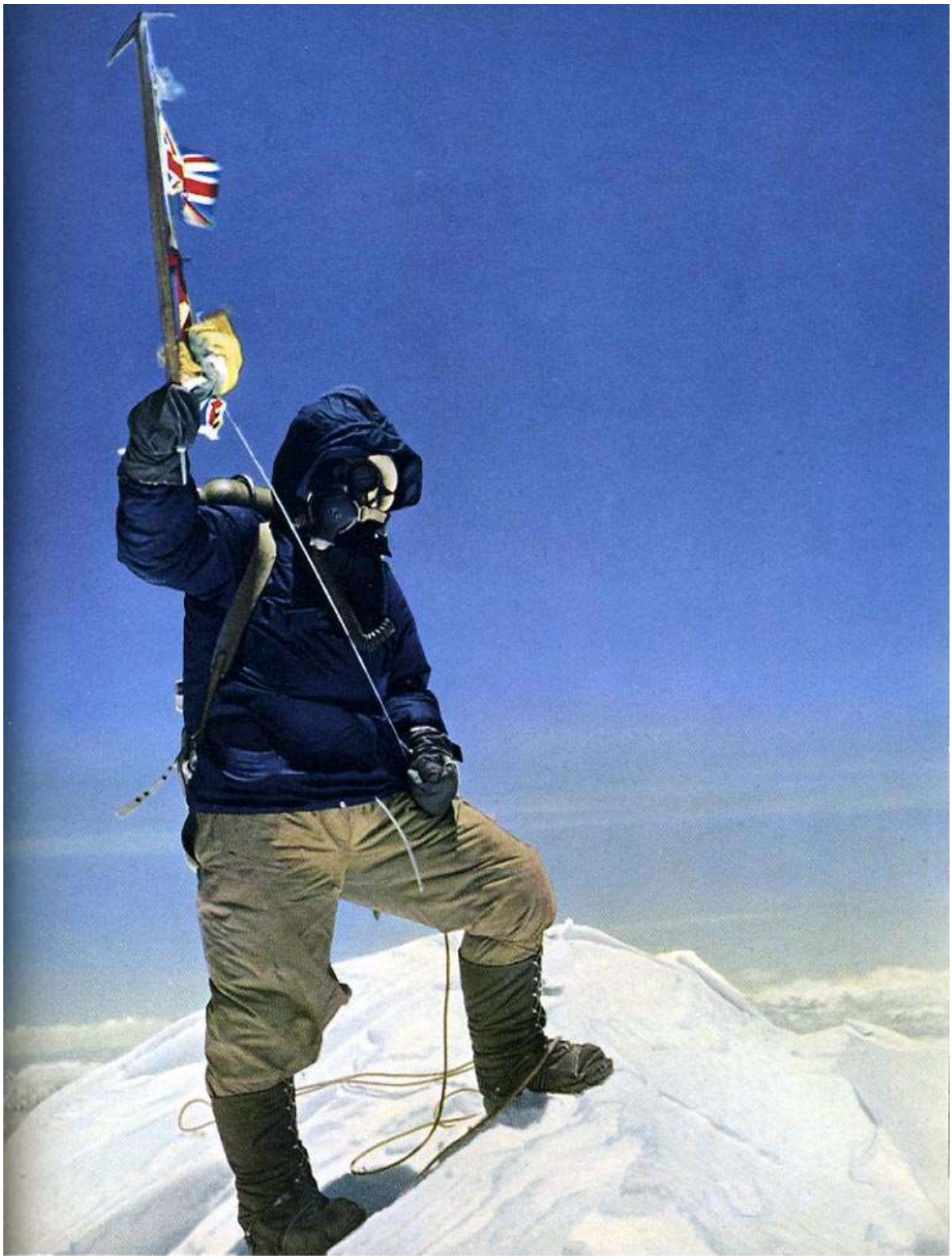
THE NORTHERN HOSPITAL

ACUTE MOUNTAIN ILLNESS - ACUTE MOUNTAIN SICKNESS (AMS)



Tenzing Norgay on the summit of Mount Everest, 11.30 a.m, May 29, 1953, photograph by Sir Edmund Hillary.

On 29 May 1953 at the age of 33, New Zealander Edmond Hillary and Nepalese Sherpa, Tenzing Norgay, became the first human beings known to have reached the summit of the world's tallest mountain, Mount Everest.

"They must be getting to camp VI", the watchers said. "They are hidden behind that serac (ice pinnacle) with the vertical crack in it – you know the one. "Two of them are sitting down; now they are up again". "Only another hour to wait. What are the odds?" At last, soon after 1.30, just as the radio was announcing the reported failure of the assault, the party emerged above a rise in the ground 300 yards or so above the camp, their blue windproof jackets sharp and cheerful against the glistening snow. Hillary and Tenzing were leading. All at once it was through the camp by the magic wireless of excitement that Everest had been climbed"

James Morris,
Times Correspondent with the Hillary Everest Expedition, May 1953.

"I had carried my camera, loaded with colour film, inside my shirt to keep it warm, so I now produced it and got Tenzing to pose for me on the top, waving his ice-axe on which was a string of flags - British, Nepalese, United Nations, and Indian. Then I turned my attention to the great stretch of country lying below us."

Sir Edmond Hillary and Tenzing Norgay, on Mount Everest, May 1953

ALTITUDE ILLNESS - ACUTE MOUNTAIN SICKNESS (AMS)

Introduction

The 3 principal **high altitude illnesses** are:

- Acute Mountain Sickness (AMS)
- High Altitude Cerebral Oedema (HACE)
- High Altitude Pulmonary Oedema (HAPE)

HAPE and HACE are both life threatening medical emergencies.

Prevention strategies are important

Definitive treatment of serious high altitude illness involves descent.

The risk for and severity of altitude illness are most importantly related to:

1. **The rate of ascent**
2. Individual susceptibility
3. The maximum height reached
4. The duration of time spent at altitude
5. The contribution of any comorbid medical conditions

The following relates to **acute mountain sickness**

Acute mountain sickness is the most common form of altitude illness.

The diagnosis however van be very difficult to make as symptoms are ill-defined non-specific; and may be difficult to distinguish from mere exhaustion.

The Lake Louise Score was developed in an attempt to standardize the diagnostic criteria of AMS

AMS is essentially the benign early mild end of the spectrum of cerebral changes that may lead to the severe life-threatening end of the spectrum known as HACE.

See separate documents for:

- **Acclimatization (in Environmental folder)**
- **High Altitude Cerebral Oedema (HACE)**

- **High Altitude Pulmonary Oedema (HAPE)**

History

A mountain is a landform that extends above surrounding terrain with a peak. There is no accepted standard definition of what height constitutes a "mountain".

Typically mountain height is reported as the **elevation above mean sea-level**.

Historically mountains were climbed for reasons such as:

- Worshiping deities and sacrificing to them.
- For laying territorial claims.

Then during the Enlightenment, for scientific reasons:

- Before balloon flight the only way to measure barometric pressure above sea level was by climbing mountains (which **John Dalton** did in his meteorological studies).

In more modern times, however, a shift occurred towards exploration for its own sake. On May 29, 1956 the ultimate climb was achieved by the New Zealander **Sir Edmund Hillary** and the Nepalese, **Tenzing Norgay**, who conquered the summit of the tallest mountain on Earth, **Mt Everest**.

In the 21st century *recreational* mountaineering has become a mega-million dollar industry.

Classification of Altitude

ALTITUDE	HEIGHT ABOVE SEA LEVEL	PHYSIOLOGY
LOW	**< 1500 meters**	No significant physiological changes are expected at this level.
INTERMEDIATE	**1500 - 2500 meters**	Whilst physiological changes occur they are not usually of clinical relevance - although altitude related illness has been reported at altitudes as low as 2000 meters
HIGH	**2500 - 3500 meters**	At these heights altitude illness can occur with rapid ascent or poor acclimatisation.

VERY HIGH	**3500 - 5800 meters**	These levels lead to significant hypoxaemia and a very high risk for altitude related illness.
EXTREME	**> 5800 meters**	These levels leads to marked hypoxaemia at rest and above which life cannot be indefinitely maintained without additional oxygen being made available.

Pathophysiology

Risk factors:

Altitude illness can occur on exposure to elevations > 3000 meters. [1]

(By comparison, commercial airliners fly with a cabin pressure equal to 2500 meters).

The severity of altitude illness will be related to:

1. Inherent susceptibility of the individual
    - Genetic traits
    - Pre-existing co-morbidities, (especially cardiovascular or respiratory disease).
2. **The rate of ascent**
3. The maximum height achieved.
4. Duration of exposure

Additional points of interest include:

- Athletic or aerobic fitness does not appear to be protective
- There is little difference between children and adults
- Persons greater than 50 years of age, surprisingly, have a slightly lower risk
- Women may have a greater risk (although this may in reality be explained by the the fact that they are simply more sensibly likely to report it than men)
- There is a slight increase in risk with increasing BMI
- There is no obvious link to nutritional status

- How a traveler has responded to high altitude previously can be a guide for future trips but is **not** infallible (as many variables ore involved)

Cause:

The precise pathophysiology of **altitude illness** is complex and not fully understood.

Factors currently thought to be contributory include:

1. Relative hypoventilation leading to hypoxia.

    - But the effects can clearly not be due to hypoxia *alone*.

      Symptom onset can be delayed and there can be a lack of immediate reversal with oxygen therapy. It can also occur in people at altitudes of about 2500 meters above sea level with an SaO_2 of > 90%

2. Fluid retention

3 Cerebral vasodilation and increased capillary permeability:

    - Fundamentally there is vasogenic and cytotoxic cerebral oedema

      AMS is simply the mild end of the spectrum at the opposite end of which is HACE

4. Hypoxic pulmonary vasoconstriction and non-cardiogenic pulmonary oedema (for HAPE)

Clinical features

Diagnosis is made on the basis of the **Lake Louise Score**

This score is based on a 4 level severity stratification of the following 4 symptoms

1. **Headache**

2. GIT upset

3. Fatigue / weakness / lethargy

4. Dizziness/ light headedness

A diagnosis of **AMS** requires:

- The presence of headache

Plus:

- At least 1 other symptom

And:

- An LLS of ≥ 3

In a person with:

- Recent travel to altitude (typically > 2500 meters).

The **Lake Louise Score** is assessed as follows:

SYMPTOM	**LL SCORE**			
	0	**1**	**2**	**3**
HEADACHE	Nil	Mild	Moderate	Severe
GIT UPSET	Good appetite	Poor appetite *Or* Nausea	Moderate nausea *Or* Vomiting	Severe nausea *Or* Vomiting
FATIGUE	None	Mild	Moderate	Severe
DIZZYNESS	None	Mild	Moderate	Severe

It is important to note that the LLS was developed as a **research tool** and was never intended for use by clinicians in actively managing patients. However, it has inevitably come to be used in this manner.

Whilst the evidence for using the score in determining severity of AMS is limited authorities have suggest the following ratings:

Mild: LLS 3 - 5

Moderate: LLS 6 - 9

Severe: LLS > 9

In more general terms:

AMS typically requires at least **6 - 12 hours** to evolve after arrival at altitude.

Headache is inevitably the first symptom followed by anorexia, dizziness and then fatigue.

One of the first features noticed in an individual by fellow trekkers is an **increasing irritability** in the individual.

There may be some increased **dyspnoea** especially at rest but this of course is a common feature of simply being at high altitude.

Some people may show evidence of mild peripheral oedema (or periorbital oedema on waking).

The individual may have lower SaO_2 levels than one would expect at the given altitude.

At any given altitude the symptoms of AMS will typically peak over 2 - 3 days and then resolve by day 4 - 5.

However, if **ascent continues** especially if **rapidly** then symptoms are more likely to **persist** and potentially **worsen.**

One old study by the Indian Army took soldiers straight up from sea level to between 3300 - 5500 meters above sea level and observed them over time with the following results:

- All were symptomatic for up to 5 days.
- 40% were still symptomatic after 1 week.
- 13% were symptomatic after 1 month.
- 6 soldiers were still symptomatic after 6 months.

Whilst interesting the main take home message was probably not to join the Indian army…

Differential Diagnosis:

Important differential diagnoses that should be considered include:

1. Infectious disease
2. Intracranial pathology
3. Dehydration

4. Physical exhaustion

5. Hypothermia

6. Electrolyte disturbances

    - Hypoglycaemia
    - Hyponatraemia

7. Hangover:

    - A somewhat startling diagnosis under the circumstances - but nonetheless a not unheard of one amoung climbers.

Investigations

Investigation is obviously not possible in most high altitude illness situations.

Once the patient has been stabilized and if in a medical facility, investigations may be considered when:

- The diagnosis is unclear
- Other important differential diagnoses need to be excluded
- The patient appears very unwell

The following may be considered:

Blood tests

1. FBE
2. CRP
3. U&Es/ glucose
4. Cardiac enzymes
5. LFTs
6. VGBs/ ABGs/ lactate

ECG

This is routine for any seriously ill patient, but especially in those who have pre-existing cardiovascular disease.

The ECG typically shows a right ventricular strain pattern in HAPE

CXR

In those suspected of having HACE

There will be typical bilateral alveolar infiltrates.

CT Scan

This should be done in any case of an altered conscious state when the diagnosis is unclear.

Confused patients with presumed HACE whose symptoms resolve with treatment, will not require a CT, but if symptoms fail to completely resolve a CT will be needed.

CT cannot detect changes *specific* to HACE, but can be used to exclude other differential diagnoses.

MRI Scan

MRI will be needed in those whose symptoms of HACE have not resolved with treatment and where a cerebral CT scan has not provided a clear diagnosis.

MRI is better able than CT scan to show evidence of hypoxic brain injury and of HACE in particular.

MRIs may show **generalised cerebral oedema** with a predilection for:

- The cerebellum
- The globus pallidus
- The limbic regions
- The frontal lobes

There is both a **vasogenic** and a **cytotoxic** component to the cerebral oedema although the cytotoxic component predominates over time

Management

Prevention

The best preventive strategies for altitude illness in general are:

1. Education

2. Slow ascents

3. Prophylactic therapy with **acetazolamide**:

   Acetazolamide decreases proximal tubular reabsorption of bicarbonate leading to a loss of bicarbonate. This results in a towards metabolic acidosis which then helps further increase ventilation (and hence increases PaO_2)

   Acetazolamide is also a weak diuretic agent although diuresis per se does not appear to be a clinically significant therapeutic action in altitude illness

   The **prophylactic** dosing is:

   - Acetazolamide **125 mg orally, twice daily** commenced **24 hours prior to ascent** and discontinued after reaching peak altitude and until acclimatisation occurs (approximately 3 days) [1]

   Acetazolamide also helps decrease **periodic breathing**

4. Nifedipine:

   - The dose for prophylaxis of **HAPE** is **20 mg SR oral** tds or **30 mg SR oral b.d**

     It is generally accepted that patients with an episode of HAPE should be considered HAPE susceptible and not return to altitude in the future without taking prophylaxis.

   - Nifedipine has **no** role in the prophylaxis or treatment of AMS / HACE.

5. Salmeterol

   - Salmeterol is an inhaled long acting beta-2 agonist that causes bronchodilatation as well as dilatation of the pulmonary microcirculation.

     This can assist in the clearing of alveolar fluid and in better matching ventilation and perfusion.

     It can be used as an alternative prophylaxis for HAPE if nifedipine and sildenafil are contraindicated.

     The usual dose is 125 mcg b.d

6. Gingko:

   - A few studies have suggested that gingko might have a benefit in prophylaxis for AMS.

This has not been substantiated and other studies have found contrary findings.

It is **not** recommended for use.

7. Coca leaves:

- Coca leaves are widely chewed by the locals in the Andes.

  However, there is no evidence of any benefit in the prevention of AMS.

  They can, however, make a splendid cup of tea….

Specific treatments

For most people symptoms of AMS will resolve and can be managed by either stopping ascent for a day or two and then continuing to ascend but more slowly.

For milder cases (e.g. LLS = 3-5)

1. Stop ascent

2. Rest

3. Maintain hydration

4. Simple non-opiate analgesia

4. **Acetazolamide:**

- In a *treatment* dose of 250 mg oral 12 hourly.

  It is recommended preferentially in **mild** AMS to assist with both prevention and treatment as in this scenario the individual is likely to want to continue to ascend after symptoms improve.

If symptoms resolve consider further ascent after 24 hours with an appropriate acclimatization schedule and a **prophylactic** dose of acetazolamide of 125 mg oral 12 hourly continued until peak altitude has been reached

For more significant cases (e.g. LLS > 5):

1. Stop ascent

2. Dexamethasone:

   This is presumed to exert its main benefit by preserving cerebral vasculature endothelial integrity. It has anti-inflammatory properties and down regulates cytokine receptors.

Dexamethasone appears to have a benefit in both prophylaxis and treatment of AMS and HACE

It does not appear to offer any benefit in HAPE

- Give in a treatment dose of **4 mg 6 hourly** - this can be oral, IM or IV depending on the exact clinical scenario and availability

3. If dexamethasone is not available:

   - Give acetazolamide in a **treatment** dose of **250 mg orally 12 hourly**

     Note that acetazolamide speeds up acclimatization and so it is better for use in **prevention** rather than treatment.

     Dexamethasone is more effective for **treatment** and, therefore, should be used preferentially for more severe AMS. Some authorities however recommend using both dexamethasone and acetazolamide.

4. Arrange for the patient to descend to a lower altitude

Coca leaf tea, very popular in the Andes.

References

1. eTG - November 2018.

2. ACEM Altitude Illness Study Notes - Dr Geoff Ramin, December 2018

Dr Geoff Ramin
Dr J. Hayes

1March 2019.