Breathless and dying on Mount Everest

Published **Online** August 16, 2019 http://dx.doi.org/10.1016/S2213-2600(19)30281-4

This online publication has been corrected. The corrected version first appeared at thelancet.com/respiratory on August 21, 2019

For more on **arterial blood gases and oxygen content in climbers on Mt Everest** see *N Engl J Med* 2009; **360:** 140–49

For more on **The Himalayan Database** see https://www.himalayandatabase.com/

For more on the **causes of mortality on Mt Everest** see *BMJ* 2008; **337:** a2654

Abrupt exposure to the enormous 8848 m altitude of Mount Everest would cause loss of consciousness in less than 3 min, but a period of several weeks of progressive acclimatisation allows some humans to survive at such altitude. The functional limits of the body at such extreme hypoxia, especially in relation to lung function, alveolar and blood gases, or oxygen uptake, were established by the *Operation Everest I*, *II*, and *III-Comex* studies, in 1946, 1985, and 1997, respectively, which were done with the use of hypobaric chambers, and by the *American Medical Research Expedition to Everest* in 1981 and the British *Caudwell Xtreme Everest Expedition* in 2007, both carried out on Mt Everest itself. During the weather window that occurs on the Himalayas in mid-spring, when Mt Everest is usually climbed with a greater guarantee of success, the average barometric pressure on the summit is about 33·6 kPa and leads to a partial pressure of inspired oxygen (PiO_2) of 5·7 kPa. Just below the summit, an astonishingly low PaO_2 of about 3·3 kPa and an oxygen saturation (SaO_2) of about 55% can be reached, whereas during vigorous exercise when simulating 8848 m of altitude in a hypobaric chamber or with low inspired oxygen mixtures, some individuals do not achieve an SaO_2 higher than 40%. However, climbers do not usually exert maximum physical effort during the final ascent to the summit. Those who do not inhale supplemental oxygen maintain very low levels of SaO_2 during their advance, but the levels are not as dangerously low as those observed in altitude-simulated stress tests, avoiding the risk of such extremely low hypoxaemia.

Each year, especially over the past 10 years, Mt Everest has been overcrowded with climbers and up until the summer of 2019, about 10 000 ascents have been made since Edmund Hillary and Tenzing Norgay conquered the summit in 1953. The overall risk of death when climbing Mt Everest has declined over time, from an average of 1·8% in the previous century to 0·8% in the current one. This decline is most likely to be due to the massive popularisation of commercial expeditions, guided by Sherpas and expert mountaineers, which have significantly minimised the relative risk despite many climbers being technically underprepared. However, since the first expedition in 1921, more than 300 people have died and this number continues to rise, with 44% of deaths occurring during the past two decades. Nowadays, more than 95% of attempts to reach the summit from the highest camps are made with the aid of inhaling supplemental oxygen; without it, the risk of death is more than five times higher, according to *The Himalayan Database*.

When approaching the summit, or descending from it, fatalities are most frequently due to exhaustion, or ataxic and cognitive disorders that suggest the presence of cerebral oedema, as Paul Firth and colleagues reported in 2008. Falls, errors, faintness, self-abandonment, and hypothermia can be fatal and can be induced by heavy fatigue or brain oedema. High-altitude pulmonary hypertension, secondary to hypoxia, can induce interstitial or alveolar swelling and alter gas exchange, and subclinical pulmonary oedema is likely to be prevalent at extremely high altitude, worsening hypoxaemia and brain function. In fact, climbers most likely to succeed during these extreme-altitude attempts are those who have a lower pulmonary vasoconstrictor response to hypoxia. Those who are quicker to climb the final ascent of Mt Everest have a better physical and mental state, meaning they have a higher rate of survival; however, the congestion currently registered on the main routes causes hundreds of climbers to get stuck in long lines on the highest ridges, being forced to remain lashed by harsh weather conditions and under the deleterious effects of environmental hypoxia, especially when oxygen tanks are extinguished. Even resting at 8000 m, 2 L/min of supplemental oxygen does not restore the SaO_2 optimally, nor 4 L/min in deteriorated climbers, as recorded during *The British 40th Anniversary Everest Expedition* in 1993.

Many climbers have crowned Mt Everest repeatedly—nearly 100 of them more than five times each, with Sherpas being the majority. Himalayan natives have broken many records on this mountain; for example, one individual has made the ascent ten times without supplemental oxygen and is also the only person to date to have climbed it during the winter period without supplemental oxygen. This individual's climb during winter was a special physiological event because the small decline in barometric pressure at an altitude of 8848 m in winter compared with the rest of the year will have substantially decreased an already depleted maximal oxygen uptake (VO_2max). This climb took place on Dec 22, 1987, and we reported in 1997 that the VO_2max of

Climbers stuck in a long queue on the southeast upper ridge (8800 m) of Mt Everest in May, 2019

this elite Sherpa at sea level was high (4·37 L/min), which no doubt was a contributing factor to his historic climb. At the summit during winter, VO_2max reduces by almost 80%, despite help with extreme hyperventilation and respiratory alkalosis. With barometric pressure at the summit reaching as low as 32·4 kPa during mid-winter, a feat such as that achieved by this elite Sherpa would be very difficult because at these altitudes VO_2max is highly sensitive to minimal changes in barometric pressure; John West, a physiologist at the University of California, San Diego (CA, USA), also suggested that the reduction in VO_2max during mid-winter is one of the reasons an ascension at this particular time has not yet been achieved without supplementary oxygen. Even so, the highest point on Earth seems still just within the limits of human physiological tolerance.

Since the late 19th century, high-altitude medicine has made valuable contributions to scientific research and provides an opportunity to increase knowledge of respiratory pathophysiology and of critical care patients with hypoxaemia, helping to understand the subtle biological mechanisms involved and shedding light on potential future treatments. Specific genes have been implicated in the regulation of hypoxia, which could be important in finding out the basis of high-altitude adaptations seen in Tibetan ethnic groups. Phenotypic characteristics seen in these groups, such as minimal pulmonary hypertension, optimal ventilation or perfusion match, enhanced tissue oxygen delivery, and metabolic efficiency of cells, can be attributed to their highly differentiated genetic profile and to epigenetic modulation. Nevertheless, despite some individuals having physiological defences against the harmful effects of hypoxia, both Sherpas and foreign climbers have lost their lives near the summit of Mt Everest when they get trapped on the usual routes, which have collapsed due to overcrowding. The risk of death is a high price to pay to climb the highest mountain on Earth.

Photograph taken with a polarising filter and reproduced with permission from Josep A Pujante

Empty oxygen bottles scattered along the South Col (7906 m) of Mt Everest, with the troposphere visible after sunrise

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We declare no competing interests.

For more on **elite Sherpas** see *Med Sci Sports Exer* 1997; **29:** 937–42

For more on **human physiology at extreme altitudes** see *Science* 1984; **223:** 784–88

For an **overview of high-altitude medicine** see **Comment** *Lancet Respir Med* 2015; **3:** 12–13

For more on the **adaptations of Sherpas** see *Proc Natl Acad Sci USA* 2017; **114:** 6382–87

When is the right time to discuss ECMO?

The rise of extracorporeal membrane oxygenation (ECMO) over the past decade as a support modality provides clinicians with a powerful new therapy to care for patients with advanced cardiopulmonary disease. The growing use of this technology has required careful attention to the ethical challenges that accompany its use, including the allocation of scarce resources, determining when a trial of ECMO support is unsuccessful, managing conscious patients on ECMO without a viable pathway to recovery or destination therapy, and determining the role of ethics consult services in addressing the concerns of patients and care providers. Even as these challenges are presented and examined in the academic literature, comparatively little attention has been paid to the way clinicians communicate about ECMO. How should we broach the possibility of ECMO with patients and families in the first place?

One method, which we have encountered at our hospital, is for intensivists to introduce ECMO to families just before consulting the ECMO service or, in some cases, to defer initial discussion to the consulting ECMO service. As a patient's respiratory function declines or circulatory shock worsens, the intensivist might choose to delay discussion of ECMO until after exhausting conventional measures. Introducing ECMO early in a patient's course might place an unnecessary emotional and cognitive burden on families when alternative, less invasive therapies might suffice. Moreover, even when ECMO is the only potentially life-saving therapy remaining for a patient, the patient might be ineligible for support depending on institutional criteria. Extracorporeal support, in this respect, is akin to surgery; after evaluation, the procedural team regularly declines to initiate ECMO, regardless of the patients' or surrogates' expressed preferences, because the attendant risks of this technology

Dr Barry Slaven/Science Photo Library

Published **Online**
August 12, 2019
http://dx.doi.org/10.1016/S2213-2600(19)30278-4

For more on the **rise of ECMO** see https://www.elso.org/Registry/Statistics.aspx