SPUMS Annual Scientific Meeting 2002

Altitude illness

Trish Batchelor

Key words
Travel medicine, altitude, hypoxia, medical conditions and problems

Abstract
Travel to previously geographically remote areas of the world has increased dramatically in recent years. In particular, high altitude travel is becoming increasingly popular. High altitude travel presents a number of specific risks, including acute mountain sickness and its life-threatening forms – high altitude pulmonary oedema and high altitude cerebral oedema. These entities can largely be prevented with medications and behavioural modifications. However, travellers are often unaware of the risk posed by journeys into high altitude areas.

Introduction

Of the potential environmental hazards the traveller may face (Table 1), high altitude is one of the most common. Millions of people now travel to, or live in, high altitude areas of the world. Access to many of these areas is no longer purely the domain of the young, fit and adventurous. There are many parts of the world where it is possible to fly from sea level to over 3000 m in less than a few hours, providing a rapid altitude gain and a high likelihood of developing acute mountain sickness (AMS). High altitude is defined as an altitude above 2500 m, very high altitude is 3500–5800 m and extreme altitude is above 5800 m. Above 5800 m one cannot acclimatize and there is a gradual and chronic deterioration.[1]

Oxygen changes at altitude

The term high altitude illness is used to describe the cerebral and pulmonary syndromes that can develop in unacclimatised persons shortly after ascent to high altitude.[2] The most important effect of high altitude on physiological processes is the decrease in oxygen pressure and content in the circulating blood. The percentage of oxygen in the air remains at 21% whether at sea level or altitude. However, at 18,000 feet the atmospheric pressure is approximately 50% of that at sea level and subsequently the partial pressure of oxygen is also 50% of that at sea level – a state of hypobaric hypoxia. At the summit of Mount Everest the partial pressure is reduced to 30% of the value at sea level. To a degree, individuals can compensate for this decrease in partial pressure by increasing their pulmonary ventilation, yet this compensation is only partial.[3] Other environmental changes include a drop in ambient temperature of 2^0C for every 300 m altitude gain and an increase in UV radiation of 10% for very 1000 m in altitude gain. These factors increase the risks of hypothermia, frostbite, snow blindness and UV skin damage.

Physiological responses to high altitude

There are some known physiological responses to ascent to high altitude. In the respiratory system there is a reflex increase in ventilation, and pulmonary vasoconstriction. Initially, cardiac output, heart rate and blood pressure rise secondary to sympathetic activation, which response lasts for a few days. As a result of dehydration there is an initial increase in the haemoglobin concentration. After some weeks, there is an increase in red blood cell production secondary to an increase in erythropoietin production. All

TABLE 1
HEALTH RISKS ASSOCIATED WITH ADVENTURE ACTIVITIES
(modified from Zell[4])

High altitude trekking	Mountain climbing	Scuba diving	Freshwater kayaking/ rafting
AMS, HAPE and HACE	AMS, HAPE and HACE	DCI	Drowning
Snow blindness	Hypothermia	Barotrauma	Leptospirosis
UV damage	Snow blindness	Marine envenomation	Hypothermia
Hypothermia	Frostbite	UV damage	Enteric infections
Frostbite	Major musculo-skeletal injury	Sea sickness	
Enteric disease		Infected coral cuts	
Animal bites			

individuals will develop some degree of cerebral oedema. However there is marked individual variation in this. For reasons not yet understood, some individuals will have an excessive response to altitude and will consequently develop one of the forms of altitude sickness.

High altitude syndromes are considered to sit on a spectrum from the milder acute mountain sickness (AMS) through to the life-threatening forms of high altitude pulmonary oedema (HAPE) and high altitude cerebral oedema (HACE). Other syndromes include high altitude deterioration, and for residents of high altitude areas, chronic mountain sickness.

Acute mountain sickness

Acute mountain sickness (AMS) is a syndrome of non-specific symptoms and is therefore subjective.[2] The Lake Louise Consensus Group defined AMS as the presence of headache plus one or more of insomnia, dizziness, lassitude, fatigue, anorexia, nausea or vomiting in an individual who has recently arrived at an altitude of greater than 2500 m.[5] Symptoms of AMS typically develop within 4–12 hours of arrival at altitude and occur in 40% of individuals ascending from sea level to over 3000 m and in 75% of those ascending to altitudes greater than 4500 m. The exact process of AMS remains unknown, however it is known that hypoxia elicits neurohumoral and haemodynamic responses that result in overperfusion of neurovascular beds, elevated hydrostatic capillary pressure, capillary leakage and consequent oedema in both the brain and the lungs.

There is currently no accurate method of predicting individual susceptibility to AMS. The only clear risk factor, apart from rapid ascent, is a past history of AMS, HAPE or HACE. Prevention of AMS will logically prevent progression to the more severe forms of altitude sickness. The most effective method of prevention is graded ascent. Essentially, this involves following a conservative approach to altitude gain. Recommendations include walking in rather than flying to altitudes of > 3000 m, when above 3000m limiting daily altitude gain to 300–500 m and having a rest day after every 1000 m of altitude gain. Another useful activity is to walk or climb higher than the sleeping altitude at some time during the day.

DRUG PROPHYLAXIS FOR AMS

Drug prophylaxis can be useful if a rapid ascent is unavoidable (eg. flying to over 3000 m) or if there is a past history of AMS. Acetazolamide (Diamox) is the drug of choice under these circumstances. This carbonic anhydrase inhibitor acts to decrease the degree of alkalosis with a resulting increase in arterial pCO_2 and increase in ventilation. The ideal dose of Diamox is controversial, traditionally 250 mg BD has been recommended, however many experts now recommend a dose of 125 mg BD. This lower dose appears to be effective for most individuals and has a far lower incidence of side effects.[6,7] Another advantage of Diamox is that it reduces the sleep hypoxia and periodic breathing that is a common complaint at altitude. Diamox should be avoided in those with an allergy to sulphur-based drugs.

Alternative medications include Dexamethasone 4 mg daily, however this is rarely recommended. Promising results have been shown in two studies using the herb Ginkgo Biloba to prevent AMS in both gradual and sudden ascents to altitude.[8,9] These studies have also shown that Ginkgo Biloba provides the benefit of increased peripheral circulation, thus reducing the incidence of uncomfortably cold extremities and frostbite.

High altitude pulmonary oedema

The progression from AMS to HAPE or HACE is accompanied by the development of physical signs and symptoms. HAPE is usually preceded by AMS and in context should be suspected when an individual suffers decreased performance and dry cough. As it progresses, tachycardia, tachypnoea and respiratory distress ensue. HAPE is a non-cardiogenic pulmonary oedema associated with pulmonary hypertension and elevated capillary pressure.[2]

Those individuals who develop HAPE have a relatively poor ventilatory response to hypoxia, exaggerated hypoxic pulmonary vasoconstriction and subsequent excessive pulmonary hypertension.[2] Whilst it may seem feasible that such individuals could be predicted by laboratory testing, there is substantial overlap between susceptible and non-susceptible groups. Predictions of susceptibility in the field based on laboratory testing do not yet seem possible.

A past history of AMS is the main risk factor. Those with a history of HAPE have a 60% chance of recurrence with abrupt ascent to high altitude.[10] The priority in treating HAPE is increasing oxygenation hence the primary treatment is oxygen, followed by descent to a lower altitude. Descent may be actual or simulated in a portable hyperbaric chamber (eg. the Gamow bag). Nifedipine SR 20 mg six-hourly is used if oxygen or descent are not available.

High altitude cerebral oedema

HACE is a clinical diagnosis defined as the onset of ataxia and/or altered consciousness in someone with AMS or HAPE. It is characterised by a global encephalopathy rather than focal neurological signs. The treatments of choice are descent to a lower altitude and oxygen. Dexamethasone may also be used at a dose of 8 mg stat, then 4 mg QID.

Retrieval

Data published in 1989 on helicopter rescues in Nepal showed a rate of 75 rescues for every 100,000 trekking

permits issued. The most common reasons for rescue were AMS (36%); trauma (26%); illness (26%) and non-traumatic orthopaedic problems (10%).[11] Accurate data on exact numbers of helicopter rescues in Nepal are impossible to obtain, however it was estimated that around 370 rescues per 100,000 trekkers occurred.

Data collected at the CIWEC Travel Medicine Centre, Kathmandu, from October 1999 to October 2000 examined 140 evacuations. Of these, 46% were for AMS, 13% for infections (not diarrhoea), 10% for diarrhoea, 10% for musculoskeletal problems and 5% for chest pain. Interestingly, whilst only 20% of trekkers visit the Everest region, 80% of altitude-related evacuations were from there. This reflects the higher altitude reached by trekkers in this region. It is interesting to see the rather dramatic increase in the rate of helicopter rescues being carried out in Nepal. This is probably due to improved communications (satellite phones are now available in many locations on the major trekking trails), and to the increase in the number of private helicopter companies providing these rescue services. There were no deaths in this study cohort (Pandey P, CIWEC Clinic, Kathmandu, personal communication).

Whilst trekking is generally a reasonably safe activity, high altitude mountain climbing is a highly dangerous endeavour. An analysis of deaths in summiteers of the world's two highest mountains showed the influence of the use of supplementary oxygen.[12] On Mount Everest, one in 29 summiteers using oxygen died on descent whereas for those not using oxygen the death rate was one in 12. On K2, the death rate was one in seven of those using oxygen and one in five of those who summitted without oxygen. It is thought that the even more extreme hypoxia resulting from climbing to these altitudes without supplementary oxygen contributes to this excessively high death rate, the majority of which are caused by falls and altitude sickness.

Contraindications to high altitude travel

Some individuals should not travel to high altitude. These include those with pulmonary hypertension, pulmonary atresia, moderate to severe COPD, unstable cardiac disease, and sickle cell disease. Individuals with asthma, diabetes, epilepsy, controlled hypertension and other forms of controlled heart disease may travel to altitude as long as they have been adequately prepared by a travel medicine practitioner with experience in high altitude medicine.

The elderly and young children appear to be at no increased risk of AMS. The International Society of Mountain Medicine has published guidelines for preventing and recognising altitude sickness in children.[13] Pregnant women are generally advised to stay below 4000 m and should take great care if visiting less developed countries to avoid enteric pathogens and mosquito-borne diseases.

References

1 Ward M, Milledge JS. Griffith Pugh, Pioneer Everest physiologist. *High Alt Med Biol* 2002; 3: 77-88
2 Hackett PH & Roach RC. High-altitude illness. *N Engl J Med* 2001; 345: 107-114
3 Hultgren H. *High Altitude Medicine*. California. Hultgren Publications, 1997
4 Zell SC. Environmental and recreational health hazards associated with adventure travel. *J Travel Med* 1997; 4: 94-99
5 Roach RC, Bartsch P, Oelz O, Hackett P. Lake Louise AMS scoring consensus committee. The Lake Louise acute mountain sickness scoring system. In: Sutton JR, Houston CS, Cootes G, eds. *Hypoxia and molecular medicine*. Burlington, Vt. Charles S Houston 1993; 272-274
6 Dumont L, Mordisoroff C, Tramer CR. Efficacy and harm of pharmacological prevention of acute mountain sickness: a quantitative systematic review. *BMJ* 2000; 321: 267-272
7 Bartsch P, Schneider M. Pharmacological prevention of acute mountain sickness. *BMJ* 2001; 322: 48 (letter)
8 Maakestad K, Leadbetter G, Olson S, Hackett PH. Ginkgo Biloba reduces incidence and severity of acute mountain sickness. [abstract] *WEM* 2001; 12: 51.
9 Roncin J, Schwartz F, D'Arbigny P. Egb761 in control of acute mountain sickness and vascular reactivity to cold exposure. *Aviat Space Environ Med* 196; 67: 445-452
10 Bartsch P, Maggiorni M, Ritter M et al. Prevention of high altitude pulmonary oedema by nifedipine. *N Engl J Med* 1991; 325: 1284-1289
11 Shlim DR, Houston R. Helicopter rescues and deaths among trekkers in Nepal. *JAMA* 1989; 261: 1017-1019
12 Huey RB, Eguskitza X. Supplemental oxygen and mountaineer death rates on Everest and K2. *JAMA* 2000; 284: 181-182
13 Children at high altitude. An international consensus statement by an ad hoc committee of the International Society for Mountain Medicine. *High Altitude Med Biol* 2001; 2: 389-40

Dr Trish Batchelor, MB,BS, FRACGP, MPH (Trop Med), is the Medical Adviser to The Travel Doctor TMVC, New Zealand. Trish was the principal guest speaker at the SPUMS ASM, Port Vila, Vanuatu, May, 2002.
Currently she is working as a medical officer at the CIWEC Travel Medicine Centre, PO Box 12895,
Durbar Marg, Kathmandu, Nepal
E-mail: <trishb@mos.com.np>