Breathing at extreme altitudes. Scientific projects "EVEREST" (Second part)

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Summary

Climbing to the highest height on Earth, the Mt. Everest (8.848 m), without supplementary oxygen equipment involves a physiological demand that is close to the maximum human tolerance. Exposures at extreme altitudes drastically conditions lung function, stores of oxygen and physical performance. This review brings interesting aspects about respiration, blood gases and aerobic exercise reported by those scientific projects that have carried out physiological measurements between 8,000 m and 8,848 m above sea level, under real or simulated altitude: the Operations "Everest I" (1946), "Everest II" (1985), "Everest III-COMEX" (1997), and the Expeditions "AMREE" (1981), "British 40th Anniversary Everest" (1993), and "Caudwell Xtrem Everest" (2007). These fascinating scientific research events, along with other outstanding biomedical expeditions performed above 5,500 m, like especially the "Silver Hut" (1960-61), "Italiana all'Everest" (1973), and "British Everest Medical" (1994), including those pioneer scientific reports made on the XIX century until the most recent research projects performed, have laid the foundations and knowledge on the human tolerance to such extreme levels of hypobaric hypoxia, where the lung, breathing and respiratory chain takes on a major role requiring fine physiological adjustments to ensure cellular oxygenation. Geophysical aspects, climatic factors and other environmental conditions that limit the biological viability and can affect the respiratory health of climbers on the upper troposphere zone at the subtropical latitude where that mountain is located are likewise reviewed and analyzed. Every year, hundreds of climbers try to reach the top of Mt. Everest, but only a few of them achieved their goal without inhaling supplemental oxygen, including some exceptionally gifted Sherpa natives, protagonist on unsuspected exploits in the highest mountain on terrestrial surface, whose summit touch the physiological limit of survival for the human being.

Key words:
Altitude. Oxygen uptake. Hypoxia. Mountaineering. Atmospheric pressure. Respiration.

Respirar en altitudes extremas. Proyectos científicos "EVEREST" (Segunda parte)

Resumen

Escalar el punto más alto de la Tierra, el Mt. Everest (8.848 m), sin equipos de oxígeno conlleva una demanda fisiológica que está próxima a la máxima capacidad de tolerancia humana. Exponerse a altitudes extremas condiciona drásticamente la función pulmonar, el nivel de oxígeno y el rendimiento físico. Esta revisión reúne interesantes aspectos respiratorios, de gases sanguíneos y ejercicio aeróbico aportados por aquellos proyectos científicos que han llevado a cabo mediciones fisiológicas entre 8.000 m y 8.848 m, en altitud real o simulada, como las Operaciones "Everest I" (1946), "Everest II" (1985) y "Everest III-COMEX" (1997), y las Expediciones "AMREE" (1981), "British 40th Anniversary Everest" (1993) y "Caudwell Xtrem Everest" (2007). Estos fascinantes eventos de investigación, junto a otros destacados proyectos biomédicos realizados a más de 5.500 m, muy especialmente las Expediciones "Silver Hut" (1960-61), "Italiana all'Everest" (1973) y "British Everest Medical" (1994), incluyendo aquellas pioneras observaciones científicas llevadas a cabo en el s.XIX hasta los más recientes proyectos de investigación realizados, han sentado las bases del conocimiento sobre la tolerancia humana ante niveles de hipoxia hipobárica extrema, donde el pulmón y la cadena respiratoria adquieren un trascendente protagonismo requiriéndose de finos ajustes fisiológicos que garanticen la oxigenación celular. Asimismo, se exponen ciertos aspectos geofísicos, factores climáticos y otros condicionantes ambientales que limitan la viabilidad biológica y pueden afectar la salud respiratoria de los alpinistas situados en las cotas superiores de la troposfera a la latitud subtropical donde se encuentra ubicada dicha montaña. Actualmente cientos de alpinistas intentan alcanzar la cumbre del Mt. Everest todos los años, pero solo algunos consiguen su objetivo sin inhalar oxígeno suplementario, entre ellos algunos excepcionalmente dotados nativos Sherpa, protagonistas de insospechadas hazañas en la montaña más elevada de la superficie terrestre, cuya cima roza el límite fisiológico de supervivencia para el ser humano.

Palabras clave:
Altitud. Consumo de oxígeno. Hipoxia. Montañismo. Presión atmosférica. Respiración.

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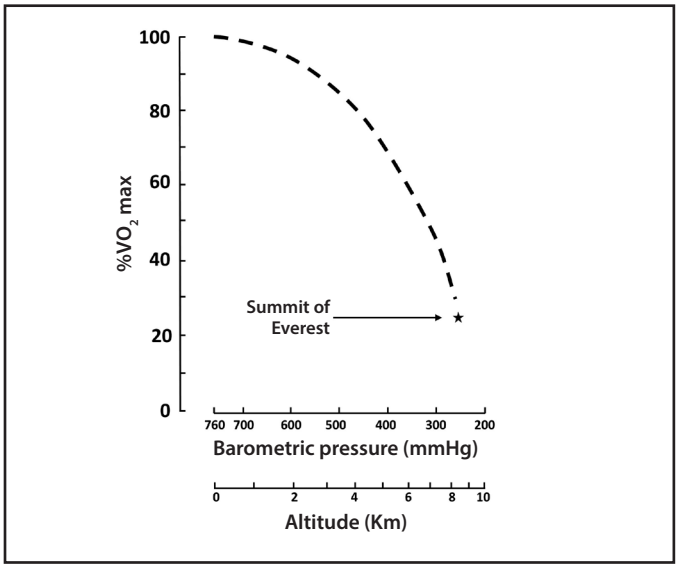
Maximum oxygen transportation and uptake faced with extreme levels of hypoxia

Physiological adaptations that occur when faced with a very low oxygen blood transportation must ensure tissue homeostasis, even during physical exercise, as this situation drastically reduces SaO_2 at extreme altitudes[31,48,63-65]. Blood samples taken at 8,400 m, descending from the summit of Everest, reveal average SaO_2 values of ~55%, with a figure below 35% detected in one of the subjects studied[36]. During maximum physical exercises, SaO_2 measurement below 50% were obtained at an altitude of 5,800 m[66], as well as at 6,300 m with FiO_2 of 14% (PiO_2 ~43 mmHg), simulating the 8,848 m of Mount Everest[48], even SaO_2 below 40% with identical PiO_2 but in a hypobaric chamber[39].

Hypoxia causes an exponential reduction in maximum aerobic power. At 7,000 m the maximum oxygen uptake (VO_2max) is reduced by ~60%[14] and at 8,848 m by 70-80% compared to sea level[39,48,67] (Figure 2). Mountaineers at an altitude that simulates the summit of this mountain have proven to develop a VO_2max ~1—1,2 L•min^{-1} in diverse studies[31,39,48,68], and some subject display slightly lower values[39,69]. Despite this, cell function must be guaranteed, via a capillary PO_2 that must be kept above 15 mmHg[35]. The physiological explanation behind this major reduction of the organic availability of oxygen is complex[67], and it seems to radiate mainly in the diffusive limitations of gases both in terms of the lungs (alveolar/capillary) and the muscles (capillary/mitochondrial), as the ventilatory and cardiac systolic functions have a lesser influence[70-72]. Two factors are key in terms of the lungs: on the one hand the effect of an interstitial oedema (initially sub-clinical) induced by the hypoxic vasoconstriction and the subsequent PHT, as mentioned before, and on the other hand the reduction of the blood transit time through the alveolar capillary bed, which is due to the increase of the cardiac output (parallel to that of the systemic circuit) and the hypoxic vasoconstriction itself, which both cause a marked increase in the flow speed of the pulmonary micro-circulation. As a result of this insufficient transit time, the haemoglobin does not reach its optimum balance when faced with very low levels of PAO_2 and the blood leaves the lung with a more reduced SaO_2 than expected. However, it is estimated that mountaineers usually ascend over 6,000 m of altitude at a physical exertion intensity of 50-75% of their VO_2max[73,74], though during the climb of the last stretch of the final pyramid of Everest, it is feasible that this ascends to 85-90% of the VO_2max[75,76].

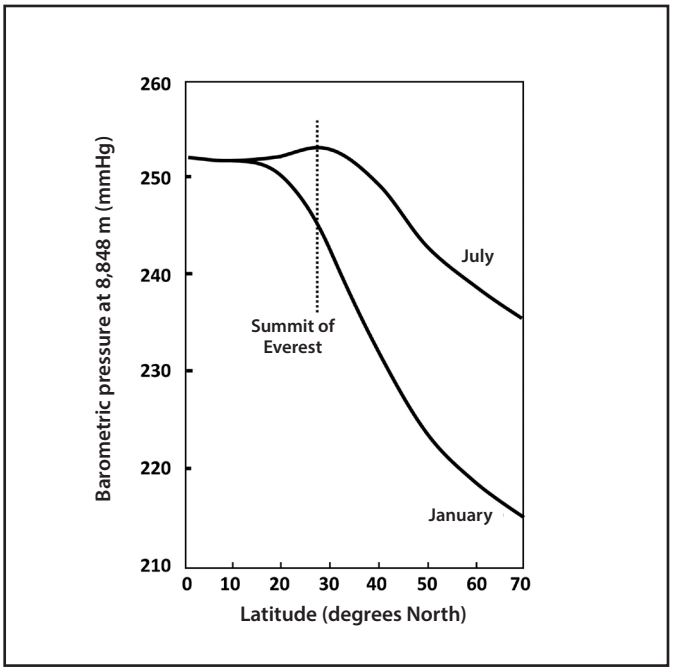
Figure 2. Percentage of maximum oxygen uptake depending on altitude and barometric pressure. Average value obtained by different authors (Based on Richalet and Herry[45]).



Assuming that 1 MET (basal metabolism) is equivalent to 3.5 mL•min^{-1}•kg^{-1}, it has been calculated that remaining at the summit of Mt. Everest without performing physical exercise requires a minimum metabolic requirement of 1.4 METs (~5 mL•min^{-1}•kg^{-1} or ~350 mL•min^{-1})[32,77]. The most important example of this being possible was the mountaineer Babu Chiri Sherpa, who in May 1999 remained on the summit of Everest for 21 hours straight without breathing supplemental oxygen. However, in order to climb to that point in summer without oxygen masks, taking into account an average VO_2max ~15.3 mL•min^{-1}•kg^{-1} with a PiO_2 equivalent to 8,848 m[31,39,48] as well as the linear relationship PiO_2-VO_2max[18,31,48] and the extremely slow ascent pace performed by Messner over the last 100 m (2 m•min^{-1}; body weight plus equipment ~150 kgm•min^{-1}), it is estimated that a minimum functional reserve of 3.5 METs (VO_2max ~12.3 mL•min^{-1}•kg^{-1}) is required at an exertion intensity of 85% of VO_2max, i.e. 3 METs (VO_2 ~10.5 mL•min^{-1}•kg^{-1})[76]. Therefore, taking into account the percentage of aerobic reduction at the summit of Everest, a VO_2max between 49 and 61 mL•min^{-1}•kg^{-1} at sea level[39,48] was proposed as a minimum metabolic requirement, climbing to ~90% of the VO_2max[75], although these values being below those previously estimated[68,78]. In fact, the first people to climb Mt. Everest that did not use supplemental oxygen showed a VO_2max of 49—66 mL•min^{-1}•kg^{-1} at low altitude[79]. In general, Caucasian mountaineers that ascend between 4,500—8,848 m reveal average VO_2max values of ~51—61 mL•min^{-1}•kg^{-1}, (range ~43—67 mL•min^{-1}•kg^{-1}) at low altitude[75,80,81]. Above 6,000 m there is a direct correlation between the maximum altitude reached and VO_2max, as the higher the value, the more guarantees there are that the climb to extreme altitudes will be successful; and not just this, it is also shown to be a physiological safety parameter for climbing heights over 7,500 m[75]. Various genetic polymorphisms are associated with a higher aerobic performance at high altitude, among which it is worth highlighting the angiotensin-converting enzyme (allele ACE-I)[82], though scientific data is still very scarce to claim sufficient proof in this respect.

The average BP on the summit of this mountain in the times of year when it is usually climbed (May and October) is 251—253 mmHg[33]. However, the BP undergoes minor daytime variations and marked seasonal oscillations[62,83], with these variations having a significant physiological transcendence on physical performance, especially on

Figure 3. Barometric pressure variation at the altitude of Mount Everest (8,848 m) in relation to different times of year and geographical latitudes of the Northern hemisphere (Based on West *et al.*[34,62]).



VO_2max[33,34,77]. Therefore, the success rate for climbing Everest without supplementary oxygen will depend largely on the BP prevailing on the day the summit is climbed. As Figure 3 shows, the BP is at its highest during the months of July and August (~255 mmHg), therefore, the VO_2max will be higher and the mountain ascent will be more physiologically accessible; the BP is at its lowest in January and February (~243 mmHg) and this is when the greatest reductions of VO_2max occur, approximately 10—12% compared to summer levels[33,62,84]. The current record of the number of ascents to Mount Everest is held by three Sherpas: Appa, Phurba Tashi and Kami Rita, with 21 ascents each. However, the Sherpa Ang Rita — known as the "snow leopard" because of his exceptional physical performance at extreme altitudes — is the only human being to ever scale this mountain on 10 separate occasions without inhaling supplemental oxygen, and is the only person ever to have performed this ascent in this way in winter. A few years after achieving this feat, performed on 22nd December 1987, we detected that his VO_2max was 66.7 $mL \cdot min^{-1} \cdot kg^{-1}$ at sea level[85], a value that is significantly higher than that measured in other internationally renowned Sherpas[86]. It is known that a higher VO_2max at sea level entails a greater VO_2max at high altitude[39], a fact that enabled Ang Rita to scale the summit of Everest in winter without oxygen equipment, despite the drastic reduction of ~80% which his VO_2max presumably experienced at the altitude of 8,848 m. A VO_2max ~13.3 $mL \cdot min^{-1} \cdot kg^{-1}$ would align with the minimum value needed of 3.9 METs, which has been estimated to complete the ascent under these conditions[76]. On the day that Ang Rita achieved this winter climb, the BP was 247 mmHg at the same altitude and latitude of Mount Everest, i.e. 9 mmHg less than during the ascent performed by Messner on 20th August 1980, but 4 mmHg higher than the levels registered on the summit during mid winter, a fact that was decisive in the success of the sporting achievement accomplished by this sherpa[84].

Despite minimum BP changes entailing very significant variations of VO_2max at such extreme altitudes, it is worth highlighting that other advantageous factors associated with the chronic hypoxic adaptation shown by people from Tibetan lineage also influence their best physical performance at such high altitudes. These natives present a lower reduction of maximum heart rate compared to levels observed in Caucasians; a larger surface of pulmonary gas exchange and alveolar-capillary diffusion capacity; minimal PHT and right ventricular overload; greater HVR and a lack of, or lower rate of central apnoea and Cheyne-Stokes-type respiratory rhythms during sleep; less erythrocytosis and blood viscosity with higher SaO_2 values[87-96]. These adaptive mechanisms found in Tibetans, from whom phylogenetically the Sherpas descended[97], are the best anthropologic example of adaptation to altitude, because their permanent exposure to environmental hypoxia originates from the Upper Palaeolithic Era, surpassing the Andean highlander natives by thousands of years[89,96]. More than ten genes have been described that are involved in their extraordinary adaptive response to high altitude, though a few of them specially related to blood-oxygen transportation, seem to play a particularly important role: allotype EPAS1 (HIF-2α) and allotype PHD2/EGLN1, its negative regulator[98-100]. A higher frequency of allele ACE-I has also been reported in Sherpas[101].

In general, aside from climatic aspects, three physiological mechanisms are particularly decisive in being able to climb to 8,848 m of altitude without oxygen equipments: the extreme hyperventilation generated reduces the $PACO_2$ stabilising the PAO_2, the acute hypocapnic alkalosis facilitates the oxyhaemoglobin saturation in the lungs, and the BP increased, despite being minimal, significantly raises the VO_2max[48]. Despite these extraordinary adaptations, which enable survival at such extremely high, human beings have limiting physiological factors in terms of physical performance in this environment, despite this being a controverted aspect. Within this limitation, BP has a relevant role to play, as well as the energy expenditure of hyperventilation, the gas diffusion capacity through the alveolar-capillary membrane, the peripheral PO_2 and the transfer of oxygen to active muscles[67,71,102,103]. Moreover, the on-going sympathetic stimulus caused by chronic hypoxia inhibits the maximum chronotropic response, a fact that has been fundamentally linked to a progressive desensitisation of the beta-adrenergic myocardial receptors[104]. Recently, particular emphasis has been placed on the sporting success of the two mountaineering pioneers – Messner and Habeler - who reached the summit of Mount Everest in 1978 without supplemental oxygen. Whilst they did not have exceptional VO_2max at low altitude[76], they would have undergone transcendental physiological changes in the increase of their muscular capillary density, and therefore, an optimum periphery diffusion of oxygen during that historical ascent[105]. While VO_2max is drastically reduced at extreme altitudes, the anaerobic capacity of the skeletal-muscle paradoxically reveals also a great reduction when faced with chronic exposure to hypoxia[106]. This is due to the major depletion of plasma bicarbonate upon compensating the respiratory alkalosis, with minimal increments of lactate being detected when faced with intense physical exertions in altitudes over 7,500 m[31,102].

Geophysical aspects, extreme climatic and environmental factors on aerobic performance and the health of mountaineers

If we take the standard atmosphere model of BP as a reference, at the summit of Everest there would be a value of 236 mmHg, about 17 mmHg less compared to real atmospheric measurements taken in springtime, and this would cause a reduction of the PiO_2 from 43 mmHg to 39.5 mmHg[33]. Because the logPiO_2—VO_2max relationship is very pronounced (~63 mL•min^{-1}•mmHg^{-1}), this loss of just 3.5 mmHg would produce a reduction of the VO_2max of ~222 mL•min^{-1}, i.e. adding ~21% more reduction to the altitude of 8,848 m, with a viable ascent to the summit of this mountain under these conditions seeming unlikely, as calculated by West *et al*.[33]. A BP of 236 mmHg in a real atmosphere is equivalent to an approximate altitude of ~9,350 m at the latitude where this mountain is located, in other words, half a kilometre higher than Mount Everest. Due to the contraction shown by the troposphere towards the Earth's polar regions, this BP at this time of year would correspond to Everest being geographically located on a more northerly latitude. Therefore, despite the extreme hypobaric hypoxia that prevails at the summit of Everest, given its subtropical position (28ºN), mountaineers are favoured by the equatorial dilation of the troposphere[34,62]. As shown in Figure 3, the BP at around 8,850 m during mid-winter in the Artic polar circle (66ºN) reaches ~214 mmHg[34], i.e. this mountain would have a simulated elevation of a thousand meters higher.

Bailey calculated that where Mount Everest was located, and according to the BP that prevailed there at different times of year, an altitude of ~9,970 m could be the theoretical limit where the PiO_2 would ensure a VO_2max of 3.5 METs in summer, as well as ~9,660 m of 3.9 METs in winter, and at the altitude of ~11,900 m it would equal 1 MET[76]. *A priori*, the first and second figures would exceed already exposed estimations, and the third would exceed the experiment performed by Angelo Mosso, who simulated an altitude of 11,650 m in a hypobaric chamber. He reached a BP of 192 mmHg, equivalent to ~10,800 m of standard atmosphere[107], though taking into account the slightly oxygen-enriched mixture that he inhaled during this experiment (FiO_2: 29.2%)[25], it would have given him a PiO_2 corresponding to ~8,850 m, similar to the summit of Everest. It is worth mentioning that during the final phase of Operation Everest I, a simulated altitude of ~15,400 m was achieved whilst acclimatised subjects breathed 100% oxygen, with these subjects revealing a certain degree of momentary tolerance to this atmosphere. Non-acclimatised flight pilots do not usually tolerate altitudes of 13,000 m, despite inhaling supplemental oxygen[38]. In fact, being exposed to altitudes over ~12,000 m, even while resting and breathing maximum concentration oxygen, the SaO_2 drops drastically, and does not guarantee the cell viability of the organism, therefore pressurised equipment is needed[108].

The Himalayan mountain range rises slowly but constantly, due to the compression between the tectonic plates of India and Eurasia. Due to this fact, it is intriguing to consider speculations regarding the amount of time that it will be possible to ascend Mount Everest without using oxygen equipment. Considering that the annual elevation rate is ~3 cm, Bailey calculated that it will not be until approximately the year 29,000 and 39,000 - in winter and summer respectively - when Everest will reach an altitude at which the PiO_2 will ensure the physiological limit that is compatible with developing a VO_2max ~3.5 METs at its summit, and the year ~104,000 when it would equal ~1 MET[76]. However, it is clear that the precise future behaviour of continental platforms is unknown, as well as the exact characteristics of the troposphere in thousands of year's time, due to the effect of global warming or climate changes that modify the composition of atmospheric gases. While there are numerous speculations regarding the effect of global warming on the Himalayan region, certain measurements reveal that since 1948 the troposphere has been increasing its pressure on average by ~1,8 mmHg each decade, therefore, in keeping with this trend it could be possible that progressively developing a greater VO_2max on the summit of Mount Everest may be feasible, and climbing it may become more accessible aerobically in the future. However, within this environmental context, mountaineers will have to face new technical difficulties caused by the thermal increase, such as the risk of potential avalanches and rock-climbing sections caused by the disappearance of the ice on a large part of the routes[109].

The wind that strikes the edges and walls of this great geological formation could reduce the BP due to the Venturi effect, though calculations using the Bernoulli equation reveal that local BP changes would be minimal, below 1 mmHg, for which it would have a very minor physiological impact on the VO_2max[34]. This is particularly true before and after monsoons, when there is greater meteorological stability, and winds do not usually exceed 55 km•h^{-1}, a time that is chosen to attempt the ascent with the highest possible chance of success and safety[83]. However, during these brief anti-cyclonical windows of opportunity occurring in May and October, sudden gales occur, and the high layer of the troposphere can even be fed with great masses of subtropical water vapour which interact with the stratospheric jet stream current, causing convective vortexes with strong hurricane-force storms over Everest[110]. During winter, this jet-stream current moves towards southern Central Asia, completely covering Himalayan mountain range, and cyclonic winds with speeds reaching around 300 km•h^{-1} can hit the peaks of over 8,000 m of altitude. Days with strong gales are avoided by mountaineers given the high risk of accidents that these conditions entail, as well as the chill factor associated with low temperatures that the wind has on the body. On the summit of Mount Everest, temperature of minus 60˚C can be reached when masses of Siberian polar air penetrate the Tibetan plateau, though during the climatic windows in May and October, the average wind temperature is usually around minus 26˚C[111], though the chill factor can give values below minus 50˚C[83], with warmer temperatures more usual throughout the day during these periods[34]. The atmospheric factors largely explain why only 25% of mountaineers that attempt to climb Everest in spring or autumn actually manage to reach the summit, and why this success rate drops to just 4% of those that attempt it in winter[112], though the first percentage seems to have risen in recent years.

Breathing low-temperature air increases the risk of suffering from a pulmonary pathology, and at extreme altitudes upper respiratory tract problems are frequent, which improve upon descent[113,114]. The low relative humidity in the high layers of the troposphere cause a large loss of water vapour through the lungs, exceeding 60—80 $mL \cdot h^{-1}$ during moderate-intensity physical exercise[62]. The nasal inspiratory flow increases with altitude, but at 8,000 m it is lower than expected in accordance with the prevailing low air density[115]. Episodes of bronchospasm are infrequent at high altitude, especially among well-controlled asthma suffers[116], as atmospheric pollution reduces in high mountain regions and this environment has been shown to be clearly beneficial to these patients[117]; even exposures at extreme altitude appear to be safe in cases of moderate-intensity asthma sufferers[118]. With the exception of bronchospasm episodes that are not directly related to pollen allergens (breathing cold and dry air, vigorous physical activity), it has not been proven that bronchial spasm episodes are favoured, specifically, by the hypoxia and hypocapnia of altitude, given that the oxygen supplement does not seem to increase ventilatory flows at extreme altitudes[59]. The stratospheric ozone, whose existence has been detected at a level very close to Mount Everest, represents a potential added health-risk for mountaineers, as it causes the inflammation of the mucous membranes of the airways, bronchoconstriction, coughing and/or dyspnoea, deteriorating pulmonary function[62,119,120]. The upper respiratory tracts quickly re-heat low-temperature air inhaled, for which it reaches optimum thermal graduation at the level of the alveolar, clearly not constituting a reason to support high-altitude pulmonary oedema (HAPE), secondary non-cardiogenic oedema to PHT, already relatively frequent at heights below 5,000 m[72,121-125]. It is difficult to estimate the HAPE rate at altitudes over 8,000 m, but the presence of plasma at interstitial level caused by subclinical HAPE would be frequent, which would alter the gas exchange[69,126], as observed in athletes at much lower altitude[127-131]. Even with no oedema, the low gradient alveolar-capillary oxygen pressure (because of the reduction of BP and excessive pulmonary blood transit) increases the degree of hypoxemia especially during physical activity[131], an exercise capacity that is already limited by the PHT[132].

Aside from the hypoxemia caused by a HAPE, just breathing in an oxygen-deprived atmosphere causes deterioration to the central nervous system and the development of cerebral oedemas that could cause an accident on the mountain, or cause the mountaineer to lose consciousness[10]. Neuropsychological alterations are frequent[133,134], some are transitory and are associated with a focal reduction of cerebral blood flow caused by extreme hypocapnia[135]. Paradoxically, subjects with a higher HVR - therefore a greater PaO_2 and better adaption to hypoxia – suffer from more residual cognitive dysfunctions, possibly due to the reduction of blood flow caused by very low levels of $PaCO_2$[136]. The possibility of permanent encephalic hypoxic damage was suggested in Himalayan mountaineers[137] and although the exact mechanism of brain damage in these subjects has not been well clarified[138-141], the fact that Sherpas appear to have certain neurological protection against extreme altitudes is very suggestive[142].

Conclusions

Since the pioneering scientific experiments from the 19th century, which used hypobaric chambers to attempt to establish if a "hypoxia"[26] or "acapnia"[25] respiratory situation was the result of certain limitations and pathological disorders that appear in high mountain areas, numerous later studies, especially the fascinating projects carried out in the Mount Everest region or simulating its altitude have established the basic concepts on the respiratory response mechanisms in humans exposed to this extreme environmental setting. Pulmonary adaptation to severe levels of hypoxia, especially during physical exercise, is astonishing, yet the diffusion capacity of gas exchange at an alveolar-capillary level seems to play a determining role as a limiting factor of oxygen availability in the body. However, evidence reveals that pulmonary respiration can guarantee cell respiration, even faced with such levels of reduced partial pressure of oxygen, and on the summit of Mount Everest these levels are very close to the limit of human physiological tolerance. The subtropical geographic latitude upon which the Himalayas are found enable an ascent up to the highest point without inhaling supplemental oxygen, due to the geoid effect or equatorial convexity of the troposphere, a fact labelled "cosmic coincidence"[143] by the prestigious physiologist John West. Even so, three physiological mechanisms will be particularly decisive in managing to reach the 8,848 m of altitude in this way: extreme hyperventilation, accused respiratory alkalosis, and favourable atmospheric pressure; factors that facilitate and increase the saturation, transportation and oxygen uptake during the day that the climb is attempted.

Some respiratory pathologies, such as asthma episodes or HAPE, must always be contemplated when at high altitude, as they may evolve seriously, and they may be difficult to control in such a hostile environment, potentially endangering lives. Breathing hypoxic air frequently entails the appearance of neuropsychological disorders, with these being one of the main causes behind the accident rate at extreme altitudes. Despite the risks involved in attempting to climb the colossal pyramid of rock and ice that forms Mount Everest, its inhospitable summit is a coveted goal year after year for hundreds of mountaineers from all over the world. Achieving such a major challenge without any supplementary breathing equipment is only possible for very few people, even for natives with thousands of years of adaptation to high altitude, such as those from the Tibetan lineage, with their exceptional genetics and physiological capacity to endure environmental hypoxia.

The testimony of the mythical mountaineer Reinhold Messner is highly compelling, as he describes the sensations he experienced when climbing the final metres of Mount Everest, becoming, alongside Peter Habeler, the first to climb it without using oxygen equipment: "*…I have the feeling I am about to burst apart. As we get higher, it becomes necessary to lie down to recover our breath… Breathing becomes such a strenuous business that we scarcely have strength felt to keep moving forward… I am nothing more than a single, narrow, gasping lung, floating over the mists and the summits*"[144]. Undoubtedly, overcoming the onslaught of the forces of nature inherent to the highest point on Earth represents a first-degree physiological challenge, a dangerous sporting feat that takes place on a colossal backdrop far from the public applause.

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