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LESSONS FROM HISTORY

# Extreme Altitude: Words From on High

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Medical science has its own objective language for describing the effects of high altitude. Mountaineers' words and metaphors tell the story with subjectivity and feeling. This essay will include only limited physiology about lowlanders and high altitude. Instead, the focus will be literary, using the quotations of 20th-century mountaineers and mountaineer physicians to provide color commentary about the hardship. These are Words From on High.

*Key Words:* altitude, mountaineering, literature, quotations, Himalaya, Everest

## Introduction

Medical science has its own objective language for describing the effects of high altitude. Mountaineers' words and metaphors tell the story with subjectivity and feeling. Many of the Words From on High quotations in this essay are from the penultimate risk area in the Himalaya where "all places above 8,000 meters (26,246 feet) belong to the dead."[1(p20)]

This essay will include only limited physiology about lowlanders and high altitude, acute mountain sickness (AMS), decreased exercise capacity at altitude, high altitude pulmonary edema (HAPE), high altitude cerebral edema (HACE), the Death Zone, and high altitude deterioration. Instead, the focus will be literary using the quotations of 20th-century mountaineers and mountaineer physicians to provide color commentary about the hardship in these categories.

## HIGH ALTITUDE AND AMS

High altitude may be defined as 2440 m (8000 feet), with extreme high altitude at greater than 5490 m (18,000 feet).[2(pp4–5)] High altitude illnesses occur at or even below the lowest of those levels, beginning with AMS, a constellation of symptoms including headache, malaise, nausea, and difficulty sleeping. AMS and its complications are often diagnosed by the Lake Louise Score,[3] and much of the physiology in this essay is summarized from the text of West et al.[4] The *sine qua non* of AMS is headache, probably related to early cerebral edema whose much rarer end point is HACE. Severe headache stops many hikers and mountaineers. American John Roskelley on Nanda Devi complained that "[e]very muscle was throbbing, especially the one between my ears."[5(p81)]

Abnormal breathing at night persists with further ascent, even in the acclimatized.[4(pp202–215)] Lowland sleepers at high altitude have poor-quality sleep and a central sleep disturbance with instability in the feedback systems that control ventilation. Canadian surveyor E. O. Wheeler in Tibet on the 1921 British Expedition to Everest describes it this way: "I find that I am on the point of dropping to sleep…when I seem to hold my breath and wake up gasping."[6(p267)] British 1930s Himalayan veteran and prolific writer Frank Smythe is his usual dramatic self here, exaggerating for his lay audience: "Cheyne-Stokes breathing is experienced by those who climb high.…The fact that at lower levels it is only seen in dying persons is proof in itself of the borderline conditions on Everest."[7(p613)] Periodic breathing at high altitude was well known to both mountaineers and scientists by then, but the average reader would have only been familiar with oscillatory or agonal breathing patterns in terminal illnesses. On the other hand, Smythe may not have been overstating. He was familiar with the highest altitudes, where severe hypoxemia with nighttime apneic spells is impressive, and the rule "climb high, sleep low" is wise. Smythe wisecracks that "[t]o sleep well at high altitudes a man must be very tired."[7(p584)]

Altitude acclimatization is a physiological process involving respiratory, circulatory, hematologic, metabolic,

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and other changes whereby the body partially adjusts to reduced partial pressure of oxygen, but there is variable individual susceptibility, response, allowable ascent rate, and limit. Only previous experience is a useful indicator of who is at risk. British Himalayan explorer Eric Shipton notes that "[o]ne man acclimatises quickly, another slowly.…The whole process appears to be analogous to sea-sickness about which predictions are impossible."[8(p376)] Like acclimatization, deacclimatization is variable as well. One rule of thumb is that after descent it takes about as long to lose acclimatization as it does to gain it, but some individuals have more "carryover" than that, anecdotally perhaps several weeks.[2(p230),4(p62)] The other half of the Shipton-Tilman explorer duo, H. W. Tilman, stretches it wildly to 3 years on his Kilimanjaro climb: "I was beginning to feel the effects of altitude…but it was very noticeable that these symptoms were not nearly so well marked as in 1930, substantiating the well-known fact that acclimatization is retained to a certain degree over long periods."[9(p103)] More likely, he benefitted from different conditions and improved recognition and management at high altitude, including pace, workload, and hydration.

## DECREASED EXERCISE CAPACITY

Aerobic exercise capacity decreases as altitude increases and differs from the more protracted condition of high altitude deterioration. Even with acclimatization and despite strikingly greater ventilation, lowlanders can only partially improve to submaximal exercise levels and endurance compared with their own performance at sea level.[4(pp164–184)] Even simple activities become arduous, especially at great altitudes. The following quotations may reflect a combination of many factors, including both decreased exercise capacity and deterioration, about which more will be said later. Here is Tilman again: "I had mountaineer's foot—inability at times to put one in front of the other."[10(p525)] Joe Tasker, who with Pete Boardman disappeared on the Northeast Ridge of Everest in 1982, portrays the suffering: "It was as if we were runners on a race track we knew with a ball and chain on each foot,"[11(p23)] and he gauges that "[a] load of 30 lbs is crippling above 20,000 feet."[12(p66)] At the top of Everest, with acclimatization but without supplementation, maximal oxygen uptake of only 1 L/min is so low, just above the basal metabolic rate, that a climber uses it for muscles to breathe and to stay warm, with only little left over for ambulation, or thinking.

## HIGH ALTITUDE PULMONARY EDEMA

High altitude pulmonary edema (HAPE) is a life-threatening form of noncardiogenic pulmonary edema occurring in otherwise healthy people at high altitude with individual predisposition, cold, workload, and other factors contributing. Historical landmarks of the modern era include a 1960 HAPE case report[13] by Charles Houston, MD, mountaineer and author of K2 fame and Operation Everest hypobaric chamber studies. In the same year, Herb Hultgren, MD of Stanford reported even more cases of HAPE[14] and in 1964 gave catheterization results from his South American studies,[15] thus helping define the circulatory dynamics of HAPE and advancing the still-accepted pathophysiology of hypoxia-induced inhomogeneous pulmonary artery precapillary constriction and hypertension, with overperfusion of lesser-affected segments causing pulmonary capillary leakage and edema.[16,17]

Tasker applies some British understatement in *Savage Arena*: "Apart from the difficulty of performing any action with such a low oxygen intake, there is also an unquantifiable risk of contracting pulmonary or cerebral oedema, the sickness of high altitude which fills the lungs or brain with fluids. This, at best, is incapacitating and at worst is fatal."[11(p179)] Neurologiist Charles Clarke, MBBCh, in *Everest the Hard Way* in 1976 recognizes that " '[d]isproportionately breathless' is the key to it all.…Clearly, he was in incipient pulmonary edema.…Slowly, it became abundantly obvious that he must go down."[18(p92)] The diagnosis was not so obvious in previous times. Here is Physician Raymond Greene, MBBCh, in 1974 looking back to Everest in 1933: "My diagnosis then was pneumonia, but now, forty years later, I realize that he had the mysterious disease of great altitude we call acute pulmonary oedema. I gave him oxygen all night and the next day sent him down."[19(p146)]

## HIGH ALTITUDE CEREBRAL EDEMA

High altitude cerebral edema (HACE) is usually associated with even higher and longer stays. It is perhaps the end point of unbridled AMS and is immediately life-threatening. Incompletely understood, HACE is a vasogenic and cytotoxic brain swelling that usually occurs in the setting of AMS or HAPE.[4(p300),20] Presentation may be initially subtle, with ataxia and mental status changes dominating, but unconsciousness can follow rapidly in just hours.

The central nervous system is exquisitely sensitive to hypoxia, with or without edema. The authors here and the next section probably had dull brains from "only" simple hypoxia or hypothermia and exhaustion. Then again, many climbers have come down stumbling and mumbling, thus meeting the ataxia and altered mental status criterion for at least early HACE. In severe HACE, once coma has developed, death is much more likely,[20]