ALTITUDE SICKNESS

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November 4, 2025

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Primary Disciplinary Field(s): High-Altitude Physiology, Emergency Medicine, Environmental Health, Aerospace Medicine

1. Core Definition

Altitude sickness, clinically known as acute mountain sickness (AMS), represents a spectrum of non-traumatic medical conditions that arise from rapid exposure to high altitudes, typically above 2,500 meters (8,000 feet), where the ambient barometric pressure decreases significantly. This reduction in pressure leads directly to a corresponding decrease in the partial pressure of inspired oxygen (PiO2). Consequently, the body experiences a state of oxygen deprivation, or **hypoxia**, which initiates a cascade of physiological responses designed to compensate for the lack of oxygen delivery to tissues. The severity of the sickness is highly variable, depending on the speed of ascent, the absolute altitude attained, individual genetic predisposition, and the level of physical exertion during the ascent.

The condition is fundamentally a failure of the body to adequately acclimatize to the hypobaric environment in a timely manner. While the percentage of oxygen in the atmosphere remains constant at approximately 21% up to extremely high altitudes, the number of oxygen molecules available per breath diminishes dramatically due to reduced barometric pressure. This diminished availability triggers symptoms ranging from mild discomfort, such as headache and nausea--the classic signs noted in the source material--to life-threatening edema of the brain or lungs. The primary goal of managing altitude exposure is to facilitate effective physiological **acclimatization**, a complex process involving increased ventilation, changes in blood chemistry (e.g., buffering the resultant respiratory alkalosis), and modifications to cardiac output and tissue blood flow.

The common reference to the "nosebleed section" in sports arenas, as noted in the original source, illustrates a minor manifestation related to altitude exposure. While the highest sections of seating in many large arenas are only slightly elevated compared to sea level, this colloquialism speaks to the historical association between high places and minor physical symptoms, such as the spontaneous rupture of capillaries in the nasal mucosa (epistaxis), potentially exacerbated by dry, pressurized air conditioning systems, mimicking the effects observed at truly high altitudes. However, true, clinically significant altitude sickness requires much higher elevations than typical building structures reach.

2. Etiology and Pathophysiology

The underlying cause of all forms of altitude sickness is **hypobaric hypoxia**. As an individual ascends, the barometric pressure drops exponentially. In response to the reduced PiO2, the body

immediately attempts to restore oxygen homeostasis through hyperventilation, a primary compensatory mechanism mediated by the carotid bodies. This increased breathing rate elevates the partial pressure of oxygen in the alveoli (PAO2) but simultaneously results in an excessive removal of carbon dioxide (CO2). The reduction in blood CO2 leads to **respiratory alkalosis**, where the blood pH rises, initially inhibiting the respiratory drive and complicating the acclimatization process.

Pathologically, the severe forms of altitude sickness--High Altitude Cerebral Edema (HACE) and High Altitude Pulmonary Edema (HAPE)--are driven by capillary leakage. In HACE, hypoxia causes cerebral vasodilation, increasing blood flow to the brain in an attempt to maintain oxygen supply. However, this increased flow, coupled with changes in endothelial permeability, allows plasma fluid to leak into the interstitial space of the brain, leading to swelling and elevated intracranial pressure. This edema is responsible for the severe neurological symptoms, including ataxia and cognitive impairment, which the source content identifies as "handicapped cognitive abilities."

In HAPE, the mechanism involves a unique and inhomogeneous vasoconstriction within the pulmonary vasculature, caused by low oxygen levels. This constricted blood flow forces blood into the remaining open capillaries at extremely high pressures, causing mechanical stress that damages the capillary walls. Fluid, including plasma proteins, subsequently leaks into the alveoli, severely impairing gas exchange. HAPE is particularly dangerous because it significantly compromises the primary mechanism (breathing) required for acclimatization, creating a rapidly worsening feedback loop of hypoxia and fluid accumulation in the lungs.

3. Classification and Forms

Altitude sickness is categorized into three distinct syndromes, reflecting increasing severity, all rooted in the same hypoxic exposure: Acute Mountain Sickness, High Altitude Cerebral Edema, and High Altitude Pulmonary Edema. These conditions often occur sequentially or concurrently, though they represent distinct clinical entities demanding specific management protocols. Recognition of the subtle progression from AMS to HACE or HAPE is critical for survival at altitude.

Acute Mountain Sickness (AMS) is the most common and mildest form, resembling a severe hangover. Diagnosis relies on the presence of a headache in an unacclimatized person who has recently ascended above 2,500 meters, accompanied by one or more symptoms from a list including gastrointestinal distress (nausea, vomiting), fatigue, dizziness, or difficulty sleeping. AMS is generally self-limiting, resolving with rest and halting further ascent, but it serves as a crucial warning sign that the body is struggling to adapt. The symptoms of queasiness and general malaise noted in the original description are characteristic features of AMS.

High Altitude Cerebral Edema (HACE) represents a life-threatening progression of AMS,

characterized by severe neurological dysfunction due to cerebral swelling. Key diagnostic indicators include changes in mental status (confusion, lethargy) and, most importantly, **ataxia** (loss of coordination), which renders the victim unable to walk a straight line or perform tandem gait tests. HACE is a medical emergency requiring immediate descent, as delayed treatment can result in coma and death. The "handicapped cognitive abilities" described in the source material are early signs of this dangerous condition.

High Altitude Pulmonary Edema (HAPE) is the most common cause of death related to altitude sickness. It involves the abnormal accumulation of fluid in the lungs, leading to severe difficulty catching one's breath, even at rest. Early signs include persistent dry cough, reduced exercise tolerance, and increasing breathlessness. As HAPE progresses, individuals may produce frothy, blood-tinged sputum, and display signs of severe respiratory distress. Unlike HACE, HAPE can occur without prior severe AMS symptoms, although it frequently accompanies moderate to severe AMS.

4. Key Characteristics and Symptoms

The onset of symptoms typically occurs within 6 to 12 hours after arrival at high altitude, peaking on the second or third day. Recognizing the key characteristics is essential for both self-assessment and group monitoring in remote environments. The spectrum of signs reflects systemic distress caused by hypoxia and compensatory physiological shifts.

Headache: A defining feature of AMS, often throbbing, worsening with activity or lying down, and unresponsive to standard doses of common painkillers unless descent is initiated.
Gastrointestinal Distress: Symptoms such as **nausea**, loss of appetite (anorexia), and occasional vomiting are highly prevalent, contributing to overall weakness and dehydration.
Respiratory Distress: Initially manifesting as difficulty catching one's breath (dyspnea) on exertion, this can progress to severe dyspnea at rest, indicative of developing HAPE.
Neurological Impairment: Mild AMS causes fatigue and insomnia; more severe cases (HACE) involve confusion, disorientation, impaired judgment, and ataxia.
Peripheral Edema: Swelling of the face, hands, and feet is common but not necessarily predictive of severe cerebral or pulmonary edema.
Epistaxis (Nosebleeds): While often minor and related to low humidity and capillary fragility at altitude, persistent or severe nosebleeds may occasionally accompany other symptoms of altitude stress, referencing the common historical association.

The severity of symptoms is assessed using standardized tools, such as the Lake Louise Consensus Score. This scoring system quantifies the presence and intensity of common AMS symptoms, including headache, gastrointestinal symptoms, fatigue, and sleep disturbance. A high Lake Louise score mandates halting ascent and often necessitates immediate descent or

treatment with medications like **acetazolamide**, a carbonic anhydrase inhibitor that speeds up the acclimatization process by inducing metabolic acidosis and stimulating ventilation.

5. Prevention and Acclimatization Strategies

Prevention is the cornerstone of safe high-altitude travel. The most effective preventative measure is a slow, graded ascent, allowing the body adequate time for natural acclimatization. Above 3,000 meters (10,000 feet), experts recommend limiting the daily sleeping elevation gain to no more than 300 to 500 meters (1,000 to 1,600 feet), and incorporating a rest day for every 1,000 meters ascended. This "climb high, sleep low" strategy optimizes acclimatization by exposing the body to higher altitudes during the day while allowing recovery at a lower, more oxygen-rich elevation overnight.

Pharmacological prophylaxis plays a crucial role for individuals who cannot adhere to a slow ascent schedule or who have a history of altitude sickness. Acetazolamide (Diamox) is the drug of choice, typically started 24 hours prior to ascent. It works by increasing the kidney's excretion of bicarbonate, thereby causing a mild metabolic acidosis. This acidosis tricks the brain into believing the CO2 levels are too high, stimulating increased respiration and enhancing the delivery of oxygen. Dexamethasone, a powerful corticosteroid, is also used, primarily for individuals with known severe reactions or as a rescue medication for AMS symptoms, although it masks rather than cures the underlying problem.

Other preventive measures include maintaining adequate hydration, avoiding excessive physical exertion immediately upon arrival at high altitude, and crucially, abstaining from alcohol and sedative medications. Alcohol and sedatives depress the respiratory drive, particularly during sleep, which significantly impairs the body's primary defense mechanism against hypoxia and increases the risk of developing severe forms of altitude sickness. Proper diet, featuring easily digestible carbohydrates, also supports the body's higher metabolic demands at altitude.

6. Treatment and Management

The definitive treatment for all forms of altitude sickness is **descent**. Mild AMS may be treated in place by resting and halting further ascent, but moderate to severe AMS, HACE, or HAPE necessitates immediate and rapid descent to a lower elevation (ideally at least 500 to 1,000 meters). Descent reverses the underlying hypobaric hypoxia and allows the body's equilibrium to restore itself.

For AMS, management includes rest, hydration, and prophylactic medication continuation. For HACE, rapid descent is mandatory, supplemented by high-dose dexamethasone to reduce cerebral edema. Supplemental oxygen, if available, must be administered immediately. If descent is impossible due to weather or injury, a **Gamow Bag**--a portable hyperbaric chamber--can provide

temporary relief by simulating a lower altitude, stabilizing the patient until descent can be safely initiated.

HAPE management is equally urgent and demanding. Treatment involves immediate descent, supplemental oxygen, and the use of medications designed to reduce pulmonary artery pressure, such as **Nifedipine** (a calcium channel blocker) or phosphodiesterase inhibitors (e.g., Sildenafil). These drugs help relax the constricted pulmonary arteries, thereby lowering the pressure that drives fluid into the alveoli. Due to the high risk of mortality, HAPE is always treated as a medical emergency requiring evacuation and specialized care.

Further Reading

Altitude Sickness (Wikipedia)
Altitude Sickness (Mayo Clinic)
High Altitude Travel & Altitude Sickness (CDC)
The Lake Louise Consensus on the Definition and Quantification of Altitude Illness (Wilderness & Environmental Medicine Journal)