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# Effects of High Terrestrial Altitude on Military Performance

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An old military expression advises "Take the high ground" since elevated tactical positions have natural advantages for observation, aimed fire, and defense of surrounding terrain. Unfortunately, high ground is often rugged, lacking developed routes of transportation, and sparsely provided with food, water, and fuel. Besides the problems caused by the terrain and generally inhospitable climate there are also adverse effects on health and functional capabilities at altitudes above 3000 m due to inadequate oxygen which can adversely affect military performance (Disease threat, 1979; U.S. Army, 1975).

Much information of military importance about high altitude has been learned from mountain climbing expeditions, early balloon and aircraft flights (without supplemental oxygen), as well as experimental studies and medical disorders with oxygen insufficiency. Physiological changes after ascent may result in debilitating symptoms, adverse moods, and impaired ability to do physical work or perform mental tasks. Sleep and nutrition may be compromised.

This chapter describes the impact of high terrestrial altitudes above 3000 m upon an individual's ability to live and function. Emphasis is given to impairments resulting soon after ascent since they are usually greatest and can degrade military operations. Factors promoting 'success' at altitude such as medications, psychological strategies, and nutrients are also described.

## HIGH-ALTITUDE ENVIRONMENTS

Many people visit, work, or temporarily inhabit high-altitude regions because they enjoy the physical and psychological challenges. Such environments may offer tangible advantages as well; observatories on mountains provide clearer views of the stars and planets (Cudaback, 1984).

Physical Characteristics. At high altitude, several adverse physical characteristics make coping, functioning, and survival very difficult: hypoxia, cold, wind, dryness, solar radiation, and ultraviolet radiation (Heath,

Williams, & Harris, 1981). The proportion of oxygen in the air is constant at 20.93%, even at altitudes up to 100,000 m. The decreased atmospheric pressure at high altitudes results in a proportional reduction of the partial pressure of oxygen ($PO_2$). This lessens the amount of oxygen carried by the blood. The barometric pressures of two regions at the same elevation may differ, since atmospheric pressure also depends on latitude, thickness of the earth's crust, and weather (Mazess, 1975). The relationship between altitude and atmospheric pressure is described in tables which include adjustment factors for specific locations and conditions (Manual International, 1968).

High altitude regions are cold. Temperature drops about 1°C for each 150 m increase in elevation, independent of latitude (Heath et al., 1981). Increasing wind velocity greatly reduces body temperature and comfort. Dryness also influences heat loss and subjective comfort; at extreme altitudes, low ambient humidity increases respiratory water loss and can cause cracking of the skin. Solar radiation increases with higher altitudes. Solar radiation is 50% greater at 5800 m and snow can increase it by 50-65%. Bright reflective snow and clear, clean air also increase the ultraviolet load. At 4000 m, it can be 1.5-2.5 times greater, requiring appropriate eye and skin protection.

About 10% of the world's population lives in mountainous regions; however, only about 2% live above 3000 m (Mazess, 1975). Mazess feels the lack of food sources is responsible, while Heath et al. (1981) think the poor quality of sleep at high altitudes determines habitation preferences and limits. People will work as high as 5945 m in mines but they usually will not live above 5485 m (Houston, 1987).

Functioning in Extreme Environments. The unusual demands of a military training exercise in a moderately high altitude (2100-3050 m) and cold environment have been described (Davis, Curtis, & Bachinski, 1982).

> "The high altitude, snow-covered environment is unique in the demands imposed on marines. Failure to recognize and consider the implications of this environment can drastically distort operational requirements. It is difficult to convey all the encumbrances associated with maintenance of basal tasks in this environment." (Davis et al., 1982, p. 37)

More than 100 deaths, averaging two per expedition, resulted on Mt. Everest from falls, accidents in icefalls, or exhaustion (West, 1986a). These are reminders of the arduousness of all efforts, the extra attention necessary to complete even simple tasks and procedures, and the dire consequences of poor judgments, omissions, and lack of preparation.

The fact people live, work, and play at high altitude testifies to their adaptive capabilities. Adaptation to stressful environments results from behavioral, psychological, and biological compensations (Bachrach, 1982). Prior familiarity with the environment and its physiological effects aids accomplishment of mission objectives. Training also fosters success since one learns how to minimize many risks inherent in the environment by using protective equipment and clothing and rehearsing emergency procedures. Confidence results from safely testing the limits of human capabilities.

## HYPOXIA AND HYPOXEMIA AT HIGH ALTITUDES

Immediately upon ascent to high altitude, there is a decrease in the amount of oxygen carried by the blood (hypoxemia). The reduced partial pressure of oxygen in the ambient air lowers the rate at which oxygen can diffuse into the blood. This reduces the oxygen supply to the brain, working muscles, and other parts of the body and has profound effects on sensory processes, mentation, sleep, and physical work capacity. For example, Ashkenazi, Ribak, Avgar, & Klepfish (1982) found the circadian rhythms of physiological and performance parameters were shifted after a 3 min exposure to 7620 m. The higher one climbs and the harder one works, the more severe the hypoxemia.

The brain is very demanding of oxygen since it consumes 20% of the oxygen required by the body (Gibson, Pulsinelli, Blass, & Duffy, 1981). Before the dangers of hypoxemia were understood, balloon flights were sometimes fatal (Ernsting, 1978), and there were injuries and deaths of civilian recreational glider pilots (Fischer, 1982-1983). Errors in judgment and altered mental performance, caused by hypoxemia, are believed to have been a major cause of deaths to climbers on Mt. Everest (West, 1986a). Visual hallucinations of a "phantom companion", food, or clouds have been described with profound hypoxia (Heath et al., 1981; West, 1984b). Near the summit, "treacherous euphoria" may threaten life by increasing impulsiveness and bad judgments.

Oxygen Transport at High Altitudes. There are excellent summaries which describe the body's adaptive responses to high altitude (Fulco & Cymerman, 1988; Heath et al., 1981; Lahiri, 1977; Luft, 1972; Reeves et al., 1987; Young & Young, 1988). These responses, some immediate and some longer term, reduce hypoxemia and its effects; they are collectively termed "acclimatization". The body's first line of defense is increased ventilatory rate which ultimately elevates the oxygen concentration in the blood stream. This response to hypoxia occurs immediately, increases slowly over the first 1-2 weeks at altitude, and persists throughout the exposure period (Lahiri, 1977). If one climbs higher, respiratory ventilation increases even more. Other physiological mechanisms increase the efficiency of oxygen transport and utilization at altitude. Some compensations require several months to develop fully, but most are well developed within two weeks. Then, individuals are physically and mentally nearly as ready as they will ever be to live and work at a particular altitude. Even one week at altitude is sufficient to induce a large fraction of the eventual acclimatization (Hackett, 1980).

Medical Problems. There are three principal medical problems at high altitude and a host of minor ones which impact on physical and mental performance. The most common is acute mountain sickness (AMS). The symptoms of AMS are headache, loss of appetite, sleeplessness, lassitude, nausea, vomiting, and general malaise (Hackett & Rennie, 1979; Hackett, Rennie, & Levine, 1976; Heath et al., 1981; Houston, 1987; Hultgren, 1979; Meehan & Zavala, 1982). AMS begins in susceptible individuals within a few hours to a day. Fortunately, the symptoms of uncomplicated AMS resolve spontaneously within a few days to a week as the body acclimates to that particular altitude, but they can reoccur upon further ascent. Susceptibility to AMS is generally persistent (Forster, 1984); both genders seem to suffer equally, although it may lessen with age (Hackett et al, 1976). The incidence and severity of AMS symptomatology are directly related to the rate and height of ascent (Hackett et al., 1976; Hackett & Rennie, 1979; Miller, Pincock, & Wright, 1983); the faster and higher one climbs, the greater the chances of being affected. With rapid ascent a few individuals will be affected at about 3000 m, about 40% at 3650 m, about 75-80% at 4300 m (Burse, Rock, & Fulco, 1986), and nearly everyone to some extent at 5000 m (Hackett et al., 1976). Up to 50% of those affected at each altitude will be sick enough to interfere with the performance of military duties, at least for a few days because of headache, nausea, and malaise (Dusek & Hansen, 1969; Maher, 1981; Malconian & Rock, 1988). Numerous publications outline these effects, their amelioration (Foulke, 1985; Hackett, 1980; Hackett & Rennie, 1979; Heath et al., 1981; Houston, 1987; Hultgren, 1979; Meehan & Zavala, 1982; Mountain, 1987; Ward, 1975), and assessment (Fletcher, Wright, Jones, & Bradwell, 1985; Sampson, Cymerman, Burse, Maher, & Rock, 1983; Wright, Jones, Fletcher, Mackintosh, & Bradwell, 1985).

There are two potentially fatal medical problems, high altitude pulmonary

edema (HAPE) and high altitude cerebral edema (HACE). In HAPE, the lung capillaries appear to become leakier in response to hypoxia. Some of the blood plasma is forced into the lung, especially during exercise (Foulke, 1985; Gray, 1983; Mountain, 1987; Schoene, 1987). This interferes with oxygen transport, rendering the victim severely hypoxemic and compromising the capillaries even more. Unless treated as soon as possible by descent to lower altitude, the lungs continue to fill until the victim gets too little oxygen to survive. The most obvious signs of HAPE are coughing up frothy, pink-tinged sputum and rales, the crackling sounds heard in the lungs on inspiration (Foulke, 1985; Hackett et al., 1976). The incidence of HAPE is 1-15% above 4000 m (Hackett et al., 1976; Singh, et al., 1969).

The signs of HACE result from the effects of increased pressure within the skull. Increased intravascular pressure, cerebral blood flow, cerebrospinal fluid pressure, or leaky cerebral capillaries are suspected causes (Hackett, Rennie, Grover, & Reeves, 1986; Hamilton, Cymerman, & Black, 1986; Wohns, 1981). Autopsy shows swollen, spongy brain tissue and multifocal brain hemorrhages (Singh et al., 1969). Onset of HACE is indicated by mental confusion, slurred speech, or an uncoordinated walking gait, all of which may be hard to detect initially. These signs can progress rapidly from hallucinations, ataxia, and somnolence to paralysis, coma, and death (Hackett, 1980; Hackett & Rennie, 1979; Pines, 1978). The first appearance of these signs should be regarded as life-threatening and cause for immediate descent and oxygen therapy. The victim usually requires assistance, which may jeopardize mission accomplishment. Others must be prepared to intervene if the victim is leader and asserts authority to prevent or delay descent.

Peripheral edema is quite common at altitude and is different from HACE. It causes no particular difficulty beyond the puffy face, ankles, and wrists,

but it may indicate a predisposition to AMS. Those with peripheral edema at 2800 m had a 64% incidence of AMS at higher altitudes, compared to only 43% in those without peripheral edema (Hackett & Rennie, 1979).

AMS, HAPE, and HACE can be prevented in most cases by ascending slowly to 3000 m, preferably on foot, and then climbing less than 500 m at a time, allowing one or two days to acclimatize at each altitude (Hackett, 1980). Shuttling the components of the next camp up 300-500 m over a day or two, and returning to sleep at a lower camp each night ("Climb high, sleep low".) takes advantage of this method. In 1980, a woman team climbed as high as 6800 m by slow ascent on foot without significant medical problems (Kramer, Drinkwater, Folinsbee, & Bedi, 1983). If AMS occurs, more time can be given for additional acclimatization and symptom subsidence. Signs and symptoms of HAPE, HACE, or AMS that do not resolve are indications for immediate descent to low altitude.

Minor retinal hemorrhages also occur at high altitude, especially at altitudes above 5200 m (McFadden et al., 1981). Normally, there is no interference with vision and the hemorrhage resolves upon descent (Houston & McFadden, 1979). Other medical problems also occur at high altitude: cold urticaria (hives), frostbite, sunburn and snow blindness, dehydration from inadequate water intake, and respiratory and gastrointestinal infections (Disease threat, 1979). These problems can be minimized with proper clothing, equipment, and hygiene.

Work Performance. The physiology of work and exercise performance at high altitude and extreme high altitude is detailed in several sources (Fulco & Cymerman, 1988; Reeves et al., 1987; Sutton, Jones, Griffith, & Pugh, 1983; Torrance, Lenfant, Cruz, & Marticorena, 1970/1971). The most important effect of high altitude on physical performance is the reduction in the maximum rate at which the body can use oxygen to perform physical work (maximum aerobic ca-

pacity). This means not only is the maximum work rate less at high altitude, and progressively more so the higher one goes, but also the endurance time for submaximal work is reduced and greater effort is required.

Aerobic work capacity is progressively diminished at altitudes greater than 1500 m (Buskirk, 1969; Hartley, 1971; Squires & Buskirk, 1982). Recent hypobaric chamber experiments simulating a climb of Mt. Everest showed loss in aerobic capacity to be a monotonically increasing function of altitude (Cymerman et al., in press). These data also validate the linear approximation developed by Squires and Buskirk (1982) and Hartley (1971) of an average 10% loss in capacity for each 1000 m increase in elevation above 1500 m, showing it to have little error up to 5500 m. The decrement in maximal capacity persists as long as the individual remains at altitude (Young & Young, 1988). There is no more than 6% recovery, no matter how complete the acclimatization (Boutellier, Marconi, DiPrampero, & Cerretelli, 1983; Dua & Sen Gupta, 1980; Saltin, Grover, Blomqvist, Hartley, & Johnson, 1968). The decrement disappears either immediately upon (Buskirk, Kollias, & Picon-Reatigue, 1967) or shortly after (Cymerman et al., in press) descent to sea level.

The decrement in submaximal endurance is less predictable, as it appears to depend on time at altitude and rate of work in addition to motivational factors. When physical work load is reduced at altitude to match the percentage of aerobic capacity taxed at sea level, average endurance times are approximately the same. The energy costs of grade walking at various speeds and loads can be predicted by the same equations as at sea level (Cymerman, Pandolf, Young, & Maher, 1981). Of course, the amount of external work (loads carried, rate of ascent) that can be accomplished is much less, because the total work rate includes the added work of faster breathing plus that imposed by any environmental clothing and climbing equipment. It is not clear how much

submaximal endurance increases with prolonged acclimation to altitude; contradictory reports exist, ranging from a small change after several weeks to large improvements up to 60% (Horstman, Weiskopf, & Jackson, 1980; Maher, Jones, & Hartley, 1974). After 18 days' acclimatization to 4300 m, less muscle glycogen is used up and less lactic acid is produced in response to 30 min bouts at 85% of maximum capacity (Young et al., 1982); this implies greater submaximal aerobic metabolism, despite exercising at the same percentage of a relatively unchanged aerobic capacity.

There appears to be no loss in the isometric strength of large muscle groups, at least as high as 4300 m (Young, Wright, Knapik, & Cymerman, 1980). There is one report of reproducible gains in isometric handgrip strength of 5-15% at 4300 m (Burse, Cymerman, & Young, 1987), which persisted for at least two weeks, but disappeared after 2 days at sea level.

## SENSORY, NEUROLOGICAL, AND OTHER EFFECTS

Early scientific descriptions of the effects of high altitude are noteworthy and still cited (e.g, McFarland, 1932; Tune, 1964). Cudaback's (1984) review of symptoms, moods, the senses, various task performances, and selected job performances is informative. The senses are affected by altitude before cognitive and psychomotor performances.

Vision. Sensitivity to light, visual acuity, and color discrimination decrease at altitudes above 3000 m, and vision is the first sense affected by hypoxemia. Some performance decrements resulting from hypoxemia are due, in part, to changes in visual sensitivity. Fowler, White, Wright, and Ackles (1982) demonstrated reaction times were increased by hypoxemia; reaction times at altitude to low intensity visual stimuli were even greater. Cahoon's (1970) signal detection study showed decreased detectability of a visual stimulus resulted from a change in the perceptual sensitivity of the visual system (d')

rather than a change in the observer's response criterion (Beta).

Dyer (1988) summarized many classic references and reviewed the effects of altitude upon vision (dark adaptation, acuity, contrast sensitivity, depth perception) and visual structures. Altitude's effects upon vascular, neural, and electrophysiological processes were also considered. High-altitude exposure produces changes in dark adaptation which are likely neural in origin (Kobrick, Zwick, Witt, & Devine, 1984; Luria & Knight, 1987). Acute exposure (eq. 5790 m) for 15 min changed scotopic, i.e. rod receptor, sensitivity in dark-adapted subjects; exposure to a lesser level of hypoxia (eq. 3810 m) for 3 h did not (Luria & Knight, 1987). Similar exposure for 72 h also did not change scotopic sensitivity or field-of-view (Luria & Morris, 1988). At 4300 m, sensitivity was decreased during the first 10 min of dark adaptation (Kobrick et al., 1984). Effects were evident after the first day and most pronounced after 4-9 days, a time when symptoms and other effects were minimal (Houston, 1987; Shukitt & Banderet, 1988). After 10 days, a descent to 3200 m for 8 h with subsequent re-ascent to 4300 m resulted in some recovery. Dark adaptation degraded again with increased time at 4300 m. This study also demonstrates that visual impairments may persist after recovery from symptoms, adverse moods, and impaired performances. Small refractive errors have been documented in a male climber on two separate occasions after descent from 6000 m; however, their cause is unknown (Hamilton, 1982). Surprisingly, contrast sensitivity was unaffected after gradual ascent to 7620 m (Kobrick et al., 1988).

Such effects were carefully considered by the Australian Defense Department when they recommended visual standards for military free-fall parachuting (Gordon, 1982). Proposed standards were deliberately conservative because of some of the anticipated visual impairments resulting from hypoxemia during

free-fall and the parachutist's mission.

A light-amplification, night vision system was evaluated in subjects that were hypoxemic (Leber, Roscoe, & Southward, 1986). Measures of visual acuity from subjects using the system were compared with acuity measures from their unaided eyes. Measures of unaided visual acuity were impaired by hypoxemia; whereas measures with the night vision system were unaffected. This also suggests that vision in aviators flying at 2400-3000 m, without supplemental oxygen, may benefit from the night vision system.

Audition. Sensitivity appears unaffected (Heath et al., 1981).

Mood States. Exposure to high altitude may initially produce euphoria and errors in judgment. Euphoria can be very dangerous since its effects are usually not recognized by the affected person (Houston, 1987). Air Force personnel, assigned flying duties, experience a hypobaric chamber demonstration of the effects of hypoxia as part of their training (Beatty, 1986). The insidious nature of hypoxia and its role in errors, bad judgments, and accidents are emphasized. Unfortunately, a person's reaction to acute hypoxemia is usually a poor predictor of his reaction to chronic hypoxemia (Houston, 1987).

Even moderate levels of altitude alter metabolism of biogenic amines (Francesconi, 1988; Mazess, 1975), so it is not surprising that changes in mood and affect result (Katz, 1982). After 1-4 h at 4300 m subjects rated themselves as less 'friendly' and 'clear thinking'; they were also more 'sleepy' and 'dizzy' (Shukitt & Banderet, 1988). Surprisingly, subjects were also 'happier', perhaps related to the initial euphoria of high altitude. Most of these moods have a characteristic time course at high altitude similar to that of AMS. After 48 h, all moods adversely affected by high altitude had recovered. At 1600 m, increased 'sleepiness' was detected initially but it returned to sea level values after 18 h. No morning-evening differences in mood

were found on any mood factor at 200, 1600, or 4300 m. In a separate study at 3840 m 'sleepiness' and 'dizziness' increased the first day of exposure (Shukitt, Burse, Banderet, Knight, & Cymerman, 1988). Moods and symptoms appeared more sensitive to the effects of low altitude than did measures of cognitive performance; mood was not affected at 1800 m.

Two of the six mood factors on the Profile of Mood States varied with altitude in Operation Everest II (Houston, Sutton, Cymerman, & Reeves, 1987). With increased altitudes, vigor decreased and fatigue increased. Mood profiles were similar to that of soldiers who volunteer for military training and duty in mountainous terrain and world class athletes; such profiles were different than those of typical adult males.

Personality Changes. Sixteen men and four women were tested during an expedition to Mt. McKinley (Nelson, 1982). At 3800 m, personality characteristics and cognitive functioning were similar to that at sea level. At 5000 m, undesirable personality changes were evident (increased paranoia, obsessive compulsiveness, depression, hostility) and cognitive functioning was also dramatically impaired. Ryn (1988) described mental disturbances in climbers related to the level of altitude and duration of stay at high altitude. The neurasthenic syndrome, characterized by fatigability, lack of motivation, feeling inadequate, and psychosomatic symptoms, is common at 3000-4000 m. The cyclothymic syndrome occurs at 4000-7000 m and involves alternating depressed or elevated mood. Acute organic brain syndromes occur above 7000 m and result from structural or functional defects in the central nervous system. The climber's personality, emotional atmosphere associated with climbing, high degree of risk, and other biological and psychological factors are important in the etiology of such mental disturbances.

Neurological Functioning. Mountain climbers and balloonists have known that high altitude produces changes in affective behavior, judgment, and cognitive performance. Some neurochemical researchers suggest the acute effects of hypoxemia, and even subsequent AMS, result from changes in neurotransmitter utilization and concentrations (Gibson et al., 1981). One of the primary neurotransmitters affected is acetylcholine, since its metabolism is very oxygen-dependent. Serotonin and norephinephrine are also important so it is not surprising that hypoxemia alters neurological functioning. At high altitude, sympathetic arousal increases, e.g. increased heart rate and cardiac index (Holness, Fraser, Eastman, Porlier, & Paul, 1982). Body movement and posture change, and finger tremor and anteroposterior (forward-backward) sway increase (Holness et al., 1982). Sway is greater with a rapid onset of hypoxia or with eyes closed; tremor is not affected by either factor. Lateral sway increases when the eyes are closed but is not affected by the rate of onset of hypoxia.

There is growing concern some undesirable effects of altitude exposure may be long-term. Hemorrhages of the retina, nails, and gastric mucosa are observed at high altitude; they may also occur in the brain (Heath et al., 1981). Also, Habeler still suffered nightmares and memory lapses a few years after he and Messner climbed Everest without supplemental oxygen. Medical personnel, some who are also climbers, express concerns that extreme climbs (almost resulting in death or completed without supplemental oxygen) may produce long-term or even permanent behavioral changes (West, 1986a).

New trends in mountain climbing increase the risk of damage to the nervous system (Rennie, 1976; Ryn, 1988; West, 1986a). Climbers may ascend with little staging time, without supplemental oxygen, during the winter, or without securing ropes. Such practices increase the challenge of climbing high mountains but they also provoke two other concerns. Some neurological effects

of extreme exposures to high altitude may be latent and have minimal impact until the brain is subsequently traumatized by another injury or stressor (Ewing, McCarthy, Gronwall, & Wrightson, 1980). If so, an earlier impairment from climbing could combine with the degradation from a new trauma and magnify it. Secondly, climbing attracts many professionals with livelihoods dependent upon exceptional cognitive and psychomotor capabilities (West, 1986a).

Data suggest long-term or permanent effects are equivocal. Over 100 climbers who spent time above 5500 m indicated by survey in the mid-1970's they did not feel they or their peers were permanently impaired by altitude (Houston, 1987). Over 20 individuals climbing above 5300 m were carefully studied with several psychological tests; these data supported Houston's earlier findings of no long-term effects (Clark, Heaton, & Wiens, 1983).

Recent studies provide the strongest evidence for long-term effects. Perhaps there is a greater incidence of such effects, advances in behavioral assessment yield more sensitive measures, and climbers are taking risks more likely to compromise the nervous system. Ryn (1988) documented personality and mental effects in 80 Polish Alpinists from 1960-1985. In some climbers, he inferred permanent injury to the central nervous system because of the persistence of mental changes long after the climb.

Townes, Hornbein, and Schoene (1985) studied 51 climbers from five expeditions to Mt. Everest. Various neuropsychological tests suggested transient changes in verbal expression and possibly memory, depending upon how soon people were tested after the climb. Most remarkable and persistent was a bilateral motor impairment even one year after the climb. These changes at high altitude and immediately after descent were replicated in Operation Everest II. Cavaletti, Moroni, Garavaglia, & Tredici (1987) studied memory in seven people who climbed to 7100 m without oxygen. Even 75 days after the climb, memory

impairments persisted. Regard, Oelz, Brugger, Biol, and Landis (1989) measured small impairments in concentration, short-term memory, and ability to shift concepts and control errors in five of the eight world class climbers that they studied. Some 2-10.5 months earlier, all climbers had been at altitudes above 8500 m for 48-358 h (Regard et al., 1989). However, others feel the physical trauma, dehydration, and weight loss, e.g. climbers studied by Ryn, are responsible for such changes (Clark et al., 1983; Houston, 1987). West (1986a), a climber and physician, sounded a cautionary alert emphasizing the possibility that long-term changes may result from climbing under conditions too extreme. He calls for increased awareness of these phenomena since climbers, physicians, and educators cannot appraise the risks and choose conditions appropriate for long-term well-being unless they are informed.

Although such data support the possibility that permanent adverse effects may result from exposure to extreme altitudes, a recent study yielded negative findings. Jason, Pajurkova, & Lee (1989) assessed climbers who spent time above 7200 m; two climbers even reached the summit of Mt. Everest. Impairments only resulted above 5200 and after descent there was no evidence of any impairments. The absence of impairments below 5200 m or after descent suggests the assessment battery lacked sensitivity or potential 'long-term impairments' were confounded with practice effects.

Sleep. Mountain climbers, hikers, and tourists at high altitude have reported disturbed and fitful sleep. At high altitude, respiratory periodicity with apnea (actual pauses in breathing for 10-20 sec or more) and sleep disruption occur (Anholm, Powles, Houston, Sutton, & Bonnett, 1989; Sutton, 1982; Sutton et al., 1979; White et al., 1987). Self ratings of 'sleepiness' increase after acute ascent to 1600-4700 m (Banderet, 1977; Shukitt & Banderet, 1988) or gradual ascent to 7000-7600 m (Houston et al., 1987). Even af-

ter a full night of sleep, one is seldom rested.

In 1972, sleep at high altitude was studied with electroencephalography for the first time in a military research lab on top of Pikes Peak, CO (Reite, Jackson, Cahoon, & Weil, 1975). Sleep stages 3 and 4 decreased and periodic breathing occurred; however, total sleep time did not change. During the 12 days at 4300 m, electroencephalogram (EEG) characteristics, the number of awakenings during sleep, and subjective ratings of sleep quality returned to sea level values. At altitude, there was a disparity between objective and subjective measures of sleep quality. Although Reite et al. (1975) suggested the difference was related to the frequency of arousals, they concluded the subjects' intense complaints were disproportionate for the situation. It appears they did not recognize that the disruptiveness of such arousals might also influence subjective appraisal of sleep quality. Powles, Anholm, Houston, & Sutton (1988) recognized traditional scoring of EEG records would not detect brief arousals of 3-4 sec since such arousals are not predominant in a 20-30 sec scoring epoch. Looking closely at brief events in the EEG records, Powles et al. (1988) found a relationship between number of arousals during nighttime sleep and deficits in daytime performance.

At night there were several episodes of marked hypoxemia and 3-4 sec arousals, e.g. 22-84 arousals/h, during Operation Everest II (Powles et al., 1988). Such arousals were not observed in all apneic cycles but all subjects experienced them. The number of arousals and the degree of apnea-induced hypoxemia during sleep seemed better predictors of daytime cognitive impairments than sleep stages. At altitude, subjects also found it more difficult to go to sleep and to stay asleep. Indirect measures of arterial $PO_2$ during nighttime sleep were less than those observed in the daytime (Powles et al., 1988; White et al., 1987); such acute $PO_2$ in the daytime would probably be lethal in an

unacclimatized subject. Sleep efficiency and number of awakenings during sleep did not change above 4572 m; however, subjects were less active behaviorally above 6100 m and spent more time napping (Anholm et al., 1989). Rapid eye movement sleep was decreased by 70% during hypoxemia, but slow wave sleep did not change. Various sleep stages did not produce any significant changes in arterial $PO_2$. Apnea, at high altitude, is of central origin (Anholm et al., 1989; Powles et al., 1988).

Sleep quality, quantity, and self ratings are improved by acetazolamide (Sutton et al., 1979; Sutton, 1982). Sleep at high altitude is generally deteriorated by sedatives since they depress ventilation and relax the muscles of respiration (Harper, 1983; Sutton et al., 1979; Sutton, 1982). This can further increase hypoxemia and complicate sleep apnea at hign altitude. Improvements in ventilatory and blood-gas measures during acclimatization have also been described for various stages of sleep and wakefulness (White et al., 1987).

Currently, there is a controversy as to whether daytime performance impairments in sleep apneics result from severe hypoxic episodes induced by apnea during sleep or from disrupted EEG sleep stages and fragmented sleep (e.g. Bonnet, 1985). The best predictor of the hypoxic aspects of apneic episodes in a study of patents with sleep apnea was the number of desaturations during sleep showing a change of 4% or more (Berry, Webb, Block, Bauer, & Switzer, 1986). This criterion correlated with several of the patients' daytime performance measures but did not predict changes in sleep stages. Daytime cognitive impairments in an experimental study of sleep apneics were found to result from hypoxemic episodes during the evening and to a lesser extent from sleep fragmentation (Potolicchio, Hu, & Kay, 1988). Thus, it appears the degree of hypoxemia during sleep, rather than changes in sleep

stages, is correlated with daytime performance changes.

Unexpectedly, Hornbein, Townes, Schoene, Sutton, and Houston (1989) found subjects with the greatest ventilatory responses at high altitude desaturated most at night during apneic episodes. White et al. (1987) showed that periodic breathing at night is also more common in such individuals. Such individuals also had the greatest performance decrements on cognitive tasks during the daytime. Hence, subjects best able to adapt to hypoxemia and perform physical work during waking hours were most impaired in their cognitive performances because of ventilatory responses during sleep (Hornbein et al., 1989).

## COGNITIVE AND PSYCHOMOTOR EFFECTS

Minimal Altitudes with Effects upon Performance. Knowing the effects of varying levels of high altitude is of critical importance for many military and civilian activities. Astronomers determine if oxygen enhancement is required in observatories above 3800 m (Cudaback, 1984). Requirements for supplemental oxygen or pressurization (1800-2438 m eq.) in aircraft are based upon high altitude research and experience. Guidelines are re-evaluated periodically to ensure human capabilities are not compromised in especially demanding environments or that the structural specifications, weight, and safety systems of aircraft are not excessive (Ernsting, 1984).

Cognitive performance is usually more vulnerable to hypoxemia than is psychomotor performance (Berry et al., 1986). Complex performances are usually affected before simpler performances (Cahoon, 1970; Woodward & Nelson, 1974), and activities requiring decisions and strategies are more vulnerable than automatic processes (Defayolle, 1985). Performances involving visual input of shapes, patterns, and contours are thought to be more vulnerable to impairments at altitude than those involving numbers, words, or characters.

Tune (1964) suggested that an altitude of 3048 m or greater will cause

perceptual-motor impairments. However, Denison, Ledwith, and Poulton (1966) found increased reaction times on a spatial task while subjects exercised at an altitude 600 m less than that specified by Tune. Denison et al. (1966) suggested that task novelty, resulting from learning of new task information, made performance more vulnerable. Guided by these data, it was recommended cabin altitudes in aircraft be maintained below 2438 m eq. to ensure the performance of aircrews (Ernsting, 1978). Recently, Fraser, Eastman, Paul, and Porlier (1987) reported increased postural sway at 1521, 2438, and 3048 m; however, Hamilton (1988) challenged their analyses and interpretation.

Other investigators showed varied performances are more robust to the effects of hypoxemia than this earlier finding suggested. Fowler, Paul, Porlier, Elcombe, & Taylor (1985) attempted to replicate Denison et al. (1966) and found greater altitudes were required to produce effects. Their investigation also showed the importance of controlling arterial $PO_2$ rather than atmospheric pressure or gas mixtures to ensure comparable hypoxemia. They concluded the findings of Denison et al. (1966) probably resulted from an unusually low arterial $PO_2$, caused by the resistance of breathing through a facial mask which became significant during hypoxia, exercise, and hypoventilation. Hence, Fowler, Paul, Porlier, Elcombe, & Taylor (1985) concluded 2438 m eq. was substantially lower than the threshold altitude that affects performance. Also, new learning is not more vulnerable to the effects of hypoxemia.

Performance on the Grammatical Reasoning Test was not significantly impaired by 2440 or 3050 m of altitude in four groups of 30 civilians (Green & Morgan, 1985). Performance rates did not change. Greater error rates at 3050 m were attributed to apprehension. In a clinical study, 7 subjects were exposed to 3048 m for 6.5 h. Their cognitive performances appeared affected; however, the largest effects of a few subjects were emphasized and inferential statis-

tics were not used.

To demonstrate the minimum altitude that causes changes on perceptual motor performance, Fowler, Elcombe, Kelso, & Porlier (1987) established an altitude-response curve for the serial choice reaction time task at two different levels of stimulus brightness. Arterial $PO_2$ was manipulated by having 6 subjects breath low-oxygen gas mixtures so that varied levels of hypoxemia (2712 to 3475 m eq.) were produced. Response times slowed in an altitude-dependent manner; the minimum threshold for effects was estimated at 2972 m. These data from a choice reaction time task are strong support for Tune's assertion that the minimum threshold for altitude effects on most performance tasks is approximately 3050 m. Hence, 2438 m eq., an earlier estimate of the minimum threshold altitude that produces performance impairments and its implied aircraft pressurization requirements, were unnecessarily conservative and should be revised (Fowler, Paul, Porlier, Elcombe, & Taylor, 1985; Fowler, Elcombe, Kelso, & Porlier, 1987).

Speed-accuracy tradeoffs. Performance impairments at high altitude can result from increased errors, slowing of performance, or a combination of these degradations. Banderet et al. (1986) studied cognitive tasks requiring processing of numbers, words, and patterns under conditions of cold, dehydration, and hypoxia. Before the experiment, subjects practiced these tasks extensively, received performance feedback, and maintained low error rates. During exposure to a stressful environment, the rate of problem solving decreased; some errors resulted but they contributed little to the impairments. These findings illustrate a common functional strategy where the rate of performance is sacrificed for accuracy (Berry et al., 1986; Woodward & Nelson, 1974). At 4600 m, performance recovered to prior rates with time after ascent; there was also a slight decrease in the number of errors. Other analyses of

these data suggested the rate of baseline performance does not predict the magnitude of performance impairments resulting from altitude or other environmental stressors.

Cognitive data, collected at altitudes from 5500-7600 m in the Operation Everest II study, also exhibited a slowing of response rate for computer-administered tasks (Kennedy, Dunlap, Banderet, Smith, & Houston, 1989) and paper and pencil tasks (Banderet et al., 1986). Tapping, using fingers on the preferred and nonpreferred hands, was not affected and is consistent with the notion hypoxemia affects cognitive functioning more than motor functioning (Kennedy et al., 1989).

Mackintosh, Thomas, Olive, Chesner, & Knight (1988) studied 20 climbers from The Birmingham Medical Research Expeditionary Society on two separate climbs to 5000 m. There were increased reaction times in climbers ill with AMS. Such increased latencies were interpreted as due to hypoxemia rather than the lethargy associated with AMS. Increased errors were also observed but they were not related to altitude or to the symptomatology. A slowed processing model incorporates trends for altitude exposure, aging (Hale, Myerson, & Wagstaff, 1987), and nitrogen narcosis (Fowler, Ackles, & Porlier, 1985).

Fine and Kobrick (1978) found increased errors at high altitude on tasks such as receiving information from radio transmissions. This was expected since task characteristics influence the quality of performance and number and types of errors (Woodward & Nelson, 1974). Errors are more likely to occur when tasks are paced by external conditions, e.g. assembly line or receiving radio transmissions that cannot be "said again". Under stressful conditions when the pace of the task is set by the subject, e.g. paper-and-pencil tasks, response rates are likely to be sacrificed for low error rates.

The P300 waveform, a positive, endogenous, event-related brain poten-

tial, may provide a new tool for investigating performance impairments. Decreasing levels of arterial $PO_2$ increased P300 latencies and reaction times; hypoxemia had no effect on P300 amplitude (Fowler, Kelso, Landolt, & Porlier, 1988). Measures of P300 latency and reaction time were highly correlated; whereas, P300 amplitudes and reaction times were not. It is thought this measure reflects the processes of evaluation rather than those involved with selecting or executing a response. So, increased P300 latencies indicate hypoxemia slows stimulus evaluation processes. Another study demonstrated that both the reaction time and movement components of a reaction time task are affected by hypoxemia (Fowler, Taylor, & Porlier, 1987).

Time Course of Performance Impairments. Performance impairments are not constant with time at high altitude. At 4600 m, impairments on various performance tasks were greatest immediately after ascent (Banderet et al., 1986). In other studies for up to 7 h at 4200 and 4700 m, a variety of performances were affected soon after ascent to altitude (Banderet & Lieberman, 1989). From 2-7 h the performances improved slightly or remained impaired. After exposure to 4600 m, performance recovered to baseline levels on most cognitive tasks in 14 h; however, some performances remained impaired even after 38 or 43 h (Banderet et al., 1986). In contrast, the symptoms of AMS and most adverse mood changes were greater with durations up to 24-38 h. These data emphasize the dissociation between performance impairments and the symptoms of AMS. Performance was most impaired before the onset of AMS; later, when symptoms were maximal, performance was improved or fully recovered.

## *FACTORS INFLUENCING 'SUCCESS' AT HIGH ALTITUDE*

Predicting individuals who will be asymptomatic and functional above 3000 m or climbers likely to reach the summit of a mountain at extreme altitude is of great interest. At high altitude, severely affected individuals may

experience symptoms which may be life threatening unless the condition is treated. Affected individuals are difficult to evacuate; they also strain personnel and logistical resources. Also, outcomes, such as mission accomplishment or life and death, may be determined by critical judgments and actions of each participant.

A number of biological, motivational, attitudinal, and personality variables appear related to 'success' at high altitude. Greater ventilatory responses to hypoxia result in higher arterial $PO_2$ values (Hornbein, 1983); however, others are skeptical of this advantage (Griffith, Pugh, & Sutton, 1983). In fact, individuals with the greatest daytime ventilatory drives may experience the largest oxygen desaturations at night during sleep (Hornbein et al., 1989). They also experience the most impaired cognitive and psychomotor performances in the daytime. Many wonder how Peter Habeler and Reinhgold Messner climbed Mt. Everest in 1978 without oxygen or how Messner was able to make an even more remarkable climb of the north slope without supplemental oxygen two years later (Griffith et al., 1983; West, 1986b). These climbers were carefully compared with Edmund Hillary and Tenzing Norgay, the first climbers of Mt. Everest on 29 May 1953. Biological variables were evident, but many differences between these climbers resulted from training, work patterns, motivation, and exceptional behavioral tolerance of adverse conditions.

Personality variables are also important. Climbers on the 1983 expedition to Karakorum (8610 m) had different personality characteristics than similar nonclimbing personnel (Magni, Rupolo, Simini, DeLeo, & Rampazzo, 1985). Climbers were more detached, had weaker super egos, and were less anxious. The personalities of climbers showed good functional integration, which enabled them to perform unusual endeavors away from their families and routine activities. Defayolle (1985) classified subjects into a "good" or "bad" group, based

on an interview and personality measures, and found their moods and performances differed during military operations when they were exposed to freezing temperatures at 2000 m altitude. "Bad" subjects had traits such as immaturity and impulsiveness and were predisposed to greater difficulties in adjusting, coping, and performing in prolonged stressful situations. In demanding environments such as high altitude, excessively emotional, activated, or motivated people may become liabilities since their ability to detect, process, and discriminate critical information are likely to be decreased.

Motivation and attitude are usually exceptional in the successful climber. Heath et al. (1981) cited the parallels between military campaigns and mountain expeditions, e.g. assault, conquest, and defeat. The mountain and competitor climbers are the enemy. Nationalism can also be a powerful, but sometimes unhealthy, motivator. Other motivating forces are less specific. George Leigh-Mallory, who vanished with his climbing partner Andrew Irvine while climbing the northeastern ridge of Mt. Everest, described his motive for pursuing the mountain with the simple words, "Because it's there!" (Heath et al., 1981, p. 71). Petiet, Townes, Brooks, & Kramer (1988) studied eight women who attempted sites in the Himalayas up to 6250 m. Psychosocial variables and gender seemed to result in different appraisals of mission objectives and strategies for the climb. Although these climbers were unsuccessful and there were isolated behaviors reflecting individual goals, the collective well-being of the team was predominate and optimized. Other personal qualities cited are desire, singleness of mind, intensity of purpose, ability to exploit opportunities, climbing virtuosity, motivation, and luck (Hornbein, 1983). Mission training and experience also contribute greatly to success (Bachrach, 1982).

Health and lifestyle variables also influence 'success'. Heavy consumption of tobacco or alcohol, heart or lung disease, anemia, or chronic med-

ical illness are liabilities in this stressful environment (Johnson & Rock, 1988). Physical exertion at 3658 m eq. does not impair mental and psychomotor performances (Lategola, Lyne, & Burr, 1982). A study at 3810 m simulated altitude demonstrated mental and psychomotor performance was impaired by altitude and/or higher workloads; performance was impaired less by sleep deprivation. Sleep deprivation and altitude exposure interacted, especially at increasing cognitive workloads (Mertens & Collins, 1986). Sleep deprivation or altitude did not appear to interact with age, although age-related performance impairments were observed. In a second study at the same altitude neither age nor altitude changed urinary 17-ketogenic steroid excretion rates or Fatigue Check List responses (Mertens, Higgins, & McKenzie, 1983). Altitude per se did not affect cognitive performance.

## REDUCING THE ADVERSE EFFECTS OF ALTITUDE

Increased Oxygen Availability. Supplemental oxygen reduces or defers most effects of high altitude. Pilots in high performance fighter aircraft use it as do some mountain climbers (Houston, 1987; West, 1986a). Its main limitations are logistical, i.e. weight, bulk, and the difficulty of transporting it. A recent invention, a large inflatable nylon bag, offers promise to individuals severely afflicted with altitude disorders (Geer & Gamow, in press). Individuals critically affected are put into the bag, which is then inflated with a hand pump to increase the atmospheric pressure inside the bag, effectively lowering the altitude. This temporary treatment offers many of the advantages of supplemental oxygen while the afflicted individual is transported to a lower altitude, but imposes fewer logistical requirements.

Acclimatization. Prolonged stay at high altitude results in physiological acclimatization which produces adaptive effects upon oxygen delivery and regulation of metabolites (Heath et al., 1981; Hultgren, 1979; West, 1984a; Young

& Young, 1988). Beneficial effects occur within days to weeks, depending upon the altitude, rate of ascent, and the measures selected. With enough time at altitude, there is usually a dramatic reduction in symptoms, adverse mood changes, and behavioral impairments.

Staging. Acclimatization can be induced in many ways. Mountain climbers and others use the strategy of slow ascent to high altitude, providing time for acclimatization and symptoms to subside (Foulke, 1985; Houston et al., 1987; Hultgren, 1979). Others try staging, where people stay at one altitude for a few days before ascending further (Houston et al., 1987; Hultgren, 1979). Wearing a rebreathing device at sea level which produced a hypoxic breathing mixture was not beneficial (Burse & Forte, 1988). Another strategy was evaluated in personnel working in observatories at 4200 m (Forster, 1985). Some personnel had intermittent exposures to high altitude since they lived and slept at low altitude but drove to the top to work. These personnel with experienced AMS symptoms for more days than personnel who stayed continuously at high altitude.

Medications. Selected medications also improve wellbeing and functioning at high altitude. Acetazolamide is the medication of choice (Birmingham Medical, 1981; Foulke, 1985; Hultgren, 1979; Lassen & Severinghaus, 1987; McIntosh & Prescott, 1986; Mountain, 1987). Acetazolamide, a carbonic anhydrase inhibitor, stimulates ventilation and partially corrects acid-base and gas exchange imbalances in the blood, without impairing cognitive performance (White, 1984). At high altitude it increases arterial $PO_2$ (Sutton, 1982), improves sleep, and reduces symptoms (Fulco & Cymerman, 1988) and adverse moods (Banderet, 1977). It reduces symptoms best when combined with staging (Evans, Robinson, Horstman, Jackson, & Weiskopf, 1976). Dexamethasone, a powerful anabolic steroid, is even more effective than acetazolamide (Foulke, 1985; John-

son et al., 1984; Johnson & Rock, 1988; Rock et al., 1989). Dexamethasone also reduces performance impairments and mood changes without adverse psychological effects (Jobe, Shukitt Hale, Banderet, & Rock, unpublished). However, it is controversial since some assert it may have side effects (Ellsworth, Larson, & Strickland, 1987; Zell & Goodman, 1988). Tyrosine, a precursor of the neurotransmitter norepinephrine, reduces some of the adverse effects of high altitude and cold (Banderet & Lieberman, 1989). Tyrosine resulted in improvements on measures of symptoms, moods, and various performances in those subjects who showed average or greater than average adverse effects during another environmental challenge when they were treated with placebo.

Furosemide (Hultgren, 1979), phenytoin (Burse, Landowne, Young, & Maher, 1982; Wohns, et al., 1986), and naproxen (Meehan et al., 1986) are ineffective or possibly harmful. Sleep preparations, alcohol, and sedatives should be avoided. Stimulants may increase the symptoms of AMS and impair performances requiring subtle discriminations or judgments.

Psychological Strategies. The fact man lives, works, and plays in adverse environments testifies to his adaptive capabilities (Bachrach, 1982). Psychological factors can facilitate coping and functioning in high-altitude environments (Heath et al., 1981; Hornbein, 1983). Experience in stressful environments minimizes performance impairments because of adaptive changes in behavioral arousal, attentional capacity, or controlled vs. automatic processing of the task (Bachrach, 1982; Fowler, Ackles, & Porlier, 1985; Hancock, 1985). Involvement in a performance task during high altitude exposure apparently decreases the types and intensities of discomforts reported (Nesthus, Bomar, Holden, & O'Connor, 1987). Experiencing th symptoms and discomforts of a stressful environment will facilitate subsequent coping in similar situations (Bachrach, 1982; Fowler, Ackles, & Porlier, 1985; Stretch, Heslegrave, & An-

gus, 1987). If personnel are aware of the nature and time course of altitude effects, they can devote extra attention to tasks, create checks for errors, manage tasks in ways ensuring redundancy, and develop other compensatory strategies (Defayolle, 1985; Druckman & Kraman, 1988; Nesthus et al., 1987).

Other Strategies. Common antacid tablets reduced altitude effects (Roach et al., 1983), although earlier data suggested they were ineffective (Hultgren, 1979). Supplemental intake of potassium is contraindicated (Heath et al., 1981). Limited physical exertion at high altitude appears to minimize symptoms (Mountain, 1987); however, individuals with superb physical conditioning are just as likely to experience the effects of high altitude and the symptoms of AMS (Hultgren, 1979).

## OTHER DYNAMICS AT HIGH ALTITUDE

Nutrition. High-altitude conditions, especially above 6000 m, cause anorexia, a self-induced starvation (Potera, 1986). This condition is also thought to contribute to burnout, accidents, and even death, e.g. 40% of the casualties on Mt. Everest occurred as climbers descended. Anorexia is aggravated by climbing activities and fatigue since it takes time to prepare and eat 5000-6000 kcal of food per day (e.g. 20 slices of bread, 20 ounces of meat, and 8 cups of milk). The hedonic qualities of foods may be less at high altitude since the basic flavors of taste are reduced (Heath et al., 1981). Anorexia has been observed in long-term altitude studies in the lab, even when preferred and highly nutritious foods were provided (Rose et al., 1988).

Personnel in high-altitude environments require increased calories and special nutrients. High altitude, cold, and extreme physical activities can dramatically increase caloric demands (Potera, 1986). Food sources supplying carbohydrates are preferred to fats since carbohydrates replace depleted muscle glycogen and require less oxygen for metabolism (Hornbein, 1983; Potera,

1986). Extra vitamins (due to decreased food intake) and up to 5 liters of water per day may be required. A balanced diet at high altitude is 52% carbohydrates, 33% fats, and 15% proteins (Consolazio & Schnakenberg, 1977).

The caloric intake of a 4-man climbing team decreased 35% at altitudes below 6000 m and 57% during the final ascent; they lost 7% of body weight (Guilland & Klepping, 1985). They preferred carbohydrates over fats at high altitudes. Four other climbers during a Himalayan expedition also preferred carbohydrates; AMS was not affected by the type of ration (Gray & Milne, 1986). Six men studied during Operation Everest II decreased their caloric intake by 42% during ascent from sea level to 8848 m (Rose et al., 1987, 1988). Their carbohydrate intake decreased significantly from 62 to 53%. Although these men lost 9% of their initial body weight, caloric intake increased during "staging", 2-3 day scheduled intervals at constant altitudes. Rose et al. (1988) noted appetite suppression and decreased caloric intake can last for several weeks; however, acclimatization can minimize these effects somewhat. They suggested that hypoxemia per se was responsible for the weight losses.

Compounding the Effects of High Altitude. Some substances interact with high altitude to depress vital functions, decrease arterial $PO_2$, or increase toxicity. Carbon monoxide from cigarette smoking, weapons firing, or vehicle exhausts in semi-enclosed spaces may aggravate hypoxemia at high altitude (Denniston, Pettyjohn, Boyten, Hiott, & Piper, 1978). Animal studies have shown central nervous system stimulants such as the sympathomimetic amines can be lethal at high altitude. Such stimulants were ten times more toxic in mice at 5791 m than at sea level (Robinson, Baumel, & Blatt, 1969). Stimulants also impair subtle discriminations and judgments in humans (Defayolle, 1985).

Alcohol increases high altitude symptomatology and can adversely affect thermoregulation, motor control, and judgment (Foulke, 1985). Time to uncon-

sciousness in animals at 7620 m was decreased by 38% after ingestion of 0.5 ml of 100% alcohol per pound of body weight (Heath et al. 1981). Surprisingly (Collins, Mertens, & Higgins, 1987) did not find alcohol potentiated the effects of 3566 m of simulated altitude on various measures of performance; alcohol and altitude yielded separate impairments of similar magnitudes, but there were no synergistic effects. At altitude, sleeping pills and other depressants reduce ventilation aggravating hypoxemia and degrading the quality of sleep (Sutton, 1982). Long acting sedatives may impair coordination and judgment during the next day before they are completely excreted.

Possible interactions of chemical warfare pretreatment drugs and altitude have been evaluated (Krutz, Burton, Schiflett, Holden, & Fischer, 1987; Schiflett, Stranges, Slater, & Jackson, 1987). Pyridostigmine bromide, a reversible cholinesterase inhibitor, did not interfere with pulmonary functioning, impair overall performance, or interact with altitudes of 2438 or 3962 m.

Contact Lenses. Wearing contact lenses at high altitude may cause discomfort and even opacities of the cornea (Castren et al., 1985; Eng, Rasco, & Marano, 1978). Type of lens, atmospheric pressure (Castren et al., 1985), dryness (Eng, Harada, & Jagerman, 1982), and inadequate diffusion of oxygen into the cornea (Eng et al., 1978) have been implicated. Two climbers have successfully worn contact lenses above 7300 m (Clarke, 1976).

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