however, is also thought to be adequate for confirming diagnosis.^{5,6} In this study, we attempted to determine whether infiltrative processes (such as HAPE) shown on chest radiographs are consistently associated with lower blood oxygen saturation.

Methods

The 126 subjects for this study were all patients who came to the Summit Medical Center Emergency Department or to the Frisco Medical Center. Both units serve Summit County, Colorado. The base elevation of Summit County ranges from roughly 9000 to 11,000 feet (2743 to 3354 m). Between 1 November 1990 and 26 January 1991, a record was maintained of the age, sex, room air pulse oximeter measure of oxygen saturation (S₂O₂), chest radiograph findings, and final diagnoses of all patients who underwent a chest radiograph examination.

There were 152 patients who underwent chest radiography during the study period. Twenty-six patients were excluded from the study: 18 had no oxygen saturation measurement taken or recorded, 7 had conditions that required more intensive treatment and were referred to an affiliated hospital in Denver, so their radiographs were not read by the Summit Medical Center staff radiologists, and 1 patient had both reasons for exclusion.

Pulse oximetry was performed using a Nellcor N-200[™] pulse oximeter to obtain room air S₂O₂ levels. Chest radiographs were read by 2 staff radiologists who were blinded to the clinical data, including pulse oximetry data.

Results

After exclusions, 126 subjects remained in the study. Their ages ranged from 2 months to 89 years; mean age was 38.6±21 years. Ninety-eight patients (78 percent) were male. There were 23 subjects who were considered to have HAPE; 15 had other infiltrative processes, all of which were diagnostic of pneumonia; and the remaining 88 had no infiltrates seen on chest radiography. The diagnoses in those without infiltrates included rib fractures and contusions, pericarditis, asthma, angina, hiatal hernia, and acute mountain sickness.

In those patients who had a diagnosis of HAPE, the mean S₂O₂ was 73.7 percent, standard devia-

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tion 13.2 percent. The ages of those with HAPE ranged from 14 years to 54 years; the mean was 29.9 years, standard deviation 12.6 years. In patients with pneumonia, the mean S_aO_2 was 82.3 percent, standard deviation 11.8 percent. The mean S_aO_2 in the group with no pulmonary infiltrate was 90.6 percent, standard deviation 6.2 percent. These results are summarized in Table 1. Table 2 presents the number of patients in each group according to particular S_aO_2 ranges.

Analysis of variance (F-test) was applied to the data in Table 1. F-value (2123 df) was 41, and therefore significant at P = 0.01.

Discussion

At high altitude, changes in oxygen saturation are a sensitive reflection of changes in total oxygen tension (P₂O₂) of the blood, whereas at altitudes of less than 8000 feet, larger changes can occur in P_aO_2 without accompanying change in S_aO_2 . This phenomenon is attributable to the sigmoidal shape of the oxyhemoglobin dissociation curve. Consequently, measurement of S₂O₂ by pulse oximetry is of far greater value at high altitude than at low altitude in assessing the adequacy of a patient's oxygenation. The advantages of measuring S₂O₂ by pulse oximetry rather than direct measurement of PaO2 by arterial blood gases are several. Pulse oximetry is painless and noninvasive, the results are instantaneous and continuous, no laboratory work-up is required, and the procedure is relatively inexpensive.7 Technical limitations of the pulse oximeter, however, must always be considered when evaluating results. Both carboxyhemoglobin and methemoglobin interfere with the accuracy of pulse oximeter readings.8 Also, the accuracy of the pulse oximeter, which is ±4 percent between values of 80 percent to 100

Table 1. Oxygen Saturation, as Measured by Pulse Oximetry, of Those Patients with HAPE, with Pneumonia, and No Infiltrates by Chest Radiography.

Radiograph Results	No.	Mean S _a O ₂ †	Standard Deviation 13.2	
HAPE*	23	73.7		
Pneumonia	15	82.3	11.8	
Negative	88	11.8	6.2	

^{*}HAPE = High-altitude pulmonary edema.

Table 2. Number of Patients with HAPE, Pneumonia, and No Infiltrates by Chest Radiography That Fall within Certain Oxygen Saturation Ranges.

Radiograph Results	S₂O₂†				
	< 70%	70-79%	80-89%	90-100%	Total
HAPE*	8	3	11	1	23
Pneumonia	2	2	5	6	15
Negative	0	6	25	57	88

*HAPE = High-altitude pulmonary edema.

†S₂O₂ = Oxygen saturation.

percent, is much lower when S₂O₂ is less than 80 percent.

Our study shows that there is less than a 1 percent chance that differences in S₂O₂ among HAPE, pneumonia, and no infiltrate patients are attributable to random error. Infiltrative processes seen on radiographs are associated with decreased blood saturation, and HAPE victims are more severely desaturated than those with pneumonia. As Table 2 illustrates, however, high S_aO₂ values do not effectively rule out a diagnosis of HAPE. Although low S_aO_2 values (e.g., < 70 percent) are useful for predicting an infiltrative process on a chest radiograph, they do not distinguish HAPE from pneumonia. This consideration is not trivial, because both pneumonia and HAPE can cause dyspnea, cough, and fever in the hypoxic patient. In a study of 27 patients in Japan, the authors noted that mild elevations of peripheral white cell counts were also common in HAPE victims.9 Chest radiographic findings ultimately distinguish pneumonia from HAPE in questionable cases. In many highaltitude health care settings, chest radiography is not available to confirm a diagnosis of HAPE. In such locations, the availability of pulse oximetry is a useful adjunct to history and physical findings in the presumptive diagnosis and follow-up evaluation of treatment of HAPE. As this study suggests, pulse oximeter measurements of S₂O₂ can bear close relation to findings on chest radiograph.

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 $[\]dagger S_a O_2 = Oxygen saturation.$

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