Altitude Related Emergencies

"Beyond the Road"

Environmental Emergencies
for
Emergency Services Providers

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File name: 2nd ed JB Altitude.doc 06/23/01 10:52 PM

Objectives

1. Discuss the etiology of high altitude illnesses, to include the physics of gases and how they apply to the ascending climber.

2. Discuss the pathophysiology of HAPE, HACE, and HARH.

3. Discuss the principles of a staged ascent.

4. Discuss the basis for chemoprophylaxis of altitude illnesses. Include the effects of diet and exertion in prophylaxis of altitude illnesses.

5. Discuss the natural progression of altitude illnesses and why this mandates early descent.

6. Discuss both field and hospital therapy of all of the common altitude-related illnesses.

7. Discuss the epidemiology of high altitude illness in order to best predict who should not ascend rapidly.

Introduction

As long as man has climbed high places for reasons of trade, war or just enjoyment, he has recorded tales of the difficulties that are encountered at altitude. Prior to the 1960s, only hearty adventurers would trek to Mt. Everest. This trek involved 3 weeks of arduous approach that acclimated all in the party. With an increase in free time and disposable income, there has been a corresponding increase in the numbers of those who have gone to visit faraway high places. During the last 2 decades, this increase in recreation in mountainous areas has caused a corresponding increase in the prevalence of altitude related illnesses.

Today, over 5000 travelers each year in the Himalayas alone reach altitudes that are potentially dangerous. Millions more will trek to stateside mountain resorts for skiing, hiking, and, of course, climbing. Some 3600 people visit the top of Pike's Peak (14,213 feet, 4332 M) each summer day. A major difference between those early trekkers and contemporaneous adventurers is that the modern trekker may fly to the resort, spend only a few days skiing or hiking, and then return to sea level. This further increases the potential for altitude-related illnesses.

Altitude-related illnesses are not discrete separate entities but, rather a spectrum of illness caused by inappropriate compensation to continuing relative hypoxia. The diseases may be lethal, usually occur when man ascends above 8000 feet (2400 M), and are best treated with descent to lower altitude.

The usual illnesses that accompany travel into high altitudes include the following:

1. Acute Mountain Sickness (AMS). This is a common disease that affects up to 25% of travelers that ascend rapidly to any altitude greater than 8500 feet (2590 meters). Headache, nausea, vomiting, dyspnea, fatigue, work intolerance, and sleep disturbances are common. AMS is usually a self-limited disease that requires only supportive therapy or descent to lower altitude.

2. High Altitude Pulmonary Edema (HAPE). This unusual but always serious disease is rarely seen above 8,500 feet (2590 meters). The common early symptoms include headache, rales, dyspnea, fatigue, and a non-productive cough.

3. High Altitude Cerebral Edema (HACE). This rare, life-threatening, illness is manifested by headache, fatigue, ataxia, confusion, and hallucinations. If not treated, this disease will rapidly progress to coma and subsequent death.

4. High Altitude Retinal Hemorrhage (HARH). This is a relatively common and usually innocuous finding of travelers at the upper limits of very high altitude and beyond. It does not usually require therapy.

Definitions

Standard Atmosphere

Standard atmospheric pressure is define as 760 mm Hg at sea level. This standard pressure decreases linearly with increasing altitude at all terrestrial elevations. The actual atmospheric pressure varies somewhat with the weather, the seasons, and the latitude, although these changes seldom are greater than 10 mm Hg. The atmospheric pressure is somewhat higher at the equator and lower at the poles due to the shape and rotation of the earth and the presence of a dense cold air mass above the equatorial earth.

When completely dry, the atmosphere is composed of 20.94% oxygen, 79.02% nitrogen, 0.04% carbon dioxide, and trace amounts of noble (inert) gases. The only substantial variable component is the water vapor. Except for water vapor content, the composition of the lower levels of the atmosphere surrounding the earth is constant over its entire surface.

For medical purposes, the water vapor contribution may be presumed to be the vapor pressure of water at tracheal temperatures (47 mm Hg at 37°C). This assumption is safe, because the air is completely saturated with water vapor by tracheal moisture by the time it passes into the alveoli. The vapor pressure of water is primarily dependent upon temperature and will be higher with higher temperatures or fever. (At 39°C, water vapor pressure is 50 mm Hg.)

High altitude

High altitude is usually defined as altitudes between than 8000 feet (about 2400 M) and 14,000 feet (4300 M). About 12% of the world's population (40 million people) live at or above this altitude.[1] Because many people visit locations within this altitude range, most cases of altitude-related illnesses occur at these elevations. The incidence of altitude-related illnesses increases in frequency and severity as individuals travel higher.

Very high altitude

Very high altitude is the ranges from 14,000 feet (4300 M) to 18,000 feet (5500 M). Although most cities frequented by tourists are located lower than this, a significant number of base camps and towns exist at these heights. Indeed, the highest known continuously inhabited town on earth, Wenchuan on the Chianghe-Tibet road, is located at 16,732 feet (5100 M).

In this range of altitudes, any known altitude illness can occur. Rapid ascent to these heights without prior acclimation is dangerous.

Extreme altitude

Any altitude above 18,000 feet (5500 M) is considered extreme. There is a substantial risk of altitude related illnesses at these higher elevations. Fortunately, most climbers venturing to this altitude have been properly acclimated by the approach journey. Trekkers who are susceptible to altitude illnesses have usually turned back or have been evacuated. Ascent to these heights without substantial preparation is exceedingly dangerous.

Above 18,000 feet (5500 M), it is not possible to become acclimated to the altitude. Rather, there is a gradual decrease in physical conditioning and a progressive mental deterioration.

Partial Pressures

When we discuss the atmosphere, we are primarily concerned with the amount of available oxygen. The laws of partial pressures describe the relationship between oxygen and the other gasses found in the atmosphere.

The pressure exerted by each component of a mixture of gases is termed the partial pressure of that gas. The total pressure of a gas mixture is the sum of the partial pressures of the individual gas components of that mixture. The partial pressure of each component can be given by the formula:

$$P_P = (\%\ \text{gas}) \times (\text{total pressure})$$

As noted previously, the air is fully saturated with water at the level of the alveolus. In the medical case, therefore, the above equation must be modified to account for the vapor pressure of water. The resultant equation for the partial pressure of oxygen at the level of the alveolus is expressed by:

$$P_{O_2} = (0.21) \times (760\text{-}47) = 149 \text{ mm Hg}$$ at sea level

Since the barometric pressure decreases as the altitude increases, the partial pressures of the component gases will also decrease. The oxygen partial pressure at 18,000 feet (5500 M) drops to about half that at sea level. An inevitable consequence of an ascent to high altitude is a decreased amount of available oxygen.

Effects of Exposure to Altitude

As was just noted, when we breathe air at higher altitude, we have less available oxygen. Because the oxyhemoglobin dissociation curve is sigmoid (s-shaped), we can travel to a considerable altitude before the arterial oxygen saturation falls to below 90%. The precise elevation required to produce this fall varies among individuals, and is influenced by concurrent or pre-existing illnesses.

Hypobaric or Hypoxia?

It is widely accepted that hypoxia is the major environmental stress at altitude and is the main factor causing altitude illness. Unfortunately, the acceptance of this "truth" may be misleading. Descent - which both increases the pressure and the available oxygen concentration will improve the symptoms of any of the altitude illnesses more rapidly than supplemental oxygen alone. Other studies have shown that hypoxia of the alveolus will not produce pulmonary edema or changes in the lung tissue that are found in high altitude pulmonary edema. Only with BOTH hypobaria and hypoxia can human-like high altitude illness be reproduced in experimental animals.

Feedback Mechanisms

The carotid body chemoreceptors sense the decrease in arterial oxygen saturation and stimulate an increase in both depth and rate of breathing. In patients with a decreased sensitivity to hypoxemia, such as those with chronic obstructive pulmonary disease, breathing rates may not rise and the hypoxemia will be worsened.

Work of Breathing

When we climb, the maximum useful and productive work that we can do decreases markedly as we go higher. The reasons for this reduction are related to the hypoxia of altitude. The resting oxygen requirement of the body remains relatively constant. Unfortunately, there is a decreased amount of oxygen available per inhalation due to

the decreased atmospheric pressure. The respiratory rate must increased in an effort to maintain a constant amount of oxygen, even at rest. An increased respiratory rate leads to an increase in oxygen consumption by the respiratory muscles, leaving less oxygen available for the rest of the tissues.

The increased respiratory rate causes a vicious cycle that consumes more of the available oxygen.

Maximum cardiac output remains constant for an individual at a given time, but a larger proportion of the cardiac output is now used "merely" to provide oxygen to the tissues. This leaves less cardiac output available for "useful work". At some elevation, the cardiac output becomes less than required to provide oxygen. The climbers who are breathing ambient air near the summit of Mt. Everest are very close to the hypoxic limits of human survival.[2]

Even during relatively heavy exercise at low altitude, a healthy person's arterial oxygen saturation does not change. On the other hand, exercise at high altitude causes in a distinct fall in arterial oxygen saturation. This decrease in the oxygen available to the tissues is thought to be due to two factors:

1. At high elevations, our hemoglobin oxygen saturation is on the steep part of the oxyhemoglobin dissociation curve. This means that a small further decrease in the arterial oxygen tension will cause a large decrease in the oxygen saturation of the hemoglobin.

2. As the respiratory exchange, and cardiac rate increases, the limits of maximum pulmonary diffusing capacity are reached. In fact, it is possible to make a useful estimate of the diffusing capacity of the lung at high altitude from the fall in oxygen saturation during exercise at high altitude.[3]

Altitude Tolerance

The climbers who have reached the summit of major peaks have been found to have certain physiologic attributes not predictable by sea level studies or performance at lesser altitudes. Some of these attributes include (1) high ventilatory response to hypoxia,[4] (2) effective cerebral function during severe hypoxia, (3) effective muscle function during severe hypoxia,[28] (4) high pulmonary diffusing capacity, (5) unusual ability to perform sustained physical work.[5] Again, these attributes have been noted in successful climbers at extreme altitudes and the physiologic basis is not yet understood.

It should be emphasized that fitness alone does not protect the individual from the problems encountered at high altitudes.[6] Indeed, by allowing a rapid ascent, a high level of fitness may predispose a subject to altitude-related illnesses. In this aspect, the older more sedentary trekker or skier who takes but a single holiday per year may be at an advantage over the younger and more fleet of foot - if both are equally acclimated.

Effects on Sleep

Sleep can be profoundly disturbed by the decreased oxygenation at high altitude. Our respiratory drives decrease during sleep, leading to a slight, but normal, reduction in the arterial oxygen saturation. At high altitude, this effect is greatly magnified.

Periodic breathing (Cheyne-Stokes respirations) may occur during sleep, but may also be seen at rest in non-active, awake subjects visiting higher altitudes. During sleep with periodic breathing, the oxygen saturation of the hemoglobin may drop as low as 50%.[7] This periodic breathing and subsequent hypoxia may lead to severe sleep disturbances. The resultant exhaustion may cause judgment errors, poor concentration, and irritability.

Even though fatigued, many patients with altitude-related illnesses have difficulty sleeping. They wake frequently and have bizarre, unpleasant dreams. Sleeping at altitude seems to predispose climbers to the genesis of all of the illnesses of high altitude. People who spend their days above 8000 feet (5500 M) but sleep below this level have a decreased incidence of altitude-related symptoms, giving rise to the skier's adage of "ski high - sleep low".

Sedatives given to induce sleep will worsen the hypoxemia by decreasing the sensitivity of the oxygen chemoreceptors.[8] Reversal of the respiratory alkalosis with drugs, such as acetazolamide, appears to result in improved oxygenation and better sleep[7].

Effects on Diet

Not only is the shortness of breath a problem, but the sojourner to high altitudes will frequently be somewhat nauseated. The taste of foods may change markedly, and there is often a craving for carbohydrates.[9] [10] Fatty foods may be particularly unappealing, despite the fact that these foods provide more useful energy in a smaller

package. Medications given to decrease or modify the effects of altitude illnesses may further impair tastes.

Adaptation to Life at High Altitude

When the partial pressure of oxygen is reduced abruptly, our compensation mechanisms do not have time to adapt. If a aircraft abruptly loses pressurization at 28,000 feet, the occupants

A slow ascent to the same altitude allows a gradual compensation and carries less ominous outlook. Without this acclimation process, Messner and Habeler would never have been able to ascend to the summit of Mt. Everest without supplementary oxygen in 1978.[11] The body can adapt and acclimate in multiple ways to the decreased availability of oxygen up to about 18,000 feet. We start to show the physiological effects of acclimation at altitudes greater than 6562 feet (2000 M). Acclimation is not physically possible when we travel higher than 18,000 feet.

Respiratory Compensation and Effects

Initially, our bodies try to adapt to the higher altitude by increasing the respiratory rate and volume, but it takes several days at the target altitude before a new constant value is achieved. This change begins at about 3200 feet (1000 M) and makes more oxygen available. Until the adaptation is complete, shortness of breath is experienced during exercise.

Respiratory Alkalosis

A natural consequence of the increased respiratory rate is a respiratory alkalosis. This coincides with the increasing hypoxemia during the first 24 to 48 hours after an ascent. The hyperventilation and subsequent respiratory alkalosis is profound at the extreme terrestrial elevations, with measured alveolar pCO^2 about 7 torr and calculated pH of 7.70 in one subject at the summit of Mt. Everest.[3] [12] Reversal of alkalosis with drugs, such as acetazolamide, that induce the kidney to excrete bicarbonate result in improved oxygenation.

Ventilatory Drive

Long visits at high altitude lead to a decrease in the ventilatory drive to breathe, particularly in response to hypoxemia.[13] Ventilation in native mountain dwellers is only one-half that of acclimated lowlanders and the ventilatory response to exercise is lower.[14] When exercising, the highland natives will experience substantially less dyspnea than a lowlander.

Oxyhemoglobin Dissociation Curve

A shift to the right on the oxyhemoglobin dissociation curve will occur shortly after ascent to high altitude. This is due to an increased level of 2,3-diphosphoglycerate (2,3-DPG) and a resultant decreased affinity for hemoglobin.[15] [16] The immediate effect is thought to be improved oxygen unloading from hemoglobin at the tissue level. This may be offset by a decreased bonding of hemoglobin to oxygen resulting from the increased pH (the Bohr effect), so the net effect is uncertain. Certainly, oxygen saturation is maintained within narrow ranges up to an altitude of 20,000 feet (6300 M).[17]

Chronic Adaptation

During chronic exposure to altitude, the pulmonary arterial pressures are elevated.[18] This will "force" open the capillaries in all portions of the lung and maximize perfusion to improve the capacity of the pulmonary circulation to absorb oxygen. While little effect on diffusion and pulmonary circulation is seen in lowland visitors to high altitude, a fully adapted native will show improved diffusion, increased pulmonary capillary bed size, and a relative redistribution of blood flow to the upper lobes of the lung.[19]

Cardiovascular Compensation

As a person is gradually exposed to decreasing barometric pressure and hypoxia, the cardiac rate and cardiac output increase.[20] The work of breathing increases markedly as noted above, causing more cardiac output to be diverted to life-sustaining processes. The maximum cardiac output, of course, is unchanged. This hyperventilation adaptation serves, in moderate hypoxemia, to oxygenate systemic organs. Needless to say, as the hypoxic insult is increased, target organs such as the brain become hypoxic despite the adaptive mechanisms.

The increased resting cardiac output at high altitude appears to be primarily a result of tachycardia rather than a change in the volume of blood ejected with each beat (stroke volume). As the cardiac rate increases with exercise, the net effect is to further decrease the stroke volume. This decreases the ability of the patient to accomplish useful work at high altitude.[21]

Renal Compensation

After about 36 hours at altitude, an alkalotic diuresis usually occurs as a partial compensation for the respiratory alkalosis. This respiratory alkalosis is never completely compensated, even in high altitude natives.[22] [23]

As bicarbonate is excreted, an obligatory fluid loss occurs, predisposing the patient to dehydration and a decreased plasma volume.[24] During the 1981 American Medical Research Expedition to Mt. Everest (AMREE II), this fluid loss was studied. At 6300 M, the urine output is significantly greater than it is at 5400 M or at sea level. Osmolar clearance, creatinine clearance, and the urinary excretion of vasopressin were not changed. The increase in excretion despite a higher serum osmolarity may be due to a decrease in osmoreceptor sensitivity or a decrease in release or synthesis of vasopressin.[25]

In the same study, electrolytes and osmolality were also observed with changes in altitude. At an altitude of 5400 M the serum concentration of chloride is above the upper limits of normal and the total CO_2 is below the lower limits of normal. At 6300 M, the serum concentrations of chloride were significantly greater than at lower altitudes. The total CO_2 concentration was significantly lower at 6300 M than at 5400 M. This retention of salt may in part be due to a blunted response to the rennin-angiotensin-aldosterone system noted after 2 weeks or more at extreme altitudes.[26]

In other studies, test subjects at altitude who had the most severe symptoms of acute mountain sickness appear to have the greatest decrease in aldosterone secretion.[27] [28] The reason for this change in the rennin-angiotensin-aldosterone system is not known. Japanese investigators have felt that the increase in osmolarity may be attributable to a fluid shift from extra to intracellular spaces, a shift mediated in part by increased cortisone and aldosterone.[29]

In addition to the fluid shifts, the investigators of AMREE II noted the lack of subjective complaints of thirst that would be normal with a serum osmolarity of 302 mOsm/kg. These observations document that the threat of dehydration for mountaineers working at extreme altitudes is potentially serious.

Hematologic Compensation

As a chronic adaptive response, the total red cell mass increases. Any chronic hypoxia will stimulate production of the hormone erythropoietin by the kidneys. This hormone stimulates the bone marrow to increase the production of red blood cells. The response occurs over several weeks and is reflected in the higher hematocrit of the long duration high-altitude dweller [14]. An extremely high hematocrit (greater than 60%) may also be found with continued stimulation. This excess is associated with chronic mountain sickness. Over a longer period of time, increased capillary density, intracellular mitochondria, and cytochrome oxidase, enable more efficient use of available oxygen stores.[30]

Acute Altitude Illnesses

The consequences of rapid ascent for the unacclimated individual can be broadly grouped into three categories: Acute Mountain Sickness, High Altitude Pulmonary Edema, and High Altitude Cerebral Edema

Acute Mountain Sickness

Epidemiology

Acute mountain sickness is the most common manifestation of the altitude-related illnesses. Some symptoms are likely to appear in any lowlander who ascends higher than 12,000 feet (3660 M). Severe symptoms rarely occur below 8000 feet (2440 M). As with the other altitude-related illnesses, the incidence of acute mountain sickness appears to be exacerbated by rapid ascent and vigorous exercise undertaken soon after arrival at altitude.

Some people become sick each time they visit at moderate altitude, no matter how slowly they travel. Others appear to be unaffected until extreme elevations are reached. Infants and adolescents are more vulnerable than adults. The reasons for the individual differences in sensitivity to altitude changes are not well understood

Physiology

It has been suggested that the headache of acute mountain sickness is due to cerebral edema, but cerebral edema in other conditions is not associated with headache. A

more plausible suggestion is that the headache of AMS is due to hypocapnia along with either concomitant spasm of the cerebral blood vessels or hypoxia.

Presentation

The most common initial symptom is headache, which may vary from a mild light-headedness to a severe, incapacitating pain. Lassitude, drowsiness, malaise, weakness, and dyspnea on exertion are also common. Anorexia is a frequent complaint and, particularly in children, nausea and vomiting may occur. Observers may note facial pallor and cyanosis of lips and nail beds in those exercising and in susceptible individuals at rest. After arrival at altitude, a feeling of warmth and flushing of the face may be noted for the first 24 to 48 hours. Slight physical effort may produce an annoying dyspnea, even in the physically fit. Weakness, tachycardia, and palpitations may also be associated with effort.

In addition to symptoms acquired with acute mountain sickness, sudden increases in altitude may also be accompanied by decreased performance in athletic endeavors. Such changes were observed in athletes at the 1964 Olympic games in Mexico City (elevation 6000 feet or 1830 M). The degree of athletic conditioning appears to have little or no influence upon the occurrence of symptoms if the change in altitude has been rapid.

Sleeping problems at altitude may be a major source of annoyance. Sleep, particularly for the first few nights, is difficult, with frequent periods of wakefulness and, at the higher altitudes, Cheyne-Stokes breathing may be present. During the apneic phase of the periodic breathing, the episodes of hypoxia may be quite profound.

Complications

Sleep Hypoxia

Occasionally, recent arrivals to high altitude are found to be very weak and drowsy, or apparently sleeping. They may be found in a semicomatose state, hallucinating or behaving in an irrational pattern. The other members of the trek may note that the new arrival is deeply cyanotic. Improvement can be quickly achieved by awakening the person and encouraging deep breaths. Because of decreased respirations during sleep, an individual may be suffering from profound hypoxia. If the climber is awakened and encouraged to breathe deeply, symptoms may decrease.

HAPE and HACE

It needs to be emphasized that acute mountain sickness is part of a spectrum of altitude-related diseases and shares many of the attributes of the more serious high altitude cerebral edema and pulmonary edema. Indeed, it is difficult to differentiate between minimal high altitude cerebral edema and AMS on a windswept slope in the snow. For these reasons, it is clear that evacuation of the patient with serious acute mountain sickness is preferable to allowing progression of the patient's cerebral edema or pulmonary edema. This patient is a distinct liability to the group, no matter what the final diagnosis is found to be.

It is often difficult to tell the trekker who has invested considerable time and money in a "once in a lifetime" trip that he or she is going "down the hill." This is also a lot easier than later having to tell the bereaved family that you did not think the illness was serious.

High Altitude Pulmonary Edema

Epidemiology

High altitude pulmonary edema occurs with rapid ascent to altitudes greater than 8000 feet (2440 M). The incidence of clinically significant HAPE ranges from about 0.6% to 4% following a rapid ascent to 12,000 feet (3660 M) in those who are older than 20 years.[31] In those who are younger than 20, the incidence of HAPE during the same climb is six-fold higher than in adults (from 2.5% to as high as 38%).[32] The higher figures are derived from populations that live at high-altitude and visit sea level periodically. Such high altitude dwellers seem to be at increased risk of developing HAPE upon return to high altitude.

HAPE usually begins 24 to 72 hours after ascent to an altitude of greater than 10,000 feet (3050 M). Fatal cases have been noted as low as 8000 feet (2440 M). Cases are rare on the day of arrival and very few cases are reported after more than 10 days at altitude. Frequently the onset of symptoms of HAPE is noted at night, but it is uncertain whether this is connected with the sleep disturbances of altitude or whether this is just when the tent mate first notes the cough that keeps both patient and companion awake. Indeed, you may often wonder if your tent mate is going to take another breath and may muse over which course of action will get you the most sleep.

Predisposing Factors

HAPE most often occurs in young individuals who rapidly ascend to altitude, and then engage in strenuous exercise at altitude.

Rapidity of Ascent

Rapid ascent is the most important predisposition to HAPE. The only known protective factor for HAPE is slow acclimation to ambient altitude. Indeed, in the early phases of the Sino-Indian border wars of 1962, Indian troops traveled by rail to altitude from sea level and suffered enormous casualties from HAPE.[33]

Age and Sex

Children under 5 are thought to be more susceptible to HAPE, although the incidence in newborns appears to be lessened. There appears to be no great difference between sexes, although more males are climbers or skiers and, hence, exposed to the etiologic conditions more frequently. Since there are more male climbers than females, HAPE is reported more frequently in men.

Exertion

Multiple studies have shown that heavy exertion at altitude carries a marked predisposition to HAPE, although the actual increase in incidence is not known. The incidence of HAPE is thought to be increased by the increase in cardiac output and subsequent increase in the flow through the pulmonary vessels.

Cross-country skiers, backpackers, and mountaineers who carry heavy loads and ascend rapidly are particularly at risk. Downhill skiers, who are not encumbered with packs and can rest while ascending have lesser incidence of HAPE.

Geographical Incidence

There may be some geographic variation in the incidence of HAPE, as there appears to be a somewhat greater incidence on Denali and in Peru than in other places. This apparent geographic variation may also be due to the difficulty in reaching other highlands and the subsequently slower ascent.(RB Schoene, unpublished data)

Reascent to High Altitude

The highest incidence of HAPE is among the acclimated highlanders who return home after a short trip or vacation at lower elevations. HAPE may occur after a low altitude trip of only a day. A low altitude stay of 2 to 7 days has the highest incidence upon return to the highlands.[34]

Prior History of HAPE

There is good evidence that the condition has a relatively high recurrence rate. One study reported that 20% of HAPE victims had repeated episodes.[36] The mechanisms of this apparent predisposition have not yet been determined.

Pulmonary artery atresia

A very high rate of development of HAPE has been reported among persons with absence of the right pulmonary artery.[35] This is of more interest to the academic than practical in the field. (Of course, don't your patients present with their pulmonary arteriograms microfilmed and in their backpacks?)

Menstrual Cycle

Women in the premenstrual water-retaining phase may be more vulnerable to HAPE.[36] This has not been substantiated in the modern literature or in the author's experience.

Pathophysiology

High altitude pulmonary edema has been described in the past as pneumonia or congestive heart failure but was recognized as a non-cardiogenic pulmonary edema in the early 1960s. Cardiac catheterization studies show that HAPE is a non-cardiogenic form of pulmonary edema associated with high pulmonary artery pressures and normal pulmonary capillary wedge pressures.[37] It does not have hemodynamics consistent with heart failure.

Recent data indicate that HAPE is a high-protein, high-permeability type of pulmonary edema, similar to that found in Adult Respiratory Distress Syndrome (ARDS).[38] In studies with cardiac catheterization and lung scans, hypoxia has been

shown to have an exaggerated increase in the pulmonary artery pressure in susceptible individuals. It is theorized that it is in part due to hypoxia and inordinately high pulmonary vascular pressures that may lead to vascular leakage.

Unlike patients with ARDS, HAPE victims improve rapidly with proper treatment and may return to climb under close supervision within 10 to 14 days of the original insult.

Symptoms

The onset of symptoms of HAPE is somewhat slower than in AMS. Normally, the onset of symptoms occurs within 24 to 96 hours after arrival at altitude. The most common symptoms are a nonproductive cough, gradually increasing tachypnea and dyspnea, and fatigue. Dyspnea becomes marked, even with only modest exertion. Headache, anorexia, nausea, and vomiting are frequently present, particularly in children.

The individual with pulmonary edema is usually more tired than other members of the climbing or skiing party. He or she may have a sense of "tightness in the chest" or a feeling of impending suffocation at night. Occasionally, a patient might note wheezing sounds with breaths and a burbling feeling in the chest.

An important indication of the severity of HAPE is the level of mental acuity. Confusion is frequently noted and may be due to either the hypoxia or some component of HACE.

Signs

Signs are both cardiac and respiratory in nature. Tachycardia and tachypnea are always seen. The pulse rate is usually rapid (110 to 160 / minute), even after several hours of rest. The respirations are usually greater than 20 per minute (typically 20 to 40). Cardiac signs of pulmonary hypertension may include a right ventricular heave and an increased intensity of the pulmonic component of S2.

Rales are considered diagnostic of the condition and may best be heard in the early course about the right axilla (Hackett - Unpublished communications). As the disease progresses, the rales become audible to the tent mates and may be heard several feet away. The cough is usually dry and intermittent at first but then becomes persistent and productive of a white, watery or frothy material. Late in the course, the sputum becomes pink tinged or bloody.

The temperature may be slightly elevated but not markedly so. Cyanosis may be marked.

Clinical Course

The severity of HAPE ranges from mild dyspnea at rest to severe respiratory embarrassment with production of frothy pink sputum. Rarely, coma or alteration of consciousness is seen as the first sign of HAPE, with little or no respiratory manifestations. Both signs and symptoms often become worse at night. Progression of the disease may be quite rapid and frequent examination of patients who present with minimal signs is important. Any worsening should mandate immediate oxygen therapy and descent.

If the patient becomes unconscious, death will follow in 6 to 12 hours unless prompt descent or oxygen therapy are initiated. In the Sino-Indian conflict, mortality of HAPE was decreased from 27% to 4% by rapid evacuation to lower altitude.[39]

Grading the severity of high altitude pulmonary edema is advocated by many and may be useful. Patients with lesser severity (grade I) HAPE may have only mild symptoms, such as fatigue or exertional dyspnea. These patients typically recover after only a day or so at lower altitude and may proceed with the climb after a week or so. Patients with higher grades of pulmonary edema should be considered as major medical emergencies and require prompt treatment and evacuation to lower altitudes.

Laboratory and X-ray findings

Although radiographs and electrocardiograms are not found beyond the roadhead, the findings illustrate the differences between HAPE and congestive heart failure. In the field environment, therapy is not usually dependent upon these studies.

Radiological findings include patchy infiltrates and alveolar infiltrates common to other adult respiratory distress syndromes . There are usually multiple small, irregular infiltrates with poorly defined margins in a patchy asymmetrical distribution. The infiltrates may be unilateral and appear first in the upper lobes, although in severe disease, infiltrates are often confluent and extensive. The pulmonary vascular pattern is often widened. Heart size appears normal and will not usually change with the clearing of infiltrates. With recovery, there is rapid clearing of the infiltrates.

The X-ray will not show the "bat-wing" distribution and cardiomegaly found in the patient with congestive heart failure. Pulmonary vascular congestion is also not found.

Electrocardiographic findings are those of acute right heart strain with right ventricular overload. It is often difficult to differentiate the findings of HAPE from those of a pulmonary embolus.

Pulmonary arterial catheters will show severe pulmonary hypertension, normal capillary wedge pressure, and normal left atrial pressure.

Only a few abnormal laboratory findings occur in patients with HAPE. The white blood cell count is frequently elevated with a leftward shift. Severe hypoxia is generally present with HAPE, and arterial blood gases will reflect this hypoxia. Administration of supplementary oxygen will improve the abnormalities but often does not completely correct them.

Differential Diagnosis

When the history of high altitude exposure in a young athlete is known and the typical findings of a non-cardiogenic pulmonary edema are noted, the diagnosis of high altitude pulmonary edema is relatively easy. When the symptoms develop on the mountainside, the whole party is usually aware of the potential for HAPE and acts accordingly. Indeed, the dry hacking cough that is often caused by the relative dehydration at altitude may trigger over zealous expedition leaders to order a descent.

The diagnostic problem occurs when the patient with cough, slight fever, and signs of pulmonary congestion presents to a practitioner AFTER returning from the ski trip or short climbing expedition. The pattern of right ventricular strain on the electrocardiogram and the clinical signs of pulmonary hypertension may also suggest a pulmonary embolism. History of recent trips to high altitude MUST be obtained for an accurate diagnosis when the still-suffering patient has returned to the lowlands.

When the patient is a resident of high altitude who has a short stay at lower altitudes and then returns to the heights, the diagnosis may be missed if he is not returning to home.

CHF From Other Causes

Another diagnostic dilemma is the older patient who has traveled to high altitude to visit relatives. This patient may have good reasons to have pulmonary edema even at LOW altitude. Differentiation of CHF from HAPE is not easy and requires an uncommon judgment. These patients require prompt evaluation at a well-equipped hospital facility. Fortunately, these patients are usually not found far from the roadhead.

Pneumonia

A young patient with dyspnea, tachypnea, chest pain, rales or Rhonchi, sputum production, and fever may well have a pneumonia. A very high fever is uncommon in HAPE. The radiographic picture of the two diseases is often the only difference in early presentation. If there is any doubt, have the patient descend with supplemental oxygen. Appropriate antibiotics should be given in consultation with a physician.

Prevention

The single best way to prevent the problem is by acclimation to altitude as explained above.

Many outdoors persons are afflicted with strange (large) senses of ego that encourage them to press on despite all personal discomforts. Others see the plans of a trek disappearing in coughing spasms and vow to make the best of their hard-earned vacation money. The climber, cross-country skier, or hiker who reacts in this manner is likely to reach a state of collapse before his illness comes to the attention of his companions.

Early recognition is essential in dealing with high altitude pulmonary edema because if it is diagnosed and treated early, the disease is more reversible. The trip physician should examine every member of the party every day at high altitude. This should include noting whether they are consistently falling behind or if they have a headache, shortness of breath, or unusual weakness. The physician should listen to the chest and check for resting pulse and heart rate. If the heart rate is above 110 in a fit climber at altitude, or the respiratory rate is above 20 or rales are present, the patient should be restricted to bed rest, observed closely, and evacuation considered. If mildly affected individuals are returned to lower altitudes promptly, the incidence of serious complications and death should be reduced to almost zero.

High Altitude Cerebral Edema

In 1959, Chiodi described a patient who had neurologic signs and symptoms with exposure to high altitude in the Andes.[40] The signs and symptoms disappeared promptly with descent. A cerebral angiogram and flow studies were normal. This syndrome has been found repeatedly since that time. Data from small numbers of patients have shown that increased cerebral spinal fluid pressure, retinal hemorrhages, and papilledema have been present. Cerebral edema was verified in one instance by craniotomy performed for a suspected subdural hematoma.[41]

Although the term high altitude cerebral edema has been used for this complex of symptoms and signs, a better term might be high altitude encephalopathy. Cerebral edema may well be a consequence of the profound cerebral hypoxia, and not the cause of the various clinical manifestations of this syndrome.

Epidemiology

High altitude cerebral edema is the least common but most severe of the high-altitude illnesses. It almost always occurs at an elevation of greater than 12,000 feet. It is quite rare in the United States. If not treated promptly, it can result in death or permanent neurological injury.

It normally presents within 2 to 3 days of arrival at altitude. In rare instances, high altitude pulmonary edema may appear as coma or unconsciousness without cough or shortness of breath and may simulate cerebral edema.

Symptoms

Headache is the usual presenting symptom, although in some patients, it is curiously absent. The usual headache is a dull severe constant ache that requires narcotics for relief. The patient may complain of loss of memory, hallucinations, and confusion. Companions may note an inability to use proper judgment or psychotic behavior.

Signs

The first sign noted on physical examination is usually ataxia. This may be followed by ataxia of gait, mental confusion and somnolence. The climber may also "just want to stay in bed." Loss of memory, hallucinations, and lethargy may also be noted by the examiner. In the most severe cases, the patient can not be aroused and does not respond to verbal or even painful stimuli. Death will follow shortly if treatment is not emergently provided.

Fundoscopic examination may reveal papilledema and retinal hemorrhages. On neurologic examination, the patient may have focal neurologic deficits and upgoing Babinski's reflexes. Ataxia is the single most consistent neurological sign. The most common form of ataxia is truncal, where the patient is unable to maintain balance. In severe cases of ataxia, the patient cannot walk or stand without falling and may be unable to hold a cup or plate. Increasing heart rate, respiratory rate, and central cyanosis may signal concomitant HAPE.

Complications

The neurologic deficits that are seen with this disorder will usually resolve slowly with the increase in oxygen pressure and supportive treatment. Recent studies do show that climbers of Mt. Everest who do not use oxygen will have residual hypoxic neurological deficits, even if they did not have any symptoms of HACE. The longer that the high altitude cerebral edema victim remains at high altitude, the higher the propensity for permanent nervous system damage or death.

The heart and respiratory rate should be determined and the chest examined carefully in all cases, to ensure that the rare case of HAPE with coma is not missed. A chest x-ray should be obtained as soon as practical.

Prevention

Any confusion or ataxia combined with a severe headache should raise the possibility of cerebral edema in a member of a climbing or skiing party. These individuals should be assisted to descend, with oxygen if possible. Assistance is mandatory in view of the ataxia and the possibility of rapid progression with subsequent falls and injuries. The unconscious patient needs to be evacuated and hospitalized as soon as possible.

A simple test for early cerebral edema that may be performed even by lay trek leaders is the heel-to-toe gait test. The victim is observed as he walks a straight line with a heel-to-toe gait and is observed. If the victim loses balance and falls over, then he or she must descend. This test will help to detect the trekker who conceals symptoms in order to continue.

Treatment

Treatment of patients with diseases caused by an acute ascent to altitude follows uniform principles regardless of the underlying manifestation: AMS, HAPE, or HACE.

Emergency Descent

If the patient's symptoms worsen despite therapy, the patient should be returned to a lower altitude. The picture of a nauseated, vomiting, coughing, complaining climbing or hiking associate is nearly enough to motivate the homicidal. With the added stimulus of insomnia, Cheyne- Stokes breathing, and snoring respirations, one often can hope that one's tent mate simply doesn't take another breath.

Throughout this and subsequent discussions, it should be remembered that although emergency descent is often recommended, it is more easily proposed than performed. If the patient can travel under his or her own power, descent may be managed with 2 people for observation and aid. If a litter is required for a prolonged descent over rough terrain, no less than 12 will suffice for transportation and more will be appreciated by all. The leader MUST make the decision to descend early in the patient's course, before the individual is incapacitated. If there is no supplemental oxygen available, this decision must be made much earlier.

Gamow Bag

A small portable "hyperbaric" chamber has been developed by Dr. Igor Gamow. This appears useful in treatment of HAPE and has been approved for such use by the FDA. It is best suited for rescue situations where immediate evacuation is not feasible or the patient is too unstable. This 15 pound device will yield a drop in elevation of about 6000 feet. Symptoms of AMS will improve within 45 minutes and will last for about 12 hours when the patient has a 2-4 hour treatment. HAPE and HACE will require longer treatment (4 and 6 hours respectively) for full effect.

The Gamow bag is inflated to 2 psi with a foot pump that also serves as the air-exchange ventilation. A pressure-limited 2 psi pop-off valve allows the bag's contents to be vented while maintaining the desired pressure. It requires about 10-15 "pumps" per minute to keep the bag inflated.

A Gamow bag is constructed of 10 ounce polyurethane coated fabric and weighs about 15 pounds with necessary accessories. The fully inflated bag is about 2.5 meters long and .5 meters in diameter. It has two observation windows and a quick release air-tight zipper. The Gamow bag can be obtained by contacting Hyperbaric Mountain Technologies, 186 Canon Park, Boulder, CO 80302.

Supplemental Oxygen

Supplemental oxygen will decrease the effects of all of the acute altitude illnesses and should be considered to be part of the standard therapy of HAPE victims. With full flow oxygen, the respiratory and heart rates drop, and the patient will feel better. Unfortunately, very few parties carry enough oxygen to treat a victim of HAPE with 6 to 10 liters of oxygen flow per minute for 12 to 48 hours. Even if the available oxygen supplies will not last that long, it may permit a completely debilitated patient enough recovery to allow him to ambulate to a lower altitude. It should be considered as a temporizing measure and not a be used as a substitute for or delay prompt descent.

Steroids

Dexamethasone has been recently used for treatment of AMS with excellent effects and little incidence of side effects during the relatively short courses needed for this disease.[42] The mechanism of action is uncertain. When the medication was discontinued, the symptoms returned. Unfortunately, at least one study also showed that there was no OBJECTIVE improvement in the pathophysiology of exposure to high altitude. Dexamethasone may certainly be used to decrease symptoms and allow the patient to be evacuated in an upright position.

The recommended dose of dexamethasone (Decadron) is 8 mg IV or PO initially followed by 4 mg every 6 hours. This medication has interesting promise and needs further study to ensure the safety of the patient when the patient is to stay at altitude. The patient should be simultaneously treated with supplemental oxygen (if available) and either descent or hyperbaric pressure.

Specific Therapy

Acute Mountain Sickness

Moderate or mild symptoms of AMS are best treated by rest, increased fluid intake, and symptomatic relief. For severe symptoms of AMS, descent and supplemental oxygen are recommended.

Headache may be treated with aspirin, acetaminophen, mild narcotics such as codeine or propoxyphene, or combinations of any of these. A light diet should be followed, but anorexia and nausea may preclude any solid food intake. Alcohol and smoking should be strictly avoided.

Barbiturates or stronger narcotics should be avoided. Either class of drugs may depress respiratory efforts with subsequent catastrophic results. Barbiturates may make rest and sleep possible but may increase symptoms upon awakening. Oversedation may mask HACE. Diazepam may occasionally cause hypoxia, disorientation, and hallucinations. It has been suggested that if sleeping medications are to be used, the benzodiazepam class should be avoided; a medication such as Halcion (tm) is preferable because of the short duration of action.

The use of antiemetics may be helpful. There is some evidence that prochlorperazine (Compazine) may be one of the few drugs of its category that does not dull the respiratory response to hypoxia. Calcium carbonate tablets have also been advocated for the treatment of AMS. There appears to be little additional gain by the use of

Rolaids(tm), but there also seems to be no harm done by this medication. Some temporary relief of AMS may be achieved by forced voluntary hyperventilation. Ten to twelve deep breaths every 10 to 15 minutes will suffice. If the patient overdoes the "remedy," the symptoms of hyperventilation syndrome with dizziness and paresthesias about lips and distal extremities, soon appear.

HAPE

Once high-altitude pulmonary edema is diagnosed, the best treatment is immediate descent. As in acute mountain sickness, a relatively small descent of 1000 meters can improve the patient's status immensely. The patient with HAPE should be considered to have a reversible physiologic disorder. All except those with the most advanced cases will recover with descent, rest, and oxygen unless iatrogenic complications ensue. The Gamow bag has been shown to provide substantial relief for these patients, but the evacuation of the patient should still be scheduled - even with substantial improvement.

Long-term, high-altitude residents who have returned to altitude after a visit to the lowlands represent a special case. These patients may be treated with rest and low-flow oxygen. They do not need to be evacuated to a lower elevation unless their illness is critical.

Drugs other than oxygen and dexamethasone are not of much use in treating HAPE. Since the pathophysiology is that of a leaky membrane pulmonary edema that is similar to ARDS, it can be expected that morphine will have only limited effect, and this is borne out by clinical experience. The vasodilator effects of morphine may precipitate hypotension, although this is usually only postural. Morphine and other opiates carry the risk of decreasing respiratory drive with catastrophic results. In short, morphine carries severe complications in these patients. Digitalis derivatives and rotating tourniquets are likewise ineffective since the left atrial pressure is normal.

HACE

The optimum treatment for HACE is not yet well established but appears to be similar to that of HAPE. Prompt descent, high flow oxygen, and bed rest are the mainstays of therapy. As in HAPE, the best, mandatory, and most therapeutic treatment is descent while the disease is in an early phase. The Gamow bag has proven quite useful as a temporizing measure and may even allow the patient to descend with minimal help.

Based upon long-standing recommendations of our neurosurgical colleagues, hyperosmolar solutions have been commonly employed. Unfortunately, there are no good controlled studies, and only anecdotal observations bolster these clinical studies. Mannitol and glycerol may reduce the cerebral blood flow and worsen HACE's effects.

Non-Recommended Therapy

Furosemide

Furosemide has been recommended and frequently employed for the prevention and treatment of AMS and HAPE. The drug does not reduce the symptoms of AMS.[43] In normal persons, it has been noted that even small doses of furosemide may result in a substantial incidence of postural hypotension and near syncope at high altitudes.[44] Furosemide is contraindicated in the young and otherwise healthy patient with pulmonary symptoms at altitude. Of course, the complex presentation of an elderly patient who is visiting a high altitude and develops pulmonary edema may require the use of furosemide or other diuretics for complete therapy.

A profound diuresis is often necessary in the treatment of pulmonary edema of cardiogenic origin, and a clinician may be tempted to employ furosemide for HAPE. Unfortunately there is little rational basis for the use of furosemide in the non-cardiogenic pulmonary edema of altitude. The etiology of HAPE appears to be from a leaky capillary membrane and is most certainly not from volume overload. Furosemide will have no effect on the membrane leak of HAPE and therefore will not substantially help in its treatment. Indeed, furosemide may precipitate either a profound orthostatic hypotension or a significant non postural hypotension as a result of the brisk diuresis. Orthostatic hypotension with use of furosemide may be so debilitating as to prevent the patient from walking, thus complicating the already formidable problem of evacuation.

High Altitude Retinal Hemorrhage

Epidemiology

High altitude retinal hemorrhage occur in half of all people who travel above 17,000 feet (5200 M).[45] [46] Retinal hemorrhages are probably associated with an increase in retinal blood flow measured at high altitude.[47] They are uncommon below 15,000 feet (4570 M), but may be precipitated even at lower levels by strenuous activity.

Symptoms

In most cases, the retinal hemorrhages of HARH cause no visual difficulty and are seldom symptomatic. Only when the macula is affected is the individual aware of a scotoma.

Signs

To an examiner with an ophthalmoscope, the hemorrhages appear as small, flame-shaped markings or as large pools or blobs. Rarely, cotton-wool retinal edema markings are noted. The vessels show increases in tortuosity and in diameter. Hyperemia near the disk is also seen. Rarely, vitreous hemorrhages have been noted.[48]

Etiology

The etiology of retinal hemorrhages is not known. Theories proposed include a defect in vasoregulation, increased venous pressure due to Valsalva maneuvers with strenuous exercise, and a decrease in intraocular pressure associated with exercise.[49] [50] Available evidence suggests that retinal hemorrhages are due to an increased capillary permeability. Capillary permeability is increased as confirmed by fluorescein leakage in the region about the optic disk in 8 of 20 subjects.[45]

Treatment

No treatment or preventative medication is known. Acetazolamide does not appear to prevent HARH, but slow ascent or long stays at altitude do appear to reduce the daily incidence of retinal hemorrhages.[44] [46] Macular hemorrhages and cotton wool spots with scotomas probably should mandate descent. Retinal hemorrhages are not seen as contraindications to ascent, have not been shown to mark a hemorrhagic diathesis, and do not portend an abnormal vascular fragility.

The smaller lesions resolve without sequelae, but cotton wool spots and macular hemorrhages often leave a permanent scotoma. Most lesions resolve within 6 weeks after descent to sea level.

Prevention

There is no apparent ability to predict or prevent this disorder of altitude. Gradual ascent appears to reduce the rate at which HARH develops, but it is unclear as to whether it will reduce the incidence.[44]

Miscellaneous Altitude-related Problems

High Altitude Flatus Expulsion

HAFE is the natural consequence of trapped intestinal gases following the dictates of the combined gas laws. The combined gas laws state that the volume of a gas is directly proportional to the pressure and inversely proportional to the temperature of the gas. Since we are homeothermic and the temperature in our abdomen is relatively constant, this component can be neglected. The result is that as we ascend, our bowel gas is subject to less pressure and, hence, has greater volume. This can result in intraabdominal discomfort and the passage of large amounts of flatus. The syndrome has been associated with colonic rupture in at least one case.[51]

Fortunately the gastrointestinal (GI) tract has an egress to the outside world, so that the trapped gas may escape and not rupture the relatively fragile intestines.[52] Our tent mates and comrades may not always agree. Simethicone may provide some relief. Descent is curative.

Thromboembolic Disease

Venous Thromboembolism

When the dehydration that is common at high altitude, the polycythemic response to hypoxia of altitude, and the inactivity noted during storms and when preparing for the climb are added together, the high altitude sojourner has developed a high risk for thromboembolic disease.

The most frequently noted sites are the leg veins but the deep veins of the upper extremities and the pelvis have been involved. As is true for lowlanders, pulmonary embolism is a risk for patients with deep vein thrombosis. Under field conditions, little can be done for the patient with a pulmonary embolus except to administer oxygen if available. For the patient who has been evacuated to a hospital, the standard therapies for pulmonary embolus should not be modified.

Cerebral Thrombosis

Thrombosis of the venous cerebral circulation is an unusual complication of high altitude. Clinically it is quite similar to HACE, and the two are easily confused.[53] Japanese authors feel that the cause is due to polycythemia and a disturbance of the intracranial pressure due to AMS. Dehydration may be a contributing factor.

This disease must be diagnosed by CT scanning and a field diagnosis is simply not possible. Appropriate management is rapid evacuation to a hospital with suitable diagnostic equipment.

Dysbarisms

Dysbarisms are disorders that are related to changes in atmospheric pressure. Although they are not related to the hypoxia of high altitude, they are discussed in this section for completeness. Most physicians will see these disorders in patients who have been flying recently, not climbing, skiing, or hiking. The disorders are loosely grouped into 2 classes: barotrauma and decompression sicknesses. (See Chapter on Diving Emergencies).

Barotrauma

Barotrauma in general involves what is known as the "trapped gas" phenomenon. Undissolved gas within body cavities that has no exit to the ambient pressure will follow the physical laws governing gases. During ascent, the gas in these areas will expand. If the opening to the outside world is occluded, the increased gas pressure will cause pain and occasionally damage. During descent, the enclosed gas will contract. This is not usually a problem unless a thin membrane, such as the tympanic membrane, collapses.

Barotitis Media

As noted above, as the patient ascends, the gas trapped behind the tympanic membrane will attempt to equalize with the ambient pressure. Usually, this gas can easily escape through the eustachian tube. As the patient descends, the eustachian tube, if partially blocked, will collapse, and the venting will not occur. The difference in pressure is felt with increasing pain and pressure on the tympanic membrane.

On physical examination, the tympanic membrane is retracted in mild cases. In severe cases, the tympanic membrane is ruptured and blood is noted in the canal. The pain

may be quite intense until the tympanic membrane ruptures. Hearing may be disrupted.

Treatment is usually a long-acting nasal decongestant such as Afrin or cocaine. If practical, ascent to higher altitude and subsequent slow descent is often helpful. Swallowing, chewing gum, or yawning may open the eustachian tube and allow equilibration. Valsalva's maneuver (forced expiration against a closed mouth and nose) will also often help.

Barosinusitis

Poor drainage of the paranasal sinuses may lead to the trapped gas phenomenon within the sinuses. The frontal and maxillary sinuses are most frequently involved. Predisposing factors include an underlying chronic or acute sinusitis, edema of the nose from trauma or allergies, and a deformity of either septum or turbinates. This condition frequently occurs on descent and the patient will bitterly complain of pain about the affected sinuses. Blood may be present in the nose or mouth, and the sinuses will be markedly tender to palpation. X-ray studies will show the opacified sinus if fluid or blood is present.

Treatment consists of administration of both oral and nasal decongestants. The use of cocaine or an other equally powerful vasoconstrictive nasal decongestant is the quickest way to relieve the pain.

Barodontalgia

Those patients who have had a recent filling or capping of a tooth may have trapped air between the filling and the nerve root. As the atmospheric pressure is decreased, the increased pressure of the trapped air on the nerve root causes an exquisite pain. This pain is readily relieved with descent. Patients with barodontalgia should be advised to return to the dentist and have the filling or cap redone at leisure after the pain has been relieved by repressurization.

Rarely, the patient with an apical tooth abscess or severe caries may develop a similar pain. This dental pathology must be corrected as soon as practical.

Gastrointestinal Barotrauma

Ascent causes the intraluminal bowel gases to expand. The use of carbonated beverages or "beans" (legumes) prior to ascent will serve only to exacerbate the condition (see also HAFE). Belching and flatus are typical complaints. Since the GI

tract is open at both ends, the condition is self-correcting - to the consternation of those next to the patient and the embarrassment of those afflicted. The condition is an annoyance only at terrestrial elevations but may be serious at over 30,000 feet (9000 M) or in those who have had recent bowel surgery. If the patient has an ileus or has had a recent bowel anastomosis, the expansion of the trapped gas may cause a rupture of the affected portion of the intestinal tract.

Treatment is descent to the usual ambient elevation for the patient. Those with recent bowel surgery should not fly.

Peripheral Edema

Systemic edema of high altitude is seen after 4 to 10 days at high altitude. The overall incidence in one study of peripheral edema at 14,000 feet (4243 M) was 18%.[54] It appears in females more frequently than males but is not related to the menstrual cycle. The condition appears to be due to salt and water retention, which disappears spontaneously after returning to lower levels. The disorder is usually seen with both facial and peripheral swelling. It may make the patient quite uncomfortable but is not dangerous. It is often recurrent in the same patient each time he or she returns to high altitude.

Treatment may be delayed in most cases, and the edema will resolve after the trek is over. If the edema is marked, small doses of furosemide (40 mg daily) may be cautiously employed if there is no evidence of cardiac disease.

Prevention Of Altitude Illnesses

Extensive investigations into the altitude-related illnesses have included attempts to predict susceptible individuals and to define the early indications of the more severe forms of altitude-related illnesses. These efforts have included written graded examinations, physical examinations, psychometric testing, self-assessment, leader review and peer review of the performance of the subjects.[55]

In general, physical examination is rather unrewarding in prediction and in noting the EARLY effects of any of the altitude-related illnesses. Of course, it is an effective measure of more severe forms of any of the diseases. Heel-to-toe gait (as a test for ataxia) has been noted previously as a good objective test for the presence of HACE. It is easily performed by even lay leaders and is accurate as long as the subject is not on skis or crampons. Auscultation for the presence of rales is a similar test that is easily performed with a minimum of equipment and is relatively objective. Likewise, if a

subject develops fatigue that is out of proportion to exercise and altitude or lassitude, these symptoms are easily observable. Subjects with any of these findings should be accompanied to a lower altitude. The literature is replete with references to tragedies of patients who were sent alone to lower altitudes but did not arrive.

Recent work by Fletcher and colleagues showed that peer review, interview, and self-assessment all seemed equally effective at rating the effects of high-altitude illness but are good only for identifying the more severely and least severely affected individuals [54]. Daily interviews and examinations by a qualified medical observer were felt to be the safest procedures by which to identify illness.

Prevention

Acclimation

The single most effective method of prevention of altitude related-illnesses is by appropriate acclimation to altitude. This may be done in one of three ways:

Intermediate Staging

Prior to ascending to exercise at an altitude above 12,000 feet (3660 M), a program of gradually increasing exercise at an intermediate stop (6000 to 8000 feet or 1800 to 2400 M) for 3 to 4 days will provide sufficient acclimatization to prevent most problems at the higher altitude. For climbers to the 15,000 to 18,000 foot (4570 to 5500 M) range, a second acclimation stage at 12,000 to 13,000 feet (3660 to 4000 M) is helpful.

Graded Ascent

In many areas of the Himalayas, a long approach march is made to reach the high elevations. During the classic approach to Mt. Everest, for example, both acclimation and physical conditioning take place. At altitudes above 14,000 feet (4200 M), ascents should be limited to 500 to 1000 feet (150 to 300 M) between sleeping elevations, and every third or fourth day should be a rest day.

One Stage Ascent

For the trekker with no time to waste, an ascent directly to 12,000 to 14,000 feet (3660 to 4200 M) without stopping is usually planned. The trekker should plan on spending

at least 3 days at the altitude before engaging in strenuous activity. The risk of all altitude illnesses after such an ascent is high, and heavy exertion prior to acclimatization is foolhardy. It is usually more pleasant to spend the 3 to 4 days planned for acclimation at the lower altitudes.

Acetazolamide

Acetazolamide is a carbonic anhydrase inhibitor that increases the urinary excretion of bicarbonate, sodium, and potassium. The metabolic consequence of the forced bicarbonate diuresis is acidosis. This metabolic acidosis is felt to promote the sensitivity of the peripheral oxygen-sensitive chemoreceptors and stimulation of the medullary chemoreceptors. The beneficial effects of this increased sensitivity to hypoxia include increased ventilation at altitude, increased alveolar oxygen tension, and possibly improved sleep patterns.

Acetazolamide may be given in a dose of 125 to 250 mg once or twice a day beginning on the day prior to ascent. It should be continued until the patient is at the same or lower altitude for 5 days. Treated persons at high altitude have higher arterial oxygen tension, a lower arterial carbon dioxide tension, and a lower pH than control subjects.[56] Field studies of troops show that AMS at 14,200 feet (4330 M) can be ameliorated by a combination of staging at 5500 feet (1675 M) and administration of acetazolamide for the last 2 days of staging and the first 2 days of the new altitude. Hornbine also demonstrated an overall higher success rate in achieving the summit of Denali in those individuals using acetazolamide as compared to placebo.[57] This was apparently due to a combination of decreased symptoms of AMS and improved ability to breathe. It does not appear to improve exercise performance at extreme altitudes.

Side effects of acetazolamide are common and may be incapacitating for a climber, although less troublesome for a trekker or skier. The most common side effects include paresthesias and hypoesthesias about the lips and fingertips. The paresthesias in a climber who is depends upon touch for climbing ability or to clutch an ice ax or ski pole may be exceedingly troublesome. This symptom complex is attributed to a transient peripheral neuropathy that may vary from a mild discomfort to a major annoyance. Other side effects such as a flat taste to beer and cola drinks and myopia may occur after a few days of medication.

Who should not go high

In general, persons who are active at sea level, who have no uncontrolled illnesses, who are sensible enough to be careful, slow, and alert to the problems discussed should not be banned from the mountains. They may not be able to tolerate the expedition to the very high and extreme altitudes, however.

A number of diseases that occur at low altitude will be predictably worsened by the hypoxia of high altitude. There are four groups of patients for whom this hypoxia is a major consideration.

Chronic Hypoxemia

Individuals with chronic pulmonary disease, congestive heart failure, or cyanotic congenital heart disease with chronic hypoxemia and low arterial oxygen saturation will have a marked risk associated with ascent. The presence of hypoxemia at the lower altitude appears to be a major risk factor for all of the above diseases. This is due to the lack of reserves available for the extra work of breathing and by the shape of the oxyhemoglobin dissociation curve.

The arterial oxygen desaturation caused by the relative hypoxemia of the disease places the patient at a downslope on the oxyhemoglobin dissociation curve. Once the blood is less than 90% saturated with oxygen, the curve is steeper and even small changes in the oxygen available will cause a marked change in the amount of oxygen that the blood can deliver to the tissues.

Even among permanent residents at high altitudes, mortality is higher in the mountains than in the nations as a whole. Deaths from emphysema occur at a lower age, after shorter durations of illness, and more commonly involve cor pulmonale than at lower altitudes, where pneumonia is a more common cause of death.[58]

Cardiovascular Disease

Another group of patients who should not ascend are those patients with severe angina or heart failure. At the same time that the patient has decreased his available oxygen supply by ascending, the work of breathing is increased, and consequently the myocardial oxygen consumption has increased. This may have disastrous results for the patient with marginal coronary blood flow.

Pulmonary Hypertension

Patients with primary pulmonary hypertension may be at the highest risk. In addition to the arterial desaturation noted in the other risk groups, these patients have a primary disease process that is worsened by the normal vascular response to high altitude. In fact, one of the highest risk factors for HAPE is the presence of only one pulmonary artery.

Sickle Cell Crisis

Sickle cell disease is well known to be worsened by hypoxemia of any cause. Needless to say, the hypoxemia of high altitude can precipitate a sickle cell crises. Certainly at 15,000 feet (4570 M), the arterial oxygen tension is low enough to precipitate a massive sickle cell crisis, but crises have been observed at altitudes as low as 11,500 feet (3500 M).

Summary

Four major variables appear to determine the susceptibility to altitude illnesses: rate of ascent, altitude reached, length of stay, and amount of vigorous exercise. The altitude sicknesses are a continuum of symptoms that are grouped into three major categories: AMS (headache, nausea, and dyspnea); HAPE (dyspnea, cough, and coma); and HACE (headache, ataxia, and coma). High altitude retinal hemorrhages and peripheral edema are encountered at the higher altitudes but do not carry the serious connotations of the first three illnesses.

Rate of ascent, if adjusted to the individual is an excellent preventative measure. Acetazolamide may aid in acclimation to altitude and decrease symptoms.

Descent at an early stage of serious illness is the best therapy. Indeed, if descent is early, it may be the only therapy needed.

The final and most important point is that climbing or skiing at altitude is a sport of risks, and the individual responsibility and education of the climber or skier is the single most important preventative measure taken. This educational preventative is best taken at leisure prior to the climb, not learned in the schools of hard experience proctored by Mother Nature.

Questions

1. Which of the following statements is true:

   A. Cheyne-Stoke's breathing due to altitude illness occurs only above 12,000 feet.
   B. Sedatives are the drug of choice for prevention of AMS.
   C. Sedatives may decrease oxygenation.
   D. Oxygenation is better during sleep at altitude.
   E. Acetazolamide can worsen HAPE.

2. Following the climber's adage of "climb high - sleep low" decreases:

   A. The severity of hypoxemia.
   B. The likelihood of severe AMS.
   C. The likelihood of developing severe high altitude pulmonary edema.
   D. All of the above.
   E. None of the above.

3. A staged ascent includes all of the following *except*:

   A. Spending 2 nights at the same altitude for every 2,000 foot gain above 10,000 ft.
   B. Proceeding higher than the night's camp during the day and descending to sleep.
   C. Moving more slowly under conditions of extreme cold, heat, or severe exertion.
   D. Ensuring a high fat diet is available to provide extra energy sources for high exertion.
   E. Gradually increasing the sleeping altitude (1-2,000 feet per night)

.

4. Which of the following are NOT symptoms of AMS?

   A. Coma.
   B. Anorexia.
   C. Nausea.
   D. Headache.
   E. Insomnia

5. Which of the following are NOT symptoms of HACE?

   A. Coma.
   B. Ataxia.
   C. Deterioration in judgment.
   D. Cough.
   E. Hallucinations.

6. Which of the following are NOT symptoms of HAPE?

   A. Cough.
   B. Dyspnea on exertion.
   C. Fever.
   D. Ataxia.
   E. Impaired consciousness.

7. Treatment of HAPE should include the following:

   A. Lasix (furosemide) 80 mg IV stat.
   B. Acetazolamide 250 mg IV stat.
   C. Diazepam 5 mg IV stat.
   D. Descent to lower altitude immediately.
   E. Dexamethasone 6-8 mg IV stat.

Answers

1. **Answer: C**. The reason not to give sedatives to people with acute altitude illnesses is that they may markedly decrease oxygenation - particularly during sleep. Acetazolamide has not been shown to worsen HAPE and is a preventative measure for AMS. Cheyne-Stokes breathing has been seen as low as 8,000 feet.

2. **Answer: D.** It is precisely because sleeping low decreases hypoxemia that it helps to prevent both AMS and HAPE.

3. **Answer: D.** Actually, a low fat, high carbohydrate diet is often better tolerated at altitude. A diet of at least 70% carbohydrates decrease the symptoms of AMS by about 30%.

4. **Answer: A.** AMS does not characteristically include coma as a symptom. When coma intervenes, the illness is classified as either HAPE or HACE.

5. **Answer: D.** HACE (High Altitude Cerebral Edema) does not include coughing as a symptom.

6. **Answer: C.** HAPE (High Altitude Pulmonary Edema) does not cause a fever. It is normally simply a leaky membrane pulmonary edema.

7. **Answer: D.** None of the drugs listed are appropriate for the treatment of HAPE (High Altitude Pulmonary Edema). Descent is the treatment of choice.

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