Chapter 26

ADDITIONAL MEDICAL PROBLEMS IN MOUNTAIN ENVIRONMENTS

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INTRODUCTION

Acute mountain sickness (AMS), high-altitude cerebral edema (HACE), and high-altitude pulmonary edema (HAPE), which were discussed in the two previous chapters of this textbook (Chapter 24, Acute Mountain Sickness and High-Altitude Cerebral Edema, and Chapter 25, High-Altitude Pulmonary Edema), are probably the most familiar and notorious altitude-associated medical problems to mountaineers and medical personnel. These disorders occur only in high mountains, are common, and are potentially life-threatening. From a military standpoint they are important because they increase the rate of disease and nonbattle injuries and degrade performance, both of which can jeopardize a military unit's mission. These well-known altitude illnesses are not the only medical problems that occur in mountain terrain, however, and certainly are not the only ones to have significant potential impact on soldier performance. The complex tableaux of terrain and climatic features of mountains can cause a variety of medical problems in visitors and permanent inhabitants alike. Although many of these problems are not caused by hypoxia and, therefore, are not unique to high altitude (ie, hemorrhoids), the specific *constellation* of problems is characteristic of high mountain environments. Steele's[1] lively description of the medical support of the 1971 International Himalayan Expedition, for instance, provides an example of the scope of injury and illness that can be inflicted by the mountain environment. To be adequately prepared to care for people in the mountains, military and civilian medical personnel should be familiar with the whole range of problems that can occur.

The purpose of this chapter is to describe the types of illness and injury other than AMS, HACE, and HAPE that commonly occur in high mountain environments. The intent is to facilitate an awareness of the characteristic constellation of problems so that medical personnel supporting military units deploying to mountain regions are prepared to treat more than the familiar altitude illnesses. Problems that are unique to mountains (ie, those directly related to hypobaric hypoxia) are described in some detail. These include peripheral edema, altitude-related eye problems, sleep disturbances, thromboembolic disorders, suppression of immune function, wound healing, high-altitude pharyngitis and bronchitis, and exacerbation of preexisting medical conditions. Conditions that occur in mountains but may be more characteristic of other environments (eg, cold injury, carbon monoxide poisoning) are described in less detail. Readers are referred to other chapters in this volume or different volumes in the *Textbook of Military Medicine* series for more-extensive discussion of those problems, and to Exhibit 19-1 in Chapter 19, Mountains and Military Medicine: An Overview, for definitions of the categories of visitors to high mountains.

MEDICAL PROBLEMS CAUSED BY HYPOXIA

The spectrum of hypoxia-related medical problems other than "classic" altitude illness is a potpourri, affecting different body systems and ranging from benign to serious in consequence and from common to rare in occurrence. All of these problems can have a negative impact on military operations by diminishing unit manpower or degrading individual performance. Because they are related to hypoxia, they tend to be increasingly frequent and more severe at higher elevations. Most are rare and mild at altitudes under 3,000 m and quite common at altitudes over 5,000 m. As with AMS, HACE, and HAPE, the most effective treatment for all of these hypoxia-related problems is descent to lower altitude, a treatment option that may not always be available during bad weather or combat.

High-Altitude Peripheral Edema

Given that the progressive hypobaric hypoxia in high mountain environments can cause edema in the brain and lungs, it should not be surprising that edema occurs in other tissues as well. "High-altitude peripheral" or "systemic" edema refers to edema of the face, extremities, or both during altitude exposure in the absence of other causes of edema (eg, congestive heart failure, cirrhosis, kidney failure). Two manifestations of altitude-related peripheral edema are described in the literature, one associated with altitude illness, the other occurring as a seemingly isolated clinical condition. Additionally, "stasis" edema (which is not caused by hypoxia and can be seen in other settings) can occur in the arms and legs due to impeded venous blood return by constriction from climbers' bulky, multilayered clothing or from tight straps on rucksacks and climbing harnesses.

Although the Moguls in central Asia apparently described "swelling of the hands and feet" as a sign of mountain sickness in the 14th century,[2] and

climbers have been aware of the condition for many years, the first descriptions of high-altitude peripheral edema in the modern medical literature were in the 1960s and early 1970s. Waterlow and Bunjè[3] recorded "oedema of the hands and face" in a single volunteer test subject whose symptoms were consistent with HACE. Singh and colleagues[4] reported lower-extremity edema occurring in many soldiers who had symptoms of altitude illness. Shortly thereafter, a seemingly benign form of peripheral edema affecting the face and extremities while climbing a mountain in the tropics was reported by Sheridan and Sheridan.[5] Although some literature describes this peripheral edema without accompanying altitude illness as if it were an independent entity,[6] the consensus is that all altitude edemas are manifestations of the same pathological process, a concept first proposed by Hackett and colleagues.[7]

Signs and symptoms of high-altitude peripheral edema include facial edema, often prominent periorbital edema, and/or edema in the upper and lower extremities (Figure 26-1). It is associated with a weight gain of 4 to 6 kg (6–12 lb),[6] or as much as 6% of body weight.[8] Steele[1] described a young woman whose peripheral edema at 5,300 m (17,800 ft) was accompanied by a weight gain of 8.6 kg (19 lb), or approximately 20% of her normal body weight. The weight gain associated with peripheral edema is often apparent as a bloated feeling or uncomfortable tightness of clothing. Facial edema may become apparent as creases left in the skin by sun goggles or headbands. Likewise, edema of the extremities may cause skin indentations left by rings, watch bands, gloves, or socks and boots.

The incidence of high-altitude peripheral edema has not been well delineated but may be substantial even at lower altitudes. Hackett and Rennie[9] documented a 16% to 18% incidence in trekkers at 4,243 m in the Himalayan mountains, while Bärtsch and colleagues[10,11] reported a 40% to 70% incidence in male mountaineers at 4,559 m in the Alps. High-altitude peripheral edema appears to be more common in women than men but is not clearly associated with the fluctuation of menstrual cycle hormones or with ingestion of birth control pills or estrogen replacement therapy.[9,12] It also is much more common in individuals with altitude illness. Bärtsch and colleagues[11] found facial edema in 73% of individuals with AMS, compared with 28% in individuals without. Hackett and Rennie[12] reported some form of peripheral edema in 64% of individuals with AMS and 43% of individuals without.

There is a general consensus that the underlying pathophysiology of all the altitude-related edemas is similar,[7] and various postulated mechanisms are presented in Chapter 21, Human Adaptation to High Terrestrial Altitude; Chapter 24, Acute Mountain Sickness and High-Altitude Cerebral Edema; and Chapter 25, High-Altitude Pulmonary Edema. Low arterial oxygen saturation (Sa_{O_2}) from relative hypoventilation probably initiates increased capillary and small-vessel leakage[8] through activation of permeability mediators.[13,14] Increased blood flow and blood pressure in specific organs and tissues, mediated through increased sympathetic nervous activity, accelerates edema formation in the brain, lungs, and peripheral tissues.[15] Insufficient peripheral arterial chemoreceptor-mediated diuresis and natriuresis,[16] changes in renal blood flow,[17] and perturbations in the fluid-volume regulatory hormones (renin-angiotensin-aldosterone system, arginine

Fig. 26-1. High-altitude peripheral edema manifested by periorbital and facial swelling is seen in a woman at about 4,400 m during a mountain-climbing expedition. Other common signs of this condition include body-weight gain and swelling in the distal portions of the arms and legs. Altitude-related peripheral edema is often associated with altitude illness, and this woman had symptoms of acute mountain sickness. High-altitude peripheral edema may be more common in women than men but is not related to either the menstrual cycle or hormone therapy. Photograph: Courtesy of Peter Hackett, MD, Seattle, Washington.

vasopressin, and atrial natriuretic peptide) due to hypoxia,[10] exertion, or both probably also contribute.[11,18] The extent and distribution of edema formation is a function of the complex interaction of all these factors and possibly more.

Diagnosis of high-altitude peripheral edema is based on the presence of signs and symptoms of edema in the face or extremities following ascent to a higher altitude. A history of previous episodes in mountain terrain helps to strengthen the diagnosis. Some individuals seem to develop peripheral edema consistently above a certain "trigger" altitude.[6,12] It is important to rule out other potential causes of peripheral edema, especially edema associated with life-threatening medical conditions. The differential diagnosis includes the usual causes of edema seen at sea level such as congestive heart failure, cirrhosis, renal failure, and so forth, none of which is likely in a relatively young, healthy, military population. Anand and colleagues[19] reported congestive heart failure in young soldiers deployed to extreme altitude for long periods of time, however. Most of the conditions included in the differential diagnosis can be ruled out through clinical history and physical exam. Stasis edema due to pressure of pack straps or constrictive clothing is identified by its resolution after the constriction is removed. Given that most altitude-induced peripheral edema is associated with other forms of altitude illness, its presence should prompt an evaluation for HAPE or HACE.

Recommended treatment of uncomplicated high-altitude peripheral edema is poorly defined because no controlled studies have been reported. The usual treatment is salt restriction and induction of a mild diuresis. Adequate salt restriction can usually be achieved by avoiding foods with high sodium content (eg, meat jerky, junk foods, some ethnic foods, many military rations) and by not adding salt to foods during preparation or consumption. When necessary, appropriate diuresis can be achieved with low-dose furosemide (20 mg/d) or another diuretic. Prophylaxis consists of starting salt restriction several days prior to ascending and starting a diuretic during the ascent. Prophylaxis for AMS using acetazolamide will also prevent peripheral edema and may be the best choice for individuals with a history of AMS accompanied by peripheral edema.[20]

The clinical course of altitude peripheral edema is not well documented, but the prognosis appears relatively good if it is not associated with more menacing forms of altitude illness. Many untreated individuals remain edematous for the entire time they are above their trigger altitude but diurese rapidly when they return to lower elevations.[6] Others may eventually diurese at high altitude as they acclimatize. Most are uncomfortable enough while edematous that they prefer not to remain at altitude waiting to acclimatize. Treatment and prophylaxis are said to be nearly universally effective, but in susceptible individuals, edema may recur on every ascent if not treated. Given the benign but predictably recurrent nature of this condition, susceptible military personnel do not need to be medically restricted from high altitude but should probably receive prophylactic treatment when the mission takes them into mountain terrain.

Problems in the Eye

Several problems can occur in the eyes during high altitude exposure. Both the retina and the cornea are affected by hypoxia. Snowblindness from exposure to high levels of ultraviolet (UV) radiation is also a potential problem. Although most altitude-related eye problems are temporary, changes in visual acuity can increase the risk for accidents and put a soldier at great risk during combat.

High-Altitude Retinopathy

The term high-altitude retinopathy (HAR) describes a spectrum of retinal changes associated with exposure to prolonged hypobaric hypoxia at high altitude. The changes include retinal hemorrhage, vitreous hemorrhage, cotton wool spots, and optic disc edema. HAR is common during altitude exposure and has been well documented for several decades. In the 1930s, Sedan[21] described retinal hemorrhages in hypertensive subjects at 3,650 m. Singh and colleagues[4] reported papilledema and engorgement of retinal veins and vitreous hemorrhages in Indian troops exposed to high altitude in 1969. In 1970, Frayser and colleagues[22] reported a 37.5% incidence of high-altitude retinal hemorrhages (HARHs) among otherwise healthy climbers at 5,360 m on Mount Logan in Canada. McFadden and colleagues[23] first reported cotton wool spots in the retina at high altitude in 1981. Numerous other reports have described high-altitude retinal changes in studies of climbers,[24–31] at high-altitude laboratories,[22,23] and in a hypobaric chamber.[32]

The first retinal changes associated with altitude exposure are usually disc hyperemia and an increase in retinal vascular diameter and tortuosity (Figure 26-2).[22–26,33–36] These changes are almost universally observed at altitude and probably represent a normal

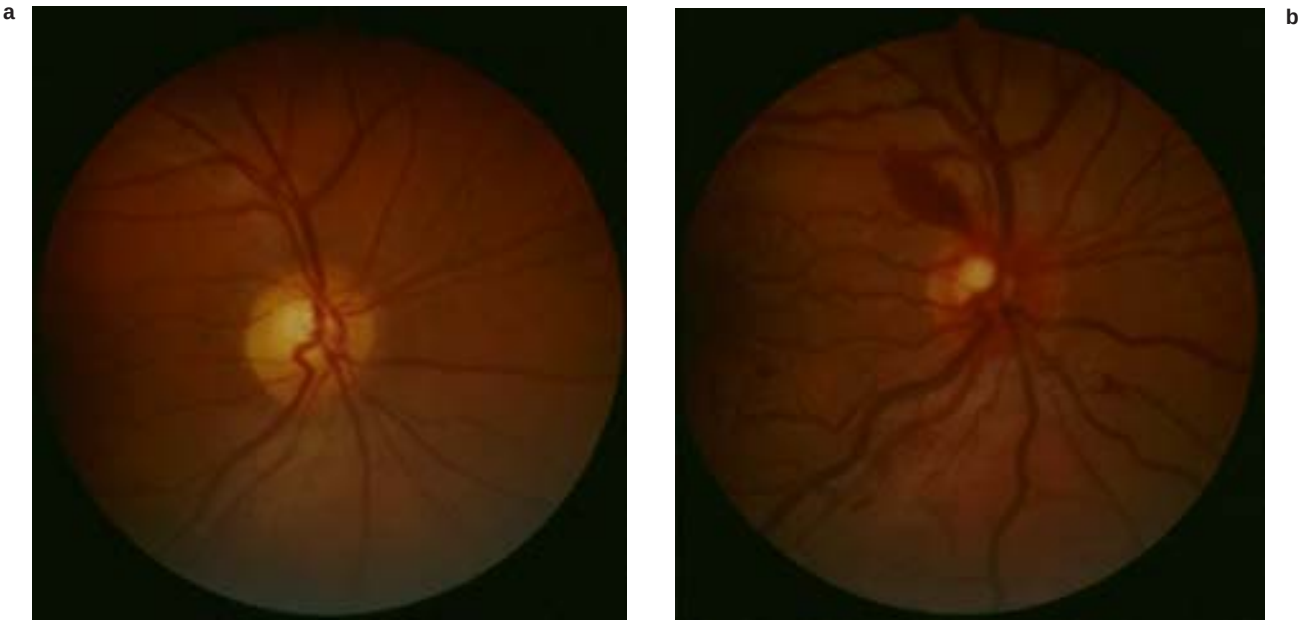

Fig. 26-2. High-altitude retinopathy in the right eye of a 26-year-old male volunteer test subject (**a**) at sea level and (**b**) at 7,620 m altitude-equivalent pressure in a hypobaric chamber following a gradual decompression over 4 weeks.[1] At high altitude, the vessels are engorged and tortuous. There is a large, flame-shaped hemorrhage in the peripapillary region. There are smaller flame-shaped hemorrhages further away from the optic disk and a "dot-blot" hemorrhage near the macula. There is slight papilledema. The retinopathy did not cause symptoms and resolved completely over several weeks following descent. (1) Rock PB, Meehan RT, Malconian MK, et al. Operation Everest II: Incidence of retinal pathology during simulated ascent of Mt Everest [abstract]. *Federation Proceedings*. 1986;45:1030.

response to hypoxia (as opposed to a true pathological condition). Although apparently benign, retinal vascular dilation and tortuosity may be striking on examination, and the mechanism of these changes may provide some insight into the early retinal vascular response to altitude exposure. The overall blood supply to the retina is from the terminal branches of the internal carotid artery. The retina itself has a dual blood supply. The central retinal artery branches from the optic disc to supply the inner layers of the retina, and its diameter is controlled by autoregulation. The choroid, which supplies the outer retina, is a spongy vascular plexus without autoregulation. With increasing altitude, there is a precipitous drop in ocular oxygenation.[37] As the level of oxygenation decreases, autoregulation causes observable dilation of retinal arteries and veins. In conjunction with the increase in retinal vascular diameter, there is a concurrent lengthening of the vessel. Because the vasculature is tethered to the retina and cannot elongate longitudinally, this lengthening results in vascular tortuosity. These changes progress with increasing altitude and may be prominent at extreme altitude. The changes are purely vascular, however, and reverse with decreasing altitude, leaving no residual effects.

The most widely recognized manifestation of HAR is HARH. These retinal hemorrhages have been reported extensively in the medical literature, particularly since the early 1970s. Although one report[32] described HARH in subjects exposed to hypobaric hypoxia in an altitude chamber, all other reports have involved subjects exposed to high altitude in mountain laboratories[22,23] or during mountaineering expeditions.[21,22,24–31]

The exact appearance and position of the hemorrhages varies somewhat but most are flame-shaped, "dot and blot," or splinter hemorrhages and appear in the peripapillary area or near or between the vascular arcades. Fundus photographs suggest that the hemorrhages can be preretinal,[22,23] intraretinal,[29,36] or extend into the vitreous.[4,29] Fluorescein angiography of established hemorrhages has demonstrated blockage of underlying fluorescence with no leakage in the hemorrhagic area.[23]

Hemorrhages occurring outside the macula are usually asymptomatic, self-limited, and resolve without sequelae. HARHs are *not* always transient and innocuous, however. Macular hemorrhages may cause a sudden loss of central vision.[27] Al-

though some degree of recovery can be anticipated, a permanent decrement in central visual acuity or a pericentral scotoma can occur following macular hemorrhage.[22,23,31]

There is no clear threshold altitude for occurrence of HARHs, and they have been reported at altitudes as low as 3,650 m.[21] The reported incidence is extremely variable, probably owing to differences in altitude, rates of ascent, method of documentation, and duration of altitude exposure. In general, however, the higher the ascent, the higher will be the incidence and severity of HARH. HARHs occur in approximately one third to one half of subjects exposed to altitudes in excess of 5,000 m.[23,26,28,30] They occur in nearly 100% of those ascending above 6,500 m.[25]

Although HARH has been observed in both healthy climbers and those with AMS,[22] HARH may be more common in those with AMS. Schumacher and Petajan[28] reported a progressive rise in the incidence of retinal hemorrhages correlated with increased intensity of high-altitude headaches. Hackett and Rennie[9] found that of 13 mountain trekkers with HARH, 11 had AMS, 7 had pulmonary rales, and 4 had HAPE or HACE. Wiedman and Tabin[38] reported a statistically significant correlation between HAR and HACE in climbers who reached altitudes between 4,800 and 8,854 m on Mount Everest; although they reported a high incidence of HACE, however, they did not report the criteria that they used for diagnosing HACE.

There is conflicting information regarding the effect of acclimatization, as reflected by slower ascent rate, on the incidence of HARH. In a study[9] of more than 200 trekkers at 4,243 m in the Himalayan mountains examined for retinal hemorrhage, no difference was found in the incidence of HARH in subjects who flew to an altitude of 2,800 m and then ascended on foot compared with those who walked from 1,300 m and, therefore, were better acclimatized. One other report[22] also suggests no relation between HARH and rate of ascent. This is in contrast with studies that suggest that the incidence of HARH was decreased in subjects who had undergone gradual, as opposed to more rapid, ascent.[23,28,29]

HARH may be exacerbated by exercise. During a study at 5,360 m on Mount Logan in Canada, McFadden and colleagues[23] observed new hemorrhages in 7 of 34 subjects after maximum exertion on a cycle ergometer. Additionally, peripapillary fluorescein leakage associated with exercise-induced hemorrhages was noted in 8 of 20 subjects examined with fluorescein. Whether the retinal vasculature leakage was due to increased mechanical pressure from increased blood flow, exercise-induced hypoxemia, or a combination of both, was unclear.

The pathogenesis of HARH is a subject of debate but is probably due to the complex interaction of several factors. The hypoxia-induced increase in retinal blood flow at high altitude is accompanied by retinal vascular dilation. With dilation, linear velocity of the blood decreases and lateral pressure increases, causing more mechanical stress on the vessel wall.[24] It is possible that while the vasculature is distended, increases in venous back pressure may cause fragile vessel walls to rupture. Valsalva stress associated with coughing, defecating, and strenuous exercise may cause an elevation in reflex venous pressure in the eye and increase the likelihood of vessel rupture.[24,26,28] The finding of fluorescein leakage following vigorous exercise at 5,360 m reinforces this hypothesis.[23] The hypothesis is also supported by reports of retinal hemorrhages similar in appearance to HARH associated with Valsalva stress in the absence of hypoxia.[39,40]

Hypoxia and hypocapnia may have a direct effect on the vascular endothelium, causing increased vascular permeability.[22–24,41] Microscopic examination of retinal tissue from a climber who died as a result of HACE after an ascent to 5,330 m documented endothelial damage to the retinal vasculature at the nerve fiber layer level in an area of macroscopic retinal hemorrhage.[42] Based on their findings, Lubin, Rennie, and Hackett[42] hypothesized that hypoxia may cause a defect in capillary endothelial integrity, leading to extravasation of blood. They concluded that the hemorrhages were from both retinal capillaries and retinal veins. The fact that HARHs frequently occur away from major vessels also supports a capillary origin. Because most retinal hemorrhages occur in the nerve fiber layer within the distribution of the radial peripapillary capillaries, it is possible that some HARHs may also occur in this vascular net. The radial peripapillary capillaries are a distinct strata of capillaries occupying the superficial portion of the nerve fiber layer in the peripapillary retina.[43–46]

Many other possible causes for HARH have been suggested. Decreased intraocular pressure (IOP) reported in some altitude studies has been hypothesized to set the stage for HARH, presumably due to the decreased tamponade effect of IOP on the retinal vasculature.[31,35,36,47] However, not all studies have found a decrease in IOP at altitude.[29,48] Systemic hypertension has also been implicated as a causative factor.[24] Other possible predisposing factors include hemoconcentration and blood hyperviscosity caused by fluid shifts and dehydration at

altitude.[31,42,49] These conditions may lead to increased coagulability and decreased oxygen transport, which could contribute to the pathogenesis of HARH.

Some researchers suggest that high-altitude exposure may lead to low-grade cerebral edema, increased cerebrospinal fluid pressure, mild papilledema, and compression of retinal veins, all of which may predispose a climber to HARH.[31,42] However, in experimentally produced intracranial hypertension in rhesus monkeys, Hayreh and Hayreh[50] documented that venous dilation is a comparatively late phenomenon and should not be used as a criteria for early papilledema. Even eyes with marked optic disc edema showed no capillary dilation or retinal hemorrhages.[50]

The actual contribution of any of the proposed factors predisposing to HARH is open to question. Additionally, a wide spectrum of retinal changes is noted in individuals with nearly identical climbing profiles (ie, altitude exposures). Thus, retinal vascular response to altitude appears to be diverse, suggesting that different factors may be more or less important in different individuals.

Other than avoiding high-altitude exposure, there are few agreed-on measures to prevent HARH. Although anecdotal information suggests some protection from previous exposure to high altitude, there appears to be no significant correlation between preclimb altitude exposure and the incidence of HARH.[29,30] Acetazolamide decreases the symptoms and incidence of AMS but has not been conclusively demonstrated to lessen the incidence of HARH.[22,23] Although evidence is scanty, gender and race probably have no impact on the rate of occurrence or extent of HARH.[23] Since HARH can be associated with AMS, it seems logical to follow the same general guidelines that are used to lessen the symptoms of AMS (see Chapter 24, Acute Mountain Sickness and High-Altitude Cerebral Edema). Avoidance of vigorous exercise and performing Valsalva maneuvers at altitude also may be prudent.

There is no specific treatment for HARH, and the hemorrhages usually resolve in 2 to 3 months.[24,31] Administration of oxygen may alleviate symptoms of AMS, but it does not necessarily prevent or decrease the extent of retinal hemorrhages. Hemodilution, which may decrease blood viscosity and coagulability in dehydrated climbers, has been reported[49] as a treatment for retinal hemorrhages associated with visual decrement. Documentation of the best corrected baseline visual acuity to provide a comparison for any subsequent visual loss is prudent. Any unexplained visual complaints during altitude exposure should prompt bilateral, dilated fundus examination to search for intraocular pathology. As with all altitude illness, the most effective measure to prevent further hemorrhage is evacuation to a lower altitude, preferably lower than 3,000 m. Asymptomatic HARHs, those not involving the macula, are usually not considered significant enough to dictate descent to a lower altitude.[23] It may be prudent to evacuate individuals with macular or vitreous hemorrhages to a lower altitude, because such changes may interfere with central vision and could worsen with increasing altitude. It is also advisable that individuals with previous symptomatic retinal hemorrhages not return to high altitude because of the possibility that hemorrhage will recur to the same area.

Several authors have hypothesized that owing to similarities in the cerebral and the retinal circulation, changes observed in the retina may also occur simultaneously in the brain.[25,42] Such changes were documented by Lubin, Rennie, and Hackett[42] in autopsy studies. From these studies, Wiedman and Tabin[25] concluded that (1) HARH may be a prognostic indicator for altitude illness and (2) those ascending to high altitude should be monitored by funduscopic exam for HARH as an early warning sign of impending, more-serious altitude illness. Because HARH may occur in healthy individuals or those with minimal symptoms of AMS, it is probably not necessary to do serial dilated exams on all asymptomatic individuals at high altitude. This obviously would be very difficult in a military unit deployed in a tactical situation. However, Wiedman and Tabin[25] recommend that if a person has symptoms or signs of cerebral edema, then careful serial fundus examinations may provide useful additional information.

Edema of the optic disc at high altitude has been well described, and it occurs as a result of HACE. Ocular manifestations include pronounced disc hyperemia and swelling. It should be emphasized that, using direct ophthalmoscopy, it can be difficult to distinguish prominent disc hyperemia and vasodilatation from true early disc edema. Binocular examination by a 78-diopter (D) or 90 D lens at a slitlamp is probably the most reliable method of documentation. Because the facilities for such examinations are rarely available at altitude, serial examinations using a hand-held direct ophthalmoscope are usually the most practical option. Regardless of specific etiology, *true disc edema represents the ocular manifestation of increased intracranial pressure and should be considered a life-threatening condition.*

Immediate evacuation to a lower altitude is mandatory, with treatment as detailed in Chapter 24, Acute Mountain Sickness and High-Altitude Cerebral Edema.

High-altitude exposure may predispose a climber to other sight-threatening conditions. Although not common, cotton wool spots (an area of infarction of the nerve fiber layer[51]) have been observed at high altitude.[23,29] Since systemic hypoxemia would not be expected to cause localized areas of ischemia, it is hypothesized that these may occur as a result of microembolization. Microemboli may be formed by platelet aggregates, which have been reported to develop during hypoxic conditions.[23,52] Cotton wool spots cause scotoma in the visual fields corresponding to the size and location of the lesion. McFadden and associates[23] documented a persistent visual field loss 2 years after a well-documented occurrence of a cotton wool spot during a high-altitude laboratory study. There is no specific treatment for cotton wool spots, but descent to a lower altitude is never inappropriate.

Central retinal vein occlusion with permanent visual loss has also been reported at altitude. Butler and colleagues[29] described a patient whose vision, after what was thought to be a vitreous hemorrhage, deteriorated to "finger counting" following ascent to 5,909 m. Following resolution of the vitreous hemorrhage, a widespread area of capillary nonperfusion was noted by fluorescein angiography, consistent with ischemic central retinal vein occlusion. The patient subsequently underwent panretinal photocoagulation. Although central retinal vein occlusion at high altitude is extremely uncommon, this report underscores the potential for permanent visual loss associated with high-altitude exposure.

In summary, HAR consists of retinal changes ranging in severity from relatively asymptomatic retinal hemorrhages to potentially sight-threatening conditions such as macular hemorrhages and nerve fiber layer infarcts. Life-threatening HACE may be manifest in the eye as papilledema. All of the retinal changes occur as the result of hypobaric hypoxia associated with exposure to increased altitude. Individuals vary widely in their susceptibility to high-altitude retinopathy, and other than avoiding altitude exposure, there are no sure means to prevent it. There is no definitive treatment. Individuals who develop symptoms should be evacuated to lower altitude to avoid worsening of any visual decrement.

High Altitude and Refractive Surgery

Since the mid 1980s, refractive surgery for the correction of myopia has become increasingly popular. In the military it is an attractive alternative to eyeglasses or contact lenses in the field and eliminates the necessity for optical inserts in protective masks. Historically, the most popular procedure for the correction of myopia has been radial keratotomy (RK), which is widely available, heavily advertised, and performed extensively in active young people wishing to rid themselves of glasses. The procedure normally consists of four to eight radial incisions made in the periphery of the cornea at approximately 90% corneal depth, leaving the central 3 to 4 mm of the cornea untouched. These incisions cause the cornea to steepen peripherally and flatten centrally, thus decreasing myopia and improving distance visual acuity. The procedure requires minimal instrumentation to perform, is comparatively inexpensive, causes only mild discomfort, and usually results in excellent, predictable vision almost immediately following surgery.

Evidence suggests that some radial keratotomy patients may experience visual difficulties following exposure to changing oxygen concentration.[48,53–56] In the late 1980s and early 1990s, two case reports described a hyperopic (farsighted) shift in refraction and increased corneal flattening in radial keratotomy patients exposed to altitudes of 2,743 m to 3,048 m.[53,57] At the time, the authors of the 1988 report[57] hypothesized that hypobaria alone was responsible for the observed changes.

In 1994, research confirmed a significant increase in spherical equivalence in four eyes that had undergone RK surgery (ie, four RK eyes) exposed to altitude compared with four "normal" controls.[54] The average increase in spherical equivalent, cycloplegic refraction from sea level to 3,657 m was 1.03 ± 0.16 D and from sea level to 5,182 m was 1.94 ± 0.26 D. A flattening of the cornea that increased with altitude was also noted. The subjects who had undergone RK surgery (ie, RK subjects) did not have a subjective change in visual acuity until after an overnight stay at either altitude. This study also documented a prominent decrease in near and far visual acuity in a 49-year-old RK subject at 5,182 m. The individual was unable to read his watch, operate a combination lock, or even assemble a portable cookstove at this altitude, and his distance visual acuity was reduced from 20/20 at sea level to 20/50 bilaterally at altitude. Fortunately, this se-

vere visual impairment occurred in an indoor research facility rather than in an environment where such visual changes could have proved dangerously debilitating. The same year, Ng and colleagues[56] failed to find a hyperopic shift in nine RK subjects following 6 hours at a simulated altitude of 3,660 m in an altitude chamber. This extended time to produce visual changes suggests a metabolic origin, as opposed to a purely pressure-related phenomenon.

A more-definitive study was performed in 11 eyes of six RK subjects, 12 eyes of six subjects who had undergone photorefractive keratectomy (PRK), and 17 eyes of nine myopic controls during a 72-hour exposure to 4,270 m.[48] Cycloplegic refraction, IOP, keratometry, computerized video keratography, and corneal pachymetry were measured every 24 hours. Although no measurable change occurred on day 1, RK eyes had a significant shift by day 3, with a 3.75 D shift documented in one patient. Keratometry confirmed a 1.29 D corneal flattening by day 3 in the RK group. There was no significant change in cycloplegic refraction or keratometry in the PRK or control groups. No change in IOP was found in any group, but all three groups had a statistically significant increase in peripheral corneal thickness. A myopic shift back to baseline refraction occurred several days after return to sea level. This study firmly established that a progressive increase in hyperopia and corneal thickness occurred in RK eyes during 72 hours of high-altitude exposure. It also established that PRK corneas were stable at high altitude.

In 1996, 20 eyes in 10 subjects who had undergone RK surgery and 20 eyes of 10 myopic controls were studied at sea level to determine whether the changes seen at altitude were due to hypoxia or hypobaria.[55] Subjects were fitted with airtight goggles in which one eye was exposed to anoxic conditions (humidified nitrogen, 0% oxygen), and the other eye was exposed to humidified room air. There was a significant hyperopic shift of 1.24 D and corneal flattening of 1.19 D after only 2 hours in the RK eyes exposed to nitrogen. Radial keratotomy eyes exposed to humidified room air had no refractive change, nor did non-RK eyes exposed to either nitrogen or room air. Both RK and non-RK eyes exposed to nitrogen had a significant increase in corneal thickness. These findings strongly supported the hypothesis that hypoxia causes metabolic alterations within the RK cornea that result in refractive change. Because all the changes occurred at sea level, hypobaria could not be the cause.

The specific physiological mechanism responsible for refractive changes in RK eyes is not proven. However, research suggests that when the normal corneal architecture is weakened by radial incisions, the hypoxic cornea may expand in two dimensions (both thickness and circumference) perpendicular to the keratotomy incisions.[48,54,55,58] Perhaps even a small amount of hypoxia-induced increase in stromal hydration near the RK sites may cause a circumferential expansion and subsequent elevation of the cornea peripheral to the optical zone. This annular band of corneal expansion could lead to central corneal flattening within the optical zone and a resultant hyperopic shift. The cornea of an RK eye appears to adjust constantly to changing environmental oxygen concentration, producing a new refractive error over a period of 24 hours or more. The data suggest that both the hyperopic shift and the corneal flattening in radial keratotomy subjects (1) are progressive with increasing altitude and (2) return to normal after reexposure to sea level.[48,54–56,58]

The hypoxia-induced changes in RK eyes result in delayed visual difficulties at high altitude.[59] All reports have documented that approximately 24 hours of altitude exposure are needed to cause the hyperopic shift and associated visual changes.[48,53,54,58,59] Thus visual changes might not be observed immediately on arrival at a new altitude. A soldier or pilot could arrive at a new elevation with normal vision but awaken the following morning to a prominent change in visual acuity. The additional hypoxic insult of lid closure during sleep may add to the magnitude of the change.

The magnitude of postoperative refractive error is an important factor in predicting visual problems in RK eyes during high-altitude exposure. The starting point of the hyperopic shift is as important a determinant of visual changes as the absolute amount of shift.[58] For example, if an RK subject with 2 D of residual myopia at sea level has a 2-D hyperopic shift at altitude, then he will note an improvement in distant visual acuity. However, an RK emmetrope (no refractive error) may observe a very noticeable decrease in visual acuity associated with a 2-D hyperopic shift. Additionally, older subjects with diminished accommodative amplitude may experience more profound visual changes than younger subjects with greater amplitude.

There is no known practical method to reverse the hyperopic shift that can occur in RK eyes during altitude exposure. However, soldiers who have undergone RK may be aided by plus lenses (read-

ing glasses) at high altitudes for crisp near and far vision. One RK climber has successfully used such lenses during ascents to altitudes in excess of 6,000 m.[58] Unfortunately, it is very difficult to predict the exact lens power needed at a given altitude. Thus, if travel to extreme altitude is planned, multiple spectacles with increasing plus power should be considered.

Since the mid 1990s, two additional methods of refractive surgery (*las*er *i*n situ *k*eratomileusis [LASIK] and photorefractive keratectomy [PRK]) for correcting corneal abnormalities to improve myopia and hyperopia have largely replaced the RK technique. Both LASIK and PRK use laser light to reshape the overall cornea more uniformly than is possible with the few narrow radial incisions made in the cornea during RK. Theoretically, a cornea reshaped with these techniques should swell uniformly when it becomes hypoxic at high altitude and, thus, not change its refractive index as drastically as corneas subjected to RK. LASIK, however, incorporates a circumferential surgical flap in the cornea that might affect hypoxia-induced swelling in a manner similar to RK. Indeed, anecdotal reports and results of initial studies suggest that the vision of individuals who have undergone LASIK is affected by hypoxia, while individuals who have undergone PRK may not experience the vision problems.

A case report[60] of a woman with low myopia 2 years after LASIK stated that she had stable refraction, uncorrected visual acuity, and near-point conversion between 3,300 and 4,800 m altitude for 1 to 10 days. However, when Nelson and colleagues[61] studied 40 eyes in 20 subjects who had undergone LASIK and 40 eyes in 20 myopic control subjects who had not undergone LASIK, using air-tight goggles to expose one eye of each subject to humidified nitrogen to create ocular surface hypoxia and the other eye to humidified air (normoxia) for 2 hours, they found increased central corneal thickness in all eyes, which was associated with a myopic shift in refraction in the post-LASIK subjects. They attributed the changes to a combination of mechanical and metabolic mechanisms.

Unlike RK and LASIK, post-PRK eyes may not be greatly affected by exposure to high altitude. Mader and colleagues[62] studied six subjects (12 eyes) who had undergone PRK at sea level and during 72 hours exposure to 4,300 m and compared them with six subjects (11 eyes) who had undergone RK and nine unoperated myopic control subjects (17 eyes) undergoing the same altitude exposure. They reported significant hypoxia-induced corneal thickening in all individuals, but only the post-RK individuals demonstrated a shift in refractive error. On the basis of these results of the studies reported to date, it appears that PRK is preferable to both RK and LASIK for correcting acuity related to corneal shape abnormalities in individuals who may spend time at high altitudes.

Sleep Disturbances

"Poor sleep" is a nearly universal experience during a trip to high mountains. Many mountain travelers attribute their sleep problems to travel-related factors such as jet lag, cold temperature, an unfamiliar bed, sleeping on the ground, and other causes of "insomnia" that are common at low altitude and very common during military deployments. While such factors may contribute, the primary cause of sleep disturbances at high altitude is hypobaric hypoxia. The distinction is important, for mistaking the cause of altitude-induced sleep problems may lead to a certain complacency. In the context of civilian recreational mountaineering, poor sleep is more a nuisance than a significant disability. For the military, however, sleep deprivation from any cause during deployment may affect mission success through the performance decrements it causes. In high mountains, where hypoxia generates significant performance deficits by itself (see Chapter 22, Physical Performance at Varying Terrestrial Altitudes, and Chapter 23, Cognitive Performance, Mood, and Neurological Status at High Terrestrial Elevation), the addition of sleep deprivation acts synergistically[63] and could cause disaster for a soldier and his or her unit. Given their prevalence and potential, altitude-related sleep disturbances are not a problem that can be safely ignored during military operations.

The four common manifestations of high-altitude sleep disturbances include

1. periodic breathing with prolonged periods of apnea,
2. sleep "fragmentation" with frequent arousals and alterations of sleep stages,
3. decreased arterial oxygen content, and
4. cardiac rhythm disturbances.

Most unacclimatized individuals experience all of these manifestations to some degree following initial ascent. The symptoms are so frequent that poor sleep often has been considered a part of the AMS syndrome,[64–66] but at present, altitude-related sleep problems are considered to be a separate entity from AMS (see Chapter 24, Acute Mountain Sickness and High-Altitude Cerebral Edema). Many believe that

sleep disturbances may play a causal role in altitude illnesses, however. This opinion stems from the observations that AMS symptoms often increase in severity in the early morning after sleeping[64,67–69] and that the onset of HAPE is often after an overnight sleep.[70] The relationship is thought to be due to worsening during sleep of the already low Sao_2.[66,67,71] That concept was called into question during a study by Eichenberger and colleagues,[72] who suggested that the periodic breathing during sleep at altitude in HAPE-susceptible individuals may be caused by their profound hypoxemia rather than a cause of their HAPE.

Periodic Breathing and Apnea

Altitude-induced periodic breathing is probably the most overtly apparent of the altitude-induced sleep disturbances. The most characteristic pattern is cyclic increase and decrease in tidal volume over two to six breaths[73,74] followed by a prolonged period of apnea lasting from 6 to 20 seconds.[65,71,74] Each breathing cycle lasts from 15 to 24 seconds,[64,68] and the cycles get shorter with increasing altitude.[71,75] Another pattern consists of cyclic fluctuations of respiration without periods of apnea. In contrast to these regular cycles, a few individuals have a very disorganized breathing pattern with irregular frequency and tidal volume.[76,77] The percentage of sleep time consumed by altered breathing, especially periodic breathing with apnea, increases with increasing altitude.[71,75,78] Although periodic breathing and prolonged apnea can be alarming to a casual observer, such as someone sharing the tent, it does not seem to cause the sleeper distress unless it is associated with arousal (see below). Sleepers who are awakened during apnea often describe a sensation of suffocation, or dreams of being smothered by a collapsing tent or an avalanche.[65]

Periodic breathing during sleep occurs in nearly everyone above 3,000 m and is universal above 6,000 m.[20,71,79] Periodic breathing has been documented in some awake individuals at rest at 2,440 m,[75] so presumably it might also occur during sleep at that altitude. Individuals vary significantly in their pattern and amount of periodic breathing at altitude.[72,78–81] Some of the variation may be related to differences in respiratory drive among individuals, for several researchers have described a positive association with respiratory drive as measured by hypoxic ventilatory response (HVR)[77,79,81–83] and hypercapnic ventilatory response (HCVR).[79,84] Others have failed to find a strong association with either HVR or HCVR.[65,71] Periodic breathing may be less frequent in women,[20] but as yet no supporting data exist. Although periodic breathing can occur during all stages of sleep at altitudes greater than 4,600 m,[72,78,85] it is much more common during non–rapid eye movement (REM) sleep.[74,80,86]

Fragmented Sleep

"Sleep fragmentation" in mountain environments refers to hypoxia-induced changes in normal sleep architecture. The overall sleep/wakefulness pattern in mountain expedition settings is often a function of living in the field without electricity or other amenities, and it tends to conform to the seasonal light/dark cycle.[87] This may not be true in a military setting where operations are driven by tactical considerations. However, even if an adequate amount of time is available for sleep in mountain environments, changes in the length of sleep stages and more-frequent awakening and arousals (ie, brief periods of alpha-wave activity on electroencephalographic recordings or changes to a lighter sleep stage) cause more time awake compared with lower altitudes in most,[74,78,88] but not all,[86,89] individuals. Usually REM sleep decreases.[78] "Deep" or "slow-wave" sleep (Stages 3 and 4) may also decrease, although that change has not been consistently observed.[78,89] As a result of the decrease in deep and REM sleep, there are often increases in "transition" and "light" sleep (Stages 1 and 2, respectively)[66,74,89] and/or in wakefulness.[74,78,88] The symptoms caused by changes in sleep stages are vivid dreams or nightmares, a feeling of poor or unrefreshing sleep, or a feeling of not having slept much at all.[66,74,78,90] Daytime fatigue is common. The awakening and arousals are thought to be the most responsible for the subjective symptoms of poor sleep in the mountains.[91]

Like periodic breathing, sleep fragmentation is nearly universal in altitude sojourners,[73] especially at altitudes higher than 5,000 m.[78] Sleep fragmentation may be common at lower altitudes, but there are few data to delineate the lower limit for the effect. One study found little effect at 3,200 m,[89] while another found evidence for sleep disturbance between 1,100 m and 2,750 m.[85]

Decreased Arterial Oxygen Content

Slight hypoxemia due to decrease in ventilation during sleep at low altitude is a well-known and "normal" phenomenon.[92] It also occurs during sleep at high altitude. Sao_2 measured with a peripheral oximeter during sleep at high altitude shows a cyclic pattern that tracks periodic breathing, although the peak and trough levels characteristically lag

peak and trough ventilatory excursions.[80,83] Mean saturations are somewhere between 4% and 10% below the awake values.[66,71–73,78] That amount of desaturation from the already low values caused by the hypobaric hypoxia can result in profoundly low blood oxygen levels during sleep.

The absolute amount of Sa_{O_2} during sleep is directly related to altitude,[71,78] as is the amount of desaturation relative to awake values.[78] There is a great deal of individual variability in the amount of desaturation that occurs.[72,78,93,94] Some evidence indicates that individuals with higher HVRs have less desaturation,[77,81] although others have not found a significant relationship.[66,73]

Cardiac Arrhythmias

Cardiac rhythm during sleep in high-altitude sojourners is characterized by an overall relative tachycardia compared with that found at lower altitudes[74,95] and by an almost universal sinus arrhythmia, often with profound periods of bradycardia. The bradycardia at extreme altitude can go as low as 20 to 30 beats per minute with beat-to-beat (R–R) intervals as long as 3.6 seconds.[96,97] The bradycardia may be followed by sudden acceleration of heart rate in the range of 130 to 135 beats per minute.[97,98] Like Sa_{O_2}, the sinus arrhythmia tracks periodic breathing with increasing bradycardia during apneic periods and the sudden increase in heart rate with the resumption of respirations.[71,74,81,97] The association is so characteristic that changes in heart rate are sometimes used as surrogate markers for periodic breathing during sleep studies at high altitude, because heart rate measured by continuous electrocardiogram is easier to obtain in extreme field settings than are measures of respiration.[71]

In addition to relative tachycardia and sinus arrhythmia that occur during sleep at high altitude, other arrhythmias have been noted but seem to be fairly rare. Premature atrial and ventricular contractions have been observed,[71,86] as well as atrial bigeminy[98] and sinus arrest with junctional escape.[97] Given that these arrhythmias can also be seen in healthy individuals at low altitude, they are probably not very physiologically significant or medically portentous.

Pathophysiology of Altitude Sleep Disturbances

The pathophysiological mechanisms of hypoxia-related sleep disturbances are not precisely known, nor are the relationships between the various sleep phenomena well defined. A useful approach to understanding sleep at high altitude is to consider periodic breathing to be the primary problem, with the other phenomena (ie, arterial desaturation, sleep fragmentation, and cardiac arrhythmias) related to that abnormal breathing pattern. This approach is useful because it provides a focus for treatment option; in other words, if the concept is correct, then treating periodic breathing would also treat the other problems.

The most prominent theory of the cause of periodic breathing during sleep at high altitude is that it results from an hypoxia-induced instability in the feedback system that controls ventilation.[99,100] Hypoventilation occurs during sleep at both low and high altitude owing to (1) the loss of a suprapontine stimulus associated with the awake state and (2) an increase in upper airway resistance due to reduced phasic stimulation of upper airway musculature. At high altitude, the combination of hypobaric hypoxia and hypoxia due to the relative hypoventilation associated with sleep effectively increases the gain of the peripheral (carotid) chemoreceptors, which respond primarily to arterial oxygen pressure (Pa_{O_2}) without altering the apneic threshold of the central (medullary) chemoreceptors to carbon dioxide pressure (P_{CO_2}) or hydrogen ion concentration [H^+]. Hyperventilation driven by the peripheral chemoreceptors lowers the P_{CO_2} below the central chemoreceptor threshold, causing a centrally mediated apnea in which all respiratory efforts cease. The apnea causes an increase in P_{CO_2} at the central chemoreceptors and a decrease in Pa_{O_2} at the peripheral chemoreceptors, which stimulates resumption of breathing. Owing to the increased gain of the peripheral chemoreceptors, hyperventilation then occurs, which lowers the arterial carbon dioxide pressure (Pa_{CO_2}) and the P_{CO_2} below the apneic threshold again, and the cyclic pattern of alternating hyperventilation and apnea is perpetuated.[99,100] Additional factors that have been postulated to contribute include (1) decreased body stores of oxygen and carbon dioxide, which may change the relative response times of the chemoreceptors, and (2) the loss of a central, chemoreceptor-independent ventilatory drive ("afterdischarge"), which normally functions to dampen ventilatory depression after hyperventilation.[101]

Many people view reduced oxygen content of the arterial blood as the most significant consequence of periodic breathing during sleep at high altitude, because low oxygen levels drive many pathophysiological processes. Sa_{O_2} during sleep at altitude is a function of the following:

1. the underlying level of hypobaric hypoxia associated with the decreased partial pressure of oxygen at a specific altitude (see Chapter 22, Physical Performance at Varying Terrestrial Altitudes), combined with
2. the normal 4% to 6% desaturation associated with sleep-induced hypoventilation,[92] and
3. any desaturation due to the apneic phase of altitude-associated increases in periodic breathing.

The contribution of underlying hypobaric hypoxia to sleep desaturation increases with increasing altitude. Desaturation owing to sleep-associated hypoventilation remains relatively fixed but assumes more and more physiological significance as the underlying hypobaric hypoxia positions the individual further down on the steep portion of the oxyhemoglobin dissociation curve (see Chapter 21, Human Adaptation to High Terrestrial Altitude). The contribution of periodic breathing to the overall level of hypoxia is somewhat controversial, however. It makes sense that recurrent apnea would cause arterial desaturation, and it does. As previously noted, Sa_{O_2} measured during sleep with peripheral oximeters shows a cyclic pattern of decreasing and increasing saturation that tracks cyclic breathing. West and colleagues[71] found that apneas at very high altitudes were associated with the lowest Sa_{O_2} measured during the 24-hour day. The question is whether the *mean* saturation during periodic breathing is low. Some investigators found that it was,[76,93,94] while others found little relationship between Sa_{O_2} and amount of periodic breathing.[72,73] Masuyama and colleagues[77] found a negative correlation between the amount of periodic breathing and the degree of Sa_{O_2} during sleep. On the basis of their findings, they suggested that individuals with high ventilatory drives maintain higher saturation during sleep by means of periodic breathing. The findings of Lahiri and colleagues[102] that acclimatized climbers from low altitudes had higher HVRs, more periodic breathing, and higher Sa_{O_2} during sleep than high-altitude, native-born Sherpas, who had a blunted hyperventilatory response, no periodic breathing, and lower saturation, also suggests that periodic breathing might have an adaptive value for maintaining saturation during sleep in altitude sojourners.

The sleep fragmentation that occurs at high altitude is generally believed to be related to periodic breathing.[65,68,74,78] The mechanism is thought to be the triggering of arousal and awakenings by periods of apnea. The arousals or awakening functions to stimulate respiration and terminate the apnea. Arousal may sometimes occur in the absence of apnea,[78] which suggests that the body may respond more to the level of desaturation than to the absence of feedback from respiratory movements. There is little information in the literature to suggest that altitude-induced hypoxemia has any effect on central mechanisms that control sleep architecture.

As noted previously, the sinus arrhythmias seen during sleep at high altitude are so consistently related to periodic breathing that the recurrent episodes of bradycardia have been used as surrogate markers for periods of sleep apnea.[71] The cyclic variation in heart rate has been suggested to be mediated through alterations in the predominance of the sympathetic versus the parasympathetic influence of the autonomic nervous system.[95,97] The other bradyarrhythmias that have been observed undoubtedly result from parasympathetic activity on the sinoatrial node through the vagus nerves.[97]

Diagnosis and Treatment of Sleep Disturbances at Altitude

The diagnosis of altitude-related sleep disorder is made by noting the signs and symptoms of disordered sleep in an individual who has recently ascended to moderate or high altitude, or at any time in an individual at very high and extreme altitudes. It is useful to try to differentiate altitude-related sleep disturbance from other causes to ensure appropriate treatment. As noted below, inappropriate treatment of altitude-induced sleep problems with hypnotic drugs (ie, sleeping pills) can be dangerous, owing to depression of ventilation. Other possible causes of poor sleep in the setting of a military deployment to mountain terrain include environmental factors other than hypoxia (eg, cold, unfamiliar bedding or sleeping surface, crowded sleeping quarters, etc) and disruption of circadian rhythms by work schedules or moving across time zones (ie, jet lag).

Preexisting pathological sleep disorders should be rare in military populations, which generally consist of relatively young, healthy individuals who have often been medically screened as a condition of entering military service. However, acquired sleep disorders are possible in the command and senior noncommissioned officer populations, which are traditionally older. Preexisting sleep-disordered breathing occurs in 4% to 9% of middle-aged persons[103] and may be a cause of chronic hypertension.[104] Most sleep problems that are not directly related to altitude can be identified on the basis of

clinical history. Medical officers should remember, however, that the signs and symptoms of many preexisting conditions of sleep-disordered breathing, including Cheyne-Stokes respiration in chronic heart failure and obstructive sleep apnea syndrome, are similar to those seen in altitude periodic breathing.

Raising Arterial Oxygen Content During Sleep

Because hypobaric hypoxia is the ultimate cause of altitude-related sleep disturbances, treatment is based on increasing the oxygen content of the blood. This can be accomplished either by raising the inspired oxygen or by increasing ventilation to raise lung alveolar oxygen content. Conversely, anything that decreases respiration will worsen altitude sleep disturbances and is potentially harmful at altitude.[66] Transient or short-term insomnia related to deployment and environmental factors other than hypobaric hypoxia can be treated by changing the sleep environment, sleep schedule, or both. Use of sleeping pills that are routinely used at low altitude is possible at high altitude so long as those treatments do not depress respiration.

Descent. The most reliable method to raise inspired oxygen during sleep is to descend in elevation, which effectively increases the oxygen content of the ambient air. Mountaineers codified this principle many years ago in the axiom "climb high, sleep low." Ideally, one should descend to a level at which the partial pressure of oxygen is sufficient to eliminate periodic breathing during sleep. Given individual variability, this altitude will differ for different individuals, and some individuals exhibit periodic breathing at sea level.[103] However, descent below 2,500 m will significantly reduce altitude-induced sleep problems for the vast majority of people.

Supplemental Oxygen. Sleeping low, or at least at low-enough elevations to decrease sleep problems related to hypoxia, is not always a viable option in mountain environments where severe weather conditions and rugged terrain can significantly limit mobility. Tactical situations may also limit descent during military deployment. When descent is not possible, the inspired oxygen levels can be raised using supplemental oxygen administered by mask or nasal cannula. Supplemental oxygen has been demonstrated to eliminate apnea and eliminate or attenuate periodic breathing depending on the altitude and the amount of oxygen supplied.[71,74,78–81] The mountain climbing community has known about the benefits of low-flow, supplemental oxygen during sleep and has used it for years at high camps and bivouacs.[105]

The biggest problem with the use of supplemental oxygen is obtaining a sufficient supply. Metal oxygen tanks are heavy, cumbersome, and cold in an environment where those characteristics are potential liabilities. The high camps on Mount Everest (and other mountains that are popular to climb) are littered with used and abandoned oxygen tanks, because the difficulty of transporting them down outweighs any perceived utility. Additionally, it would require vast amounts of oxygen and equipment to supply the personnel of any unit bigger than a squad or platoon for any period of time. Bottled oxygen is also an explosive hazard if it is struck by bullets or fragments. In a static situation or a relatively fixed site with adequate power supplies, molecular-sieve oxygen concentrators may be more cost effective than transporting bottled oxygen. The use of portable, cloth, hyperbaric chambers or fixed-facility hyperbaric chambers to sleep in will also allow an increased inspired oxygen level owing to the increase in barometric pressure. Both of these solutions could present a supply problem for larger units, however.

Pharmacological Aids to Sleep. Increasing ventilation during sleep at altitude is accomplished most practically by using pharmacological respiratory stimulants. Drugs that have been evaluated in that role include carbonic anhydrase inhibitors, almitrine, and progestogens. Of these, acetazolamide, a carbonic anhydrase inhibitor, is the best studied and probably the most useful.

Acetazolamide is not generally thought of as a respiratory stimulant, but it is effective in this role during sleep at high altitude at a dose that is also effective for prophylaxis of AMS (ie, 250 mg/8 h).[65,68,83,85,94] It increases the oxygen in arterial blood and decreases periodic breathing, arousals, and awakenings without changing the HVR (Figure 26-3).[65,83] Another carbonic anhydrase inhibitor, benzolamide, has been shown to have similar actions.[106] The drugs probably stimulate respiration by inducing a metabolic acidosis, which counteracts the respiratory depression effect of the altitude-induced respiratory alkalosis (see Chapter 21, Human Adaptation to High Terrestrial Altitude). They may also have a stimulatory effect on the central respiratory chemoreceptor.[106] Regardless of the mechanism of action, acetazolamide is relatively effective and safe for preventing sleep problems at high altitude. Its additional advantage—that of being safe and effective for preventing AMS—make it the drug of choice for both indications.

Other respiratory stimulants that have been evaluated during sleep at high altitude include

almitrine and methyl progesterone.[65,76,83] Almitrine increases periodic breathing by raising the HVR and increases Sa_{O_2} about as effectively as acetazolamide.[83] To the extent that periodic breathing contributes to frequent arousals or awakening, almitrine is not as effective as acetazolamide in treating altitude sleep problems. Methyl progesterone, on the other hand, decreases periodic breathing but only moderately increases Sa_{O_2} in altitude sojourners. It increases Sa_{O_2} during sleep more effectively in altitude residents with chronic mountain polycythemia (Monge's disease) and prevents transient episodes of severe desaturation that characterize this group of patients.[65,76] Given its relative ineffectiveness and potential side effects, progesterone is probably not a good option for treating altitude-related sleep disturbances in young, healthy, military personnel.

Use of hypnotic medications such as benzodiazepines to treat sleep problems at high altitude is relatively contraindicated due their ability to depress respiration and worsen sleep hypoxemia. Nonetheless, they have been suggested as appropriate therapy for sleep problems that are not directly related to hypobaric hypoxia.[107] In reality, individuals who are not medically trained tend not to differentiate among various causes of insomnia and treat all sleep problems, including those related to hypoxia, with "sleeping pills." It is well known that mountain climbers often use hypnotic medications to help them sleep at high altitude,[82] sometimes with disastrous results.[108]

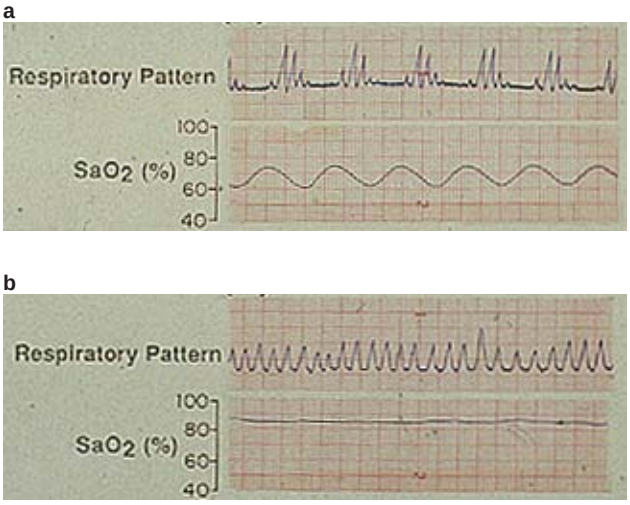

Fig. 26-3. Periodic breathing during sleep in a volunteer test subject at 4,400 m on Mount McKinley in Alaska. Each panel contains a tracing of respiratory excursion and arterial oxygen saturation (Sa_{O_2}, %) measured by pulse oximeter. (**a**) During treatment with placebo the subject had periodic breathing and low arterial oxygen saturation. (**b**) Acetazolamide treatment decreased periodic breathing and improved oxygen saturation. Adapted with permission from Hackett PH, Roach RD, Harrison GL, Schoene RB, Mills WJ Jr. Respiratory stimulants and sleep periodic breathing at high altitude: Almitrine versus acetazolamide. *Am Rev Respir Dis*. 1987;135:897.

Few studies of hypnotic medication use at high altitude have been reported, and all the studies were of benzodiazepines. Diazepam in a 10-mg dose was found to decrease Stage 3 sleep and increase hypoxemia[66]; the authors suggested that this drug might be dangerous to use at high altitude. Triazolam 0.125 mg was used to promote sleep at 7,600 m in a hypobaric chamber without apparent harmful effects, but it was not formally studied.[109] One milligram of loprazolam did not alter sleep architecture or increase periodic breathing at 4,800 m.[82] Likewise, 10 mg of temazepam reduced sleep-onset latencies and increased sleep efficiency without increasing periodic breathing above 4,000 m but was more effective when taken in addition to 500 mg acetazolamide per day.[85] This approach of combining a hypnotic with acetazolamide, which stimulates respiration during sleep, seems to offer a method for avoiding respiratory depression. Diphenhydramine 50 to 75 mg has been recommended as a means to treat insomnia at high altitude without resorting to benzodiazepines,[106] but no studies have been reported.

For most people, the prognosis for untreated hypoxia-induced sleep problems is good at altitudes under 5,000 m because they will improve with acclimatization.[74,94,110] Additionally, the sleep problems can be successfully treated with acetazolamide or supplemental oxygen as described above. At altitudes higher than 5,000 m, periodic breathing and its associated sleep disturbances appear to persist indefinitely; although treatment may improve sleep somewhat at very high and extreme altitudes, sleep is never normal.[71]

The attitude of many civilians who venture into the mountains for recreation is that altitude-induced sleep problems are "a nuisance rather than a disability."[93(p172)] As previously noted, sleep problems cannot be safely ignored in a military deployment because of their detrimental effects on performance. Strong consideration should be given to treating all military personnel deployed to mountain environments above 3,000 m to prevent altitude-related sleep problems during their initial ascent. At altitudes under 5,000 m, treatment can be discontinued for most individuals as they successfully acclimatize. At higher altitudes, treatment

should be continued indefinitely. At present, the safest and most practical treatment for use in military operations in the mountains is probably acetazolamide in the same dosages that are effective for preventing AMS (ie, 125–250 mg twice daily, or 500 mg slow-release formulation once a day in the evening.)

Blood Clotting Disorders

Exposure to high mountain environments is associated with intravascular blood clot (thromboembolic) problems and minor bleeding phenomena that suggest a possible altitude-induced disruption of normal hemostatic mechanisms. Thromboembolic events include pulmonary embolism and stroke in healthy, young individuals, who do not ordinarily develop such catastrophic problems. Given that most military units are composed largely of relatively young individuals, effective medical support for deployment to high altitude should include some provision to manage thromboembolism.

Case reports and anecdotal descriptions of thromboembolic phenomena are surprisingly frequent in the mountaineering literature. Superficial thrombophlebitis[111] and deep venous thrombosis (DVT) in the lower extremity,[1] DVT with subsequent pulmonary embolus (PE),[112–116] transient ischemic attack (TIA),[117] and thromboembolic stroke[113,115,118,119] have all been reported as occurring in the high mountains. Additionally, blood clots have been observed in the small vessels of the lungs and other organs of persons who died of altitude illness.[113,120,121] Those autopsy findings were the basis of a hypothesis that HACE results from an altitude-induced coagulopathy,[122] a theory that is not widely held at present (see Chapter 24, Acute Mountain Sickness and High-Altitude Cerebral Edema).

Although reports of thromboembolic phenomena in high mountains are surprisingly common, the actual incidence of such events is not known. The number of reports could merely reflect an exaggerated interest in these phenomena; however, the consensus is that thromboembolic phenomena at high altitude are not unusual. The events tend to occur at very high altitudes (> 4,500 m), however. Proposed risk factors include either preexisting or altitude-induced coagulopathy, polycythemia, dehydration, cold, climbing without supplemental oxygen, and long periods of forced inactivity in small tents owing to bad weather.[20]

The clinical signs and symptoms of altitude-related thromboembolic disease are the same as those that occur at low altitude:

- peripheral venous thrombi can cause pain, swelling, and warmth in the affected limb;
- pulmonary emboli can cause chest pain, dyspnea, hypoxemia, cough, and hemoptysis; and
- cerebral thrombi and emboli can cause transient or permanent, focal or global neurological abnormalities; coma; and death.

Diagnosis of thromboembolic disease in a high mountain environment usually relies solely on signs and symptoms because the sophisticated diagnostic equipment that is available in fixed medical facilities is lacking. If the patient can be evacuated to appropriate facilities, an attempt to confirm the diagnosis can be made using standard methods (eg, doppler ultrasound, roentgenography, angiography, various scans). The differential diagnosis must include other altitude-related conditions that can have similar manifestations. HACE, HAPE, and cellulitis resulting from altitude-related suppression of immune function (see below) can cause signs and symptoms similar to those of thromboembolism and should be considered in the differential diagnosis.

The most common type of abnormal bleeding at high altitude is retinal hemorrhage, which was described earlier in this chapter. Other bleeding phenomena that have been reported include "splinter" hemorrhages under the fingernails thought to be caused by hand trauma associated with technical climbing,[123,124] rare instances of unexplained gross gastrointestinal and urinary tract hemorrhage that resolved with descent,[125] and subarachnoid hemorrhage.[113,126] Diagnosis of hemorrhage based on direct observation of retinal or significant gastrointestinal or urinary bleeding is relatively uncomplicated. As with cerebral thromboembolism, the signs and symptoms of subarachnoid hemorrhage may be similar to those of HACE, and the definitive diagnosis will require evacuation and more extensive study.[126]

The pathophysiology of thrombosis and abnormal bleeding at high altitude has not been well established. Because bleeding problems at altitude seem relative minor, most attention has been directed toward explaining the mechanisms responsible for the abnormal clotting. The concept of Virchow's triad suggests that intravascular blood clot formation is due to the interaction of at least two of three factors: stasis of blood flow, injury to the vessel epithelium, and changes in blood coagulability.[127] Stasis of blood due to constrictive clothing, cold temperatures, and forced inactivity in cramped quarters during bad weather are well-recognized risk factors in mountain environments.

Likewise, the possibility of vessel injury due to trauma, including pressure trauma from pack straps, climbing ropes, and harnesses, or due to cold injury are also recognized risk factors. What has been the most intriguing to researchers, however, is the possibility of a hypoxia-induced hypercoagulable state. Results of many early investigations, including the autopsy findings of extensive clotting in individuals who died from altitude illness, seemed to support that possibility.[113] In the aftermath of the Sino–Indian border conflict of the early 1960s, researchers from India did a series of studies of the effects of high altitude on coagulation and platelet function, which suggested that both coagulability and fibrinolysis increased with altitude exposure in normal individuals, but that coagulation was activated without a compensatory increase in fibrinolysis in patients with HAPE.[128,129] Platelet adhesiveness was found to be increased in individuals at high altitude with symptoms of altitude illness and stroke.[130,131] Studies since that time have not provided consistent evidence for a hypercoagulable state or abnormal platelet function induced by altitude (see Cucinell and Pitts[132] for a summary of the work up to 1987).

Bärtsch and colleagues[133–135] did an extensive series of studies of coagulation in individuals with and without AMS and HAPE. They found no significant alteration of coagulation or platelet activity in individuals without AMS, and partial activation of coagulation without fibrin formation in individuals with AMS and individuals who later developed HAPE. Once HAPE was established, there was activation of coagulation with production of fibrin and decreased fibrinolysis. They concluded from these results that the hypercoagulation associated with HAPE is an epiphenomenon *resulting* from, not *causing*, HAPE.[135]

A hypercoagulable state does not have to be induced by hypoxia to cause problems at high altitude. Congenital hypercoagulable states caused by deficiencies of proteins C and S or antithrombin III, or by activated protein C resistance due to the factor V Leiden mutation or the prothrombin G20210A mutation may not result in thrombosis in young adults until middle age, or until they are exposed to the many risk factors for intravascular clot formation found in the mountain environment. The Leiden mutation is prevalent in Caucasian populations[136] and has been hypothesized to contribute to frequent DVT formation in aviators.[137] This and similar undetected hereditary defects in coagulation are likely to play a role in thromboembolic events at high altitude.

Pulmonary embolus arising from a DVT is a well-recognized clinical entity and probably explains most of these cases in high mountains. The origin of cerebral venous thrombosis *at high altitude* has been suggested to occur from the combination of hemoconcentration causing a functional hypercoagulable state combined with stasis of blood in the brain owing to HACE.[118] A preexisting hypercoagulable state could also play a role. Thromboembolic stroke has been suggested to result from emboli traveling through the foramen ovale, which is opened by increased right atrial pressure from hypoxia-induced pulmonary hypertension.[20] The causes of subarachnoid hemorrhage and hemorrhagic stroke at high altitude are not known. An effect of hypoxia-related increase in cerebral blood flow and cerebral blood pressure on preexisting aneurysms or arteriovenous malformations may play a role.[126]

Treatment of thromboembolic disease involves

1. preventing the formation of further blood clots or emboli through anticoagulation and elimination of risk factors, and
2. restoring blood circulation to affected areas using pharmacological thrombolysis, surgical extirpation, or waiting for collateral circulation to develop.

The technical equipment and facilities for adequate treatment are not usually available in mountain field settings, where thromboembolic events may occur. Consequently, treatment virtually always requires descent and evacuation to higher-echelon medical support with the capabilities to provide a more-definitive diagnosis and to monitor anticoagulation and thrombolytic therapy. Unfortunately, evacuation from high mountains can often be difficult. Houston and Bates[112] give a gripping account of the attempted evacuation of an individual with DVT and PE from near the summit of K2, an attempt that led to one of the most famous accidents in mountaineering history.

Supportive treatment in the field prior to and during evacuation includes bed rest and ensuring adequate hydration and oxygenation by whatever means are available. The patient should be kept warm, but care should be taken to ensure that clothing, blankets, or equipment do not constrict blood flow. Anticoagulation with heparin or warfarin at high altitude while awaiting evacuation is generally considered to be dangerous without the ability for monitoring coagulation parameters to guide dose adjustments.[20,107] Subcutaneous low molecu-

lar weight heparin, which can be used with relative safety in standard doses without the necessity of monitoring coagulation parameters, provides a means of beginning anticoagulation while awaiting evacuation.[20] Anticoagulation should not be started in cases of possible cerebral thromboembolism without a definitive diagnosis to identify possible subarachnoid hemorrhage or hemorrhagic stroke.

Prophylactic measures to prevent thromboembolic disease consists of eliminating risk factors for abnormal blood clot formation. This includes ensuring adequate hydration, protection from cold temperatures, and avoiding situations that promote restriction of blood flow. Prophylactic use of anticoagulation or antiplatelet therapy runs the risk of exacerbating HARHs or other altitude-related bleeding and is not generally recommended.[107]

The prognosis for thromboembolic events occurring at high altitude is the same as at sea level, assuming that the patient can be evacuated for treatment in a timely fashion. Mortality and morbidity are very high if the patient remains at high altitude without treatment.[20] During military deployment to mountain environments, thromboembolic events other than superficial thrombophlebitis create functional casualties, for the afflicted unit members cannot be returned to duty in the time frame of most military operations. The limitations on return to duty include the necessity for prolonged anticoagulant therapy, neurological deficits from stroke, and decreased lung function from pulmonary emboli. The incidence of recurrent thromboembolic disease during subsequent altitude exposure in individuals who have had a previous event in the mountains is not known. Presumably, individuals with a preexisting coagulopathy would be at increased risk. There are anecdotal reports of mountain climbers returning to high altitude successfully after recovering from an altitude-related thromboembolic event. However, military members with a previous history of thromboembolic phenomenon at high altitude should probably be restricted from deployment to the mountains to limit potential impact of a recurrent event on unit strength.

Suppression of the Immune System

Infectious diseases can be a serious medical problem during any military deployment. Consequently, anything that compromises immune function of personnel in a military unit must be viewed as a medical threat. Hypobaric hypoxia at high altitude has been shown[138] to degrade immune function in experimental conditions, although the clinical significance of that finding has not been determined. Nonetheless, military medical personnel should be alert for possible adverse consequences related to effects of altitude on immune function.

Speculation about a possible effect of high altitude on the immune system was originally generated from reports of apparent differences in the occurrence of specific infectious diseases between Indian soldiers stationed at high and low altitudes,[139] and from accounts of persistent skin infections and cellulitis associated with minor trauma in climbers on Mount Everest.[1,140] Although these specific phenomena could have other causes than immune suppression, subsequent research has documented some degree of hypoxia-related immune system dysfunction.

Exposure to high altitude for prolonged periods can depress cell-mediated immunity but seems to have little effect on humoral immune function. Effects on cell-mediated immunity that have been observed include (1) a decreased ability for in vitro monocyte activation despite an increase in monocyte numbers, and (2) a decrease in measures of natural killer cell cytotoxicity.[140,141] The intraindividual variability in cell-mediated response effects was large.[141] These effects are postulated to be the result of altitude-induced increases in adrenal corticosteroid hormone levels.[141] Measures of humoral immune function, including serum immunoglobulin levels[141,142] and B cell response to vaccination with T cell–independent antigen, remain intact at high altitude.[143] Additionally, there is no depression of mucosal immunity, as measured by lysozyme and immunoglobulin A content in nasopharyngeal washings.[141]

Clinical importance of altitude-induced immune suppression appears limited to an increase in skin infections and cellulitis that may not respond well to treatment while the patient remains at high altitude. Poor personal hygiene in the always-difficult mountain environment may contribute to the incidence of these infections.[140] Nagging minor infections can affect morale and performance and should not be dismissed as insignificant by medical personnel. Additionally, infectious complications of all wounds and burns should be anticipated,[107] although there is little information in literature on the subject. Careful attention to wound care and appropriate antibiotics should be attempted to prevent or resolve infections. When possible, descent to a lower altitude or continuous supplemental oxygen therapy will improve immune function and facilitate resolution of the infections.

Wound Healing

Casual observations during climbing expeditions suggest that superficial wounds heal slowly at altitudes higher than 5,000 m.[1,105] No field studies have been reported to confirm this; however, experimental evidence suggests that very low oxygen levels may slow wound healing.[144] Whether levels of oxygen in tissues reach low-enough levels to slow healing as a result of hypobaric hypoxia alone is hard to say, but it is a theoretical possibility at very high altitudes. Certainly the combination of low tissue saturation and cold temperatures at high altitude might decrease the circulation in the extremities enough to be a factor. Whether wound healing would be a problem with surgical and deep soft-tissue wounds is not known but is also a theoretical possibility. The prudent course is to evacuate wounded patients to low altitude or provide them with supplemental oxygen therapy.

High-Altitude Pharyngitis and Bronchitis

Pharyngitis and bronchitis are very common at altitudes above 4,500 m.[1,107,140] The cause is thought not to be infection but rather irritation of the mucosal lining of the respiratory passages by an increased volume of cold, dry air moving across them from the hypoxia-induced increased ventilation.[107] The condition is characterized by sore throat and a dry, nonproductive, hacking cough that can occur in paroxysms. At extreme altitudes, the coughing spells can be severe enough to fracture ribs.[1,30] Throat lozenges and cough suppressants are the mainstays of treatment. The condition resolves without permanent effects after descent, but it may take days to several weeks to completely resolve. Preventive measures include adequate hydration and breathing through a balaclava or face mask to retain moisture. Chronic cough could cause significant problems in a tactical situation by degrading performance and giving the enemy auditory clues to the location of the individual or the unit.

Exacerbation of Preexisting Medical Conditions

Although most military personnel who deploy to high mountain terrain will be relatively healthy, some could have preexisting medical conditions that would be made worse by the environment. Because physical standards and medical screening prevent individuals with significant medical problems from entering or remaining in active-duty military service, the types of medical problems that could be present in a military unit at deployment do not represent the full spectrum seen in the civilian sector. At worst, most military units could be expected to have some acute illnesses and injuries and few chronic health problems. Officers and noncommissioned officers at higher ranks are selected for their experience and training, however, and are older and more likely to have chronic, age-related medical problems. Often they have received waivers for their medical problems to enable them to be retained in active military service for their skills and leadership.

Few studies have been done of preexisting medical problems at high altitude, but the number of anecdotal reports is growing owing to increasing numbers of civilians with medical problems traveling to mountains for recreational purposes. Even in the absence of good data, the progressive decrease in oxygen content of ambient air at altitude provides a basis for prediction of potential problems. The significance of a preexisting medical problem depends largely on its interaction with hypobaric hypoxia. Hypothetical outcomes to that interaction include (1) the medical condition could increase the degree of hypoxia, (2) hypoxia could worsen the medical condition, and (3) no significant effect of either. (In reality, most preexisting medical conditions both increase the degree of hypoxia at altitude and are worsened by it, but the conceptual framework is probably useful.)

Any condition that interferes with oxygen transport will increase the degree of hypoxia experienced at any altitude. Thus chronic heart and lung diseases could be problems at high altitude. Most active-duty military personnel do not have significant heart and lung disease, however. Conditions involving the heart or lungs that might be found in military populations include mild chronic obstructive pulmonary disease (COPD), elevated carbon monoxide levels, or both, due to smoking tobacco; mild asthma; and mild or moderate sleep apnea.

Individuals with mild COPD or mildly elevated carbon monoxide do have lower Sa_{O_2} at altitude,[145,146] but this might not cause significant clinical problems or greatly impact the individuals' performances except at higher altitudes. If individuals with COPD develop significant problems or performance decrements, they should be redeployed to lower elevations, for it is unlikely that they can be adequately managed at high altitude. Elevated carbon monoxide levels will resolve with cessation of tobacco smoking, and these individuals may be able to remain at high altitude if they discontinue smoking.

Asthmatics are at increased risk for bronchoconstriction at high altitude due to inhalation of cold air and airway drying from hypoxia-stimulated ventilation. However, they may also benefit from a decrease in allergen load and lower density of the air. Asthma that is mild and well-enough controlled to allow staying in the military is probably not a major contraindication for deployment to high altitude.

Sleep apnea increases nocturnal hypoxemia and also the risk of pulmonary and systemic hypertension, cardiac arrhythmia, and daytime performance decrements at both high and low altitudes.[104] The effect will be greater at high altitude owing to altitude-induced periodic breathing (see above). *Individuals known to have sleep apnea should not be deployed to high elevations.* Those with occult sleep apnea may do poorly at high altitude, but sleep apnea will be difficult to diagnose in the presence of the typical altitude-induced periodic breathing.

Sedatives and any other medications that depress ventilation will increase hypoxia at high altitude and should be avoided. All medications being taken by deploying unit members should be screened prior to deployment to identify those that could worsen hypoxia. This could be a significant problem if unit members are allowed to bring their own supplies of medications during deployment, especially sleeping pills. Medications that depress ventilation should be discontinued prior to deployment or given only under medical supervision.

Hypobaric hypoxia can worsen many chronic medical conditions through direct or indirect effects. The exacerbation of chronic congestive heart failure at mountain resorts due to hypoxia-stimulated increase in catecholamine levels is the classic example of this phenomenon.[20] Congestive heart failure is not prevalent in active-duty military populations, however. Conditions that can be found in military populations and could be made worse by hypoxia include essential hypertension, sickle cell trait, occult coronary artery disease, pulmonary hypertension, migraine headache, and pregnancy.

Blood pressure increases in almost everyone at high altitude prior to acclimatization, owing to autonomic nervous system activation.[147] In normal individuals, the level of increase is not enough to warrant treatment, but patients being treated for hypertension at low altitude will have to continue or increase their medication at altitude to control their blood pressure.

Individuals with sickle cell trait can have splenic syndrome or vaso-occlusive crisis at high altitude.[20,148] Given that many military members may have sickle cell trait, medical personnel should be prepared to treat that condition. Occasionally, splenic syndrome or a vaso-occlusive crisis during altitude deployment may be the first manifestation of an otherwise occult sickle cell trait condition.[20]

Other occult conditions in military populations that could be worsened by hypobaric hypoxia include coronary artery disease, pulmonary hypertension, and seizure disorders. The evidence that exists on established coronary artery disease at high altitude suggests that there is little increased risk for acute events, possibly because of a protective effect of altitude-related limitations of physical work.[149] Whether this situation would hold during combat at high altitude is questionable.

Pulmonary artery pressure increases with altitude exposure and may contribute to HAPE (see Chapter 25, High-Altitude Pulmonary Edema). Individuals with known pulmonary hypertension should be restricted from altitude exposure, but they normally would not be serving on active duty. Likewise, individuals with known seizure disorders would not be serving on active duty. New onset of seizures at high altitude requires that the individual be evacuated to low altitude for evaluation.

Migraine headaches have been reported at high altitude,[150] but the exact incidence and significance are not well established. Worsening of migraine frequency or severity in the mountains is an obvious indication for redeployment to low altitude and future deployment restrictions.

Women living at high altitude have an increased incidence of hypertension and preeclampsia syndrome during pregnancy, and low-birth-weight babies.[151] Pregnant women visiting high altitude may have increased bleeding complications and preterm labor.[152] Although there is little evidence for other significant problems associated with pregnancy at high altitude, pregnant women in the military probably should not be deployed to altitudes greater than 2,500 m.

MEDICAL PROBLEMS CAUSED BY OTHER ENVIRONMENTAL FACTORS

Although hypobaric hypoxia is unique to the high mountains, it is not the only factor in that environment that can cause medical problems. A significant number of other factors play a role, often in conjunction with hypoxia. Some, like cold injury, may be obvious, because most people associate high mountains with snow and cold. Others, like hemorrhoids, may not be expected, but medical units

that deploy to high altitude without provisions to treat hemorrhoids will not be able to provide adequate care for a common problem. In many instances, these hypoxia-*un*related problems may be far more clinically significant than those caused by hypoxia itself (eg, a lightning strike will cause more disability than retinal hemorrhages.) Often they will be more frequent than hypoxia-related problems (eg, cold injuries usually outnumber cases of HACE). Although not unique to the high-altitude environment per se, these medical problems are significant contributors to the constellation of environmentally related medical problems that affects military units during deployment to the mountains, and the medical support system must be prepared for them.

Trauma

A substantial amount of trauma occurs in high mountain environments. It is likely that the incidence of nonballistic injury due to accidents in units deploying to mountain terrain could be relatively higher than that in other environments, but few data exist to substantiate that supposition. Certainly, the array of traumatic injuries associated with civilian recreation activities in the mountains suggests that a significant threat exists. (Interested readers can also consult the Pictorial Atlas of Freezing Cold Injury by William J. Mills, Jr, MD, which is appended to *Medical Aspects of Harsh Environments, Volume 1*.[153] In addition to freezing cold injuries per se, the Atlas contains numerous descriptions and photographs of trauma in freezing cold and high terrestrial environments.)

The combination of rugged topography with hypoxia-impaired judgment and reasoning abilities is a major contributing factor to traumatic injury in high mountain regions. Other environmental factors may also play a role. Hypothermia can contribute to decrements in judgment and mental processes, and cold fingers and toes may not have the sensitivity needed for safe technical climbing maneuvers. Snow, rain, and ice make footing and handholds more tenuous than when the weather is dry.[154] Bad weather can also make vehicular travel, which is often precarious in the mountains, treacherous. Mountains can inflict trauma directly through rockslides and avalanches. In military operations, the trauma due to environmental factors is compounded by the well-known, early-entry stresses of traveling and a novelty location's causing more accidents early in any deployment.

The traumatic injuries seen in mountain environments form a pattern that results from the interactions among environment, activity, and equipment (Figure 26-4). The combination of rugged terrain and the effects of hypobaric hypoxia on performance cause falls to be a major contributor to the tally of injuries. The injuries caused by falls are characteristic because of their types. Owing to steep terrain, falls tend to be long and uncontrolled, causing multiple blunt trauma. Abrasions are generally not seen because of the protective effect of heavy clothing worn to protect against cold. In the military setting, the wearing of field uniforms also helps protect against abrasion. Falls in mountain terrain are often complicated by specialized equipment used for technical climbing or skiing. Equipment with sharp points and surfaces, such as ski poles and ice crampons, can cause lacerations and penetrating injuries during a long fall.[155] Climbing harnesses and ropes meant to prevent serious falls can cause specific types of injury if misused or if they fail. Free-falling equipment can also inflict injury on personnel at lower levels who are not directly involved in the fall. Blunt trauma and lacerations caused by runaway skis with sharp edges are well-known injuries in recreational ski areas, for example. Weapons carried by military personnel in tactical operations could add an additional hazard during falls.

Different mountaineering activities are associated with different types and locations of injuries. Downhill skiing generates many lower-extremity injuries, but it also causes serious injuries to the head and upper body due to high-speed impacts. Owing to slower speeds, falls during cross-country skiing are associated with fewer serious head injuries. Cross-country skiers tend to have both upper- and lower-extremity injuries, however, owing to falling with backpacks.

Serious trauma is less-often associated with hiking and backpacking. Whereas the higher speeds associated with skiing tend to break bones and tear muscle tissue, hiking and backpacking are associated with strains and sprains of joints and other soft-tissue injuries. Upper-extremity neuropathy due to brachial plexus injury from pack-strap trauma (a form of Erb palsy) can occur due to shifting of rucksack position during climbs and descents.[156] Overuse injuries are common in the mountains owing to traveling up and down steep slopes. Technical climbing, which relies heavily on the arms and hands, is associated with a characteristic set of upper-extremity soft-tissue injuries, including serious hand trauma.[157]

Avalanches and rockslides cause multiple trauma. They often bury individuals, causing crush injury

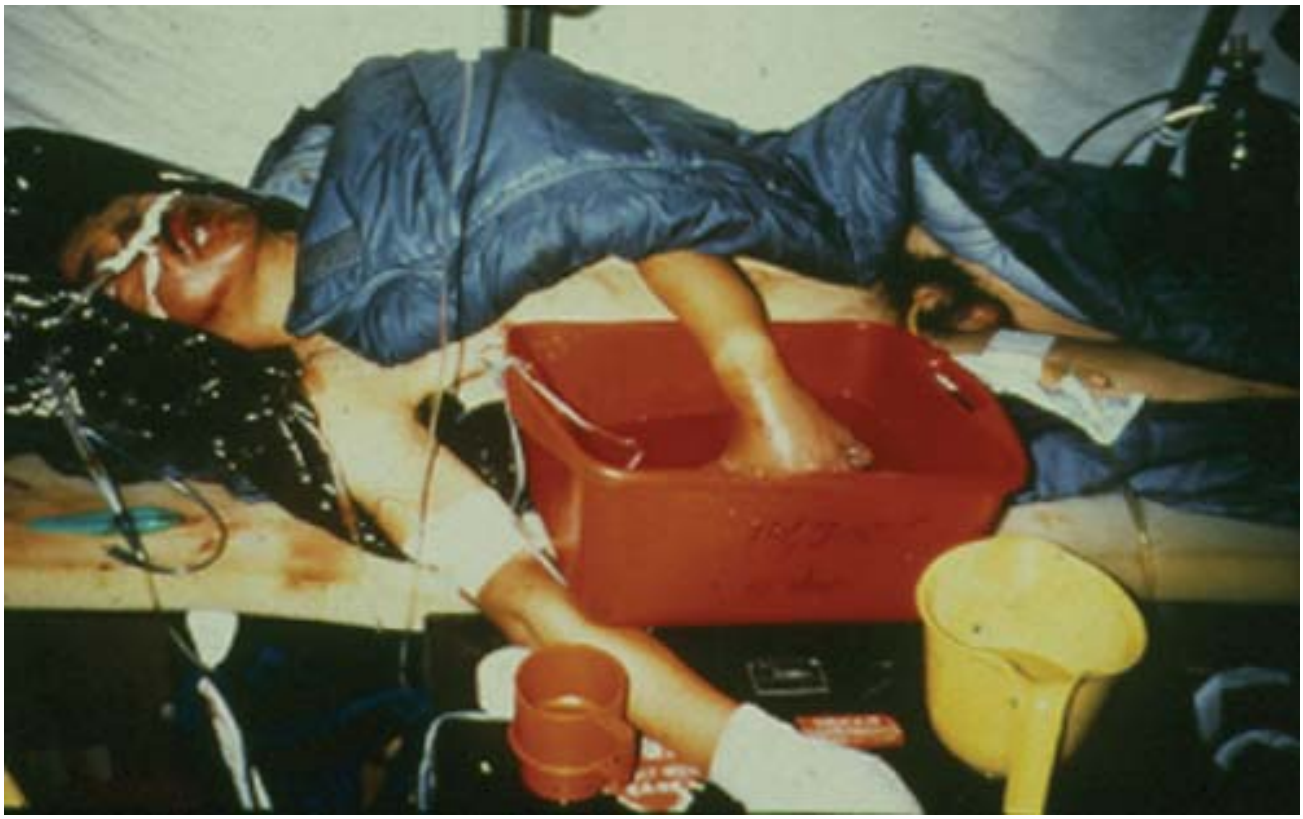

Fig. 26-4. A mountain climber with multiple environmental injuries receives initial treatment in a tent on Mount McKinley, Alaska. The patient had high-altitude pulmonary edema (HAPE), dehydration, multiple trauma from a fall, and severe frostbite injury to his hands. The fall was presumed to be the result of decreased physical and mental functioning caused by hypoxia and altitude illness. Multiple environmental injuries are common in mountain environments. Photograph: Courtesy of Peter Hackett, MD, Seattle, Washington.

and asphyxiation. It is not unusual for whole groups to be caught and injured in a avalanche or rockslide, creating a mass casualty situation.

Diagnosis and treatment of trauma in high mountains is the same as at sea level, with the admonition that transport of the patient may present problems due to rough terrain. Adequate protection of the patient from harsh climatic conditions, especially cold, is also important. Specific treatment of traumatic injuries is not the subject of this chapter but is covered in standard textbooks on orthopedics and sports medicine.

Prevention of trauma in mountain terrain requires appropriate training and equipment. Training should include proper use of equipment such as skis and climbing harnesses. Use of hiking sticks seems to prevent falls during recreational hiking in the mountains even though their use does not ease the strain on the legs and back in most individuals.[158] Unit personnel should also be familiarized with relevant environmental hazards, such as avalanches and rockslides, and know how to avoid being caught in them. Good physical conditioning helps prevent accidents in the mountains by lessening the contributions of fatigue and lack of strength to accidents.

Thermal Injury and Illness

It is no surprise to medical personnel that cold injuries occur in mountain environments, for most people associate mountains with cold and snow. The concept that heat stress can also occur in mountain environments is less obvious but nonetheless true (for an example, see the Preface in *Medical Aspects of Harsh Environments, Volume 1*[153]). Consequently, medical personnel supporting troops deployed to high altitude must anticipate the full spectrum of thermal injuries, although cold injuries are much more frequent. Prevention of thermal injuries through increased awareness and training should be a high priority. Increasing awareness in unit and

command personnel has been shown to decrease the incidence of cold injuries in the setting of military deployment to high altitude.[159]

Cold Injuries

Cold injuries are very frequent at high altitude. Mean ambient temperature drops approximately 2°C to 6°C for each 1,000 m increase in elevation, as a function of the inability of the decreased mass of atmospheric gas to absorb or retain significant amounts of heat. Progressively lower temperatures, combined with the high winds and precipitation that are frequent components of mountain weather, create significant cold stress. At the same time, the effects of hypobaric hypoxia on the body and on psychological function increase vulnerability to the cold. Altitude-induced increases in red blood cell mass and peripheral vasoconstriction from decreased plasma volume and dehydration significantly increase the risk of frostbite or freezing injury.[115] Hypoxia-related errors in judgment and blunted reasoning ability hinder the normal behaviors that protect against cold stress. One of the most common scenarios in the mountains is that of climbers taking off their gloves and, owing to hypoxia, forgetting to put them back on, causing severe freezing injuries to their hands.[115]

Diagnosis and treatment of cold injuries in the high-altitude environment is the same as at low altitude and is discussed extensively in *Medical Aspects of Harsh Environments, Volume 1*,[153] particularly Chapter 14, Clinical Aspects of Freezing Cold Injuries; Chapter 15, Nonfreezing Cold Injuries; and Chapter 16, Treatment of Accidental Hypothermia. Evacuation or descent to a lower altitude, or use of supplemental oxygen will help in treating cold injury that occurred at high altitude.

Heat Illness

Heat illness is much less common than cold injury in the mountains, but it does occur in certain situations. It is usually due to a combination of factors that increases an individual's heat load. In high mountain environments, the most common factors are increased solar load due to lack of shade above timberline; increased light reflection off of rock, snow, and ice surfaces (albedo); dehydration; and increased metabolic heat production due to the increased physical effort needed to accomplish work while hypoxic. Heat exhaustion due to these conditions is often referred to as "glacier lassitude" and has been chronicled during climbing expeditions for many years.[105]

The full spectrum of heat illness is possible at high altitude, but severe forms are not common. Diagnosis and treatment of heat injuries in the mountains is the same as at low altitude and is described in *Medical Aspects of Harsh Environments, Volume 1*,[153] particularly Chapter 8, Clinical Diagnosis, Management, and Surveillance of Exertional Heat Illness. Because hypoxia does not appear to make a significant contribution to the pathophysiology of heat injury, there is no reason to believe that descent or the provision of supplemental oxygen would be of any significant benefit in treating heat injury itself. Increased Sa_{O_2} would certainly not be harmful to a patient with heat injury, however, and might be beneficial for any coexisting altitude illness.

Ultraviolet Radiation Injury

Excessive UV radiation from sunlight (UV-A and UV-B; UV-C is almost entirely filtered out except at the polar regions) is a serious threat to military personnel operating in high mountain regions during daylight hours, because several factors in the environment increase its damaging potential. The decreased UV filtering by the thinner atmosphere at high altitude causes an increased intensity of exposure (~ 4% for every 300 m) and a shift of the UV spectrum to more-harmful short wave lengths.[107] Reflection from snow and ice not only significantly increases the intensity of exposure (~ 75% of the incident radiation is reflected) but also creates different directions for sources of exposure (UV radiation in sunlight usually comes from overhead; reflected UV radiation can come from the side or below). UV exposure can cause significant short-term disability to military personnel through severe sunburn and snow blindness. It can also cause discomfort through mild sunburn and reactivation of cold sores on the lips.

Prevention is the key to lessening the impact of solar radiation injuries on military operations. Prevention is facilitated by threat awareness and the use of sunscreen, protective clothing, and UV protective eyewear. As with most preventive measures, command emphasis may be very successful in ensuring compliance with preventive measures.

(Interested readers may also wish to explore two other volumes in the *Textbook of Military Medicine* series: *Occupational Health: The Soldier and the Industrial Base*,[160] and *Military Dermatology*,[161] in which sunburn, solar keratitis, and other damaging effects of UV exposure are also discussed.)

Sunburn

Sunburn is a well-known hazard of outdoor activity in high mountains. It occurs more rapidly than at lower altitudes, and is often more severe. Because people at high altitude are usually well clothed for protection from the cold temperatures, the sunburn is often confined to the exposed skin of the face, especially the nose and lips. The pattern of the burn can include the underside of the nose, chin, and eyebrows, owing to light's being reflected up from ice, snow fields, and large rock surfaces. This pattern is similar to that seen in people who spend considerable time on large bodies of water, where light is reflected up from the water surface, or in arctic areas, where it is also reflected off the snow and ice. In areas of the mountains where the ambient temperatures are warmer and people hike or work in short pants or take off their shirts, extremities and torsos can be burned in addition to the face. Individuals are often lured into a false sense of security about sunburn because the ambient temperature is still cool, although relatively warmer than at night or at higher altitudes.

The diagnosis and treatment of sunburn at high altitude is the same as that at low altitude. Due to the increased albedo, sunburn may occur in unusual areas such as inside the ears and nostrils, around the eyes, and under the chin. In the setting of military deployment where a decrement in manpower due to sunburn may have an adverse impact on the mission, aggressive treatment with systemic corticosteroids might be considered, to limit the period of disability.

Prevention of sunburn at high altitude relies on covering as much skin as possible with clothing and using sunblocking creams and lotions on exposed skin during the day. At high altitude, special attention should be given to protecting areas of skin that are vulnerable to albedo.

Snow Blindness

In addition to sunburned skin, the increased UV-A and UV-B radiation and reflection off snow and ice in high mountain environments can cause snow blindness (corneal keratitis and uveitis) similar to that seen in arctic regions. This condition is not only very painful, but the effective visual decrement resulting from severe light sensitivity (ie, photophobia) also makes ambulation or vehicle operation by the afflicted individual dangerous, if not impossible, in rugged mountain terrain. It would be similar in a combat situation. Snowblindness, like sunburn, can easily be prevented and usually occurs as the result of failure to take adequate preventive measures. Consequently, the incidence is quite variable.

Diagnosis and treatment of snow blindness is not different in the high-altitude setting than in the arctic. Signs and symptoms include severe eye pain (due to exposure of corneal nerve endings by sloughing of the damaged outer corneal layer), photophobia with constricted pupil and spasm of swollen and/or sometimes blistered eyelids, profuse tearing, and prominent blood vessels in the sclera and conjunctiva. Symptoms are often maximal 6 to 8 hours after exposure and resolve in 48 to 72 hours.[162] Patching the eye and controlling pain are the mainstays of treatment. Dilating the pupil and using ocular antibiotics are helpful. Pain control may require oral analgesics, sedatives, or both. The prognosis is generally good, although in severe cases permanent damage can occur.[163]

Prevention of UV injury to the eye is achieved by use of protective eyewear that filters UV light (ie, effective sunglasses). To prevent injury from reflected light, protective eyewear should have side shields. Additionally, the eyewear should have a safety cord or strap to prevent its being lost accidentally.

Cold Sores

The occurrence of cold sores on the lips due to reactivation of latent herpes simplex virus infection by UV light exposure is a common and well-known phenomenon at high altitude. Lesions can be treated with local application of an antiviral ointment such as acyclovir. There is probably little utility in treating with an oral antiviral medication in a healthy individual during a military deployment to high altitude. Likewise, there is probably no reason to use oral antiviral medication prophylactically. Prevention of UV exposure by using a sunblock such as zinc oxide is generally effective in preventing cold sores at altitude.

Lightning Strikes

Lightning strikes are a significant seasonal hazard in the mountains, especially in exposed areas above timberline. The thunderstorms that generate lightning are more frequent over high mountains, and many people are killed or injured by lightning in mountain regions.[164] Dusek and Hansen[165] reported a fatality and several injuries in soldiers struck by lightning during maneuvers in the Colorado mountains.

Lightning can strike a person in several ways.[166]

It can strike a person directly or strike an object that the person is touching or holding. These strikes tend to cause the most severe injuries. Lightning can jump from a nearby object that has been struck (ie, splash) or travel through the ground or water (ie, step voltage), types of lightning strikes that are often less severe than direct ones. Military personnel in close proximity to metal vehicles, armament, and communication equipment are at risk for splash strikes. Step voltage can cause injury to large groups with a single strike; military units moving in close formation are at risk for this kind of indirect strike. All strikes can cause blunt trauma if the person struck is thrown by violent muscle contraction or falls while unconscious.[167]

The electrical properties of lightning are those of massive voltage and current applied for a very short time. The current tends to run over the outside of the body (ie, flashover) so that there are fewer deep burns with lightning compared with high-voltage injuries from man-made sources, where the current is long-lasting.[166,168]

Lightning strikes usually cause initial cardiopulmonary arrest due to massive depolarization of muscle tissue and the central nervous system. The heart often resumes its normal beating rapidly, while the respiratory function lags behind due to prolonged paralysis of the diaphragm and the respiratory control areas in the of the brainstem. If victims get cardiopulmonary resuscitation support until the heartbeat and breathing resume, they can often survive. Other injuries include neurological abnormalities, burns, tympanic membrane rupture, ocular injuries, and blunt trauma from falling or being thrown. Neurological problems include confusion, anterograde amnesia, and sometimes transient seizures.[169]

Superficial burns can be linear or in a feathery pattern called Lichtenberg's ferning (also called Lichtenberg's flower). Linear burns are due to the current's following areas of sweat concentration. The mechanism of ferning is not known and it may not cause actual burns, for they fade within hours. Ferning is pathognomonic of lightning injury. Punctate burns through the skin are common with lightning strikes.[168] Second- and third-degree burns occur when metal objects such as rings or coins on the skin are heated by the lightning. Rupture of one or both tympanic membranes due to the blast effect of the lightning strike is common. Ocular injuries are also common, and many individuals will develop cataracts within a few days of being struck.[168] The extent of blunt trauma a victim sustains is a function of the distance that he or she falls or is thrown.

Initial treatment of lightning injury consists of cardiopulmonary resuscitation support. In multiple casualty situations, the apparently dead individuals should be resuscitated *before* medical officers attend those whose breathing and cardiac function are spontaneous,[170] a reversal of usual triage procedure. Once the heart and respiratory function return, treatment of the other injuries is by standard methods. Unlike victims of other high-voltage burns, lightning victims usually do not need massive amounts of fluid because they generally have few internal burns.[167] Tetanus prophylaxis is required in individuals with burns or open wounds.[167]

The prognosis for victims of lightning strikes who survive is good, initially, but after they recover from their acute injuries, victims may experience long-term effects, including posttraumatic stress disorder and other neuropsychological symptoms such as sleep and memory difficulties.[167]

Measures to prevent lightning strikes are essential for military units deployed to mountain terrain, and medical personnel should brief units appropriately. Where the pattern of thunderstorms can be predicted (eg, afternoon storms caused by the adiabatic water cycle), it may be possible to avoid high-risk areas (eg, above timberline). This may not be possible in combat situations. Personal protection includes staying at least 5 m away from tall and large metal objects, and from electrical and communication equipment. The recommended protective position in open terrain is crouching in a low spot with feet and legs pressed together. The interior of fully enclosed metal vehicles offers protection because a strike will flow over the outer surface of the vehicle.

Infectious Disease

It is a basic tenet of military medicine that infectious disease is a serious threat during any deployment. To effectively counter that threat, unit medical personnel must anticipate the general pattern of diseases associated with the environment to which the unit is being deployed.

The pattern of infections seen in high mountain areas appears different from that at low altitude,[139,140,159] and the cause of that difference has been speculated to be hypoxia-induced immune depression (discussed above). However, incidence of infection is a function of both the opportunity for exposure and the susceptibility of the population exposed. Consequently, factors other than immune function alone must be involved in determining the

pattern of infectious diseases that occurs in the mountains. Some of those factors that are characteristic of mountain environments are discussed below. A more-extensive discussion of infectious disease in military deployment is presented in another volume in the *Textbook of Military Medicine* series, *Military Preventive Medicine: Mobilization and Deployment*.[171]

Potential exposure to infectious diseases at high altitude is greatly affected by two factors: (1) the absence of many arthropod vectors and (2) the generally low socioeconomic status of many indigenous populations of mountain regions, with concomitant poor sanitation and public health. Additionally, some viral and parasitic infections seem to be found only in specific mountain regions of the world.

Arthropods serve as vectors to transmit a variety of infectious diseases. In mountain regions their populations decrease with increasing elevation,[172] probably as a function of the decrease in humidity and ambient temperature rather than the lower oxygen content of the air. Free-living insect vectors such as mosquitoes are limited to low altitudes except in regions of higher humidity.[173] As a result, the malaria they transmit is not generally seen at higher altitudes. Commensal arthropods such as fleas, lice, and mites, however, range to higher elevations because they inhabit man-made microhabitats. Infrequent bathing owing to harsh, cold conditions probably also contributes to the frequency of these vectors. Diseases spread by these pests, such as louse-borne typhus, can be expected to occur in high mountain regions. At high-enough altitude, even commensal arthropods cannot survive, possibly due to desiccation of their eggs. Eggs and oocysts of arthropod vectors and other parasites (eg, *Toxoplasma gondii*, the organism that transmits toxoplasmosis) are particularly vulnerable to low-humidity cold and increased UV radiation at high altitude. For example, the low prevalence of toxoplasmosis in Colorado is thought to be due to the effects of the mountain environment on the survival of oocysts in the soil.[174]

Poor sanitation and personal hygiene are common in both indigenous populations and sojourners at high altitude owing to cold climate, harsh living conditions, and low socioeconomic conditions (Figure 26-5). Fecal–oral transmission of viruses, such those causing hepatitis and bacterial enteritis, are common.[175,176] Fecal contamination of climbing routes on popular mountains such as Mount Everest and Denali is well known to climbers and trekkers. Given the significant role that these diseases have played in military operations in all environments, fecal contamination would be expected to also be a problem in deployment to high altitude. Anecdotal reports suggest that it is. Additionally, early in deployment, unit members may manifest diseases that were acquired at low altitude.[159]

Some infectious diseases seem fairly localized to specific mountain regions. Carrion's disease, which is caused by *Bartonella bacilliformis* and includes Oroya fever and characteristic skin lesions called verruga peruana, is the classic example. It is transmitted by a sandfly that is found only in river valleys of the Andes mountains.[177]

Diagnosis and treatment of infectious diseases at high altitude are the same as at low altitude and rely on antibiotics, supportive care, or both. Descent may improve immune function, but whether by relieving hypoxia or improving general health and nutrition status is hard to say.

Fig. 26-5. "*Ya must've missed Headquarters on the way up. It's halfway between where the timber stops growin' an' the sojers start shavin'*." There is less concern with personal hygiene in high mountain terrain, as this Bill Mauldin cartoon from World War II illustrates. Causes include inclement weather, limited water supply, and the effects of hypoxia on judgment and motivation. Poor hygiene may contribute to increased infections and the prevalence of disease-carrying arthropod pests. Copyright 1944. By Bill Mauldin. Reprinted with permission by Bill Mauldin.

Poor Hydration and Nutrition

Hypohydration

The poor hydration status of unit personnel is a potential problem in most military deployments, but it is a consistent occurrence during deployments to high mountain environments (interested readers should consult Marriot and Carlson's book[178] on this subject). At high altitude, hypohydration is nearly universal, and it degrades performance and contributes to various environmentally related injuries. Medical personnel in support of units that are deploying to mountain terrain need to be prepared to prevent and to treat problems related to inadequate hydration. The physiological mechanisms involved in altitude-related perturbations of hydration are described in the chapter by Montain in *Medical Aspects of Harsh Environments, Volume 3*.[179]

Fig. 26-6. "*I don't haul no water up no crummy mountain fer luxuries*." Cartoonist Bill Mauldin documented the problem of limited water supplies in mountain terrain during World War II. Hypohydration is almost universal in high mountain environments, owing to insufficient fluid intake and increased loss of body water. Limited access to sufficient water supplies is a major factor causing inadequate fluid intake. Copyright 1944. By Bill Mauldin. Reprinted with permission by Bill Mauldin.

Hypohydration in high mountain environments is the result of fluid intake inadequate to compensate for the high rate of fluid loss. Fluid loss is increased over that at low altitude owing to low ambient humidity and the body's response to hypobaric hypoxia and cold. The avenues of fluid loss include increased insensible loss through the skin and respiratory tract and a cold and hypoxia-induced diuresis. Insufficient fluid intake is due to a combination of decreased thirst and volitional behaviors. Thirst is suppressed by both hypoxia and cold. Hypoxia-induced malaise and effects on judgment and reasoning can further limit intake. Obtaining enough fluid to drink in the high mountains requires effort, for it must be either carried on the person and protected from freezing or acquired from melting snow and ice. When an individual is hypoxic, the effort to obtain and consume sufficient water may not seem important (Figure 26-6). Additionally, a conscious decision may be made to limit water intake to avoid having to remove clothing to urinate in cold weather and dangerously rugged terrain (ie, voluntary dehydration).

Diagnosis of hypohydration in a high-altitude setting is somewhat complicated, because many of the common signs and symptoms (eg, malaise, anorexia, dizziness, tachycardia, headache) could be due to hypobaric hypoxia or AMS. Even dry oral mucous membranes could be due to increased ventilation rather than a sign of disordered fluid status. Facilities for laboratory evaluation to support a diagnosis are seldom available during initial deployment or in a field setting. However, because hypohydration is so common at high altitude, it is probably appropriate to consider everyone at altitude to be at least mildly "dry" until proven otherwise. At altitudes below about 5,000 m, many individuals will regain normal hydration status after several weeks. Above that altitude, most will remain chronically hypohydrated.

Hypohydration, while very common at high altitude, is not inevitable. Short of providing continuous supplemental oxygen and adequate protection from cold, little can probably be done to decrease fluid losses. Therefore, prevention of dehydration in mountain environments relies on increasing fluid intake to match losses.

Poor Nutrition

Poor nutrition is a potential problem during deployment to many environments, including high terrestrial altitude. The consistent observation of

body weight loss during high-altitude climbing expeditions suggests that inadequate nutrition is an inevitable consequence of altitude exposure, and some research results support that concept. Consequently, unit medical personnel must be prepared to confront nutrition problems during deployment to mountain regions.[178]

Loss of body weight during sojourns to very high altitude (> 5,000 m) is so common it has been labeled "climbers' cachexia" (Figure 26-7). Weight loss at altitude is the result of hypohydration and negative energy balance. Negative energy balance is the result of decreased intake of food due to anorexia and behavioral phenomena combined with a possible increased energy expenditure. Anorexia can be the result of AMS early in deployment but is probably due to hypohydration and the effects of hypoxia later in exposure and at very high altitudes. Hypoxia-induced malaise and effects on judgment and reasoning can limit intake of food and fluid. Additionally, carbohydrates and fat from food that is consumed at very high altitude may be malabsorbed, although this has not been well demonstrated. Increased energy balance is the result of increased metabolic rate and increased energy expenditure to accomplish physical work. Initial weight loss at lower altitudes is largely due to mobilization of fat stores, but both fat and muscle protein are lost at high altitude and during chronic exposure.

Fig. 26-7. Altitude-induced cachexia in a mountain climber after spending 4 weeks above 6,400 m on Mount Everest. This individual lost approximately 12 kg. Wasting of fat and muscle tissue occurs in everyone at altitudes greater than 5,500 m and is termed "climbers' cachexia." It is caused by the combination of chronic hypoxia, inadequate caloric intake, and hypohydration. Photograph: Courtesy of Peter Hackett, MD, Seattle, Washington.

Weight loss is the primary sign of inadequate nutrition during deployment to high altitude. Most healthy individuals tolerate the loss fairly well and recover the weight when they redeploy to lower altitude. Although manifestations of poor nutrition other than weight loss are possible, they are not often seen in the time frame of usual climbing expeditions (ie, 6–8 wk). It has been demonstrated that high-altitude weight loss can be prevented at lower altitudes[180,181] and lessened at very high altitudes.[182]

Carbon Monoxide Poisoning

Carbon monoxide poisoning is known to occur in mountain environments. The actual incidence at high altitude is unknown, but anecdotal reports suggest that it is not uncommon.[183] The potential for exposure to hazardous levels of carbon monoxide during deployment to high terrestrial elevations has been recognized by the US Army for many years.[172] Exposure is most often due to increased production in a closed space. Carbon monoxide production by vehicle engines, cook stoves, and the like is increased owing to inefficient combustion caused by the lower oxygen content of the air. Outside of shelters, this increased production does not lead to increased exposure unless a person is in a position to breathe exhaust fumes. Because of the cold conditions at high altitude, people often try to stand close to engines and stoves to find warmth, and in doing so may be exposed to exhaust. Additionally, they may deliberately operate engines and stoves in poorly ventilated, enclosed areas (eg, tents, caves, and vehicles) to create heat. Increased levels of carbon monoxide from cooking stoves have been documented in tents, igloos, and snow caves at altitude,[184,185] and the carbon monoxide levels in snow caves were shown to increase blood levels of

carboxyhemoglobin in people inside the cave.[185] Foutch and Henrichs[183] reported that a small, backpacking-type stove caused the deaths of two men by carbon monoxide poisoning in a closed tent during a snowstorm at 4,300 m.

Carbon monoxide binds to hemoglobin in red blood cells much more tightly than does oxygen and thereby decreases the amount of oxygen that the blood can carry. It also shifts the oxyhemoglobin dissociation curve to the left[186] and decreases 2,3-diphosphoglycerate (2,3-DPG) in the red blood cell[187]—changes that decrease oxygen delivery to tissues. At high altitude, where the amount of oxygen in the blood is already limited, it takes less carbon monoxide to cause more significant tissue hypoxia than at lower altitudes. Because low levels of carbon monoxide exposure cause measurable decrements in physical performance at low altitude, they probably have increasingly significant effects at high altitude.[188]

Because the mechanism of injury in carbon monoxide poisoning is hypoxia, the early signs and symptoms are similar to those of AMS and HACE. They include headache, nausea, malaise, and shortness of breath, and they progress to coma. Retinal hemorrhages similar to HARH are also seen in carbon monoxide poisoning.[189] Consequently, it may be difficult at times to distinguish carbon monoxide poisoning from AMS or HACE in unacclimatized individuals during early altitude exposure. A high index of suspicion for carbon monoxide poisoning is appropriate in this setting. Evidence of exposure to possible sources of carbon monoxide should be sought. It is also important to not to rule out carbon monoxide poisoning based on the lack of cherry red skin color, as that is a late and inconsistent clinical sign.

The initial step in treatment is to prevent further exposure by removing the source of carbon monoxide or removing the patient from the source. Treatment then is similar to that at low altitude and consists of administration of 100% oxygen and providing appropriate supportive care. Since descent in elevation increases oxygen pressure, it is helpful to evacuate the patient to lower altitude. Individuals who recover without neurological damage can return to high-altitude duty without limitation.

Prevention of carbon monoxide poisoning in the military setting entails educating people to the danger and identifying the situations and behaviors to avoid.

Constipation and Exacerbation of Hemorrhoids

Constipation and exacerbation of hemorrhoids are so common in the high mountains[1,159] that the problems have achieved the status of medical lore in the climbing community. The common saying is that "the only chronic diseases of climbers are bedsores and hemorrhoids." "Bedsores" refers to a situation that climbers often face, that of being confined to small tents in a recumbent position during prolonged periods (days to a week or more) of inclement weather. Although the climbers find the confinement to be uncomfortable and frustrating, they do not actually develop bed sores. The combination of cold, blood stasis, and elevated hematocrit can cause thromboembolic events, as discussed earlier in this chapter.

Unlike bedsores, however, constipation and hemorrhoids are very real problems at high altitude. Although the phenomenon is well know anecdotally, the exact incidence is not reported. The cause is thought to be multifactorial: a combination of dehydration, low intake of fiber, behavioral avoidance of defecation due to the cold, the inconvenience of removing layers of protective clothing, and lack of suitable private sanitary facilities above timberline. Constipation is a well-known problem during military deployments in general and may be exacerbated during deployment to high mountains. Diagnosis and treatment are the same as at low altitude. While not usually not life-threatening, the condition can become severe enough to affect morale and performance. Preventive measures include increase in fiber in the diet, proper hydration, and provision of warm and protected sanitary facilities.

SUMMARY

In addition to the classic altitude illnesses (ie, AMS, HACE, and HAPE), the constellation of medical problems associated with deployment to mountain regions of the world includes a number of other conditions, some of which are related to hypoxia and others that are not but all of which medical support personnel will have to manage. Although many seem relatively benign, all can contribute to the disease and nonbattle injury decrement in unit fighting strength and degrade military performance. Environmental factors other than hypoxia that contribute to injury and illness in mountainous areas include rugged terrain, thermal stress, low humidity, and often violently inclement weather. Medical problems in which hypoxia plays a significant role include peripheral edema, high-altitude eye prob-

lems, sleep disturbances, thromboembolic disorders, suppression of cell-mediated immunity, and high-altitude pharyngitis/bronchitis with coughing severe enough to cause rib fractures. Chronic medical conditions that are present prior to deployment can be exacerbated by the hypoxia at high altitude, or they can increase the degree of hypoxia and worsen altitude illness. All hypoxia-related conditions can be improved by descent or supplemental oxygen. Medical problems that are not directly related to hypoxia but are common in mountain terrain include trauma, cold injury, sunburn and snowblindness, lightning strikes, carbon monoxide poisoning, some infectious diseases, poor hydration and nutrition, and constipation. Because hypoxia is not the major cause of these conditions, they are generally managed at high altitude in the same manner as at low altitude.

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