# CLINICAL REVIEW

# Acute altitude illnesses

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Acute altitude illnesses are potentially serious conditions that can affect otherwise fit individuals who ascend too rapidly to altitude. They include high altitude headache, acute mountain sickness, high altitude cerebral oedema, and high altitude pulmonary oedema. The number of people travelling to altitude for work (soldiers, miners, construction workers, and astronomers) or for recreation (skiing, trekking, mountain biking, and climbing) is rising, and increased media attention towards these activities has also raised the profile of altitude related illness. Typical scenarios in which such illness might occur are a family trek to Everest base camp in Nepal (5360 m), a fund raising climb of Mount Kilimanjaro (5895 m), or a tourist visit to Machu Picchu (2430 m). Awareness of potential altitude related problems is important even for healthcare practitioners working at lower altitude, because patients may ask for advice about the safety of a proposed journey and how to prevent illness at altitude.

## What is high altitude and why is it a problem?

The definition of high altitude varies and depends not only on the absolute altitude attained but also on the rate of ascent. A useful and widely accepted classification of altitude is presented in the box.

Rate of ascent to altitude is as important as the absolute altitude itself. Although the percentage of oxygen in the atmosphere remains constant at 20.94% as altitude increases, the barometric pressure falls steadily, mirrored by a fall in inspired or available oxygen (fig 1).

Acclimatisation is the process by which an individual adapts to high altitude and includes a number of physiological changes that occur over a variable time course (fig 2).[4] Some changes take place within minutes while others take several weeks. At intermediate to high altitudes, a significant degree of acclimatisation takes place over two to four days. With a few exceptions, most altitude related illnesses occur when there has been insufficient time to acclimatise at altitudes over 3000 m.

## What are the altitude related illnesses?

The common altitude specific illnesses are high altitude headache and acute mountain sickness. Much rarer, but more serious, are high altitude cerebral oedema and high altitude pulmonary oedema.

### High altitude headache

High altitude headache is defined by the International Headache Society as a headache that develops within 24 hours of ascent above 2500 m and resolves within 8 hours of descent.[w2] The headache often worsens during the night and with exertion. Unlike a common migraine, it resolves after 10-15 minutes of supplementary oxygen therapy (2 L per minute). A prospective observational study estimated that 80% of people who ascend to high altitudes are affected by high altitude headache.[w3] Most high altitude headaches resolve with analgesic treatment (paracetamol or ibuprofen). Ascent to altitude often results in dehydration, owing to exercise, hyperventilation, and limited access to water,[w2] so an important early step is to ensure adequate hydration. The person may need to stop the ascent or descend to lower altitude if the headache does not improve with simple analgesia.

### Acute mountain sickness

Acute mountain sickness is a symptom complex characterised by headache and at least one of nausea/vomiting, fatigue, dizziness, and difficulty sleeping, appearing 6-12 hours after arrival at high altitude and usually resolving within 1-3 days.[5] Many people who usually live at sea level are surprised on their first encounter with acute mountain sickness by debilitating tiredness, which may be compounded by sleeping difficulties. Individuals may note decreased urine output independent of

**Summary points**

- High altitude headache and acute mountain sickness often occur a few hours after arrival at altitudes over 3000 m

- Occurrence of acute mountain sickness is reduced by slow ascent, and severity can be modified by prophylactic acetazolamide

- Mild to moderate acute mountain sickness usually resolves with rest, hydration, halting ascent, and analgesics

- Occasionally people with acute mountain sickness develop high altitude cerebral oedema with confusion, ataxia, persistent headache, and vomiting

- Severe acute mountain sickness and high altitude cerebral oedema require urgent treatment with oxygen if available, dexamethasone, possibly acetazolamide, and rapid descent

- High altitude pulmonary oedema is a rare but potentially life threatening condition that occurs 1-4 days after arrival at altitudes above 2500 m; treatment should include oxygen if available, nifedipine, and rapid descent to lower altitude

- Treat for both high altitude pulmonary and cerebral oedema if in doubt

**Sources and selection criteria**

We searched Medline and Google Scholar with no date limitations using the term "acute mountain sickness" to obtain references that form the basis of this article. We included only articles that were well written, had high clinical importance, and (where relevant) were referenced from soundly conducted clinical trials. However, field studies are often difficult to perform, and numbers of study participants are often small. Hypoxic chamber studies yield reliable results on the acute effects of hypoxia but are often limited to a few hours and results are not always applicable to high altitude. Comparing studies is difficult because of differing ascent profiles, altitudes achieved, and uncontrolled exercise and diet.

**Definitions of altitude and associated physiological changes[1]**

*Intermediate altitude: 1500-2500 m*

 Physiological changes detectable. Arterial oxygen saturation >90%. Altitude illness rare but possible with rapid ascent, exercise, and susceptible individual

*High altitude: 2500-3500 m*

 Altitude illness common when individuals ascend rapidly

*Very high altitude: 3500-5800 m*

 Altitude illness common. Arterial oxygen saturation <90%. Marked hypoxaemia during exercise. 5800 m is altitude of the highest permanent habitation

*Extreme altitude: >5800 m*

 Marked hypoxaemia at rest. Progressive deterioration despite maximal acclimatisation. Permanent survival is not thought to be possible

*"Death zone": >8000 m*

 Prolonged acclimatisation (>6 weeks) is essential. Most mountaineers require supplementary oxygen to climb safely. Arterial oxygen saturations about 55%.[2] Rapid deterioration is inevitable and time spent above this altitude is strictly limited[w1]

Adapted from *The High Altitude Medicine Handbook*[1]

fluid intake.[w4] The prevalence of acute mountain sickness depends on an individual's susceptibility, the rate of ascent, and the absolute altitude achieved. In a prospective observational study 84% of people who flew to 3740 m developed acute mountain sickness,[w5] while about 50% of trekkers who walk to altitudes higher than 4000 m in the same region over five or more days develop acute mountain sickness.[w6 w7]

## High altitude cerebral oedema and pulmonary oedema

The cerebral effects of ascent to high altitude have been reviewed recently.[3] In an observational field study, the fall in cerebral oxygenation (assessed with near infrared spectroscopy) was similar to the fall in arterial oxygenation.[6] Progression of acute mountain sickness to high altitude cerebral oedema is marked by altered mental status, including impaired mental capacity, drowsiness, stupor, and ataxia. Coma and ultimately death may occur as soon as 24 hours after the onset of these symptoms. These features usually allow a confident diagnosis of cerebral oedema, although mental confusion and ataxia can also present in patients with hypothermia, hypoglycaemia, or alcohol intoxication and should be ruled out.

High altitude pulmonary oedema manifests as a non-cardiogenic form of pulmonary oedema and is not necessarily preceded by acute mountain sickness. It usually occurs between one and four days after arrival at altitudes above 2500 m.[7] Patients may report a greater reduction in exercise tolerance than might be expected for the altitude (a consequence of impaired alveolar gas exchange) followed by a dry cough, which then becomes productive with blood stained sputum. Crackles may be present on auscultation of the chest. Profound hypoxaemia and death may occur if the condition is not treated.

## Who gets acute altitude illness?

Prospective observational studies and anecdotal reports have shown that some individuals are more prone to high altitude illness than others, and variants of at least eight genetic polymorphisms show positive associations suggesting that acute mountain sickness is an environmentally mediated polygenic disorder.[8] Because of the polygenic nature of the human response to hypobaric hypoxia, several genetic loci, each with a small contribution, probably define the phenotype.[w8] Possible candidate genes include D7S477, the erythropoietin response gene, HIF-1$\alpha$, NOS3, which encodes for nitric oxide synthase III, and CYP 11B2, which encodes aldosterone synthase and angiotensin converting enzyme polymorphism.[3w9]

An individual's past performance at altitude is the main predictor of their future performance. A recent prospective observational study found that risk of developing acute mountain sickness was associated with better aerobic capacity, younger age, and higher body mass index.[9] An observational field study suggested that young people are more affected by altitude related illness than their parents,[10] but other studies show no such differences.[w10, w11] Obesity was associated with development of acute mountain sickness in a chamber study,[11] and the additional exertion required of obese individuals under field conditions could be an aggravating factor. No clinical feature or test has been shown to predict an individual's susceptibility with any reliability. Arterial oxygen saturation determined 20-30 minutes after exposure to simulated hypoxia, equivalent to that at 2300-4200 m altitude, seems to be the best predictor of susceptibility.[12] Anaemia is not associated with an increased risk of acute mountain sickness.[w4] However, in a prospective observational field study inducing anaemia reduced exercise performance.[w12]

High altitude cerebral oedema is rare and usually occurs only at altitudes over 4000 m. In an observational field study of 1925 soldiers studied at altitudes from 3350 to 5000 m, 23 men (1.2%) developed high altitude cerebral oedema.[13] Similarly, in another study only five (1.8%) of 278 trekkers were diagnosed with high altitude cerebral oedema at 4243 m.[14] The only study to directly measure intracerebral pressures at high altitude was performed by the late Brian Cummings during a climbing expedition to Kishtwar. The data were lost for years and only recently discovered and published.[w13] The three climbers studied showed normal intracranial pressures at rest at high altitudes. However, at 4725 m the youngest climber had a striking rise in intracranial pressure during any form of mild exertion.

In observational field studies, the prevalence of high altitude pulmonary oedema was shown to be 0.2% in a general population ascending to altitude,[w14] and rose to 4% among trekkers and climbers ascending at a rate of 600 m per day[w15]; 7% of climbers without a history of high altitude pulmonary oedema developed symptoms shortly after arrival at 4559 m, whereas 62% of climbers with a history of radiographically proved high altitude pulmonary oedema developed the condition.[w16] Risk factors include individual susceptibility (as shown by a previous episode), pulmonary hypertension, structural abnormalities of the pulmonary circulation (such as unilateral absence of a pulmonary artery), exercise, and exposure to cold. A rise in pulmonary artery pressure is a normal physiological response to hypoxia and exercise. Individuals susceptible to high altitude pulmonary oedema seem to have a more exaggerated rise in pulmonary artery pressure, and, more importantly, regional pulmonary blood flow becomes non-uniform as a result of a mismatch between the ventilation and perfusion of the lungs (V/Q mismatching).[7w4 w17]

## How does altitude related illness develop?

The exact pathogenesis of acute mountain sickness is unknown.[5] Because headache, nausea, lethargy, and mild ataxia are common features of more severe acute mountain sickness and of early high altitude cerebral oedema, it has been suggested that the two conditions represent different points on the same continuum.[3 5] The hypoxic drive to breathe was proposed as the key to acclimatisation and an indicator of susceptibility to altitude illness, but further study by independent groups showed that although it is better to breathe more deeply and frequently at high altitude, this response varies greatly and is not a sole predictor of either acclimatisation or of risk of altitude illness.[w18 w19]

Exercise and ascent to altitude can affect overall fluid balance. Plasma volume has been shown in field and chamber studies to decrease with exposure to altitude.[w20 w21 w22] In a chamber study, hypoxia led to an acute rise in haemoglobin concentration, indicating an initial increase in vascular permeability with subsequent leakage of water out of the vascular space.[w23] As a consequence, generalised and dependent tissue oedema may develop.

Increased sympathetic drive results in raised heart rate and blood pressure. In a prospective observational chamber study, cerebral vascular autoregulation was impaired by hypoxia, but this does not seem to have an important role in acute mountain sickness.[15] Cerebral oedema begins to develop as the severity of acute mountain sickness increases.

## How is acute mountain sickness diagnosed?

In 1991, a committee of experts proposed a consensus scoring system for assessing the severity of symptoms, known as the Lake Louise acute mountain sickness scoring system (table 1).[16] It was originally designed as a research tool but has been more widely adopted for use in the field. The Lake Louise questionnaire has limitations; it may not always be understood by those whose first language is not English and may be too complex for use with young children. Fewer symptoms of acute mountain sickness were reported using the standard Lake Louise questionnaire compared with a questionnaire using age appropriate language or visual representations.[17] A simple visual analogue score has also been reported to be a simple effective measure of the severity of acute mountain sickness.[18]

Symptoms of acute mountain sickness are commonly misattributed to viral infection, alcohol hangover, exhaustion, or dehydration. However, fever is often absent in acute mountain sickness. Use of alcohol or other drugs should be excluded when taking a history. If rest and rehydration do not improve symptoms, fatigue and dehydration are unlikely to be the primary cause. A patient with relevant symptoms who has recently ascended to a new altitude is likely to have altitude sickness, and we suggest that such patients should be treated for altitude sickness until another disease process is proved.

## Can acute altitude illness be prevented?

### Special advice for certain groups

There is limited evidence to support giving special advice to any particular group of people travelling to high altitudes. Since acute mountain sickness tends to recur in susceptible individuals, a positive history may prompt a warning about possible recurrence. *Travel at High Altitude: a Guide to Staying Healthy*

includes advice for patients with pre-existing chronic medical conditions.[19]

## Slow ascent

Prospective observational studies suggest that ascending slowly and allowing time for acclimatisation is the best way of preventing acute mountain sickness. The Wilderness Medical Society[w24] advises that individuals travelling above 3000 m should ascend at less than 300 m per day, with a rest day for every 1000 m climbed.[20] In one study, the benefits of acclimatisation after five days at 4200 m were lost within a few days of returning to a lower altitude;[21] however, others suggest a more attenuated decline.[w25] Recently an uncontrolled observational study of people doing construction work at high altitude reported that repeated seven month exposures to high altitude protected lowlanders against acute mountain sickness with increasing effect over time, even when interspaced with five month periods spent at low altitude.[22]

## Hypoxic preconditioning

There is no evidence to suggest physical fitness protects against acute mountain illnesses. However there is a growing interest in the use of hypoxic pre-conditioning for the prevention of acute mountain sickness, although currently the supporting evidence base is limited. In an uncontrolled intervention study in which individuals underwent daily pre-acclimatisation using a hypobaric chamber for three weeks and were re-exposed eight days later to altitudes of 4300 m, subsequent symptoms of acute mountain sickness were attenuated.[23] More recently the same group found increased exercise tolerance at 4300 m after six days of pre-acclimatisation to 2200 m altitude conditions[24] and reduced incidence and severity of acute mountain sickness during subsequent rapid ascent to 4300 m.[25] In a recent double blind placebo controlled trial hypoxic preconditioning was effective at reducing the incidence of acute mountain sickness on ascent to 3611 m but there was no observed benefit on further ascent to 4559 m.[26]

## Pharmacological agents

Improving oxygenation with drugs such as carbonic anhydrase inhibitors (for example, acetazolamide), or attenuating cytokine and inflammatory responses (for example, with glucocorticoids or antioxidants) are the two main pharmacological approaches to the prevention of acute mountain sickness and high altitude cerebral oedema.[5w24]

### Carbonic anhydrase inhibitors

Acetazolamide leads to bicarbonate diuresis through inhibition of renal carbonic anydrase. The resulting metabolic acidosis stimulates ventilation. It is also thought to reduce the production of cerebrospinal fluid, which may contribute to the beneficial effects. Meta-analysis and systematic reviews of observational field studies have shown that prophylactic acetazolamide in doses between 125 mg and 1 g per day (usually prescribed as 125 mg or 250 mg twice daily) reduces symptoms of acute mountain sickness.[27 28] However, the drug does not prevent symptoms of acute mountain sickness when ascent is too fast; for example 500 mg/day may not prevent acute mountain sickness in some individuals climbing at the typical rate of ascent on Kilimanjaro. Climbers must begin taking acetazolamide at least one day before ascent and continue until descent has begun. Side effects of acetazolamide—which include parasthesia, diuresis lasting 24-48 hours, and an unpleasant metallic taste to carbonated drinks—are usually well tolerated,

and allergic reactions are rare. We advise anyone who plans to use acetazolamide at high altitude to obtain the medication from a reliable source and to test their tolerance of the drug by taking a trial dose at sea level before travelling.

### Glucocorticoids

The exact mechanism of action of glucocorticoids such as dexamethasone is unknown, but some authors have speculated that they reduces capillary permeability and cytokine release.[29] Dexamethasone 8 mg daily in divided doses has been used to prevent acute mountain sickness; lower doses are less effective. Because the potential side effects of glucocorticoids are thought to outweigh the benefits, they are not normally justified for prophylaxis. Exceptions are if acetazolamide is contraindicated or when a very rapid effect is required—for example, when rescue workers are called on to ascend very quickly.

### Ginko biloba

Ginko biloba is a traditional Chinese medicine that contains flavonol glycosides and terpene lactones, which, among many effects, scavenge excess free radicals. There is conflicting evidence of its effectiveness in the prevention of acute mountain sickness. A prospective randomised placebo controlled study showed a benefit of ginko biloba (80 mg twice daily starting 24 hours before altitude exposure).[w26] However, a larger randomised placebo controlled study found that ginko biloba (120 mg twice daily) was not effective compared with acetazolamide (250 mg twice daily) and placebo.[w27] The lack of a standardised chemical preparation for gingko biloba may partly account for the variation in results.[30]

## How is acute mountain sickness treated?

When practical, accompanied descent by 300-1000 m remains the most effective treatment for all high altitude illnesses. Other treatments for acute mountain sickness reflect the varying severity of the clinical symptoms. For milder cases (Lake Louise score 3-4) rest and maintenance of hydration are best combined with symptomatic relief of headache with ibuprofen. For moderate to severe acute mountain sickness (Lake Louise score 5 or more) acetazolamide, glucocorticoids, or both are the best acute therapy, provided that the patient has not taken these drugs prophylactically (fig 3).[w24 w28] A randomised placebo controlled field study of acute therapy with acetazolamide showed improvement in overall acute mountain sickness scores, although relief of symptoms may take 24 hours and headache may worsen temporarily.[w29] In 2010, the Wilderness Medical Society recommended acetazolamide 250 mg every 12 hours for the treatment of acute mountain sickness in adults.[w24] The medication can be discontinued once symptoms resolve. They advise that children may take 2.5 mg/kg body weight every 12 hours.

Dexamethasone (8 mg initially and 4 mg every six hours orally or parenterally) often improves the symptoms of acute mountain sickness sufficiently to allow safe evacuation. The ideal duration of treatment after the patient has descended is unknown. We advise that when dexamethasone is given alone it should be continued while the risk of acute mountain sickness or high altitude cerebral oedema remains. The combination of acetazolamide 500 mg with dexamethasone 4 mg twice daily was more effective than acetazolamide alone at treating acute mountain sickness in a small group of participants.[31] When 31 people were randomised to either a simulated descent of 2250 m in a portable hyperbaric chamber for one hour or dexamethasone for the treatment of acute mountain sickness, 8

mg dexamethasone initially and 4 mg six hourly resulted in longer term clinical improvement and reduction in Lake Louise scores.[32]

In field studies, raising arterial oxygen saturation rapidly improves the symptoms of acute mountain sickness. Portable hyperbaric therapy and continuous positive airway pressure ventilation both raise the relative inspired pressure of oxygen, assisting gas exchange and resulting in improved arterial oxygen saturations.[w30 w31 w32] Recent novel approaches to enhance individuals' oxygen saturation include oxygenating train cars on the Qinghai-Tibet railway (maximum altitude 5072 m)[33] and 6% oxygen enrichment of room air in a high altitude observatory.[34]

## How is high altitude cerebral oedema treated?

The cranial vault is a rigid structure with a fixed volume, containing the brain, cerebral blood vessels, and cerebrospinal fluid, all which have variable volumes. Some individuals can "buffer" or accommodate much larger volume changes than others, and they are described as having compliant brains. Others, in whom a relatively small volume change causes an early rise in intracerebral pressure, are said to have tight brains. Ross[35] proposed that variation between individuals in neuroaxis compliance (the ability to cope with brain swelling) accounted for the variability in susceptibility to acute mountain sickness. High altitude cerebral oedema is a life threatening condition in which the individual's neuroaxis compliance has been exceeded as intracerebral pressure rises rapidly (fig 4). Management consists of immediate accompanied descent (300-1000 m) ideally using mechanised transport. Administration of oxygen (2-4 L/m) and dexamethasone (8 mg immediately followed by 4 mg orally or parenterally every 6 hours) is recommended.[w24] If descent is not possible and the airway can be protected, a portable hyperbaric chamber could be used.

## How is high altitude pulmonary oedema managed?

High altitude pulmonary oedema (fig 5) is often, but not always, preceded by acute mountain sickness. It is a potentially life threatening condition requiring urgent medical intervention. When practical, accompanied descent by 300-1000 m is the most effective treatment, again ideally by being carried down. The physiological rise in pulmonary artery pressure in response to hypoxia is likely to be an important factor, and drugs that lower pulmonary artery pressure are beneficial. Nifedipine 30 mg sustained release twice daily is effective for both prevention and treatment.[w24 w33] Tadalafil 10 mg twice daily and sildenafil 50 mg three times a day are effective in prevention.[w24 w33] In a randomised placebo controlled study the β agonist salmeterol (125 mg twice daily) decreased the incidence of high altitude pulmonary oedema.[w34] In a field study, expiratory positive airway pressure improved arterial oxygenation in individuals with high altitude pulmonary oedema.[w35] There is no evidence to support the use of diuretics in the treatment of high altitude pulmonary oedema.

## What is the role of supplementary oxygen in treatment of high altitude illness?

Supplementary oxygen is an important adjunct to the treatment of high altitude illnesses. Bottled oxygen is light and portable but requires suitable connectors and can run out. Portable oxygen concentrators can be powered by solar or hydroelectric energy, allowing them to be used in remote clinics or on large expeditions. Portable hyperbaric chambers—lightweight airtight bags that simulate descent—are now carried by many larger expeditions. Patients are zipped into the bag and the air pressure within the bag is raised using a continuous foot or hand pump.[4] Pressurising the bag to approximately 200 mBar simulates a descent of about 2000 m.[w37] Any patient placed in the bag needs to be able to protect their airway and equalise their middle ear pressure.

## Conclusion

Acute mountain sickness is a relatively common condition that can affect any otherwise fit individual who ascends to altitude too quickly. The clinical course is usually self limiting, responding to simple measures. However, failure to descend or, worse still, further ascent is likely to exacerbate the condition and can precipitate much more serious and potentially fatal conditions: high altitude cerebral oedema and high altitude pulmonary oedema. Gradual ascent, or using acetazolamide to aid in acclimatisation when time is short, should lead to healthy travel at high altitude.

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**Additional educational resources**

*For doctors*

- Luks AM, McIntosh RE, Grissom CK, Auerbach PS, Rodway GW, Schoenr RB, et al. Wilderness Medical Society consensus guidelines for the prevention and treatment of acute altitude illness. *Wilderness and Environmental Medicine* 2010;21:146-55—a contemporaneous overview citing the levels of evidence behind each recommendation

- Pollard AJ, Murdoch DR. *The High Altitude Medicine Handbook*. 3rd ed. Radcliffe Publishing, 2003—a practical short text, with an excellent section on children at altitude. A pocket sized edition is available in Nepal

- Johnson C, Anderson S, Dallimore J, Winser S, Warrell DA. *Oxford Handbook on Expedition and Wilderness Medicine*. Oxford University Press, 2008—a pocket sized book to take on expeditions

- Milledge JS, West JB, Schoene RB. *High Altitude Medicine and Physiology*. 4th ed. Hodder Arnold, 2007—one of the major reference texts

- Hornbein T, Schoene R. *High Altitude—an Exploration of Human Adaptation*. Informa Healthcare, 2001: p 982—one of the major reference texts; a new edition is due out shortly

*For climbers and travellers*

- Medex (www.medex.org.uk)—free handbook *Travel at High Altitude* available to download, aimed at lay people and small enough to take away travelling

- List of mountain medicine diploma holders (http://medex.org.uk/diploma/diploma_holders.php)—many are available for advice

- Altitude advice sheets from the British Mountaineering Council (www.thebmc.co.uk/feature.aspx?id=4156)—specific information for the most popular high altitude peaks in the world, as well as general advice on altitude illnesses

**Ongoing research and unanswered questions**

Can a sea level test be developed to predict individuals' susceptibility to acute mountain sickness?

Which genetic factors predispose to acute mountain sickness?

Is there an optimal and effective hypoxic training regimen in the prophylaxis of acute mountain sickness?

What is the optimal dose of acetazolamide for prevention and treatment of acute mountain sickness?

What causes high altitude cerebral oedema?

What is the most effective drug prophylaxis for high altitude pulmonary oedema?

Why do the Sherpas and other people indigenous to high altitudes excel in these environments?

**A patient's perspective**

We climbed Mount Kilimanjaro during our medical elective. Although it is a straightforward trek, with no technical climbing, the rapid ascent rate makes altitude sickness a common problem. We took the longer Machame (or "whisky") route, which included an extra day to acclimatise to reduce the chance of altitude sickness. The trek begins at 1490 m and finishes at the summit at 5895 m on the fifth day, giving an ascent rate of almost 800 m a day.

We first began to feel the effects of the altitude at the final base camp at 4550 m, which we reached at around 15:00 on the fourth day. With nine hours to rest before the final ascent, it is important to eat and sleep at this point, but the altitude suppressed our appetites and made sleeping very difficult. Once we began the final climb, the gain in altitude led to further unpleasant symptoms, including shortness of breath, headache, and nausea. We reached the summit in a daze, with heads pounding and our food and drink frozen. The combination of altitude sickness and the low temperature meant we quickly turned round to descend. Thankfully, the relief from the symptoms of altitude sickness was rapid with each step down the mountain, clearing our heads and easing our breathing.

David O'Connor

**Tips for non-specialists**

- Unexplained symptoms after recent altitude gain should be treated as acute mountain sickness until proved otherwise

- Advising patients about the use of acetazolamide off licence can be difficult; it is more important to advise a sensible rate of ascent

- If prescribing acetazolamide for use at altitude, suggest that the person try it at sea level before going away, to make sure that they can tolerate the side effects

- On expeditions where individuals may be separated from the medical kit or medical officer, consider supplying each climber with an emergency medical kit and instructions on how to treat altitude related illnesses

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# Table

**Table 1**| **Lake Louise acute mountain sickness questionnaire. An individual has acute mountain sickness as assessed by the Lake Louise self-assessment scoring system if they fulfil the following criteria (A) recent ascent in altitude, (B) headache present, and (C) total symptom score above 3**

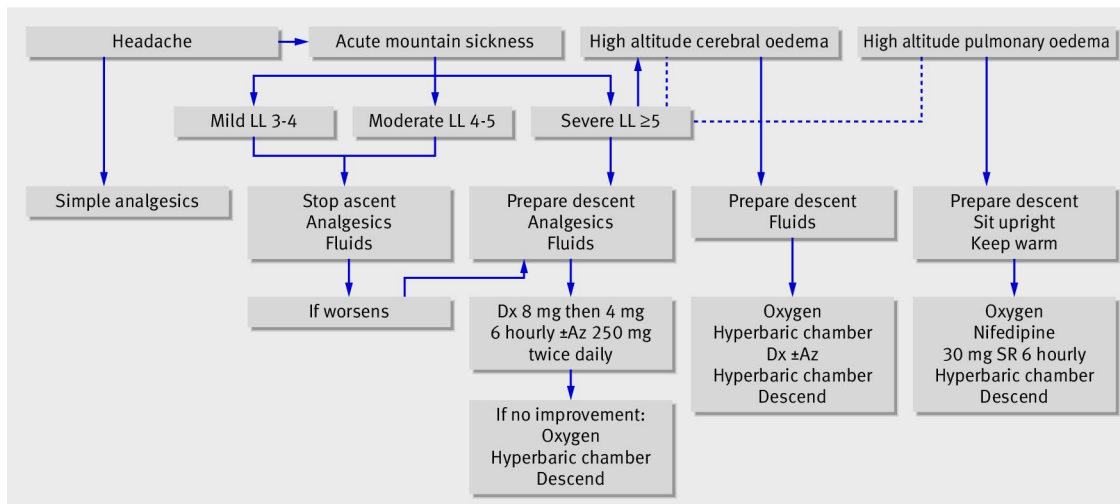|  | Score |
|---|---|
| **Symptom** | |
| Headache | |
| No headache | 0 |
| Mild headache | 1 |
| Moderate headache | 2 |
| Severe, incapacitating | 3 |
| Gastrointestinal symptoms: | |
| No gastrointestinal symptoms | 0 |
| Poor appetite or nausea | 1 |
| Moderate nausea or vomiting | 2 |
| Severe nausea and vomiting incapacitating | 3 |
| Fatigue/weakness: | |
| Not tired or weak | 0 |
| Mild fatigue/weakness | 1 |
| Moderate fatigue/weakness | 2 |
| Severe fatigue/weakness, incapacitating | 3 |
| Dizzy/light-headedness: | |
| Not dizzy | 0 |
| Mild dizziness | 1 |
| Moderate dizziness | 2 |
| Severe, incapacitating | 3 |
| Difficulty sleeping: | |
| Slept well as usual | 0 |
| Did not sleep as well as usual | 1 |
| Woke many times, poor night's sleep | 2 |
| Could not sleep at all | 3 |
| **Clinical assessment** | |
| Change in mental status: | |
| No change | 0 |
| Lethargy/lassitude | 1 |
| Disoriented/confused | 2 |
| Stupor/semi consciousness | 3 |
| Ataxia (heel to toe walking): | |
| No ataxia | 0 |
| Manoeuvres to maintain balance | 1 |
| Steps off line | 2 |
| Falls down | 3 |
| Can't stand | 4 |
| Peripheral oedema: | |
| No oedema | 0 |
| One location | 1 |
| Two or more locations | 2 |

# Figures
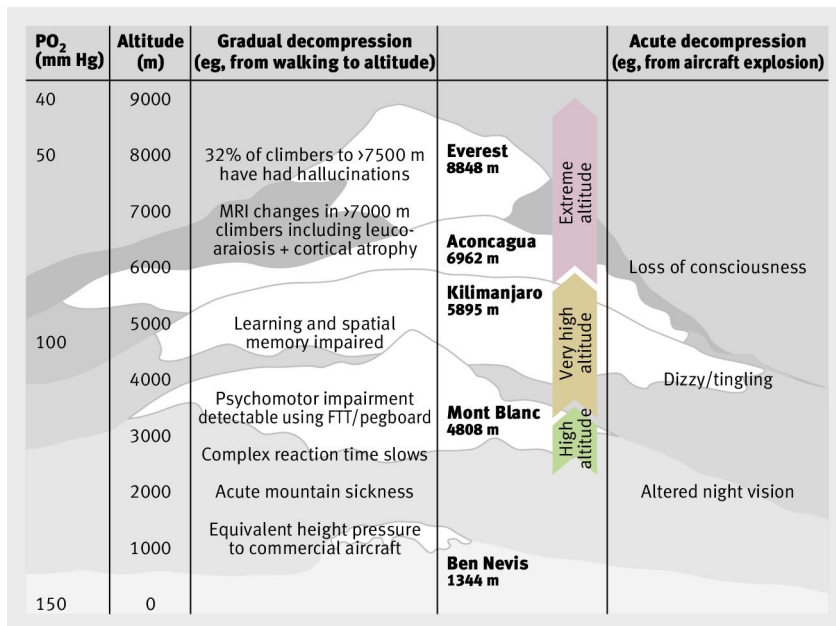


**Fig 1** Changes in partial pressure of inspired oxygen (P0$_2$) and clinical effects of exposure with increasing altitude. High altitude cerebral oedema is more common at higher altitudes, although it has been reported at 2500 m. FTT=finger tapping test. Adapted from Wilson et al[3]
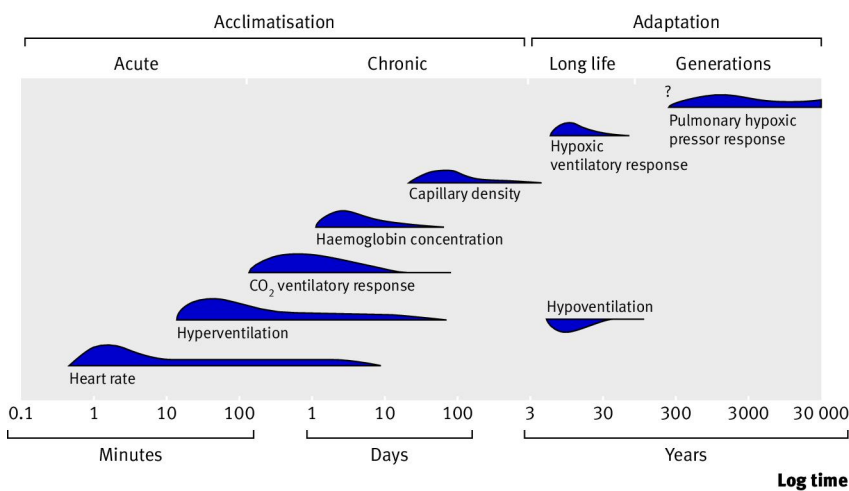


**Fig 2** Physiological adaptations to altitude and time course. Time course of acclimatisation and adaptive changes plotted on log time scale. The curve of each response denotes the rate of change. Adapted from Peacock AJ[4]

**Fig 3** Treatment algorithm for high altitude illness. LL=Lake Louise score, Dx=dexamethasone, Az= acetazolamide, SR=sustained release. Adapted from Davis PR, Pattinson KT, Mason NP, Richards P, Hillebrandt D. High altitude illness. *J R Army Med Corps* 2005;151:243-9
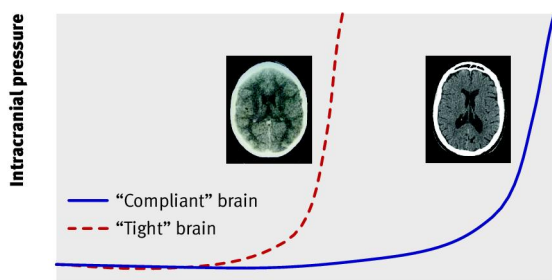


**Fig 4** "Tight fit hypothesis" of high altitude cerebral oedema.[3] Lack of cerebrospinal fluid buffer is thought to result in a predisposition to earlier rise in intracranial pressure when there is oedema formation or increased blood volume. Adapted from Wilson et al[3]
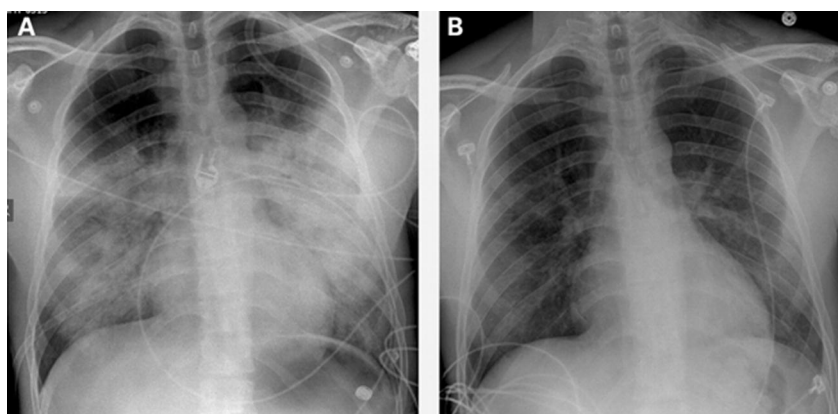


**Fig 5** Chest radiograph showing high altitude pulmonary oedema. Progressive improvement was noted on chest radiographs after 3 h (A) and 5 h (B) of treatment with positive airway pressure, dexamethasone, nifedipine, and sildenafil in the emergency department before admission to intensive care. From Gregorius et al, with permission[w36]