Osteopathic Family Physician (2011) 3, 202-206

ELSEVIER

acofp Osteopathic Family Physician

High-altitude exposure and its effects on special populations

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KEYWORDS:
High-altitude illness;
High-altitude cerebral edema;
Mountain sickness;
Hypobaric hypoxia;
Monge disease

Altitude illness is a spectrum of conditions that affect the majority of travelers to high elevations (>8200 ft [2500 m]). The rapid decrease in arterial oxygen saturation with increasing altitude is the physiologic basis for this group of disorders. Many travelers to high altitudes will experience symptoms of acute mountain sickness including headache, fatigue, dyspnea, and dizziness. Prolonged exposure can result in more serious pulmonary, central nervous system, and circulatory disorders. Core treatments for most altitude-related illnesses include descent, rest, medications, and supportive care. Children and travelers with chronic diseases are more susceptible to altitude illness compared with the general population. Pregnant women residing at high altitudes are at increased risk for pregnancy-induced hypertension, preeclampsia, and low-birth-weight infants. Early recognition and treatment of altitude illness can prevent life-threatening situations. Primary care physicians should counsel patients traveling to high altitudes about altitude illness and discuss medications available for mountain sickness prophylaxis.

High-altitude syndromes are a variety of conditions that affect as many as 90% of travelers to high altitudes. The severity of acute altitude illnesses range from self-limited to life-threatening and encompass a wide range of clinical and physiologic conditions. The key to treatment and prognosis of altitude illness is prompt identification of symptoms and modification of activity. Family physicians should counsel patients traveling to high elevations about the risks of altitude illness and discuss safe practices and available medications for prevention of symptoms (Table 1).

Physiology: the effects of increasing altitude

The percentage of oxygen in the atmosphere remains constant (about 21%) at all elevations. As altitude increases and barometric pressure decreases, there is a proportional decrease in the partial pressure of oxygen.[1] The decrease in average barometric pressure from 760 mm Hg at sea level to 523 mm Hg at 10,000 ft (3048 m) translates into a decrease in arterial oxygen saturation in non-acclimated healthy individuals from 97% to 90%. At 20,000 ft (6096 m), non-acclimated healthy individuals will average an oxygen saturation of 73%.[2] This hypobaric hypoxia is the basis for most altitude-related illnesses.

Immediate compensation mechanisms after ascent include modest increases in cardiac output and increased pulmonary ventilation. Over time, the human body adapts to hypoxemia and individuals are able to function with less hypoxic effects. This multifactorial process involves physiologic changes over days, weeks, and months and is referred to as acclimatization. Factors contributing to acclimatization include increased red blood cell mass, increased vascularity of tissues, and increased diffusion capacity in the lungs.[2]

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doi:10.1016/j.osfp.2010.10.003

Table 1 Sample locations with elevations

Location	Elevation
Los Angeles, California, USA	233 ft (71 m)
Dallas, Texas, USA	430 ft (131 m)
Ayers Rock, Australia	2831 ft (863 m)
Lake Louise, Alberta, Canada	5449 ft (1661 m)
Mexico City, Mexico	7349 ft (2260 m)
Vail, Colorado, USA	8022 ft (2445 m)
Machu Picchu, Peru	8200 ft (2500 m)
Mauna Kea Observatory, Hawaii, USA	13 800 ft (4205 m)
South Mount Everest Base Camp, Nepal	17 590 ft (5360 m)

For the non-acclimated traveler, exposure to modest altitude may result in headache, tachypnea, and dizziness. These symptoms can develop within hours and are usually self-limited. Symptoms generally resolve in a few days without intervention. Exposure to higher altitudes for healthy individuals without proper acclimatization can result in mental status change, cerebral edema, pulmonary edema, coma, or death. For patients with certain cardiac and pulmonary conditions, exposure to modest altitudes may increase risks of complications from these diseases.

High-altitude sleep disturbance (HASD) and periodic breathing

Most travelers to high altitude report altered patterns of sleep. Frequent or recurrent waking is the most common complaint. This is probably caused by alternating hyperventilation as a result of hypoxia and hypoventilation as a result of hypocapnia, a condition termed *periodic breathing*.[3]

Prophylactic acetazolamide (Diamox, Sigma Pharmaceuticals, Monticello, IA) may help maintain night-time oxygenation and improve sleep quality by decreasing periodic breathing.[3]

Acute mountain sickness

Acute mountain sickness (AMS) is a condition that affects individuals who rapidly ascend to altitudes above 6500 ft (2000 m). Hypobaric hypoxia leads to a cascade of physiologic changes that alter capillary permeability, leading to mild dependent edema and most likely a minimal degree of cerebral edema. Diagnosis of AMS involves the development of headache in a person who has recently arrived at the higher altitude plus one of the following symptoms: fatigue, dizziness, anorexia, nausea, vomiting, or dyspnea on exertion.[4] For those ascending from sea level to 6500 ft (2000 m), the risk of symptoms is 25% for adults.[4] Above 14800 ft (4500 m), risks of AMS symptoms increase to more than 50%.[5]

In general, risk factors for AMS include rapid ascent, final altitude attained, and age younger than 60. Gender, physical fitness, and recent respiratory infections do not appear to contribute significantly to AMS.

A slow rate of ascent and time for acclimation are the best ways to prevent AMS.[5] Travelers should be advised to stop ascent and rest at the onset of symptoms and to descend if symptoms do not improve or they worsen. Acetazolamide, a carbonic anhydrase inhibitor, promotes the excretion of bicarbonate from the kidneys, decreases $PaCO_2$, and increases PaO_2.[6] It has been shown to decrease AMS symptoms. The usual recommended dose is 125 to 250 mg twice daily, starting at least 24 hours before ascent and continued until descent has begun.[7]

High-altitude pulmonary edema

High-altitude pulmonary edema (HAPE) is the most lethal acute syndrome encountered at high altitudes. It occurs in about 4% of travelers above 8200 ft (2500 m), depending on individual adaptability and the ascent rate. Rapid ascent (more than 9800 ft [3000 m] in 3 days) is associated with a higher incidence of HAPE. Previous episodes of HAPE are associated with recurrence rates greater than 50%. Symptoms of HAPE usually appear between one and four days after reaching high altitude and include decreased exercise tolerance and dry cough progressing to productive cough with clear or blood tinged mucous.[8] HAPE can develop with or without preceding AMS symptoms.

Hypobaric hypoxia can cause heterogeneous areas of pulmonary vasoconstriction and vasodilatation.[8] Concurrently, worsening hypoxia leads to increased pulmonary artery pressure. Under these conditions, areas of pulmonary vasodilatation can develop capillary leakage and alveolar hemorrhage. The resulting pulmonary edema appears as patchy infiltrates on chest radiographs.[9]

Arrangements for descent should be made immediately once HAPE is suspected, and pressurized suits and supplemental oxygen can be used if descent is delayed. Medications that can be considered for prophylaxis and treatment of HAPE include calcium channel blockers like nifedipine (Procardia, Pfizer, New York, NY) to prevent or blunt the hypoxia-induced rise in pulmonary artery pressure, and phosphodiesterase-5 inhibitors like tadalafil (Adcirca, United Therapeutics, Silver Spring, MD) to induce pulmonary arterial vasodilatation.[9] There is some evidence that acetazolamide may prevent calcium ion influx into pulmonary artery smooth muscle cells, leading to reduced hypoxic vasoconstriction. There is conflicting evidence whether glucocorticoids should be a first-line treatment for HAPE.[6]