ACUTE MOUNTAIN SICKNESS

Authored by

mohammad looti

November 12, 2025

ACUTE MOUNTAIN SICKNESS

Primary Disciplinary Field(s): Wilderness Medicine, High Altitude Physiology, Travel Medicine

1. Core Definition and Etiology

Acute Mountain Sickness (AMS) is a non-infectious, self-limiting disorder that represents a failure of the human body to adequately acclimatize to the decreased availability of oxygen at high altitudes. This condition primarily affects individuals--including **mountain climbers**, **backpackers**, and **winter sport enthusiasts**--who ascend too rapidly above 2,500 meters (approximately 8,000 ft), particularly those starting their ascent from near sea level. The fundamental etiological factor is **hypobaric hypoxia**, defined by the reduction in both atmospheric pressure and the partial pressure of inspired oxygen (PO2). This physiological stressor initiates a cascade of responses that, if insufficient, lead to the characteristic symptoms of AMS, influencing the central nervous system, respiratory system, and cardiovascular function. The severity of AMS is directly proportional to both the rate of ascent and the maximum altitude achieved; the faster the incline and the higher the elevation, the more pronounced and dangerous the symptoms become.

The core mechanism underlying AMS involves the body's struggle to maintain adequate tissue oxygenation when the barometric pressure drops significantly. As atmospheric pressure decreases, the pressure driving oxygen into the lungs and subsequently into the bloodstream diminishes, leading to hypoxemia (low oxygen in the blood). While the body attempts to compensate through increased ventilation (hyperventilation), this process often lags behind the rate of ascent, resulting in cellular stress. This stress can manifest in various organ systems, causing the debilitating symptoms that characterize mild to moderate AMS. Recognition of these early signs is paramount, as failure to halt the ascent and descend can lead to life-threatening complications.

2. Pathophysiology: The Role of Hypobaric Hypoxia

The atmosphere exerts less pressure as altitude increases; consequently, even though the fractional concentration of oxygen remains constant at 21%, the total number of oxygen molecules available for respiration decreases dramatically. At sea level, the partial pressure of oxygen (PO2) is approximately 159 mmHg, but this drops to around 70 mmHg at 5,500 meters (18,000 ft). This substantial pressure gradient reduction impairs the diffusion of oxygen across the alveolar-capillary membrane into the pulmonary circulation, causing arterial oxygen saturation levels to fall. This hypoxemia is the trigger for the body's physiological adjustments, a process known as acclimatization, which involves multiple complex respiratory, cardiovascular, and hematological adaptations over time.

Initially, the primary response to low oxygen is the activation of peripheral chemoreceptors, particularly those in the carotid bodies, which stimulate the respiratory center to increase the rate and depth of breathing (hyperventilation). This compensatory hyperventilation helps to raise the alveolar PO2, but it also causes excessive washout of carbon dioxide (CO2), leading to respiratory alkalosis. The resulting alkalotic state inhibits further respiratory drive, creating a cycle where adequate breathing is difficult, especially during sleep. Furthermore, hypoxia induces vasodilation in certain vascular beds, particularly within the cerebral circulation, increasing cerebral blood flow. While this is initially protective, facilitating better oxygen delivery to the brain, it also raises hydrostatic pressure within the capillaries, contributing to the development of cerebral edema, which underpins the most severe neurological symptoms of AMS.

Beyond the central nervous system, hypoxia affects other critical systems. The cardiovascular system responds by increasing heart rate and cardiac output to move oxygenated blood more rapidly throughout the body. However, the pulmonary vasculature reacts differently; hypoxia causes generalized **pulmonary vasoconstriction**. This response diverts blood flow away from poorly ventilated areas of the lung, but in a generalized hypoxic environment, this leads to an increase in pulmonary artery pressure. This heightened pressure contributes to fluid leakage into the lung tissues, which is the mechanism underlying the potentially fatal complication known as High Altitude Pulmonary Edema (HAPE).

3. Clinical Presentation and Classification

AMS typically manifests within 6 to 12 hours following a rapid ascent, but symptoms can emerge up to 24 hours later. The presentation is highly variable but generally involves a set of non-specific symptoms that mimic influenza or a severe hangover. The most universal symptom, essential for the diagnosis of AMS, is a **headache**. This headache is often refractory to standard over-the-counter analgesics, typically described as throbbing or frontal, and worsens with exertion or bending over. It must be present alongside at least one other symptom from the common list to meet diagnostic criteria for AMS.

The widely accepted standard for diagnosing and grading the severity of AMS is the **Lake Louise Scoring System** (Lake Louise Scoring System). This system assesses symptoms across five main categories: headache, gastrointestinal distress (nausea, vomiting, anorexia), fatigue/weakness, dizziness/lightheadedness, and difficulty sleeping. Mild AMS is characterized by mild headache and one other minor symptom, typically resolving with rest and minor medication. Moderate AMS involves more severe symptoms that impair normal activity and performance, often requiring pharmacological intervention and mandatory halting of further ascent.

Specific symptoms indicate distress in the targeted physiological systems mentioned in the source content. Involvement of the nervous system is evidenced by dizziness and confusion.

Gastrointestinal symptoms, such as nausea and anorexia, reflect visceral responses to hypoxia. Fatigue and generalized muscle weakness are results of overall metabolic strain and reduced oxygen delivery to muscle groups. Sleep disturbances, often manifesting as periodic breathing patterns (Cheyne-Stokes respiration), are a hallmark of initial acclimatization failure and contribute significantly to overall malaise and daytime fatigue.

4. Risk Factors and Susceptible Populations

While AMS can affect anyone ascending quickly above the critical threshold of 8,000 feet, several factors increase an individual's susceptibility. The single most important predictor of AMS is the **rate of ascent** and the altitude attained, underscoring that a fast incline significantly increases the risk compared to a gradual, staged climb. Altitude exposure history is also critical; individuals who have previously suffered from AMS are more likely to experience it again upon subsequent rapid re-exposure. However, fitness level is a poor predictor, as even highly conditioned athletes can develop severe AMS if they ignore the principles of gradual acclimatization.

Pre-existing medical conditions, particularly those affecting pulmonary or cardiovascular function, such as chronic obstructive pulmonary disease (COPD) or congenital heart defects, can exacerbate the hypoxic stress and increase risk. Age plays a complex role; while historically thought to spare the elderly, younger individuals (especially children) may be at higher risk due to potentially greater exertion or inability to articulate symptoms effectively. Furthermore, certain behaviors and environmental conditions contribute significantly to risk, including dehydration, alcohol consumption, use of sedatives (which further depress respiratory drive), and the presence of extreme cold, which increases metabolic demands. The ascent profile, particularly the "sleep altitude" (the elevation at which one spends the night), is considered a more significant risk indicator than the maximum elevation reached during the day.

5. Progression to Severe Complications (HACE and HAPE)

While mild AMS is common and usually resolves easily, failure to recognize the worsening symptoms and initiate descent can lead to severe, life-threatening forms of altitude illness: High Altitude Cerebral Edema (HACE) and High Altitude Pulmonary Edema (HAPE). As noted in the source material, in a small but critical percentage of individuals--approximately 2 out of every 10,000 ascending to extreme altitudes--edema takes place surrounding the human brain, prompting bafflement and potentially leading to comatoseness. This condition is HACE.

High Altitude Cerebral Edema (HACE) represents the end stage of severe AMS. It is caused by widespread swelling of the brain tissue due to increased capillary permeability and fluid leakage (vasogenic edema) within the skull, exacerbated by increased cerebral blood flow as the body attempts to compensate for hypoxia. Early signs of HACE include severe ataxia (inability to

coordinate voluntary muscle movements, leading to gait instability), severe personality changes, extreme lethargy, and progressive confusion (bafflement). Untreated, HACE rapidly progresses to stupor, seizures, and ultimately, coma and death. HACE is a true medical emergency requiring immediate descent and administration of high-dose corticosteroids, such as Dexamethasone.

The other major complication is **High Altitude Pulmonary Edema (HAPE)**, which involves the accumulation of fluid in the air sacs (alveoli) of the lungs, severely impairing gas exchange. HAPE symptoms include severe shortness of breath at rest, a persistent, wet cough (sometimes producing pink, frothy sputum), chest tightness, and profound fatigue. HAPE is caused by non-uniform, severe pulmonary vasoconstriction leading to extremely high pulmonary artery pressures, forcing fluid out of the capillaries and into the lung interstitium. Both HACE and HAPE can occur independently or concurrently, and both necessitate immediate, mandatory descent as the definitive treatment, often combined with supplemental oxygen and specific medications (Nifedipine for HAPE; Dexamethasone for HACE).

6. Prevention and Acclimatization Strategies

Prevention is the most effective approach to managing Acute Mountain Sickness. The cornerstone of prevention is **gradual staged ascent**, allowing the body sufficient time to undergo the necessary physiological adaptations--a process known as acclimatization. Standard recommendations suggest that above 2,500 meters (8,000 ft), climbers should limit their daily gain in sleeping altitude to no more than 300 to 500 meters (1,000 to 1,600 ft). Additionally, a mandatory rest day should be taken every 3 to 4 days, or for every 1,000 meters gained, during which the individual should "climb high, sleep low," meaning they hike to a higher altitude during the day but return to a lower altitude to sleep.

Pharmacological prophylaxis plays a significant role, particularly for those who must ascend quickly or who have a known history of AMS. The most widely used prophylactic medication is **Acetazolamide** (Diamox), a carbonic anhydrase inhibitor. Acetazolamide works by increasing bicarbonate excretion in the urine, creating a mild metabolic acidosis. This acidosis stimulates the peripheral chemoreceptors, promoting increased ventilation and accelerating the body's natural acclimatization process, thereby improving nocturnal oxygen saturation levels and reducing AMS symptoms. Treatment usually begins 24 hours prior to ascent and continues for the first few days at altitude.

Beyond staged ascent and medication, maintaining adequate **hydration** and nutrition is crucial. Since the air at altitude is cold and dry, respiratory water loss is significantly increased. Dehydration can exacerbate symptoms of headache and fatigue, often masking true AMS symptoms. Individuals should avoid depressants, such as alcohol and sedatives, especially during the first 48 hours at altitude, as these substances depress the respiratory drive and worsen

nocturnal hypoxia, significantly increasing the risk of severe altitude illness.

7. Treatment and Management Protocols

The management of AMS is dictated by its severity. For mild AMS, treatment typically involves rest at the current altitude, hydration, and symptomatic relief using mild analgesics (like ibuprofen or acetaminophen) for the headache. Crucially, the individual should not ascend further until all symptoms have resolved. Resolution usually occurs within 24 to 48 hours if no further ascent is attempted.

For moderate AMS, immediate pharmacological intervention is necessary, often involving Acetazolamide (if not already used prophylactically) and Dexamethasone, a powerful corticosteroid. Dexamethasone reduces inflammation and cerebral edema, rapidly alleviating AMS symptoms. The single most important management principle for moderate or severe AMS (HACE or HAPE) is **descent**. A descent of even 500 to 1,000 meters (1,600 to 3,300 ft) often results in a dramatic and immediate improvement in symptoms because of the exponential increase in oxygen availability at lower elevations.

In emergency situations, particularly involving HACE or HAPE where immediate descent is impossible, supplementary treatments must be initiated. These include the administration of **supplemental oxygen** to raise arterial saturation above 90% and the use of portable hyperbaric chambers (like the Gamow Bag). These chambers simulate a descent of several thousand feet by increasing the ambient pressure around the patient, offering a temporary, life-saving measure until a physical descent can be achieved. Failure to achieve rapid relief or resolution of neurological symptoms (confusion, ataxia) demands immediate, urgent evacuation to a lower altitude medical facility.

Further Reading

Acute Mountain Sickness - Wikipedia
Altitude Illness: Acute Mountain Sickness, HACE, and HAPE - CDC
International Society for Mountain Medicine (ISMM) Prevention Guidelines
Mountain sickness: Symptoms and causes - Mayo Clinic