LESSONS FROM HISTORY

High Altitude Deterioration: A Historical Essay

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This wilderness essay about high altitude deterioration will explore the historical mountaineering and medical literature with a limited discussion of physiology. The writings of mountaineers and physician-mountaineers provide an evocative supporting narrative to illustrate one of the problems of living at altitude.

Keywords: acclimatization, metabolism, expedition, lassitude, Everest

Introduction

European observation and scientific writing on high altitude deterioration (HAD) started around the turn of the 20th century when protracted siege-style attempts and extreme altitude exposures began on expeditions to the Andes and especially the Himalaya, known as the Greater Ranges. One definition came from Michael Ward (1925–2005), mountaineer and surgeon on the 1951 and 1953 British Everest expeditions: “High altitude deterioration means a gradual diminution in man’s capacity to do work at great heights. This is associated with insomnia, lack of appetite, loss of weight, and increasing lethargy. These symptoms appear after a prolonged stay above [5486 m] 18,000 ft and there is great individual variation.”¹

Ward, along with British physician James Milledge (1930–) and Australian-American physiologist John West (1928–) wrote the first edition of *High Altitude Medicine and Physiology*.² The fifth edition contains a broader definition of HAD: “weight loss, poor appetite, slow recovery from fatigue, lethargy, irritability and an increasing lack of will power to start new tasks. There is slowing of mental processes, dulling of affect and impaired cognitive function.”³

The threshold range for HAD is about 4570 m (15,000 ft) to 6000 m (19,685 ft), and there is considerable individual variation.^{1–4} This altitude coincides with the definition of extreme altitude as being at about 5500 m (18,050 ft), where atmospheric pressure is roughly half that of sea level.⁵ HAD advances dramatically faster as one goes higher, and even

with adequate warmth, hydration, and nutrition it inexorably overcomes acclimatization.³

There have been many varied and extraordinary performances and outcomes at extreme altitude, where some live and some die. However, HAD may have less written about it than some other high altitude disorders. For example, only 2 of 12 pages in a chapter titled “Altitude acclimatization and deterioration” are devoted to HAD alone.³ For comparison, a chapter on acute mountain sickness (AMS) has 20 pages.³ As another example of the disparity, the diagnosis of AMS, high altitude pulmonary edema, and high altitude cerebral edema have the Lake Louise consensus score with a self-grading and clinical assessment portion, but there is no such method for the determination of HAD.⁶

HAD may lack a grading system because it is not one simple disorder but rather has variable manifestations from the threat of hypoxia but also cold exposure, workload, right ventricular strain, caloric deficits, and other high altitude factors. Excessive height and exposure time are major risks for rapid development of HAD, particularly in the *Todeszone* (Death Zone), so named in 1953 by the Swiss physician-mountaineer Edouard Wyss-Dunant.⁷ Today, the threshold of the Death Zone tends to be rounded to 8000 m (26,247 ft). Survival there, for those lucky to be alive at all, is limited to hours or days. For example, in 1986 during a multiple fatality disaster high on K2, Austrians Kurt Diemberger and Willi Bauer spent 5 days camped at 8500 m (27,900 ft), with Bauer setting the over-8000 m survival record of 10 days.⁸

HAD-related signs and symptoms may not fit neatly into a syndrome as do AMS, high altitude pulmonary edema, and high altitude cerebral edema. HAD may overlap with the effects of severe hypoxia alone. The onset of HAD and the height at which it begins vary with individual

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sensitivity.^{1–4} HAD is an issue separate from the much longer stays in high altitude residents with chronic mountain sickness. Chronic mountain sickness is a maladaptation to high altitude characterized by hypoventilation, hypoxemia, excessive erythrocytosis, and pulmonary hypertension progressing to right heart failure.^{3,9}

This wilderness essay on HAD focuses on quotations from mountaineers and physician-mountaineers in the historically oriented “Words from on High” style of the author.^{10–16} Use of the mountaineering literature provides an evocative supporting narrative to illustrate one of the problems of living at altitude. An extensive review of the current understanding of high altitude physiology or its application to those living with chronic hypoxia from medical or environmental conditions is beyond the scope of this essay. What follows is not a summary of high altitude physiology but only a few selected topics. After that will follow the medical and historical observations from the past.

A limited review of some of the current understanding of high altitude physiology

How much of the multisystem problems of HAD are caused by hypobaric hypoxia, malnourishment, metabolic changes, or other factors was only partially recognized years ago. Even the understanding of hypoxia itself is relatively recent. One author indicated that “the term hypoxia rather than anoxia was not used until 1940.”¹⁷

Lack of adequate nutrition might seem at first to be the simplest component of HAD to understand. Loss of weight can be a prominent feature during both expeditions and scientific studies at high and extreme altitudes.^{4,18} Muscle mass loss can be greater than that in simple starvation, suggesting an adverse effect of hypoxia on protein metabolism.³ Today, it is thought that exposure to hypobaric hypoxia alone will suppress appetite and caloric intake, but it is only part of the subsequent and multifactorial loss of body mass during lengthy stays.¹⁹ Some current authors suggest that this effect, rather than being a negative, acts instead as a protective or adaptive feature (at least for a while) by making amino acids and ketones available as metabolic substrates and as metabolic modulators protecting cells from hypoxia.²⁰

Aerobic exercise capacity decreases as altitude increases, but the more protracted condition of HAD causes not just a reduction in muscle function but also wasting. With prolonged exposure to severe hypoxia, muscle tissue undergoes structural and functional deterioration and has a delayed recovery from exercise, a decrease in muscle proteins and enzymes, and a reduction in muscle mass. In addition, with neuroendocrine and other factors affecting muscle microcirculation, there are changes in nutritional blood flow to the tissues, with changes in both aerobic and anaerobic metabolism.^{21–23}

Subcellular mechanisms have also recently become better understood. A family of transcription factors named hypoxia inducible factors (HIFs) was discovered. One of the first clues about HIFs was found in 1988 during the identification of a hypoxia response element in the enhancer gene for erythropoietin, a hormone that stimulates erythrocyte proliferation and is part of the response to hypoxia and high altitude.²⁴ The HIF-1 alpha signaling cascade mediates responses to reduced oxygen availability in multicellular animals, with upregulation of target genes not only for the generation of more red blood cells but also for more blood vessels. Many, many other functions are affected by HIFs, just one example being the genes that control mitochondrial oxygen consumption.²⁵

Hypoxia challenges oxidative phosphorylation at the inner mitochondrial membrane. With subacute hypoxia exposure, a coordinated downregulation of mitochondrial biogenesis occurs, with resultant reduced mitochondrial density. It is theorized that this matches better the muscle oxygen demand to the decreased supply. In addition, a shift in the balance from oxidative to glycolytic metabolism prevents excessive generation of reactive oxygen species and protects the mitochondria but does so at the cost of impaired efficiency. Despite a loss of mitochondria, successful acclimatization to high altitude over several weeks can improve skeletal muscle energy dynamics both at rest and with exercise up to a certain altitude. More sustained hypoxia exposure further suppresses mitochondrial biogenesis and oxidative metabolism, with profound changes in both intermyofibrillar and subsarcolemmal mitochondria.^{25,26}

Lastly, progress in genomics to identify molecular, physiological, and developmental genetic variants provides an improving knowledge of both adaptive evolution in native highland populations and in nonadaptive responses to hypoxia.²⁷ This limited review of physiology will now give way to the historical part of the essay in roughly chronological order.

Historical observations

The term HAD has been attributed to members of the British Everest expeditions of the 1920s. Whatever the term, the condition was already somewhat recognized. As an example, during 5 months in the Andes in 1880, English explorer Edward Whymper (1840–1911) informally tried to distinguish between the deteriorating effects of high altitude alone vs acclimatization issues and multiple privations such as cold, overwork, and insufficient nutrition. While climbing Chimborazo 6263 m (20,549 ft), Cotopaxi 5897 m (19,347 ft), and numerous other peaks, his team spent 208 nights above 2348 m (8000 ft); 21 of those nights were above 4570 m (15,000 ft) and 12 were above 4877 m

(16,000 ft). At these latter heights they were at risk for HAD. Whymper's comments dwelt mostly on what we call AMS, acclimatization, and individual variation, but he also noted that with prolonged stay the team reached a point of less vigor.²⁸ This remark and the heights and time suggest he may have been referring to at least some degree of HAD.

Whymper had correctly suggested the additional deleterious effects of atmospheric pressure below 55.9 kPa (16.5 in Hg)—that is, above 5079 m (16,664 ft). Later, HAD was suspected in another location in the Andes. In 1935, a team including American physiologists Ancel Keys and DB Dill observed the workforce at near 5950 m (19,520 ft) in sulfur mines in Chile. Miners had abandoned a camp at 5639 m (18,501 ft), preferring to descend to the 5300 m (17,500 ft) town of Aconquilcha. With limitations imposed by HAD and other factors, this altitude was thought to be the highest habitable altitude at that time.^{29–31} There were many observations by others over the years, but it would not be until 1960–1961 that a comprehensive study on altitude physiology was performed by expedition members spending months at high and extreme altitudes during the “Silver Hut” Himalayan Scientific and Mountaineering Expedition.⁴

Exploration of the Greater Ranges in the early 20th century brought outstanding mountaineering literature. Physicians and nonphysicians alike suffered, observed, and wrote memorably about the debilitating effects of long stays at altitude. For example, when the Italian Duke of Abruzzi explored the Karakorum Himalaya in 1909, his chronicler Filippo de Filippi recorded that 2 months above 5334 m (17,500 ft) caused an “evil effect” with decreased appetite, lowered nourishment, reduced vitality, and loss of flesh.³²

At about the same time, descriptions came from British physician-mountaineer Thomas Longstaff (1875–1964), well known for the first ascent of 7120 m (23,359 ft) Trisul in 1907. Longstaff's medical book of 1906 recapped earlier reports of many explorers including Whymper and British Army officer/explorer Charles Granville Bruce (1866–1939) and made a distinction between mountain sickness (today called AMS) and the separate effects of prolonged stays. Longstaff attributed to Bruce the term *mountain lassitude*, defined as “diminution in the strength of a man due to diminished atmospheric pressure. This weakness is progressive, and affects the physical, mental, and will powers.”³³ With symptoms and settings different from mountain sickness, mountain lassitude sounds like HAD. Longstaff reiterated this distinction more than once, noting “loss of appetite and deterioration of the mental powers” and emphasizing with italics that “when climbers have spent many *consecutive* days at pressures of half an atmosphere [5486 m / 18,000 ft] it cannot escape notice that their physical condition seems to have deteriorated.”³³

Up high, the human experience in the early 20th century might have been more understandable if various aspects of high altitude physiology (not just HAD) were more easily disseminated in the medical literature. There were 3 examples. First, and directly addressing HAD, was one of the most experienced high altitude adventurers. Scottish chemistry lecturer Alexander Kellas (1868–1921) unfortunately perished on the approach to Everest in 1921. Parts of his manuscript were published in French but lay dormant in archives for years until rediscovered by West and published in English in 2001.³⁴ Kellas was referring to HAD when he described a loss of vitality that was more than mountain sickness: “‘Mountain lassitude’ has a variable effect above the limits of permanent acclimatisation [5182 to 6096 m] (17,000 to 20,000 ft).”³⁴ This, indeed, was HAD.

A second clue was about a manifestation of prolonged stay that was not HAD. It was known in the veterinary literature but less so in the human medical literature. In Colorado, George Glover and Isaac Newsom described brisket disease.³⁵ Right ventricular syndromes can occur at relatively low elevations in cattle (brisket disease) or at higher elevations in humans and can occur with subacute or chronic exposure to low atmospheric pressure and hypoxia. At altitude, there are only mild effects on the cardiac muscle of the left ventricle, but because of hypoxic pulmonary vasoconstriction there can be pronounced effects on the right ventricle (RV).³⁶ In the early 20th century, this effect on the human RV was not well understood and was called by the medical term “dilated heart.”¹³ A third example was also different from HAD. Like RV failure in brisket disease, RV failure also occurs in humans with chronic mountain sickness, but Peruvian physician Carlos Monge's 1928 *The Disease of the Andes* was written in Spanish.³⁷

No British mountaineer is more associated with Everest than George Leigh Mallory (1886–1924) with his astute observations and beautiful prose but untimely demise. The 3 expeditions to Everest in the 1920s were fraught with new dangers. For one example, a peculiar form of heat-related illness at altitude called “glacier lassitude” was coined by Mallory.¹² For another example, extended time and loss of conditioning above 6096 m (20,000 ft) prompted him to correctly surmise: “It is difficult to account for this deterioration, unless we suppose that altitude, though it may have no immediate effect, takes its toll at length.”³⁸ He rightly concluded that “The whole machine in fact, was running down.”³⁹ On the second expedition and climbing much higher, Mallory said there was only one solution: “At a high altitude even the strongest might suffer this loss of muscular power; and *he will not recover up there.*”⁴⁰ This was HAD.

Other team members in 1922 included surgeon T. Howard Somervell (1890–1975) and medical officer Longstaff. Bruce, the leader of the expedition, described Somervell as “absolutely untireable.”⁴¹ Full of energy or not, he, like Mallory, cautioned against overexertion at extreme altitude for fear of prolonging their recovery. Somervell also recalled that with loss of appetite, change in mental condition, and loss of determination that, “approaching [8230 m] 27,000 ft I remember distinctly that I cared very little whether we reached the top of Everest or not” and that some of the team “actually seemed to deteriorate in condition while staying at great height.”⁴² According to Ward of the 1951 and 1953 expeditions, Bruce in 1922 attributed to Somervell the term “high altitude deterioration.”^{43,44} In the official book on the 1922 expedition, with the term deterioration mentioned in sections by Bruce, Mallory, Somervell, and others, it is difficult to say which one of them “invented” the now widely used phrase.

Somervell returned on the 1924 Everest expedition along with Irish physician RWG Hingston (1887–1966). Hingston was the medical officer and, despite his inexperience, went as high as 7000 m (22,970 ft) at Camp IV and helped rescue snow blind leader Teddy Norton after he and Somervell nearly died in their summit attempt. In Norton’s *The Fight for Everest 1924*, Hingston recognized the competing forces, advising that “while acclimatisation is in progress there may be physical deterioration at the same time.” He also saw “after-effects consequent on residence at the high camps. . . . All were debilitated. All had wasted.”⁴⁵ So great was the wasting that climber Bentley Beetham in the chapter “The Return Journey” colorfully illuminated that “The last reserves had been used up long ago, and our limbs would have moved a cannibal chief to tears.”⁴⁶ They had all recognized the competing forces of acclimatization and deterioration. More than a half century would pass for it to be given a familiar catchphrase.

Echoing the 1920s, there were 4 official British attempts on Everest in the 1930s; other teams, including the Germans, attempted other 8000 m (26,247 ft) peaks, Kanchenjunga and Nanga Parbat. By 1933, partial acclimatization was thought possible perhaps up to 7010 m (23,000 ft), but higher up deterioration was even more rapid.^{47–49} The 3 concerns were about getting there in good health, the balance between acclimatization and deterioration, and the inevitable decline. Physician-mountaineer and raconteur C. Raymond Greene (1901–1982) on Kamet warned about first things first: “At this height, [7010 m] 23,000 ft, you start tired out.”⁵⁰ On Everest 3 times in the decade, famed explorer Eric Shipton (1907–1977) took it a step further: “At high altitudes (in my opinion above [6400 m] 21,000 ft) the wastage of muscle tissue is so rapid that it is well to start with a fairly large reserve of flesh.”⁵¹ Mountaineer

and wit Frank Smythe (1900–1949) said the same, adding that “The effect of prolonged residence at high altitudes—we had spent a month above [6400 m] 21,000 ft—had been to reduce not only flesh but muscle.”⁵² So pronounced was this impact on stamina and reserve that Shipton proclaimed, just like Mallory, that “‘Desperate efforts’ are not made above [6705 m] 22,000 ft without great exhaustion, from which it is not possible wholly to recover without a prolonged rest at a very much lower altitude.”⁵¹

In 1933, Everest leader Hugh Ruttledge (1884–1961) was well aware of the problem: “There is no doubt that the long residence at Camp IV [6950 m (22,800 ft)] and above allowed deterioration to set in. . . the limit of beneficial acclimatisation had been passed.”⁴⁸ Shipton summarized the problem nicely: “The real bone of contention was how long climbers could stay at high altitudes. The opposing factors were ‘Acclimatisation’ and ‘Deterioration,’ and as we had very little data from which to argue the debate waxed exceedingly fierce. . . . These two processes are going on at one and the same time, and the problem is to strike the optimum mean between them.”⁵¹ It was like the proverbial math formula of the bathtub filling and draining, acclimatization peaking while deterioration was advancing.

The use of the term “wasting” continued. Here is Ruttledge: “a great deal of wasting occurs as a result of prolonged residence at high altitudes.”⁴⁸ Then Shipton expands on the subject: “‘High-altitude deterioration,’ a kind of creeping paralysis of our limbs and faculties, was by far the most unpleasant manifestation of life above the North Col. It was like a wasting disease, causing rapid degeneration of our muscles and progressive weakness and lethargy.”⁵³ Shipton, with a nod to the tactics of the future, concluded that “in 1933 too much emphasis was laid upon acclimatisation. . . . the whole policy had been one of slow advance. When we returned to Base Camp we were terribly emaciated. It was a standing joke that we looked like a collection of famine-stricken refugees.”⁵¹ Finally, there was Greene reaffirming Beetham from 9 years before: “I took off my clothes for the first time in six weeks and had my first bath. I was horrified by the wasted muscles.”⁵⁰

In 1950 on Annapurna IV, HW Tilman (1898–1977) complained like Mallory that with lengthy stays “Contrary to expectation the machine begins to run down.”⁵⁴ The 1953 Everest leader, John Hunt (1910–1998), cautioned that “There was a great urge to do nothing—the danger sign of deterioration.”⁵⁵ Ward, doing physiological work on Everest in 1953 with Griffith Pugh (1909–1994), would write in 1954 the definition used in this essay’s introduction and other articles with Pugh later.^{47,56} In 1953, mental and physical decline was noticed not only on Everest but also on K2 by American physician-mountaineer Charles Houston (1913–2009). His team had spent nights well above

5060 m (16,600 ft) for a very lengthy 68 days, and of those 45 were above 5852 m (19,200 ft). While at their 10-day stormbound maximum height of 7772 m (25,500 ft), they had been far above the threshold for HAD for a long time, prompting Houston to write about those last camps: “Apathy grows strong on high peaks, the body become feeble, the force to advance declines.”⁵⁷ As a protagonist in the investigation of high altitude, Houston would later add a famous phrase: “deterioration outstrips acclimatization.”⁵⁸

Most celebrated for his climb with Tensing Norgay to the top of Everest in 1953, Ed Hillary was climbing leader at the Silver Hut in 1960–1961. Hillary admitted that “I had always expected Makalu to be the final testing ground of my acclimatization theories and believed that the long periods my men spent at [5790 m] 19,000 ft and above would make them so adjusted to living in rare atmospheres that Makalu would not be too formidable an objective for them even without the use of oxygen. . . . But now I realize that. . . these long periods at high altitudes were possibly our undoing.”⁵⁹ Science leader Pugh and his team, including Ward, Milledge, and West, had already suspected this. They knew from Keys and Dill that Andean miners could only stay that high for so long.^{29,30} Pugh concluded from the Silver Hut that “[5790 m] 19,000 ft was too high for complete adjustment [to allow permanent habitation] and that [5330 m] 17,000 ft, or in some cases [4570 m] 15,000 ft, would be nearer the limit.”⁴ When they did leave the Silver Hut to go higher on Makalu, the team collapsed at 8351 m (27,400 ft). One climber reported that only after being rescued by West with supplemental oxygen were they able to “realize how badly we had all deteriorated.”⁶⁰

In 1978 Italian Reinhold Messner (1944–) and Austrian Peter Habeler (1942–) became the first persons to successfully ascend Everest without supplemental oxygen. Later in 1980 Messner repeated the feat solo from the north side. These record-shattering ascents ushered in a new era for a select few, culminating in other “oxygen-less” ascents and speed climbs never thought possible.

Although the scientific community uses some elaborate words, the conclusion of this essay on HAD can be reduced to simplicity from the mountaineer and physician-mountaineer literature. British climber Pete Boardman exclaimed in *Sacred Summits* that “We had left a lot of muscle on the mountain.”⁶¹ The last clinical declaration comes from Houston. With this sentence, worth repeating, his words remained forever in the medical lexicon: “Above [5791 m] 19,000 ft, however, deterioration outstrips acclimatization.”⁵⁸

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